BEFORE THE UNITED STATES
ENVIRONMENTAL PROTECTION AGENCY

ENVIRONMENTAL TOBACCO SMOKE:

A GUIDE TO WORKPLACE SMOKING POLICIES

EPA/400/6-90/004

RESPONSE OF R.J. REYNOLDS TOBACCO COMPANY

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VOLUME I

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RESPONSE TO THE EPA PUBLIC REVIEW DRAFT DOCUMENT: "ENVIRONMENTAL TOBACCO SMOKE: A GUIDE TO WORKPLACE SMOKING POLICIES"

SUBMITTED BY:

R. J. REYNOLDS TOBACCO COMPANY

I. INTRODUCTION

On June 25, 1990, the Environmental Protection Agency (the "EPA" or the "Agency") released a draft document titled Environmental Tobacco Smoke: a Guide to Workplace Smoking Policies, EPA/400/6-90/004 (Public Review Draft) (the "Policy Guide" or "Guide"). By Federal Register Notice, dated June 25, 1990, the EPA solicited comments regarding the "manner in which EPA characterizes in this document the scientific information available on environmental tobacco smoke" [Federal Register, 1990]. The Notice also states that the Agency wants to ensure that the Policy Guide does not oversimplify the state of scientific knowledge on environmental tobacco smoke (ETS). R. J. Reynolds Tobacco Company ("RJRT" or the "Company") submits this response to address the scientific accuracy, the presentation of scientific information, and the policy implications of the Guide.[1]

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[1] Appended to this Response are individual comments, prepared by scientists of RJRT's Research and Development Department. Specific technical inaccuracies, mischaracterizations, omissions and oversimplifications are addressed by these scientists according to their area of expertise.

Addressing these concerns is hindered by the Guide's gross oversimplification of ETS science. This gross oversimplification results in both lost accuracy and incompleteness. The Guide presents as fact (i) inaccurate scientific arguments, (ii) assertions which are technically correct, but altogether irrelevant to a discussion of ETS and (iii) statements that portray a one-sided, selective view of current scientific understanding. The Guide's inadequate presentation of the science may be partially attributed to the scarcity of credible ETS research related to health. In a recent review of ETS and health, Mahajan commented that "no other aspect of the tobacco and health issue has had more shoddy research and less reproducible results published" [Mahajan, 1990, p. 87]. The Guide has unfortunately relied upon much of this shoddy research, but has ignored many well-conducted studies that are relevant to an evaluation of ETS in the context of indoor air quality. This selective review of the science apparently is designed to justify predetermined policy conclusions.

The EPA states that "[t]he Guide to Workplace Smoking Policies is intended to provide government and private sector decision makers with information on the technical basis for controlling involuntary nonsmoker exposure to environmental tobacco smoke and to describe the range of technical and policy options for instituting effective smoking policies" [Federal Register, 1990]. To accomplish this goal, the Guide must present an accurate, objective and complete review of ETS science. The Guide must also present a comprehensive and objective discussion of all policy options available to decision-makers -- including the option of no formal smoking policy. The Guide falls far short of the EPA's intended goals, and thus the EPA does not meet its obligations to decision-makers.

In a peculiar abrogation of Agency responsibility, the EPA retained outside contractors, Robert Rosner and Robin Simons of the Smoking Policy Institute (the "Institute"), to develop the Guide. Rosner and Simons have indisputable vested interests that raise questions about their objectivity. They and their Institute stand to benefit significantly if employers follow the limited options promoted by the Guide -- options that would enjoy the EPA's imprimatur if the Guide were finalized in its present form. Agency employees have strict conflict of interest guidelines. The involvement of these outside authors is wholly inappropriate and raises the question of impropriety. The EPA was established to provide a neutral, objective forum for the evaluation of science and for the development of national policy. This Guide does not meet even minimal standards of objectivity and accuracy.

The Guide incorrectly presumes that ETS is a proven cause of lung cancer. The authors present as established fact conclusions drawn from the Agency's draft document: Health Effects of Passive Smoking: Assessment of Lung Cancer in Adults and Respiratory Disorders in Children, EPA 1600/6-90/006A (the "Health Assessment") -- now under initial review. The authors leap from a superficial review of the Health Assessment and other federal government documents [NRC, 1986; USPHS, 1986] to conclude that ETS causes lung cancer. Despite the Guide's many summary statements, ETS has not been proven to cause lung cancer in nonsmokers.[2] Many scientists have reached conclusions contrary to the Agency [Adlkofer, 1989; Layard, 1990; Letzel, 1988; Thornton, 1989; Uberla, 1988; Uberla,

[2] RJRT's position is described fully in comments on the Health Assessment submitted to the Agency separately and attached to these comments.

1987; Wynder, 1990]. Neither the Health Assessment nor any other review has presented a persuasive case that ETS is an unconfounded risk factor for lung cancer in nonsmokers -- let alone that there is a causal relationship. It is an abuse of discretion for the Guide to be based on preliminary and questionable findings contained in the Health Assessment.

The Guide selectively reviews and incorrectly characterizes ETS scientific literature. Many important studies are ignored; others are distorted or overinterpreted. For example, studies presenting real-world concentrations of ETS constituents are omitted [Carson, 1988; Crouse, 1990; Crouse, 1989; Oldaker, 1990a; Oldaker, 1990b; Oldaker, 1986; Sterling, T., 1988] and limitations regarding ETS exposure measurements are ignored. Most troubling, the Guide blurs the distinction among ETS, mainstream smoke (MS) and sidestream smoke (SS). The result is a distorted perspective of ETS science. In short, the Guide presents a quasi-scientific apologia to justify predetermined, but unfounded, policy positions.

The policy recommendations rest upon this flimsy scientific foundation. The Guide emphasizes two extreme policy options -- a total ban on smoking in the workplace and the construction of specially designed, separately ventilated rooms. Logically, these two policies follow directly from the EPA's assumptions that ETS is a proven cause of lung cancer in nonsmokers and that no safe exposure level exists.[3] The Guide, however, fails to inform the reader of the uncertainties pervading these assumptions. The failure to discuss other options can withstand scrutiny only if these assumptions are correct. Different assumptions would suggest different policy preferences. The Guide's recommendations fail to satisfy the EPA's

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[3] The Company does not offer a detailed critique of the presumption of no threshold limits for carcinogens because the Agency has failed to demonstrate that ETS is a carcinogen.

stated goal: "to describe the range of technical and policy options for instituting effective smoking policies" [Federal Register, 1990]. The promotion of extreme policy choices based on uncertain and questionable assumptions raises serious questions about the EPA's objectivity.

Finally, the EPA has exceeded its authority by developing the Guide. The Guide constitutes *de facto* rulemaking. The Occupational Safety and Health Administration (OSHA) is the only federal agency with authority to regulate exposures to substances found in the workplace. The EPA's justification for its Policy Guide is based upon a companion Health Assessment which itself is arbitrary and capricious and an abuse of the Agency's discretion.

RJRT's response is presented in seven sections.[4] Section A describes the bias of the Guide's principal authors. Section B examines the Guide's treatment of the science of ETS. Section C describes the role of ETS in indoor air quality. Section D considers the extreme policy options presented in the Guide and describes several others that should be included. Section E discusses the EPA's resort to extraneous information to buttress its rationale that extreme workplace smoking policies are necessary. Section F discusses EPA's lack of substantial evidence upon which to base the Guide's conclusions and recommendations. Finally, Section G explains that the Guide is an inappropriate exercise of authority by the EPA.

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[4] This response is not a line-by-line correction of the Guide. That type of response could only be made effectively if the Guide contained only minimal errors amenable to correction.

II. RESPONSE

A. The Authors Of The Policy Guide Are Biased And Have Conflicts Of Interest

The primary authors of the Policy Guide are Robert Rosner and Robin Simons of the Smoking Policy Institute (the "Institute") in Seattle, Washington. A review of the Institute's mission and Rosner's activities reveals bias and conflict of interest.

Although billed as a non-profit organization, the Institute, which began in the University of Washington's School of Business, routinely consults with companies to "eliminate smoking in the workplace" [Estill, 1988]. In addition, the Institute develops and markets materials produced by the Bureau of Business Practice, a division of Simon and Schuster.

Rosner, whose antipathy for cigarette smoking is well-known, is the director of the Institute. He has publicly voiced strong antismoking sentiments that preclude any objectivity in drafting a smoking policy guide [*e.g.*, Estill, 1988]. As early as 1982, smoking cessation featured prominently in his social agenda [Reynolds, 1990]. In 1984, Rosner established the consulting firm of Rosner, Weiss, and Lowenberg to assist private employers with the development and implementation of workplace smoking policies [Oates, 1985]. Rosner continued his antismoking efforts as Executive Director of the Institute for Occupational Smoking Policy [Greene, 1985]. Today, Rosner remains very active in promoting smoke-free workplaces [Reynolds, 1990].

Despite the name of his organization, or its tax-exempt status, the business of Rosner's Institute is to create an environment in which employers and employees feel a need for workplace smoking restrictions and then to cater to that perceived need. Rosner

personally consults with such commercial groups as "Smoke-Stoppers" and assists them in promoting smoking-cessation seminars to corporations. As Rosner aptly noted, the "pitch" for corporate clients makes economic sense for him and for the companies he represents: "Getting together a room full of strangers requires extensive ads, but if you get one hit at a company, you can make $2,000 with 12 people in the room" [Hammer, 1988, p. 42].

The Guide is tantamount to a vehicle for promoting additional interest among private employers to institute smoking policies. In fact, it reads more like a marketing brochure than a policy guide. Rosner has publicly stated his belief and hope that "[i]f widely adopted, these [workplace smoking] policies might have a dramatic effect on the nation's smoking habits. Theoretically, they would encourage people to quit smoking by increasing the social pressure against it and by restricting the time available for it" [Martin, 1986, p. 647]. The potential benefits of the Guide to Rosner and his Institute are obvious. The EPA would not permit agency personnel with such clear conflicts of interest to draft policy. The same standard should be expected of EPA contractors.

B. The Policy Guide Presents A Selective, Distorted And Misleading Review Of ETS Science

The scientific evaluation that forms the basis for the recommendations in any government policy guide should be accurate, complete, impartial and appropriate for a non-technical audience. The Guide should not represent to the public as established fact assertions that are the subject of substantial disagreement in the scientific community. It also should clearly identify points of scientific debate and questionable assumptions. Furthermore, the Guide should present information about ETS within the greater context of indoor air quality, rather than singling out ETS.

The polemical path taken by the Guide relies upon an inadequate review of the available data. Indeed, many statements and implications in the Policy Guide conflict with data reported by the EPA itself, as demonstrated below.

1. ETS Is Not Equivalent To Sidestream Cigarette Smoke

Throughout Chapter 1, titled "What is ETS?," the EPA treats ETS as if it were a well-described, stable mixture of SS[5] and exhaled MS[6]. The Guide purports to discuss the chemistry of ETS, but includes data only for SS and MS. The authors make no effort to discuss published data regarding ETS chemistry; pertinent studies of ETS in the workplace are ignored [Carson, 1988; Crouse, 1990; Crouse, 1989; Oldaker, 1990a; Oldaker, 1990b; Sterling, T., 1988]. Instead, the authors choose to give the false impression that the properties of ETS are equivalent to sidestream smoke. ETS is neither stable nor equivalent to sidestream smoke.

ETS differs significantly both from SS alone and from a simple combination of SS and exhaled MS. ETS is "an aged, dilute mixture of sidestream and exhaled mainstream smoke from combustion of tobacco products such as cigarettes, cigars, etc." [Ogden, 1988, p. 77]. The Guide ignores this distinction. One scientist who has studied cigarette combustion processes and cigarette smoke generation for more than two decades stated, "Several scientists have incorrectly assumed that concentrated, fresh SS is the same as ETS and have

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[5] Sidestream smoke is that smoke emitted by a cigarette between puffs plus a small amount of smoke escaping during the puff.

[6] Exhaled MS is not the same as MS generated by smoking machines for analysis. In addition to the aging effects, some of the smoke inhaled is retained by the smoker.

consequently made invalid conclusions concerning the properties of ETS" [Baker, 1989, p. 2]. EPA scientists have themselves concluded that "ETS differs both chemically and physically from the precursor sidestream smoke, presumably due to chemical and physical transformations that occur as the mixture is diluted and aged" [Lofroth, 1989, p. 610]. The Guide does not acknowledge the existence of literature that describes the unique physical and chemical properties of ETS [Baker, 1990; Baker, 1989; Eudy, 1986; Harke, 1974; Hulka, 1988; Ingebrethsen, 1989; Proctor, 1988; Reasor, 1987; USPHS, 1986].

The Guide's discussion of ETS composition [The Guide, pp. 7-10] disregards dissipative forces, *e.g.*, air currents, ventilation, diffusion, surface sorption, that greatly dilute sidestream and exhaled mainstream smoke. Some studies [Mahajan, 1990; Oldaker, 1989] indicate that ETS is diluted 100- to 1000-fold, compared to SS. Actual ETS constituent concentrations often fall below detection limits of current scientific detection techniques [Carson, 1988; Crouse, 1989; Oldaker, 1990a; Oldaker, 1990b; Oldaker, 1986]. Consequently, characterization of ETS by relying solely on SS and MS data is fallacious.

ETS is a dynamic mixture of aged and diluted SS and exhaled MS. "[O]nce the smoke has left the cigarette, cigar or pipe, its physical nature and relative chemical composition will change dramatically" [Proctor, 1988, p. 58]. During aging, chemical reactions and physical processes change the chemical composition as well as the distribution of compounds between gaseous and particulate phases.

> A variety of studies have shown that nicotine in ETS is almost entirely in the vapour phase. This contrasts sharply to fresh mainstream smoke where nicotine is almost entirely in the particulate phase, and to exhaled mainstream smoke where there is actually little nicotine left. In fresh sidestream smoke, about 80% of the nicotine is in the vapour phase. Thus, as

> sidestream smoke is diluted and ages to form ETS, nicotine evaporates out of the particles. Other quite different studies have shown that matter evaporates from sidestream particles as they age to ETS. Ingebrethsen has estimated that 20-30% of the original matter in sidestream smoke particles is lost in this way. This is accompanied by a shrinkage of the particles.
>
> In addition to physical changes, chemical changes also occur as ETS ages. Thus, for example, nitric oxide slowly oxidizes over minutes and hours to nitrogen dioxide in ETS. However, these chemical changes, and indeed the particle size changes described above, will be far outweighed by the physical effects of air movement which occur in real indoor environments [Baker, 1989, p. 5].

Chapter 1 of the Guide contains a table, "Toxic and Cancer-Causing Agents in Mainstream and Sidestream Cigarette Smoke" [The Guide, p. 10], that is not worthy of publication. It reports only sidestream and mainstream data and ignores available and more pertinent data on ETS constituents in real-world environments. The table is inaccurately labelled and not all compounds listed have been reported to be "Toxic and Cancer-Causing Agents." Units in the table are incorrect in some cases and missing in one instance. Finally, cigarette types are unidentified and analytical procedures are unspecified. As a result, it is impossible to judge the accuracy of the concentrations reported.

Like the table, the authors ignore fundamental principles pertaining to the physical and chemical properties of ETS. The Guide does not include any original data on the composition of ETS, although there are peer-reviewed reports in the literature. Contrary to the authors' misstatements and mischaracterizations:

1. ETS is a complex, constantly evolving mixture of SS and exhaled MS. Implied equivalence with SS is false and deceptive.

2. ETS is hundreds to thousands of times more dilute than either SS or MS.

Further, the Guide's insinuation that nonsmokers are exposed via ETS to higher concentrations of smoke constituents than smokers via MS is patently ridiculous. There is no authority for such a proposition.

2. Constituents Of ETS Provide No Basis To Reach Conclusions Regarding ETS And Health

In discussing the alleged "Hazardous Constituents in ETS," the Guide states that "[m]any of the chemicals in ETS are known carcinogens, mutagens, toxins or irritants" [The Guide, p. 8]. The Guide then suggests that ETS is more hazardous than MS by stating that "sidestream smoke is known to have significantly higher concentrations of carcinogens and mutagens than mainstream smoke" [The Guide, p. 9]. Finally, the Guide relies on individual constituent studies to support its assertions about the alleged biological properties of ETS.

These points are based on the false premise that the physicochemical properties of ETS are equivalent to those of SS, MS or a combination of the two. The Guide makes no effort to discuss the chemical composition of ETS, except in the inappropriate context of SS and MS. As described above, ETS is not identical to SS or to a simple combination of SS and MS. Because of dilution and dissipative forces, ETS is much more dilute [Mahajan, 1990; Oldaker, 1989]. **The concentration of ETS and its constituents in the workplace environment is the true issue.** The Guide's failure to take into account and describe facts about ETS constituent concentrations deceives the reader.

The Guide fails to place the magnitude of real-world ETS exposures in a meaningful context. Paracelsus pointed out a fundamental axiom of toxicology 500 years ago: "All substances are poison; there is none which is not a poison. The right dose differentiates a poison and a remedy" [Klaassen, 1986, p. 1]. Recently Ames paraphrased the principle in

a more specific context: "Thus, a high percentage of all chemicals might be expected to be carcinogenic at chronic, near-toxic doses and this is exactly what is found" [Ames, 1990, p. 970]. Therefore, any discussion of "hazardous constituents" that does not consider dose (hence, concentration) is meaningless from a toxicological perspective.

The EPA's presumption that, in the absence of evidence for a threshold, any exposure to a cancer-causing agent increases the risk of cancer, does not change this toxicologic principle. Virtually every food (*e.g.*, cooked meats, black pepper, coffee, mustard, celery, parsley, basil, oranges) and material (*e.g.*, air, water, sunlight, our own bodies) known to man contains substances that are carcinogenic, mutagenic, or toxic. Ames has noted that the foods in every meal contain many mutagens and carcinogens:

> The cooking of food produces thousands of pyrolysis products, and we estimate that dietary intake of these products is roughly 2000 mg per person per day. Few of these have been tested; for example, of 826 volatile chemicals that have been identified in roasted coffee, only 21 have been tested chronically, and 16 are rodent carcinogens; caffeic acid, a non-volatile carcinogen, is also present. A cup of coffee contains at least 10 mg (40 ppm) of rodent carcinogens (mostly caffeic acid, catechol, furfural, hydrogen peroxide, and hydroquinone). Thus, very low exposures to pesticide residues or other synthetic chemicals should be compared to the enormous background of natural substances [Ames, 1990, p. 971].

In fact, no substance has unequivocally been shown to be free of carcinogenic or mutagenic properties.[7] The Guide makes no effort to report the concentrations at which any of the "hazardous constituents" are present, if detectable at all, in ETS.

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[7] For example, the only substance that has been certified by the International Agency for Research on Cancer ("IARC") as being non-carcinogenic to man, caprolactam, is mutagenic in several widely used test systems [Ashby, 1985].

The authors next rely on the unproven assumption that MS or its individual constituents cause lung cancer in humans. Chemists, toxicologists, and epidemiologists agree that it is not possible to identify any particular MS constituent(s) as the alleged cause of disease.

> [U]nderstanding [of the] components which may act on the cardiovascular and respiratory system is embarrassingly lacking, if not outright contradictory. . . . Uncertainty about the specific attribution of risk to individual smoke components may be greater than ever now, . . . [Gori, 1980, p. 355].

Even vocal anti-tobacco researchers concede that no particular constituent of mainstream smoke has been shown to cause any specific disease in smokers [Hoffmann, 1982]. Tests conducted on individual constituents of smoke, at concentrations far greater than their concentrations in smoke, do not alter these conclusions. Pinpointing specific "causative agents" in a mixture as complex as cigarette smoke is of questionable use in predicting the mixture's biological properties:

> Cigarette smoke contains over 3,800 chemicals . . . . The biologic effects of this galaxy of compounds are virtually impossible to predict from a knowledge of the individual constituents, not only because of their huge number, but also because transient chemicals generated through the processes of pyrolysis, oxidation, and free-radical formation might dissipate or change with time and temperature [NRC, 1988, p. 28].

One simply cannot predict the biological effects of a mixture based on knowledge of individual constituents within the mixture, or based on knowledge of another mixture, even when the mixtures are similar and well characterized.

MS and ETS, however, are not similar. The Surgeon General's 1986 Report concluded that knowledge of cigarette smoke composition is of limited utility in predicting ETS-exposure effects.

> Comparison of the relative concentrations of various components of SS and MS smoke provides limited insights concerning the toxicological potential of ETS in comparison with active smoking. As described above, SS characteristics, as measured in a chamber, do not represent those of ETS, as inhaled by the non-smoker under nonexperimental conditions. Further, the dose-response relationships between specific tobacco smoke components and specific diseases are not sufficiently established for the necessary extrapolations from active smoking to environmental tobacco smoke exposure for individual agents [USPHS, 1986, p. 24].

Nonetheless, the Policy Guide attempts to bolster its arguments by focusing on a number of biologically active compounds that have been reported in MS. The Guide describes health hazards associated with these individual compounds, then completely ignores at least three critical considerations: (i) some of these compounds have never been detected in realistic concentrations of ETS; (ii) the species' minute concentration (dose) in ETS (as well as in MS); and, (iii) interactions with other species in the smoke mixture (*e.g.*, chemical reaction or solution effects). As the NRC concluded:

> Because the physicochemical nature of ETS, MS, and SS differ, the extrapolation of health effects from studies of MS or of active smokers to nonsmokers exposed to ETS may not be appropriate . . . . Laboratory studies in conjunction with epidemiologic investigations are needed to clarify possible health effects of exposure to ETS in nonsmokers [NRC, 1986, p. 8].

The fact is that ETS has never been shown to be carcinogenic in any animal species. Furthermore, ETS has not been shown to be mutagenic in any animal or cell culture system

when tested at or near ambient concentrations. Also, none of the individual chemicals in ETS has ever been shown to be carcinogenic or mutagenic when tested at its concentrations in ETS.

Following are examples of the document's misleading characterization of supposed hazards associated with ETS constituents.

a. Carbon Monoxide

The Guide states that carbon monoxide (CO) "is a gas that interferes with the ability of the blood to carry oxygen," and that increased levels of CO in the presence of smokers add to "the body burden of carbon monoxide from other environmental sources" [The Guide, p. 9]. These statements misrepresent the impact of ETS CO on humans or the environment.

The American Conference of Governmental Industrial Hygienists (ACGIH) has established a threshold limit value (TLV) of 50 ppm for CO [ACGIH, 1987]. OSHA has established a TLV of 35 ppm for CO, measured as a time-weighted average over an 8-hour period [Federal Register, 1989]. The EPA itself has established an outdoor CO standard of 9 ppm. In comparison, ETS CO concentrations are trivial.

The maximum CO concentration attributed to ETS in a major survey of 31 offices, where smoking was occurring, was 6.6 ppm [Carson, 1988]. Arithmetic mean concentration for ETS CO in this study was 0.2 ppm. The maximum indoor CO concentration, including all sources, was 8.7 ppm, which was still below the EPA's standard. Outdoor CO concentrations were reported for 23 of the 31 offices. Of these, 13 indoor locations

possessed lower CO levels than outdoors. Straightforward statistical analysis of these data confirms that CO concentrations in offices where smoking occurs are not significantly different from outdoor levels.

Fischer reported results from measurements of CO in three restaurants [Fischer, 1978]. He also measured CO in the air outside the restaurants to account for the outdoor contribution to indoor CO concentrations. He estimated CO associated with ETS by subtracting results of outdoor measurements from results of indoor measurements; the author reported these concentration results in terms of dCO ("delta carbon monoxide").[8] Average dCO's for two of the restaurants were 0.1 and 1.1 ppm; average dCO's in the smoking and nonsmoking sections of the third restaurant were 0.6 and 0.0 ppm, respectively. The average outdoor CO concentrations were 4.8, 1.5, and 0.4 parts per million.

Biological effects associated with exposure to high concentrations of CO are not germane to the low CO concentrations found in ETS. Blood carboxyhemoglobin (COHb) concentrations resulting from normal human metabolism are comparable to those found in the blood of nonsmokers exposed to ETS [Jarvis, 1984]. Furthermore, COHb concentrations associated with exposure to as much as 9 ppm CO do not alter cardiovascular function, as measured by maximal aerobic capacity testing [Horvath, 1989]. In an EPA-funded study in which subjects' COHb concentrations were five times higher than those in Horvath, Sheps concluded that there was no change in the "time to onset" or duration of angina [Sheps, 1987]. Electrocardio-physiologic parameters were also unchanged.

Thus, ETS does not make a significant contribution to indoor air CO concentrations

[8] The term dCO is the indoor CO concentration minus the outdoor CO concentration.

and it does not, as the Guide implies, starve nonsmokers of oxygen [The Guide, p. 9].

b. Hydrogen Cyanide

The Guide's discussion of hydrogen cyanide (HCN) is oversimplified and inappropriate [The Guide, p. 9]. Again, concentration is ignored. High levels of HCN have indeed been reported in *in vitro* studies to impair ciliary function, produce lung irritation, and inhibit oxygen uptake [USEPA, 1981]. However, HCN produces none of these effects at levels found in ETS. In fact, all reported HCN levels in ETS [Ball, 1986; Hoffmann, 1984; Klus, 1986] fall below or just within the "no effect" range [Ball, 1986]. Reported ETS HCN levels range from 0.009 ppm [Klus, 1986] to 0.11 ppm [Ball, 1986]. The "no effect" range is 0.1 to 0.9 ppm [Ball, 1986]. Despite these facts, the authors elected to juxtapose a toxicologically accurate characterization of HCN (as "more potent than carbon monoxide in its ability to starve one of oxygen)" [The Guide, p. 9] next to scientifically unsubstantiated and inflammatory statements about ETS CO exposure. In short, the toxicity of undiluted HCN is unquestioned, but the authors' comments in the context of ETS are deceptive.

c. Ammonia

The Guide characterizes ammonia (NH_3) as "a powerful eye and respiratory irritant" [The Guide, p. 9]. By ignoring concentration, the Guide implies that ETS will irritate the eyes and respiratory system because it contains ammonia. The irritation threshold for ammonia has been reported to be 104 ppm [Ruth, 1986]. According to the 1986 Surgeon General's Report, NH_3 concentrations in ETS range from 1 to 4.6 ppm [USPHS, 1986, p. 230]. A more recent study reported concentrations ranging from "not detectable" to 9.5 ppm, with the median reported as "not detectable" [Sterling, E.M., 1988]. Even at the

maximum reported value (9.5 ppm), ammonia concentrations in ETS are an order of magnitude less than the irritation threshold [Ruth, 1986]. Given levels found in typical real-world environments and irritation threshold levels, ETS ammonia cannot be characterized as an irritant.

d. Nicotine

The Policy Guide states "Nicotine, a poison, is also the addictive agent in tobacco smoke" [The Guide, p. 9]. This statement is pejorative, unsubstantiated and misleading. There is absolutely no evidence that ETS nicotine is toxic. Consequently, characterizing nicotine as a poison (in this context) is emotional rhetoric, not scientific fact. Similarly there is no evidence that ETS nicotine is addictive.

Relevant data ignored by the Policy Guide demonstrate that nicotine, at ETS concentrations, is not a poison. OSHA has established permissible exposure limits (PEL) and short term exposure limits (STEL) for nicotine at 500 $\mu g/m^3$ [Federal Register, 1989]. One 1984 study reported a 13.2 $\mu g/m^3$ mean nicotine concentration in public buildings occupied by one to five smokers [USPHS, 1986, p. 156] -- less than 3% of the OSHA standards. A more recent survey of offices containing smokers reported average nicotine levels of 4.8 $\mu g/m^3$ [Oldaker, 1990a] -- 100 times less than OSHA standards. Moreover, nicotine concentrations in ETS frequently are not detected by sensitive analytical methods -- even when smoking is occurring [Carson, 1988; Oldaker, 1986; Oldaker, 1990a; Oldaker, 1990b].

Characterizing nicotine as "the addictive agent in tobacco smoke," is inflammatory and is irrelevant to ETS. This gratuitous comment must have been included for propaganda

purposes because no one remotely suggests that exposure to ETS causes anyone to be addicted to cigarettes.

e. Summary

Without explanation, the authors ignore the central, inescapable conclusion reached by researchers at the EPA in 1989: "Data are lacking, however, on the presence and concentration of potentially toxic and carcinogenic components in tobacco-smoke polluted indoor environments" [Lofroth, 1989]. The Agency emphasized this conclusion in its 1990 Report to Congress on Indoor Air Quality:

> Information on exposure in homes and buildings is limited to a very few pollutants and groups of pollutants. In addition, virtually nothing is known about cancer and noncancer health effects due to low level respiratory exposures to multiple chemical contaminants [USEPA, 1989a, p. 1].

In light of pertinent data and the Agency's recent statements, the Guide's simplistic, misleading and inflammatory discussion of ETS constituents can only be viewed as a clear abuse of the EPA's statutory authority.

3. The Policy Guide Trivializes The Complexities Of Exposure Assessment

An accurate measurement of exposure is critical to assess the potential impact of ETS on human health. Current technology for measuring ETS concentrations in the air provides only upper bound estimates for some ETS components. Current biomarker technology for measuring ETS dose in the body is inadequate. The EPA recently stated that "air quality problems are pervasive in a wide spectrum of buildings, but the prevalence of such problems, the nature of their sources and the amount of human exposure attributable to these sources remains virtually unknown" [USEPA, 1990a, p. 3].

After acknowledging the uncertainties inherent to ETS exposure measurement [The Guide, pp. 11-14], the authors rationalize the use of several problematic measures. The Guide wrongly implies that ETS exposure has been "measured" and promotes several inappropriate surrogate "markers." The authors never discuss the severe limitations of these markers and never acknowledge the absence of an accepted quantitative method for them.

a. Air Monitoring -- RSP

The Guide recognizes that several surrogates, or tracers, are necessary to estimate ETS exposure, but relies primarily on respirable suspended particles (RSP) as a marker in its discussion of air monitoring with respect to ETS. As Ogden and Maiolo have noted: "RSP, although a necessary indicator of indoor air quality in general, is an inappropriate tracer of ETS . . . ." [Ogden, 1990, p. 420].

The National Research Council's Committee on Passive Smoking has established four criteria to assess the reliability of a proposed ETS tracer:

- unique or nearly unique to the tobacco smoke so that other sources are minor in comparison,
- a constituent of the tobacco smoke present in sufficient quantity such that concentrations of it can be detected in air, even at low smoking rates,
- similar in emission rates for a variety of tobacco products, and
- in a fairly consistent ratio to the individual contaminant of interest or category of contaminants of interest (e.g., suspended particulates) under a range of environmental conditions encountered and for a variety of tobacco products [NRC, 1986, p. 70].

Respirable suspended particles fail to meet two of these criteria. First, RSP is <u>not</u> "uniquely attributable to tobacco" and RSP measurements overestimate ETS exposure.

> Although total RSP concentration can be reliably determined, it too is not specific to tobacco smoke . . . . As a result, total RSP can overestimate exposure to ETS, although the magnitude of this bias can be reduced by determining a quantity identified as ultraviolet-particulate matter (UV-PM). Based on an empirical relationship between RSP and its UV absorption established on particulate matter collected in an environmental chamber containing only ETS, the contribution of ETS to RSP in indoor environments can be estimated. Since this procedure relies on measuring the total absorbance at 325 nm of the methanol extract of filters used to collect RSP, the tendency is still toward overestimating the ETS contribution to RSP [Ogden, 1988, pp. 77-78] (Emphasis added).

Many sources contribute to RSP in indoor environments -- clothing, furniture, office equipment, paper, carpet, and dust. Humans themselves are a major source of particles. "Virtually everything we use in buildings sheds particulate matter and/or produce gases" [Robertson, 1990, p. 334]. Consequently, according to DiNardi, "the measured increase in RSP due to ETS can only be reported if the occupied and nonsmoking RSP background is determined and compared to the occupied and smoking level of RSP" [DiNardi, 1986, p. 94].

Second, RSP is not in constant ratio to other constituents of ETS "under a range of environmental conditions encountered." As the Guide correctly notes, ETS is a two-phase mixture, consisting of particulate and gaseous phases [The Guide, p. 7]. However, the Guide fails to note that both phases are in a continuous and complex state of flux. At any given moment, constituents of either phase are evolving chemically, thermodynamically and mechanically according to laws of aerosol physics and chemistry. One important aspect of this disequilibrium is decay. Constituents within each phase of ETS deplete at different

decay rates (*e.g.*, by condensation, evaporation, nucleation, sedimentation, sorption, chemical reaction, selective filtration) [Kay, 1990; Nelson, 1990a; Nelson, 1990b]. The relative concentrations are never static.

Consequently, no single component, including RSP, can be in constant ratio with all other components in a dynamic two-phase mixture. Measurement of RSP does not produce an accurate estimate of ETS exposure. The Guide's virtually exclusive reliance upon RSP as a marker is therefore unwarranted and arbitrary.

Even if RSP were a reliable ETS surrogate, the Guide incorrectly implies that ETS levels in the workplace are high. The authors ignore pertinent workplace data and manipulate other, irrelevant data to exaggerate the contribution of ETS to indoor air quality.

The Guide relies exclusively on studies of home, as opposed to workplace, environments. Workplace environments differ dramatically from home environments in terms of smoking behavior, ventilation, RSP sources and other important parameters. Workplace exposure levels are generally very low [Oldaker, 1990a]. Inexplicably the Guide never acknowledges these important differences.

The authors contend both that ETS is "the major contributor to RSP in indoor air," and that "each smoker generates 25 to 35 micrograms of RSP per cubic meter of air ($\mu g/m^3$)" [The Guide, p. 12].[9] Neither contention is supported by the data. The Guide describes a

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[9] The Guide also claims that "[h]omes with two or more heavy smokers frequently exceed the federal 24-hour outdoor particle standard of 260 $\mu g/m^3$" [The Guide, p. 12]. This statement contains an egregious error, because there is no such federal standard. EPA abandoned that standard in 1986, replacing it with a standard for particulate matter known as PM-10. The authors of the Guide should at least be held to modest accuracy in such assertions.

study by Spengler which reports mean indoor RSP concentrations of 24.4 $\mu g/m^3$ for 35 homes with no smokers, and 36.5 $\mu g/m^3$ for 15 homes with one smoker [Spengler, 1981, Table 2]. If one assumes conservatively that tobacco smoke particulate matter accounts for the entire observed difference, then a simple calculation shows that ETS contributes at most 12.1 $\mu g/m^3$ to the RSP concentration in homes with one smoker. At least twice as much RSP comes from other sources.

Other studies not cited in the Guide confirm that ETS does not constitute the bulk of RSP in indoor air. For example, Proctor demonstrated in a field study that ETS contributed <6% to 40% of the total RSP of one modern, air-conditioned office building in England. The bulk of particulate matter in the offices surveyed came from non-ETS sources [Proctor, 1989]. Similarly, Oldaker reported that, based on UV-PM[10] and RSP measurements taken in a survey of indoor air quality in four major cities, ETS contributed at most 21% and 30% to the total RSP in offices and restaurants, respectively [Oldaker, 1990a].

b. Air Monitoring -- Nicotine

The most commonly used surrogate for ETS monitoring is nicotine. But nicotine, a vapor-phase component [Eudy, 1986] also fails to meet a critical NRC criterion. Like RSP it is not present in a constant ratio to other ETS constituents, even within a single controlled environment -- much less, among a wide variety of environments as mandated by the NRC

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[10] UV-PM is an estimate of ETS respirable suspended particles. "Based on an empirical relationship between RSP and its UV absorption established on particulate matter in an environmental chamber containing only ETS, the contribution of ETS to RSP in indoor environments can be estimated" [Ogden, 1990, p. 415]. Ogden reports that UV-PM overestimates the contribution of ETS to RSP by 26%.

[Kay, 1990; Nelson, 1990a; Nelson, 1990b; Oldaker, 1990a]. In fact, nicotine is particularly elusive as a marker:

> The peculiar decay behavior of nicotine, which is believed to be caused by its ready adsorption onto and desorption from surfaces, causes the ratio of nicotine concentrations to the concentrations of other ETS constituents such as CO, volatile organic compounds, and particulate matter to be highly variable. By itself, the large variation in nicotine/constituent ratios can lead to large errors in the estimation of exposure to ETS. If nicotine concentrations are measured for a long time following smoking, the concentration of ETS components other than nicotine may decay to background levels before nicotine. Under these circumstances, if nicotine is used to predict exposure to other ETS constituents or to calculate an "equivalent number of cigarettes smoked" the numbers obtained can greatly overestimate actual exposures [Nelson, 1990a, p. 371].

Plausible ETS exposure estimation must integrate all the NRC tracer acceptability criteria. Additionally, it must include accurate quantification of several ETS-specific constituents -- *i.e.*, representative cross-sections of each phase. Few studies to date have measured more than one surrogate [Carson, 1988; Crouse, 1990; Oldaker, 1990a; Oldaker, 1990b]. Inexcusably, the Guide ignores these complications.

The measurements of ETS nicotine and particulate matter have no proven relationship to health effects. The best measurement techniques in use merely indicate an upper bound for the contribution of ETS to the concentrations of certain components found in indoor air.

c. Air Monitoring -- Dynamics

The Guide states that "ETS diffuses rapidly through buildings . . . ." [The Guide, pp. 3, 12]. This statement reveals a technical misunderstanding of the term "diffuse" and perpetuates a false, pernicious notion of ETS dynamics.

First, the Guide's authors confuse the term "diffuse," which has a specific physical/mathematical meaning, with the generic terms "disperse" or "dissipate" throughout the document. ETS disperses in buildings by convective and diffusive mechanisms. ETS is removed by the simultaneous mechanisms of deposition, impaction, filtration, sorption, and ventilation. For the particulate phase, convective forces dominate; for the gaseous phase, diffusion may be equally important. Diffusion coefficients may differ by orders of magnitude among particulate- and vapor-phase species [Reist, 1984].

Second, the authors falsely imply that ETS rapidly pervades all confines of a building at like concentration, regardless of distance from the source. In reality, concentration depends critically on location and time. Sterling examined the influence of ventilation and smoking-rate parameters on ETS concentrations in indoor environments [Sterling, T., 1989]. His data indicated minimal recirculation of ETS between smoking and nonsmoking areas -- areas on different floors or within different sections of a single large room. In his study, smoking and nonsmoking areas of a cafeteria (which were neither separately ventilated nor physically separated) could be distinguished by nicotine or RSP measurements. In this case, different concentrations of ETS components were measured within the same room. Concentrations were lower in areas further from the cafeteria, even though served by the same ventilation system [Sterling, T., 1989]. Sterling's work supports Kim's conclusions

regarding concentration gradients within the same room [Kim, 1990]. Sterling also found that nicotine concentrations were below the detection limit in nonsmoking offices -- offices that shared recirculated air from the cafeteria. RSP concentrations were also low and apparently unaffected by ETS [Sterling, T., 1989].

Hedge measured ETS constituent concentrations in smoking and nonsmoking areas of a smoking restricted 40-story office building [Hedge, 1990]. Both nicotine and UV-PM were monitored. Results are presented in Table 1. From morning to afternoon, UV-PM levels in nonsmoking areas remained constant (or decreased slightly), while increasing in smoking areas. Clearly, concentrations of both species depend upon time and location. This result, coupled with the absence of detectable nicotine in nonsmoking areas, refutes the Guide's implication that concentration gradients are transitory or nonexistent. The data also suggest that ETS has a negligible impact in the nonsmoking areas of properly ventilated buildings.

Table 1: Nicotine and UV-PM Concentrations

	CONCENTRATION ($\mu g/m^3$)			
	SMOKING		NONSMOKING	
	a.m.	p.m.	a.m.	p.m.
Nicotine	27.2	41.2	0.0	0.0
UV-PM	120	185	9.0	7.0

The Guide's statement that ETS "persists for long periods after smoking ends . . ." [The Guide, pp. 3, 12] is misleading. In fact, most ETS constituents are removed rapidly from building interiors by dilution with fresh air introduced by the ventilation system [Arfi, 1989; Kim, 1990]. Several studies have demonstrated that removal of most ETS constituents (for which measurements exist) from room environments is proportional to the air exchange

rate [Baker, 1990; Nelson, 1990a; Nelson, 1990b]. Other studies have shown that ETS concentrations decrease so rapidly that it is often difficult to determine whether, or where, smoking has occurred [Carson, 1988; Oldaker, 1990a; Oldaker, 1986].

The Guide therefore oversimplifies the science of air monitoring and ignores important relevant data. A more comprehensive review of the literature compels the following conclusions:

1. Because of dilution and decay, ETS is generally present in extremely low levels in the workplace; often ETS constituents are not detectable by current scientific methods;

2. Removal of ETS from an interior environment is strongly dependent on air exchange rate;

3. If a building is properly ventilated, the dilution process is both rapid and efficient; and

4. Under proper ventilation conditions, ETS recirculates to a very small extent between smoking and nonsmoking areas.

d. Biomarkers -- Cotinine

The Guide's section on "Biomarker Studies" [The Guide, p. 13] creates an impression of manifest scientific verity by oversimplification, false assumptions and mischaracterizations. This section fails to acknowledge the complexities of biomarker science and the scarcity of hard data regarding ETS biomarkers. It assumes incorrectly that cotinine levels in body fluids are reliable indicators of ETS exposure.

Quantitative measures of ETS exposure are necessary to evaluate whether health effects may be attributed to ETS exposure. To be useful, biomarkers must indicate whether exposure to a specific chemical or mixture of chemicals has occurred, and be related to health. Ideally, a biomarker should (i) be specific to the agent being studied; (ii) serve as

an index of exposure; (iii) give an indication of the health risks, if any, of exposure; and (iv) be measurable at low levels [Reasor, 1990]. Several ETS species (individual chemical compounds, as well as RSP) have been proposed as biomarker candidates, but none meets these criteria [Reasor, 1990].

Biomarkers can sometimes provide accurate estimates of exposure to individual chemical species. Nicotine and cotinine in body fluids, for example, indicate exposure to nicotine. Neither can serve as surrogates for exposure to other ETS constituents [Kay, 1990; Nelson, 1990a; Nelson, 1990b]. Consequently, the Guide's statement that observation of a single biomarker (cotinine) indicates that subjects "have inhaled and retained ETS" is an oversimplification [The Guide, p. 13].

i. Dietary Nicotine

The Guide contends that cotinine, a nicotine metabolite, is "totally unique to tobacco" and therefore "is a reliable indicator of ETS exposure" [The Guide, p. 13]. The assumption follows that anyone with cotinine in their blood has been exposed to ETS.

> Positive cotinine concentrations in three out of four nonsmokers, including persons reporting no exposure to tobacco smoke in the measuring period (up to a few days, depending on the body fluid tested) demonstrate the ubiquity of ETS exposure in nonsmokers [The Guide, p. 13].

This conclusion and the assumptions on which it is based are speculative and probably false.

Nicotine is present in a variety of foods (solanaceous vegetables including green peppers, tomatoes and eggplants). As a result, the observation of cotinine in body fluids does not confirm exposure to ETS [Castro, 1986; Idle, 1990; Sheen, 1988]. It only indicates

exposure to nicotine -- which may or may not have come from tobacco. Cotinine, therefore, is not always a reliable indicator of ETS exposure. Significantly, Castro have concluded:

> The data we have obtained thus far suggests that the amount of nicotine ingested from dietary sources closely parallels or may exceed that ingested directly or indirectly from the smoke of nationally advertised 'low-yield' cigarettes [Castro, 1986, p. 94].

The Guide's statement that "[c]otinine has also been measured in the urine of people who were unaware they had been exposed" [The Guide, p. 13] is entirely consistent with ingestion of nicotine through dietary sources. Dietary nicotine, particularly the presence of nicotine in tea [Sheen, 1988] might explain why, as the Guide notes, "in several British studies, nearly all nonsmokers had measurable cotinine levels, regardless of reported exposure" [The Guide, p. 13].

Moreover, as a consequence of nicotine's unique environmental "decay" behavior, the presence of cotinine in nonsmokers exposed to tobacco-smoke nicotine may be of little relevance to overall ETS exposure. Because nicotine tends "to linger in the environment longer than other ETS constituents" [Nelson, 1990a], it is not possible to correlate cotinine levels with direct exposure to ETS. It may, for example, have originated from the clothing of a smoker or interior surfaces of the room [Nelson, 1990a; Nelson, 1990b]. In addition, a vapor-phase marker cannot be used to estimate particulate-phase exposure.

ii. Cotinine Measurement Errors

The low concentrations found in nonsmokers' body fluids seriously diminish its viability as a biomarker. Contrary to the Guide's claim [The Guide, p. 13], cotinine is very difficult to measure in any biological fluid. In fact, cotinine levels in nonsmokers' body fluids are near the detection limits of most reported analytical methodologies [Watts, 1990].

At present, no standardized, validated method exists to quantify such low concentrations. Researchers have developed several different methods. Reproducibility of results is a problem. In a comparative study, Biber found that nonsmoker cotinine levels varied significantly among laboratories [Biber, 1987]. He also found that several laboratories were unable to detect cotinine in serum from exposed nonsmokers. Concentrations reported by some laboratories bore no relationship to estimated ETS exposure, or were unrealistically high. In two separate studies, Haley and Schepers reported a problem in immunoassays of cotinine due to cross-reactivity between the commonly-used anticotinine antibodies and trans-3-hydroxycotinine [Haley, 1990; Schepers, 1988].

In addition, cotinine measurement in urine can be subject to error because of caffeine interference. Cummings [the Guide's reference 37] used an HPLC method [Machacek, 1986] to screen the urine of 663 never-smokers and exsmokers for nicotine and cotinine [Cummings, 1990]. The reported mean urinary cotinine concentrations appear unusually high (9.5 ng/ml) for people who claimed they had not been exposed to ETS. These levels of cotinine may be explained by the caffeine interference inherent in this HPLC method. Thuan found that caffeine eluted between cotinine and the phenylimidazole internal standard, causing interference with the cotinine determinations [Thuan, 1989]. In contrast, Matsunga offered a better estimate of nonsmoker exposure to environmental nicotine, using gas chromatography to determine nicotine and cotinine [Matsunga, 1989]. He reported mean urinary cotinine concentrations of 0.4 ± 0.2 ng/ml in nonsmokers claiming no ETS exposure, and 3.5 ± 2.1 ng/ml in nonsmokers with confirmed ETS exposure.

Finally, the measurement of urinary cotinine concentrations is complicated further

because cotinine is present in smokers' urine in two chemically distinct forms: the free base and as a conjugate [Curvall, 1989]. Either form can predominate and there are considerable intra- and inter-individual differences. Current methods for the quantification of cotinine only determine the free-base form, therefore providing an incomplete measure of the cotinine present.

For these reasons, the Guide's ETS exposure estimates based on cotinine concentrations are arbitrary and misleading. Because of potential dietary sources of nicotine, the inability of nicotine or its metabolite, cotinine, to predict exposure to other ETS constituents, and the potential for error in measuring cotinine, ETS exposure estimates based upon cotinine have serious limitations.

e. Other Surrogates

The Guide's discussion of "Other Surrogates" [The Guide, p. 13] perpetuates the errors of preceding sections. Specifically, it reinforces the false impression that RSP, nicotine, or other individual constituents when measured independently, are reliable indicators of ETS exposure. It also reinforces the unproven assertion that ETS presents a major health threat to nonsmokers.

The Guide states: "Air monitor studies have shown nicotine levels considerably greater in homes with smokers than in homes without" [The Guide, p. 13]. This is another example of the authors' reliance on data from non-workplace environments to build a case for workplace smoking bans. Moreover, as previously discussed, several studies have shown that nicotine overestimates ETS exposure, because of its unique decay behavior and other properties [Baker, 1990; Nelson, 1990a; Nelson, 1990b].

The discussion of benzene [The Guide, p. 13] is particularly inaccurate and misleading. By juxtaposing such statements as: "[Benzene] causes leukemia in humans at occupational levels," with "ETS is a significant source of benzene exposure" [The Guide, p. 13], the Guide leaves a strong, but totally false, impression that nonsmokers develop leukemia from ETS exposure.

Benzene exposure from ETS is negligible. Automotive fuel is, by far, the largest, most pervasive source of benzene exposure. In 1989, the U.S. Department of Health and Human Services estimated that 1 billion pounds of benzene were released into the atmosphere from the refueling and operation of approximately 130 million motor vehicles in 1976 [NIEHS, 1989]. This translates into 7.8 pounds of benzene per vehicle per year. In contrast, a pack-per-day smoker would generate approximately 0.008 pounds of benzene per year, assuming that, at most, 0.5 mg of benzene is generated from one cigarette (MS plus SS) [Hoffmann, 1990]. Based on these estimates, an average person is potentially exposed to 1,000 times more ambient benzene from one automobile than from a smoker in a given year. The Guide's characterization of ETS as "a significant source of benzene exposure" is plainly wrong.

This mischaracterization, coupled with the false implication that ETS benzene is a proven cause of leukemia, is yet another example of the Guide's distortion of data in its campaign to persuade the reader that ETS presents a major health threat to nonsmokers -- an implication that has not been proven and that is not supported by scientific data.

f. Questionnaires

The Guide's "Questionnaires" section [The Guide, p. 14] identifies some limitations of using questionnaires to quantify ETS exposure:

> First, questionnaires usually only can address short term exposure and do not provide an indication of dose over a lifetime. Second, most questionnaires have limited the discussion of ETS exposure to the home, while a large amount of the exposure may take place away from the home (at work or in public places). Third, it is often difficult to measure and quantify ETS exposure at work [The Guide, p. 14].

These limitations are by no means all the shortcomings of questionnaires. In fact, the Guide ignores many important provisos to their use in developing statistical data -- including respondent bias and subsequent misclassification [Jarvis, 1984]. Experts repeatedly question the reliance on questionnaires to assess ETS exposure [*e.g.*, Coultas, 1989]. The Surgeon General identified several additional problems with questionnaires:

> In studies of the effect of ETS exposure, two types of misclassification are of concern: misclassification of current or former smokers as never smokers and misclassification of the extent of ETS exposure. [USPHS, 1986, p. 35].

Because of the many drawbacks to questionnaires, RJRT disagrees with the Guide's statement that "questionnaires have proved to be an effective tool to distinguish between populations that receive a high level of exposure and those that receive a smaller level" [The Guide, p. 14]. Questionnaire responses do not reliably predict ETS exposure levels. Questionnaires may be of some value as supplements to other exposure indicators (such as appropriate biomarkers), but they cannot be used blindly or alone.

g. Mathematical Models

The Guide's extremely brief discussion of mathematical modeling -- and its conclusion

that "[m]athematical models ... have proved to be a reasonable way to estimate ETS exposure" [The Guide, p. 14] -- leave the reader with the impression that accurate and reliable mathematical models have been developed to predict ETS exposure. The Guide fails to describe any specific model used to estimate ETS exposure, the many factors that must be considered in any such model, and the relative significance of these various factors. By failing to discuss the many assumptions, approximations and unknowns inherent in the model-building process, the Guide exaggerates the ability of mathematical models to predict ETS exposure. The Guide should include a full and fair assessment of model types, their applicability and their many limitations.

A typical, drastic assumption which afflicts many ETS models in the literature is that of instantaneous, perfect mixing [Repace, 1982; Ryan, 1988]. Yamamoto identified several problems with models which assume rapid diffusion and good mixing in indoor air spaces:

> Numerous models have been developed to calculate airflow distribution and concentration profiles, many of these being supplied by the clean room research community. However, these models do not adequately account for various parameters such as supply/return air duct location and source location. Especially, the contaminant concentration in the occupants' breathing zones (1.0 to 1.5 m from the floor) will be an important consideration for evaluating the indoor air quality and the ventilation effectiveness [Yamamoto, 1990, p. 243].

Kim has addressed some of these concerns in a more rigorous aerodynamic model [Kim, 1990]. His work indicates that ETS is rapidly removed from an office space once smoking has stopped.

Many experts question the utility of mathematical models to predict ETS exposure. The accuracy of ETS models is limited both by the precision of data that are used in the

model and by the sophistication of the model itself. Several types of mathematical models have been used to estimate ETS exposure. Each type is characterized both by its own level of physicochemical and mathematical sophistication and by its utility in predicting ETS exposure. Some models in the literature are suitable only for very restricted, unrealistic environments (*e.g.*, environments at thermodynamic, chemical or mechanical equilibrium) [Repace, 1982; Ryan, 1988]. Others can be applied more generally, but are severely limited by computational requirements [Horstman, 1988]. None, however, can be applied universally. The Guide ignores this important qualification. Contrary to the impression created by the Guide, accurate prediction of ETS exposure in workplace environments by mathematical modeling has never been achieved.

4. The Purported Relationship Between ETS And Certain Health Effects Is Dubious

Throughout Chapter 3, "Health Effects of ETS" [The Guide, pp. 15-18], the Guide makes sweeping assertions regarding purported health effects relative to ETS. These assertions are based in part on the biased and inaccurate Health Assessment. The Guide's discussion of alleged health effects is selective; when contrary data are acknowledged, they are minimized. In addition, the Guide reviews data that are largely irrelevant to the workplace.

a. Irritation

Many statements in the Guide's section "Irritation" [The Guide, p. 15] are unsubstantiated. The authors do not discuss workers' responses to ETS in the workplace. For instance, there are undocumented statements such as: "[f]or many people the effect is annoying; for some it can be incapacitating" and the "result [of ETS irritation] is generally

a sore throat or cough" [The Guide, p. 15]. In poorly ventilated areas, occupants may consider high levels of ETS to be annoying. These complaints are most frequently associated with inadequate ventilation [Oldaker, 1989] or to indoor air contaminants other than ETS [Robertson, 1990].

There are no studies of "incapacitation" due to ETS exposure. Subjective perceptions of urge to cough and sensations of sore throat have been studied. Winneke found that subjects' responses to ballot terms: "urge to cough," "sore throat," "breathing is impaired" or "my eyes are burning" were not significantly different from control responses at or below 5 ppm dCO [Winneke, 1984]. Subjects' responses were significantly different from control responses at 15 ppm dCO. To put these data in perspective, dCO values above 5.0 ppm were found in only one out of 23 offices in a survey of smoker-occupied offices [Carson, 1988]. The mean dCO for the 23 offices was 0.2 ppm; the range was -4.3 to 6.6 ppm. Even ignoring the likely potential irrelevance of CO measurements to ETS concentrations, these data indicate that workplace ETS levels are below the threshold levels at which subjects experience discomfort.

There are no data to support the suggestion that irritation results from ETS levels found in the workplace. Researchers typically expose subjects to levels of ETS much greater than those encountered in real environments in order to elicit significant responses. Kay found that high levels of ETS were required to produce subjective complaints.

> Exorbitantly high ETS levels, 13 to 65 times greater than the approximately 5 to 12 $\mu g/m^3$ nicotine levels found in field studies, were selected for this study so that quantifiable and significant differences in subject responses could be obtained [Kay, 1990, p. 275].

Conclusions about irritation from ETS as a problem in the workplace are unwarranted.

b. Lung Cancer

The Guide follows several lines of evidence to conclude that ETS is a proven cause of human lung cancer: epidemiologic studies of lung cancer and spousal smoking, the 1986 reports by the Surgeon General [USPHS, 1986] and the National Research Council [NRC, 1986], and the EPA's draft Health Assessment[11]. Evaluations of ETS and lung cancer that either reach alternate conclusions [*e,g.*, Layard, 1990; Letzel, 1988; Uberla, 1988; Uberla, 1987], or suggest alternate explanations for the data [Koo, 1989; Lee, 1987] are ignored. Contrary to the discussion presented in the Guide [The Guide, pp. 15, 16, 19], neither the Health Assessment nor any other review has presented a persuasive case that ETS is a risk factor for lung cancer -- let alone that ETS is a proven cause of lung cancer.

The flaws in the Guide's conclusion regarding ETS and lung cancer [The Guide, pp. 15, 16, 19] and in the EPA's draft Health Assessment are discussed in detail in RJRT's response to the Health Assessment. Nevertheless, several points are worthy of emphasis here.

The epidemiologic studies are primarily of spousal smoking (not ETS) and lung cancer. Over 80% of the studies relied upon by the EPA's Health Assessment found no statistically significant increased incidence of lung cancer for nonsmokers married to a smoker. The observed associations in the few remaining studies are weak. Those studies were conducted in countries where lifestyles, diet and environmental factors are very

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[11] RJRT has submitted comments on the Health Assessment. Those comments contain a more complete review of ETS and lung cancer studies and discuss the many flaws in the EPA's Health Assessment.

different from those in the United States. In contrast to these foreign studies, no U.S. study of spousal smoking and lung cancer, including the largest study performed to date [Varela, 1987], has reported a statistically significant association. Bias and the many confounding factors cannot be excluded in any of the studies.

The authors ignore epidemiologic studies which investigated ETS exposure in the workplace. To date, six studies [Garfinkel, 1985; Kabat, 1984; Lee, 1986; Shimizu, 1988; Varela, 1987; Wu, 1985] have examined workplace ETS exposure and lung cancer. None found a statistically significant risk of lung cancer associated with exposure to ETS in the workplace. Results from three of these studies are summarized below.

Varela failed to find an association between exposure to ETS in the workplace and lung cancer (OR = 0.99, 95% CI = 0.97, 1.01) [Varela, 1987]. Variations in duration of exposure did not affect the results. Garfinkel examined the odds ratio for lung cancer and workplace ETS exposure for women [Garfinkel, 1985]. No association with workplace exposure was observed (OR = 0.88, 95% CI = 0.66, 1.18; OR = 0.93, 95% CI = 0.73, 1.18, for 5 years and 25 years workplace exposure, respectively). Wu found that nonsmokers exposed to ETS in the workplace did not have a statistically significant increased risk of developing lung cancer, compared to nonsmokers not exposed to ETS in the workplace (RR =1.2, 95% CI = 0.8, 2.2) [Wu, 1985].

In summary, the EPA's conclusions regarding ETS and lung cancer are based on a biased and incorrect interpretation of epidemiologic studies of spousal smoking and lung cancer. The Guide does not adequately address the uncertainty and limitations associated with these studies.

c. Spurious Health-Effect Claims

The authors buttress their allegations regarding irritation and lung cancer with discussions of other health concerns which are unrelated to the workplace or for which current data do not permit conclusions to be drawn.

i. Children

Nearly one-third of Chapter 3, "Health Effects of ETS" [The Guide, pp. 15-18], discusses respiratory diseases in children. This section concludes with the following bold-lettered statement: "**it is prudent to eliminate ETS exposure from the environments of small children**" [The Guide, p. 17]. In the Health Assessment the EPA correctly recognizes that a causal relationship between ETS and respiratory diseases in children has not been established. The Guide fails to acknowledge this point or to describe how tenuous are the observed associations.

Most important, a discussion of childhood respiratory disease has no bearing on whether smoking should or should not be allowed in workplaces. Few would seriously argue that workplaces are "the environments of small children" [The Guide, p. 17]. For these reasons, the Guide's discussion of respiratory problems in children is irrelevant and should be eliminated.

ii. Asthma, Heart Disease, Allergies And Other Cancers

A significant portion of Chapter 3 relates to alleged effects of ETS on asthmatics, people with heart disease and people with allergies [The Guide, pp. 17, 18]. The Guide describes how ETS increases symptoms, aggravates the conditions or brings on allergic reactions and then acknowledges that "data are too limited," [The Guide, pp. 17, 18] or that

it "is still too early" [The Guide, p. 18] to draw conclusions. These sections appear designed to establish guilt by association. The reader will be misled by the implication that there are health concerns when, as the Guide acknowledges, no solid evidence for such conclusions exists.

The section, "People with Heart Disease," suggests that exposure to CO from ETS results in alterations of cardiovascular function in individuals already compromised with coronary vascular disease [The Guide, p. 17]. CO concentrations in ETS are trivial and are unlikely to affect those with heart disease. This section leaves the reader with the impression that ETS aggravates heart disease. Nonetheless, the authors admit that "[a] complete analysis of the data linking ETS and heart disease has not been conducted by the EPA" [The Guide, p. 17]. The Guide should state clearly that ETS exposure has not been demonstrated to aggravate existing heart disease.

Similarly, the authors discuss "Cancer at Other Sites" [The Guide, p. 18] ("brain tumors, nasal sinus cancers, genital, breast and endocrine cancers, and cervical cancer" in "nonsmoking women"). They concede that "the data are too limited to be conclusive," and note that "additional research is needed in this area" [The Guide, p. 18]. They do not, however, state clearly that ETS exposure in the workplace has never been implicated in these chronic diseases.

In short, sections on asthma, heart disease, allergies and other cancers are filled with suppositions [The Guide, pp. 17, 18]. The Guide's treatment of these issues appears contrived to mislead the reader.

C. Smoking Bans Do Not Effectively Address Indoor Air Quality Problems

The Guide creates the false impression that smoking bans will eliminate a wide variety of potentially toxic compounds from indoor air when, in fact, ETS is only one aspect of indoor air quality. Many pollutants other than ETS, *e.g.*, asbestos, formaldehyde, ozone, volatile organic compounds, cotton fibers, fungi, bacteria, and dust mite excreta, have a significant impact on indoor air quality [Molina, 1989; Robertson, 1990].

Virtually everything used in the interior of a building, including building materials, furnishings, equipment and supplies, is a source of airborne particles and/or gases [Kirk, 1988; Robertson, 1990; Wanner, 1984]. Some particles and gases are released quickly, while others are released at a slow rate for years. Office supplies and equipment, especially generators, duplicators, copiers and ovens have been found to release potentially hazardous chemicals. Formaldehyde is released from bulk paper supplies. The human body is also a major contributor to indoor air pollution. Each person sheds literally millions of particles, primarily skin scales, per minute. Many of these scales carry microbes; the bulk of which are short-lived and harmless. Some persist, however, and contribute to the particulate burden in an indoor environment. As stated by Kirk:

> There are many sources of indoor air pollutants (both gaseous and particulate) including the use of gas stoves and fires, coal, coke and wood fires, house plants, cooking, cleaning, painting, and the adoption of a variety of household and office products including cleaning agents, glues, correction fluids, plastics, and varnishes. In addition, the simple act of movement resuspends particulate matter whilst building materials and furnishings, especially when new, may release a variety of organic materials into the indoor atmosphere. Release of formaldehyde from cavity wall insulation, furniture and fabrics are all examples of such indoor air pollutants and are of considerable public concern [Kirk, 1988, p. 99].

Although cleaning processes such as sweeping, vacuuming, and dusting normally remove the larger particles, they often increase the airborne concentrations of the smaller particles [Robertson, 1990].

Low relative humidity causes complaints similar to those ascribed to ETS [Jaakkola, 1990; Reinikainen, 1990]. Temperature, noise, poor lighting, and stress can also result in similar complaints [Molina, 1989]. Indoor air quality complaints are referred to as sick building syndrome[12] (SBS).

Investigators of building-related complaints have found multiple causes of SBS and have reported ETS to be a negligible source of complaints about indoor air quality [Collett, 1989; Melius, 1985; Robertson, 1990]. For example, from 1981 through 1987, one company studied indoor air quality in 223 different buildings, accounting for over 39 million square feet of property, and found that ETS was a significant concern in only 10, or 4%, of the buildings [Robertson, 1989]. In another SBS database, smoking was implicated as a major contributor to complaints in only 12 of 408 (<3%) of the buildings surveyed [Collett, 1989]. The National Institute for Occupational Safety and Health (NIOSH) investigated more than 200 "sick" buildings and found that tobacco smoke was the source of claimed discomfort in only 2% of the buildings investigated. Ventilation problems caused half the complaints; outdoor air was considered a bigger problem than ETS [Melius, 1985]. Other investigators

[12] Sick building syndrome comprises a group of symptoms which are reported by people working predominantly in air conditioned buildings [Molina, 1989]. Typical symptoms are eye and nose irritation, fatigue, coughing, rhinitis, nausea, headaches, sore throats and general respiratory problems [Robertson, 1988].

concluded that bacterial and fungal contamination is a major source of indoor air problems [Collett, 1989; Nevalainen, 1990; Robertson, 1990, Strom, 1990]. Inadequate ventilation exacerbates all indoor air quality problems.

Ignoring the broader issue of indoor air quality, the Guide recommends only extreme solutions. The implication that banning smoking or establishing smoking policies will make indoor air "safe" is not justified. Crawford concluded: "The prohibition of smoking in the workplace will not reduce the adverse health effects of industrial processes. It will remove the telltale signs of inadequate ventilation" [Crawford, 1988, p. 203]. Similarly, Robertson confirmed that ETS is blamed for poor indoor air quality because it can be seen and smelled:

> In reality, ETS is merely a symptom of an invisible problem, not a cause. Without question, the leading cause of sick buildings is inadequate ventilation. If visible pollutants like smoke accumulate inside a building, so too do pollutants that are invisible. The symptoms often wrongly attributed to ETS can be caused by a veritable garden of contaminants. Scores of nontobacco pollutants such as formaldehyde, carbon monoxide, oxides of nitrogen, ozone, fungal and bacterial spores, cotton fibers and fiberglass fragments -- coupled with poor ventilation -- are generally found on investigation to be the real cause of the problems reported by building occupants.
>
> The Surgeon General's 1986 report on 'involuntary smoking' questioned the effectiveness of ventilation as a mitigation strategy for ETS. We have found, however, that proper ventilation quickly dissipates ETS. Indeed, ventilation experts frequently gauge the operational efficiency of ventilation systems by using smoke tests. If smoke persists, it is a clue that a serious ventilation problem exists. Once the underlying problem of poor air circulation is corrected, so too is the high level of ETS [Robertson, 1990, pp. 333-334].

It is ironic that the Guide disregards the role of ventilation. An EPA headquarters building itself was "the subject of significant occupant complaints of inadequate ventilation and poor indoor air quality" [USEPA, 1989b, p. 23]. In fact, "[a]pproximately six EPA employees have been advised by their physicians not to reenter the building. Some of these employees exhibit signs of a heightened sensitivity to the air pollutants present in the complex" [USEPA, 1989b, p. 23]. Smoking was not permitted in the building. The Agency's own experience shows that smoking bans do not effectively address indoor air quality problems.

D. The Guide Should Objectively Discuss The Entire Range Of Possible Workplace Policies, Not Just Extreme Options

The EPA has stated that it intends, in the Guide, "to describe the range of technical and policy options for instituting effective smoking policies" [Federal Register, 1990]. The Guide identifies seven policy options, but summarily dismisses all but two: outright prohibition of smoking in the workplace or separately ventilated smoking rooms. Since the latter option will be prohibitively expensive for many employers, only the former option -- total smoking bans -- is likely to be considered. As currently written, the Guide could appropriately be titled: "Guide to Workplace Smoking Prohibition."

The rationale for these two options depends upon the EPA's conclusion in the Health Assessment that ETS is a proven Group A carcinogen. As previously discussed, the data do not support that conclusion. Consequently, the rationale for including only two extreme policies fails.

The Agency must appropriately evaluate additional alternatives. Simple accommodation, emphasizing flexibility, common courtesy and interactive management has

proven viable [BNA, 1987]. Studies indicate that ETS levels in offices where smoking is permitted are low [Carson, 1988; Proctor, 1989].

Other studies show that ETS does not significantly recirculate between smoking and nonsmoking areas of buildings. Even in the same room, smoking and nonsmoking sections effectively mitigate nonsmoker exposure to ETS [Kim, 1990]. Not surprisingly, therefore, separating smokers and nonsmokers also has proven viable [Hedge, 1990; Sterling, 1989]. The Guide fails to include even one case study on these less extreme alternatives.

The authors also fail to consider effective ventilation as an alternative. Improved ventilation will not only reduce nonsmoker exposure to ETS, but will also improve the overall quality of indoor air. In this regard, the Agency should not ignore the experience of the American Society of Heating, Refrigerating and Air-Conditioning Engineers (ASHRAE) in formulating a national ventilation standard.

In 1973, ASHRAE published its original ventilation standard. Standard 62-73 incorporated two criteria: a minimum ventilation rate (typically 5 cfm per person) to accommodate energy conservation and a recommended rate for comfort (typically 15 cfm per person) [Burroughs, 1988]. In 1981, ASHRAE published a revised standard (Standard 62-1981) that required a dual ventilation standard of 5 cfm of fresh air per person in a smoke-free environment and 25 cfm in a smoking environment. In addition to "no smoking," other criteria, such as air cleaners, were required for designing a system utilizing 5 cfm [Burroughs, 1988, p. 532].

ASHRAE revised the standard in 1989 to provide for a single, standard ventilation rate of 20 cfm per person in offices with 15 cfm as a minimum. The new standard

recognizes that the 1981 standard's 5 cfm rate for nonsmoking areas was inadequate to ensure an acceptable degree of comfort in light of the many other -- imperceptible -- sources of indoor air contamination. A ventilation rate of 20 cfm is sufficient to deal with normal smoking activities as well as the full range of contaminants in buildings [Burroughs, 1988].

Perceptible ETS is a symptom of poor indoor air quality rather than a cause. Smoking bans do nothing to address the overall causes of poor indoor air quality. Thus, a manager who bans smoking in the belief that such a ban will resolve indoor air quality problems undertakes a false economy. Those businesses that adopt the Guide's recommendation to ban smoking, but fail to improve ventilation, will eliminate only a visible symptom of an indoor air quality problem without doing anything to mitigate the problem itself.

One example illustrates vividly the magnitude of the Guide's omission in its discussion of appropriate policy alternates. In 1987, The Bureau of National Affairs (BNA) published a special report which was designed to provide information on workplace smoking issues to private sector decision-makers [BNA, 1987]. BNA polled 623 employers and reported that 54 percent had smoking policies in effect and that another 21 percent were considering such policies. Of the employers with smoking policies in effect, only 12 percent had instituted complete smoking bans.

Like the Guide, the BNA report included a number of case studies. Unlike the Guide, the BNA report included nine case studies to cover a range of alternate policies -- from a flexible policy emphasizing accommodation (IBM) to a smoking ban also included in the Guide (Pacific Northwest Bell). The BNA report stands in stark contrast to the

Guide, which completely fails to include examples of more moderate policy options. When compared with the BNA report, the Guide is embarrassingly incomplete.

E. Discussion Of Litigation, Economics And Public Opinion Is Extraneous And One-Sided

The Guide includes one-sided treatments of public attitudes about ETS, the economic costs of workplace smoking, and the threat of private litigation. These sections generate heat but no light on an important and potentially far-reaching public policy statement.

The section on litigation [The Guide, pp. 28-31] merits special attention because it illustrates the insidious approach taken by the Policy Guide. Supposedly, the Policy Guide is designed to disseminate relevant information on ETS to government and private-sector decision-makers. RJRT questions the propriety of including a section on litigation in an EPA policy guide. Moreover, the Guide's discussion is particularly unbalanced and inevitably leads the reader to conclude that employers will get sued if workplace smoking bans are not instituted. The section leaves the impression that such lawsuits will generally prevail. This is not surprising, because the authors essentially present an advocate's brief, not an objective discussion of the regulatory and legal issues surrounding workplace smoking (*i.e.*, the merits of the case). Here, the EPA is advocate, judge and jury in its effort to stimulate smoking bans. The EPA has no authority to require smoking bans. By providing such an unbalanced treatment, the Agency strays far from its mission and statutory authority.

The section, "Cost Savings Related to ETS Reduction" is similarly inappropriate. The Guide suggests with undue certainty that smoking increases business costs. The cost data presented by the EPA are based on anecdotal opinion surveys, not on objective facts. Indeed, the Guide notes: "There has been relatively little research in this area and most of

the studies that have been conducted only minimally document the direct and indirect costs of ETS" [The Guide, p. 37]. This begs the question whether the documentation is minimal or whether documented costs are minimal.

Whether smoking in the workplace raises -- or lowers -- business costs is a subject of much debate but little evidence. Despite this uncertainty, the Guide presents the issue as proven. The EPA should not rely on opinion and guesswork to justify its policy recommendations. Claims that nonsmoker exposure to ETS in the workplace increases business operating costs must be quantified and verified. Without such objective information, the public cannot adequately assess this issue and is left with the impression that workplace smoking automatically raises the costs of doing business.

The section, "Public Attitudes Towards ETS" [The Guide, p. 41] attempts to validate the Guide's contentions regarding the health effects of ETS and the need for workplace smoking bans by citing public opinion surveys. This is sophistry of the highest order. The fact that people may believe ETS to be harmful does not make it so. Public opinion surveys are not pertinent to an objective analysis of the issues. Rigorous scientific investigation frequently demonstrates public opinion to be misinformed. Popular belief does not relieve the EPA of its obligation to provide an accurate and objective scientific foundation for its policy recommendations regarding ETS in the workplace. Moreover, dissemination of accurate scientific information can often result in a better informed public. That should be the EPA's objective.

F. The Policy Guide Should Be Based On Substantial Evidence

The Policy Guide should be objective and based on substantial, relevant evidence. Public policy and proper administrative procedure require nothing less. The Guide's inadequate review of ETS science and its frequent inclusion of extraneous issues to justify policy recommendations raise serious questions about its objectivity. As discussed below, the Guide's historical development, coupled with the bias and interests of its principal authors, raises similar questions about the EPA's motivations.

Perhaps the most provocative aspect of the Policy Guide is its relation to the Health Assessment and the EPA's still-unfinished Technical Compendium on Environmental Tobacco Smoke [USEPA, 1989c]. The Policy Guide was intended to be a simplified version of the Technical Compendium [Axelrad, 1989]. Curiously, the Technical Compendium has not been released for public comment and review by the Agency's Science Advisory Board. Instead, the draft Policy Guide has been released first for public comment. In other words, the Policy Guide has been developed and released in advance of the originally intended support document. Moreover, the Guide now relies extensively on a document of far more limited scientific scope, the draft Health Assessment. As a consequence, several serious questions regarding procedural regularity, objectivity and utility of the Policy Guide are raised.

In short, the Policy Guide represents a conclusion -- based largely on the predilections of the authors -- in search of a rationale. The scientific rationale chosen by the authors of the Guide may fit the policies recommended by those authors, but it does not reflect an accurate summary of the science relating to ETS. Science should not be forced to fit the

policies set forth in the Guide; the opposite should be true -- policy recommendations should follow from the science. In these circumstances, the EPA should return to the predicate task -- a complete and objective review of ETS science -- before issuing policy recommendations.

G. The Policy Guide Is An Inappropriate Exercise Of Authority By EPA

In 1986, Congress enacted Title IV of the Superfund Amendments and Reauthorization Act (SARA) to establish a research effort to characterize the extent of indoor air quality and evaluate potential mitigation strategies [SARA, 1986]. Title IV of the Radon Gas and Indoor Air Quality Research Act authorized EPA to investigate indoor air quality. Section 403 of SARA Title IV ("Title IV" or the "Title") directs EPA to:

1. Research all aspects of indoor air quality and its impact on health;

2. Coordinate federal, state, local and private research and development, and disseminate information related to improving indoor air quality, and;

3. Assess potential mitigation strategies at the federal level.

The EPA's ETS-related activities have sprung from this directive. Section 404 of Title IV, however, states expressly that the Title is not to be construed to authorize any regulatory programs or activities other than research, development, coordination and information dissemination. Notwithstanding this express restriction, the EPA has engaged in *de facto* regulation in developing this Guide. The EPA has selectively reviewed existing research, combined science with policy considerations and issued recommendations tantamount to workplace smoking regulations.

The Agency's activities are precisely those that one would expect if the Agency had regulatory authority. The Guide is clearly designed to promote workplace smoking bans in lieu of regulation because the EPA lacks authority to regulate the workplace. In other words, the EPA is hoping to accomplish extralegally what it is has not been granted the authority to do by Congress.

Further, the statute authorizes the EPA to study overall indoor air quality. Yet the EPA singled out one component of indoor air -- ETS -- and condemned it through an unacceptable review of the science. Significantly, the Guide purports to analyze the issue of ETS in the workplace, but instead relies primarily on selective data on ETS in homes. Furthermore, the EPA has relied on authors who will benefit financially from workplace smoking restrictions.

There is no substantial, relevant, scientific evidence to justify the Guide's policy recommendations. Absent substantial scientific evidence and a more objective evaluation, the EPA should not issue the Policy Guide. The Policy Guide, therefore, represents an abuse of discretion by the EPA.

Finally, the need for the Guide must be questioned. OSHA is required by statute to regulate workplace health hazards. On September 1, 1989, OSHA denied a petition by Action on Smoking and Health to ban or limit workplace smoking. The Agency concluded that the data do not establish a "grave danger" from tobacco smoke at workplace exposure levels [OSHA, 1990]. On July 9, 1990, OSHA committed to study the issue of environmental

tobacco smoke in the workplace [OSHA, 1990]. Therefore, the EPA should refrain from promulgating the Policy Guide. To do otherwise constitutes an improper exercise of administrative power, an abuse of Agency authority, and a waste of taxpayers' money.

III. CONCLUSION

The EPA has exceeded its statutory authority, abused its discretion, and compromised its scientific integrity by developing this Guide. RJRT submits that the Guide is irreparably flawed in its presentation of ETS science. In the absence of a scientific basis and legislative authority, the authors present irrelevant and misleading information in a transparent attempt to stimulate workplace smoking bans. Assuming that any action is required, the Guide fails to consider all viable policy options. For these reasons, the Agency's development of the Guide should be terminated. The public would be better served if the EPA adheres to its primary charge -- to research all aspects of indoor air quality and its impact on health. Exercises in creative writing, such as this Guide, will then be superfluous.

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