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Commentary

Obesity and Inflammation: One Size Never Fits All

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Chronic, low-grade adipose tissue inflammation has long been associated with obesity, and pro-inflammatory adipokines can contribute to circulating levels of inflammatory markers (Frikke-Schmidt et al., 2016; Xu et al., 2003). Excess visceral adipose tissue is an independent risk factor for cardiovascular disease and type 2 diabetes and represents a stronger correlate to these diseases than subcutaneous adipose tissue (Frikke-Schmidt et al., 2016). Rakotoarivelo and colleagues challenge the observations that adipose tissue inflammatory cytokine profiles correlate with obesity and/or diabetes status in a manuscript in *EBioMedicine* (Rakotoarivelo et al., 2018). In this study, the visceral (omental) and subcutaneous adipose tissue cytokine profiles of 89 bariatric surgery patients were assessed and compared to the profiles of 13 control patients. Consistent with previous observations, they observed that BMI was highly significantly correlated with waist circumference and that cytokine profiles differed between subcutaneous and omental fat. However, in contrast to other studies, they report an overall lack of correlation between pro-inflammatory cytokine profiles and diabetes and/or obesity status. Protein and gene expression levels of several cytokines associated with obesity were examined, and no differences were observed between control and obese patients in either fat depot. Although *IL1B* expression was significantly elevated in the omental fat of some obese patients, it did not correlate with their diabetes status. Despite the observed increase in *IL1B* expression in some patients, other obese patients did not express *TNF*, *IL6*, or *IL1B* in their visceral fat. Based on these observations, Rakotoarivelo and colleagues propose that the chronic adipose tissue inflammation in human obesity is "…not predicted by a single mediator, but rather includes a large spectrum of possible profiles (Rakotoarivelo et al., 2018)." This assertion is certainly accurate, as the scientific community has realized for some time that the molecular and cellular effects of obesity are manifested differently from individual to individual.

While the conclusions provided by Rakotoarivelo and coworkers have merit, readers must also consider the limitations of the current study. Perhaps the most obvious limitation of the current study is the timing of biopsies in the obese patients. The omental and subcutaneous biopsies were performed at the time of bariatric surgery, after the patients had (presumably) been on a pre-operative very-low calorie diet for two weeks. This pre-operative diet is used for several reasons, including intrahepatic fat reduction, and therefore, reduction of the risk of surgical complications (Colles et al., 2006; Van Nieuwenhove et al., 2011). In addition to reducing intrahepatic fat, the very-low calorie diet also reduces the volume of the visceral and subcutaneous fat depots (Colles et al., 2006; Collins et al., 2011). If the patients in this study demonstrated a loss of fat mass, it is possible that the tissue inflammatory profiles were improved, thereby influencing the results. Another fact to note is that omental fat comprises a small proportion of total visceral adipose tissue mass (Frikke-Schmidt et al., 2016). At least 75% of the obese diabetic patients were on medications for hypertension and/or diabetes, which could definitely affect adipose inflammatory status. The lack of suitability of a 'one size fits all' approach for treating obesity and its comorbidities is further highlighted by these results. Additional, well-controlled studies are required to determine whether the lack of correlation observed in this study is independent of pre-operative alterations in adipose tissue metabolism.

Disclosure

The author declared no conflicts of interest.

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