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on Environmental Tobacco Smoke

Mr. Chairman and Members of the Committee. We would like to thank you for the invitation to appear before you today to discuss the statistical basis for estimates of the health effects of passive smoking.

Please note that we are trained as economists and our area of expertise relates to economic analysis and the associated areas of statistical inference and quantification of effects for purposes of cost-benefit analysis and related economic policies. We do not have technical expertise in the physiological and biological transmission mechanisms of disease causing agents.

Our involvement in this issue was a result of a research paper we prepared on the proposed cigarette tax. This paper, which is now completed, is entitled Cigarette Taxes to Finance Health Care Reform: An Economic Analysis (CRS Report 94-214 E). In order to assess the economic efficiency of the proposed tax, it was necessary to examine the magnitude of any costs that smokers might impose on nonsmokers; the health effect of passive smoking is one aspect of this cost calculation. This led us to a review of the methodology used to assess the

scientific evidence on passive smoking.

Our evaluation of that evidence led to two conclusions: first, the evidence that passive smoking causes disease is far less certain than the effects for active smoking; second, the health costs of these potential passive smoking effects, which we translated into a tax per pack, are likely to be quite small.

The claim that passive smoking results in damage to the health of nonsmokers is based upon both theory and empirical analysis. If the theoretical case for the existence of passivesmoking effects is considered to be sound, it leads investigators to expect to find empirical support for the proposition. This theoretical case can be summarized in three steps: (1) environmental tobacco smoke has many of the same components as smoke inhaled by smokers;

(2) there is physical evidence of some absorption by passive smokers of these components; and

(3) a positive relationship exists between active smoking and additional disease and health costs, with no threshold observed.

Questions have been raised about this entire chain of reasoning, but the focus in our evaluation is the third link in the chain. This link is based upon evidence on active smokers who report different amounts of smoking. The difficulty with this theory is that even the lightest active smokers experience far greater exposure to and absorption of potential diseasecausing agents than do passive smokers. Thus the statistical evidence on active smoking, including evidence of greater damage as smoking increases, is a necessary but not a sufficient condition for establishing a link between passive smoking and health risks. That is, a threshold effect may exist between the lowest levels of active smoking studied and the levels of exposure in passive smoking.

Since the theory is not certain, one approach to studying passive smoking effects is to examine epidemiological ("epi") studies -- statistical studies of the incidence of diseases in human populations. Given the small risks that are often found for passive smoking, the statistical problems inherent in epidemiological studies are of far greater concern for passive smoking than for active-smoking studies. That is, when the effects are small, it is more likely that some error in design or specification could be responsible for the results. Given this greater uncertainty, consistency of the results with alternative evidence becomes more critical as a reality check.

An alternative method of estimating passive smoking effects is to extrapolate from active smoking studies based on the relative levels of physical exposure, using some type of biomarker which measures the absorption of substances in the body. This approach, sometimes called the 'cigarette-equivalent' approach, suggests a strong possibility that the relationship between passive smoking and disease incidence found in epidemiological studies is larger than expected, and that the statistical problems of the epi studies may be attributing disease incidence to passive smoking that is attributable to other factors. Thus, the combination of the greater statistical uncertainty of passive-smoking epi studies and the potential inconsistency of those results with physical exposure models is responsible for our conclusion that the finding of increased risk from passive smoking is "uncertain."

The remainder of this testimony provides the analysis upon which this conclusion is based. It begins with a discussion of the lung cancer evidence on passive smoking, first discussing the epi evidence. This is followed by a discussion of the physical exposure approach and its potential inconsistency with the epi results. The testimony then turns to a comparison of the epidemiological evidence and physical exposure approach for estimating the risk of heart disease from passive smoking, along with a brief mention of non-lung cancer and respiratory illness in children.

LUNG CANCER

Epidemiological Evidence

A number of epidemiological studies have assessed the effects of environmental tobacco smoke on specific diseases, with the largest body of research focusing on lung cancer among nonsmoking wives of smokers. Based upon these studies, several Government agencies have, in the last few years, taken the position that environmental tobacco smoke causes lung cancer in nonsmoking adults, including the Office of the Surgeon General and the Environmental Protection Agency's (EPA's) 1992 risk assessment that classifies environmental tobacco smoke as a cancer-causing agent.¹ Despite the controversy surrounding this latter report, the estimates of the risk of lung cancer deaths from passive smoking by the EPA are relatively small, amounting to a lifetime risk of death from lung cancer due to passive smoking of from one-tenth to two-tenths of a percent. The positions taken on passive smoking's effects on health by Government agencies and by the EPA 1992 assessment in particular have been subject to criticism by the tobacco industry and by some researchers.²

Our discussion draws on the evidence presented on both sides of the passive smoking issue with regard to the statistical and scientific evidence, but pays particular attention to the latest summary of this evidence, the EPA study.³ The EPA study analyzed and summarized 30 studies of passive smoking lung cancer effects. (1) Critics have questioned how a passive-smoking effect can be discerned from a group of 30 studies of which six found a statistically significant (but small) effect, 24 found no statistically significant effect, and six of the 24 found a passive smoking effect opposite to the expected relationship.

U.S. Department of Health and Human Services, *The Health Consequence of Involuntary Smoking*, 1986, Surgeon General Report, DHHS Publication Number (CDC) 87-8398; and United States Environmental Protection Agency, *Respiratory Health Effects of Passive Smoking. Lung and Other Cancer Disorders*, December 1992.

² A group of tobacco growers and manufacturers has filed a lawsuit challenging the EPA assessment as not being supported by the evidence. Among the issues raised is the use of empirical work based upon exposure in the home to draw inferences about health effects from exposure in the workplace.

EPA attempted to standardize this diverse group of studies to account for statistically important differences in their methodologies. In this process, EPA reduced the standard for statistical significance from the usual standard, and the one generally used in the original studies. It is unusual to return to a study after the fact, lower the required significance level, and declare its

results to be supportive rather than unsupportive of the effect one's theory suggests should be present, but our conclusion about the "uncertainty" of the EPA results is not dependent upon this change in significance levels.

However, the issue raised by the change in the statistical significance standard should not be ignored. The test of statistical significance used in these studies answers the following question: How large a chance, statistically speaking, is society willing to take that it accepts a conclusion that a passive-smoking effect exists when in fact a passive-smoking effect does not exist? In effect, EPA changed the standard from a two-and-a-half percent chance to a five percent chance of accepting an incorrect conclusion. The implication for policy is that society has accepted a greater chance of focusing resources on an unjustified intervention (from an efficiency standpoint).

3 These sources include the U.S. Department of Health and Human Services, Surgeon General Reports for 1986 and 1989; United States Environmental Protection Agency (1992), which detail the rationales for their positions. These reports also summarize the epidemiological studies on environmental tobacco= smoke, especially on lung cancer and childhood respiratory illness. The reader is also referred to a hearing at which researchers who both supported and criticized the EPA study appeared: U.S. Congress, House Committee on Agriculture Subcommittee on Specialty Crops and Natural Resources, Review of the U.S. Environmental Protection Agency's Tobacco and Smoke Study, 103rd Congress, 1st Session, July 1993. For a view that questions the passive-smoking hazard, focusing particularly on lung cancer, and that is written for the layman, Gary L. Huber, Robert E. Brockie and Mahajan, "Passive Smoking: How Great a Hazard?" Consumers' Research, July 1991, 10-16, 33-34. Huber, et al. wrote a companion paper on cardiovascular "Passive Smoking and Your Heart," Consumers' Research, Apra 1992, pp. 13-19, 33-34. Also, see Kyle Steenland, "Passive Smoking and the Risk of Heart " Journal of the American Medical Association, January 1, 1992, Vol. 267, pp. 94-99. These last two articles provide capsule summaries of epidemiological studies on passive smoking and heart. Finally, see The Tobacco Institute, EPA Report Scientifically Deficient for a summary of the industry's criticism of the EPA report. Some critics of the that passive smokes causes have also raised questions about institutional bias in the Government or in the professional journal; those issues are not addressed here.

(2) One important difference among the studies is the chance of accepting the absence of a passive-smoking effect when in fact a passive-smoking effect exists. The smaller the size of the sample (number of observations, or people, for whom data were available), the greater the chance of making such a mistake. To correct for these differences, EPA adjusted (weighted) the estimate of the passive-smoking effect in each study. This has the effect of reducing the importance of studies with small sample size, studies that would tend to find less significant effects for passive smoking, and increasing the relative importance of studies with large sample size, studies that would tend to find more significant effects for passive smoking.

(3) EPA adjusted the results of each study for misclassification bias (classifying smokers or former smokers as never-smokers). It also made subjective judgments about the extent to which the studies suffered from a variety of other statistical problems, such as confounding (failure to consider the influence of other factors that might increase lung cancer risk). Those that fared poorly in this analysis were placed in a "Tier 4" category and

excluded from the analysis of joint significance of the studies. This procedure allowed EPA to "emphasize those studies thought to provide better data..." (EPA, p. 6-61). After making all these adjustments, EPA combined the studies to conclude that, as a group, the remaining studies indicate that exposure to passive smoke produces a statistically significant increase in lung cancer among nonsmokers.

(4) Another test the EPA conducted was to examine the included studies for evidence of a positive relationship, within each study, between risk and degree of exposure (number of years the husband smoked, or number of cigarettes he smoked per day). They found such a relationship in 10 of the 14 studies for which such data were available. They also found that the highest-exposure-level group had higher risks than other groups combined, which was statistically significant in 9 of 16 comparisons. These results increased EPA's confidence in the integrity of the data, making it more willing to draw conclusions. This confidence comes from the fact that these results conformed to expectations. From our perspective, these results also are consistent with expectations about the functional form of the passive-smoking dose/response relationship. We will return to this issue in the section on the physical exposure approach.

(5) In addition, there are several potential statistical problems. These studies do not have (and indeed cannot have) very precise estimates of exposure from environmental tobacco smoke. The data are based on interviews of the subjects or their relatives. If errors in measurement occur in a systematic way that are correlated with development of the disease, the effect would be to bias the results. An example would be if those individuals who developed lung cancer (or relatives of those individuals) remembered or perceived their exposure differently from those who did not develop the disease.

Another concern is the possibility that some subjects classified as nonsmokers are actually current or former smokers and that such current or former smokers are more likely to be married to husbands that smoke. While EPA made some adjustment for this effect, it is not possible to correct precisely for this problem. That is, it remains possible that a relationship observed might reflect the effects of active rather than passive smoking.

In addition, while EPA considered the presence of certain confounding factors in its evaluation of some of the studies, this issue is not laid to rest. If wives of smokers share in associated poor health habits or other factors that could contribute to illness and that are not or cannot be controlled for, statistical associations found between disease and passive smoking could be incidental or misleading. Such an error could also render a relationship between risk and degree of exposure spurious.

In fact, there is evidence, as discussed in our cigarette tax study, that smokers are greater risk takers than nonsmokers and that they tend to engage in many other lifestyle habits that are not favorable to health. If smokers tend to be less concerned in general about health risks and engage in other behaviors (e.g. diet, lack of preventive health care) that might be shared with their spouses, these factors may be responsible for some share of the estimated increased health effects.

Such limitations of studies are often inevitable, but they impart some degree of uncertainty to the results, especially when small risks are estimated.

(6) Two epidemiology studies that each covered a large number of observations were published in 1992 after the cutoff date for inclusion in the EPA report. The one with the largest number of observations found no overall increased risk of lung cancer among nonsmoking spouses of smokers, the other found an increased, but statistically not significant, lung cancer risk. Both studies looked at exposure levels within their samples and both found a statistically significant increased risk among the highest exposure group in some categories. In smaller exposure groups, the first study found an unexpected negative relationship between passive smoking and disease and the second found a positive, but not a statistically significant, relationship. It has been pointed out that in large studies where the data are broken into several subsets and each is analyzed separately, some associations may be statistically significant as a matter of chance. 4 Ross C. Brownson, Michael C. R. Alava, Edward T. Hock, and Timothy S. Loy, "Passive Smoking and Lung Cancer in Women," American Journal of Public Health, November 1992, vol. 82, pp. 1525-1529. Heather G. Stockwell, Allan L. Goldman, Gary H. Lyman, Charles I. Nose, Adam W. Armstrong, Patricia A. Pinkham, Elizabeth C. Candelora, and Marcia R. Brusa, "Environmental Tobacco Smoke and Lung Cancer Risk in Nonsmoking Women," Journal of the National Cancer Institute, September 16, 1992, vol. 84, pp. 1417-1422.

Physical Exposure Relationship

An alternative approach to estimating the effects, if any, of passive smoking through statistical studies is through a physical exposure extrapolation approach. We believe a discussion of this approach will shed some light on why one might be concerned about the certainty of the epidemiological estimates. A physical exposure approach was discussed in our cigarette tax paper, and it was also contrasted with the statistical approach in a memorandum prepared by the CRS. We elaborate on those discussions.

As noted earlier, even the lightest smokers studied among active smokers experience far greater exposure to and absorption of tobacco smoke based on common biomarkers than do passive smokers. Therefore, such evidence on active smokers is necessary but not sufficient to conclude that a similar relationship exists for passive smokers. It is entirely plausible that the (unknown) dose/response function rises very little over the range of exposure (dose) levels for passive smokers and begins to rise rapidly as the exposure levels experienced by active smokers are approached.

The existence of an exposure threshold for disease onset below which many passive smokers fall is not implausible. Most organisms have the capacity to cleanse themselves of some level of contaminants. It is for this reason that public policy usually does not insist that every unit of air or water pollution be removed from the environment: the damage of low levels of pollutants is sufficiently small that removal is not cost effective. In fact, strongly nonlinear relationships in which health effects rise with the square of exposure, and more, have been found with respect to active smoking (see Surgeon General's Report, 1989, p. 44). Were these relationships projected backward to construct the lower (unknown) portion of the dose/response function, the observed relationship might lead researchers a priori to expect no empirical relationship.

In fact, the EPA report dismisses linear extrapolation from the

active-smoking dose/response relationship to estimate passive smoking effects. Numerous reasons are given for the decision not to make such an inference. The most interesting reason is a suggestion that extrapolation might underestimate the response, exactly the opposite of what the discussion above suggested. That is, if the relationship were such that disease rose with the square or more of exposure, or if there were a threshold, a linear extrapolation would overstate the response. The support for this position that linear extrapolation would underestimate the response is based upon a paper by Remmer⁶ that suggests small amounts of carcinogenic substances are large enough to begin the disease process but are too small to activate the body's defenses against the disease. In effect, this suggests there is no threshold for disease onset, but there is a threshold for the body's automatic disease fighting mechanisms. Thus, depending upon the relative strengths of the disease and immune responses as dosage increases, marginal disease per unit of dosage could cause the observed average dose/response relationship to increase, decrease, or remain the same as dosage increases for the dosage range that includes passive smoking.

If this is the case, one wonders why EPA's confidence in the epi lung cancer studies was increased by its investigation of the dose/response relationship within the individual studies. Referring back to the discussion in the epi section of this testimony, EPA's theory about a threshold effect for the immune response to exposure should have lead them to expect no particular dose/response relationship.

How do the actual numbers estimated using the different approaches compare? The epi studies indicate an additional risk for lung cancer due to marriage to a smoking spouse for female never-smokers of about 30 percent. That is, according to their analysis of the statistical studies, nonsmoking wives of men who smoke have 30 percent more lung cancer than nonsmoking wives of men who do not smoke. This risk is, in turn, only a tiny fraction of the risk from active smoking (probably around 3 percent)⁷. The risk in the United States epi studies was slightly lower, only about 20 percent. Based on the epi studies' 20 percent risk factors for the U.S., the EPA estimated 3000 lung cancer deaths from passive smoking, 2000 for those who are never-smokers and 1000 for former smokers.⁶ H. Remmer. *Passively Inhaled Tobacco Smoke: A Challenge to Toxicology and Preventive Medicine*. Archives of Toxicology, vol. 61, pp. 89-104.

Extrapolation based on physical evidence yields smaller effects. According to data in the EPA report, measures of cotinine in the urine indicate that, overall, passive smokers have about 1/2 of one percent of the level of active smokers. Or, to put it another way, given that the average smoker smokes about 20 cigarettes a day, the passive smoking effect is equivalent to smoking a tenth of a cigarette a day.

In comparing the physical exposure extrapolation approach to the epi estimates, it is simpler to compare the effects on never-smokers. Since the number of current and former smokers are the same as the number of never-smokers, the estimated premature deaths annually from passive smoking for never-smokers would be about 600 using a linear extrapolations This number is considerably less than the EPA's estimate of 2000 never smoker deaths."

⁷ The risk of lung cancer in smokers and ex-smokers depends on intensity, duration, and, in the case of exsmokers, time elapsed since quitting. Passive

smoking would involve three percent of the risk of active smoking if there is a ten fold active-smoking risk (i.e. smokers have an additional estimated risk of lung cancer that is ten times the rate of nonsmokers) which is typical of current estimates of the risk for women as reported in the 1989 Surgeon General's Report, Reducing the Health Consequences of Smoking. U.S. Department of Health and Human Services, DDHS Publication No. (CDC) 89-8411). In generating the estimates of deaths from passive smoking, the EPA actually used the additional risk (of wives married to smokers as compared to wives married to nonsmokers) in the U.S. passive smoking studies, which was about 20 percent. If studies from all countries are considered, the estimated risk from these studies was 30 percent.

8 Actually, the only number that was directly estimated from the epi study was the calculation of slightly under 500 deaths due to increased risks for women married to nonsmokers. This number was, in fact, extrapolated backward using a linear physical exposure method to calculate an additional 1000 deaths from other sources of environmental exposure (e.g. workplace, animal), for a total of 1500 female never-smoker deaths. A further extrapolation yielded 600 additional deaths for male never-smokers, and another 1000 for both male and female former smokers. Lung cancer deaths attributable to passive-smoking are two to three percent as large as the estimated 113,000 lung cancer deaths attributed to active-smoking. For data on widely accepted estimated active smoking deaths, see C. Stephen Redhead, Mortality and Economic Costs Attributable to Smoking and Alcohol Abuse, Congressional Research Service Report 93-SPR, April 20, 1993.

There are potential problems with the physical exposure measure as well as with the epi approach. The physical extrapolation method used above assumes a linear relationship between the incidence of a disease and exposure. Based on evidence from the pattern for active smoking, however, a linear method may not be correct. There is some evidence that disease rises with square of the exposure or even with higher powers in the case of lung cancer." If the disease were to rise with the square of exposure, then the estimate based on cotinine levels would be only 3 people rather than 600 people. Thus, in this case the epi studies suggest 2000 deaths of never smokers and the physical exposure measure suggests 3 deaths and the contrast between the two approaches is even greater.

It is also possible that cotinine is not the best measure of exposure; as discussed in the EPA study some exposure measures show larger and some show smaller effects. It is worth noting that the EPA chose the epidemiological studies as a basis of their approach, but they nevertheless relied on the cotinine measures for several aspects of their estimates (such as extrapolating from the effects on spouses of smokers to the population in general).

HEART DISEASE AND OTHER CANCER

Many of the statistical concerns raised above with regard to lung cancer are relevant to respiratory effects in children and heart disease in adults. Indeed, the conclusions by these 113,000 deaths attributable to active smoking.

9 To extrapolate, multiply the ratio of cotinine (.005) by the ratio of never-smokers to ever smokers.

10 There is also a section in the EPA study that extrapolations based on the physical exposure to passive smoking-, these estimates also tend to be smaller -- in some cases, much smaller--than the epidemiological estimates.

11 Surgeon General's Report, p. 44.

Government agencies about passive smoking and lung cancer are generally not extended to heart disease or other health effects in adults. The presence of other factors that may be related to these illnesses that are not controlled for are particularly important in the case of heart disease, general respiratory illness, and cancers in other parts of the body, where the link between active smoking and the disease is not as strong as in the case of lung cancer.

In addition, the differences between deaths estimated from epi studies and from physical exposure extrapolations are much more pronounced in the case of heart disease estimates.

Recall that the EPA estimate of lung cancer deaths from passive smoking was 3000. There has also been widespread reference to an estimate of 50,000 deaths attributable to passive smoking. The 50,000 estimate has been circulated by non-government organizations, and was mentioned in testimony by the American Medical Association which stated that passive smoking "may kill as many as 53,000" Americans annually." This statement in turn appears to be ultimately traceable to an article by Wells published in 1988 in Environment International. This article used existing epidemiological studies to estimate these deaths which included, under one set of calculations, 3,700 lung cancer deaths, 12,300 deaths from other cancers, and 37,400 deaths from heart disease. (Wells actually reported estimates ranging from 38,000 to 53,000, with a preferred estimate of 46,000)."

12 Statement of the American Medical Association, Health and the Environment Subcommittee, House Committee on Energy and Commerce, Re Adverse Health Effects of Exposure to Environmental Tobacco Smoke, July 21, 1993.

13 A Judson Well, An Estimate of Adult Mortality in the United States from Passive Smoking. Environment International. Vol. 14, No. 3, 1988, 249-265.

14 Each issue of Environment International contains an editorial; the one in the issue containing the Wells article was directed at that article. The editorial indicated that the study received mixed reviews from referees (two recommended publication after revision and the third recommended against publication on the grounds that it was too speculative); the editors chose to publish the paper. In the following three years there were a series of critiques and rejoinders related to this paper. Letters from Alan W. Katzenstein, Peter M. Lee, and Larry Holcolm criticizing the Wells results; a clarifying letter from Takeshi Hirayama, a rebuttal to Katzenstein and Lee from James L. Repace and Alfred H. Lowrey, and a response from Wells were published in 1990 (Vol. 16, no. 2, pp. 175- 193). In 1991, a letter from Stanton A. Glantz criticizing Lee was published along with Lee's reply (Vol. 17, no. 1, pp. 89-91). Later in 1991, a response of Lee to the 1990 letters of Repace and Lowrey, and Wells, a letter from Muin J. Khoury clarifying a point raised in Lee's letter, a joint letter from Glantz and Lee clarifying an issue raised earlier.

While the estimates from at least some of the epidemiology studies of lung cancer are significantly larger than the estimates based on physical exposure, these results are not magnitudes apart. The same cannot be said, however, for the Wells estimates of deaths from heart disease. Using the same type of linear physical exposure extrapolation as in the previous section would result in 700 deaths from coronary disease for never smokers, and perhaps another 350 for former smokers, with a total of about 1000. The comparable portion of the Wells' 53,000 estimate from the epidemiological studies, even for several years ago, is 37,000, a number that is enormously larger.

This large estimate occurs because the epidemiological studies, on the whole, show a very high risk estimate for passive smoking relative to active smoking for heart disease as compared to lung cancer. For example, Wells indicates a 30 percent additional risk for heart disease for males and a 20 percent rise for females resulting from exposure to passive smoke, as compared to a 70 percent risk for smokers. The passive-smoking deaths associated with these relative risks are immense compared to both the physical exposure extrapolation estimates for heart disease and to either method for lung cancer. Note that although the risk ratios are not that different from lung cancer, the absolute risk estimates are much larger. The risk of lung cancer for nonsmokers is very low, and any percentage of a small number is still a small number. The estimated risk for heart disease is much larger initially, and therefore any significant percentage change in the risk is larger. Put another way, even the epidemiological studies of lung cancer produced passive-smoking deaths of less than 3 percent of active-smoking deaths, while the heart disease studies produced estimates that were 26 percent of estimated active-smoking deaths.

The biological plausibility of passive smoking effects on cardiovascular diseases has been the subject of some discussion.¹⁵ A likely explanation of these apparent large risks from passive smoking found in epidemiological studies for heart disease is, however, the absence of control for other factors. There are many important causes of heart disease (e.g. diet, lack of exercise, lack of preventive health care) that may be engaged in by smokers. There is much evidence that smokers tend to be less concerned about health risks in general. In general, studies do not, and perhaps cannot, control for many of these factors. If smokers' wives share in these behaviors, the relationships found in the epidemiological studies are spurious.

The Wells estimate of passive-smoking deaths from cancers other than lung cancer is even larger relative to active-smoking deaths than is the case of heart disease—about 50 percent. Again, these cancers are influenced by many other factors, and the same general criticisms can be made about these epidemiological estimates as in the case of heart disease.

In sum, this analysis suggests that the Wells estimates are so high relative to measures of physical exposure that they seem implausible. It also suggests that the absence of controls or the inability to control for other factors may be a major problem in relying on epidemiological estimates of the health effects of passive smoking. To restate this criticism, if wives or children of smokers share in poor health habits or other factors that could contribute to illness, statistical associations found between disease and passive smoking could be incidental or misleading.

¹⁵ The argument has been made for a relationship in which passive smoking

can have large effect relative to active smoking in some laboratory settings, which is largely attributed to increased sensitivity of some nonsmokers. See Stanton Glantz and W Parmley, *Passive Smoking and Heart*, vol. 63, no. 1, January 1991, pp. 1-12.

16 This position is taken by Gary L. Hubert, Robert E. Brockie, and Vbay K Mahajan in a paper written for the layman: *Passive Smoking and Your Heart*, *Consumer8 Research*, vol. 75., April 1992, pp. 13-19, 32. These authors consider the results in the Wells study and the simflar heart diseases study by Steenland (1992) biologically implausible, and also note that six of the nine epidemiological studies show relative risks for passive smokers that are in excess of risks estimated for active smokers and that most have very few controls for the other factors that might affect heart disease.

CONCLUSION

Our assessment of the existing evidence on passive smoking was made as a basis for drawing conclusions about the efficiency justifications for an increase in the cigarette tax. Based on that evidence, as indicated in this testimony, our evaluation was that the statistical evidence does not appear to support a conclusion that there are substantial health effects of passive smoking. This finding flows from an analysis of the statistical methodology employed in assessing such health effects and purports to no technical research or conclusion on the physiology of disease-causing agents.

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