



Role of Fireplaces in the Measurement of Exhaled Carbon Monoxide in Two Smokers with Chronic Obstructive Pulmonary Disease

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ABSTRACT

Carbon monoxide (CO) concentration in the body will be decreased after smoking cessation. However, confounding factors may influence the results. Exhaled CO (eCO) assessment of lung is a simple, noninvasive tool, but confounder factors such as gas fireplaces might influence results. We thus quantified the effect of them in two smokers with chronic obstructive pulmonary disease (COPD). We described one of these confounder factors in two smokers with COPD in a clinical trial study setting with IRCT201609271457N11 coding in IRCT. The amounts of eCO and carboxyhemoglobin of these patients rose while the average daily smoking decreased (in the first patient) or stopped smoking (in the second patient). We found that they had used the fireplace to heat their home. These measures decreased in both patients by discontinuing the use of the gas fireplace. The gas fireplaces influence the results of eCO assessments in smoking cessation programs.

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Introduction

Nearly 2 million people die prematurely from illness attributable to indoor air pollution from household fuel use (1). Exhaled carbon monoxide (eCO) concentration is measured to confirm the reduction of

smoking through self-reporting in tobacco weaning programs (2, 3). CO in exhaled air can be confounded by many factors including variations in diet, physical exercise, exposure to atmospheric pollution, time of day, time since the last cigarette, and last but not least,

and environmental tobacco smoke exposure (ETS) (4). We described one of these confounder factors in two smokers in our clinical trial research.

The presented cases were observed in a clinical trial entitled “efficacy of guided self-change add-on nicotine replacement therapy for cigarette smoking cessation in chronic obstructive pulmonary disease (COPD) patients: a randomized controlled clinical trial.” The study was conducted in three groups involving 45 cases using guided self-change (psychological intervention), nicotine replacement therapy, and both, applying block randomized clinical trial. The eCO was assessed by a smokerlyzer (bedfont company, England) to confirm self-reported smoking by the patients. These values are in non-smokers 0-6 ppm, danger zone 7-10 ppm, smokers 11-15 ppm, infrequent smokers 16-25 ppm, addicted smokers 26-35 ppm, heavily addicted smokers 36-50 ppm, and in dangerously addicted smokers higher than 50 ppm (5). These patients were presented because of unexpected confounders of eCO for the researchers in this field to identify important confounders such as the gas fireplace. They were resident of urban area and used fireplaces with stove pipe and without glass cover.

Case Report

Case 1: The first patient was a COPD 63-year-old man with a 45-year history of smoking (one packet per day of average) treated via guided self-change. In the first CO measurement by smokerlyzer (bedfont company, England), the amounts of eCO and carboxyhemoglobin (COHb) were 21 and 4.8 ppm, respectively. In the following week, the average daily smoking decreased to 4 cigarettes by self-reporting, but CO and COHb were 47 and 8.2 ppm at the end of the 2nd week, whereas, the average time exposure to the fireplace was 8 days before the last performing CO measurement.

To evaluate the unexpected eCO, we found that the patient had used the fireplace to heat the home. A week later, the patient reported that his average daily smoking was two cigarettes. Moreover, by discontinuing the use of the gas fireplace, the amounts of CO and COHb at the end of the 3rd week were reported 9 and 2.1 ppm, respectively.

Case 2: The second patient was a COPD 48-year-old man with a smoking history of 28 years (16 cigarette/day) who was treated via a combination of guided self-change and nicotine replacement therapy. In the first measurement, the amounts of eCO and COHb were 18 and 3.5 ppm. In the following week, he completely stopped smoking, and the amounts of eCO and COHb decreased to 11 and 2.4 ppm. In the 3rd and 4th week, he did not smoke, and his wife approved it, also, the amounts of eCO and COHb at the end of the 3rd week were reported to be 5 and 1.4 ppm. However, at the end of the 4th week, these amounts reached 22 and 4.2 ppm. It was found that the patient had used the gas fireplace to heat his house for 7 days. By discontinuing the use of the fireplace, the eCO and COHb concentrations at the end of the next week declined to 10 and 2.2 ppm, respectively.

Discussion

Indoor air pollution from solid fuel use is strongly related to COPD (1) such as fireplaces. Assessing eCO concentration has been routinely used in smoking cessation treatments for 15 years (6). It has been introduced as a valuable noninvasive biomarker of tobacco smoke daily consumption, passing through most validation studies (7). Furthermore, since the determination of eCO is non-invasive, inexpensive, and is based on immediate results, it is considered as a method of choice for clinical studies (8). As it stands, eCO is a 5-or 10-second measurement that responds to most issues related to any tool: easy to do, no contraindication, no expertise requirement,

harmlessness, and low cost (6). CO is typically measured in exhaled air, has a half-life of 4-5 hours, and a high sensitivity and specificity (7). COHb can be assessed in blood and has a half-life of 1-4 hours (9). Compared to the other techniques mentioned, eCO has several advantages. The first is the possibility to provide immediate feedback to the user. Other methods require more time-consuming chemical processing. In addition, the level of CO seems to be higher in individuals with an inflammatory airway disease such as COPD (4). Nevertheless, despite the so-called possible confounders, subjects can be successfully classified into broad categories of smoking activity by eCO levels (10). Sato et al. (11) concluded 11 ppm to be the optimal cut-off point for COPD patients. Self-reported smoking status and cigarette consumption among patients with COPD were highly consistent with eCO results (12). The prescribed 9 ppm cut-off point of the breath CO generates a sensitivity of 68% and 42% for COPD patients and healthy people, respectively. Using the prescribed cut-off point (10 ppm), the smokerlyzer produces 56% sensitivity for COPD patients and 23% for healthy people. Both monitors generate 100% specificity in both groups (13). The CO concentration of over 10 ppm shows that participants are smokers; 6-10 ppm represents sporadic smoking, and concentration of < 6 represents non-smoking (2, 3). CO half-life is dependent on environmental CO (14, 15). ECO can be confounded by many factors including exposure to atmospheric pollution, time of day, time since the last cigarette, and last but not least, ETS (4) and the fireplace was one of the environmental CO producers that confounded in eCO assessment in our study. The strong agreement between self-reported smoking and eCO indicates that self-reported smoking can be considered a reliable measure (16), but when self-reported measurements are incompatible with eCO results, confounders should be considered. Although the investigation of eCO in cigarette smoking

assessment has high reliability, unexpected confounders of eCO such as gas fireplaces and plays a large role on increasing it.

Conflict of Interests

Authors have no conflict of interests.

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References

1. Sood A. Indoor fuel exposure and the lung in both developing and developed countries: An update. *Clin Chest Med* 2012; 33(4): 649-65.
2. Cropsey KL, Jackson DO, Hale GJ, Carpenter MJ, Stitzer ML. Impact of self-initiated pre-quit smoking reduction on cessation rates: Results of a clinical trial of smoking cessation among female prisoners. *Addict Behav* 2011; 36(1-2): 73-8.
3. Korte KJ, Capron DW, Zvolensky M, Schmidt NB. The Fagerstrom test for nicotine dependence: do revisions in the item scoring enhance the psychometric properties? *Addict Behav* 2013; 38(3): 1757-63.
4. Montuschi P, Kharitonov SA, Barnes PJ. Exhaled carbon monoxide and nitric oxide in COPD. *Chest* 2001; 120(2): 496-501.
5. Bedfont® Scientific Ltd. Smokerlyzer® Range [Online]. [cited 2017]; Available from: URL: <https://www.bedfont.com/file/2488-LAB679%20Smokerlyzer%20Manual%20Issue%203.pdf>
6. Silkoff PE, Stevens A, Pak J, Bucher-Bartelson B, Martin RJ. A method for the standardized offline collection of exhaled nitric oxide. *Chest* 1999; 116(3): 754-9.
7. Monnikhof E, van der V, van der Palen J, Mulder H, Pieterse M, van Herwaarden C, et al. The effect of a minimal contact smoking cessation programme in out-patients with chronic obstructive pulmonary disease: a pre-post-test study. *Patient Educ Couns* 2004; 52(3): 231-6.
8. Javors MA, Hatch JP, Lamb RJ. Cut-off levels for breath carbon monoxide as a marker for cigarette smoking. *Addiction* 2005; 100(2): 159-67.
9. Fortmann SP, Rogers T, Vranizan K, Haskell WL, Solomon DS, Farquhar JW. Indirect measures of cigarette use: Expired-air carbon

- monoxide versus plasma thiocyanate. *Prev Med* 1984; 13(1): 127-35.
10. Etzel RA. A review of the use of saliva cotinine as a marker of tobacco smoke exposure. *Prev Med* 1990; 19(2): 190-7.
 11. Sato S, Nishimura K, Koyama H, Tsukino M, Oga T, Hajiro T, et al. Optimal cutoff level of breath carbon monoxide for assessing smoking status in patients with asthma and COPD. *Chest* 2003; 124(5): 1749-54.
 12. Wilson JS, Elborn JS, Fitzsimons D, McCrum-Gardner E. Do smokers with chronic obstructive pulmonary disease report their smoking status reliably? A comparison of self-report and bio-chemical validation. *Int J Nurs Stud* 2011; 48(7): 856-62.
 13. Christenhusz L, de Jongh F, van der V, Pieterse M, Seydel E, van der Palen J. Comparison of three carbon monoxide monitors for determination of smoking status in smokers and nonsmokers with and without COPD. *J Aerosol Med* 2007; 20(4): 475-83.
 14. Biochemical verification of tobacco use and cessation. *Nicotine Tob Res* 2002; 4(2): 149-59.
 15. Kauffman RM, Ferketich AK, Murray DM, Bellair PE, Wewers ME. Measuring tobacco use in a prison population. *Nicotine Tob Res* 2010; 12(6): 582-8.
 16. Maclaren DJ, Conigrave KM, Robertson JA, Ivers RG, Eades S, Clough AR. Using breath carbon monoxide to validate self-reported tobacco smoking in remote Australian Indigenous communities. *Popul Health Metr* 2010; 8(1): 2.