Obesity, Weight Loss, and Cardiovascular Health: Is Oxidative Capacity a Missing Link?

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Obesity is a risk factor for hypertension, atherosclerotic heart disease, and a major cause of morbidity and mortality throughout the world. In the United States, the incidences of obesity and insulin resistance have risen dramatically in the past decade with an adult prevalence of 32%. Public awareness of the “obesity epidemic” has resulted in interest in dietary weight loss with 30–40% of Americans using diet to lose weight. Although the mechanistic link(s) between fat metabolism, weight loss, and cardiovascular risk are largely unknown, visceral fat accumulation is associated with cardiovascular disease risk factors including insulin resistance and hypertension. Multiple mechanisms linking weight loss to reduced cardiovascular disease have been postulated. Visceral fat reduction is associated with blood pressure lowering and it is likely that reduced inflammatory cytokines and adipokines released from fat contribute to the improved profile. Although, high low-density lipoprotein cholesterol, hypertension, and hyperglycemia are also improved with weight loss, other data indicate that obesity contributes independently to coronary disease development suggesting an independent relationship between fat and heart disease.

Other evidence suggests that decreased oxidative capacity driven by elevated energy expenditure with accumulating fat mass may represent a common link between weight gain, hypertension, and other cardiovascular disease. In this regard blood lactate is a simple, noninvasive marker of oxidative capacity. In this issue of the American Journal of Hypertension, Crawford et al. investigated the relationship between lactate and blood pressure during sudden weight loss in 40 subjects with metabolic syndrome. In this study, weight loss was achieved by providing a very low–calorie diet (VLCD; 600–800 kcal) daily for 12–20 weeks. The authors found that blood lactate was higher in obese (with and without metabolic syndrome) compared to lean subjects and was reduced by 31% after VLCD in metabolic syndrome. Interestingly, there was a strong association between the change in blood pressure (diastolic and mean) and the magnitude of lactate reduction induced by VLCD after correcting for age, gender, body mass index change, and baseline lactate. In contrast to others, there was no relationship between lactate and changes in the other risk factors associated with obesity (i.e., insulin resistance, glucose, and triglycerides), suggesting an independent relationship between lactate and body weight.

Taken together, these results support the conclusion that insufficient oxidative capacity contributes to obesity hypertension and may represent an important, mechanistically unexplored link between weight loss and risk reduction. Importantly, the strengths of the study are derived from the ability to control for variability and compliance with a feeding trial design for weight loss. In this context, all subjects were given meals of similar caloric content. However, it is difficult to interpret whether subjects developed differing catabolic responses to the diet because these diets were not designed relative to baseline caloric need, macronutrient content, and energy expenditure as it changes with weight loss. Thus, future studies comparing the catabolic consequences of the different modes of weight loss (dietary and surgical) that may impact oxidative capacity and hence blood pressure regulation during metabolic syndrome are warranted.

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