Editorial Commentary

Aging

How lifestyle changes could prove to be an effective medicine for the aging cardiovascular system

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Aging is a growing medical concern. Despite more people living longer, the economical impact is likely to be severe, and pressures on healthcare providers are constantly growing to treat aging-related conditions and disorders. It is estimated, that in the US, life expectancy will go from ~76 years of age in 2010 to 80 years in 2030 (1), a pattern that is similar to a host of industrialised countries. Indeed in the United Kingdom, recent analysis has shown that male life expectancy has increased from 69 years in 1970 to 78 years in 2010, and female life expectancy from 75 years to 82 years in the same time period (2). As a result, incidence of aging-related diseases such as cardiovascular disease (CVD), dementia, and cancer are likely to increase substantially (3). It is no surprise, therefore, that research into medical advancements and lifestyle behaviours and the impact of these on aging populations has gathered a lot of interest in the academic community, as seen by the Physiological Society announcing a year of '*Understanding Ageing*' in 2015. This has led to the growth of research in aging, and also specifically cardiovascular aging.

Lifestyle choices can have significant influence on how our cardiovascular system ages. We see individuals who are fitter and regularly exercise live longer than unfit/sedentary individuals

(4, 5), which could be due to changes in our cardiovascular system. Such favourable changes in our cardiovascular system with simple modifications in our lifestyle thus offers a significant promise in helping to counteract the impact of aging on our bodies. This small collection of reviews, encompasses the effects of diet, exercise and physical activity on the '*aging cardiovascular system*', and provides some novel insights into the mechanisms by which these affect our vascular and cardiac tissues.

The first review (6) puts forward the argument that resistance exercise could affect vascular aging via modulation of arterial stiffness and matrix metalloproteinases (MMP). Older individuals display elevated levels of arterial stiffness (as measured by pulse wave technology) compared to younger individuals (7), with recent evidence supporting the view that exercise can reduce arterial stiffness in humans (8). Knox demonstrates some data to suggest that arterial remodelling with exercise may be a result of altered MMP regulation, but more work is needed to observe this link in humans.

The second review (9) discusses the role of aging on the body's endogenous endothelial repair capacity, via the reduction and dysfunction of endothelial progenitor cells, and how exercise and dietary behaviour modulate both number and function of these cells. The effects vary from improving bone marrow niche environment and pro-apoptotic stimuli to thus promoting our own stem/progenitor cell repair mechanisms on our ageing vasculature.

The subsequent review, by Beaumont et al (10) uses masters athlete's as a model of lifelong exercisers, and the differences in cardiac structure and function between master's athlete's and inactive older people. We observe that masters athletes display better diastolic function than sedentary age-matched controls, which appears to be maintained into the 8^{th} decade of life. Interestingly, the review does not demonstrate much evidence for differences in systolic function, and a much debated measure of systolic function, ejection fraction, does not appear

to be different between sedentary controls and masters athletes, being suggestive that lifelong exercise does not improve cardiac health via modulations in ejection fraction.

The final review in our collection, by Peace et al. (11), provides an insight into carotid and coronary arterial function, with subsequent focus on the role of advancing age on both, and the potential for exercise to act as a countermeasure for the aging-associated deleterious effects. They also purport that, since coronary artery function is difficult and incredibly invasive to measure and quantify, that we can use carotid artery function as a surrogate, as it appears to show similar structural and functional changes with disease conditions, offering clinicians and researchers a non-invasive global measure of arterial function that could be used to determine risk of myocardial infarction, stroke, and other atherosclerotic-mediated events.

Together, these reviews, provide evidence for the importance of lifestyle modifications and behaviour change to treat age-associated cardiovascular problems, which could subsequently have significant effects on healthcare burden and quality of life of patients. However, translation of such work into practice is often poor at best, and thus strategies must be put into place by governing bodies, healthcare providers, and clinicians to encompass such lifestyle strategies to treat disease.

Disclosures

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