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Ethane as a marker of lipid peroxidation

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Keywords

COPD, peroxidation

Introduction

Oxidative stress is believed to play a role in the pathogenesis of COPD due to the number of inflammatory cells found in the airways of these patients. Alkanes, pentane and ethane (hydrocarbon byproducts of lipid peroxidation) can be measured in exhaled breath. In this study, the authors not only measure ethane in the exhalate of patients with COPD but measure nitric oxide (NO) and carbon monoxide (CO) as well. They detail a simple single breath method for doing the ethane measurement that is portable and requires little equipment, thus making it acceptable for field tests.

Comments

This paper is important for two reasons. Firstly, it describes the utility of a simple, single breath test for ethane measurement. Most investigators in the past have been concerned with fluctuations in the ambient air ethane concentration; however, by eliminating deadspace air, the authors may have found a way around the ambient air ethane quandary. A direct comparison with previously used methods would be interesting. Secondly, this paper not only supports previous work that shows elevated ethane in the breath of patients with chronic obstructive pulmonary disorder (COPD), it also confirms the negative correlation between lung function (as measured by FEV_1) and the amount of ethane exhaled

The authors note that ethane is produced in many organ systems and may be transported to the lung for elimination. Credence that exhaled ethane is a reflection of lung oxidant activity comes from results that show a reduction in ethane in the presence of inhaled steroids applied topically and have little systemic effect. The use of exhaled breath analysis to measure inflammatory activity in the airway is appealing since these tests may lead to the determination of which patients will progress to clinically

significant lung disease or those amenable to therapy. This would permit more selective targeting of this population for intervention.

Methods

Twenty-two stable, ex-smoking patients with COPD, who did not manifest response to bronchodilators on spirometric testing, were enrolled in the study. Ten of these subjects had received either inhaled steroids or oral steroids over the past 3 months. Fourteen healthy, but admittedly younger, subjects who had never smoked were used as controls. Smoking status of the participants was assessed using a urine test for nicotine.

Flow and pressure controlled exhalation was made into a reservoir, the anatomic dead space gas being discarded. The remaining sample was tested for ethane using gas chromatography. Intra- and extra-session reproducibility was 5.4% and 6.2%, respectively. Ethane concentration was flow dependent, with higher flows yielding lower results. All samples were collected between 10 and 11 LPM. Exhaled CO and NO were measured as usual with appropriate pressure and flow controls.

Results

In the nonsteroid COPD group, there was a negative correlation between exhaled ethane and FEV_1 ($r = -0.67$, $P < 0.05$). Steroid naive subjects had more than twice the ethane in their exhalate as compared to controls (2.77 ± 0.25 vs 0.88 ± 0.009 ppb: $P < 0.05$). The steroid treated patients had ethane levels of 0.48 ± 0.05 ($P < 0.01$ vs steroid naive). CO levels between steroid (5.99 ± 0.50 ppm) and nonsteroid treated (5.96 ± 0.63 ppm) were similar, but the controls were lower (2.88 ± 0.2 ppm: $P < -0.05$). The NO values for nonsteroid patients and steroid patients were 11.86 ± 1.27 and 9.11 ± 0.53 ppb, respectively (not significant). These values were higher than for the controls (6.7 ± 0.5 ppb, $P < 0.05$).

Discussion

The elevated ethane levels in patients with COPD correlated negatively with degree of obstruction as measured by FEV_1. In addition, steroid treatment reduced the ethane exhaled. These data suggest that ethane is a marker for airway inflammation and oxidative stress in patients with COPD. The failure of steroids to reduce NO and CO may relate to the lack of a steroid suppressive effect on neutrophils or the lack of eosinophilic inflammation. CO and NO may only be indirect measures of oxidative stress. Ethane on the other hand directly measures lipid peroxidative activity. Combined use of these three gases may be needed in order to better define the presence of ongoing inflammation in the airway which

may have implications for the treatment and prevention of inflammation-induced structural damage to the airways.

Additional information

References

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