Fitness Versus Fatness: the Debate Continues

To the Editor:

We read with interest the articles about the effect of exercise training on C-reactive protein (CRP) levels\(^1\) and the accompanying editorial.\(^2\) Okita et al\(^1\) showed that a 2-month period of exercise training in women with a wide range (20.4 to 39.0) of body mass index (BMI) reduced CRP concentrations independently of the magnitude of weight loss. Obisesan et al\(^2\) reported that gene variants of CRP are not related to reduction of CRP levels induced by exercise training (24 weeks) in 63 sedentary men and women (prevalence of obesity not specified). Surprisingly enough, reduction was greater in the first study (3 kg on average) than in the second study (range of weight loss 1.1 to 1.8 kg). In none of the two studies was there any statement about the cut of daily calories at the end of the observation. This seems particularly strange as losing weight with exercise alone is more difficult because of the accompanying increase in free fat mass (muscle) which may counterbalance the decrease of fat mass. Another analysis missing was enucleating the obese group to see for a particular response of CRP levels. So, the reader continues to suspect that part of the effect of exercise training on CRP levels is related to the concomitant weight loss. Okita et al\(^1\) stated, and we agree, that there might be an optimal pace of exercise and weight loss to optimize the inflammatory status which needs to be examined in long-term intervention studies. In any case, two such studies escaped his attention.

We have demonstrated in two intervention studies of 2-year follow-up in obese women\(^3\) and men\(^4\) that a multidisciplinary program of changes in lifestyle designed to obtain a substantial reduction in body weight through caloric restriction and increased physical activity was associated with reduced inflammatory status. The pooled data for the two studies comprising 60 women and 55 men are given in the Figure. The average body weight decrease was 14 kg, aerobic physical activity increase was 130 minutes, and CRP levels decrease was 1.1 mg/L. Spearman rank correlation coefficients revealed significant associations between changes in CRP levels after intervention and changes in both body weight (\(r=0.31\)) and physical activity (\(r=-0.29\)). The healthful behavior were not extreme, as for the most the physical activity criterion was met by half an hour of walking (moderate intensity) daily.

From the standpoint of preventive medicine, the debate of the relative importance of fitness versus fatness in reducing the inflammatory status seems largely academic. Sedentary behavior and excess body weight both seem to contribute to increased inflammatory status: regular physical activity is a common treatment of low fitness and excess weight, and also is a critical component of long-term weight management. Western societies actually spend a huge part of their health care costs on chronic disease treatment and interventions for risk factors. Promotion of healthful lifestyles for primary prevention among individuals at all ages would yield great benefits and reduce the burden of chronic diseases, primarily cardiovascular diseases.

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In response:

Esposito and colleagues raised a number of interesting questions relative to our recent publication “C-reactive protein genotypes affect baseline, but not exercise training-induced changes, in C-reactive protein levels.”\(^5\) First and foremost, Esposito and colleagues address the age-old issue of whether fitness or fatness (or in this case, change in fitness or fatness) is the primary mechanism underlying the 14% reductions in C-reactive protein (CRP) levels that we found with exercise training. We believe very strongly that in our study the change in fitness was the primary physiological exercise training–induced adaptation that accounted for the training-induced reductions in plasma CRP levels. We base this belief on two lines of evidence. First, the reductions in body weight and total body fat in response to the 6-month exercise training intervention were minimal, averaging 1.5±0.3 kg and 1.2±0.3% fat, respectively. Though both of these reductions were highly statistically significant, because nearly all subjects reduced both of these variables as a result of training, they are hardly of the magnitude that one would expect to elicit direct and substantial clinical improvements in other cardiovascular disease risk factors. Second, and more directly to the point, the changes in plasma CRP levels with exercise training we observed in our total population maintained their statistical significance even after we covaried for the effects of training-induced changes in body composition. Because of these two lines of evidence, we believe that the reductions in plasma CRP levels our subjects elicited with exercise training are the result of their increase in fitness and are not due to their rather minimal changes in fatness, in terms of body weight or percent total body fat.

Our subjects did not lose larger amounts of body weight and body fat during the long-term 6-month exercise training intervention because our study was designed to maintain a constant caloric intake through the use of strict dietary monitoring. Dietary records indicate this program was very effective in maintaining a constant caloric intake throughout the entire duration of the study. This important design feature ensured that...
any weight lost during the intervention was the result of the increased caloric expenditure associated with the exercise the subjects were undergoing, and not the result of an absolute caloric restriction. Thus, our subjects did not “cut” the number of calories they were ingesting during the intervention, and this is undoubtedly the reason why our average weight loss was substantially less than that of Okita and colleagues with their shorter exercise training intervention that was designed to bring about weight loss.2 Furthermore, our data do indicate, as Esposito and colleagues point out, that it is difficult to lose substantial amounts of body weight using only exercise as an intervention, irrespective of specific alterations in the fat and fat free components of body composition.

We thank Esposito and colleagues for raising these important issues.

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In response:

We were very pleased to read the letter from Esposito et al concerning our recent article demonstrating the effect of exercise training with weight loss on blood CRP levels.1 In their letter, they mention the extent of weight loss with exercise in the subjects in our study. Our subjects were all women, and it is unlikely that they increased their muscle mass through endurance exercise. Indeed, muscle mass evaluated by ultrasound tomography showed no change after our weight reduction program. Therefore, fat reduction might simply reflect weight reduction in our subjects. We used a weight reduction protocol with exercise training, and only during the baseline period did we provide simple nutritional education (see our Methods section1) suitable for the subjects of our exercise training program. We believe this to be a general and useful protocol in a clinical situation. As Després appropriately suggests, it may be that some of our subjects were under caloric restrictions.3 We agree with the editorial comment that the magnitude of weight loss was greater than could reasonably be expected from the estimated energy deficit caused by the exercise. Under our exercise protocol, the expected weight loss should have been within 2 kg. It would have been better to estimate caloric intake by a validated and more consistent method.

There are many factors other than the intensity and amount of exercise which contribute to changes in weight with exercise. Increased physical activity is usually accompanied by increased activity in the whole body metabolism, which could lead to further caloric consumption. Additionally, daily caloric intake could change. Also, the initial weight and gender of the subjects are important factors; women have more fat than men. More initial fat and/or weight potentially cause more caloric consumption, especially with exercise on the ground, because the heavier weight causes a larger workload. Although our study was not strictly designed to determine the effects of exercise training on CRP levels, we observed a significant decrease in CRP in subjects who experienced little weight loss (within 1 kg), from 0.61 (0.24 to 1.43) to 0.38 (0.12 to 0.80) mg/L (n=28, P<0.05). In any case, the relative importance of increased physical activity versus fat loss in reducing inflammatory status has not yet been fully elucidated. Sedentary thin subjects with high CRP values should also be studied.

Esposito et al3 demonstrated the remarkable effects of weight loss with a low-energy diet and increased physical activity (ie, lifestyle changes) on inflammatory markers in obese women (BMI, 35 on average). Both exercise and nutrition are important components of a healthy lifestyle. Clinically, weight reduction should be performed with an integrated weight reduction program, because caloric restriction empirically seems to be more difficult than exercise. We recently completed a randomized clinical trial designed to examine the differences between the effects of gym-based supervised exercise training and lifestyle modification on cardiovascular risk factors including CRP in ~500 subjects. The results may provide further insights into the clinical practice of preventive medicine.

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