REVIEW ARTICLE

Cardiovascular benefits of controlled isometric training may be due to an improvement in endothelial function via up regulation of nitric oxide synthase through shear stress induced transcriptional mechanisms.

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Abstract Essential hypertension is associated with a reduced functioning of endothelial release of nitric oxide. Impaired endothelium-dependent vascular relaxation has been documented in virtually all cardiovascular disorders and appears to occur early in the course of cardiovascular disorders such as arteriosclerosis, diabetes mellitus, hypercholesterolemia, hypertension, and heart failure.

Endothelium-dependent vasodilatation is most actively driven by the release of nitric oxide (NO) in response to shear stress on the arterial wall. The exposure to shear stress is also a regulating factor in the transcription of endothelial nitric oxide synthase (eNOS); an increase in shear stress upregulates the production of eNOS. These regulating mechanisms are initiated at the onset of an increase in shear stress, and produce structural changes through a signaling cascade that involves transcription factor activation, gene regulation and protein synthesis, and ends in a remodeling of the vessel to align with the applied shear stress.

If there is a deficiency of eNOS activity/availability, then the basal release of NO will not properly dilate the arteries, and the basal sympathetic activity will allow the vascular smooth muscle to constrict the vessel to a smaller diameter. This results in a net increase in total peripheral resistance (TPR), increase in systemic blood pressure, a net increase in wall shear stress, and eventually a balanced regulation of eNOS and release of NO to hold the dilation to the new diameter. This feed back provides homeostasis, but at the price of elevated systemic pressures.

A transient increase in systemic mean arterial pressure (MAP) increases the mean shear stress in vivo throughout the entire arterial tree. Sustained, brief (one minute), increases in MAP may be able to initiate the signaling cascade that leads to an upregulation in eNOS in the entire arterial tree, an improved basal release of NO, a reduction in total peripheral resistance (TPR), and a reduction in arterial blood pressure.

A simple method for eliciting a transient increase in MAP is through the use of acute controlled isometric efforts greater than 15% maximum voluntary contraction (MVC). During such a sustained effort, MAP rises constantly (pressor response). At higher levels of sustained force, MAP rises faster. A predictable level of MAP increase can be obtained by holding a set percentage of MVC for a set amount of time. This type of sustained isometric effort has been shown to reduce blood pressure over a four-week period and may be due to a systemic upregulation in eNOS. This type of training also maintains the benefit of reduced blood pressure, probably through maintenance of the new level of eNOS activity.

The cardiovascular benefits of chronic controlled isometric training may be due to an improvement in endothelial function; a common treatment goal for conditions such as arteriosclerosis, diabetes mellitus, hypercholesterolemia, hypertension, and heart failure.

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