components to the air that are qualitatively similar to the smoke taken into smokers' lungs. The levels of these constituents to which nonsmokers are exposed are much lower levels to which smokers are exposed, and the levels vary depending on the amount of smoking around the nonsmoker, the architecture and ventilation of the structure, other aspects of air quality (e.g., humidity), and chemical and physical changes that take place in the air. Nevertheless, the major components of tobacco smoke have been repeatedly detected in enclosed spaces in which there has been smoking, at higher levels than occur in the absence of smoking. Biologic measurements of tobacco smoke constituents and their metabolizes in nonsmokers provide direct, convincing evidence that nonsmokers do have measurable internal exposure to environmental tobacco smoke, and that levels of exposure are related to the number of cigarettes and/or smokers to which they are exposed.

HEALTH EFFECTS: INTRODUCTION

It is now accepted by most scientists and endorsed by several Surgeons General of the United States that cigarette and other tobacco smoking is the cause of most lung cancer and a substantial number of cancers at other sites, a large share of cardiovascular disease, and most chronic obstructive lung disease (COLD) in the United States. OTA estimated that, in 1982, about 314,000 deaths in the United States were related to smoking, amounting to about 16% of all deaths in that year. The exact mechanisms by which tobacco smoking induces disease and the specific components of tobacco smoke that are harmful are not all known. It has been shown, however, that many of the individual constituents of tobacco smoke are carcinogenic in animals.

The mountain of evidence against tobacco smoking that has accumulated since the 1950's indicates that, among smokers, the level of health risk for the major effects increases with increasing dose. The age when a person starts smoking, the number of years of smoking, and

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the amount smoked per day all play a part in determining a smoker's risk of smoking-related disease or death. No level of smoking is thought to be "safe." This "dose-response" relationship, which is a commonly accepted tenet in assessing the effects of toxic chemicals, is one reason that investigations of possible health effects of passively inhaled smoke have been undertaken. Passive smoking results in much lower doses than smokers get, so nonsmokers' health risks, per person, should be smaller than the risks of smokers. The number of passively exposed individuals is larger than the number of smokers, however, so even at low levels of risk, a large number of people might be harmed through passive smoking. A particular concern of some investigators has been the possibility that some subgroups in the population, for instance children and those with preexisting lung disease or other chronic diseases, might be more sensitive to the effects of cigarette smoke than would be predicted from studies of smokers.

Much research has been directed at trying to characterize the risks from passive smoking, to determine whether they are or are not important public health concerns. Since the late 1970's, the pace of research on the health effects of passive smoking has increased considerably, and the body of literature now available is adequate, at least in some areas, to draw reasonable conclusions about the importance of passive smoking to the health of nonsmokers. This Staff Paper concentrates on published experimental and epidemiologic studies. Such studies are not available to document many of the specific kinds of symptoms that people experience and report to physicians, such as various allergic reactions. Survey results support the fact that most smokers and nonsmokers are "annoyed" by tobacco smoke, annoyance undoubtedly taking in physical as well as psychologic effects (Roper Organization, 1978).

The health effects that have been investigated most extensively in relation to passive smoking in adults are lung cancer and alterations in lung function. There is a small literature concerning the relationship of passive smoking to cardiovascular symptoms and to death from

ischaemic heart disease.[1] Although there are isolated reports of a variety of conditions not known to be associated with active smoking in the passive smoking literature, these have not been confirmed, and are unlikely to be important. In general, such associations are not biologically plausible.

A relatively large number of investigations of respiratory infections and lung function in babies and children have drawn links associating parental smoking habits with adverse effects. OTA did not review these studies. However, two critical literature reviews of passive smoking health studies (Weiss et al., 1983; Higgins, 1985) considered the literature on both respiratory illness and lung function, and the 1984 Surgeon General's report (USDHHS, 1984) examined studies of lung function.

In the following pages, the literature on lung cancer, COLD, cardiovascular disease, and irritation is reviewed. The material presented relies to some extent on other published reviews, which are identified in the appropriate sections.

Lung Cancer and Passive Smoking

The first major studies linking passive smoking to lung cancer, one a study of Greek women (Trichopolous et al., 1981), the other a study of Japanese women (Hirayama, 1981), were published in 1981. Since then about a dozen other studies, of various designs and in different parts of the world, have been completed and the Greek and Japanese studies have been updated (Trichopolous, Kalandidi, and Sparros, 1983; Hirayama, 1984). The study populations are made up mainly, though not exclusively, of women. Studies that have a significant focus on passive smoking and lung cancer have been carried out in Hong Kong (Chan & Fung, 1982; Koo, Ho &

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[1] **Ischaemic heart disease (IHD) describes a spectrum of conditions caused by insufficient oxygen supply to the heart muscle; IHD is the leading cause of death in the United States. The most common manifestations of IHD are angina, acute myocardial infarction (heart attack), and sudden death.**

Saw, 1983; Koo et al., 1983; 1984), Germany (Knoth, Bohn, and Schmidt, 1983) and in different parts of the United States (Garfinkel, 1981; Correa et al., 1983; Kabat & Wynder, 1984; Wu, et al., 1985; Garfinkel, Auerbach, & Joubert, 1985). Table 1 lists these studies and their salient features, as well as several other studies that have some information about passive smoking and lung cancer, but which do not include sufficient data to be considered in an evaluation of this specific question. Epidemiologic study of lung cancer and passive smoking continues, with at least two other studies nearing publication and a heightening of interest among researchers.

In the United States, an estimated 9,000 to 11,000 nonsmokers die of lung cancer each year, out of a total of about 100,000 lung cancer deaths. About one-third of the nonsmokers who die of lung cancer are men and two-thirds are women. The percentages of different cancer types (mainly adenocarcinomas and squamous cell carcinomas) differ between smokers and nonsmokers, suggesting at least some different causes in nonsmokers. Passive smoking may account for a portion of these deaths among nonsmokers, but there also are other as yet unknown, causes.

Most of the studies listed above have reported results consistent with approximately a doubling in the risk of lung cancer among nonsmokers heavily exposed to environmental tobacco smoke compared with nonsmokers who were not regularly exposed; some report larger increased risks, some smaller, and two studies found no increase. Passive smoking exposure may vary considerably around the world because of social customs and living conditions, so it is not unreasonable to expect risks to differ among studies. In five studies, statistically significant increased risks are reported.

The International Agency for Research on Cancer (IARC), a unit of the World Health Organization, has recently reviewed the published studies (the study by Garfinkel, Auerbach, and Joubert, described below, had not yet been published when the IARC review took place) as part of a monograph about the carcinogenic effects of smoking, currently in press (IARC,

1986). They note that the risk estimates could actually be somewhat higher or lower than were calculated because of the uncertainties in measurements of passive exposure to cigarette smoke, as well as to other exposures that might have contributed to the development of lung cancer. Because the results could have been influenced by these uncertainties, they conclude that each study is compatible with either an increase or an absence of excess risk of lung cancer from passive exposure to tobacco smoke, even though statistically significant results were reported.

A recent case-control study, published in September 1985, is generally consistent with the results of the other studies, and it is described here in some detail for illustrative purposes. In this study by Garfinkel, Auerbach, and Joubert (1985), the passive smoking histories of 134 nonsmoking women with lung cancer were compared with the passive smoking histories of 402 nonsmoking women with colon-rectum cancer (cancers not known to be associated with smoking). Information was collected about several different aspects of passive exposure to cigarette smoke: current smoking habits of husbands or other cohabitants; number of cigarettes smoked per day at home by the cohabitant smokers; number of years the husband or cohabitant smoked; average number of hours per day the women had been exposed to smoke of others during the past five and 25 years at home, at work, or elsewhere, and during childhood.

Data were analyzed using a variety of standard statistical methods. In almost all cases, the women with lung cancer were somewhat more likely to have been passively exposed to cigarette smoke than were the controls, the women with colon-rectum cancer. Most of the differences were not statistically significant, meaning that, using generally accepted statistical standards, the results could be plausibly explained by chance alone. Several comparisons, however, did produce statistically significant results. For those results, chance alone is an unlikely explanation of the findings.

The strongest evidence for an effect of passive smoking in this study is from an analysis of risk related to the number of cigarettes smoked by the husband per day in total, and the

Table 1: Epidemiologic Studies of Passive Smoking and Lung Cancer in Nonsmokers[1]

Reference	Type of Study: Study Population	Study Subjects	Exposure	Risk Estimate (with p values and 95% confidence limits when available)	Comment
Trichopoulos et al 1981; Trichopoulos, Kalandidi, and Sparros, 1983 (update)	Case-Control: Greek nonsmoking women	77 lung cancer cases excluding adenocarcinomas and terminal bronchial carcinomas 225 orthopedic patient controls, "similar" demographic and socioeconomic profiles	Husband nonsmoker ex-smoker 1 to 21 cig/day 21+ cig/day	 1.0 1.9 2.4 3.4 p~0.01 for trend (2-tailed)	
Kurayama, 1981; 1984 (update)	Prospective cohort: Japanese nonsmoking wives	200 lung cancer deaths among 91,540 wives 40 or older in 1966, followed through 1981	Husband nonsmoker ex-smoker 1 to 21 cig/day 21+ cig/day	 1.0 1.36 1.45 p=0.0337 1.91 p=0.0012 p=0.00178 for trend	
Garfinkel, 1981	Cohort: U.S. nonsmoking married women in American Cancer Society "million person study"	153 lung cancer deaths among 176,739 women followed 1959/60 to June, 1971, 35 to 89 years	Husband nonsmoker <20 cig/day 20+ cig/day	 1.0 1.27 (c.l.: ~.85-1.89) 1.10 (c.l.: ~.77-1.61)	
Chan and Fung, 1982	Case-control: nonsmoking Hong Kong Chinese women	84 lung cancer cases 139 orthopedic patient controls	Husband nonsmoker smoker	 1.0 0.75 (calculated from data in paper)	
Knoth, Bohn, & Schmidt: 1983	Case-population prevalence: nonsmoking German women	39 lung cancer cases	Prevalence of smoking husbands among cases Smoking prevalence of men aged 59-60 in census of 2 million German citizens	61.5% 22.4%	No proper control group.

Table 1 (continued

Correa et al ; 198≡	Case-control: nonsmoking Louisiana ever-married men and women	10 male lung cancer cases (8 in analysis) 25 female lung cancer cases (22 in analy≡i≡ 180 male and 133 female control patients with diagnoses other than chronic lund diseases or smoking related cancers.	Wife nonsmoker smoker Husband nonsmoker smoker 1-40 pack-yrs 41+ pack-yrs Spouse om ined nonsmoker 1-40 pack-yrs =1+ pack-yrs	1.ꝏ 2·ꝏ 1.0 2.07 1.18 3.52 1.0 1.48 3.11	 p<ꝏ 05 p<ꝏ 05	Small number of cases; 15% of controls had cardio-vascular diagnosis.
Gill≡s et al , 1983	Prospective cohort: Scottish nonsmoking men and women in "partnerships"	6 lung cancer cases am ng 827 men 8 lung cancer cases am 1917 women All followed 1972/76 through 1982	Partner (usually spouse) nonsmoker smoker male cases female cases	 1.0 3.3 1.0		Too few cases for reliable analysis Foll wup ongoing.
Kabat & Wynder, 1984	Ca≡e-control: U.S. nonsmoking men and women	25 male and 53 female lung cancer cases. Equal number of controls matched on age, sex, race, hospital, interview date; with non-tobacco-related diagnoses.	Exposed at home male cases female cases Exposed at work male cases female cases Spouse smokes male cases female cases	 1.3 0.9 3.3 0.7 1.0 0.8	 p=0.05	Risk estimates calculated from data in report. Authors consider data on passive smoking "preliminary."
Koo, Ho, & Saw, 1983; Koo, Ho, & Saw, 1984; Koo, et al., 1985 (abstract only)	Case-control: nonsmoking Hong Kong Chinese females	88 lung cancer cases 137 district contro≡	no exposure exposed at home, at work, or both pl≡ces ≤35,ꝏo hrs exposure >35,ꝏꝏ hrs exposure	1.0 1.24 1.28 0.96	 p≤0.49 p≤0.44	Data from Koo, Ho, & Saw, 1984. Koo et al , 1985 presents results for same study subjects categorized by husbands' smoking; risk estimates for highest categories of total yrs exposure and number of cigarettes/day were 2 to 3, compared with no exposure at home

Table 1 (continued)

Miller, 1984	Case-control: nonsmoking Pennsylvania [illegible] om	123 lung cancer deaths 414 controls; deaths from other causes	Spouse nonsmoker smoker smoker married to unemployed case	 1.0 1.4 1.9		Did not control age differences between cases and controls; association probably invalidated with age adjustment.
Wu et al, 1985	Case-control: California [illegible] ng white women.	31 lung cancer cases 31 neighborhood controls controls matched on birth date and other selection criteria	No exposure Either parent smoked Spouse smokes Exposed at work	1.0 0.6 1.2 1.3	 (c.l.: [illegible].5-3.3) (c.l.: [illegible].5-3.3)	Analysis based on 29 adenocarcinomas: 2 squamous cell carcinomas too few to analyze.
Sandler, Everson, and Wilcox, 1985; Sandler et al, 1985	Case-control: U.S. nonsmoking women	2 lung cancer cases	Too few cases to evaluate			Too few cases to evaluate. Part of larger study of passive smoking cancers at all sites in male and female smokers and nonsmokers; exposure in childhood and adulthood.
Garfinkel, Auerbach, and Joubert, 1985	Case-control: nonsmoking New Jersey and Ohio women	134 lung cancer cases 402 colon-rectum cancer controls matched on age and hospital All diagnosed 1971-81	Exposed over last 5 years Exposed over last 25 years Husband [cohabitant smoked Husband cohabitant mo at home	1.28 1.13 1.22 1.31	(c.l.: 0.96-1.70) c.l.: 0.60-2.14) (c.l : [illegible] 97-1 71) (c.l.: 0.94-1.83)	
			Hrs/day exposed the last: 5 years: 0 1-2 3-6 ≥7 All>0 25 years: [illegible] 1-2 3-6 ≥7 all>0	 1.0 1.59 1.39 0.94 1.28 1.0 0.77 1.34 1.14 1 12	 (c.l.: 0.9-2.72) (c.l.: 0.96-2.03) (c l : 0.69-1.28 (c l : [illegible] 98-1 66) (c.l.: [illegible].60-0.99) (c.l.: [illegible].96-1.87) (c l : [illegible] 83-1 57) (c l : [illegible] 81-1 42)	

Table 1 (continued

Husband [cohabitant			
Nonsmoker	1.0		
<20 cig/day	0.84	(c.l.: 0.61-1.16)	p<0.025 for trend
20-39 cig/day	1.08	(c.l.: 0.81-1.44)	
≥40 cig/day	1.99	(c.l.: 1.13-3.50)	
cigar/pipe	1.13	(c.l.: 0.78-1.62)	
all smoking	1.23	(c.l.: 0.94-1.60)	
Husband [cohabitant] smoking at home			
none	1.0		
<10 cig/day	1.15	(c.l.: 0.84-1.58)	p<0.025 for trend
10-19 cig/day	1.08	(c.l.: 0.76-1.54)	
≥20 cig/day	2.11	(c.l.: 1.13-3.95)	
pipe/cigar	1.17	(c.l.: 0.80-1.70)	
all smoking	1.31	(c.l.: 0.99-1.73)	
Logistic regression, continuous dose-response model			
5 yr exposure-- 10 hr/day	0.93		
25 yr exposure-- 10 hr/day	0.85		
Husband 20 cig/day at home	1.70	p=0.032	
Husband smoked 20 cig/day outside home	1.26		

[1]For several of the studies listed, this topic is not the only one investigated. For those studies, only the data relevant to this question are included

Source: Office of Technology Assessment.

number smoked at home. The risks for women whose husbands smoked more than 40 cigarettes per day (2 packs) total, or more than 20 cigarettes per day (1 pack) at home were significantly higher than the risks for women whose husbands did not smoke. More importantly, there was a trend of increasing risk that rose significantly with higher categories of husband's daily cigarette consumption.

As expected, the level of increased risk is much lower than the substantial increase in the risk of lung cancer incurred by smokers. Lifetime smokers are on the order of 10 to 15 times more likely to develop lung cancer than are lifetime nonsmokers (see OTA, 1981). Data from the study by Garfinkel and colleagues described here indicate that the risk of lung cancer among women passively exposed to the smoke of 20 cigarettes per day smoked at home by their husbands is somewhat greater than two times the risk of nonsmoking women not passively exposed to cigarette smoke.

All the lung cancer studies have some methodologic weaknesses, and these have been pointed out in some cases by the authors themselves, and by others (Baiter et al., 1986). A small number of the studies include so few lung cancers or have such major flaws that they are essentially disregarded in OTA's overall appraisal of the literature. The studies that can be evaluated vary greatly in design and the populations studied vary, yet the results are generally consistent with an increased risk of lung cancer from passive smoking, even taking into account these weaknesses. This consistency across studies lends weight to an overall evaluation that no single study can achieve.

One specific criticism of some of the studies (mainly applying to the case-control studies) is that misclassification of a smoker as a nonsmoker would cause the risk of disease to appear higher than it is. The prospective studies (Hirayama, 1984; Garfinkel, 1981) do not generally suffer from this problem, and the case-control study of Garfinkel and colleagues (1985) described above, went to great lengths to verify smoking history and status for this

reason. There is also potential for misclassifying people as to their exposure to environmental tobacco smoke. There is evidence, for example, that assigning passive smoking status to Americans based on the smoking habits of their spouses can result in considerable misclassification (Friedman, Petitti, and Bawol, 1983). Not all smokers smoke very much around their spouses, and people with nonsmoking spouses may be heavily exposed in other environments, particularly the workplace. This type of bias generally tends to make the risk appear less than it actually is. The identification of potential biases and methodologic problems is important for improving future research. Recent studies, such as the one by Garfinkel and colleagues, appear to have benefited from criticism of early studies.

Repace and Lowrey (1985a) have recently generated two widely-quoted quantitative estimates of the number of lung cancer deaths likely to be attributable to passive smoking per year in nonsmokers in the United States. These investigators did not conduct a specific study; the two estimates are derived using two independent methods, and different sources of data, including several epidemiologic studies, surveys, and estimates of nonsmokers' exposure to tobacco tar from passive smoking. One method produced an estimate of 500 nonsmoker lung cancer deaths per year attributable to passive smoking, the other, 5,000 such deaths per year.

Some of Repace and Lowrey's assumptions are inappropriate. In particular, in the method yielding the higher number, they assumed that the entire difference between the lung cancer death rate in a group of nonsmoking Seventh Day Adventists and in a group of nonsmoking (non-Seventh Day Adventist) Southern Californians was attributable to passive smoking. Mortality rates for cancers at other sites are also lower in that Seventh Day Adventist population, and the exact reasons for the differences are not all known. Seventh Day Adventists clearly have a low overall lung cancer death rate because there are few smokers in the population. Repace and Lowrey assume a lower rate of passive smoking than in the general population as well, which is probably justified. However, they use no specific information

about the rate of passive smoking in either population they compared. At best, one can conclude that some part of the difference between the two populations may be due to differences in passive smoking rates, but the assumption that it is reasonable to attribute the entire difference to passive smoking is unjustified. The effect of these and other flaws on the final estimates calls into question the reliability of either of these numbers.

Effects of Passive Smoking on Lung Function

The 1984 Surgeon General's report (USDHHS, 1984) examined the relationship of direct smoking to chronic obstructive lung disease (COLD), which killed more than 66,000 Americans in 1983. The report states:

> ...the experimental and epidemiologic evidence leaves no room for reasonable doubt on the fundamental issue: cigarette smoking is the major cause of COLD in the United States.

The 1984 Surgeon General's report also reviewed the studies of the relationship between passive smoking and COLD and lung function published to that time. The information in this section is taken largely from the Surgeon General's report and from two other recent critical reviews of the literature concerning health effects of passive smoking, by Weiss and colleagues (1983) and by Higgins (1985).

In general, COLD refers to the narrowing of the airways of the bronchial tree and loss of elasticity in the lungs, with a resultant loss of airflow driving pressure. Increased secretion of mucous and an increase in the size of mucous glands, as well as inflammation, abnormal cell types, ulceration, and a variety of other changes in the cellular makeup and condition of lung and bronchial tissue are also signs of COLD. Emphysema, characterized by specific pathologic changes in lung tissue, is the type of COLD most closely associated with smoking. While most

diagnoses of COLD are in middle-aged or older people, a diagnosis is preceded by pathologic changes and measurable declines in lung function, which may occur over a period of decades.

The pertinent questions in regard to passive smoking are 1) whether passive smoking contributes to the development of COLD; and 2) whether passive smoking exacerbates the symptoms of or has long-term adverse effects on people with preexisting COLD.

The studies in this area take two basic forms: 1) laboratory-based experiments in controlled chambers, in which the endpoints are short-term changes in lung function, and 2) epidemiologic studies of the relationship between passive exposure to cigarette smoke and either measures of lung function or morbidity. Most of the epidemiologic studies focus on children, classified according to parental smoking. Investigators have studied the exposure of 1) healthy people, to find out whether those passively exposed to tobacco smoke are more likely to develop respiratory problems than those not exposed; and 2) those with respiratory conditions, particularly asthma, to see whether exposure exacerbates those conditions.

OTA's review concentrates on studies of adults, the main targets of workplace smoking policies. However, children may be passively exposed to tobacco smoke in Federal offices, for example, in agencies where Federal workers deal directly with the public. In addition, at least a portion of the adult population may be as sensitive as children to the effects of passive smoking. Other reviewers have evaluated the evidence for respiratory system effects of passive smoking in children. Weiss and colleagues report that "several studies suggest important increases in severe respiratory illness in very young (less than 2 years old) children of smoking parents." They also cite evidence of respiratory symptoms in older children exposed to environmental tobacco smoke. Higgins concludes, "The evidence linking passive smoking with acute respiratory illnesses, chronic respiratory symptoms and mild impairments of pulmonary function in children is quite strong."

EXPERIMENTAL STUDIES OF HEALTHY SUBJECTS

A few investigations have been conducted on subjects exposed to tobacco smoke in laboratory chambers, in which the environment can be carefully monitored. Measurements of lung function and, in some cases, measurements of carboxyhemoglobin levels (a measure of carbon monoxide uptake) are carried out at specific times during the experiment. The pulmonary function tests used in these experiments consist largely of measuring the volume of air that is moved in and out of the lungs under different conditions. Two of the three such studies cited by the Surgeon General reported measurable decreases from initial levels in some measures of lung function after exposure of healthy volunteers to tobacco smoke (Pimm, Silverman, & Shephard, 1978; Shephard, Collins, & Silverman, 1979). In the third study (Dahms, Bolin, & Slavin, 1981), which included both healthy volunteers and asthmatics, no statistically significant change in lung function after exposure to environmental tobacco smoke was found in the healthy subjects.

EPIDEMIOLOGIC STUDIES OF HEALTHY ADULTS

Four epidemiologic studies of pulmonary function in healthy adults classified as to their passive smoking history are reported in the 1984 Surgeon General's report. Two of the studies (Schilling et al., 1977; Comstock et al., 1981) found no effect on pulmonary function as a function of spouses' smoking status (the study by Comstock and colleagues included only men exposed to wive's smoking). In both studies, however, the study populations were relatively young and might not have had long-term passive exposure to cigarette smoke.

Two other studies have reported statistically significant, small losses in pulmonary function related to passive smoking. In one (White & Froeb, 1980), tobacco smoke at work was used as the measure of exposure, so it was really a study of current exposure, not necessarily

representative of long-term exposure. The second was a study of adults in France (Kauffman, Tessier, & Oriol, 1983). In this study, nonsmoking women married to smokers had lower values for one measure of pulmonary function than did similar women married to nonsmokers, but the effect did not become apparent until the women had reached age 40. The findings are not ascribable to differences among the women in social class, educational levels, exposure to air pollution, or family size. According to Weiss and colleagues (1983), the results of this study "lend credence to the possible effect of long-term exposure in adult life."

STUDIES OF ADULTS WITH ASTHMA

Two experimental studies of asthmatic adults, conducted in controlled environmental chambers, are cited in the 1984 Surgeon General's report. In one study (Dahms, Bolin, & Slavin, 1981), 10 patients with asthma and 10 healthy controls were exposed to environmental tobacco smoke. Similar increases in blood carboxyhemoglobin levels were found in both groups. The asthmatics, however, experienced worsening pulmonary function over the course of the one-hour experiment, while no change was detected among the controls. In a similar study of pulmonary function (Shephard, Collins, & Silverman, 1979), no such differences were found in objective measures, but in the asthmatic group subjective symptoms--wheezing and chest tightness--were reported.

A recent study (Wiedemann et al., 1986) of nine asthmatics with normal or nearly normal lung function who were asymptomatic at the time of the test, found no significant change in lung function tests after one hour of tobacco smoke exposure in an experimental chamber. In addition to lung function tests, these investigators performed a test to determine whether tobacco smoke exposure increased the reactivity of the subjects' lungs when exposed to a chemical that causes a reaction in the airways. A high degree of reactivity is characteristic of asthmatics, which explains much of their sensitivity to many external agents. After exposure to

environmental tobacco smoke, the asthmatics in the study were slightly less sensitive to the chemical than they had been before exposure, though they were still more sensitive than were a group of nonasthmatics.

SUMMARY: EFFECTS ON LUNG FUNCTION

There is currently a small literature on the effects of passive smoking on lung function in healthy adults, though there are no longitudinal studies, i.e., studies that follow adults over a period of years to look for changes in lung function or disease status. The experimental studies used a variety of tests and selected participants in different ways, some based on a self-assessment of adverse effects of passive smoking. There is significant heterogeneity among the total population with some form of COLD, and the experimental studies that have been done have looked at small, selected groups which may not represent either the "average" or the most sensitive individuals in that population. It is difficult, therefore, to generalize from these results to the total population with COLD. (For methodologic critiques of these studies, see e.g., Witorsch, 1986.)

The assessment of effects of passive smoking on lung function and disease in healthy and compromised individuals would benefit greatly from further research. However, the studies to date do suggest a small acute effect of passive smoking on lung function in healthy adults and the study by Kauffman, Tessier, and Oriol (1983) suggests a long-term adverse effect. The studies relating passive smoking to acute effects in adult asthmatics are at some variance. Higgins summarizes the evidence by saying, "There is insufficient evidence to permit conclusions about acute effects of passive smoking on patients with asthma or chronic obstructive lung disease but it is likely that some unknown proportion of them will be adversely affected."

Passive Smoking and Cardiovascular Disease

Smoking is estimated to have contributed to 123,000 deaths from cardiovascular disease in the United States in 1982 (OTA, 1985). This strong relationship underlies much of the concern about the potential for passive smoking to increase the cardiovascular disease risk of nonsmokers.

EPIDEMIOLOGIC STUDIES

In a study of adults in two populations in Scotland, Gillis and colleagues (1983) found no association of cardiovascular conditions with passive smoking in men or women. Hirayama (1983, cited in Higgins, 1985), in his study of about 91,000 nonsmoking women, found small, statistically significant, increases in the risk of death from ischaemic heart disease among wives of smokers and exsmokers. In a study in the United States, Garland and colleagues (1985) found an increased risk of death from ischaemic heart disease among wives of current or former smokers, but the result was not statistically significant.

EXPERIMENTAL STUDIES

The focus of experimental studies has been to determine the effect of acute exposure to carbon monoxide and environmental tobacco smoke on patients with angina. The literature identified by OTA consists of a series of experiments by Aronow and various colleagues, and a study by Anderson and colleagues. This literature is summarized in the 1979 Surgeon General's report, *Smoking and Health* (USDHHS, 1979). These studies showed that, after exposure to environmental tobacco smoke, angina pain began sooner than it did in the absence of exposure. Aronow's work, however, has been questioned (Aviado, 1986) and it is unclear whether the results of his experiments are valid. While there is agreement that an increase of about 5 percent in carboxyhemoglobin levels can measurably shorten the time to onset of anginal pain,

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there is still insufficient evidence to determine whether an increase in carboxyhemoglobin caused by passive smoking, which has been measured in the range of 2 to 3 percent, can be sufficient to produce an effect. According to Higgins (1985), new studies are under way to investigate the relationship of carbon monoxide exposure to onset of anginal pain.

SUMMARY: CARDIOVASCULAR EFFECTS

The available epidemiologic data point to an increased risk of death from ischaemic heart disease among nonsmokers exposed to environmental tobacco smoke, but there are too few studies to make final judgments. The experimental evidence suggests that patients with ischaemic heart disease could suffer a worsening of symptoms with exposure to environmental tobacco smoke.

Irritation

The most widespread acute physical effects of passive exposure to cigarette smoke are various types of "irritation." Eye irritation is the commonest complaint, but headaches, coughs, and irritation of the nose are also commonly reported. In one study cited in the 1984 Surgeon General's report, 69 percent of subjects reported eye irritation at some time in response to cigarette smoke (Speer, 1968). In one experimental chamber study (Weber, 1984), both a subjective and an objective measure of eye irritation were recorded. After an hour of exposure at smoke levels similar to those found in many public places, including offices, study participants reported increased eye irritation, and the objective measure, the rate of eye blinking, also increased. Eye irritation is also reported incidentally in various experimental studies of passive smoking. In a recent experimental study of adult asthmatics, the authors noted that, "Marked eye irritation was a universal finding," and that nasopharyngeal irritation was also common (Wiedemann et al., 1986). After several minutes in the experimental chamber, most subjects chose to wear goggles offered to protect their eyes from smoke.

There is sufficient evidence from surveys and observational studies that most people, including many smokers, are physically irritated by tobacco smoke. The means to test this belief is limited and few studies have done so, but the effect is generally accepted.

Health Effects: Summary

Taken piece by piece, much of the evidence for adverse health effects related to passive smoking is equivocal. As is the case for nearly every other body of health effects literature, there are few ''definitive" studies that by themselves change scientific thinking. Conclusions are drawn by examining the aggregate of studies and weighing their designs, flaws, and findings. In the case of passive smoking, the available evidence taken together supports stronger conclusions than do the individual studies.

Studies of respiratory effects suggest that people with asthma can be harmed by environmental tobacco smoke. Healthy adults may experience measurable disturbances of pulmonary function from passive smoke exposure. There is evidence that environmental tobacco smoke is an acute respiratory irritant, and an eye irritant. While the acute, short-term effects of passive smoking are by themselves relevant for a study of workplace smoking policies, their long-term health implications are less clear.

Evidence linking passive smoking to cardiovascular disease and symptoms is still rather scanty, but some studies suggest both acute exacerbation of angina pain and an increased risk of death from heart disease. The plausibility of these conclusions is supported by the known cardiovascular effects of direct smoking.

The epidemiologic evidence from a number of studies is generally consistent with the biologically plausible hypothesis that passive exposure to tobacco smoke can cause lung cancer. Taken together, the evidence points to a carcinogenic effect smaller than that observed for direct cigarette smoking. The published studies to date have not been free of flaws in