Trauma and Psychosis: synthesising evidence, network modelling and expanding into an interventionist-causal paradigm to investigate mediating mechanisms

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By

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Declaration

The work in this thesis has not been submitted for any other degree or professional qualification. The thesis is the result of the student's own independent work.

Signed: 

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Abstract

The link between childhood trauma and the development of psychosis in adulthood is already well established, but factors which explain this relationship are currently less well understood. This thesis firstly aims to review the current state of evidence, then contribute novel empirical findings to help expand the understanding of how early traumatic experience leads to the development of psychosis. Information about causal factors is essential to the future development of effective therapeutic interventions for psychosis.

Firstly an extensive systematic review of studies which examines potential mediating mechanisms between trauma and psychosis is undertaken. Data from 37 studies were used to analyse 232 mediation models, taking into account the magnitude and significance of effects, along with study quality. Judgements are offered on the strongest areas of evidence, and implications for future research are discussed.

The first empirical study uses network analysis to generate a data-driven model of trauma, sub-clinical psychotic experiences and other relevant factors using data gathered from an online survey in a general population sample. Exploratory analyses were undertaken to derive a hypothetical model, which was then analysed statistically using structural equation modelling. The model hypothesis was pre-registered then prospectively tested in a second sample of data. Results and implications are discussed in the context of psychological models of psychosis.

The second empirical study uses an interventionist-causal paradigm to conduct a randomised controlled trial in a clinical psychosis population with experience of paranoia. An emotion regulation skills intervention was tested against an active control condition, with participants providing pre- and post- data, along with experience sampling data collected using mobile phones for analysis of individual and group change. Although limited by small sample size, findings are discussed in terms of acceptability, feasibility and implications for research and practice.

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Chapter 1 – Introduction to the thesis

1.1 Overview of chapter

This chapter introduces and defines the key terms ‘psychosis’, ‘childhood trauma’ and ‘emotion regulation’ which are used throughout this thesis. A broad overview of the evidence linking trauma and psychosis is discussed, followed by a description of relevant theories and models of trauma, psychosis and hypothesised mediating mechanisms. A rationale is provided for focusing on emotion regulation as a potentially important causal factor. Gaps in the current evidence are identified, some of which will be addressed by the empirical studies within this thesis.

Recurring problems with methodological practice and the reporting of evidence in psychological research are highlighted, and more contemporary research paradigms which seek to overcome these issues are described. This is followed by a summary of the overall aims of this thesis and an overview of the chapters.

1.2 Psychosis

Psychosis is a collective term used to refer to a set of symptoms present in a number of complex and severe psychiatric conditions. Under the umbrella term, more specific diagnoses including schizophrenia, schizoaffective disorder, delusional disorder, schizophreniform disorder, brief psychotic disorder, substance induced psychosis and unspecified schizophrenia spectrum disorder or other psychotic disorder are included (Diagnostic and Statistical Manual of Mental Disorders DSM 5, American Psychological Association, 2013). Affective psychosis (bipolar disorder) is distinct from the above, non-affective psychosis diagnoses. This thesis focuses on non-affective psychosis, hereafter ‘psychosis’.

Throughout the literature descriptions and definitions vary, however there is widespread acknowledgement that psychosis causes severe impairment in functioning across a number of areas including interpersonal relationships, family and work (Mueser & Marcello Duva, 2011).

Schizophrenia is classified as a low-prevalence disorder globally (Baxter et al., 2013), however it does affect approximately 1% of the worldwide population (McGrath et al., 2004) and large scale systematic reviews have noted prevalence rates of 3.89 to 4.60 per 1000 people (Moreno-Kustner, Martin & Pastor, 2018; Saha et al., 2008). Estimates were however found to be confounded by study quality, with lower estimates consistently found in better quality studies (Moreno-Kustner, Martin & Pastor, 2018). Regardless of this 'low-prevalence' label, the social and healthcare costs are high with significant levels of individuals with a diagnosis psychosis being unemployed (Marwaha et al., 2007). The early onset and long duration of the illness, along with the fluctuating nature of symptoms which can often lead to periods of hospitalisation, means that a proportion of patients will require healthcare input for most of their life (Hafner & an der Heiden 2003 & 2008).

Onset is typically in late adolescence or early adulthood, and is relatively balanced across genders, however males typically have earlier onset and poorer treatment outcomes, with greater symptom severity (Burton, 2012; Mueser & Marcello Duva, 2011 Emsley, Chiliza & Schoeman, 2008; Canuso et al., 2000; Murray & van Os, 1998). A lack of insight, which may lead to poorer engagement with treatment, was found to predict poorer outcomes, as was a longer duration of untreated illness, higher negative symptoms at first presentation, and higher premorbid difficulties (Diaz-Caneja et al., 2015; Lysaker, Lancaster, Davis & Clements, 2003, Perkins, 2005).

Diagnostic criteria for the various specific psychotic conditions vary, but broad categories of positive, negative, disorganised and cognitive symptoms are applicable across the spectrum. These are described further below. Researchers argue that these symptoms occur across a continuum from very mild everyday occurrences, to the more severe clinical presentations (Bentall, 2003; Van Os, Hanssen, Bijl & Rivelli, 2000; Van Os et al., 2009; Esterberg & Compton, 2009; Shevlin et al., 2016). Although patients must fulfil a number of criteria to attract a clinical diagnosis of a psychotic illness, research tends towards a more nuanced approach, where subclinical symptoms or psychotic-like experiences can be widely studied in

the general population to help inform clinical theory and research (Krabbendem, Myin-Germeys, Bak & van Os, 2005).

1.2.1 Positive Symptoms

Positive psychotic symptoms are additive phenomena, which alter an individuals' experience of reality, and which would preferably be absent (Bentall, 2003; Crow, 1980). These are often most prominent in early stage of the illness and may be triggered by heightened levels of stress and distress (Burton, 2012). This category of symptoms includes hallucinations and delusions, one or both of which are essential in order for a diagnosis of psychosis to be made according to both DSM 5 and the International Classification of Diseases – Tenth revision (ICD-10; World Health Organization, 1992) criteria.

1.2.1.2 Hallucinations

Hallucinations are perceptions which occur without the presence of corresponding environmental stimuli and may or may not involve a level of awareness of their unreal nature (Arciniegas, 2015). The most common form of hallucination in psychosis is auditory, where the hearer is aware of voices which may be either recognisable or unknown to them. Voices may be singular, multiple, conversing, or manifest as a running commentary; and they may give instructions or be critical of the hearer (Arciniegas, 2015; McCaffrey, Lynch & Westerveldt, 2011; Morrison, 2001; Mueser, Belack & Brady, 1990). Visual hallucinations are less common than auditory, but research has recently recommended that future studies consider the possibility of multi-modal hallucinatory experiences (i.e. those which occur across more than one format, for example seeing an image which also speaks or 'touches' them) (Dudley et al, 2018).Tactile (touch sensations); gustatory (taste) and olfactory (smell) hallucinations are less common in psychosis (McCaffrey, Lynch & Westerveldt, 2011), however when taken together, hallucinations which combine two or more of these modalities tend to be more realistic, and therefore more challenging to expose as non-real, which has implications for insight and conviction (Dudley et al., 2019).

1.2.1.3 Delusions

Delusions are fixed false beliefs about the world which are not amenable to change regardless of exposure to contradictory evidence, and are indicative of an individual's impaired reality testing (Arciniegas, 2015). Delusional beliefs may be situated in a real world context, in which an individual holds firm beliefs or fears that others intend to cause them harm (paranoid or persecutory beliefs); or beliefs may be more 'bizarre' and situated outside of the realm of reasonable possibility (Langdon & Coltheart, 2000; Cermolacce, Sass & Parnas, 2010; Arciniegas, 2015). Persecutory beliefs (being the actual or intended victim of harm) and referential beliefs (beliefs that the words, gestures and actions of other people or events are being directed at the individual) are the most commonly observed in psychosis, however other types of delusions do occur (Startup, Freeman & Garety, 2007; Vorontsova, Garety & Freeman, 2013). These include thought-based delusions, including thought broadcasting (a belief that others can hear their thoughts); thought insertion (believing that thoughts are being introduced by an external entity), thought extraction (where thoughts are being removed by an external entity), thought control (where thoughts are being externally controlled) and mind reading (where others are aware of the person's thoughts without active communication). Religious content is also common in delusions. Individuals may believe they have been given a special mission from God, or have special powers, or may believe themselves to be embodiments of God or the devil, or good or evil. Other types of delusional content include beliefs that one is infested with insects, one is dead (Cotard's delusion), loved ones have been replaced by impostors (Capgras syndrome), or that one is particularly rich, famous or important (grandiose delusions). Generally these are less common but do occur in psychosis, and particularly in delusional disorder (Paolini, Moretti & Compton, 2016; Arciniegas, 2015; Langdon & Coltheart, 2000).

1.2.2 Negative Symptoms

Negative psychotic symptoms reflect a reduction in, or an absence of, what would otherwise be considered desirable behaviour (Mueser & Marcello Duva, 2011; Crow, 1980). This

category of symptoms includes reduced emotional expression (affect flattening), a lack of motivation or drive (avolition), reduced ability to experience pleasure (anhedonia); social withdrawal, and poverty of thought and speech (Burton, 2012; Arciniegas, 2015; Lyne et al., 2018). A number of these symptoms may initially be misattributed to depression, and in the long term, negative symptoms have been found to be more persistent than positive symptoms (Lyne et al., 2018).

1.2.3 Disorganised symptoms

Also referred to as 'formal thought disorder', disorganised symptoms include disorganised patterns of thought, tangential thought or speech, perseverative thinking, clear illogical thought, thought blocking (abruptly switching between topics) and neologisms (the creation often incomprehensible new terms which may include truncating words, or splicing them together) *(*Arciniegas, 2015; Mueser & Marcello Duva, 2011; Minzenberg et al., 2003). Further symptoms include disorganised, chaotic or poorly planned behaviour (Frith, 1996; Andreasan, 1995) and communication which is difficult to comprehend due to disorganised syntax (sometimes termed 'word salad') in combination with the disorganised thought processes above (Marvel, 2006).

1.3 Childhood trauma

The definition of what constitutes 'childhood' varies throughout the literature, as do definitions of 'trauma' and 'adversity'. This thesis will consider 'childhood' to include events which occur before the age of 18. 'Trauma' will be considered as highly negative events which occur at a single time point, or repeatedly, which have a negative effect on the individual experiencing them. This thesis will focus on trauma which is interpersonal in nature, described in further detail below. It will not include events which are often classified as 'adversities'. These include natural disasters, accidents or illnesses which occur without a 'perpetrator' (Gibson et al., 2016). The subjective interpretation of these events may be qualitatively differently to events which are inflicted with harmful intent (van Nierop et al, 2014; Burgermeister, 2007).

The DSM-5 definition of trauma requires the experience of “actual or threatened death, serious injury, or sexual violence” (American Psychiatric Association, 2013), thus setting trauma apart from other life stressors such as marriage breakdown or job loss (Pai, Suris & North, 2017). These events comprise a category of the most severe trauma exposure, however there are a number of further abusive and neglectful experiences which may provoke similar negative responses in those who are exposed to them (Gibson et al., 2016).

Emotional or psychological abuse includes shaming, embarrassing and insulting; emotional neglect involves rejection and being emotionally distant. Bullying is sometimes included within emotional abuse, and involves similar actions such as name calling, harassing and intimidation, but some actions can also overlap with physical abuse. Physical trauma includes acts of physical violence such as slapping, smacking, punching or kicking; and physical neglect involves a lack of provision for basic needs including food, clothing, and warmth, and may also include ignoring or locking away in isolation. Sexual abuse includes sexual talk, touching and intercourse (Gibson et al., 2016; Varese et al., 2012; Bernstein, Fink, Handelsman & Foote, 1998; Gray et al., 2004; Higgins & McCabe, 2001; Burgermeister, 2007).

Parental separation and institutional care are somewhat disputed in the literature. Some classify this as a traumatic event (Bentall, Wickham, Shevlin & Varese, 2012), others classify it as an adversity due to the absence of a direct perpetrator (Gibson et al, 2016). The review chapter in this thesis includes parental separation and institutional care, along with emotional, psychological, physical and sexual trauma as described above. Experience of these life events in childhood are hypothesised to have negative social, developmental, cognitive and emotional consequences for the individuals who are exposed to them (Williams et al., 2018; Velikonja et al., 2015; De Sousa et al., 2014; Ackner et al, 2013). The current evidence for this is systematically reviewed in **Chapter 2**

1.4 Childhood trauma and psychosis

The experience of early life trauma has been reliably linked to the development of psychosis in later life (Kelleher et al., 2013; Varese et al., 2012; Schafer & Fisher, 2011; Larkin & Read, 2008; Read, van Os, Morrison & Ross, 2005). The severity of psychotic symptoms in individuals diagnosed with a psychotic disorder have also been correlated with the frequency and severity of reported early trauma (Schenkel et al., 2005). There are a number of theoretical models hypothesising how trauma leads to psychosis. These include a number of biological, psychological and social factors which may act as mediators of this relationship. These are described below.

Models suggest hypersensitisation to stress (Read et al, 2001) and impaired downregulation in response to stressors (Barker et al., 2015) can result from chronic exposure to acute or ongoing adversity, and this may leave an individual vulnerable to developing psychosis (Myin-Germeys et al., 2001). The model by Barker and colleagues also suggests that insecure attachment is a vital mediating factor, having both emotional and interpersonal consequences. A more contemporary integrative model suggests genetic factors, environment and trauma interact and combine to increase psychosis liability (Misiak et al., 2017). The model branches into biological and psychological pathways which both progress towards psychosis. The biological path reflects the mediators identified by Read's traumagenic model (2001), and the hypothesised psychological mechanisms also include insecure attachment, along with dysfunctional cognitive schemas, affect dysregulation and dissociation.

These psychological mechanisms are mirrored in other established models of positive symptom development. Bentall and Fernyhough (2008) suggest that victimisation and insecure attachment lead to negative self-esteem and a tendency to externally attribute blame for negative events. This has a potentially protective function for the individual, but can have negative consequences by creating hypervigilance to threat, and eventually leading to persecutory beliefs. The model suggests that the cognitive 'jumping to conclusions' bias which is common in psychosis (Moritz et al., 2005; Dudley et al., 2016; Lincoln et al., 2017) further

augments these persecutory beliefs. Although Garety's cognitive model (Garety, 2001) does not directly hypothesise about the effect of childhood trauma, it does highlight the importance of environmental stressors and the responses they provoke. The model brings together emotional and cognitive factors such as belief inflexibility, beliefs about diagnosis, and social factors including isolation, as other potential mediating mechanisms. These factors may lead to the misinterpretation of anomalous experiences as threatening, which may then contribute to symptom development and maintenance (Freeman et al., 2002; Garety, 2001). Morrsion (2001) instead describes positive symptoms as resulting from culturally unacceptable interpretations of intrusive experiences. The model implicates self and social awareness in dysfunctional patterns of interpretation; and emotional and cognitive factors which maintain the cycle of intrusions through dysfunctional coping mechanisms such as selective attention and avoidance.

It is clear from these models that a number of factors are believed to be highly relevant to the trauma-psychosis pathway. Gibson, Alloy and Ellman (2016) describe the concepts of 'multifinality' and 'equifiniality'. Multifinality is where the same predictor can cause a variety of outcomes – in this case trauma may lead to psychosis, but also to eating disorders, substance use disorders, post-traumatic stress and personality disorders. Equifiniality on the other hand is where multiple predictors lead to a single diagnostic outcome – in this case various environmental factors, developmental mechanisms and biological vulnerabilities will be implicated in the final diagnosis.

Other authors describe the inherent heterogeneity of constructs such as trauma and psychosis. Bentall et al (2014) propose specific pathways from early trauma to specific psychotic symptoms, explaining that the trauma-psychosis pathway is highly complex and likely to be mediated by multiple factors. Specific pathways through childhood sexual abuse to hallucinations are described, with proposed mediation by dissociation as a result of trauma and distress, and by cognitive deficits in discriminating between internally and externally

generated stimuli. Evidence for a pathway from neglect or parental separation to persecutory delusions, via insecure attachment, cognitive biases and threat anticipation is also described.

In line with this specificity, some researchers advocate a 'complaint orientated' approach (Bentall, 2006) in which patients highlight their most problematic or distressing symptoms so these can be specifically targeted for treatment. This approach is positioned as an alternative to more traditional psychiatric practices which tend towards diagnostic labels to characterise an individual's symptom profile. Bentall (2006) suggests that research has suffered inconsistencies due to too rigid adherence to this Kraepelin framework which fails to appreciate the idiosyncrasies of individual patients. As a result, studies may have missed potentially salient findings because their clinical groups may have been too heterogeneous (Longden & Read, 2016; Read 2013). Therefore, it is recommended that potential mechanisms are studied in isolation to better understand their individual contributions, whilst maintaining an awareness that the overarching aetiological framework is likely to be highly complex and interconnected (Gibson, Alloy & Ellman, 2016).

More recently, researchers have begun to investigate emotion regulation as a potentially important mechanism (Chapman et al., 2020; Liu et al., 2020; Ludwig, Mehl, Krkovic & Lincoln, 2020; Lincoln, Hartmann, Kother & Moritz, 2015; O'Driscoll et al, 2014; Khoury & Lecomte, 2012; Livingstone, Harper & Gillanders, 2009). Emotions feature in a number of the models described above, but until recently little research attention has been devoted solely to emotion dysregulation in psychosis (see Chapter 2). Recent studies have found early trauma to be implicit in the development of emotion regulation difficulties in individuals who go on to develop psychosis (Wallace & Docherty, 2020). Further studies have found evidence to suggest that emotion dysregulation mediates post-traumatic symptoms and psychosis, and maintains depression, positive symptoms, and symptom-related distress (Liu et al., 2020b; Lincoln et al., 2017). Greater levels of reported trauma in clinical and high-risk samples are associated with elevated dysfunctional emotion regulation, as well as higher negative, and lower positive affect (Oorschot et al., 2013; Liu, Subramaniam, Chong & Mahendran, 2019; Pries et al., 2020). The

next section defines emotion regulation, and the following section discusses its potential relevance to trauma and psychosis research.

1.5 Emotion regulation

'Emotion regulation' refers to the set of processes by which emotional arousal can be modified and controlled to maintain balance and preserve goal directed functioning (Gross, 2001; Beauchaine, 2015). The process model (Gross, 1998) and the extended process model (Gross 2015) describe the basis for emotional processing and regulatory action. In order to modulate affect and continue progressing towards a goal, individuals may take regulatory action based on the environmental context of their perception of it. These models describe how this may occur (Gross, 2015; Gross, Sheppes & Urry, 2011)..

The five stages of the original process model are focused on cognitive emotion regulation. This includes 'situation selection' and 'situation modification' where an individual modulates exposure to potentially arousing scenarios which may provoke an emotional response. Associated with this is 'attention deployment', whereby the individual can influence the aspects of the situation they attend to. This often becomes relevant when the situation can no longer be actively altered, and can include dysfunctional strategies such as worry and rumination (O'Driscoll et al., 2014). Mindfulness is considered to be a positive attentional deployment strategy where individuals focus on elements of the present moment with conscious acceptance of affective and cognitive states, without active engagement or resistance (O'Driscoll et al., 2014; Chambers, Gullone & Allen, 2009). 'Cognitive change' is the next sequential stage, in which the emotional significance of the situation can be modified by using cognitive reappraisal (Gross & Thompson, 2007). These first four stages enable the use of antecedent-focused regulatory strategies based on the situation and the cognitive assessment of the stimulus. This occurs prior to the emotional reaction. The final stage is described as 'response modulation' which can include a variety of different regulatory actions including suppression, avoidance and distraction, which are classified as negative or inhibitory emotion regulation strategies (Gross, 1998; O'Driscoll et al., 2014); or acceptance which is considered

to be a more adaptive response (Hayes et al., 2006). However, all of these are response-focused strategies, following the activation of the emotional response, by which time it is more challenging to re-establish a state of equilibrium (Gross, 1998).

Building on the original model, Gross (2015) added three further stages which are important to the processing of emotional arousal. Firstly 'identification' which involves the recognition of the emotional state and the cognitive interpretation of whether there is a need to regulate. Secondly 'selection' where a regulatory strategy which is believed to be appropriate is chosen; and finally 'implementation' where the strategy is put into action. A subsequent iteration of the model includes the ongoing monitoring of the success of the chosen strategy over time (Sheppes, Suri & Gross, 2015). This extended model may be of particular relevance to clinical patients with a diagnosis of psychosis as they have been identified as lacking in areas of emotion perception (Ruocco et al., 2014; Thaler et al., 2013; Kring & Elis, 2013), emotion identification or description (alexithymia) (Heshmati et al., 2010; Van't Wout, Aleman, Bermond & Khan, 2007; Maggini & Raballo, 2004) and emotional expression (Phillips & Seidman, 2008; Henry et al., 2007; Kring, 1999).

There is still debate in the research literature about what the term 'emotion regulation' encompasses (Kross 2015; Gross, Sheppes & Urry 2011). Some researchers believe the concept is too broad, and should be split into more distinct components, or reduced and used to only describe processes of emotional response (Kross, 2015; Koole & Veenstra, 2015). Others argue that this would be an oversimplification which risks limiting the understanding of emotion regulation as a process in its entirety (Phillips & Seidman, 2008; Gross, 2015). The processing of emotions may begin as early as the initial contact with the environmental stimulus. This is then followed by the cognitive processes which implicated in the interpretation of the emotional experience, its intensity and how the individual interacts with it. These stages may be concurrent or overlapping, or at the very least not distinct form one another (Gross, 2006; Phillips & Seidman, 2008; Gross, 2015). This makes emotion regulation a challenging concept to define and measure. The majority of research in psychosis has centred on the use

of suppression and reappraisal as the most common cognitive regulatory strategies. In this thesis both empirical studies will make use of the Cognitive Emotion Regulation Questionnaire (CERQ, Garnefski Kraaij & Spinhoven, 2001) which measures nine emotion regulation strategies which can be summed into 'positive' and 'negative' regulation strategy totals. This provides the flexibility to look at specific strategies along with more broad functional and dysfunctional strategy use. Details of the reliability and validity of the CERQ measure can be found in Chapter 3.

1.6 Trauma, emotion regulation and psychosis

Evidence suggests that emotion regulation difficulties in psychosis are linked with the development of positive, negative and disorganised symptoms, and are correlated with symptom severity (Berenbaum et al., 2006; Strauss et al., 2013). Higher emotion dysregulation has also been associated with poorer treatment outcomes in psychosis (Wallace & Docherty, 2020). A number of emotion-related difficulties in psychosis have been investigated. These include the more prevalent use of sub-optimal regulation strategies, particularly suppression (Gross & Levenson, 1997; Van der Meer, Van't Wout & Aleman, 2009); and a tendency to display blunted affect or reduced outward emotional expression (Kring, 1999; Henry 2007; Phillips & Siedman, 2008). Further studies highlight other problematic areas including difficulties identifying feelings and verbalising emotional information, particularly during times of high arousal (Van't Wout et al, 2007; Van der Meer, Van't Wout & Aleman, 2009; Gaweda & Krezolek, 2019); and the reduced self-reporting of positive emotions, along with a bias towards reporting increased negative emotional experience (Cohen & Minor, 2010; Philips & Siedman, 2008).

Exposure to early trauma may leave individuals more sensitive to the effects of stress in daily life (Read, 2005. Zubin & Spring, 1977) which may in turn lead to ongoing difficulties in managing and regulating emotions, and increased anxiety (Myin Germeys et al., 2003). Higher instances of negative emotions coupled with an inability to down-regulate emotions may form part of a cycle which leads to the development and maintenance of paranoid ideation (Lincoln

et al., 2009). Attempts to regulate negative emotions with cognitive reappraisal appears to be inhibited by both stress-sensitivity and cognitive biases, such as negative beliefs about others, or jumping to conclusions, both of which may reinforce beliefs that others intend to cause harm (Opoka et al., 2020; Ludwig, Werner & Lincoln, 2019; Lincoln, Hartmann, Köther, & Moritz, 2015a). Unsuccessful reappraisal can result in elevated negative affect and heightened stress, which still cannot be adequately reappraised and downregulated, resulting in a distressing cycle (Westermann et al.; 2012; Freeman et al., 2002; Gross & Levenson, 1997). Therefore it is suggested that in patients with paranoia, chronic levels of stress and negative affect, coupled with the inability to regulate these emotions, may enhance the severity of symptoms and associated distress (Strauss et al., 2013; Garety et al., 2001). Whilst negative affect and emotion regulation are factors in a number of theoretical models and hypotheses, there has been little research into emotion regulation as a potential causal mechanism (see Chapter 2). In cross-sectional research emotion regulation has been found to predict positive symptoms and in particular, paranoid thinking (Westermann et al., 2013; Westermann & Lincoln, 2011) but this has yet to be tested experimentally. Since both theoretical and correlational research indicate this may be an important factor, this thesis will aim to investigate its importance using an experimental paradigm (see Chapter 5). Further details are provided below about the need for experimental research into potential causal mechanisms.

1.7 Research into causal factors

As is clear from the evidence above, studies in the area of trauma and psychosis are abundant, but the quality of the evidence varies and as the majority of studies are observational, any causal claims based on their findings will be limited.

The Bradford Hill criteria for causality describe qualities which should be present in order to support causal claims based on research findings (Hill, 1965). Firstly the association must be strong, generally indicated by a large effect size (Hill, 1965; Boffetta, 2010), although Hill does state that a small effects does not rule out the possibility of an effect being causal. The association must also be consistent and specific, as evidenced by a body of research, in either

a specific population or in the case here, between specific types of adversity and symptoms. Temporality must be considered, where the hypothesised cause must precede the outcome, and when the cause is absent or removed, the effect should not occur – this is referred to as reversibility (Hill, 1965; Giordano & Lindström, 2016). However temporality and reversibility are challenging to consider when only cross-sectional evidence is available. Hill also describes a 'dose-response' whereby greater exposure to the cause will lead to greater severity of the outcome (Hill, 1965; Weiss, 1981). The cause must also be plausible, meaning it must make sense as a potential cause of the outcome, with all other potentially confounding variables being controlled (Shimonovich, Pearce, Thomson, Keyes, & Katikireddi, 2020). Methodology is also important. In order to fulfil the criteria around coherence and experimental evidence, researchers must consider where the existing evidence has come from (Höfler, 2005). Cohesion between laboratory findings and larger population-based studies can strengthen causal claims, as can evidence from specifically designed experimental work, however this is not available in all fields (Shimonovich, Pearce, Thomson, Keyes, & Katikireddi, 2020)..

Currently the evidence suggesting that trauma causes psychosis is strong, however the evidence of other contributory causal factors is less conclusive. The systematic review chapter (Chapter 2) provides information about the magnitude and significance of the mediation effects in the current literature. These are discussed alongside an assessment of study quality, and details of the causal inferences which can be made at present. It also highlights a number of limitations and weaknesses in the current evidence base, including a lack of transparency and poor reporting practices. The repercussions of these problems, and the risks they pose to evidence integrity are described below.

1.8 Replicability crisis and the Open Science Movement

For a number of years flaws in the methods and reporting of scientific research have been identified and highlighted as areas in need of improvement. A variety of research practices have been criticised leading to widespread calls for changes to be made to enhance the credibility and utility of evidence (Baker, 2016; Nosek et al., 2015; Bare, 2014; Ioannidis,

2005). A decade ago, attempts were made to begin replicating findings from a number of psychological studies published in respected journals (Bohannon, 2015; Open Science Collaboration, 2015). Only 36% of attempts found significant effects, as compared with 97% in the original studies, and effect sizes were found to be around half the size of those originally reported.

1.8.1 Reasons for lack of replication

A number of potential reasons for this lack of replicability have been suggested. Meta-analyses have noted that psychological research suffers widely from low power, small effect sizes and small sample sizes (Stanley, Carter & Doucouliagos, 2018; Munafo et al., 2017; Button et al., 2013). Some studies stop recruitment ahead of target sample sizes if statistical tests show significance, whilst others may instead use selective reporting, where certain data or variables are ignored in order to manipulate findings. This increases the likelihood that findings will not be replicated in subsequent samples (John, Lowenstien & Prelec, 2012). Data dredging or 'p-hacking' is linked to this, where researchers conduct multiple analyses to find significant results and report these without acknowledgement of the multiple non-significant results which may have been discarded along the way (Head et al., 2015; John, Lowenstein & Prelec, 2012). Similarly, outliers may be manipulated, either by removal or retention depending on the desired results, but in studies where methods and intentions are not specified ahead of analyses being undertaken, this sort of flexibility may go unnoticed (Simmons, Nelson & Simonsohn, 2011). In fact, many studies combine these factors with post-hoc changes which remain undisclosed in their manuscripts. This enables the presentation of exploratory analyses as if they were confirmatory, and often means findings are represented as having credibility far beyond what is truly reasonable (Allen & Mehler, 2018). This has also been termed as HARK-ing, or 'hypothesising after results are known' (Kerr, 1998) where researchers 'specify' their hypotheses and report their studies as if these were the original aims.

Concato & Horwitz (2019) claim that meta analyses should be treated with caution when the authors have a particular vested interest in the topic of the review (e.g. psychotherapies) or where they stand to gain from evidence which demonstrates the efficacy or effectiveness of treatments. Kahneman (2014) states that research is less well replicated when study protocols are registered in advance. Publication bias is a further threat to replicability and reliability. Historically it has been challenging to have null findings published, particularly in small studies, leading to an elevated risk of false positive results (Simmons, Nelson & Simonsohn, 2011). This may be partly based on an incorrect assumption that a significant result indicates that the study had sufficient power to detect effects. It should be clear from the above that an alternative is needed to improve the credibility and usefulness of future research.

1.8.2 Open Science as an alternative methodological framework

The Open Science Movement offers an alternative framework for the conduct and reporting of scientific research, designed to improve trust, transparency and verifiability of findings. Open Science seeks to increase the accessibility of scientific research, quickly and efficiently without the delays of the traditional peer review and publication process, whilst maintaining open peer review principles for others to comment on, and form conclusions about the work (Bare, 2014). This should result in better collaboration, more streamlined data sharing and more citations between works, enabling accelerated progress towards answers for complex research questions (Munafo et al., 2017; Priem, 2013; Nosek, 2012). Open science also places value upon the replication of prior findings, and advocates the use of open source software which is accessible by all, as well as the pre-registration of aims and intentions before studies commence (Nosek, Ebersole, Haven & Mellor, 2018; Munafo, et al., 2017; Open Science Collaboration, 2015; Chambers, 2013).

However, Open Science is not without its own challenges as highlighted by Allen & Mehler, (2018). These include the inflexibility which follows pre-registration, which is not a challenge in itself, however it does reduce the scope for revising and improving methods after the original statement of a planned protocol. The authors advocate openness about perceived errors and

subsequent changes, with an emphasis on transparency, which will permit the reader to make their own decisions about the motives of the research team. This sort of research does takes more time, requires larger samples and may have greater financial implications which may at first seem prohibitive, particularly for early career researchers. There are also concerns over the misuse and misinterpretation of data and findings in a realm where science is more widely available to a lay public (Bare, 2014), however some of this risk should be negated by adhering to good reporting standards. In spite of these challenges, the advantages of adopting an open science approach outweigh the risks and will ultimately lead to better quality and more replicable research.

1.8.3 Pre-registration

One of the most crucial elements of open science is the emphasis placed on pre-registration in order to ensure studies are rigorously planned and transparently reported. Studies should be registered in the public domain before the research takes place means researchers must thoroughly plan their procedures and clearly record their intentions ahead of undertaking their work (Yamada, 2018). Pre-specifying the method and analysis plan leaves researchers little scope for deviation, regardless of what their study finds. Some journals have recently begun to offer 'results-blind peer review' where a commitment is made to publish the research based on the rigour of the pre-registered protocol, rather than the study findings (Findley et al., 2016; Greve, Broder & Erdfelder, 2013). A recent study found that 61% of studies published in this way reported null findings, in contrast to traditional publishing where only 5-20% of findings are null (Allan & Melher, 2018).

Pre-registration has a number of benefits including improving transparency and helping to protect against bias (Munafo et al., 2017); improving wider awareness of research and encouraging data-sharing (Wicherts et al., 2006); avoiding duplication of research topics so funds can be diverted into novel areas, whilst promoting collaboration, enhancing the reputability of research and researchers, inspiring confidence in findings and making research more accessible to wider audiences via open science infrastructure (Allan & Melher, 2018).

For all of these reasons, all empirical work in this thesis has been pre-registered in the public domain. Any changes to the protocol are described in detail along with thorough rationale within each chapter. The aim of this was to be clear about what the study hypotheses were, and which analyses were exploratory and confirmatory. Efforts were made to adhere to the original research plan wherever possible, despite the challenges encountered. Details of registrations and changes can be found in each methodology section, and the registered protocols are available in the appendices (see appendix 1, 2 and 3).

1.9 Interventionist causal research

Whilst open science approaches aim to improve transparency and integrity in research practice and reporting, studies in psychology still suffer from a number of challenges in terms of methodological limitations. It is difficult to infer causality from observational research, and from traditional experimental studies which use analogues or non-clinical samples. Even RCT research can be limited by the testing of complex multi-faceted interventions which cannot always point to the exact cause of any recorded improvements. Observational studies tend to be popular as they are frequently more feasible, accessible and cost-effective. More complex experimental designs can be costly and time consuming. Both types do have value, but a combination of both are required to complete the evidential picture.

The field of trauma research is also rightly constrained by ethical boundaries, whereby trauma or adversity cannot be readily induced or manipulated (Brand, Rossell, Bendall & Thomas, 2017). Some studies have attempted to induce trauma using films (Holmes, Brewin & Hennessey, 2004; Holmes & Bourne, 2008) but arguably this passive viewing of traumatic content is not truly akin to in vivo trauma exposure (James et al, 2016) and therefore is not always seen as an adequate representation of 'trauma' which can be experimentally investigated.

A paradigm shift is essential in order to gather evidence of causal factors. As a result studies have begun to use 'interventionist-causal' designs in the study of psychosis (Freeman, 2011; Garety & Freeman, 2013; Farrely et al., 2016; Reininghaus et al., 2016; Garety et al., 2017;

Gollwitzer, Wilczynska & Jaya, 2018). This involves the identification and experimental manipulation of a hypothesised 'causal factor' in a controlled manner, to investigate whether changing the proposed mechanism causes changes in the outcome of interest (Brand, Rossell, Bendall & Thomas, 2017).

1.9.1 The Interventionist Model

Kendler & Campbell (2009) describe an 'Interventionist Model' (IM) which they claim can help researchers pursue causal evidence beyond the more typical association-based research. They suggest the IM provides an enhanced framework for understanding conceptual or theoretical models by moving away from busy multi-factor models, and instead focusing only on simple, concrete demonstrable causal connections between variables.

Essentially the IM uses an intervention to target and change a hypothesised causal factor, in order to observe any subsequent changes in the outcome. To demonstrate causality, three conditions must be met: the intervention must be the only systematic attempt to change the hypothesised cause; the intervention must not affect the outcome in any other way which is not directly via the hypothesised causal variable, and the intervention itself must not be influenced by any other factors which may also influence the outcome without routing through the hypothesised cause (Kendler & Campbell, 2009; Woodward & Hitchcock, 2003, Pearl, 2000). By randomising individuals to either the active intervention or a placebo control in a highly controlled manner, it should be possible to uncover whether the hypothesised cause truly does fulfil a causal role (Kendler & Campbell, 2009; Pearl, 2000). Randomisation should balance any observed and unobserved variables which might otherwise exert a confounding effect on either of the groups (Kendler & Campbell, 2009). For these reasons, Brand and colleagues (2017) assert that an interventionist-causal design is perfectly placed to partition out the complex mechanisms at play in psychological pathways, which may help identify more specific treatment targets, leading to improved outcomes in the long term. Further information about interventionist causal research can be found in Chapter 4.

1.10 Aims of the thesis

This thesis aims to investigate factors which mediate the relationship between childhood trauma and the positive symptoms of psychosis. To do so, it firstly reviews and appraises the state of current knowledge about mediation mechanisms involved in this relationship. Chapter two presents the methodology, results and discussion of a systematic review of the literature in this area.

Secondly an empirical research study in a general population sample collected data to develop and subsequently test a model of trauma, subclinical psychotic experiences and potential mediating factors. This study used a robust two-stage method of data collection and analysis in which an exploratory phase was first used to develop and iteratively improve a hypothetical model, followed by a confirmatory phase where the model was tested in a new data sample. Chapter three presents the methodological protocol for the study, and Chapter five presents the exploratory and confirmatory results.

Thirdly, a small randomised controlled trial was undertaken in a population of individuals with a clinical diagnosis of non-affective psychosis, emotion dysregulation and paranoia. An interventionist-causal design was adopted, and participants were randomised to either an emotion regulation skills intervention, or an active control condition. Primarily the study aimed to identify whether the intervention helped to improve paranoid symptoms, and whether emotion regulation ability was improved via the intervention group. The experience sampling method, an ecologically valid repeated-measures method of data collection, was used. This was delivered using a mobile phone interface which participants used to complete questionnaires in their own environment several times per day. Chapter four describes the methodological protocol for the trial, and chapter six presents the results and evaluation.

Chapter seven brings together the findings from the empirical studies and contextualises the novel findings in terms of existing knowledge. The implications for future research and clinical practice are discussed.

Chapter 2 –A systematic review of mediators of the pathway between childhood trauma and positive psychotic symptoms

2.1 Overview of chapter

This chapter presents the rationale, method and results of a systematic review of the current research into mechanisms which may mediate the pathway from childhood trauma to psychosis.

The chapter will first describe the rationale for reviewing this area, followed by a description of mediation research and its importance in terms of making causal claims about the mechanisms underlying psychological processes. This is followed by a brief discussion of effect size and its importance in the interpretation of mediation evidence. Previous reviews in this area have been unable to fully interpret mediation effect sizes and therefore this forms the key point of difference in this review. The specific aims of this review are stated, followed by a description of the systematic review process.

Results are presented in categories of similar mechanisms, and are contextualised using the magnitude and significance of the effects, along with information about the study quality. Key findings are highlighted, along with an appraisal of the strengths and weaknesses of current research evidence. This is followed by a discussion of the theoretical, clinical and research implications of the findings from this review. The limitations of the existing literature, and of this study, are discussed.

2.2 Research rationale

There is strong evidence from clinical studies indicating that patients who experience psychosis in adulthood have experienced higher rates of early life trauma than the general population (Ashcroft, Kingdon & Chadwick, 2012, Varese et al., 2012). Studies suggest that over 80% of individuals with a diagnosis of schizophrenia have experienced at least one traumatic event (Mueser et al., 1998) and that rates of victimisation are found to be ten times

higher in those with a severe mental health diagnosis as compared with the general population (Walsh et al., 2003).

Researchers have proposed the concept of a 'global cumulative adversity model' which suggests that more severe traumatic experiences can lead to poorer long term outcomes including more severe positive psychotic symptoms (Ackner et al., 2013; Longden & Read, 2016; Barker at al., 2015); increased antipsychotic resistance (Hassan & De Luca, 2015) and a negative impact on therapeutic alliance leading to poorer overall treatment outcomes (Barker et al, 2015). There is also evidence in support of a 'dose-response' relationship. This suggests that individuals who receive a greater 'dose' of trauma either through repeated traumatic events or ongoing abuse will have an increased chance of developing psychosis (Read et al., 2005; Janssen et al., 2005; Larkin & Read, 2008; Longden & Read, 2016, Hardy et al., 2016). Demonstrating a dose response relationship is one of the Bradford Hill criteria for causality (Hill, 1965). See further details of causal criteria in Chapter 1.

To date, the trauma-psychosis pathway is supported by a great deal of evidence, however the factors which influence this are less well understood. A more thorough understanding of mechanisms which are important to this pathway may help optimise treatment development. Although it is seldom possible to intervene at the point the trauma is occurring, it may be possible to develop interventions which will better equip individuals to cope with its effects. By establishing which factors cause the development of psychosis following trauma exposure it may be possible to target these at an earlier stage, and in a more effective manner, before individuals develop positive symptoms.

Two recent reviews have attempted to synthesise the existing evidence of mediation mechanisms on the trauma-psychosis pathway (Williams et al., 2018; Alameda et al., 2020), however neither has been able to offer a comprehensive appraisal of current knowledge. Williams et al. (2018) focus on the main finding from each paper, and discuss results based on significance values. Little attention is given to secondary or non-significant findings, and the statistical information provided is incomplete. They do identify issues with study conduct

and quality, but these appear to be largely confined to the limitations section rather than used to influence the interpretation of the accuracy of reported effects. Alameda et al. (2020) do take into account non-significant findings, and they make efforts to quantify the proportion mediated however, this can be challenging to interpret without firstly quantifying the overall effect, and without the computation of confidence bounds it is difficult to gain a sense of precision (see further below). An assessment of evidence quality is undertaken, however this information is pooled to describe the overall body of evidence as 'fair'. Beyond this, there is only one reference to study quality in the discussion of outcomes. Without taking into account the methodological rigour of the studies generating the evidence it is not possible to assess the validity of claims and the reliability of findings.

Although there are a number of theories about how the experience of early trauma can lead to psychosis (see Chapter 1), it remains unclear where the strongest evidence lies regarding the most promising potential mediators of the relationship. Only by comprehensively reviewing the current evidence in terms of effect size and quality, will it be possible to gain an understanding of which mechanisms are most important. Doing so will also serve to highlight areas where there is little evidence at present; and will help to highlight gaps in the literature which have yet to be investigated.

Firstly, in order to make sense of the evidence being reviewed, it is essential to understand what a mediator is, and what mediation research is attempting to show. A description of this is provided, followed by details of effect size measures and their importance to the understanding and comparison of mediation models. This should further highlight the limitations of the existing literature reviews and demonstrate how this review intends to enhance the current understanding of the evidence base.

2.3 Mediation research

Before reviewing the current research, it is important to understand what is meant by mediation, and what this type of research is investigating. This section presents a description of what a mediating variable is, and how mediation models are constructed and tested. There

are several methods of testing these models, each are described briefly below and further details can be found in appendix 4.

Mediation models are those which map the transmission of effects from predictor variables to outcome variables both directly, and through intervening variables known as mediators (Lachowicz et al., 2018, Kenny, 2015). Mediation modelling reveals patterns of association between variables which may form grounds for hypotheses of causal effects. Additional assumptions are required to be met before any causal claims can be made (Lachowicz et al., 2018; Cohen, Cohen, West & Aiken, 2013) and the Bradford Hill criteria for causality (Hill, 1965) should be considered. These include the strength, consistency, specificity, dose-response gradient, reversibility, temporal sequencing, plausibility and experimental support for the effect, and whether other potential causes can be ruled out (see Chapter 1 for more detail). Not all types of studies can make causal claims, however they may offer evidence which fulfils some of these criteria, and therefore can help to identify factors which may merit further investigation as potential causal factors.

The simplest mediation models contain three variables; the predictor variable, the outcome variable and the mediator (see figure 2.1). The connection between the predictor (X) and the outcome (Y) is termed the 'total effect', denoted as '*c*'. This is the representation of the model in its unmediated form. When the mediator (M) is added, the total effect is decomposed into the indirect pathway, denoted as '*ab*'; which represents the effect of X on Y as mediated by M, and the direct pathway, denoted as '*c*' ' which represents the residual effect between X and Y when M is entered into the model; essentially this is the effect left over when controlling for the mediator. The indirect path further decomposes into two regression coefficients, one between X and M (the *a*-path) which is the correlation between X and M when controlling for covariates and any other X values; and the other between M and Y (the *b*-path) which is the correlation between M and Y, controlling for covariates and any other X or M values (Kenny, 2015). The indirect effect is therefore the product of these two coefficients and their associated error terms.

The very well established Baron and Kenny (1986) stepped approach has been an influential method of mediation testing, however it has been criticised in more recent years for being overly simplistic. As methodological knowledge has progressed, alternative methods have been developed which highlight some weaknesses in the stepped approach. It has a tendency to oversimplify complex relationships between variables by demanding the presence of a significant total effect before any mediation testing can be undertaken (MacKinnon et al, 2000; Shrout & Bolger, 2002). The approach does not quantify the total indirect effect (Preacher & Hayes, 2008), nor does it take into account the possibility of suppression effects, whereby a positive and a negative effect within the model essentially cancel each other out (MacKinnon et al, 2000).

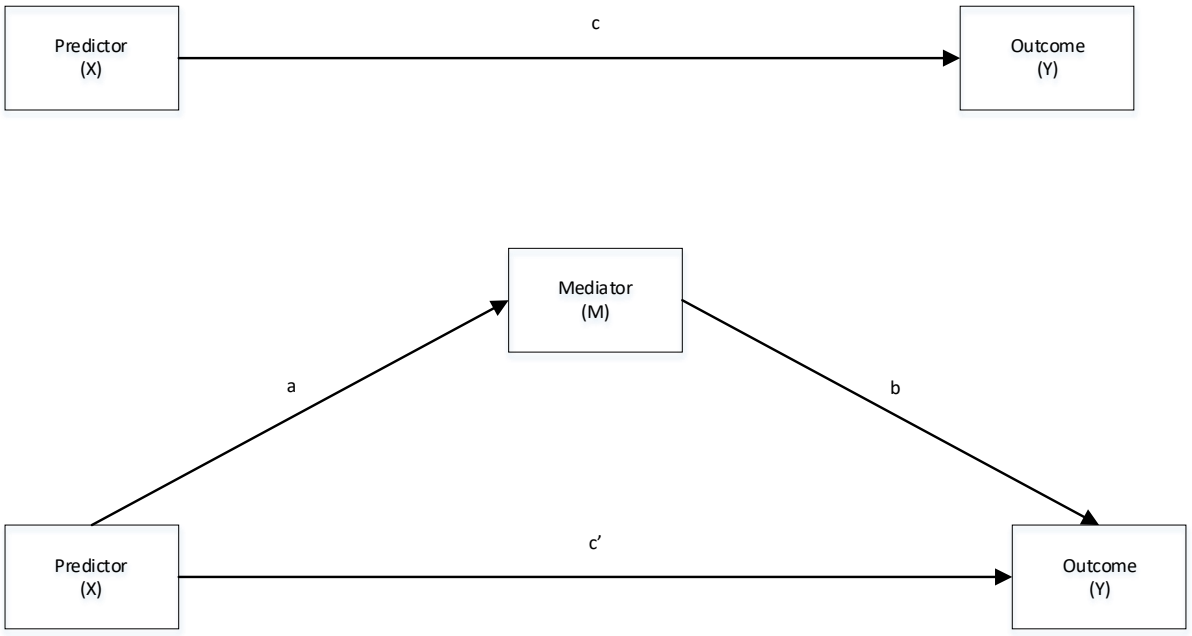


Figure 2.1 The unmediated model depicting the total effect (c), and a simple mediation pathway incorporating the indirect effect (ab) and the direct effect (c').

The Preacher and Hayes (2008) bootstrap method of mediation testing offers an alternative. Bootstrapping is a non-parametric method of resampling the original data set multiple times to generate a more accurate estimates of the true effect, and associated confidence bounds

(Preacher & Hayes 2008). This method also enables significance testing of the indirect pathway.

More recent advances in statistical understanding and in computational power have enabled the simultaneous testing of multiple mediators. These models test and control for all other included variables, and may therefore present a more realistic picture of the complex interrelationships of interest in psychological research (Preacher & Hayes, 2008). However this introduces the risk of multicollinearity where variables in the model are not independent from one another. This may negatively impact the unique contribution of each mediator represented in the model, and will be reflected in the standard errors associated with each point estimate (Alin, 2010). Despite this complexity, Preacher and Hayes (2008) recommend that researchers endeavour to test theoretically sound multiple mediator models as a preference over single mediator models wherever possible. Further details about the Baron and Kenny method, bootstrapping, multiple mediator models and multicollinearity can be found in appendix 4.

2.4 Effect sizes in mediation research

Critics suggest that without an effect size, accompanied by confidence intervals and adequate discussion, mediation results bear little practical usefulness (Lachowicz et al., 2018; Preacher & Kelley, 2011). Existing reviews of mediation research in the area of trauma and psychosis have not contextualised findings in terms of the magnitude of effects, and instead have relied upon statistical significance or proportional mediation results which do not always accurately reflect the size and importance of the effects (Alameda et al., 2010; Williams et al., 2018). This section describes what qualities are desirable in an effect size indicator, and highlights the unique challenges mediation models bring to effect size expression. This information is then used to highlight the interpretative limitations brought about by the indices used by previous reviews.

Effect size reporting is vital in mediation research to facilitate the interpretation of results and to enable comparisons across studies, independent of design characteristics. Effect sizes also

assist with evidence synthesis, for example in meta-analysis (Lachowicz et al., 2018; Wen & Fan, 2015). The magnitude and direction of the effect are equally, if not more important than the significance value, however despite significant growth in mediation research, there is no universally established or recommended metric for the reporting of effects across the literature (Lachowicz et al., 2018).

Effect sizes should make use of an interpretable scale; have associated confidence intervals; be consistent, unbiased, and efficient, and retain their parameter values regardless of resampling (Lachowicz et al., 2018; Fan & Konold, 2010; Williams & MacKinnon, 2008; Preacher & Kelley, 2011; Wen & Fan, 2015). Further information about each of these qualities, along with details of frequently used indices can be found in appendix 5.

In terms of effect size expression, the reporting of the indirect effect brings a level of complexity beyond that associated with more standard indices (Lachowicz et al., 2018; Preacher & Kelley, 2011). As the product of two path coefficients and their associated error terms, they are more challenging to interpret, and researchers must consider how best to communicate such results in a manner which enables the comparison of their highly specific results with other similar research. Perhaps because of this, effect size reporting is variable throughout the empirical literature, and neither of the two recent systematic reviews of mediation research in trauma and psychosis have attempted to compare the magnitude of effects. Williams et al., 2018 report only the primary findings from each included study, and effects are presented in terms of their significance without acknowledgement of how this may be affected by varying sample sizes. Alameda et al (2020) present 'proportion mediated', however this too risks being misleading as it is possible to find large proportions of very small effects being mediated (see further below). Essentially, it remains challenging to compare results across studies without gaining an adequate sense of the overall effect sizes, along with the mediation proportions.

Proportions are simple to compute and are the most frequently reported metric, however they are not recommended for use (Lachowicz et al., 2018; Miočević et al., 2017). They can be unstable in small samples, particularly those of less than N=500 (Lachowicz et al., 2018;

Preacher & Kelley 2011; MacKinnon et al, 1995) and in multiple mediator models individual mechanisms can misleadingly return proportions which are suggestive of 'complete' mediation. They are unbounded and therefore have no ceiling value to denote complete mediation, meaning they are not truly proportions of a 'whole' (Preacher & Kelley, 2011). Proportions can show a large percentage of the effect as being mediated, however if the overall effect is of very small magnitude, the mediation effect may be trivial (Preacher & Kelley, 2011). Without contextualising the proportion mediated against the full effect, it is not possible to adequately interpret and compare proportions. It is however possible to compute confidence intervals for the proportion mediated, which can assist with interpretation and gives an indication of precision (Preacher & Kelley, 2011), yet this is a notable omission from the review by Alameda and colleagues (2020). Neither review offers collated evidence of reported effects, both significant and non-significant, presented in a manner which allows comparison and interpretation across the body of research.

As an alternative to proportion mediated, the completely standardised indirect effect (CSIE) was sought in this review. The CSIE is an estimate which facilitates comparison across studies regardless of the original measurement scales used (Lachowicz et al., 2018). Standardising against both the IV and DV scales, means the CSIE can be simply interpreted as representing the expected standard deviation change in Y for one standard deviation change in X, as mediated through M. The 'complete' standardisation facilitates easy comparison across studies in different populations or where different scales have been used for variable measurement (Cheung, 2009). The CSIE does not depend on sample size, and confidence intervals can be easily derived to determine the precision of the effect (Preacher & Kelley, 2011). However, without the context of direct and total effects, there is still some potential for misinterpretation, but standardisation makes this a more readily comparable metric for use between studies as compared with proportions, as above. As a metric on a standard scale, a larger value will always represent a larger effect estimate and standardised effects are

generally unbiased, consistent and efficient in both single and multiple mediator models (Miocevic et al., 2017; Cheung, 2009).

Partial standardisation, which standardises the effect against the scale of the outcome only, is most often used in cases where the predictor is binary or already has interpretable levels (Lachowicz et al, 2018). It refers to the change in the outcome when there is a unit change in either the mediator or the a-path (MacKinnon, 2008). This was used as an alternative in studies where the CSIE was not possible to derive due to categorical predictors. Further details of the effect sizes sought in this review can be found below (see Extraction hierarchy), and full details of effect size indices and their relative advantages and disadvantages can be found in appendix 5.

2.5 Aims of the study

This study aims to review all existing research which has examined potential mediating mechanisms between childhood trauma and positive psychotic symptoms.

Outcomes will be interpreted by taking into account several factors: the magnitude and reported significance of each effect, the quality of each estimate, and the overall study quality.

The study aims to reach a balanced and transparent judgement of the current state of the evidence, and identify which potential causal mediators show promise as future targets for research or therapeutic intervention.

2.6 Methods

2.6.1 Search strategy

Four electronic databases (PsycINFO, MEDLINE, EMBASE and PILOTS) were searched initially in July 2017 and repeated in November 2018 using search terms including (psychosis OR schizo*) AND (trauma OR neglect OR abuse OR maltreatment). Searches were expanded to specify ‘childhood abuse’ and ‘adult survivors’ as the target population. Searches were adapted to each individual database and were designed to be sensitive and inclusive. The search strategy was developed in collaboration with an information specialist and detailed

copies of the search terms can be found in appendix 6. In addition, the reference lists of relevant review articles were hand searched.

Duplicate titles were removed and a list of remaining titles were scrutinised for ineligible articles. The abstracts of the remaining articles were screened for further exclusion criteria. Following this, the full text versions of all remaining articles were examined. Any uncertainties throughout the process were checked by a second reviewer.

2.6.2 Study selection and inclusion

All studies investigating psychological mediating mechanisms accounting for the relationship between the experience of early interpersonal trauma and the later experience of clinical, sub-clinical or attenuated positive psychotic symptoms were included. Data from clinical, at-risk and non-clinical populations were included. For inclusion, clinical groups were required to have a minimum of 50% of participants with a diagnosis of non-affective psychosis. Cross-sectional, cohort studies, case-control studies, prospective studies and trials were included where other inclusion criteria were also met. Both published and unpublished studies (e.g. theses) were eligible for inclusion provided sufficient data and a full text report were available, or could be obtained from authors. Where articles matched inclusion criteria but contained insufficient data for analysis, authors were contacted for additional information. Only studies published in English were included.

2.6.3 Outcomes

The primary outcomes for this review were the magnitude, direction, statistical significance and quality of the mediators of the trauma-psychosis pathway. Similar mediation mechanisms were grouped into categories and each category was assessed for its overall importance.

The categories were created by listing all of the potential mediators from included studies, and identifying similarities between them. This involved consideration of the description of the potential mediator, and the tools used in the study for its measurement. Initially this was undertaken by one researcher. Confirmation of the groupings and discussion of uncertainties

involved two further researchers. The final categories were (i) PTSD symptoms and dissociation; (ii) cognitive beliefs and appraisals; (iii) attachment style; (iv) mood and anxiety; (v) emotion regulation and stress sensitivity; (vi) social defeat. Further details of group assignment can be found in the results section below.

2.6.4 Data extraction

Study characteristics and participant characteristics were recorded. This included the type of study, the country where the research took place, the groups included in the review (e.g. clinical, high risk) and the number of participants per group. Sample characteristics included age, gender, ethnicity, education and employment.

The variables included in each mediation pathway were recorded, as were the tools used to measure these. Details on how diagnosis was confirmed was also noted where appropriate. The type data provided for each variable was logged (either continuous or binary) as this was relevant to understanding the effect size metrics provided in each paper. Statistical information was extracted for each mediation relationship. Primarily information about the magnitude and significance of the indirect (mediation) effect was sought, but direct and total effect data was recorded where available as this provided context, and also assisted with computing and converting effects where required.

Papers reported a variety of different effect size indicators including standardised and unstandarised regression coefficients, point estimates, odds ratios and logodds ratios. An extraction hierarchy was developed (see section below and figure 2.2) to prioritise the estimation of effects.

2.6.5 Extraction hierarchy

Despite the high frequency of proportions and ratios being reported, a hierarchy of desirable metrics for extraction and comparison was developed (see Figure 2.2). The completely standardised indirect effect (CSIE) was prioritised as this has been shown to be interpretable across studies where different scales have been used for measurement. Where this was not

readily presented, various methods were employed to derive the CSIE and associated confidence intervals -see section 2.6.7 below. In the case of a dichotomous predictor variable, partial standardisation was undertaken to produce the partially standardised indirect effect.

Studies with dichotomous predictor or outcome variables often expressed effect sizes as odds ratios (OR); these formed the third level of the hierarchy. The odds ratio for the indirect effect was extracted, along with the direct and the total effect to provide context. Confidence intervals (CIs) were extracted or computed, and efforts were made to convert logodds values to odds ratios for comparison.

In any studies where adequate information to extract or derive the above metrics was not provided, the proportion mediated was used. Again this was contextualised using the direct and total effects wherever possible. Where ratio or proportion data were used, the interpretation of effects was cautious due to the instability of the metric in small samples and the potential bias this may introduce. See appendix 5 for further details of the disadvantages of using proportion mediated as an outcome.

Finally, if the data provided in the paper were not in a format which allowed for direct extraction or conversion to any of the above metrics, the study was retained for narrative inclusion only. In studies where this was the case, authors were contacted for the additional information required for full inclusion. Where this data was not provided, studies which met all other inclusion criteria were retained in the interests of completeness, and their findings were described as reported in the paper, with the clear caveat that the effect sizes could not be quantified or compared.

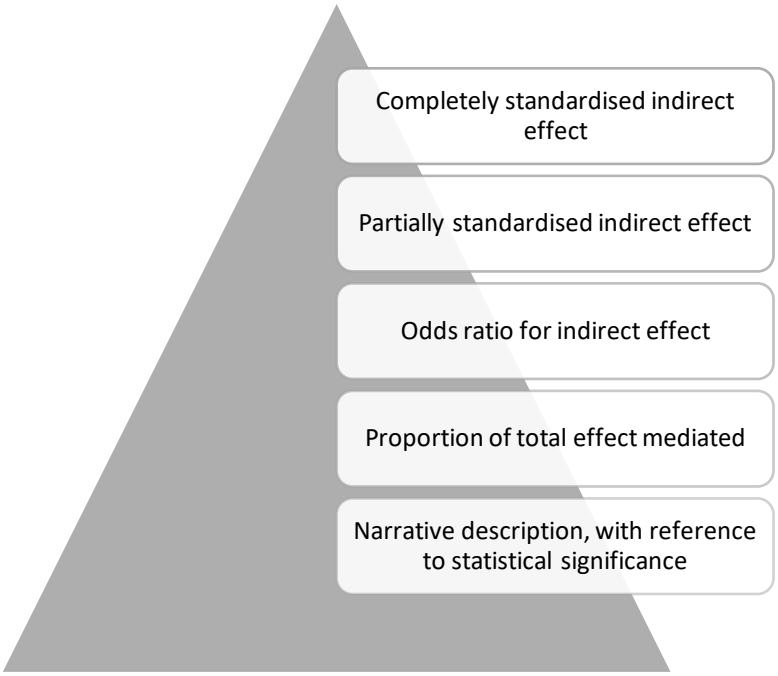


Figure 2.2 - Hierarchy of effect sizes

2.6.6 Assessment of study quality

2.6.6.1 Agency for Healthcare Research and Quality (AHRQ) tool

In line with other recent meta-analyses (Taylor et al., 2015; Dudley et al., 2016; Larkin & Hutton, 2017), an adapted version of the Agency for Healthcare Research and Quality (AHRQ) tool was used to assess methodological quality at an individual study level. The adapted version of the tool is available in appendix 7. Several adjustments were made to ensure the tool was adequately specific to the studies under review, and to ensure the quality assessment was balanced and transparent.

Studies were rated on up to nine discrete criteria including participant matching, controlling for confounding variables and the use of a-priori power calculations. The tool aims to distinguish between methodological quality and the quality of study reporting, and considers multiple factors within each criterion before applying a rating. Adaptations to the original tool were based on sample size; as many studies were secondary analyses of large data sets, it was often the case that an a-priori calculation was not provided, but samples were sufficiently large to detect the presence of effects and therefore it would be unreasonable to downgrade a study

on these grounds. Similarly, with the handling of confounding variables, caveats were added to clarify that studies would not be downgraded for not reporting confounders if their mediation modelling techniques implied that potential confounders were automatically controlled. Criteria assessing baseline differences between groups, and the measurement or confirmation of psychotic symptoms were only applied to relevant studies which had multiple and/or clinical groups.

In the interests of rigour a second reviewer independently rated a random sample of the included studies (35%). Cohen's Kappa was 0.66 indicating substantial agreement according to interpretative thresholds (Cohen, 1960). Raw percentage agreement was 84%. Discrepancies were resolved through discussion until consensus was reached. The agreed rating was then checked with a third reviewer.

In order to incorporate study quality as a metric in the overall assessment of each category, a quality score was computed for each study. For each criterion studies were awarded three points for a rating of 'yes', two points for a rating of 'partial', one point for a rating of 'unclear' (which suggests there is reason to believe the criteria may have been satisfied but is not clearly reported in the text) and zero points for 'no'. Each study total score was divided by the number of criteria relevant to the assessment (range 7-9). Quality ratings were as follows: very low quality = 1.4 – 1.9; low quality = 2.1 – 2.3; moderate-low quality = 2.3 - 2.49; moderate quality = 2.5 - 2.69; moderate-high quality = 2.7 - 2.79 and high quality = 2.8 – 3.0 (range 1.4 - 2.88).

2.6.6.2 Grading of Recommendations Assessment, Development and Evaluation (GRADE) criteria

The Grading of Recommendations Assessment, Development and Evaluation (GRADE) provides a structured and replicable framework for the transparent reporting of healthcare evidence. Originally designed for use with clinical trials or interventions research, many of the criteria are also relevant to other types of studies, to appraise outcomes in response to

different types of research questions. A number of factors are considered which may lead to the upgrading or downgrading of each outcome.

Outcomes may be upgraded for finding large effect sizes, providing evidence of a dose-response gradient, or dealing well with potentially confounding variables (Dijkers, 2013). Outcomes may be downgraded for risk of bias (brought about by study design and conduct); inconsistency (unexplained heterogeneity); imprecision (uncertainty around effect estimates); indirectness (degree of generalisability to wider populations) and potential publication bias (Guyatt, et al., 2011; Dijkers, 2013; Schünemann, 2013; McMaster University, GRADE online learning modules, n.d.). Further details about GRADE and each of the criteria can be found in appendix 8.

Factors are additive, but must be considered on a continuum rather than as points on a quantitative scale. Reviewers are encouraged to consider all criteria relevant to each outcome, and the severity of their impact before reaching a judgement (Schünemann, 2013). Judgements are required to be transparent and explicit, and any borderline decisions or uncertainties should be highlighted as such (Schünemann, 2013). Outcomes are then placed into one of four categories: high, moderate, low or very low quality (Dijkers, 2013). The GRADE ratings were applied to each outcome category in this review and judgements can be found in Table 2.14.

2.6.7 Analysis

All reported mediation data was extracted for both significant and non-significant pathways where available. Favour was given to simple models as there was less ambiguity about the partitioning of effects.

To facilitate comparisons between studies, effect sizes were required to be converted into comparable metrics. Following the extraction hierarchy (see above) the CSIE was sought in studies where continuous data was used. Odds ratios were used in studies where one or more

variables were binary. See Figures 2.3 and 2.4 for continuous and binary data processing flowcharts.

The majority of studies reported unstandardised beta regression coefficients and confidence intervals which were standardised following the formula provided by Preacher and Kelley (2011). Where confidence intervals (CI) were not provided, standard error (SE) was used, or derived, in order to compute them (Lipsey & Wilson, 2001).

In all cases where more extensive computations were required, authors were contacted for information which would enable simpler calculations to be performed – i.e. those which would require fewer assumptions. These alternative methods included using normative Z-value tables to help derive SE for CI computations; and the use of ratios between reported standardised and unstandardised effects which were applied to compute estimates of indirect effects and associated confidence intervals See appendix 9 for further details.

In papers where binary data was used the odds ratio (OR) and 95%CIs for indirect effects were considered essential for extraction, and the direct and total effects were required for context wherever available. A number of papers presented logodds which were converted to OR. If confidence intervals were not provided, they were derived using SE values.

More complex papers required more statistical conversions to be undertaken. Where certain paths were provided in a different format (for example in Bebbington 2011, where the *a*-path is presented as a continuous value whilst all other paths are expressed as odds ratios). Standard deviation for the *a*-path was derived using reported confidence intervals and sample size (Deeks et al, 2011). This was used to convert the reported *a*-path effects to *d* using the Campbell effect size calculator (Wilson, n.d.). This d value was then converted to logodds following methods in Borenstein (2011) and standard errors were computed using a CMA. These were treated as the *a*-path estimates. These, along with reported values for the b-paths were input into formulas specified in Iacobucci (2012) to compute Z-values for first the *a* and *b* paths individually. These were then multiplied to derive a Z-value for *ab*. The SE was derived

and the overall Z for mediation was computed following Iacobucci (2012). This Z was converted to *r* following Rosethal and DiMatteo (2001). Next *r* was converted to *d* and 95% CI for *d* using formulae from Lipsey and Wilson, 2001. Finally, the values were converted from *d* to OR. The final values were double-checked using the Campbell effect size calculator (Wilson, n.d.) to ensure accuracy of the formulae applied.

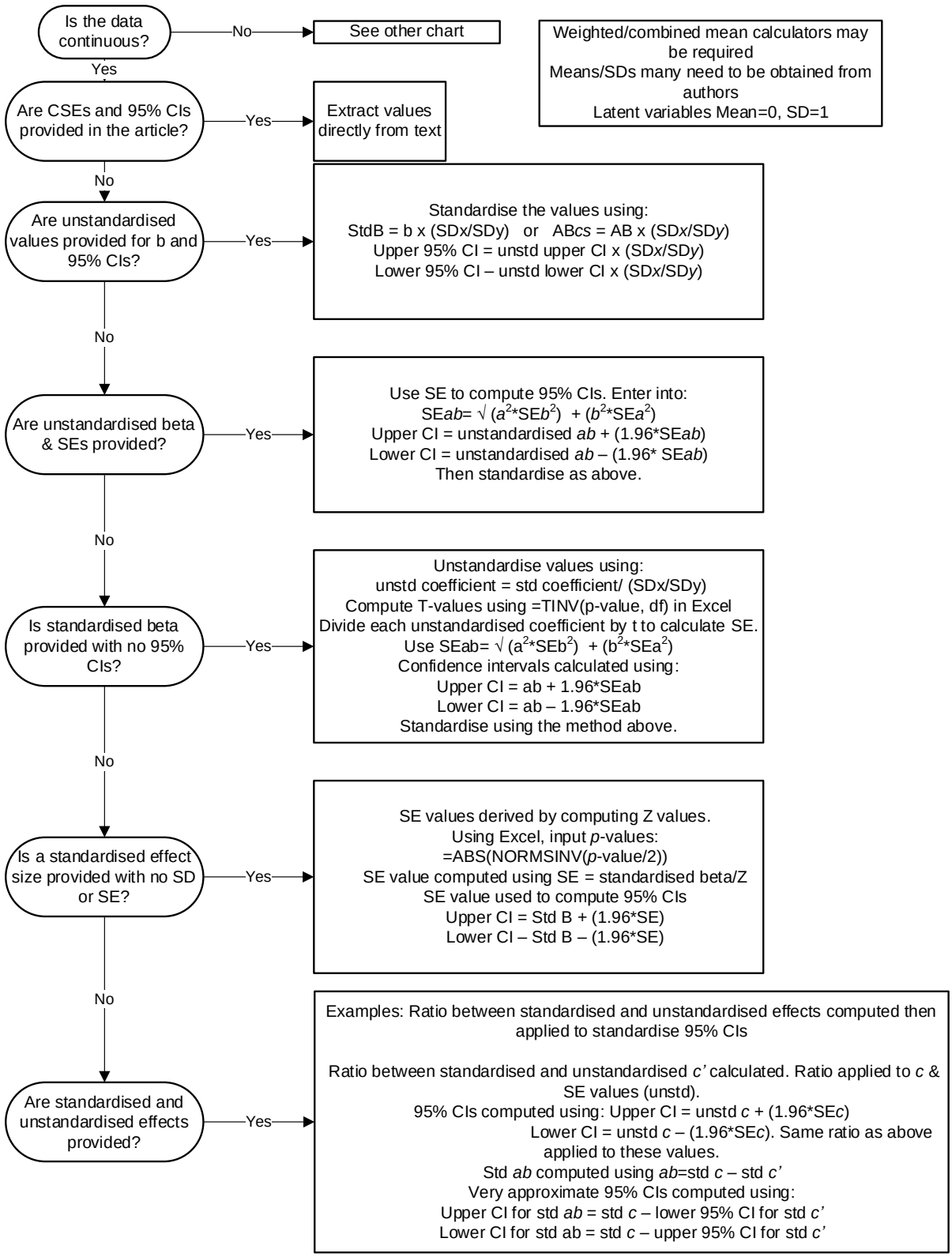


Figure 2.3 - Continuous data decision flowchart

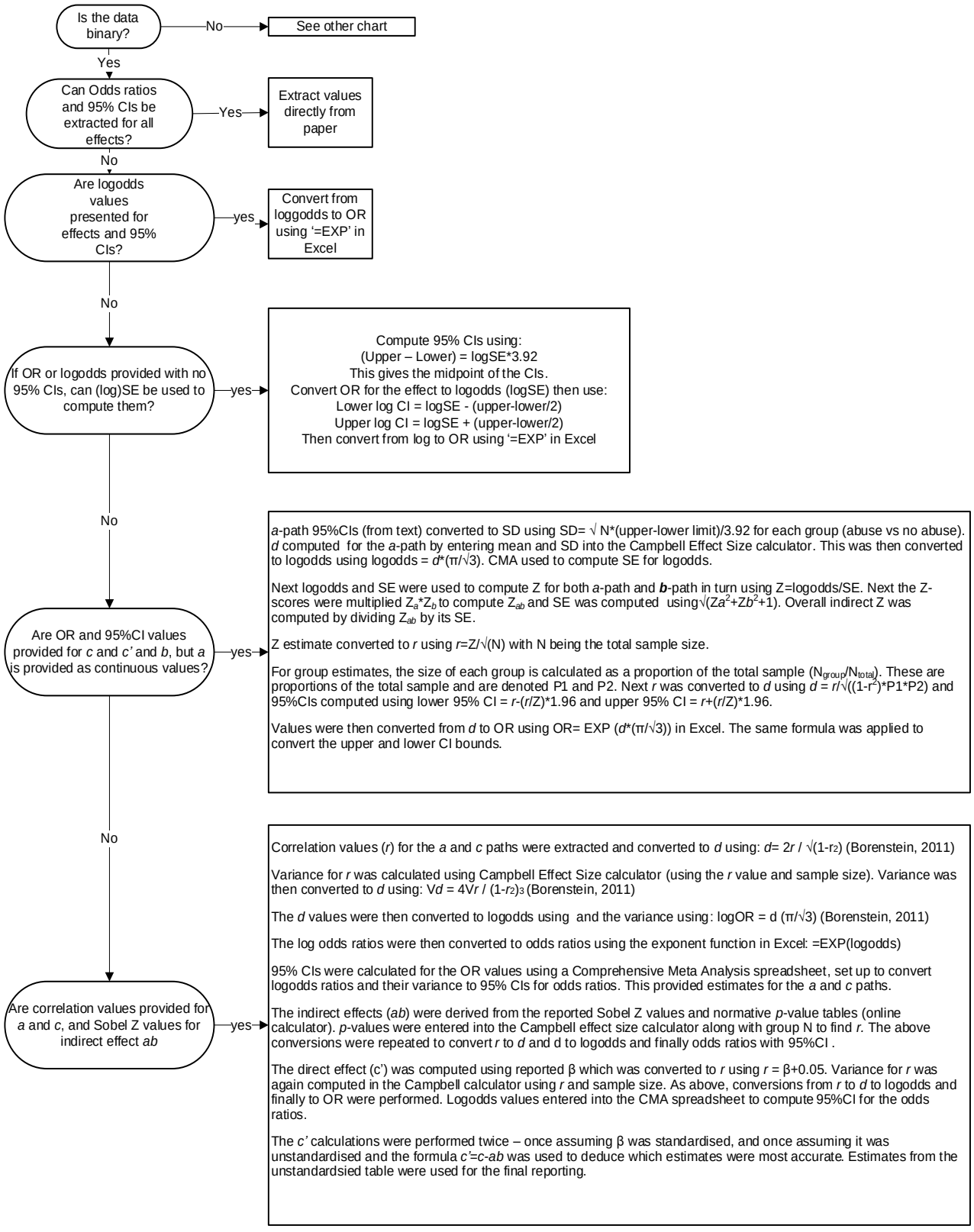


Figure 2.4 - Binary data decision flowchart

Where only select pathways (*a*-path and *c*-path) were reported (Berenbaum et al., 2008), correlations were extracted from the paper and the *r* values and their variance (computed using the Campbell effect size calculator) were converted to *d* and variance, then logodds and variance and finally OR following Borenstien (2011).Comprehensive Meta-Analysis (CMA) (version 3, Borenstein et al.) was used to compute 95% CIs for OR using logodds and variance.

The indirect effect (*ab*) estimates were derived using Sobel Z-scores. Exact *p*-values were identified for the reported Z-scores using an online calculator (https://www.socscistatistics.com/pvalues/normaldistribution.aspx) and entered along with sample size into CMA to compute *r* and variance for *r*. The above steps were followed to convert *r* to *d* and *d* to logodds, and finally odds ratios with 95% CIs. For the direct effect (*c'*), reported β values were converted to r and variance then the process above was followed to convert to OR. 95% CIs for the direct effects were computed using CMA.

The *c'* conversions were performed twice - once assuming the β stats provided were already standardised, the other assuming they were unstandardised.

The formula *c'=c-ab* was used to check the estimates. The figures from the unstandardised table were more closely matched, suggesting that the reported β values were unstandardised estimates. Therefore, figures from the 'assumed unstandardised' table were used in the final reporting.

The appropriate calculations were performed per study, and were independently checked by a second reviewer. Forest plots for CSIE and OR for each category were created in order to visually inspect effect sizes. Interpretative thresholds were sought in the literature. CSIE were interpreted as null (0), small (0.14), medium (0.36) and large (0.51) (Cheung, 2007) and odds ratios were null (1), small (1.68), medium (3.47) and large (6.71) (Chen, Cohen & Chen, 2010). For the latter, these were the suggested cut-off scores when the incidence rate of the condition (psychosis) is 1% in the 'non-exposed' (no-trauma) group. As the vast majority of odds ratio

papers were conducted in general population samples, it was felt this incidence rate was representative. A recent study examining data from 1990-2017 found an incidence rate of 1130.5 new cases globally per 100,000 head of population (95%CI 1000.6-1281.9) (James et al, 2018). One study reported odds ratio data in a high-risk population (Thompson et al., 2016) and one study in a clinical population (Hardy, 2016). The effect sizes from each were considered in terms of the above criteria for a 1% incidence rate, but also for a 10% incidence rate in the non-trauma-exposed group.(small 1,46; medium 2.50; large 4.14) as incidence or transition rates would be expected to be higher (Chen et al., 2010).

An additional graph was created to enable visual comparison of the magnitude and significance of effect sizes, along with study quality (See Figure 2.6). Details of all extractions and computations per study can be found in appendix 9.

2.6.8 Protocol Registration

The protocol for this study was registered in advance with the International Prospective Register of Systematic Reviews (PROSPERO), registration number CRD42017072428. A copy of the protocol is included see appendix 1.

2.6.9 Amendments to protocol

Ahead of commencing the review, a brief scoping review was undertaken in order to develop the protocol. At this time it was not clear how complex and diverse the existing evidence was. The protocol specified that in addition to the magnitude, direction and significance of effects, the amount of variance explained by each mediator would be recorded. This was not always possible, particularly in cases where multiple mediator models were reported. In light of methodological evidence above which suggests that proportions are unstable unless they are based on data from very large samples (Lachowicz et al., 2018; Preacher & Kelley 2011), and the fact it was not possible to obtain complete information across the studies, the recording of variance explained by each mediator was not undertaken. Similarly, the protocol stated the intention to extract and report correlations between change in mediator and reciprocal change

in positive symptoms. In many cases this data was not reported, and due to the array of different mediators and outcomes, this information would have been of little comparative usefulness. It was also stated in the original extraction list that confounding variables would be recorded – this formed part of the AHRQ quality assessment.

The original protocol specified that findings would be assessed against the Bradford Hill criteria for causality. This was not formally undertaken in the planned format. Existing evidence was found to be almost exclusively cross-sectional, and therefore unable to offer information about potential causal direction and reversibility. This review considered aspects of the criteria, including the strength of the relationship (magnitude of the effect size), the consistency (overall evidence per category) and plausibility of relationships (theoretical basis for investigating the mediator, and the implications of the evidence), and discusses findings in this context. However, it was not thought to be beneficial to apply rigid assessment criteria when the current evidence base is unable to support causal claims.

2.7 Results

2.7.1 Search results

As shown in Figure 2.5, database searches returned 8639 articles, with hand searches identifying a further 16 articles for review. Of these 8655 articles, 890 were identified as duplicates and removed. A further 7375 articles were rejected following title and abstract examination. The full-text reports of 390 articles were reviewed further, with 353 of these failing to meet inclusion criteria. The most common reason for rejection at the full text stage was insufficient provision of mediation data (56.4%). In total 37 articles were included but effect sizes could only be calculated for 33 of these. The remaining four papers were retained for narrative inclusion only. Studies excluded at the full text stage are listed with brief reasons for exclusion in appendix 10.

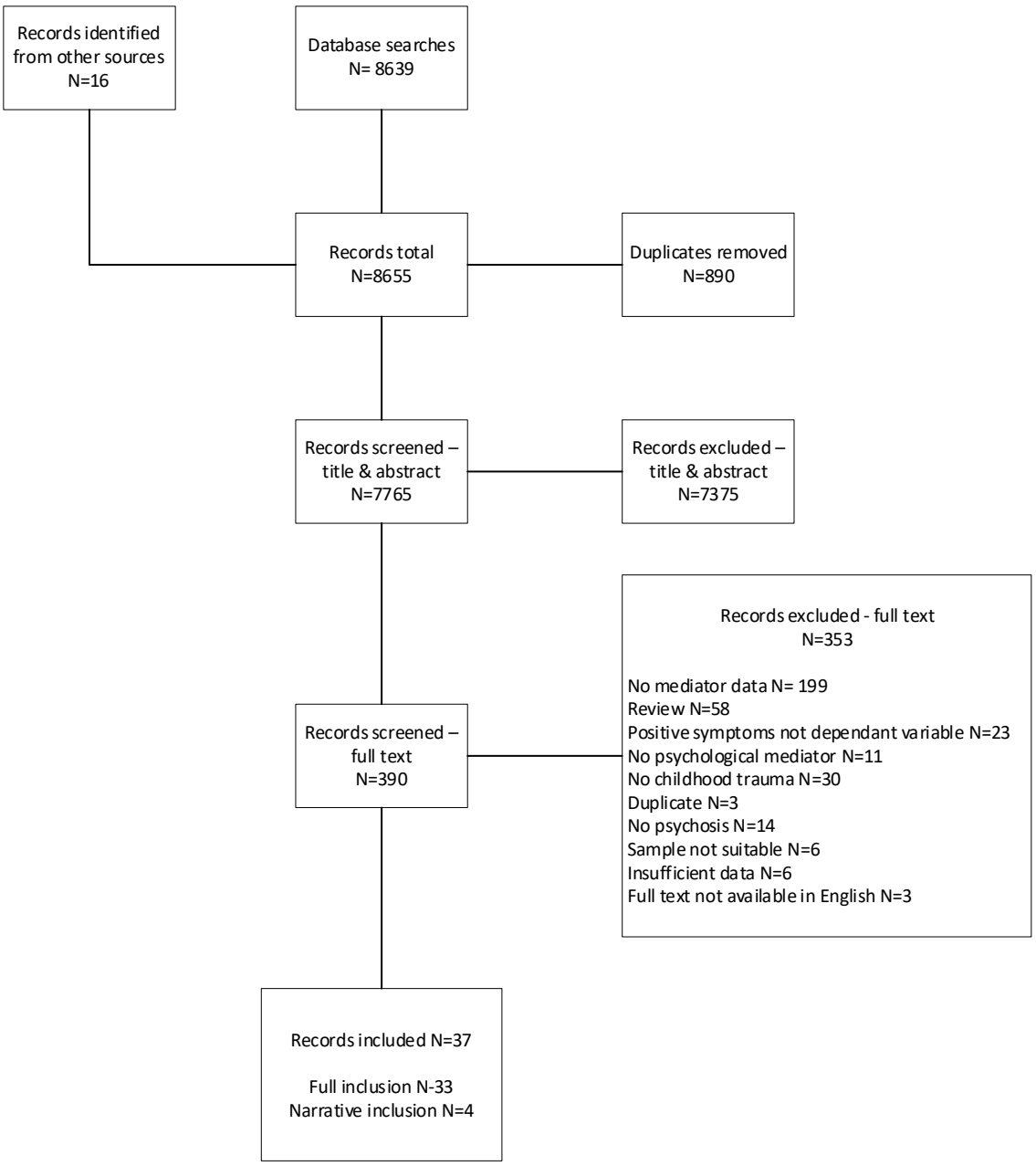


Figure 2.5 - PRISMA flowchart

2.7.2 Categories

The final included studies investigated a range of mediation mechanisms. Effect size estimates were grouped under the following six categories of mediator: (i) PTSD symptoms and dissociation (*k*=12); (ii) cognitive beliefs and appraisals (*k*=13); (iii) attachment style (*k*=9); (iv) mood and anxiety (*k*=7); (v) emotion regulation and stress sensitivity (*k*=6); (vi) social defeat (*k*=4).

The ‘PTSD and dissociation’ category included overall dissociation (Choi, 2017; Pearce et al., 2016; Thompson et al.,2016; Evans et al., 2015, Varese, Barkus & Bentall, 2012), and its component parts: dissociative amnesia, absorption, depersonalisation (Cole et al., 2016; Perona-Garcelan et al., 2012 & 2014; Berenbaum et al., 2008). Post-traumatic stress based mediators included intrusive memory; avoidance and numbing; and hyperarousal (Hardy et al., 2016;) or more broadly categorised total PTSD symptoms (Choi et al., 2015 & 2017; Powers et al., 2016).

‘Cognitive beliefs and appraisals’ included negative schemas and beliefs about the self and others (Appiah-Kusi, 2017; Hardy 2016; Ashford 2010; Fisher, Appiah-Kusi & Grant, 2012; Jaya, Ascone & Lincoln 2017); self-esteem (Morgan et al., 2014); self-concept clarity (Evans, 2015); beliefs in a ‘just world’ (Wickham & Bentall, 2016); self-disturbance; cognitive biases (Gaweda, 2018 a & b) and metacognition (Østefjells, 2017; Goldstone, Farhall & Ong, 2011 & 2012[1]).

‘Attachment style’ focused largely on anxious and avoidant insecure attachment (Pilton et al., 2016; Goodall et al., 2015; Sitko, Bentall, Shevlin & Sellwood, 2014; Van Dam, et al., 2014[2]). Other studies partitioned this into more specific attachment styles (e.g. enmeshed, angry-dismissive, fearful) (Pearce, 2016; Sheinbaum, Kwapil & Barrantes-Vidal, 2014; Sheinbaum et al., 2015). Interpersonal sensitivity (McDonnell, 2018) and sensitivity to rejection (Ashford, 2010) were also included in this category.

In the ‘mood and anxiety’ category the majority of studies assessed both anxiety and depression (Ashford, 2010; Marwaha & Bebbington, 2015; Thompson et al., 2016; Fisher et al., 2012; Bebbington et al., 2011; Østefjells et al., 2017). Other mood related mechanisms were mood instability (Marwaha et al., 2014); mood swings, and mania (Thompson et al., 2016).

[1] Goldstone, Farhall & Ong 2011 & 2012 did not contribute effect size data, and were not included in the GRADE analysis. Evidence was narratively included and study quality was assessed.
[2] Van Dam et al. 2014 was included narratively, as above.

In the ‘emotion regulation and stress sensitivity’ category potential mediators generally had stress response at their core. These included emotion regulation and dysregulation (Lincoln, Marin & Jaya 2017; Van Nierop et al., 2014); experiential avoidance (classified as a negative emotion regulation strategy) (Goldstone, Farhall & Ong, 2011 & 2012[1]); stress sensitivity (Rossler et al., 2016); and mindfulness (Perona-Garcelan et al., 2014).

The ‘social defeat’ category focused on psychological and emotional aspects of social dynamics, and incorporated both objective and subjective measures of potential mechanisms. These included loneliness (Boyda 2015; Jaya, Ascone & Lincoln, 2017); social rank (Jaya, Ascone & Lincoln, 2017); social defeat (Van Nierop, 2014); adult disadvantage, and a lack of attainment and educational qualifications (Morgan et al., 2014).

Study characteristics and sample demographic details can be found in Table 1. The variables included in each mediation model and the tools used to measure these can be found in the appendix 11. The quality of studies in each category is discussed and used to inform the interpretation of effect size. Quality ratings can be found in Tables 2-7. Hereafter in the text and tables, for reasons of clarity, each study is identified by the name of the primary author and date only.

Table 2.1 - Characteristics of included studies and baseline demographics

Study Ref	Groups included in review	N participants	Country	Age, mean (SD)	Proportion male	Education	Ethnicity	Employment
Appiah Kusi 2017	UHR for psychosis group	30	UK	23.93 (4.77)	53%	GCSE (16.7%) Degree started (26.7%) Degree Completed (13.33) Other (43.27%)	White (36.7%) Black (20%) Mixed (23.3%) Other (20%)	Full time (30%) Part time (10%) Out of work (16.7%) Student (16.7%) Other (23.3%)
Ashford 2010	Subclinical (students)	135	UK	Range 18-44	12	Not Reported	White British (77.7%) White other (7.4%) Chinese (5.2%) Caribbean (4.4%) Indian (3%) Mixed/Other (9.7%)	Student 100%
Bebbington 2011 & Marwaha & Bebbington 2015	General population	642 5689	UK (APMS 2007)	16+	Not reported	Not reported	Not reported	Not reported

Study Ref	Groups included in review	N participants	Country	Age, mean (SD)	Proportion male	Education	Ethnicity	Employment
Berenbaum 2008	Higher 'odd beliefs' subsample from original study	303	USA	43.2 (17.6)	46.9%	College degree 53.8% College education 33.8% High school diploma 11.0% Didn't finish high school 1.4%	European American 78.9% African American 9.4% Asian 5.4% Latino 2.7% Bi-racial 1.7% Native American 1.3% Other 0.6%	Not reported
Boyda 2015	Community sample	7403	UK (APMS 2007)	46 (18.6)	49%	No qualifications 24%	Minority 13.7%	Age, gender, education, ethnicity, low socioeconomic status, drug dependence.
Choi 2015	SMI with psychotic features	126	Korea	36.14 (13.79)	44%	Not reported	Not reported	Not reported
Choi 2017	Clinical outpatients	169	Korea	35.53 (16.59)	57%	Not reported	Not reported	Not reported

Study Ref	Groups included in review	N participants	Country	Age, mean (SD)	Proportion male	Education	Ethnicity	Employment
Cole 2016	Community sample (undergraduate students)	200	UK	19.96 (2.18)	17%	Not reported	White British 69% White Other 7.5% Indian 4.5% Other 19%	Not reported
Evans 2015[1]	Clinical	29	UK	Range 18-38	66%	None 4 GCSE 0 A Levels 4 HND/HNC 12 Degree 9	White British 25 Other 4	Employed 8 Other 21
	Non-clinical	31			61%	None 1 GCSE 2 A Levels 4 HND/HNC 16 Degree 8	White British 29 Other 2	Employed 22 Other 9
Fisher 2012	Community sample	212	UK	27.0 (8.4)	35%	Not reported	White British 26.6% Other 73.4%	Employed 36.0% Studying 56.5%
Gaweda 2018 a.	Healthy individuals (students)	650	Poland	23.3 (3.97)	20%	Not reported	Not reported	Not reported

Study Ref	Groups included in review	N participants	Country	Age, mean (SD)	Proportion male	Education	Ethnicity	Employment
Gaweda 2018 b.	Healthy individuals (students)	653	Poland	22.24 (3.14)	29%	Not reported	Not reported	Not reported
Goldstone 2011 & 2012[1]	Clinical	100	Australia	18-25 13.1% 26-35 33.3% 36-45 31.3% 46+ 23.3%	56%	Incomplete secondary 53.6% Complete Secondary 10.3% Degree 36%	Not reported	Full time 0% Part time 10.1% Student 3% Unemployed 73.7%
	Non Clinical	133		18-25 45.0% 26-35 32.8% 36-45 8.4% 46+ 13.7%	41%	Incomplete secondary 4.5% Complete Secondary 6% Degree 89.5%		Full time 38.3% Part time 21.1% Student 30.1% Unemployed 10.5%
Goodall 2015	Non-clinical group	283	UK	26.8 (9.28)	28%	Not reported	Not reported	58% students
Hardy 2016	Clinical group	228	UK (sample from PRP trial)	38.24 (11.11)	72%	Not reported	White 73% Black African 10% Black Caribb.7% Other 10%	Unemployed 80%

Study Ref	Groups included in review	N participants	Country	Age, mean (SD)	Proportion male	Education	Ethnicity	Employment
Jaya 2017	Community sample	2350	USA, Germany & Indonesia	32.53 (11.38)	62.2%	Not reported	Not reported	Not reported
Lincoln 2017	Community sample	562	USA, Germany & Indonesia (subsample from Jaya 2017 study)	35.99 (12.77)	50.7%	Not reported	Not reported	Not reported
Marwaha 2014	Community sample	7403	UK (APMS 2000 & 2007 surveys)	Range 16-74 16+	Not reported	Not reported	Not reported	Not reported
McDonnell 2018	CHR sample	64	UK	22.5 (4.0)	59%	Mean 13.25 years (SD 2.3)	Black 29.7% White British 35.9% White Other 17.2% Other 17.2%	Employed 25% Student 18.8% Unemployed 56.2%
Morgan 2014[2]	Clinical group	390	UK (from AESOP study)	30.5 (10.8)	55.9%	No qualifications 32% Any qualifications 68%	White British 45.4% White Other 7.2% African Caribb. 27.4%	Not reported

Study Ref	Groups included in review	N participants	Country	Age, mean (SD)	Proportion male	Education	Ethnicity	Employment
	Control group	391		37.3 (12.5)	41.2%	No qualifications 18.3% Any qualifications 81.7%	Black African 11.0% Asian 5.6% Other 3.3% White British 61.4% White Other 10.7% African Caribb. 18.9% Black African 5.6% Asian 2.1% Other 1.3%	
Østefjells, 2017[3]	Total clinical sample	261	Norway	30.2 (09.6)	53.6%	Mean 13.0 years (SD 2.5)	Caucasian 78.5%	Unable to work or study 19.8%
Pearce 2016	Clinical group	112	UK	40.26 (12.50)	27%	GCSEs or less 23% A levels 16% Undergraduate degree 33% Postgraduate degree or above 28%	White Caucasian 89% Other 11%	Unemployed 35% Working 41% Studying 24%

Study Ref	Groups included in review	N participants	Country	Age, mean (SD)	Proportion male	Education	Ethnicity	Employment
Perona-Garcelan 2012	Clinical group	71	Spain	Men 38.63 (9.15) Women 40.53 (8.5)	76%	Not reported	Not reported	Not reported
Perona Garcelan 2014	Students	318	Spain	21.41 (5.78)	21%	Not reported	Not reported	Not reported
Pilton 2016	Clinical group	55	UK	42.16 (11.33)	80%	None 40% GCSE 45% A-Level 10.9% Higher 3.6%	White British 83.6% Black British 5.5% Mixed 7.3%	Not reported
Powers 2016	Clinical group	328	USA	40.84 (12.04)	14.6%	Not reported	African American 96.0% White 1.8% Hispanic 0.3% Mixed 1.8%	Not reported
Rossler 2016	Psychosis subsample (subclinical)	663[4]	Swizerland (from ZInEP study)	31.52 (6.77)[5]	47.4%	Not reported	Not reported	Not reported
Sheinbaum 2014	Undergraduate student sample	546	Spain	20.6 (4.1)	16.8%	Not reported	Not reported	Not reported
Sheinbaum 2015	Non-clinical group	214	Spain	21.4 (2.4)	22%	Not reported	Not reported	Not reported

Study Ref	Groups included in review	N participants	Country	Age, mean (SD)	Proportion male	Education	Ethnicity	Employment
Sitko 2014	Community sample	5877	USA (from NCS)	Range 15-54yrs	Not reported	Not reported	Not reported	Not reported
Thompson 2016	UHR group	233	Australia (from PACE study)	Range 15-30yrs	41.2%	Secondary school 76.6% Higher23.4%	Not reported	Not reported
Van Dam 2014[1]	Patients	131	Netherlands (GROUP)	31.19 (10.58)	84%	Not reported	Caucasian 81% Other 9% Mixed 10%	Not reported
	Siblings	123		30.89 (8.12)	47%		Caucasian 85% Other 4% Mixed 11%	
Van Nierop 2014	Extended psychosis phenotype group	384		43 (13.1)	36%	Low/no education 7% Lower secondary 27% Upper secondary 37% Higher/Professional 29%	White European 89% Other 11%	Paid work 69%

Study Ref	Groups included in review	N participants	Country	Age, mean (SD)	Proportion male	Education	Ethnicity	Employment
	Psychotic disorder group	43		41 (12.9)	40%	Low/no education 7% Lower secondary 42% Upper secondary 28% Higher/Professional 23%	White European 72% Other 28%	Paid work 47%
Varese 2012	Schizophrenia spectrum disorder group	45	UK	44.71 (12.79)	53%	13.0 years (SD=2.78)	Not reported	Not reported
	Healthy Control Group	20		39.5 (14.6)	55%	16.1 years (SD=3.1)		
Wickham 2016[6]	Patient group	72 (50 for mediation but info given for full sample)	UK	43.46 (11.17)	63.9%	Secondary 72.2% Vocational training 5.6% Higher education 12.5% No info 9.7%	Not reported	Not reported

1 Studies included in narrative analysis

2 N = 240 cases and N=295 controls provided data for self-esteem analysis

3 Combined clinical sample consisted of psychosis N-163 and bipolar N=98 groups. Psychosis comprised >50% of the sample. Mediation was undertaken on the combined group.

4 Paper reports N=820 but only N=663 in analysis

5 Paper provides age mean & SD by group – combined mean calculator used to calculate for full sample

6 N=50 after listwise deletion for the mediation analysis

2.7.3 Quality overview

2.7.3.1 AHRQ assessment

Study quality was negatively affected by a number of factors. Only 50% of studies included unbiased samples, with 45% of studies failing to adequately describe sample demographics, and only 27% of studies minimising baseline differences between groups in studies where multiple groups were included. Close to one quarter of studies did not use a validated psychosis measure (24%) but 90% of studies did use valid means of measuring potential mediators. Confounding variables were not controlled in 37% of studies, and in 39% of studies missing data was not adequately handled. Quality ratings for individual studies can be found in Tables 2-7, grouped by category.

2.7.3.2 GRADE assessment

The GRADE ratings for each category varied and ratings can be found in Table 2.14. The majority of downgrading was due to risk of bias in sample selection and study design. The AHRQ criteria in the tables highlights which individual studies and categories were most affected by this. One category was downgraded for inconsistency (attachment) as significant heterogeneity was found in the findings. One category was downgraded for imprecision (mood and anxiety) due to marked variation in effect estimates and confidence intervals which suggested a lack of certainty. One category was downgraded for indirectness (social defeat) as the mediators and populations were diverse and at present there was an insufficient number of studies to negate the potential impact of this.

Table 2.2 - Quality ratings – PTSD symptoms & dissociation

Study	Unbiased selection of cohort	Minimises baseline differences	Adequate sample size/power	Adequate description of cohort	Valid psychosis measure	Valid measures of variables	Controls for confounders	Few instances of missing data/ adequate handling	Analytical methods appropriate
Berenbaum 2008	No	N/a	Yes	No	Partial	Yes	Yes	Partial	Yes
Choi 2015	Yes	N/a	Unclear	Partial	Yes	Yes	Unclear	Unclear	Yes
Choi 2017	Yes	N/a	Unclear	Partial	Yes	Yes	Yes	Yes	Partial
Cole 2016	No	N/a	No	Yes	N/a	Yes	No	Partial	Partial
Evans 2015*	Yes	Yes	No	Yes	Yes	Yes	Yes	Yes	Yes
Hardy 2016	Yes	N/a	No	Yes	Unclear	Yes	Yes	Yes	Yes
Pearce 2016	No	N/a	No	Yes	N/a	Yes	No	Partial	Partial
Perona Garcelan 2012	Yes	N/a	Unclear	No	Yes	Yes	Partial	Yes	Yes

Perona Garcelan 2014	Partial	N/a	No	Partial	N/a	Yes	No	Yes	Yes
Powers 2016	No	N/a	Yes	Yes	N/a	Yes	Yes	Unclear	Yes
Thompson 2016	Yes	N/a	Unclear	Partial	Yes	Yes	No	Partial	Partial
Varese 2012	Yes	Partial	No	Yes	Yes	Yes	Unclear	Yes	Yes

*Narrative inclusion only – did not contribute to GRADE

Table 2.3 - Quality ratings – Cognitive beliefs and appraisals

Study	Unbiased selection of cohort	Minimises baseline differences	Adequate sample size/power	Adequate description of cohort	Valid psychosis measure	Valid measures of variables	Controls for confounders	Few instances of missing data/ adequate handling	Analytical methods appropriate
Appiah-Kusi 2017	Yes	N/a	No	Yes	Yes (UHR)	Yes	Yes	Yes	Partial
Ashford 2010	No	N/a	Yes	Yes	N/a	Yes	Yes	Yes	Yes
Evans 2015*	Yes	Partial	No	Yes	Yes	Yes	Yes	Yes	Partial
Fisher 2012	Unclear	N/a	No	Yes	N/a	Yes	Yes	Unclear	Yes
Gaweda 2018 (a)	Partial	N/a	Yes	Partial	N/a	Yes	Yes	Yes	Yes
Gaweda 2018 (b)	Partial	N/a	Yes	Partial	N/a	Yes	Yes	Yes	Yes
Goldstone 2011*	Partial	No	No	Yes	Yes	Yes	Unclear	Yes	Yes
Goldstone 2012*	Partial	No	No	Yes	Yes	Yes	Unclear	Yes	Yes
Hardy 2016	Yes	N/a	No	Yes	Unclear	Yes	Yes	Yes	Yes

Jaya 2017	Partial	N/a	Yes	Partial	N/a	Yes	Unclear	Unclear	Yes
Morgan 2014	Yes	No	Yes	Yes	Unclear	Yes	Yes	Yes	Yes
Østefjells, 2017	Yes	N/a	Yes	Partial	Yes	Yes	Yes	Yes	Yes
Wickham 2016	Partial	No	Unclear	Yes	Yes	Yes	Yes	Yes	Yes

*Narrative inclusion only – did not contribute to GRADE

Table 2.4 - Quality ratings – Attachment style

Study	Unbiased selection of cohort	Minimises baseline differences	Adequate sample size/power	Adequate description of cohort	Valid psychosis measure	Valid measures of variables	Controls for confounders	Few instances of missing data/ adequate handling	Analytical methods appropriate
Ashford 2010	No	N/a	Yes	Yes	N/a	Yes	Yes	Yes	Yes
Goodall 2015	Partial	N/a	Yes	Yes	N/a	Yes	Yes	Yes	Yes
McDonnell 2018	Yes	N/a	Unclear	Yes	Yes	Yes	Unclear	Yes	Yes
Pearce 2016	Yes	N/a	No	Yes	N/a	Yes	No	Partial	Partial
Pilton 2016	Yes	N/a	No	Yes	Yes	Yes	Unclear	Yes	Partial
Shienbaum 2014	No	N/a	Yes	Partial	N/a	Yes	No	Yes	Yes
Shienbaum 2015	Partial	N/a	No	Partial	N/a	Yes	Yes	Unclear	Yes
Sitko 2014	Yes	N/a	Unclear	No	N/a	Yes	Yes	Unclear	Yes
VanDam 2014*	Unclear	No	No	Partial	Yes	Yes	Yes	Unclear	Yes

*Narrative inclusion only – did not contribute to GRADE

Table 2.5 - Quality ratings – Mood & anxiety

Study	Unbiased selection of cohort	Minimises baseline differences	Adequate sample size/power	Adequate description of cohort	Valid psychosis measure	Valid measures of variables	Controls for confounders	Few instances of missing data/ adequate handling	Analytical methods appropriate
Ashford 2010	No	N/a	Yes	Yes	N/a	Yes	Yes	Yes	Yes
Bebbington 2011	Yes	Unclear	Yes	No	Partial	Yes	Yes	Partial	Yes
Fisher 2012	Unclear	N/a	No	Yes	N/a	Yes	Yes	Unclear	Yes
Marwaha 2014	Yes	N/a	Yes	No	Yes	Partial	Yes	Yes	Yes
Marwaha & Bebbington 2015	Yes	N/a	Yes	No	Partial	Yes	Yes	Unclear	Yes
Østefjells, 2017	Yes	N/a	Yes	Partial	Yes	Yes	Yes	Yes	Yes
Thompson 2016	Yes	N/a	Unclear	Partial	Yes	Yes	No	Partial	Partial

Table 2.6 - Quality ratings – Emotion regulation and stress sensitivity

Study	Unbiased selection of cohort	Minimises baseline differences	Adequate sample size/power	Adequate description of cohort	Valid psychosis measure	Valid measures of variables	Controls for confounders	Few instances of missing data/ adequate handling	Analytical methods appropriate
Lincoln 2017	Partial	N/a	Yes	Yes	N/a	Yes	Yes	Yes	Yes
Perona Garcelan 2014	Partial	N/a	No	Partial	N/a	Yes	No	Yes	Yes
Rossler 2016	Yes	N/a	Yes	Partial	N/a	Partial	Yes	Unclear	Yes
van Nierop 2014	Yes	Yes	Yes	Yes	Yes	Partial	Yes	Unclear	Yes
Goldstone 2011*	Partial	No	No	Yes	Yes	Yes	Unclear	Yes	Yes
Goldstone 2012*	Partial	No	No	Yes	Yes	Yes	Unclear	Yes	Yes

*Narrative inclusion only – did not contribute to GRADE

Table 2.7 - Quality ratings - Social

Study	Unbiased selection of cohort	Minimises baseline differences	Adequate sample size/power	Adequate description of cohort	Valid psychosis measure	Valid measures of variables	Controls for confounders	Few instances of missing data/ adequate handling	Analytical methods appropriate
Boyda & McFeeters 2015	Yes	N/a	Yes	Partial	Yes	Partial	Yes	Yes	Yes
Jaya 2017	Partial	N/a	Yes	Partial	N/a	Yes	Unclear	Unclear	Yes
Morgan 2014	Yes	No	Yes	Yes	Unclear	Yes	Yes	Yes	Yes
van Nierop 2014	Yes	Yes	Yes	Yes	Yes	Partial	Yes	Unclear	yes

2.7 4 Category results

2.7.4.1 PTSD symptoms and dissociation

The majority of samples in this category included clinical (N=1108) or high-risk (N=536) groups with fewer sub-clinical participants than other categories (N=569). Effect size estimates were generally trivial to small with narrow confidence intervals suggesting precision. Study quality varied from very low to moderate, with the majority of studies being of low quality. Overall the category was downgraded in the GRADE assessment for risk of bias (see Table 2.14).

As Figure 2.6 shows this category has the highest frequency of studies with non-trivial effect sizes (a combination of both significant and non-significant results), however the effects must be carefully considered due to the poor quality of the studies.

The strongest evidence came from Varese (2012) where significant pathways from sexual abuse and total trauma to hallucination-proneness were mediated by dissociation in the clinical group (sexual abuse CSIE=0.32, 95% CI 0.13-0.54 total trauma: CSIE-0.31, 95% CI 0.17-0.49). These effects are approaching moderate size and are based on moderate-low quality evidence. Small significant effects were detected in their combined sample for these predictors, as well as neglect and emotional abuse (see Table 2.8). Further clinical studies found dissociation to mediate between trauma and aberrant salience (Choi, 2017, moderate quality evidence) and both voices and paranoia (Pearce, 2012, very low quality evidence) but effects were of small magnitude. However Thompson (2016) did not find dissociation to significantly mediate between childhood sexual abuse and transition to psychosis in a UHR population (OR=0.99, 95%CI 0.97-1.01) but this was a null effect size based on very low quality evidence.

When the individual components of dissociation were tested, depersonalisation along with dissociation total score mediated between trauma and hallucinations (clinical sample, Perona Garcelan 2012) and hallucination-proneness (sub-clinical sample, Perona Garcelan 2014). Absorption also mediated this pathway in the sub-clinical sample but all effects were small

and based on low and very low quality evidence. In a further sub-clinical sample dissociation total score and absorption were found to mediate between both trauma and delusions, but depersonalisation did not (Cole, 2016). Absorption, but not depersonalisation also mediated trauma and schizotypy in an at-risk sample (Berenbaum 2008). All effects were small and based on low to very low quality evidence. Dissociative amnesia had a small inverse relationship with delusions in a clinical sample (Perona Garcelan 2012) and in a sub-clinical sample with both delusions and hallucinations (Cole, 2016) however effect sizes were small and did not reach significance.

Childhood sexual abuse and hallucinations were mediated by both post-traumatic hyperarousal (OR=1.44, 95%CI 1.00-2.06) and post-traumatic avoidance and numbing (OR 1.48, 95%CIs 1.02-2.13) in a clinical sample, but intrusive trauma memory was not (Hardy 2016). These were small but precise effects as interpreted at both the 1% and 10% incidence rates (see section 2.6.7 above) and were based on moderate-low quality evidence

Further clinical evidence showed childhood abuse and current psychosis were significantly mediated by PTSD (OR 1.90, 95%CIs 1.13-3.19) (Powers 2016, low quality); and trauma and persecutory delusions were significantly mediated by post-traumatic stress symptoms (CSIE=0.13, 95%CI 0.06-0.22) (Choi 2017, moderate quality). However an earlier study found no significant mediation effect by post-traumatic stress symptoms between childhood abuse and psychotic symptoms (Choi 2015, low quality). Full results can be found in Table 2.8 and Figures 2.7 and 2.8.

2.7.4.2 Cognitive beliefs and appraisals

In this category, the majority of participants came from community samples (N=4555) however there was a relatively large clinical population (N=1058) and a small UHR group (N=30). Across the category, effects were consistently trivial to small, and confidence intervals were narrow suggesting precision (see Table 2.9 and Figures 2.9 and 2.10). Quality ranged from very low to high, with the majority of studies being rated as moderate. This category was rated

as high quality using the GRADE criteria as mediators were consistently defined, missing data was transparently handled and confounders were well controlled.

Figure 2.6 clearly shows this category as having the largest proportion of better quality studies, but these yield a combination of trivial and non-trivial effect sizes. Despite some of the effects being of very small magnitude, there are comparatively few effects based on poor quality evidence.

Research in this category largely focused on paranoia as an outcome, with emotional abuse or neglect as the predictor. There was also greater similarity in the mediators being investigated. As a result this category was more consistent than the others.

In a high risk sample, Appiah-Kusi (2017) found a significant path from emotional neglect to paranoid ideation through negative self beliefs (CSIE=0.18, 95%CIs 0.06-0.33). This was a small effect based on moderate quality evidence. Wickham (2016) also found emotional neglect and suspiciousness to be significantly mediated by personal beliefs in a just world (CSIE=0.09, 95%CI 0.03-0.20). This was based on moderate-low quality evidence. Hardy (2016) found negative beliefs about others acted as a mediator between childhood emotional abuse and persecutory delusions (OR 1.36, 95%CIs 1.04-1.77), based on moderate-low quality evidence. Østefjells, (2017) also found an effect for metacognitive beliefs, in a multiple mediator model with mood factors which significantly mediated between emotional abuse and positive symptoms (partially standardised indirect effect 0.05, 95%CI 0.02-0.10), based on high quality evidence. Both of these were null to small effect sizes, detected in clinical samples. These four studies taken together, suggest that emotional trauma and cognitive factors are important in the development of positive symptoms, particularly paranoia, in clinical populations.

Gaweda (2018 a&b) found cognitive biases to significantly mediate between traumatic life events and psychosis-proneness (CSIE=0.22, 95%CIs 0.13-0.30); and to significantly mediate along with self disorders between traumatic life events and psychotic-like experiences

(CSIE=0.24, 95%CIs 0.20-0.28). These effects were of small magnitude, detected in non-clinical samples, based on moderate-high quality evidence. Further sub-clinical evidence was mixed with few significant effects (Fisher, 2012; Evans, 2015; Ashford, 2010) suggesting cognitive mediators may be less salient in subclinical populations.

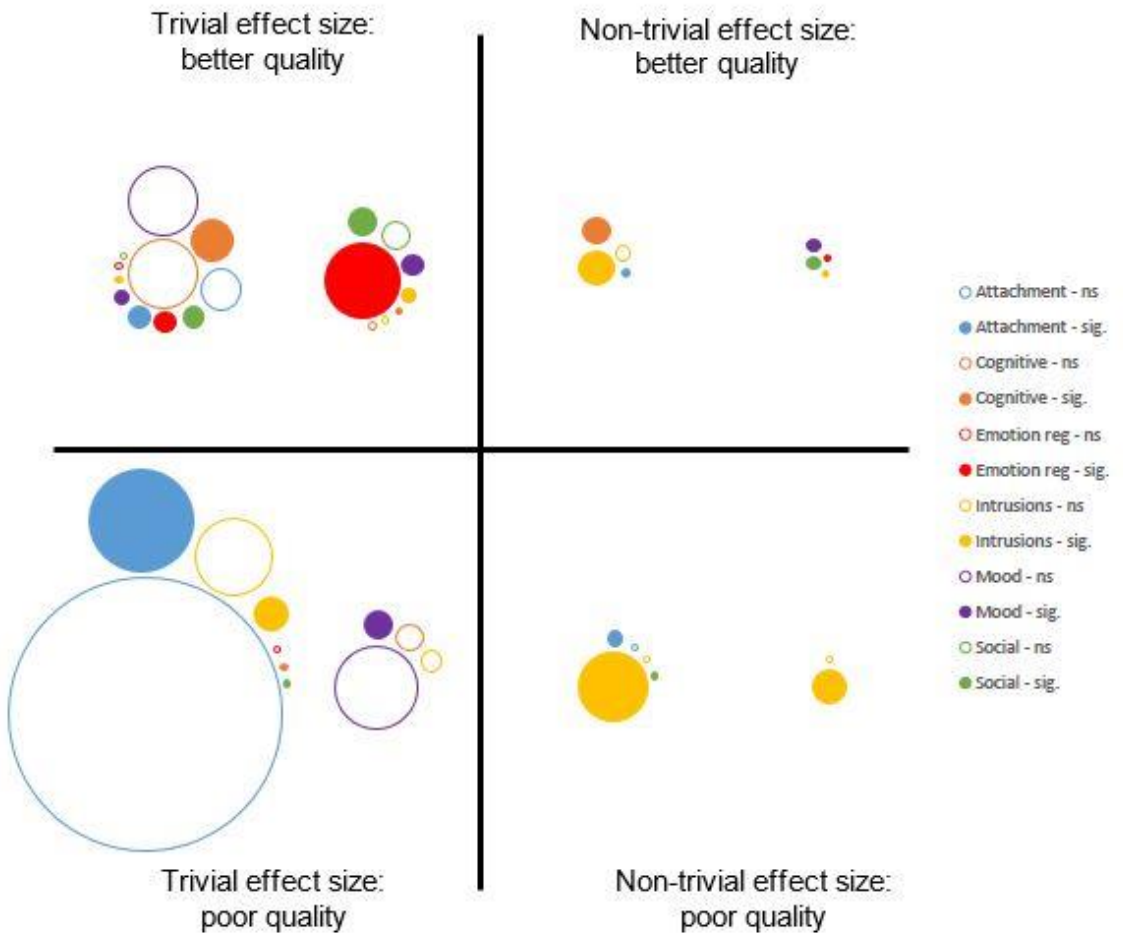


Figure 2.5 - Effect size and quality of CSIE and OR estimates of mediation. The size of each circle denotes number of effect sizes falling in this category.

Note: CSIEs are on left-hand side of each quadrant, with ORs on the right.

2.7.4.3 Attachment style

In this category samples were derived from various populations including small clinical (N=308) and at-risk populations (N=64) and one very large community sample from the National Comorbidity Survey in the USA (Sitko, 2014, N=5877) which comprised the majority

(78%) of participants in this category. Most effects were null to small with few exceptions. Estimates of small-moderate magnitude had confidence intervals amongst the widest in the category, suggesting imprecision (see Table 2.10 and Figure 2.11). Study quality ranged from very low to high and the outcome was downgraded in the GRADE assessment for overall risk of bias mainly due to sample bias and lack of transparency around data handling; and inconsistency due to heterogeneity across the category.

As indicated in Figure 2.6 significant effects are of trivial size and better quality studies often found non-significant mediation effects. Although studies in clinical samples found some of the larger effects, these were of small magnitude and are compounded by their lack of study quality and small sample sizes. There is a degree of consistency throughout a number of the detected effects which suggests attachment is not an important mediator between trauma and psychosis.

The best evidence in this category comes from McDonnell (2016) who identified a pathway from secondary school bullying to paranoid ideation through interpersonal sensitivity in a clinical high risk group (CSIE=0.18, 95% CIs 0.05 – 0.37), based on moderate quality evidence. However the confidence intervals are notably wider than for many of the other estimates[3]. The variables are also unique to this study, this is the only high risk sample, and the sample size is small. Narratively included evidence found attachment style to mediate between childhood maltreatment and the severity of positive symptoms in clinical participants and their siblings, however the mediation effect was notably stronger when negative symptoms were the outcome variable (Van Dam, 2014).

A high quality sub-clinical study found marginal effects for anxious and avoidant attachment mediating between emotional abuse and schizotypy (anxious CSIE= 0.06, 95%CI 0.02-0.11; avoidant CSIE= 0.04, 95%CI 0.01-0.08) (Goodall, 2015), and moderate quality evidence from

[3] It should be noted that 95%CI values were derived by applying the ratio of standardised:unstandardised beta to unstandardised 95%CI values provided in the text. The paper indicates that significant effects were found, but the computed CIs contain zero indicating non-significance. These values should be interpreted with caution as they are approximate.

Ashford (2010) found trivial effects for verbal and physical aggression being mediated by rejection sensitivity in the development of subclinical delusions of reference and persecution in a student sample. Similarly Sitko (2014) found anxious and avoidant attachment to mediate only 1% and 3% of variance respectively in a model with trauma and hallucinations. This study had a large subclinical sample (N=5877) so it is notable that only null effects were found, however the traumatic events were rare (incidence range 2.5-8.8%) and study quality was low[4].

2.7.4.4 Mood and anxiety

Samples in this category were moderate to large and were almost exclusively drawn from the general population (N=14,081), with one clinical (N= 261) and one high-risk group (N=233). Almost half of the studies used sub-samples from the Adult Psychiatric Morbidities Survey (APMS), 2000 and 2007 (Bebbington, 2011; Marwaha, 2014; Marwaha, 2015) which comprised 94% of the total participants in this category. Quality ranged from very low to high and the category was downgraded for imprecision and risk of bias in the GRADE assessment (see Table 2.14).

The majority of effects were null to small and had narrow confidence intervals suggesting reasonable precision. Most of the effects in this category fell into the trivial effect size, better quality quadrant of Figure 2.6. Some of the direct and total effects from the studies using APMS sub-samples were moderate to large, but the indirect effects remained small. Confidence intervals for these effects were wide suggesting a lack of precision. See Table 2.11.

Marwaha (2014) found mood instability to mediate between childhood sexual abuse and probable psychosis (OR= 2.30, 95%CI 1.60-3.29) with elevated odds ratios for hallucinations (OR=1.44, 95%CIs 1.23-1.63) and paranoia (OR=1.63, 95%CIs 1.37-1.93). All effects were

[4] It should be noted that effect sizes and 95%CIs were derived using ratio of standardised:unstandardised effects due to lack of provision of data in the paper. This is described in detail in appendix 9**,** but 95%CIs values are very approximate and should be interpreted as such.

significant but small and based on moderate quality evidence, however mood instability was measured using only a single questionnaire item. There was a slightly larger reported effect for childhood sexual abuse (intercourse) and psychosis being dual-mediated by anxiety and depression (OR=2.41 95%CIs 1.61-3.61) however this was based on low quality evidence (Marwaha, 2015).

In the only clinical sample, high quality evidence found a small significant mediation pathway through anxiety and depression to positive symptoms in a multiple mediator model with metacognitive beliefs (partially standardised effect 0.05, 95%CI 0.02-0.10) (Østefjells, 2017).

2.7.4.5 Emotion regulation and stress sensitivity

In this category samples were predominantly drawn from the general population (N= 1833), with only one study which included clinical (N=143) and high-risk groups (extended psychosis phenotype, N=384).One study used a longitudinal design (Lincoln, 2017), all other evidence was cross sectional.

Although a number of the effects in this category were significant, they were all of null to small magnitude (see Figure 2.6). Confidence intervals were narrow, suggesting precision. Study quality varied, from low to high, but the majority of studies were of higher quality and the GRADE rating for this category was high.

High quality evidence found very small effects for emotion regulation mediating between childhood trauma and overall distress (CSIE=0.003, 95%CIs 0.00-0.01) paranoia-related distress (CSIE=0.02, 95%CIs 0.00-0.04) and paranoia frequency (CSIE=0.02, 95%CIs 0.01-0.03) (Lincoln, 2017). Similarly Van Nierop (2014) found a path from childhood trauma to extended psychosis phenotype group membership to be mediated by affect regulation (CSIE 0.04, 95%CIs 0.02-0.07) in a high risk sample, based on moderate quality evidence. See Table 2.12and Figures 2.14 and 2.15 for full results.

2.7.4.6 Social defeat

Samples in this category were moderate to large and included clinical (N=433), high risk (N=384) and non-clinical (N=10,144) groups. There were comparatively few studies in this area, and all effects were null to small. Confidence intervals suggested effect estimates were precise. See Table 2.13 for full results. Most effects fall into the trivial effect size and better quality quadrant in Figure 2.6.

The strongest evidence comes from Boyda (2015) for an indirect effect for loneliness between emotional neglect and psychotic-like experiences (OR=1.89, 95%CIs 1.41-2.54) and a smaller effect when sexual abuse was entered as the predictor (OR=1.37, 95%CIs 1.12-1.66). Both effects were significant and based on moderate-high quality evidence, however there was a negative effect of loneliness in a model from social adversity to positive symptoms (CSIE= -0.09, 95%CIs -0.13 - -0.06) (Jaya, 2017). This is a trivial effect based on low quality evidence in a small sample but it is derived from a specific loneliness scale (UCLA Loneliness scale, v1), whereas the loneliness data in Boyda (2015) was collected using a single questionnaire item. Full results can be found in Table 2.13 and Figures 2.16 and 2.17.

Table 2.8 Results – PTSD symptoms and dissociation

Study ref	N	Population	Predictor	Mediator(s)	Outcome	Effect size(s)	95% CIs (lower, upper)	Effect type	Quality	Forest plot reference
Choi 2015	126	Severe clinical group	Childhood abuse	Post-traumatic stress symptoms	Psychotic symptoms	0.172	-0.023, 0.366	CSIE	Low	Choi 2015 1.
Choi 2017	169	Clinical	Trauma	Post-traumatic stress symptoms	Persecutory ideation	0.13	0.06, 0.22	CSIE	Moderate	Choi 2017 1.
	169	Clinical	Trauma	Dissociation	Aberrant experience	0.13	0.02, 0.23	CSIE	Moderate	Choi 2017 2.
Cole 2016	200	General (students)	Childhood maltreatment	Dissociation (total model)	Hallucinations	0.224	0.122, 0.362	CSIE	Very low	Cole 2016 1.
	200	General (students)	Childhood maltreatment	Dissociative amnesia	Hallucinations	-0.075	-0.182, 0.002	CSIE	Very low	Cole 2016 2.
	200	General (students)	Childhood maltreatment	Absorption	Hallucinations	0.196	0.114, 0.311	CSIE	Very low	Cole 2016 3.
	200	General (students)	Childhood maltreatment	Depersonalisation	Hallucinations	0.055	-0.070, 0.197	CSIE	Very low	Cole 2016 4.
	200	General (students)	Childhood maltreatment	Cambridge depersonalisation scale	Hallucinations	0.120	-0.002, 0.262	CSIE	Very low	Cole 2016 5.
	200	General (students)	Childhood maltreatment	Dissociation (total model)	Delusional ideation	0.208	0.114, 0.340	CSIE	Very low	Cole 2016 6.
	200	General (students)	Childhood maltreatment	Dissociative amnesia	Delusional ideation	-0.071	-0.183, -0.001	CSIE	Very low	Cole 2016 7.
	200	General (students)	Childhood maltreatment	Absorption	Delusional ideation	0.139	0.055, 0.258	CSIE	Very low	Cole 2016 8.
	200	General (students)	Childhood maltreatment	Depersonalisation	Delusional ideation	0.122	-0.014, 0.345	CSIE	Very low	Cole 2016 9.
	200	General (students)	Childhood maltreatment	Cambridge depersonalisation scale	Delusional ideation	0.085	-0.057, 0.250	CSIE	Very low	Cole 2016 10.
Pearce 2017	112	Clinical	Childhood trauma	Dissociation	Voices	0.189	0.071, 0.401	CSIE	Very low	Pearce 2017 1.

Study ref	N	Population	Predictor	Mediator(s)	Outcome	Effect size(s)	95% CIs (lower, upper)	Effect type	Quality	Forest plot reference
	112	Clinical	Childhood trauma	Dissociation & fearful attachment	Voices	0.283	0.132, 0.497	CSIE	Very low	Pearce 2017 2.
	112	Clinical	Childhood trauma	Dissociation	Paranoia	0.249	0.108, 0.465	CSIE	Very low	Pearce 2017 3.
	112	Clinical	Childhood trauma	Dissociation & fearful attachment	Paranoia	0.356	0.186, 0.573	CSIE	Very low	Pearce 2017 4.
Perona Garcelan 2012	71	Clinical	Childhood trauma	Dissociation (simple model)	Hallucinations	0.187	0.080, 0.339	CSIE	Low	Perona Garcelan 2012 1.
	71	Clinical	Childhood trauma	Dissociation (simple model)	Delusions	0.070	0.000, 0.209	CSIE	Low	Perona Garcelan 2012 2.
	71	Clinical	Childhood trauma	Dissociative amnesia	Hallucinations	0.009	-0.136, 0.088	CSIE	Low	Perona Garcelan 2012 3.
	71	Clinical	Childhood trauma	Absorption	Hallucinations	-0.0178	-0.105, 0.176	CSIE	Low	Perona Garcelan 2012 4.
	71	Clinical	Childhood trauma	Depersonalisation	Hallucinations	0.170	0.046, 0.344	CSIE	Low	Perona Garcelan 2012 5.
	71	Clinical	Childhood trauma	Dissociative amnesia	Delusions	-0.020	-0.141, 0.071	CSIE	Low	Perona Garcelan 2012 6.
	71	Clinical	Childhood trauma	Absorption	Delusions	0.040	-0.091, 0.246	CSIE	Low	Perona Garcelan 2012 7.
	71	Clinical	Childhood trauma	Depersonalisation	Delusions	0.050	-0.041, 0.190	CSIE	Low	Perona Garcelan 2012 8.
Perona Garcelan 2013	318	Community (students)	Childhood trauma total	Depersonalisation	Hallucination proneness	0.088	0.039, 0.151	CSIE	Very Low	Perona Garcelan 2013 1.
	318	Community (students)	Childhood trauma total	Absorption	Hallucination proneness	0.037	0.007, 0.093	CSIE	Very Low	Perona Garcelan 2013 2.
Varese 2012	65	Combined sample	Childhood trauma total	Dissociative tendencies	Hallucination proneness	0.258	0.129, 0.480	CSIE	Moderate-low	Varese 2012 1.

Study ref	N	Population	Predictor	Mediator(s)	Outcome	Effect size(s)	95% CIs (lower, upper)	Effect type	Quality	Forest plot reference
	65	Combined sample	Sexual abuse	Dissociative tendencies	Hallucination proneness	0.244	0.090, 0.401	CSIE	Moderate-low	Varese 2012 2.
	65	Combined sample	Physical abuse	Dissociative tendencies	Hallucination proneness	0.161	-0.017, 0.347	CSIE	Moderate-low	Varese 2012 3.
	65	Combined sample	Neglect	Dissociative tendencies	Hallucination proneness	0.238	0.101, 0.385	CSIE	Moderate-low	Varese 2012 4.
	65	Combined sample	Emotional abuse	Dissociative tendencies	Hallucination proneness	0.218	0.086, 0.365	CSIE	Moderate-low	Varese 2012 5.
	45	Clinical sample	Childhood trauma total	Dissociative tendencies	Hallucination proneness	0.314	0.171, 0.485	CSIE	Moderate-low	Varese 2012 6.
	45	Clinical sample	Sexual abuse	Dissociative tendencies	Hallucination proneness	0.315	0.132, 0.535	CSIE	Moderate-low	Varese 2012 7.
	45	Clinical sample	Physical abuse	Dissociative tendencies	Hallucination proneness	0.073	-0.180, 0.329	CSIE	Moderate-low	Varese 2012 8.
	45	Clinical sample	Neglect	Dissociative tendencies	Hallucination proneness	0.160	-0.037, 0.344	CSIE	Moderate-low	Varese 2012 9.
	45	Clinical sample	Emotional abuse	Dissociative tendencies	Hallucination proneness	0.170	-0.014, 0.381	CSIE	Moderate-low	Varese 2012 10.
Hardy 2016	118	Clinical	Sexual abuse	Intrusive trauma memory	Auditory hallucinations (absence vs presence)	indirect= 1.20 direct = 2.44 total = 2.39	0.925, 1.559 0.980, 6.065 1.145, 7.489	OR	Moderate-low	Hardy 2016 1.
	118	Clinical	Sexual abuse	Post-traumatic avoidance & numbing	Auditory hallucinations (absence vs presence)	indirect= 1.48 direct = 2.05 total = 3.03	1.020, 2.132 0.809, 5.206 1.151, 7.952	OR	Moderate-low	Hardy 2016 2.
	118	Clinical	Sexual abuse	Post-traumatic hyperarousal	Auditory hallucinations (absence vs presence)	indirect= 1.44 direct = 2.10 total = 3.03	1.003, 2.064 0.826, 5.359 1.15, 7.791	OR	Moderate-low	Hardy 2016 3.
Thompson 2016	233	Ultra- high risk	Sexual abuse	CAARMS-Dissociation	Transition to psychosis	indirect = 0.99 direct = 1.09 total = 1.08	0.97, 1.01 0.99, 1.19 0.98, 1.18	OR	Very low	Thompson 2016 1.

Study ref	N	Population	Predictor	Mediator(s)	Outcome	Effect size(s)	95% CIs (lower, upper)	Effect type	Quality	Forest plot reference
Powers 2016	328	Clinical	Childhood abuse	PTSD	Current psychosis	indirect =1.896 direct = 1.840 total = 3.525	1.128, 3.190 0.546, 6.204 1.176, 10.566	OR	Low	Powers 2016 1.
Berenbaum 2008	142	High odd beliefs men (subsample)	Childhood maltreatment	Absorption	Schizotypal symptoms	Indirect = 2.10 Direct = 5.23 Total = 6.90	1.14, 3.86 1.27, 4.74 3.77, 12.62	OR	Very low	Berenbaum 2008 1.
	142	High odd beliefs men (subsample)	Childhood maltreatment	Dissociation	Schizotypal symptoms	Indirect = 1.33 Direct = 6.29 Total = 6.90	0.73, 2.42 1.80, 7.67 3.77, 12.62	OR	Very low	Berenbaum 2008 2.
	142	High odd beliefs men (subsample)	Childhood maltreatment	Lifetime PTSD	Schizotypal symptoms	Indirect = 1.27 Direct = 6.29 Total = 6.90	0.70, 2.31 3.33, 22.17 3.77, 12.62	OR	Very low	Berenbaum 2008 3.
	142	High odd beliefs men (subsample)	Childhood maltreatment	Current PTSD	Schizotypal symptoms	Indirect = 2.03 Direct = 5.23 Total = 6.90	1.11, 3.74 2.31, 11.31 3.77, 12.62	OR	Very low	Berenbaum 2008 4.
	171	High odd beliefs women (subsample)	Childhood maltreatment	Absorption	Schizotypal symptoms	Indirect = 2.15 Direct = 1.96 Total = 2.55	1.21, 3.81 1.23, 4.16 1.45, 4.49	OR	Very low	Berenbaum 2008 5.
	171	High odd beliefs women (subsample)	Childhood maltreatment	Dissociation	Schizotypal symptoms	Indirect = 1.70 Direct = 2.24 Total = 2.55	0.97, 3.00 1.00, 3.24 1.45, 4.49	OR	Very low	Berenbaum 2008 6.
	171	High odd beliefs women (subsample)	Childhood maltreatment	Lifetime PTSD	Schizotypal symptoms	Indirect = 2.03 Direct = 2.32 Total = 2.55	1.15, 3.59 1.52, 5.45 1.45, 4.49	OR	Very low	Berenbaum 2008 7.
	171	High odd beliefs women (subsample)	Childhood maltreatment	Current PTSD	Schizotypal symptoms	Indirect = 2.39 Direct = 2.02 Total = 2.55	1.34, 4.25 1.82, 6.95 1.45, 4.49	OR	Very low	Berenbaum 2008 8.

Figure 2.7 – Forest plot – completely standardised indirect effects - PTSD symptoms and dissociation

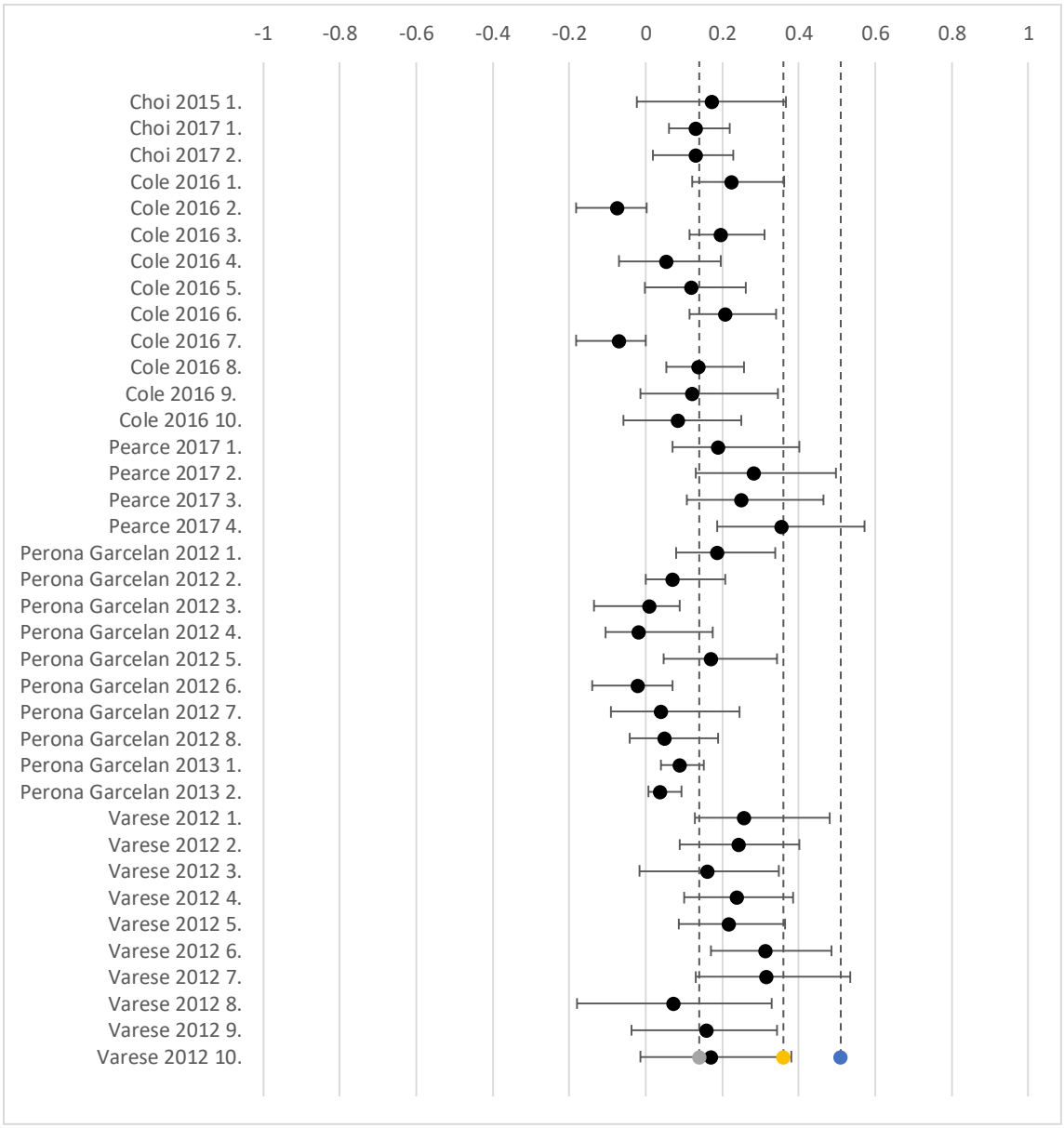


Figure 2.8 – Forest plot – odds ratios – PTSD symptoms and dissociation

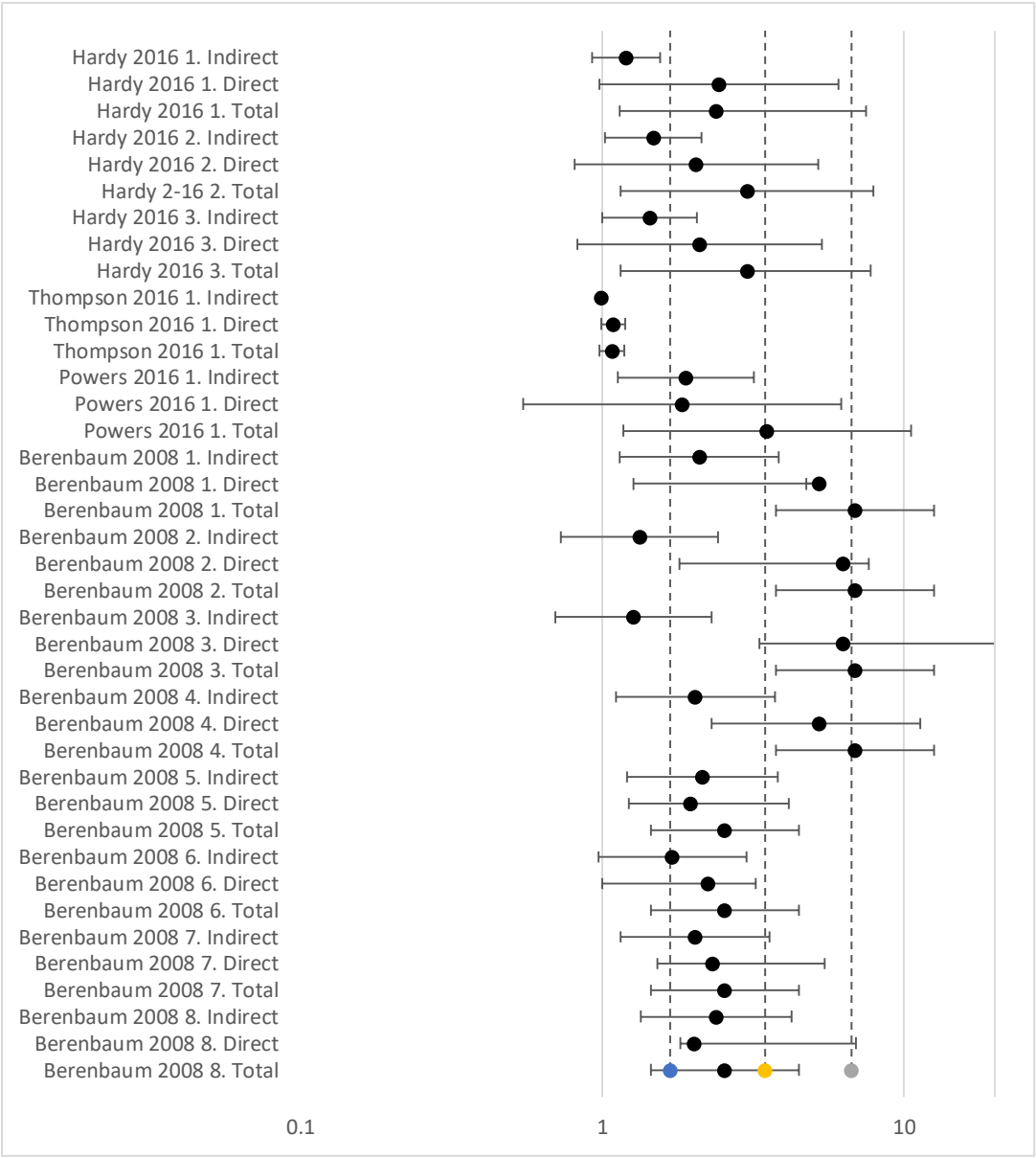


Table 2.9 Results - Cognitive beliefs and appraisals

Study ref	N	Population	Predictor	Mediator(s)	Outcome	Effect size(s)	95% CIs (lower, upper)	Effect type	Quality	Forest plot reference
Østefjells 2017		Clinical	Childhood trauma	Metacognitive beliefs, anxiety, depression	Positive symptoms	0.046	0.020, 0.100	CSIE	High	Østefjells 2017 1.
Appiah-Kusi 2017	30	Ultra-high risk	Emotional neglect	Negative self-schema	Paranoid ideation	0.176	0.057, 0.332	CSIE	Moderate	Appiah-Kusi 2017 1.
Ashford 2010	135	General (students)	Indirect aggression	Negative self beliefs	Ideas of social reference	0.0780	0.014, 0.202	CSIE	Moderate	Ashford 2010 1.
	135	General (students)	Indirect aggression	Negative beliefs about others	Ideas of social reference	-0.0004	-0.048, 0.070	CSIE	Moderate	Ashford 2010 2.
	135	General (students)	Direct verbal aggression	Negative self beliefs	Ideas of social reference	-0.0221	-0.125, 0.022	CSIE	Moderate	Ashford 2010 3.
	135	General (students)	Direct verbal aggression	Negative beliefs about others	Ideas of social reference	0.0808	0.019, 0.170	CSIE	Moderate	Ashford 2010 4.
	135	General (students)	Direct physical aggression	Negative self beliefs	Ideas of social reference	0.0016	-0.044, 0.060	CSIE	Moderate	Ashford 2010 5.
	135	General (students)	Direct physical aggression	Negative beliefs about others	Ideas of social reference	0.0471	-0.009, 0.116	CSIE	Moderate	Ashford 2010 6.
	135	General (students)	Indirect aggression	Negative self beliefs	Persecution	0.1085	0.020, 0.302	CSIE	Moderate	Ashford 2010 7.
	135	General (students)	Indirect aggression	Negative beliefs about others	Persecution	-0.0002	-0.038, 0.046	CSIE	Moderate	Ashford 2010 8.
	135	General (students)	Direct verbal aggression	Negative self beliefs	Persecution	-0.0308	-0.179, 0.029	CSIE	Moderate	Ashford 2010 9.
	135	General (students)	Direct verbal aggression	Negative beliefs about others	Persecution	0.0484	-0.004, 0.154	CSIE	Moderate	Ashford 2010 10.

	135	General (students)	Direct physical aggression	Negative self beliefs	Persecution	0.0022	-0.054, 0.093	CSIE	Moderate	Ashford 2010 11.
	135	General (students)	Direct physical aggression	Negative beliefs about others	Persecution	0.0283	-0.004, 0.095	CSIE	Moderate	Ashford 2010 12.
Jaya 2017	2350	General (community)	Social adversity	Negative schemas	Positive symptoms	0.037	0.000 – 0.074	CSIE	Moderate-high	Jaya 2017 1.
Wickham & Bentall 2016	50	Clinical group	Emotional neglect	General beliefs in a just world	Suspiciousness	0.037	-0.044 – 0.118	CSIE	Moderate-low	Wickham 2016 1.
	50	Clinical group	Emotional neglect	Personal beliefs in a just world	Suspiciousness	0.090	0.025 – 0.204	CSIE	Moderate-low	Wickham 2016 2.
Gaweda 2018 (1)	650	General (students)	Traumatic life events	Self disorders	Psychotic-like experiences	0.151	0.061, 0.240	CSIE	Moderate-high	Gaweda 2018a 1.
	650	General (students)	Traumatic life events	Cognitive biases	Psychotic-like experiences	0.024	0.010, 0.038	CSIE	Moderate-high	Gaweda 2018a 2.
	650	General (students)	Traumatic life events	Self disorders, cognitive biases, anxious attachment	Psychotic-like experiences	0.242	0.196, 0.284	CSIE	Moderate-high	Gaweda 2018a 3.
Gaweda 2018 (2)	653	General (students)	Traumatic life events	Self disturbances	Psychosis-proneness	0.089	0.054, 0.126	CSIE	Moderate-high	Gaweda 2018b 1.
	653	General (students)	Traumatic life events	Cognitive biases	Psychosis-proneness	0.217	0.131, 0.303	CSIE	Moderate-high	Gaweda 2018b 2.
Hardy 2016	190	Clinical	Emotional abuse	Negative other beliefs	Auditory hallucinations (absence vs presence)	indirect= 1.18 direct = 2.34 total = 2.76	0.973-1.429 1.097-5.003 1.274-5.992	OR	Moderate-low	Hardy 2016 1.
	190	Clinical	Emotional abuse	Negative other beliefs	Persecutory delusions (absence vs presence)	indirect= 1.36 direct = 1.89 total = 2.57	1.041-1.774 0.940-3.795 1.229-5.366	OR	Moderate-low	Hardy 2016 2.
	190	Clinical	Emotional abuse	Negative other beliefs	Delusions of reference	indirect= 1.18 direct = 1.95 total = 2.303	0.988-1.412 0.998-3.812	OR	Moderate-low	Hardy 2016 2.

					(absence vs presence)		1.171-4.529			
Morgan 2014	240 295	Clinical Controls	Parental separation	Self esteem	Unaffected vs psychosis	Indirect – 1.01 direct = 1.6 total = 6.41	0.96 – 1.08 0.97-2.65 2.44-16.86	OR	Moderate-low	Morgan 2014 1.
	390 391	Clinical Controls	Parental separation	No qualifications & self esteem	Unaffected vs psychosis	Indirect = 0.98 direct = 1.6 total = 6.41	0.92 – 1.05 0.97-2.65 2.44-16.86	OR	Moderate-low	Morgan 2014 2.
	390 391	Clinical Controls	Parental separation	Adult disadvantage & self esteem	Unaffected vs psychosis	Indirect = 1.01 direct = 1.6 total = 6.41	0.99 – 1.02 0.97-2.65 2.44-16.86	OR	Moderate-low	Morgan 2014 3.
	390 391	Clinical Controls	Parental separation	No qualifications, adult disadvantage & self esteem	Unaffected vs psychosis	Indirect = 1.02 direct = 1.6 total = 6.41	1.00 – 1.05 0.97-2.65 2.44-16.86	OR	Moderate-low	Morgan 2014 4.
Fisher 2012	212	General (community)	Emotional abuse	Negative self-beliefs	Paranoia	Indirect = 1.05 direct = 1.16 total = 1.32	0.99 – 1.14 0.96 – 1.40 1.09-1.59	OR	Very low	Fisher 2012 1.
	212	General (community)	Emotional abuse	Negative other beliefs	Paranoia	Indirect = 1.02 direct = 1.16 total = 1.32	0.97 – 1.11 0.96 – 1.40 1.09-1.59	OR	Very low	Fisher 2012 2.
	212	General (community)	Physical abuse	Negative self-beliefs	Paranoia	Indirect = 1.03 direct = 1.21 total = 1.29	0.98 – 1.08 1.00 – 1.44 1.07-1.55	OR	Very low	Fisher 2012 3.
	212	General (community)	Physical abuse	Negative other beliefs	paranoia	Indirect = 1.01 direct = 1.21 total = 1.29	0.98 – 1.05 1.00 – 1.44 1.07-1.55	OR	Very low	Fisher 2012 4.

Figure 2.9 – Forest plot – completely standardised indirect effects – Cognitive beliefs and apparaisals

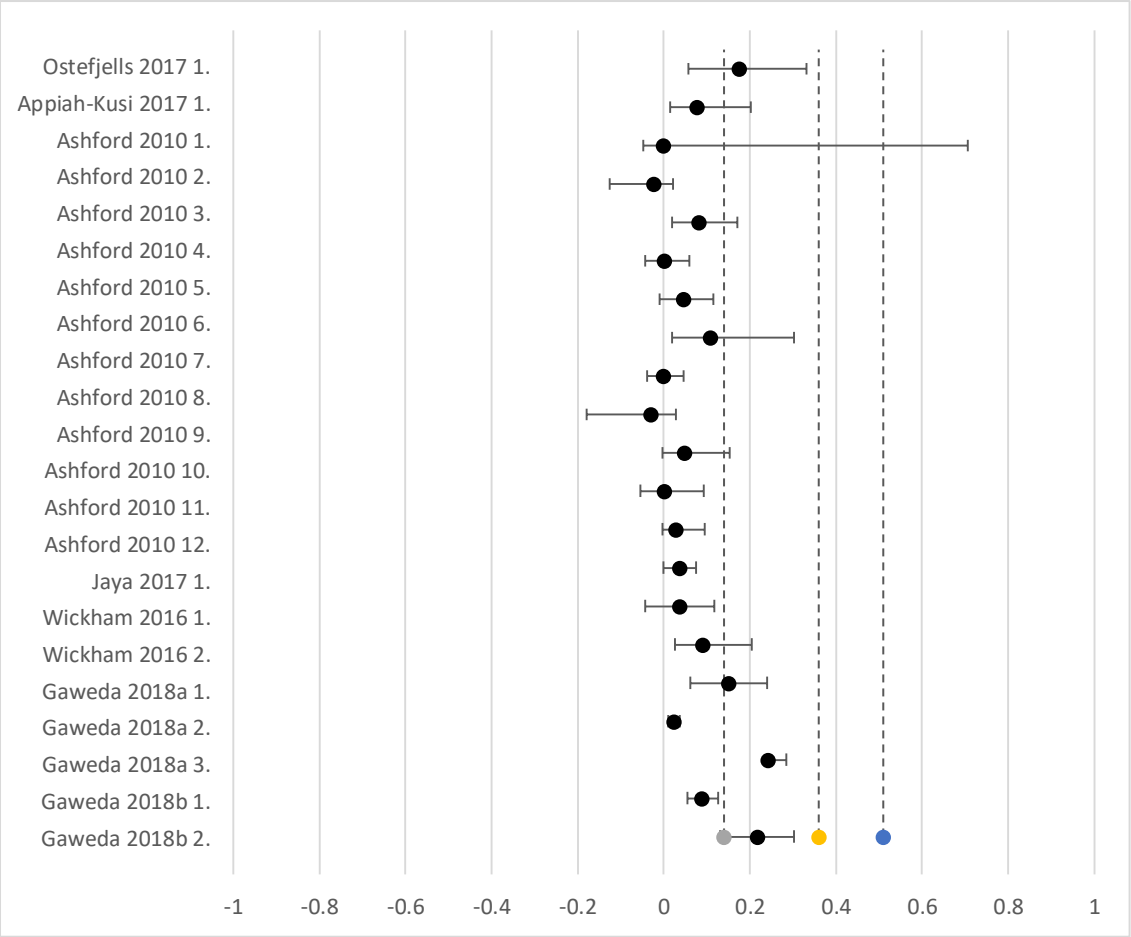


Figure 2.10 - Forest plot – odds ratios – Cognitive beliefs and apparaisals

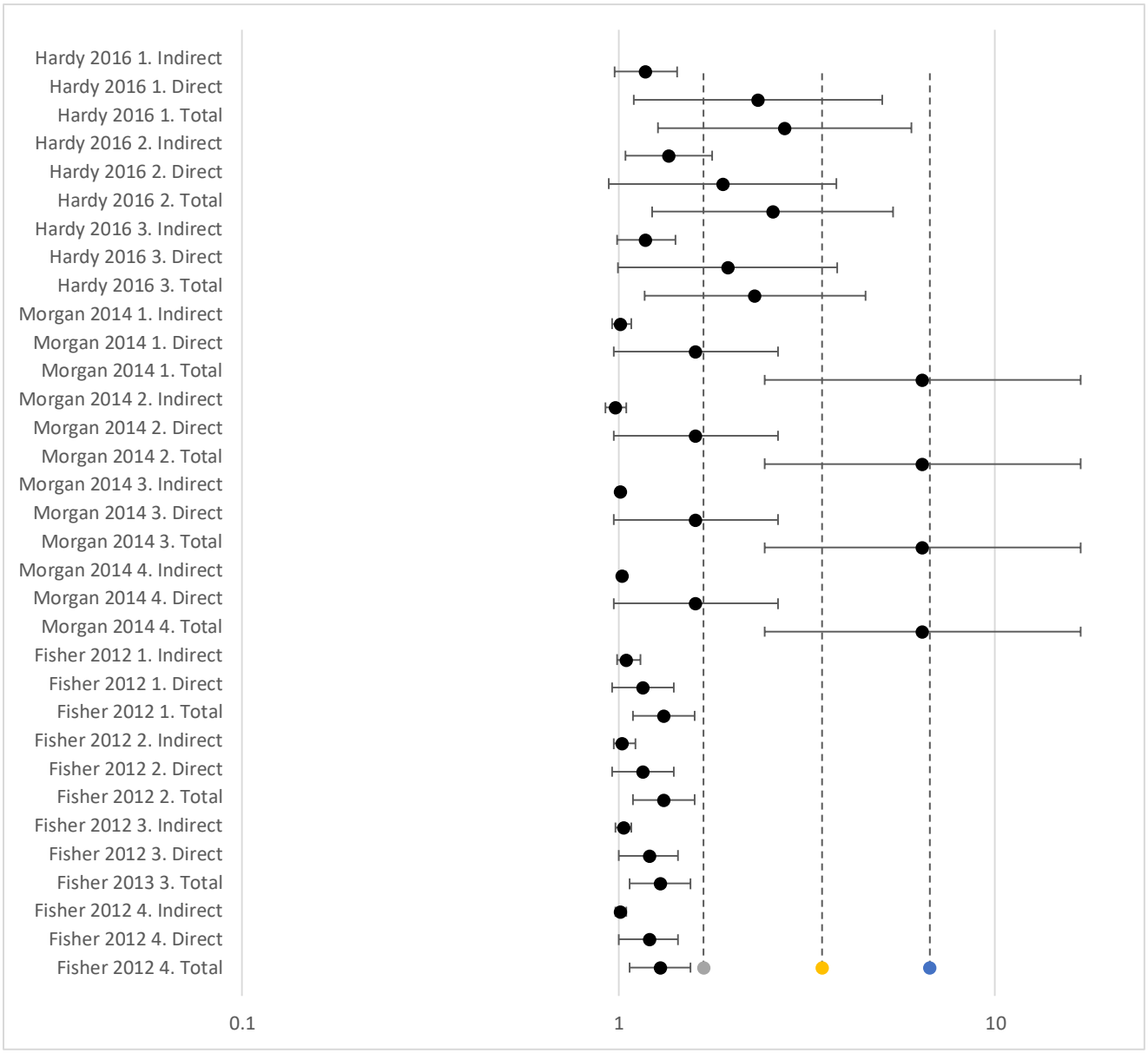


Table 2.10 Results –Attachment style

Study ref	N	Population	Predictor	Mediator(s)	Outcome	Effect size(s)	95% CIs (lower, upper)	Effect type	Quality	Forest plot reference
Ashford 2010	135	General (students)	Indirect aggression	Rejection sensitivity	Ideas of social reference	0.0298	-0.032, 0.132	CSIE	Moderate	Ashford 2010 1.
	135	General (students)	Direct verbal aggression	Rejection sensitivity	Ideas of social reference	0.0066	-0.013, 0.078	CSIE	Moderate	Ashford 2010 2.
	135	General (students)	Direct physical aggression	Rejection sensitivity	Ideas of social reference	-0.0137	-0.071, 0.014	CSIE	Moderate	Ashford 2010 3.
	135	General (students)	Indirect aggression	Rejection sensitivity	Persecution	-0.0538	-0.174, 0.007	CSIE	Moderate	Ashford 2010 4.
	135	General (students)	Direct verbal aggression	Rejection sensitivity	Persecution	-0.0119	-0.095, 0.0165	CSIE	Moderate	Ashford 2010 5.
	135	General (students)	Direct physical aggression	Rejection sensitivity	Persecution	0.0249	-0.002, 0.094	CSIE	Moderate	Ashford 2010 6.
Goodall 2015	283	General (community)	Emotional abuse	Avoidant attachment	Schizotypy	0.0383	0.010, 0.077	CSIE	High	Goodall 2015 1.
			Emotional abuse	Anxious attachment	Schizotypy	0.0575	0.019, 0.105	CSIE	High	Goodall 2015 2.
Pearce 2017	112	Clinical	Childhood trauma	Fearful attachment	Voices	0.058	-0.002, 0.165	CSIE	Very low	Pearce 2017 1.
	112	Clinical	Childhood trauma	Dissociation & fearful attachment	Voices	0.283	0.132, 0.497	CSIE	Very low	Pearce 2017 2.
	112	Clinical	Childhood trauma	Fearful attachment	Paranoia	0.0846	0.016, 0.186	CSIE	Very low	Pearce 2017 3.
	112	Clinical	Childhood trauma	Dissociation & fearful attachment	Paranoia	0.356	0.186, 0.573	CSIE	Very low	Pearce 2017 4.
Pilton 2016	55	Clinical	Sexual abuse	Anxious attachment	AH total	0.138	0.024, 0.253	CSIE	Low	Pilton 2016 1.

Study ref	N	Population	Predictor	Mediator(s)	Outcome	Effect size(s)	95% CIs (lower, upper)	Effect type	Quality	Forest plot reference
	55	Clinical	Sexual abuse	Anxious attachment	Malevolence	0.086	0.001, 0.171	CSIE	Low	Pilton 2016 2.
	55	Clinical	Sexual abuse	Anxious attachment	Omnipotence	0.09	0.004, 0.176	CSIE	Low	Pilton 2016 3.
	55	Clinical	Sexual abuse	Anxious attachment	Resistance	0.156	0.026, 0.287	CSIE	Low	Pilton 2016 4.
	55	Clinical	Sexual abuse	Anxious attachment	Voice dominance	0.09	−0.006, 0.187	CSIE	Low	Pilton 2016 5.
	55	Clinical	Sexual abuse	Anxious attachment	Voice intrusiveness	0.051	−0.012, 0.114	CSIE	Low	Pilton 2016 6.
	55	Clinical	Sexual abuse	Anxious attachment	Hearer dependence	0.112	0.007, 0.216	CSIE	Low	Pilton 2016 7.
	55	Clinical	Sexual abuse	Anxious attachment	Hearer distance	0.063	-0.028, 0.154	CSIE	Low	Pilton 2016 8.
	55	Clinical	Emotional abuse	Anxious attachment	AH total	0.138	0.024, 0.253	CSIE	Low	Pilton 2016 9.
	55	Clinical	Emotional abuse	Anxious attachment	Malevolence	0.086	0.001, 0.171	CSIE	Low	Pilton 2016 10.
	55	Clinical	Emotional abuse	Anxious attachment	Omnipotence	0.09	0.004, 0.176	CSIE	Low	Pilton 2016 11.
	55	Clinical	Emotional abuse	Anxious attachment	Resistance	0.156	0.026, 0.287	CSIE	Low	Pilton 2016 12.
	55	Clinical	Emotional abuse	Anxious attachment	Voice dominance	0.09	0.005, 0.187	CSIE	Low	Pilton 2016 13.
	55	Clinical	Emotional abuse	Anxious attachment	Voice intrusiveness	0.051	−0.012, 0.114	CSIE	Low	Pilton 2016 14.
	55	Clinical	Emotional abuse	Anxious attachment	Hearer dependence	0.112	0.007, 0.216	CSIE	Low	Pilton 2016 15.
	55	Clinical	Emotional abuse	Anxious attachment	Hearer distance	0.063	−0.028, 0.154	CSIE	Low	Pilton 2016 16.

Study ref	N	Population	Predictor	Mediator(s)	Outcome	Effect size(s)	95% CIs (lower, upper)	Effect type	Quality	Forest plot reference
	55	Clinical	Physical neglect	Anxious attachment	AH total	0.156	0.003, 0.310	CSIE	Low	Pilton 2016 17.
	55	Clinical	Physical neglect	Anxious attachment	Malevolence	0.085	−0.016, 0.186	CSIE	Low	Pilton 2016 18.
	55	Clinical	Physical neglect	Anxious attachment	Omnipotence	0.101	−0.004, 0.206	CSIE	Low	Pilton 2016 19.
	55	Clinical	Physical neglect	Anxious attachment	Resistance	0.197	−0.007, 0.310	CSIE	Low	Pilton 2016 20.
	55	Clinical	Physical neglect	Anxious attachment	Voice dominance	0.095	−0.022, 0.213	CSIE	Low	Pilton 2016 21.
	55	Clinical	Physical neglect	Anxious attachment	Voice intrusiveness	0.064	−0.020, 0.143	CSIE	Low	Pilton 2016 22.
	55	Clinical	Physical neglect	Anxious attachment	Hearer dependence	0.124	0.005, 0.243	CSIE	Low	Pilton 2016 23.
	55	Clinical	Physical neglect	Anxious attachment	Hearer distance	0.082	−0.039, 0.204	CSIE	Low	Pilton 2016 24.
Sheinbaum 2015	214	General (community)	Parental antipathy	Total attachment	Positive symptoms	0.0677	-0.003, 0.175	CSIE	Very low	Sheinbaum 2015 1.
	214	General (community)	Parental antipathy	Enmeshed	Positive symptoms	0.0288	-0.009, 0.1421	CSIE	Very low	Sheinbaum 2015 2.
	214	General (community)	Parental antipathy	Fearful	Positive symptoms	0.005	-0.009, 0.049	CSIE	Very low	Sheinbaum 2015 3.
	214	General (community)	Parental antipathy	Angry-dismissive	Positive symptoms	0.0426	0.002, 0.108	CSIE	Very low	Sheinbaum 2015 4.
	214	General (community)	Parental antipathy	Withdrawn	Positive symptoms	-0.0088	-0.061, 0.004	CSIE	Very low	Sheinbaum 2015 5.
	214	General (community)	Role reversal	Total attachment	Positive symptoms	0.045	-0.007, 0.135	CSIE	Very low	Sheinbaum 2015 6.
	214	General (community)	Role reversal	Enmeshed	Positive symptoms	0.0272	-0.006, 0.122	CSIE	Very low	Sheinbaum 2015 7.
	214	General (community)	Role reversal	Fearful	Positive symptoms	0.0016	-0.014, 0.036	CSIE	Very low	Sheinbaum 2015 8.

Study ref	N	Population	Predictor	Mediator(s)	Outcome	Effect size(s)	95% CIs (lower, upper)	Effect type	Quality	Forest plot reference
	214	General (community)	Role reversal	Angry-dismissive	Positive symptoms	0.0194	-0.005, 0.066	CSIE	Very low	Sheinbaum 2015 9.
	214	General (community)	Role reversal	Withdrawn	Positive symptoms	-0.0032	-0.044, 0.011	CSIE	Very low	Sheinbaum 2015 10.
Sheinbaum 2014	546	General (students)	Emotional & physical trauma	Total attachment	Psychotic-like experiences	0.033	0.007, 0.068	CSIE	Very low	Sheinbaum 2014 1.
	546	General (students)	Emotional & physical trauma	Dismissing	Psychotic-like experiences	0.0005	-0.006, 0.011	CSIE	Very low	Sheinbaum 2014 2.
	546	General (students)	Emotional & physical trauma	Preoccupied	Psychotic-like experiences	0.0179	-0.002, 0.046	CSIE	Very low	Sheinbaum 2014 3.
	546	General (students)	Emotional & physical trauma	Fearful	Psychotic-like experiences	0.0147	0.003, 0.035	CSIE	Very low	Sheinbaum 2014 4.
	546	General (students)	Emotional & physical trauma	Total attachment	Suspiciousness	0.0489	0.016, 0.088	CSIE	Very low	Sheinbaum 2014 5.
	546	General (students)	Emotional & physical trauma	Dismissing	Suspiciousness	-0.0005	-0.010, 0.006	CSIE	Very low	Sheinbaum 2014 6.
	546	General (students)	Emotional & physical trauma	Preoccupied	Suspiciousness	0.02	-0.001, 0.049	CSIE	Very low	Sheinbaum 2014 7.
	546	General (students)	Emotional & physical trauma	Fearful	Suspiciousness	0.0295	0.010, 0.058	CSIE	Very low	Sheinbaum 2014 8.
	546	General (students)	Emotional & physical trauma	Total attachment	Positive Schizotypy	0.0373	0.011, 0.072	CSIE	Very low	Sheinbaum 2014 9.
	546	General (students)	Emotional & physical trauma	Dismissing	Positive Schizotypy	0.004	-0.001, 0.017	CSIE	Very low	Sheinbaum 2014 10.
	546	General (students)	Emotional & physical trauma	Preoccupied	Positive Schizotypy	0.02	-0.001, 0.049	CSIE	Very low	Sheinbaum 2014 11.
	546	General (students)	Emotional & physical trauma	Fearful	Positive Schizotypy	0.0133	0.003, 0.033	CSIE	Very low	Sheinbaum 2014 12.
Sitko 2014	5877	General (community)	Witness injury/killing	Attachment	Paranoia	0.0034	-0.028. 0.035	CSIE	Very low	Sitko 2014 1.
	5877	General (community)	Rape	Attachment	Paranoia	0.0147	-0.025, 0.055	CSIE	Very low	Sitko 2014 2.

Study ref	N	Population	Predictor	Mediator(s)	Outcome	Effect size(s)	95% CIs (lower, upper)	Effect type	Quality	Forest plot reference
	5877	General (community)	Sexual molestation	Attachment	Paranoia	0.0017	-0.030, 0.034	CSIE	Very low	Sitko 2014 3.
	5877	General (community)	Physical attack/assault	Attachment	Paranoia	-0.0012	-0.040, 0.037	CSIE	Very low	Sitko 2014 4.
	5877	General (community)	physical abuse	Attachment	Paranoia	0.0056	-0.040, 0.052	CSIE	Very low	Sitko 2014 5.
	5877	General (community)	Neglect	Attachment	Paranoia	0.0179	-0.033, 0.069	CSIE	Very low	Sitko 2014 6.
	5877	General (community)	Held captive/threatened with a weapon	Attachment	Paranoia	0.0125	-0.024, 0.049	CSIE	Very low	Sitko 2014 7.
	5877	General (community)	Witness injury/killing	Attachment	Hallucinations	0.0023	-0.030, 0.035	CSIE	Very low	Sitko 2014 8.
	5877	General (community)	Rape	Attachment	Hallucinations	0.0124	-0.032, 0.057	CSIE	Very low	Sitko 2014 9.
	5877	General (community)	Sexual molestation	Attachment	Hallucinations	0.0040	-0.036, 0.044	CSIE	Very low	Sitko 2014 10.
	5877	General (community)	Physical attack/assault	Attachment	Hallucinations	-0.0031	-0.047, 0.041	CSIE	Very low	Sitko 2014 11.
	5877	General (community)	physical abuse	Attachment	Hallucinations	0.0042	-0.039, 0.048	CSIE	Very low	Sitko 2014 12.
	5877	General (community)	Neglect	Attachment	Hallucinations	0.0116	-0.035, 0.058	CSIE	Very low	Sitko 2014 13.
	5877	General (community)	Held captive/threatened with a weapon	Attachment	Hallucinations	0.0086	-0.033, 0.050	CSIE	Very low	Sitko 2014 14.
McDonnell 2018	64	Clinical high risk	Severity of bullying (primary school)	Interpersonal sensitivity	Paranoid ideation	0.129	0.029, 0.284	CSIE	Moderate	McDonnell 2018 1.
			Severity of bullying (secondary school)	Interpersonal sensitivity	Paranoid ideation	0.179	0.047, 0.366	CSIE	Moderate	McDonnell 2018 2.

Figure 2.11 – Forest plot comlpetely standardised indirect effects – Attachment style

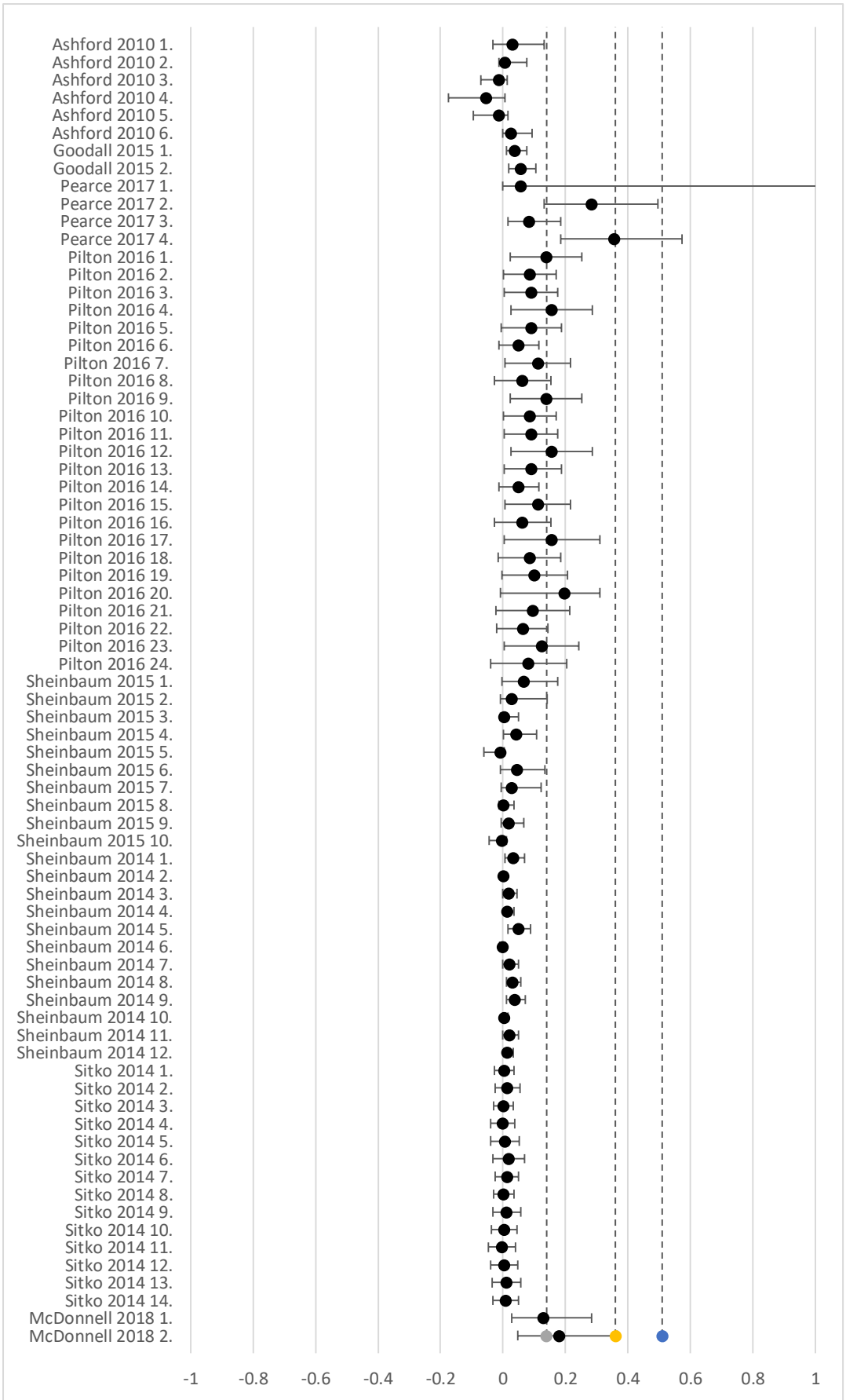


Table 2.11 Results –Mood and anxiety

Study ref	N	Population	Predictor	Mediator(s)	Outcome	Effect size(s)	95% CIs (lower, upper)	Effect type	Quality	Forest plot reference
Osterfjells 2017	261[5]	Clinical	Childhood trauma	Anxiety, Depression, Metacognitive beliefs	Positive symptoms	0.046	0.020, 0.100	CSIE	High	Osterfjells 2017 1.
Ashford 2010	135	General (students)	Indirect aggression	Anxiety	Ideas of social reference	0.0525	-0.001, 0.1669	CSIE	Moderate	Ashford 2010 1.
	135	General (students)	Indirect aggression	Depression	Ideas of social reference	0.0521	0.001, 0.1603	CSIE	Moderate	Ashford 2010 2.
	135	General (students)	Direct verbal aggression	Anxiety	Ideas of social reference	0.0019	-0.075, 0.090	CSIE	Moderate	Ashford 2010 3.
	135	General (students)	Direct verbal aggression	Depression	Ideas of social reference	0.0005	-0.077, 0.084	CSIE	Moderate	Ashford 2010 4.
	135	General (students)	Direct physical aggression	Anxiety	Ideas of social reference	0.0195	-0.0315, 0.1053	CSIE	Moderate	Ashford 2010 5.
	135	General (students)	Direct physical aggression	Depression	Ideas of social reference	0.0077	-0.046, 0.083	CSIE	Moderate	Ashford 2010 6.
	135	General (students)	Indirect aggression	Anxiety	Persecution	0.0493	-0.004, 0.170	CSIE	Moderate	Ashford 2010 7.
	135	General (students)	Indirect aggression	Depression	Persecution	0.0617	-0.002, 0.201	CSIE	Moderate	Ashford 2010 8.
	135	General (students)	Direct verbal aggression	Anxiety	Persecution	0.0017	-0.077, 0.089	CSIE	Moderate	Ashford 2010 9.

[5] Total clinical sample used for mediation analysis – BPD and Psychosis

Study ref	N	Population	Predictor	Mediator(s)	Outcome	Effect size(s)	95% CIs (lower, upper)	Effect type	Quality	Forest plot reference
	135	General (students)	Direct verbal aggression	Depression	Persecution	0.0006	-0.091, 0.101	CSIE	Moderate	Ashford 2010 10.
	135	General (students)	Direct physical aggression	Anxiety	Persecution	0.0183	-0.030, 0.102	CSIE	Moderate	Ashford 2010 11.
	135	General (students)	Direct physical aggression	Depression	Persecution	0.0091	-0.052, 0.114	CSIE	Moderate	Ashford 2010 12.
Marwaha & Bebbington 2015	5689	General (community)	Sexual abuse (intercourse)	Mood	Unaffected vs Probable psychosis	indirect = 2.41 direct = 4.08 total = 9.84	1.61-3.61 1.41-11.79 3.48-27.85	OR	Low	Marwaha 2015 1.
	5689	General (community)	Sexual abuse (contact)	Mood	Unaffected vs Probable psychosis	indirect = 1.60 direct = 2.14 total = 3.42	1.32-1.94 0.91-5.04 1.46-8.02	OR	Low	Marwaha 2015 2.
Marwaha 2014	7403	General (community)	Sexual abuse	Mood instability	Unaffected vs Probable psychosis	indirect = 2.30 direct = 4.83 total = 11.09	1.60-3.29 1.96-11.90 4.62-26.62	OR	Moderate	Marwaha 2014 1.
	7403	General (community)	Sexual abuse	Mood instability	Unaffected vs Paranoid ideation	indirect = 1.63 direct = 2.52 total = 4.10	1.37-1.93 1.71-3.72 2.80-6.00	OR	Moderate	Marwaha 2014 2.
	7403	General (community)	Sexual abuse	Mood instability	Unaffected vs Auditory hallucinations	indirect = 1.41 direct = 2.79 total = 3.94	(1.23-1.63) (1.51-5.14) (2.13-7.29)	OR	Moderate	Marwaha 2014 3.
Thompson 2016	233	Ultra- high risk	Sexual abuse	HAM-Anxiety	Transition to psychosis	indirect = 1.00 direct = 1.15 total = 1.15	0.99-1.01 1.02-1.31 1.02-1.31	OR	Very low	Thompson 2016 1.
	233	Ultra- high risk	Sexual abuse	HAM-Depression	Transition to psychosis	indirect = 1.00 direct = 1.06 total = 1.06	(1.00-1.01) (1.00-1.13) (1.00-1.13)	OR	Very low	Thompson 2016 2.

Study ref	N	Population	Predictor	Mediator(s)	Outcome	Effect size(s)	95% CIs (lower, upper)	Effect type	Quality	Forest plot reference
	233	Ultra- high risk	Sexual abuse	CAARMS Mood	Transition to psychosis	indirect = 1.00 direct = 1.05 total = 1.05	(0.99-1.01) (0.99-1.13) (0.99-1.13)	OR	Very low	Thompson 2016 3.
	233	Ultra- high risk	Sexual abuse	CAARMS anxiety	Transition to psychosis	indirect = 1.00 direct = 1.07 total = 1.06	(0.98-1.01) (1.00-1.14) (0.99-1.14)	OR	Very low	Thompson 2016 4.
	233	Ultra- high risk	Sexual abuse	CAARMS mood swings	Transition to psychosis	indirect = 1.00 direct = 1.09 total = 1.09	(0.99-1.01) (1.01-1.17) (1.01-1.17)	OR	Very low	Thompson 2016 5.
	233	Ultra- high risk	Sexual abuse	CAARMS mania	Transition to psychosis	indirect = 1.01 direct = 1.11 total = 1.12	(0.98-1.04) (1.01-1.22) (1.02-1.24)	OR	Very low	Thompson 2016 6.
Fisher 2012	212	General (community)	Emotional abuse	Recent anxiety[6]	Paranoia	Indirect = 1.05 direct = 1.16 total = 1.32	 1.0 - 1.13 0.96 – 1.40 1.09-1.59	OR	Very low	Fisher 2012 1.
	212	General (community)	Emotional abuse	Depression[1]	Paranoia	Indirect = 1.01 direct = 1.16 total = 1.32	 0.93 – 1.11 0.96 – 1.40 1.09-1.59	OR	Very low	Fisher 2012 2
	212	General (community)	Physical abuse	Recent anxiety[1]	Paranoia	Indirect = 1.02 direct = 1.21 total = 1.29	 0.97 – 1.10 1.00 – 1.44 1.07-1.55	OR	Very low	Fisher 2012 3.
	212	General (community)	Physical abuse	Depression[1]	Paranoia	Indirect = 1.01 direct = 1.21 total = 1.29	 0.97 – 1.06 1.00 – 1.44 1.07-1.55	OR	Very low	Fisher 2012 4.
Bebbington 2011	642	General (community)	Sexual abuse (contact)	Revictimisation & anxiety	Probable psychosis	Indirect = 1.35 Direct = 3.2 Total = 4.5	1.16 - 1.56 1.3 – 8.1 2.0 – 10.2	OR	Low	Bebbington 2011 1.

[6] Multiple model with negative beliefs about self and others

Study ref	N	Population	Predictor	Mediator(s)	Outcome	Effect size(s)	95% CIs (lower, upper)	Effect type	Quality	Forest plot reference
	642	General (community)	Sexual abuse (contact)	Revictimisation & depression	Probable psychosis	Indirect = 1.52 Direct = 2.8 Total = 4.5	1.31 - 1.76 1.1 – 7.7 2.0 – 10.2	OR	Low	Bebbington 2011 2.

Figure 2.12 - Forest plot – comlpetely standardised indirect effects – Mood and anxiety

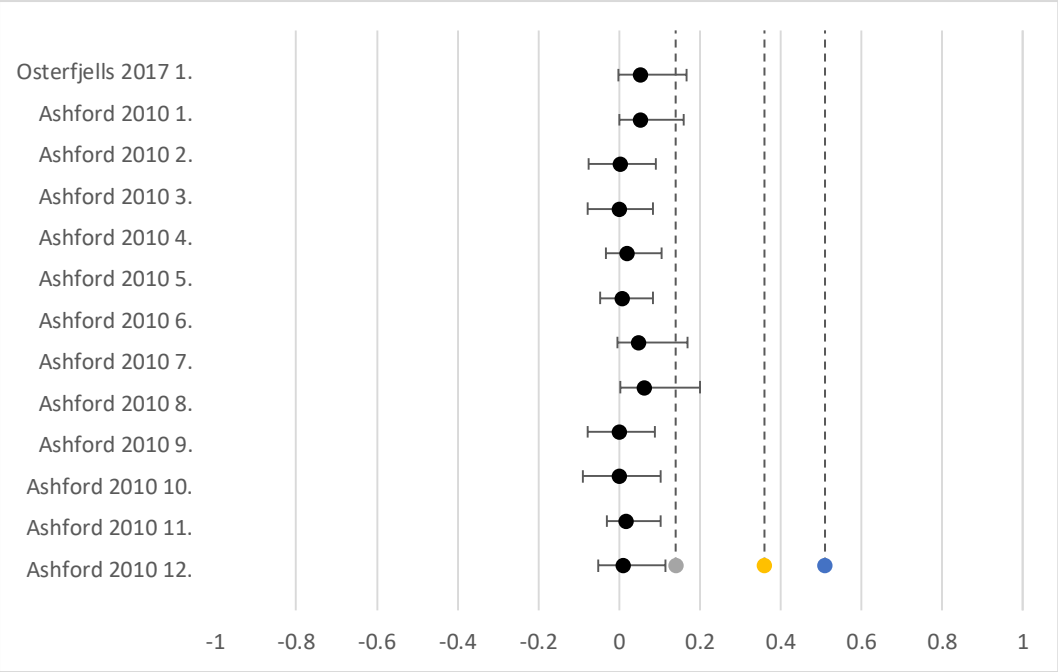


Figure 2.13- Forest plot – odds ratios – Mood and anxiety

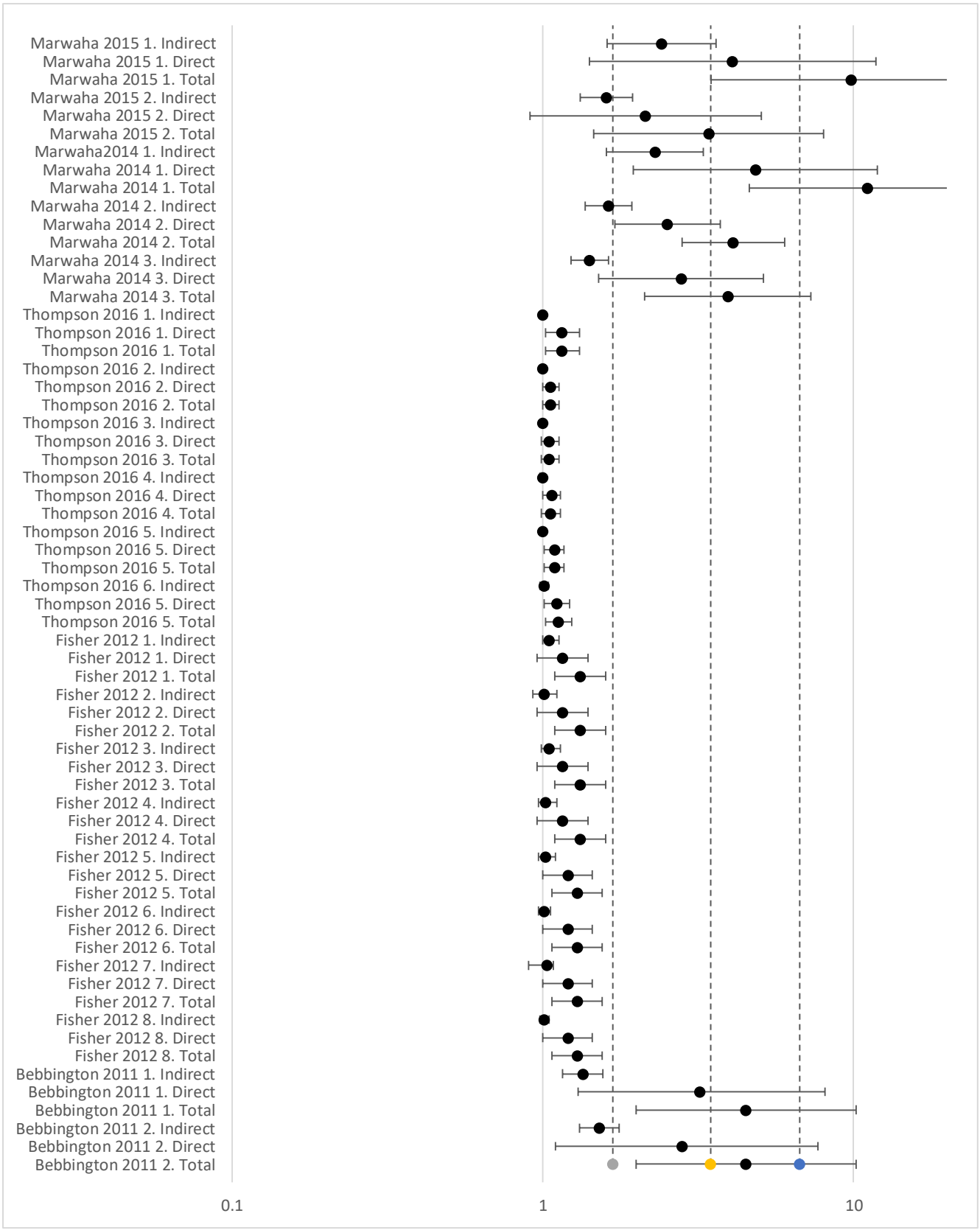


Table 2.12 Results - Emotion regulation and stress sensitivity

Study ref	N	Population	Predictor	Mediator(s)	Outcome	Effect size(s)	95% CIs (lower, upper)	Effect type	Quality	Forest plot reference
Van Nierop 2014	384	Extended psychosis phenotype	Childhood trauma	Affect dysregulation	Psychotic experiences	0.04	0.015, 0.065	CSIE	Moderate	Van Nierop 2014 1.
	384	Extended psychosis phenotype	Childhood trauma	Combined affect dysregulation & social defeat	Psychotic experiences	0.07	0.027, 0.111	CSIE	Moderate	Van Nierop 2014 2.
	43	Psychosis	Childhood trauma	Affect dysregulation	Psychotic symptoms	0.01	-0.023, 0.043	CSIE	Moderate	Van Nierop 2014 3.
	43	Psychosis	Childhood trauma	Combined affect dysregulation & social defeat	Psychotic symptoms	0.04	0.016, 0.063	CSIE	Moderate	Van Nierop 2014 4.
Perona Garcelan 2013	318	General (students)	Childhood trauma	Mindfulness	Hallucination-proneness	0.0093	-0.007, 0.0302	CSIE	Very low	Perona Garcelan 2013 1.
Lincoln 2017	562	General (community)	Childhood trauma	Emotion regulation	Overall distress	0.0033	0.0001, 0.0065	CSIE	High	Lincoln 2017 1.
	562	General (community)	Childhood trauma	Emotion regulation	Paranoia distress	0.0203	0.0014, 0.0391	CSIE	High	Lincoln 2017 2.
	562	General (community)	Childhood trauma	Emotion regulation	Paranoia Frequency	0.0157	0.0016, 0.0314	CSIE	High	Lincoln 2017 3.
Rossler 2016	663	General (enriched)	Childhood trauma total	Stress sensitivity	Unaffected vs anomalous perceptions	direct = 0.98 indirect = 1.22 total = 1.17	0.70, 1.38 1.10, 1.34 0.84, 1.64	OR	Low	Rossler 2016 1.
	663	General (enriched)	Emotional abuse	Stress sensitivity	Unaffected vs anomalous perceptions	direct = 0.78 indirect = 1.24 total = 1.01	0.48, 1.25 1.10, 1.39 0.70, 1.46	OR	Low	Rossler 2016 2.
	663	General (enriched)	Emotional neglect	Stress sensitivity	Unaffected vs anomalous perceptions	direct=1.04 indirect=1.18 total=1.20	0.68, 1.57 1.07, 1.30 0.91, 1.59	OR	Low	Rossler 2016 3.

Study ref	N	Population	Predictor	Mediator(s)	Outcome	Effect size(s)	95% CIs (lower, upper)	Effect type	Quality	Forest plot reference
	663	General (enriched)	Physical neglect	Stress sensitivity	Unaffected vs anomalous perceptions	direct=1.34 indirect=1.11 total=1.39	0.98, 1.82 1.03, 1.19 1.09, 1.77	OR	Low	Rossler 2016 4.
	663	General (enriched)	Childhood trauma total	Stress sensitivity	Unaffected vs odd beliefs and behaviours	direct=1.73 indirect=1.49 total=2.15	1.36, 2.21 1.29, 1.70 1.71, 2.70	OR	Low	Rossler 2016 5.
	663	General (enriched)	Emotional abuse	Stress sensitivity	Unaffected vs odd beliefs and behaviours	direct=1.37 indirect=1.60 total=2.24	0.98, 1.90 1.37, 1.84 1.80, 2.80	OR	Low	Rossler 2016 6.
	663	General (enriched)	Emotional neglect	Stress sensitivity	Unaffected vs odd beliefs and behaviours	direct=1.31 indirect=1.44 total=1.95	0.92, 1.86 1.25, 1.64 1.59, 2.39	OR	Low	Rossler 2016 7.
	663	General (enriched)	Physical neglect	Stress sensitivity	Unaffected vs odd beliefs and behaviours	direct=0.98 indirect=1.26 total=1.50	0.73, 1.30 1.11, 1.42 1.24, 1.82	OR	Low	Rossler 2016 8.
	663	General (enriched)	Childhood trauma total	Stress sensitivity	Unaffected vs anomalous perceptions and odd beliefs	direct=1.55 indirect=1.65 total=2.06	1.09, 2.20 1.34, 1.95 1.50, 2.83	OR	Low	Rossler 2016 9.
	663	General (enriched)	Emotional abuse	Stress sensitivity	Unaffected vs anomalous perceptions and odd beliefs	direct=1.60 indirect=1.76 total=2.39	0.96, 2.65 1.41, 2.11 1.77, 3.23	OR	Low	Rossler 2016 10.
	663	General (enriched)	Emotional neglect	Stress sensitivity	Unaffected vs anomalous perceptions and odd beliefs	direct=0.74 indirect=1.56 total=1.66	0.41, 1.32 1.28, 1.83 1.20, 2.31	OR	Low	Rossler 2016 11.

Study ref	N	Population	Predictor	Mediator(s)	Outcome	Effect size(s)	95% CIs (lower, upper)	Effect type	Quality	Forest plot reference
	663	General (enriched)	Physical neglect	Stress sensitivity	Unaffected vs anomalous perceptions and odd beliefs	direct=1.31 indirect=1.32 total=1.64	0.85, 2.01 1.12, 1.52 1.23, 2.18	OR	Low	Rossler 2016 12.

Figure 2.14 - Forest plot – comlpetely standardised indirect effects – Emotion regulation and stress sensitivity

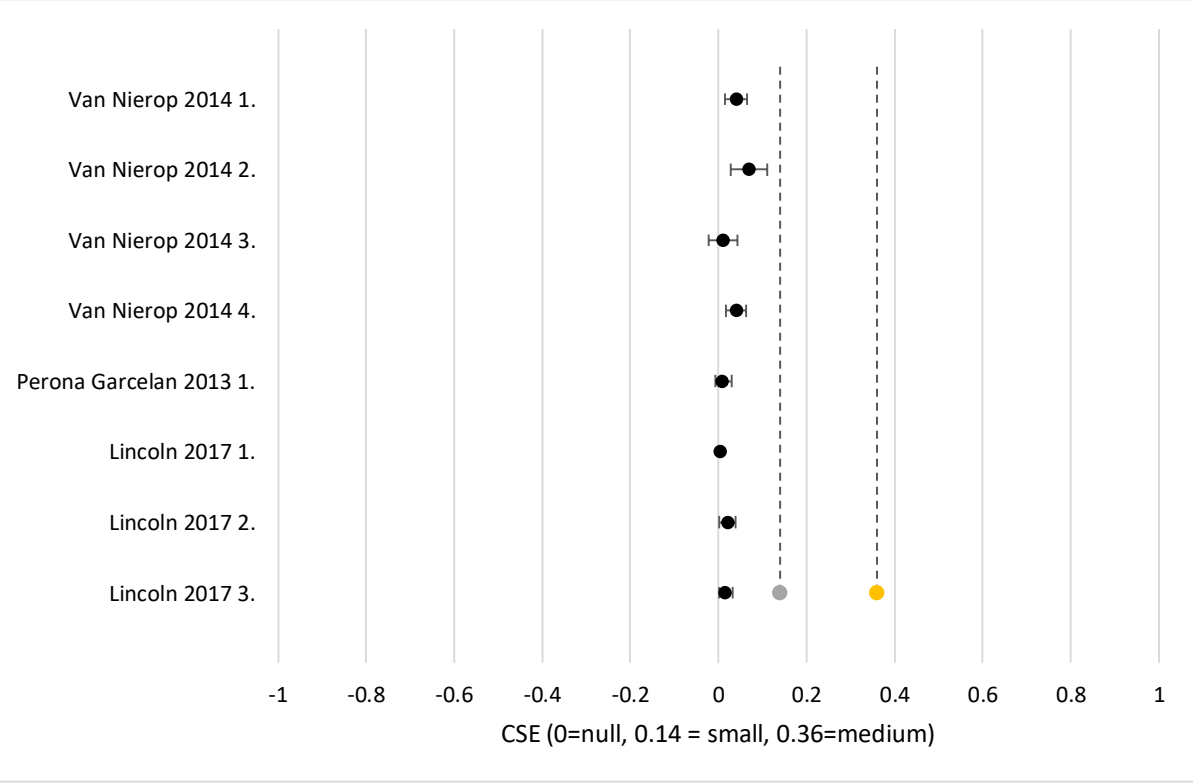


Figure 2.15 - Forest plot – odds ratios – Emotion regulation and stress sensitivity

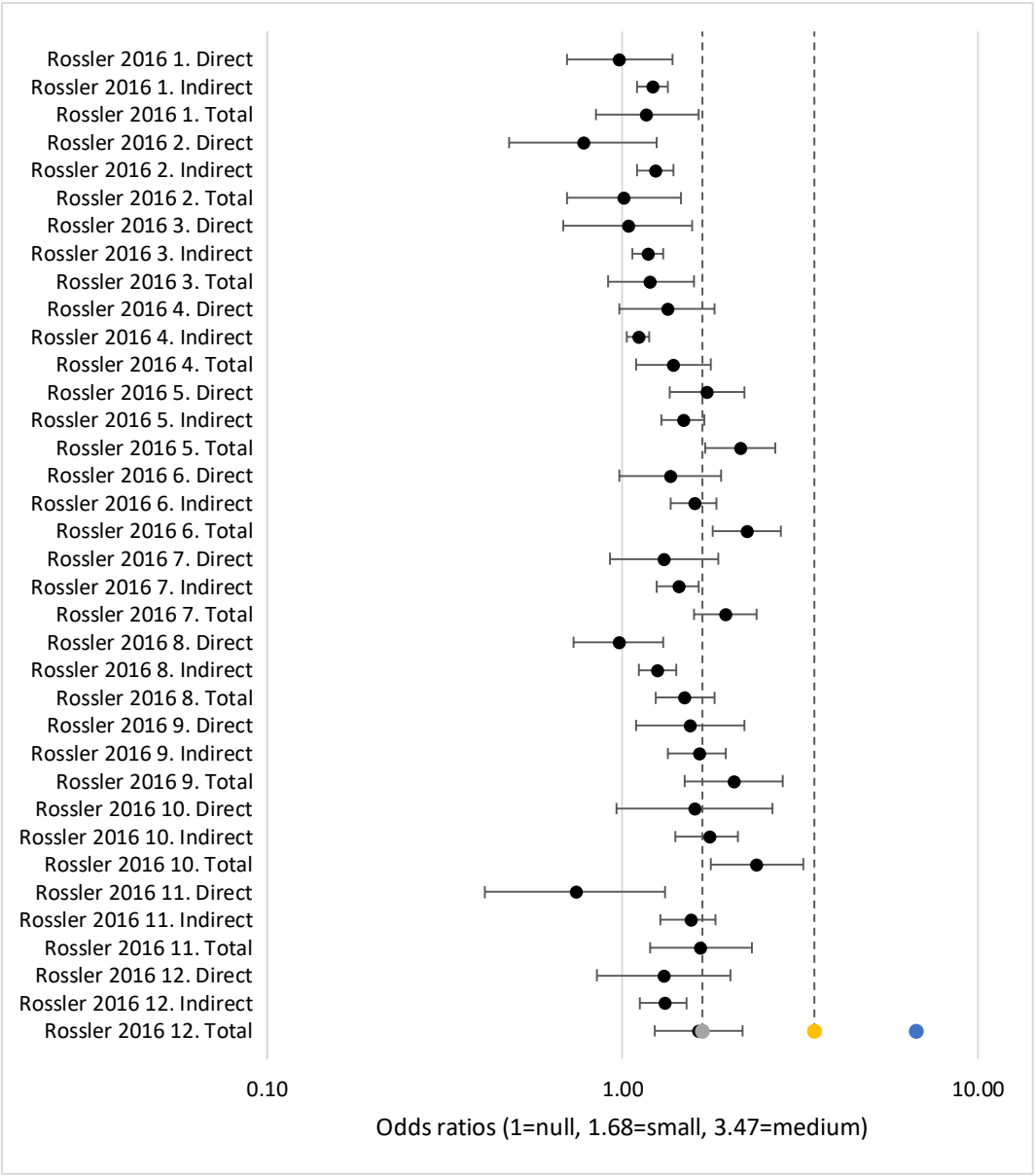


Table 2.13 Results - Social defeat

Study ref	N	Population	Predictor	Mediator(s)	Outcome	Effect size(s)	95% CIs (lower, upper)	Effect type	Quality	Forest plot reference
Jaya 2016	2350	General (community)	Social adversity	Social rank	Positive symptoms	0.222	0.148, 0.315	CSE	Low	Jaya 2016 1.
	2350	General (community)	Social adversity	Loneliness	Positive symptoms	-0.093	-0.130, -0.056	CSE	Low	Jaya 2016 2.
Van Nierop 2014	384	Extended psychosis phenotype	Childhood trauma	Social defeat	Psychotic experiences	0.03	-0.004, 0.064	CSE	Moderate	Van Nierop 2014 1.
	384	Extended psychosis phenotype	Childhood trauma	Combined affect dysregulation & social defeat	Psychotic experiences	0.07	0.027, 0.111	CSE	Moderate	Van Nierop 2014 2.
	43	Psychosis	Childhood trauma	Social defeat	Psychotic symptoms	0.04	0.013, 0.067	CSE	Moderate	Van Nierop 2014 3.
	43	Psychosis	Childhood trauma	Combined affect dysregulation & social defeat	Psychotic symptoms	0.04	0.016, 0.063	CSE	Moderate	Van Nierop 2014 4.
Boyda & McFeeters 2015	7403	General (community)	Sexual abuse	Activities in daily living	Unaffected vs Psychotic-like experiences	Indirect =1.326 Direct =1.38 Total =1.60	1.008-1.744 0.936-2.056 1.117-2.317	OR	Moderate-High	Boyda 2015 1.
	7403	General (community)	Sexual abuse	Loneliness	Unaffected vs Psychotic-like experiences	Indirect =1.366 Direct =1.38 Total =1.60	1.123-1.662 0.936-2.056 1.117-2.317	OR	Moderate-High	Boyda 2015 2.
	7403	General (community)	Emotional neglect	Loneliness	Unaffected vs Psychotic-like experiences	Indirect =1.889 Direct= 0.94 Total = 1.40	1.408-2.535 0.668-1.329 1.012-1.898	OR	Moderate-High	Boyda 2015 3.

Study ref	N	Population	Predictor	Mediator(s)	Outcome	Effect size(s)	95% CIs (lower, upper)	Effect type	Quality	Forest plot reference
Morgan 2014	390 391	Clinical Controls	Parental separation	No qualifications	Unaffected vs psychosis	Indirect = 2.15 direct = 1.60 total = 6.41	1.08 - 4.26 0.97-2.65 2.44-16.86	OR	Moderate-low	Morgan 2014 1
			Parental separation	Adult disadvantage	Unaffected vs psychosis	Indirect = 1.17 direct = 1.60 total = 6.41	0.95 - 1.45 0.97-2.65 2.44-16.86	OR	Moderate-low	Morgan 2014 2.
			Parental separation	No qualifications & adult disadvantage	Unaffected vs psychosis	Indirect = 1.54 direct = 1.6 total = 6.41	1.10 – 2.14 0.97-2.65 2.44-16.86	OR	Moderate-low	Morgan 2014 3.
			Parental separation	No qualifications & self esteem	Unaffected vs psychosis	Indirect = 0.98 direct = 1.6 total = 6.41	0.92 – 1.05 0.97-2.65 2.44-16.86	OR	Moderate-low	Morgan 2014 4.
			Parental separation	Adult disadvantage & self esteem	Unaffected vs psychosis	Indirect = 1.01 direct = 1.6 total = 6.41	0.99 – 1.02 0.97-2.65 2.44-16.86	OR	Moderate-low	Morgan 2014 5.
			Parental separation	No qualifications, adult disadvantage & self esteem	Unaffected vs psychosis	Indirect = 1.02 direct = 1.6 total = 6.41	1.00 – 1.05 0.97-2.65 2.44-16.86	OR	Moderate-low	Morgan 2014 6.

Figure 2.16 Forest plot – comlpetely standardised indirect effects – Social defeat

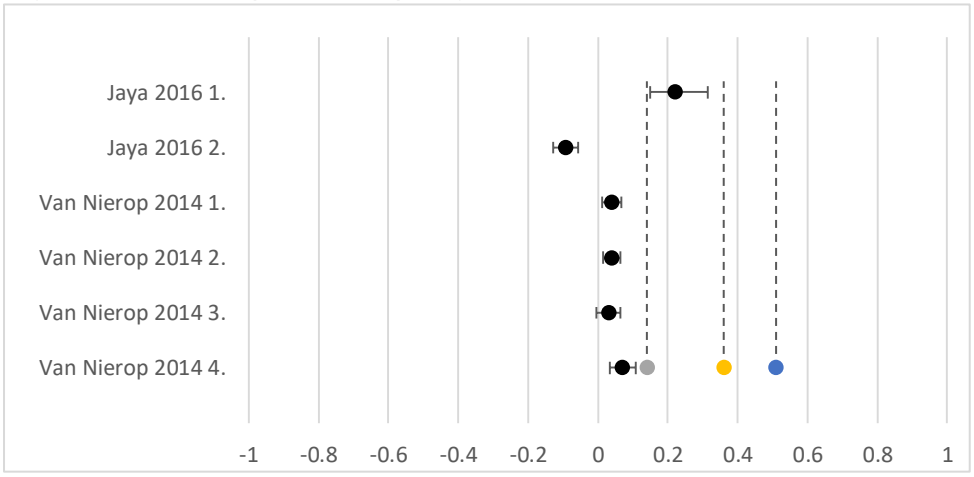


Figure 2.17 - Forest plot – odds ratios – Social defeat

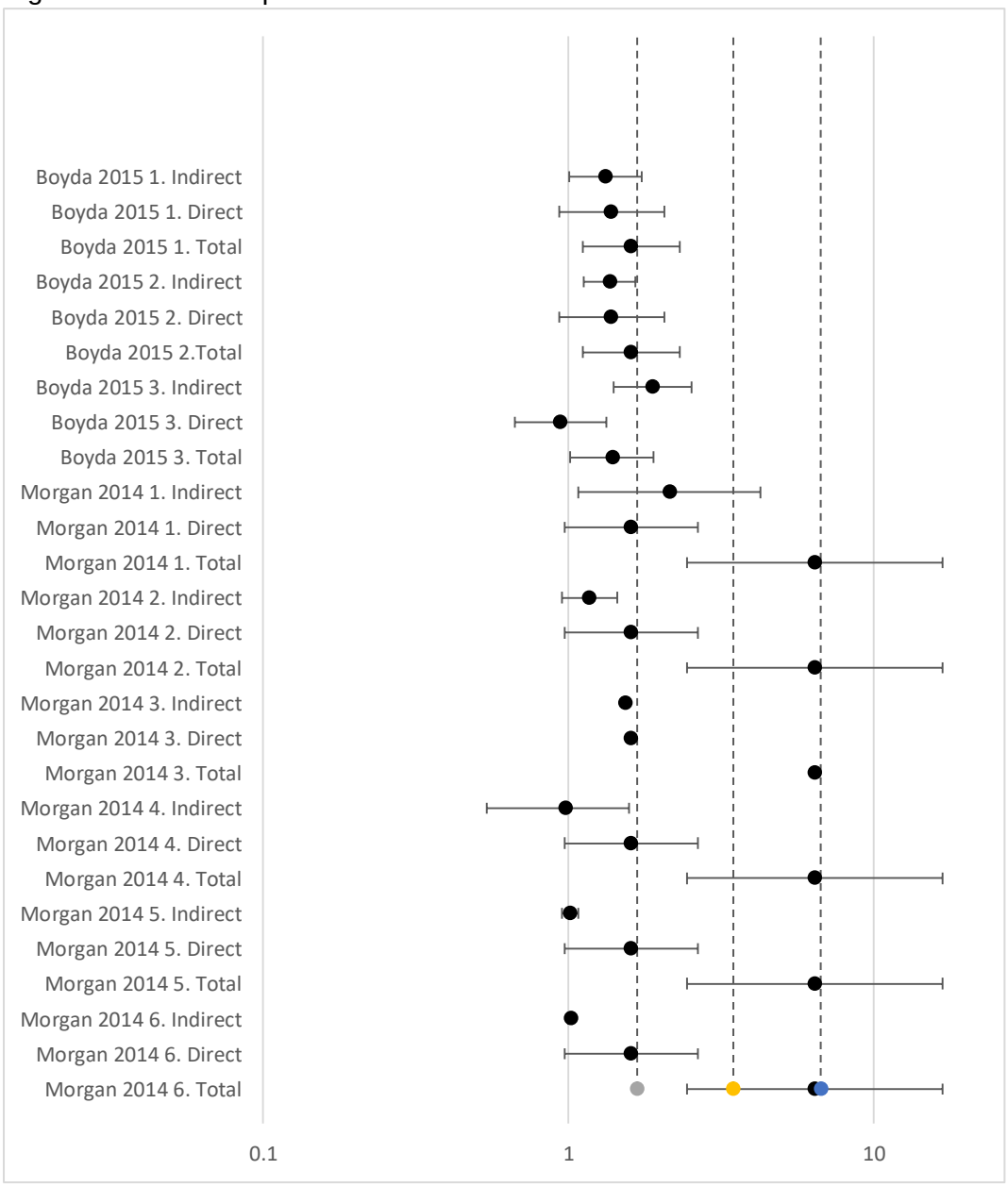


Table 2.14 GRADE ratings for all categories

Category	GRADE rating	Criteria
PTSD symptoms and dissociation	Moderate	-1 Risk of bias
Cognitive beliefs and appraisals	High	
Attachment style	Low	-1 Risk of bias -1 Inconsistency
Mood and anxiety	Low	-1 Risk of bias -1 Imprecision
Emotion regulation and stress sensitivity	High	
Social defeat	Moderate	-1 Indirectness

2.7.5 Summary of key findings

Overall, this review serves to highlight a number of limitations in mediation studies in this area. Poor reporting and inconsistent study quality caused the evidence synthesis to be challenging. The most common issues were potential sample bias and inadequate description of sample characteristics. In over one third of the included studies potential confounding factors were poorly controlled and missing data was inadequately, or not transparently handled. At present the evidence suggests that effect sizes are almost universally trivial to small in magnitude, with larger effects being negatively affected by imprecision.

Of the categories in this review, cognitive beliefs and appraisals has the greatest incidence of good quality evidence finding trivial and non-trivial effect sizes– see Figure 2.6. PTSD and dissociation as a category finds a number of significant effects, some of which are of non-trivial size, however study quality in this area is markedly poorer. Effects in both of these categories were small but relatively consistent, and with adequate precision as compared with other categories. The research into cognitive factors is based on greater sample sizes, whereas the studies assessing PTSD and dissociation are largely confined to smaller clinical samples. These categories at present constitute the best evidence in support of factors which act as mediators between childhood trauma and psychosis.

Both the mood and anxiety, and attachment style categories have effects spread across the quadrants of Figure 2.6. Each category has only one study in the non-trivial, good quality quadrant, both of which are significant, however the majority of studies fall into the trivial effect size quadrants. Taking into account the effect sizes and confidence bounds, at present there is little evidence to support the importance of either mood and anxiety factors, or attachment style as significant mediators.

It is notable that emotion regulation and stress sensitivity, although one of the smaller categories, has three good quality studies, all of which find significant effects of both trivial and non-trivial size. Although current evidence suggests only very small mediation effects, this is

a category which may benefit from further investigation, particularly in clinical samples where evidence is currently lacking.

Social defeat follows a similar pattern to attachment and mood, however the mediators in this category are more disparate in definition, and at times poorly measured. At present, evidence shows only very small effects, however this category remains under-researched and future studies using better quality measures are required.

Despite the prevalence of research into cognitive factors, metacognition and cognitive biases have received little attention. Cognitive biases are frequently included in models of psychosis development (Coltheart et al., 2011; Freeman, 2007; Garety et al, 2005). These biases are hypothesised to contribute to belief inflexibility, which has an effect on the ability to make alternative appraisals and attributions of anomalous experiences (Kuipers et al., 2006), and have been regularly associated with positive symptoms (Gaweda, Prochwicz & Cella, 2015; Peters et al., 2014; Moritz et al., 2010). Metacognitive beliefs may also be relevant to appraisals and the subjective interpretation of positive symptoms, for which they may act as maintaining factors (Morrison et al., 2001). The types of tasks used to measure cognitive biases are not always amenable to administration as part of a questionnaire battery, which may explain why they have seldom been researched, but this is an area which researchers should consider in future.

No studies to date have investigated theory of mind or alexithymia. Theory of mind, the ability to understand the mental states of self and other, and an awareness of how these may differ (Brune, 2005; Frith, 2004), has been found to be associated with trauma, attachment, and the tendency to make misattributions (Bentall 2006; Scherzer et al., 2012), and therefore it may be fruitful to take this into consideration in future cognitive mediation studies. Likewise, alexithymia, a difficulty with identifying and verbalising emotional states, (Sifneos, 1972; Suslow & Donges, 2017), has been found to be associated with negative emotions, but also with impaired emotional recognition and communication (O'Driscoll et al., 2014; Cohen &

Minor, 2010) and therefore may be important to assess alongside mood and emotion regulation factors.

2.8 Implications

2.8.1 Theoretical implications

Findings highlight the importance of cognitive factors which largely supports cognitive models of psychosis development, specifically those which take into account environmental risks such as trauma. Models suggest that early interpersonal adversity leads to the development of negative schemas about the self and others (Read et al., 2005), as well as deficits in processing information and emotions (Kuipers et al., 2006). This may lead to intrusive thoughts or experiences which are either misattributed to external sources, or misinterpreted as threatening (Kuipers et al., 2006; Fowler et al., 2006, Holmes et al., 2004). The consistent small effects reported by studies reviewed here for negative schemas and beliefs about others lend a degree of support to this.

Morrison (2001) suggests that internally generated thoughts which are mistakenly attributed to external sources, due to faulty self-beliefs and dysfunctional social understanding, may give rise to persecutory delusions. These faulty beliefs may have their roots in early trauma which can lead to elevated threat anticipation, and sensitivity to stress; leaving the individual vulnerable to interpreting anomalous experiences in distressing ways (Broome et al., 2005; Garety et al., 2007). Models propose that biases such as jumping to conclusions (making decisions based on limited or incomplete evidence) or confirmatory bias (rejecting any evidence which does not fit with a theory) are logical precedents to paranoid or persecutory thinking (Moritz et al., 2010). Rigidity of thought patterns and the refusal or inability to consider alternative explanations means fleeting intrusions may lead to rumination and fixation on fears of being harmed. As yet, the jumping to conclusions bias has not been examined as a potential mediating factor, and it is notable that these cognitive models often include reference to emotions, emotions regulation or sensitivity to stress which have, thus far, attracted little research attention.

Clearly there is further work to be done to fully understand how cognition may interact with emotion-based factors in multiple mediator models. Current evidence does suggest that cognitive factors are a plausible mechanism, with consistent effects, however the other Bradford Hill criteria (dose-response, reversibility and experimental support) will require evidence from future studies.

Although there is support for PTS symptoms and dissociation acting as mediators, there is still further work required to understand the temporal sequence, an important criteria when considering causality. Studies have found evidence that PTSD symptoms mediate between abuse and dissociation (Terock et al., 2016), but also that dissociation mediates between complex childhood trauma and PTSD (van Dijke et al., 2015). Without studies which can offer evidence for causal sequencing, it is not possible to establish whether PTS symptoms and dissociation are causally implicated in the development of psychosis following childhood trauma. This category does show support for specificity which is another indicator of potential causality (Hill, 1965). There is good evidence that sexual abuse as well as total trauma lead to hallucinations via dissociation (Varese, 2012; Perona-Garcelan 2012 & 2014; Choi, 2017; Pearce, 2012; Cole, 2016, Evans, 2015). However the decomposition of dissociation into component parts degrades the clarity of the relationship, with inconsistent findings around depersonalisation and absorption (Perona Garcelan 2012 & 2014).

Difficult childhood environments have also been proposed to lead to insecure attachment styles and maladaptive social functioning (Berry, Barrowclough & Wearden, 2008). The social and emotional repercussions of adverse environments may include heightened sensitivity to threat, and an avoidant coping style; along with cognitive biases which can negatively impact on attributions and the contextual processing of anomalous experiences (Freeman, Garety, Kuipers, Fowler & Bebbington 2002; Gumley & Schwanneur, 2006). Further models implicate insecure attachment on the pathway from childhood adversity to paranoid thinking (Bentall & Fernyhough, 2008); and in the development of low self-esteem, which along with an external attribution bias may lead to threat beliefs and ultimately to paranoia. However, the results of

this review do not support the importance of insecure attachment as a mediator. There are currently no high quality studies in clinical populations, and non-clinical samples consistently find little evidence of mediation. This corresponds with findings in one previous review (Alameda et al., 2020), but conflicts with Williams et al (2018) who claim there is consistent evidence to support the role of attachment as a mediator. Effects indeed are consistent, but trivial, and this serves to highlight the risk of synthesising evidence without regard for effect magnitude over more simplistic significance values. It may be worthwhile for future studies to consider how attachment may instead function as an interim step in a complex multiple mediator model rather than continue to pursue evidence of outright mediation.

2.8.2 Clinical implications

Given that the findings here appear to support cognitive models of psychosis, it may be the case that traditional cognition-focused interventions are the most useful for individuals with a diagnosis of psychosis who are experiencing positive symptoms, and who have a history of childhood trauma. There is already an evidence base finding support for the benefits of cognitive behavioural therapy delivery for a number of psychosis populations using a variety of delivery formats: individuals at high risk of developing psychosis (Hutton & Taylor, 2014); individuals with a clinical diagnosis (Turner, van der Gaag, Karyotaki & Cuijpers, 2014); individuals who have been considered to have 'medication resistant' psychosis (Burns, Erickson & Brenner, 2014); brief CBT for psychosis (CBTp) (Naeem et al., 2016; Hazell, Hayward, Cavanagh & Strauss, 2016) and group CBTp (Lecomte, Leclerc & Wykes, 2012). Whilst the effectiveness of CBT for psychotic symptoms, distress and quality of life, are still disputed by some (Jauhar et al., 2014; Laws et al., 2018) the findings from this review support the importance of cognition as a target for intervention.

Conversely, interventions with an attachment-focus may require reconsideration. Some researchers recommend that the attachment profiles of patients can be utilised to indicate which types of therapy may be of greatest benefit (Mallinckrodt, 2000; Berry, Barrowclough & Wearden, 2007). It is suggested that difficult experiences have an impact on working models

of interpersonal attachment, and that the beliefs and emotions surrounding these may form good targets for psychological intervention (Mallinckrodt, 2000; Berry, Barrowclough & Wearden, 2007). At present, the evidence from this review does not support the role of attachment as a causal mediator in the pathway from trauma to psychosis. However evidence does suggest an association between insecure attachment and positive psychotic symptoms (Lavin, Bucci, Varese & Berry, 2019; Carr, Hardy & Fornells-Ambrojo, 2018; Berry, Barrowclough & Wearden, 2007). So whilst it may not act as a causal mediator, maintaining an awareness of attachment traits and the potential therapeutic implications associated with this may still be important when delivering any psychological intervention. Developing strong therapeutic relationships can maximise adherence to therapy and minimise clinician-patient conflict (Barrowclough et al., 2001) whilst also providing a safe base from which patients can begin to challenge their difficult cognitive and self-esteem issues such as negative self-views (Freemen, Freeman & Garety, 2016) and self-critical inner dialogues (Hutton et al., 2013).

2.8.3 Implications for research

A number of limitations were identified within the existing studies. These limitations have implications for future mediation research, to ensure it is transparent and informative. Firstly, study design was problematic in a number of cases. Evidence is almost exclusively cross-sectional which limits any strong claims around causality. Based on the Bradford Hill criteria (Hill, 1965), this study has gathered evidence on the strength of associations by considering the magnitude of effect sizes, along with the consistency of findings and the plausibility, or degree to which findings align with existing knowledge. However, temporal sequencing and reversibility cannot be examined in cross-sectional data sets. In order to generate evidence for this, a review of experimental and longitudinal designs will be required, but at present these types of studies are scarce.

Almost half of studies were based on data from samples which presented a risk of bias, and sample characteristics were not always adequately described. The quality and reliability of measures was inconsistent, with some studies using single or few items to measure important

constructs. This carries a risk of inaccurate measurement, and also limits the comparability of findings across studies. Future research should look to move beyond cross-sectional designs, and test potentially mediators in an experimental paradigm. Interventionist-causal research (see Chapter 1 and Chapter 4) would enable data to be collected about whether the measured mechanisms perform causal functions. Study measures should be carefully considered, and selected based on their validity and reliability; samples should be recruited in unbiased ways wherever possible; and studies should perform and report sample size calculations to demonstrate their research has adequate power to detect the effects of interest.

Ultimately, too few studies are pre-registered in the public domain prior to their conduct. This leaves researches with the freedom to interpret and report their results without a framework which is pre-specified and bound by their genuine intentions. Although effect sizes in this area are almost exclusively null to small, the lack of pre-registration raises questions around data handling. It therefore cannot be ruled out that some of the reported effects were discovered by chance, and subsequently reported as if the discovered effect had been the original research target. Pre-registration enables researchers to demonstrate their rigor and transparency, whilst also helping to minimise repetitive research into areas which produce null findings. All future studies should endeavour to pre-register their work, and make this explicit in their reports.

Future research should also aim to clarify the definition of terms used to describe variables to ensure findings are comparable across studies and populations. Attachment in particular is poorly defined throughout the literature, and measures refer to similar constructs under different titles (Berry, Barrowclough & Wearden, 2007). To avoid inconsistencies between studies, consensus should be reached regarding the definition and gold standard measure for such variables. If this proves to be impossible, contentious terms should be described and analysed with a broad awareness of differences throughout the wider literature.

Quality parameters including transparency around attrition rates, the handling of missing data, and potential confounding factors require significant improvement, as do the overall reporting

of results. The most prominent challenge in this review was finding a method to facilitate the comparison of effects across studies when reporting varied so widely. It is not currently the norm to report effect sizes in mediation research (Miočević, O'Rourke, MacKinnon & Brown, 2018), and in many cases only coefficients and significance values were offered. To improve future evidence synthesis, mediation research should endeavour to present results in a standardised manner which is meaningful to the study itself, but also comparable across wider research. This variability in reporting has thus far concealed the extent of trivial effects, presenting the illusion that mediators of trauma and psychosis are supported by stronger evidence than is truly the case. Understanding which variables mediate this pathway is highly important to the development of effective future treatments for individuals with a diagnosis of psychosis, and those at increased risk of developing the condition. Transparent, high quality research is essential to enhance knowledge in this area and without this there is a risk that treatment development will be delayed.

2.9 Limitations

Whilst this review has attempted to synthesise effects and transparently assess the state of current evidence, it is not without limitations. All database searches, initial title and abstract exclusions and the majority of full-text exclusions were undertaken by one researcher. Uncertainties were discussed with a second researcher and an inclusive approach was maintained throughout. In the case of uncertainty, articles were retained until ineligibility was confirmed. This required two authors to agree the exclusion, for reasons including lack of available information, or following failed attempts to obtain information from the authors.

Only one third of quality ratings were cross-checked by another researcher. Agreement was substantial and discrepancies were discussed until consensus was reached, and decisions were checked with a third reviewer. The source of discrepant judgements was considered in terms of the clarity and specificity of the AHRQ criteria. Refinements were made and the remaining articles rated again by the original researcher to make any necessary changes.

The categories of variables were devised through discussion between researchers. Another review may place the same articles into different groupings, and this would likely have an impact on the distribution of effects and quality ratings per group. Category titles were intended to be broad and inclusive, but distinct enough to allow contrast and comparison of different putative mechanisms across the range of psychological mediators which have been studied to date.

In some papers it was challenging to extract or convert the information required. Efforts were made to favour simple single mediator models over multiple mediation models to enable more direct comparison between studies. Some of the reported statistics required conversion between different effect size metrics. Reliable methods for converting between effect sizes were sought in the literature, however these extra steps will have increased the likelihood of human error. All conversions and analyses were double-checked by a second reviewer. In some studies assumptions had to be made or approximate estimates derived using the best methods available to the reviewer. The methods used are summarised above and in Figures 2.3 and 2.4, and are reported in detail in appendix 9. Transparency around the data used and conversions performed should assist the reader with interpretation, replication and comprehension.

2.10 Conclusion

This review highlights the relative weakness of current evidence for the effects of mediators operating on the pathway from childhood trauma to psychosis. Whilst cognitive factors and post-traumatic stress symptoms and dissociation have well established evidence bases, and small but consistent effects, other categories have significantly less supporting evidence. Attachment style, and mood and anxiety do not have evidence supporting their role as mediators, whilst social defeat, and emotion regulation and stress sensitivity require a great deal more evidence to be generated before judgements can be made about their potential importance. Currently research is limited by poor quality design and poor reporting of results, and studies are almost exclusively cross-sectional. In order to develop this area of research,

future studies should look to test potential mediators in experimental research designs, and all studies should pre-register their hypotheses and protocol in the public domain in advance to improve scientific rigour and transparency in the field, and increase the robustness of their findings

Chapter 3 – Methodology for study one - the development and testing of a mediation model of childhood trauma and subclinical psychotic experiences in a large online general population sample

3.1 Overview of chapter

The previous chapters have outlined the research context, and established the state of current evidence. They have also identified a number of issues with typical research methodology. This chapter will describe the methodological protocol for an online study in a general population sample, which aims to develop a theoretical model of pathways between childhood adversity and sub-clinical psychosis using a robust two-stage analysis process.

This chapter will first describe the rationale for the study including why specific variables have been selected, and it will justify the research design. The study aims are outlined and details of the recruitment, target sample and procedures are described. The assessment measures are listed and details about the pre-registration of the study protocol are presented. Finally the three stages undertaken to develop, refine and test the model are described in detail.

3.2 Research rationale

Taking into consideration the findings from the systematic review (Chapter 2), few variables currently have extensive evidence supporting their importance as mediators on the pathway from childhood trauma to psychosis. This empirical research study was designed to gather data from a general population sample, using a large online survey, in order to generate a model hypothesis of how childhood trauma may lead to subclinical psychotic symptoms through relevant mediators. Numerous studies have investigated mediation models to date, however it was clear in the systematic review that the quality of both the methodology and reporting varied widely across studies, and no studies described a robust two-stage method where models were first developed, and then later tested, as is used here.

In order to build upon the findings from the review, variables which have been less frequently investigated were selected for inclusion. Cognitive factors are already well researched, as are dissociation and PTSD symptoms, and although effect sizes appear to be small, evidence is fairly consistent in support of their importance. The latter have been more widely studied in clinical samples and are arguably more relevant to this population. Instead, variables were selected from the categories where the evidence of mediation effects was less clear, or those which have had little research attention to date. These are briefly described below.

Insecure attachment was selected for inclusion as it is frequently included in theoretical models of psychosis, however current evidence from the review suggests that attachment variables do not mediate the relationship between trauma and psychosis. Associations have been found between insecure attachment and subclinical positive symptoms (Berry et al., 2006; Carr, Hardy & Fornells-Ambrojo, 2018) and mediation studies have found some small effects in non-clinical samples (Goodall et al., 2015; Sheinbuam et al., 2014), however evidence is inconsistent and other research has not found attachment factors act as mediators mediate (Ashford, 2010; Sitko et al., 2014). Further studies have not found attachment to be predictive of either positive symptoms (MacBeth et al, 2011) or symptom recovery (Gumley et al., 2014). As the evidence is currently inconsistent it was important to gather further data here.

Mood and emotion regulation have been examined by a small number of studies but as with attachment, findings remain inconsistent. The review found some evidence that mood factors may function alongside other variables in multiple mediator models, and as mood factors are relatively common across a spectrum of severity in the general population it was pertinent to include them here. Multiple studies included in the review drew their evidence from the APMS data sets and therefore it is also essential to test these potential mediators in a novel general population sample.

Emotion regulation has been examined by higher quality studies, however these are few in number, and evidence is therefore still lacking. Childhood trauma has been found to be associated with elevated emotion dysregulation in both clinical and subclinical samples (Oorschot et al., 2013; Liu, Subramaniam, Chong & Mahendran, 2019; Pries et al., 2020) and dysregulated emotion has been found to predict symptom severity (Strauss et al., 2013), however investigation into its potential role as a mediator is sparse.

Alexithymia, a difficulty with identifying and verbalising emotional states, along with a tendency towards very concrete, externally oriented thinking (Sifneos, 1972; Suslow & Donges, 2017; Lopez-Munoz & Pérez-Fernández, 2020) has yet to be studied as a potential mediator. Alexithymia has been associated with affect dysregulation (McLean et al., 2006); higher levels of anxiety and depression (Bagby et al., 2020); greater severity of negative symptoms (van't Wout et al, 2007; Stanghellini & Ricca, 1995) and subclinical delusion-proneness (Larøi, Van der Linden & Aleman, 2008). It has also been found to positively correlate with both childhood trauma (Seghers, McCleery & Docherty., 2011; Joukamaa et al., 2008; Bermond et al., 2008; Zlotnick, Mattia & Zimmerman., 2001; Berenbaum 1996) and insecure attachment (Seghers et al., 2011; Goldsmith & Freyd, 2005). This evidence along with the identification of alexithymia as relevant to clinical individuals with a diagnosis of schizophrenia (O'Driscoll et al, 2014), mark it out as a factor worthy of further investigation as a potential mediator.

Although cognition and PTSD have supporting evidence of their mediation effects, related variables were selected for inclusion here. Hypervigilance was included as a subclinical factor relevant to PTSD. This is a state of alertness, or excessive attention to and awareness of surroundings, motivated by the perception (or fear) of risk of harm (Alsawy, Wood, Taylor & Morrison, 2015). The majority of research into PTSD symptoms as a mediator has been undertaken in clinical samples however it may be more appropriate to measure hypervigilance in a general population sample who do not meet criteria for PTSD. The data gathered may be useful in indicating whether early PTS symptoms are relevant to the trauma-psychosis relationship in non-clinical individuals.

Despite the evidence supporting the role of cognition as a mediator, only two studies to date have considered metacognition as a potential mechanism (Østefjells et al., 2017; Goldstone 2012). As beliefs about the self and others were highlighted as particularly important, it was of interest to assess whether subjective thoughts about beliefs could also be relevant, potentially as a precursor to symptom development. In this study it was beliefs about paranoid thoughts which were measured.

Establishing a more comprehensive evidence base of factors which do and do not mediate the trauma-psychosis relationship is vital to the development of effective treatments. Greater knowledge and understanding about the causal sequence following the experience of early trauma will highlight potential targets for interventions which could be administered at an earlier time point, thus reducing or preventing the development of severe symptoms and associated distress. By investigating links in a general population, hypotheses can be generated and tested before collecting data from clinical samples which can be more challenging to recruit.

In order to access an adequately large sample size, the research was conducted online in a general population sample. The online platform enables participants to remain anonymous, which can be advantageous when asking questions about sensitive topics, and can promote honest disclosure (Bouchard, 2016). Although the study is cross-sectional in nature, and therefore cannot make causal claims, the data gathered were subjected to a robust dual-stage analysis to test the hypothesised model in a second data sample. This sets the study apart from existing mediation research in this area, none of which describes subsequent confirmatory testing of the reported models. A large proportion of mediation studies make use of data from large surveys (e.g. the Adult Psychiatric Morbidity Survey, 2007) yet without prior specification of aims and hypotheses, it is difficult to rule out potential data mining or the reporting of models which are over-fitted to the specific data sample. This could be overcome by pre-registering the study in advance, however this is seldom undertaken.

This study used exploratory analytical methods in the first data sample to identify salient pathways between variables. Network analysis was used to identify patterns in the raw data. This is a data-driven method which visually maps the connections between variables, and offers indices of their relative importance. This was followed by structural equation modelling, using the information gathered from the network models to develop a full exploratory model with the additional significance testing of a hypothesised indirect (mediation) path. Following this phase the hypothesised model was pre-registered in the public domain with details of the study hypotheses and the planned analyses. Pre-registration took place before the second data sample was collected. The pre-registration of a study protocol prevents any post-hoc manipulation of aims and outcomes, misrepresentation of results, of misleading presentation of exploratory findings as confirmatory.

The procedure for the exploratory and confirmatory phases are described in detail below. The findings from both phases are presented in Chapter 5, along with the interpretation of results and a discussion of the implications of the unique methodology can be found in Chapter 7.

3.3 Aims of the research

This study aims to determine which factors mediate the relationship between childhood adversity and subclinical paranoid symptoms in a general population sample. To achieve this, exploratory analyses will be undertaken to firstly hypothesise a structural model, which will then be tested in a subsequent data sample. Secondly, confirmatory analyses will be undertaken in this second data sample. If the model is not well replicated, or can be improved, a modified model will be specified. Finally, the model will be tested on the combined dataset and re-specified for further testing in a future sample, if modifications are required.

3.4 Method

3.4.1 Study design summary

This study used a series of online questionnaires to measure childhood trauma, subclinical psychotic symptoms and a number of potential mediators in a self-referring general population

sample. The study was undertaken in two parts. Data from the first sample (the 'exploratory sample') was used in exploratory network analysis to identify patterns in the data. Findings were then used to develop a structural equation model based on hypothesised mediation mechanisms, which could then be tested in a second sample of data (the 'confirmatory sample'). Exploratory analyses were completed before the confirmatory sample was collected. The structural model was tested in the confirmatory sample for goodness of fit and replicability of the indirect path. Potential model modifications were examined and applied where they had both a positive effect on the model, and a sound theoretical basis for inclusion. A final model was then tested on the combined data sets.

3.4.2 Recruitment and sampling

3.4.2.1 Participants

Participants were recruited online, primarily using social media adverts on Facebook and Twitter, and by circulating the study recruitment poster via email. NHS contacts, university postgraduate communities and relevant charitable organisations were sent the study poster and the link to the survey, and were encouraged to share widely with their network.

A study website (https://emotioninpsychosisstudy.wordpress.com/) was created to provide further information to potential participants. The link to the survey interface was only available via the participant information sheet, to ensure participants had downloaded the information. A copy of this information sheet is available in appendix 12. A form of snowball sampling was used which involves each participant helping to expand the reach of the research by passing on the study details to their networks or by recommending individuals who may be eligible and willing to take part (Vogt & Johnson, 2011; Cohen & Arieli, 2011). In addition to emails and social media posts, research related tags were used on Twitter in an attempt to expand the accessibility and reach of the study.

3.4.2.2 Inclusion/exclusion criteria

As the study was interested in past experience of childhood adversity, participants were required to be minimum 18 years of age. They were required to be resident in the UK, and be able to read and communicate in English.

The questionnaire measures had been validated in English so restricting the sample to UK residents avoided any confusion over the cultural-specificity of any questions. Due to limited resources, it was not possible to offer the survey in translation. The often uncontrollable snowball effect of online research (Jack & Egan, 2015; Waters, 2013) meant the survey would be accessible by those outside of the UK, but the support services signposted in the participant debrief sheet were UK-specific. It was not possible to verify whether comparable services would be available in other locations, and thus for ethical reasons participants outside of the UK were asked to self-exclude.

Potential participants were also asked to self-exclude if they had any developmental or learning disabilities, or organic brain impairments such as dementia or acquired brain injury. As a number of the topics in the survey were considered sensitive in nature, participants who could be considered particularly vulnerable to distress, either with a history of, or ongoing psychiatric illness were asked to self-exclude.

Following the protocol of Moritz et al (2016), any participants who entered identical values throughout the questionnaires were excluded. Participants who contributed less than 50% data to any one of the questionnaires were excluded from the analysis of that measure. Multiple submissions from the same computer were also excluded. Such submissions were identified using the computers internet protocol (IP) address which were recorded by the Novi Survey platform.

3.4.3 Sample Size

3.4.3.1 Exploratory sample

Minimum sample size for structural equation modelling is often based on 'N per parameter', however in exploratory modelling, the number of parameters to be estimated is not clear until the latter stages of model development. This study comprised 10 questionnaire measures with a total of 32 subscales, each of which was treated as a separate variable. There has been debate in the literature over whether an absolute N is required, or whether a subject-to-variable (STV) ratio is preferable (Wolf & Brown, 2013). A large scale review of factor analytical studies suggested a minimum STV ratio of 3.25:1 with almost 88% of the 60 included studies using a ratio of 5:1 or above (Henson & Roberts, 2006). A further review found the majority of studies (over 48%) used STV ratios of between 2:1 and 10:1 (Costello & Osborne, 2005). In SEM studies there are a number of suggested ways to compute sample size based on the number of participants per parameter, however these vary widely from a conservative five per parameter (Hair et al., 1998) through 10 (Jackson, 2003) up to 15 (Bentler & Chou, 1987). A widely used rule of thumb in SEM is a minimum sample of 200 participants (Kline, 2011). With 32 variables, the minimum requirement for a 5:1 ratio was 160, and the maximum for a 10:1 ratio was 320. However, the requirement to recruit two distinct samples for analysis meant targets had to be reasonably constrained to avoid under-recruitment in the latter sample.

The nature of online research often results in missing data. Hoerger (2010) suggests up to 10% of participants may drop out immediately after commencing the study, and cautions researchers to potentially expect a further 2% attrition per 100 questions in the survey. Therefore a target sample size of 220 was used for the first round of data collection as satisfies suggested minimum requirements for exploratory and SEM research, whilst taking into account the potential attrition rate experienced in online psychological research.

3.4.3.2 Confirmatory sample

A sample of 223 responses was gathered for the initial exploratory sample, with 205 of those completing consent and demographic information. However individual survey completion rates

ranged from N=169 -190. This was lower than anticipated and comprised 82-92% of the eligible sample. In order to buffer against further data loss, the target sample for the confirmatory phase was increased to 240.

3.4.4 Procedure

A series of questionnaires were collated into a large online survey. Participants were directed to the study website where they could access study information and complete the consent form and demographic questionnaire. Participants then completed the standardised measures. The consent form was the only page where responses were mandatory and therefore participants were free to miss out any questionnaire items which they did not wish to provide an answer for. A debrief sheet was provided to all participants who completed or exited the survey, detailing sources of support should they feel distressed by the content of any of the questions.

3.4.5 Assessment measures

3.4.5.1 Participant demographics

The demographic information was collected using an electronic multiple-choice questionnaire. Age, gender, ethnicity, years in education, employment status and relationship status were recorded. This information was used to characterise the sample. The use of snowball sampling introduces potential for bias so demographic details were used to quantify how generalisable the results may be, and also to compare the exploratory and confirmatory samples for any systematic differences which may complicate the interpretation of results. Details were also used as covariates in the analyses to rule out confounding due to selection biases associated with age, gender and sociodemographic factors.

3.4.5.2 Standardised research measures

All participants completed the surveys using the Novi Survey platform which does not allow for random questionnaire sequencing so all participants completed measures in the same order. To minimise burden and encourage completion, the short-form versions of measures

were used wherever available and when validity and reliability were demonstrated to be comparable with the full length questionnaires. The measures are described below.

3.4.5.2.1 Independent variable - childhood trauma

Two measures of trauma were used to collect information on interpersonal adversity in childhood. Subscales from these measures were treated as independent variables in the analyses.

Child Abuse and Trauma Scale (CATS), Sanders & Becker-Lausen (1995).

CATS is a 38 item self-report questionnaire which measures the occurrence of adverse childhood events falling into three categories: sexual abuse (6 items); negative home environment (14 items) and punishment (6 items). The remaining 12 items load only onto the total score, which comprises all 38 items. Items are rated on a five point scale from 'never' (0) to 'always' (4). The items are worded 'gently' to avoid causing distress despite asking about emotive topics (Sanders & Becker-Lausen, 1995). For this reason CATS was considered useful for online research where participants will be completing questions without direct access to support.

CATS has been used widely in research and has been validated in general population and clinical samples (Sanders & Becker-Lausen, 1995; Kent & Waller, 1998). It has been found to have good internal consistency (Cronbach's alpha = .63 - .90); good test-retest reliability (r = .71 - .91) and has been shown to correlate well with depression and dissociation self-report scores - other factors which have been reliably linked to childhood adversity (Sanders & Becker-Lausen, 1995).

Comprehensive Child Maltreatment Scale for Adults (CCMS-A), Higgins & McCabe, 2001.

The CCMS-A is a self-report questionnaire about adults' perceptions of their childhood experiences before the age of 13, and the frequency with which they occurred. The full measure contains 5 subscales; sexual abuse, physical abuse, witnessing family violence, psychological maltreatment and neglect. Only the latter two subscales were used to avoid

duplication with CATS. The psychological maltreatment and neglect subscales contain three items each and are scored on a five point scale from ‘never/almost never’ (0) to ‘very frequently/always’ (4).

The measure was originally developed and tested in an adult community sample. The psychological maltreatment and neglect subscales were found to strongly correlate with CATS negative home environment and sexual abuse subscales (r = .70-.80, all p< .001) showing good concurrent validity (Higgins & McCabe, 2001). Reliability was also shown to be high (Cronbach’s alpha = .76 - .84). Test-re-test reliability at 6-8 weeks was acceptable for the neglect subscale (.62) and high for psychological maltreatment (.84).

3.4.5.2.2 Dependent variable - sub-clinical psychotic symptoms

A number of measures have been created or adapted to record subclinical psychotic symptoms in the general population. Measures assessing symptoms across a spectrum of severity were selected to more accurately capture the range of experiences across the general population sample. Subscales from these measures were treated as dependent variables in the analyses.

Community Assessment of Psychic Experiences (CAPE-15), Capra et al. (2015).

CAPE-15 is a self-report questionnaire in which participants indicate lifetime experience of psychotic phenomena. It consists of 15 items which are rated on a four point scale from ‘never’ (0) to ‘nearly always’ (3). Participants indicate how often, if ever, each of the experiences has occurred.

The CAPE-15 is a short version of the full CAPE-42, which focuses exclusively on positive symptoms. Experiences are categorised into three subscales; persecutory ideation (5 items), perceptual abnormalities (3 items) and bizarre experiences (7 items). This three factor structure has been tested extensively and has been found to be replicable and reliable across multiple languages and in student, community and high-risk samples (Schlier et al., 2015; Mark & Toulopoulou (2016); Haque, Jacobson, Bowie & Munhall, 2017). Internal consistency

has been found to be high (Cronbach's alpha = .79) with a stable internal structure despite being a short form version (Capra et al., 2015).

General Paranoia Scale for adults (GPS), Barreto Carvalho et al. (2014).

The GPS is a 20 item self-report questionnaire, which asks participants to indicate the frequency of paranoid thoughts. The questionnaire was originally created by Fenigstein & Vanable (1992), loading on to a single paranoia factor, however the revised three factor structure was used in this study (Barreto Carvalho et al., 2014). The revised subscales are mistrust feelings (8 items); persecutory ideas (8 items); self-deprecation (4 items). Items are scored on a five point scale from 'never' (1) to 'always' (5). Higher scores indicate more frequent experience of paranoid thoughts.

The measure was devised specifically for use in the general population, but the revised version, originally tested in adolescents has subsequently been tested and used in community adults and clinical samples (Barreto Carvalho et al., 2017; Barreto Carvalho et al., 2018). The three-factor structure is more indicative of a paranoid-thinking hierarchy from general thoughts of distrust towards others, up to more severe fears of persecution (Freeman et al., 2005). Subclinical samples tend to endorse more mistrust and self-deprecating items, whereas clinical samples have been found to endorse more severe items on the persecutory ideas subscale (Barreto Carvalho et al., 2017). This spectrum of endorsement is also indicative of the cross-population effectiveness of the measure, and of the discriminant validity of the subscales.

Internal consistency has been found to be high in adolescent and adult samples with subscale alpha of; mistrust = .79 -.84; persecutory ideas = .84 -.90 and self-deprecation = .71 -.72 (Barreto Carvalho et al., 2014, 2017, 2018). The original study found Cronbach's alpha of .84 (Fenigstein & Vanable, 1992).

3.4.5.2.3 Potential mediating factors

Potential psychological mediators were identified from the existing research literature as detailed in the study rationale, above. The questionnaires selected to measure each of these variables are detailed below.

Brief Hypervigilance Scale, Bernstein et al. (2015)

The BHS is a five-item scale originally developed for use in an undergraduate student sample using items from the 52-item Hypervigilance Questionnaire (Knight, 1993). Items are scored on a five point scale from 'never true' (0) to 'always true' (4).

Internal reliability had been shown to be good (Cronbach's alpha = .81) and the five items load on to a single 'hypervigilance' factor explaining 57% of variance (Bernstein et al., 2015). Convergent validity was moderately related to PTSD symptoms and positively related to all 6 forms of trauma experience measured by the Brief Betrayal Trauma Survey (Goldberg & Freyd, 2006).

Depression, Anxiety & Stress Scale (DASS-21), Lovibond & Lovibond (1995)

The DASS-21 is a self-report questionnaire, which asks participants to rate the severity of various experiences over the past week. The 21-item scale contains three subscales; depression (7 items); anxiety (7 items) and stress (7 items), all rated on a four point scale from 'never' (0) to 'almost always' (3). The DASS-21 is the short form version of the full 42-item DASS. Both versions have been tested in multiple large sub-clinical samples (Imam, 2008; Henry & Crawford, 2011; Sinclair, Sifert & Slavin-Mulford, 2011 Osman et al., 2012), with the short form being found to have higher mean loadings and fewer cross-loading items in contrast to the full version, resulting in a cleaner overall factor structure (Antony et al., 1998). The DASS-21 has shown strong correlations with the Beck Depression Inventory (r =.79) and the Beck Anxiety Inventory (r =.85) (Antony et al., 1998).

Some studies have found a single common factor to be underlying depression and anxiety (Osman et al., 2012); others have identified a four factor solution with a 'psychological distress'

or ‘negative affect’ factor in addition to the original factors (Henry et al., 2005). However, the original three factor solution shows good internal consistency across studies with subscales achieving Cronbach’s alpha scores of .88-.94 for depression; .82-.87 for anxiety and .90-.91 for stress (Antony et al., 1998; Crawford & Henry, 2003; Henry et al., 2005).

Cognitive Emotion Regulation Questionnaire – short version (CERQ-short), Garnefski & Kraaij (2007)

The CERQ-short is an 18-item version of the original 36-item CERQ (Garnefski, Kraaij & Spinhoven, 2002). It seeks to measure cognitive strategies used by participants to regulate emotions in stressful situations. Items are measured on a five point scale from ‘almost never’ (1) to ‘almost always’ (5). The short form reduces the number of items per subscale from four to two, and was specifically developed for use in clinical populations or in large psychometric batteries when time and space are at a premium. There are nine subscales, which can be categorised into adaptive emotion regulation strategies; ‘acceptance’, ‘positive refocusing’, ‘refocus on planning’, ‘positive reappraisal’ and ‘putting into perspective’; and maladaptive strategies; ‘self-blame’, ‘rumination’, ‘catastrophizing’, ‘blaming others’.

The CERQ-short has been found to have acceptably high reliability with alpha scores for subscales ranging from .62 - .87 (Garnefski et al., 2006, Garnefski & Kraaij, 2007). Further studies have found the nine factor solution to be better supported in the CERQ-short than in the full CERQ, however high correlations (>.70) between factors have been noted indicating overlap in the measured variance. It has been suggested that some of the latent variables would be better collapsed together due to this conceptual similarity (Ireland et al., 2017).

Psychosis Attachment Measure (PAM), Berry et al. (2006)

The PAM is a self-report questionnaire which asks participants to report on their thoughts and feelings about their interpersonal attachment relationships. It contains 16 items across two subscales: anxious attachment (8 items) and avoidant attachment (8 items), rated on a four point scale from ‘not at all’ (0) to ‘very much’ (3). PAM was developed from existing attachment

measures with the removal of items asking about romantic relationships, thus increasing its relevance across both clinical and subclinical populations (Brennan et al., 1998). Higher scores are indicative of higher anxious/avoidant attachment.

A review of measures of attachment in psychosis concluded that both subscales show good internal consistency ranging from .70 to .86 for the anxious subscale, and .60 to .91 for the avoidant attachment subscale across nine included studies (Gumley et al., 2014). Subscales were not found to correlate highly with each other, suggesting they measure discrete factors (Berry et al., 2008). Subscales were also significantly associated with similar dimensions in the Relationship Questionnaire, demonstrating good concurrent validity (Berry et al., 2008).

Beliefs about Paranoia Scale (BAPS) short form, Gumley, Gillan, Morrison & Schwannauer, (2011)

The BAPS is a self-report questionnaire measuring metacognitive beliefs about paranoid thoughts. The short form version was used in this study which contains 18 items loading on to three subscales; paranoia as a survival strategy (6 items); negative beliefs about paranoia (6 items); normalising beliefs about paranoia (6 items). Items are reported on a four point scale from 'not at all' (1) to 'very much' (4). Higher subscale scores indicate greater endorsement of each type of belief.

Internal consistency has been found to be good and stable in both subclinical and clinical samples with Cronbach's alphas ranging from .87-.89 for survival strategy; .87-.88 for negative beliefs and .89-.93 for normalising beliefs (Gumley et al., 2011, Murphy et al., 2017).

Toronto Alexithymia Scale short form (TAS-20), Bagby, Parker & Taylor (1994)

The TAS-20 is a self-report questionnaire designed to measure participants' ability to identify and describe their own emotional states, and the emotions of others. The scale contains 20 items, which load on to three factors: difficulty identifying feelings (DIF); difficulty describing feelings (DDF) and externally oriented thinking (EOT). Items are measured across a five point

scale from ‘strongly disagree’ (1) to ‘strongly agree’ (5). Higher scores indicate greater difficulty in each area.

Despite the TAS-20 being the most widely used measure of alexithymia, translated into 18 different languages and validated cross-culturally (Taylor et al, 2003), the factor structure has been highly contested (Grabe et al., 2009; Parker et al., 2003; Taylor et al., 2003; Gonzales-Arias et al, 2018; Bagby et al., 2020). The original authors found the current three-factor structure to be superior to a unifactorial model, or a two factor model (in which DIF and DDF were collapsed into a single factor). Cronbach’s alpha was .81 for the overall scale with individual scales at .78 for DIF; .75 for DDF and .66 for EOT (Bagby et al., 1994). The three factor structure has been replicated across a number of general population studies, however different structures have been identified in clinical samples (Kooiman et al., 2002; Parker et al., 2003; Meganck et al., 2008) and studies have highlighted the instability of the EOT subscale (Bagby et al., 2020; Goerlich, 2018). Other research has indicated a preference for the two factor model described above (Muller et al., 2003; Güleç et al., 2009) whilst others have added a fourth factor, importance of emotional introspection, which explained greater variance (Franz et al., 2008). The majority of studies do however support the three factor solution, particularly in large community samples, so this structure will be used on this study, however results will be considered with these debates in mind.

3.4.6 Approval and pre-registration

The study was given a favourable opinion by the Edinburgh Napier University Ethics Committee. Following the exploratory analysis, and the development of a hypothesised model, the study method and hypotheses were pre-registered on the Open Science framework at: https://osf.io/k49pj/ before collecting the confirmatory data sample.

3.5 Analysis

3.5.1 Data preparation and missing data handling

Novi Survey holds answers as text responses, and does not allow for reverse scoring, so this was undertaken using Microsoft Excel. This also enabled screening of responses for exclusion purposes. The Excel data was then transferred into IBM SPSS Statistics for Windows, Version 26.0 (IBM Corp, 2019) for descriptive analyses and missing data screening. Participant records where consent and/or demographics were incomplete, and cases where no survey answers had been provided were removed. Participants contributing less than 50% data to any individual questionnaire were removed from the analysis for that measure.

Missing data is a common problem in research of this type. Cohen et al. (2013) suggest that a tolerance threshold of 10% missing values should be applied. Below this, a variable should be retained and missing data handled for inclusion in analyses. As the analysis here was of an exploratory nature, the maximum possible level of data was retained. There are several approaches available for handling missing data, each of which brings different issues. Listwise deletion is the simple removal of cases with missing items, regardless of whether the variable is included in the active analysis (Kline, 2011). Pairwise deletion only deletes the case when the variable with missing data is included in the analysis. However, both of these approaches can have a significant impact on sample size and pose a risk of bias (Byrne, 2016). Many statistical packages default to either listwise or pairwise deletion as many types of analysis require a complete data set for all included variables (Fox-Wasylyshyn & El-Masri, 2005).

Alternatively, imputation techniques can be used to preserve cases with missing values. Sample mean substitution can be used in cases of missing interval data, however this reduces the variance of the variable in question, and therefore impacts its correlation with other variables in the analysis (Byrne, 2016). Mean imputation is only recommended in cases where missing data is low (Roth, 1994). Expectation maximisation is a model-based technique which uses an iterative procedure to first estimate parameter values and then estimate missing data values based on these. It leads to less bias than mean imputation, however as with any single

imputation technique it treats imputed values as if they are 'known' rather than estimated (Fox-Wasylyshyn & El-Masri, 2005).

Multiple imputation (MI) circumvents this disadvantage. MI estimates missing data based on multiple simulated complete data sets. These are based on the original data and missing values are filled using a random number generator. Ordinarily this is undertaken three to five times, after which each complete data set is analysed and the results pooled (McCleary, 2002). This method is more reflective of the genuine uncertainty of missing values, whilst still making use of all available original data.

Missing data here were investigated using Little's Missing Completely at Random (MCAR) test to identify whether data could be assumed to be missing at random. Mean imputation was then used to complete subscales with few missing values (<10%). Where higher instances of missing data were found, MI was used at the SEM stage to include all possible participants. The network analysis program (JASP) does not support the use of imputed data and therefore cases with higher levels of missing data were excluded from the network modelling analysis, but were retained for the SEM analyses.

Summed subscale scores were tested for normality in SPSS. Histograms and Normal Q-Q plots were visually inspected. Variables deviating from the expected bell-curve (histogram) or line of best fit (Q-Q plots) were identified. Descriptive statistics tables were inspected for skewness and kurtosis values to identify potential outlier variables. Established cut off scores (skew values of >2 or <-2 and kurtosis values of >7 or less than -7) were used to identify non-normal distribution of data (Byrne 2010, Hair 2010). Results of normality testing highlighted outlier variables which were considered for exclusion, and confirmed the requirement for non-parametric analyses to be pursued in both data samples.

3.5.2 Phase 1 - Model development

3.5.2.1 Network Analysis

Network analysis is a data-driven method of investigating connections between variables in a complex arrangement. The network reveals the pattern of interactions between the variables by estimating a statistical model directly from the data which highlights strong and significant pathways using a partial correlation network (PCN) (Epskamp, Borsboom & Fried., 2018). In the output, measured variables are represented as 'nodes', depicted as circles, and the estimated statistical relationships between them are represented by 'edges', depicted as lines of varying boldness to represent the strength of connection.

The PCN shows the remaining connection between variables after controlling for all other known information (Epskamp & Fried, 2018). The partial correlations are similar to multiple regression coefficients, but have the advantage of being multi-directional, revealing what predicts the independent variables as well as the specified dependent variable, as in multiple regression. They can also indicate, but not confirm causality. The pathway between two variables can only be non-zero, and thus retained in the model, when the variables are causally dependant on each other (in either direction), or they are mutually caused by another variable in the network (Epskamp & Fried. 2018). PCNs can also reveal 'predictive mediation' where two variables are indirectly linked by a third, mediating variable (Epskamp & Fried, 2018). This would not be clear in multiple regression, and highlights why the PCN is more useful in exploratory analyses.

However, this ability to assess multiple pathways between all variables brings a degree of risk. The number of parameters to be estimated escalates quickly and in order to retain adequate power, the analysis may demand far more observations than are available (Epskamp, Borsboom & Fried et al., 2018). To remedy this, a regularization technique called 'least absolute shrinkage and selection operator' (LASSO) can be used (Tibshirani, 1996).

Regularization involves reducing the variance in the model, shrinking the small parameter estimates towards absolute zero, at which point they will drop out of the network. This reduces

the overall flexibility of the model, but also reduces the uncertainty, and thus returns a more conservative model with fewer edges making it clearer and easier to interpret (Epskamp, Borsboom & Fried, 2018; Bickel et al., 2006). Pathways which are sufficiently strong (i.e. different from zero) will be retained. The LASSO effectively selects the best fitting model whilst simultaneously estimating the model parameters.

The LASSO makes use of a tuning parameter to control the application of regularization, known as lambda (λ). This imposes a penalty to regulate how many parameters are used to estimate the network. When lambda is assigned a value of zero, the model is not penalised at all, but as lambda increases, fewer edges will be estimated and included (Wysocki & Rhemtulla, 2019). Several iterations of the model are estimated, using different values of lambda. The tuning parameter used in this study is the Extended Bayesian Information Criterion (EBIC) which has been shown to return models with good specificity (not estimating edges which are not included in the 'true' model) (Epskamp & Fried, 2018), and moderate sensitivity (accurate estimation of edges which are included) even when minimised (Wysocki & Rhemtulla, 2019). The EBIC has been found in some cases to produce inconsistent results using psychological data, as it can be influenced by the sample size and the characteristics of the population and data (Wysocki & Rhemtulla, 2019). However, alternative penalty parameters fall foul of similar issues and therefore as the EBICglasso estimation method has been most widely applied in psychology to date, this method was preferred.

The parameter estimates of the model will increase in accuracy with increasing sample size, and bootstrapping can be applied to further test the accuracy of the edge weights. This can be undertaken in parametric or non-parametric ways, depending on the data available (Epskamp, Borsboom & Fried, 2018). Non-parametric bootstrapping involves simulating several model data sets based on replacement values which can then be used to estimate the potential sampling distribution. Confidence intervals can then be estimated based on the minimum and maximum values of the bootstrapped samples. These CIs are not a significance test, but function to demonstrate the accuracy each edge weight in the model, and facilitate

comparisons between edges in terms of their accuracy (Epskamp, Borsboom & Fried., 2018). Parametric bootstrapping requires resampling with values from the original data and is contingent on the normality of that data, whereas non-parametric bootstrapping can always be applied (Epskamp et al., 2018).

Centrality measures are directly linked to edge weights, and imprecise CIs tend to also result in poor accuracy of these indices (Epskamp, Borsboom & Fried, 2018). Increasing the number of bootstraps should improve the accuracy of the CI estimates, and latterly improve the accuracy of centrality indices. Several indices are available which can be used to inspect the importance of each node to the overall network. There are briefly described below.

'Degree', also sometimes referred to as 'strength', totals all the edge weights (as absolute values) from the one-step connections associated with the node (Epskamp & Fried, 2018). More connections, and/or stronger connections will result in higher scores, which suggest the node is more directly influential, as opposed to operating through other mediators (Costantini et al., 2015). 'Closeness' quantifies the inverse of this. It is based upon the distance between the node in question and all other nodes in the network, computed using the inverse of the edge weights. This is summed for all of the shortest paths between the selected node and others in the network, and a score is assigned on this basis. Higher scores suggest the node may more rapidly influence or be influenced by changes in other closely connected variables. (Constantini et al., 2015; Epskamp & Fried, 2018). Betweenness' is a measure of how often the node features on the shortest paths between other nodes (Constantini et al., 2015). Higher centrality scores indicate the greater relative importance of a node to the overall network (Epskamp, Borsboom & Fried, 2018.).

In this study, network analysis was used to allow the data to drive the hypotheses about potentially salient mediators. Theory driven modelling in this area has thus far largely failed to reach consensus on which mediators are most important. Mediation models always contain some degree of specificity to the sample from which they are derived (MacKinnon, Fairchild & Fritz, 2007) but allowing the data to inform the hypotheses removes some of the risk that

salient connections may have been overlooked. By utilising network analysis it was possible to visualise which variables and connections were dominant within the sample, and thus structural models could be built and tested using this information.

Subscale data was imported into JASP version 0.13.1 (Jasp Team, 2020) for the above network analysis.

3.5.3 Phase 2 - Model refinement

3.5.3.1 Structural equation modelling

Further analysis was undertaken using lavaan Version 0.6-6 for SEM (Rosseel, 2012) in R version 3.6.3 (R Core Team, 2020). Structural equation modelling (SEM) is a theory-driven approach in which data models are generated using theoretical reasoning and applied to observed data sets to assess applicability and goodness of fit. Model hypotheses were generated using the salient connections indicated in the network analysis from which various models were built and tested to find the best fitting, and most parsimonious version.

SEM has a number of advantages over other multivariate techniques such as regression. It is a confirmatory technique where the pattern of variables must be pre-specified, enabling hypothesis testing. This sets it apart from regression which is a more descriptive process (Byrne, 2016). SEM takes into account measurement error which is regularly missed or ignored in other forms of multivariate testing. In the case of independent variable error in particular, failure to account for measurement error can have a significant impact on model accuracy (Byrne, 2016). SEM also allows multiple independent and dependent variables to be entered, and all pathways to be simultaneously tested in order to determine how well the model fits the data (Tarka, 2018).

SEM is being used in this study to investigate potential mediation pathways derived from the network analysis. There are a number of established methods for testing whether variables act as mediators between independent and dependent variables. Despite its shortcomings, the Baron & Kenny (1986) method is still the most widely used. This requires models to satisfy

several ‘causal steps’ before mediation claims can be made. The independent variable (IV) and dependent variable (DV) must be correlated, as must the IV and potential mediator (M). M must have an effect on the DV, whilst controlling for the IV. And finally the relationship between IV and DV must be either reduced (partial mediation) or disappear completely (full mediation) when the mediator is entered. This method has come under a great deal of scrutiny and subsequent criticism for failing to adequately test and quantify the magnitude of the indirect effect; for having low statistical power and for its inability to cope with models which have inconsistent mediation (Memon et al., 2018; Zhao et al., 2010; Hayes, 2009), all potentially resulting in inaccurate or misleading results.

Instead researchers suggest the strength of the mediator should be interpreted in terms of the magnitude and significance of the indirect effect, rather than the negation of the direct effect (Zhao et al., 2010). Having a zero-order relationship between IV and DV should not be considered essential, particularly when there is a possibility that effects may occur in opposite directions and effectively cancel each other out. For this reason Zhao et al (2010) delineate different types of mediation: ‘complementary’ in which the indirect and direct pathways have the same sign; ‘competitive’ in which the indirect path and the direct path have opposing signs; and ‘indirect only’ where there is an indirect mediating effect, but no direct effect. Consideration of these types of mediation should safeguard against missing effects which are present and important, but not immediately obvious using more traditional approaches.

In addition to this, applying Preacher & Hayes (2008) bootstrapping method should further strengthen the accuracy of the output. Bootstrapping in mediation analysis is similar to its application in edge weight accuracy testing (above). The original data set is resampled several times using case-substitution. All simulated data sets are based on the original data, but in a variety of iterations. Any case, once drawn, is added back to the pool and can be drawn again. For each resampled distribution the indirect effect is estimated and plotted, and the resulting total distribution should approximate the ‘true’ distribution of the indirect effect (Hayes, 2009). Regardless of the normality of this distribution, the top and bottom estimates can be discarded

to form 90% or 95% confidence intervals (R provides 90% CIs by default). Inspection of the CIs should reveal whether zero is included. If not, the researcher can be confident in the claim that there is an effect. Hayes (2009) recommends a minimum of 1000, but preferably 5000 bootstrap resamples are drawn for analysis. Bootstrapping is a powerful technique which relies on fewer assumptions than the causal steps approach. For the analysis here, potential indirect pathways were specified for bootstrap testing.

3.5.3.2 Model fit

Model fit was assessed using various indices to understand how well the data fit the model. These indices are described below, along with the threshold values applied when iteratively developing the exploratory model.

Firstly the chi-squared (X^2) statistic was required to be non-significant (Kline, 2011). The X^2 test is a test of significance which assesses whether the predicted model is significantly different from the observed data. It indicates the magnitude of difference between the expected covariance matrix, and the sample covariance matrix (Kline, 2011). Smaller values indicate a better fit, and non-significance is desirable as this would indicate that the model prediction does not significantly differ from the observed data. The test is considered useful in samples of 75-200, but is heavily influenced by sample size and is almost always significant in larger samples (N=400+) (Kenny, 2015). For this reason it is essential to also consider alternative fit indices.

Secondly the Comparative Fit Index (CFI) was used to compare the specified model to a baseline model. It is an incremental measure of model fit, which indicates the proportional improvement of model fit against the baseline (Hu & Bentler, 1999). The CFI is normed and so has a range of 0-1 and values of > 0.95 are considered a good fit, 0.90-0.95 as a marginal fit, and <0.90 as poor (Kenny, 2015; Hu & Bentler, 1999).

Thirdly, the Root Mean Square Error of Approximation (RMSEA) was used. This is an absolute measure of model fit. It has a known distribution and compares the sample data to an a-priori

model. It has been shown to suffer greater error in models with few degrees of freedom, or in small-N sample which can artificially inflate the estimate (Kenny, 2015). There are discrepancies in desirable cut-off levels in the literature. Kline (2011) suggests <0.09 as a good fit; Hu & Bentler (1999) consider <0.06 as a good fit; MacCallum, Browne & Sugawara (1996) suggest < 0.05 as a good fit and <0.01 as excellent. Confidence intervals for RMSEA are computed by default in R at 90%. Kenny (2015) suggests that the lower bound confidence value should include or be very near 0.00, and the upper value should be no higher than 0.08 as this would be indicative of precision in the point estimate. Wider intervals should be interpreted with greater caution.

Finally the Standard Root Mean Square Residual (SRMR) was used. This is another absolute measure of model fit which compares the sample data to an expected a-priori distribution. The SRMR is relatively independent of sample size (Chen, 2007). A value of zero would indicate 'perfect' fit, but again there is discord in the literature when offering rule of thumb cut-off scores. Kline (2011) indicates a value of less than 0.10 as essential, and under 0.05 as a good fit, whereas Hu & Bentler (1999) suggest anything less than 0.08 can be considered a well-fitting model.

For the analysis here, the most stringent cut off criteria for each fit index were applied. As the analysis is exploratory it is primarily concerned with identifying the best possible fitting model for testing in the subsequent data sample.

3.5.4 Phase 3 - Model Testing

The hypothesised SEM model was tested in the confirmatory data sample. The model fit was assessed, then modification indices were investigated to identify potential areas where the fit could be improved. Modification indices test all available parameters in the model and provide estimated parameter change (EPC) scores. Larger values indicate greater potential improvement in model fit, however reasoning must be applied to ensure model modification can be theoretically justified beyond the statistical implications.

3.5.4.1 Final model specification

The final modified SEM model was applied to a collapsed data set, using all data from both the exploratory and confirmatory samples. Further potential model modifications are discussed in the Results chapter, as are implications of the findings for future research.

Chapter 4 – Methodology for study two - an interventionist-causal randomised controlled trial of an emotion regulation skills intervention in a sample of individuals with a diagnosis of psychosis who were experiencing paranoia

4.1 Overview of chapter

This chapter describes the methodological protocol for a randomised interventionist-causal trial of an emotion regulation intervention in a sample of people with a diagnosis of psychosis who are currently experiencing paranoia. Following an interventionist-causal design (Kendler & Campbell, 2009; Pearl, 2000), the study intervention attempted to isolate and change emotion regulation as a potential mediating mechanism between the experience of childhood adversity and paranoid symptoms and associated distress. Outcomes included testing whether the intervention was successful in changing the target mechanism, and whether the groups experienced change in paranoid symptoms.

This chapter briefly describes the proposed relevance of emotion regulation to psychosis, and more specifically paranoia. It goes on to introduce the interventionist causal research paradigm and the novel design used in this study, including a description of the experience sampling methodology which was used for data collection.

Study aims and research questions are stated along with a brief overview of the study, before more detailed description of the study design and procedures are provided. Information about the pre-registered protocol and subsequent amendments are detailed.

Study assessment measures are listed and described, along with the planned analyses to address each of the research questions. Further changes to the planned protocol are highlighted, and additional analyses are outlined.

4.2 Introduction

Emotion regulation and its potential importance to the relationship between childhood trauma and psychosis is described in detail in the introduction to this thesis. The introduction also highlights a paucity of theories focused on emotion regulation in psychosis, and the systematic

review demonstrates the lack of empirical research into whether emotion regulation acts as a mediator.

There is however evidence which suggests emotion regulation may be an important factor in psychosis. Studies have reported that emotion dysregulation is associated with higher levels, and greater severity of positive symptoms (Wallace & Docherty, 2020, Berenbaum et al., 2006), and dysregulated emotion has been repeatedly found to predict paranoid thinking in cross sectional research studies (Liu et al., 2020b; Westermann, Boden, Gross & Lincoln, 2013; Westermann & Lincoln, 2011).

Individuals with a diagnosis of psychosis are more likely to use less adaptive and more maladaptive emotion regulation strategies, particularly suppression and avoidance (Ludwig, Werner & Lincoln, 2019), and studies have found participants to report increased negative, and reduced positive emotional experience (Oorschot et al., 2013; Liu, Subramaniam, Chong & Mahendran, 2019; Pries et al., 2020). Individuals with experience of trauma also report higher levels of dysregulated emotion (Oorschot et al., 2013; Pries et al., 2020), and the strongest evidence identified by the systematic review found very small effects for emotion regulation acting as a mediator between childhood trauma and both paranoia distress and frequency, in a non-clinical sample (Lincoln et al., 2017).

Paranoid symptoms in particular may be linked to dysregulated emotions. The use of adaptive cognitive reappraisal strategies to downregulate negative emotions appears to be impaired by heightened sensitivity to stress and by cognitive biases (Westermann et al.; 2012; Freeman et al., 2002). These biases include negative beliefs about others, and the jumping to conclusions bias which may predispose an individual to thoughts or fears about others wishing to cause them harm (Lincoln, Peter, Schafer & Moritz, 2009 & 2010; Garety, Hemsley & Wessley, 1991). Further distress caused by these fears cannot then be adequately regulated, leading to a maintenance cycle of stress and negative affect, ultimately supporting or strengthening paranoid beliefs (Westermann et al.; 2012; Garety et al., 2001; Freeman et al., 2002; Gross & Levenson, 1997).

Understanding the importance and potential influence of emotion dysregulation is essential to researchers aiming to reduce symptoms and distress in individuals with a diagnosis of psychosis. Although currently one of the lesser studied potential mediators between trauma and psychosis, further research into emotion regulation as a mechanism should help uncover whether it is indeed relevant. If it is found to be an important factor, it can then be targeted by specific interventions. Current evidence in the area is largely of moderate to high quality (see Chapter 2), but predominantly comes from cross-sectional studies in general population samples. Only one study has investigated emotion regulation in a small clinical sample. Therefore, more research is required, particularly in clinical populations. In order to move research beyond the existing cross-sectional studies, there is a need to look to more novel experimental paradigms.

This study makes use of an interventionist causal design (see below). This type of research aims to isolate a potentially important mechanism and change it. In this case, the intervention targets the use of cognitive emotion regulation strategies and teaches mindful acceptance of difficult emotions. The intervention content is intended to highlight the disadvantages of typical response-focused regulation, and introduce the idea of antecedent-focused responding (Gross, 1998), thus bridging between different components of the emotion regulation process (see Chapter 1 for more details about the process model of emotion regulation). Further details of the interventionist-causal paradigm, and the experience sampling method which will be used to collect data are described in further detail below.

4.2.1 Interventionist causal research

Interventionist causal (IC) research involves selecting, isolating and reducing a single hypothesised mechanism using an intervention designed to incite change (Kendler & Campbell, 2009). The use of this paradigm in the context of a randomised controlled trial (IC-RCT) can provide evidence of whether the hypothesised mechanism is a causal factor in the relationship of interest. In order to do so, the results must demonstrate not only an association between the variables, but also that they follow a temporal sequence in which the cause

precedes the outcome, whilst ensuring potential confounding factors are adequately controlled (Reininghaus et al., 2016; Brand, Rossell, Bendall & Thomas, 2017). Randomising participants helps to ensure both the baseline covariates and the treatment effects are independent of potential confounding (Emsley, Dunn & White, 2010, VanderWeele 2015; Lee et al., 2019). Researchers do however caution that it is still not possible to rule out potential confounding between the mediator and the outcome, as the mediator itself is not randomised, only the exposure to the intervention, and therefore there remains the possibility of unmeasured confounds and post-treatment bias (Emsley, Dunn & White, 2010, Lee et al., 2019). This type of design can provide more valuable information than many traditional RCTs of complex interventions which seek to change multiple mechanisms, or observational research designs where the data gathered cannot be used to make causal inferences.

The use of an IC -RCT enables the degree of within group change to be compared between groups, first to investigate the effectiveness of the intervention in altering the target mechanism, and secondly to assess whether changes in the target mechanism caused changes in the outcome of interest. In doing so, and in contrast with induction research, the principle of attempting to change a mechanism in a positive way in order to further understand its workings is ethically sound and should be mutually beneficial for participants and researchers alike (Kendler & Campbell, 2009; Brand, Rossell, Bendall & Thomas, 2017).

For the IC-RCT used here, inclusion criteria were more stringent that other RCTs in the field of psychosis. It was essential that participants had at least minimal levels of both emotion dysregulation and paranoia. The intervention was an emotion regulation skills training, delivered as a series of group sessions, and the control condition was a skills-based art group. The control group matched non-therapeutic elements of the intervention as closely as possible so that any positive benefits experienced by participants could be interpreted more clearly as either attributable to the intervention, or as a result of non-specific therapeutic effects such as empathy or attention. The use of an active control also mitigates the risk of bias brought about by a wait-list or treatment as usual control condition. As it is essentially impossible to conduct

double-blind research in trials of psychological interventions, it is vital that intervention effects are not falsely enhanced by participant expectations, and vice versa for the control condition.

Experience sampling method

The experience sampling method (ESM) has been increasingly used in psychology research, indicating an attempt to move away from more traditional single time point questionnaire measures. In populations with psychosis, it has been suggested such measures may be negatively affected by memory and reporting biases (Kihlstrom, Eich, Sandbrand & Tobias, 1999; Kimhy et al., 2012; Jacobsen et al., 2019). This will have implications for the quality and accuracy of the data which can be collected.

Experience sampling involves collecting data from participants several times per day, over multiple days. Responses are collected using a remote device such as mobile phone, and answers are provided by participants in their own environment, as they go about their daily life (Kimhy & Vakhrusheva, 2019; Palmier-Claus et al., 2011). There are a number of advantages to collecting data in this way, including greater volumes of data per participant, and more accurate representations of the constructs being measured, and their potential fluctuations. It may also help identify patterns between measured variables (Palmier-Claus, Haddock & Varese., 2019). This means ESM is potentially a powerful tool for use in IC designs as it provides a sensitive measure of change, and enables the reporting of these changes to occur in a real-world setting (Freeman 2011; Reininghaus, Depp & Myin-Germeys, 2016; Palmier-Claus, Haddock & Varese, 2019). Time-lagged analyses also permit the assessment of variable changes over time which can help establish evidence for temporality, but these analyses are complex and instead many studies choose to focus upon concurrent analyses (Reininghaus, Depp & Myin-Germeys, 2016).

To date a number of non-RCT empirical studies in psychology have used ESM as an alternative to traditional pre-post questionnaire measures (Mulligan et al., 2016; Hartley et al., 2014a; Kasanova et al., 2020). Several RCTs have also made use of ESM to deliver all or part

of the intervention itself, across a range of different mental health conditions (Knippenberg et al, 2018 - dementia care; Kramer et al, 2014; Simons et al., 2020 - depression; Garrison et al., 2020 – mindfulness for substance use; Bucci et al., 2015 – early psychosis; Garety et al, 2017 - paranoia). These studies have generally shown that ESM is an accessible and efficient means to deliver interventions to, and collect data from participants in a convenient manner, with good adherence rates (Bell et al., 2020; Reininghaus, Depp & Myin-Germeys, 2016).

More recently, researchers have begun using ESM as a measure of change following interventions in clinical psychosis populations (Pot-Kolder et al., 2018; Jongeneel et al., 2018; Bell et al, 2018; Pos et al., 2017). Repeated measures such as the pre, post and follow up ESM surveys used in these studies can help establish temporal ordering and the directionality of effects, as well as enhancing the external and ecological validity of evidence (Reininghaus, Depp & Myin-Germeys, 2016). This study will utilise ESM in line with the psychosis studies above, at both pre and post intervention time points to measure the target mechanism and the symptom related outcomes of interest.

4.3 Study aims

4.3.1 Primary research questions

The primary aims of this study were:

1. To test whether improving emotion regulation skills can reduce experience of paranoia.[7]

2. To test whether there is a positive association between emotion dysregulation and paranoia[8] dynamically over time.

3. To test whether self-report of early emotional abuse and neglect predicts levels of emotion dysregulation during the first sampling period.

[7] The original analysis plan intended to make use of ESM data on both paranoia occurrence and associated distress as outcomes, however due to high levels of missing data the GPTS persecutory ideation subscale was used as the outcome instead.

[8] Data was available for use on paranoia occurrence and associated distress as planned for a limited number of participants. Additional data points were constructed using baseline and end of treatment scores to facilitate the inclusion of all participants – see Analysis Plan below.

4.3.2 Secondary research questions

The secondary aims of this study were:

4. To test whether emotion regulation mediates the trauma-psychosis association at baseline and over time.

5. To investigate how the results from momentary assessment methods compare to those from standardised questionnaire measures.

If improvements in emotion regulation did not mediate the effect of the intervention on paranoia, then analyses of other potential mediators were planned, namely hypervigilance, attachment, metacognitive beliefs about paranoia, and emotional distress (anxiety and depression).

The original rationale for this trial was to answer the research questions specified above. However it was concluded that the small sample size and missing data levels limited the extent to which this was possible. It may therefore have been more appropriate to focus on questions relating to the feasibility and acceptability of trial procedures (Bugge et al., 2013; Shanyide, Pickering & Weatherall, 2011). Acknowledgment of this must be balanced against the risk of selective reporting bias created by a post hoc switch to feasibility assessment alone. For this reason, it was decided to report analyses in accordance with the original questions, but also report an additional post hoc feasibility assessment, based on the criteria outlined by Shanyide, Pickering & Weatherall (2011). See further details below on changes to the study protocol.

4.4 Procedure

4.4.1 Study summary

This study randomised a sample of individuals with a diagnosis of non-affective psychosis to receive either an emotion regulation skills intervention or an active control task. Participants were screened to ensure they were currently experiencing paranoid symptoms and had at least minimal levels of emotion dysregulation before entering the study. Assessments were

undertaken at baseline and post-intervention in both written questionnaire, and smartphone-enabled experience sampling survey formats.

Between group change was assessed to determine the effectiveness of the intervention for the primary research outcome. Regression analyses were used to test this, and also whether emotion regulation ability and other potential mechanisms showed evidence of group change (Farmus, Arpin-Cribbie & Cribbie, 2019; Field, 2009; Lecomte et al., 2008). Selected variables were also tested as potential mediators between group and paranoia (Steenkamp et al., 2019; Kleiman, 2017 McNeish, 2017; Preacher, 2011).

Multilevel modelling techniques (Kleiman, 2017; Schneider et al., 2017; Verhagen et al., 2016; Myin-Germeys et al., 2003) were used to examine whether emotion dysregulation predicted experience of paranoia occurrence and associated distress; and also to test whether trauma predicted reported levels of emotion dysregulation during the first ESM period. Finally mediation analyses were used to examine whether emotion dysregulation mediated between trauma and paranoia.

4.4.2. Design and participants

4.4.2.1 Sample Size

Calculating an adequate sample size for research which uses experience sampling is complicated by the 'nested' nature of the data points which do not meet normal assumptions of independence (Carter & Emsley, 2019). Complex Monte Carlo simulations are sometimes used to derive sample size estimates but these too involve a number of assumptions and unknown factors (Arend & Schafer, 2019).

Several steps were undertaken in order to compute an adequate sample size for the study. A provisional G*Power calculation based on a large correlation between data points (r = 0.5) was performed. In order to detect a large between group effect (Cohen's d = 1) with 80% power, a sample size of 32 would be required (16 per group). This is supported by guidelines

by Snijders & Bosker (2011) who suggest that samples of n >30 are adequate for multilevel analysis of treatment effects using fixed effects models.

A review of experience sampling studies in acute mental health research provided further evidence that the method is feasible in small samples, and several studies in similar populations have used modest sample targets (Palmier-Claus et al., 2012; Humber, Emsley, Pratt & Tarrier, 2013; Hartley et al., 2014a; Nittel et al., 2018). Researchers considered to be experts in the use of experience sampling in clinical research were consulted for their guidance, and feasibility was also discussed with them. The project would be recruiting from a single site, with a part-time researcher, over a one year period. Researchers and clinicians familiar with the hospital site were also consulted and asked for their input based on their experience of admissions and referrals in similar research projects.

A target sample size of 34 individuals was set. Based on the information gathered this was both adequate for the planned analyses and feasible within the time available for recruitment, and also allows for ~10-15% attrition as is common in randomised controlled trials of psychological interventions (Wright, Mughal, Bowers & Meiser-Stedman, 2020; Ong, Lee & Twohig, 2018; Dumville et al., 2006; Hewitt et al., 2005).

4.4.2.2 Inclusion and exclusion criteria

Participants were required to have a primary diagnosis of non-affective psychotic disorder (schizophrenia, schizoaffective disorder, schizophreniform disorder, delusional disorder or non-affective psychosis) and were required to experiencing symptoms at the time of referral. Diagnoses were confirmed by the participant's clinical team.

As the research was interested in the past experience of childhood adversity, all participants were required to be adults aged 18 or over. Evidence suggests adults over the age of 65 are more prone to the onset of memory and cognitive difficulties (Murman, 2015; Salthouse 2009). For this reason an upper age limit of 65 was imposed to minimise the risk of confounding brought about by either inaccurate reporting on questionnaires or by distorting the group

treatment effects. All participants were required to have the capacity to consent to participate in research. This was monitored throughout the duration of their participation (see below).

Participants were required to be able to read and communicate in English. All study measures had been validated in English and the intervention would be delivered verbally in English. The researcher was available to read aloud any material as required, and to ensure comprehension, particularly of the participant information and consent form documents. These documents can be found in appendix 13 and 14.

A short screening tool was developed and administered to all potential participants, to confirm they had a minimum level of emotion dysregulation and paranoia. Items were selected from validated measures and participants were required to answer yes to at least one statement per section of the screening tool. Emotion regulation items were taken from the Emotion Regulation Questionnaire (Gross & John, 2003) and included 'When I am feeling negative emotions, I make sure not to express them' and 'I keep my emotions to myself'; and paranoia items were taken from the General Paranoia Scale for Adults (Barreto-Carvalho et al., 2017) and included 'I often wonder what hidden reason another person may have for doing something nice for me', 'someone has it in for me' and 'it is safer to trust no-one'. The full screening tool can be found in appendix 15. Trauma history was discussed with clinical team ahead of the first assessment appointment, however there was no baseline threshold for this.

Participants were excluded if they had developmental or learning disabilities, such as Autism Spectrum Disorder; or organic brain impairments such as dementia or acquired brain injury. Participants were also excluded if their primary diagnosis was not non-affective psychosis, or was primarily substance-induced psychosis.

Due to the nature of the data collection method (experience sampling using a mobile phone application), any participants who were not permitted access to their mobile phone at the time of consent were excluded. Study mobile phone devices were loaned to participants who did not own a mobile phone or did not wish to download the app onto their own device.

4.4.2.3 Study design

4.4.2.3.1 Baseline

After ensuring potential participants satisfied the required inclusion criteria they were invited to attend an initial appointment where they provided written informed consent and completed a demographic details questionnaire. A set of baseline measures was then administered and a second meeting was scheduled. The participant allocation was checked by the researcher at the end of the first meeting as the randomisation sequence was stored in the data locker where consent forms and paper questionnaires were filed. During the second meeting the PANSS interview was undertaken and the individual was introduced to the experience sampling app. Participants were also informed if they would be attending the intervention or control group sessions at the end of this meeting.

4.4.2.3.2 Experience sampling

The experience sampling questionnaire was delivered via a smartphone app called *PsyMate*^{TM} (Maastricht University, www.psymate.eu). This could be downloaded on to a participant's own phone, or a study phone could be loaned to them for the duration of the sampling.

Following similar research and recommendations in the literature, the questionnaires were delivered ten times per day over six consecutive days (Myin-Germeys et al., 2001), using a semi-random sampling schedule. This provides a more complete picture of daily life, whilst also avoiding anticipation (or 'expectancy effects') and minimising reactivity and boredom (Varese et al., 2019; Palmier-Claus et al., 2011). Alternative sampling strategies include 'interval sampling' where the questionnaires are delivered at regular intervals throughout the day (Myin-Germeys et al., 2001). Whilst this regularity can help establish a routine of responsiveness, it does introduce the risk of intentional avoidance (Palmier-Claus et al., 2011). An alarm signalled the availability of each questionnaire, and participants had ten minutes to complete it. After this period the survey was considered 'missed'. This was designed to prevent back-filling of missed alarms which would have a detrimental effect on the validity of the in-vivo data.

The semi-random schedule involved alarms sounding between selected hours, primarily to avoid disturbing participants at times when they may be asleep, but also to avoid data clustering. Recommendations ordinarily suggest 7.30am to 10.30pm (Myin-Germeys et al,. 2009) however as acute patients often have disrupted sleeping patterns, other authors have suggested this time frame should be adjusted to better suit the participant's routine (Palmier-Claus et al., 2011). Alarms were programmed to sound between the hours of 10am to 10pm and participants were asked to complete as many as possible each day. Missing data is to be expected when using ESM but offering more questionnaires per day should enhance the likelihood that sufficient data will be obtained (Palmier Claus et al., 2011).

The researcher guided participants through a familiarisation with the app and the questionnaire at an in-person appointment. The questionnaire was first demonstrated to the participant, then they completed a trial version for themselves. They were also shown how to charge the device (if not using their own handset), and given details of how to contact the researcher in the case of technical difficulties. Each participant was called by the researcher within the first 24-48 hours of the sampling period to check comprehension and compliance (Palmier-Claus et al., 2011; Kimhy & Vakhrusheva, 2019). Participants were also shown how to silence the alarms if required; for example during their clinical appointments; and clinical staff were informed of their participation in the study.

4.4.2.3.3 Group sessions

After this sampling period participants started to attend weekly group sessions. There were four sessions in total and each lasted 60 minutes. Participants had up to twelve weeks to attend in case of missed sessions.

4.4.2.3.4 End of treatment assessment

Following completion of the four group sessions, participants repeated selected questionnaires and the PANSS interview before commencing a further six day period of experience sampling surveys. Any participants who were discharged early and were unable to attend the full group program were asked to attend a final meeting to complete the measures

where possible. Control group participants were offered the opportunity to attend the intervention sessions after their participation in the study had ended. This was not part of the research and no further data was collected from them.

4.4.2.3.5 Ongoing treatment

Throughout the study all participants continued to receive routine care, and all were made aware they could withdraw from the study at any time, without giving a reason, and this would have no effect on their ongoing care.

4.4.3 Recruitment and sampling

4.4.3.1 Participant recruitment

A clinical sample was recruited through NHS contacts in the acute psychiatric wards at the Royal Edinburgh Hospital. The study was presented to each of the five acute inpatient ward teams at the weekly ward round meeting, then followed up on at least a weekly basis to gather referrals. Outside of these times, nursing staff were contacted in person, and the ward psychiatrists were regularly contacted by email for referrals. The ward rounds facilitated access to intensive home treatment team staff, as well as occupational therapy, junior doctors and student nurses, all of whom were invited to refer appropriate patients. Recreational nursing staff, music and art therapists were also contacted for potential referrals, particularly as they had a familiarity with how potential participants would behave in a group setting. Staff at the activity centre where the groups took place were encouraged to discuss the study with regular attendees. If interested, the staff obtained permission to pass contact details on to the researcher.

Outpatients were also eligible to be referred into the study. Presentations were given to community mental health services and the psychologists from these teams were contacted to gather referrals. Community groups including Hearing Voices groups and a local advocacy service were contacted with posters and information about the study to pass on to their service users. Individuals who self-referred were asked to provide details of their care team, and

consent for the researcher to contact them in order to conduct a risk assessment prior to their entry into the study. When contacted, risks of violence towards the researcher and any perceived risks of participation (i.e. risks of distress, possible suicidal intent) were discussed

4.4.3.2 Random sequence generation and concealment

The randomisation sequence was generated in advance using an online randomisation service (http://www.randomization.com/). The randomiser made use of randomly permuted blocks of sizes two to four. Blocking balances allocations to the intervention and control groups by the end of each block (Herbert, 2005). This ensures groups do not end up unbalanced, particularly if the full sample size is not achieved. As the target sample here was small, block sizes were minimised to re-balance allocation more frequently. The use of small block sizes enhances the predictability of allocation which can be problematic (Efird, 2010; Herbert, 2005), however having two different block sizes minimises the possibility of guessing the allocation sequence. The specification of block sizes and the generation of the randomisation sequence were undertaken before it became clear that researcher blinding would not be possible (see amendments to protocol below).

At the time of generation, participant ID codes (provided by the app management company, *PsyMate*^{TM}) were assigned in sequential order to each of the group allocations on the list before any participants had been recruited. These participant IDs were then allocated in order of admission to the study. This was tracked and verified by date of initial assessment so the pre-specified sequence could not be altered in any way once the study had commenced.

No stratification was used. Stratification involves further balancing participant allocations based on an additional variable such as gender, age, ward or other demographic factors. As the research took place within a single adult service on one site, it was not considered necessary to include stratification.

4.4.4 Intervention and control procedures

Both the intervention and the control task were delivered as a series of four workshop sessions, each of 60 minutes duration. In keeping with the interventionist-causal design, the intervention and control procedures were matched as far as possible for contact time, host environment, participatory elements and homework-setting. Both were skills-based and involved an element of learning, but the control task had no direct therapeutic element and no references to emotion or regulatory techniques. Participants were required to attend a minimum 'dose' of one session. The number of sessions attended was recorded for use as a covariate in the analysis.

4.4.4.1 Intervention

The intervention was 'Living Well with Emotions' (Lennon, 2015). This is a group-based emotion regulation skills training originally developed for use in an acute adult inpatient setting. It incorporates elements adapted from Dialectical Behaviour Therapy (DBT) (Linehan, 2013) and includes mindfulness principles and acceptance techniques from Acceptance and Commitment Therapy (ACT) (Hayes, Strosahl & Wilson, 2009). It aims to teach participants to recognise and label emotions, and equips them with relaxation and self-awareness skills to encourage more proactive and masterful emotion regulation. The group was facilitated by a clinical psychologist from the acute psychology department in the hospital.

4.4.4.2 Original pilot study

The Living Well with Emotions group was piloted in an acute inpatient sample based on the same site as this study. The group was offered on a rolling basis over a five month period and was able to recruit eight participants from across the acute and rehab inpatient services into the case series (an additional 20 participants attended the group but declined to take part in the research assessments). It was unclear from the report how many participants has been referred to the group in total. The case series was transdiagnositic and accepted patients with a range of diverse diagnoses, including schizophrenia and psychosis.

The group was offered as six sessions, with mean attendance of 2.4 sessions for the acute patients, and 4.75 for the rehab patients. On average each group session had three attendees (range 2-6). It was concluded that it was therefore less feasible to offer the group as a six session program for acute inpatients. In this study the group program was reduced to four sessions.

Attrition rates were low. A similar study experienced an attrition rate of close to 34% (Heriot-Maitland et al., 2014), however this pilot experienced only 8% attrition. Several difficulties with recruitment were highlighted, including short admission, rapid discharge, ward transfers and reluctance to take part in research. As a result this study opted to open recruitment to outpatients in the community as well as inpatients, in an attempt to recruit a larger sample.

The pilot noted positive change on quantitative emotion regulation outcomes of small to large magnitude of effect, however the difficulties in obtaining a full range of measures for each participant was highlighted. The pilot study attempted to assess participants at every session in addition to pre and post intervention, however this proved too demanding. In the pilot study the group facilitator was also the researcher. By having separate group facilitators in this study, additional time and resource should be available for the completion of planned study measures.

These positive effect findings were complimented by qualitative interviews where participants asserted their enjoyment of the group. They describe feelings of validation in terms of their difficult emotions; experiences of social inclusion, and improvements in their management of distress as areas of positive improvement for participants. Overall the pilot concluded that there was support for both the acceptability and feasibility of this intervention in a transdiagnostic sample of inpatients in a Scottish hospital setting.

4.4.4.3 Intervention design and content

The four-session outline is as follows:

Session 1 – *Identifying and labelling emotions*. This session aims to build awareness of suppression and avoidance as dysfunctional regulation strategies. Participants are asked to think about their own experience of difficult feelings. The facilitator reassures participants that suppression is a reasonable response, but one which may cause later problems. A mindful breathing exercise is performed with a focus on self-awareness. Homework is set to practice this before the next session.

Session 2 – *Reducing vulnerability to overwhelming emotions*. This session introduces the idea of acknowledging and observing an emotion without necessarily acting on it. Practical strategies for self-care are discussed (diet, sleep, exercise, avoiding substances). The mindfulness based practice includes controlled breathing and focusing on making space for, and accepting painful emotions.

Session 3 – *Self-soothing*. This session introduces strategies for the development of self-compassion and positivity. Participants are encouraged to engage all of their senses in the present moment whilst being unmindful of worries, and distracting themselves from negative anticipatory thoughts. The mindfulness practice involves the use of a self-soothing object selected from a box of items during the session. This acts as an anchor to focus the participant on their bodily and sensory awareness. Homework from this session includes mindful breathing and an attempt to increase positive activities to enhance positive emotions.

Session 4 – *Emotions, urges and acting opposite*. This session introduces the strategy of 'acting opposite' as a response to the often difficult behavioural urges which can occur with painful emotions. The mindfulness practice includes bringing together awareness of bodily sensations, controlled breathing and emotional awareness and acceptance. Homework from this session is again mindfulness practice and an effort to work on acting opposite.

The sessions are designed to be stand-alone and they do not need to be attended in any specific order, however the later content does build upon skills and ideas introduced in the earlier sessions. This creates a course which works to expand participants' knowledge and skills during each weekly session, but also allows new participants to commence the group program at any point. The group was delivered on a rolling basis and attendance logs were maintained by the clinical psychologist leading the group.

4.4.4.4 Control

The control group was an arts and crafts workshop which included elements of practical skills-based learning and group interaction which mirrored the activities set out in the intervention sessions. Activities included clay-modelling, drawing and painting and 'room styling' which enabled participants to make small items such as photo frames or cushions for their ward room or home. There was no emotion-based content included in the group sessions. This group was selected as an adequate control as it was facilitated by qualified and trained staff members from the activity centre, and provided attendees with the social aspect of taking part in a group. For these reasons it was considered superior to simply controlling with 'treatment as usual'.

4.5 Approvals & Pre-registration

The project received a favourable ethical opinion from South East Scotland Research Ethics Committee 02 (SES REC) and from NHS Lothian Research & Development (NHS R&D). REC reference 18/SS/0065. R&D No: 2018/0202. IRAS reference 229624.

Due to GDPR-based changes in data protection which came into force during the application process, and due to the nature of data collection using a mobile phone app, the study was referred to the Caldicott Guardian for review, and subsequently approved. Caldicott application number: CRD18086.

Edinburgh Napier University also provided a favourable ethical opinion. Reference number SHSC 0022.

As described in the introduction to this thesis, it was essential that the study was pre-registered in the public domain before any recruitment related activity commenced. The original study protocol was registered on the Open Science Framework at DOI: https://osf.io/vywc7/ and subsequent protocol updates based on amendments submitted to the SES REC for ethical approval were uploaded sequentially. Changes to the study protocol are discussed below and in Chapter 6. These are detailed in a manner which is transparent and supported by robust rationale.

4.6 Amendments to study protocol

A number of amendments were submitted following protocol registration. These were submitted to the SES REC and NHS R&D for scrutiny and implemented only after favourable opinions had been received. Significant changes to protocol are discussed below, and further minor changes are detailed in appendix 16.

4.6.1 Blinding

It was not possible to maintain researcher blinding in the study due to staffing constraints. It would have been necessary for nursing staff to escort participants to the group each week to prevent the researcher discovering each individual's group allocation. This was not feasible due to a shortage of nursing staff in the wards, and instead the researcher facilitated all patient escorts unless an additional escort was required for safety reasons. Whilst this may have increased potential for bias, it also prolonged contact time with participants, improved rapport and potentially enhanced retention in the study due to trust, confidence and accountability.

4.6.2 Adverse events

Consultation with the Data Monitoring and Ethics Committee (DMEC) (see below) resulted in a substantial amendment to request the addition of further measures to gather information about adverse events which may have occurred during the duration of the study (see Adverse Events below for further details.

4.7 Assessment measures

4.7.1 Consent and demographic details

4.7.1.1 Capacity to consent to research

The researcher was trained to assess whether a participant had the capacity to consent to take part in research. Potential participants' care teams were contacted prior to any appointments being scheduled and research decision-making was discussed.

As capacity is not a static construct, there was a need to continually assess this on a regular basis. For inpatients this was discussed with ward staff, and for outpatients this was based on informal conversation before the session started each week. Any concerns were highlighted to study supervisors and clinical care teams.

Any participant who was judged to have lost capacity but wished to continue taking part in the group was encouraged to do so. No data was collected from them during this period.

4.7.1.2 Informed consent

Potential participants received an information sheet with details of the study, and they were permitted a minimum of 24 hours to read and consider this. Their understanding of the information, and their willingness to take part was checked through discussion with the researcher, before the formal consent form was completed.

4.7.1.3 Demographic questionnaire

Demographic details were gathered from participants using a short questionnaire. This included age, gender, ethnicity, years of education, marital and employment status. Details of diagnosis were collected, along with the number of years since their first mental health diagnosis, medication details, and whether they had received psychological therapies in the past. This information was used to characterise the sample.

4.7.2 Research measures

Asking participants about sensitive topics will always carry a level of risk, particularly when the study population are considered vulnerable. However, all of the questionnaire measures used

in the study are routinely used in research and healthcare practice without adverse effects. The study did involve questions about sensitive topics including the experience of childhood abuse and psychotic symptoms which participants may have been reluctant to disclose, however measures were completed in person with the researcher present as far as possible, who could monitor for signs of distress.

4.7.2.1 Primary efficacy outcomes

The primary outcomes for the study were the experience of paranoid symptoms and associated distress. These outcomes were measured using both the experience sampling app, and a standardised questionnaire. Originally the ESM data was intended for use in the primary outcome analyses, but due to high levels of missing data, the primary analyses were conducted using the questionnaire data only.

Green Paranoid Thoughts Scale (GPTS) Green, Freeman, Kuipers, 2008

The GPTS consists of two subscales; ideas of social reference (16 items) and persecutory ideation (16 items). Items are rated on a five point scale from 'not at all' (1) to 'totally' (5) and scores range from 16 to 80 per subscale, with higher scores indicating greater incidence of paranoid thinking. The measure was designed to be applicable to both clinical and non-clinical groups, with the ability to assess the strength of beliefs, preoccupation and associated distress, which are not adequately measured by earlier paranoia questionnaires, which have instead tended to focus on a broader conceptualisation of paranoid thought (Green et al., 2008).

The measure has good reliability in clinical populations (Cronbach's alpha = .95), with strong correlations with relevant measures (other paranoia measures, anxiety and depression) showing strong concurrent and convergent validity (Green et al., 2008). The measure showed sensitivity to change at 6 months, prompting the authors to recommend its use in empirical research (Green et al., 2008). A review of self-report measures of paranoia identified the GPTS as offering the best estimate of paranoia prevalence, whilst highlighting the uniqueness

of the assessment of paranoia appraisal afforded by the items asking about distress, an element which is markedly absent from alternative measures (Statham et al., 2019).

The GPTS has undergone revisions since it was listed for use in this study. The study, based on pooled data from ten years of GPTS use in research, questions the adequacy of the original factor structure (Freeman et al., 2019). Factor analysis suggested that the ideas of social reference scale was negatively affected by items which loaded on to both its own, and the persecutory ideation factor, suggesting a lack of coherent internal structure. These items were removed, along with items from the persecutory ideation subscale where the wording had been identified as confusing. Overall the revised measure was reduced to only 18 items, with a cleaner factor structure which explained 69% of variance (Freeman et al., 2019). The authors maintain that the persecutory ideation scale, that which is most often endorsed in a clinical population, was and remains a good standalone measure in clinical populations and as this forms the primary outcome for this study, the original scale will be used. The study protocol was registered prior to the above research being published, and as the persecutory ideation scale has undergone only minor revisions, it was reasonable to adhere to the original pre-specified plan.

ESM survey

Paranoia occurrence and associated distress were measured using the ESM questionnaire. Research guidance recommends that the questionnaire contains no more than 30-60 items and takes no longer than three minutes to complete in order to minimise participant burden (Palmier-Claus et al., 2011). The questionnaire used in this study consisted of 24 items scored on a scale from ‘not at all’ (1) to ‘very’ (7). Questions were based on items used in previous similar studies (Collip et al., 2011; Sitko et al., 2016; Lüdtke, Kriston, Schröder, Lincoln & Moritz , 2017; Ben Zeev, Ellington, Swendsen & Granholm, 2011) and items from the CERQ and GPTS measures (see appendix 17).

The aim was to measure participants' paranoid thoughts at the time of, and since the last alarm, along with their levels of associated distress and their use of regulatory strategies to cope with this. All items were standardised across the same seven point scale to avoid confusion and input errors. There were an additional three items at the end of the questionnaire which reported contextual information about where the participant was, who they were with, and what they were doing at the time of the alarm. Each alarm contained the same questions in the same order for all participants. When piloted the questionnaire took on average 2.40 minutes to complete (range 2.11 minutes to 3.09 minutes).

Following consultation with the app designers, *PsyMate*, the order of items was set to prioritise current 'state' information ahead of items relating to the interim period since the previous alarm, and finally more general contextual questions. This is in contrast to the guidance in the literature which suggests placing contextual items ahead of interim information (Palmier-Claus et al., 2011; Myin-Germeys et al., 2009). *PsyMate* recommended this sequencing based on their experience with developing similar questionnaires. They suggested that in terms of congruency it was pertinent to record the most immediate information first ('Right now I feel suspicious') leading into information about each participant's thoughts in the interim period ('Since the last alarm I have thought that others are plotting against me') and associated distress ('This was distressing'), before finishing with contextual questions. As the context is unlikely to have changed in the minutes taken to complete the questionnaire, it made sense to record this information at the end. Participants were required to complete a minimum of 50% of the questionnaires to be included in the analysis.

4.7.2.2 Secondary outcomes

4.7.2.2.1 Emotion regulation

As emotion regulation was the target for the intervention in the study, the full length CERQ was used at pre and post-intervention time points. It was important to establish a baseline for each participant, and to measure emotion regulation after the study using the CERQ in

addition to the experience sampling app to ensure each participant had adequate data for analysis.

This data was used to assess whether changes had occurred in emotion regulation during the course of the study, and to investigate whether changes were correlated with changes in paranoia. Emotion regulation was also investigated as a potential mediating mechanism between trauma and paranoid symptoms.

Cognitive Emotion Regulation Questionnaire (CERQ), Garnefski Kraaij & Spinhoven (2001).

The 36-item version comprises the same subscales as the short-form version (see Chapter 3) but in the full length version each subscale contains four items, rather than two. Studies report that alpha in studies using the full length version has been found to range from .70 to over .80 with good factorial validity, discriminant validity and construct validity (Jermann et al., 2006; Garnefski et al., 2002; 2006). The full length version has been found to regularly be more reliable than the short form, likely due to the enhanced number of items per subscale (Garnefski et al., 2006). The full length CERQ has acceptable test-retest reliability of .41-.59 (Garnefski et al., 2001).

4.7.2.2.2 Childhood trauma

The same childhood trauma measures were used here as in the online study (see Chapter 3). These measures were only administered at baseline as reporting is based on past experiences which research has shown to be relatively consistent, and not influenced by symptom severity (Fisher et al., 2011). The two measures cover several types of interpersonal trauma (sexual, physical, emotional/psychological and neglect). Scores will be used to characterise the sample, and in mediation analyses.

Child Abuse and Trauma Scale (CATS), Sanders & Becker-Lausen, 1995

Comprehensive Child Maltreatment Scales for Adults (CCMS-A), Higgins & McCabe, 2001

4.7.2.2.3 Psychotic symptoms

The PANSS interview was used as a secondary measure of psychotic symptoms to establish a baseline for each participant and to assess change. The PANSS interview measures all aspects of psychotic symptomatology – positive, negative and general psychopathology, and thus enables a more complete understanding of a participants' presentation. Simultaneously, the interview format offers an opportunity for the researcher to engage with the participant on a level beyond that which is possible with paper questionnaires.

Positive and Negative Syndrome Scale (PANSS), Kay, Fiszbien & Opler 1987.

The PANSS is a structured interview which assesses 30 items of psychopathological symptoms. There are three subscales; positive symptoms (7 items); negative symptoms (7 items) and general psychopathology (16 items). All items are rated across a seven point scale of severity from 'absent' (1) to 'extreme' (7). The ratings are made by the interviewer and involve interpretation of dialogue as well as observation of behaviour during the interview. A number of items also require input from clinical staff. The interview takes 30-45 minutes, starting with open questions, and becoming increasingly structured and directive (Kay et al., 1989).

Initial studies found good internal reliability for all scales; Cronbach's alpha positive = .73, negative = .83 and general = .79 (Kay, Opler & Lindenmayer, 1989). Test-retest scores were stable in participants who were non-responsive to treatment at three to six months; positive = .80, negative = .68 and general = .60 (Kay, Opler & Lindenmayer, 1989). Criterion validity was supported as scales correlated with the Scales for the Assessment of Symptoms – Positive and Negative (SAPS and SANS) and the Clinical Global Impression Scale (Kay et al., 1988 & 1989).

The PANSS has been used widely in research but in more recent years, alternative factor structures have been investigated. Critical reviews have suggested a five-factor model may be more appropriate in explaining the multidimensionality of the disorder (Lehoux et al., 2009;

Wallwork et al., 2012). The five factor model retains the 'positive' and 'negative' subscales but these comprise different items to the original factor structure (Lehoux et al., 2009). The other subscales are 'cognitive/disorganised', 'excitability/hostility' and 'depression/anxiety'. Other models have been suggested, including a sixth factor of 'suspiciousness/persecution' however there is currently little statistical support for this (Wallwork et al., 2012).

Lancon et al. (2000) found the five factor model consistent across two clinical samples, one chronic and one relapsing. The five factor solution explained 62.1% and 64.3% of variance respectively, with internal consistency >.70 on all scales with the exception of cognitive in the chronic population. The five factor model has been used in treatment-response research, as well as studies into functioning and insight (Wallwork et al., 2012). In this study the PANSS was not a primary outcome measure and so the original factor structure, as per the pre-specified protocol, was retained for both its simplicity and for its widespread use in research, making results from this study more easily comparable with other studies.

4.7.2.2.4 Alternative mechanisms

Mechanisms which were hypothesised to be potentially important to the main outcomes were identified and measured. In the event of reduced distress but unchanged emotion regulation, these measures would be used to help identify any other mechanisms which may be working to incite the change in symptoms. These measures were completed at the beginning of the study to characterise the sample, and again at the post-study time point to enable comparison. Further details about the measures can be found in Chapter 3.

Brief Hypervigilance Scale (Bernstein et al., 2015)

Depression, Anxiety & Stress Scale (DASS-21), Lovibond & Lovibond (1995)

Beliefs about Paranoia Scale (BAPS) short form, Gumley et al. (2011)

Psychosis Attachment Measure (PAM), Berry et al. (2006)

4.7.2.3 Adverse events

Guidelines were set out for the definition, recognition and reporting of adverse events (AE) or serious adverse events (SAE) occurring during the study. These incorporated standardised NHS Health Research Authority criteria for SAEs along with additional criteria specified in the protocol documents of similar previous research (Griffiths et al., 2019; Pyle et al., 2016). Adverse events are not uncommon when undertaking research in clinical populations, however it is vital for trials to record and report their incidence.

The HRA defines SAEs as an untoward occurrence that:

(a) results in death;

(b) is life-threatening;

(c) requires hospitalisation or prolongs existing period of hospitalisation;

(d) results in persistent or significant disability or incapacity;

(e) consists of a congenital anomaly or birth defect; or

(f) is otherwise considered medically significant by the investigator.

In addition to this (i) suicide attempt; (ii) suicidal crisis without attempt (rating of 2 on item 8 of the Calgary Depression Rating Scale for Schizophrenia; CDSS) (Addington, Addington, & Maticka-Tyndale, 1993); and (iii) severe symptom exacerbation (rating of ≥6 on the patient or researcher-rated CGI and CGI-I) were added as additional events which would require immediate action.

Non-severe adverse events were defined as a score of ≥3 (agree ‘quite a lot’ or ‘a lot’) on any relevant item (e.g., subjectively worsening mental state, heightened stigma, increased medication use, increased conflict) on the patient-rated 26-item Adverse Events Questionnaire (see below) (Griffiths et al., 2019; Pyle et al., 2016, Morrison et al., 2018).

AEs and SAEs could be identified during any study activity. In particular, the researcher was vigilant during assessments and the escorting of participants to groups. Group facilitators were regularly asked about participant condition during the groups, and likewise clinical ward staff were asked to highlight any changes in condition which may have been prompted by involvement in the study. As an additional safeguard, the patient-rated 26-item Adverse Events Questionnaire (Griffiths et al., 2019; Pyle et al., 2016) was added. This was administered to all participants at the end of their study involvement. There are two versions; one for study completers and another for those who withdraw early (defined as any point before the completion of the end of treatment questionnaire battery).

Additionally, a further document was added which included three researcher rated items, and two participant-rated items from the Clinical Global Impression Scale (Guy, 1976) and the Calgary Depression Scale (Addington et al., 1993) collated into a single brief measure. Scores of ≥ 6 would be considered adverse effects as these indicated severe illness or worsening of a participant's condition throughout the study. These were administered with the final questionnaires at the end of treatment assessment. Scores from these documents were used to highlight any potential distress, deterioration in condition or potential SAEs which required reporting to the DMEC and then, if required, to the NHS REC.

Events would be reported to the NHS REC if they were deemed to be both related to the study – that is, having resulted from administration of, or participation in, any of the research procedures, and unexpected – that is, not listed in the study protocol as an expected event. The DMEC members would offer their judgement on whether any event fit these criteria and thus was required to be passed on to the NHS REC.

Events were required to be highlighted to the study supervisor within 24 hours, and a written report submitted to the DMEC within 48 hours. The DMEC would respond and advise within seven days and if required the event would be reported to the NHS REC within 15 days of the event occurring. The REC would then reply within 30 days and any required action would be taken. Details of adverse events will be reported in the study results chapter.

4.8 Analysis Plan

The original analysis plan set out in the pre-registered protocol was not feasible due to high levels of missing data on the ESM measure. As a result a number of changes were implemented, including how missing data was handled and which statistical tests could be undertaken. Each of the changes are described in detail below, and the revised analyses used to address each of the original study aims are outlined.

4.8.1 Changes to missing data handling

The protocol stated that multiple imputation (MI) would be used to handle missing data, however the revised analysis plan (below) meant this was not possible. Multiple imputation creates several iterations of the data set imputed with missing values which are then pooled across the sets. This type of data set is not compatible with a number of statistical tests or packages. The original revised analysis plan included a series of ANCOVAs in SPSS and mediation analyses using PROCESS. Neither of these can be undertaken using a multiple imputation data file. Therefore the final revised analyses were performed using linear regressions and mediation analyses in lavaan in R version 3.6.3.

Full information maximum likelihood (FIML) missing data mechanisms were used in lavaan for both regression and mediation testing, along with an 'MLR' robust maximum likelihood estimator in order to deal with the non-normality of variables. Maximum likelihood methods are statistically more efficient than MI, and involve fewer uncertainties (Allison, 2012). With MI the number of data sets and required iterations must be decided based upon levels of missing data, and imputed values are drawn at random to fill the empty data fields (Mallinckrodt, Clark & David, 2001). ML does not directly generate substitute data to fill these empty fields, but rather bases its estimates upon patterns in the obtained data. For this reason, ML will always return the same results for any given data set, whilst MI will often return marginally different results based on the randomness of the data draws (Allison, 2010). The potential for differentiation in MI can be reduced by increasing the number of imputed sets (Siddiqui, 2011) but ML avoids this issue entirely by creating only one data model, meaning that the 'analysis

model' and the 'missing data model' are one and the same (Allison, 2010). MI specifies the missing data model separately, leaving the method open to conflict between the missing data model and the analysis model.

This change was implemented after creating a MI dataset and discovering it was not possible to complete the ANCOVA and PROCESS analyses. The changes were specified blind to results as it was not possible to undertake the planned statistical tests. The missing data in the ESM data set was judged too extensive to be filled with imputation so this data was used in its original form to provide additional data points for the individuals who did engage with the measure (see further details below).

4.8.2 Changes to outcome data

4.8.2.1 Experience sampling subscales

The items on the ESM measure corresponded with selected items from the CERQ and GPTS which were administered at baseline and end of treatment. Regardless of whether participants engaged with the ESM measure, it was possible to construct 'pseudo-ESM' data points using the data collected during these assessment sessions. This enabled all participants to be included in the analysis. This 'reduced GPTS' subscale was constructed from eight items - five paranoia-occurrence items, and three paranoia-distress items. The 'reduced CERQ' data comprised seven items – four negative regulatory strategy items, and three positive.

4.8.2.2 Baseline and end of treatment data

Where analyses were based solely on baseline and/or end of treatment data, scores from the full GPTS persecutory delusions subscale were used as the main outcome. The GPTS subscale is valid and reliable when administered at a single time point. The reduced GPTS subscale – consisting only of the items used in the ESM measure (above) has not been validated in the same way and therefore the full subscale score was preferable in this instance. The use of the reduced GPTS subscale was considered to be acceptable in a repeated measures context.

The research questions were therefore addressed using a combination of baseline and end of treatment data, with ESM data incorporated wherever possible. The revised analysis plan is described below with details of the included variables used in each statistical test.

4.8.3 Revised analysis plan

4.8.3.1 Primary Research Questions

4.8.3.1.1 Aim 1 – To test whether improving emotion regulation skills can reduce experience of paranoia

The primary research aim was to determine whether the intervention caused changes in the experience of paranoia. The original analysis had intended to use the pre and post intervention ESM data to investigate group change. Due to poor compliance with the data collection method only one participant reached the prespecified minimum data threshold of 50%. A further eight participants provided data points during both the pre and post intervention sampling periods, but missing data levels were high (missing data range 54.2-100%). In hindsight the minimum data level should have been lower, and a number of studies use 30-33% completion as a threshold (Palmier Claus et al, 2011). More recent research has used minimum thresholds of below 30% (Klippel et al., 2017).

Effect of the intervention on paranoia

Linear regressions were run to examine whether group allocation had an effect on paranoia scores at the end of treatment. The primary outcome variable was score on the GPTS persecutory ideation subscale. Secondary analyses were undertaken to examine any effects of group allocation using the GPTS referential delusions subscale, GPTS total score, and the PANSS suspiciousness item as further outcomes. All analyses controlled for baseline paranoia scores on the relevant outcome measures. All regression models were re tested using completer data only as a means of sensitivity analysis.

Effect of the intervention on emotion regulation

Linear regressions were run using CERQ positive and negative emotion regulation subscales to investigate whether group allocation had an effect on emotion regulation at the end of treatment. Regression models were re-tested using completer data only as a means of sensitivity analysis.

Mediation analyses

Mediation models were tested in lavaan using group allocation as the predictor variable and GPTS persecutory ideation as the outcome. Two models were specified to examine whether emotion regulation acted as a mediator; one using the positive and one using the negative emotion regulation subscale as the potential mediation mechanism.

4.8.3.1.2 Aim 2 - To test whether there is a positive association between emotion dysregulation and paranoia occurrence and distress dynamically over time.

Mixed model regression analyses (also known as multilevel models, or MLM) were used to investigate whether there was a positive association between emotion dysregulation and paranoia over time. These models account for data which have variance on a number of levels (Aarts et al., 2014). Here data came from the individual ESM alarms, nested within days, nested within participants, giving the analysis a hierarchical structure. Multilevel models account for the non-independence of data points which are nested in this way (Aarts et al., 2014).

The models were specified using the 'lme4' package for R (Bates, Maechler, Bolker & Walker, 2014a). Restricted maximum likelihood estimation (REML) was used, with fixed effects significance testing undertaken using t-tests via Satterthwaite's method (Luke, 2017). Typically the REML method is used in studies where fixed effects are being tested, to determine whether the predictor is related to the outcome (Bates et al., 2014b). R automatically uses the Satterthwaite method which adjusts degrees of freedom in order to

compute t-test statistics to check the significance of fixed effects, whilst reducing the risk of Type 1 error (Luke, 2017; Kuznetsova, Brockhoff & Christensen, 2017).

An unconditional random-intercept model was first specified and tested. The grouping variable and the outcome are entered as random effects into the model, with no independent variable, to investigate whether there is sufficient variation between participants to merit the use of MLM (Kleiman, 2017). A significant *p*-value for the random intercept in the model indicates adequate variation. The unconditional models were specified separately for paranoia occurrence and paranoia-related distress as outcomes.

After confirming the suitability of each unconditional model, the full multilevel models were constructed. First the independent variable (emotion dysregulation) was grand mean centred. Centring variables by their grand mean involves subtracting the sample mean from each individual's response(s). This has the effect of shifting the variable scaling so the value of the intercept is now the expected value of Y when X is at its original mean, as opposed to when X=0 (Algina & Swaminathan, 2011). This also means the 'centred variable' can now be interpreted in terms of each participant's deviation from the mean (Kleiman, 2017).

The model was then run to test each outcome, with group allocation and emotion dysregulation as independent variables. These were entered as fixed effects, with both participant and day entered as random intercepts, as the data were nested within days and then within participants.

All available ESM data were used in the analyses, as were the pseudo-ESM time points constructed from baseline and end of treatment scores (see above). Sensitivity analyses were run to examine whether there were any effects of the inclusion of multiple data types (ESM and questionnaire data).

4.8.3.1.3 Aim 3 - To test whether self-report of early emotional abuse and neglect predicts levels of emotion dysregulation during the first sampling period.

The original protocol described plans to test two way interactions (emotion regulation x trauma) by group to determine whether associations between emotion regulation and paranoid symptoms were modified by exposure to early trauma (Reininghaus et al., 2016). Interaction effects would then be tested between baseline trauma scores and emotion regulation scores (pre and post intervention) to investigate whether participants with higher levels of childhood trauma were more prone to higher levels of emotion dysregulation, and whether this was more amenable to change using the intervention. This was not possible due to the issues with sample size and levels of obtained data.

Mixed model regression was instead used to test whether the experience of early trauma predicted levels of emotion dysregulation. The trauma questionnaires were only completed at baseline as trauma is understood to be a stable construct over time (Fisher et al., 2011). The two trauma subscales, total trauma (CATS) and neglect (CCMS-A), were entered simultaneously as predictors. Emotion regulation data from both the baseline assessment (the reduced CERQ subscale – see above) and scores from the first ESM sampling period were incorporated into the analyses.

4.8.3.2 Secondary Research Questions

4.8.3.2.1 Aim 4 - To test whether emotion regulation mediates the trauma-psychosis association at baseline and over time.

Mediation models were specified to test whether emotion regulation mediates the association between childhood trauma and paranoia over time. Trauma scores from the baseline questionnaire subscales were used in both models. Baseline emotion regulation and paranoia scores were use in the first model to test whether there was a mediation relationship at baseline. A second mediation model was specified using baseline trauma scores, and end of treatment emotion regulation and paranoia scores, controlling for baseline scores.

4.8.3.2.2 Aim 5 - To investigate how the results from momentary assessment methods compare to those from standardised questionnaire measures.

It was originally intended that analyses would be undertaken to investigate the similarities or differences in data collected using momentary methods, and those collected using questionnaires at baseline and end of treatment. It was of interest to assess the utility of using multiple short measures delivered over several days against more detailed questionnaires collected at a single time point. Due to the lack of ESM data obtained it was not possible to analyse this in the current study.

4.8.3.3 Exploratory analyses

Other potential mediators of the effect of the intervention were examined. Hypervigilance, attachment, metacognitive beliefs about paranoia, and emotional distress (anxiety and depression) were measured at baseline and post-treatment time points. These were first tested for group differences at the end of treatment time point, using linear regressions and controlling for baseline scores. Mediation models were then specified to test whether any of these variables acted as mediators between group allocation and paranoia.

4.8.3.4 Post hoc analysis – feasibility assessment

Elements of the trial were analysed and reported using criteria for the evaluation of a feasibility pilot study (Shanyide, Pickering & Weatherall, 2011; Bugge et al., 2013). This provides the opportunity to consider aspects of the research in a context which may be of use to future studies, and enables reflection on the areas which did not progress as planned. These post-hoc analyses are presented in the results chapter, following the presentation and discussion of the statistical analyses.

4.9 Data Monitoring and Ethics Committee

A Data Monitoring and Ethics Committee (DMEC) was formed to track the progress of the study. The DMEC had input into the monitoring and reporting of adverse events, tracking recruitment progress and advising on any ethical amendments. The committee comprised four members in addition to the researcher. All DMEC members had extensive experience in

clinical practice and/or mental health research and policy. Meetings were held quarterly throughout the study with additional contact available when required, particularly in the reporting of adverse events.

The DMEC monitored adherence to the study protocol and were consulted for their input ahead of amendments being submitted to the NHS Research Ethics Committee (NHS REC). Trial progress was assessed at each meeting to maximise the likelihood of the study concluding on time and with an adequately sized sample having been recruited. Input was received during particularly challenging periods of low bed occupancy on the wards and low rates of referral. An annual report was also submitted to the NHS REC in the 12 months following initial study approval, detailing study progress.

The safety and wellbeing of participants is of the utmost importance to any study. The DMEC scrutinised all patient facing documents (consent form, demographics, participant information sheet) for clarity and suitability. The DMEC was also responsible for reviewing any new information or evidence which may be released during the duration of the study which would have an impact on the continuation of the trial. They would advise in the case of risk information, or any other details which may affect the ability of the study to continue in its original format. Nominated members of the DMEC reviewed all adverse events reports and reached consensus as to whether any such events could be attributed to the study, and whether they required escalation to the NHS REC. The DMEC also had the authority to suspend the trial should they have believed this to be necessary at any time.

Chapter 5 – Results – Study One

5.1 Overview of chapter

This study aimed to investigate potential mediation mechanisms between childhood trauma and subclinical paranoia. Participants from the general population completed a set of online questionnaires. Two separate samples were recruited. Data from the first sample (N=205; the 'exploratory sample') were used for exploratory network analysis using JASP Version 0.13.1 (JASP Team, 2020) to generate a model hypothesis. Further model development was undertaken using lavaan for Structural Equation Modelling in R (version 3.6.3, R Core Team, 2020). Amendments were made until the model fit was good and the details of the final model hypothesis were pre-registered online as was the confirmatory analysis plan.

Data from the second sample (N=237; the 'confirmatory sample') were used to test the model generated by the exploratory phase using a confirmatory approach, and amendments were made based on modification indices. Finally, the amended model was tested in a combined sample using all data from the exploratory and confirmatory samples together.

This chapter reports the details of each analytical phase, along with the results of the exploratory model development, followed by the confirmatory testing. Subsequent model modifications are described, and key findings are highlighted. A full discussion of the implications is available in Chapter 7.

5.2 Phase 1 – Model development

5.2.1 Summary of phase

Phase 1 aimed to observe patterns in the data using network analysis, which visually maps the strength of connections between variables and can be used to identify potential mediation relationships (see Chapter 3). The importance of each variable (referred to as a 'node') in the network is depicted visually in a plot, and also represented in the centrality measures output. This enables key pathways to be identified and hypotheses can begin to be generated about potentially salient pathways.

5.2.2 Exploratory sample characteristics

Data was collected from 223 individuals using the Novi Survey platform. Records with incomplete consent declarations were excluded (N=18). The remaining records (N=205) provided data which could be included in the analysis. Sample characteristics can be found in Table 5.1. Close to three-quarters of the sample (71.2%) were female, and almost all of the participants were white (97.5%). Almost half of participants were in the 22-31 years age bracket (45.9%). Most were employed (79.5%) and the majority were either married (33.7%) or in a relationship (39.5%).

5.2.3 Missing data

Missing data were assessed using the missing values analysis function in IBM SPSS Statistics for Windows, Version 26.0. Little's MCAR test checks for systematic differences between cases with missing data and those without. It provides a chi-square statistic and significance value to be tested against a null hypothesis that data are 'missing completely at random'. This data set indicated that the data were not missing completely at random; X^2=131.43, DF=99, *p*=0.02, with missing data ranging from 0-11.1% per scale.

Further missing data analyses were undertaken to investigate this. Twenty-three variables had incomplete response data, with 11 of these showing missing values in greater than 10% of cases. The missing data was found to come from a minority of cases (N=21) and overall 93.6% of data was complete. Detailed inspection of pattern graphs indicated an increased attrition rate in later questionnaires. When subscales were ranked in order of missing data, from fewest to most, they followed the sequence of measures as they were delivered in the survey. This pattern of missing data is common in online research where no researcher is present, and may be attributable to boredom, fatigue, distraction, technical issues with the survey or loss of internet connection (De Leeuw, 2001; Barnett, McElwee, Nathan, Burton & Turrell, 2017)

Table 5.1 - Sample characteristics of the exploratory sample

		Sample 1 N=205	Sample 2 N=237	Independent samples t-test
Gender	Male Female	28.8% 71.2%	31.2% 68.3% 0.5%	t = 0.457 (p=0.648)
Age	18-21 22-31 32-41 42-51 52-65	8.8% 45.9% 23.9% 10.2% 11.2%	3.8% 41.4% 26.6% 15.2% 13.0%	t = -2.165 (p=0.031)
Ethnicity	White Other	97.5% 2.5%	94.5% 5.5%	t = -1.661 (p=0.097)
Employment status	Employed Unemployed Retired Student Unable to work	79.5% 2.4% 2.0% 14.6% 1.5%	82.7% 3.4% 8.9% 5.0% 0.0%	t = 0.851 (p=0.395)
Relationship status	Single Married Divorced In relationship Widowed Other	23.9% 33.7% 2.4% 39.5% 0.5% 0.0%	21.1% 37.6% 2.5% 36.3% 0.8% 1.7%	t = 0.534 (p=0.594)

- Note: 'other' in sample 1 included N=2 Black; N=1 Asian; N=2 mixed; sample 2 included N=8 Asian; N=5 mixed; N=1 Black

Where participants had completed at least 50% of the items in a subscale, mean imputation was used to generate total scores which could be used in the network analysis. This varied by subscale but comprised only 0.08 – 1.6% of the total data. JASP does not have facility to account for, or impute missing data; and it is not possible to import a multiple imputation data set from another statistical package for use in network modelling. Participants with more than 50% data missing on any given subscale were excluded pairwise from the analyses of that

subscale. The number of participants contributing data to each subscale, along levels of missing data and with skew and kurtosis values can be found in appendix 18. Maximum likelihood estimators which allow for the inclusion of cases with incomplete data were used in the SEM analyses as reported below.

5.2.4 Data screening

The distribution of scores for each subscale was inspected visually in IBM SPSS Statistics for Windows, Version 26.0, using histograms and Q-Q plots. All variables appeared to deviate from a normal distribution with the exception of TAS3 'externally oriented thinking'.

Descriptive statistics tables highlighted four variables with extreme values for skewness (>2 or <-2) and kurtosis (>7 or < -7) (George & Mallory, 2016). These were sexual abuse (CATS1), neglect (CCMS2), perceptual abnormality (CAPE2) and bizarre experiences (CAPE3). For each of these subscales, the number of non-zero responses was inspected. After running initial exploratory analyses (see Network 1, below), two of these variables were excluded due to very low incidence (<20%) in the surveyed population (non-zero responses: bizarre experiences N=9, sexual abuse N=34). The other variables had higher non-zero response rates (perceptual abnormality N=64, neglect N=89) and thus were retained. All analyses were performed following non-parametric or robust estimation methods to account for non-normality.

5.2.5 Network Analysis

Network analysis provides information about the strength of pathways identified within data using visual mapping. It makes use of a partial-correlation network (PCN) which controls for all other known information (Epskamp & Freid, 2018), and depicts variables as circular nodes, connected by 'edges', the boldness of which indicates the strength of the connection (or partial correlation). This type of analysis was chosen because it is data driven and enables researchers to scrutinise prominent pathways in the data and identify which of these may be relevant to their research questions.

The dataset was imported into JASP for the exploratory network analysis. All models were run using the Extended Bayesian Information Criterion Graphical Least Absolute Shrinkage and Selection Operator (EBICglasso) estimator. This applies a regularising penalty which returns a conservative model by treating directed paths (positive and negative) equally, and shrinking all zero and near zero edges out of the model (Epskamp, Borsboom & Fried, 2018). This improves model interpretability as a smaller number of edges are presented, and only the strongest paths are retained.

This estimator was used as it is recommended in relatively small sample sizes, and in models with a high number of estimation parameters (Golino & Epskamp, 2017). The original model including all subscales has 32 nodes and 32*(31/2) pairwise association parameters requiring estimation. This is a total of 496 parameters from a sample size of only N<190, therefore the EBICglasso estimator was the most appropriate for use.

The EBICglasso estimator was run using 'npn' (non-paranormal) correlation methods for continuous non-normal data, with normalised centrality measures and with missing values excluded pairwise (https://jasp-stats.org/2018/03/20/perform-network-analysis-jasp/). The maximum sample size was used for each analysis, which included all available data for each subscale. The numbers included in each subscale varied due to missing data and exact numbers contributing to each subscale can be found in appendix 18.

5.2.5.1 Exploratory modelling summary

Five networks were estimated. These are described in turn below.

Network 1 - For the preliminary network, data from all subscales were input, and a simple model generated to observe the raw connections within the data. Variables were grouped by scale in each of the models, with each scale being assigned a colour for ease of visual identification (see Figure 5.1).

Network 2 – This model, and all subsequent network models made use of 5000 bootstrap resamples. In addition to using NPN correlation methods for non-normal data, bootstrapping

was used to produce more robust model estimates (Epskamp, Borsboom & Fried, 2018). In non-parametric bootstrapping the data is resampled using random draws from the original data set to generate additional plausible data sets upon which model estimates can be based. From this, edge weight stability can be assessed using the computed 95% confidence intervals based on the bootstrapped data (Epskamp, Borsboom & Fried, 2018). Bootstrapping is also adequate for use on regularised outputs generated using EBICglasso estimation (Hastie, Tibshirani & Wainwright 2015). Network 2 presents the results of Network 1 with the addition of bootstrap resampling (see Figure 5.2).

Network 3 – Due to very low incidence rates in the sample, the bizarre experiences (CAPE3) and sexual abuse (CATS1) subscales were identified as potentially having an unreliable influence within the network. As a result, both were excluded from further analyses. Network 3 presents the revised model with these variables excluded (see appendix 19).

Network 4 – This network presents a sensitivity analysis where the CERQ subscales were summed into 'positive' and 'negative' regulatory strategies and tested as two subscales as opposed to nine (Network 4a). They were also removed entirely (Network 4b), and changes to the overall network structure were considered.

The final network model (Network 5) is then described in the context of previous models and the exploratory evolution of the analysis.

5.2.5.2 Network 1

The initial model showed 158/496 non-zero edges with a sparsity score of .68. A sparsity score is computed using the number of zero edges, divided by the total number of edges in the matrix (Duff, Erisman & Reid, 2017). A higher sparsity score indicates a more sparse network. Non-zero edges indicate the number of pathways between variables which are strong enough to be retained after the application of the regularizing penalty; and therefore the sparsity score reflects how well connected the network is (Epskamp, Borsboom & Freid, 2017). The score of .681 in the Network 1 indicates that 68% of the network is made up of zero-scores. The LASSO

estimator used here assumes a sparse network structure, to enhance interpretability (Epskamp, Kruis & Marsman, 2017). Visual inspection of the output network (below) suggested that data were, as expected, clustering within their respective scales. The emotion regulation (CERQ) subscales appeared to be disconnected from the rest of the model. The two trauma variables were not closely correlated, and links with the symptoms subscales were weak, with the exception of sexual abuse (CATS1) and bizarre experiences (CAPE3) which, as described above, were based on very limited non-zero scores.

The mood (DASS), hypervigilance (BHS), alexithymia (TAS) and attachment (PAM) variables appeared to be positioned as potential mediating mechanisms, but this model was based only on obtained data from the relatively limited sample, and further exploratory analyses with bootstrapping were required to establish which edges should be retained in order to identify a stronger network and build a sound model.

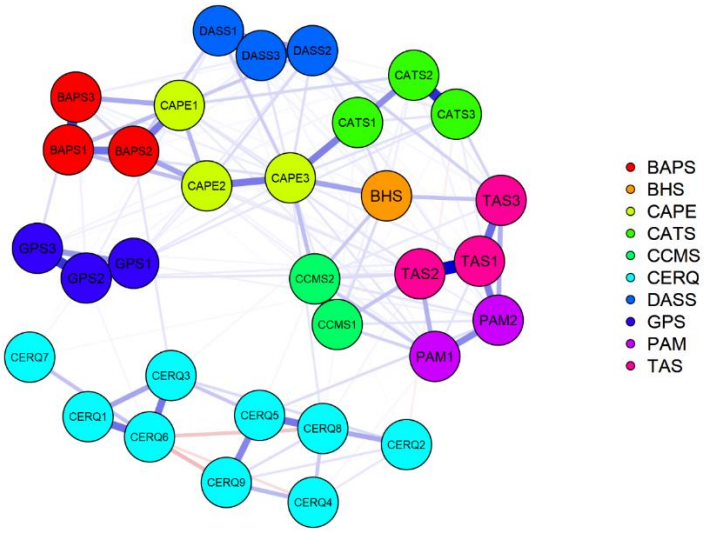


Figure 5.1– Network 1

Note: BAPS Beliefs About Paranoia Scale; BHS Brief Hypervigilance Scale; CAPE Community Assessment of Psychic Experiences; CATS Childhood Abuse and Trauma Scale; CCMS Comprehensive Child Maltreatment Scale for Adults; CERQ Cognitive Emotion Regulation Scale; DASS Depression, Anxiety and Stress Scale; GPS Green Paranoia Scale; PAM Psychosis Attachment measure; TAS Toronto Alexithymia Scale

5.2.5.3 Network 2

Network 2 was generated using the same raw data set, but with the addition of 5000 bootstrap resamples. As no variable changes were made, the output remains as above: 158/496 non-zero edges and a sparsity score of 0.68, however the additional use of bootstrapping serves to visually clarify the salient pathways in the network. As can be seen in Figure 5.2, the stronger connections remain bold, but other pathways which at first appeared sufficiently strong in Network 1, have now faded. This suggests they are not robust to the resampling process. It is clear that the emotion regulation (CERQ) and paranoia (GPS) variables are still peripheral to the main model, but the overall structure remains similar to Network 1.

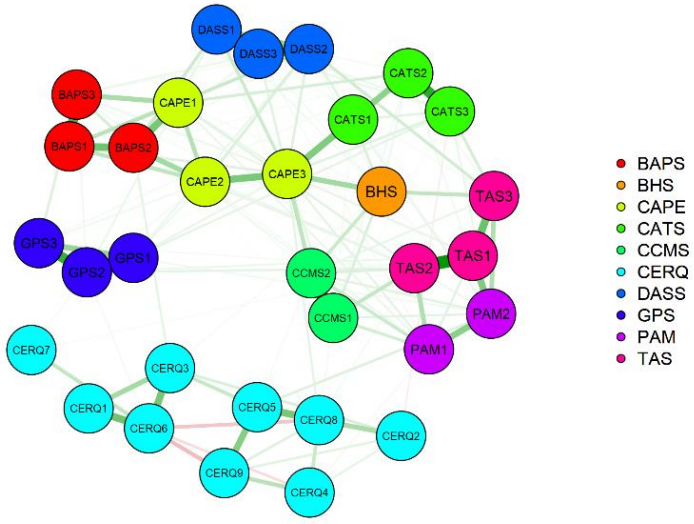


Figure 5.2 – Network 2

5.2.5.4 Network 3

Once the overall network structure had been established, potential network modifications were considered. The bizarre experiences (CAPE3) and sexual abuse (CATS1) subscales were identified in the data screening stage as having very low incidence rates in the data. This along with the inspection of centrality indices suggested they may having undue influence over the network. The centrality measures indicate the importance of each node to the overall network (see Chapter 3). Associated centrality plots for each of the networks described in this chapter can be found in the appendix 19.

Bizarre experiences (CAPE3) in particular did appear to be important to the network with centrality measures indicating a betweenness score of 3.57, a degree score of 2.27 (the highest of any node), and a score of 1.96 for closeness (second highest score). However as the data for bizarre experiences (CAPE3) and sexual abuse (CATS1) are drawn from such a small proportion of the participant sample, they may be unduly influencing the network structure. As a result, both were excluded from further analyses.

After these exclusion the network was reduced to 30 nodes, with 145/435 non-zero edges and a sparsity score of 0.67 (5000 bootstraps). Compared to Networks 1 and 2, there was some redistribution of other nodes in this network, but the overall layout remained largely similar (see appendix 19).

5.2.5.5. Network 4a and 4b – sensitivity analysis

Following the removal of the above nodes, the emotion regulation variables appeared further isolated from the rest of the network. The emotion regulation (CERQ) nodes represent subscales comprising only two items each, potentially resulting in reduced variance or poorer measurement accuracy. This was identified as a potential contributory factor to their disconnection from the rest of the model, and the relative weakness of edges. Sensitivity analyses were conducted to investigate different ways of including the CERQ in the network. Alternative networks were tested with CERQ subscales summed into 'positive' and 'negative' emotion regulation strategy subscales, and with the CERQ removed entirely.

Network 4a with positive and negative subscales (see appendix 19) comprised 23 nodes with 113/253 non-zero edges and a sparsity score of 0.55. Network 4b without CERQ (see appendix 19) comprised 21 nodes with 95/210 non-zero edges and a sparsity score of 0.548. The clustering of variables and the nodes which had the highest betweenness scores remained similar regardless of the handling of CERQ data. In particular, difficulty describing feelings (TAS1) and difficulty identifying feelings (TAS2); persecutory ideation (GPS2) and anxiety (DASS2) nodes in both models returned high betweenness scores, and externally

oriented thinking (TAS3) and negative beliefs about paranoia (BAPS2) returned moderately high centrality scores in the second model (see appendix 19 for network and centrality plots)

5.2.5.6 Network 5 - final network

It was decided that the network depicted in Figure 5.3 was the most parsimonious. Based again on 5000 bootstraps, this network included data from all subscales with the exception of those excluded above. The positive and negative emotion regulation subscales were retained, but these along with the paranoia (GPS) subscales remain peripheral to the rest of the network.

The network plot suggests the trauma variables (CCMS & CATS), considered here as the independent variables, are connected to alexithymia (TAS) and attachment (PAM) variables. The symptom variables (CAPE & GPS) form the outcome variables of interest, however, as noted above, the GPS subscales are largely disconnected from the main network. The CAPE variables are most closely connected to metacognitive beliefs (BAPS) and mood (DASS), with a degree of connection to alexithymia (TAS) and hypervigilance (BHS). CAPE also appears to have direct connections to the trauma variables.

A number of these connections are supported by the centrality measures which mark out alexithymia as being important to the network model, scoring highly on betweenness, closeness and degree (See Figure 5.3, below), as do the CAPE symptom variables. Despite the strength of connections between metacognition and symptoms, there do not appear to be edges between metacognition and trauma, suggesting the metacognitive variables are unlikely to be acting as mediators between trauma and symptoms. Similarly, the mood variables share notable connections with symptoms, but not trauma.

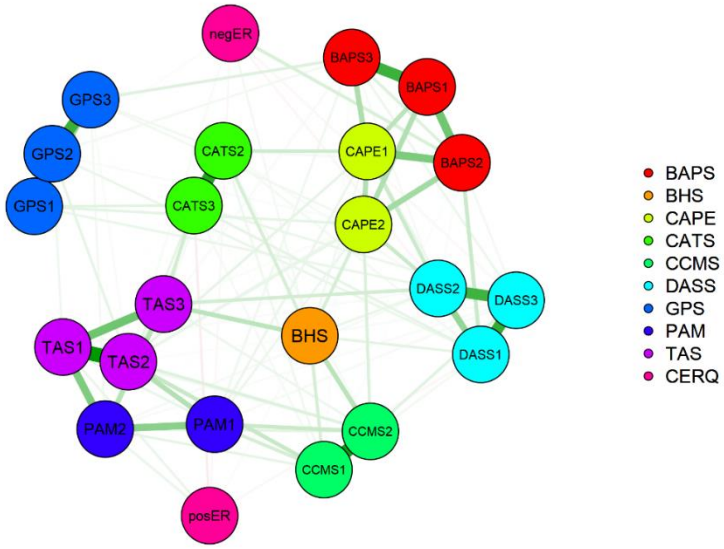


Figure 5.3 – Netwok 5

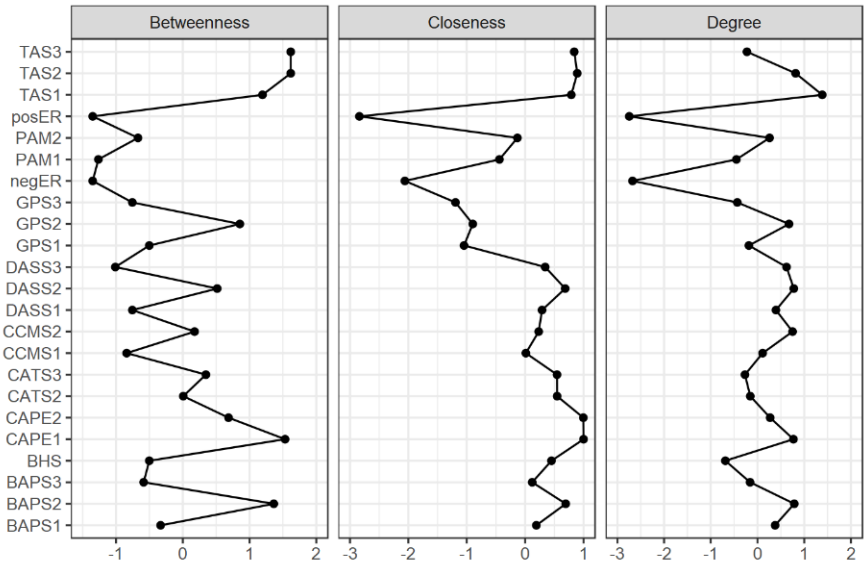


Figure 5.4 Centrality indices for Network 5

5.2.6 Summary of findings

Phase 1 of the analysis established the layout and interconnectedness of the subscale variables within the data. Paranoia and emotion regulation variables were shown to be peripheral to the main network, and two variables with extremely low response rates were removed. The remaining variables do show good connectivity and despite the iterative network testing, much of the structure and layout has remained consistent. As the focus of the study is trauma and subclinical psychotic experiences, these variables are treated as the predictor

and outcome variables in the next phase of analysis. Both alexithymia and attachment were identified as potential mediators, whilst metacognition and mood show relationships with the outcome, but not the predictor. These pathways are further investigated below, using structural models.

5.3 Phase 2 – Model refinement: development of network connections into a hypothetical model

5.3.1 Summary of phase

This phase involved the development of a structural equation model based on the findings from the network analysis. A hypothetical model was constructed using all potentially important variables and potential model modifications were investigated. A final model was selected and pre-registered ahead of testing in a second data set. All analyses were performed using lavaan Version 0.6-6 for SEM in R (Rosseel, 2012).

5.3.2 Sample

This is phase two of the exploratory analysis, so the same sample was used as with the network analysis above. Sample characteristics can be found in Table 5.1.

5.3.4 Theoretical considerations & the sequence of model development

The data-driven nature of network modelling is one of the primary benefits of the method, however theoretical reasoning must be applied before further statistical analyses can be undertaken. This section describes theoretical considerations relevant to the variables included in the models, and how these shaped the various stages of overall model development.

5.3.4.1 Alexithymia

Alexithymia is characterised by difficulties with emotional awareness and communication, relevant to both the self and the emotional states of others, and as a result can have a negative impact on the development and maintenance of interpersonal relationships (Van der Velde et al., 2015; O'Driscoll, Laing & Mason, 2014). There is debate in the literature about whether

the components ‘difficulty identifying feelings’ (DIF) and ‘difficulty describing feelings’ (DDF) should be combined into a single latent factor in TAS-20 data (Erni et al., 1997; Grabe et al., 2009) however a recent review of the measure and its use over the last 25 years suggested that the majority of confirmatory factor analytic research has found the original three-factor structure to be the best fitting (Bagby et al., 2020). For this reason the three subscales were retained as separate variables rather than creating a latent variable.

5.3.4.2 Model 1

The first model was generated using all of the variables identified as important in the in the network analysis. Two trauma variables, psychological abuse and neglect, were entered as predictors. The attachment variables were entered as parallel mediators, followed by the alexithymia variables, with the symptoms variables, persecutory ideation and perceptual abnormality, as the outcomes. Depression and beliefs about paranoia were entered as exogenous predictors of the outcomes.

5.3.4.3 Model 2

Although metacognitive beliefs about paranoia were highlighted by the network analysis, conceptually it made more sense to expect these beliefs to arise following the experience of paranoid symptoms. Therefore these variables may form part of a subsequent maintenance loop which may occur after the initial experience of subclinical paranoia. As this study was interested in mediators between trauma and symptoms, a second model was estimated without the metacognitive beliefs (BAPS) variables.

5.3.4.4 Demographic confounding

Age, gender and ethnicity were entered as potential confounding factors, however ethnicity showed very little variation in the sample (only N=4 were not ‘white British/Other’) so this was withdrawn as a covariate.

5.3.4.5 Model 3

The final model was specified by taking into account the prior exploratory changes described above, along with the relevant covariates.

5.3.5. Procedure

Maximum likelihood estimation with robust standard errors and scaled test statistic was used to investigate model fit. This is suitable for use with incomplete data sets and produces adjusted fit indices for non-normal data (Rosseel, 2012 & 2020). This is a form of missing data handling akin to multiple imputation, and enables the estimation of a full, unrestricted model based on a 'complete' data set (Rosseel, 2012).

Specified indirect pathways were then estimated using bootstrapping (with 2000 resamples). It is suggested that this is the minimum number of bootstrap resamples required to produce stable results (Mooney et al., 1993). Bootstrap testing of indirect effects is not compatible with the missing data estimator, so these analyses were conducted separately on completer data only by way of sensitivity analysis. The bootstrap indirect effect sizes and 95% confidence bounds were considered alongside the indirect estimates from the robust maximum likelihood models – see Table 5.2.

5.3.6 Results

5.3.6.1 Model 1

The model returned a moderate fit; X^2 (7) = 34.21, p=0.00, CFI = 0.95, RMSEA= 0.17, 95% CI: 0.12-0.23, SRMR = 0.08. However in accordance with the criteria set out in Chapter 3, section 3.5.3.2, the RMSEA was higher than the desirable threshold of <0.05 which would indicate good fit (MacCallum, Browne & Sugawara, 1996), and confidence bounds were wider than is recommended to be acceptable (Kenny, 2015). RMSEA is notably affected in models with few degrees of freedom, such as this one. The chi-squared value is also significant where a non-significant value would be desirable (Kline, 2011). The CFI indicated good fit (>0.95) and the SRMR meets the essential threshold of <0.1, but is higher than the 0.05 which would

indicate good fit (Kline, 2011). Although this model fit reasonably well there was room for further improvement to be made. Figure 5.5 illustrates the input model. The overall model fit statistics, along with parameter estimates are listed in Table 5.2.

The specified indirect path within this model is shown in Figure 5.6. This path from psychological abuse (CCMS) to persecutory delusions (CAPE) was sequentially mediated by avoidant attachment (PAM) and difficulty identifying feelings (TAS). The selection of this path was guided by the significant parameters identified in the lavaan output. No significant paths were detected from neglect to attachment, however the model fit statistics were negatively impacted by the removal of this variable. The same was true for perceptual abnormalities, the other outcome variable. Depression showed a significant relationship with perceptual abnormalities, but the potential mediators did not. As with neglect, removal negatively impacted model fit statistics. In Model 1 the specified indirect path was non-significant: B=0.02, SE= 0.01, Z=1.57, *p*=0.12, β=0.04).

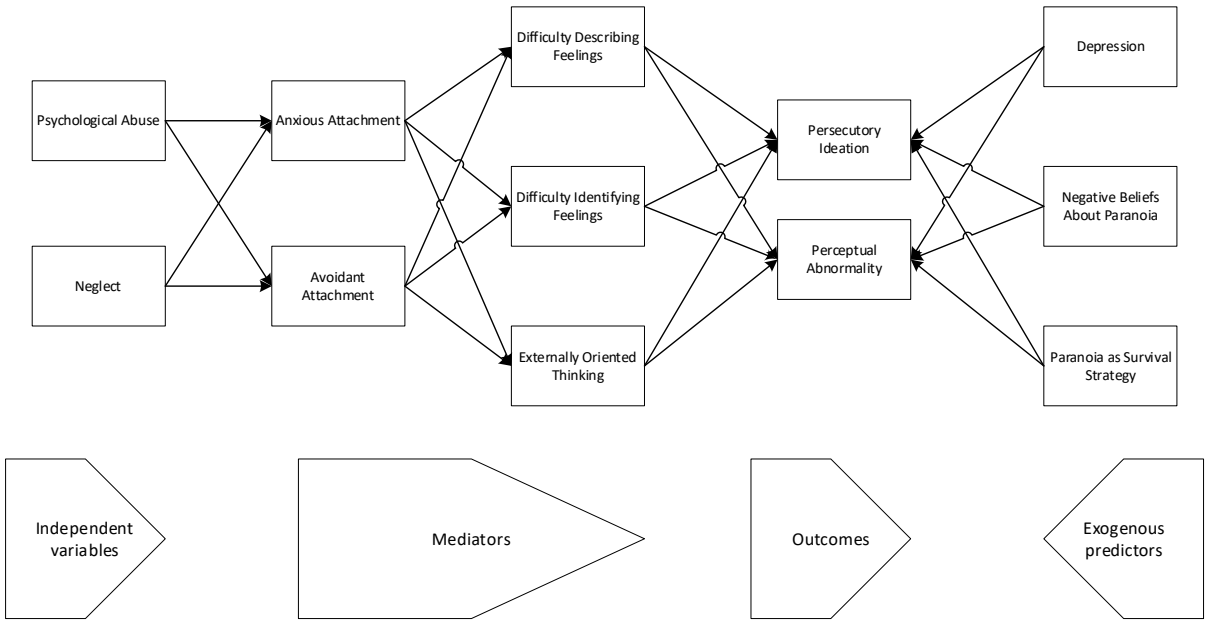


Figure 5.1 Model 1 – the original structural model based on network analysis findings

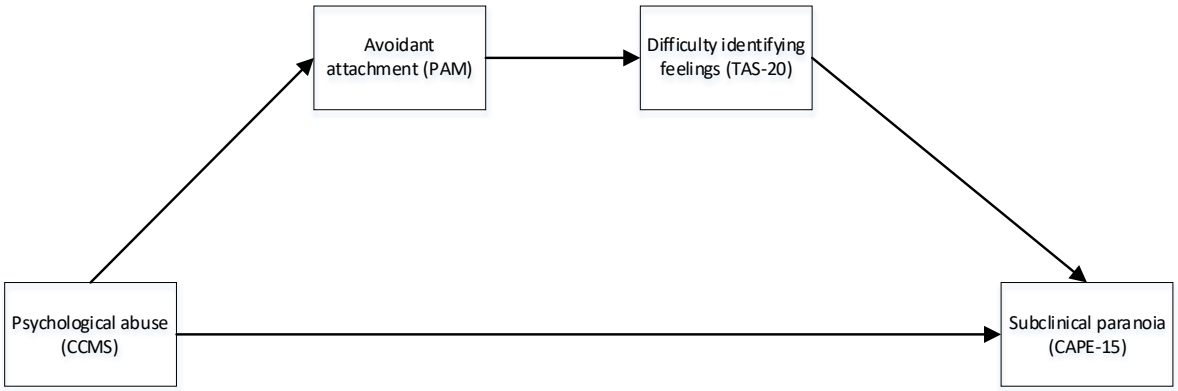


Figure 5.6 The specified indirect path for all models

5.3.6.2 Model 2

Upon removal of the metacognitive beliefs variables, the model fit improved X^2 (6) = 12.26, *p*=0.06; CFI=0.98; RMSEA=0.08 (0.00-0.14), SRMR=0.04. The chi-squared test was no longer significant and the RMSEA was much more acceptable according to the threshold criteria. CFI and SRMR values were also improved. The specified indirect path was significant in this model: B=0.05, SE=0.02, Z=2.38, *p*=0.02, β=0.10.

5.3.6.3 Demographic confounds

No effect was found for either age or gender was detected, however both were retained as covariates within the model.

5.3.6.4 Model 3 – Final Model

The final model, depicted in Figure 5.7, was considered the most parsimonious and best fitting. This model returned an excellent fit: $X^2(7) = 5.62$, *p*=0.59, CFI = 1.00, RMSEA = 0.00 (90% CI 0.00-0.08), SRMR = 0.02. The specified indirect pathway was highly significant (B= 0.06, SE = 0.03, Z = 2.40, *p*=0.02, β = 0.12).

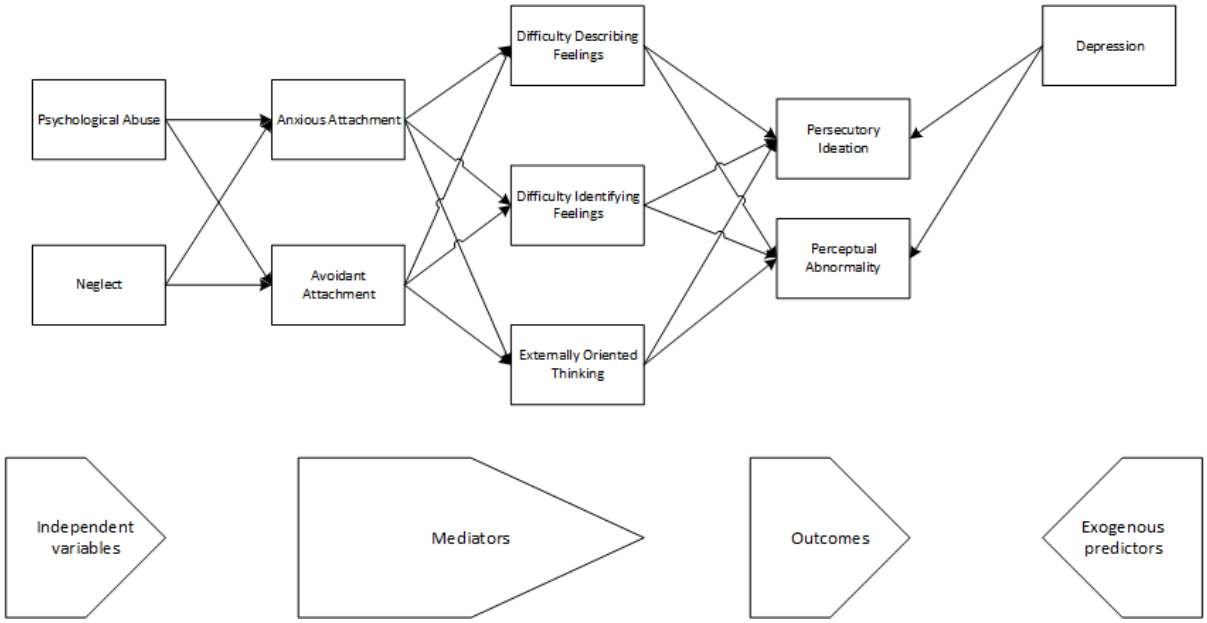


Fig. 5.7 – Final hypothesised model. Age and gender were included as covariates.

5.3.7 Summary of results

This phase involved the iterative testing of structural models based on pathways highlighted by the network analysis. Variables were considered theoretically as well as statistically, and changes were made to the models on this basis. The model itself was refined and demographic variables were tested and included. The final model hypothesis was pre-registered ahead of the collection of the confirmatory data sample.

5.4 Phase 3 – Confirmatory model testing

5.4.1 Summary of phase

This phase tested the model hypothesis developed in phases 1 and two above, in a new data sample. The hypothesised model and planned analyses were pre-registered (see Chapter 3). SEM analyses were again undertaken using lavaan in R. Potential model modifications were investigated.

5.4.2 Sample characteristics

The confirmatory sample consisted of data from 256 individuals with 19 failing to complete consent and demographics. These were removed from the analysis and of the 237 remaining

responses, 185 provided data for all surveys. This sample was composed of over two thirds female (68.3%) and again participants were predominantly white (94.5%). The most common age bracket of participants was 22-31 years (41.4%), most were employed (82.7%) and were either married (37.6%) or in a relationship (36.3%). See Table 5.1.

When the exploratory and confirmatory samples were compared in SPSS, Levene's test for homogeneity of variance showed that for most variables the group variances were equal (indicated by non-significant *p*-values). Ethnicity was highlighted as the only variable with unequal variance (p=0.001). Independent samples t-tests revealed no significant difference between the samples with the exception of age (p=0.031). See Table 5.1 for details.

5.4.3 Missing data

Missing data were investigated using the missing values analysis in SPSS. Little's MCAR test in this sample was non-significant, suggesting that data were missing completely at random (X^2=69.74, DF=75, p=0.65). Missing data ranged from 17.7-19.8% per scale. When cases contributing no data to each scale were removed, missing data was found to be <1% per scale. These missing values were filled using mean imputation as in the exploratory sample.

Further inspection of missing data analyses showed that all variables had some level of missing data, but these were attributable to a minority of cases (N=47). In total 81.8% of data values were complete. Inspection of the pattern graphs indicated a similar trend to the exploratory sample, with greater attrition on the later scales. Before re-issuing the online survey to collect data for the confirmatory analysis, the order of questionnaires was rearranged to prioritise the measures featured in the model. All of the questionnaires were still included, but those most relevant to the hypothesised model were placed at the beginning of the survey to minimise missing data caused by attrition towards the end of the battery. As in the exploratory sample, mean imputation was used to complete the individual missing items in each scale.

5.4.4 Data Screening

Normality testing was again performed using SPSS. Visual inspection of histograms suggested variables did not conform to a normal distribution and Shapiro-Wilk outputs confirmed all variables to be non-normal with the exception of avoidant attachment (p= 0.148). As with the exploratory sample, all analyses were performed using robust estimation methods to account for non-normality.

Descriptive statistics tables highlighted the same four variables as in the confirmatory sample which exceeded cut-off values for skewness and kurtosis: sexual abuse, neglect, perceptual abnormality and bizarre experiences (see Table 2). The proportion of non-zero responses was investigated for each of these and the same two variables as in the exploratory sample had very low incidence (bizarre experiences N=18, sexual abuse N=40). Neglect (N=59) and perceptual abnormality (N=82) had greater non-zero responses and were therefore retained and included in the model.

5.4.5 Model testing

The Model 3 syntax was applied to the confirmatory data set using the robust maximum likelihood estimator. The model was replicated in the confirmatory sample, with the hypothesised model showing adequate fit: $X^2(7) = 25.112$, p=0.00, CFI = 0.98, RMSEA = 0.10 (90% CI 0.06-0.14), SRMR = 0.04. The fit is poorer than in the exploratory sample but this is to be expected, as the model was derived directly from the exploratory data and is therefore likely to fit less well in a completely new data set.

The Chi-square fit statistic is significant where it would ideally be non-significant. This index does tend towards significance in larger samples, however this usually applies to samples of over 400. The other fit indices were good. The specified mediation pathway was however non-significant: B=0.01, SE=0.01, Z=1.18, p=0.24, β=0.02. See Table 5.2 for comparisons.

5.4.6 Modification indices – Model 4

Modification indices were investigated to look for potential areas where model fit could be improved. These indices test all possible fixed paths within the model and provide information about what influence they may have on model fit if they were added to the model (Kline, 2015; Jorgensen, 2017). Higher modification index scores suggest larger potential improvements, however there needs to be a theoretical justification for adding any of the proposed pathways into the model. Without this there is a risk of overfitting to the data, rendering the model too specific and unlikely to replicate in subsequent samples. This impacts on generalisability.

The only theoretically feasible pathway was regressing subclinical paranoia on to anxious attachment. This resulted in a good fit (Model 4), but minimal model improvement: $X^2(6) = 14.57$, p=0.03, CFI = 0.99, RMSEA = 0.07 (90% CI 0.03-0.12), SRMR = 0.04. The indirect path remained non-significant. See Table 5.3 for model comparisons.

Table 5.3 – Model fit statistics

	Model 1	Model 2	Model 3	Model 3 confirmatory sample	Model 4	Model 4 combined sample	Model 5
N	169	169	169	237	237	442	440
Chi-Squared (DF) *p-value*	34.209 (7) *p=0.000*	12.256 (6) *p=0.056*	5.615 (7) *p=0.585*	25.117 (7) *p=0.001*	14.566 (6) *p=0.025*	4.529 (4) *p=0.339*	4.572 (4) *p=0.334*
CFI	0.952	0.984	1.00	0.981	0.991	1.00	0.999
RMSEA (90% CI)	0.170 (0.115-0.228)	0.079 (0.000-0.140)	0.000 (0.000-0.083),	0.099 (0.059-0.143),	0.074 (0.025-0.124)	0.018 (0.000-0.077)	0.019 (0.000-0.080)
SRMR	0.075	0.039	0.018	0.043	0.041	0.008	0.008
Indirect path B (SE) Z *p-value* β	0.020 (0.013) 1.571 *0.116* 0.043	0.045(0.019) 2.380 ***0.017*** 0.095	0.060 (0.025) 2.402 ***0.016*** 0.121	0.012 (0.010) 1.177 *0.247* 0.022	0.008 (0.008) 1.063 *0.288* 0.015	0.019 (0.009) 2.073 ***0.038*** 0.038	0.019 (0.009) 2.063 ***0.039*** 0.037

5.4.7 Exploratory analysis on combined sample

The two data sets were then combined into a single set. Model 4 was tested in this combined sample and returned a very good fit $X^2(4) = 4.53$, p=0.34, CFI = 1.00, RMSEA = 0.02 (90% CI 0.00-0.08), SRMR = 0.01, with a significant specified indirect path B=0.02, SE=0.01, Z=2.073, p=0.04, β=0.04.

5.4.8 Influential cases

The model was tested for influential cases using the influence.SEM package for lavaan (Pastore & Altoe, 2018). This calculates log-likelihood distances for each case in the model to determine whether any individual cases are exerting undue influence. Plots were inspected by eye to identify any influential cases. Two such cases were identified (see Figure 5.8) but when these were removed the resulting model, Model 5, remained largely similar to Model 4 (see Table 5.3). Two previously significant pathways, from neglect to both anxious and avoidant attachment did become non-significant. Neglect and anxious attachment reduced from B=0.32 (p=0.03) to B=0.30 (p=0.08); neglect and avoidant attachment reduced from B=0.35 (p=0.02) to B=0.31 (p=0.06), however the change in the beta regression weights was minimal, and model fit statistics remained largely similar. This suggests the model itself is generally robust to these influential cases, yet the pathways which did show change must be treated with caution as the removal of only two cases had such an impact on their significance values.

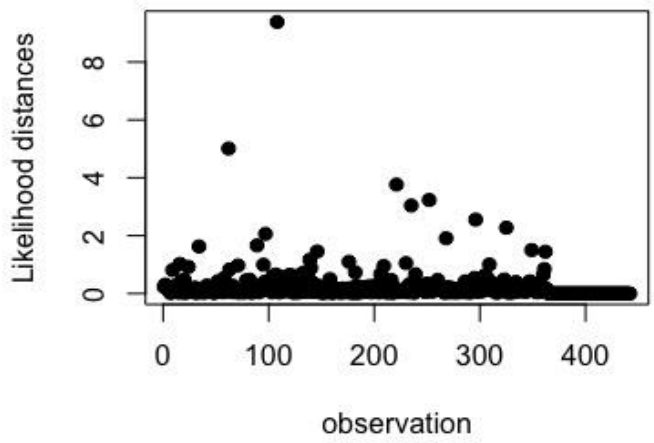


Fig 5.8 - Log-likelihood distances per case in combined sample

5.5 Overall summary of key findings

Results did not suggest that the experiences of childhood psychological abuse and subclinical paranoid thinking were reliably mediated by insecure attachment and alexithymia. The indirect pathway was not replicated in the confirmatory sample, despite good overall model fit across all data sets. In all models depression appeared to be important to subclinical paranoid thinking, however it did not have a strong relationship with traumatic experiences in these data samples.

The use of a two-stage analytical process was a clear strength, and enabled transparent development of the original model before progressing to confirmatory testing. This a process which appears to be omitted by similar studies in the area. Few studies mention the pre-registration of a hypothesis or analysis plan, and therefore it is possible that null findings or alternative models go unreported. This may be one reason for the lack of replication of findings amongst the studies included in the systematic review (Chapter 2). It is clear from the findings of this study that very different conclusions would be drawn if only the exploratory phase has been undertaken.

5.6 Limitations

This study was limited by its use of cross-sectional evidence which cannot be used to infer causal links between variables. A sequence of effects is being hypothesised, but the data as it stands cannot express whether the sequence is in the correct order. Although it seems reasonable to assume that childhood trauma will have occurred early in life, it is not possible to rule out reverse causality amongst other variables, or that other unmeasured variables may account for the observed associations. Broadening research into experimental paradigms where mechanisms are isolated and altered is required before causal hypotheses can be investigated.

The data collection method may leave the study open to common method bias, whereby the measures used introduce bias into the data (Podsakoff, MacKenzie & Podsakoff, 2012). Common method bias is likely to derive from participants' lack of ability or motivation to respond accurately to the questions, perhaps due to fatigue brought about by similarity in the measures and response scales, or by the ambiguity in the wording of certain items. This can either inflate or shrink the estimates of relationships between variables, increasing the risk of type I or type II error. Equally, in some cases it can have no effect at all, and it can often be difficult to discern which of these is relevant to the data in question (Siemsen, Roth & Oliveira, 2010). Efforts were made to use well established research measures presented in an order which would not enhance the likelihood for bias as the participant made progress through the sequence i.e. the trauma questionnaires were separated so recalled information from one trauma questionnaire was not immediately salient when answering the next.

The sample itself may have introduced elements of bias. Individuals were self-referring volunteers from the general population. Although snowball sampling was used in an attempt to widen the reach of the study, the sample consisted mainly of females of white ethnicity. The findings may not be generalizable to the wider general population, or to clinical populations. Further research will be required to test whether the mechanisms may be relevant to a larger

general population sample, or to a clinical sample with experience of childhood trauma and paranoid symptoms.

The confirmatory sample was collected in May 2020 during the COVID-19 pandemic lockdown. It is not possible to rule out confounding by potential contextual factors affecting understanding, attention, perception or stress which are not adequately captured or controlled for in the second model. It is impossible to know how this may have affected the data, but it is worth noting that the data collection context is very different between the two samples.

5.7 Implications and recommendations

As identified in the systematic review (Chapter 2), there are currently no studies which assess alexithymia as a potential mediating mechanism between childhood trauma and positive psychotic symptoms. Whilst the evidence of mediation was not replicated in the confirmatory sample, the network analysis offers preliminary evidence of the potential importance of alexithymia. Future studies may wish to investigate alexithymia further in large general population samples, or in individuals with a clinical diagnosis. Whilst it can be difficult to hypothesise about potential clinical implications based on data drawn from a general population sample, the data itself is free from the potentially confounding effects brought about by medication or other treatment which is common in clinical samples (Tiliopoulos & Goodall 2009). Therefore, conducting early research in non-clinical samples, which are largely easier to recruit may be useful at this preliminary stage of investigation.

All indirect effects, both significant and non-significant were of trivial magnitude, which is in keeping with the findings of the systematic review, particularly in terms of attachment. Correlational evidence to date does suggest that both insecure attachment and alexithymia may be important the development of psychotic symptoms, however causal mediation is not indicated. Future research may wish to examine metacognition as this did appear to be relevant in the network model in this study, however the variables were hypothesised to be more pertinent to the maintenance of paranoid thoughts, rather than their onset. Studies

investigating the mediation potential of metacognitive beliefs are scare, with only one study adequate for assessment in the systematic review[9] (Østefjells et al., 2017). The study did find a mediation effect between abuse and positive symptoms in a multiple mediator model with depression, in a clinical sample. Both of these variables were identified as potentially salient in this study, however more evidence is required before conclusions can be drawn.

In terms of the AHRQ criteria applied to studies in the systematic review, this study has made efforts to recruit an adequate sample size and use analytical methods appropriate to this; describe the demographic details of both unique samples; use valid research measures for all variables, particularly those which have been validated in non-clinical samples; control for potential confounding variables and handle missing data in a transparent way. There is however still potential for sample bias due to the use of snowball sampling, and ethnic diversity in the recruited sample was limited. However this method was selected with an awareness of the limited time available for the study to collect data and therefore this compromise was made in order to enhance the likelihood of recruiting a sufficiently large sample.

5.8 Conclusion

This study used a robust two-stage approach to developing and testing a model of childhood trauma and subclinical psychotic symptoms. The study aimed to identify mediators of this relationship. Network modelling was used to first identify potential mechanisms of interest, then these were considered theoretically to conceptualise how this pathway may develop in an individual over time. It was reasoned that childhood adversity may have a negative impact on attachment relationships, which may inhibit the development of an ability to recognise one's own emotions and those of others, possibly due to a lack of exposure to adaptive emotional engagement and regulation. However this is speculative as the indirect mediation pathway was not replicated in the confirmatory data sample. No further claims regarding their

[9] Goldstone, Farhall & Ong 2011 & 2012 also investigate metacognitive beliefs but these studies could only be included narratively due to lack of data.

importance can be made without further evidence. A full discussion of the study results and theoretical implications can be found in the **General Discussion** (Chapter 7).

Chapter 6 – Results – Study Two

6.1 Overview of chapter

This study aimed to evaluate a psychological intervention to improve emotion regulation skills in a sample of patients with a diagnosis of non-affective psychosis who were experiencing paranoia. The study followed an interventionist-causal design (Kendler & Campbell, 2009; Pearl, 2000) and participants were randomised to either the intervention or a control condition which was designed to match the intervention for duration and attention, without being specifically therapeutic. The study investigated whether emotion regulation skills were improved by the intervention, and whether this had an effect on the experience of paranoid symptoms. Experience sampling questionnaires (ESM) were delivered via a mobile phone app, and these along with more standard questionnaire measures were used to evaluate change.

A number of the original outcomes were affected by poor engagement with the ESM app which resulted in inadequate data to conduct the planned analyses. The recruited sample also fell below the target sample size which had an impact on power. Regardless of these issues, the original research questions were answered using amended analysis methods where applicable (see Chapter 4, section 4.8). The original protocol was pre-registered, and all subsequent changes are reported here transparently, with care taken to maintain the ethical and scientific integrity of the original research plan (van der Zee & Reich, 2018).

This chapter therefore presents the results of the planned analyses, with explanation of, and rationale for, any changes which were made. Additional post hoc analyses are described. This is followed by a summary of the key findings and a description of the study limitations. Full discussion of the implications of the study results can be found in Chapter 7.

6.2 Sample

6.2.1 Participant flow

During the study recruitment period, 83 referrals were received. Of these, sixteen were judged to be ineligible. The most common reason for this was the individuals' symptoms being judged too acute to engage with research.

In total 67 individuals were approached with information about the study, with 41 of these declining to take part. Reasons for declining to participate were recorded informally. These included having other commitments at group times (e.g. library group, regular community visits); not wishing to feel 'experimented on'; not wishing to participate in group-based therapy; concerns over data confidentially; being reluctant to leave the ward; perceiving the research measures as requiring too much effort; and feeling too tired or too unwell. The remaining 26 individuals provided consent to take part in the study (39% of eligible referrals).

Two individuals were discharged after baseline measures were completed, before they could engage with the group sessions, and a further four participants were unable to be contacted to complete end of treatment assessments after taking part in the group sessions. No participants formally withdrew their consent during the study, however one participant was withdrawn due to deterioration in their condition. Details can be found in Figure 6.1.

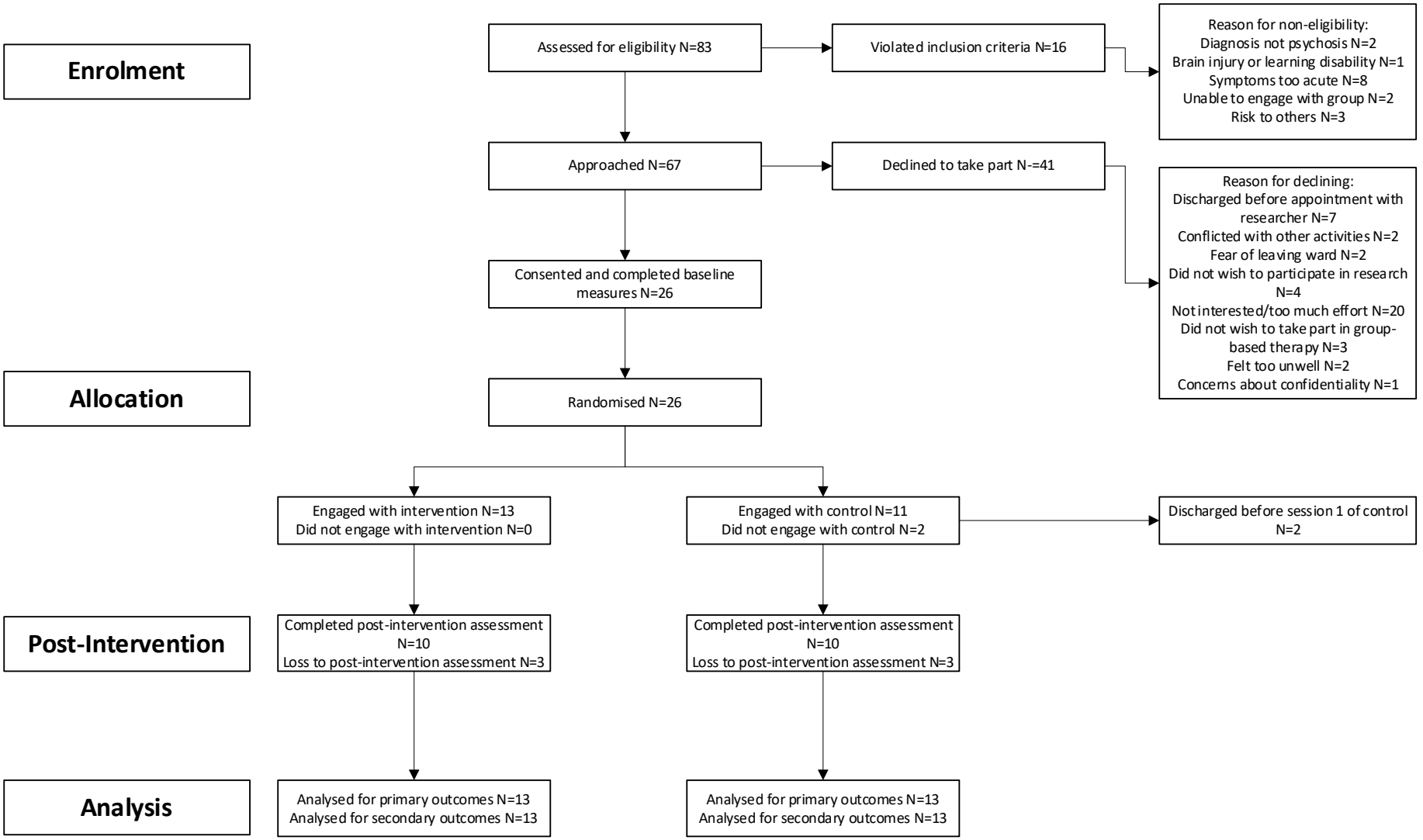


Figure 6.1 CONSORT diagram

6.2.2 Sample characteristics

During the recruitment period, 26 individuals took part in the study, N=13 were randomised to receive the intervention and N=13 to the control group. Demographic details can be found in Table 6.1. The majority of participants were inpatients (N=21, 81%) and of these, the majority were referred by a clinician (N=20, 77%). All outpatients (N=5), and one further inpatient entered the study via self-referral using the contact details on the recruitment poster. There was an equal gender split with N=12 males and females, with N=2 participants who did not identify with either gender. The majority were white (N=22, 86%). 54% of participants had a diagnosis of schizophrenia (N=14), and 38% had their first psychiatric diagnosis over 10 years ago (N=10). All participants were taking antipsychotic medication at the time they entered the study, and 58% had prior experience of psychological therapies (N=14). No significant differences were detected between the participant groups on demographic details.

Table 6.2 shows the means and SDs for the intervention and control group on all measured variables in the completer data. The only significant difference identified between groups at baseline using an independent samples t-test was in CCMS Neglect (t= 1.990, p=0.047; d=0.82, 95% CI 0.02, 1.62).

6.3 Intervention acceptability and safety

6.3.1 Acceptability

Dropout rates from both the intervention and control groups were low. Two participants in the control arm were discharged before they could engage with the group sessions, and they were unable to be contacted thereafter. One further participant from the control arm was lost to post-intervention follow up as they could not be reached following discharge.

Three participants in the intervention group were also lost; one participant was withdrawn due to worsening of symptoms (see below); one participant was discharged out of area; and one participant could not be followed up due to COVID-19 restrictions in the hospital, and was unable to be contacted thereafter.

Table 6.1 - Participant demographics

		Intervention group (N=13)	Control group (N=13)	Independent samples t-test
Gender	Male	61.5%	30.7%	t= -1.947
	Female	38.5%	53.8%	p= 0.063
	Other		15.5%	
Age range	18-21	7.6%	0%	t= -0.156
	22-31	30.8%	38.4%	p= 0.877
	32-41	15.4%	23.1%	
	42-51	30.8%	15.4%	
	52-65	15.4%	23.1%	
Years in education	Mean	14.77	15.85	t= -1.171
	SD	2.35	2.34	p= 0.253
Employment status	Employed	7.7%	23.1%	t= 0.978
	Unable to work	53.8%	23.1%	p= 0.341
	Unemployed	30.8%	23.1%	
	Retired	7.7%	7.6%	
	Student	0%	23.1%	
Relationship status	Single	61.5%	69.2%	t= -0.922
	Married	0%	15.4%	p= 0.366
	In a relationship	15.4%	0%	
	Divorced	15.4%	15.4%	
	Widowed	7.7%	0%	

Ethnicity	White British Asian British Other	92.3% 7.7% 0%	69.2% 23.1% 7.7%	t= -1.569 p= 0.136
Diagnosis	Schizophrenia Schizoaffective disorder Non-affective psychosis Delusional disorder	53.8% 30.8% 7.7% 7.7%	53.8% 38.5 7.7% 0%	t= 0.480 p= 0.635
Years since first diagnosis	0-1 1-3 3-5 5-10 10+	38.4% 15.4% 0% 15.4% 30.8%	15.4% 7.7% 7.7% 23.1% 46.2%	t= -1.398 p= 0.175
Medication	Yes No	100% 0%	100% 0%	No difference between groups
Psychological Therapy	CBT Counselling CAT DBT Unknown None	30.8% 7.7% 61.5%	30.8% 15.4% 7.7% 7.7% 15.4% 23.0%	t= -0.754 p= 0.464

Duration (of those who have had therapy)	< 3 months 3-6 months 6-12 months Over 12 months Unknown	20% 0% 20% 20% 40%	0% 50% 30% 10% 10%	t= 1.155 p= 0.269
Number of sessions attended	Mean SD	3.38 1.04	2.00 1.29	t=3.007 p= 0.006

Table 6.2 Group and total sample means on all measures

	Intervention group (N=13) Mean (SD)	Control group (N=13)	Total sample (N=26)
PANSS Positive	18.67 (4.62)	17.82 (5.46)	18.26 (4.94)
PANSS Negative	17.67 (7.57)	15.08 (7.88)	16.43 (7.66)
PANSS General psychopathology	41.83 (8.26)	42.91 (10.42)	42.35 (9.15)
PANSS Total	78.18 (16.95)	75.82 (18.28)	77.04 (17.23)
GPTS Persecution	49.69 (20.35)	40.92 (16.83)	45.31 (18.83)
GPTS Reference	47.69 (17.97)	43.77 (17.25)	45.73 (17.37)
GPTS Total	97.38 (35.22)	84.69 (30.03)	91.04 (32.71)
PAM Anxiety	11.38 (6.53)	12.42 (6.61)	11.88 (6.45)
PAM Avoidant	12.77 (3.44)	13.42 (4.48)	13.08 (3.90)
PAM Total	24.15 (9.41)	25.83 (10.04)	24.96 (9.55)
DASS Depression	21.66 (14.42)	23.29 (13.86)	22.47 (13.88)
DASS Anxiety	20.00 (10.89)	15.78 (9.89)	17.89 (10.42)
DASS Stress	22.99 (11.04)	21.14 (13.10)	22.06 (11.91)
DASS Total	64.64 (33.43)	60.20 (33.94)	62.42 (33.09)
CATS Sexual abuse	4.71 (7.11)	3.37 (4.47)	4.04 (5.86)
CATS Negative home environment	25.10 (14.68)	23.58 (11.89)	24.34 (13.11)
CATS Punishment	12.60 (4.50)	11.13 (4.41)	11.87 (4.43)
CATS Total	62.86 (34.88)	57.25 (26.72)	60.06 (30.58)
BHS	9.69 (6.03)	8.77 (6.10)	9.23 (5.96)
BAPS Survival	14.46 (5.62)	12.62 (5.39)	13.54 (5.48)
BAPS Negative	17.35 (5.35)	14.39 (5.41)	15.87 (5.48)
BAPS Normalising	14.69 (5.53)	15.89 (5.52)	15.29 (5.46)
CERQ Positive	64.54 (20.09)	57.55 (16.18)	61.04 (18.22)
CERQ Negative	49.46 (11.06)	50.66 (12.66)	50.05 (11.66)
CCMS Physical abuse	3.92 (3.17)	2.23 (2.83)	3.08 (3.07)
CCMS Psychological abuse	6.00 (3.89)	5.09 (3.96)	5.54 (3.88)
CCMS Neglect*	4.38 (4.23)	1.77 (2.13)	3.08 (3.54)

*CCMS neglect was the only significant difference in independent samples t-test of group mean differences (t= 1.990, p=0.047; mean difference = 2.165, SE = 1.314, 95% CI 0.040, 5.191)

Participants in the intervention attended significantly more sessions than those in the control arm (intervention mean: 3.38 sessions; control mean: 2 sessions; t=3.007, p=0.006). Overall 69% of participants completed all intervention group sessions (N=9) whereas only one participant attended all control group sessions (7.7%). All control participants were offered access to the intervention group after completing the study. Four participants attended at least two sessions of the intervention, with one further participant being interested in attending before COVID19 restrictions forced cancellation.

6.3.2 Safety

Serious adverse events were defined in line with NHS Health Research Authority criteria, with the addition of suicide attempt, suicidal crisis without attempt, or severe symptom exacerbation (see Chapter 4, section 4.7.2.3 for full criteria and definitions). Two serious adverse events were recorded during the study, both of which involved deterioration in the condition of the participants; one involving readmission after a short period of discharge. Reports were submitted to the Data Monitoring and Ethics Committee (DMEC) for consideration, but neither event was judged to be attributable to involvement in the research study. One participant was withdrawn from the group due to the severity of symptoms, the other continued to attend and responded positively about being permitted to continue during the end of treatment measures session.

Non-serious adverse events were defined in line with published research in similar populations, using an Adverse Events Questionnaire (Griffiths et al., 2019; Morrison et al., 2018; Klingberg et al., 2011). This was added via ethics amendment (see Chapter 4) after the study had commenced so data was not available for all participants, however no scores of ≥3 (indicating ‘quite a lot’ or ‘very much’ of a negative effect attributed to taking part) were recorded on the measure. This indicated that participants’ ratings of symptoms, distress and medication use were not negatively impacted by taking part in the study. No participants reported adverse effects as indicated by scores of ≥6 on the CGI items (see Tables 6.3 and 6.4).

Table 6.3 Clinical Global Impressions Scale and Calgary Depression Scale measures

	Intervention mean (SD)	Control Mean (SD)	Mean difference *Cohen's d*	95% CI (*p-value*)
CGI Severity (participant rated)	2.800 (0.837)	2.333 (1.033)	0.491	-0.713, 1.696 (*0.44*)
CGI Severity (researcher rated)	3.200 (0.447)	2.833 (0.753)	0.578	-0.634, 1.789 *(0.37)*
CGI Improvement (participant rated)	3.200 (1.483)	2.667 (1.033)	0.425	-0.775, 1.6254 *(0.49)*
CGI Improvement (researcher rated)	3.400 (0.894)	3.167 (1.169)	0.221	-0.970, 1.411 *(0.72)*
Calgary Depression Scale (researcher rated)	0.40 (0.548)	0.333 (0.516)	0.126	-1.062, 1.314 *(0.84)*

Table 6.4 Participant responses to the Adverse Events Questionnaire

	Intervention N=5					Control N=6				
Taking part…	0	1	2	3	4	0	1	2	3	4
1 hasn't helped me with my problems[10]	4	0	1	0	0	2	2	0	1	0
2 made my problems worse	4	1	0	0	0	5	1	0	0	0
3 made me feel more anxious	5	0	0	0	0	4	1	1	0	0
4 took up too much time	5	0	0	0	0	5	1	0	0	0
5 led to my mood becoming very low	4	0	1	0	0	6	0	0	0	0
6 made me feel more angry and irritable	5	0	0	0	0	5	1	0	0	0
7 I didn't feel ready to talk about my problems	4	1	0	0	0	5	1	0	0	0
8 made me think too much about bad things that have happened in the past	4	0	1	0	0	4	2	0	0	0
9 meant I stopped looking after myself properly	5	0	0	0	0	6	0	0	0	0
10 made me feel more suspicious	5	0	0	0	0	6	0	0	0	0
11 required too much energy or motivation	5	0	0	0	0	4	2	0	0	0
12 increased my thoughts of killing myself	5	0	0	0	0	6	0	0	0	0
13 I didn't feel listened to or believed by the study staff	5	0	0	0	0	6	0	0	0	0
14 made my voices or visions worse	5	0	0	0	0	6	0	0	0	0
15 was making me fall out with my family or friends	5	0	0	0	0	6	0	0	0	0

[10] One participant in the control group did not answer this question. Two other participants commented that the wording of this item was confusing. Higher scores indicate improvement, but the negative wording is difficult to interpret.

15 was making me fall out with my family or friends	5	0	0	0	0	6	0	0	0	0
16 was having a bad effect on my self-esteem	5	0	0	0	0	6	0	0	0	0
17 was making me want to harm myself	5	0	0	0	0	6	0	0	0	0
18 I didn't like or feel I could trust the research team members	5	0	0	0	0	6	0	0	0	0
19 I felt embarrassed talking about my problems with people I had not met before	4	1	0	0	0	5	0	1	0	0
20 made me have thoughts of harming other people	5	0	0	0	0	6	0	0	0	0
21 was making me feel hopeless about the future	5	0	0	0	0	5	1	0	0	0
22 meant I had to increase my medication in order to cope	5	0	0	0	0	6	0	0	0	0
23 involved too much hard work.	5	0	0	0	0	5	1	0	0	0
24 made me worry that people would think badly of me because of my diagnosis	5	0	0	0	0	3	2	1	0	0
25 made me fall out with my doctor or care team	5	0	0	0	0	6	0	0	0	0
26 made me worry about losing control of my mind	5	0	0	0	0	6	0	0	0	0
27 My problems have improved to the point I no longer feel I need help[11]	2	1	1	1	0	5	0	1	0	0

[11] Higher scores indicate improvement

6.4 Primary Research Questions

6.4.1 Aim 1 - To test whether improving emotion regulation skills can reduce experience of paranoia

6.4.1.1 Effect of intervention on paranoia

In order to test for differences in post-treatment paranoia scores, a series of linear regression models were constructed in lavaan. Group allocation was set as the predictor and end of treatment GPTS persecutory delusions subscale scores used as the outcome, co-varying for baseline scores. A small significant negative group effect was detected B= -11.45, SE=4.95, *p*= 0.02, ß= -0.37. Both groups reported reductions in paranoia on the GPTS persecutory delusions subscale at post treatment, but reductions were greater for participants in the control arm.

Secondary analyses were undertaken on the GPTS delusions of reference subscale, and GPTS total score, both of which showed negative effects favouring the control arm, but these did not reach significance. No effect was found when the PANSS suspiciousness item was tested as the outcome. Full results can be found in Table 6.5. Sensitivity analyses were run using 2000 bootstrap resamples on completer-only data for each of the paranoia outcomes. It is suggested that this is the minimum number of bootstrap resamples required to produce stable results (Mooney et al., 1993). For the previously significant GPTS persecutory delusions model, change in the regression coefficient was minimal, but was sufficient to marginalise the significance of the effect B= -11.48, SE=5.85, 95% CI -22.93, -0.02, *p*=0.05, ß= -0.37. Sensitivity analysis results can be found in Table 6.5.

As specified in the original study protocol, a per-protocol analysis was undertaken to investigate if there was an effect when the analysis was restricted to individuals who received a minimum 'dose' (i.e. attended at least one group session). This returned very similar results to the original analysis B= -11.46, SE=4.95, *p*= 0.02, ß = -0.37. When number of sessions was added as a covariate to the original analysis of the primary outcome the effect became non-significant B= -7.66, SE=4.30, *p*=0.08, ß= -0.24.

Table 6.5 – Results of linear regression models to examine group effects on paranoia and emotion regulation

Model	Variable	Unstandardised coefficients *b* (SE)	95% CI for *b* (lower, upper)	*p*-value	Standardised coefficients, *ß*
Model 1: Persecutory ideation (GPTS) $R^2 = 0.365$	Constant	32.005 (10.663)	11.106, 52.904	**0.003**	2.078
	Baseline	0.330 (0.177)	-0.016, 0.676	0.062	0.396
	Treatment allocation group	-11.450 (4.950)	-21.177, -1.773	**0.020**	-0.373
Sensitivity analysis – completer data only $R^2 = 0.365$					
	Constant	32.005 (12.479)	7.546, 56.464	**0.010**	2.078
	Baseline	0.330 (1.149)	0.039, 0.621	**0.026**	0.414
	Treatment allocation group	-11.475 (5.846)	-22.933, -0.017	0.050	-0.366
Sensitivity analysis – Number of sessions as covariate $R^2 = 0.413$					
	Constant	16.447 (13.229)	-9.482, 42.376	0.214	1.047
	Baseline	0.335 (0.166)	0.009, 0.661	**0.044**	0.394
	Treatment allocation group	-7.658 (4.298)	-16.082, 0.766	0.075	-0.244
	Effect of session attendance	3.027 (1.893)	-0.684, 6.738	0.110	0.261
Sensitivity analysis with per protocol attendees only $R^2 = 0.361$	Constant	32.005 (10.663)	11.106, 52.904	0.003	2.086
	Baseline	0.330 (0.177)	-0.016, 0.676	0.062	0.401
	Treatment allocation group	-11.475 (4.950)	-21.177, -11.773	**0.020**	-0.373
	Constant	21.319 (10.840)	0.074, 42.565	**0.049**	1.659

Model	Variable	Unstandardised coefficients *b* (SE)	95% CI for *b* (lower, upper)	*p*-value	Standardised coefficients, *ß*
Model 2: Referential delusions (GPTS) R^2 = 0.180 Sensitivity analysis - completer data only R^2 = 0.180	Baseline	0.315 (0.160)	0.002, 0.628	**0.048**	0.418
	Treatment allocation group	-1.018 (5.251)	-11.310, 9.274	0.846	-0.040
	Constant	21.319 (11.361)	-11.3199.283	0.061	1.659
	Baseline	0.315 (0.154)	0.014, 0.616	**0.040**	0.418
	Treatment allocation group	-1.018 (5.256)	-11.319, 9.283	0.846	-0.040
Model 3: Paranoia total (GPTS) R^2 = 0.303 Sensitivity analysis - completer data only R^2 = 0.303	Constant	55.183 (17.488)	20.906, 89.460	**0.002**	2.190
	Baseline	0.326 (0.157	0.017, 0.634	**0.039**	0.414
	Treatment allocation group	-14.60 (8.983)	-32.217, 2.997	0.104	-0.290
	Constant	55.183 (21.444)	13.154, 97.213	**0.010**	2.190
	Baseline	0.326 (0.139)	0.053, 0.598	**0.019**	0.414
	Treatment allocation group	-14.610 (9.865)	-33.946, 4.726	0.139	-0.290
Model 4: Suspiciousness (PANSS) R^2 =0.006 Sensitivity analysis - completer data only R^2 **=** 0.006	Constant	2.613 (1.032)	0.591, 4.636	**0.011**	3.244
	Baseline	0.059 (0.197)	-0.328, 0.445	0.765	0.082
	Treatment allocation group	0.034 (0.435)	-0.818, 0.034	0.938	0.021
	Constant	2.613 (0.236)	0.300, 4.927	**0.027**	3.244
	Baseline	0.059 (0.208)	-0.349, 0.467	0.778	0.082
	Treatment allocation group	0.034 (0.454)	-0.856, 0.923	0.941	0.021

Model	Variable	Unstandardised coefficients *b* (SE)	95% CI for *b* (lower, upper)	*p*-value	Standardised coefficients, *ß*
Model 5: Negative emotion Regulation (CERQ) R^2 = 0.315 Sensitivity analysis - completer data only R^2 = 0.315	Constant	18.838 (7.478)	4.182, 33.494	**0.012**	1.622
	Baseline	0.569 (0.133)	0.309, 0.829	**0.000**	0.561
	Treatment allocation group	0.134 (4.227)	-8.150, 8.418	0.975	0.006
	Constant	18.838 (11.878)	-4.442, 42.117	0.113	1.622
	Baseline	0.569 (0.186)	0.205, 0.933	**0.002**	0.561
	Treatment allocation group	0.134 (4.308)	-8.309, 8.577	0.975	0.006
Model 6: Positive emotion Regulation (CERQ) R^2 = 0.397 Sensitivity analysis - completer data only R^2 = 0.397	Constant	33.190 (8.972)	15.606, 50.774	**0.000**	2.312
	Baseline	0.481 (0.174)	0.140, 0.822	**0.006**	0.599
	Treatment allocation group	-3.195 (4.373)	-11.765, 5.376	0.465	-0.111
	Constant	33.190 (13.051)	7.611, 58.769	**0.011**	2.312
	Baseline	0.481 (0.138)	0.211, 0.751	**0.000**	0.599
	Treatment allocation group	-3.195 (5.101)	-13.193, 6.803	0.531	-0.111

6.4.1.2 Effect of intervention on emotion regulation

In order to test for differences in post-treatment emotion regulation scores, further linear regressions were constructed. CERQ positive and CERQ negative scores were used in turn as outcomes, with group allocation entered as a predictor. There were small mean decreases in the use of both negative and positive emotion regulation strategies by participants in both groups, however no significant effects were found. Sensitivity analyses using completer data were again performed but no significant effects were found. See Table 6.5 for results.

6.4.1.3 Mediation analyses

Mediation models were specified in lavaan to investigate any evidence of emotion regulation mediating between group allocation and GPTS persecutory ideation. Neither negative nor positive emotion regulation showed evidence of mediation. Additional models were tested using GPTS delusions of reference and GPTS total score as outcomes, and all models were subject to sensitivity analyses using completer-only data. No evidence of mediation was found. See Table 6.6 and appendix 20 for details.

Table 6.6 Mediation models (group effects)

Model		**Estimate (SE)**	**95% confidence bounds**	**p-value**	**Standardised effect**
Negative emotion regulation (CERQ) and paranoia (GPTS-persecution)	a-path	0.134 (4.227)	-8.150, 8.418	0.975	0.006
	b-path	0.125 (0.267)	-0.399, 0.649	0.639	0.095
	Indirect effect	0.017 (0.537)	-1.036, 1.070	0.975	0.001
	Direct effect	-11.447 (5.027)	-21.300, -1.593	**0.023**	-0.390
	1000 Bootstrap indirect effect *	-0.042 (1.657)	-4.742, 2.550	0.980	-0.001
Positive emotion regulation (CERQ) and paranoia (GPTS-persecution)	a-path	-3.195 (4.373)	-11.765, 5.376	0.465	-0.113
	b-path	-0.452 (0.170)	-0.786, -0.118.	**0.008**	-0.401
	Indirect effect	1.444 (2.230)	-2.926, 5.814	0.517	0.045
	Direct effect	-13.560 (4.610)	-22.595, -4.524	**0.003**	-0.457
	1000 Bootstrap indirect effect*	1.319 (2.573)	-1.885, 9.326	0.608	0.041

*Bootstrap indirect effects are computed using completer data only as the missing data estimator (FIML) is not compatible with the bootstrap process

6.4.2 Aim 2 - To test whether there is a positive association between emotion dysregulation and paranoia occurrence and distress dynamically over time.

6.4.2.1 Emotion dysregulation and paranoia occurrence

The unconditional model using paranoia occurrence as the outcome, along with both participant and day as grouping variables (random intercepts) was highly significant (p=<0.001) with an intraclass correlation coefficient (ICC) of 0.74. The ICC represents the ratio of the between-group variance and the total variance. This essentially indicates the amount of variance in the outcome which is accounted for by the data clustering or by the grouping structure (Musca et al., 2011; Hox, 2002). It is calculated by dividing the between-group variance by the sum of the between and within group variance (Kleiman, 2017).In this case, the ICC of .74 indicates 74% of variance occurs between participants.

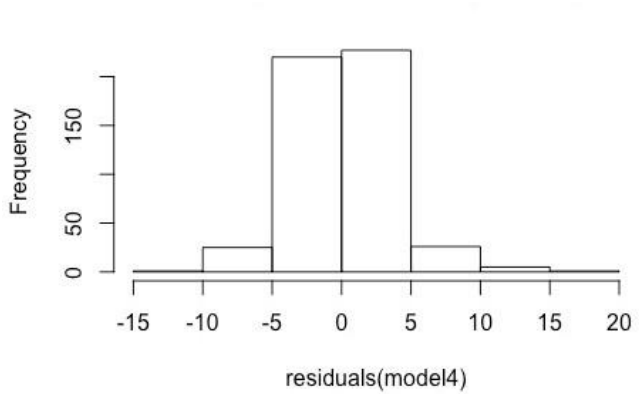
The full multilevel model showed a significant effect of emotion dysregulation on paranoia occurrence B=0.63, SE= 0.05, df=485.57, t=13.89, p<0.001. The model residuals were checked for normality by visually inspecting histogram and Q-Q plots (see Figure 6.2). The graphs suggested the residuals followed a normal distribution.

6.4.2.2 Emotion dysregulation and paranoia related distress

Multilevel modelling was next used to investigate the association between emotion dysregulation and distress scores. The unconditional model was again highly significant, (p<0.001), confirming the suitability of the data for use in MLM. ICC in this model was 0.65 indicating the variance is mostly between-participants.

Group allocation and emotion dysregulation were then added as fixed effects. This model showed a significant effect of emotion dysregulation on distress B=0.48, SE=0.33, df=358.938, t=14.74, p<0.001. The model residuals were checked for normality by visually inspecting histogram and Q-Q plots (see Figure 6.3). Again the graphs suggested the residuals followed a normal distribution.

Figure 6.2 Residuals plots for MLM paranoia occurrence



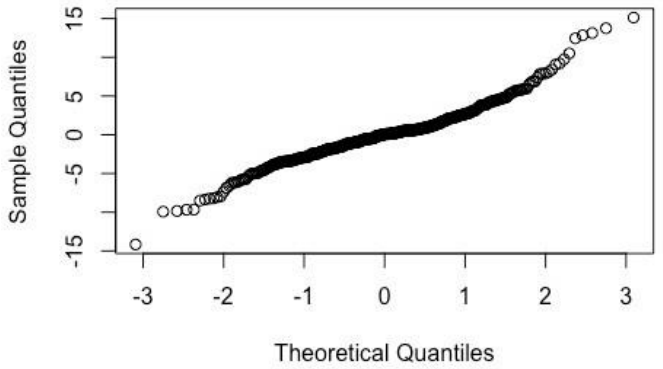
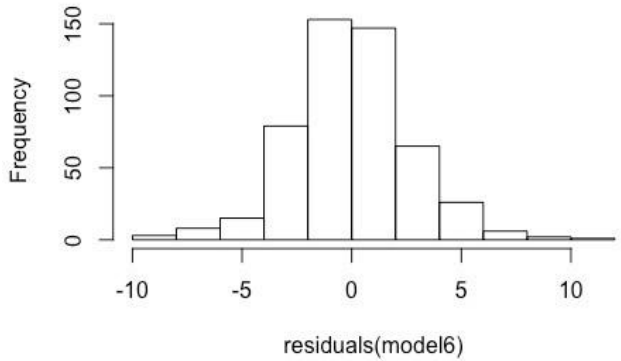
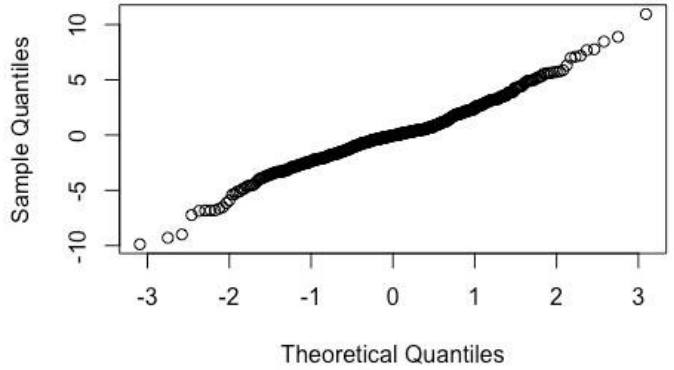


Figure 6.3 Residuals plots for MLM paranoia distress





Sensitivity analyses were undertaken to investigate whether data type had an effect on the model. Although the questions on both the questionnaire and ESM data were the same, the method of collection differed. Questionnaires were completed with the researcher, ESM questions were completed alone, using the mobile phone app. It was important to check if the method of collection had an impact on the results. The two MLM above were re-run including 'type of data' as a covariate.

6.4.2.3 Sensitivity analysis – paranoia occurrence model

There was no significant effect of data type B=1.18, SE=1.01, df=52.09, *t*=1.17, *p*=0.25, and the regression coefficient of emotion regulation was only minimally changed B=0.63, SE=0.05, df=480.68, *t*=13.99; *p*<0.001.

6.4.2.4 Sensitivity analysis - distress model

There was no significant effect of data type on this model B= -0.03, SE=0.65, df=25.90, *t*=-0.04, *p*=0.97, and as with the paranoia occurrence model, the regression coefficient of emotion dysregulation was robust to the addition of data type as a covariate B=0.48, SE=0.03, df=358.51, *t*=14.59, *p*<0.001.

6.4.3 Aim 3 - To test whether self-report of early emotional abuse and neglect predicts levels of emotion dysregulation during the first sampling period.

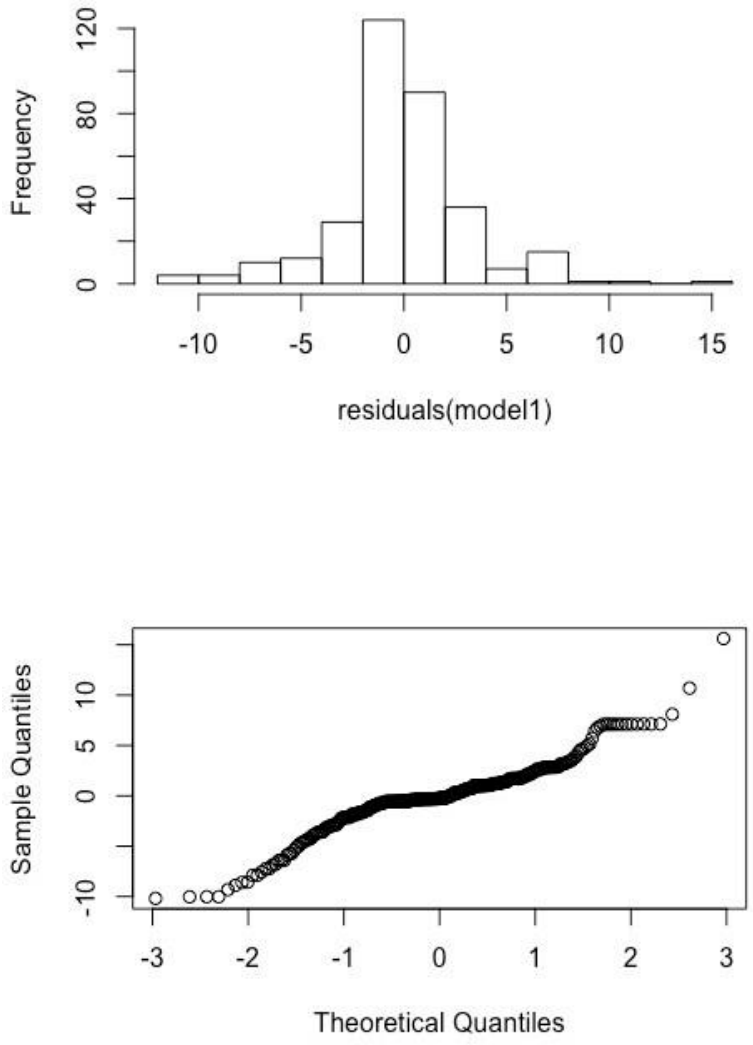
6.4.3.1 Results of model

The unconditional model was highly significant, indicating that the data were suitable for use in a multilevel model (*p*<0.001).ICC in this model was 0.56.

The independent variables in this model were 'trauma' using the CATS total score, and 'neglect' using the CCMS neglect subscale. Both independent variables were grand mean centred, then the multilevel model was specified with both predictors simultaneously. No significant effect of trauma on emotion dysregulation was detected: trauma B=0.03, SE=0.03, df=21.56, *t*=0.76, *p*=0.46; neglect B=0.11, SE=0.30, df=21.14, *t*=0.38, *p*=0.71. Model residuals

were checked for normality by visual inspection of a histogram and Q-Q Plot which showed model residuals were normally distributed, see Figure 6.4.

Figure 6.4 Residuals plots for MLM



6.5 Secondary Research Questions

6.5.1 Aim 4 - To test whether emotion regulation mediates the trauma-psychosis association at baseline and over time.

6.5.1.1 Results of models

Mediation models were tested to investigate whether emotion regulation mediates between childhood trauma and paranoia. Two models were specified, both using the baseline trauma

total (CATS) and neglect (CCMS) scores, entered simultaneously. The first model used baseline scores from the negative emotion dysregulation (CERQ) and persecutory ideation (GPTS) subscales; and the second model used end of treatment scores for these variables, controlling for baseline. As with the above analyses, MLR and FIML were used.

As reported in Table 6.7, the direct effects between trauma and persecutory ideation were significant for all models with the exception of CATS total and post-intervention persecutory ideation. There was however no significant evidence of mediation in either the FIML or the completer-only data sets. The results of sensitivity analyses using completer data only are available in appendix 21.

6.5.2 Aim 5 - To investigate how the results from momentary assessment methods compare to those from standardised questionnaire measures.

In the original protocol, it was stated that analyses would be undertaken to investigate any differences in ESM data and questionnaire data. However, due to high levels of missing data in the ESM measure it was not possible to analyse this. Missing data in the baseline and post-intervention questionnaires was notably low (14.44% overall), with the majority of participants providing both baseline and post intervention data (N=20, 77%). However, ESM missing data was much higher. Data was only obtained from 61.5% of the total sample (N=16). Of the responders, only 56% provided pre and post study data for comparison (N=9) and only one of these participants provided data on at least 50% of the ESM questionnaires, and amongst other participants missing ESM data ranged from 54.2-100%.

Table 6.7 – Trauma and emotion regulation mediation model testing

Predictor		FIML data set b (SE)	p-value	Std B
Model: Trauma total (CATS) – pre intervention				
Control variables	Age (baseline)	1.497 (2.386)	0.530	0.098
	Gender (fixed)	4.872 (4.077)	0.232	0.167
	Education (baseline)	0.205 (1.212)	0.866	0.027
Effects	Trauma (CATS – total) – Emotion regulation (CERQ – negative)	0.133 (0.096) 95%CI (-0.056, 0.322)	0.167	0.349
	Emotion regulation (CERQ-negative) – Paranoia (GPTS – persecution)	-0.038 (0.197) 95% CI (-0.423, 0.348)	0.848	-0.023
	Trauma (CATS-total) – Paranoia (GPTS-persecution)	0.372 (0.077) 95%CI (0.222, 0.523)	**0.000**	0.608
	Total indirect effect	-0.005 (0.025) 95%CI (-0.055, 0.045)	0.843	-0.008
Model: Trauma total (CATS) – post intervention				
Control variables	Age (baseline)	0.497 (1.814)	0.784	0.041
	Gender (fixed)	-5.054 (2.690)	0.060	-0.217
	Education (baseline)	-1.119 (0.861)	0.194	-0.178
	Treatment allocation	-6.404 (5.797)	0.269	-0.221
	Baseline emotion regulation (CERQ - negative)	-0.323 (0.329)	0.327	-0.257
	Baseline paranoia (GPTS – persecution)	0.333 (0.199)	0.093	0.422
Effects	Trauma (CATS-total) – Emotion regulation (CERQ – negative)	0.097 (0.070) 95%CI (-0.041, 0.236)	0.167	0.250
	Emotion regulation (CERQ-negative) – Paranoia (GPTS – persecution)	0.166 (0.329) 95%CI (-0.480, 0.812)	0.615	0.133
	Trauma (CATS-total) – Paranoia (GPTS-persecution)	0.045 (0.143) 95%CI (-0.235, 0.324)	0.753	0.092
	Total indirect effect	0.016 (0.028) 95%CI (-0.039, 0.071)	0.563	0.034

Predictor		FIML data set b (SE)	p-value	Std B
Model: Neglect (CCMS) – pre intervention				
Control variables	Age (baseline)	-0.341 (2.569)	0.894	-0.022
	Gender (fixed)	5.900 (4.736)	0.213	0.200
	Education (baseline)	0.053 (1.466)	0.971	0.007
Effects	Trauma (CCMS-neglect) – Emotion regulation (CERQ – negative)	-0.471 (0.663) 95%CI (-1.769, 0.828)	0.478	-0.143
	Emotion regulation (CERQ-negative) – Paranoia (GPTS – persecution)	0.368 (0.243) 95%CI (-0.109, 0.844)	0.131	0.228
	Trauma (CCMS-neglect) – Paranoia (GPTS-persecution)	2.148 (0.991) 95%CI (0.206, 4.090)	**0.030**	0.404
	Total indirect effect	-0.173 (0.251) 95%CI (-0.665, 0.319)	0.491	-0.033

Predictor		FIML data set b (SE)	p-value	Std B
Model: Neglect (CCMS) – post intervention				
Control variables	Age (baseline)	-1.405 (1.953)	0.470	-0.119
	Gender (fixed)	-10.171 (3.818)	**0.008**	-0.446
	Education (baseline)	0.233 (0.995)	0.815	0.038
	Treatment allocation	-0.359 (6.538)	0.956	-0.012
	Baseline emotion regulation (CERQ - negative)	-0.055 (0.323)	0.858	-0.047
	Baseline paranoia (GPTS – persecution)	0.224 (0.207)	0.279	0.291
Effects	Trauma (CCMS-neglect) – Emotion regulation (CERQ – negative)	0.354 (0.465) 95%CI (-0.558, 1.265)	0.447	0.112
	Emotion regulation (CERQ-negative) – Paranoia (GPTS – persecution)	0.103 (0.309) 95%CI (0-0.501, 0.708)	0.738	0.084
	Trauma (CCMS-neglect) – Paranoia (GPTS-persecution)	2.575 (1.203) 95%CI (0.217, 4.934)	**0.032**	0.665
	Total indirect effect	0.037 (0.079) 95%CI (-0.119, 0.192)	0.644	0.009

6.6 Exploratory Analyses

6.6.1 Other potential mediators of the effect of the intervention on the primary measure of paranoia

As paranoia did show change, but emotion regulation did not, secondary exploratory analyses were performed on other measured variables to investigate potential untargeted mechanisms of change. The effects of treatment allocation on attachment, metacognitive beliefs about paranoia, hypervigilance and emotional distress were tested in a further series of regressions.

Significant effects were detected for positive beliefs about paranoia. Individuals in the intervention group reported significant reductions in both beliefs that paranoia is a survival strategy B= 4.97, SE=1.31; *p*=0.00 and in normalising beliefs about paranoia B= 5.57, SE=1.45, p=0.00. Negative beliefs about paranoia showed slight decreases in both groups. Models were again subject to the same sensitivity analyses as above, using completer-only data. Effects were found to be robust: survival strategy B= 4.97, SE=1.42, *p*=0.00; normalising beliefs B=5.57, SE=1.49, *p*=0.00. No effects were detected for attachment, hypervigilance or emotional distress (anxiety and depression). Significant models are reported in Table 6.8 and full results can be found in appendix 22.

Mediation analyses

Mediation models were tested using these other measured variables as potential mediators. Group allocation was entered as the predictor. Models were tested using GPTS persecutory delusions, GPTS delusions of reference, and GPTS total score as the outcomes. Potential mediation mechanisms were tested individually in simple mediation models.

In the model using GPTS persecutory delusions as the outcome, both BAPS survival strategy and BAPS normalising beliefs showed significant evidence of mediation (survival strategy: B=8.39, SE=4.16, *p*=0.04; normalising beliefs B=14.33, SE=7.00, *p*=0.04). Both subscales also showed significant mediation of GPTS total scores (survival strategy B=20.14, SE=7.105, *p*=0.01; normalising beliefs B=25.94, SE=11.39, *p*=0.02), and survival strategy also

significantly mediated between group and GPTS delusions of reference (B=8.87, SE=3.22, *p*=0.01). There was no evidence of mediation by attachment, hypervigilance or emotional distress (anxiety and depression).

Sensitivity analyses were conducted using 2000 bootstrap resamples on completer-only data. Where GPTS persecutory delusions was used as the outcome, the mediation effects of both survival strategy and normalising beliefs lost significance (survival strategy bootstrap indirect effect: B=8.59, SE=4.98, 95% CI 0.84, 19.75, *p*=0.09; normalising beliefs bootstrap indirect effect: B=16.00, SE=9.06, 95% CI 2.78, 39.59, *p*=0.08).

In the other models where significant mediation effects were found, the indirect pathways retained significance when tested using completer data. In the models using GPTS total score, sensitivity analyses indicated that survival strategy and normalising beliefs still mediated the effect of group allocation (survival strategy bootstrap indirect effect B=20.60, SE=9.22, 95% CI 7.27,43.86, *p*=0.03; normalising beliefs bootstrap indirect effect B=28.97, SE=13.92, 95% CI 7.13, 61.50, *p*=0.04. Survival strategy also still significantly mediated between group and GPTS delusions of reference in the completer dataset (bootstrap indirect effect B=8.87, SE=4.26, 95% CI 2.56, 21.45, p=0.04). See Table 6.9 for the results of the models discussed here. There was no evidence of mediation by any of the other measured variables.

Table 6.8 - Exploratory analyses - significant linear regression results – group effects

Model	**Variable**	**Unstandardised coefficients *b* (SE)**	**95% CI for *b* (lower, upper)**	**p-value**	**Standardised coefficients, *ß***
Survival beliefs (BAPS) R^2 = 0.576	Constant	-3.929 (2.350)	-8.535, 0.677	0.095	-0.833
	Baseline	0.566 (0.127)	0.317, 0.814	**0.000**	0.644
	Treatment allocation group	4.971 (1.313)	2.398, 7.544	**0.000**	0.527
Sensitivity analysis - Completer data only R^2 = 0.576					
	Constant	-3.929 (3.259)	-10.315, 2.458	0.228	-0.833
	Baseline	0.566 (0.141)	0.289, 0.843	**0.000**	0.636
	Treatment allocation group	4.971 (1.421)	2.185, 7.757	**0.000**	0.555
Normalising beliefs (BAPS) R^2 = 0.430	Constant	4.912 (2.965)	-0.899, 10.724	0.098	1.120
	Baseline	0.087 (0.164)	-0.234, 0.407	0.596	0.106
	Treatment allocation group	5.573 (1.446)	2.739, 8.407	**0.000**	0.635
Sensitivity analysis - completer data only R^2 = 0.430 (0.414)					
	Constant	4.912 (3.462)	-1.873, 11.697	0.156	1.120
	Baseline	0.087 (0.149)	-0.205, 0.378	0.559	0.100
	Treatment allocation group	5.573 (1.487)	2.658, 8.487	**0.000**	0.644

Table 6.9 – Exploratory analysis – significant mediation models

Model		**Estimate (SE)**	**95% confidence bounds**	**p-value**	**Standardised effect**
Normalising beliefs (BAPS) and paranoia (GPTS-persecution)	a-path	5.573 (1.446)	2.739, 8.407	**0.000**	0.636
	b-path	2.570 (0.809)	0.984, 4.157	**0.001**	0.704
	Indirect effect	14.325 (7.001)	0.604, 28.046	**0.041**	0.447
	Direct effect	-27.576 (6.642)	-40.594, -14.557	**0.000**	-0.895
	1000 Bootstrap indirect effect*	15.999 (9.055)	2.778, 39.588	0.077	0.514
Survival strategy (BAPS) and paranoia (GPTS-persecution)	a-path	4.971 (1.313)	2.398, 7.544	**0.000**	0.513
	b-path	1.688 (0.568)	0.575, 2.801	**0.003**	0.568
	Indirect effect	8.392 (4.164)	0.231, 16.553	**0.044**	0.280
	Direct effect	-19.820 (5.129)	-29.873, -9.768	**0.000**	-0.670
	1000 Bootstrap indirect effect*	8.585 (4.980)	0.837, 19.747	0.085	0.289
	a-path	5.573 (1.446)	2.739, 8.407	**0.000**	0.636
	b-path	4.655 (1.203)	2.297, 7.014	**0.000**	0.776

Normalising beliefs (BAPS) and paranoia (GPTS-total)	Indirect effect	25.943 (11.388)	3.622, 48.264	**0.023**	0.493
	Direct effect	-43.621 (10.124)	-63.465, -23.778	**0.000**	-0.829
	1000 Bootstrap indirect effect*	28.974 (13.919)	7.128, 61.499	**0.037**	0.565
Survival strategy (BAPS) and paranoia (GPTS-total)	a-path	4.971 (1.313)	2.398, 7.544	**0.000**	0.510
	b-path	4.051 (0.787)	2.509, 5.594	**0.000**	0.786
	Indirect effect	20.137 (7.105)	6.212, 34.062	**0.005**	0.401
	Direct effect	-34.230 (7.708)	-49.337, -19.123	**0.000**	-0.681
	1000 Bootstrap indirect effect*	20.601 (9.221)	7.296, 43.862	**0.025**	0.419
Survival strategy (BAPS) and paranoia (GPTS-reference)	a-path	4.971 (1.313)	2.398, 7.544	**0.000**	0.528
	b-path	1.784 (0.579)	0.648, 2.919	**0.002**	0.669
	Indirect effect	8.866 (3.223)	2.549, 15.184	**0.006**	0.353
	Direct effect	-8.343 (5.702)	-19.519, 2.834	0.143	-0.332
	1000 Bootstrap indirect effect*	8.866 (4.255)	2.564, 21.449	**0.037**	0.366

*Bootstrap indirect effects are computed using completer data only as the missing data estimator (FIML) is not compatible with the bootstrap process

6.6.2 Feasibility assessment

The evaluation of the study against pilot feasibility criteria was added as a post-hoc assessment due to the numerous challenges faced throughout the trial. The criteria developed by Shanyide, Pickering & Weatherall (2011) address a number of areas including randomisation, adherence, acceptability and safety. Details relevant to this trial are summarised below and evaluated further in the discussion.

Of the 83 referrals received 81% were eligible, of whom 39% consented and were randomised. The most common reasons for declining to participant were perceiving participation as involving too much effort, not wishing to be involved in research, and not wishing to take part in group-based therapy. The study start date was delayed by five months, and the recruitment period was extended by one month due to the time taken to apply for, and receive, approval from the NHS Caldicott Guardian (who governs access to confidential patient data, and information sharing).

The study recruitment rate was 2.0 participants per month, which was less than the required and expected rate of 2.6 per month. Potential reasons for this include poor engagement from a number of potential referrers and a reduction in researcher availability (i.e. a move to part-time hours). This meant only 76% of the planned target sample size was achieved. There were no formal withdrawals from the study, but four participants withdrew or were withdrawn from therapy group, and six participants withdrew or were withdrawn from the control. The most common reasons for participants leaving the study early was due to hospital discharge and being unable to attend the remaining sessions due to either issues with access to transport or a return to work. All participants completed baseline measures but five participants (19%) did not provide data for the end of treatment assessments. These were all participants who has been discharged and could not be contacted thereafter.

There were some methodological limitations in relation to randomisation and blinding. The randomisation sequence was generated in advance of the study, however due to staff shortages in the wards the researcher had to escort participants to the sessions each week,

and therefore group allocation could not be concealed. It is not possible to rule out researcher bias due to this lack of blinding. Allocation was revealed to participants following the baseline PANSS interview, however later stage randomisation following the ESM data collection would have been more advantageous to minimise missing data and avoid unnecessary attrition.

Participant feedback overall was positive, but there were greater rates of non-attendance for the control group sessions. No data were collected on cost at this stage, however delivery of the intervention involved at least 70 hours of a clinical psychologist's time, and the art group required at least 70 hours of an activity centre staff member's time (75 minutes per session including the 60 minute group and 15 minutes set-up time).

It was feasible to complete questionnaires and conduct the PANSS interviews with participants, and missing data rates were low with 85.6% of data being complete. The ESM was not feasible in this sample with missing data rates being much higher (49% to 100% per participant). There was no evidence of trial-attributable adverse events. Two events were referred to the DMEC for consideration but neither was attributed to trial participation. Of the participants who completed the adverse events questionnaires, no further events were identified, although notably the emotion regulation intervention group had higher reported rates of paranoia than the control group at the end of treatment assessment. This was a single-site trial so multi-site issues have yet to be considered.

In summary, a definitive trial should be based on a recruitment rate of at least 2.0 per month per 0.5 FTE researcher, a treatment and control engagement rate of 100% and 77%, and a missing post-treatment data rate of 19%. ESM cannot be recommended as a primary outcome, but the GPTS could be used as an alternative. If such a trial were to go ahead, it should carefully monitor safety, and should also consider whether a third TAU arm is necessary to determine the relative contributions of specific and non-specific treatment effects to any group differences.

6.7 Summary of key findings

There was no evidence that the intervention was effective in changing emotion regulation ability in attendees. Post-intervention scores suggest the intervention may have had a negative impact on paranoid symptom recovery, with the control arm experiencing greater self-reported symptom reductions than individuals in the intervention group. It is therefore not possible to draw any conclusions about the relevance of emotion regulation to paranoia in this sample, and whether improving emotion regulation would lead to reductions in paranoia.

The exploratory analyses suggested a potential indirect benefit through metacognitive beliefs, particularly beliefs that paranoia is a survival strategy, and normalising beliefs about paranoia, however the presence of large negative direct effects somewhat cancels out the impact of the small indirect effects. The confidence intervals for the significant indirect effects are notably wide, and despite several moderate effect sizes, this clear imprecision cannot be ignored.

The use of ESM data for the primary outcome analysis was not feasible due to high levels of missing data. The self-report questionnaires and interview measures were well received and missing data levels were much lower. The study recruitment rate was lower than anticipated but retention was high, with the majority of participants attending at least one group session and providing end of treatment data. Full discussion of the study findings can be found in Chapter 7.

6.8 Limitations

The study had a number of limitations and should have been designed as a pilot-feasibility trial. Outcomes should have focused on recruitment, retention and levels of data completion, as opposed to testing efficacy hypotheses. Data should have been gathered directly on the acceptability of the intervention and control sessions, along with the formal recording of reasons for declining to participate, and reasons for withdrawal. It also would have been useful to gather information from participants and clinical staff about improvements which could be made to study processes, including the referral process and the intervention itself.

The study was negatively affected by a lack of staff availability in the hospital. Initially the referral process was slow potentially due to a large proportion of nursing bank staff who were unfamiliar with which patients may be eligible. Some may have been reluctant to make referrals on wards where they did not regularly work. The referral process improved with more frequent attendance by the researcher at ward meetings, and greater familiarity with staff. Any future study should aim to establish relationships with key staff early in the recruitment process, and maintain a presence in the ward environment regularly.

The sample recruited was at risk of bias in a number of ways. The majority of referrals were for patients who were relatively well given the acute context. This may have been an artefact of staff nervousness in making referral, or may have been an attempt to only refer individuals who the staff members perceived as willing and able to engage in group sessions. Some individuals did decline on the basis of the intervention being group-based, yet overall the group appeared to be well received, and evidence does suggest there are advantages to engagement with group therapy for individuals with a diagnosis of psychosis (Braehler, Harper & Gilbert, 2013, Burlingame, Strauss & Joyce, 2013). A group program has the advantage of replicability if the content is manualised; it can reach a greater number of participants in a short period making it efficient and cost effective for staff, and often outcomes are well defined and measureable as all participants will follow a very similar schedule of goals (Peters & Kanas, 2015).

The recruited sample was small and although randomisation was a strength, the lack of blinding was a significant change from the original protocol and it is not possible to rule out potential researcher bias. The study did benefit from an active control condition as opposed to treatment as usual, however a negative effect of the intervention was found, which conflicted with informal qualitative feedback received from participants. Small trials such as this one tend to produce varied results and regardless of whether positive or negative effects are found this cannot be assumed to be representative of the ‘true effect’. Only a larger-scale and adequately

powered study will be able to offer evidence of this, as will a meta-analysis of numerous smaller well conducted studies.

This study was given approval to include participants for a period of up to 16 weeks. This flexibility was a clear strength, enabling participants to complete the full group program regardless of conflicting clinical or tribunal appointments, days where participants less well or less motivated to attend, and also allowed for clinician absence. However group attendance could be erratic, and the attendees and group size regularly varied, which may have prevented the building of group familiarity and trust.

Diagnoses were confirmed by clinical staff or case notes, as opposed to a clinical research interview and it may be the case that individuals who did not wish to complete the measures or discuss their past adversity, symptoms or difficult emotions may have declined to participate on this basis (Legerski & Bunnell, 2010). As there was no formal recording of reasons for declining, it is not possible to rule out self-selection bias.

6.9 Implications and recommendations

The results of this study serve as a reminder that it should not be assumed that psychological interventions can only beneficial, and that all treatment must be evaluated for safety as much as efficacy. The findings here raise a number of considerations which should be taken into account by future studies looking to use a similar group-based intervention.

Both groups did experience reductions in paranoia following the intervention, however analyses detected a greater improvement in symptoms for the individuals in the control group. This is suggestive that the intervention group was not beneficial, and may have even been harmful, to the recovery of attendees. A degree of regression to the mean is expected, particularly in a clinical sample, however the delayed rate of recovery in the intervention groups is concerning on safely grounds, and should be taken into account before any future research is undertaken using this intervention.

Firstly, this may have been attributable to the small sample size. The study may have been just as likely to find a positive effect of the intervention. It is well recognised that meta-analyses often record an array of different effects from small-N studies, from large positive effects, through null effects to large negative effects, often accompanied by wide confidence intervals. Imprecision is common in such studies, and this must be taken into account when considering study results. This indeed is the reason for conducting such meta-analytical reviews, in order to gain a more thorough understanding of the 'true' effect. Nonetheless, patient safety must be prioritised, and since a negative effect has been found, the intervention must be carefully considered before any future use.

Secondly, evidence suggests that patients with a diagnosis of psychosis habitually make greater use of suppression as an emotion regulation strategy (van der Meer et al., 2009; Kimhy et al 2012). Asking participants to bring to mind and discuss times they have experienced difficult emotions, which they would ordinarily have suppressed, will have forced them to confront some of these difficult emotions. Despite the weekly relaxation and mindfulness aspects, the more emotionally challenging elements of the group content may have exceeded what participants felt able to cope with, particularly as some were inpatients experiencing moderate to severe ongoing symptoms. Some guidance suggests that participants should be offered adjunctive individual therapy to deal with issues of trauma and distress following a course of group-based treatment (Braehler, Harper & Gilbert, 2013) or that groups should avoid dealing directly with any issues which provoke anxiety or elevate negative affect (Kanas, 1996). Others assert the advantages of group therapy as part of a more integrative therapeutic approach (Kanas, 1996 & 2020; Pearson & Burlingame, 2013).

The group setting itself may also have introduced an additional level of difficulty for some participants. Patients with paranoid and persecutory ideation have been found to avoid social situations due to stress, suspiciousness and hypervigilance (Michail & Birchwood, 2018; Pot-Kolder et al, 2018). Studies have found busy environments to increase paranoia and distress in individuals with existing symptoms (Veling et al., 2016; Pot-Kolder et al., 2018), and have

suggested that environmentally induced stress can be linked to both the onset and relapse of psychotic phenomena (Alvarez-Jimenez, Priede & Hetrick, 2012; Phillips, Francey, Edwards & McMurray, 2007). There are a number of hypothesised reasons for this social anxiety, including self-stigma or low self-confidence, which leads to negative social comparisons and low self esteem (Freeman et al., 2014: Atherton et al., 2016). Understandably this may have an impact upon an individuals' willingness or ability to engage with a group. Although the control condition also involved group attendance, there was no therapeutic content, nor any discussion of illness factors.

The use of safety behaviours, which are common in those with paranoid thoughts can also block the gathering of social information from others due to avoidance or lack of communication; and often work to enhance or maintain paranoid beliefs about the threat posed by others, by preventing the collection of disconfirmatory evidence (Freeman, 2007; Simpson, MacGregor, Cavanagh & Dudley, 2012; Pot Kolder et al., 2018). In a group which relies upon engagement from attendees, these social challenges may have had an effect on the conduct of the group and the dissemination of content. If participants were reluctant to fully engage, this may have had a negative impact on the utility of the content for all group members. As a result the group content should be reviewed and carefully considered for its effectiveness and any potential ways it could be enhanced for use with this population before proceeding with its use in a future patients with acute psychosis.

As emotion regulation was not improved in participants in the intervention group, it is not possible to speculate on whether improved emotion regulation capacity will lead to improvements in the symptoms of paranoia and any associated distress. Future research efforts will be required to establish effective interventions to target and improve emotion regulation in individuals with a diagnosis of psychosis, before any evaluation of its effectiveness in reducing paranoid symptoms can be undertaken.

The results do have implications for future research in practice this area. In this sample, the ESM proved not to be feasible, however this conflicts with other research studies in similar populations using comparable research designs where completion rates off over 70% have been reported (Kimhy et al, 2010; Hartley et al, 2014b). Consultations with experts in the method failed to identify any obvious oversights in equipment, explanation, implementation and monitoring, however they did confirm adherence in this study was below the level they would expect. Missing data was notably higher in the post-intervention period which may reflect a lack motivation to continue contributing to the research after having completed the group sessions. A number of participants were discharged during their time in the study with some returning to work or study, impacting access to their mobile device, and others may have been keen to distance themselves from their inpatient experience. Future studies should carefully consider the risk of missing data when using ESM in acute populations, given the comprehensive attempts made in this study to mitigate this. Acceptable levels of data completion should be specified a-priori and sample sizes should be calculated to ensure adequate data can be collected for analysis. Late randomisation, following the first period of ESM data collection, may be beneficial as this would allow the exclusion of individuals who provide insufficient data.

Fundamentally this research, in its current form should not be taken forward to a definitive trial. Further work is required to first identify an intervention which is safe and efficacious in changing emotion regulation capacity in this population. Potentially a third 'treatment as usual' arm should be added alongside an active control task to allow comparison. This would assist with the identification of factors which contribute to improvements, which are not attributable to the therapy provided. It may also be useful to include a qualitative element to gather information about participants' experiences of the intervention and control groups. Informal discussions with participants in this study suggested both groups were well received and beneficial, however this was not directly reflected in the end of treatment scores. There may have been a degree of socially desirable responding in this dialogue with the researcher, but

a pre-planned qualitative element would have enabled greater exploration of subjective participant appraisals. A full discussion of the implications of this study can be found in Chapter 7.

6.10 Conclusion

This study fundamentally should have been designed as a pilot feasibility study with appropriate outcomes for such a research design. Owing to the small sample size and lack of researcher blinding, it is important to avoid attempting to draw conclusions from the analyses which were performed as per the original pre-registered protocol. It is also however important to note the negative effect of the intervention which appears to have delayed improvements in paranoid symptoms for participants in the intervention group. Further work will be required to establish and test an alternative intervention which is effective in reducing emotion dysregulation in individuals with a diagnosis of psychosis. The ESM data suggested there was a relationship between emotion dysregulation and paranoia occurrence and distress. This is echoed by wider research literature, so this relationship merits further investigation. The tentative evidence suggesting metacognitive beliefs may act as mediators should also be investigated by future studies to determine whether this is a replicable finding. The low rates of engagement with the ESM should offer a note of caution to future researchers intending to use this form of data collection, however the high rates of attendance, particularly in the intervention group suggest that research of this type is of interest to, and is found to be acceptable by individuals with a diagnosis of psychosis.

Chapter 7 - General discussion

7.1 Overview of chapter

This thesis was concerned with identifying mediators of the trauma-psychosis relationship. The preceding chapters have described the results of a systematic review of the current evidence, along with the rationale, procedure and results of two empirical studies which aimed to address gaps in existing knowledge. This chapter will review the findings from all studies, and will consider these in the context of theoretical models of trauma and psychosis. The questions answered by this thesis will be discussed, as will further questions emerging from the results. Remaining gaps in understanding will be identified as targets for potential future research.

7.2 Summary of main findings

The aim of this thesis was to investigate the factors which mediate the relationship between childhood trauma and the development of the positive symptoms of psychosis in adulthood. In order to fulfil this aim, an extensive systematic review of the current research literature in this area was conducted. The review found that whilst mediation studies are numerous, no mediation mechanisms have evidence of moderate to large effects. All effect sizes are trivial to small and study quality varies widely. Cognitive mediators were well supported with consistent small effects based largely on good quality evidence. PTSD symptoms and dissociation also had consistent evidence of their role as mediators but evidence was of poorer quality and was limited to small clinical studies. Other categories were less well supported, or less well researched. The evidence for mood factors, and attachment style did not suggest evidence of mediation. The majority of effects were trivial to small and narrow confidence intervals suggested these effects to be precise. Social defeat and emotion regulation were lesser studied and therefore require further research before any firm conclusions can be drawn.

A number of methodological concerns were also highlighted. The current evidence showed signs of biased samples which were often inadequately described. Potential confounding factors were poorly controlled, missing data was poorly handled or not addressed, and there was a lack of transparency around power calculations. There was also little evidence of pre-registration of study protocols in the public domain ahead of data collection taking place. These methodological issues have a significant impact on the quality and integrity of the evidence. With this in mind, the empirical studies in this thesis were designed to overcome some of these challenges and limitations.

The first empirical study aimed to generate a mediation model of childhood trauma and psychosis in a general population sample, using an online questionnaire. A number of potential mediators were measured, based on categories identified by the systematic review. The study design set this work apart from the majority of studies included in the review as it involved a more robust two-stage process which transparently compartmentalised the exploratory phase of model development as being separate from the model testing phase. Network analysis was used, followed by structural equation modelling to develop a hypothetical model. This was then pre-registered ahead of the second round of data collection. The original exploratory model showed a significant mediation pathway from psychological abuse through avoidant attachment and difficulty identifying feelings, to subclinical persecutory ideation, however this pathway was not significant in the second sample.

The second empirical study aimed to investigate whether an intervention targeting emotion regulation would help to reduce paranoid symptoms in a sample of individuals with a clinical diagnosis of psychosis. An interventionist-causal RCT design was used, in which participants were randomised to attend either the intervention or an active control group. Data collection was partially undertaken using ESM delivered using a mobile phone app. High levels of missing data on the ESM measure meant analyses had to be performed using alternative data, but the study protocol was adhered to as far as possible. All changes were reported transparently, accompanied by a rationale for making such changes. The target sample size

was not achieved and it was not possible to maintain researcher blinding. The analyses indicated a small negative effect of the intervention and there was no evidence to suggest that the intervention sessions had caused changes in emotion regulation. Additional post-hoc analyses were undertaken to consider the study against criteria for a pilot feasibility trial.

7.3 Key findings

This section will bring together the key findings from this thesis, and discuss them in the context of wider evidence and theory. Each potential mediator, or category of mediators, is discussed in turn. The findings from the research across this thesis are brought together in an attempt to answer the research question 'how does trauma cause psychosis?'. Further research questions arising from this research are highlighted, as are potential future targets for research.

7.3.1 Cognitive factors

Findings from the systematic review indicated the strongest evidence in support of mediation came from studies of cognitive factors. This category contained a larger number of studies and investigated potential mechanisms across heterogeneous populations. Although effect sizes were small, evidence is consistent in support of cognitive beliefs and appraisals acting in a mediating role.

This evidence largely supports cognitive models of psychosis development, particularly those which also take into account environmental risk factors such as childhood trauma. Models suggest that early interpersonal adversity leads to the development of negative schemas about the self and others (Read et al., 2005), as well as deficits in processing information and emotional arousal (Kuipers et al., 2006). This may lead to intrusive thoughts or experiences which are either misattributed to external sources, or misinterpreted as threatening (Kuipers et al., 2006; Fowler et al., 2006, Holmes et al., 2004). Morrison (2001) suggests that internally generated thoughts which are mistakenly attributed to external sources, due to faulty self-beliefs and dysfunctional social understanding, can give rise to persecutory delusions. These

faulty beliefs may have their roots in early trauma which may then lead to elevated threat anticipation, and sensitivity to stress; leaving the individual vulnerable to interpreting anomalous experiences in distressing ways (Broome et al., 2005; Garety et al., 2007). The models make clear that biases such as jumping to conclusions (making decisions based on limited or incomplete evidence) or confirmatory bias (rejecting any evidence which does not 'fit' with a theory) are logical precedents to paranoid or persecutory thinking (Moritz et al., 2010). Rigidity of thought patterns and the refusal or inability to consider alternative explanations means fleeting intrusions may lead to rumination and fixation on fears of being harmed.

There is some evidence of specificity between emotional or interpersonal trauma and the development of persecutory beliefs (Ered & Ellman, 2019; Gibson, Alloy & Ellman, 2016; Shevlin, McAnee, Bentall & Murphy, 2014; Bentall & Fernyhough, 2008). This is important when considering psychosis under the single symptom approach (Bentall, 2003; Kuipers et al., 2006, Garety et al., 2001) and attempting to understand how specific traumatic experience may lead to the development of specific symptoms. The theoretical models discussed above are supported by the evidence from this category, suggesting that cognitive factors are a plausible mediation mechanism, however the other Bradford Hill criteria (dose-response, reversibility and experimental support) will require further studies to be undertaken. Other recent reviews of the mediators of trauma and psychosis concur that cognitive factors are important (Alameda et al., 2020; Williams et al., 2018), however gaps remain in the knowledge of whether other potentially important factors such as metacognition and the jumping to conclusions bias mediate these pathways in similar ways to negative beliefs. Cognitive factors were not measured in the empirical studies in this thesis, as the evidence is already relatively strong, however both studies did measure metacognition, and results are discussed below.

7.3.2 Metacognition

Both empirical studies found some evidence to suggest that metacognitive beliefs about paranoia may have an important role in the trauma-psychosis relationship. The exploratory

network analysis in the online study highlighted beliefs about paranoia as salient, and subsequent SEM did support this with a number of significant paths. Theoretically, it was reasoned that beliefs about paranoia would manifest after the development of paranoid thoughts, and therefore may act as maintaining factors, whereby an individual believes that their paranoid thoughts are protecting them from harm. As the study was focused on mediators between childhood trauma and psychosis, metacognition was not investigated further in the model, however it may prove to be an important future target for further research.

In the clinical study there were significant group mediation effects detected for positive and normalising beliefs about paranoia. BAPS normalising beliefs mediated between group allocation and both persecutory delusions and paranoia total score, with only the total score pathway remaining robust to sensitivity analyses. BAPS survival strategy mediated between group and persecutory delusions, delusions of reference and total score, however the persecutory delusions pathway lost significance in the sensitivity analysis. This highlights the fragility of a number of these effects, and emphasises the need to use extreme caution when interpreting the data from analyses of such small samples.

In terms of correlational evidence, elevated positive and negative beliefs about paranoia have been found to be associated with increased suspiciousness (Morrison & Wells, 2003; Murphy et al., 2017) and to be predictive of paranoia frequency and distress (Gumley et al., 2011). Conversely, normalising beliefs have not been found to be associated with paranoid ideation (Morrison et al., 2005; Gumley et al, 2011; Murphy et al., 2017). Interestingly, the intervention group here reported much greater decreases in positive and normalising beliefs in contrast with the control participants, despite retaining higher levels of reported persecutory ideas. The linear regression results were robust to the sensitivity analyses using completer data only, however as above, all results must be subject to cautious interpretation due to the small sample and lack of power. Both groups experienced slight decreases in negative beliefs about paranoia, but these were non-significant.

These findings are unexpected as positive beliefs about paranoia have been viewed as a potential maintenance factor (Morrison et al., 2011). These may be part of a learned response, initially developed as a means of self-protection following the experience of early life adversity (Bentall & Fernyhough, 2008; Varese et al, 2012). This may once have been adaptive, and is now potentially disruptive and damaging to everyday life (Murphy et al., 2017). It has been proposed that individuals who view paranoia as a survival strategy may be disinclined to relinquish those beliefs which they perceive as protective against threats of harm. This can lead to a difficult cycle of engagement with, and subsequent attempted suppression of, paranoid thoughts. As these grow stronger they bring elevated distress (Morrison et al., 2011). It is interesting that participants in the intervention group appear to have reduced their perceptions of paranoia as protective following the intervention, yet corresponding decreases in paranoid symptoms were not detected. It may be the case that the content of the group highlighted the dysfunctionality of paranoia as means of self protection, and that participants have internalised some of these beliefs, but this has been insufficient to significantly influence their experience of symptoms. The decrease in positive beliefs was not matched by an increase in negative beliefs about paranoia either, so it does not appear that the participants in the intervention have increased their metacognitive awareness of the potential harms brought about by paranoid thinking.

Studies of cognitive therapies have found that normalising beliefs increase over the course of an intervention in line with decreases in other factors such as self-stigma, feelings of shame, and fears about the uncontrollability of paranoid thoughts (Morrison et al., 2016, Murphy et al., 2017). The intervention in this study appears to have had the opposite effect, with normalising beliefs being reduced. Whilst the intervention may have had a beneficial effect on reducing positive beliefs about paranoia, this simultaneous reduction in normalising beliefs may have had a deleterious effect on that benefit.

7.3.3 PTSD symptoms and dissociation

PTSD symptoms and dissociation have also attracted significant research attention. This is perhaps unsurprising since PTS symptoms often emerge as a response to traumatic life experiences, and have been reliably correlated with psychotic symptoms (Aakre et al., 2014; Buckley, Miller, Lehrer & Castle, 2009; Kilcommons & Morrison, 2005). However the majority of studies have been conducted in small clinical samples and effect sizes are consistently significant but small.

Some researchers have suggested there may be a specific dissociative subtype of psychosis, and a psychotic subtype of PTSD (Moskowitz et al., 2009; Moskowitz, Barker-Collio & Ellson, 2005; Ross, 2006). However this raises questions around temporal ordering, an important criteria for understanding causality, in which the hypothesised cause must precede the outcome (Hill, 1965). Studies have found evidence that PTSD symptoms mediate between abuse and dissociation (Terock et al., 2016), but also that dissociation mediates between complex childhood trauma and PTSD (van Dijke et al., 2015). Without studies which can offer evidence for causal sequencing, it is not yet possible to establish whether PTS symptoms and dissociation are causally implicated in the development of psychosis following childhood trauma.

7.3.4 Attachment

Both the systematic review and the online empirical study found no evidence that insecure attachment acts as a mediator between childhood trauma and psychosis. This corresponds with findings in Alameda et al. (2020), but conflicts with Williams et al (2018) who claim there is consistent evidence in support of attachment as a mediator. Effects indeed are consistent, but of trivial magnitude with relatively precise confidence bounds. This serves to highlight the risk of synthesising evidence without regard for effect magnitude over more simplistic significance values. There were also no high quality studies in clinical populations, however subclinical studies consistently found very small effects and therefore it may be worthwhile for

future studies to consider how attachment may instead function as an interim step in a complex multiple mediator model rather than pursue evidence of outright mediation.

Whilst insecure attachment may not be a mediation mechanism, it is present in a number of theoretical models and there is a great deal of correlational evidence of its importance to both trauma and psychosis. Associations have been found between insecure attachment and subclinical positive symptoms (Berry et al., 2006; Carr, Hardy & Fornells-Ambrojo, 2018); and paranoid thinking in an early psychosis sample (Korver-Neiberg, et al., 2013). Higher instances of insecure attachment have also consistently been detected in clinical samples (Korver-Nieberg et al, 2015; Carr, Hardy & Fornells-Ambrojo, 2018; Chatziioannidis et al, 2019). However not all evidence is consistent, with other clinical studies finding no significant association between attachment and positive symptoms (MacBeth et al., 2011), nor did insecure attachment predict symptom recovery (Gumley et al, 2014).

It has been suggested that early attachment styles continue to influence social and interpersonal functioning into adulthood (Berry et al., 2006). Disrupted early attachment due to traumatic experiences such as abuse, separation or loss may lead to the development of insecure attachment styles, leaving individuals with reduced social support in adulthood (Moreira et al., 2003). They are also more likely to make use of using less adaptive coping mechanisms (Ludwig, Werner & Lincoln, 2019; Nittel et al., 2018; Laloyaux et al., 2015). The early cognitive templates of caregiver and self may persist into adult life, influencing behaviour and inhibiting the understanding of the behaviour of others (Berry et al., 2007). Anxious attachment reflects a negative self-concept, with positive beliefs about others meaning negative events are more readily internally attributed in a self-blaming manner (Wickham, Sitko & Bentall, 2014). Conversely, an avoidant attachment style typically conceptualises the self in a more positive manner, with negative beliefs about others (Bartholomew & Horowitz, 1991). Thinking negatively about others can lead to social withdrawal, isolation and further misattributions in a cycle which may promote the maintenance of paranoid thinking (Freeman et al, 2002). Patients with avoidant attachment styles have been found to have both higher

paranoia symptom ratings and higher interpersonal hostility (Berry et al., 2008). This links with the 'poor me' style of paranoid thinking, in which individuals feel unjustifiably victimised or persecuted by others, and the intentions of others are mistakenly assumed to be malevolent (Melo & Bentall, 2013).

The network analysis did highlight an association between psychological abuse and avoidant attachment in the exploratory sample. Individuals who experience psychological abuse, consisting of actions such as shaming, taunting and humiliating, may develop uncertainty around caregivers' intentions and behaviours. This may lead them to internalise beliefs that others are threatening. From this, an insecure attachment bond is likely to form, and various developmental processes may be negatively affected, including mentalization abilities (Korver-Nieberg et al, 2014). Mentalization is the process by which an individual recognises their own internal mental states and understands that these may differ from those of others (Fonagy, 2012). It has been described as a form of social cognition through which an individual can attribute meaning to behaviour and can function effectively in interpersonal relationships (Korver-Nieberg et al., 2014). Faulty mentalization abilities may arise as a consequence of these early life adverse experiences including abuse and neglect, in which caregivers fail to engage with the child in a meaningful manner so as to model these skills. Poor mentalization abilities have been detected in patients with psychosis who have insecure attachment styles, and has been found to mediate the association between attachment and psychosis (MacBeth et al., 2011). Whilst avoidant attachment was not a reliable mediator in the online study here, evidence suggests the influence of both attachment and mentalization on attributional style (Kover-Nieberg et al., 2014; Bentall et al., 2009; Berry et al., 2008) may be worthy of further investigation.

7.3.5 Alexithymia

Although mentalization ability was not measured in the online study, its potential influence may be highly relevant to alexithymia. This is a difficulty with identifying and verbalising

emotional states, along with a tendency towards very concrete, externally oriented thinking (Sifneos, 1972; Suslow & Donges, 2017; Lopez-Munoz & Pérez-Fernández, 2020). It has been found to correlate with both positive and negative psychotic symptoms and comprises both cognitive and affective elements (Seghers et al., 2011), however it had not been tested as a potential mediator by the time the systematic review was undertaken.

Alexithymia has been associated with affect dysregulation (McLean et al., 2006); higher levels of anxiety and depression (Bagby et al., 2020); greater severity of negative symptoms (van't Wout et al, 2007; Stanghellini & Ricca, 1995) and subclinical delusion-proneness (Larøi et al., 2008). It has also regularly been found to positively correlate with both childhood trauma (Seghers et al., 2011; Joukamaa et al., 2008; Bermond et al., 2008; Zlotnick et al., 2001 Berenbaum 1996) and insecure attachment. (Seghers et al., 2011; Godsmith & Freyd, 2005). Findings also indicate that alexithymia and PTSD are strongly associated (Frewen, Dozois, Neufeld & Lanius, 2008) and that elements of both may function as regulatory strategies for individuals to cope with negative thoughts and emotions (Terock et al., 2016). Thus far, evidence is still unclear, with some evidence of alexithymia mediating between PTSD and dissociation (Powers et al., 2015), but not between childhood trauma and dissociation (Terock et al., 2016). Importantly, no studies had investigated alexithymia as a potential mediator between childhood trauma and positive psychotic symptoms, so testing this in the online study in this thesis was novel.

In the hypothetical model from this study it was difficulty identifying feelings from the alexithymia measure (TAS-20) which was identified as the most prominent subscale between psychological abuse and paranoid thinking. Other studies have found higher difficulty identifying feelings in patients with schizophrenia, versus controls (Van der Meer et al., 2009; Fogley, Warman & Lysaker, 2014); difficulty identifying feelings to be significantly associated with trauma, even after controlling for other psychopathology (Joukamaa et al., 2008); and to be associated with a lack of emotional awareness which is particularly impactful in the experience of psychosis (Kimhy et al, 2020). However as the mediation pathway identified in

the exploratory sample was not signficant in the confirmatory data, there remains no evidence of DIF acting in a mediating role.

Regardless of the findings from the online study here, there does appear to be sound theoretical reasoning for the further investigation of the role of alexithymia. An inability to identify emotional states negatively affects the ability to engage with, and where needed, down-regulate emotional arousal (Moyal, Henik & Anholt, 2014). Experiences of abuse in childhood, without adequate modelling of emotional coping strategies may leave emotional disengagement as the only option to safeguard against distress. Much like the insecure attachment styles discussed above, this may start out as protective, but quickly becomes dysfunctional when it persists into adulthood. Learned disengagement may chronically inhibit the ability to recognise emotions, and may negatively affect self-regulation, empathy with others, and the ability to understand social cues (Weijers et al, 2016). As a result, individuals may 'hypermentalize', meaning they excessively misattribute the intentions of others, through a lack of ability to interpret and understand. This may lead to heightened threat-awareness which may eventually prompt the transition to paranoid thinking (Weijers et al., 2016).

If future evidence was found to support the importance of alexithymia, interventions which are known to target this could be tested in samples of individuals with paranoid symptoms and a history of childhood trauma. Individuals may benefit from mentalization based treatment (MBT) of which there has been one recent RCT in psychosis (Weijers et al., 2020a). This study found no difference at post-treatment as compared with a treatment as usual condition, however significant positive effects were detected for the MBT group at follow up.

The same authors also propose a new theory of non-affective psychotic disorder. This is highly complex and incorporates a number of factors, but it does include a potential pathway from childhood trauma, through insecure attachment and impaired development of metalization abilities, leading to difficulties with emotion regulation. This is proposed, along with social factors, to lead to prodromal psychotic symptoms and eventually to florid psychotic disorder

(Weijers et al, 2020b). Although there is currently no evidence to support alexithymia as a mediator, this along with mentalization require more research before it will be clear whether theoretical models such as the one by Weijers and colleagues can be supported.

7.3.6 Emotion regulation

Emotions and emotion regulation already feature in a number of theoretical models of the development of psychosis, however there is markedly little evidence available of its potential role as a mediator. The systematic review did find some evidence of very small significant effects, however the overall volume of evidence was lacking. Emotion regulation was not identified as an important potential mechanism in the online study and findings from the empirical clinical study were equivocal.

The intervention failed to change self-reported emotion regulation ability in the attendees, and both the intervention and control group experienced only minimal changes in emotion regulation over the course of the study. Given the lack of effectiveness in changing emotion regulation as the target mechanism, there is no evidence to support the use of this intervention further, however, all results from this study must be interpreted cautiously due to the very small sample size and inadequate power. The limited data collected in the study showed no evidence of emotion regulation acting as a mediator between trauma and psychosis in this sample.

The ESM data did however suggest that the use of negative emotion regulation strategies was associated with both the occurrence of paranoia, and the accompanying elevated levels of distress. The multilevel modelling analysis suggested these associations were highly significant, which is consistent with previous research (see below), however this must also be interpreted cautiously due to the very small sample, and the high levels of missing data on the original outcome measure. Further multilevel modelling did not indicate a relationship between childhood trauma and emotion regulation ability at either baseline or at the end of treatment assessment.

Poor emotion regulation has been found to be associated with an enhanced likelihood of experiencing paranoia (Westermann & Lincoln, 2011; Westermann, Kesting & Lincoln, 2012; Westermann, Boden, Gross & Lincoln, 2013). Individuals with a diagnosis of schizophrenia tend to use more dysfunctional emotion regulation strategies such as suppression; and fewer functional regulatory strategies such as acceptance, when compared with healthy controls (Gross, 1998; Gross & John, 2003; van der Meer et al., 2009; Perry 2011). However, studies have also found evidence to suggest that patients have comparable regulatory abilities to those of healthy controls when prompted with which regulation strategy to use (Opoka et al., 2020; Ludwig, Werner & Lincoln, 2019; Lincoln, Hartmann, Köther & Moritz, 2015a). These studies suggest that the difficulty instead lies in the awareness or understanding of the emotion in order to select the optimal response. This raises the question of insight, and whether participants will have had the ability to accurately report on their emotion regulation use if emotional awareness is negatively affected by their condition. Although alexithymia did not consistently mediate between trauma and symptoms in the non-clinical study, it may be pertinent for future research to consider how alexithymia may confound the reporting of emotions and emotion regulation in clinical research participants.

7.3.7 Social defeat

The systematic review highlighted the current lack of research into social factors as potential mediating mechanisms. All effect sizes were very small and confidence intervals did suggest precision, but studies varied in the quality of their measurement of each potential mediator. However the wider research literature does suggest that social factors are important to the development of psychosis. Maladaptive social functioning is likely to derive from difficult childhood environments, particularly those which lead to insecure attachment styles (Berry, Barrowclough & Wearden, 2008). The social and emotional repercussions include heightened sensitivity to threat, and an avoidant coping style, along with cognitive biases. These may negatively impact on attributions and the contextual processing of anomalous experiences (Freeman & Garety, 2002; Gumley & Schwanneur, 2006). Issues with social engagement,

potentially brought about by theory of mind deficits, can negatively affect how individuals communicate, build and maintain relationships. This may result in social withdrawal and isolation which have been associated with low self-esteem, increased positive symptoms and poorer treatment outcomes (Kuipers et al., 2006; Barrowclough et al., 2003).

Low self-esteem is also highlighted by cognitive models as co-occurring with negative self-beliefs (Kuipers et al., 2006; Garety et al., 2001). It is recognised to have a detrimental impact on interpersonal experience, self-stigma and the development and maintenance of social networks, all of which are relevant to the concept of 'social defeat' (Selten, van Os, Cantor-Graae, 2016; Selten, van der Ven, Rutten & Cantor-Graae, 2013; Selten & Cantor-Graae, 2005; Romm et al., 2011). Without supportive social environments, it is unlikely that negative affect or negative beliefs will improve, and disconfirmatory evidence cannot be sought about the occurrence and interpretation of intrusive experiences, which are then more likely to be interpreted in a distressing way (Morrsion, 2001). Whilst social factors do not currently have strong evidence of causal mediation, there are few studies which have examined this to date. However theory does suggest there may be merit in investigating potential cognitive and social mechanisms in multiple mediator models.

7.3.8 Mood factors

Previous research suggests that mood factors may function alongside social isolation in the maintenance of positive psychotic symptoms, rather than acting directly as mediators (Smith et al., 2006). Studies have repeatedly shown mood to be associated with the severity of positive symptoms (Hartley, Barrowclough & Haddock, 2013; Smith et al., 2006; Huppert & Smith, 2005). However it is not clear whether mood symptoms develop as a response to the onset of psychotic symptoms, or to the self-stigma, feelings of hopelessness and distress which often follow (Watson et al., 2006). In contrast to both Alameda et al. (2020) and Williams et al. (2018) who assert the important of affect factors, the systematic review found only trivial effects, based on large samples with relatively precise confidence bounds. There was consistency across the mechanisms being tested, and whilst significant effects were returned,

current evidence does not indicate that anxiety and depression are sufficient single of combined mediators in this relationship. As mood symptoms are common to many mental health conditions, further evidence of temporal sequencing is required, as will further studies in clinical samples.

7.4 Evaluation of methodology

One key strength of this thesis is the robustness of the methodology used across all three studies. The strengths and limitations of each individual study are discussed in the results chapters, however this section offers a broader overview of the novel methods applied throughout this thesis and highlights implications for future research.

The systematic review identifies the importance of effect size when attempting to draw comparisons between studies, as opposed to focusing only on significance. Equally it highlights study quality as an important parameter for consideration when interpreting study results. This sets the review apart from recent reviews of the same topic, and helps to identify not only gaps in the current evidence, but also systemic limitations in study conduct, quality and reporting.

The online study performed a robust confirmatory testing process after an iterative model development phase. Research in this area does not appear to have utilised such an approach to date. It may the case that some published research has presented the results of exploratory modelling as confirmatory findings, particularly given the lack of replication across studies in the systematic review categories. Efforts were made to meet the quality assessment criteria which were used to evaluate studies in the systematic review. Although the sample was open to bias due to the sampling method, all other criteria were met. It is notable in this study that the significant indirect pathway from the exploratory model was not found to be significant in the confirmatory data sample, despite a well-fitting data model overall. The results therefore do not support either insecure attachment or alexithymia as causal mediators in this sample and further exploratory work is required to better understand the connections between these variables.

The clinical study attempted to use a novel paradigm to investigate emotion regulation as a potential mediator by isolating and manipulating it in a controlled study design. The ESM was not a feasible means of data collection for use in this sample, despite having been used in a number of similar samples previously. Consultations with those experienced in using the method did not highlight any areas of oversight, and therefore this study can offer a note of caution to researchers hoping to employ the method in similar samples in future.

The intervention used in this study did not achieve the expected change in the potential mechanism. This raises questions about whether the intervention itself was problematic, in terms of format, content or delivery; or whether the measurement of the mechanism was inadequate. Evidence of potential harm from a small study such as this is not, in itself, inhibitory, but does serve to highlight the importance of recording adverse effects and events in clinical research. Regardless, the study findings cannot offer evidence about whether changing emotion regulation ability has an effect on the experience of paranoid symptoms.

Group therapy studies in clinical patients with psychosis have found positive effects for a number of group-based treatments including compassion focused therapy (Braehler et al., 2013); mindfulness based groups (Chadwick, Taylor & Abba, 2005) and cognitive behavioural therapy (Lecomte et al., 2008), although positive symptoms had returned to pre-treatment levels after 12 months (Lecomte et al., 2012). Review evidence also suggests positive effects on both psychotic symptoms and interpersonal and social factors as a result of group interventions (Burlingame, Strauss & Joyce, 2013). However, others have found no difference in group versus individual therapy (Hutton & Taylor, 2014), and some evidence indicates the efficacy of group therapy for psychosis may only be relevant to individuals in very specific phases of their illness, particularly those who are in recovery from an episode, as opposed to those who are still more acutely unwell (Kanas, 2003; Braehler, Harper & Gilbert., 2013). Some individuals did explicitly decline to take part as they did not wish to attend group therapy and this should be taken into consideration by future researchers considering undertaking similar research. However the benefits of group delivery should also be taken into account.

There is a reduction in time required, more participants can be included, and costs should be minimised in comparison with one to one therapies.

The other findings from the clinical study which suggest metacognitive beliefs about paranoia do function as mediators, and that emotion dysregulation is associated with paranoia occurrence and distress, should be interpreted very carefully given the limited data, sample size and power. Such findings will require replication in subsequent, adequately powered studies before they can be accepted.

Although a great deal more demanding than cross-sectional research, IC-RCTs offer a means of experimentally testing potentially causal mechanisms. This will generate evidence which satisfies the Bradford-Hill criterion of 'experimental evidence', which is often lacking. IC-RCTs are however demanding to conduct and less cost effective than cross-sectional alternatives. Arguably, evidence from a small number of well conducted IC-RCTs could bolster the importance of a causal mechanism in a way which could not be achieved by much larger volumes of cross-sectional research. For this reason it would be a worthwhile investment in time and research funding for future researchers to commit to using such experimental paradigms to generate more thorough causal evidence.

7.5 Conclusion - Implications of the thesis

This thesis aimed to identify mediators of the trauma-psychosis pathway. The systematic review highlighted the consistently small effects detected across the body of existing research. Even those mediators which are supported by significant evidence still only appear to mediate a very small proportion of the overall relationship.

Without an evidence synthesis which quantified the magnitude and quality of all detected effects, it would have been easy to assume mediation evidence was in a much stronger state than is truly the case. This raises questions about what researchers may be missing. If there is such a strong link between childhood trauma and psychosis, why is current mediation evidence unable to account for more of the causal effect? The systematic review highlighted

several quality and reporting weaknesses across the studies reviewed, which may have negatively affected the evidence. The online study made efforts to overcome these methodological limitations, and demonstrate the rigorous development and testing of a mediation model.

The clinical study was unable to offer evidence about the causal importance of emotion regulation as a mediator in the trauma-psychosis relationship. In this sample emotion regulation was not associated with childhood trauma, however it was associated with paranoid symptoms. Given the instability of the analyses based on such a small sample, these findings will require further investigation in larger samples. An intervention which can be shown to effectively change emotion regulation should be identified and trialled in a similar IC-RCT format. However there may be some merit in testing alexithymia in future clinical samples, to gain an understanding of how this may affect or even conflict with emotion regulation attempts.

There is currently no evidence to support the role of insecure attachment as a mediator. The systematic review reflects this, as does the lack of significant mediation effect in the confirmatory testing in the online study. The clinical study found no group differences or any effects of mediation when insecure attachment was tested. Given its frequent inclusion in theoretical models of psychosis development, this is highly important. Advocates of attachment theory may highlight that this apparent lack of evidence may be a result of inconsistent definition of terms and measurements throughout the field of research. To test whether this is the case, a fundamental shift is required to unify definitions and improve the measurement tools used to gather data. This should generate more efficient, generalizable and reliable research results. If the same lack of supporting evidence is found when research practices are improved, then firm conclusions can be drawn that attachment style does not mediate between trauma and psychosis.

Given the issues with low statistical power which include effects being missed or identified errantly as significant; and results being more difficult to reproduce, it is clear that it was misconceived to run analyses of group differences on the sample size recruited in the clinical

study (Button et al., 2013). As acknowledged in the results chapter, the study should have been designed as a pilot feasibility trial from the beginning. However the problems encountered in the study serve to highlight systemic problems within clinical trials for treatment development. Although initial phases primarily focus on establishing safety (Yao, Zhu, Jiang & Xia, 2013), using such small sample sizes not only increases the chance of finding false positives, suggesting efficacy, but also false negatives. This may prompt discontinuation on the grounds of potential harm, when in fact this is simply representative of one of a whole range of possible effects which could have been detected.

Ultimately psychological research needs to improve its accountability and transparency. By following different methodological principles, the reporting of findings from this thesis may have been entirely different. However the main aim of the research, as it should be in all cases of scientific research, was utilise rigorous methods to generate evidence which may help to answer complex questions. Although some tentative claims have been made based on the findings in this thesis, it is instead the methodological process which offers the most significant results. By making use of pre-registration for each study, and following through on planned analyses this thesis has been able to demonstrate the value in open science and the profound impact it can have on the analysis and interpretation of results.

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Appendix 1 – Registered protocol for Systematic Review

Systematic Review Protocol

Review Questions

To identify and quantify the magnitude of the effect size of the mediation mechanisms underpinning the trauma-psychosis link, and assess the degree to with the mechanism reflects underlying causal relationships.

To assess the methodological quality of existing evidence and evaluate the implications for future research.

Searches

Electronic databases (PsycINFO, EMBASE, MEDLINE and PILOTS) and the grey literature will be searched.

The reference lists of key papers (e.g. relevant reviews) will be hand searched for further eligible articles.

The databases will be searched using the keywords (psychosis OR schizo*) AND (childhood trauma OR early adversity) AND (mediat* OR structural equation model OR path analysis). The search strategy will be amended appropriately for each database.

Types of study to be included

All studies which have investigated mediation mechanisms between early trauma and the development of psychosis. Cross-sectional, cohort studies, case-control studies, prospective studies and trials will be included where other inclusion criteria are met.

Condition or domain being studied

Psychological mechanisms which mediate the relationship between childhood trauma and early adversity, and the positive symptoms of non-affective psychosis.

Participants/ population

Both clinical and sub-clinical participant populations will be included if they have experience of positive psychotic symptoms (i.e. voice hearing, hallucinations, delusions, paranoia).

Intervention(s), exposure(s)

N/A

Comparator(s)/ control

(first aim….?)

Study findings will be assessed against Bradford Hill criteria for causality.

Context

Included studies will assess potential mediation mechanisms between the experience of early trauma and adversity, and the development of positive psychotic symptoms.

Outcomes

Primary outcomes:

The magnitude and significance of mediation mechanisms linking early trauma to the positive symptoms of psychosis and the degree to which these meet Bradford Hill criteria for causality.

It is anticipated that mechanisms will be classified into meaningful groups: emotion regulation, mood, attachment, cognitive, social defeat and intrusions. These groups will be assessed for impact and significance, and evidence quality will be examined.

Data extraction, (selection and coding)

Methodological data such as the type of study, quality-related parameters, sample size and nature, and relevant demographic information will be recorded.

The mediator(s) being measured will be listed, along with relevant statistical information, for example the magnitude of the indirect and direct effect, the amount of variance explained by the mediator, and the correlation between the change in mediator and the change in experience of positive symptoms of psychosis. Other parameters will be examined where available. Any potential confounds will be noted and judgements regarding method and rigour will be generated.

Risk of bias (quality) assessment

Scores will be assigned using an adapted version of the Agency for Healthcare Research and Quality (AHRQ) tool. If possible two researchers will grade each study independently and scores will be recorded and compared. Interrater reliability will be monitored and any significant discrepancies will be arbitrated by the third researcher.

The overall quality of the final outcome will be assessed using an adapted version of the Grading of Recommendations Assessment, Development and Evaluation (GRADE) approach.

Strategy for data synthesis

The mediation statistics will be extracted from individual studies, and results will be evaluated in terms of the anticipated groupings: emotion regulation, mood, attachment, cognitive, social defeat and intrusions. Judgements about theoretical significance and causal status will take into account the quality and rigour of the studies in each group.

Analysis of subgroups or subsets

N/A

Dissemination plans

The completed review will be submitted for publication in a peer-reviewed journal.

Appendix 2 – Registered protocol for online study

Background

There is strong evidence from clinical studies that patients with psychosis have a higher incidence of early trauma than is regularly found in the general population (Ashcroft et al., 2012, Varese et al., 2012) and there is good evidence to support a dose-response relationship (Larkin & Read, 2008; Longden et al., 2016, Hardy et al., 2016) suggesting that ongoing trauma, or the experience of multiple traumatic events further increases the chances of developing psychosis.

Adversity in childhood may lead to cognitive vulnerabilities which may then lead to reasoning biases, inciting paranoid thoughts and contributing to their conviction and maintenance (Garety, 2001 & 2005). Negative thoughts about the self and about others, feeding into an external attribution bias (where negative events are blamed on others), along with low self-esteem or self-worth may contribute to the development of paranoia and persecutory delusions (Melo & Bentall, 2013; Fornells-Ambrojo & Garety, 2009). Metacognitive beliefs about the utility of paranoid thinking may also be important to the maintenance of such beliefs (Morrison et al., 2005; Murphy et al., 2017).

Others suggest that a sense of overwhelm, or an enhanced sensitivity to stress may provoke exaggerated responses to events and information (Pavic, 2003; Ford & Courtois, 2014, Lincoln et al., 2015). This may be augmented by an inability to down-regulate stress as a result of faulty emotion regulation abilities. Patients with psychosis have been found to experience higher levels of negative emotion and lower levels of happiness when compared with healthy controls, and to use fewer functional and more dysfunctional emotion regulation strategies in dealing with these (Livingstone et al., 2009).

Clinical samples have also been found to experience reduced awareness of, and ability to tolerate emotions (Lincoln et al., 2015). Alexithymia, a difficulty with identifying and describing emotions, is prominent in patients with psychosis who as a result may then face challenges in applying cognitive emotion regulation strategies (van der Meer et al., 2009; O'Driscoll et al., 2014).

It has been suggested that there may be a developmental or attachment basis for this deficit, where those with psychosis do not learn how to adequately identify or describe emotions due to early trauma or being raised in under-stimulating environments (O'Driscoll et al., 2014). Insecure attachment has been linked not only to early trauma and psychosis, but also to greater interpersonal difficulties, more frequent use of avoidant coping strategies, more severe positive and negative symptoms, and higher incidence of affective symptoms (Gumley et al., 2013). These affective symptoms, depression in particular, have been found to have links with low self-worth and negative interpersonal self-concepts (Lincoln et al., 2010) which impact attachment relationships and negatively impact socioemotional functioning (Seghers et al., 2015). Depression has also been implicated in the maintenance of persecutory ideation (Vorontsova et al., 2013).

Although a substantial body of research exists there is no complete model which considers all of these potentially relevant factors and how they may combine to in the transition from early abuse or neglect, to the development of positive psychotic symptoms. This study proposes to examine a number of potential mediators in a sub-clinical population to conceptualize a model of trauma and psychosis with relevant mediating relationships.

The study began with an exploratory analysis of data from a general population sample (N=190). Data were subject to network analysis to identify salient connections, followed by structural equation modelling to generate a model hypothesis which could be tested in a subsequent data sample. The strongest mediation relationships were from psychological abuse to persecutory ideation through avoidant attachment and alexithymia (difficulty

identifying and describing feelings). Depression also acted as an exogenous predictor of persecutory ideation. These pathways will be tested in the subsequent sample in a confirmatory analysis stage to test whether the model is replicated and retains goodness of fit.

Research Question

To identify mediating mechanisms in the relationship between childhood trauma and subclinical experiences of positive psychotic symptoms.

Primary and Secondary Aims

Primary:

To test the hypothesized model (Fig. 1) generated from exploratory analysis of preliminary data for replicability and goodness of fit.

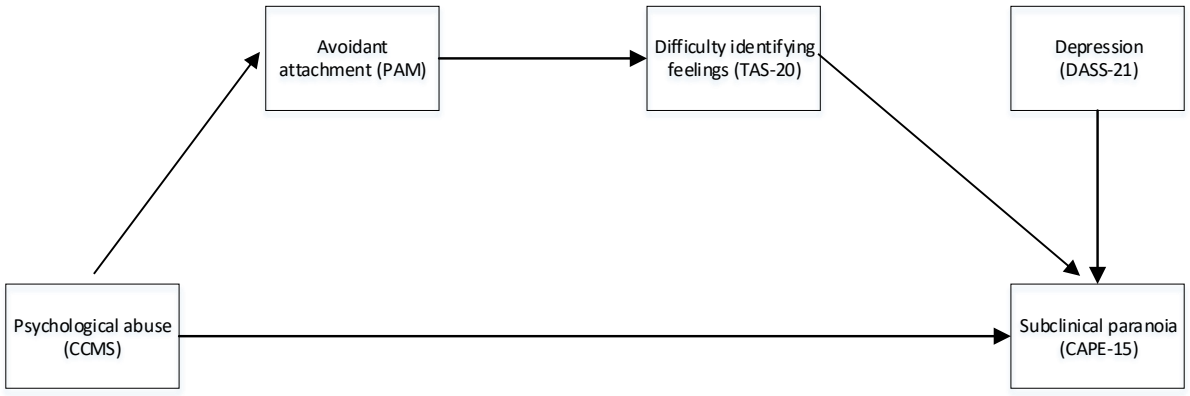


Fig. 1 – hypothesized mediation model. Covaried for age & gender.

Secondary Aim:

To develop and test an extended model based on the model above, but incorporating distress (measured by DASS-21) as the ultimate outcome variable, and using the subclinical paranoia symptoms (measured using CAPE-15) as the interim independent variable with metacognitive beliefs about paranoia (measured using BAPS) as potential mediators.

To perform further network modelling and generate alternative hypotheses based on a collapsed sample, if the original hypothesised model does not hold in the second data sample. Hypotheses from this model will be tested in future research.

Outcome Definitions/Data Points Collected

Variables will be measured using the questionnaires in Table 1. The trauma variables are treated as the independent variables and the symptom measures as the dependent variables in the modelling analyses. Each questionnaire subscale is treated as a separate variable.

Table 1 – Variables collected in online survey

Variable	Measure	Subscales
Trauma (independent variable)	CCMS-A	Psychological abuse* Neglect
	CATS	Sexual abuse Negative home environment Punishment
Symptoms (dependent variable)	GPS	Distrust Persecutory ideas Self-deprecation
	CAPE-15	Persecutory ideation* Perceptual abnormality Bizarre experiences
	BHS	Hypervigilance
Potential mediators		
Emotion regulation	CERQ	Positive reappraisal Suppression Refocus on planning Rumination Self-blame Acceptance Positive refocus Other blame Putting into perspective
Mood	DASS-21	Depression* Anxiety Stress
Attachment	PAM	Anxious Avoidant*
Metacognition	BAPS	Survival strategy Negative beliefs Normalising beliefs
Alexithymia	TAS-20	Difficulty identifying feelings* Difficulty describing feelings Externally oriented thinking

CCMS-A Comprehensive Childhood Maltreatment Scale for Adults; CATS Child Abuse and Trauma Scale; GPS General Paranoia Scale; CAPE-15 Community Assessment of Psychic Experiences; BHS Brief Hypervigilance Scale; CERQ Cognitive Emotion Regulation Scale; DASS-21 Depression, Anxiety & Stress Scale; PAM Psychosis Attachment Measure; BAPS Beliefs About Paranoia Scale; TAS-20 Toronto Alexithymia Scale.

*Asterisked items are the variables used in the hypothesized model (Fig. 1)

Study Design

Online survey, cross sectional.

Target Population

Participants will be a UK general population sample, recruited online.

Inclusion/Exclusion Criteria

Inclusion:

- Adults ages 18 and over
- Able to read and communicate in English
- Resident in the UK (the survey will be accessible to those outside of the UK online but the support services detailed in the debrief sheet will be UK only, and the research team cannot be certain that comparable services will be available in other locations)

Exclusion:

- Developmental or learning disabilities, organic brain impairments such as dementia or ABI, or a history of severe psychiatric illness.
- Responses contributing less than 50% data to any single questionnaire will be excluded, as will multiple submissions from the same IP address and responses entering identical values throughout the survey.

Sampling Method/Recruitment Process

All study recruitment and promotion will take place online. The survey will be promoted using social media adverts and emails, and after completion participants will be encouraged to share the link to encourage wider participation.

The survey will be accessible through the study website (links to this will be provided in all promotional adverts).

Study Retention/Withdrawal

Participants can skip any individual questions/surveys they do not wish to answer, and can withdraw from participation at any point. It is made clear in the consent form that it will not be possible to withdraw any data which has already been submitted.

Study Procedures

The study comprises 10 standardised questionnaire measures collated into a large online survey. These provide 32 subscale variables for analysis, from which the model hypotheses were generated. All responses are anonymous (aside from IP address, which will be retained to check for duplicate submissions) to encourage honesty in responding. Questionnaires are completed in the same order for all participants, and all questions/questionnaires are set up to allow non-response without termination.

The study is being undertaken in two parts, both of which required a general population sample of 200 self-referring volunteers. Data from the first 200 participants was used to perform exploratory analyses and generate a mediation model hypothesis.

Data from the second participant sample will be used to test this specified model for replicability and goodness of fit.

All data will be collated on the NOVI Survey platform and will be transferred to Microsoft Excel for cleaning before descriptive and normality testing in SPSS. Data will then be transferred to JASP for network analysis and lavaan in R for structural equation modelling.

Sample Size

The first sample target was N=200. 223 responses were received, 190 of which were viable for inclusion in the analysis (85%). In order to achieve the required sample of 200 viable responses, assuming a similar attrition rate, a target sample size of N=240 participants will be required.

Data Analysis

Data from the first sample has been subject to network analysis in JASP using the EBICglasso estimator using the npn correction for non-normal continuous data. Maximum sample size was used for all iterations of the model. Several exploratory versions of the model were generated with the final, most parsimonious version indicating strong connections between psychological abuse, attachment, alexithymia, and persecutory delusions.

The lavaan package in R was used to build SEM models based on the network analyses. Mediation models were constructed using the robust ML indicator function. Various models using multiple indicators and outcomes, with parallel and sequential mediators were tested. Bootstrapped testing of indirect effects indicated the above model (Fig. 1) to be the best fit and most parsimonious. Age, gender and ethnicity were entered as covariates. Gender and ethnicity showed no effect but a very small significant effect was detected between age and difficulty describing feelings (TAS-20) whereby younger participants experienced higher levels of difficulty.

In the second data set, the above network analysis will be repeated to examine whether the data follows a similar structure. Regardless of outcome, the SEM model will be tested in lavaan.

The independent variable will be psychological abuse (measured using the CCMS-A subscale). Sequential mediators will be added, first avoidant attachment (measured using the PAM), followed by difficulty describing feelings (measured using TAS-20). The dependent variable will be subclinical paranoia (measured using the persecutory ideation subscale of the CAPE-15) with depression (measured using the DASS-21) as an exogenous predictor.

The indirect path through the sequential mediators will be tested for significance, as will depression as an exogenous predictor of subclinical paranoia. Age will again be entered as a covariate to examine whether the borderline effect on difficulty describing feelings is replicated. Gender and ethnicity will also be tested as potential confounders.

The other variables collected in the survey, listed in Table1, will be used for the secondary analyses – both the extension of the tested model, and in the exploratory analyses (see Secondary Aims above).

Use of Study Results

Study results will form part of a doctoral thesis and will be submitted for publication in a peer reviewed journal.

Appendix 3 - Registered protocol for clinical study (version 4 – updated with ethics substantial amendments)

A randomised controlled experimental investigation of an emotion regulation skills training on paranoia and related distress in a clinical psychosis population: Research study protocol

Introduction

In recent years there has been increased recognition that psychosis is likely to develop from a number of interacting factors (Freeman & Fowler, 2009; Garety et al, 2001; Garety et al, 2007). One psychological factor that has recently gained prominence within the research literature is the influence of childhood trauma (Freeman & Fowler, 2009). Childhood trauma refers to a collection of difficult events including physical, sexual and emotional abuse, and neglect. Studies have found good evidence for a causal link between trauma and later psychosis (Varese et al., 2012) with severity and frequency of trauma being linked to the severity of psychotic symptoms (Schenkel et a, 2005).

Studies have found a link between high levels of emotional abuse and neglect and persecutory delusions (Ashcroft, Kingdon & Chadwick, 2012; Ucok & Bikmaz, 2007). Evidence shows emotional abuse to be the most commonly reported type of trauma in both clinical and subclinical populations (Ucok & Bikmaz, 2007; Bentall et al., 2014; Hardy et al., 2016;).

Maladaptive emotion regulation (or emotion dysregulation) has been linked with both clinical and sub-clinical positive symptoms (Westermann & Lincoln, 2011). There are a number of theories about why emotion dysregulation leads to paranoid symptoms in particular. Studies suggest emotional changes develop in the context of adverse life experiences and anomalous conscious experiences (Garety, 2001 & 2005). Adversity in childhood may lead to a cognitive vulnerability which in turn may lead to reasoning biases, inciting paranoid thoughts and contributing to their conviction and maintenance. Others suggest that a sense of feeling overwhelmed, particularly in social situations, coupled with the inability to down-regulate emotional hyperarousal may provoke exaggerated responses to information and events (Pavic, 2003; Ford & Courtois, 2014, Lincoln et al., 2015). It has also been suggested that delusions themselves may exist as dysfunctional regulation strategy, in an attempt to deal with overwhelming threat-related information (Lincoln et al., 2015).

It appears to follow that if emotional needs are not met in childhood, due to either abuse or neglect, that the developmental attachment processes are disrupted, prompting failed development of emotion regulatory processes and the inability to down-regulate threat affect. This, coupled with the hypothesised cognitive biases experienced by psychosis patients may combine to incite and maintain paranoid symptomatology. As yet there is no model which draws together all of these proposed factors. Therefore, this experimental research study hopes to establish whether a causal pathway exists between emotion regulation and psychosis, and assess whether enhancing an individual's emotion regulation capacity can have a positive impact upon the experience of paranoia related distress which in turn should improve overall quality of life.

Primary Research Questions:

To test whether there is a positive association between emotion dysregulation and paranoia related distress dynamically over time.

To test whether improving emotion regulation skills can reduce experience of paranoia related distress.

To test whether self-report of early emotional abuse and neglect predicts levels of emotion dysregulation during the first sampling period.

Secondary Research Questions:

To test whether emotion regulation mediates the trauma-psychosis association at baseline and over time.

To investigate how the results from momentary assessment methods compare to those from standardised questionnaire measures.

Methodology

Study summary:

This study aims to investigate potential causal mechanisms underpinning the link between experiences of childhood adversity and the later development of the positive symptoms of psychosis. It will do so by trialling an emotion regulation skills training intervention against an active control in a randomised clinical sample. The sample will be asked to provide information about childhood adversity, paranoid psychotic symptoms and related distress.

The study will be open to any patient with a diagnosis of non-affective psychosis of non-organic and non-substance induced origin. Both inpatients and outpatients will be eligible as long as they are in regular contact with a clinical care team. Participants will be involved in the study for a period of 4-16 weeks in total.

The experience sampling method will be used to collect data about paranoia-related distress. This involves short questionnaires delivered via a mobile phone app at ten intervals per day for six consecutive days. This provides a more accurate measure of the fluctuation in symptoms and distress over time, and is ecologically valid. It has been used widely in psychosis and other severe mental health conditions in recent years (Lardonois et al., 2007; Myin-Germeys et al., 2009; Hartley et al., 2014; So et al., 2015; Reininghaus et al., 2016) and has been shown to be safe and easy to follow, provided participants are thoroughly briefed.

The intervention task is a series of emotion regulation skills workshops drawn from mindfulness and dialectical behaviour therapy. It has been piloted in NHS Lothian in both inpatients and outpatients and was met with a positive response from attendees, both in terms of acceptability and effectiveness (Lennon, 2015). Workshops will run for 60—75 minutes for 4-6 sessions and will be delivered by trained NHS care professionals.

The active control task will be an arts and crafts skills-based workshop. The workshop will matched for contact time, duration and attentional demands, as far as possible. The control task will make no reference to emotions or emotion-management based content.

Both the intervention and control task will take place in the same activity centre.

It is hoped that by isolating emotion regulation as a relevant mechanism, targeting and improving it will enhance the existing understanding of relevant causal mechanisms and whether they are amenable to change. This will inform future research and reveal potential target mechanisms for treatment development. This should facilitate the reduction of patient distress and improve overall quality of life.

Recruitment & Consent

Participant selection

A clinical sample will be recruited through NHS contacts and associated community mental health teams (CMHTs). Participants will currently be experiencing symptoms of psychosis and will be engaged with these treatment and support teams.

The primary recruitment pathway will be via clinical referral. Psychiatrist, psychologist and CMHT colleagues may identify and approach potential participants about the study if they satisfy the inclusion criteria. Clinical staff will be provided with information about the study and a list of the particular inclusion and exclusion criteria. The clinician will discuss the study with the potential participant, answer any questions, give the potential participant an information sheet and, if the participant remains interested in the study, seek their verbal consent to pass on their details to the research team. The researcher shall contact clinicians to gather contact details for potential participants.

A secondary route for recruitment will be through self-referral. Study posters and information will be displayed at various NHS sites; hospital premises - inpatient wards and outpatient departments, and other relevant NHS sites including GP surgeries. Participants will contact the research team via telephone or email. The researcher will obtain verbal consent to contact their keyworker/ care-coordinator to ascertain whether they meet inclusion criteria for the study. The researcher and the clinical team will collaboratively complete a risk assessment.

After speaking with their clinical team, a brief screen will be administered via telephone to ensure potential participants have a minimum level of emotion dysregulation and paranoid thinking. Should participants meet all inclusion criteria for the study and continue to express an interest in taking part, the researcher shall arrange an appointment to meet with them. All potential participants will have a minimum of 48 hours to consider the information sheet prior to being contacted by the researcher.

Potential participants shall be made aware that participation is entirely voluntary and that deciding not to take part in the study will have no impact upon the clinical care they receive. In the event the potential participant still wishes to participate in the study, they will be asked to give their informed written consent by completing a Consent Form. Once this consent form is complete the study shall commence.

Inclusion Criteria

1. Adults aged 18-65 years with a diagnosis of schizophrenia, schizoaffective disorder, schizophreniform disorder, delusional disorder or non-affective psychosis.

2. Individuals will be in current contact with NHS Mental Health outpatient services.

3. Individuals will be able to read, understand and communicate in English.

4. Individuals will have a minimum level of emotion dysregulation and paranoid symptoms as measured by the brief screening tool developed for this study.

5. Individuals will have the capacity to consent to take part in research.

Exclusion criteria

1. Individuals who do not meet the minimum required score on the screening tool.

2. Individuals with developmental or learning disabilities, such as Autism Spectrum Disorder.

3. Individuals with psychosis caused by an organic brain impairment such as dementia or acquired brain injury.

4. Individuals not currently in contact with NHS Mental Health Services or without a keyworker/ care co-ordinator.

5. Individuals who lack the capacity to consent to participate in research.

6. Individuals with a primary diagnosis of substance-induced psychosis.

7. Individuals who have a level of English ability that prevents completion of questionnaires and/or interviews.

8. Individuals who are not permitted access to a smartphone throughout the duration of the day.

Informed Consent & Capacity

Potential participants will be asked to give their written informed consent by completing a Consent Form. Once this consent form is complete the study shall commence. If at any time during the study a participant is deemed to lose the capacity to consent to research, data collection will cease, but access to the intervention will not be withdrawn if the participant feels it is of benefit to them.

Participants who re-gain the capacity to consent before the end of the study will be re-consented and data collection will recommence. Participants who do not recover capacity to consent by the end of the study will have their identifiable data withdrawn.

Procedure – baseline measures

After providing informed consent, participants will complete a number of baseline measures which will characterise the sample and provide information about each participants' experiences. A list of the measures is included in the *assessment and data collection* section below.

Procedure – experience sampling (ESM)

After completion of baseline measures, participants will be introduced to the experience sampling app (PsyMate) which will be explained fully, and demonstrated, by the researcher who will also be able to answer any questions they may have. They will be shown how to silence the alarms and informed that surveys will remain 'live' for ten minutes after the alarm sounds to give them time to complete each one. They will be provided with contact details in case they have further questions or encounter any technical issues after leaving the research centre. Researchers will follow up with the participant within the first two days of ESM data collection to ensure participants are managing to fill in the surveys, and to address any technical difficulties.

The ESM has been chosen as it enables data to be collected from participants in their own environment, using their mobile phone, increasing ecological validity. Capturing 'in the moment' data several times a day also increases accuracy and measures fluctuations in symptoms over the sampling period. Participants who do not own a smartphone will be provided with an internet-enabled handset which will enable them to complete the measures.

The app works by sounding an alert at ten random time points during the day to prompt the participant to complete a short survey. The survey questions are derived from paranoia and distress related standardised measures. Answers are transmitted via the internet but can be stored locally on the participant's handset until a Wi-Fi connection is established.

Participants will complete an initial six day period of experience sampling which will establish a baseline for each participant. They will be required to complete a minimum of 50% of these surveys in order to be included in the analysis. Each survey will take 1-2 minutes to complete and will ask for 'in the moment' information about the participant's current thoughts and feelings, or thoughts and feeling in the interim since the previous alarm. Answers are provided on a Likert style rating scale. By taking multiple measures from each participant (60 measures per week, 120 in total across the duration of the study) the subtle fluctuations in symptomatic experience should become clear. This should also reduce the overall error within each group and provide a more accurate estimate of change post-intervention. Additionally, the method

reduces the number of participants required for a viable analysis. Given the time scale and relative rarity of the complex condition, the use of this method makes it more likely that recruitment targets will be met and adequate data gathered.

Following the initial six-day experience sampling period participants will be randomised using a computer program (www.randomizer.org) and notified of when their workshops will take place. Randomisation will be entirely electronic and will not rely on a pre-determined sequence. Each participant will be entered into the program only after then have completed the experience sampling. The research supervisor (PH) will do the electronic randomisation and will contact the participants in order to ensure the researcher (AW) remains blind to treatment allocation. *Allocation of participants will no longer be blind. This was not feasible in practice due to limited staff members to facilitate the escorting of participants to the therapy & control groups. The randomisation sequence was generated during the pilot of the study, and the researcher (AW) will escort participants to the groups.*

The method circumvents a number of the issues of using single time-point questionnaires and interviews, predominantly memory biases (Kihlstrom et al., 2000; Holmes et al., 2016; Barry et al., 2004). These affect the quality and reliability of the data which can be gathered. The method has been used widely in psychosis and other severe mental health conditions in recent years and has not shown any adverse reactivity (Lardonois et al., 2007; Myin-Germeys et al., 2009; Hartley et al., 2014; So et al., 2015; Reininghaus et al., 2016). It has been shown to be safe, acceptable and easy to follow.

Procedure – intervention/control

Both the intervention and the control task will be delivered as a series of 4-6 workshops of 60-75 minute duration. In the interests of equipoise these will be matched for contact time, participatory elements and homework as far as possible. Both will be skills based and will involve an element of learning, but the control task will bear no reference to emotion regulation abilities. Participants will be required to attend at least one session. Attendance will be recorded and entered as a covariate in the analysis to assess whether any changes (or lack thereof) can be connected with the duration of their attendance.

The emotion regulation skills training (Lennon, 2015) has been derived from dialectical behaviour therapy and aspects of mindfulness. It aims to teach participants to recognise and label emotions, and equip them with relaxation and awareness skills to encourage more proactive and masterful regulation strategies. It has been successfully piloted in small inpatient and outpatient samples in Scotland. Both groups showed good adherence and participants found the training useful and acceptable.

The control task will comprise of a series of arts and crafts workshops. These workshops will match the duration and design of the intervention tasks as far as possible. There will be elements of skills-based learning and group interaction which will mirror the activities set out in the intervention workshops, but there will be no emotion-based content. Group members will have access to activities such as clay modelling, drawing and painting and 'room styling' which allows them to make small objects such as photo frames, mirrors and cushions for their rooms/homes. This group was selected as an adequate match as it already runs weekly at the Hive and is facilitated by qualified and trained staff members. It teaches participants skills and should be enjoyable and useful to those who attend.

Procedure – after the intervention

After the intervention, participants will complete a further six day experience sampling period, designed to assess change in their paranoia and related distress. After this, participants will complete a number of the questionnaires from the first meeting in order to assess change. This is intended to capture whether any change in paranoia and related distress is due to improvement in emotion regulation ability. The trauma measures will not be repeated as these are based on past events and will not be influenced by the intervention.

This will mark the completion of their participation in the study. Those participants who have been part of the control group will be offered the opportunity to take part in the emotion regulation skills workshops but no further data will be collected from them.

Procedure – assessments and data collection

Brief Screening Tool – Administered via telephone prior to participants attending any meetings with researcher. This will be used to confirm that participants meet inclusion criteria for the study. The measure will determine whether participants have minimum levels of both paranoia and emotion dysregulation. The screening tool was developed using selected items from the General Paranoia Scale for Adults (GPS) Carvalho et al., 2015 and the Emotion Regulation Questionnaire (ERQ) Gross & John, 2003. If participants answer 'yes' to one or more items from each scale, they are considered to be eligible to participate in the study.

Participant Demographics Sheet – short question sheet facilitating the characterisation of the sample. This information will also provide data which will be used in controlling for potential confounding variables in the study analysis. This will be administered after completion of the informed consent form at the start of the study.

Measures – baseline and change

These measures will be completed at the beginning of the study, to characterise the sample, and again at the end of the study to assess change.

Cognitive Emotion Regulation Questionnaire (CERQ) Garnefski Kraaij & Spinhoven (2001), This will measure participants' use of both positive/adaptive and dysfunctional emotion regulation strategies. 36 items, 10 minutes to complete.

Positive and Negative Syndrome Scale (PANSS) (Kay, Opler & Fiszbien, 1987). 45 minute interview conducted by the researcher, assessing three scales – positive symptoms, negative symptoms and general psychopathology.

Green Paranoid Thoughts Scale (GPTS), Green et al., 2008. Two scales measuring persecutory ideation and ideas of social reference. Incorporates measurements of conviction, preoccupation and distress. 10 minutes to complete. This will be used to assess participants' paranoid thoughts, along with their associated conviction, preoccupation and distress.

Measures – confounders and moderators

These measures will be completed at the beginning to characterise the sample and again at the end to assess change. In the event that distress is found to be reduced, but emotion regulation remains unchanged, these measures will help to detect any other mechanisms which may be working to incite the change in symptoms.

Brief Hypervigilance Scale (Bernstein et al., 2015). Measures proneness to hypervigilance. This very brief questionnaire measures paranoid/anxious thinking, sensory sensitivity and threat-based behaviours. Hypervigilance has been associated with previous experience of trauma. 5 items, 2 minutes to complete.

Depression, Anxiety & Stress Scale (short form) (DASS-21) (original Lovibond & Lovibond, 1995). Measures participants' experience of depressive, anxious and stress related symptoms over the past week. Takes 5-10 minutes to complete.

Beliefs about Paranoia Scale (short form) (BaPS) Short form: Gumley et al (2011). This will be used to understand how the participants view their experience of paranoid symptoms, and whether this is adaptive or protective in any way. Measures metacognitive beliefs about paranoia across 3 factors: negative beliefs about paranoia, paranoia as a survival strategy & normalising beliefs about paranoia. Takes 5-10 minutes to complete.

Psychosis Attachment Measure (PAM) Berry et al (2006). Measures attachment style across two sub-scales – anxiety & avoidance. Takes under 10 minutes to complete. This will be used to measure participants' subjective experience of their attachment relationships which may be pertinent to past experience of emotional abuse/neglect. As a trait measure this will only be administered as part of the initial questionnaire battery.

Measures – trauma

These measures will only be administered at the beginning of the study as they are based on concrete past experiences which will remain unaffected by the intervention in this study. Two measures have been selected here to ensure all trauma types are covered (sexual, physical, emotional/psychological and neglect). The sexual abuse subscale from CCMS-A will be removed to avoid duplication. The trauma measures will be used to characterise the sample. Combined, these measures will take 10 minutes to complete.

Child Abuse and Trauma Scale (CATS) (Sanders & Becker-Lausen, 1995) Measures subjective perception of the degree of stress or trauma present in childhood. Sums scores from three subscales: Sexual Abuse, Punishment, Neglect and Negative Home Environment. 38 items rated on a 0-4 response scale.

Comprehensive Child Maltreatment Scales for Adults (CCMS-A) (Higgins & McCabe, 2001) Measures perception of exposure to childhood abusive, neglectful behaviours, witnessing violence before the age of 13. Contains five subscales: Sexual abuse, physical abuse, psychological maltreatment, neglect, violence. The separate response scale for sexual abuse (rated 0-5; never – more than 20 times) will be removed. Other scales scored 0-4 (never to very frequently).

Measures – experience sampling

Experience Sampling Survey (PsyMate*)* - this is a brief measure which will take 2-3 minutes to complete. The measure has been developed using a combination of items taken from previous similar research, and standardised measures. Information will be firstly be collected about immediate context and feelings, to capture data about any transient states. Questions will then ask about the interim since the previous alarm – any paranoid/delusional thinking, along with associated distress and coping strategies. The measure will be administered ten times per day for six days and will supply data on real time fluctuations in participants' rich symptoms and coping.

Measures – adverse events

The patient-rated 26-item Adverse Events Questionnaire (Hutton, Byrne, Pyle, & Morrison, 2015; Pyle et al., 2016) will be administered to all participants at the end of their study involvement. There are two different versions; one for those who complete the study and another for those who choose to withdraw from the study before the end.

Additionally, there are 3 researcher rated items, and two participant-rated items from the Clinical Global Impression scale and the Calgary Depression Scale. Scores on these items will be used to assess how useful the groups have been, and will serve to highlight any potential serious adverse events which will be reported first to the DMEC and then, if required, to the NHS REC (see below).

Serious adverse events criteria

Using an approach applied previously (Murphy et al., 2019; Pyle et al., 2016), serious adverse events were defined as: (i) death by suicide; (ii) suicide attempt; (iii) suicidal crisis without attempt (rating of 2 on item 8 of the Calgary Depression Rating Scale for Schizophrenia; CDSS) (Addington, Addington, & Maticka-Tyndale, 1993); (iv) severe symptom exacerbation

(rating of ≥6 on the patient or researcher-rated CGI and CGI-I). Non-severe adverse events were defined as a score of ≥3 (agree 'quite a lot' or 'a lot') on any relevant item (e.g., subjectively worsening mental state, heightened stigma, increased medication use, increased conflict) on the patient-rated 26-item Adverse Events Questionnaire (Hutton, Byrne, Pyle, & Morrison, 2015; Pyle et al., 2016). Severe adverse events will be reported to the DMEC for consideration and, if required, will be submitted to the NHS REC for review within 14 days.

Sample Size

Sample size and power calculations for use in the experience sampling method are highly complex due to the 'nested' nature of the data. The method provides multiple measures from each participant. Standard deviation margins will therefore be minimised, leading to a more accurate representation of the true effect. If a large correlation is assumed (r = 0.5), a provisional G*power calculation to detect a large effect size (Cohen's d = 1) with 80% power would require a sample size of 32 participants – 16 per group. This is supported by Snijders & Bosker (2012) who suggest an n > 30 is adequate for non-biased significance tests of fixed effects. We will aim to recruit a sample size of 34 which will take into account that ~10% of participants may drop-out.

Analysis

Primary outcome measure

The primary outcome measure for this study is mean score on paranoia and distress items in the Experience Sampling questionnaire measure, over the post-therapy sampling week.

Secondary outcome measures

Paranoia and associated distress will be measured using the PANSS interview and the GPTS. Results from these will allow analysis of change within participants. Scores will also be compared with the scores on the experience sampling questionnaire to determine the extent of similarity or disparity between the results. This will be useful methodological information - directly comparing single time-point measures with momentary data.

Changes in emotion regulation ability will be measured using the CERQ at baseline and post-intervention. These scores will confirm whether emotion regulation ability is a mechanism which has been affected by the intervention.

Changes in other scores between baseline and post-intervention will be measured to characterise the sample and will be checked as close analogues or other mechanisms relevant to emotion regulation. If there are changes in paranoia and distress levels, but no change in emotion regulation scores, these other mechanisms may be involved in the change in symptoms and distress.

These other mechanisms are:

Hypervigilance as measured by the BHS

Attachment as measured by the PAM

Beliefs about paranoia as measured by the BAPS

Mood as measured by the DASS-21

Statistical Analysis

Analysis of ESM data is challenging because of its 'nested' nature. Multiple observations from the same participant cannot be considered entirely independent from each other. This necessitates the use of multilevel modelling.

Participants' responses to the ESM surveys will be compared pre- and post-intervention, and any changes in overall paranoia severity (frequency & distress) will be compared against their own baseline scores. This will generate within-participant data, which can be summed into a group mean. Standardised mean differences will then be compared between groups for both pre- and post-intervention scores to interpret the effect of the intervention versus control task.

Following this, the method outlined by Renninghaus et al. (2016) will be employed to determine whether associations between emotion regulation and paranoid symptoms were modified by exposure to early trauma. Two way interactions will test (emotion regulation x trauma) by group, for the intervention and control participants. Potential confounding factors will be controlled for, as detailed in the demographics sheet (i.e. gender, age, employment status, years in education, ethnicity). This analysis will require the use of Stata. The further information gathered in the questionnaire battery will be available for sub-group correlation analyses.

Interaction effects between baseline trauma scores (as measured by CCMS-A and CATS) and emotion regulation scores (pre and post intervention) will be analysed to investigate whether participants with higher levels of childhood trauma are more prone to higher levels of emotion dysregulation, and whether this is more amenable to change using the intervention.

Efforts will be made to keep participants engaged throughout the study so as to avoid large amounts of missing data. Phone calls are scheduled as part of the experience sampling protocol. Participants who show consistent trends towards non-respond or non-attendance will be contacted to check whether they still wish to participants. This will alert the research team to any potential issues with the technology or the running of the groups.

Analysis will be performed on an intention to treat basis with multiple imputation for missing data values. However, due to the large volume of data collected using the ESM method, adequate data should be received from most participants without requiring missing data manipulation. There is a recruitment buffer to safeguard against potential attrition in the sample size which should preserve statistical power in the event of drop-outs.

Knowledge Exchange

This study will be written up for submission in doctoral thesis format for submission to Edinburgh Napier University. The results will be written up for submission to a peer reviewed journal. After completion, the results of this study will be made available to any participants who wish to receive them.

Appendix 4 - Mediation Research

Early methods of mediation testing (Baron & Kenny, 1989)

Earlier methods for establishing whether mediation effects were present in a model, focused on sequential steps which needed to be satisfied in order to progress to the next stage of the analysis (Baron & Kenny, 1989; James & Brett, 1984) Firstly there is required to be a relationship between X and Y, to confirm there is an effect which could be mediated. Following that, the model must prove there is a link between X and M to show the predictor is linked with the mediator. Third, there must be a connection between M and Y, shown by regressing X and M on Y, to show that M and Y are not mutually caused by X as a single predictor. The final step in the Baron and Kenny (1986) sequence is that the path between X and Y should be reduced to zero to demonstrate complete, or 'perfect' mediation. If the other steps are satisfied with the exception of the fourth, then partial mediation can be claimed.

This method has come under a great deal of criticism in more recent years, for a number of reasons. The method implies that the strength of mediation be judged by the absence or substantial reduction of a direct effect, as opposed to by the size of the indirect effect (ab) (Zhao et al., 2010). Other researchers have argued that there need not be a significant total effect present before mediators can be considered (MacKinnon et al, 2000; Shrout & Bolger, 2002) and that mediation should be judged on the presence or absence of a mediation effect regardless of the strength of the initial connections (Zhao et al., 2010). The stepped approach also fails to adequately quantify and test the indirect effect, it merely provides the a-path and b-path coefficients without calculating a point estimate for the overall indirect effect, or the accompanying standard error (Preacher & Hayes, 2008). It further fails to consider the potential impacts of suppression effects, particularly in multiple mediation models, where some M variables which do truly mediate the effect may be 'cancelled out' but other M variables acting in the opposite direction (i.e. with the opposite sign)(MacKinnon et al, 2000). In this case the indirect effect can appear vastly reduced or entirely negated, potentially leading to erroneous conclusions about the presence or absence of mediation effects, due to an oversight in model testing methodology.

Baron and Kenny (1986) advocate the use of the Sobel test (Sobel 1982), also referred to as the 'product of coefficients' or the 'delta method', as means of evaluating the significance of the indirect effect, however this is not explicit in their stepped approach and as a result has often been overlooked by researchers, resulting in claims of mediation which are not substantiated by any significance testing of the indirect effect. The Sobel test computes the ratio of ab to its standard error from which it derives a p-value based on a normal distribution. The finding of a significant p-value would indicate support for a significant effect of mediation (Preacher & Hayes, 2008).

The Sobel test has also been widely criticised for its lack of power, its conservatism and its assumption that ab follows a normal distribution when in reality it is almost always skewed (Kenny, 2015; MacKinnon, Warsi & Dwyer, 1995). The original Sobel test was designed with very large samples in mind and thus should only be utilised in sufficiently large samples or where effects are large and assumptions of normality are met (Preacher & Hayes, 2008; Shrout & Bolger, 2002). More contemporary mediation methods overcome these shortcomings by employing more robust methods of interrogating the indirect effect, and subsequently attempting to report it using meaningful metrics.

Bootstrap mediation testing (Preacher & Hayes, 2008)

The now well established Preacher & Hayes (2008) method involves bootstrapping – a non-parametric technique which involves resampling the dataset with replacements. This is done repeatedly, through thousands of iterations (Preacher & Hayes 2008 recommend a minimum

of 1000 bootstrap resamples) and for each simulated data set a sample distribution is generated. After running all requested iterations, the point estimates for the indirect effect(s) which are generated, are ranked from low to high. In order to derive confidence bounds, the top and bottom percentages of these are discarded. For an alpha = .05 significance level the top and bottom 2.5% will be discarded to form 95% confidence intervals around the point estimate. Thus the confidence interval and indirect effect point estimate are based on a largely inflated sample, but one which is still derived from the original data. This can be viewed as a proxy for the way in which most scientific research is undertaken – researchers endeavour to derive a population estimate based on data from a sub-sample of a given population. Repeating research in the sub sample a number of times should bring the result closer to a 'true' population estimate.

Preacher & Hayes (2008) explain that the point estimate will not always sit centrally in the confidence bounds as they are derived from multiple sample distributions which are likely to be skewed, however Efron & Tibshirani (1993) caution researchers against manually altering the confidence bounds as this can result in inclination towards Type I error and issues with power when artificially adjusted. Bootstrapping also facilitates significance testing of the indirect effect and the derivation of a p-value and/or standard errors without the reliance upon the assumption of normality.

Multiple mediator models

Added complexity arises in cases of multiple mediators. Statistical understanding and computational power have improved in recent years enabling the investigation of multiple mediator models. These models have the added benefit of simultaneously testing several potential mediators in one single model. Conceptually this makes good sense as it is unlikely that psychological relationships can be reduced to single strings of cause and effect (Preacher & Hayes, 2008), and by entering several potential mechanisms into a single model, they are tested in conjunction with each other, controlling for one another's effects. This enables the derivation of a total indirect effect, as well as specific indirect effects through each mediator in turn, controlling for the others and any additional covariates. This also reduces any risk of parameter based bias due to omitted influential variables (Preacher & Hayes, 2008). In contrast, testing multiple single mediator models would likely yield a different, and much less accurate picture of the overall effect(s). This is likely to enhance the impact of any omitted variables, and artificially inflate the relative magnitude of each mediator due to lack of controlling.

Multiple testing is not without its own risks. When entering multiple putative mediators into a model, the researcher must consider the potential for multicollinearity and the influence this may have on the results. Multicollinearity refers to predictors in a regression model which are correlated rather than independent. The effects of this would be manifest in the b-paths from M to Y diminishing them by the degree to which correlation occurs (Preacher & Hayes, 2008). If the correlation is substantial, the individual mediators may not demonstrate their full effect as their unique influence in terms of mediation is impacted. Whilst the point estimates should be unaffected by this, the standard errors will reflect the increased uncertainly in the model (Alin, 2010). Although multicollinearity cannot strictly be overcome without the addition of more data (Alin, 2010), Preacher & Hayes (2008) recommend that researchers select distinctive putative mediators which theoretically have a reduced chance of overlap. They encourage the two-stage testing of indirect effects; firstly test the mediator set to see if it has an effect on the X to Y relationship before secondly testing, if appropriate, predictions about individual mediators within the context of the multiple mediator model. Despite the complexities, they do maintain that research should consider multiple mediator models as a preference over single mediator models wherever possible.

Appendix 5 – Effect sizes in mediation research

The definition of effect size has also been debated throughout the literature. Preacher & Kelly (2011) describing effect size as the quantification of a phenomenon of interest in addressing a specified research question. This captures both sides of the argument; that the magnitude of the effect size relative to a specified null hypothesis is important; but also that the interpretation of the practical importance to the research in question must be considered.

Effect sizes should ideally fulfil a number of desirable qualities to ensure their usefulness. These include the use of an interpretable scale to enable to cross-study comparison of results, regardless of the design characteristics of each study. Standardised measures fulfil this criteria as they are resistant to any linear transformation of variables (Lachowicz et al., 2018; Fan & Konold, 2010). Confidence intervals should be available in order to gauge the accuracy of the effect point estimate. Effects will not always accurately reflect the population values due to sampling error, but confidence intervals will reveal the distribution of values and the extent of uncertainty (MacKinnon, 2008; Preacher & Kelley, 2011). The effect should also be 'consistent', meaning that the point estimate itself should be representative of the true population value, and thus should not change, but as the sample size increases in the research, the confidence bounds should converge demonstrating improved accuracy (Lachowicz, 2018; Preacher & Kelley, 2011). Ideally effect sizes should be 'unbiased' and should retain their parameter values, even over infinite sampling and in order to do so they should be 'efficient', meaning they should have low overall sampling variability (Preacher & Kelley, 2011).

Wen & Fan (2015) also discuss the importance of 'monotonicity', whereby the effect size, essentially an index or representation of the effect, should increase 'monotonically' with any increase in the indirect effect. When all else is held constant (i.e. the total effect), both the indirect effect and the effect size index should change in the same direction (MacKinnon, 2008; Preacher & Kelley, 2011). This is a property that has been tested and holds true in a number of traditional effect sizes including Cohen's *d* (Cohen, 1988); Hedge's *g* (Hedges, 1981), R^2, etc. Without this, the preservation of rank is lost and the effects become difficult to interpret and explain relative to their original metrics. When monotone effect sizes are used larger values will indicate stronger effects, making comparisons and interpretations easier.

Despite the range of effect sizes available to researchers, finding consensus on what constitutes an adequate effect size to represent the indirect effect has proved challenging. The derivation of the indirect effect itself is straightforward, particularly in models using continuous variables. The 'product of coefficients' approach involves multiplying the a-path and b-path coefficients together, or alternatively the 'difference in coefficients' approach involves subtracting the direct effect (c') from the total effect (c) (Rijnhart et al., 2019; Kenny 2015). Mediation proportions are equally simplistic to compute using either indirect/total or 1-(direct/total). These methods should yield the same results, however in cases where any of the variables are dichotomous, this does not hold and alternative methods must be applied (Rijnhart et al., 2019). In most cases logistic regression is used and effects are reported as odds ratios as opposed to regression coefficients (see **Odds ratios** below).

The reporting of effect sizes for indirect paths carries a level of complexity beyond the expression of more standard effects such as standardised mean difference or proportion of variance explained (Lachowicz et al., 2018; Preacher & Kelley, 2011).. The indirect effect is composed of two regression coefficients. Researchers must therefore consider how to represent their findings in a meaningful and interpretable way, which also facilitates comparison with other research. Throughout the literature, effect size reporting is inconsistent and researchers have selected a number of different indices to communicate their results, the most common of which are discussed in turn below.

Narrative description

In its most basic form, the effect size for mediation can be reported narratively to express the degree of mediation present in the model. Researchers coin different terms for the level of mediation present, including 'complete' mediation in cases where the direct effect is nullified when the mediator is added, essentially re-routing the effect of X on Y completely through M (James & Brett, 1984). Similarly, and most prominently, Baron & Kenny (1986) discuss 'perfect' mediation following their stepped approach to testing, where the direct path between X and Y is reduced to zero when controlling for the mediator and 'partial' mediation where the direct effect is reduced, but not to zero, therefore indicating the presence of a mediation effect, but one which is not sufficient to entirely negate the direct effect. There are a number of reasons why these descriptions are insufficient (see **Mediation research**, above), not least that descriptions of effects, particularly those using terms such as 'perfect' or 'complete' can be highly misleading.

Proportion and ratio

The indirect effect is often expressed as a percentage of the overall effect of X on Y, highlighting the proportion of the full effect which is mediated (Miocevic et al., 2018). Alternatively, some studies express this as a ratio which compares the indirect effect with either the total (Alwin & Hauser, 1975) or the direct (Sobel, 1982) effect. Ratios and proportions are simple to compute, but are minimally informative particularly in multiple mediation models where single mediators can return values of close to 1, giving the incorrect impression of 'complete' mediation and implying that other potential mediators need not be investigated .This is misleading for a number of reasons, as proportions are unbounded and thus a value of 1 is not a ceiling value; and there is the potential for additional mediators in the model to partition the effect (Preacher & Kelley, 2011)

Although the proportion mediated is the most frequently reported metric (Lachowicz et al., 2018; Miocevic et al., 2017), closely followed by the ratio, these effect sizes are not recommended for use as they tend to be unstable unless they are based on large sample sizes or large effects (Lachowicz et al., 2018; Preacher & Kelley 2011). MacKinnon et al, 1995 suggest for proportion effect size derivation samples should be a minimum of N=500, and for ratio N=5000. The fragility of the ratio estimates is clear when considered in context of the computational method. Even small changes in the denominator value (c') or the numerator (ab) can potentially have a profound effect on the overall effect estimate. As precision is already a challenge in small samples it is clear why much larger samples are required to improve the reliability of this metric. Despite these limitations, confidence intervals can be computed to indicate precision, and the decomposition of the total, direct and indirect proportions can be of value in cross-study comparisons (Preacher & Kelley, 2011)

Unstandardised and partially standardised indirect effects

Unstandardised effects are reported in their raw form, and are interpretable in relation to the scales used in the original study. The unstandardised indirect effect simply illustrates the decrease in outcome Y per unit change in predictor X, which occurs through the mediator (Preacher & Kelley, 2011). The indirect effect can be computed as the product of regression coefficients for the a-path and b-path in the model, and provided that the metrics of X and Y are meaningful and interpretable, the unstandardised indirect effect may be sufficient to illustrate the effect present in the model. It is however of limited comparative utility if X and Y are unique to the study, or are measured on scales which are not widely used (Preacher & Kelley, 2011).

In contrast, partially standardised effects are effect size metrics standardised by the scale of the dependent variable. This is ordinarily undertaken in cases where the independent variable retains interpretable levels, for example group membership; 'trauma' and 'no trauma'. This

quantifies the magnitude of the effect in terms of the SD change in Y, for a change in either M or the specified a-path (MacKinnon, 2008) Although a potentially useful metric, partial standardisation falters similarly to the unstandardised effect if X is continuous or is not meaningfully comparable across studies (Lachowicz et al, 2018)

Completely standardised indirect effect

The completely standardised indirect effect (CSIE), also referred to as the 'index of mediation' (Preacher & Hayes. 2008), is an effect size estimate which enables the comparison of effects across research regardless of the methodological idiosyncrasies of each individual study (Lachowicz et al., 2018). It has a number of advantages over the unstandardised or partially standardised estimates discussed above. Standardising against both the IV and DV scales, means the CSIE can be simply interpreted as representing the expected SD change in Y for one SD change in X, as mediated through M. The 'complete' standardisation facilitates easy comparison across studies in different populations or where different scales have been used for variable measurement (Cheung, 2009). As with the unstandardised or partially standardised effect estimates, the CSIE does not depend on sample size, and confidence intervals can be easily derived (Preacher & Kelley, 2011). Although the CSIE is not bounded in the same way a proportion is, it retains its interpretability even if the b-path is greater than 1, or if either path is negative (Preacher & Kelley, 2011). As a metric on a standard scale, a larger value will always represent a larger effect estimate and standardised effects are generally unbiased, consistent and efficient in both single and multiple mediator models (Miocevic et al., 2017; Cheung, 2009). In terms of power, risk of Type I error, overall balance and precision, bootstrap interval estimates have repeatedly been found to be superior to both ratio and proportion based effect estimates (Miocevic et al, 2017; Williams & MacKinnon, 2008 Briggs, 2006). Standardised indirect effects and their associated bootstrap CIs are therefore highly desirable in mediation effect size reporting.

Odds ratio

An odds ratio expresses the likelihood of an event occurring, versus the likelihood of it not occurring (which stands in contrast to a probability which references the chance of the event occurring against all total possible outcomes). For dichotomous outcomes, such as risk of or transition to psychosis, an odds ratio for the indirect effect can be computed. The accompanying standard errors can be used to compute confidence bounds, but the effect requires interpretation alongside the odds ratios for both the direct and total effects in order to provide context for understanding the relative magnitude of mediation. Odds ratios remain constant across different levels of X, i.e. as X increases by one unit, the odds ratio will increase proportionally in tandem to indicate the resultant change in Y (Szumilas, 2010). Some studies present log odds ratios which are logarithmic odds ratio values, centred on zero. These prevent infinite skew in odds ratio values as an artefact of their original scale. Log odds conversion truncates the scale equidistantly in both directions from zero and normalises the distribution (Eckel et al., 2008).

To deal with the added complexity of non-continuous variables different approaches must be used to estimate the indirect effect and the mediation proportion. The product of coefficients approach (axb), or the difference between coefficients approach (c-c') commonly used with regression coefficients for continuous variables are unreliable in models with dichotomous outcomes (Rijnhart et al., 2019; Mackinnon et al., 2007). Logistic regression is routinely used as it allows for multiple independent variables of either continuous or dichotomous origin to predict a binary outcome. However, an alternative computation method is required to compute the indirect effect or the proportion mediated as a result of the non-concordant scales across the included variables (Rijnhart et al., 2019). Researchers have called again for standardisation to ensure comparability across studies. Options include 'full standardisation' (Kenny, 2008) where the SD of both X and Y is set to 1 and coefficients are standardised by

multiplying by the SD of X and dividing by the SD of Y. This method cannot be applied if X is also dichotomous and as such only has two levels (Rijnhart et al., 2019). An alternative is 'Y-standardisation' (MacKinnon & Dwyer, 1993), similar to partial standardisation (above) where the Y-scale is standardised to have an SD =1 and coefficients represent the expected change in Y per 1SD change in X. this enables comparison across studies provided they share dependent variables. However, a recent review found that standardising coefficients before computing indirect effect estimates did improve bias or efficiency over simpler forms of estimation (i.e. a*b) (Rijnhart et al., 2019).

Alternatives - Kappa-squared (κ^2)

Preacher & Kelley (2011) reviewed existing effect size measures and offered a new alternative which, at the time, they believed to be highly effective. Kappa squared (κ^2) represents the ratio of the indirect effect derived from the sample data to the maximum possible indirect effect value given the specific characteristics and constraints of that data. They suggest that there are limits to the range of potential regression weight values, and what may appear to be a small or trivial effect may be revealed as substantial given the range of possible values. This would enable more accurate interpretation of indirect effects within the context of what is maximally possible in the data set. κ^2 is bounded between 0 (no linear indirect effect) and 1 (the maximum possible value), it is independent of sample size and bootstrap confidence bounds can be derived. However, methodological research by Wen & Fan (2015) undermined the metric, with the foremost criticism centring on the lack of rank preservation and monotonicity. Since an effect size is an expressed metric representing an original value relevant to the research question, it should be monotonic, and should therefore change in the same direction as the effect which it represents. κ^2 does not always do so and may even reduce in magnitude as the corresponding indirect effect increases, and in multiple mediation κ^2 can return values inferior to one single pathway (Wen & Fan, 2015). They also suggest that mathematically, the maximum possible effect size is infinite and that this, as a criteria for establishing an effect size is ill-defined and liable to lead to contradictory and misleading results, particularly in multiple mediation models (Wen & Fan, 2015)

Appendix 6 – Search strategy for systematic review

Search terms for database searches. This example is from Embase, but the same or equivalent terms were used to build the search in each of the databases listed in the chapter.

1. child abuse/

2. psychotrauma/

3. neglect/

4. emotional abuse/

5. emotional stress/

6. family violence/

7. child parent relation/

8. child sexual abuse/

9. physical abuse/

10. violence/

11. stress/

12. life event/

13. or/1-12

14. psychosis/ or acute psychosis/ or paranoid psychosis/

15. schizophrenia/ or paranoid schizophrenia/

16. 14 or 15

17. 13 and 16

18. limit 17 to (human and english language and yr="2000 -Current")

19. survivor*.mp. [mp=title, abstract, heading word, drug trade name, original title, device manufacturer, drug manufacturer, device trade name, keyword, floating subheading word]

20. 18 and 19

Appendix 7 – Agency for Healthcare Research and Quality (AHRQ) Quality Indicators (adapted)

1. Unbiased selection of cohort
i) Are inclusion/exclusion criteria clear?
ii) Is study recruitment strategy clearly described? (Criteria for inclusion in clinical groups and comparison groups is clear where applicable)
iii) Is recruitment relatively free from bias? (bias is considered when newspaper/poster adverts used/students with extra credit)

Yes- all of the above
Partial – if two criteria are met
No – if only one criteria met, and/or recruitment strategy is deemed to be at risk of bias
Unclear – if one or more is not clear

2. Selection minimises baseline differences
i) list as n/a if study consists of only one single group
ii) was control group selection appropriate?
iii) Are groups matched on key differences? (e.g. gender, age, employment status, education, ethnicity)

Yes - no significant differences
Partial – significantly different on one
No - significantly different on 2 or more criteria
N/A – study contains a single group

3. Sample size
i) Are there indications that an a priori power analysis was conducted? If yes, do projected/intended targets match with those recruited? (10% tolerance)
ii) If no power analysis conducted, are sample numbers adequate to detect effects at the desired level?

Yes – power calculation provided and fulfilled or adequate sample size for detection of effects
Partial – power calculation given but recruitment targets not achieved
No – no power calculation and/or sample size too small to be adequately powered
Unclear – not possible to tell if adequate power was achieved

4. Adequate description of cohort
i) Baseline characterisation of sample: age, sex, education, ethnicity, employment

Yes - 4-5 provided (if only 4 reported, the omission must be ethnicity/education/employment)
Partial - 2-3 reported
No - report 1 or less

5. Valid measures used to ascertain psychosis?
i) Are valid and reliable measures used to confirm diagnosis?

Yes – uses established measures
No – measures which are not standardised/validated
Partial – established measures used unconventionally, or split application (e.g. in the case of combining large data sets where different measures have been used)

Unclear – does not describe how diagnosis was confirmed
N/A – where no clinical participants were included

6. Valid measures used for variables?
i) Are valid and reliable measures used to obtain data?

Yes – uses established measures
Partial – combination of measures used, some of which may not be standardised/validated or newly developed measures used (study specific, etc)
No – measures which are not standardised/validated, or variables measured using 2 or less items from questionnaires

7. Adequate handling of missing data
i) Are the details of missing data clearly reported? Is it clear how any missing data was accounted for, or how it was handled in the analysis?
ii) if not reported is there a reason to assume data may have been missing?
iii) if reported, did missing data exceed 20%
iv) if missing data was present were steps taken to minimise bias?

Yes – reporting of no missing data; reporting of low levels of missing data (<10%); no reason to suspect missing data; thorough and transparent description of how missing data were handled (and appropriate strategies applied)
Partial – missing data exceeding 20% and/or without explicit description of handling
No – no description or unsatisfactory handling; reason to suspect missing data had impact on analysis
Unclear – no reference to missing data

8. Analysis controls for confounding factors?
i) If multiple groups, did analysis control for baseline differences?
ii) Does study identify and control for important confounding variables?

Yes – one or both as appropriate
Unclear – no reference to controlling (*downgrading here will be undertaken on a study by study basis; some multiple mediator models imply they are controlling for the other variables in the model and thus are not explicitly referenced in the text*)
No – explicit mention that confounders were not controlled for, or no reference to controlling in studies where baseline differences are highlighted

9. Analytic methods appropriate?
i) Was the analysis appropriate for the type of outcome data? (continuous/categorical)
ii) Was the number of variables appropriate for the sample size (do they take account of small sample size, clustering, etc)

Yes – both
Partial – i) only (with ii being unclear)
No - neither

Appendix 8 – Grading of recommendation assessment, development and evaluation (GRADE) outcome assessment

The Grading of Recommendations Assessment, Development and Evaluation is a working group set up to target the inconsistency of reporting of healthcare evidence (https://www.gradeworkinggroup.org/). Having been adopted by the World Health Organisation and 100 organisations across 19 countries globally, the GRADE assessment

criteria are considered the gold standard in appraising the quality of evidence in clinical trials and interventions research in healthcare. The criteria provide a structured, replicable and comparable framework for the transparent review of outcomes. Here the ratings will be used in conjunction with the AHRQ quality scores and the magnitude of effect sizes to provide a holistic interpretation of the strength and credibility of evidence in each area. The GRADE criteria were applied to each category of mediator, taking into consideration the quality of studies present in the category.

The GRADE criteria are designed to be used to assess an outcome, rather than each individual study. Cross-sectional research starts the rating process as 'low quality' evidence (as opposed to randomised controlled trials which start as 'high' quality) (Dijkers, 2013). There are a number of separate factors which result in the upgrading or downgrading of evidence, depending on their relevance to each outcome.

Outcomes may be upgraded for finding large effect sizes, as this increases the likelihood of there being a true effect; or for demonstrating a dose-response gradient as this, particularly across various studies in an outcome offers strong evidence of the reliable and predictable presence of an effect. In the case of confounding variables, outcomes may be upgraded where all plausible confounders would reduce an overall effect, and an effect is found; or where all possible confounders would suggest a spurious effect but none is found (Dijkers, 2013).

Outcomes may be downgraded for demonstrating serious risk of bias. This refers to elements of study design and conduct which may reduce confidence in the authenticity or accuracy of effects, for example sampling bias where study participants are either non-random, self-referral or of unknown origin. The potential impact of this will depend on the target population of the studies in the outcome. The response rate and how each study declares and handles missing data forms another potential arena for the introduction of bias, as does the selection of instruments of measurement, and their respective reliability and validity ratings. A lack of a priori specification of aims or analyses, or evidence of post hoc testing are further grounds to consider downgrading.

Inconsistency, or unexplained heterogeneity across included studies, which may reduce certainly about the magnitude or direction of effects within outcome is another criteria for consideration (Guyatt et al., 2011). Studies are not upgraded for consistency of results within an outcome, but may be rated down for serious inconsistency, defined primarily in terms of the spread of point estimates within the outcome and the extent to which their associated confidence intervals overlap (Guyatt et al., 2011). Widespread point estimates with non-overlapping confidence intervals would suggest inconsistency. The reviewer must also consider whether the outcome was similarly defined in all studies across the outcome, and whether the conduct of the studies is similar in terms of quality.

Imprecision refers to the relative uncertainly about an effect estimate. The guidelines refer to the 'optimal information size' (OIS) criterion, which should be considered alongside the confidence intervals for effects. The OIS suggests that the sample size contributing to an outcome in a review should be equal to, or greater than that required for a single adequately powered trial (Schünemann, H. (Ed.), 2013). It is suggested that unless the OIS in met or that samples are sufficiently large (N=2000+) that studies are downgraded for imprecision (Schünemann, H. (Ed.), 2013). Similarly, if confidence intervals are wide and/or include zero (i.e. the potential that there is no effect), outcomes should be downgraded. Studies with larger samples sizes are more likely to yield precise estimates but the importance of considering studies across the outcome is emphasised, rather than penalising for a single imprecise study (McMaster University, GRADE online learning modules, https://cebgrade.mcmaster.ca/, n.d.).

Indirectness is linked to the likelihood of there being a true effect, and the confidence with which the existence of an effect can be claimed. Confidence in an estimate is enhanced when the evidence is direct – it comes from the population of interest, and provides evidence for the specific outcome addressed in the research question, as opposed to a proxy or surrogate

(Schünemann, H. (Ed.), 2013). Direct evidence is more generalizable, transferrable and applicable in an external context (McMaster University, GRADE online learning modules, n.d.). Outcomes which include evidence from different populations (clinical, at risk, community) will be considered for levels of indirectness.

The final criteria to be considered is the potential for publication bias. Ordinarily this would refer to selective publication of studies into an outcome. Positive findings and studies with larger samples are traditionally more likely to be published, whereas small studies and null or negative findings often are not. In this review, as studies were largely cross-sectional there is less risk of publication bias affecting estimates, however outcomes will be scrutinised for asymmetries towards large samples.

Appendix 9 – Details of effect size data extraction and conversion per study

This section describes the extraction of data from each study, and the conversions applied where required. Studies are listed in alphabetical order.

Appiah-Kusi 2017

Provided: unstandardized beta coefficients for indirect and direct effects with associated 95% CI's

Calculations: completely standardised indirect effect computed using ab(cs) = ab * (SDx/SDy). Confidence intervals standardised using upper CI (cs) = upper CI * (SDx/SDy) and repeated to compute the lower CI (cs) value.

Notes: Authors were contacted for the mean and SD for the emotional neglect subscale from the CTQ – this was provided.

Ashford 2010

Provided: Point estimates with bias-corrected and accelerated 95% CI's

Calculations: point estimates treated as unstandardized effect estimate. Completely standardised indirect effect calculated using the formulas as in Appiah Kusi (2017), applied to both the estimates, and the 95% CIs.

Bebbington 2011

Provided – ORs and 95% CIs for c and c' for anxiety and depression with contact abuse. a-path data provided as continuous values, not ORs.

Insufficient data available for anxiety and depression with non-consensual intercourse (ab and a paths not reported. May have been possible to estimate *ab* by subtracting log odds *c'* from log odds *c* but may be unreliable

Calculations – The values which enable the calculation of the a-path (and therefore ab) are provided in the text as continuous values (scored 0-8 for anxiety and 0-9 for depression). Firstly confidence intervals were converted to SD following the Cochrane handbook formula (Deeks et al, 2011) for depression and anxiety in those with and without contact abuse.

$$SD = \sqrt{N} \times (\text{upper limit} - \text{lower limit})/3.92$$

These values can then be used to compute *d* by entering the means (provided) and the SDs (calculated here) into the Campbell effect size calculator (Wilson, n.d.) before next converting to logodds values using logodds = d* ($\pi/\sqrt{3}$) (Borenstein, 2011).

Comprehensive Meta-Analysis (version 3, Borenstein et al.) was then used to compute the standard errors for the logodds values – these are interpreted as the a-path estimates.

The log odds values and their standard errors were then input into Microsoft Excel using formulas specified in Iacobucci (2012). This involved computing Z-values for the a-path and b-path in turn (dividing the log odds value for a by its logSE, and likewise for b), then multiplying the Z results to obtain the Z-value for ab, the full indirect path. The SE for this was computed using the formula: $\sqrt{(}Za^2+Zb^2+1)$. The overall Z for mediation was computed as above, taking the Z-value for ab and dividing by the SE for ab (Iacobucci, 2012), and finally the Z-estimate was converted to *r* using $r=Z/\sqrt{(N)}$, N being the overall sample size, here N=7299 (Rosethal & DiMatteo, 2001).

In this case the groups have unequal Ns so further computations are required to establish estimates per group. Firstly, the size of each group is calculated as a proportion of the total sample size this was done by simply dividing the N for the group in question by the study total N. P1 = $N_{group}/N_{total.}$. These proportions are labelled P1 and P2

Following this *r* was converted to *d* using formulae from Practical Meta-analysis (Lipsey & Wilson, 2001): $d = r/\sqrt{((1-r^2)*P1*P2)}$ and 95% confidence intervals for *r* were computed using: lower 95% CI = $r-(r/Z)*1.96$ and upper 95% CI = $r+(r/Z)*1.96$

Finally, the values were converted from *d* to OR using OR =EXP ($d*(\pi/\sqrt{3})$). The same formula was applied to both 95% CI estimates too, in order to convert to CI for OR. In each case the *d* for effect size was substituted for the lower or upper 95% CI value for *d* (Lipsey & Wilson, 2001). The values derived in this final step were checked using the Campbell effect size calculator to ensure accuracy of the formulae applied in Microsoft Excel.

The OR and 95% CI values for c and c' paths were provided in the paper. These were extracted and used to contextualise the computed indirect effects.

Note: Actual N may be higher, because 'contact abuse' comprised sexual touching and sexual intercourse, but the exact Ns in this composite category are not provided. It was therefore assumed that all those who experienced sexual intercourse prior to 16 had also experienced unwanted sexual touching prior to 16.

Berenbaum 2008

Provided – correlations (*r*) between maltreatment and mediators (paths *a* and *c*); Sobel Z values for indirect effect (*ab*)

Calculations – the correlation values for the a-path and c-path were extracted from table 5. The *r* values were converted to *d* using: $d = \frac{2r}{sqrt(1-r^2)}$ (Borenstein, 2011).

Variance for *r* was calculated using the Campbell Effect Size Calculator, inputting the *r*-value and the sample size. This variance was then converted to variance for *d* using: $Vd = \frac{4Vr}{(1-r^2)^3}$

The *d* values were then converted to logodds ratios using: $LogOR = d\left(\frac{\pi}{sqrt3}\right)$ and the variance using: $VLogOR = Vd(\frac{\pi^2}{3})$

The log odds ratios were then converted to odds ratios using the exponent function in Excel: =EXP(logodds)

95% CIs were calculated for the OR values using a Comprehensive Meta Analysis spreadsheet, set up to convert logodds ratios and their variance to 95% CIs for odds ratios (Borenstein, 2011). The above steps yielded OR and CI values for both the a-path and the c-path.

The indirect effect estimates were derived using the reported Sobel Z mediation statistics in Table 8. Each Z statistic was used to find the corresponding *p*-value using an online calculator (https://www.socscistatistics.com/pvalues/normaldistribution.aspx).

This *p*-value was input into the Campbell effect size Calculator (https://www.campbellcollaboration.org/escalc/html/EffectSizeCalculator-R7.php) along with the sample size for each group to find *r*.

The above steps were followed again to convert *r* to *d* and *d* to logodds, and finally odds ratios with 95% CIs.

The 'remaining contribution (β)' column in table 8 was used to calculate c'. β was converted to *r* using *r* = β+0.05 (Peterson & Brown, 2005) and the variance for *r* was again computed using the Campbell Calculator, inputting the *r*-value and the sample size.

Following the same procedure as above, *r* + variance was converted to *d* + variance, and *d* to log odds + variance, and finally to odds ratio. Again the variance for log odds ratio was input into the CMA spreadsheet to compute 95% confidence intervals for the odds ratios.

The c' conversions were performed twice - once assuming the β stats provided were already standardised, the other using the SDx/SDy formula to 'standardise' them, assuming they were unstandardised.

The formula c'=c-ab was used to double-check the estimates. The figures from the unstandardised table were more closely matched, suggesting that the β from Table 8 were unstandardised estimates. Therefore, figures from the 'assumed unstandardised' table were used in the final reporting.

Boyda & McFeeters 2015

Provided – logistic coefficients for the indirect effect (logodds), standard error (SE) and 95% CIs in Table 4. Direct and total effects reported as odds ratios with associated 95% Cis in Table 3.

Calculations – The odds ratios for the direct effect (PSQ c') and total effect (PSQ c) and associated confidence intervals were simply extracted from Table 3. These were already presented in the odds ratio format and thus did not require further conversion.

Since the beta coefficients contain 1, and are significant, and the outcomes are binary, it is reasonable to assume the values in Table 4 are logodds values (and 95% CIs). These were converted to OR using the '=exp' function in Microsoft Excel.

Choi 2015

Provided – standardised path coefficients, individual subscale means and SDs for trauma, mean and SD for psychotic symptoms.

Calculations –The standardised path coefficients were unstandardised using the SD of psychotic symptoms in Table 1 and the combined SD for the trauma variable computed above. The formula 'unstandardised coefficient = standardised coefficient / (SD of x / SD of y)' was used.

Second, the N and inexact p-values in the diagram to compute t-values using excel function '=TINV (p-value, degrees of freedom)'

Third, each unstandardised coefficient was divided by t to compute SE.

Fourth, the unstandardised coefficients and unstandardised SEs were entered into the

$$SE_{ab} = \sqrt{a^2 SE_b^2 + b^2 SE_a^2}$$

equation

This provides an unstandardised SE for ab. This can be used to compute to lower and upper bounds of the (unstandardized) CIs, using the formula $ab \pm 1.96 SE_{ab}$

Finally, values were re-standardised by using the formula from Appiah-Kusi (above).

Note: Means and SDs were reported per subscale for the CTQ (Physical abuse, emotional abuse and sexual abuse). A combined latent variable 'child abuse' was used in the mediation model therefore a combined mean and SD were computed using the information provided.

Choi 2017

Provided – standardised indirect effects and bootstrapped 95% CIs

Calculations – extraction from text – data already provided in a standardised format

Cole 2016

Provided – unstandardised beta coefficients for simple and multiple mediation models; means and standard deviations for CATS total and PDI. Authors were contacted for the LSHS mean and SD omitted from the paper which they provided. Unstandardised 95% Cis are provided for the indirect effects in Table 2.

Calculations - Completely standardised indirect effect computed using ab(cs) = ab * (SDx/SDy). Confidence intervals standardised using upper CI (cs) = upper CI * (SDx/SDy) and repeated to compute the lower CI (cs) value.

Evans 2015 (narrative inclusion)

Provided – median and IQR for variables; indirect effect estimates expressed as log odds, with 95% CIs.

Calculations – It was not possible to compute 95% CIs for the total and direct effects – this would have involved too many assumptions, and therefore was not undertaken. The indirect effects and 95% CIs were converted from the reported logodds values in Table 3 (dissociation) and Table 4 (self-concept clarity) using the '=EXP' function in Microsoft Excel.

Fisher 2012

Provided – Odds ratios (adjusted for confounders) and 95% CIs

Calculations – Odds Ratios and 95% CIs are reported directly in Table 1. These are partitioned out into the four different mediator contributions, along with an 'indirect total' effect, as well as a total and direct effect for each model.

Gaweda 2018 a

Provided – standardised regression weights, inexact p-values, SD for independent and dependent variables.

Calculations – The model presented is an integrated model so indirect effects were computed using the standardised regression weights for the 'a' and 'b' paths for each mediator in Fig 3. It was noted that in the multiple mediator model, the indirect effect estimates for each mediator are controlling for all other variables in the model.

In order to compute confidence intervals for these indirect effect estimates, the standardised values had to be first unstandardised using the formula: unstandardised ab = standardised

ab/(SDx/SDy). T-values were computed using the =TINV(p-value, degrees of freedom) formula in Excel.

Standard error of ab was computed using SEab = unstandardised ab/t-value.

The 95% confidence intervals were computed using the formulas:

Upper CI = unstandardized ab + (1.96xSEab)

Lower CI = unstandardized ab - (1.96xSEab)

The unstandardised ab value and 95% CIs were then re-standardised using the formula ab(cs) = ab * (SDx/SDy).

Gaweda 2018 b

Provided – standardised path coefficients, inexact p-values, SD for trauma.

Calculations – parallel mediator model with a and b paths provided for each mediation relationship.

Standardised indirect effect computed by multiplying the path coefficients a * b.

Unstandardised path coefficients calculated using the formula: unstandardised ab = standardised ab/(SDx/SDy). The reported SD for trauma was used, along with an SD of 1 for the latent trauma variable (see Sheinbaum, 2014)

t-values were computed, as above, using N-1 for degrees of freedom and the inexact p-values provided. An approximate SE for each individual path was then computed using unstandardised path coefficient/t-value.

The unstandardised path coefficient values were then squared, as were the approximate SE values and these were entered into the formula:

$$SE_{ab} = \sqrt{a^2 SE_b^2 + b^2 SE_a^2}$$

This yields a standard error estimate for the full indirect path which was then used as in Gaweda 2018 1. above to compute 95% CI upper and lower bounds. The unstandardised ab and 95% CIs were then standardised using the formula: ab(cs) = ab * (SDx/SDy).

Note – this may be an overlapping sample with the above study.

Goldstone 2010 (narrative inclusion)

Calculations – insufficient information to be included in the mediation analysis

Goldstone 2011 (narrative inclusion)

Calculations - insufficient info to be included in the mediation analysis

Goodall 2015

Provided – unstandardised parameter estimates with SE and 95% bias-corrected confidence intervals. Mean and SD for IV and DV in table 1.

Calculations – unstandardised parallel multiple mediator parameter estimates and associated 95% CIs are given in Table 3. These were standardised using the SDs provided for IV and DV in Table 1, entered into the formula: ab(cs) = ab * (SDx/SDy).

Confidence intervals were standardised using upper CI (cs) = upper CI * (SDx/SDy) and repeated to compute the lower CI (cs) value.

Hardy 2016

Provided – odds ratios for direct, indirect and total effect, SE and p-values

Calculations – Odds ratios for indirect, direct and total effects are provided in Table 5. The SE values are logSE values – these were required to compute 95% CIs for the effects.

95% CIs were calculated by entering the logSE into the formula (upper CI-lower CI)=logSE x 3.92.

For the lower CI of the logodds values for ab, c and c', half of this value was subtracted from the logodds of the point estimate for the effect. For the upper CI value, half of this value was added to the logodds for the point estimate.

These values were then converted to OR for the 95% CIs using '=EXP' in Excel.

Jaya 2016

Provided – unstandardised regression coefficient estimates and confidence intervals. Mean and SD for positive psychotic symptoms reported in Table 1. Authors contacted for Social Adversity mean and SD which was provided. This is a latent variable and therefore Mean = 0, SD = 1.

Calculations – as the SD for both the independent and dependant variables were available, the rounded indirect effect estimates from Table 3 were standardised using the formula: ab(cs) = ab * (SDx/SDy).

Unstandardised confidence intervals for the indirect effects were also provided. These were standardised using: upper CI (cs) = upper CI * (SDx/SDy) and repeated to compute the lower CI (cs) value.

Lincoln 2017

Provided – standardised path coefficients & approximate significance values; variable means and SDs (by item). Authors contacted for means and SDs of total scores.

Calculations – Provide complex time-lagged models with path coefficients however it was not possible to partial these out, so the simple models explained in the text were used. The unstandardised effects and confidence intervals were reported in the text. These were extracted directly.

To work out SDs for paranoia frequency and distress the dataset provided by Wusten 2018 (N=7141) was used. The distress and frequency scores for items 2,6,7,10 and 22, as discussed in Schlier 2015, which Lincoln 2017 cite when referring to their 'paranoia subscale' were computed. The SDs for mean total for paranoia, using Wusten 2018, and SDs for mean total for CT and Psychotic symptom distress provided by author were used in the calculations.

These SDs were then used to standardise both the effect and the confidence intervals using the formula: ab(cs) = ab * (SDx/SDy).

Marwaha 2014

Provided – odds ratios, SE, p values and 95% CIs for each mediation model

Calculations – Odds ratios for total ('reduced'), direct ('full') and indirect ('difference') pathways are reported in Table 5. These were extracted, along with their 95% confidence intervals. The odds ratio and 95% CI for the indirect effect are of primary interest, but the direct and total effects were required to contextualise the magnitude of the effect size.

Marwaha & Bebbington 2015

Provided – odds ratios, standard error and confidence intervals for direct, indirect and total effects

Calculations – odds ratios and 95% CIs were extracted from the paper for the indirect effect (and the direct and total effect in order to contextualise the mediation pathway). Both sets of results are from parallel mediation models with depression and anxiety, therefore results for each independent mediator are not available.

McDonnell 2018

Provided – standardised effect, unstandardised path coefficient with bootstrapped 95% CIs, p-values.

Calculations – as only the median and IQR were reported for paranoid ideation (and further details could not be obtained from the research team) the ratio between the standardised and unstandardised effect was computed using: standardised effect/unstandardised beta coefficient, then multiplying each unstandardised 95% CI by the result in order to find approximate standardised CI values.

Note: the obtained values are approximate

Morgan 2014

Provided –odds ratios (unadjusted and adjusted for confounders), 95% CIs, p-values, percentage of total effect.

Calculations – odds ratios, adjusted for age, gender, ethnicity, study centre and parental history of psychosis were extracted along with their 95% CIs. These were taken from the complex multiple mediation model (Fig. 3), based on the total sample. The pathways are expressed as controlling for the other variables in the model.

Pearce 2017

Provided - unstandardised regression pathway coefficients (assumed unstandardised as PROCESS package used) with 95% CIs and p-values reported in text. SD for IV and DVs provided.

Calculations – both models contain parallel mediators and the authors state that each outcome is controlling for the effect of the alternative outcome (voices/paranoia).

Completely standardised indirect effect sizes were calculated using the paths described in the text for each individual mediator in the model, controlling for the other mediator (dissociation/fearful attachment)

Unstandardised path coefficients were entered into the formula: ab(cs) = ab * (SDx/SDy). The SDs in Table 2 were used.

Confidence intervals were standardised using upper CI (cs) = upper CI * (SDx/SDy) and repeated to compute the lower CI (cs) value.

Note: total sample = 112 clinical psychosis (77 used in mediation – no details about the specific subsample, but no sig differences between those who completed the surveys and those who did not complete the full battery)

Perona Garcelan 2012

Provided – Unstandardised beta coefficients with 95% CIs. SDs for childhood trauma, hallucinations and delusions measures provided in Table 2.

Calculations – details were extracted for both the simple mediation models, and for the complex multiple mediator models. All beta coefficients and 95% CIs were unstandardised so SDs provided for the IV and DV were used to compute the completely standardised indirect effect using the formula: ab(cs) = ab * (SDx/SDy).

Confidence intervals were standardised using upper CI (cs) = upper CI * (SDx/SDy) and repeated to compute the lower CI (cs) value.

Perona Garcelan 2013

Provided – non-standardised beta coefficients for a, b and c and c' paths. Bootstrapped indirect effect with 95% CIs

Calculations – When contacted, the authors kindly provided the means and SDs for high, middle and low HP groups for the LSHS measure. The text states that a total of 143 individuals from the total sample reported traumatic experiences, with a mean of 1.62 and an SD of 1.03. As the mediation analysis was performed on the full sample, a weighted mean calculator was used to compute a mean and SD for the full sample, based upon N=143, mean 1.62, SD = 1.03 and N=175, mean = 0, SD = 0 (as the rest of the sample reported no trauma). The result shows this to be a skewed sample, but this matches the narrative as over half of the same reported having no traumas.

The SD for trauma calculated here, and the SD for hallucination-proneness were used, along with the provided unstandardised indirect effects reported in Table 5 in the formula: ab(cs) = ab * (SDx/SDy).

The unstandardised confidence intervals provided in the same table were standardised using upper CI (cs) = upper CI * (SDx/SDy) and repeated to compute the lower CI (cs) value.

Pilton 2016

Provided – authors confirmed the effects reported in Table 3 are standardised, with 95% CIs

Calculations – As indirect, direct and total effects were all standardised the values were directly extracted from Table 3 in the paper.

Powers 2016

Provided – logistic regression coefficients (logodds), standard error, odds ratios and 95% CIs, p-values

Calculations – logodds values for the a and b paths were extracted from the diagram (Fig 1). These were multiplied (axb) to obtain a logodds estimate for the indirect effect. Confidence intervals for this pathway were listed in the text. These values were converted to odds ratios using the '=EXP' function in Excel.

In order to provide context for indirect paths expressed as odds ratios, the odds ratios and 95% CIs for the total and direct effects were also required. The loggodds values and SE were extracted from Table 3.

The respective SE values were entered into the formula: upper-lower logCI = 3.92*logSE. This yielded a mid-point estimate for the confidence intervals.

The lower bound was computed using the formula: lower log CI = beta(log)-(upper-lower log CI /2) and the upper bound using: upper log CI = beta(log)+(upper-lower log CI /2).

Log values (beta and CIs) were then converted to odds ratios, as with the indirect effect, using the '=EXP' function in Excel.

Rossler 2016

Provided - Odds ratios and 95% CIs

Calculations – no conversions were required. Odds ratios and associated 95% CIs were directly reported and thus extracted. Values of the direct (c') path and the indirect (ab) path were lifted from Table 4, and the Total (c) path from Table 3.

Sheinbaum 2014

Provided – unstandardised parameter estimates, SE, 95% bias corrected confidence intervals. Article supplement provides means and SDs for measures.

Calculations – after contacting the authors for trauma mean and SD, they confirmed that a composite latent variable had been used with its mean standardised to 0 and an SD of 1. Means and SDs were extracted from supplementary tables for the outcome variables.

These means were entered along with the unstandardised parameter estimates for the indirect effects into the formula: ab(cs) = ab * (SDx/SDy).

Confidence intervals were provided for each indirect effect estimate. These were standardised using upper CI (cs) = upper CI * (SDx/SDy) and repeated to compute the lower CI (cs) value.

Sheinbaum 2015

Provided – raw parameter estimates, SE, 95% and 99% confidence intervals. Means and SDs provided for IV and IV in Table 1.

Calculations – All models contained multiple mediators and thus provided indirect effect estimates which are controlled for the other mediators in the model (and depression, entered as a covariate).

The SDs from Table 1 were entered along with the raw parameter estimates into the formula: ab(cs) = ab * (SDx/SDy).

95% Confidence intervals were standardised using upper CI (cs) = upper CI * (SDx/SDy) and repeated to compute the lower CI (cs) value.

Sitko 2014

Provided - unstandardised beta coefficients provided for a, b c and c' paths separately, along with their SEs. Unstandardised 95% CIs provided for c' paths.

The b-paths in table 3 do not make it clear which trauma types they map on to. The means and SDs of the measures are not available and the paper which is referenced as containing this information, does not.

Calculations –The ratios between the standardised and unstandardised c' paths in Table 4 were computed. The ratios were applied to the unstandardised c and SE values reported in Table 1. This ratio was also applied to standardise the 95% CIs reported in Table 4. Estimated 95% CIs were computed for the estimated standardised c paths using the formulas:

Upper CI = unstandardized c + (1.96xSEc)

Lower CI = unstandardized c - (1.96xSEc)

Standardised ab values were then computed using: ab = standardised c – standardised c'. It was possible to apply this to mediation models for each distinct trauma type, but not individual attachment styles due to absence of information - 'overall attachment' was used as the mediator.

Very rough estimates of the 95% CIs for ab were computed using:

Lower 95% CI for std ab = std c – upper 95% CI for std c'

Upper 95% CI for std ab = std c – lower 95% CI for std c'

This method was used as subtracting upper CI of c' from c should indicate the lowest possible value of ab – the higher the direct effect, the lower the indirect effect. The upper CI of c' indicates what the lowest bound of ab might be.

This does not take into account variance in standardised c, and in order to compute 95% CIs for ab, the b and SE values are required to be accurate.

Note: The steps used to calculate the confidence intervals for the ab paths led to non-significant results as the CIs included zero. However, the text indicates that significant pathways were found (e.g. for neglect and paranoia).

Thompson 2016

Provided – odds ratios, robust SE, p-values, 95% CIs

Calculations – Odds ratios and associated 95% confidence intervals are provided in Table 1. These were extracted directly for the indirect effect, along with the total and direct effects for context.

Van Dam 2014 (narrative inclusion)

Provided – regression analyses but insufficient mediator data

Calculations – insufficient data to compute indirect effects – tests of association rather than mediation

Van Nierop 2014

Provided – standardised indirect effect size, p-value and proportion of effect (%).

Calculations – standardised effect sizes were extracted from table 3. Z-values were computed by entering the provided p-values into Microsoft Excel using the formula =ABS(NORMSINV(p-value/2)).

This Z-value was then used to calculate standard error: SE = standardised beta/Z

The SE value was then used to determine confidence intervals using the formula: Upper CI = standardised beta + (1.96*SE) and lower CI = standardised beta – (1.96*SE).

This method involves some assumptions and thus the 95% CIs are approximate. Results were sense-checked against the narrative description and the proportion of effect reported in the paper.

Varese 2012

Provided – point estimates for mediated direct and total effects with their 95% CIs

Calculations – mediation analyses was conducted on the aggregate sample (clinical and control groups combined) and on the clinical group alone. Results for the clinical group were presented as three separate groups for current, remitted and no experience of hallucinations. A combined mean calculator was used to calculate the clinical group total mean and SD (summed from the three sub-groups), and an aggregate sample mean and SD for both the hallucinations measure, the overall trauma measure and each of the trauma subscales from Table 2.

These SDs were then used to standardise the point estimates and associated confidence intervals provided for the indirect effect in Table 4.

Wickham 2016

Provided - standardised and unstandardised regression coefficients, SE, means and SDs for variables

N = clinical N=72, control N=72. Mediation analysis performed on N=50 of the clinical sample after listwise deletion, although results tables still state N=72 (Table 3)

IV - childhood sexual abuse/ childhood emotional neglect (measures using CTQ) continuous

DV – hallucinations/ paranoia (measured using PANSS) continuous

Calculations – Means and SDs for the IV and DV variables are provided in Table 2

Indirect effect information was only available for two complete models, both using childhood emotional neglect as the IV and paranoia as the DV.

The a and b path betas and their respective SEs provided in the text were entered into the formula:

$$SE_{ab} = \sqrt{a^2 SE_b^2 + b^2 SE_a^2}$$

This yields a standard error estimate for the full (unstandardised) indirect path which was then used to calculate upper and lower bounds for 95% CIs.

Upper CI = unstandardized ab + (1.96xSEab)

Lower CI = unstandardized ab - (1.96xSEab)

The unstandardised ab value and 95% CIs were then standardised using the formula ab(cs) = ab * (SDx/SDy).

Appendix 10 – Table of excluded studies from systematic review

Excluded studies

The following table details studies or reports excluded after inspection of the full-text report, or via correspondence with authors. Studies or reports excluded on basis of title or abstract alone are not detailed as these are too numerous and the vast majority were of different conditions or were otherwise unrelated to the review question.

Study	Reason for exclusion
Aas et al (2011)	No mediator data
Aas et al (2012)	No mediator data
Aas et al (2013)	Review
Addington et al. (2013)	No mediator data
Alameda et al (2014)	No mediator data
Alameda et al (2015)	No mediator data
Alameda et al (2016)	Positive symptoms not dependent variable
Allen et al (2005)	No mediator data
Alvarez et al (2015)	No mediator data
Amr et al (2016)	No mediator data
Armando. M (2012)	No mediator data
Axelrod et al (2006)	No mediator data
Aydin et al (2016)	Positive symptoms not dependent variable
Bae et al (2010)	No mediator data
Bak et al (2005)	Positive symptoms not dependent variable
Barker et al (2015)	No psychological mediator
Barker-Collo et al (2011)	No mediator data
Barrantes-Vidal (2013)	No mediator data
Baudin et al (2016)	No mediator data

Beards & Fisher (2014)	Review
Beards et al (2014)	No childhood trauma
Beards et al (2015)	No childhood trauma
Bebbington et al (2004)	No mediator data
Bebbington et al (2009)	Review
Bebbington et al (2011)	No mediator data
Begeman et al (2016)	No mediator data
Behrendt et al (2005)	No childhood trauma
Ben Zeev et al (2011)	No childhood trauma
Bendall et al (2008)	Review
Bendall et al (2010)	Review
Bendall et al (2011)	No mediator data
Bendall et al (2012)	No psychosis
Bendall et al (2013)	No mediator data
Bentall et al (2007)	Review
Bentall et al (2012)	No mediator data
Bentall et al (2014)	Review
Berenbaum 2003	No mediator data
Berg et al (2014)	Duplicate
Berg et al (2014)	No psychological mediator
Berry et al (2009)	No mediator data
Berry et al (2012)	No childhood trauma
Berry et al (2015)	Positive symptoms not dependent variable
Berry et al (2017)	Review

Bhavsar et al. (2017)	No psychological mediator
Bhavsar, V (2015)	No mediator data
Bhui et al (2003)	No mediator data
Bilgi et al (2017)	No mediator data
Bob & Mashour (2011)	Review
Bob et al (2007)	No mediator data
Bortolon et al (2014)	No childhood trauma
Boyda et al (2015)	No childhood trauma
Boyette et al (2014)	Positive symptoms not dependent variable
Braehler et al (2013)	No mediator data
Bratlien et al (2014)	No mediator data
Briere et al (2007)	No mediator data
Briere et al (2010)	No psychosis
Broussard et al (2013)	No mediator data
Bucci et al (2017)	No mediator data
Burns et al (2010)	No psychosis
Butjosa et al (2016)	No mediator data
Calem et al (2017)	No mediator data
Calvert et al (2008)	No mediator data
Campbell et al (2007)	Sample not suitable
Catalan et al (2017)	No mediator data
Catone et al (2015)	No childhood trauma
Chaumette et al (2016)	Review
Choi et al (2014)	No psychosis

Clarke et al (2012)	No mediator data
Corcoran et al (2003)	Review
Cotter et al (2015)	Review
Cristobal-Narvaez et al (2016)	No mediator data
Cunningham et al (2016)	Review
Cutting & Docherty (2000)	No mediator data
Daalman et al (2012)	No mediator data
Daly, M (2011)	No mediator data
Davies et al (2014)	Insufficient data
Davis et al (2016)	Review
de Leede-Smith & Barkus (2013)	Review
Debbane et al (2016)	Review
DeCou et al (2017)	No psychological mediator
Denenny et al (2015)	No mediator data
DeRosse et al (2014)	No mediator data
DeVylder et al (2013)	No mediator data
DeVylder et al (2016)	No mediator data
Dorahy et al (2004)	No mediator data
Dorahy et al (2009)	No mediator data
Ered et al (2017)	No childhood trauma
Etain et al (2015)	No mediator data
Etain et al (2017)	No psychological mediator
Fallon, P (2008)	Review
Fisher et al (2009)	No psychological mediator

Fisher et al (2011)	No mediator data
Fisher et al (2013)	No mediator data
Fowler et al (2011)	No childhood trauma
Fowler et al (2015)	No childhood trauma
Freeman & Fowler (2009)	No childhood trauma
Frias Ibanez et al (2014)	No mediator data
Frissen et al (2015)	No mediator data
Fryers et al (2013)	Review
Gallacher et al (2016)	No mediator data
Galleti et al (2017)	No mediator data
Galletly et al (2011)	No mediator data
Galletly et al (2016)	No mediator data
Garcia et al (2016)	No mediator data
Garety et al (2007)	No mediator data
Gayer-Anderson et al (2014)	Insufficient data
Gayer-Anderson et al (2015)	No mediator data
Gibson et al (2013)	No childhood trauma
Gibson et al (2014)	No childhood trauma
Gibson et al (2016) 1	No mediator data
Gibson et al (2016) 2	Review
Gibson et al (2017)	No mediator data
Gil et al (2009)	No mediator data
Glasova et al (2004)	No mediator data
Goldsmith et al (2013)	No psychosis

Goodman et al (1997)	No mediator data
Groman et al (2013)	No mediator data
Guloksuz et al (2016)	No mediator data
Gumley et al (2014)	Review
Harder, S. (2014)	No mediator data
Hardy et al (2005)	No mediator data
Haug et al (2015)	No mediator data
Heins et al (2011)	No mediator data
Hesse et al (2015)	No mediator data
Holowka et al (2003)	No mediator data
Holtzman et al (2012)	No mediator data
Holtzman, C. (2016)	Review
Houston et al (2008)	No psychological mediator
Howes & Murray (2014)	Review
Hutchinson & Hassen (2004)	No mediator data
Ibanez et al (2014)	Review
Ira et al (2014)	No psychological mediator
Isvoranu et al (2016)	No mediator data
Jaffee, S.R. (2017)	Review
Janssen et al (2005)	No mediator data
Jaya et al (2015)	Duplicate
Jennissen et al (2016)	No psychosis
Johnson et al (2014)	No mediator data
Joukamaa et al (2008)	No mediator data

Kamsner et al (2000)	No psychosis
Kanamuller et al (2014)	No psychosis
Kapfhammer et al (2012) 1	Review
Kapfhammer et al (2012) 2	Review
Kelly et al (2016)	No mediator data
Kennedy et al (2013)	No mediator data
Kilcommons & Morrison (2005)	No mediator data
Kilcommons et al (2008)	No childhood trauma
Kilian et al (2017)	Positive symptoms not dependent variable
Klewchuck et al (2007)	No mediator data
Kocsis-Bogar et al (2014)	Review
Kotlicka-Antczak et al (2008)	Full text not available in English
Kraan et al (2015)	No mediator data
Kraan et al (2017)	No mediator data
Kramar, J. (2015)	No mediator data
Kramer et al (2012)	No psychological mediator
Kramer et al (2014)	No mediator data
Laddis & Dell (2012)	No mediator data
Laloyaux et al (2016)	No childhood trauma
Lang & Becker (2014)	No mediator data
Lardinois et al (2011)	No mediator data
Larkin & Read (2008)	Review
Lataser et al (2013)	No childhood trauma
Leask, S.J. (2004)	Review

Lecomte et al (2005)	No childhood trauma
Lennon, R (2015)	No childhood trauma
Leonhardt et al (2015)	No mediator data
Li et al (2015)	No mediator data
Li et al (2017)	No mediator data
Lincoln et al (2009)	No childhood trauma
Lincoln et al (2010)	No childhood trauma
Liotti & Gumley (2008)	Review
Loewy et al (2011)	Sample not suitable
Loewy, R. (2012)	No mediator data
Lommen & Restifo (2009)	No mediator data
Longden et al (2012)	Review
Longden et al (2016) 1	No mediator data
Longden et al (2016) 2	Review
Lopes et al (2013)	No mediator data
Lovatt et al (2010)	No mediator data
Luutonen et al (2013)	No mediator data
Lysaker et al (2001) 1	No mediator data
Lysaker et al (2001) 2	No mediator data
Lysaker et al (2002)	No mediator data
Lysaker et al (2005) 1	No mediator data
Lysaker et al (2005) 2	No mediator data
Lysaker et al (2007)	No mediator data
Lysaker et al (2008)	No mediator data

Lysaker et al (2011)	No mediator data
Magaud et al (2013)	No mediator data
Malcolm et al (2015)	No mediator data
Mathews et al (2016)	Review
Matos et al (2012)	No childhood trauma
Maziade et al (2014)	Sample not suitable
McCabe et al (2012) 1	No mediator data
McCabe et al (2012) 2	Insufficient data
McCabe et al (2013)	No mediator data
McCarthy Jones & Longden (2015)	No mediator data
McLaughlin et al (2010)	No mediator data
McNeill et al (2015)	Positive symptoms not dependent variable
Melle & Dazzan (2014)	No mediator data
Michail & Birchwood (2014)	No mediator data
Mier & Kirsch (2017)	Review
Millan et al (2017)	Review
Misdrahi, D. (2016)	Full text not available in English
Miskiak et al (2017)	Review
Moffa et al (2017)	No mediator data
Morgan & Fisher (2007)	Review
Morgan & Gayer-Anderson (2016)	Review
Morgan et al (2013)	No mediator data
Morgan et al (2014)	No mediator data
Morgan et al (2016)	No mediator data

Morgan, C. (2012)	No psychological mediator
Morris et al (2011)	No mediator data
Morrison et al (2003)	No mediator data
Morrison et al (2005)	Review
Moskowitz et al (2008)	Review
Mrizak et al (2016)	No mediator data
Muenzenmaier et al (2015)	No mediator data
Mueser et al (2002)	No mediator data
Mueser et al (2004)	No psychosis
Mueser, K.T. (2013)	No mediator data
Mujica-Parodi et al (2013)	No childhood trauma
Murphy et al (2013) 1	No mediator data
Murphy et al (2013) 2	No childhood trauma
Murphy et al (2013) 3	No childhood trauma
Murphy et al (2014)	No mediator data
Murphy et al (2015)	Sample not suitable
Myin-Germeys et al (2003)	No childhood trauma
Newman et al (2010)	No mediator data
Nugent et al (2013)	No mediator data
Offen et al (2003) 1.	Positive symptoms not dependent variable
Offen et al (2003) 2.	Insufficient data
O'Hare et al (2013)	Positive symptoms not dependent variable
O'Hare et al (2015)	No mediator data
Oliver et al (2012)	No childhood trauma

Ostefjells et al (2014)	No mediator data
Outcalt & Lysaker (2012)	No mediator data
Paksarian et al (2015)	No mediator data
Palmier-Claus et al (2016)	Positive symptoms not dependent variable
Pec et al (2015)	No mediator data
Peleikis et al (2013)	No mediator data
Pena-Salazar et al (2012)	Full text not available in English
Pena-Salazar et al (2016)	No mediator data
Perona-Garcelan et al (2010)	No mediator data
Phillips et al (2000)	No mediator data
Picken et al (2011)	No mediator data
Pietrek et al (2013)	No mediator data
Pinhiero et al (2015)	Review
Pos et al (2016)	Positive symptoms not dependent variable
Rajkumar, R.P. (2014)	No mediator data
Ramsay et al (2011)	No mediator data
Rapisarda et al (2014)	No mediator data
Read et al (2001)	Review
Read et al (2003)	No mediator data
Read et al (2005)	Review
Read et al (2008)	Review
Read et al (2009)	Review
Read et al (2014)	Review
Read, J. (1997)	Review

Read, J. (1998)	No mediator data
Read, J. (2013)	Review
Reininghaus et al (2016)	No mediator data
Renard et al (2012)	Positive symptoms not dependent variable
Resnick et al (2003)	No mediator data
Ringer et al (2014)	No mediator data
Roper et al (2015)	No mediator data
Rosen et al (2017)	No mediator data
Ross & Keyes (2004)	No mediator data
Rossler et al (2014)	No mediator data
Russo et al (2014)	No mediator data
Sar et al (2010)	No mediator data
Schafer & Conus (2012)	No mediator data
Schafer & Fisher (2011) 1	Review
Schafer & Fisher (2011) 2	Review
Schafer et al (2006)	No mediator data
Schafer et al (2012)	No mediator data
Schalinski & Teicher (2015)	No mediator data
Schalinski et al (2015)	No mediator data
Schalinski et al (2016)	No psychosis
Scheller-Gilkey et al (2004)	No mediator data
Schenkel et al (2005)	No mediator data
Schmidt et al (2015)	Positive symptoms not dependent variable
Scholes et al (2010)	Review

Schomerus et al (2008)	No childhood trauma
Schreuder et al (2017)	Positive symptoms not dependent variable
Schroeder, K. (2016)	No mediator data
Scott et al (2007)	No mediator data
Sellwood et al (2012)	No mediator data
Selten & Cantor-Graae (2014)	Review
Selten et al (2013)	Review
Shack et al (2004)	No mediator data
Shannon et al (2009)	No mediator data
Sheinbaum et al (2012)	Duplicate
Sheinbaum et al (2015)	Review
Sheinbaum et al (2017)	Insufficient data
Shevlin et al (2011)	No mediator data
Sideli et al (2013)	No mediator data
Sideli, L. (2012)	No mediator data
Siracusano & Rubino (2010)	No mediator data
Skehan et al (2012)	Review
Smeets et al (2015)	No mediator data
Sowden et al (2012)	No mediator data
Sporle et al (2011)	Positive symptoms not dependent variable
Stain et al (2012)	No mediator data
Stain et al (2013)	No mediator data
Stain et al (2014)	No mediator data
Stevens et al (2013)	No psychosis

Stevens et al (2017)	No mediator data
Stowkowy et al (2016)	Positive symptoms not dependent variable
Subica et al (2011)	No psychosis
Suchoka et al (2016)	No mediator data
Sweeney et al (2015)	No mediator data
Tikka et al (2013)	No mediator data
Trauelsen et al (2016)	No mediator data
Trotman et al (2014)	No childhood trauma
Trotta et al (2013)	No mediator data
Tull et al	No psychosis
Ucok & Sahin (2010)	No mediator data
Ucok et al (2015)	Positive symptoms not dependent variable
Upthegrove et al (2016)	No mediator data
Valmaggia et al (2010)	No mediator data
Valmaggia et al (2012)	Positive symptoms not dependent variable
Valmaggia et al (2015)	No mediator data
van Dam et al (2012)	No mediator data
Van Dam et al (2014)	No mediator data
van Dam et al (2015)	No mediator data
Van Der Valk et al (2012)	No childhood trauma
van Nierop et al (2013)	No mediator data
van Nierop et al (2014)	No mediator data
van Nierop et al (2015)	No mediator data
van Nierop et al (2016)	Positive symptoms not dependent variable

Van Os & Reininghaus (2016)	Review
Van Winkel et al (2013)	Review
Van Winkel, R. (2015)	No mediator data
Velikonja et al (2012)	No mediator data
Velikonja et al (2014)	Insufficient data
Veling et al (2016)	No mediator data
Vogel et al (2006)	Positive symptoms not dependent variable
Vogel et al (2009)	No mediator data
Vogel et al (2011)	No mediator data
Voitenko et al (2013)	No mediator data
Wang et al (2013)	No mediator data
Weber et al (2008)	No mediator data
Weibel et al (2017)	Positive symptoms not dependent variable
Weijers et al (2018)	Positive symptoms not dependent variable
Whitfield et al (2005)	No psychological mediator
Wolff et al (2016)	No psychosis
Wolke et al (2014)	Sample not suitable
Won et al (2014)	Positive symptoms not dependent variable
Woodberry et al (2016)	Review
Yamasaki et al (2016)	Sample not suitable
Yung et al (2015)	No mediator data
Zincir et al (2011)	No mediator data
Zincir et al (2014)	No mediator data

Appendix 11 – Table of mediation model variables and tools for measurement

Study	Trauma measure		Mediators		Mediator measure(s)	Psychosis measure
Appiah-Kusi 2017	CTQ	Continuous	Cognitive schemas	Continuous	BCSS	PSQ, GPTS
Ashford 2010	DIAS (modified)	Continuous	Rejection sensitivity Anxiety, depression Negative beliefs	Continuous	IPSM HADS BCSS	GPTS
Bebbington 2011	3 items from APMS about sexual talk, touching and intercourse	Binary	Anxiety, depression	Continuous	CIS-R	medication, inpatient stay, self-reported diagnosis, one question from PSQ and SCAN
Berenbaum 2008	CTI	Continuous	Dissociation, absorption, PTSD	Continuous	Life Events Checklist; CAPS, Dissociative Processes Scale; Curious Experiences Scale, SCID, DES	Personality Disorder Interview-IV (Schizotypal personality)
Boyda & McFeeters 2015	Items from domestic violence and abuse section of APMS 2007 (sexual abuse - 2 items; emotional neglect 7 items)	Binary	Activities in daily living (social functioning), loneliness	Binary	Composite measure of difficulties with personal care, transport, money and household; single item on loneliness	PSQ
Choi 2015	Korean-CTQ	Continuous	PTSD	Continuous	IES-R-K	Psychoticism scale of the PSY5 factor scale from the MMPI-2

Choi 2017	Korean-CTQ	Continuous	PTSD, dissociation	Continuous	IES-R-K, K-PDEQ	RC6 (ideas of persecution) and RC8 (aberrant experiences) scales of MMPI-2
Cole 2016	CATS	Continuous	Dissociation	Continuous	DES-II, CDS	LSHS-R, PDI
Fisher 2012	CTQ	Binary (by cut-off points)	Anxiety, depression, negative self/other schemas	Binary	BAI, BDI, BCSS	PSQ (2 items)
Gaweda 2018 1	TEC	Continuous	Attachment, cognitive biases, self-disturbances	Continuous	PAM, DACOBS, IPASE	PQ-16
Gaweda 2018 2	TEC	Continuous	Cognitive biases, self-disturbances	Continuous	DACOBS, IPASE	CAPE

Goodall 2015	CTQ	Continuous	Attachment	Continuous	ECR-R	SPQ-B
Hardy, 2016***	THQ	Categorical	PTSD, Schemas; depression	Continuous	Self-report scale for PTSD; BCSS (negative self & negative other subscales); BDI-II	SAPS
Jaya 2016	latent social adversity variable comprised of 14 subscales/items	Continuous	Loneliness, core schemas, social rank	Continuous	UCLA Loneliness Scale v1, BCSS, SCS	CAPE
Lincoln, 2017***	Adapted scale from NEMESIS study	Continuous	Emotion regulation	Continuous	ERSQ	CAPE
Marwaha & Bebbington 2015	APMS 2007 items on sexual abuse	Categorical combined intercourse & touching into 'contact abuse'	Anxiety, depression	Continuous	CIS-R	diagnosis of probable psychosis; ratings of paranoia/hallucinations from PSQ, continuous paranoia score (questions from PSQ and SCID-II)
Marwaha 2013	APMS 2007 items on sexual abuse	Categorical	Mood instability	Binary	SCID-II (1 item)	diagnosis of probable psychosis; ratings of paranoia/hallucinations from PSQ, continuous paranoia score (questions from PSQ and SCID-II)
McDonnell 2018	RBQ	Continuous	Interpersonal sensitivity	Continuous	IPSM	SSPS

Morgan 2013	MRC Sociodemographic schedule	Binary	Self esteem	Continuous	Rosenberg Self-Esteem Scale	SCAN
Osterfjells 2017	CTQ-SF	Binary	Anxiety, depression, metacognitive beliefs	Continuous	PANSS depression/anxiety subscale (3 items); MCQ-30 (MCQ-UD subscale used – negative belief about uncontrollability/danger of thoughts, 6 items)	SCID for DSM-IV; PANSS positive subscale (4items)
Pearce 2017	Brief Betrayal Trauma Survey	Continuous	Dissociation, attachment	Continuous	DES-R, RQ	CAPE (paranoia and hearing voices subscales)
Perona Garcelan 2012	TQ	Continuous	Dissociation	Continuous	DES-II	PANSS
Perona Garcelan 2013	TQ	Continuous	Mindfulness, dissociation	Continuous	TAS, CDS, Southampton Mindfulness Questionnaire	LSHS-R
Pilton 2016	CTQ	Continuous	Attachment	Continuous	PAM	PSYRATS-AH, BAVQ, VAY

Powers 2016	CTQ	Categorical	PTSD	Continuous	CAPS	MINI
Rossler 2016	CTQ-SF	Categorical	Stress sensitivity	Continuous	PSS, PANAS-N, SSCS	SIAPA SPQ-B German version; Paranoia Checklist; SCL-90-R, Subscales for schizotypal signs & schizophrenia nuclear symptoms scale; CEQ
Sheinbaum 2014	CTQ	Continuous	Attachment	Continuous	RQ	CAPE (positive symptom subscale)
Sheinbaum 2015	CECA Interview	Continuous	Attachment , depression	Continuous	ASI, BDI-II	CAARMS, SCID-II
Sitko 2014	Life Event History module of UM-CIDI	Continuous	Depression, attachment	Continuous	Sadness module of UM-CIDI, AAQ	Beliefs & Experiences module of UM-CIDI

Thompson 2016	CTQ-SF	Continuous	Anxiety, depression, dissociation, mood swings, mania	Continuous	HAM-A, HAM-D, CAARMS subscales	CAARMS, BPRS, CASH
van Nierop 2014	Items from NEMESIS-1 – measuring 5 types of trauma	Continuous	Social defeat, affect	Continuous	ten symptoms indexing self-devaluation; 21 general affective symptoms	CIDI interview, endorsement of 1+ lifetime psychotic experiences; SCID-I. Additional questions to rate frequency and severity of symptoms.
Varese 2012	CATS	Continuous	Dissociation	Continuous	DES	PANSS
Wickham & Bentall 2016	CTQ-SF	Continuous	Beliefs in a just world	Continuous	General Beliefs in a Just World Scale; Personal Beliefs in a Just World	PANSS

Trauma: CATS – child abuse and trauma scale; CECA – childhood experience of care and abuse interview; CTI – childhood trauma interview; CTQ (-SF)– childhood trauma questionnaire (short form); DIAS – Direct and indirect aggression scales; MRC Sociodemographic schedule – Medical Research Council sociodemographic schedule; RBQ – retrospective bullying questionnaire; TEC – traumatic events checklist; THQ – trauma history questionnaire; TQ trauma questionnaire; UM-CIDI - University of Michigan composite international diagnostic interview. **Mediators**: AAQ – adult attachment questionnaire; ASI – attachment style interview; BAI – Beck anxiety inventory; BCSS – brief core schema scale; BDI – Beck depression inventory; CAARMS – comprehensive assessment of at risk mental states; CAPS – clinician administered PTSD scale; CDS – Cambridge depersonalisation scale; CIS-R – clinical interview schedule revised; DACOBS – Davos assessment of cognitive biases scale; DES – dissociative experiences scale; ECR-R – experiences in close relationships revised; ERSQ – emotion regulation skills questionnaire; HAM-A – Hamilton anxiety scale; HAM-D – Hamilton depression scale; IES-R-K – impact of events scale revised, Korean version; IPASE – inventory of psychotic-like anomalous experiences; IPSM – interpersonal sensitivity measure; K-PDEQ – peritraumatic dissociation questionnaire – Korean; PAM – psychosis attachment measure; PANAS – positive and negative affect scale; PSS – perceived stress scale; RQ -relationship questionnaire; SCID – structured clinical interview for DSM; SCS – social comparison scale; SSCS – screening scale for chronic stress; TAS – Tellegren absorption scale. **Psychosis**: BAVQ – beliefs about voices questionnaire; BPRS – brief psychiatric rating scale; CAPE – community assessment of psychic experiences; CASH – comprehensive assessment of symptoms and history; CEQ – creative experiences questionnaire; GPTS – Green paranoid thoughts scale; HADS – Hospital anxiety and depression scale; LSHS – Launay-Slade hallucinations scale; MINI – mini international neuropsychiatric interview; MMPI – Minnesota

multiphasic personality inventory; PDI – Peters delusions inventory; PQ – prodromal questionnaire; PSQ – psychosis screening questionnaire; PSYRATS-AH – psychotic symptoms rating scales – auditory hallucinations; SAPS – scales for the assessment of positive symptoms; SCID – structured clinical interview for DSM; SCL-90-R – symptom checklist 90 – revised; SIAPA – structured interview for assessing perceptual abnormalities; SPQ-B schizotypal personality questionnaire – brief; SSPS – state social paranoia scale; VAY – the voice and you

Appendix 12 – Participant information sheet – online study

Information Sheet

Thank you for expressing interest in our study. Please read through the information on this page before deciding whether or not to take part. The survey will take 20-30 minutes to complete.

What is the study about?

This study is interested in a link between negative experiences in childhood and how this can have an effect on people's mental health later in life. We hope to gather information about whether the different ways people manage their emotions may influence their experience of anxious or fearful thoughts. Studying this in the general population will help us to identify patterns which may be useful in the improvement of healthcare for people who experience clinical symptoms.

Who can take part?

Any person who is 18 years old or over, resident in the UK, with a good understanding of written and spoken English can participate in this survey. People who have been diagnosed with issues or illnesses which affect their brain are asked not to participate at this time. This includes brain injury, dementia, autism spectrum disorders and any severe psychiatric conditions (such as schizophrenia, bipolar, major depressive disorder, etc.)

Participation is completely voluntary and you can leave the survey at any time by closing the survey tab. Due to the completely confidential nature of the study, we will be unable to remove any data which has been submitted up until the point of exit, as it will not be identifiable to any particular individual. If you have any questions about the study, before deciding whether or not to take part, please contact the research team at emotioninpsychosisstudy@gmail.com. Any email communication will not be linked to your data if you do decided to participate in the study – your answers will remain completely anonymous and confidential,

What do I have to do?

You will first be asked to complete a short online consent form, to make sure you are satisfied with the information provided for the study, and wish to take part. You will then be asked to complete a set of ten questionnaires about different topics. These will include negative childhood events, your response to these events, your mood and emotions, your relationships and your quality of life. Questions cover a range of negative experiences from the very common to more severe events which may have been very upsetting (e.g. experiences of abuse or neglect). You will not be asked to provide any detail about any events which you do disclose, and all information will be completely confidential and not attributable to any individual.

If you decide to participate, please answer each question as honestly as possible in order for us to gather accurate and meaningful data. We would like you to complete all questions, however, you can choose to not answer any specific question that you find too sensitive, upsetting or inappropriate. We are interested in your own personal experience so there are no right or wrong answers.

Due to the anonymous nature of the survey, it will have to be completed in one sitting. By closing down the browser window before completion of the survey, you are signalling your wish to withdraw. As above, any information which you have submitted up to the point of withdrawing will not be removed as it will not be attached to any specific identifying information.

What will I get for taking part?

As the study is completely anonymous, we are unable to offer any financial incentive for taking part. You will be able to sign up to receive an electronic copy of the results when the study is complete. The email address will not be linked to any of the data you have submitted.

Whilst there is no immediate direct benefit to taking part, you will be contributing to research which will hopefully inform future clinical research and the development of mental health treatments.

What are the possible disadvantages of taking part?

It is possible that the questionnaires may cover topics that are sensitive or difficult for you to think about. If you feel any distress you can stop the questionnaire at any stage. Any feelings of distress will usually disappear quickly, but should you feel unable to manage these, you are encouraged to contact your GP. We will also provide support numbers at the end if you feel you would like to talk to someone about any of the topics covered.

Will my information be kept confidential?

All of your answers will be held anonymously with no identifying information. We will ask for some general demographic information such as age, gender, etc, but we will not ask for any information which would allow the data to be identified as yours (e.g. name, location). All data you provide will be stored on secure password protected servers within the university, only accessible to the research team. There are strict laws which safeguard your privacy at every stage.

E-mail addresses will be stored in a separate database and cannot be linked to questionnaire responses; your data will remain anonymous.

Your anonymised data will be stored for ten years in accordance with Edinburgh Napier University guidelines.

If you have any complaints or concerns about this study, please contact the research team at emotioninpsychosisstudy@gmail.com.

The study is being co-ordinated by **Amanda Woodrow**, PhD candidate in Psychology at Edinburgh Napier University.

The supervisory team are:

Dr Paul Hutton - Associate Professor of Therapeutic Interventions and Lead for Postgraduate Research in the School of Health and Social Care at Edinburgh Napier, and Honorary Consultant Clinical Psychologist at NHS Lothian.

Prof. Thanos Karatzias - Professor of Mental Health and Director of Research at Edinburgh Napier University, and a Clinical & Health Psychologist at the Rivers Centre for Traumatic Stress, Edinburgh.

Dr Mick Fleming – Associate Professor in the School of Health and Social Care at Edinburgh Napier University.

Dr Sean Harper - Consultant Clinical Psychologist and Lead Psychologist for Psychosis and Complex Mental Health in NHS Lothian.

The independent contact at Edinburgh Napier University for this project is Associate Professor **Liz Adamson** who can be reached at l.adamson@napier.ac.uk or on 0131 4455696.

This study has been approved by the Edinburgh Napier University Ethics Committee.

Thank you very much for reading this and for your consideration of this study. Please follow this link to proceed to the survey https://survey.napier.ac.uk/n/zz31b.aspx

This information sheet can be downloaded as a PDF and retained for your records.

Appendix 13 – Participant info sheet – clinical study

Participant Information Sheet

Childhood experiences, emotion regulation and paranoia

You are being invited to take part in a research study. Before you decide whether or not to take part, it is important for you to understand why the research is being done and what it will involve. Please take time to read the following information carefully and talk to others about the study if you wish. Please feel free to contact us if there is anything that is not clear, or if you would like more information.

What is the purpose of the study?

This study hopes to test a new therapy which tries to help people cope better with strong emotions or feelings – this is called 'emotion regulation'. The study will also help us collect information about difficult experiences people may have had in their childhood. We hope to find out whether the things people go through in childhood have an effect on how they cope with their feelings as adults.

Why have I been asked to take part?

You have been asked to take part because:

- you have been diagnosed with a mental health condition called 'psychosis' or 'schizophrenia'.
- you are in contact with NHS Lothian mental health services
- you are worried that people wish to harm you

Do I have to take part?

No, it is up to you whether or not to take part. If you do decide to take part, you will be able to ask the researcher any questions you may have. They will ask you to sign a consent form before the study begins. You are free to withdraw from the study at any time and without giving a reason. Deciding not to take part or withdrawing from the study will not affect the care you receive, or your legal rights.

How do I take part?

There are two ways you can register your interest in the study.

1. You can contact the research team using the email address or contact number given on the posters advertising the study. The researcher will then contact you by phone to discuss the study in more detail and answer any questions you might have.

The researcher will ask you a few questions to make sure you are able to take part, and they will ask your permission to speak to your care team too.

2. A member of your mental health team may ask if you would be interested in taking part. If you agree, they will pass your contact details on to the researcher who will telephone you to discuss the study.

You will always have at least 48 hours to think about whether you would like to take part before the researcher contacts you again.

If we are unable to include you in the study, the researcher will contact you to explain why.

What will happen if I take part?

The researcher will contact you to answer any questions and see whether you would like to take part. They will then arrange a meeting with you. At this meeting they will ask you to sign a consent form.

The first part of the study will involve completing some questionnaires and answering some questions about your experiences. The researcher will ask you some questions about your childhood. They will also you about your current worries, including worries about being harmed. They will also ask you are feeling at the moment (e.g., sad, happy) and how you cope with unpleasant feelings. You do not have to answer any question(s) you do not want to. This will take around 1 to 1.5 hours. You will be able to take as many breaks as you want.

The researcher will then introduce you to a mobile phone 'app', which will be used to record your experiences over a 6-day period. This mobile phone app is designed to send you some brief questions, several times each day. Answering these questions by text or email takes about 2 minutes each time. This is called 'experience sampling' and will allow us to understand how your experiences change during the day.

After these 6 days, you will be invited to attend four group meetings. Other people with similar difficulties will also be invited to these. Each meeting lasts about 75 minutes. These will be delivered by trained staff and will be run on a drop-in basis once per week at The Hive. This is an activity centre based at the Royal Edinburgh Hospital. There are two types of group. One will discuss ways in which you can cope better with your feelings. The other will teach you arts and crafts skills.

The group you end up in will be decided 'randomly' by a computer. This means we cannot choose which group you will be able to join. This is important for finding out how helpful the groups are.

Once the groups are finished, we will ask you to use the mobile phone app for a further 6-days. This will help us find out whether the group has helped you or not. After that, we will invite you back to meet with the researcher and complete some final questionnaires.

What are the possible benefits of taking part?

Taking part will help us find out if the groups are helpful to people with similar problems to you.

Other similar research has found that people who take part in the workshops about feelings have learned new skills and have experienced improvements in how they deal with strong emotions. We do not know for sure that the workshop 'causes' these changes, but this is what we are trying to investigate in this study.

Participants in the arts & crafts group will learn potentially useful practical and creative skills.

What are the possible disadvantages and risks of taking part?

You will be asked about difficult experiences from your past. You may find it upsetting to think about these. The researcher will check how you are feeling throughout the meeting. Extra time is scheduled so you are able to take breaks at any time.

The questionnaires we will use do not usually cause people to become upset or distressed. But if you do require any extra support, the researcher will stay with you and contact your care team.

You may find the mobile phone app difficult to use or time-consuming. The research team will support you to use it. The surveys are very short and you can turn off the app when you need to – e.g., if you are driving.

Will my taking part in the study be kept confidential?

We will let your GP and your care team know that you are taking part. However everything else you tell us will be confidential, unless we are worried about your safety or the safety of someone else. If this happens, then normally we would contact your key worker or care team to let them know what we are worried about. The researcher will always aim to let you know if they decide they need to do this. They will always explain the reasons for their decision, and who they will share the information with.

What will happen to my data?

Your name and contact details will be stored securely in a locked filing cabinet at the Royal Edinburgh Hospital. None of your personal information will be removed from hospital. The other information you give us (e.g., your answers to the questionnaires) will be stored separately at Edinburgh Napier University. It will not have your name on it, or any other details about who you are.

You are able to leave study at any time. If you choose to stop taking part, this will have no effect on your ongoing medical care.

If you decide to leave during the study, you can ask for any information you have given us to be deleted. You don't need to give us a reason for this. However once the study is finished and the results are published, we won't be able to remove any information you have given us.

Sometimes people can find it difficult to understand information and make decisions. Occasionally this can mean they are no longer able to make their own decisions about taking part in research. If this happens to you, you won't be able to continue taking part in the study. However if your ability to decide to take part improves and the study is still running, then we will ask you if you want to re-join. We will ask you complete the consent form again. If your ability to make decisions doesn't improve by the time the study is finished, then all the information you have given us will be deleted.

What will happen to the results of the study?

The results will form part of a long document called a 'doctoral thesis'. The thesis will be reviewed by Edinburgh Napier University. The results will also be included in academic papers which are published in scientific journals.

We will send you a summary of findings from the study, if you tell us you would like this.

Who is doing this study?

The researcher is called Amanda Woodrow. She is a PhD candidate at Edinburgh Napier University. This study is part of a series of studies she is doing for her PhD.

Amanda is being supervised Dr Paul Hutton who is an Honorary Consultant Clinical Psychologist with NHS Lothian and an Associate Professor in Therapeutic Interventions at

Edinburgh Napier University. She is also being supervised by Dr Sean Harper who works as a Consultant Clinical Psychologist and is the Lead Psychologist for Psychosis and Complex Mental Health in NHS Lothian.

What if there is a problem?

If you have a concern about any aspect of this study please contact Amanda Woodrow at amanda.woodrow@napier.ac.uk or 07425900165. She will do her best to answer your questions.

In the unlikely event that something goes wrong and you are harmed during the research and this is due to someone's negligence then you may have grounds for legal action for compensation against NHS Lothian but you may have to pay your legal costs. The normal National Health Service complaints mechanisms will still be available to you (if appropriate).

Who is organising the research?

The research has been designed and is being carried out by Amanda Woodrow, who is a PhD candidate at Edinburgh Napier University. The study is being supervised by Dr Paul Hutton (Honorary Consultant Clinical Psychologist, NHS Lothian and Associate Professor in Therapeutic Interventions, Edinburgh Napier University) and Dr Sean Harper (Consultant Clinical Psychologist and Lead Psychologist for Psychosis and Complex Mental Health, NHS Lothian). Funding and sponsorship has been provided by Edinburgh Napier University.

Who has reviewed the research?

The study has been reviewed by representatives from Edinburgh Napier University and NHS Lothian. All research in the NHS is looked at by an independent group of people, called a Research Ethics Committee. A favourable ethical opinion has been obtained (IRAS registration number 229624). NHS management approval has also been obtained.

Who can I contact if I have a complaint?

You are free to discuss any concerns about the study with the researcher (contact details at the end of this leaflet) who will do her best to address your concerns. If you remain unhappy and wish to complain formally, you can do this by contacting:

NHS Lothian Complaints Team

2nd Floor, Waverley Gate, 2-4 Waterloo Place

Edinburgh

EH1 3EG

Tel: 0131 536 3370

Email: feedback@nhslothian.scot.nhs.uk

Who can I contact about this study?

If you would like any further information about the study or think you might like to take part, please contact the researcher:

Amanda Woodrow, Doctoral Candidate, Edinburgh Napier University

Email [redacted]

Tel: [redacted]

If you would prefer, you can ask a member of your care team to contact the researcher on your behalf.

If you wish, you can also contact the supervisors of the study, Dr Paul Hutton or Dr Sean Harper at:

[redacted]

[redacted]

If you would like to discuss this study with someone independent of the research team, please contact:

Dr Maria Truesdale a [redacted]

Thank you for taking the time to read this information sheet.

Appendix 14 – Consent from – clinical study

CONSENT FORM

Childhood experiences; emotion regulation and paranoia

Participant ID:
Centre ID:

Amanda Woodrow (PhD Student)
Email: [redacted] Tel: 0[redacted]

Dr Paul Hutton (Academic Supervisor)
Email: [redacted]
Tel: [redacted]

Dr Sean Harper (NHS Supervisor)
[redacted]
Tel: [redacted]

Please initial each box

1. I confirm that I have received, read and understood the information sheet for the above study, and have had the opportunity to consider the information, ask questions and have these answered satisfactorily. ☐

2. I understand that my participation is voluntary and that I am free to withdraw at any time, and I may ask for my data to be removed without giving a reason, and without my medical care or legal rights being affected. ☐

3. I understand that all the information I provide in the study will be anonymous and confidential. I understand however that if I reveal information about possible harm to myself, or others, this information will be passed on to a member of my current care team. This will most likely be my keyworker/ care-coordinator. ☐

4. I agree that my anonymised data can be stored securely on a password protected database, on a password protected computer on NHS and/or Napier University premises. ☐

5. I agree to my Mental Health Care Team being informed of my participation in the study. ☐

6. I agree to my GP being informed of my participation in the study. ☐

7. I understand that the information collected about me will be used to support other research in the future, and may be shared anonymously with other researchers. ☐

8. I understand that if I lose the capacity to consent to research, my data will be retained until the end date of the study. I understand that if I regain the capacity to consent before the end of the study, another consent form will be completed and my data will be used. If I do not regain capacity before the end of the study, my data will be withdrawn. ☐

9. I agree to take part in the above study. ☐

_____	_____	_____
Name of Participant	Date	Signature

_____	_____	_____
Name of Person taking consent	Date	Signature

Appendix 15 – Screening tool for clinical study

Brief Participant Screening Questions:

Screening will take place over the phone to determine participant eligibility before they attend. Participants must answer at least one 'yes' from each section to be deemed eligible for inclusion.

From Carvalho et al. (2015) General Paranoia Scale for Adults (GPS) Answer Yes/No

- I often wonder what hidden reason another person may have for doing something nice for you.
- Someone has it in for me
- It is safer to trust no-one
- I have often felt that strangers are looking at me critically
- I sometimes feel as if I am being followed
- I tend to be on my guard with people who are more friendly than I expected

From Gross & John (2003) Emotion Regulation Questionnaire (ERQ) [Suppression questions]

Answer Yes/No

- When I am feeling negative emotions, I make sure not to express them.
- When I am feeling positive emotions, I am careful not to express them
- I control my emotions by not expressing them.
- I keep my emotions to myself.

Appendix 16 – Amendments to protocol – clinical study

A number of amendments were submitted to the NHS SES REC and NHS R&D following protocol registration. Changes were implemented only after favourable opinions had been received. Substantial changes are discussed in the methodology chapter, and further minor changes are described in turn below.

Electronic data protection

Following submission to the Caldicott Guardian for review, several additional documents were requested, including a data management plan and data transfer contract between Edinburgh Napier University and the app provider. The majority of these additions did not affect the protocol, but an App Information Sheet had to be generated to be transparent about the relative data risks associated with participants opting to use their own mobile phone handset to complete the app-based surveys. Security risk is still low, but it was not possible to guarantee ongoing privacy of their device IP address or participant number as these would be transferred to the app central storage outside of the UK (University of Maastricht, Netherlands). None of their personal identification details (name, age, sex, etc) would be linked to the data regardless of the device used. The document clarified minimal data security risks and thus was acceptable to the REC.

Additional changes

The SES REC requested that we add some form of compensation for participant time and commitment. This was due to be delivered as £5 at both the pre and post measurement meetings, however University regulations do not allow cash to be offered. This was revised to a single £10 value supermarket voucher which would be distributed in the final participant meetings.

Due to a five month delay awaiting Caldicott Guardian approval, a request was made to extend the end date of the study recruitment period by four months.

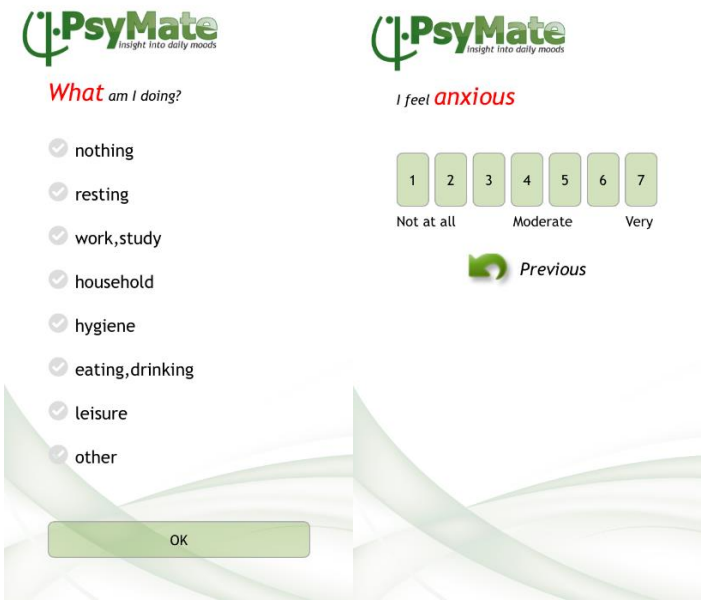
Appendix 17 – ESM questionnaire from clinical study

PsyMate questions and origins

Question	Measure	Subscale	Actual wording
Right now I feel suspicious			
Right now I feel safe			
Right now I feel that others dislike me	GPTS B Q1	Persecutory delusions	Certain individuals have had it in for me
Right now I feel that others might hurt me	GPTS B Q7	Persecutory delusions	I was sure someone wanted to hurt me
Since the last alarm I have thought that others are trying to cause me harm	GPTS B Q3	Persecutory delusions	People have intended me harm
This was distressing	GPTS B Q8	Distress total	I was distressed by people wanting to harm me in some way
Since the last alarm I have thought that others are plotting against me	GPTS B Q6	Persecutory delusions	I was convinced there was a conspiracy against me
This was distressing	GPTS B Q11	Distress total	I was distressed by being persecuted
Since the last alarm I have thought that something strange is going on	GPTS A Q7	Delusions of reference	I believed that certain people are not what they seemed
This was distressing	GPTS A Q15	Distress total	I was worried by people's undue interest in me
To cope with things I thought about something more positive	CERQ Q22	Positive	
To cope with things I tried not to think about it		Negative	
I thought about it in a way that helped me to stay calm		Positive	
I thought about how negative the situation was	CERQ Q35	Negative	
I thought about how I can best cope with the situation	CERQ Q14	Positive	
I kept my feelings to myself		Negative	
I noticed my feelings and carried on		Positive	
I am to blame for the situation	CERQ Q1	Negative	
Others are to blame for the situation	CERQ Q9	Negative	
I feel powerless to change the situation	CERQ Q20	Negative	
I am in control of my emotions		Positive	

I can change my mindset by thinking positively	CERQ Q33	Positive	
When I try not to think about my emotions they get stronger		Negative	
Right now I am (doing) Nothing Resting Work, study Household Hygiene Eating, drinking Leisure Other			
I am with… No-one Partner Friends Strangers Parents Family members Colleagues Acquaintances			
I am (where) Home Work Hospital Public place outdoors Public place indoors On transport Other			

Note – select items are adapted from items in the GPTS paranoia and CERQ emotion regulation questionnaires. These items are used to construct the 'pseudo ESM' data points for the revised analysis plan.



Screens from the PsyMate app questionnaire

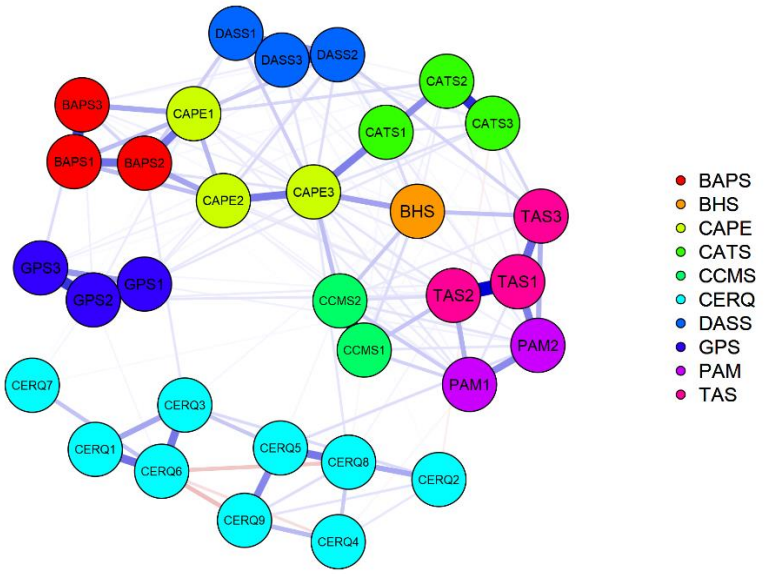
Appendix 18 – Missing data from exploratory and confirmatory samples

			Exploratory sample			Confirmatory sample		
Measure	**Subscale**	**No of items**	**No of respondents**	**skew**	**kurtosis**	**No of respondents**	**skew**	**kurtosis**
CERQ	Self blame	2	190	0.954	0.314	196	0.816	0.04
	Acceptance	2	190	0.059	-0.975	196	0.234	-1.073
	Rumination	2	190	0.242	-0.712	196	0.385	-0.731
	Positive refocus	2	190	0.649	0.012	196	1.044	0.611
	Planning	2	190	0.096	-0.687	196	0.061	-0.316
	Catastrophising	2	190	0.847	0.176	196	0.835	-0.051
	Other blame	2	190	1.401	3.314	196	1.628	3.813
	Positive reappraisal	2	190	0.207	-1.088	196	0.031	-0.806
	Put into perspective	2	190	0.15	0.764	196	0.234	-0.466
GPS	Distrust	8	182	0.255	-0.518	191	0.701	0.106
	Persecutory. Ideas	8	182	0.81	0.981	191	0.904	0.537
	Self deprecation	4	182	0.834	0.918	191	1.066	0.433
CATS	Sexual abuse	6	177	4.069	18.834	192	4.02	19.026
	Negative home enviro	14	177	1.31	1.666	192	1.19	0.903
	Punishment	6	177	0.412	0.076	192	0.394	-0.173
TAS	Describe feelings	5	169	0.164	-1.024	196	0.315	-0.884
	Identify feelings	7	169	0.89	0.123	196	0.607	-0.639
	External thinking	8	169	-0.052	-0.371	196	0.284	-0.323
PAM	Anxious	8	169	0.475	-0.859	196	0.572	-0.344
	Avoidant	8	169	-0.212	-1.157	196	-0.048	-0.48
CCMS	Psychological abuse	3	169	1.634	2.252	196	1.334	1.514
	Neglect	3	169	3.096	9.667	196	3.498	13.753
BHS	n/a	5	169	1.858	4.231	191	1.607	2.14
DASS	Depression	7	171	1.16	2.665	196	1.334	1.514
	Anxiety	7	171	1.735	3.618	196	1.835	4.185
	Stress	7	171	0.991	0.683	196	0.949	1.055
CAPE	Persecutory ideation	5	172	1.653	4.564	196	1.35	1.927
	Perceptual abnormality	7	172	4.046	17.522	196	3.164	13.261
	Bizarre experiences	3	172	6.028	39.013	196	6.11	45.246
BAPS	Survival strategy	6	174	1.075	0.418	194	1.039	0.583
	Negative beliefs	6	174	1.503	1.891	194	1.729	3.168
	Normalising beliefs	6	174	0.478	-0.015	194	0.565	-0.001

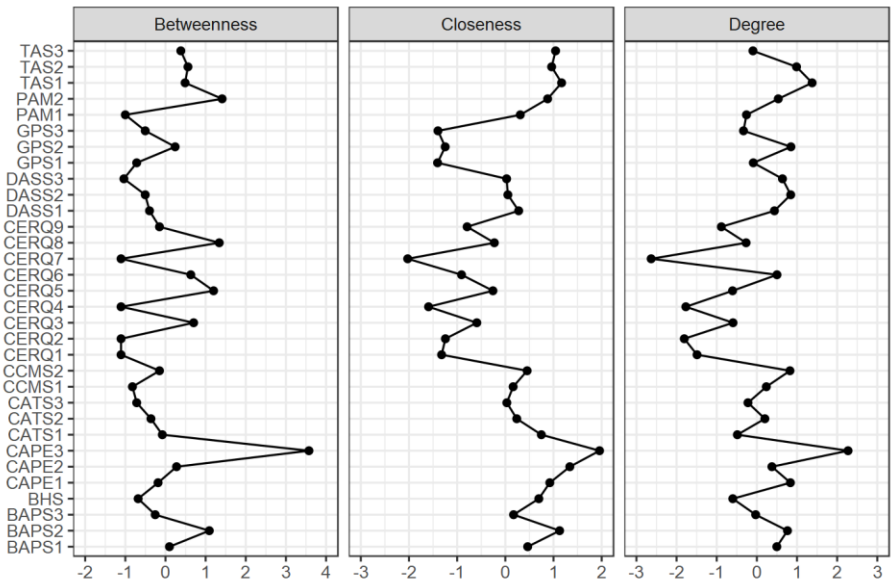
Appendix 19 – Network analysis information

Table of variable codes for network nodes

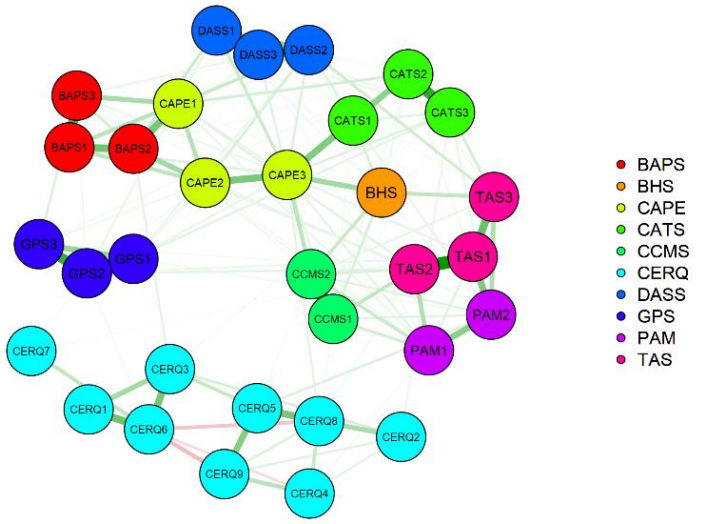
Variable code	Variable
BAPS1	Survival strategy (positive beliefs)
BAPS2	Negative beliefs about paranoia
BAPS3	Normalising beliefs
CAPE1	Persecutory ideation
CAPE2	Perceptual abnormality
CAPE3	Bizarre experiences
DASS1	Depression
DASS2	Anxiety
DASS3	Stress
BHS	Hypervigilance
CCMS1	Psychological abuse
CCMS2	Neglect
PAM1	Anxious attachment
PAM2	Avoidant attachment
TAS1	Difficulty describing feelings
TAS2	Difficulty identifying feelings
TAS3	Externally oriented thinking
CATS1	Sexual abuse
CATS2	Negative home environment
CATS3	Punishment
GPS1	Distrust
GPS2	Persecutory ideas
GPS3	Self-deprecation
CERQ1	Self-blame
CERQ2	Acceptance
CERQ3	Rumination
CERQ4	Positive refocusing
CERQ5	Refocus on planning
CERQ6	Catastrophising
CERQ7	Other blame
CERQ8	Positive reappraisal
CERQ9	Putting into perspective



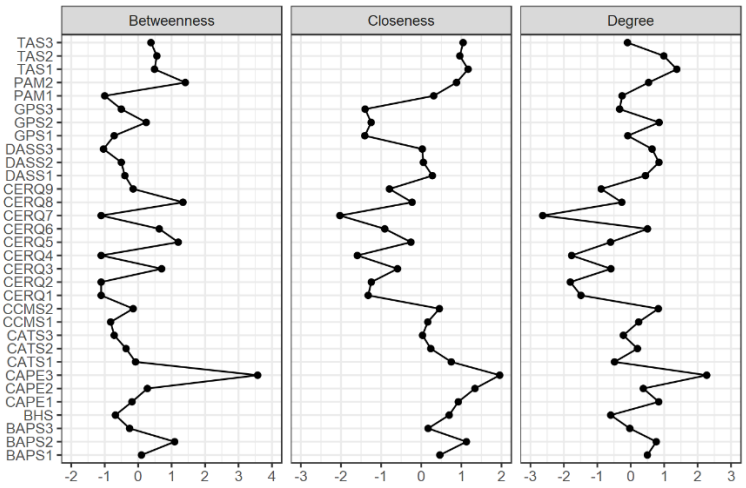
Network 1 – Raw data



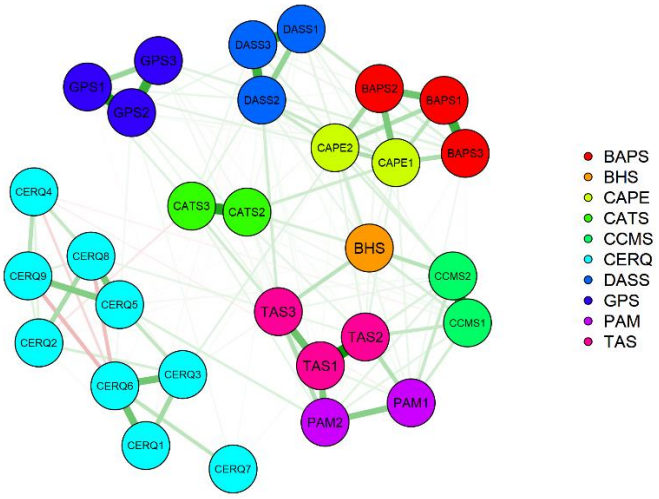
Centrality plot for Network 1



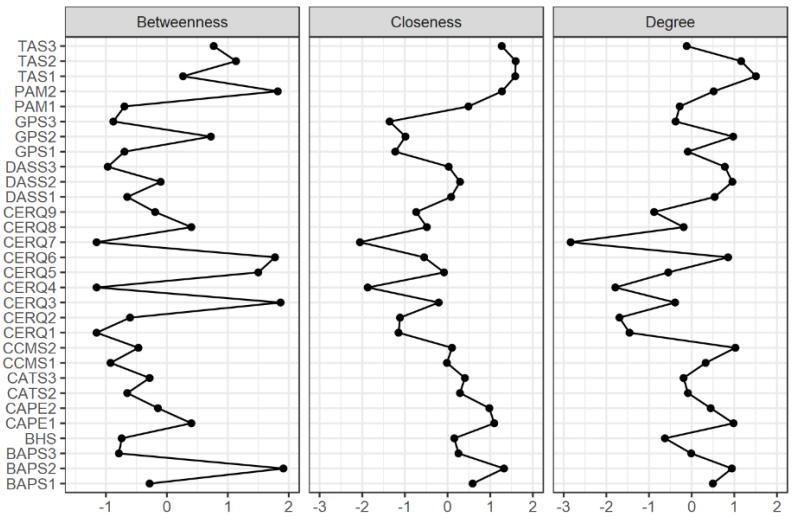
Network 2 (5000 bootstraps)



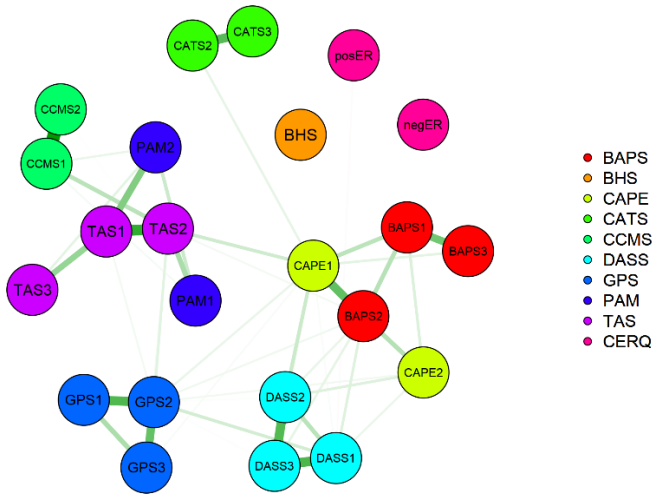
Centrality Plot for Network 2



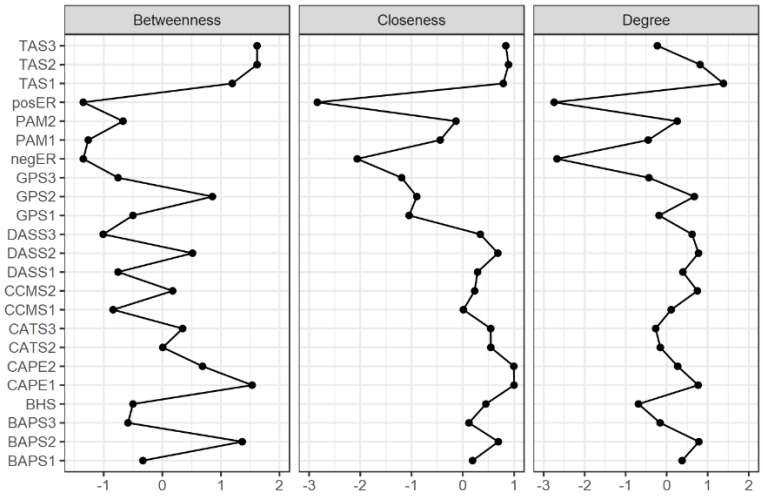
Network 3 (5000 bootstraps)



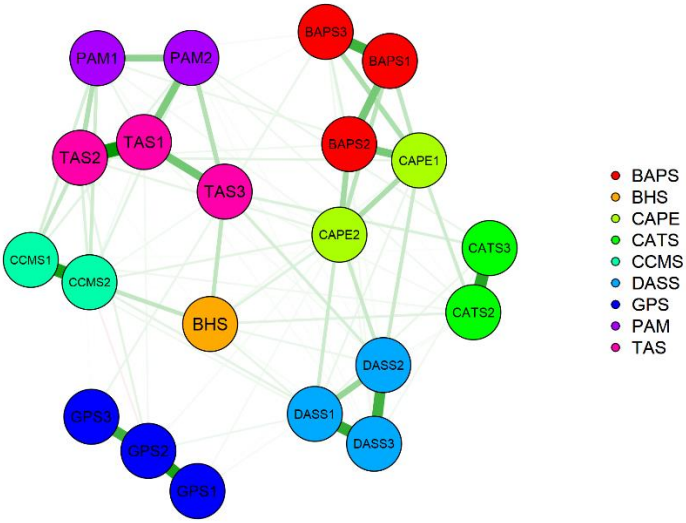
Centrality plot for Network 3



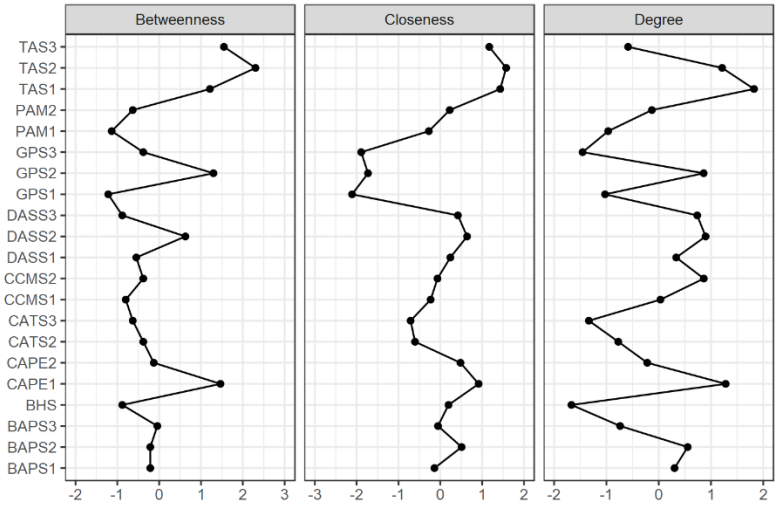
Network 4a (5000 bootstraps)



Centrality plot for Model 4a



Network 4b (5000 bootstraps)



Centrality plot for Model 4b

Appendix 20 – Results of mediation models (group effects)

This table contains the results of all mediation analyses using group allocation as the independent variable, including non-significant models and sensitivity analyses.

Model		Estimate (SE)	95% confidence bounds	p-value	Standardised effect	Completer data only Estimate (SE)	Completer data only 95% confidence intervals	Completer data only std effect size (p-value)
Negative emotion regulation (CERQ) and paranoia (GPTS-persecution)	a-path	0.134 (4.227)	-8.150, 8.418	0.975	0.006	-0.366 (4.527)	-9.209, 8.537	-0.014 (0.941)
	b-path	0.125 (0.267)	-0.399, 0.649	0.639	0.095	0.125 (0.237)	-0.338, 0.589	0.096 (0.596)
	Indirect effect	0.017 (0.537)	-1.036, 1.070	0.975	0.001	-0.042 (0.573)	-1.165, 1.080	-0.001 (0.941)
	Direct effect	-11.447 (5.027)	-21.300, -1.593	**0.023**	-0.390	-11.447 (5.614)	-22.450, -0.444	-0.383 (0.041)
	1000 Bootstrap indirect effect					-0.042 (1.657)	-4.742, 2.550	-0.001 (0.980)
Negative emotion regulation (CERQ) and paranoia (GPTS-reference)	a-path	0.134 (4.227)	-8.150, 8.418	0.975	0.006	0.134 (4.307)	-8.307, 8.576	0.006 (0.975)
	b-path	0.441 (0.207)	0.035, 0.846	**0.033**	0.413	0.441 (0.202)	0.044, 0.837	0.240 (**0.029**)

	Indirect effect	0.059 (1.863)	-3.592, 3.710	0.975	0.002	0.059 (1.898)	03.660, 3.778	0.002 (0.975)
	Direct effect	-1.119 (4.742)	-10.413, 8.175	0.813	-0.045	-1.119 (4.763)	-10.455, 8.217	-0.045 (0.814)
	1000 Bootstrap indirect effect					0.059 (2.247)	-4.040, 5.771	0.002 (0.979)
Negative emotion regulation (CERQ) and paranoia (GPTS-total)	a-path	0.134 (4.227)	-8.150, 8.418	0.975	0.006	-0.336 (4.527)	-9.209, 8.536	-0.014 (0.941)
	b-path	0.506 (0.441)	-0.358, 1.370	0.251	0.240	0.506 (0.393)	-0.264, 1.275	0.239 (0.198)
	Indirect effect	0.068 (2.148)	-4.142, 4.278	0.975	0.001	-0.170 (2.294)	-4.665, 4.325	-0.003 (0.941)
	Direct effect	-14.478 (8.749)	-31.625, 2.669	0.098	-0.303	-14.478 (9.320)	-32.744, 3.789	-0.295 (0.120)
	1000 Bootstrap indirect effect					-0.170 (3.428)	-7.927, 6.749	-0.003 (0.960)
Positive emotion regulation (CERQ) and paranoia (GPTS-persecution)	a-path	-3.195 (4.373)	-11.765, 5.376	0.465	-0.113	-2.919 (5.260)	-13.227, 7.390	-0.104 (0.579)
	b-path	-0.452 (0.170)	-0.786, -0.118.	**0.008**	-0.401	-0.452 (0.171)	-0.788, -0.116	-0.406 (**0.008**)
	Indirect effect	1.444 (2.230)	-2.926, 5.814	0.517	0.045	1.319 (2.429)	-3.442, 6.081	0.042 (0.587)
	Direct effect	-13.560 (4.610)	-22.595, -4.524	**0.003**	-0.457	-13.560 (4.897)	-23.158, -3.961	-0.446 (**0.006**)

	1000 Bootstrap indirect effect					1.319 (2.573)	-1.885, 9.326	0.041 (0.608)
Positive emotion regulation (CERQ) and paranoia (GPTS-reference)	a-path	-3.195 (4.373)	-11.765, 5.376	0.465	-0.112	-3.195 (5.098)	-13.188, 6.798	-0.110 (0.531)
	b-path	-0.341 (0.131)	-0.597, -0.084	**0.009**	-0.372	-0.341 (0.168)	-0.670, -0.011	-0.376 (**0.043**)
	Indirect effect	1.088 (1.691)	-2.226, 4.403	0.520	0.042	1.088 (1.818)	-2.475, 4.652	0.041 (0.549)
	Direct effect	-3.200 (4.865)	-12.734, 6.335	0.511	-0.122	-3.200 (4.955)	-12.910, 6.511	-0.122 (0.518)
	1000 Bootstrap indirect effect					1.088 (1.807)	-1.795, 5.703	0.041 (0.547)
Positive emotion regulation (CERQ) and paranoia (GPTS-total)	a-path	-3.195 (4.373)	-11.765, 5.376	0.465	-0.113	-2.919 (5.288)	-13.283, 7.445	-0.102 (0.581)
	b-path	-0.738 (0.284)	-1.294, -0.181	**0.009**	-0.413	-0.738 (0.297)	-1.321, -0.155	-0.403 (**0.013**)
	Indirect effect	2.357 (3.696)	-4.887, 9.601	0.524	0.047	2.153 (3.997)	-5.680, 9.987	0.041 (0.590)
	Direct effect	-18.161 (8.387)	-34.600, -1.722	**0.030**	-0.359	-18.161 (8.813)	-35.434, --0.889	-0.347 **(0.039)**
	1000 Bootstrap indirect effect					2.153 (4.527)	-3.623, 15.396	0.041 (0.634)
Normalising beliefs (BAPS) and paranoia (GPTS-persecution)	a-path	5.573 (1.446)	2.739, 8.407	**0.000**	0.636	6.224 (1.397)	3.485, 8.963	0.717 (**0.000**)
	b-path	2.570 (0.809)	0.984, 4.157	**0.001**	0.704	2.570 (0.707)	1.185, 3.956	0.716 (**0.000**)
	Indirect effect	14.325 (7.001)	0.604, 28.046	**0.041**	0.447	15.999 (5.679)	4.868, 27.129	0.514 (**0.005**)
	Direct effect	-27.576 (6.642)	-40.594, -14.557	**0.000**	-0.895	-27.576 (6.146)	-39.622, -15.529	-0.923 (**0.000**)

	1000 Bootstrap indirect effect					15.999 (9.055)	2.778, 39.588	0.514 (0.077)
Normalising beliefs (BAPS) and paranoia (GPTS-reference)	a-path	5.573 (1.446)	2.739, 8.407	**0.000**	0.635	5.573 (1.486)	2.661, 8.485	0.642 (**0.000**)
	b-path	1.057 (0.926)	-0.757, 2.872	0.253	0.364	1.057 (0.742)	-0.397, 2.512	0.358 (0.154)
	Indirect effect	5.893 (5.825)	-5.523, 17.309	0.312	0.231	5.893 (4.425)	-2.779, 14.566	0.230 (0.183)
	Direct effect	-6.880 (8.668)	-23.896, 10.109	0.427	-0.270	-6.880 (6.477)	-19.575, 5.815	-0.268 (0.288)
	1000 Bootstrap indirect effect					5.893 (6.918)	-3.773, 25.498	0.230 (0.394)
Normalising beliefs (BAPS) and paranoia (GPTS-total)	a-path	5.573 (1.446)	2.739, 8.407	**0.000**	0.636	6.224 (1.397)	3.485, 8.963	0.717 (**0.000**)
	b-path	4.655 (1.203)	2.297, 7.014	**0.000**	0.776	4.655 (1.161)	2.380, 6.931	0.788 (**0.000**)
	Indirect effect	25.943 (11.388)	3.622, 48.264	**0.023**	0.493	28.974 (9.723)	9.917, 48.031	0.565 (**0.003**)
	Direct effect	-43.621 (10.124)	-63.465, -23.778	**0.000**	-0.829	-43.621 (10.203)	-63.618, -23.625	-0.851 (**0.000**)
	1000 Bootstrap indirect effect					28.974 (13.919)	7.128, 61.499	0.565 (**0.037**)
Survival strategy (BAPS) and paranoia (GPTS-persecution)	a-path	4.971 (1.313)	2.398, 7.544	**0.000**	0.513	5.085 (1.456)	2.231, 7.939	0.535 (**0.000**)
	b-path	1.688 (0.568)	0.575, 2.801	**0.003**	0.568	1.688 (0.591)	0.530, 2.847	0.540 (**0.004**)
	Indirect effect	8.392 (4.164)	0.231, 16.553	**0.044**	0.280	8.585 (3.883)	0.975, 16.195	0.289 (**0.027**)
	Direct effect	-19.820 (5.129)	-29.873, -9.768	**0.000**	-0.670	-19.820 (5.294)	-30.198, -9.443	-0.681 (**0.000**)

	1000 Bootstrap indirect effect					8.585 (4.980)	0.837, 19.747	0.289 (0.085)
Survival strategy (BAPS) and paranoia (GPTS-reference)	a-path	4.971 (1.313)	2.398, 7.544	**0.000**	0.528	4.971 (1.417)	2.193, 7.748	0.580 (**0.000**)
	b-path	1.784 (0.579)	0.648, 2.919	**0.002**	0.669	1.784 (0.539)	0.728, 2.840	0.632 (**0.001**)
	Indirect effect	8.866 (3.223)	2.549, 15.184	**0.006**	0.353	8.866 (3.682)	1.649, 16.084	0.366 (**0.016**)
	Direct effect	-8.343 (5.702)	-19.519, 2.834	0.143	-0.332	-8.343 (4.633)	-17.423, 0.738	-0.345 (0.072)
	1000 Bootstrap indirect effect					8.866 (4.255)	2.564, 21.449	0.366 (**0.037**)
Survival strategy (BAPS) and paranoia (GPTS-total)	a-path	4.971 (1.313)	2.398, 7.544	**0.000**	0.510	5.085 (1.462)	2.219, 7.951	0.541 (**0.001**)
	b-path	4.051 (0.787)	2.509, 5.594	**0.000**	0.786	4.051 (0.860)	2.367, 5.736	0.775 (**0.000**)
	Indirect effect	20.137 (7.105)	6.212, 34.062	**0.005**	0.401	20.601 (7.362)	6.172, 35.030	0.419 (**0.005**)
	Direct effect	-34.230 (7.708)	-49.337, -19.123	**0.000**	-0.681	-34.230 (8.118)	-50.142, -18.319	-0.697 (**0.000**)
	1000 Bootstrap indirect effect					20.601 (9.221)	7.296, 43.862	0.419 (**0.025**)

Appendix 21 – Results of mediation models – emotion regulation

This table includes the results of the mediation models testing whether emotion regulation mediated between trauma and paranoia scores. Sensitivity analysis results are included.

Mediation models – Childhood trauma total as independent variable. Pre-intervention scores.

Predictor		FIML data set b (SE)	p-value	Std B	Completer data b (SE)	p-value	Std B
Control variables	Age (baseline)	1.497 (2.386)	0.530	0.098	1.497 (2.471)	0.606	0.098
	Gender (fixed)	4.872 (4.077)	0.232	0.167	4.872 (4.893)	0.996	0.165
	Education (baseline)	0.205 (1.212)	0.866	0.027	0.205 (1.368)	0.150	0.027
Effects	Trauma (CATS – total) – Emotion regulation (CERQ – negative)	0.133 (0.096) 95%CI (-0.056, 0.322)	0.167	0.349	0.133 (0.070) 95%CI (-0.004, 0.270)	0.058	0.349
	Emotion regulation (CERQ-negative) – Paranoia (GPTS – persecution)	-0.038 (0.197) 95% CI (-0.423, 0.348)	0.848	-0.023	-0.038 (0.265) 95%CI (-0.556, 0.481)	0.887	-0.023
	Trauma (CATS-total) – Paranoia (GPTS-persecution)	0.372 (0.077) 95%CI (0.222, 0.523)	**0.000**	0.608	0.372 (0.108) 95%CI (0.161, 0.584)	**0.001**	0.604
Indirect effects	Total indirect effect	-0.005 (0.025) 95%CI (-0.055, 0.045)	0.843	-0.008	0.005 (0.035) 95%CI (-0.074, 0.064)	0.887	-0.008

Predictor		FIML data set b (SE)	p-value	Std B	Completer data b (SE)	p-value	Std B
	Bootstrap indirect mediation effect (completer data only)	-	-	-	-0.005 (0.058) 95% CI (-0.154, 0.081)	0.931	-0.008

Mediation models – Childhood trauma total as independent variable. Post-intervention scores.

Predictor		FIML data set b (SE)	p-value	Std B	Completer data b (SE)	p-value	Std B
Control variables	Age (baseline)	0.497 (1.814)	0.784	0.041	0.497 (2.269)	0.827	0.041
	Gender (fixed)	-5.054 (2.690)	0.060	-0.217	-5.054 (5.735)	0.378	-0.217
	Education (baseline)	-1.119 (0.861)	0.194	-0.178	-1.119 (1.286)	0.384	-0.178
	Treatment allocation	-6.404 (5.797)	0.269	-0.221	-6.404 (7.026)	0.362	-0.221
	Baseline emotion regulation (CERQ - negative)	-0.323 (0.329)	0.327	-0.257	-0.323 (0.284)	0.256	-0.257
	Baseline paranoia (GPTS – persecution)	0.333 (0.199)	0.093	0.422	0.333 (0.196)	0.088	0.422
Effects	Trauma (CATS-total) – Emotion regulation (CERQ – negative)	0.097 (0.070) 95%CI (-0.041, 0.236)	0.167	0.250	0.097 (0.069) 95%CI (-0.039, 0.234)	0.161	0.250

Predictor		FIML data set b (SE)	p-value	Std B	Completer data b (SE)	p-value	Std B
	Emotion regulation (CERQ-negative) – Paranoia (GPTS – persecution)	0.166 (0.329) 95%CI (-0.480, 0.812)	0.615	0.133	0.166 (0.302) 95%CI (-0.425, 0.757)	0.582	0.133
	Trauma (CATS-total) – Paranoia (GPTS-persecution)	0.045 (0.143) 95%CI (-0.235, 0.324)	0.753	0.092	0.045 (0.135) 95% CI (-0.220, 0.310)	0.740	0.092
Indirect effects	Total indirect effect	0.016 (0.028) 95%CI (-0.039, 0.071)	0.563	0.034	0.016 (0.032) 95%CI (-0.046, 0.078)	0.609	0.033
	Bootstrap indirect mediation effect (completer data only)	-	-	-	0.016 (0.128) 95%CI (-0.131, 0.224)	0.902	0.034

Mediation models – Childhood neglect as independent variable. Pre intervention scores.

Predictor		FIML data set b (SE)	p-value	Std B	Completer data b (SE)	p-value	Std B
Control variables	Age (baseline)	-0.341 (2.569)	0.894	-0.022	-0.341 (2.798)	0.903	-0.022
	Gender (fixed)	5.900 (4.736)	0.213	0.200	5.900 (5.564)	0.289	0.200

Predictor		FIML data set b (SE)	p-value	Std B	Completer data b (SE)	p-value	Std B
	Education (baseline)	0.053 (1.466)	0.971	0.007	0.053 (1.630)	0.974	0.007
Effects	Trauma (CCMS-neglect) – Emotion regulation (CERQ – negative)	-0.471 (0.663) 95%CI (-1.769, 0.828)	0.478	-0.143	-0.471 (0.639) 95%CI (-1.723, 0.782)	0.461	-0.143
	Emotion regulation (CERQ-negative) – Paranoia (GPTS – persecution)	0.368 (0.243) 95%CI (-0.109, 0.844)	0.131	0.228	0.368 (0.306) 95%CI (-0.231, 0.966)	0.229	0.228
	Trauma (CCMS-neglect) – Paranoia (GPTS-persecution)	2.148 (0.991) 95%CI (0.206, 4.090)	**0.030**	0.404	2.148 (1.076) 95%CI (0.040, 4.256)	**0.046**	0.404
Indirect effects	Total indirect effect	-0.173 (0.251) 95%CI (-0.665, 0.319)	0.491	-0.033	-0.173 (0.275) 95% CI (-0.713, 0.367)	0.530	-0.033
	Bootstrap indirect mediation effect (completer data only)	-	-	-	-0.173 (0.408) 95% CI (-1.522, 0.373)	0.672	-0.033

Mediation models – Childhood neglect as independent variable. Post-intervention scores.

Predictor		FIML data set b (SE)	p-value	Std B	Completer data b (SE)	p-value	Std B
Control variables	Age (baseline)	-1.405 (1.953)	0.470	-0.119	-1.405 (2.211)	0.525	-0.119
	Gender (fixed)	-10.171 (3.818)	**0.008**	-0.446	-10.171 (5.614)	0.070	-0.446
	Education (baseline)	0.233 (0.995)	0.815	0.038	0.233 (1.292)	0.857	0.038
	Treatment allocation	-0.359 (6.538)	0.956	-0.012	-0.359 (6.967)	0.959	-0.012
	Baseline emotion regulation (CERQ - negative)	-0.055 (0.323)	0.858	-0.047	-0.058 (0.285)	0.840	-0.047
	Baseline paranoia (GPTS – persecution)	0.224 (0.207)	0.279	0.291	0.224 (0.167)	0.180	0.291
Effects	Trauma (CCMS-neglect) – Emotion regulation (CERQ – negative)	0.354 (0.465) 95%CI (-0.558, 1.265)	0.447	0.112	0.354 (0.586) 95%CI (-0.795, 1.502)	0.546	0.112
	Emotion regulation (CERQ-negative) – Paranoia (GPTS – persecution)	0.103 (0.309) 95%CI (0-0.501, 0.708)	0.738	0.084	0.103 (0.262) 95%CI (-0.411, 0.617)	0.693	0.084
	Trauma (CCMS-neglect) – Paranoia (GPTS-persecution)	2.575 (1.203) 95%CI (0.217, 4.934)	**0.032**	0.665	2.575 (1.215) 95% CI (0.193, 4.957)	**0.034**	0.665
Indirect effects	Total indirect effect	0.037 (0.079) 95%CI (-0.119, 0.192)	0.644	0.009	0.037 (0.111) 95%CI (-0.181, 0.254)	0.741	0.009

Predictor		FIML data set b (SE)	p-value	Std B	Completer data b (SE)	p-value	Std B
	Bootstrap indirect mediation effect (completer data only)	-	-	-	0.043 (0.582) 95% CI (-0.647, 1.831)	0.941	0.012

Appendix 22 – Results of linear regression models

This table contains the results from all linear regression analyses conducted using the data. The primary outcome results are reported in models1-6, and exploratory analyses are reported in models 7-17.

Model	Variable	Unstandardised coefficients *b* (SE)	95% CI for *b* (lower, upper)	p-value	Standardised coefficients, *ß*
Model 1: Persecutory ideation (GPTS) R^2 = 0.365	Constant	32.005 (10.663)	11.106, 52.904	**0.003**	2.078
	Baseline	0.330 (0.177)	-0.016, 0.676	0.062	0.396
	Treatment allocation group	-11.45 (4.950)	-21.177, -1.773	**0.020**	-0.373
Sensitivity analysis – completer data only R^2 = 0.365	Constant	32.005 (12.479)	7.546, 56.464	**0.010**	2.078
	Baseline	0.330 (1.149)	0.039, 0.621	**0.026**	0.414
	Treatment allocation group	-11.475 (5.846)	-22.933, -0.017	0.050	-0.366
Model 2: Referential delusions (GPTS) R^2 = 0.180	Constant	21.319 (10.840)	0.074, 42.565	**0.049**	1.659
	Baseline	0.315 (0.160)	0.002, 0.628	**0.048**	0.418
	Treatment allocation group	-1.018 (5.251)	-11.310, 9.274	0.846	-0.040
Sensitivity analysis - completer data only R^2 = 0.180	Constant	21.319 (11.361)	-11.3199.283	0.061	1.659
	Baseline	0.315 (0.154)	0.014, 0.616	**0.040**	0.418
	Treatment allocation group	-1.018 (5.256)	-11.319, 9.283	0.846	-0.040
Model 3: Paranoia total (GPTS)	Constant	55.183 (17.488)	20.906, 89.460	**0.002**	2.190
	Baseline	0.326 (0.157	0.017, 0.634	**0.039**	0.414
	Treatment allocation group	-14.60 (8.983)	-32.217, 2.997	0.104	-0.290

Model	Variable	Unstandardised coefficients *b* (SE)	95% CI for *b* (lower, upper)	p-value	Standardised coefficients, *ß*
R^2 = 0.303					
Sensitivity analysis - completer data only R^2 = 0.303	Constant	55.183 (21.444)	13.154, 97.213	**0.010**	2.190
	Baseline	0.326 (0.139)	0.053, 0.598	**0.019**	0.414
	Treatment allocation group	-14.610 (9.865)	-33.946, 4.726	0.139	-0.290
Model 4: Suspiciousness (PANSS) R^2 =0.006	Constant	2.613 (1.032)	0.591, 4.636	**0.011**	3.244
	Baseline	0.059 (0.197)	-0.328, 0.445	0.765	0.082
	Treatment allocation group	0.034 (0.435)	-0.818, 0.034	0.938	0.021
Sensitivity analysis - completer data only R^2 **=** 0.006					
	Constant	2.613 (0.236)	0.300, 4.927	**0.027**	3.244
	Baseline	0.059 (0.208)	-0.349, 0.467	0.778	0.082
	Treatment allocation group	0.034 (0.454)	-0.856, 0.923	0.941	0.021
Model 5: Negative emotion Regulation (CERQ) R^2 = 0.315	Constant	18.838 (7.478)	4.182, 33.494	**0.012**	1.622
	Baseline	0.569 (0.133)	0.309, 0.829	**0.000**	0.561
	Treatment allocation group	0.134 (4.227)	-8.150, 8.418	0.975	0.006
Sensitivity analysis - completer data only R^2 = 0.315					
	Constant	18.838 (11.878)	-4.442, 42.117	0.113	1.622
	Baseline	0.569 (0.186)	0.205, 0.933	**0.002**	0.561
	Treatment allocation group	0.134 (4.308)	-8.309, 8.577	0.975	0.006
Model 6: Positive emotion Regulation (CERQ)	Constant	33.190 (8.972)	15.606, 50.774	**0.000**	2.312
	Baseline	0.481 (0.174)	0.140, 0.822	**0.006**	0.599
	Treatment allocation group	-3.195 (4.373)	-11.765, 5.376	0.465	-0.111

Model	Variable	Unstandardised coefficients *b* (SE)	95% CI for *b* (lower, upper)	p-value	Standardised coefficients, *ß*
R^2 = 0.397					
Sensitivity analysis - completer data only R^2 = 0.397	Constant	33.190 (13.051)	7.611, 58.769	**0.011**	2.312
	Baseline	0.481 (0.138)	0.211, 0.751	**0.000**	0.599
	Treatment allocation group	-3.195 (5.101)	-13.193, 6.803	0.531	-0.111
Model 7: Survival beliefs (BAPS) R^2 = 0.576	Constant	-3.929 (2.350)	-8.535, 0.677	0.095	-0.833
	Baseline	0.566 (0.127)	0.317, 0.814	**0.000**	0.644
	Treatment allocation group	4.971 (1.313)	2.398, 7.544	**0.000**	0.527
Sensitivity analysis - Completer data only R^2 = 0.567					
	Constant	-3.929 (3.259)	-10.315, 2.458	0.228	-0.833
	Baseline	0.566 (0.141)	0.289, 0.843	**0.000**	0.636
	Treatment allocation group	4.971 (1.421)	2.185, 7.757	**0.000**	0.555
Model 8: Normalising beliefs (BAPS) R^2 = 0.430	Constant	4.912 (2.965)	-0.899, 10.724	0.098	1.120
	Baseline	0.087 (0.164)	-0.234, 0.407	0.596	0.106
	Treatment allocation group	5.573 (1.446)	2.739, 8.407	**0.000**	0.635
Sensitivity analysis - Completer data only R^2 = 0.430					
	Constant	4.912 (3.462)	-1.873, 11.697	0.156	1.120
	Baseline	0.087 (0.149)	-0.205, 0.378	0.559	0.100
	Treatment allocation group	5.573 (1.487)	2.658, 8.487	**0.000**	0.644
Model 9:	Constant	5.091 (4.558)	-3.42, 14.024	0.264	0.932

Model	Variable	Unstandardised coefficients *b* (SE)	95% CI for *b* (lower, upper)	p-value	Standardised coefficients, *ß*
Negative beliefs	Baseline	0.498 (0.160)	0.184, 0.811	**0.002**	0.490
(BAPS)	Treatment allocation group	0.221 (2.259)	-4.207, 4.648	0.922	0.020
R^2 = 0.235					
Model 10:	Constant	-0.389 (3.726)	-7.691, 6.913	0.917	-0.066
Hypervigilance	Baseline	0.281 (0.179)	-0.070, 0.633	0.117	0.280
(BHS)	Treatment allocation group	3.175 (2.331)	-1.393, 7.744	0.173	0.270
R^2 = 0.140					
Model 11:	Constant	2.245 (2.904)	-3.447, 7.937	0.439	0.407
Anxious attachment	Baseline	0.527 (0.185)	0.165, 0.889	**0.004**	0.604
(PAM) R^2 = 0.386	Treatment allocation group	1.119 (1.946)	-2.965, 4.934	0.565	0.101
Model 12:	Constant	2.527 (3.219)	-3.782, 8.835	0.432	0.552
Avoidant attachment	Baseline	0.606 (0.239)	0.137, 1.075	**0.011**	0.507
(PAM)	Treatment allocation group	1.339 (1.730)	-2.052, 4.731	0.439	0.146
R^2 = 0.291					
Model 13:	Constant	5.279 (4.870)	-4.265, 14.824	0.278	0.590
Total attachment	Baseline	0.548 (0.193)	0.170, 0.927	**0.005**	0.574
(PAM)	Treatment allocation group	2.456 (3.222)	-3.859, 8.770	0.446	0.137
R^2 = 0.362					
Model 14:	Constant	10.928 (8.329)	-5.396, 27.252	0.190	0.698

Model	Variable	Unstandardised coefficients *b* (SE)	95% CI for *b* (lower, upper)	p-value	Standardised coefficients, *ß*
Depression (DASS)	Baseline	0.858 (0.158)	0.548, 1.168	**0.000**	0.746
R^2 = 0.571	Treatment allocation group	-5.419 (4.428)	-14.097, 3.260	0.221	-0.173
Model 15:	Constant	14.300 (8.301)	-1.970, 30.569	0.085	1.381
Anxiety (DASS)	Baseline	0.401 (0.170)	0.69, 0.733	**0.018**	0.396
R^2 = 0.239	Treatment allocation group	-4.481 (4.283)	-12.876, 3.913	0.295	-0.216
Model 16:	Constant	7.507 (8.359)	-8.877, 23.891	0.369	0.598
Stress (DASS)	Baseline	0.598 (0.178)	0.250, 0.946	**0.001**	-0.556
R^2 = 0.320	Treatment allocation group	-1.717 (4.683)	-10.895, 7.461	0.714	-0.068
Model 17:	Constant	27.283 (22.684)	-17.177, 71.743	0.229	0.763
DASS total (DASS)	Baseline	0.685 (0.159)	0.373, 0.998	**0.000**	0.622
R^2 = 0.419	Treatment allocation group	-10.246 (12.312)	-34.377, 13.885	0.405	-0.143

All analyses were conducted using FIML missing data mechanism unless otherwise specified.