was not addressed by Twedt and colleagues, as it was not a focus of their study. Further research on this topic is clearly needed.

We agree with Dr. Kawada that the effect of mental health in moderating the relationship between sleep and metabolic disease is an important topic for future research; however, this topic was beyond the scope of our recent work.

Finally, Dr. Song draws attention to the potential for obesity to modify the association between sleep apnea and glycemia. To investigate this, we first constructed a model including an obesity × sleep apnea interaction. Although both sleep apnea and obesity were predictors of glycemic status, we found no evidence of an obesity × sleep apnea interaction ($P = 0.68$). Furthermore, the ethnicity × sleep apnea interaction persisted ($P = 0.03$) after adding the obesity × sleep apnea interaction, suggesting any obesity × sleep apnea effect does not explain the effect modification by ethnicity that we found. Finally, we found no evidence that the ethnicity × sleep apnea interaction varies by obesity, although we were almost certainly underpowered to detect such a three-way interaction. ■

Author disclosures are available with the text of this letter at www.atsjournals.org.

Jessie P. Bakker, Ph.D.
Jia Weng, Ph.D.
Rui Wang, Ph.D.
Susan Redline, M.D., M.P.H.
Brigham and Women's Hospital
Boston, Massachusetts
and
Harvard Medical School
Boston, Massachusetts

Naresh M. Punjabi, M.D., Ph.D.
Johns Hopkins University School of Medicine
Baltimore, Maryland

Sanjay R. Patel, M.D., M.S.
Brigham and Women's Hospital
Boston, Massachusetts
and
Harvard Medical School
Boston, Massachusetts

References

1. Bakker JP, Weng J, Wang R, Redline S, Punjabi NM, Patel SR; The Multi-Ethnic Study of Atherosclerosis. Associations between obstructive sleep apnea, sleep duration, and abnormal fasting glucose. *Am J Respir Crit Care Med* 2015;192:745–753.
2. Kripke DF, Hahn EK, Grizas AP, Wadiak KH, Loving RT, Poceta JS, Shadan FF, Cronin JW, Kline LE. Wrist actigraphic scoring for sleep laboratory patients: algorithm development. *J Sleep Res* 2010;19:612–619.
3. Signal TL, Gale J, Gander PH. Sleep measurement in flight crew: comparing actigraphic and subjective estimates to polysomnography. *Aviat Space Environ Med* 2005;76:1058–1063.
4. Wang D, Wong KK, Dungan GC II, Buchanan PR, Yee BJ, Grunstein RR. The validity of wrist actimetry assessment of sleep with and without sleep apnea. *J Clin Sleep Med* 2008;4:450–455.
5. Gottlieb DJ, Punjabi NM, Newman AB, Resnick HE, Redline S, Baldwin CM, Nieto FJ. Association of sleep time with diabetes mellitus and impaired glucose tolerance. *Arch Intern Med* 2005;165:863–867.
6. Ayas NT, White DP, Al-Delaimy WK, Manson JE, Stampfer MJ, Speizer FE, Patel S, Hu FB. A prospective study of self-reported sleep duration and incident diabetes in women. *Diabetes Care* 2003;26:380–384.
7. Twedt R, Bradley M, Deiseroth D, Althouse A, Facco F. Sleep duration and blood glucose control in women with gestational diabetes mellitus. *Obstet Gynecol* 2015;126:326–331.

Comment on Expression of Concern: c-Kit Is Essential for Alveolar Maintenance and Protection from Emphysema-like Disease in Mice

To the Editor:

We would like to comment on the concerns about our article that was originally published in the June 15, 2011, issue of *American Journal of Respiratory and Critical Care Medicine* (1).

In that article, we provided genetic, morphologic, histologic, and physiological evidence that c-Kit is critical for alveolar maintenance. After the publication of the article, we became aware that there may have been technical problems with the physiological assessment. The main concern was with our reported finding that pulmonary compliance increased in c-Kit mutant mice at 14 weeks. This led to the publication of an Expression of Concern in the *Journal* (2).

We, therefore, had an independent laboratory at Duke University, under the supervision of Herman Staats, Ph.D., repeat the experiment in question. The independent laboratory has replicated the key observation that c-Kit–deficient mice have increased lung compliance at 14 weeks of age (C57BL/6J control mice = 0.091 ± 0.001 ml/cm H_2O and c-Kit mutant mice = 0.122 ± 0.002 ml/cm H_2O, values are mean + SEM, n = 4/group). This difference is statistically significant ($P < 0.001$) as determined by analysis of variance followed by all pairwise multiple comparison procedures (Holm-Sidak method). These values are similar to those reported in the original article for 14-week-old mice (C57BL/6J control mice = 0.095 ± 0.003 ml/cm H_2O and c-Kit mutant mice = 0.132 ± 0.005 ml/cm H_2O, values are mean + SEM, n = 5/group). Thus, these findings validate the originally reported findings in the article.

Taking into consideration the other data presented in the article, we believe the data are valid and confirm the original conclusions of the article. ■

John W. Hollingsworth, M.D.
Duke University Medical Center
Durham, North Carolina

George Leikauf, Ph.D.
University of Pittsburgh
Pittsburgh, Pennsylvania

James Y. Lindsey, M.D.
University of Chicago
Chicago, Illinois

Holger Schulz, Ph.D.
German Research Center for Environmental Health & Comprehensive Pneumology Center Munich (CPC-M)
Munich, Germany

References

1. Lindsey JY, Ganguly K, Brass DM, Li Z, Potts EN, Degan S, Chen H, Brockway B, Abraham SN, Berndt A, *et al*. c-Kit is essential for alveolar maintenance and protection from emphysema-like disease in mice. *Am J Respir Crit Care Med* 2011;183:1644–1652.
2. Expression of concern: TLR4 is necessary for hyaluronan-mediated airway hyperresponsiveness after ozone inhalation; c-Kit is essential for alveolar maintenance and protection from emphysema-like disease in mice. *Am J Respir Crit Care Med* 2015;192:771.

Retraction: Maternal Exposure to Particulate Matter Increases Postnatal Ozone-induced Airway Hyperreactivity in Juvenile Mice

The authors of an article published in 2009 (1) have informed the *Journal* that, as a result of an institutional inquiry, they had reexamined the raw data used in their article and determined that the results reported in Figures 2, 4, and 5 were unreliable. They were unable to repeat the studies reported in Figures 2 and 4 due to lack of materials. Therefore, the authors have requested that the paper be retracted. The authors apologize to the *Journal* and to its readers. ■

Reference

1. Auten RL, Potts EN, Mason SN, Fischer B, Huang Y, Foster WM. Maternal exposure to particulate matter increases postnatal ozone-induced airway hyperreactivity in juvenile mice. *Am J Respir Crit Care Med* 2009;180:1218–1226.