Letter to the Editor

# Exhaled nitric oxide and disease progression in chronic obstructive pulmonary disease

**Mauro Maniscalco**

Section of Respiratory Medicine, Hospital S. Maria della Pietà, Casoria, Naples, Italy
*Correspondence to:* Mauro Maniscalco, M.D, Ph.D. Largo delle Mimose 1, 80131 Napoli, Italy. Email: mauromaniscalco@hotmail.com.

Submitted Dec 10, 2014. Accepted for publication Dec 11, 2014.
doi: 10.3978/j.issn.2072-1439.2015.01.19
**View this article at:** http://dx.doi.org/10.3978/j.issn.2072-1439.2015.01.19

***Dear Editor,***

I have read the review article with great interest from Dr. Show and colleagues published on the November issue of this Journal (1). In their article the Authors described several radiological and biological markers (biomarkers) that may be useful in predicting disease progression in chronic obstructive pulmonary disease (COPD). Surprisingly, exhaled nitric oxide (FeNO) was not mentioned in this review. It is well known that the NO signal magnitude in COPD patients is considerably smaller than in asthma. This is essentially due to cigarette smoke, the major causative agent, which dramatically masks any tendency toward a disease-related rise in FeNO levels. However, in some COPD phenotypes this exhaled biomarker has been suggested as a potential tool in monitoring the stability of the disease, the response to the therapy and the possible progression to COPD of other diseases (2). In fact, in a recent study FeNO was shown to be a good biomarker of eosinophilic inflammation in COPD patients with exacerbation (3). Furthermore, in COPD patients with a deficiency of Alpha-1 antitrypsin (AAT), FeNO levels are related to the reduced concentration of AAT in plasma, which is associated to pulmonary function impairment (4,5). In these patients, exhaled NO might be very important to monitor a possible progression of airways inflammation. Finally, monitoring FeNO levels may be useful to detect conditions associated to COPD and related to the severity of the disease such as pulmonary arterial hypertension (6).

In conclusion, although routine monitoring of FeNO in COPD is less established than in other inflammatory diseases such as asthma, I believe that this biomarker should be mentioned as a potential tool to detect disease progression in COPD.

## Acknowledgements

*Disclosure:* The author declares no conflict of interest.

## References

1. Shaw JG, Vaughan A, Dent AG, et al. Biomarkers of progression of chronic obstructive pulmonary disease (COPD). J Thorac Dis 2014;6:1532-47.
2. Malerba M, Radaeli A, Olivini A, et al. Exhaled nitric oxide as a biomarker in COPD and related comorbidities. Biomed Res Int 2014;2014:271918.
3. Soter S, Barta I, Antus B. Predicting sputum eosinophilia in exacerbations of COPD using exhaled nitric oxide. Inflammation 2013;36:1178-85.
4. Malerba M, Clini E, Radaeli A, et al. Exhaled nitric oxide in patients with alpha 1 antitrypsin (AAT) deficiency. Monaldi Arch Chest Dis 2001;56:175-6.
5. Malerba M, Ragnoli B, Radaeli A. Exhaled nitric oxide levels in alpha-1-antitrypsin PiMZ subjects. J Intern Med 2009;265:382-7.
6. Malinovschi A, Henrohn D, Eriksson A, et al. Increased plasma and salivary nitrite and decreased bronchial contribution to exhaled NO in pulmonary arterial hypertension. Eur J Clin Invest 2011;41:889-97.

**Cite this article as:** Maniscalco M. Exhaled nitric oxide and disease progression in chronic obstructive pulmonary disease. J Thorac Dis 2015;7(3);E59. doi: 10.3978/j.issn.2072-1439.2015.01.19