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INDOOR CARBON DIOXIDE CONCENTRATIONS AND SBS IN OFFICE WORKERS

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ABSTRACT

Relationships between indoor carbon dioxide (CO₂) levels and mucous membrane and lower respiratory sick building syndrome (SBS) symptoms are explored in 41 office buildings from the US EPA BASE study. Elevated indoor CO₂ concentrations may indicate inadequate ventilation per occupant and elevated indoor pollutant concentrations, leading to SBS symptoms. Two CO₂ metrics were constructed: average workday indoor minus average outdoor CO₂ (dCO₂, range 6-418 ppm), and maximum indoor one-hour moving average CO₂ minus outdoor CO₂ concentrations (dCO₂MAX). Multivariate logistic regressions quantified dCO₂/SBS symptom associations, adjusting for personal and environmental factors. A dose-response relationship (p<0.05) with odds ratios per 100 ppm dCO₂ ranging from 1.2 to 1.5 for sore throat, nose/sinus, tight chest, and wheezing was observed. The dCO₂MAX/SBS regression results were similar. Implications: large increases in ventilation rate or improvements in ventilation effectiveness and/or indoor pollutant source control would be expected to decrease the prevalence of selected symptoms by up to 70-85%.

KEYWORDS: dose-response, logistic regression, sick building syndrome, ventilation.

INTRODUCTION

The primary indoor source of CO₂ in office buildings is the respiration of the building occupants. CO₂ concentrations in office buildings typically range from 350 to 2500 ppm [1]. At the concentrations occurring in most indoor environments CO₂ buildup is thought to be a surrogate for other occupant-generated pollutants, particularly bioeffluents, and for ventilation rate per occupant, but not a causal factor in human health responses. The Threshold Limit Value for 8-hour time-weighted-average exposures to CO₂ is 5000 ppm [2]. The current American Society of Heating, Refrigeration, and Air-conditioning Engineers (ASHRAE) recommended minimum ventilation rate for offices is 10 Ls⁻¹ per person, corresponding to an approximate steady state indoor concentration of 870 ppm [3], based on the assumptions that outdoor CO₂ is 350 ppm and indoor CO₂ generation rate is 0.31 L/min-person.

Sick Building Syndrome (SBS) is used to describe a set of symptoms with unidentified etiology frequently reported by workers in office buildings. The individuals who suffer from SBS report that the symptoms occur when they spend time indoors, particularly in office buildings and that the symptoms lessen while away from the building [4,5]. In this paper we concentrate on upper respiratory and mucous membrane (MM) symptoms (i.e., irritated eyes, nose, sinus, or throat), and lower respiratory (LResp) irritation (i.e., cough, tight chest, wheeze, or difficulty breathing).

CO₂ and SBS studies in the literature

In a recent review [1], about one-half of 22 studies of SBS symptoms in office buildings found that increased indoor CO₂ levels were positively associated with a statistically significant increase in the prevalence of one or more SBS symptom. SBS symptoms associated with CO₂ included headache, fatigue, eye symptoms, nasal symptoms, respiratory tract symptoms, and total symptom scores. Seventy percent of studies of mechanically ventilated and air conditioned buildings found a significant association between an increase in CO₂ and SBS symptoms. Building ventilation were also associated with SBS symptoms.

METHODS

The BASE Study

The data analyzed in this paper were collected in 41 large U.S office buildings from 1994 to 1996, a subset of 100 randomly-selected buildings studied from 1994-1998 by the U.S. Environmental Protection Agency in the Building Assessment Survey and Evaluation (BASE) study [6,7,8]. These buildings were at least partially mechanically ventilated and air conditioned.

BASE buildings were studied during one-week periods of the winter or summer. The BASE protocol is discussed fully elsewhere [9,10]. A questionnaire collected information on the occupants' perceptions of their workplace environments, job characteristics, and health and well-being (including symptoms associated with SBS). Environmental data were measured during the week of questionnaire administration.

At each office building, CO₂, volatile organic compounds (VOCs), temperature, relative humidity (RH), were measured at three indoor locations and outdoors. CO₂ and indoor temperature were collected as 5-minute averages. VOC canister samples were collected and analyzed by gas chromatograph-mass spectrometry for 56 VOC species. We calculated spatial-average pollutant concentrations and average temperatures based on data from the three measurement sites. Two CO₂ metrics were calculated. One metric (dCO₂) is the time-averaged workday difference between the indoor and outdoor CO₂ concentrations. The second metric (dCO₂MAX) is the maximum indoor one-hour moving average CO₂ minus average outdoor CO₂ concentrations.

A thermal exposure variable (°C-hours) was calculated as the integrated difference between 5-minute-average-temperature and 20°C, normalized to 10 hours of exposure. The indoor workday-average RH was calculated. The eight buildings with RH < 20%

were excluded from the regression analyses discussed below, since by definition MM or LResp symptoms due to very low RH would not be considered SBS symptoms.

Associations between BASE VOCs and SBS symptoms have been discussed elsewhere [11]. One VOC, 1,2,4 trimethylbenzene (TMB), found in infiltrating outdoor air and originating from automotive sources, was found to have statistically significant associations with a number of mucous membrane and lower respiratory symptoms. Other sources of TMB in office buildings may include carpet, undercarpet, and building materials [11]. TMB was selected as a covariable in the regression models presented in order to adjust for the potential affects of ambient automotive sources on the SBS symptoms. The geometric mean TMB concentration across the 41 buildings was 1.2 ppb and the geometric standard deviation was 3.0.

The BASE questionnaire confidentially collected occupant information, including gender, age, smoking status, job characteristics, perceptions about the indoor environment, and health and well-being. The symptoms elicited from the questionnaire included: irritation of eyes, nose, and throat; chest tightness, difficulty breathing, cough, or wheezing; fatigue; headache; eyestrain; and dry or itchy skin. To qualify as a SBS symptom in the analyses presented here, the occupant must have had a reported symptom occurrence of at least 1-3 days per week during the month previous to the study and that the symptom must have shown improvement when he/she was away from work.

Statistical Methods

Logistic regression was used to calculate prevalence odds ratios (OR) and Wald Maximum Likelihood (WML) statistics [12]. Crude and adjusted multivariate logistic regression (MLR) models were constructed using either continuous dCO₂ or dCO₂MAX data as an independent variable and an SBS symptom as the dependent variable. Covariates used in the MLR models to control for confounding were age, gender, smoking status, carpet in workspace, thermal exposure, RH, and TMB.

Multivariate trend as an indicator of dose-response was tested in additional MLR models using a single categorical CO₂ variable, with five levels (10th and 90th percentile values, and three groups split between them). These levels were coded using the bin-mean dCO₂ or dCO₂Max value for each CO₂ level. The WML statistic and associated p-value for this categorical variable were used as a measure-of-fit of the dose-response relationship for the adjusted categorical associations between CO₂ measures and SBS symptoms [12].

The median dCO₂ and dCO₂MAX concentrations were 140 and 350 ppm, and the ranges were 6 - 418 ppm and 120 – 716 ppm, respectively. In no case were the indoor average or the peak indoor CO₂ concentrations extraordinarily high, with only one building having absolute indoor CO₂ concentrations routinely above 1000 ppm.

The dCO₂ and dCO₂MAX ORs are reported in units per-100 ppm and per-250 ppm, respectively, chosen to scale with the ratio of their median values (i.e., 250/100=350/140). This selection of OR units for CO₂ – SBS symptom associations

provides a basis of relative comparability between the measures of association derived using dCO₂ and dCO₂MAX.

RESULTS

Logistic Regression Results

Table 1 presents both crude and adjusted ORs and 95% confidence intervals (CI) using dCO₂ data. The crude dCO₂ ORs for Sore Throat, Nose/Sinus, and Wheeze ranged from 1.1 to 1.5 per 100 ppm increase in dCO₂. After controlling for confounding variables, statistically significant associations were found between 100 ppm dCO₂ and Sore Throat, Nose/Sinus, Tight Chest, and Wheeze, with ORs ranging from 1.1 to 1.4. Almost all ORs exceeded unity, even though many of the relationships were not statistically significant. The WML statistic (not included in Table 1) indicated a statistically significant increasing trend in OR for all three MM symptoms, as well as Tight Chest and Wheeze ($p < 0.05$).

Table 1 also presents both crude and adjusted ORs and 95% CIs using the dCO₂MAX data (per 250 ppm). The unadjusted and adjusted ORs for the association between dCO₂MAX and Sore Throat was 2.0 and 2.3 per 250 ppm, respectively ($p < 0.005$). In addition, Nose/Sinus (OR=1.4) and Wheeze (OR=1.9) symptoms were significantly associated with dCO₂MAX in the adjusted models. A statistically significant increasing trend in OR was measured for all MM symptoms in the dCO₂MAX analyses ($p < 0.05$).

Table 1. Calculated crude and adjusted associations for dCO₂, dCO₂MAX and selected MM and LResp SBS symptoms. ORs at maximum observed CO₂ levels are also shown.

SBS Symptom	dCO ₂ (per 100 ppm)		dCO ₂ MAX (per 250 ppm)		Risk at Maximum CO ₂ ^b Adjusted Odds Ratios	
	Crude	Adjusted	Crude	Adjusted	dCO ₂	dCO ₂ MAX
MM						
Dry eyes	1.1 (0.9-1.2)	1.1 (1.0-1.2)	1.1 (0.9-1.4)	1.2 (1.0-1.5)	1.5 (0.9-2.5)	1.7 (1.0-3.1)
Sore Throat	1.5 (1.2-1.9)*	1.5 (1.2-1.9)*	2.0 (1.4-2.8)*	2.3 (1.6-3.2)*	6.2 (2.5-15)*	10.2 (3.6-29)*
Nose/sinus	1.1 (1.0-1.3)	1.2 (1.0-1.4)	1.2 (1.0-1.5)	1.4 (1.1-1.8)	2.1 (1.1-4.1)	2.7 (1.4-5.6)
LResp						
Chest tight	1.2 (0.9-1.7)	1.5 (1.1-2.2)	1.3 (0.8-2.2)	1.6 (1.0-2.8)	4.9 (1.2-21)	4.2 (0.9-19)
Short breath	0.9 (0.6-1.3)	1.3 (0.9-2.1)	0.9 (0.5-1.4)	1.6 (0.8-3.0)	1.3 (0.3-6.5)	1.4 (0.2-8.3)
Cough	1.0 (0.7-1.2)	1.1 (0.8-1.2)	1.0 (0.7-1.4)	1.2 (0.8-1.7)	1.0 (0.4-2.7)	1.2 (0.4-3.6)
Wheeze	1.4 (1.0-2.0)	1.4 (1.0-2.0)	1.6 (0.9-2.7)	1.9 (1.1-3.4)	4.5 (1.1-18)	6.3 (1.2-34)

^aAll associations in bold are 95% significant or higher. Values in parentheses are the 95% confidence interval.

^{*} $p \leq 0.005$ ^b Scaled so ORs are per maximum of observed dCO₂ (418 ppm) and dCO₂MAX (716 ppm)

Table 1 also shows the adjusted odds ratios for the risk of having SBS symptoms scaled to the maximum dCO₂ and dCO₂MAX values observed in the 41 BASE buildings. These ORs are based on the other two sets of adjusted analyses shown in Table 1. This recasting of the analyses puts the SBS symptom risks into clear perspective. The implication is that, on average, office buildings with average indoor CO₂ concentrations of roughly 800 ppm (or 1h maximum concentrations of about 1000 ppm) may have from 1.5 to 6.2 times the prevalence of MM and LResp symptoms as compared to buildings with about 400 ppm CO₂.

DISCUSSION

The results of these analyses indicate a clear association between elevated indoor CO₂ levels and increases in certain MM and LResp SBS symptoms. Analyses conducted using average and maximum indoor CO₂ had similar findings. These findings were generally evident in the crude regression models, and were strengthened through adjustment for a number of potential confounders.

Both the adjusted dCO₂ and dCO₂MAX ORs indicated increased risk of MM and LResp symptoms. The units of the dCO₂- and dCO₂MAX-based ORs were designed to match each other, but the measures are not exactly equivalent. Assuming that the two approaches are close to equivalent, it appears that the associations of dCO₂MAX with symptoms are slightly stronger. It is unknown whether this is a real difference or merely an artifact. One potential explanation is that the dCO₂MAX metric tracks the peak indoor concentrations of other pollutants and SBS responses may be due to episodic peak concentrations. Further, the larger uncertainties (greater CIs) seen in the dCO₂MAX analysis results may be due to dCO₂MAX being based upon shorter-term, and hence, more variable data than the dCO₂ (e.g., peak 1-hour average vs. 3 workday average). These observations may have bearing on the methods used to measure office building CO₂ in SBS studies.

The odds ratios for the associations of symptoms with the maximum observed difference between indoor and outdoor CO₂ concentrations may indicate the maximum potential to reduce selected SBS symptoms in typical office buildings. The maximum values of dCO₂ and dCO₂MAX are 418 and 716 ppm, respectively. Considering only the significant associations, the ORs range from 1.5 to 6.3 with an extreme of 10.2 for dCO₂MAX/sore throat. Based on these ratios, the implied potential maximum reductions in symptom prevalences of these symptoms are up to roughly 70% to 85%. This reduction could come through large increases in ventilation rates, improved effectiveness in providing fresh air to the occupants' breathing zone, or through identification of the symptom-causing agents in the indoor air and control of their sources.

CONCLUSIONS

After adjusting for confounding, we found important and statistically significant associations of mucous membrane and lower respiratory SBS symptoms with increases of dCO₂ and dCO₂MAX when workday average CO₂ levels were always below 800 ppm. Odds ratios for statistically significant associations of symptoms with 100-ppm increases in dCO₂ were 1.1 to 1.5, and for 250-ppm increases in dCO₂MAX they were 1.3 to 2.3. Statistically significant dose-response relationships were found between dCO₂ and the following symptoms: sore throat, irritated nose/sinus, combined mucous membrane symptoms, tight chest, and wheeze.

Implications: These results suggest that increases in the ventilation rates per person among typical office buildings will, on average, significantly reduce prevalences of several SBS symptoms, even when these buildings meet the existing ASHRAE ventilation standards for office buildings. The magnitude of the reduction will depend on the magnitude of the increase in ventilation rates, improvement in ventilation

effectiveness, or reduction in sources of SBS-causing pollutants. Very large increases in ventilation rates, sufficient to reduce indoor CO₂ concentrations to approximately outdoor levels, would be expected to decrease prevalences of selected symptoms by up to 70 to 85%. There is no direct causal link between exposure to CO₂ and SBS symptoms, but rather CO₂ is approximately correlated with other indoor pollutants that may cause symptoms.

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