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ECONOMIC BENEFITS OF AN ECONOMIZER SYSTEM: ENERGY SAVINGS AND REDUCED SICK LEAVE

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ABSTRACT

This study estimated the health, energy, and economic benefits of an economizer ventilation control system that increases outdoor air supply during mild weather to save energy. A model of the influence of outdoor air ventilation rate on airborne transmission of respiratory illnesses was used to extend the limited data relating ventilation rate with illness and sick leave. An energy simulation model calculated ventilation rates and energy use versus time for an office building in Washington, D.C. with fixed minimum outdoor air supply rates, with and without an economiser. Sick leave rates were estimated with the disease transmission model. In the modelled 72-person office building, our analyses indicate that the economizer reduces energy costs by approximately $2000 and, in addition, reduces sick leave. The annual financial benefit of the decrease in sick leave is estimated to be between $6,000 and $16,000. This modelling suggests that economizers are much more cost effective than currently recognized.

INTRODUCTION

The effects of ventilation rates (i.e., rates of outdoor air supply) on human responses has been reviewed by Seppänen at al. (1999) and Wargocki et al. (2001). Most studies reviewed, plus a new study from school classrooms (Shendell et al. 2003) indicate that higher ventilation rates -- or the resulting lower CO_2 concentrations -- are associated with smaller prevalences of some communicable respiratory diseases or sick leave. Fisk (2000) provides an expanded discussion of the results of these studies.

The prevalence of respiratory illnesses may diminish with increased ventilation rate because the higher rate of ventilation leads to a lower indoor airborne concentration of small particles that contain infectious virus or bacteria. These particles are often called droplet nuclei and are produced during coughing and sneezing. It is known that influenza and some common colds, such as those caused by the human rhinovirus, can be transmitted through an airborne route, as well as via direct person-to-person contact and indirect contact via surfaces (e,g., Couch et al. 1966, Dick et al. 1987, Gwaltney et al. 1978; Gwaltney and Hendley 1982). Airborne transmission may be short range, e.g., when a person sneezes directly at another nearby person, or long range, e.g., due to droplet nuclei transport over distances of at least several meters. Ventilation would be relatively or totally ineffective in reducing short-range airborne transmission, or transmission via direct or indirect contact; thus, the available empirical data indicate that at least a significant portion of respiratory disease transmission is due to long range transport of infectious aerosols.

An economizer control system is an energy efficiency measure that increases ventilation rates, i.e., rates of outdoor air supply, during mild weather to reduce the need for mechanical cooling. Because economizers increase average ventilation rates, they should decrease respiratory illnesses and sick leave. The economic benefits of the decreases in sick leave have not normally been recognized; therefore, economizers may be underutilized. This paper builds upon the work of Fisk et. al. (2003), provides a model for estimating how ventilation rates influence illness and sick leave, and another model to estimate how an economizer affects building energy use. The total financial benefits of the economizer are then calculated.

METHODS

A quantitative relationship between ventilation rate and sick leave was estimated using a model of airborne disease transmission. The model was fit (i.e., calibrated) with results data obtained from several epidemiologic studies performed in sets of occupied buildings. We started with the Wells-Riley equation (Nardell et al. 1991) developed previously for a space with well-mixed indoor air to estimate the effect of ventilation rates on airborne transmission of infectious respiratory diseases.

$$P = \frac{D}{s} = 1 - \exp\left[- \frac{ipqt}{Q} \right] \quad (1)$$

where: P = proportion of new disease cases among the susceptible persons; D = number of new disease cases; s = number of susceptible persons; i = number of infectors; p = breathing rate; q = the rate at which an infector disseminates infectious particles; t = time that infectors and susceptibles share a confined space or ventilation system; Q = rate of supply of outdoor air. Rewriting equation (1) we obtain

$$P = \frac{D}{s} = 1 - \exp\left[(-\frac{ipqt}{V}) \Big/ (n_v) \right] \quad (2)$$

where: V = indoor air volume; i/V = infectors per unit volume; $n_v = Q/V$ = ventilation rate. Equation 1 neglects the removal of infectious particles by filtration and by deposition on room surfaces, which are significant processes in removing airborne particles from room air. These removal processes can be expressed with effective removal rates per unit volume n_f and n_d , yielding the equation

$$P = \frac{D}{s} = 1 - \exp\left[(-\frac{ipqt}{V}) \Big/ (n_v + n_f + n_d) \right] \quad (3)$$

where: n_f is the removal rate of infectious particles by filtration, equal to the product of the recirculation air flow rate and the filter efficiency; and n_d is the removal rate of particles due to deposition on room surfaces.

For the subsequent example calculations, we estimated n_f and n_d assuming the aerodynamic diameter of infectious particles is either 1 μm or 3 μm (Gerone et. al 1966; Duguid 1946); however, the actual size distribution of these particles is very poorly understood. It is probable that droplet nuclei in a broad size range are produced. Most larger droplet nuclei, e.g., those greater than a few micrometers in aerodynamic diameter, will quickly be removed from indoor air by settling on surfaces. For example, a 5 μm and 10 μm unit density particles will fall 1 m in 20 minutes and 330 s respectively (Hinds 1982) thus, these larger droplet nuclei are less likely to contribute to long-range airborne disease transmission and their concentrations will be only modestly affected by ventilation rates. The smaller droplet nuclei can remain suspended in air for hours. Thus, for modeling the effects of ventilation rate on long-range airborne disease transmission, one can neglect the large droplet nuclei that settle too rapidly to participate in long range transmission.

With an assumed aerodynamic diameter of 1 μm, the estimated value of n_f was 0.8 h^{-1}, based on a recirculation rate of 4 h^{-1} through the air handling system's filters which is typical of a commercial buildings in the U.S. This value of n_f assumed the filters have a particle removal efficiency of 20% for 1 μm particles[1]. Based on the review of particle deposition rate data by Thatcher et al. (2001), we assumed that n_d = 0.3 h^{-1} for 1 μm particles. With an assumed diameter of 3 μm for the infectious particles, the corresponding values of n_f and n_d are 2.4 h^{-1} and 1.5 hr^{-1}, respectively.

In equation 3, the term $ipqt/V$ is the unknown. The value of this term will vary over time; however, effective time-average values of the term can be estimated using the data from various epidemiologic studies that provide sufficient information to determine a lower and a higher reference ventilation rate (denoted $n_{v,low}$ and $n_{v,ref}$) and a relative risk (RR), which indicates the prevalence of the illness at the lower ventilation rate divided by the prevalence at the reference ventilation rate. For each study, we computed a value of $ipqt/V$ at the reference ventilation rate, denoted $i_{v,ref}\,pqt/V$, using the equation

1 Based on curves in Fisk et al (2002), a filter with a 20% efficiency at 1 μm will have an ASHRAE MERV Rating of approximately 9 or a Dust Spot Rating of approximately 40%

$$RR = \left[1 - \exp\left[-\frac{\left(\left(\frac{i_{v,low}\, pqt}{V}\right)\right)}{\left(n_{v,low} + n_f + n_d\right)}\right]\right] \Big/ \left[1 - \exp\left[-\frac{\left(\frac{i_{v,ref}\, pqt}{V}\right)}{\left(n_{v,ref} + n_f + n_d\right)}\right]\right] \quad (4)$$

The value of "i", which is the number of infectious people in the building, should, in general, increase as the ventilation rate decreases. If there were no introduction into the building of infectious individuals who became infected outside of the building, $i_{v,low}$ would equal the product of RR and $i_{v,ref}$. If all individuals who became ill due to exposures inside the building were instantaneously removed and, thus, unable to infect others, and infections of building occupants were due only to the introduction of infectious individuals who became infected outside of the building, $i_{v,low}$ would equal $i_{v,ref}$. In real buildings, the situation is between these extremes. As a first approximation, we assume that half of the infectious individuals introduced in the building became infected outside of the building and half became infected inside the building; thus, $i_{v,low} = i_{v,ref}\,(1 + RR)/2$.

Table 1 provides the values of $n_{v,low}$, $n_{v,ref}$ and RR obtained from published studies, with a few assumptions[2] required. Once the value of $i_{v,ref}\, pqt/V$ was known, equation 4 was used to calculate RR for a range of ventilation rates between 0 and 4 h^{-1}, with the reference ventilation rate being $n_{v,ref}$. Finally, all values of RR were normalized by the value of RR computed for no ventilation. For comparison to the disease transmission model represented by equation 4, we also used a much simpler model in which the disease prevalence is proportional to reciprocal of the total infectious particle removal rate

$$P \propto 1 \Big/ \left(n_v + n_f + n_d\right) \quad (5)$$

This model is consistent with the assumption that the disease prevalence in the building is proportional to the indoor concentration of infectious particles.

Equations 4 and 5 utilize the ventilation rate per unit indoor volume denoted by n_v. To enable predictions of how disease prevalence varies with ventilation rate per person, one can replace the n_v terms with corresponding n_p terms, using the equation

$$\eta_v = \eta_p \, B \Big/ V \quad (6)$$

where: n_p is the ventilation rate per person, B is the number of persons in the space, and V is again the indoor volume.

To estimate the economic costs of different disease prevalences, we assumed that short term sick leave is proportional to the prevalence of respiratory illness. With hourly predictions of ventilation rates (described below), a seasonal average value of P was calculated. From the data from Milton et al. (2000), we assumed that the baseline short-term sick leave rate was 2% with a ventilation rate of 0.45 h^{-1}, enabling a calculation of the annual average sick leave rate. Finally, a day of sick leave was valued at $200, based on annual total salary plus benefits of $50,000 and 250 work days per year.

[2] To calculate values of n_v based on the ventilation rate data from Milton et al. (2000), we assumed 2900 ft^3 of indoor volume per occupant based on data from a survey of 100 office buildings (Burton et al 2000). To calculate values of n_v based on the ventilation rate and other data in Drinka et al. (1996), we assumed a total air supply rate of 4 indoor air volumes per hour and also computed weighted average values of percentage outside air and floor space per person for a set of three lower-ventilation rate buildings. The weighting factors were the numbers of occupants in each building.

Table 1

Data used in equation 4 and resulting value of $i_{v,ref}$ pqt/V if infectious droplet nuclei have an aerodynamic diameter of 1 μm.

Reference	$n_{v,low}$ (h^{-1})	$n_{v,ref}$ (h^{-1})	RR	$I_{v,ref}$ *pqt/V*
Milton et al. (2000), short term sick leave	0.43	0.86	1.5	0.453
Brundage et al. (1988), illness all years	0.15	1.0	1.5	1.651
Brundage et al. (1988), illness 1983 data	0.15	1.0	1.9	0.841
Drinka et al. 1996, illness	1.6	4.0	2.2	1.870
Drinka et al. (1996), influenza	1.6	4.0	4.7	0.358
Hoge et al. (1994), pneumonia	0.68	1.0	2.0	-0.49

The disease transmission models were applied to hourly predictions of outside air ventilation rates in a hypothetical moderate-size two-story office building located in Washington, DC. The ventilation rate predictions and associated HVAC energy use predictions were made with a very widely-used building energy simulation program, which is described by Winkelmann et al. (1993). The program's simulation of economizers is decribed in York ete al. (1981). Key building characteristics include: 22,000 ft^2 (2000 m^2) floor area; 200,000 ft^3 (5670 m^3) conditioned volume; 72 occupants; an internal heat generation of 6.3 BTU hr^{-1} ft^{-2} (20 W m^{-2}) from lights and equipment; and an air infiltration rate of 0.3 h^{-1}. The building had a variable air volume HVAC system; thus, the supply flow rate was modulated to control indoor temperature, with a design maximum flow rate of 0.81 cfm per ft^{-2} (4.1 L s^{-1} per square meter) of floor area. Additional information on the modeling of this prototypical office building is provided by Huang et al. (1991) Simulations were performed assuming minimum outdoor air supply rates by the HVAC system during occupancy of 21, 32, and 42 cfm (10, 15, and 20 L s^{-1}) per person. Simulations were performed with and without a temperature-based economizer control system that introduced 100% outdoor air whenever the outdoor air temperature was less than the return air temperature, thus, providing "free" cooling. The HVAC system operated between 06:00 and 21:00 on weekdays, with the assumed percent of total occupancy versus time of day was as follows: 25% at 08:00; 75% at 09:00; 95% at 11:00 – 12:00; 75% at 13:00; 95% at 14:00 – 16:00; 75% at 17:00; 50% at 18:00; 35% at 19:00, 10% at 20:00, and 5% at 21:00. The HVAC system operation and occupancy were assumed to be shorter on Saturdays, and we assumed no HVAC operation on Sundays and holidays. Annual energy costs were calculated using prices [see www.eia.doe.gov] during 2001 in Washington, D.C. for electricity and natural gas of $0.076 per kWh and $1.15 per therm ($10.87 per GJ), respectively.

RESULTS

The right hand column of Table 1 provides the calculated values of $i_{v,ref}$ *pqt/V* when we assumed that the droplet nuclei have an aerodynamic diameter of 1 μm. Application of the disease model (Equation 4) to the results of Hoge et al. (1994) yielded a negative value of $i_{v,ref}$ *pqt/V,* which is physically impossible. Application of the model to the influenza data of Drinka et al. (1996) yielded a positive value of $i_{v,ref}$ *pqt/V;* however, the subsequent calculations yielded some negative relative risks with ventilation rates near zero, which are also impossible. The disease model cannot account for the high reported relative risks and associated ventilation rates in these studies. When we assumed that the diameter of droplet nuclei was 3 μm, application of Equation 4 yielded a negative value of $i_{v,ref}$ *pqt/V* for all but one study. Thus, with this assumed particle diameter, the disease model is largely unable to represent available empirical data. Therefore, all subsequent calculations assumed that the infectious droplet nuclei were 1 μm in diameter.

Figure 1 plots the calculated values of illness or short-term sick leave versus ventilation rate, normalized by the illness or sick leave rate predicted with no ventilation. All predictions show the expected decrease in illness over time; however, the rate of decrease varies dramatically for low ventilation rates, with the prediction based on the data of Drinka et al. (1996) appearing as an outlier. The simple particle concentration model (Equation 5) provides a mid-range prediction.

To illustrate how the illness or absence rate is predicted to vary with ventilation rate per person in an office building, Figure 2 provides a re-plot of two of the curves in Figure 1, assuming an occupant density

of 2900 ft^3 (83 m^3) per person, which was derived using data from a survey of 100 U.S. office buildings (Burton et al. 2000).

Figure 1. *Predicted trends in illness or sick leave versus ventilation rate per unit volume.*

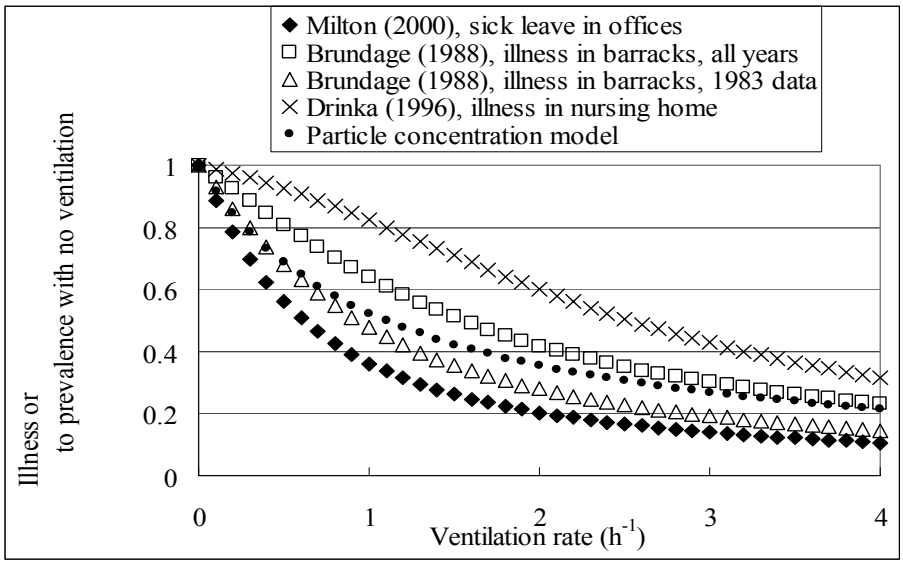
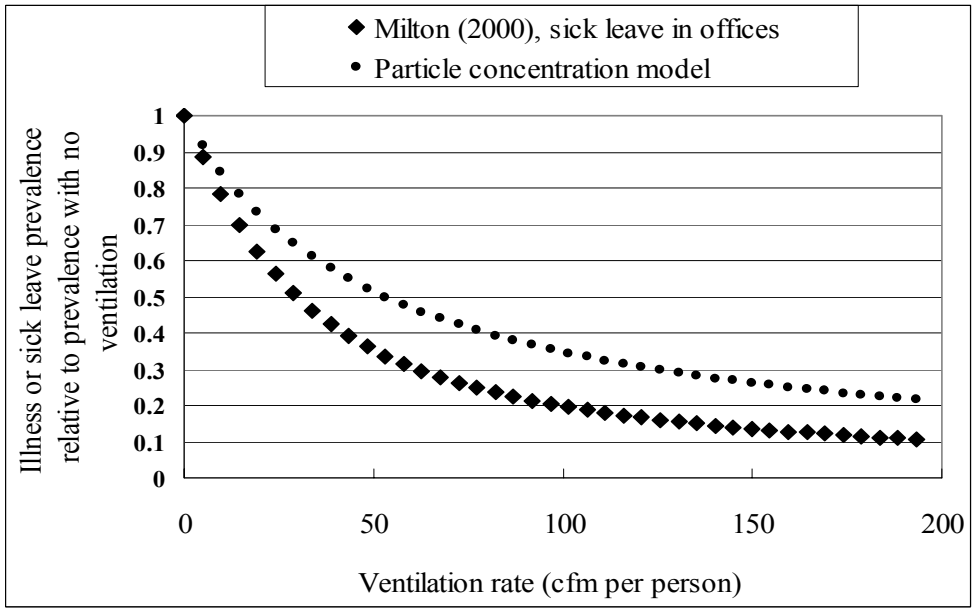


Figure 2. *Predicted trends in illness or sick leave versus ventilation rate per person*



The predicted annual HVAC energy use, ventilation rate, days of sick leave for the workforce accounting for periods with and without economizer operation, and the associated costs of energy and sick leave are provided in Table 2. The economizer was predicted to operate during 22% of all occupied hours. The upper and lower estimates of sick leave were based on the curves in Figure 1 for Milton and Drinka, respectively. The economizer system reduces annual HVAC energy costs by approximately $2,000. The estimated savings due to reduced sick leave with the economizer ranges from $6,000 to $16,000.

Table 2.
Predicted annual HVAC energy use, ventilation rates, and sick leave

Min Vent*	Vent Rate#	Econo-mizer	Annual HVAC Energy			Lower and Upper Estimate of Annual Sick Leave			
L s^{-1}	h^{-1}	Y or N	Elec. MWh	Gas Therm (GJ)	Total $US	Lower days	Lower $	Upper days	Upper $
10	0.74	N	298	6390 (674)	30000	264	53000	340	68000
10	1.46	Y	269	6690 (706)	28000	186	37000	274	55000
10	Savings from economizer				**1900**	**78**	**16000**	**66**	**13000**
15	0.96	N	303	6630(699)	31000	216	43000	321	64000
15	1.56	Y	272	6850 (723)	29000	162	32000	267	53000
15	Savings from economizer				**2100**	**54**	**11000**	**54**	**11000**
20	1.18	N	308	6960 (734)	31000	180	36000	298	60000
20	1.67	Y	276	7130 (752)	29000	150	30000	259	52000
20	Savings from economizer				**2200**	**30**	**6000**	**39**	**7700**

*per person #yearly average Note: Numbers may not add precisely due to rounding

DISCUSSION

In a recent paper, Rudnick and Milton (2003) modeled how indoor airborne disease transmission is predicted to vary with indoor carbon dioxide concentration, based on the assumption that indoor exposures to infectious aerosols will vary in proportion to indoor exposures to carbon dioxide released by occupants. Their modeling neglected the indoor losses of infectious particles by deposition on surfaces and filtration. The model is used to determine a critical indoor CO_2 concentration, below which an infectious individual will, on average, infect less than one other building occupant – implying that the individual will not cause an outbreak of disease among the occupants of the building. This critical CO_2 concentration varies with the type of respiratory infection. Although their results are not presented in a format that allows any precise quantitative comparison to our modeling, qualitatively the modeling by Rudnick and Milton (2003) suggests less benefit from increasing ventilation rates to very high levels. There are some fundamental differences in the two modeling approaches that may explain the different predictions. Our modeling accounts for the potential ongoing introduction of infectious individuals who became ill outside of the building; i.e., for the curves presented in Figures 1 and 2, we assume that half the infectious individuals were infected outside of the building. Rudnick and Milton assess whether the entry into the building of an infectious individual will cause a disease outbreak. Thus, the two papers focus on somewhat different but related questions. Our modeling uses theory to define the shape of the ventilation-disease relationship, but relies on empirical data to determine the actual quantitative relations. Due to the reliance on empirical data, our model results (but not the model form) reflect, albeit inaccurately, the potential disease transmission that occurs in the building by short-range airborne transmission and direct and indirect contact. These additional mechanisms of disease transmission would help to maintain the chain of disease transmission in the building, including maintaining the long-range airborne transmission that is influenced by ventilation rate. Because the model of Rudnick and Milton is more purely theoretical, its results do not account for these other mechanisms of disease transmission. Both models, however, suggest that ventilation rates can have a large impact on disease prevalence.

There are many sources of uncertainty in the model we have used to relate ventilation rates to sick leave. There is even uncertainty regading the proper form of the disease transmision model. Our model is derived from the Wells Riley equation. Nicas (1996) argues that this equation implicity assumes that the deposition of a single infectious organism in the respiratory tract definitely causes infection. Rudnick and Milton (2003) disagree, arguing that the Wells Riley equation simply assumes that the infection probability is a function of the quantal dose, where the quantum of infection (number of organsims necessary to cause infection) could be one organism or many organisms. In the present paper, by calibrating the model with empirical data we effectively incorporate a constant in the relationship beween dose and infection prevalence, thus, our modeling assumes that assumes that the quantum of infection may be more than one

organism. Another source of uncertainty is the highly limited empirical data available to calibrate and evaluate the model.

In addition, there are large uncertainties in the size of infectious particles, and in the associated rates of droplet nuclei loss by filtration and deposition on indoor surfaces. Our example calculations assume that infectious droplet nuclei have an aerodynamic diameter of 1 μm. We were unable to match empirical data when we assumed a 3 μm diameter for droplet nuclei. There is certainly evidence of droplet nuclei larger than 1 μm (e.g., Loudon and Roberts 1967); however, the presence of larger particles should have a minor impact on the relationship of ventilation rate with rate of long-range airborne disease transmission. For example, the rates of removal of 3 μm diameter particles from indoor air by deposition and filtration would be 1 to 1.5 h^{-1} and 3 to 4 h^{-1}, respectively, for a combined removal rate of 4 h^{-1} to 5.5 hr^{-1}. The changes in ventilation rate, between low and high rates, in most of the empirical studies (see Table 1) are less than 1 h^{-1}. Because the indoor droplet nuclei concentration will be proportional to the recriprocal of the total removal rate, the changes in ventilation rate in the empirical studies will have only a small, e.g., 1/5 or 20%, impact on indoor concentrations of droplet nuclei, which is insufficient to explain the high observed values of relative risk. Hence, even if large droplet nuclei are present, it is likely that the observed associations of ventilation rate with disease prevalance and absence are a consequence of the influence of ventilation rate on the small droplet nuclei.

Also, the natural loss of viability of airborne infectious particles has not been accounted for in the model due to a lack of information on the survival times of the airborne virus and bacteria that cause respiratory diseases. If better information were available, viability loss could be incorporated in the model as filtration and depositional losses were incorporated.

The rate at which an infector disseminates infectious particles will likely vary among illnesses. The susceptibility to infection will vary with the age, health status, and immunizations of the occupants of the building. It is likely that these and other factors, including different amounts of time spent in different types of buildings, partially explain the different curves shown in Figure 1.

The disease transmission model represented by Equations 1-4 is theoretically superior to the model represented by equation 5. However, given the limited empirical data available to calibrate and evaluate the complex model, and the wide range of associated predictions, the complex model may not, at present, be any more useful than the simple model represented by Equation 5.

Despite these large sources of uncertainty, a rough accounting of the influence of ventilation rates on sick leave may lead to better decisions about building design and operation than totally neglecting this issue. Clearly, individual decision makers will have to decide whether or not to consider uncertain but potentially large benefits. When we do account for our range of estimates of the reduced sick leave from an economizer system, the economizer becomes much more attractive than it appears based on energy savings alone. The predicted financial value of the sick leave reduction from economizer use is three to eight times as large as the estimated energy cost savings. In the U.S., minimum ventilation requirements for offices are generally 20 cfm (10 L s-1) per person; thus, the most relevant estimates of the related benefits from economizer use in this building are $1,900 for energy and $13,000 to $16,000 for sick leave reductions. Even if the sick leave savings are a factor of ten smaller than predicted, they would still be comparable to the energy cost savings. The influence of economizer use on illness would need to be extremely small to make the related savings negligible. There is one recent study (Myatt et al. 2002) that failed to find an effect of ventilation rate on sick leave; however, the majority of the limited evidence available indicates that ventilation rate does affect sick leave. It is clear that more research is warranted to elucidate this issue.

The data in Table 2 enable a comparison of economizer use to higher values of ventilation rates in HVAC systems without economizers. Based on the estimates in this paper, adding an economizer to a HVAC system with a minimum ventilation rate of 20 cfm (10 L s-1) per person (which saves energy), would bring about larger sick-leave-related savings than increasing the minimum ventilation rate to 30 cfm (15 L s-1) per person. When both energy and sick leave-related savings are considered, the economizer option with a 20 cfm (10 L s-1) per person minimum ventilation rate is predicted to be more economical

than a fixed 40 cfm (20 L s-1) per person minimum ventilation rate. However, we caution the reader that other possible impacts of ventilation rates on health or productivity or equipment costs have not been considered.

Currently economizers are often not considered cost effective for smaller HVAC systems. Economizer performance failures are also common. This modeling suggests that properly functioning economizers may be much more cost effective than currently recognized. The benefits of other energy efficiency measures that increase ventilation rates would also be higher than currently recognized. Examples include evaporative air conditioning systems for dry climates that use 100% outside air, and the use of heat recovery systems together with higher ventilation rates. Also, if the observed reductions of respiratory illness with increased ventilation are a consequence of increased removal infectious particles, the same benefits might be achieved by improving filter efficiencies, which can have a negligible impact on HVAC energy use (Fisk et al 2002).

CONCLUSIONS

- The majority of existing literature indicates that increasing ventilation rates will decrease respiratory illness and associated sick leave. The model prediction in Figure 1 indicate diminishing benefits as ventilation rates increase.

- A disease transmission model, calibrated with empirical data, has been used to estimate how ventilation rates affect sick leave; however, the model predictions have a high level of uncertainty.

- Financial benefits of the use of an economizer system were estimated considering both the energy savings and the value of reductions in sick leave. The estimated financial value of the sick leave reduction from economizer use is three to eight times as large as the estimated energy cost savings. Thus, economizers may be much more cost effective than currently recognized.

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