Exercise Generates Lactate and Fluid Intake: Effects on Mitochondrial Function in Heart and Vascular Smooth Muscle

To the Editor:

We were very interested to read the 2 editorials and 3 articles1–3 published in a recent issue of Hypertension concerning exercise training and cardiovascular disease. Taking, eg, the article by Garciarena et al.,1 we were encouraged to see that the concepts of a Letter to the Editor published recently4 could apply equally well here. The main topic discussed is what is happening in exercise and why this improves symptoms of cardiovascular disease. We suggested for diabetes mellitus and ageing that the problem was one of lowered substrate supply for metabolic function, thus creating mitochondrial dysfunction. In many cardiovascular-related problems, dysfunction of mitochondrial metabolism has been observed both in the heart and throughout the circulatory system.5,6 Thus, as we suggest, exercise increases the availability of the monocarboxylate family of lactate transporters and lactate production in skeletal muscles. The lactate passes into the blood and becomes a substrate that penetrates easily into cells via the monocarboxylates and improves mitochondrial biogenesis and possible function in cardiac and vascular smooth muscle cells. Although this is just a hypothesis, it would not appear to be difficult to confirm.

There is, however, another aspect of the physiology presented in the articles in this volume of Hypertension that is completely overlooked, and that is fluid ingestion. No measures of water intake are given; however, exercise, or physical activity, is associated with fluid loss and, thus, cellular and extracellular dehydration. This condition stimulates the release of renin forming angiotensin II, which is also a risk factor for cardiovascular disease. Exercise and angiotensin II should stimulate thirst and, thus, water intake. An increased fluid consumption has been recommended to decrease the incidence of not only cardiovascular disease but also diabetes mellitus and obesity through a possible mechanism of increased tissue perfusion.

Given then that exercise training improves cardiovascular function in rats and humans, it could be proposed that exercise-induced lactate production activates mitochondrial activity, along with the drinking-induced increase in tissue perfusion. These 2 actions would return toward normal cardiac and smooth muscle, as well as endothelial and cell metabolic functions, which could potentially allow improved cardiac activity and decreased arterial tree stiffness.

Disclosures

None.

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Hypertension. 2009;54:e14; originally published online June 29, 2009;
doi: 10.1161/HYPERTENSIONAHA.109.134742
Hypertension is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0194-911X. Online ISSN: 1524-4563

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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