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BEFORE THE UNITED STATES
FEDERAL AVIATION ADMINISTRATION

Petition To Initiate Rulemaking To Amend Federal Aviation Regulations: Certification and Operation (Subpart K -- Instrument and Equipment Requirements)	))))))	Petition Docket No. 26566

COMMENTS OF THE R. J. REYNOLDS TOBACCO COMPANY ON PETITIONERS' REQUEST TO PROHIBIT TOBACCO SMOKING ON ALL U.S. CARRIERS

Filed: September 16, 1991

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INTRODUCTION

On July 19, 1991, the Federal Aviation Administration ("FAA") published a summary notice of a Petition (the "Petition") submitted by the Union of Flight Attendants, the Independent Union of Flight Attendants, the Independent Federation of Flight Attendants, and the Association of Professional Flight Attendants (collectively referred to as "Petitioners"). 56 Fed. Reg. 33213. The Petitioners request, among other things, that the FAA prohibit tobacco smoking on all U.S. carriers. 56 Fed. Reg. 33214. They base this request on the assertion that prohibiting tobacco smoking on all U.S. airliners would protect airliner crew and passengers from health risks caused by environmental tobacco smoke ("ETS") exposure.[1] FAA solicits comments on the Petition by September 16, 1991. 56 Fed Reg. 33213.

These comments are submitted by the R. J. Reynolds Tobacco Company ("RJR"). Section I of these comments discusses considerations of separation of powers, delegation of authority, and comity with sister agencies counselling that FAA should summarily reject the Petition. Section II responds to Petitioners' incorrect allegations regarding the level of ETS in airline cabins. A review of the literature demonstrates that ETS levels in airline cabins are extremely low and not significant. In Section III, Petitioners' allegations regarding ETS exposure and health are addressed. Those allegations are largely conclusory in nature with minimal reference to supporting data. Petitioners reiterate a partial list of conclusions of other reviewing organizations, without pointing out inaccuracies and subsequent research which cast doubt on those conclusions, and rely on an unbalanced subset of individual studies which have made sensational claims. They make no attempt to provide FAA with a helpful

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[1] The United States Congress has prohibited smoking on virtually all flights of six hours or less duration between domestic points. 49 U.S.C.A. App. § 1374. Petitioners request that FAA extend that prohibition to all flights by U.S.-based carriers, regardless of the flight's origin or destination.

review of the existing data on ETS and health. These comments address the Petitioners' specific allegations regarding ETS. Additional information can be supplied upon request. Section IV addresses smoking on airliners and fire safety. Finally, Section V critiques the Petitioners' limited and deceptive discussion of mitigation options and cost/benefit relationships.

I. FAA SHOULD REFRAIN FROM EXERCISING AUTHORITY TO BAN SMOKING ON ALL U.S. CARRIERS

The Petitioners devote an inordinate amount of space to the proposition that FAA should ban smoking on all flights because of its "responsibility for ensuring a healthy and safe aircraft working environment." Regardless of FAA's authority to address occupational safety and health issues, the Petitioners ignore an important point: Congress attempted to balance competing considerations in creating existing airline smoking restrictions, and only Congress should disturb that balance.

Congress has banned smoking on virtually all flights within the United States. Conspicuously absent, however, is a smoking ban on international flights. Congress has not instructed FAA to go beyond the statutory proscriptions, and FAA should not exercise such authority independently. In fact, Congress rejected legislation which would have banned smoking on both domestic and international flights. Petitioners should not be allowed to lure FAA into a regulatory arena that Congress considered and avoided. For these reasons alone, FAA should summarily reject this Petition.

FAA should also consider the fact that the Occupational Safety and Health Administration ("OSHA") is now considering indoor air quality issues, including workplace smoking. Unlike OSHA, FAA has limited expertise evaluating claims in the nature of those that Petitioners make. These factors counsel strongly in favor of rejecting the Petition.

II. PETITIONERS HAVE NOT DEMONSTRATED THAT ETS CONCENTRATIONS ON INTERNATIONAL FLIGHTS REQUIRE REGULATION

A. The Levels of ETS in Aircraft Cabins Are Low

The Petitioners attempt to create the impression that they are exposed to high levels of ETS. The data do not support this assertion. The overall literature on ETS in aircraft cabins shows that ETS levels are low.

This literature extends back to 1971 when the Department of Transportation ("DOT") and the Department of Health, Education and Welfare ("DHEW") reported ETS measurements in passenger cabins of military and domestic aircraft. This research was conducted before segregation of smoking and nonsmoking passengers was required. Concentrations of respirable suspended particulates ("RSP") averaged 40 $\mu g/m^3$ in the DOT/DHEW study.[2] The DOT/DHEW report concluded that the levels of ETS in aircraft were below recommended standards:

> The results of environmental sampling revealed very low levels of each constituent measured, much lower than those recommended in occupational and environmental air quality standards.
>
> These combustion products [ETS] were judged not to represent a hazard to the nonsmoking passenger, based on environmental levels and expected dosage-response relationships of contaminants.

(DOT/DHEW, 1971).

In 1985, Japanese researchers measured nicotine in smoking and non-smoking sections of Japanese domestic flights and found nicotine concentrations ranging from 6.28 to

[2] The NRC report discussed below erroneously states that the average RSP concentration observed in the DOT/DHEW study was 140 $\mu g/m^3$.

28.78 $\mu g/m^3$. (Muramatsu, 1985). To put these levels in perspective, FAA should note that the U.S. occupational standard for nicotine is 500 $\mu g/m^3$ for an eight-hour work day.

Oldaker and Conrad reported more than seventy measurements of nicotine in smoking and nonsmoking boundary sections of Boeing 727 and 737 aircraft on domestic flights in the United States. (Oldaker, 1987). Average nicotine concentrations were 22.4 $\mu g/m^3$ in the smoking section and 9.3 $\mu g/m^3$ in the nonsmoking boundary. These data are comparable to those reported by Muramatsu, et al.

Malmfors, et al. measured nicotine in DC9 and MD80 aircraft on Scandinavian Airlines System ("SAS") flights connecting European cities. (Malmfors, 1989). These researchers reported average concentrations of nicotine in nonsmoking sections of Tourist and Business Classes of service. The nicotine concentrations were comparable to those reported earlier by other researchers. Of particular significance in the context of this Petition, the authors found that ETS levels declined significantly as the duration of a flight increased. The implication of this finding is that levels of ETS exposure for international flights can be expected to be less than those for domestic flights.

Data reported by Drake and Johnson from an investigation in B747s on four flights connecting New York, Tokyo and Hong Kong confirm this expectation. (Drake, 1990) They reported that average nicotine concentrations on these flights were 10.48 $\mu g/m^3$ in the smoking sections and 2.54 $\mu g/m^3$ in the nonsmoking sections. These measurements are lower than the measurements reported by Oldaker and Conrad and Muramatsu, et al., for domestic flights in the United States and Japan, respectively.

In sum, nicotine levels aboard international and domestic flights are low. Actual measures of ETS in airline cabins on domestic flights show that the nicotine levels are well

below the existing occupational standard, and the ETS levels on international flights are even lower than those on domestic flights.

B. Smoking Sections Do Not Create High Levels of ETS

Petitioners rely on two isolated references ("The Airline Cabin Environment: Air Quality and Safety," NRC, 1986a; "Passive Smoking on Commercial Airlines," Mattson, et al., 1989) for the propositions that:

(1) Because ETS can be detected in nonsmoking sections of aircraft, all flight attendants on smoking flights are exposed to ETS; and

(2) Ventilation aboard aircraft is inadequate to allow smoking.

With respect to the first proposition, the Petitioners' argument can be reduced to the tautology that where smoking is permitted, ETS can be detected. The issue for FAA, however, is not whether any indicators of ETS exposure can be detected, but whether those exposures are significant.

Mattson, et al. undertook their study:

> (1) to measure nicotine levels in ambient air during flights . . . and urinary cotinine levels at various points during the three days after the flights, and (2) to determine if these exposure and excretion measurements correlate with each other and with acute symptoms experienced during the flights.

(Mattson, 1989). What their data demonstrate is unremarkable.

Flight attendants and passengers participating in the study wore sampling devices that collected airborne nicotine during four flights. Like other researchers, Mattson, et al. found low concentrations of airborne nicotine. For attendants assigned to smoking sections, nicotine concentrations ranged from 0.5 to 10.5 $\mu g/m^3$ with an average of 5.0 $\mu g/m^3$. For attendants assigned to nonsmoking sections, nicotine concentrations ranged from 0.1 to 9.8

$\mu g/m^3$ with an average concentration of 4.3 $\mu g/m^3$.[3] The authors reported that exposure among attendants did not statistically differ from the passengers, though the highest individual readings were among the passengers.[4]

Interpretation of the urinary cotinine (a metabolite of nicotine) measurements made by these researchers is difficult because they do not provide the raw data for the subjects, as they do for the airborne nicotine measurements. (Insofar as attendants' exposures aboard smoking flights is concerned, it is intriguing to note that 11 of 16 attendants' urine samples were rejected from some analyses because of other nonflight airborne nicotine exposures.) The authors reported that urinary cotinine could be detected in the "high" exposure group.

This finding is neither novel nor surprising. Foliart, et al. (1983) measured for ETS exposure indicators in the body fluids of flight attendants on transoceanic flights. They measured nicotine and carbon monoxide in blood and concluded that there was evidence of ETS exposure. They further concluded, however, that the exposure levels found "are unlikely to have physiologic effects."

As to the "acute symptoms" studied by Mattson et al., again the authors provided no raw data. The subjects completed a simple questionnaire before and after each flight. The authors reported that dry mouth, coughing, sneezing, scratchy or sore throat, and headache were not significantly related to either nicotine exposure or their analysis of urinary

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[3] The Mattson *et al.* results for nicotine concentrations are consistent with reports, both before and since, by other scientists; *e.g.*, Muramatsu, *et al.* (1987); Oldaker and Conrad (1987); Oldaker, *et al.* (1988); Drake (1990); and Malmfors, *et al.* (1989).

[4] The authors note that "[n]o passenger subjects were placed in the center of the nonsmoking section far from the border with smoking because of the expectation that exposures there would be very low."

cotinine data. Using dubious statistical techniques, the authors claimed that changes in eye symptoms, nose symptoms, annoyance with smoking and perception of a smoky environment were related to those measures.

As noted by Crawford and Holcomb (1991), "passengers and flight attendants have complained about eye irritation, headaches, nose and throat irritation and breathing discomfort." They conclude, however, that:

> [T]he levels of ETS constituents are not high enough to result in the symptoms most commonly reported by airline attendants or passengers. These symptoms are much more likely to result from elevated levels of ozone or abnormally low relative humidity, neither of which is uncommon on long flights.

Symptoms from ozone exposure under simulated flight conditions include eye discomfort, headache, nasal irritation and throat irritation. (Crawford and Holcomb, 1991). Decreased pulmonary function and asthmatic symptoms may also occur. (Holcomb, 1988). Reported cabin relative humidity values range from 8.5 to 25% -- below the threshold of 40% for symptoms of dry mucous membranes and eye irritation. (Crawford and Holcomb, 1991).[5]

Petitioners' assertion that ventilation aboard aircraft is not sufficient to permit smoking is based upon an inadequate body of scientific information. The ventilation "findings" and concomitant recommendations drawn from the 1986 NRC report were based upon

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[5] There are many sources of air contamination in airline cabins unrelated to tobacco smoking. O'Donnell, et al. evaluated air quality in aircraft cabins of seven identical aircraft on 45 flights with durations ranging from 30 minutes to one hour. (O'Donnell, 1991). Smoking was allowed on none of the flights. Arithmetic mean concentrations of carbon dioxide, carbon monoxide, and total particulates were 719 ppm, 1.6 ppm, and 105 $\mu g/m^3$, respectively. Respective maximal concentrations were 2,170 ppm, 4 ppm, and 200 $\mu g/m^3$. A substantial number of the results from determinations of CO_2 exceeded the ASHRAE guideline of 1,000 ppm. To some extent, these results put the issue of smoking in aircraft in a new perspective, since they suggest that banning smoking has little effect on overall air quality in the cabin; thus, the measured concentrations of these indicators of air quality are comparable to those which researchers have reported for cabins where smoking was permitted.

minimal evidence drawn from the airline cabin environment. DOT commissioned the NRC to study aircraft cabin environment. The NRC's report included a recommendation that smoking be banned on domestic flights. DOT expressly refused to adopt this recommendation and concluded that further study was needed in areas such as health effects and concentrations of ETS. DOT, in fact, quoted directly from the NRC report as justification for further review:

> Empirical evidence is lacking in quality and quantity for a scientific evaluation of the quality of airliner cabin air or of the probable health effects of short or long exposure to it.

(DOT, 1987).

The assumption that aircraft ventilation is inadequate to permit smoking is belied by the fact that nicotine concentrations on aircraft are so low. Oldaker and Conrad (1987) noted that nicotine concentrations observed in their study (which included some aircraft recirculating approximately 40% of cabin air) were "substantially lower than mean levels observed in [other] environments where the density of smokers is similar." They attributed "the observed relatively low nicotine concentration levels and the absence of significant correlation with number of smokers" to the design of the aircrafts' heating, ventilation and air conditioning systems.

Mattson, et al. (1989) found that airborne nicotine levels were lower in the two flights with 100% fresh air than in the two flights with 50% recirculated air. However, the key finding of the Mattson study remains that all airborne nicotine measurements, regardless of aircraft type and regardless of ventilation system, are small by any standard. The data do not show that ventilation is inadequate to permit smoking on any type of aircraft. Theorizing that ventilation may become inadequate as the airline fleet includes more planes that recirculate cabin air does not change the data.

Petitioners' allegations simply are not supported by data. Because ETS levels are low, sensory effects reported by flight attendants and others aboard aircraft are more likely to be caused by factors other than ETS. Moreover, the Petitioners have not made a case that aircraft ventilation is inadequate to permit smoking.

III. ETS IS NOT A PROVEN CAUSE OF DISEASE IN NONSMOKERS

Petitioners paint a frightening picture about ETS and health by claiming that "[t]he scientific evidence is now abundant and irrefutable that environmental tobacco smoke causes lung cancer and other respiratory illnesses in healthy nonsmokers." As will be discussed below, Petitioners fail to acknowledge the wealth of data that suggest contrary conclusions. Their efforts to oversimplify, misuse, and obscure the relevant data should be rejected.

A. ETS and Health

The Petition includes a series of sweeping conclusions without providing any underlying data. The Petitioners would have FAA believe that these conclusions represent all the available data.[6] The Petition does not (1) describe the nature of the data; (2) analyze the data; (3) point out the selective nature of the few studies cited; or (4) provide any guidance to FAA for its review of the data.

Studies of ETS and human health have been limited largely to epidemiologic investigations. Epidemiology is the study of disease incidence in human populations. Epidemiologic studies are observational in nature; changes or differences in one factor such

[6] Over 6,000 scientific studies of ETS have been published in the past decade. More are being published at an accelerating rate. This phenomenon alone belies Petitioners' characterization of the scientific issues as resolved "beyond doubt."

as ETS exposure are studied in relation to changes or differences in another variable such as disease occurrence.

If properly conducted, epidemiologic studies can demonstrate an apparent statistical correlation between a factor and the incidence of a disease in a human population. In contrast to experimental studies, however, the investigators simply observe fluctuations in both exposure and disease incidence. This leads to the major weakness in epidemiologic studies -- the inability to control and account for other factors that could affect disease occurrence. These factors are referred to as "confounding factors." The existence of confounding factors is one of the principal reasons why epidemiologic data alone cannot establish causation. Epidemiologic data is only one part of the evidence required to draw a causal inference.

Several government agencies have formulated criteria or guidelines for assessing epidemiologic evidence. (e.g., USSG, 1964; EPA, 1986). The more important of the criteria are:

1. statistical significance and strength of the association
2. consistency of the association
3. freedom from bias and confounding
4. presence of a dose-response relationship
5. biological plausibility of the observed association

Statistical significance tests are used to determine the probability that an observed association could be attributable to chance alone. According to EPA guidelines, chance must be ruled out before a causal association can be inferred between an exposure and disease. (EPA, 1986). Only 6 of 30 epidemiologic studies of the association between lung cancer and spousal smoking (used as a surrogate of ETS exposure) have reported a statistically significant result.

Strength refers to the magnitude or size of the observed association. The weaker or smaller an association, the less reliable it is as evidence. Weak associations are more likely than strong associations to be artifacts produced by flaws in the study, such as failure to control for confounding factors, or its analysis. Although there is no precise definition of a "weak association," relative risks of less than 2.0 to 3.0 are generally considered to be weak and therefore unreliable. (Wynder, 1987; Doll, 1985.) The reported lung cancer relative risks from female spousal exposure to ETS range from 0.7 to 2.55. None of the reviewing organizations cited by Petitioner have squarely addressed the problems inherent in interpreting weak associations.

An association is consistent if it is observed in different study designs, across different populations in different time periods. Relative risks of similar magnitude should be observed for similar exposures under varying conditions. This cannot be said of the studies of ETS and lung cancer in view of the range of relative risks reported and the fact that the overwhelming majority of studies did not achieve statistical significance.

An epidemiologic study must be free from bias. Bias refers to any trend in the collection, analysis, interpretation, review or publication of data that can lead to conclusions that are systematically different from reality. Studies of ETS and health are subject to a number of sources of bias:

- Misleading differences between groups can result from the way the subjects were either selected or excluded and the way the data were collected;
- Improper matching of controls to cases is a potential source of bias in many of the case-control studies;
- Measurement errors and misclassification of the study subjects regarding one or more variables may

be systematically distorting the results in many studies; and

- Misclassification of study subjects' exposure status, health status and smoking status plagues most of the data.

(Lee, 1988).

Bias also can operate to distort the published literature apart from effects on the results of individual studies. Publication bias, for example, refers to the tendency of scientific and medical journals to accept studies reporting positive results. Studies failing to identify an association or identifying a negative association are less likely to be published. (Mann, 1990; Chalmers, 1990; Begg and Berlin, 1989). As a result, the published literature consists of an unrepresentative set of the studies actually performed.

Confounders are factors that are statistically correlated both with the exposure under study and with changes in the incidence of the disease under study. As such, they are possible alternative explanations for the observed association. Most epidemiologic studies of ETS and health employ an exposure surrogate such as spousal smoking as an index of exposure. This technique admits significant confounding, especially when one considers the recent data indicating that spouses of smokers adopt lifestyles which closely mirror the lifestyles of their smoking spouses. Those lifestyles include many reported risk factors for lung cancer including diet and lack of exercise. (Butler, 1991; Sydney, 1989; Rylander, 1989; Koo, 1989).

A dose-response relationship means that a risk is observed to change in proportion to the degree of exposure. As the extent of ETS exposure, and hence the amount of the dose received, increases, so too should the incidence of the disease, if the observed association is valid. In assessing the existence of a dose-response relationship, the non

exposed subjects should not be considered. In studies of ETS and health, this means that only nonsmokers exposed to various levels of ETS should be examined for presence of a dose-response relationship; nonsmokers not exposed to ETS should not be considered. (Layard, 1989.) Most, if not all, analyses of dose-response data from the ETS studies have inappropriately included the non-exposed group. Nonetheless, dose-response relationships have been observed infrequently.

To be biologically plausible, an observed statistical association must make sense given what is known about the biology of the agent, exposure, subject, and disease. This requirement contemplates use of mechanistic data and experimental data, knowledge of the chemistry involved, and other biological considerations. (EPA, 1986). Based on the extremely low levels of ETS encountered in everyday life, the observed associations for ETS and lung cancer are implausible and likely to be statistical artifacts. (Gori, 1991).

Epidemiologic studies of ETS and health should not be over-interpreted. Even appropriately conducted studies can demonstrate only a statistical association -- not causation. Studies of multifactorial diseases and environmental exposures, such as ETS, that produce only weak associations must be controlled rigorously for other potential risk factors. Before determining whether a valid statistical association has been demonstrated, the statistical significance, strength, consistency, specificity, presence of a dose-response relationship and methodological soundness of the study, including freedom from bias, must be assessed. The totality of the evidence, including evidence that some negative studies have not been published, must be weighed in evaluating ETS and health. The reviews of the available ETS epidemiology which have concluded that ETS is causally associated with chronic disease have not followed these guides in reaching their conclusions.

B. Lung Cancer and ETS Exposure During Childhood

The Petitioners' statement that the Janerich, et al., 1990 study demonstrates that children exposed to ETS are at a significantly increased risk of developing lung cancer during adulthood is disingenuous at best. As reflected in Table I, at least ten groups of investigators have examined whether childhood ETS exposure is associated with lung cancer. Nine of those studies found no statistically significant increase in risk. Half of the studies reported a nonsignificant decrease in risk for at least one ETS exposure index. Petitioners' attempt to infuse childhood ETS exposure and lung cancer into the debate is an appeal to emotion, not reasoned decision making.

TABLE I

Relative Risks For Exposure During Childhood

	Study	Sex	Index of Exposure	Relative Risk (95% Confidence Limits)
Case-control studies				
1.	Correa, 1983	M+F	Maternal or paternal smoking	"No significant increase in risk"
2.	Garfinkel, 1985	F	Exposed during childhood	0.91 (0.74-1.12)
3.	Wu, 1985	F	Parents in childhood	0.60 (0.2-1.7)
4.	Akiba, 1986	M+F	Maternal or paternal smoking	"No association"
5.	Gao, 1987	F	Lived with smoker during childhood	1.1 (0.7-1.7)
6.	Koo, 1987	F	Household exposure to cigarette smoke during childhood	2.07 (0.51-95.17)
7.	Pershagen, 1987	F	Parents smoked	1.0 (0.4-2.3)
8.	Svensson, 1989	F	Father smoking at child's age 0-9 Mother smoking at child's age 0-9	0.8 (0.5-1.4) 1.8 (0.5-7.0)
9.	Janerich, 1990	M+F	Smoker years of exposure before age 21 1-24 greater than or equal to 25	 1.09 (0.68-1.73) 2.07 (1.16-3.68)*
10.	Sobue, 1990	F F F	Smoking by father Smoking by mother Smoking by other family members	0.60 (0.40-0.91) 1.71 (0.95-3.10) 1.13 (0.69-1.87)

*Statistically significant at the 5% level.

C. Cervical Cancer

Petitioners rely on a single study, by Slattery et al., on ETS and cervical cancer to promote their thesis. Virtually all of the published data cast serious doubt on Petitioners' assertions about ETS and cervical cancer.

Slattery, et al. examined smoking and ETS exposure as risk factors for cervical cancer in a population-based case-control study conducted in Utah. (Slattery, 1989). They reported a relative risk of 3.43 (95% CI = 1.23, 9.54) for cervical cancer in nonsmoking women reporting over three hours of ETS exposure per day. This relative risk was the same as that reported for current cigarette smokers. The relative risk was adjusted for age, church attendance, education, and number of sexual partners (but not, surprisingly, for being Mormon). Socioeconomic status as reflected by income and education, number of sexual partners, and frequency of church attendance were found to be confounding variables with ETS exposure.[7]

An editorial comment by Layde (1989b), published in the same journal, pointed out that measures of sexual activity, such as number of sex partners, are unlikely to completely reflect exposure to the causal agent. The editorial noted that the reported relative risk probably overestimates the risk from ETS exposure. Slattery et al. acknowledged that misclassification of sexual history could result in an inflated "adjusted" risk estimate for ETS exposure. Layde also criticized the study as inadequately assessing ETS exposure and noted that the observed association was implausibly large. He concluded that the resolution of any

[7] In the case of current smokers, the unadjusted relative risk reported was 10.1, which, when compared to the adjusted relative risk reported for smoking of 3.43, illustrates the profound effect of confounding variables on an association. (The unadjusted relative risk was not reported for nonsmokers.)

relationship between ETS exposure and cervical cancer may have to wait identification of the sexually transmitted agent that appears to be the most important etiologic agent in cervical cancer.

The Slattery study was also severely criticized by Zang, et al., 1989, of the American Health Foundation. They noted that Slattery undermatched controls with regard to important risk factors such as sexual activity, religious background, and education. Zang suggested that "the adjusted odds ratios [for passive smoking] are probably no more than the left-over effect of variables controlled imperfectly by logistic regression."[8] Zang concluded that classification of ETS exposure as a risk factor for cervical cancer is premature.

The association of cervical cancer with ETS exposure reported by Slattery, et al. has not been confirmed in other studies. In particular, previous studies by Hirayama (1984), Zunzunegui (1986), Hellberg (1983), Brown (1982), and Buckley (1981) found no statistically significant risk of cervical cancer for having a husband who smoked. Although Sandler (1985) reported a two-fold cervical cancer risk among women whose husbands smoked, this result is highly suspect. Sandler collected no data on sexual activity of cases or spouses and therefore could not adjust for this important confounder.

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[8] It is widely assumed that the most important cause of cervical cancer is an infectious agent which is transmitted sexually. (Layde, 1989a). Accurately identifying noninfectious statistical risk factors for cervical cancer is highly dependent on proper control for confounding factors. (Brinton, 1990). The role of smoking in cervical cancer is notoriously difficult to evaluate because smoking is strongly correlated with sexual activity. Id. As Winkelstein has noted, in studies of cervical cancer the number of sexual partners is frequently used as a proxy for the unidentified causative infectious agent. (Winkelstein, 1990). A number of studies, however, have found little correlation between this variable and detection of suspect infectious agents. (Reeves, 1989; Villa, 1989). Even the best efforts to control for reported sexual activity and other risk factors are limited in their ability to control for confounding.

The existing data therefore suggest no direct link between husband's smoking habits and wife's cervical cancer, let alone any link between exposure to ETS in any public environment and cervical cancer.

D. Mortality Projections

Petitioners suggest that the FAA should rely on an article published by Glantz and Parmley that projects 53,000 annual deaths attributable to ETS exposure (Glantz, 1991) and a report by the Centers for Disease Control that "at least 3825 nonsmoking Americans die each year from lung cancer attributable to passive smoking." These projections are based on more assumptions than any regulatory agency or scientist should be willing to accept. With respect to their claim about heart disease deaths, one EPA staff member has even been quoted as saying that: "Thirty-seven thousand may be a figment of Stan Glantz's imagination and William Parmley's imagination . . . ."[9]

Glantz and Parmley begin their analysis by concluding that ETS exposure causes lung cancer, cancer other than lung, and cardiovascular disease in nonsmokers. (Glantz, 1991). For each disease, they then pool epidemiologic data to calculate a summary relative risk across the studies. This technique is referred to frequently as "meta-analysis." Those risks are integrated with exposure assumptions to project the number of deaths allegedly caused by a particular exposure. Many of the steps are simply adopted from Wells, 1988.[10]

Glantz and Parmley's work violates nearly every risk assessment guideline utilized by government agencies. (*See, e.g.*, EPA 1986). They identify none of the assumptions and

[9] Associated Press, "Secondhand Smoke Kills, Report Says: 53,000 Die Every Year From Passive Smoking, Study Sponsored by EPA Says," Winston-Salem Journal, May 30, 1991.

[10] Wells' work has been criticized extensively. (Katzenstein, 1990; Lee, 1991a; Holcomb, 1990).

sources of uncertainty inherent in their analysis. They assert as fact conclusions regarding issues that are vigorously disputed in the scientific literature.

A number of scientists and organizations have critically reviewed the epidemiologic studies of ETS and lung cancer and have concluded that ETS has not been shown to be a cause of lung cancer in humans. (*See, e.g.*, Feuer, 1991; Gori, 1991; Uberla, 1989; Layard, 1989; Wynder, 1988; Uberla, 1987; IARC, 1985). Many investigators have also rejected the hypotheses that ETS causes other cancers (Pershagen, 1989; Erikson, 1988; USSG, 1986; NRC, 1986A) and cardiovascular disease (Wexler, 1989; Fielding, 1988; USSG, 1986; NRC, 1986A).

Fleiss and Gross recently reviewed the application of meta-analytic techniques to epidemiologic studies of ETS. (Fleiss, 1991). They concluded that the application of the technique to ETS studies was a "mere computational exercise" and unwarranted given the "poor quality of the underlying studies."

Glantz and Parmley's and other mortality projections rest on a house of cards. The abundance of issues described above led one well-known risk assessment expert to conclude "the current data appear to be insufficient for conducting a convincing quantifiable risk assessment." (Paustenbach, 1989). Alan Gross, a professor in the Department of Biometry at the Medical University of South Carolina, concluded that performing a risk assessment on ETS -- prior to accruing proof of causation -- is a futile exercise:

> The most striking aspect of the ETS risk assessments that have been carried out thus far is that the authors have seen fit to undertake them at all. . . . [T]hese assessments have feet of clay: Because they are based on epidemiologic studies that themselves are not reliable, they lack the necessary foundation of an inference that exposure to ETS causes disease of any sort in humans.

(Gross, 1989). Peter Lee reviewed recent ETS risk assessments including Wells' and concluded that the epidemiologic-based estimates cannot be relied upon. (Lee, 1991b). In sum, Glantz and Parmley's work provides no evidentiary base for FAA to act upon.

Finally, Petitioners seek to bolster their weak Petition by pointing to the EPA's Science Advisory Board's ("SAB") acceptance of the conclusions of EPA's draft risk assessment on ETS. Unfortunately, Petitioners fail to mention that the SAB severely criticized the approach taken by EPA. Indeed, the SAB acknowledged that the EPA did not comply with its own standards for classifying an agent as a carcinogen. Nevertheless, the chairman of that panel justified the conclusion and noted that the guidelines should be changed. Unabashed political judgments of this nature have no place in a reasoned debate about proper public policies based on ETS and health. The EPA's conclusions have been criticized by numerous commentators.[11]

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[11] In contrast to EPA's approach, OSHA has resisted pressure to depart from reasoned decisionmaking. The Occupational Safety and Health Administration ("OSHA") has refused to adopt an emergency temporary standard concerning workplace smoking despite heavy pressure from antismoking organizations. Action on Smoking and Health ("ASH") first petitioned OSHA to issue such a standard in 1987. ASH filed suit in the U.S. District Court for the District of Columbia in 1989, alleging that OSHA breached its statutory duties by not issuing a satisfactory response. OSHA responded to ASH that an adoption of the emergency temporary standard was not justified.

ASH sought judicial review of OSHA's response in October 1989, calling OSHA's determination "arbitrary, capricious and an abuse of discretion." The suit was stayed in 1990, and OSHA promised to study the workplace ETS issue further. Last fall, OSHA determined that it would not promulgate the requested regulations. In early 1991, U.S. Court of Appeals for the District of Columbia declined to review OSHA's refusal to promulgate an emergency standard. (Action on Smoking and Health v. U.S. Department of Labor et al., DC Cir., No. 89-1656; see Indoor Pollution LR, March 1991, P. 748).

OSHA has announced its intention to issue a Request for Information that will address overall indoor air quality issue (including ETS). The RFI should be issued sometime this fall. It is believed that OSHA will seek answers to a series of specific questions related to indoor air quality in the workplace.

E. Workplace Data

The Petitioners request, in essence, that FAA regulate ETS as a workplace hazard. True to form, the Petition does not include any data regarding workplace exposure to ETS and health. Table II summarizes all reported data on ETS exposure in the workplace and lung cancer risk that RJR is aware of. Out of ten groups studied, only one reported an increase in risk. These data clearly provide no basis for regulatory action.

TABLE II

Reported Relative Risks for Workplace Exposure

Study	Sex	Index of Exposure	Relative Risk (95% Confidence Limits)
Case-control studies			
1. Kabat, 1984	F	Current exposure on regular basis to tobacco smoke at work	0.68 (0.32-1.47)
	M	Current exposure on regular basis to tobacco smoke at work	3.27 (1.01-10.6)*
2. Garfinkel, 1985	F	Smoke exposure at work in last 5 years	0.88 (0.66-1.18)
		Smoke exposure at work in last 25 years	0.93 (0.73-1.18)
3. Wu, 1985	F	Passive smoke exposure at work	1.3 (0.5-3.3)
4. Lee, 1986	F	Passive smoke exposure at work	0.63 (0.17-2.33)
	M	Passive smoke exposure at work	1.61 (0.39-6.60)
5. Shimizu, 1988	F	Someone at working place smokes	1.2 (0.69-2.01)
6. Janerich, 1990	M+F	150 person/years smoking in the working place	0.91 (0.80-1.04)
7. Wu-Williams, 1990	F	Passive smoke exposure at work	1.1 (0.9-1.6)
8. Kalandidi, 1990	F	Passive exposure at work	Small and nonsignificant

*Statistically significant at the 5% level.

F. The Geomet Risk Assessment

Petitioners rely extensively on a study commissioned by the DOT and conducted by Geomet, titled "Airliner Cabin Environment: Contaminant Measurements, Health Risks, and Mitigation Options" (the "Geomet Report"), to assert that ETS exposure poses a unique risk to flight attendants. (Nagda, 1989). They take out of context quotes from the Geomet Report to buttress

their contentions. Again, however, these statements, and the "risk assessment" contained in the Geomet report, presume causation. Accordingly, any conclusions should not be accepted uncritically.

Moreover, Petitioners deliberately omit an important qualification, included by Geomet itself, to this risk assessment. The omitted language reads:

> "Applying these risk estimates to the entire U.S. cabin crew population results in an estimated 0.18 premature lung cancer deaths per year for domestic flights (that is, approximately 4 premature deaths can be expected every 20 years) and 0.16 premature deaths per year for international flights."

(Nagda, 1989). As a result of this omission, the Petition distorts the conclusion that it hopes to advance.

In a 1991 review, Crawford and Holcomb specifically addressed the fundamental flaws of the Geomet risk assessment:

> [Geomet's] risk estimation, based on measured respirable suspended particulate (RSP) levels in-flight and assumed exposure durations, assumed a causal relationship between ETS exposure and lung cancer and a relative risk of 1.3 for this relationship. As has been shown earlier, both the assumption of a causal relationship and a magnitude of 1.3 for that relationship are based upon inappropriate evaluation of the studies involved and are not widely accepted within the scientific community.

(Crawford, 1991).

Crawford and Holcomb also identified -- and corrected -- errors affecting the calculations of Geomet's risk assessment. These errors included (a) use of exposed dose of RSP, rather than retained dose of RSP and (b) an incorrect respiratory rate. Crawford and Holcomb gave corrected values for the lifetime risk of lung cancer for flight attendants exposed to ETS that ranged from 0.40 to 0.57/100,000, a range substantially less than the range of 12 to 17/100,000 that Geomet

estimated incorrectly. The authors concluded that the effects of "[t]his [risk] would not be discernible within a population."[12]

The Petition does not accurately depict what is known about EST exposure and health. When viewed objectively, the overall scientific literature presents a far different picture. Epidemiologic studies of ETS and health have produced weak, inconsistent and largely statistically insignificant results. Significant sources of bias and confounding plague the data. Measured by conventional criteria, the studies do not demonstrate that ETS exposure causes chronic disease.

IV. SMOKING ON AIRCRAFT DOES NOT REPRESENT A SIGNIFICANT FIRE HAZARD

Banning smoking on international flights will do little to improve overall fire safety aboard aircraft. In 1986, the National Research Council noted that the FAA program on flammability testing was excellent, but pointed out the need for further activity designed (NRC, 1986a) to reduce the hazards of aircraft fires and the smoke produced by those fires. At that time, the FAA had already begun to increase the attention devoted to fire safety issues.

Petitioners' reference to the tragedy aboard Air Canada Flight 797 on June 2, 1983 is misleading. As noted by Richard G. Hill in a paper presented at the Fifteenth International Conference on Fire Safety, there is no conclusive evidence that the Air Canada fire was caused by a cigarette:

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[12] Subsequent to publication of its document on the study of aircraft, Geomet obtained new information relating to the effects of cosmic radiation. (Baker, 1990). In their paper, Crawford and Holcomb presented the revised risk connected with cosmic radiation: 90 to 1026/100,000. Depending on the accounting, the risk connected with exposure to cosmic radiation is from 150 to 2,500 times greater than the risk claimed for ETS exposure.

> Investigation into this accident indicated that a fire started in the hidden area of the aft lavatory. The actual ignition source or fuel was not determined. It could have been electrical in nature or it could have been caused by a cigarette and trash behind the vanity area.

(Hill, 1990).

In any event, fire safety regulations have been strengthened significantly since that accident:

> The Air Canada accident led to a number of regulatory changes in the United States. Requirements for smoke detectors in lavatories, fixed fire extinguishers in trash containers, and at least two Halon fire extinguishers onboard transport aircraft were incorporated. Also, floor proximity lighting and seat fire blocking rules were hastened in their adoption because of this accident.

(Hill, 1990). At the same conference, Constantine P. Sarkos of the FAA Technical Center reviewed the FAA's past and current activity in the area. Sarkos referred to the "unprecedented series of new standards designed to improve transport aircraft fire safety" adopted by FAA in the preceding five years and discussed the seven specific new standards which had been adopted to that date. He made the following observation in connection with his discussion of FAA's project on Hidden Fire Protection:

> The use of highly resistant materials and the responsiveness of cabin crewmembers virtually guarantees that a small in-flight fire originating in an open or accessible location will be quickly extinguished before developing into a problem.

(Sarkos, 1990).

A review of the information reproduced from FAA files on in-flight and ground fires in the NRC report reveals that in those few instances where cigarettes and lighters were implicated in the origin of fires in aircraft, the fires were quickly extinguished. Recent safety

advances and regulations further diminish the possibility of tobacco-related fires as a serious threat.[13] In-flight fires aboard aircraft deserve (and have received) serious attention by regulators, aircraft manufacturers and suppliers, and carriers. Tobacco-related fires, however, are not a serious threat to the safety of passengers and crew.

V. THE PETITION PRESENTS AN INCOMPLETE AND DECEPTIVE DISCUSSION OF THE MITIGATION OPTIONS AND COST/BENEFIT RELATIONSHIPS

In an attempt to bolster its position that smoking should be banned on all flights by U.S. carriers, the Petitioners raise issues of mitigation and cost/benefit analysis, relying exclusively on the Geomet Report. (Nagda, 1989). The Petition's treatment of these subjects is distinguished more by what it omits than by what it includes.

A. The Geomet Report Was Not Designed To Apply To International Flights

Foremost among the Petition's omissions is that it fails to indicate that Geomet's analysis of mitigation options and cost/benefit relationships is applicable to neither international carriers nor their flights. Rather, Geomet's analysis was intended to address such issues within the context of domestic flights involving short range narrow-bodied aircraft.

In their attempt to summarize the Geomet Report, the Petitioners misrepresent the results of the cost/benefit analyses. The dollar amounts that the Petition quotes for the benefits of a total ban are those that Geomet estimated might apply to domestic flights

[13] The aircraft industry has also responded with many recent advances in aircraft fire safety, with numerous presentations in the last few years at aviation and fire safety conferences on new technologies and materials. For example, an upcoming conference (Fire Safety '91, to be held November 4-7, 1991 in St. Petersburg Beach, Florida) has an entire session on Aircraft and Aerospace, with at least ten presentations scheduled on aircraft materials.

operating before passage of the law banning smoking on flights of duration less than two hours.

B. The Geomet Report Failed To Consider All The Possible Costs Of A Smoking Ban

The Petition also states: "The [Geomet] study found no costs associated with a total ban." FAA should not be misled. The cost/benefit analysis by design considered only technical costs. The authors of Geomet's document are clear on this subject: "only technical costs are considered." (See Section 9.2.3, p. 9-26).

C. The Petition Does Not Summarize Fairly the Conclusions Contained in the Geomet Report

In addressing the option "Periodic Curtailment of Smoking During Flights", the Petition provides no cost accounting for the option although it does so for the other three. (Geomet estimated that the benefit this option afforded flight attendants was only $0.9 million.) Instead, the Petition obfuscates the issue by introducing the speculative subject of compensatory smoking.

D. Modeling Techniques Employed by Geomet Have Not Been Validated

The mathematical model Geomet used to estimate the effects of mitigation options on levels of respirable suspended particles (RSP) is not validated. This is true also for the model which Ryan, et al. developed that served as the basis for the model that Geomet used. (Ryan, 1988).

Table 9-3, p. 9-14 of the Geomet report, which compares concentrations of RSP predicted by the model to concentrations of RSP that were measured, indicates errors in the

model's predictions that range from -85% to +205%. The poor quality of the model, as evidenced by its inability to predict RSP concentrations accurately, explains the absence of any validation and leads to the inescapable conclusion that the model is invalid. The invalidity of the modeled RSP concentrations extends also to the results of the analyses of cost/benefit for mitigation options because these incorporate the modeled RSP concentrations.

In addition, Geomet's modeling derives from an unacceptably small number of narrow-bodied aircraft (namely, B727 and MD-80), which are not representative of types flying international routes. Because of major differences in the design, operation, and passenger loads between narrow-bodied and wide-bodied types of aircraft, the assumption that the model's predictions extend from one type to the other is not supportable.

E. The Petition Presents an Inadequate Economic Basis Upon Which the FAA Should Act

The Petitioners conclude, but do not demonstrate, that the ban they are seeking would not have serious economic consequences to U.S. air carriers. In particular, they fail to give due attention to the most important economic impact of all, namely, the loss to foreign carriers of passengers who smoke and the revenue they represent -- to the detriment of our nation's air transport industry.

Citing the Geomet Report, Petitioners attach a figure of $0 to the impact of switchover. As is discussed above, Geomet's analysis was neither designed nor intended to apply to economic issues relating to international flights, and many additional factors must be weighed to analyze the impact of a smoking ban on international flights. FAA cannot ignore the fact that a smoking ban on international flights would have some impact on U.S. carriers. Before acting, FAA should, at a minimum, develop a record including appropriate information about the possible economic impact of such a ban.

Petitioners use the experience of Northwest Airlines as an example to support their attempt at brushing aside this important economic issue. The experience of a single carrier, voluntarily making a decision to ban smoking, cannot be extended to an entire industry, forced into the decision when competing carriers from other nations will not be so limited.

Petitioners totally fail to provide any information which might shed light on the subject of economic impacts. Petitioners claim that savings will be realized from reduced use of health benefits and reduced amounts of litigation without demonstrating that these "savings" will occur and, if so, the economic impact of these "savings." In the absence of supporting documentation, these "savings" are speculative and administrative action cannot be based upon them.

CONCLUSION

When the relevant evidence is considered in detail, there simply is no adequate basis for regulation of smoking on U.S. airlines. As put succinctly by Holcomb,

> The available scientific evidence does not support the prohibition of smoking on commercial aircraft. The data that are available reveal low concentrations of substances that can be traced to ETS in smoking sections, and even lower concentrations in non-smoking sections, thus confirming the efficacy of current in-flight smoking policies. The available data also suggest that factors or substances other than ETS may be major contributors to subjective complaints of discomfort by passengers and flight crew.
>
> Finally, given the limited and intermittent occasions for exposure, even in the case of compromised individuals and flight attendants, adverse health effects from exposure to ETS aboard aircraft are highly unlikely.

(Holcomb, 1988).

For the reasons stated above, the FAA should deny the Petition.

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