Section: Research Paper

Carbon Monoxide (CO) levels in Smokers and Non-smokers: A Review

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Abstract

Smoking has long been known to contribute to a number of respiratory disorders, both etiologically and prognostically. Many subjects still smoke nonetheless, despite knowing better.

Based on the literature review, the number of people who compiled with advice to quit smoking grew further as a result of the usage of a carbon monoxide (CO) monitor to show an immediate and potentially detrimental consequence of smoking. Levels of CO in exhaled air has been widely used as an indicator of smoking cessation. Although other sources of pollution, such as exhaust gases, induce increases in the fractional concentrations of CO in expired air, measures of exhaled CO level may offer an immediate, non-invasive technique of determining smoking status. Several research studies have been conducted using breath analyser to measure end expiratory CO concentrations in smokers and non-smokers and concluded that breath analysis was rapid to asses the risk of CO poisoning. This paper will review the various research conducted on CO levels in smokers and non-smokers.

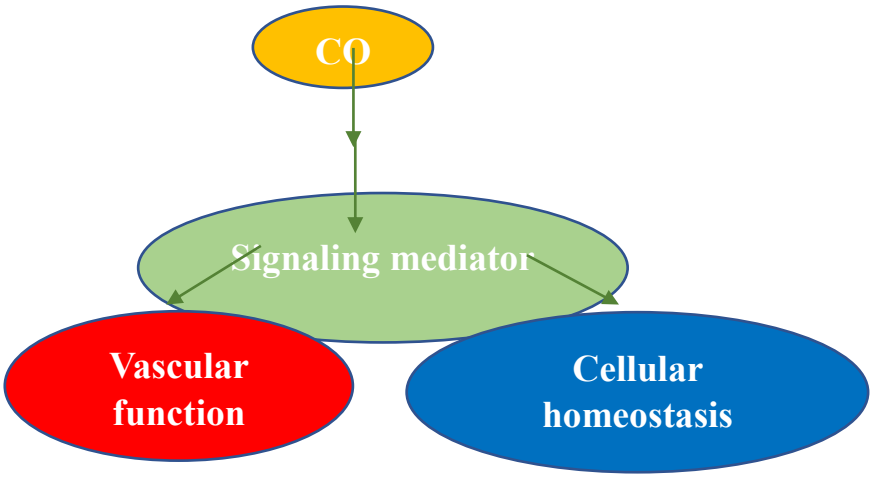
Key words: Carbon Monoxide (CO), Breath analyser, CO poisoning, Smoking Cessation

Introduction

Carbon monoxide (CO) is a low molecular weight gas, is a pervasive environmental product of organic combustion, including the burning of wood, coal, gas, and tobacco. It is also produced endogenously in the body, as the by-product of heme metabolism [1]. It binds to hemaglobin with much greater affinity than oxygen and forms carboxyhemoglobin, resulting in decreased oxygen delivery to tissues at toxicological concentrations [2]. CO levels in exhaled air have frequently been utilized as a sign of quitting smoking [3]. Tobacco smoking

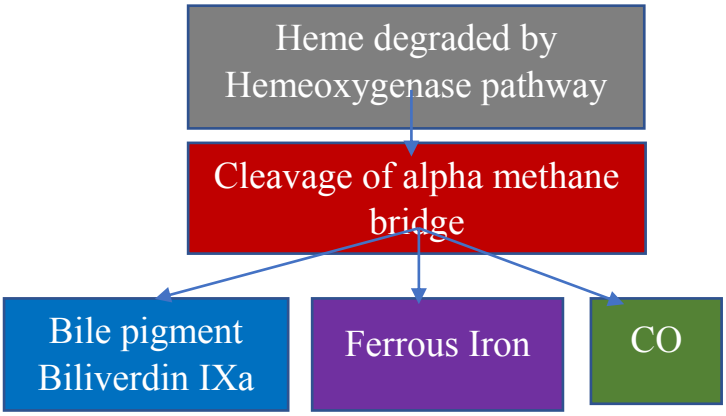
is one of the most detrimental habits in the world. There are approximately 1 billion smokers with 7 million deaths due to tobacco use every year globally [4].

Pathophysiology



Physiologically, CO acts as a signalling mediator for vascular functions and cellular homeostasis. Exhaled carbon monoxide (eCO) has been studied as a breath analysis biomarker for pathophysiological conditions, such as smoking status and inflammatory illnesses of the lungs and the other organs [5].

Biological sources of CO



There is equimolar production of CO and bilirubin for each molecule of heme degraded by cellular heme oxygenase activity. The CO thus formed diffuses into the blood and is carried via hemoglobin, and is excreted in the lungs. Therefore, CO production can be assessed clinically by measuring the rate of total body CO excretion, blood COHb levels, and end-tidal CO concentration [6]. In the absence of smoking or contaminating factors in the environment, the basal COHb in an individual is about between 0.1 and 1% [5].

Environmental CO

It is a toxic inhalation hazard, and a common contaminant of indoor and outdoor air. The main source of environmental CO is organic combustion, which includes the burning of tobacco, coal, gas and wood [7]. CO concentrations within typically range from 0.5 to 5 parts

per million (ppm), although they can go higher (up to 30ppm) [5]. Its molecular weight is similar to that of air approximately 29. It effortlessly blends with air in any ratio. It reacts strongly with oxygen, acetylene, chorine, fluorine and nitrous oxide. In the human body it reacts with hemoglobin to from carboxyhemoglobin (COHb) [8]. In densely populated urban areas, alongside roads, or close to combustion sources, ambient CO levels may be greater. CO levels can rise to 10 to 50 ppm in places with significant circulation. Cigarette smoke, which has an average CO content of 4% by volume, is a substantial source of ambient CO [5]. Smokers' exposure to high CO levels results in an average COHb of 4%, with a typical range of 3-8% [9].

Carbon Monoxide in Exhaled Breath

The origins of eCO probably reflect a process of systemic elimination where CO is diffused across the alveoli from the pulmonary circulation. Many factors may be related to CO uptake and elimination such as CO concentration, duration of exposure to CO, age, sex, exercise, minute ventilation, alveolar ventilation, total haemoglobin mass and different treatments for CO poisoning..In this regard eCO values may be related in part to HbCO values [10].

Evaluation of Smoking Status

eCO has been suggested as a useful, non-invasive diagnostic technique for medical practitioners to evaluate and track smoking status, particularly in patients who reject or underestimate their smoking status while evaluating their own health. Recent research suggests that using eCO measures instead of urine cotinine tests as a way to track smoking cessation may be more accurate and cost-effective. eCO values are typically increased in smokers with normal lung function relative to non-smokers [11].

CO levels in smokers and non-smokers

In an extensive survey by Deveci, S. E., Deveci F., Acik, Y., and Ozan, A. T. (2004) of 322 subjects (243 healthy smokers, 55 healthy non-smokers, 24 passive smokers), exhaled CO concentration was measured using the EC50 Smokerlyser. According to their findings the mean CO levels for healthy smokers was 17.13±8.50 parts per million (ppm), 3.61±2.15 ppm for healthy non-smokers and 5.20±3.38 ppm for passive smokers. In healthy smokers, there was a statistically significant positive connection between CO levels and both daily cigarette consumption and smoking duration. An overall analysis of smokers and non-smokers revealed that a threshold of 6.5 ppm had a 90% sensitivity and 83% specificity [12].

Another study conducted by Cunnington AJ, Hormbrey P (2002) using a breath analyser to measure end expiratory CO concentrations in 382 consenting subjects (Smokers & Non-Smokers) showed the range of CO concentrations in Non-Smoking group was 0-6 ppm and in the smoking group was 1-68 ppm. Smokers had a mean breath CO concentration of 16.4ppm and Non-Smokers had a mean of 1.26ppm. They concluded that breath analysis was rapid and results correlated well with CO exposure and subjects with breath CO concentrations greater than 6ppm should be assessed for the risk of CO poisoning [13].

According to Shie HG, Pan SW, Yu WK, Chen WC, Ho LI and Ko HK (2017) in a 6-year retrospective observational cohort study in smokers the amount of inhaled CO was 4.5 ppm on day 8 of an intervention programme. This significantly predicts successful smoking cessation after a year [14].

In an extensive study by Kumar R, Prakash S, Kushwah AS, Vijayan VK in 2010 compared the breath carbon monoxide (CO) levels in cigarette and bidi smokers in India. 389 smokers (241 cigarette smokers and 148 bidi smokers) had their breath CO levels tested using a portable breath CO analyser. Average breath CO levels were 15.6 +/- 7.0 ppm in smokers and 4.07 +/- 1.16 ppm in non-smokers. Average breath CO level was significantly higher in bidi smokers (18.9 +/- 7.7 ppm) compared to cigarette smokers (13.6 +/- 5.8 ppm) when total consumption of cigarette/bidi was more than five pack-years [15].

Conclusion

Use of carbon monoxide (CO) monitor to demonstrate an immediate and potentially harmful consequence of smoking can be used as a diagnostic tool for tobacco cessation program. Breath analysis is a rapid, non-invasive method and results are correlated well with carbon monoxide exposure**.**

Acknowledgements

We are thankful to Mr Raj Pandey from Vedika Infracon OPC Pvt. Ltd. for sponsoring research project titled "Efficacy of Nutritional supplement (Haras) on CO levels in smokers and Non-smokers- An observational study." This review paves the way for our future research project.

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