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EDITORIAL COMMENT

Therapeutic Cardiorespiratory Fitness to Prevent and Treat Heart Failure*

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Obesity is increasing in epidemic proportions in the United States and most of the Westernized globe (1,2). Recent studies indicate that the prevalence of obesity in the United States has now reached 38%, and more alarming, the prevalence of severe or morbid obesity, defined as body mass index (BMI) ≥40 kg/m^2, reached a prevalence of 8% (3). Obesity has adverse effects on cardiovascular (CV) hemodynamics and cardiac structure and function, including worsening systolic and, particularly, diastolic ventricular dysfunction, so it is not surprising that obesity increases the prevalence of heart failure (HF) (1–3). Because obesity also increases the risk of hypertension, left ventricular hypertrophy, coronary heart disease (CHD), and atrial fibrillation, all strongly related to HF, not only the prevalence of HF but also HF hospitalizations have been increasing (1–4).

Substantial evidence exists to show that cardiorespiratory fitness (CRF) is one of the most powerful predictors of prognosis, including CV disease (CVD) and all-cause mortality (1–5). Indeed, in patients with established CVD, including HF, CRF is a strong predictor of better prognosis.

SEE PAGE 367

In this issue of *JACC: Heart Failure*, Pandey et al. (6), using data from the Cooper Center of Longitudinal Study (CCLS) followed 19,485 patients for 10 years to determine the association between BMI and CRF and the changes in these parameters over time and subsequent risks of HF hospitalizations. As demonstrated in other studies, high mid-life BMI was significantly associated with high risk of HF hospitalization, with a 19% increase (hazard ratio [HR]: 1.19; 95% confidence interval [CI]: 1.12 to 1.26) in HF hospitalization risk for every 3 kg/m^2 higher BMI, after adjusting for other HF risk factors. However, this association was significantly attenuated, although not completely abolished, after adjusting for CRF, with only a 10% increased risk (HR: 1.10; 95% CI: 1.03 to 1.17) of HF hospitalization per 3 kg/m^2 BMI. Cardiorespiratory fitness accounted for nearly one-half of the HF risk associated with BMI. Moreover, changes in BMI over time were not significantly associated with HF risk after adjusting for changes in CRF. These data add support to the idea that CRF markedly alters the relationship between adiposity and prognosis, including CVD and, specifically, HF risk. These data also support previous data from the Aerobic Center Longitudinal Study showing that a 1 metabolic equivalent of task (MET) increase in CRF on maximal exercise tests separated by an average of 6.3 years in 14,345 men was associated with reductions in all-cause and CVD mortality of 15% and 19%, respectively (7). In addition, Lee et al. (7) observed that changes in BMI were not associated with either all-cause or CVD mortality after accounting for possible confounders and changes in CRF.

The present study by Pandey et al. (6) of a cohort of patients with HF who were mostly relatively fit, Caucasian, and male, demonstrated that higher BMI and lower CRF was progressively associated with increased risk of HF hospitalizations. As shown in other studies, progressive reductions in CRF also occur with higher BMI (1,2,5,7,8), so the obese unfit participants have a particularly higher risk of HF hospitalization. Although the highest risk of HF in this CCLS cohort occurred in those who were most

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unfit (quintile 1 [Q1] or the bottom 20 percentile of CRF), and HF risk progressively declined with higher CRF (lowest in Q4 and Q5 or the top 40 percentile of CRF), the largest incremental improvement in risk occurred with moving from the lowest Q, into Q2 and Q3, or moderate levels of CRF (between the 20th and 60th percentile) (6). Although CRF is also determined from genetic factors, a major component of CRF is regular physical activity (PA) and exercise training (ET) (8). Performing 150 min per week of moderate PA/ET or 75 min per week of more vigorous PA/ET is generally sufficient to move most individuals into at least the moderate CRF level (8). The lowest risk of HF occurs in the highest Qs of CRF (Q4 and Q5), which does not represent extremely high levels of CRF but rather levels just above the average. Many patients performing these levels of PA/ET, particularly higher intensity PA/ET, can easily reach this level of CRF, which is associated with maximal protection against HF risk. Interestingly, in the highest CRF level, Q4 and Q5, there was no significant relationship between BMI and HF risk, suggesting that high CRF completely ameliorates the adverse impact of obesity on HF risk.

Although obesity increases HF risk, unlike the situation with CHD, HF risk increases with BMI, even when metabolic health is present (1,2). Nevertheless, we and others have reported an obesity paradox, indicating better prognosis in overweight and at least mildly obese patients with established HF versus those in normal weight and, especially, underweight HF patients (1). However, as we demonstrated in CHD (2), the observed obesity paradox appears to be restricted to mostly the lower fit patients with HF (1,2). In those with moderate or high CRF, prognosis is excellent, and no obesity paradox is present (1,2). Again, these data support the fact that fitness markedly alters the relationship between adiposity and subsequent prognosis (1–3,8).

Greater efforts to increase CRF in primary and secondary prevention are needed, which can be accomplished by regular PA/ET (8). An ideal way to accomplish this is through the use of cardiac rehabilitation (CR) and ET programs in CHD and HF; thus, it is very important to increase enrollment and adherence to these programs throughout the health care system in the United States and worldwide (1,2,8). As suggested in the CCLS data and as discussed earlier (6), our data also suggest that only small improvements in CRF with CR are needed to reduce psychological stress and depression and depression-related increased mortality (8). Although maximal protection may occur at the highest levels of CRF, the biggest "bang for the buck" is observed with moving a patient out of the low CRF category into a moderate level of CRF. This gain in CRF and reduction in risk is within striking range of most adults, who are capable of performing modest levels of PA/ET as discussed above. The promotion of PA/ET is desperately needed throughout the health care system and is a cost-effective method for reducing CVD, mortality (1–5,8), and HF risk, as demonstrated by Pandey et al. (6).

Finally, further studies are needed to determine the relative benefits of PA/ET/CRF to prevent various types of HF (preserved versus reduced left ventricular ejection fraction) at various levels of BMI and adiposity. The proper amount and type of PA/ET to maximally reduce HF risk has yet to be determined. Studies are needed to assess the ideal PA/ET intensities and volumes needed to prevent HF and to lower the risk in patients with established HF.

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