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 al2 showed that in women, but not men, a lower serum adiponectin concentration is associated with asthma. Estrogen and progesterone 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 the HMW form of adiponectin. Secretion of HMW adiponectin has been reported to decline in response to testosterone.7 In a cross-sectional study of 32 healthy women (12 premenopausal, 10 postmenopausal, and 10 early pregnant), they found that serum concentrations of total and HMW adiponectin were highest in postmenopausal women and lowest in pregnant women with regard to estrogen level.8 In summary, application of more strict inclusion criteria considering sex hormones and investigating circulating oligomeric isoforms of adiponectin could help us to understand underlying mechanisms for asthma and adipokines better.

Duygu Ozol, MD
Ankara, Turkey

Affiliations: From the Department of Pulmonology, Fatih University Faculty of Medicine.

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.

Correspondence to: Duygu Ozol, MD, Department of Pulmonology, Fatih University Faculty of Medicine, Hsdere cad. no:145 06510 Ankara, Turkey; e-mail: dozol@hotmail.com
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 luteal menstrual phase on serum adipokine 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 is 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.”

Akshay Sood, MD, MPH, FCCP
Clifford Qualls, PhD
JeanClare Seagrave, PhD
Christine Stidley, PhD
Tereassa Archibeque, RRT
Marianne Berrick, PhD
Mark Schuyler, MD, FCCP
Albuquerque, NM

Affiliations: From the Department of Medicine (Drs Sood and Stidley, Ms Archibeque, and Drs Berrick and Schuyler) and the Clinical Translational Sciences Center (Dr Qualls), University of New Mexico School of Medicine; and the Experimental Toxicology Program (Dr Seagrave), Lovelace Respiratory Research Institute.

Funding: This work was supported in part by the National Institutes of Health [Grants NCI R01 CA109667 and 1 R01 HL07582-01 A1] and Grant UNM-RAC-C-2929.

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.

Correspondence to: Akshay Sood, MD, MPH, FCCP, University of New Mexico School of Medicine, Department of Medicine, 1 University of New Mexico, MSC 10 5550, Albuquerque, NM 87131-0001; e-mail: asood@salud.unm.edu

© 2010 American College of Chest Physicians. Reproduction of this article is prohibited without written permission from the American College of Chest Physicians (www.chestjournal.org/site/misc/reprints.xhtml). DOI: 10.1378/chest.09-2621

References


www.chestjournal.org

CHEST / 137 / 2 / FEBRUARY, 2010 499

© 2010 American College of Chest Physicians.

REFERENCEs


© 2010 American College of Chest Physicians.

REFERENCES