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Science of the Total Environment 354 (2006) 150–156

Science of the Total Environment
An International Journal for Scientific Research into the Environment and its Relationship with Humankind

www.elsevier.com/locate/scitotenv

End-expiratory carbon monoxide levels in healthy subjects living in a densely populated urban environment

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Received 16 February 2004; accepted 16 February 2005
Available online 7 April 2005

Abstract

Carbon monoxide (CO) has a high affinity for haemoglobin and is a common cause of poisoning in industry and the home. Exhaled CO levels in patients with respiratory disease have been reported but exhaled CO in a large cohort of healthy subjects grouped by age and gender has not been reported. Exhaled CO levels and spirometry lung function data were recorded from 1032 subjects at a university campus and two commercial plazas. Subjects were also asked to complete a respiratory symptom questionnaire. Ninety-eight subjects reported respiratory disease and were excluded from the study. Non-smoking male subjects ($n=508$) had higher exhaled CO levels (4.36 ± 2.54 ppm) [range 0–21 ppm] compared with female ($n=348$) subjects (3.72 ± 2.12 ppm) [range 0–14 ppm] ($p<0.0005$), and older subjects (>60 years) had lower exhaled CO levels compared with young subjects (<22 years) ($p=0.018$). Over 13% of non-smokers had an exhaled CO greater than 7 ppm. Smokers showed significantly higher exhaled CO levels compared with non-smokers ($p<0.0005$) and smokers who complained of frequent cough and sputum production had higher levels of exhaled CO compared with smokers without such complaints. Smoking history (pack-years) was directly related to age ($r=0.59$) but correlated inversely with forced expiratory flow in the 1st second (FEV_1) ($r=-0.29$) and peak expiratory flow rate (PEFR) ($r=-0.25$) ($p<0.05$). If a city's micro environmental CO concentrations and human activity patterns is available, regular monitoring of exhaled CO in healthy subjects has the potential to be used as a functional index of air pollution.

Keywords: Exhaled carbon monoxide; Smoking; Air-pollution; Healthy population

1. Introduction

Carbon monoxide (CO) is produced when there is incomplete combustion of organic material. In the human body, small quantities of CO are produced from catabolism of haemoglobin and present as carboxyhaemoglobin (COHb). The normal saturation of COHb in the blood is 0.4–0.7% (Stewart, 1975). CO is poisonous because it has an affinity for haemoglobin 200 times that of oxygen (West, 2003). Recent studies have shown that CO can cause increased leukocyte adherence and subsequent perox-

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doi:10.1016/j.scitotenv.2005.02.018

idation of brain lipid (Thom, 1993; Ischiropoulos et al., 1996). It is also suggested that exposure to CO can induce myocardial ischaemia in subjects with coronary artery disease (Wickramatillake et al., 1998), however early symptoms of carbon monoxide poisoning/exposure are similar to influenza, encouraging misdiagnoses (Knobeloch and Jackson, 1999; Walker and Hay, 1999).

Production of CO in the body is associated with oxidants and inflammatory cytokines (Paredi et al., 2002) and high exhaled carbon monoxide levels have been reported in patients suffering chronic obstructive pulmonary disease (COPD) (Paredi et al., 2000), primary ciliary dyskinesia, bronchiectasis and cystic fibrosis (Horvath et al., 2003) and patients with severe asthma (Yamaya et al., 2001). Exhaled CO is now considered a biomarker of some pulmonary diseases (Biernacki et al., 2001; Montuschi et al., 2001; Kharitonov and Barnes, 2002; Paredi et al., 2002), although it has also been shown that exhaled CO level in patients with asthma, allergic rhinitis and cystic fibrosis were not raised compared to subjects without inflammatory airway disorders (Zetterquist et al., 2002). Most studies report a sample size of less than 100 subjects, and a data base of exhaled CO in a large cohort of healthy subjects grouped by age and gender has not been reported. Such data may help to further exploit the role of exhaled CO as a biomarker of pulmonary dysfunction.

In an urban environment, the most common sources of CO are cigarette smoke and vehicle exhaust (Cunnington and Hormbrey, 2002). Exhaled CO in smokers is higher than in non-smokers (Cunnington and Hormbrey, 2002; Gourgoulianis et al., 2002). It has been suggested that exposure to microenvironmental CO levels should be considered when exhaled CO concentrations are determined for any subject cohort (Ott et al., 1992; Cunnington and Hormbrey, 2002). Exhaled CO with reference to CO exposure in a population has not been reported. This study aims to provide a data base of exhaled CO levels grouped by age, gender and smoking habit, in a city with reported micro environmental CO concentrations and human activity pattern data. Effort was made to connect the findings to causes and consequence, and to apply them to gender, smoking and age categories.

2. Materials and methods

Approval was obtained from the university's departmental research ethics committee, the university's student union, as well as from the management offices of two commercial plazas in densely populated residential estates. Written informed consent was obtained from each participant prior to the data collection.

2.1. Subjects

University subjects: An invitation to participate in a gratuitous lung function assessment session was sent to all staff and students at one of the universities in Hong Kong by mass electronic mailing. The assessment sessions were conducted at the University podium over a 2-week period.

Commercial plaza subjects: A booth was set up over two weekends at two commercial plazas. Banners inviting people to participate in a gratuitous lung function assessment were displayed.

2.2. Procedures

The nature of the study was explained and written consent from subjects obtained. Height and weight of each subject were measured. The subjects were then asked to complete a questionnaire which requested information on smoking history, known respiratory dysfunction, and frequency of respiratory symptoms (wheezing, shortness of breath on minimal exertion, cough, sputum).

Lung function parameters (Forced vital capacity FVC; Forced expiratory volume in 1 s FEV_1, peak expiratory flow rate PEFR) were measured using two Microlab 3300 spirometers (Micro Medical Ltd, Kent, UK) and exhaled carbon monoxide (CO) level measured with two Micro CO Meters (Micro Medical Ltd, Kent, UK). Oxygen saturation was determined by a finger pulse oximeter (Onyx 9500, Nonin Medical, Inc., Mn USA). Proper technique for lung function measurement was demonstrated to each subject and the best of three trials of measurement in the standing position was recorded. As recommended by Zetterquist and colleagues (Zetterquist et al., 2002) and in accord with the manufacturer's instructions, exhaled CO levels were measured by