Adaptive and Maladaptive Cardiac Hypertrophy: What Is the Effective Role of Heat Shock Transcription Factor 1?

To the Editor:

In the recent interesting article by Sakamoto et al.,1 the authors found that rats undergoing exercise training showed lower degree of left ventricular (LV) hypertrophy, higher expression and activity of heat shock transcription factor 1 (HSF1), and preserved LV systolic function in comparison with rats exposed to sustained pressure overload by surgical constriction of the transverse aorta (TAC). They also observed that transgenic mice expressing constitutively active HSF1 showed less evident hypertrophic response and better systolic function than their wild-type littermates 5 weeks after TAC. Conversely, HSF1-deficient mice showed similar degree of LV hypertrophy but worse systolic function in comparison with their wild-type littermates after 4 weeks of exercise or 1 week after TAC. These data support the intriguing hypothesis that HSF1 upregulation in cardiac myocytes may play a key role in preventing LV systolic impairment in the exercise-induced hypertrophy model, and in determining the adaptive or maladaptive nature of the hypertrophic response.

An important issue in the interpretation of these findings derives from the method used for the assessment of LV systolic performance. The use of unadjusted echocardiographic indices measured at the level of endocardium, such as the M-mode-derived fractional shortening, in exercise-induced and pressure overload, but further studies are needed to investigate its effective role in affecting LV systolic function and in characterizing the adaptive or maladaptive nature of LV hypertrophy in these conditions.

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