TOXIC TOXICOLOGY:

PLACING SCIENTIFIC CREDIBILITY AT RISK

PUBLIC COMMENT

Review procedures and listing criteria used in the preparation of the DHHS Report on Carcinogens (RoC)

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Independent assessment and remarks

Littlewood & Fennell is an independent public and health policy research group, with no ties whatsoever to industry or any government agency. I am here today on my own time and at my own expense to address the clear possibility that the National Toxicology Program has actively undermined the process by which risk assessments should be conducted. NTP overlooked a substantial body of evidence showing uncertainty, vagueness, and lack of statistical support of what is and is not carcinogenic. In addition, NTP conducted its assessments in a manner reminiscent of a rubber stamp proceeding, which favored politics over science.

I have included a history of our involvement with the NTP carcinogen listing process as an addendum to this paper. Briefly, we became interested in the topic of environmental tobacco smoke (or ETS) during an ongoing study of increasing rates of asthma in the U.S. Because a review of the literature indicates a negative correlation between ETS and asthma, and because ETS is physically and chemically quite different from mainstream tobacco smoke, we were curious about NTP's decision to list ETS as a carcinogen. We requested background materials from them in order to review this listing process. It was during this review that we unearthed a number of gross scientific improprieties in both procedure and conduct by a number of federal agencies. NTP is simply the latest chapter in the same sad story.

Since the topic of today's meeting is NTP's review process and procedure for listing substances as human carcinogens, I will limit most of my discussion to these matters.

There are three areas I believe are critical to the process of listing human carcinogens, none of which are addressed by NTP:

> **One: Inclusion of a reasonably expected real-life exposure to toxic or carcinogenic substances.** Exaggerated estimates of risk can themselves be toxic; inasmuch as these exaggerated estimates of risk create confusion, misunderstanding, anxiety and, inevitably, utter disdain by the general public. We call this phenomenon "Toxic Toxicology" for, in the end, it does far more harm than good.
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> **Two: Assessment of scientific research and comments based on substance, merit and quality irrespective of source -- industry, academia or government**. I contend that if you are unable to determine what is sound science without knowing the antecedents of the researcher then you are not competent to assess risk.
>
> **Three: Animal studies must be put in perspective.** Rats and mice are not little people. Nothing could make this more clear than Dr. Robert A. Weinberg's very recent success at finally inducing malignant transformation in human cells. As Gilbert L. Ross, M.D., Medical Director of the American Council on Science and Health, noted in a letter to the *New York Times* this past August: "Scientists induced such cancerous changes in rodents 15 years ago. The fact that it took so many years to accomplish this feat in humans illustrates *how differently carcinogens affect rodents than humans*… Rodents are far more susceptible to cancer induction than humans. Merely because chemicals… produce tumors in rodents does not mean that humans will be harmed."

Real-life exposure levels.

Each substance proposed for listing as a human carcinogen must be subjected to a careful and *unbiased* process of assessment for **real-life exposures**.

The compilers of reports from the National Academy of Sciences,[1] the US Surgeon General[2], and the Environmental Protection Agency have simply ***inferred the presence of ETS components by proxy***, based on the composition of the highly diffuse sidestream smoke from which ETS derives. Still, even CDC concludes, "ETS contains higher amounts of some of the components of cigarette smoke in general ***only when it is obtained in its undiluted form under laboratory conditions***" (CDC/DHSS 1989)[3].

I would propose that only those chemicals present in significant amounts – perhaps 10% of the maximum tolerated dose in rodents -- be considered. Independent laboratories could then conduct personal air monitoring for these chemicals under realistic conditions, rather than in laboratory conditions designed to exaggerate exposure risk.

CDC notes that "ETS is diluted in the air before it is inhaled and thus is less concentrated than MS (mainstream smoke)." Further, "... on the basis of urinary cotinine concentrations, the NRC [1986] concluded that non-smokers exposed to ETS absorb the equivalent of 0.1 to 1.0 cigarettes a day. On the basis of 1985 data, NIOSH estimates that each cigarette smoker in the US smokes an average of about 21 cigarettes a day. Blood and urine samples analyzed for vapor phase nicotine indicate that nonsmokers exposed to ETS absorb about 1% of the tobacco combustion products absorbed by active smokers [NRC 1986: DHHS 1986]." If these urine and blood samples were accurate, that would indicate that, at most, ETS would account for only 0.021 cigarette over exposure to 21 cigarettes on average.

In his RoC subcommittee testimony last December, Dr. Philips reported that, based on actual personal monitoring, average ETS exposure is as little as five to six cigarettes per year. I would tend to think these figures more accurate than our extrapolations from NIOSH data.

In the case of environmental tobacco smoke, such simple and rigorous personal air monitoring would have eliminated any possibility of listing ETS as a carcinogen. Most ETS components are far below the sensitivity of current analytical capabilities.[4]

Of those chemicals present in ETS, only a very few can even be classified as toxins or carcinogens. Some basic physics, a bit of chemistry and a series of rather simple mathematical calculations reveal that exposure to ETS is hardly a dangerous event. Indeed, the cancer risk of ETS to a non-smoker appears to be roughly equal to the risk of becoming addicted to heroin from eating poppy seed bagels.

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[1] NAS-NRC. Environmental Tobacco Smoke; Measuring Exposures and Assessing Health Effects. Washington, DC: National Academy Press; 1986.

[2] EPA, United States Environmental Protection Agency, Health Effects of Passive Smoking; Assessment of Lung Cancer in Adults and Respiratory Disorders in Children. Washington, DC United States Environmental Protection Agency; May 1990.

[3] Intelligence bulletin, CDC at www.cdc.gov/niosh/nasd/docs2/as73000.html.

[4] Guerin MR, Higgins CE, Jenkins RA. Measuring environmental emissions from tobacco combustion: sidestream cigarette smoke literature review. *Atmos Environ*. 1987; 21:291-297.

Calculating the non-existent risks of ETS

We have taken the substances for which measurements have actually been obtained – very few, of course, because it is difficult to even *find* these chemicals in diffuse and diluted ETS. We posit a $100m^3$ sealed and unventilated enclosure. For those of us who are metrically challenged, that is a room approximately 20-feet square with a 9-foot ceiling clearance. Taking the figures for ETS yields per cigarette directly from EPA, we calculated the number of cigarettes that would be required to reach the *lowest* published threshold for each of these substances. The results are actually quite amusing. In fact, it is difficult to imagine a situation where these threshold limits could be realized.

Our chart (see Table 1) illustrates each of these substances, but let me report some notable examples. For Benzo[a]pyrene, about which we heard so much last year, 222,000 cigarettes would be required to reach the lowest published "danger" threshold. For Acetone, 118,000 cigarettes would be required. At the lower end of the scale – in the case of Acetaldehyde or Hydrazine, more than 14,000 smokers would need to light up simultaneously in our little room to reach a threshold limit. Toluene would require 50,000 *packs* of smoldering cigarettes – given 20 cigarettes per pack.

For Hydroquinone "only" 1,250 cigarettes are required. Perhaps we could post a notice limiting this 20-foot square room to 300 rather tightly packed people smoking no more than 62 *packs* per hour?

Of course, the moment we introduce real world factors to the room – a door, an open window or two, or a healthy level of mechanical air exchange – achieving these levels becomes even more implausible.

It becomes increasingly clear to us that ETS, as well as other spurious indoor substances such as asbestos and radon, are political rather than scientific scapegoats for poorly ventilated, hermetically sealed, energy-efficient buildings, with endlessly re-circulated and poorly filtered air.

Table 1

CALCULATED NUMBER OF CIGARETTES REQUIRED TO REACH A THRESHOLD LIMIT FROM ETS IN A SEALED, UNVENTILATED 100m^3 ENCLOSURE AT STP[5]

ETS Component	CAS Number	Molecular Weight	ETS Output (mg/cigarette)[6]	Threshold Limit (ppm)	Threshold Limit (mg/m3)	Cigarettes Required
2-Toluidine	(3 isomers)[7]	107.15	0.003	2	8.7	290,000
Acetaldehyde	75-07-0	44.05	1.26	111	180[8]	14,285
Acetic acid	64-19-7	60.05	1.5	10	25	1,666
Acetone	67-64-1	58.05	1	500	1187	118,700
Benzene	71-43-2	78.11	0.24	1	3.1[9]	1,290
Benzo[a]Pyrene	50-32-8	252.30	0.00009	0.02	0.2[10]	222,000
Cadmium	7440-43-9	112.40	0.0007	0.002	0.01	1,430
Catechol	120-80-9	110.11	0.14	5	22	15,700
Dimethylamine	124-40-3	45.08	0.036	10[11]	9.2	25,555
Formic acid	64-18-6	46.02	0.525	5[12]	9.4	1,790
Hydrazine	302-01-2	32.05	0.00009(90ng)	0.01	.013	14,444
Hydroquinone	123-31-9	110.11	0.16	0.4	2	1,250
Methylamine	74-89-5	31.09	0.1	5	13	13,000
Methylchloride	74-87-3	50.49	0.88	50	103.0	11,170
Nickel	7440-02-0	58.71	0.0025	0.4	1	40,000
Phenol	108-95-2	94.11	0.25	5	19	7,600
Polonium 210[13]		210.0	0.4 pCi	na	3 pCi/liter[14]	750,000
Pyridine	110-86-1	70.10	0.39	5	16	4,100
Toluene	108-88-3	92.13	0.000035	50	375	1,000,000

These calculations are not complex. They assume a 100m^3 enclosed and unventilated space at Standard Temperature and Pressure. STP assumes 24.45 = molar volume of air in liters at STP conditions (25^{o}C. and 760 torr). Conversion equations are as follow:

$$\text{TLV in mg/m3} = \frac{(\text{TLV in ppm})(\text{gram mol wt of substance})}{24.45} \qquad \text{TLV in ppm} = \frac{(\text{TLV in mg/m3})(24.45)}{\text{gram mol wt of substance}}$$

[5] Limits expressed in ppm have been translated to mg/m^3 for the sake of clarity and volume calculation. New values have been incorporated, and the ***lowest*** threshold (irrespective of source) has been used. Unless otherwise noted, lowest threshold limit values were found in "1999 TLVs and BEIs," American Conference of Governmental Industrial Hygienists.
[6] Data from NTP RoC ETS, December 2-3, 1998, Table 1-1, pp 1-3, per EPA.
[7] Three isomers o-Toluidine [95-53-4], m-Toluidine [108-44-1], p-Toluidine [106-49-0]. Mol wt for each is 107.15, TWA/TLV is 2 ppm for each.
[8] OSHA, PEL-TWA has been raised to 200ppm, vacating a previous lower level of 180 mg/m^3. I used the lower limit.
[9] This calculation based on the lowest possible calculated OSHA/NIOSH threshold of 1 ppm.
[10] Based on coal tar pitch volatiles [65996-93-2], as benzene solubles
[11] OSHA PEL
[12] NIOSH PEL-TWA, and HSDB
[13] "Levels of polonium-210 in tobacco smoke are not believed to be great enough to significantly impact lung cancer" Hecht S, Tobacco smoke carcinogens and lung cancer. *JNCI* 1999;91:1194-1210.
[14] EPA (1990c).

Assessment of scientific research and comments based on substance, merit and quality irrespective of source -- industry, academia or government.

The tobacco industry's interest in the basic science and epidemiology of ETS may be a vested interest, but their research should be judged on its own merits -- not suppressed or ignored because the results are politically inconvenient. When scrutiny of research -- both during peer review and post-publication -- is objective and scientific it is valuable. NTP's thinly veiled hostility toward presenters finding no convincing evidence of ETS carcinogenicity is unacceptable. We found the presentations last December of varying quality, but were generally impressed with their factual and substantive nature.

As we prepared our comments earlier this year on the 9th RoC subcommittee's decision regarding ETS, we dug into the original risk assessment proceedings of the EPA. It became abundantly clear that ***the so-called "independent scientific bodies" were not independent at all.***

Rather, these groups – Scientific Advisory Boards -- were pressured by a wide variety of political and procedural forces to cast their weight (quite reluctantly in several cases) on the side of ETS as a carcinogen.

> " . . . the integrity of research sponsored by governmental or other private organizations is rarely questioned. Ignoring the possibility that the granting agencies may have specific agendas for the research they sponsor, there are substantial pressures on scientists to publish and a well-known bias against publication of negative data."
>
> Letter, *JAMA*, 1998; 280:1141

After reviewing the NTP materials forwarded to us, as well as what source documents we could acquire during the response period, ***we conclude that government and institutional bias far exceeds industry bias in the issue of ETS.***

Biological gradient (exposure or dose-response consistently exhibited over the range of the studies) is a critical factor in establishing cause and effect. There is *no clear pattern of dose/response in the majority of epidemiological studies tracking ETS and lung cancer* where quantity of exposure is measured.

We had determined that only 16.6% of the papers used in the EPA report included the odds ratios necessary to conduct a trend analysis. There was no correlation between dose increase and odds or risk increase across the range of studies. Of the 24 trend tests reported by the EPA, only 11, or 41.6% showed any evidence of upward trend – thus 58.4% of the tests for trend were non-monotonic.

Time after time we encountered actual human measurements of ETS exposure. For example: Urine cotinine measurements between ETS exposed and non-exposed women, which showed no difference.[15] Or, "in the matter of DNA adducts, personal air monitoring of carcinogenic polycyclic aromatic hydrocarbons (PAH) showed no significant difference in DNA adduct levels between non-smokers and *smokers* for RSPs of <2.5 microns after controlling for exposure to ETS via urine cotinine."[16] More recently we found that "…ETS- subjects had levels of carbon monoxide (CO) in expired air similar to that of the non-ETS nonsmokers, and significantly

[15] O'Connor TZ *et al.* Measurement of exposure to environmental tobacco smoke in pregnant women *Am J Epidemiol* 1995 Dec 15:142(12):1315-21.

[16] Binkova B *et al* DNA adducts and personal air monitoring of carcinogenic polycyclic aromatic hydrocarbons in an environmentally exposed population. *Carcinogenisis* 1995 May; 16(5):1037-46

lower than the smokers (p<= 0.05), their actual exposure (>=4 hr/day) to ETS may not have been sufficient enough to have the adverse effect."[17] While CO is not a carcinogen, it is an absolute bell cow for indoor air quality.

The fact that NTP's RoC subcommittee overlooked so much information is disturbing. It suggests extreme bias. We strongly urge that NTP's supervisory agency insist that future subcommittees employ the services of independent specialists, untainted by bureaucratic pressure, past or potential grant seeking or advocacy/activist status.

Animal studies must be put in perspective.

All substances are toxic in quantity. Many therapeutic medications are acutely toxic, but beneficial when used at a therapeutic level. Water, oxygen, and table salt are toxic in large enough doses. The mere presence of a substance does not imply toxicity.

The National Research Council and the USEPA have both recommended improvements in the risk assessment process that involve incorporating consideration of dose to the target tissue, mechanism of action, and biologically based dose-response models, including a possible threshold of dose below which effects will not occur.[18]

"Testing for carcinogenicity at near-toxic doses in rodents does not provide enough information to predict the excess number of human cancers that might occur at low-dose exposures. Testing at the maximum tolerated dose (MTD) frequently can cause chronic cell killing and consequent cell replacement (a risk factor for cancer that can be limited to high doses), and ignoring this effect in risk assessment greatly exaggerates the risks."[19]

> "...rodent bioassays provide little information about mechanisms of carcinogenesis and low-dose risk."
>
> Gold LS *et al*

Animal studies cited during the 9th RoC process (Witschi et al, various) used injectable concentrates of carcinogens, intense concentrations of tobacco smoke and skin application of tobacco smoke condensate. Not only do these studies bear no relation to inhalation of ETS; they also use concentrations that are so high that nearly any substance in these concentrations could be expected to cause deleterious effects.

In addition, Volume II of the NTP materials included animal study results (heavily relied upon according to the transcript) using concentrations of tobacco smoke that far exceed any exposure that humans could reasonably be expected to experience. Even then, despite the intense concentrations of condensates ***"Exposure to tobacco smoke had no effect on pulmonary tumor incidence or tumor multiplicity."*** Vol. II (p 47).[20]

Animal studies can be useful in identifying gross cause and effect relationships between substances and animal cancers. They should play only a minor role in the assessment of carcinogenicity in humans.

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[17] Dorsey, JL Ph.D., Meier,L B.S., and Moffatt, RJ Ph.D., M.P.H., The Effect Of Environmental Tobacco Smoke Exposure On Antioxidant Status Of Women Florida State University http://www.srnt.org/events/abstracts99/index.htm

[18] USEPA Office of Research and Development, Proposed Guidelines for Risk Assessment. Fed. Reg. 61: 17960-18011 (1996).

[19] Gold LS, Slone TH, Ames BN Overview of analyses of the Carcinogenic Potency Database. In: *Handbook of Carcinogenic Potency and Genotoxicity Databases* (LS Gold & E Zeiger, eds) Boca Raton, FL: CRC Press (1997)

[20] NTP 9th RoC/ETS materials supplied 1999.

The dose makes the poison

A basic tenet in toxicology is "the dose makes the poison." Cooking food generates thousands of chemicals.[21] There are over 1,000 chemicals reported in a cup of coffee – 19 of them are rodent carcinogens, but this does not mean that coffee is dangerous.[22] At some level, every chemical becomes toxic, but there are levels below which no adverse health effects are observed.

In addition, cancer is largely a disease of aging. Carcinogenic effects on a short-lived species such as rodents can hardly be expected to offer realistic estimates for effects in a long-lived species such as humans. High dose animal cancer testing and exaggerated risk assessment cannot be considered measures of true risks. Data on high doses in rodents simply cannot be extrapolated to low doses in humans without information on the precise mechanism of mutagenesis or carcinogenesis.

Indeed, the carcinogenic mechanisms of tobacco smoking "are not well understood."[23] What is more, cancers once "associated" with smoking are being quietly removed from the "official" lists. Stomach cancer – which is likely caused by undetected *H. pylori*, is one example; cervical cancer – in which fresh evidence suggests that human *papillomavirus* (HPV) may be the sole cause -- should soon meet the same fate. [24]

How many hundreds of millions of dollars are being wasted on senseless, useless – and quite possibly harmful -- "Blame ETS" research? How much time and talent is being diverted from useful research into chronic infections from bacteria and viruses that are major causes of cancer worldwide? How many more absurd "risk alerts" will it take before the public laughs scientists out of a position trust altogether?[25]

Even the best of epidemiological studies conclude only a very slight and easily confounded risk for *lifetime* exposure to ETS. Clearly NTP is not able to distinguish a cancer risk for the occasional exposures most of us experience during our daily activities. And just as clearly NTP must incorporate truly scientific standards and procedures before its reports deserve to be given even marginal credence.

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[21] Gold LS, Slone TH, Manley NB *et al* "Heterocyclic amines formed by cooking food: Comparison of bioassay results with other chemicals in the Carcinogenic Potency Database," *Cancer Lett.* 1994;83:21-29

[22] Gold S, Slone TH and Ames BN "Prioritization of possible carcinogenic hazards in food," in *Food chemical risk analysis,* edited by D. Tennant (Chapman & Hall Ltd. London) 1996.

[23] Ames BN, Gold LS, Willett WC. The causes and prevention of cancer. Proc Natl Acad Sci USA 1995;92:5258-65

[24] Walboomers JM *et al,* Human papillomavirus is a necessary cause of invasive cervical cancer worldwide.J Pathol 1999 Sep;189(1):12-19

[25] The carcinogenicity of ETS is not the only issue. We acknowledge that. It is, however, the sole topic of this meeting. We are increasingly skeptical of the claims made about ETS and childhood respiratory problems in light of our research into asthma. Just last week we discovered a study, presented at an international conference on indoor air pollution in Edinburgh. Researchers found that babies under six months old who were exposed on most days to air fresheners had 30 per cent more ear infections than those exposed less than once a week. Poor indoor air quality, increasingly inadequate ventilation and the growing use of perfumes, solvents and cleaners may be far greater risks than ETS.

A history of our involvement

Earlier this year, we undertook a review of the National Toxicology Program's 9th Report on Carcinogens regarding Environmental Tobacco Smoke (ETS). Our interest in ETS is an outgrowth of an ongoing project involving increased asthma rates in the U.S. What we have found is an negative correlation – smoking rates and exposure to ETS have markedly decreased while the incidence of asthma has dramatically increased.

Unconvincing background materials.

We were somewhat surprised at the background materials we received from NTP. With the exception of one study, (Bofetta *et al*., which showed a tiny and easily confounded 1.14 RR for lung cancer and ETS and *no* consistent dose-response trend) there was nothing new. The two volumes we received consisted almost exclusively of the same inadequate and tortured data used by the USEPA and CalEPA to reach the conclusion that a *lifetime* of exposure to ETS confers only a statistically marginal correlation with increased risk of lung cancer. As you know, such tiny expressions of risk are easily confounded by myriad variables.

The single animal study presented, in the words of its own authors (Witschi *et al*), "may not serve as a valid model to assess carcinogenicity in human."

RoC meeting transcript

After expressing our disappointment with these background materials, we received a verbatim transcript of the RoC subcommittee's discussion on ETS's possible listing as a human carcinogen. This was a most illuminating document. It included several informative and sound presentations from outside researchers who had made direct air measurements of ETS exposure, analyzed a variety of animal studies and assessed the raw data from several significant studies upon which the USEPA based its decision on ETS. This complete transcript is markedly different from the publicly posted, abridged version of the meeting available at the NTP website -- which completely conceals any of the twisted reasoning and political maneuvering that actually took place and ignores nearly every salient point made by the outside presenters. I will be happy to provide a copy of the entire transcript to anyone who is interested in the contorted and illogical process by which the subcommittee reached its questionable conclusions.

Unwarranted bias

During the actual meeting, subcommittee members routinely ignored convincing, well-documented presentations by outside sources – making it abundantly clear that current or past associations with industry rendered these presentations null. Several of these same subcommittee members then proceeded to base their final vote on a rather bizarre suggestion by a self-avowed anti-smoker activist (Repace) -- who proposed that a hypothetically "pure" control group be used to assess exposures. This is an appalling suggestion to those of us who understand quite clearly that case and control groups should be as *alike* as possible except for specific exposure to the substance being studied. One subcommittee member went so far as to say that she was "comforted" by this suggestion since it enabled her to vote on ETS as a carcinogen despite the fact that "relative risks in this for ETS were (from her perspective) quite low."

Another sub-committee member stated that he hoped "when we get to diesel we will get the same generous interpretation of epidemiology" enjoyed by the ETS-as-carcinogen faction. This is science? No. It is politics. And it is insupportable, unacceptable – and, quite possibly, legally actionable.

Bias and *a priori* conclusions are not sound science

In our public comments, we raised several substantive issues and found serious faults not only in the data used to reach a decision, but also with the decision making process itself. I would be pleased to provide copies of this document as well.[26] We concluded that:

> There is no convincing scientific evidence -- either epidemiologic or biologic -- that ETS is a human carcinogen.
>
> There has been and continues to be a persistent and disturbing pattern of reaching conclusions prior to rigorous scientific investigation.
>
> There has been and continues to be a pattern of 'data torturing' and scientific distortion to force an unwarranted conclusion.
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> There has been and continues to be a lack of neutrality in the formation of supposedly 'independent' scientific advisory boards convened to assess risks (or lack thereof) associated with ETS.
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> In short, we were appalled at the misrepresentation of data, willful omission of data and bias clearly displayed by the NTP subcommittee for listing ETS as a human carcinogen.

It is the result of our careful review of the NTP process regarding ETS that brings me here today – at my own time and expense. Dr. Michael R. Fox, a nationally recognized and highly respected chemist, generously assisted me in establishing and verifying real-life exposure levels for various components of ETS. I sincerely wish he were able to join us today to express his own concerns about NTP's risk assessment process.

Inadequate ventilation, not ETS, is the danger

We find continuing evidence that energy-efficient building techniques and increasingly limited ventilation – not ETS – adversely influence asthma and other upper respiratory problems – especially in children. There is an old maxim in the engineering world: "The solution to pollution is dilution." Yet building ventilation rates continue to decrease -- replaced by inadequate filtering and re-filtering systems -- and exposing and re-exposing us via re-circulated, undiluted air to contaminants strongly associated with asthma and allergies: bacteria, fungi, viruses, algae, amoebae, dust mite and cockroach feces, pollen, etc.

Since ventilation rate recommendations for buildings accommodating smokers are high enough to promote dilution of all indoor air contaminants, we have been quite careful in our assessment of risks presented by ETS. Our conclusions may be politically incorrect, but they are scientifically sound: ETS poses little if any risk to non-smokers. Indeed, it is entirely possible that buildings ventilated to a level to comfortably accommodate smokers would promote *higher* indoor air quality overall.

[26] For copies of the verbatim transcript as well as our public comments, please contact Littlewood & Fennell, 4103 Lullwood Road, Austin, TX 78722. Or by e-mail: annef@io.com. We prefer to provide electronic copies, please.

Technology, not toxic toxicology, is the solution

Rather than desperately attempting to sustain the myth that ETS is some mysteriously lethal substance, we urge an honest, open-minded look at real risk factors and real solutions for respiratory health. Rather than accepting the incremental lowering of ventilation rates for indoor air, we would insist that energy efficiency take a back seat to respiratory health.

If buildings were designed to accommodate smokers, air quality would inevitably improve for us all, since fresh air exchanges would be increased and biocontaminants such as bacteria, fungi, viruses, algae, amoebae, dust mite and cockroach feces, pollen, would be diluted. Potential toxins -- from building materials, office chemicals, cleansers, cooking, etc. could be exhausted rather than inadequately filtered and re-circulated.

NTP's Investment in Toxic Toxicology

The National Toxicology Program has become a willing and enthusiastic participant in the disturbing trend toward "Toxic Toxicology." In doing so, it undermines scientific credibility and contributes to the increasing skepticism and disdain with which the public views science in general and health warnings in particular.

NTP does so by implying that even the tiniest exposure to toxic or carcinogenic substances somehow constitutes a life threatening risk. This is patently absurd – contravening a basic tenet of toxicology: the dose makes the poison.

Animal cancer tests and worst-case risk assessment should not be considered true risks. And regulatory policy aimed at reducing minute exposures to rodent carcinogens confuses and unnecessarily alarms the public about what factors are truly important for preventing cancers.

Data torturing of epidemiological studies concerns the many responsible scientists and public health researchers who work honestly, openly and willingly in the service of sound science. We are seeing an enormous diversion of funds and talent from truly important health risks – and the real possibility that anxiety raised by false health scares is in itself a risk factor. Thus the term "Toxic Toxicology."

It is obvious that by straining at gnats, exaggerating risks and supporting researchers who are willing to do the same, the National Toxicology Program perpetuates its own existence. There is certainly nothing new about this type of metastasizing bureaucracy and empire building. What is new, is the public's increasing tendency to ignore this "sound-bite" science. Warning labels have become subjects of comics and late-night talk show hosts. Editorial cartoons poke fun at the Health Scare of the Day. Yet irresponsible risk assessment continues to be a growth industry. It is time to call a halt.

The financial and social costs of biased risk assessment are receiving increased scrutiny at both the Federal and State level. The Open Science Freedom of Information Act – making data from Federally funded studies available for independent review – is a good start. It should be followed immediately by GAO investigation of the shoddy standards and political pressures that have undermined the integrity of our nation's health research programs.