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Is passive smoking in the workplace hazardous to health?¹

by Alistair Woodward, PhD²

WOODWARD A. Is passive smoking in the workplace hazardous to health? *Scand J Work Environ Health* 1991;17:293-301. Apart from evidence on the acute effects of passive smoking, there is relatively little information available on whether breathing other people's smoke at work causes disease. However, exposure does commonly occur at work, and it does not differ qualitatively from passive smoking occurring in other settings. Therefore it appears sensible to extrapolate from what is known about health risks in settings such as the home. There is too little known to quantify precisely workplace risks due to passive smoking. However, the weight and consistency of the epidemiologic evidence, backed up by the data from active smoking, favor a causal link between passive smoking and serious disease, especially lung cancer. In the light of this evidence, enough is known to justify action to reduce smoking at work.

Key terms: heart disease, lung cancer.

Passive smoking is the inhalation of smoke from others' cigarettes, cigars, or pipes. It is not a new issue. There have no doubt been complaints by non-smokers ever since tobacco was first smoked, although the term "passive smoking" is of recent origin (1). What is new is the emergence of passive smoking as a major occupational health issue. Millions of dollars have been spent on ventilation systems around the world in order to reduce tobacco smoke pollution. Thousands of person-hours have been spent drafting workplace smoking policies. In many countries, employers are now fearful that, unless they control smoking in the workplace, they will be found liable in the courts for illnesses related to passive smoking. Is this flurry of activity well founded, or is it an overreaction to the better known risks of active smoking? Is passive smoking in the workplace hazardous to health?

Is passive smoking a cause of disease?

Evidence up to 1986

In 1986 two authoritative reviews of this subject were published in the United States, the Surgeon General's report "The Health Consequences of Involuntary Smoking" (2) and the report of the Committee on Passive Smoking of the National Research Council (3). At about the same time, in Australia, the National Health and Medical Research Council also published

a major review of passive smoking (4). There were no major discrepancies between the findings of these three reviews, which are summarized in this section.

Smoke emitted into the environment from cigarettes, pipes, and cigars contains the same wide range of toxic compounds known to be present in "mainstream" smoke (that which is inhaled directly by the smoker). These compounds are present in the air of worksites, homes, and public places, wherever cigarettes are smoked. These compounds are absorbed by nonsmokers, as shown by the presence of smoke compounds, or their metabolites, in blood and other body fluids. The levels of these substances are typically 20 to 200 times lower in passive smokers than in active smokers.

Acute irritant effects on the eyes, nose, and throat are widely reported in surveys of the effects of passive smoking. These effects have been produced under controlled conditions in laboratories, with a good correlation between ambient levels of smoke pollution, symptoms, and physiological signs of irritation. There is little evidence on whether passive smoking impairs the respiratory function of healthy adults, although exposure to smoke may exacerbate asthma.

At the time the Surgeon General's and other reports were compiled, information on passive smoking and lung cancer was available from 13 studies. The results of the individual studies varied, but, overall, passive smoking was associated with an increased risk of lung cancer. The increase in risk was considered small (approximately 30 % according to the National Research Council report) but important, nevertheless, because of the large numbers of people exposed to passive smoke.

According to these reports, there was insufficient information available, in 1986, to draw conclusions on whether passive smoking was associated with cancers other than those of the lung, or with cardiovascular disease. Preliminary findings were noted of a link

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between passive smoking during pregnancy and reduced birthweight.

Findings since 1986

Lung cancer. In 1986 Wald et al (5) pooled the findings of the 13 studies of passive smoking and lung cancer that were available at that time. They concluded that the best estimate of the risk of lung cancer due to exposure to a spouse's smoke (compared with the spouse of a nonsmoker) was 1.35 [95 % confidence interval (95 % CI) 1.19–1.54]. The United States Environmental Protection Agency carried out a similar analysis in 1990 (6). There was considerably more information then available, from a total of 22 studies, which produced a pooled relative risk (RR) of 1.41 (95 % CI 1.26–1.57).

The recent evidence bears out the pattern reported by the Surgeon General. Nonsmokers married to smokers have an increased risk of lung cancer compared with those whose spouses do not smoke. The size of the increase is small, and in most studies the probability that the result is a chance finding is greater than the conventional criterion of statistical significance (5 %). The increase in risk was more consistently evident and more likely to be statistically significant in the larger studies, as one would expect if there was an underlying association between exposure to passive smoking and lung cancer.

Other cancers. There is little information on other cancers to add to that reviewed in the 1986 reports. Hirayama (7) has reported, from his Japanese cohort study, that nonsmoking wives with heavily smoking husbands (20 or more cigarettes daily) have increased risks for a range of cancers, including brain (RR 4.78, nasal sinus (RR 3.29), and breast (RR 1.73) malignancies, and also leukemia (RR 2.04). A recent case-referent study (8), also from Japan, has reported an exposure-response relation between the number of smokers in the household and the occurrence of squamous cell neoplasms of the maxillary sinus in nonsmoking women [overall odds ratio (OR) 5.43, $P < 0.05$]. Slatery et al (9) examined passive smoking in relation to cervix cancer in a case-referent study among women in Utah. When adjusted for other known risk factors for the disease, the relative risk for nonsmoking women exposed to passive smoke three or more hours per day was 3.43 (95 % CI 1.2–9.5). The association with passive smoking was strongest for women with few other risk factors (ie, young women with few sexual partners) and was stronger for exposure to smoke at home than for exposure away from home. The relative risk for current smoking (RR 3.42, 95 % CI 2.10–5.57) was virtually the same as that for passive smoking.

Heart disease. The association between heart disease and passive smoking has been reviewed recently by

Beaglehole (10), who concluded that the evidence now suggests that passive smoking does cause heart disease, and Glantz & Parmley (11), who were more emphatically positive in their summing up. These reviews drew on a number of substantial studies of passive smoking and heart disease that had been reported since the 1986 reports. Of particular interest, because it included an analysis of workplace exposure, is the so-called MRFIT study. Svendsen et al (12) reported death and disease rates among nonsmoking married men in the Multiple Risk Factor Intervention Trial (MRFIT), comparing those whose spouses smoked ($N = 286$) with those whose spouses did not smoke ($N = 959$). After an average of seven years of follow-up, the men whose wives smoked were observed to suffer 30 % more fatal or nonfatal heart disease events (adjusted RR 1.32, 95 % CI 0.95–1.84). The risk increased with the amount smoked by the wife, the frequency of fatal or nonfatal heart disease events per 1000 person-years being 7.3 (nonsmoking wives), 8.7 (wives smoking < 20 cigarettes/d), and 12.7 (wives smoking ≥ 20 cigarettes/d). Participants who reported that most of their co-workers were smokers had higher rates of heart disease than those who reported that most of their co-workers were nonsmokers (RR 1.4, 95 % CI 0.8–2.5). (This latter risk estimate was adjusted for the wife's smoking status.)

Other studies have investigated possible mechanisms of an effect of passive smoking on coronary heart disease. Davis et al (13) studied healthy nonsmokers before and after passive smoking in a natural setting and reported that both endothelial cell count and platelet aggregate ratio were affected in a manner similar to that observed with active smoking. Allred et al (14) studied the influence of carbon monoxide on individuals with coronary artery disease, stressed by treadmill testing. They observed that the time to ischemic electrocardiographic changes and angina was reduced when the atmospheric level of carbon monoxide was raised to 117 ppm. [The 1986 Surgeon General's report included results from 18 studies which measured carbon monoxide in real-life settings. The highest level reported was 42 ppm (in a heavily smoke-polluted night club), but most worksite levels were less than 10 ppm.]

Reproductive effects. Two studies (15, 16) published at the time of the 1986 reports observed an association between exposure of the mother during pregnancy to other people's cigarette smoke and lowered birthweight. A similar finding was reported later from Southampton, England (17). A survey of 518 couples found that babies whose father smoked were lighter at birth by an average of 113 (95 % CI 8–216) grams. This analysis controlled for mother's smoking habit, social class, parity, maternal age, and alcohol consumption in pregnancy. A prospective Swedish study (18) found that, among nonsmoking women, exposure to tobacco smoke at work was associated with spon-

taneous abortion in the first trimester (RR adjusted for other risk factors 2.16, 95 % CI 1.23—3.81). Passive smoking was not associated with low birthweight, and there were no apparent effects of exposure to smoke at home.

Effects on the respiratory tract. In the west of Scotland (19) life-long nonsmokers who lived with a smoker suffered increased rates of respiratory symptoms (infected sputum, persistent sputum, dyspnea, and hypersecretion). The increased frequency of symptoms was not statistically significant ($P > 0.05$), but a dose-response relation was observed with amount smoked by the co-habitee. In an analysis of the United States Health Interview Survey, Ostro (20) found an association between passive smoking in the home and acute respiratory illness among adults and children. After adjustment for socioeconomic factors, it was estimated that a pack-a-day smoker increased (by 20 %) the number of days on which a nonsmoking spouse was restricted by respiratory illness.

The study of MRFIT participants (12) reported that men whose wives smoked had lower pulmonary function, as measured by the maximum forced expiratory volume in 1 s. The mean difference at the base-line examination was 98.9 ml (95 % CI 5.4—192.4). Masi et al (21) examined multiple parameters of lung function in 293 nonsmoking adults, aged 15 to 35 years, by self-reported exposure to passive smoke at home and at work. There was a trend towards lower levels of function with increasing passive smoke exposure, but the differences were mostly small, and not statistically significant ($P > 0.05$). Urch et al (22) tested pulmonary function in a mixed group of asthmatic and nonasthmatic nonsmokers exposed to passive smoke in a laboratory setting, with pollution levels close to those observed under natural conditions. They observed dose-response changes in symptoms, pulmonary function (forced vital capacity and forced expiratory volume in 1 s) and nasal inspiratory resistance.

Summary. The results of recent studies of passive smoking and lung cancer resemble those of earlier research. The increase in lung cancer associated with living with a smoker is small (generally in the range of 20—100 %), and the statistical confidence interval around this estimate of risk is broad, due to the small number of cases included in most series. There are still few data on cancers of sites other than the lung.

Relatively few studies of passive smoking and heart disease have been completed, but their findings are consistent with a small increase in risk. What is known about the effects of carbon monoxide and other smoke compounds on the cardiovascular system suggests that a causal association between passive smoking and coronary heart disease is biologically plausible.

Otherwise, there is further evidence that passive smoking is associated with adverse pregnancy out-

comes, that passive smoking in adults is associated with an increased frequency and severity of acute respiratory illnesses, and that passive smoking is associated with impairments of pulmonary function, although the association is small and of uncertain clinical significance.

Is the association of passive smoking with disease causal?

Most of the published scientific papers concerning lung cancer, heart disease, and acute respiratory effects report a positive association with passive smoking. It is unlikely that this pattern is due solely to publication bias (the tendency of scientific journals to favor papers with a positive result) (23, 24). However, association does not necessarily imply causation. Other explanations include chance, confounding, and bias.

Many of the studies have reported associations of passive smoking with disease which fail the usual standard of statistical significance ($P = 0.05$). For example, Glantz & Parmley (11) reviewed 11 published studies on passive smoking and heart disease. While all but one of these studies reported an increase in risk, only 3 out of 10 positive findings were statistically significant ($P < 0.05$). In isolation, the findings of individual studies might be judged to be due to chance alone. However, when the overall pattern of research findings is considered, it seems unlikely that the consistent association of passive smoking with lung cancer and with heart disease can be attributed to chance. This view is supported by formal analyses of the combined results (5, 25).

Confounding exists where a third factor is associated with the exposure variable and is itself an independent cause of the outcome. There is evidence that passive smoking tends to be linked with adverse health factors. In the population of Maryland in 1963 individuals with exposure to household smoke were less likely to be married, to be in good quality housing, to live in rural areas, and, in the case of women but not men, to be well-educated (26). In a sample of nonsmoking Hong Kong women, wives whose husbands had never smoked tended to have healthier life-styles, in a number of respects, compared with wives of smokers (27). Friedman et al (28) reported a correlation of self-reported passive smoking with alcohol consumption in the United States. Another United States study (29) of the Kaiser Permanente Health scheme found that members exposed to passive smoke at home had a lower mean carotene intake.

In the case of lung cancer, confounding by life-style is unlikely to have a major influence, with the possible exception of diet. [There is evidence that the intake of carotenoid sources of vitamin A is inversely related to the risk of lung cancer (30).] Only one study of passive smoking and lung cancer has reported risk estimates adjusted for dietary intake. Kalandidi et al (31) found that a low consumption of fruits was a risk

factor for lung cancer, but the associations of lung cancer risk with passive smoking and reduced fruit intake were independent of one another. The situation with heart disease is more complicated, since the known risk factors explain only about half the variation in disease. There may be subtle unmeasured differences in the lifestyles and social circumstances of families that smoke that contribute to the risk of heart disease.

Another consideration is the presence of a dose-response effect reported in many (but not all) passive smoking studies. Such an effect (of increasing frequency of disease with increasing exposure) does not rule out a noncausal explanation, but it makes such an explanation more complicated, and hence less likely.

Most attention has fallen on bias as a possible explanation for the observed associations. Bias is systematic error in the collection, classification, or interpretation of data. Studies of passive smoking are particularly vulnerable to bias because they rely largely on the self-reporting of both personal smoking habit (smoker/nonsmoker) and exposure to others' smoke. Such self-reports are liable to error (32). If passive smokers and nonpassive smokers are equally likely to misreport their own smoking habits, the consequence is a risk estimate biased towards unity. However, if the groups differ in their misreporting (differential misclassification), then the risk estimate may be biased up or down.

Of particular concern are former smokers and current smokers who report themselves as nonsmokers, who will, when combined with lifelong nonsmokers, increase that group's risk of smoking-related diseases, regardless of any effect of passive smoking. There is evidence from England that smokers tend to marry smokers (33). Hence the spouses of smokers are likely to include more former smokers and more current smokers who deny their habit than the spouses of nonsmokers. Some have argued that this "aggregation bias" is sufficient to explain the observed association of passive smoking and disease (33). However, this situation is unlikely. The effect of the bias can be calculated from estimates of the extent of aggregation, the degree of misreporting, and the risk of disease experienced by former smokers. In the case of lung cancer, the errors required to explain the observed risk associated with passive smoking are implausibly high (5, 34). This misclassification bias is even less likely to be an explanation for the findings with heart disease, because the relative risk of disease due to active smoking is much lower than in the case of lung cancer.

It has been pointed out that the epidemiologic studies of passive smoking appear at odds with the biological data (35). The increase in disease risk in many studies is greater than one would expect by extrapolating from the exposures received in active smoking. The levels of smoke compounds in the bodies of passive smokers may be as low as 0.05 % of those observed for active smokers. Yet the increased risk of disease associated with passive smoking that has been reported in

the epidemiologic studies is greater than 0.05 % of the increase in risk due to active smoking. This inconsistency is the most apparent with heart disease, where active smoking is associated with an approximate doubling of risk, while increases in risk of 20–40 % are reported for passive smoking.

The differences between observed and extrapolated risks are puzzling. It is not known whether there is a straight line relation between smoke exposure and risk of disease, as assumed by the simplest risk extrapolations. Moreover, the existing biochemical measures include assumptions and opportunities for error, just as do the questionnaires used in epidemiologic studies (36). For example, levels of cotinine in saliva and urine are sometimes used to estimate unknown variables in relation to the absorption, distribution, and clearance of nicotine. Tobacco smoke contains many potentially toxic substances. It is not known which of these substances are responsible for heart disease, cancer, and other smoking-related diseases. Biochemical measures of one substance (for example, nicotine) do not necessarily give an accurate picture of exposure to other substances (such as tars). For all these reasons, caution is warranted when dosages from active smoking are extrapolated to passive smoking on the basis of biological measures.

In summary, there are many possible noncausal explanations of the association between passive smoking and disease. The available information is incomplete and often has deficiencies. However, the overall picture indicates that passive smoking does indeed increase the risk of a range of smoking-related diseases. The major reports of 1986 concluded that passive smoking was a cause of lung cancer, and the evidence that has become available since appears to strengthen that conclusion. Recent studies favor an effect of passive smoking on coronary heart disease that may be, in terms of the numbers of people affected, even more significant than the effect on lung cancer. The associations of passive smoking with reduced birthweight, and with respiratory morbidity and impaired pulmonary function, have less research weight, but they too point increasingly in the direction of true cause and effect. There is no evidence that passive smoking initiates asthma in adults, but some studies indicate that the condition may be exacerbated by exposure to smoke.

How much passive smoking occurs in the workplace?

For many people work is a major source of exposure to others' tobacco smoke. In a United States survey in the early 1980s two-thirds of adults reported they were exposed to passive smoke at least 1 h/week, and the place of exposure was most commonly work (28). Some occupational groups experience particularly high levels of passive smoke. One taxi driver in Canberra,

Australia, counted 143 cigarettes lit and smoked in his cab in one 12-h shift (37). In the most comprehensive study of this topic so far, a survey of women in 13 centers in 10 countries coordinated by the International Agency for Research on Cancer (IARC), the proportion reportedly exposed to passive smoke at work varied from 2 % (Chandigarh, India) to 67 % (Sendai, Japan) (38). In all centers, with the exception of Chandigarh, at least one-third of the women were exposed to passive smoke at work.

Several studies have been carried out of indoor air quality in relation to passive smoke. The Surgeon General's report summarizes many of these studies and concludes that passive smoking under realistic office conditions typically produces a three- to fourfold increase in ambient levels of respirable suspended particulates (2). The volume of the workplace, ventilation rates, and amount smoked are the major influences on levels of smoke pollutants. The segregation of smokers and nonsmokers is not an effective means of reducing passive smoking, as shown in a study of air nicotine levels in a commercial airliner (39). Recently studies of the effects of smoke bans at work have become available. Nicotine vapor concentrations in the Johns Hopkins Hospital fell from a weekly average of 13 $\mu\text{g}/\text{m}^3$ before a ban on smoking to 0.51 $\mu\text{g}/\text{m}^3$ (40). The levels of respirable suspended particulates in Canadian public service worksites declined by 25–50 % with the introduction of smoking restrictions (41).

There have been fewer studies of passive smoking in the workplace with biological measurements, but their findings are consistent with those already reported. For example, nonsmoking Japanese office workers who were exposed to smoke at home had higher urine levels of cotinine than those who were not exposed at home, and regardless of exposure at home, cotinine levels rose with increasing numbers of smokers in the workplace (42). Among municipal workers from nonsmoking households in the United States, self-reported exposure to passive smoke at work agreed well with mean urine cotinine levels (43). Both urine cotinine levels and symptoms of respiratory tract irritation reported by airline passengers and staff increased with ambient nicotine levels (39). Restaurant staff exposed to smoke for 40 h/week had a mean urine cotinine level of 56 ng/ml compared with a mean level of 8.3 ng/ml for staff not exposed at work (44).

Which is more important — exposure to passive smoke at work or at home? The answer depends on factors such as the proportion of the population working outside the home, the prevalence of smoking, the size of households, and housing conditions. Some have claimed that, in general, the workplace is a more important source of exposure to passive smoke (45). Recent information indicates that this may not be generally true.

In the IARC study of passive smoking there were 274 women who were exposed to smoke only at home.

The mean urine cotinine concentration for this group (9.5 ng/mg creatinine) was twice that of the 350 women exposed only at work (4.5 ng cotinine/mg creatinine). The mean for women exposed at both work and at home was 10.1 ng/mg (38). In this multinational study population of women, smoking in the home was clearly a more important cause of passive smoking than was smoking in the workplace. The same pattern has been reported in other studies that have included both men and women (43).

What are the likely health effects of passive smoking at work?

Irritation of the eyes and upper respiratory tract as a consequence of exposure to others' cigarette smoke at work is known to be a relatively common occurrence (46). However, with regard to other health outcomes, few passive smoking studies to date have separated the effects of workplace exposure from the effects of exposure to smoke in other settings.

A search of the English language journals up to 1991 revealed information on workplace exposure in 9 out of 25 passive smoking and lung cancer studies. All were case-referent studies. Garfinkel et al (47) reported an odds ratio of 0.93 for exposure to tobacco smoke at work in the last 25 years. Lee et al (48) asked whether cases and referents were exposed to smoke at work a "little" or a "lot," and reported odds ratios of 1.82 and 0.19, respectively (neither being statistically significant). Svensson et al (49) presented odds ratios for exposure to smoke at work or home (OR 1.2, 95 % CI 0.4–2.9) and for exposure at work and home (OR 2.1, 95 % CI 0.6–8.1), but no information on the effect of workplace exposure alone. Janerich et al (50) calculated smoker-years of exposure at work (number of smokers in the workplace times the number of years worked) and reported an odds ratio of 0.91 (95 % CI 0.8–1.04) for more than 150 person-years of exposure. Kabat & Wynder (51) asked simply whether persons were exposed to smoke at work and reported nonsignificant odds ratios of 0.68 (women) and 3.27 (men). Wu et al (52) calculated odds ratios of 1.2 (95 % CI 0.8–2.2) for adenocarcinoma and 2.3 (95 % CI 0.7–7.9) for squamous cell carcinoma for exposure to smoke at work. Shimuzu et al (53) reported a 20 % increase in the risk of lung cancer with passive smoking at work, although the risk was not statistically significant ($P > 0.05$) and it is not clear from the paper whether the analysis was adjusted for exposures occurring at home and elsewhere. Kalandidi et al (31) reported a case-referent study of nonsmoking Greek women, of whom three-quarters had worked outside the home. Compared with the quartile of women with the least exposure to passive smoking at work, the relative risk of the most heavily exposed quartile was 1.08 (95 % CI 0.24–4.87).

In summary, there is no direct evidence that passive smoking at work increases the risk of lung cancer, but

the data are limited. Information on workplace passive smoking is available from fewer than one-third of the studies of passive smoking and lung cancer, and often the numbers within each study are small since many participants had not worked outside the home. The measures of exposure at work varied greatly, and in only three studies was an attempt made to quantify passive smoking.

If passive smoking at work does cause lung cancer, one would expect the effect to be strongest in groups such as hotel staff and waiters, who have the greatest exposures to passive smoking. Indeed, it has been observed frequently that these occupational groups do experience increased rates of respiratory cancer (54, 55). It is difficult to interpret this finding since the increases in the rate of disease have been relatively small and workers in the entertainment industry tend to smoke more than the general population. The relative risk estimates reported for bartenders for respiratory cancer, compared with the general population of the same country, range from 1 to approximately 1.5 (54–56). However, bartenders are also heavy “active” smokers. In the United States in 1970, 68 % of the bartenders were estimated to be current smokers (ie, more than one and a half times the proportion of current smokers in the national white male population) (57). The greater prevalence of active smoking among bartenders, compared with that of the general population, could by itself account for an increase in the risk of respiratory cancer of up to 30–40 % among bartenders (58). There is one study reported so far that has attempted to control for confounding by active smoking. This study of bartenders in California reported 69 cancers of the trachea, bronchus, and lung from 1979 to 1981, compared with 45.4 cancers expected given the age, gender, and smoking habits of the study population (55). That is, after allowance for the effect of active smoking, the risk of lower respiratory tract cancer was increased by approximately 50 %.

Since there have been few studies that included measures of relevant exposures, assessment of the likely health effects of passive smoking at work has relied chiefly on extrapolation from risks of passive smoking in other settings. An example is a paper that estimated the deaths in New Zealand attributable to passive smoking in the workplace (59). The authors used pooled risk estimates for lung cancer (60) and heart disease (24) and the estimate of Repace & Lowrey (61) that passive smoking in the workplace contributes approximately four times the load of smoke particulates that results from passive smoking at home, and hence four times the excess risk of diseases related to passive smoking. On this basis, it was calculated that in New Zealand (population 3.2 million) in 1985 there were approximately 26 lung cancer deaths and 152 ischemic heart disease deaths attributable to passive smoking at work. These deaths made up 65 % of the total number of deaths estimated to be due to passive smoking.

The New Zealand analysis rests heavily on the estimate of a fourfold greater exposure to passive smoke at work than in the home, which may be an overestimate in the light of the evidence that I have already presented. As a consequence, the authors may have overstated the effect of passive smoking at work by a factor of up to three to fivefold. However, their paper does illustrate some important points. First, even a small increase in risk of serious diseases such as heart disease and cancer will cause a substantial number of deaths, if many people are exposed to the harmful agent. Second, in the past most attention has been paid to the increased risk of lung cancer, but it is possible that passive smoking causes more deaths from heart disease, since it is a more common condition in most populations.

It is important to consider groups in the population who may experience increased susceptibility to the effects of passive smoking. These include individuals with medical conditions which may be exacerbated by exposure to others’ tobacco smoke. People with a history of atopy are more likely to report eye and upper respiratory symptoms induced by passive smoke (46). Exposure to relatively low levels of smoke may cause bronchospasm in some asthmatics (62). Among individuals with preexisting coronary heart disease, exposure to increased levels of carbon monoxide under laboratory conditions hastens the onset of angina (14, 63). (There is no evidence yet as to whether this increased sensitivity applies also to workplace conditions, at lower levels of carbon monoxide.)

Children, who are particularly susceptible to the effects of passive smoking on acute respiratory illness, may be affected by workplace conditions, as clients if not employees (eg, in child care centers and schools). The association of passive smoking with reduced birth-weight indicates that pregnant women should also be considered an at-risk group.

The interaction of active smoking and certain occupational exposures, such as asbestos, is well documented, but it is not known whether passive smoking acts in the same fashion. There are suggestive findings from research in other settings. A Swedish case-referent study of lung cancer and domestic radon exposure found that risk due to passive smoking increased with increasing radon exposure (64). There is a plausible explanation for this finding, however. Radon daughters attach to smoke particles, and so the presence of passive smoke may increase the dose of radiation to the bronchial airways.

How do these effects compare with levels of so-called acceptable risk? Federal agencies in the United States have set a lifetime cancer risk of 1 in 1000 as targets for workplace exposure (65) and 1 in 100 000 for involuntary exposures affecting the public (66). The Food and Drug Administration sets a more stringent standard — one in a million lifetime risk (61). Whether or not passive smoking is regarded as an acceptable risk may depend on which standard is chosen. The cal-

culations are necessarily approximate, but the lifetime cumulative risk of lung cancer for a nonsmoker exposed to passive smoke in all settings is about 1 in 1000 (67).

There are major difficulties with the risk assessment approach. Epidemiologic studies of passive smoking are restricted by the relatively weak association of exposure with disease and by the imprecise quality of measurements of exposure to passive smoke. The biological research is frustrated by a lack of knowledge on how it is that tobacco smoke causes disease. The mechanisms of action of active smoking are not known, let alone those of passive smoking.

However, occupational health is not standing still. In Australia, it was estimated that in 1988 two-thirds of indoor workers were subject to some restriction on smoking at work, and 17 % were in workplaces that were totally smoke-free (68). What are the possible reasons?

First, passive smoke contains many potentially toxic substances. These include the obvious tars and respiratory irritants. There is also a range of carcinogens and substances shown to be genotoxic (69). For example, cigarettes are the major cause of public exposure to benzene. Passive smoking is estimated to be responsible for 5 % of the national exposure to benzene in the United States (70). (This level is orders of magnitude greater than the hazard that led recently to the recall of Perrier water contaminated with traces of benzene.)

Second, there is no known safe level of active smoking. If a lot of smoke causes a big increase in the risk of disease, it makes sense to most people that a little smoke, as inhaled by the passive smoker, will be responsible for a small but definite increase in risk.

A similar point is that there is evidence, from the epidemiologic studies, that this exposure is associated with cancer in humans. Many occupational exposures have been regulated in the past solely on the basis of animal evidence. The questions of whether this association is causal, and what the strength of the association is, are less important, in many minds, than the observation that exposure is associated with cancer in human populations.

Third, the effects of passive smoking upon the nose, throat, and eyes make the exposure obvious. Furthermore, tolerance appears to be related to frequency of exposure. As passive smoking becomes less common, nonsmokers are less willing to put up with the smell and irritation of tobacco smoke.

Finally, there are economic benefits to regulation, and few costs. The benefits not only include fewer fires and lower cleaning costs, but also lower smoking rates in the work force (68), and hence, it may be predicted, less absenteeism. The costs of regulation of smoking at work are more difficult to identify.

In a number of countries the principal costs, or feared costs, of failing to regulate will be those resulting from litigation. Where there are laws that require employers to provide safe workplaces, there will

be debate as to whether an employer who permits smoking at work is in breach of the law. There have now been several instances around the world of employees obtaining damages on the grounds that smoke-related illnesses were due to passive smoking at work (71). In Great Britain, it has been ruled that illness caused by passive smoking may be classified as an industrial accident (72). In Australia, a federal court judge has recently ruled that there was compelling scientific evidence available in 1986 that passive smoking caused disease in nonsmokers (Australian Federation of Consumer Organisations versus Tobacco Institute of Australia, 1991). All these examples indicate that the issue of legal liability is likely to speed the introduction of restrictions on smoking at work.

Conclusion

The recent scientific evidence on passive smoking is consistent with the conclusions of the major reviews of 1986, which were that breathing other people's tobacco smoke is a cause of serious disease, including lung cancer. There is relatively little information available on whether breathing other people's tobacco smoke at work causes disease. However, it appears reasonable to extrapolate from what is known about health effects of passive smoking in other settings (predominantly the home) to the likely health effects in the workplace. It is difficult to quantify the risks involved. Since hazard is a binary variable, it is easier to answer the question posed in the title of this paper: on the balance of what is now known, passive smoking at work is hazardous to health. It is hoped that further research will improve the precision of the estimated risks to health. However, there is widespread opinion that enough is already known to justify action and the restriction or prohibition of smoking at work.

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