RESPONSE TO COMMENT ON DALMAS ET AL.

Intima-Media Thickness in Severe Obesity: Links With BMI and Metabolic Status but Not With Systemic or Adipose Tissue Inflammation. Diabetes Care 2013;36:3793–3802

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We appreciate that our study has been read with such interest by Dogru et al. (1). The main objective of our study (2) was to evaluate the relationships between inflammation, especially in adipose tissue, and subclinical atherosclerosis. Here, we would like provide some clarification.

First, to avoid a confounding effect of type 2 diabetes on cytokine concentration or on adipose tissue inflammation, we have excluded diabetic subjects in the analysis of relationships between bioclinical phenotypes, inflammation, and arterial intima-media thickness (IMT).

Second, in the series of analyses showing that BMI and age are independent factors associated with IMT, dyslipidemia and the presence or absence of hypolipidemic medications was taken into account. In the multivariate model taking into account subcutaneous adipose tissue $CD68^+$ cell number, age, sex, BMI, glycemia, total cholesterol, LDL-cholesterol, and blood pressure, adding hypolipidemic medications did not influence the results.

Third, we agree that abnormal glucose tolerance could have an impact on systemic inflammation. For technical and ethical reasons, we did not perform oral glucose tolerance tests for all subjects of the cohort, especially for morbidly obese patients. This is a limitation of our study. Nevertheless, we did not find any significant correlation between surrogates of glucose intolerance, such as homeostasis model assessment of insulin resistance or HbA_{1c}, and systemic markers of inflammation.

Fourth, considering hypertension as a potential confounding factor, we mentioned the prevalence of hypertension in each group of our study. Furthermore, we have taken into account either the presence of hypertension or the blood pressure levels (systolic and diastolic) in the multivariate models. Moreover, antihypertensive medications did not influence the results of these models.

Finally, considering the importance of nonalcoholic fatty liver disease in context of systemic inflammation and its impact on cardiovascular system, we have analyzed the nonalcoholic fatty liver disease activity score (3) in liver biopsies performed during gastric surgery for most obese patients. No significant correlation was found among the grade of nonalcoholic fatty liver disease and IMT (carotid or femoral artery IMT) or markers of systemic inflammation.

We consider that our study has analyzed thoroughly the potential confounding factors present in severe obesity in the relationships between IMT and inflammation, and we found that macrophage infiltration in subcutaneous or visceral adipose tissue was not related to carotid or femoral artery IMT. However, it is undeniable that these data remain to be confirmed in other human populations, with distinct anthropomorphic, metabolic, and cardiovascular risk phenotypes.

Duality of Interest. No potential conflicts of interest relevant to this article were reported.

References

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