We now have direct and incontrovertible proof of deliberate scientific misconduct and concealment of crucial evidence exonerating smoking by the anti-smoking health establishment.

In May of this year, I sent in public comments on the California EPA ETS report. The authors of Chapters 3 (Gayle Windham and Mari Golub), 4 (Kirsten Waller), and 5 (Gayle Windham and Mari Golub) completely glossed over my criticisms. I sent them a copy of my article, "How the anti-smokers lie about smoking and pregnancy," as well as my comments on their report.

In particular, they ignored the solid epidemiologic evidence that the purported poor perinatal outcome in smokers is entirely attributable to a fatal methodological defect of the anti-smokers' own studies, namely their failure to detect chorioamnionitis because they failed to perform placentual pathological examinations. NONE OF THE ANTI-SMOKERS' STUDIES HAVE BEEN DONE CORRECTLY, BECAUSE OF THIS DEFICIENCY. And, because of their epidemiologic malpractice, they missed about 90% of cases, which they then proceeded to falsely blame on smoking.

Windham and Golub deliberately ignored this issue with their disingenuous response: "The document notes that at a minimum, maternal age, prior history of pregnancy loss, and socioeconomic status should be considered as potential confounders." My major point is that it has been demonstrated that these are insufficient.

And, "The relative contribution of these other confounders has not been established, but their distribution by ETS exposure status must vary in order them to confound the association. It is not clear why this would be so with these particular factors."
The relative contribution of chorioamnionitis has been established by the work of RL Naeye, and the anti-smokers have systematically concealed it.

Naeye: "We recently found no significant association between maternal smoking and either stillbirths or neonatal deaths when information about the underlying disorders, obtained from placental examinations, was incorporated into the analysis. Similar analyses found no correlation between maternal smoking and preterm birth. The most frequent initiating causes of preterm birth, stillbirth, and neonatal death are acute chorioamnionitis, disorders that produce chronic low blood flow from the uterus to the placenta, and major congenital malformations. There is no credible evidence that cigarette smoking has a role in the genesis of any of these disorders." Naeye's study population is the 56,000+ pregnancies of the Collaborative Perinatal Study. (RL Naeye. Disorders of the placenta, fetus, and neonate, diagnosis and clinical significance. New York: CV Mosby Co., 1992).

And, it is clear why the rates of chorioamnionitis would vary by ETS status: because those most exposed to passive smoke are those who are the most like smokers themselves: "Acute chorioamnionitis is the largest contributor to the poor pregnancy outcomes of black women and women who have low socioeconomic status," and it is "the most common cause of preterm labor wherever it has been studied," in the words of Naeye. (Acute chorioamnionitis and the disorders that produce placental insufficiency. In: Monographs in Pathology No.33, Pathology of Reproductive Failure. FT Kraus et al, eds. Williams and Wilkins 1991. Ch 10, pp 286-307).

The anti-smokers' scientific fraud is longstanding and systematic. NONE of the anti-smokers' studies or reviews address the role of chorioamnionitis. For example, it is entirely absent from the widely cited review by DiFranza (Effect of maternal cigarette smoking on pregnancy complications and Sudden Infant Death Syndrome. J Fam Pract 1995 Apr;40(4):385-394). Incidentally, DiFranza was on the witness list for the Full Committee hearing on Tobacco Restrictions and Youth, Sep 16, 1997, by Sen. John McCain. And, DiFranza ought to be interrogated about why his supposedly exhaustive research of the literature failed to turn up any work by Naeye dating after 1976.

In fact, the anti-smokers have deliberately conducted defective studies for nearly 40 years, ignoring the early work by WA Blanc which demonstrated the necessity of placental examinations (Amniotic infection syndrome. Pathogenesis, morphology, and significance in circumnatal mortality. Clin Obstet Gynecol 1959;2:705).

Windham and Golub have committed an additional element of fraud by falsely attributing perinatal illness and death to simple "low birthweight," which are actually healthy births.

Their central premise that "if the distribution of birthweight is shifted lower with ETS exposure, as it appears to be with active smoking, infants who are already compromised may be pushed into even higher risk categories. Low birthweight is associated with many well-recognized problems for infants, and is strongly associated with perinatal mortality," is bogus.

Low birth weight in the absence of chorioamnionitis or other actual disease, is of negligible clinical significance. THE KEY FACTOR IS THE PRESENCE OR ABSENCE OF DISEASE, specifically of chorioamnionitis, and statistical sleights of hand purporting to predict illness by shifting the means of bell curves are pure deceit.

It is the purpose of REAL science to distinguish false and artificial associations from genuine ones, but Windham and Golub, along with their fellow anti-smokers, have deliberately obscured and confounded these distinctions, in order to make malicious and unfounded accusations against smoking. And, the authors refused to acknowledge the evidence or address the points when these were directly presented to them, as well as continuing the ongoing conspiracy of silence about RL Naeye's work.

Elsewhere, Windham and Golub made no mention of the correction I sent in to change the words "vitamin E" to "folic acid." Nor did they mention my summary of studies of active smoking and neural tube defects, which, even in the absence of controlling for folic acid in any of them, showed no relation between active smoking and neural tube defects.

And, in other responses, Windham and Golub simply brushed off my criticisms that the existing evidence
was deficient.

For example, Helicobacter pylori infection in children, besides reducing adult height by slowing the growth spurt, could well affect fetal growth as well. But studies of this have not been done, and should be, before any conclusions are drawn. Windham and Golub merely offered an evasive, non-scientific argument from intimidation that "The evidence for an effect of maternal smoking is not detailed in this document, but it has been clearly shown in numerous studies and is accepted by medical experts." Those studies are not reputable when they leave out important factors, and the so-called "experts" embrace them out of nothing but malice.

Physical activity and violence during pregnancy are issues which should be addressed in particular for spontaneous abortion. Again, such studies have not been performed, and should be, before any conclusions are drawn.

Windham and Golub just blew them off under the same evasive response they gave for perinatal mortality: "The document notes that at a minimum, maternal age, prior history of pregnancy loss, and socioeconomic status should be considered as potential confounders. The relative contribution of these other confounders has not been established, but their distribution by ETS exposure status must vary in order them to confound the association." [Meaning, "what we don't know we don't care about, because we want to find smoking guilty."] It is not clear why this would be so with these particular factors [when socioeconomic confounding is a clear probability].

In Chapter 4, Kirsten Waller ignored my criticism that SIDS, by definition, is not a disease; it is merely a catchall for any unknown cause of death. As such, it is extremely vulnerable to confounding by uninvestigated socioeconomic factors. Like Windham and Golub, she clearly views it as acceptable to recklessly exploit confounding factors, in flagrant disregard for the requirements of good science.

Waller also refused to address the issue that Helicobacter pylori infection in children reduces adult height by slowing the growth spurt; and that more studies should therefore be done.

And, in Chapter 5, Windham and Golub summarized my objection that this report ignored its own statement that "covariates related to sexual practices are important to consider, including frequency of coitus,..." by relying on defective studies that did not consider those covariates to falsely suggest that passive smoking reduces fertility in women. And then, just as they did in their report, they simply ignored it!

They deliberately concealed the evidence I submitted from RL Naeye's study, which unlike their studies DID consider frequency of coitus, and which consequently exonerated active smoking. "However, this association completely disappeared when confounding risk factors were taken into consideration. The two confounding risk factors that were responsible for the delay that smokers experienced in becoming pregnant were being over 34 years in age and having blue-collar employment outside of the home."

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Carol Thompson 12-15-97
Smokers' Rights Action Group
P.O. Box 259575
Madison, WI 53725-9575
608-249-4568
71334.3541@compuserve.com

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