Ventilation (estimated three ways) and building-related symptoms in U.S. office buildings – The U.S. EPA BASE study

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SUMMARY

We investigated relationships between ventilation rate (VR) and building-related symptoms in office workers, using adjusted logistic regression models with data from 87 office buildings in the U.S. EPA BASE study. We based three VR estimators on: peak indoor minus outdoor carbon dioxide (CO₂), volumetric estimates of flow rates, and CO₂ ratio in airstreams. Increased VR by the first two estimators was associated with decreased lower respiratory, upper respiratory, and eye symptoms, whereas increase in the third VR estimator was associated only with decreased upper respiratory symptoms. VR from 3 to 14 Ls⁻¹ per person or more above the current 10 Ls⁻¹ per person target levels for offices were generally associated with 6-48% reduced odds for these symptoms. Low occupant density, even with adjustment for VR, was independently associated with decreased symptoms. VRs above current target levels thus might substantially reduce symptoms in office workers, with occupant density playing an additional, unrecognized role in ventilation requirements. Reliable VR measurements are necessary to clarify these relationships.

KEYWORDS

Ventilation, Symptoms, Office workers, Indoor air quality

INTRODUCTION

Adequate outdoor air ventilation in buildings is required to dilute concentrations of indoorgenerated pollutants to levels sufficiently low for the health and comfort of occupants. Increased ventilation, however, raises costs for thermal conditioning of the introduced outdoor air. Because *incremental benefits* of increasing outdoor air ventilation rates (VR) are expected to *decrease* as VR *increases*, the relationship is not expected to be linear; i.e., indoor pollutants and associated health effects should decrease much more with ventilation increased from 0-10 l/s-person⁻¹ than from 40-50 l/s-person⁻¹.

Historically, VR standards were set to control odorous pollutants emitted by occupants, based on findings from laboratory and field studies. Recently it has become clear that emissions from buildings, building contents, and ventilation systems also contribute biologic and chemical air pollutants to indoor environments that need to be controlled by ventilation (Wargocki et al., 2000). Furthermore, multiple studies, mostly in office buildings, have shown lower VR to be consistently associated with increased health symptoms in occupants (Seppanen et al., 1999). These symptoms have included eye, nose, and throat irritation, breathing problems, headache, and fatigue. The indoor pollutants that increase with lower VR and cause these symptoms might come from the occupants or their activities, or from the buildings and their contents. The biologic mechanisms might involve irritation, toxicity, allergenicity, or odor.

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In setting protective ventilation guidelines, many questions remain, including:

- 1) What are the quantitative relationships between ventilation and human health and comfort? This understanding is necessary to weigh the human benefits for each further increase in ventilation against the energy costs of ventilation (which are fairly well understood).
- 2) Since both occupants and buildings are known to emit indoor pollutants, should building ventilation guidelines reflect both the number of occupants and the amount of indoor space per occupant (as ANSI/ASHRAE Standard 62-2004 now does (ASHRAE, 2004)), and if so, how? Would scientific data support the existing judgment-based guidelines requiring less ventilation per person in more densely occupied spaces (e.g., auditoriums vs. offices)?

We report here the findings from analyses of an existing data set on VR and symptoms from a large study of U.S. office buildings. Prior analyses of this study reported associations between indoor minus outdoor carbon dioxide (CO₂) concentrations (as proxies for VR per occupant) and several building-related symptoms among occupants (Apte et al., 2000; Erdmann et al., 2002). The goal of the present analysis was to use direct estimates of VR in these buildings to analyze how occupant symptoms were related to both VR per person and occupant density. These analyses differ from those in a prior report (Mendell et al., 2005) in that present analyses use corrected rather than draft values for VR, different definitions for symptom outcomes, five instead of seven categories of VR levels, and data only from buildings with all VR estimators available.

METHODS

We used data from the Building Assessment and Survey Evaluation (BASE) Study, collected between 1994-1998 by the U.S. Environmental Protection Agency (EPA). The study included 100 representative office buildings from geographic regions throughout the U.S., and within each building one randomly selected study space with at least 50 occupants. A total of 4,326 office workers participated. The BASE data includes environmental measurements, building characterizations, and occupant responses from self-completed questionnaires. Detailed descriptions of this study and the available data have been reported previously (Brightman et al., 1999).

VR/person values for the office spaces studied in BASE were estimated (Persily and Gorfain, 2004) in three separate ways:

- "peak CO₂" method VR/person estimated using peak measured indoor minus outdoor CO₂ concentration (among mean values in each study space) and a mass balance model.
- "volumetric" method total outdoor air intake from air velocity traverse measurements in the outdoor airstreams of the air handlers, divided by the number of occupants.
- "CO₂ ratio" method total outdoor air flow based on the percent outdoor air intake (from measurements of CO₂ concentrations in the outdoor air, supply, and recirculation airstreams) multiplied by the supply airflow measured with an air velocity traverse, divided by the number of occupants.

Each of the techniques used to measure ventilation rates is subject to substantial error. The peak CO₂ method relies on unverified assumptions about the CO₂ generation rates of occupants and the assumption that the measured peak CO₂ concentration difference is equal to

the true equilibrium peak concentration difference. Both methods using velocity traverse measurements (volumetric and CO_2 ratio) are subject to large errors because velocities are often low (near detection limits of instruments), spatially very uneven, and with an unknown airflow direction (Fisk et al., 2005). The CO_2 ratio method is also subject to large errors from small inaccurately measured CO_2 concentration differences and spatial variability in CO_2 concentrations in the airstreams coupled with reliance on single point measurements.

From the total of 100 BASE buildings, analyses were restricted to the 87 mechanically ventilated, air-conditioned buildings for which all three VR estimators were available. We divided the range of each VR estimator across these buildings into five categories for analysis – the bottom (reference) category for each was defined as below approximately 10 l/s-person 1, and the remaining range for each was divided into four approximately equal numbers of observations.

We included each VR/person estimator with occupant density (the mean of occupancy counts in each space divided by floor area) as multi-categorical risk variables in a set of models. Analyses used a separate set of models for each of seven symptom-based health outcomes, of which findings for three are reported here: lower respiratory symptoms (one or more symptoms of wheezing, shortness of breath, or chest tightness,) upper respiratory symptoms (one or more symptom of stuffy or runny nose, sneeze, or sore or dry throat), and eye symptoms (dry, itching, or irritated eyes). For a study participant to be considered to have an outcome required a "weekly, work-related" symptom – reported at least once per week at work in the last four weeks and improving outside the building. Other independent variables included personal information from the occupant questionnaires on demographics (gender, age, education, smoking status), health status (asthma and allergy diagnoses), job factors (years in building, hours per week at work, job satisfaction, job demand, job conflict), indoor temperature (summarized as degree-hours above 20 °C), and mean indoor relative humidity.

We used logistic regression models with generalized estimating equations (GEE, with exchangeable covariance structure, used to adjust for potential correlation of subjects' questionnaire responses within buildings). Models for each symptom outcome estimated unadjusted and adjusted odds ratios (ORs) and 95% confidence intervals (CI) for both ventilation rate and occupant density. An OR, a measure of strength of association, indicates increased risk when >1.0 and decreased risk when <1.0, and is roughly equivalent to a relative risk (or, for common outcomes like upper respiratory or eye irritation symptoms, would slightly overestimate the distance of a relative risk above or below 1.0). Models for each of the three outcomes included unadjusted models for each ventilation rate estimate and full multivariate models including ventilation rate, occupant density, and potentially confounding personal and environmental variables.

RESULTS

Density of occupancy in the BASE buildings varied substantially, from 1.5-9.0 occupants/ 100 sq m (median 3.7). Estimated VRs were available for all 100 buildings using the peak CO_2 method, but missing in 10 buildings for the CO_2 ratio method and in 8 for the volumetric method. The three estimators for ventilation rates, even after omitting an extreme outlier, differed substantially in range (peak CO_2 , 7-60 l/s-person⁻¹; volumetric, 0-227; CO_2 ratio, 3-208) (Persily and Gorfain, 2004). By all methods, few or no buildings had very low VR (e.g., less than 5 l/s-person⁻¹). The different VR estimators also showed different patterns of association with symptoms.

Overall prevalences of work-related symptoms in the study population were: 5% for lower respiratory symptoms, 22% for upper respiratory symptoms, and 19% for eye symptoms. Findings from unadjusted analyses (not shown) were similar to those from multivariate models. In multivariate models (Table 1) controlling for occupant density and other confounding variables, prevalence of lower respiratory, upper respiratory, and eye symptoms decreased at VRs above the lowest level for both the peak CO2 and volumetric VR estimators, but only upper

Table 1. Multivariate adjusted¹ odds ratios (OR) and 95% confidence intervals (CI) for associations of symptom outcomes with occupant density (/100 sq m) and ventilation per occupant (l/s-person⁻¹).

		Work-Related Symptom Outcomes		
Risk Factors		Lower	Upper	
		Respiratory	Respiratory	Eye
		OR (95% CI)	OR (95% CI)	OR (95% CI)
	Ventilation rate			
Peak Delta CO ₂ Estimator	6.8 - 9.9	1.00	1.00	1.00
	10.0 - 13.2	0.52* (0.30-0.88)	0.73* (0.56-0.96)	0.76 (0.56-1.01)
	13.4 - 18.6	0.63 (0.36-1.10)	0.72* (0.55-0.96)	0.83 (0.62-1.11)
	18.7 - 25.4	0.51* (0.30-0.88)	0.80 (0.61-1.05)	0.80 (0.60-1.08)
	25.41 - 59.9	0.47* (0.26-0.85)	0.72* (0.54-0.96)	0.81 (0.59-1.11)
	Occupant density			
	1.5 - 2.75	1.00	1.00	1.00
	2.8 - 3.7	1.42 (0.83-2.43)	1.22 (0.94-1.57)	1.15 (0.87-1.52)
	3.71-5.1	1.40 (0.85-2.31)	1.24 (0.98-1.57)	1.27 (0.99-1.63)
	5.11-9.0	1.37 (0.83-2.25)	1.34* (1.07-1.69)	1.30* (1.01-1.68)
Volumetric	Ventilation rate			
Estimator	0.0 - 9.7	1.00	1.00	1.00
	10.0 - 21.01	0.84 (0.52-1.37)	0.94 (0.73-1.21)	0.83 (0.64-1.09)
	21.9 - 41.4	0.88 (0.55-1.41)	0.88 (0.68-1.13)	0.86 (0.66-1.13)
	42.0 - 70.7	0.86 (0.52-1.43)	0.85 (0.63-1.13)	0.80 (0.59-1.09)
	86.2 - 227	0.63 (0.37-1.09)	0.81 (0.61-1.06)	0.83 (0.62-1.10)
	Occupant density			
	1.5 - 2.75	1.00	1.00	1.00
	2.8 - 3.7	1.26 (0.74-2.13)	1.16 (0.90-1.51)	1.22 (0.93-1.60)
	3.71-5.1	1.31 (0.81-2.13)	1.22 (0.96-1.55)	1.30* (1.02-1.66)
	5.11-9.0	1.19 (0.73-1.95)	1.25 (0.99-1.58)	1.35* (1.06-1.73)
CO ₂ Ratio	Ventilation rate			
Estimator	2.9-9.9	1.00	1.00	1.00
	10.0 - 24.2	1.74* (1.09-2.77)	0.92 (0.72-1.18)	1.01 (0.77-1.32)
	24.9-38.1	1.25 (0.76-2.06)	0.84 (0.66-1.07)	1.09 (0.85-1.41)
	39.5 - 68.2	1.06 (0.63-1.77)	0.87 (0.68-1.13)	0.83 (0.63-1.09)
	70.9 - 208	1.03 (0.59-1.79)	0.89 (0.69-1.15)	1.07 (0.82-1.41)
	Occupant density			
	1.5–2.75	1.00	1.00	1.00
	2.8 - 3.7	1.47 (0.87-2.49)	1.15 (0.89-1.48)	1.07 (0.81-1.42)
	3.71-5.1	1.37 (0.84-2.24)	1.20 (0.95-1.52)	1.31*(1.02-1.69)
	5.11-9.0	1.16 (0.72-1.88)	1.25 (1.00-1.58)	1.28 (1.00-1.64)

^{*}P-value <0.05; ¹ adjusted for personal variables, temperature, relative humidity, and occupant density.

respiratory symptoms decreased in this way with the CO₂ ratio VR estimator. Continued increase in VR above levels in the second category was not consistently associated with further decrease in these symptoms. Lower occupant density, with ventilation per person

controlled for, was associated for all ventilation estimators with increased odds of symptoms at densities greater than about 2.8 persons per 100 sq m; upper respiratory and eye symptoms showed a tendency to continued increases at even higher densities.

DISCUSSION

Current ventilation standards, based historically on non-health-related criteria such as perception of odor, may not be health protective. Available reviews of the literature, in fact, suggest that VR above current minimum standards for offices may reduce symptoms among workers (Seppanen et al., 1999). In practice, health-protective VR standards should balance estimated VR/response relationships with economic costs and technologic feasibility. We should also quantify separately the amounts of ventilation necessary to remove pollutants produced by occupants and to remove pollutants produced by buildings and contents. This would allow setting appropriately different ventilation standards for indoor spaces of different occupant density, such as auditoriums and offices.

For all VR estimators used in these analyses, increased ventilation per occupant above 10 l/s-person⁻¹ showed, in models adjusted for occupant density, a consistent association with reduced upper respiratory symptoms, even within the high range of VR present in these buildings. Two of the three VR estimators also showed this same pattern for lower respiratory and eye symptoms.

Relative to the first category, VRs within the second of five VR categories in this analysis (i.e., between 10 and either 13, 21, or 24 l/s-person⁻¹) provided as much apparent benefit as VRs in the third and fourth categories. If this relationship were to be substantiated as causal, it would suggest that the most substantial benefits could be achieved from VRs elevated to within the second category. The current findings, however, do not clarify how much symptom reductions would occur with VRs increased above 10 to any *specific* level up to 13 to 24 l/s-person⁻¹. In particular, the current findings do *not* support a conclusion that 10 l/s-person⁻¹ as a minimum VR, relative to less than 10 l/s-person⁻¹, would provide as much benefit as higher VRs. Further research will be required to clarify VR/health response relationships within the range between 10 and 24 l/s-person⁻¹, using a replicable VR measurement strategy. Focused findings like these will be necessary to support specific guidelines for how far above 10 l/s-person⁻¹ VR guidelines should be set.

Interpretation of the findings on symptoms as related to VR and occupant density is complex. At least three different outcomes could have been hypothesized for these analyses. First, if symptom-related indoor contaminants that could be controlled by ventilation came only from occupants or their activities, ventilation needed would be simply proportional to number of occupants. As ventilation per person increased, symptoms would decrease. Also, with ventilation per person held constant, symptom prevalence would be unrelated to occupant density. The former was observed, but the latter was not observed. A second hypothesis is that symptom-related indoor contaminants requiring removal by ventilation might come only from building surfaces or materials proportional to the size of the occupied space, rather than from the occupants. In this case, for equal numbers of people, smaller spaces would require less total ventilation than larger spaces, and thus less ventilation per person than larger spaces. Densely populated spaces such as auditoriums should then require *less* ventilation per person than sparsely occupied spaces. Also, as occupant densities increased with ventilation per person held constant (and ventilation per area thus increased), symptom prevalence would decrease. This was not observed. A third hypothesis is that, if increasing occupant density increased symptoms in ways not reversible by ventilation, then with ventilation per occupant held constant, as occupant density increased symptoms would increase. This was observed.

Consistent across all VR estimators, with ventilation per occupant held constant, as occupant density increased, symptom prevalence increased. These results are consistent only with the third hypothesis above, and not with either of the first two hypotheses or a combination of the first two. ORs for all symptom outcomes at more than 2.8 occupants per 100 sq m showed consistent increases, with further inconsistent increases at the highest observed occupant densities. While this finding may be due to some unmeasured confounding by other aspects of buildings or jobs that are correlated with occupant density, the analysis did adjust for a wide range of factors. It is not clear what mechanism could explain this finding.

The BASE data provide the only available U.S. data on ventilation rate and occupant symptoms in a representative set of offices. Prior reported analyses of BASE buildings (Erdmann et al., 2002) have assessed relationships between symptoms and carbon dioxide-based proxies for outdoor air ventilation. Those analyses showed a dose-response relation between some building-related symptoms in unadjusted analyses using four categories of CO₂ concentrations, and a significant relationship of some outcomes to a linear term for CO₂ concentrations in multivariate models that adjusted for a number of possible confounding variables. The previous analyses did not have available for use the set of ventilation rate values based on measured airflows or CO₂ in air streams. Previous analyses of other data generally have not compared ventilation/symptom relationships at a range of multiple different VR levels, or considered possible confounding of the ventilation/symptom relationship by density of occupancy.

The BASE data set lacks very low VR, thus limiting contrasts. Also, differences between the VRs determined by different measurement methods raise questions about their relative accuracy and interpretation. The peak CO₂ estimator differed substantially from the other two estimators, which agreed more closely. Estimated uncertainty in VRs from the CO₂ ratio method was very high (Persily and Gorfain, 2004) because calculations were often based on small concentration differences. Accuracy of estimated VRs from the volumetric method is questionable, as studies by Fisk et al. (2005, 2005a) have shown that air velocity measurements in the outdoor intake section of a typical air handler are unlikely to result in accurately estimated ventilation rates. It is unclear which VR estimator is more accurate, or if any one is more accurate across the full observed range of VR. This makes it impossible to know which set of ventilation/symptom relationships found in these analyses is more accurate.

Better scientific data are essential for setting scientifically-based ventilation standards, using a traditional approach to health risk assessment. Ultimately, these standards should reflect: (1) a comparison of costs of increased outdoor air ventilation with the magnitude of health (and performance) benefits expected at different VRs, and (2) the relative need for ventilation to control person-proportional contaminants vs. space-proportional contaminants, in specifying ventilation for spaces of widely differing density of occupancy such as offices and auditoriums.

CONCLUSIONS

Findings from this study of representative, large U.S. office buildings suggest, but not with complete consistency, that increases in VR up to 3-14 l/s-person⁻¹ above the current 10 l/s-person⁻¹ target level often used in offices (i.e., to 13-24 l/s-person⁻¹) would lead to reduction in some health symptoms, and that lower occupant density is independently associated with

decreased symptoms. Additional research using more robust measures of ventilation rate is necessary to document the magnitude of health benefits from increased ventilation, considering both occupant number and density, in order to establish scientifically based health-protective ventilation guidelines that can balance health benefits with costs.

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REFERENCES

- Apte MG, Fisk WJ, and Daisey JM. 2000. Associations between indoor CO2 concentrations and sick building syndrome symptoms in U.S. office buildings: an analysis of the 1994-1996 BASE study data. *Indoor Air*, 10(4), 246-257.
- ASHRAE. 2004. *ASHRAE Standard 62.1-2004 -- Ventilation for Acceptable IAQ*. Atlanta: American Society of Heating, Refrigerating, and Air-Conditioning Engineers, Inc.
- Brightman HS, Wallace LA, Sieber WK, McCarthy JF, and Spengler JD. 1999. Comparing symptoms in United States office buildings. In: *Proceedings of the 8th International Conference on Indoor Air Quality and Climate Indoor Air '99*, Edinburgh, Scotland. Vol. 1, pp. 847-852.
- Erdmann CA, Steiner KC, and Apte MG. 2002. Indoor carbon dioxide concentrations and sick building syndrome symptoms in the BASE study revisited: analyses of the 100 building dataset. In: *Proceedings of the 9th International Conference on Indoor Air Quality and Climate Indoor Air* '02, Monterey, CA, Vol. 3, pp. 443-448.
- Fisk WJ, Faulkner D, and Sullivan D. 2005. Technologies for measuring flow rates of outdoor air into HVAC systems: some causes and suggested cures for measurement errors. *ASHRAE Transactions*, 111(pt 2), 456-63.
- Fisk WJ, Faulkner D, and Sullivan D. 2005a. An evaluation of three commercially available technologies for real-time measurement of rates of outdoor airflow into HVAC systems. *ASHRAE Transactions*, 111(pt 2), 443-455.
- Mendell MJ, Lei Q, Apte M, and Fisk WJ. 2005. Estimated ventilation rates and work-related symptoms in U.S. office buildings -- the BASE Study. In: *Proceedings of the 10th International Conference on Indoor Air Quality and Climate Indoor Air '05*, Beijing, Vol. 5, pp. 3758-3762.
- Persily AK and Gorfain J. 2004. *Analysis of office building ventilation data from the U.S. Environmental Protection Agency Building Assessment Survey and Evaluation (BASE) Study (NISTIR 7145)*. Gaithersburg, MD: National Institute of Standards and Technology. Report # NISTIR 7145.
- Seppanen O, Fisk WJ, and Mendell MJ. 1999. Association of ventilation rates and CO2 concentrations with health and other responses in commercial and institutional buildings. *Indoor Air*, 9(4), 226-252.
- Wargocki P, Wyon DP, Sundell J, Clausen G, and Fanger PO. 2000. The effects of outdoor air supply rate in an office on perceived air quality, Sick Building Syndrome (SBS) symptoms and productivity. *Indoor Air*, 10(4), 222-236.