PERSPECTIVES

Exercise and diseased kidneys: are they compatible?

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Regular endurance exercise is routinely recommended for primary and secondary prevention of cardiovascular disease due to its association with lower morbidity and mortality. The beneficial effects of regular exercise are well known and include improvements in cardiopulmonary function and reduction in coronary artery disease risk factors. Additionally, regular exercise has been shown to improve endothelial function in cardiovascular disease (Walther et al. 2004) and healthy ageing (Seals et al. 2008). Maintenance of endothelial function is important for control of arterial tone in addition to numerous other beneficial effects, including inhibition of platelet aggregation, leukocyte adhesion, and smooth muscle cell proliferation. Acute exercise is associated with an increase in cardiac output and pulse pressure resulting in exercise-induced increases in shear stress. The increased shear stress is thought to be the stimulus for endothelial adaptations to exercise training (Walther et al. 2004; Green, 2009).

Endothelial function is impaired in chronic kidney disease (Schiffrin et al. 2007) and declines with sedentary ageing (Seals et al. 2008). In addition to the risk of progressing to renal failure, chronic kidney disease is also associated with an increased prevalence of cardiovascular disease (Schiffrin et al. 2007). Exercise training would appear to be particularly beneficial in chronic kidney disease to improve endothelial function and reduce cardiovascular risk; however, data are lacking in both animal models and humans with reduced renal function. As a result exercise training is not recommended to the same extent in chronic kidney disease as it is for those with cardiovascular disease or traditional cardiovascular risk factors (Johansen, 2005).

There may be unique considerations for exercise training in kidney disease as highlighted by Moningka et al. (2011) in this issue of The Journal of Physiology. The acute increase in cardiac output during exercise impacts many vascular beds. Exercise training elicits endothelial improvements in the vasculature supplying the heart and active skeletal muscle beds but also non-exercised limbs, all of which appear to be shear dependent, suggesting endothelial adaptations are systemic (Green, 2009). However, it is well known that renal blood flow declines to levels that are well below resting values during exercise as a result of a redistribution of blood flow to meet the needs of exercising muscle. Moningka et al. (2011) proposed that this reduction in renal blood flow during exercise may reduce shear stress and exacerbate high oxidative stress and low endothelial nitric oxide synthase (eNOS) levels in the ageing kidney (Moningka et al. 2011). Therefore, they tested the novel hypothesis that treadmill exercise would exacerbate the progression of age-related kidney disease in male Fisher 344 rats. Twelve weeks of treadmill training did not result in increased renal eNOS or superoxide dismutase (SOD) abundance in the old rats as it did in young rats and did not alter age-related renal injury or function. At first glance these results may be interpreted as a failure of exercise to improve renal function in ageing. However, their findings that exercise training did not worsen renal injury or function in ageing is also significant. A worsening of kidney disease with training could ultimately have a significant impact on exercise recommendations for older adults or those with kidney disease of any age. Moningka et al. initiated treadmill training at an old age (22–24 months). One potential explanation for the lack of improvement may be that it is not possible to improve renal structure or function with exercise training once a certain degree of kidney disease has occurred. In support of this, 4 weeks of voluntary wheel running following 5/6 nephrectomy in mature (but not old) Sprague–Dawley rats did not alter renal function despite positive improvements in peripheral vascular function (Shelkovnikov et al. 2008). Importantly, exercise training did not worsen renal function in this study either.

Treadmill exercise training initiated at an old age did not worsen renal injury but it remains to be determined whether exercise training can prevent or slow age-dependent kidney disease. Studies are needed to determine if a treadmill exercise intervention instituted at a younger age or voluntary wheel running can prevent or slow deterioration of renal function in the Fisher 344 rat. The type of training may be an important consideration. Life-long voluntary wheel running resulted in less age-related renal structural damage in old male Sprague–Dawley rats; however, forced treadmill running, similar to that used by Moningka et al. (2011), had no beneficial effect on the kidney (Loupal et al. 2005). Further, forced wheel running has been shown to exacerbate kidney damage in old mice (Lichtig et al. 1987). Taken together these studies suggest that perhaps the stress of forced exercise may not improve, and potentially worsens exercise effect on renal function. While exercise training may not reverse kidney disease, it has profound potential to reduce cardiovascular risk in chronic kidney disease.

References