Changing Conclusions on Secondhand Smoke in a Sudden Infant Death Syndrome Review Funded by the Tobacco Industry

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ABSTRACT. Background. Prenatal and postnatal exposure to tobacco smoke adversely affects maternal and child health. Secondhand smoke (SHS) has been linked causally with sudden infant death syndrome (SIDS) in major health reports. In 1992, the US Environmental Protection Agency (EPA) first noted an association between SHS and SIDS, and both prenatal exposure and postnatal SHS exposure were listed as independent risk factors for SIDS in a 1997 California EPA report (republished in 1999 by the National Cancer Institute) and a 2004 US Surgeon General report.

The tobacco industry has used scientific consultants to attack the evidence that SHS causes disease, most often lung cancer. Little is known about the industry’s strategies to contest the evidence on maternal and child health. In 2001, a review was published on SIDS that acknowledged funding from the Philip Morris (PM) tobacco company. Tobacco industry documents related to this review were examined to identify the company’s influence on the content and conclusions of this review.

Methods. Tobacco industry documents include 40 million pages of internal memos and reports made available to the public as a result of litigation settlements against the tobacco industry in the United States. Between November 2003 and January 2004, we searched tobacco industry document Internet sites from the University of California Legacy Tobacco Documents Library and the Tobacco Documents Online website. Key terms included “SIDS” and names of key persons. Two authors conducted independent searches with similar key terms, reviewed the documents, and agreed on relevancy through consensus. Thirty documents were identified as relevant. Two drafts (an early version and a final version) of an industry-funded review article on SIDS were identified, and 2 authors independently compared these drafts with the final publication. Formal comments by PM executives made in response to the first draft were also reviewed. We used Science Citation Index in July 2004 to determine citation patterns for the referenced SIDS reviews.

Results. PM executives feared that SHS and maternal and child health issues would create a powerful and emotional impetus for smoke-free areas in the home, public areas, and the workplace. In response to the 1992 US EPA report on SHS, the Science and Technology Department of PM’s Switzerland subsidiary, Fabriques de Tabac Reunies, searched for “independent” consultants to publish articles addressing SHS. The first industry-funded article was a literature review focusing on smoking and SIDS, conducted by consultant Peter Lee and co-author Allison Thornton, which stated that the association between parental smoking and SIDS could have been attributable to the failure to control fully for confounders. That first review has only been cited once, in the subsequent industry-funded review.

In 1997, PM commissioned a consultant, Frank Sullivan, to write a review, with coauthor Susan Barlow, of all possible risk factors for SIDS. The first draft concluded that prenatal and postnatal smoking exposures are both independent risk factors for SIDS. After receiving comments and meeting with PM scientific executives, Sullivan changed his original conclusions on smoking and SIDS. The final draft was changed to emphasize the effects of prenatal maternal smoking and to conclude that postnatal SHS effects were “less well established.” Changes in the draft to support this new conclusion included descriptions of Peter Lee’s industry-funded review, a 1999 negative but underpowered study of SIDS risk and urinary cotinine levels, and criticisms of the conclusions of the National Cancer Institute report that SHS was causally associated with SIDS. In April 2001, the Sullivan review was published in the United Kingdom journal Paediatric and Perinatal Epidemiology, with a disclosure statement that acknowledged financial support from PM but did not acknowledge contributions from PM executives in the preparation of the review. By 2004, the Sullivan SIDS review had been cited at least 19 times in the medical literature.

Conclusions. PM executives responded to corporate concerns about the possible adverse effects of SHS on maternal and child health by commissioning consultants to write review articles for publication in the medical literature. PM executives successfully encouraged one author to change his original conclusion that SHS is an independent risk factor for SIDS to state that the role of SHS is “less well established.” These statements are consistent with PM’s corporate position that active smoking causes disease but only public health officials conclude the same for SHS. The author’s disclosure of industry funding did not reveal the full extent of PM’s involvement in shaping the content of the article. This analysis suggests that accepting tobacco industry funds can disrupt the integrity of the scientific process.

The background of this SIDS review is relevant for institutions engaged in the debate about accepting or eschewing funding from the tobacco industry. Those who support acceptance of tobacco industry funds argue that academic authors retain the right to publish their work and maintain final approval of the written product.
but this argument fails to recognize that the tobacco industry funds work to ensure that messages favorable to the industry are published and disseminated.

Clinicians, parents, and public health officials are most vulnerable to the changed conclusions of the SIDS review. The national SIDS “Back to Sleep” campaign has been very successful in reducing SIDS rates. However, estimates of SIDS risk from SHS (odds ratios range from 1.4 to 5.1) have considerable overlap with estimates of risk from prone sleep positioning (odds ratios range from 1.7 to 12.9). With the Back to Sleep campaign well underway, efforts to address parental smoking behavior in both the prenatal and postnatal periods should be intensified. The tobacco industry’s disinformation campaign on SHS and maternal and child health can be countered within clinicians’ offices. Pediatrics 2005;115:e356-e366. URL: www.pediatrics.org/cgi/doi/10.1542/peds.2004-1922; secondhand smoke, tobacco industry, sudden infant death syndrome.

**ABBREVIATIONS**. SIDS, sudden infant death syndrome; EPA, Environmental Protection Agency; SHS, secondhand smoke; PM, Philip Morris; FTR, Fabriques de Tabac Reunies; NCI, National Cancer Institute; ETS, environmental tobacco smoke.

Prenatal and postnatal exposure to tobacco smoke adversely affects maternal and infant health. Adverse outcomes that have been causally associated with prenatal tobacco exposure include premature rupture of membranes, preterm delivery, fetal growth restriction, and impaired pulmonary function among infants. Exposure of infants and children to secondhand smoke (SHS) has also been associated with a number of adverse health outcomes, including induction and exacerbation of asthma, otitis media, and sudden infant death syndrome (SIDS). National estimates of annual pediatric morbidity and mortality rates associated with SHS exposure include 1900 to 2700 SIDS deaths, 0.7 to 1.6 million office visits for treatment of otitis media, 8000 to 26 000 new cases of asthma, 400 000 to 1 000 000 asthma exacerbations, and 150 000 to 300 000 cases of bronchitis or pneumonia among infants and toddlers.

SHS has been causally linked with SIDS in a number of major health reports. The landmark 1992 report from the US Environmental Protection Agency (EPA) not only confirmed that SHS causes lung cancer among nonsmokers but also noted an association between SHS exposure and an increased risk of SIDS. Subsequent research on SIDS and smoking explored the roles of in utero exposure (prenatal), exposure to SHS after birth (postnatal), or both. In 1997, the California EPA concluded that both prenatal exposure and postnatal SHS exposure are independent risk factors for SIDS. The California EPA report was republished by the National Cancer Institute (NCI) as part of its Smoking and Health Monograph Series in 1999, with a preface by the US Surgeon General endorsing the report’s conclusions. In 1999, a World Health Organization report called for the elimination of exposure to tobacco smoke in utero and exposure to SHS in childhood. Most recently, the 2004 Surgeon General’s report on smoking concluded that there is a causal relationship between SIDS and maternal smoking during and after pregnancy.

The tobacco industry has fought to counteract the scientific consensus that SHS is dangerous since the first evidence that SHS causes disease began to emerge in the 1970s. In 1988, Philip Morris (PM), working with other multinational tobacco companies, developed an International Environmental Tobacco Smoke (ETS) Consultant Program to “keep the controversy alive” on SHS by recruiting consultants to develop and promote scientific viewpoints favorable to the industry. The industry’s law firm, Covington and Burling, managed the consultants’ work for the tobacco industry so that the consultants’ relationship with the industry could be denied or downplayed, in effect allowing consultants to be portrayed as independent third parties. The industry commissioned reports and implemented strategies to confuse or undercut the mainstream scientific literature on SHS, particularly the evidence on lung cancer. Little is known about the tobacco industry’s influence on maternal and child health issues.

In 2001, a scientific review of SIDS was published that acknowledged funding from PM. We identified and compared 2 drafts of the review in previously secret, internal tobacco industry documents that have been made available as a result of litigation against the industry, and we noted changes made after communications between the PM scientific executives and the authors. In response to the suggestions of PM, the authors removed SHS from their original conclusion that both prenatal exposure and postnatal exposure to tobacco smoke are independent risk factors for SIDS. This industry-commissioned article may undermine efforts by clinicians, parents, and public health officials to promote the elimination of infant exposure to SHS as an important prevention strategy for SIDS.

**METHODS**

Tobacco industry documents include 40 million pages of internal memos and reports made public as a result of litigation settlements against the tobacco industry in the United States. Between November 2003 and January 2004, we searched tobacco industry documents from the University of California, San Francisco, Legacy Tobacco Documents Internet site (www.tobaccohistory.ucsf.edu) and the Tobacco Documents Online web site (www.tobaccohistory.ucsf.edu). Two of the authors independently searched the documents and agreed on relevance through consensus. We determined that 30 documents were relevant, in that they provided either contextual information on PM’s interest in SIDS or were related to the commissioning and writing of the SIDS review article.

Two drafts, namely, an early version and a final version, of a SIDS review by Frank Sullivan and Sue Barlow were found in the documents. The contents of these 2 drafts were compared with each other and with the final published version, and changes in the text were noted. Formal comments by PM executives made in response to the first draft and submitted to Frank Sullivan were also reviewed. We used Science Citation Index in July 2004 to determine citation patterns for the referenced SIDS reviews.
RESULTS

PM Searches for Consultants on SIDS

The PM subsidiary Fabriques de Tabac Reunies (FTR) is located in Neuchatel, Switzerland, and has a Science and Technology Department (later renamed Scientific Affairs). The Science and Technology Department’s 1993 mission for SHS was to support PM’s corporate goals, ie, “S&T [Science and Technology] Mission: To recommend, seek approval and implement strategies for dealing with the ETS, and where appropriate, the Primary issues in support of PM global and regional business.” In 1993, Lee charged the United Kingdom Tobacco Manufacturer’s Association Advisory Council £12 000 for reviews on SHS and SIDS, childhood cancer, and middle ear disease. In February 1997, PM paid Lee at least $2120 to publish his SIDS article.

However, Lee and Thornton initially encountered difficulties. In August 1997, Lee reported to the FTR Science and Technology Department that he had submitted the article to Public Health Reviews but “they had rejected our SIDS paper ‘considering it not sufficiently unbiased.’ Because of the general difficulty of “getting tobacco industry funded studies published,” Lee suggested sending the [manuscript] to a more minor journal which is more likely to give acceptance. One possibility is Indoor and Built Environment [a journal set up by PM consultants], edited by [tobacco industry consultant] John Hoskins, whom I know, which has published review papers on ETS-related issues in the past.

In 1998, Indoor and Built Environment published the Thornton and Lee article, which acknowledged that “support was provided by several companies of the tobacco industry.” Other articles by Thornton and Lee were also published in Indoor and Built Environment. The SIDS review by Thornton and Lee has had little direct impact on the medical literature. As of July 2004, it had been cited only once, and that citation was by the next consultant hired by PM to write about SIDS, Frank Sullivan.

PM Commissions Toxicologist Frank Sullivan

In February 1997, as the California EPA presented its draft SHS report for public comment, PM began its search for a consultant to review the literature on SHS and SIDS. An attorney at Covington and Burling notified PM executives that the California EPA was moving toward concluding that there was a causal relationship between SHS and SIDS and that there was a “need to continue SA’s [PM Scientific Affairs] ongoing work in this area.” Covington and Burling sent the curriculum vitae of toxicologist consultant Frank Sullivan to Ted Sanders (FTR Scientific Affairs) and asked how PM would like to proceed.

Sanders expressed reservations about Sullivan’s qualifications to Richard Carchman (director of PM Scientific Affairs), because Sullivan had no prior experience in SIDS research. Previously, Sullivan had served in multiple other capacities as an industry consultant. He was listed in 1988 as one of the industry’s first potential “international ETS consultants,” and he participated in tobacco industry activities downplaying the health effects of SHS.

In March 1997, Sanders reported to Carchman that Sullivan had expressed an interest in conducting the SIDS review. PM and Sullivan agreed that literature searches would be supplied by PM and “[w]ork would be reviewed by the appropriate PM personnel on a periodic basis and the final manuscript would also be reviewed. However, Dr Sullivan would re-
main free to publish the final version without restriction by Philip Morris." They also agreed that “[a]ppropriate attribution for PM’s support would be made,” which in the published article was described as only financial support. In 1998, PM budgeted $50,000 to $100,000 for completion of Sullivan’s review.

The Sullivan review article was part of PM’s overall scientific strategic plan for addressing SHS and childhood health issues. In January 1998, PM Worldwide Scientific Affairs described in an “Impact Assessment” how Sullivan’s review would meet PM’s goals: “a thorough analysis of all of the literature, not simply a review of those studies which implicate ETS . . . in order to determine if it is likely that ETS truly does play a role in the etiology of SIDS.” Another Worldwide Scientific Affairs project was to review the literature on SHS in childhood and organize a database of datasets for future statistical analysis. The “Impact Assessment” for this project was as follows: “Should provide the necessary scientific background for a policy on the acceptability of smoking around children.” In 1999, PM Worldwide Scientific Affairs listed the scientific issues of greatest importance to the company and described strategies to develop reviews for establishing PM’s scientific positions. “ETS and Health” was listed as one such priority issue, with Sullivan’s review being noted as already covering SIDS. Besides SIDS and adult diseases such as lung cancer and cardiovascular diseases, other topics were being internally reviewed, including childhood respiratory disease and asthma. Otitis media had been covered previously by the publication by Thornton and Lee.

Sullivan and PM Work Together

Sullivan worked on the SIDS review from 1997 to 2000, with his coauthor Sue Barlow; Sullivan was the direct link to PM’s scientific executives. In July 1998, Sullivan wrote to Sanders that he was nearly finished with the review except for the smoking section, and the 2 met soon after. Sullivan’s initial draft was 101 pages long and reviewed 20 risk factors for SIDS. Later, in June 1999, Sanders apologized to Sullivan, as he had “just not had the opportunity to complete my work on your draft [emphasis added]” and provided detailed comments from himself and Carchman (Table 1). Regarding the summary and conclusions section, Sanders’ only comment on Sullivan’s original conclusions was in disagreement with the role of SHS: “I would like to discuss some of the wording on this page with you. It seems that you have been less cautious in interpreting the data on postnatal ETS exposure than you were with prone sleeping position [emphasis added].” Sullivan and Sanders again planned a London meeting, after which Sanders sent a July 1999 memo to tobacco industry consultant Peter Lee, requesting that Lee analyze certain sections of Sullivan’s draft. Sanders passed on Lee’s commentary to Sullivan.

In January 2000, Sullivan wrote to Sanders that he had incorporated PM’s suggestions for the final version and changed the smoking portion of the summary and conclusions section (Table 2). Sullivan’s final draft included several additions and revisions that downplayed the role of smoking and especially SHS. In the revision, Sullivan removed his original conclusion that both prenatal and postnatal effects of smoking are independent risk factors for SIDS. Instead, Sullivan concluded that postnatal SHS effects were “less well established” than prenatal smoking (described below and in Tables 1 and 2).

In March 2000, Sullivan wrote to Sanders, thanking him for his “very detailed comments on the SIDS report” and reporting that “all your comments have been incorporated except for one [comment not identified].” Sullivan also stated that he had communicated with the editor of the United Kingdom journal Paediatric and Perinatal Epidemiology about the review article and had received an enthusiastic response.

The SIDS review was published in April 2001. The disclosure statement in the final publication stated, “We are grateful to Fabriques de Tabac Reunies SA (Philip Morris), Neuchatel, Switzerland, for financial support in the preparation of this review. The opinions expressed are entirely those of the authors.”

There is no indication in the acknowledgment that PM was involved in preparing the manuscript.

Comparison of Sullivan’s Drafts

PM’s Carchman had approved the comprehensive nature of Sullivan’s initial draft in 1999. Handwritten at the end of this draft, Carchman summarized his suggestions for the review.

Overall—excellent job
—complete data capture
—I would like [sic] some more creativity—bottle/breast—age—immune infection [emphasis added]

Sullivan’s draft discussed 20 categories of SIDS risk factors. Despite Carchman’s desire for “more creativity,” Sullivan made few changes to these sections. However, in response to specific PM comments on smoking, Sullivan incorporated some suggestions, such as noting that anemia among smoking mothers who have SIDS infants might suggest “other underlying problems” of the mother (Table 1).

The comments solicited from Lee by PM were incorporated into the manuscript to downplay population attributable risks for maternal smoking in SIDS. Sullivan had described a meta-analysis of 11 studies by DiFranza and Lew and its calculation of population attributable risks for maternal smoking, but Sanders wrote to Sullivan, “Dr Carchman wondered if you could provide some individual critical analysis of the population attributable risks provided in this paragraph.” Subsequently, Sanders asked Lee for comments, which Sanders forwarded to Sullivan. Lee stated that the attributable risk from maternal smoking could be explained mostly by confounding. Sullivan revised his final version to include Lee’s comments (Table 1), but the comments were not attributed to Lee and Lee’s contributions to the article were not acknowledged. Moreover, Sullivan deleted a sentence in his conclusion stating that the population attributable risk of maternal smoking was estimated to be between 27 and 66% (Table 1).
TABLE 1. Highlights of Sullivan’s Draft Alterations in Response to PM’s Suggestions

| Page 45, paragraph 1: Dr. Carchman wondered if you could provide some individual critical analysis of the population attributable risks provided in this paragraph. | A meta-analysis of 11 studies published 1966 and 1992...estimated that the population attributable risk for maternal smoking probably ranged from 22–41%, assuming the prevalence of maternal smoking in these populations ranged from 18–27% (DiFranza and Lew, 1995). These estimates of population attributable risks are however open to question, since in many of the studies used for analysis the risk estimates were not controlled for potential confounding variables, which have already been shown to affect the risk estimates dramatically. Postnatal maternal smoking was also significantly associated with an increased risk of SIDS. Only 9 of the 39 studies specifically recorded postnatal maternal smoking habits and were thus suitable for meta-analysis. Of the 9 studies suitable for meta-analysis, the unadjusted pooled estimate for the OR was 2.80 (95% CI 2.00 to 3.93). Eight of the 9 studies which presented data on postnatal maternal smoking also included data on prenatal maternal smoking; of these, 5 reported a greater effect for postnatal maternal smoking than for prenatal maternal smoking, while 3 reported a greater effect of prenatal smoking. Four studies controlled for maternal prenatal smoking, enabling the contribution of postnatal maternal smoking to be estimated; the adjusted pooled estimate for this OR was 1.94 (95% CI 1.55 to 2.43). These results suggest separate associations between smoking and SIDS both for smoking during pregnancy and for smoking after birth, both of which are independent of birthweight. A further study mentioned in the analysis by Anderson and Cook found the effect of postnatal smoking was not significant after adjustment for prenatal smoking but no estimate of the OR was provided (Blari et al, 1996). It is unusual for a mother who smokes postnatally not to have smoked prenatally, so the [Authors’ note: “the although there is inserted into final publication] evidence that postnatal smoke exposure alone is an independent risk factor for SIDS is weak [Authors’ note: “is weak this has been difficult to quantify” inserted into final publication].

| Page 45, paragraph 4: Here again, it is probably even more important to provide information on why only 9 studies were suitable for meta-analysis. | The US National Institute of Child Health and Human Development SIDS Cooperative Epidemiological Study, however, found no dose-response relationship (Hoffman et al, 1988a)...Numbers of cigarettes smoked/day during the three trimesters of pregnancy was obtained by interview 2–5 weeks after the infant’s death. This could have biased recall in mothers of SIDS cases towards understating cigarette consumption. Deficiencies in Ventilatory Responses to Hypoxia: Chronic fetal hypoxia caused by nicotine-induced reduction in uteroplacental blood flow and elevated fetal carboxyhaemoglobin levels, leading in turn to in [sic] impaired development of the central nervous system, including the cardio-respiratory control system, have been proposed as a mechanism to explain the effect of maternal smoking during pregnancy (Bulterys et al, 1990; Lewis and Bosque, 1995). Evidence of prenatal hypoxia comes from observations of elevated haematocrit values in the cord blood of infants of smoking mothers. (reviewed by Bulterys et al, 1990). Maternal Anaemia: Stewart et al (1995b) found a slightly increased risk of SIDS in association with anaemia...but this disappeared on multivariate analysis controlling for a large number of potential confounders. The authors went on to examine the combined effect of maternal anaemia and smoking and claim to have found no interaction. However, after multivariate analysis in women smoking >20 cigarettes/day, ORs were 3.45 (95% CI 1.82 to 6.70) for anaemic women, compared with 2.04 (95% CI 1.22 to 3.43) for non-anaemic women. These results suggest there may be an interaction with anaemia in heavy smokers. However, such findings should be interpreted with caution because smoking normally causes a modest increase in red cell mass (Smith and Landaw, 1978), suggesting that mothers who smoke and are anaemic may have other underlying problems.

| Page 49, paragraph 1: The statement is made that, “This could have biased recall in mothers in SIDS cases towards understating cigarette consumption.” Is this your statement or the authors’? If it is the authors’ statement, it might be well to say so. If it is your statement, I am not in agreement with it. | Sullivan’s original draft did not include the 1998 SIDS article by Thornton and Lee, but the final draft incorporated a descriptive paragraph about the article, stating that much of the association between tobacco exposure and SIDS appeared to be attributable to confounding (Table 3). The article by Thornton and Lee stated that the relative risks from SHS could be reduced with multiple statistical adjustments, which suggests that parental smoking and SIDS are not independently associated. In making this argument, however, Sullivan ignored the fact that, whereas proper adjustment for confounders...
generates a more accurate estimate of odds ratios, including excess adjustments can lead to an over-specified model and mask real associations by diluting their statistical significance.\textsuperscript{35}

Summary and Conclusions: In considering modifiable maternal risk factors, smoking is among the most important. In the majority of the many studies that have examined smoking as a risk factor, consistent associations have been found between SIDS and maternal smoking, whether smoking habits were ascertained prenatally or postnata...
TABLE 2. Other Changes Downplaying Postnatal Effects Added by Sullivan

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<tr>
<th>Sullivan Cover Letter52 to PM for Final Draft</th>
<th>Sullivan’s Final Draft18*</th>
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<td>We [Sullivan and Barlow] have considerably revised and updated the smoking section . . . and have taken account of all the comments at our last meeting. I have also revised the smoking section in the Summary and Conclusions section to put more emphasis on the probable prenatal effect of smoking, and less on the ETS aspect, which better reflects our opinion of the data.</td>
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<td>Dose Response: Demonstrations of increasing risk a close association with increases in measures which attempt to estimate the degree of exposure of the infant, in utero or postnatally, between increased exposure to smoke products and risk of SIDS would contribute evidence of a causal, biological link. Summary and Conclusions: The relative lack of unexplained deaths during the first month of life suggests that prenatal factors alone are insufficient to cause SIDS and points to a possible pivotal role for postnatal factors interacting with some minor prenatal developmental anomaly, as during the rapid developmental changes that occur in infancy. Prenatal factors, such as low birth weight and maternal smoking during pregnancy, for example, undoubtedly influence the risk of SIDS. However, their influence seemingly does not become evident until well after the neonatal period, when some other compromising situation occurs, either of an external nature, such as sleeping in the prone position, and/or of an internal nature, such as occurs which coupled with an inadequate physiological response leads to a potentially life-threatening situation. In conclusion, the overall picture which emerges from this review of the factors involved in SIDS is that the affected babies are not completely normal in development, but possess some inherent weakness which may only become obvious when the infant is subjected to an unusual stress. Initially, there may be some minor impairment or delay in development of respiratory or cardiovascular regulatory centres in the brain or in the carotid body chemoreceptor mechanism.</td>
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* Additions are underlined. The deletions from the initial draft17 are shown as strikethrough text.

tually quite good and critical but there is a complete disconnection when they write the conclusion. . .I have expressed my feelings in our review. 52 In the final draft and publication (Table 3), Sullivan criticized the NCI report, “The NCI report concludes . . . that environmental tobacco smoke is an independent risk factor for SIDS. This conclusion seems rather surprising in the light of the critical discussion of individual studies.”18 Sullivan added criticisms that prenatal and postnatal smoking effects were too difficult to separate.

Two additional paragraphs were added to describe a 1999 Tasmanian prospective study of SIDS and cotinine (a nicotine metabolite) by Dwyer et al.56 with an accompanying editorial by Spiers,57 in which sources of SHS exposure at 1 month of age and subsequent risks of SIDS were examined. Sanders wrote to Sullivan, “I think that this paper is extremely important, and I also think that if at all possible it ought to be included in your review.”47 The study by Dwyer et al.56 in which parental smoking patterns predicted urinary cotinine concentrations among the infants but not the risks of SIDS, included only 34 cases. Nevertheless, Sullivan twice emphasized the article’s negative but underpowered findings as providing no evidence of an independent postnatal effect of smoking (Table 3). The accompanying commentary by Spiers57 discussed the difficulty of separating prenatal and postnatal smoking effects and debated the relative contributions and mechanisms of each effect. However, Sullivan cited only Spiers’ comments on the possible mechanisms for a prenatal effect (Table 3). (No documents connect Dwyer or Spiers personally with PM.)

In the summary and conclusions section of the final draft,18 Sullivan made a number of revisions that served to downplay the potential effects of SHS on the risks of SIDS. He deleted his original conclusion that SHS is an independent risk factor for SIDS and replaced it with statements emphasizing the prenatal effects of smoking (Tables 1 and 2). He deleted the sentence, “The evidence overall suggests that both prenatal and postnatal maternal smoking exert independent effects [emphasis added].” He also changed the statement, “Consistent associations have been found between SIDS and maternal smoking, whether smoking habits were ascertained prenatally or postnata ially,” by deleting the last phrase. He added the conclusion, “In considering modifiable maternal risk factors, smoking is among the most important,” but he indicated that this does not necessarily include postnatal smoking exposure and inserted criticisms of studies of postnatal SHS exposure several times in the body of his article (Tables 1 and 2). He also expanded the summary and conclusions section by almost a whole paragraph to emphasize more strongly the effects of prenatal exposure on fetal development, stating that postnatal effects were “less well established” (Table 1).18 In summary, Sullivan substantially altered his article to focus more on abnormal infant development and the role of prenatal maternal smoking, while raising doubts about the certainty of SHS as a risk factor for SIDS.
TABLE 3. New Material Added by Sullivan to Support the New Conclusions

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<th>New Sources Described</th>
<th>Sullivan’s Final Draft*</th>
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<td>Thornton and Lee’s56 review of SIDS and smoking</td>
<td>A detailed review of all of the major studies of parental smoking and SIDS has been published by Thornton and Lee (1998). The major findings of the analyses were that for maternal smoking during pregnancy, 28 of the 29 unadjusted relative risks were above 1.00 and all but 2 were statistically significant. Adjusted relative risks, when presented, were also significantly raised, but less so. In general, the more potential confounders controlled for, the greater was the reduction in risk estimate. In two of the relatively large studies (Blair et al, 1996; Mitchell et al, 1993), adjustment was made for 13 factors in each, and the reductions in excess relative risk estimate compared with unadjusted risks were 80% (4.84 to 1.78) and 79% (4.09 to 1.65), which suggests that much of the excess risk of SIDS associated with maternal smoking during pregnancy may be due to correlation of maternal smoking with other risk factors. Eight studies investigated the risks of paternal smoking, of which 6 showed significantly raised risk estimates, which were also reduced by adjustment. In one of the studies (Klonoff-Cohen et al, 1995) the relative risk when the father smoked was reported to be higher than when the mother smoked, which seems unlikely and contrary to all the other studies.</td>
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<td>NCI republication* of California EPA Report</td>
<td>This report reviewed only 10 studies on SIDS performed up to 1996 and these have already been discussed above. A critical review of each study is presented which emphasizes the difficulties of attempting to separate prenatally induced effects of maternal smoking from effects induced postnatally from ETS. The major problem as mentioned above was the fact that most mothers who smoke do so both during the pre- and the postnatal period, and the danger of drawing any conclusions based on the relatively few women who smoke only during, or only after pregnancy. Much of the evidence for a role of postnatal ETS exposure comes from studies in which the mother does not smoke but the other household members do. The evidence for an effect of paternal smoking when the mother does not smoke, however, much less consistent. The NCI Report concludes, based mainly on the results of the Klonoff-Cohen et al (1995) and Blair et al (1996) studies, that environmental tobacco smoke is an independent risk factor for SIDS. This conclusion seems rather surprising in the light of the critical discussion of the individual studies. The high correlation in mothers between smoking during pregnancy and smoking after pregnancy reduces the reliance on the maternal data so far as a role for ETS is concerned.</td>
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<tr>
<td>Dwyer study56 (in two separate sections)</td>
<td>Information on Maternal Smoking During Pregnancy and Exposure to Environmental Tobacco Smoke after Birth: In the Tasmanian case-control study (Ponsonby et al, 1995), SIDS was significantly related to maternal smoking after birth (OR 3.96, 95% CI 1.91–8.24) but smoking by other household residents was not (OR 1.31, 95% CI 0.70–2.44). It should be noted that, as with many other studies, maternal smoking after birth could not be separated from maternal smoking during pregnancy because smoking behaviour is generally similar before and after birth. Another recent prospective study in Tasmania (Dwyer et al, 1999) has used interview data and infant urinary cotinine levels at one month of age to assess relationships between subsequent SIDS (53 cases) [sic and authors’ note: only 34 cases were used for SIDS risk estimates] and sources of postnatal exposure to tobacco smoke. Postnatal smoking was dose-related (1–10 cigarettes/day, adjusted OR 2.08, 95% CI 0.79–5.48; 11–20 cigarettes/day, adjusted OR 2.15, 95% CI 0.85–5.47; &gt;20 cigarettes/day, adjusted OR 4.69, 95% CI 1.74–12.58). However, smoking by other residents had no effect on the risk for SIDS, with or without adjustment for maternal postnatal smoking. The strongest predictor of urinary cotinine levels and of SIDS was maternal smoking (prenatal and postnatal not separable), but particular smoking behaviours, such as mother not smoking in the room with the baby and presence of other smokers, were discordant with respect to cotinine levels and prediction of SIDS. This study, while of limited power, provides no evidence that sources of postnatal exposure to tobacco smoke other than maternal smoking, were associated with SIDS. Nicotine Mechanism: Urinary cotinine levels in infants at risk for SIDS were measured in the Tasmanian Infant Health Survey (Dwyer et al, 1999) when the infants were 4–5 weeks of age, in an attempt to identify the sources of postnatal exposure to tobacco smoke, and the relation to SIDS. The strongest predictor of both cotinine and SIDS was maternal smoking. The effects of pre- and postnatal smoking could not be separated (almost all smoking mothers smoked both during and after pregnancy). However, if the mother did not smoke in the same room as the baby, the cotinine level in the infants’ urine was more than halved, but this was not associated with a reduced risk of SIDS. Similarly, the presence of other adult smokers in the home was associated with a 63% increase in urinary cotinine, but not with an increase in SIDS. Although the power of the study was inadequate to detect changes in risk of less than two-fold, the authors support the suggestion that that [sic] the major effect of smoking in relation to SIDS, is a prenatal one, and not a postnatal one. The possible mechanisms involved in a prenatal effect have also been discussed by Spiers (1999) who contrasts the relative unimportance of low birthweight in SIDS deaths, compared with non-SIDS infant deaths.</td>
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* Italics were added for emphasis. OR indicates odds ratio; CI, confidence interval. Authors’ notes in brackets reflect comments of this article’s authors, not those of Frank Sullivan.
DISCUSSION
The tobacco industry has long fought to counteract scientific evidence that SHS is dangerous to health. Through examination of industry documents, we conclude that PM executives responded to company concerns about the possible adverse effects of SHS on maternal and child health by commissioning and influencing a review article on SIDS. The final publication included recognition that maternal smoking during pregnancy is a risk factor for SIDS, but the author’s original conclusion regarding SHS was changed to state that the role of SHS is “less well established.” These statements are consistent with PM’s corporate position that active smoking causes disease but only public health officials conclude the same for SHS. The 4-line disclosure statement in the published article acknowledges financial support from PM but does not describe how PM’s involvement with the author helped shape the content of the review. Moreover, the disclosure does not reflect PM’s motivating concern that “there is perhaps no other issue as powerful facing the industry” as SHS and maternal and child health issues. Three years after its publication, the SIDS review by Sullivan and Barlow had been cited at least 19 times in the medical literature and in an Institute of Medicine report on SIDS. This citation pattern suggests that PM has succeeded in creating a review that some see as authoritative and credible and that has substantial potential to influence clinical practice and public policy.

Our analysis of the Sullivan SIDS review is relevant for institutions engaged in the debate regarding accepting or eschewing funding from the tobacco industry. Those who support acceptance of tobacco industry funds argue that academic authors retain the right to publish their work and maintain final approval of the written product, but this argument fails to recognize that the tobacco industry funds work to ensure that messages favorable to the industry are published and disseminated. For example, in 1988, PM led other tobacco companies in designing and controlling an airline indoor air quality study that downplayed exposure to SHS and its potential health effects when smoking restrictions on airlines were being debated. In 1995, the industry generated and controlled the conduct and content of a study, eventually published under Peter Lee’s name, that was designed specifically to refute a landmark study on lung cancer and SHS among nonsmoking women. In addition, the industry funded a 2003 British Medical Journal article that reported no causal relationship between SHS and lung cancer, which was criticized for being conducted in a way that almost ensured negative conclusions. In each of these cases, “independent” industry consultants played a critical role in providing scientific credibility in promoting the industry’s goals.

The tobacco industry’s long and consistent history of manipulating the content and presentation of scientific results raises questions about publishing work funded by the tobacco industry. The International Committee of Medical Journal Editors has recommended that, in addition to identification of financial support, the role of the funding organization in the design and control of a study should be specified. 

Paediatric and Perinatal Epidemiology, which published the article by Sullivan and Barlow, requires that acknowledgments include “details of funding.” The Sullivan review, as well as the other cases described above, illustrates the need for journals to require complete disclosure of all sponsor involvement in the conduct of a study and the preparation of the manuscript. However, such disclosure guidelines have been demonstrated repeatedly not to be effective with the tobacco industry, which seeks actively to minimize its role.

Clinicians, parents, and public health officials may be the most vulnerable to the conclusions in Sullivan’s article that SHS does not appear to be an important risk factor for SIDS. The national SIDS prevention campaign (“Back to Sleep”), sponsored by a coalition including the National Institute of Child Health and Human Development and the American Academy of Pediatrics, has focused mainly on the infant’s sleep position. Creating a smoke-free zone around the infant is one of many secondary messages. The Back to Sleep campaign has been very successful in reducing SIDS rates, and efforts to educate parents about the importance of sleep positioning must continue. Now that the campaign is well underway, efforts to address parental smoking behavior in both the prenatal and postnatal periods should be intensified. An example of a strengthened smoke-free message is found in the US EPA’s “Smoke-Free Home Pledge” 2001 campaign to reduce children’s diseases.

Clinicians need to address SHS exposure more effectively with their patients and parents. In 2001, according to a national survey of parents, only one half of all parents who visited pediatricians or family practitioners reported that they were asked about smoking status, and less than one half of the smoking parents said they were counseled about SHS or received advice to quit. Although quit rates during pregnancy have been increasing in the past decade, one half of the women who quit later experience relapses in the postpartum period, and these rates have not changed with time. One clinician barrier may be the perception that, as an American Academy of Pediatrics task force noted in 2000, “changing [maternal smoking] behavior has been far more difficult to accomplish than changing infant sleep position.” However, estimates of SIDS risks from SHS (odds ratios range from 1.4 to 5.175) have considerable overlap with estimates of risks from prone sleep positioning (odds ratios range from 1.7 to 12.976). Therefore, efforts to promote smoking cessation and reduction of SHS exposure for infants and children, particularly in discussions of SIDS prevention, should be intensified.

Clinical interventions designed to reduce SHS exposure among infants and children may be effective. A review of 19 studies (from 1987 to 2002) to reduce SHS exposure among youth concluded that these interventions can be effective.
of a motivational intervention that included educating smoking parents, ambient nicotine concentrations in children’s homes were reduced by 25 to 30%, compared with concentrations in homes where self-help material was provided, which resulted in no change. Also encouraging is the finding that, in another study, more than one half of smoking parents tried to quit after being counseled at their children’s clinic and offered nicotine replacement therapy and quit-line referrals.

The tobacco industry’s disinformation campaign regarding SHS and maternal and child health can be counteracted within clinicians’ offices. SHS must be recognized as an established, controllable, risk factor for SIDS, like prone sleep positioning. Clinicians and public health officials should intensify their efforts to promote reducing infant exposure to SHS as an effective strategy for reducing SIDS.

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