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EDITORIAL COMMENT

Physical Activity Prevents Obesity and Heart Failure

Now What Are We Going to Do About It?*

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"Knowing is not enough; we must apply.
Willing is not enough; we must do."
—Goethe (1)

The notion that physical activity improves health and well-being exists far beyond the margins of theory and deep within the confines of fact. That exercise is an essential component of a well-rounded life is a truth widely accepted by people of all cultures, and it echoes as far back as human civilization has existed. The missing link, however, remains the exact mechanism by which exercise impacts health. Applying the theory of scientific reductionism to this question, investigators ranging from geneticists to physiologists have attempted to break down the effects of exercise on health into its constituent parts. For the most part, the details remain undiscovered.

In this issue of *JACC: Heart Failure*, Florido et al. (2) explore the relationship between physical activity, obesity, and cardiac troponins from an epidemiological perspective. They examined data from 9,427 participants in the diverse and well-described ARIC (Atherosclerosis Risk In Communities) study, classifying them into categories of physical activity (recommended, intermediate, or poor), obesity (non-obese or obese), and elevations in high-sensitivity cardiac troponin level stratified at 14 ng/l (elevated or not elevated). They found that the prevalence of elevated troponins was higher in those with lower activity levels. This association was attenuated but remained significant after correction for mediators of cardiovascular disease and biomarkers of renal function, inflammation, and myocardial stress. Furthermore, the associations remained intact across subgroups of age, sex, race, and gender. Next, the authors noted that the relationship between obesity and troponin elevations was greatly attenuated to the point of being null among participants who exercised at recommended levels, especially when individual risk factors for cardiovascular disease were included in the statistical modeling. Finally, the various combinations of physical activity and obesity showed the expected gradients of risk: low-activity or obese people had the highest risk of developing heart failure, whereas high activity/non-obese people had the lowest. Within each grouping, cardiac troponin elevations forecasted higher risk.

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From these findings, Florido et al. (2) postulate that physical activity may lessen the association between obesity and subclinical myocardial damage. Simply resting on the laurels of their analysis, this conjecture would not amount to more than hypothesis generating. Thankfully, there is a colossal amount of data that reinforces these findings. For example, we previously reported that risk variants in a gene strongly associated with obesity—the fat-mass and obesity-associated gene (FTO)—predisposed carriers to a higher risk of cardiovascular disease, but only in those who were inactive (3,4). These findings were recently expanded using a more comprehensive genetic risk score, with similar conclusions (5). Using the concept of Mendelian randomization, whereby genetic variants associated with an outcome of

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interest can resolve whether observational associations imply causation or exist due to confounding, we can safely surmise that the undesirable cardiovascular effects of obesity may be offset by physical activity. Beyond just the seemingly healthy, the salutary effects of exercise have similarly been demonstrated in patients across the spectrum of heart disease. Exercise, it appears, can prevent the onset of heart failure in those with high-risk cardiovascular disease and improve outcomes in patients with chronic, stable, heart failure. In point of fact, the landmark HF-ACTION (Heart Failure: A Controlled Trial Investigating Outcomes of Exercise Training) study of more than 2,300 heart failure patients showed that exercise training improved quality of life and exercise capacity, and reduced the risk of heart failure hospitalizations. In a triumph for the heart failure community, the results of this study played a key role in the decision by the U.S. Centers for Medicare and Medicaid Services to extend cardiac rehabilitation coverage to patients with heart failure with reduced ejection fraction.

Now we must get to the more difficult question: what are we going to do with this information? There is a widening gulf between academic research on exercise and heart disease and its translation into actionable benefits. For example, a PubMed search of *exercise* and *cardiovascular* yields almost 20,000 publications in the past decade. During this period of time, the percentage of obese adults in the United States has increased from 25% to 30%, and 80% of Americans do not meet the government's national physical activity recommendations. The impact of these worrisome developments on the health of our nation are profound: an estimated $117 billion in healthcare costs are associated with inadequate physical activity, and adults who are inactive pay $1,437 more per year in healthcare costs than physically active adults. Clearly, the health care community—in particular, cardiologists who are defined by the American College of Cardiology as doctors with special training and skill in finding, treating, and preventing diseases of the heart and blood vessels—must take some responsibility for these disturbing trends, and play an active role in reversing them.

What can be done? As a first step, we must challenge the status quo, where health care systems are financially rewarded for the increasing incidence and prevalence of heart failure via reimbursements associated with complex therapies such as implantable defibrillators, complicated revascularizations, and ventricular assist devices. Furthermore, the prestige of cardiovascular divisions is gauged by their ability to perform expensive diagnostic and therapeutic interventions with diminishing gains, rather than their impact on the heart health of the population they serve. Investigators are similarly rewarded on the basis of publications and grants, rather than their influence on healthy behavior. For example, a dozen nominal publications on exercise would be viewed far more favorably by an academic promotions committee than the creation of an app that meaningfully increases population physical activity. Cardiologists should take their lead from the editor of *Journal of the American College of Cardiology*, Dr. Valentin Fuster, who has extended his influence far beyond the confines of the academic medical center and into the realm of programs that teach children the importance of healthy habits (6). Indeed, since the development of cardiovascular disease is impacted by public policy decisions about education, diet, and exercise, we must collectively play a far more active role in their drafting and implementation.

What if we started to view heart failure as a failure, of not only of the heart to keep up with the metabolic demands of the body, but also the community of cardiologists to promote the behaviors that lead to its development? This will surely encourage a shift beyond the reactionary approaches used in contemporary clinical practice and force us into interactions with the multitude of stakeholders that play a role in improving the cardiovascular health of our communities. Such a precedent is not original: almost a century back, one of the founding fathers of American cardiology, Dr. Paul Dudley White, not only spoke widely with the lay public about the benefits of physical activity, but also created several bicycle paths, and served as the president of the Bicycle Touring League of America. His mantle of prevention through attention to the "simple rules of health" has been taken on by Dr. Fuster. We should follow his example and strive to move beyond simply knowing and reacting to actively preventing cardiovascular disease.

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