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It is well recognized that the clustering atherogenic and diabetogenic abnormalities of the metabolic syndrome are highly prevalent in our affluent, sedentary populations. Indeed, we have designed for ourselves devices as well as working and living environments that spare us from various physical activities. Unfortunately, this sedentary environment cannot protect us against the energy-dense, refined diet that has been adopted by an increasing proportion of our population, leading to the development of a positive energy balance, weight gain, and obesity.

The opinions expressed in this article are not necessarily those of the editors or of the American Heart Association.

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453
blood pressure, an atherogenic dyslipidemia (high triglycerides, low HDL cholesterol, and small LDL particles), a prothrombotic state, and an inflammatory profile (elevated C-reactive protein being the most convenient marker of inflammation).12 Furthermore, although insulin resistance is likely a key factor involved in the pathogenesis of the metabolic syndrome,9–11 it is nevertheless clear that the prevalent form of the metabolic syndrome in our sedentary overweight/obese population is most often accompanied by abdominal obesity.1,2 LaMonte et al7 cannot rule out the likely possibility that some of their subjects who eventually met the NCEP-ATP III criteria during the follow-up study were nevertheless characterized by substantial differences in key features of the metabolic syndrome at baseline, such as fasting hyperinsulinemia, excess visceral fat accumulation, increased apolipoprotein B and C-reactive protein levels, reduced adiponectin concentrations, and an increased proportion of small LDL particles, to name but a few important parameters. For instance, although the investigators stated that there was no difference at baseline in the risk factors studied, it is obvious that individuals who later developed the metabolic syndrome had initially higher waist circumference and triglyceride values and lower HDL cholesterol levels. In this regard, we previously reported in Circulation12 that a large proportion of men with waist circumference values below the male NCEP-ATP III cutoff of 102 cm (but >90 cm) showed many important abnormalities of the metabolic syndrome (including hyperinsulinemia, elevated apolipoprotein B, small LDL particles, and visceral obesity) when such an elevated waist circumference was accompanied by hypertriglyceridemia, a condition that we described as “hypertriglyceridemic waist.” The only rationale for proposing 102 cm as a waist circumference cutoff value in NCEP-ATP III was that it had been shown to be the value corresponding to an average body mass index of 30 kg/m2 in men. Recent studies have also provided support to the notion that the NCEP-ATP III waist cutoff values should be revisited,13 especially in various ethnic groups.

Despite such limitations, it is nonetheless clear from the study by LaMonte et al7 that individuals who did not yet meet NCEP-ATP III criteria and who had a poor level of fitness had a markedly increased risk of meeting these criteria in later years. These results are important in clinical practice for patients with moderate or borderline metabolic abnormalities who would simultaneously perform poorly on the treadmill test. The article by LaMonte et al7 indicates that priority and support to “recalibrate” nutritional and physical activity habits is provided to these individuals at high risk.

Cardiorespiratory Fitness and Incidence of Metabolic Syndrome: Potential Mechanisms and Pending Issues

How a high fitness level may protect against the metabolic syndrome is a topic of great public health/clinical relevance. In this regard, Blair and colleagues6–14 have published seminal papers showing that a low level of cardiorespiratory fitness (which can be simply assessed by poor performance on a maximal exercise test) was a powerful predictor of cardiovascular disease events and mortality as well as of the risk of developing type 2 diabetes mellitus. This group also pioneered the interesting notion that being apparently fat yet fit could be nevertheless associated with a substantially reduced risk of metabolic complications and of cardiovascular disease compared with unfit, normal-weight individuals.15 On the basis of these observations and considering the role of abdominal obesity and of excess visceral adipose tissue in the pathophysiology of the metabolic syndrome, some key questions require further attention. For instance, should we prioritize weight loss or should we increase energy expenditure by promoting more physical activity to reduce the risk of type 2 diabetes mellitus and cardiovascular disease and related mortality? Because exercise intensity is an important element of an exercise training program to improve cardiorespiratory fitness, should we emphasize the intensity component of the exercise prescription to optimally improve cardiorespiratory fitness and reduce cardiovascular risk? Although it is the simplest and most reliable index available in clinical practice, cardiorespiratory fitness is not only a marker of physical activity but also has a significant genetic basis.16 Some sedentary individuals with good genetic predispositions may nevertheless perform quite well on a treadmill test.16 Thus, the health benefits of cardiorespiratory fitness may also be partly mediated by some favorable genetic characteristics conferring protection against the development of the metabolic syndrome and cardiovascular disease. Standardized exercise training studies conducted in initially sedentary individuals are essential to dissociate the adaptation of a condition that we have previously referred to as “metabolic fitness” (an individual’s metabolic risk profile) from the response of cardiorespiratory fitness to increased physical activity or exercise training. Several exercise training studies conducted and published by our group have failed to show any correlation between the magnitude of increase in cardiorespiratory fitness and improvements in cardiovascular disease risk factors.5,17 The loss of body fat, especially abdominal fat, has often been found to be a significant correlate of exercise training–related metabolic improvements.17,18 Furthermore, exercise training has been shown to substantially mobilize visceral adipose tissue even in the absence of a change in body weight.19 Thus, even when perfectly matched for body mass index or total adiposity, there is evidence that fit individuals may have less visceral fat than unfit fat subjects.20

In summary, until we fully understand the biological mediators of the link between cardiorespiratory fitness and cardiovascular disease, we should not confuse a marker of risk (fitness) with a therapeutic target (improving fitness). However, this last point should be discussed in academic debates, as we will never emphasize enough: (1) the powerful prognostic value of poor fitness as a predictor of metabolic diseases and related morbidity and mortality, and (2) that a physically active lifestyle combined with healthy nutritional habits reduce the likelihood of developing abdominal obesity, features of the metabolic syndrome, type 2 diabetes mellitus, and cardiovascular disease. LaMonte et al7 should be commended for their continued and significant contribution to the
field of exercise, fitness, and cardiovascular health. It is hoped that this important study will challenge all relevant stakeholders and stimulate the creation of safe environments that allow a physically active lifestyle at home, at school, and at work. Reshaping our sedentary habits will be a huge challenge that will go beyond the capacities of our medical model because the North American urban environment has been designed to be friendlier to cars than to human beings.

References


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Our Passive Lifestyle, Our Toxic Diet, and the Atherogenic/Diabetogenic Metabolic Syndrome: Can We Afford to Be Sedentary and Unfit?

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