Cysteine as a Biomarker for Obstructive Sleep Apnea

To the Editor:

In a recent study in CHEST (February 2011), Cintra and colleagues’ nicely demonstrated the relationship between obstructive sleep apnea (OSA) and cysteine. In their study, cysteine levels were significantly elevated in patients with OSA regardless of their BMI, compared with the control group. Effective treatment of OSA with continuous positive airway pressure (CPAP) resulted in a significant reduction in cysteine levels. We congratulate the authors on their well-designed study and success in introducing a marker if deemed reproducible. A good marker in OSA will demonstrate of relapse after discontinuation of CPAP is needed. This will also provide information about a good cutoff value for the marker if deemed reproducible. A good marker in OSA will have the ability to reduce the need for expensive polysomnography as a screening tool or possibly help monitor response to, and/or compliance with, CPAP treatment. So far, cysteine lacks specificity and does not fulfill those objectives.

A reproduction of this study’s results in a large, randomized, controlled study with measurement of cysteine levels (and maybe other candidate markers) before and after CPAP treatment and demonstration of relapse after discontinuation of CPAP is needed. This will also provide information about a good cutoff value for this marker if deemed reproducible. A good marker in OSA will have the ability to reduce the need for expensive polysomnography as a screening tool or possibly help monitor response to, and/or compliance with, CPAP treatment. So far, cysteine lacks specificity and does not fulfill those objectives.

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Response

To the Editor:

I read with interest the comments of Drs Alrajab, Uysal, and Jenks about our article, and I appreciate the contribution on our work. However, I would like to clarify some important points.

First, I agree that tobacco smoking may influence homocysteine and cysteine concentration, which is the reason why smoking was considered an exclusion criterion in this study and is not considered a confounding factor in our results. Moreover, the mechanisms involved in the relationship between smoking and homocysteine/cysteine concentration are still under investigation, and cigarette smoking seems to be a strong determinant of plasma homocysteine level, but the impact on plasma cysteine level should be better demonstrated.

Second, I also agree that cysteine may be affected by many other conditions, such as arterial hypertension and age, and could be considered a risk factor for atherosclerosis in patients with hyperlipidemia. Moreover, obstructive sleep apnea (OSA) may lead to a number of cardiovascular consequences, such as arterial hypertension and atherosclerosis. Therefore, it seems to be another “chicken and egg” puzzle. In order to clarify this confusing relationship among cysteine, cardiovascular consequences, and OSA, we performed a longitudinal study with an effective continuous positive airway pressure treatment of 6 months, with a minimum usage of 5.4 h/night. Continuous positive airway pressure significantly decreased plasma cysteine levels after 6 months of treatment, suggesting that OSA directly affected cysteine plasma levels.

Third, the mechanisms explaining the increase in plasma cysteine levels and the corresponding absence of changes in homocysteine concentration and the related vitamin profiles are under investigation, but based on experiments by Perry et al.3 OSA could directly affect cysteine concentration as mentioned in the “Discussion” section of the article. In this study, the combination of sleep and OSA were signifi cantly elevated in patients with OSA regardless of their BMI, compared with the control group. Effective treatment of OSA with continuous positive airway pressure (CPAP) resulted in a significant reduction in cysteine levels. We congratulate the authors on their well-designed study and success in introducing a marker if deemed reproducible. A good marker in OSA will demonstrate of relapse after discontinuation of CPAP is needed. This will also provide information about a good cutoff value for the marker if deemed reproducible. A good marker in OSA will have the ability to reduce the need for expensive polysomnography as a screening tool or possibly help monitor response to, and/or compliance with, CPAP treatment. So far, cysteine lacks specificity and does not fulfill those objectives.

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