Exercise and vascular adaptation

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Integrative human physiological studies have demonstrated the impact of exercise training and physical conditioning on vascular adaptation. In general terms, exercise training is associated with an impressive increase in cardiac capacity which is accommodated by increased vascular conductance, such that arterial pressure is not elevated. This increase in conductance is associated with functional and structural vascular changes.

From a clinical perspective, the profound impact of exercise training, physical activity and fitness on cardiovascular and all-cause mortality is not fully explained by "secondary" effects of exercise on traditional risk factors. We have proposed that this "risk factor gap" may be explained by direct impacts of episodic exercise on the vascular wall, mediated *via* haemodynamic responses which induce changes in shear stress and transmural pressure (Joyner & Green, 2009).

Evidence from animal and human experiments which indicates that exercise training leads to changes in endothelial function, in healthy subjects and in particular those with pre-existing cardiovascular and metabolic diseases (Green *et al.*, 2004). There is substantial evidence that such effects of training on vasodilator function are not limited to the exercised skeletal muscle vascular beds (Green, Maiorana & Cable, 2008). Changes in arterial pressure and lumenal shear stress which occur with each exercise bout are likely candidates for the mechanisms responsible for functional and structural adaptation, related to changes in stress and pressure brought about by different forms of exercise, including substantial retrograde arterial flow and shear during exercise in some vascular beds (Green *et al.*, 2005).

Recent studies have manipulated shear stress in humans during exercise training, with results indicating that this stimulus may be an obligatory signal for endothelium-dependent adaptations in the function and size of large and medium sized arteries (Tinken *et al.*, 2010). Other studies indicate that functional adaptations in response to training may be superseded by structural remodelling of large and medium sized arteries, which obviate the need for ongoing functional modulation (Tinken *et al.*, 2008). Finally, studies which have addressed the effect of exercise training on arterial wall thickness in humans have produced suggestive evidence that the mechanisms responsible for changes in the size and function of conduit and resistance arteries may differ from those associated with change in wall thickness (Tinken *et al.*, 2011).

In summary, exercise training is a potent stimulus to adaptation in arterial function, size and wall thickness in humans. These adaptations are a fundamental physiological response to the repeated stress of exercise which may provide a mechanistic explanation for the anti-atherogenic benefits of exercise and physical activity.

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