The Effect of Specific Allergen Inhalation on Adipokine Level

Can Adiponectin Oligomers and Hormones Be a Factor?

To the Editor:

I read with great interest the original research article in CHEST (February 2009) by Sood et al.1 The authors searched the effect of specific allergen inhalation on adiponectin, leptin, and the adiponectin-to-leptin ratio in mild asthmatic patients and found that specific allergen inhalation had no effect on serum adipokine levels. Previously, Unal et al2 showed that serum leptin levels were significantly higher in 26 adult patients with allergic rhinitis during a symptomatic period than in a control group. Also, Radon et al3’s epidemiologic findings confirmed a linear positive association between serum leptin level and prevalence of allergic sensitization (highest vs lowest quartile odds ratio 6.7, 95% CI 2.0-22.4) especially in subjects with farm contact. In combination with experimental data in animals, these findings suggest a link between adipokines and allergy.

I wish to express my concerns about two aspects of their work. As it has been speculated that sex hormones could affect adiponectin levels, I think they sought more to minimize potential confounders. In his previous study, Sood et al4 showed that in women, but not men, a lower serum adiponectin concentration is associated with asthma. Estrogen and progestosterone may affect the adiponectin concentration by controlling adipose tissue metabolism and estrogen, which has been shown to alter the action of lipoprotein lipase and hormone-sensitive lipase in adipocytes. There is also in vitro evidence that estrogen decreased adiponectin expression in adipocytes.5 In the present study, as most of the subjects were women in the premenopausal stage, this phase of their menstrual cycle might also be important.

Second, in the present study only total adiponectin concentration had been measured. Adiponectin circulates as multiple oligomer forms such as low-molecular-weight trimer, medium-molecular-weight hexamer, and high-molecular-weight (HMW) multimer (12-18 mers).6 It has been demonstrated that the lower plasma concentration of adiponectin in men could be due to a smaller amount of HMW has been reported to decline in response to testosterone.7 In the present study only total adiponectin concentration had been measured. Adiponectin circulates as multiple oligomer forms such as low-molecular-weight trimer, medium-molecular-weight hexamer, and high-molecular-weight (HMW) multimer (12-18 mers).6 It has been demonstrated that the lower plasma concentration of adiponectin in men could be due to a smaller amount of HMW multimer than in women.8 In summary, application of more strict inclusion criteria considering sex hormones and investigating circulating oligomeric isofoms of adiponectin could help us to understand underlying mechanisms for asthma and adipokines better.

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Financial/nonfinancial disclosures: The author has reported to CHEST that no potential conflicts of interest exist with any companies/organizations whose products or services may be discussed in this article.

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Response

To the Editor:

We would like to thank Dr Ozol from Ankara, Turkey, for his insightful comments related to the role of adipokines in human asthma in general and to our published article in particular (February 2009). Although not well studied, it is possible that specific phases of the menstrual cycle may differentially affect systemic concentrations of specific adipokines in women. Therefore, for menstruating women, we limited all study testing within 3 to 14 days following the cessation of menstrual flow to minimize the potential effect of the lutal menstrual phase on serum adiponectin concentrations. However, we were not powered to study the interaction between sex hormones and inhalational allergen challenge on adipokine response in our study.

We also agree with Dr Ozol’s astute observation that our study measured total adiponectin concentrations and not the various isomeric forms. Some studies suggest that the high-molecular-weight (HMW) isoform of adiponectin may be the most biologically active form of adiponectin in regulating insulin resistance. Whether the HMW isoform of adiponectin is more active than other adiponectin isoforms for asthma is currently not known. Our study therefore was unable to assess, for instance, whether subjects with asthma had overall lower diurnal curves of HMW adiponectin isoform compared with controls.

Despite the above limitations, our study successfully met its objective to replicate the relevant mouse experiments by Shore et al. that measured serum adipokine response to allergen challenge in sensitized mice. Of note, these experiments were performed on mice of both sexes, without measurement of either systemic sex hormones or of systemic adiponectin isoforms. These issues nevertheless need additional research in both animal and human asthma in the future. Finally, we could not agree more with Dr Ozol’s conclusions that “considering sex hormones and investigating circulating oligomeric isoforms of adiponectin could help us understand the underlying mechanisms for asthma and adipokines better.”

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Financial/nonfinancial disclosures: The authors have reported to CHEST the following conflicts of interest: Dr Sood has received grants from the University of New Mexico and the National Institutes of Health. The other authors have reported no conflicts of interest exist with any companies/organizations whose products or services may be discussed in this article.

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