Against Anti-health Epidemiology:

Corporate Obstruction of Public Health via Manipulation of Epidemiology

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In response to several articles on corporate corruption of science that appeared earlier in this journal, a critic outlined an epistemological model based on an unsupported assertion that epidemiologic evidence is always required to support cause–effect relationships. This model, if adopted, would eliminate compensation to victims of toxic exposures and impede regulation of accepted hazards. Epidemiology is only one element in support of cause–effect determinations. The critic's proposal of an anti-health epidemiology was initially developed by corporations with the goal of providing defense in litigation, and is based not on science but on a corporate need to enhance profits at the expense of public health. *Key words*: epidemiology; industry influence; corporate corruption of science; cause–effect relationship.

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The October/December 2005 issue of this journal included several articles on corporate corruption of science.[1–4] Recently, Otto Wong responded to these articles' critiques of his work in a seven page "editorial" published in *Regulatory Toxicology and Pharmacology*, entitled, "The interpretation of occupational epidemiologic data in regulation and litigation: Studies of auto mechanics and petroleum workers."* In his editorial, Wong outlines an epistemological model that, if adopted, would eliminate compensation to victims of toxic exposures and impede regulation of accepted hazards of all kinds, including drugs. He bases his proposed method on the unsupported assertion that epidemiologic evidence is always required to establish cause–effect relationships. Epidemiology is only a component of the science of cause-effect determination and is supplemented by a variety of data sets, including animal studies, toxicology studies, molecular studies, case reports, studies of analogous substances, pathologic examination of tissue specimens, and various other studies.

While Wong claims to support "good epidemiological practices," these practices systematically underestimate exposure risk. As discussed below, they were initially proposed, developed, promoted, and funded by tobacco companies and the Chemical Manufacturers Association (now called the American Chemistry Council). The specific studies of asbestos and benzene risks that Wong cites were funded by various corporations with the goal of providing a "scientific" defense in litigation and a "scientific" rationale for eliminating or limiting government regulations designed to protect public health. Wong's proposal is an anti-health epidemiology, based not on science but on a corporate need to enhance profits at the expense of public health, and this need is satisfied by limiting effective regulation while leaving the injured uncompensated.

While epidemiology is often a component of the science of cause–effect determination, it is never its *sine qua non*. To determine general causation, researchers evaluate a variety of data sets, including animal studies, toxicology studies, molecular studies, case reports, studies of analogous substances, and epidemiologic case–control and cohort studies. Physicians use Koch's postulates and Hill's considerations to extrapolate a general cause–effect relationship from specific case reports.[5,6]

Koch introduced his postulates in 1890 to establish practices for determining microbial causes of disease, including chronic and occupational (anthrax) infectious diseases. Although limited in application, the postulates clearly display the importance of animal models in medical epistemology.[5]

Hill developed an expanded epistemological model that demonstrated the tobacco–cancer relationship through reliance on a number of scientific approaches.[6] Since then, Hill's "considerations" have been used in numerous instances to establish cause–effect relationships. However, it should be noted that Hill's considerations do not require the use of or

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*Dr. Gori, the editor of *Regulatory Toxicoloby and Pharmacology*, refused to publish our response, and we have, therefore, submitted it to *IJOEH*, since Dr. Wong based his editorial on criticism that was published in *IJOEH*.

espouse the superiority of epidemiologic evidence. The scope of medical evidence that substantiates his considerations is inclusive of all the available data.

Until the 1960s, pathology and its attendant clinical case reports were the primary tools used to ascertain cause–effect relationships.[7–9] The first textbook of epidemiology was published in 1960; yet physicians agreed on cause–effect relationships for a wide variety of chronic diseases long before that time.[10] For example, in 1942, the carcinogenicity of nickel carbonyl was firmly established based on case reports, pathology, and toxicology alone.[11]

Epidemiology is an important tool for assessing the likely levels of risks associated with different populations, but—as Koch and Hill's guidelines demonstrate—it is not a requisite component in the process of determining that there is a risk. Animal studies can also provide information about relative risk. Epidemiologic studies often suffer from design limitations that to do not account for the inconstant nature of workplace conditions and exposure levels, the often-delayed expression of occupational disease, which can remain latent for 50 years or longer, and the fact that studies often omit minorities and women. By the time clear and convincing large-scale epidemiologic evidence emerges, exposures may span a generation or more, with the evidence representing little more than a belated confirmatory footnote to a fully matured public health calamity. As the epidemiologist David Ozonoff has underscored, "a public health catastrophe is a health effect that is so powerful that even an epidemiologic study could detect it."[12]

WONG REJECTS CASE REPORTS IN CAUSATION

Wong rejects the use of case reports for determinations of causation: "Case reports, although useful in hypothesis-generation, are of limited value in causation assessment in chronic diseases such as cancer. Causation assessment in chronic diseases must be based on appropriately designed epidemiologic studies."[13] He fails to cite any article or empirical evidence to support this claim.

Contrary to his statement, case reports and case clusters have consistently been used to establish causation.[9] In 1939, Lanza and Vane noted, "Our knowledge of asbestosis is based on individual case reports . . . [although] too few to have statistical weight . . . the disease itself and its pathology have been well demonstrated."[14] Similarly, it is universally accepted that Wagner's landmark 1960 publication established the link between asbestos and mesothelioma in the consciousness of the scientific community.[15] That publication is simply a report of a series of cases and fails to satisfy Wong's restrictive definition of "epidemiology." Other exposure–chronic disease associations that have been ascertained by case reports and disease clusters include: aplastic anemia, as associated with radiation, benzene, radium, and trinitrotoluene exposures; peripheral neuropathy and n-hexane, methyl butyl ketone and dimethylaminopropionitrile; and chemical hepatitis from dimethylformamide, trinitrotoluene, tetrahydrofuran, tetrachloroethane, and chlorinated naphthalenes.[9]

While many of these cause-effect relationships were later confirmed with epidemiologic studies, the epidemiologic evidence was not a prerequisite to the establishment of precautionary measures or compensation. In fact, Lanes and Poole observed, "most epidemiologists found persuasive the early evidence of vinyl chloride and angiosarcoma of the liver, and between diethylstilbestrol and adenosarcoma of the vagina...The acceptance of these two cancer-exposure relations was firmly established on case reports alone."[16] Thus, case reports have contributed to the determination of causation and, at times, have supplanted the need for epidemiologic studies. Furthermore, an epidemiologic study is not required to establish a harmful effect in each and every occupation where similar exposures to established carcinogens occur. Inferences can be made based on exposure data alone, especially if the cohort is exposed to amounts of the carcinogen comparable to those seen in cohorts already studied, as is the case with brake mechanics.

IGNORING OTHER TYPES OF SCIENTIFIC INFORMATION—ANALOGY AND PATHOLOGY

Wong cannot and does not dispute the fact that asbestos causes mesothelioma. He cannot and does not dispute that no one has ever been able to scientifically establish a level of exposure to asbestos that eliminates the risk of cancer. With these two premises established and unchallenged, determining cause and effect for a particular individual with mesothelioma relies on the demonstration of exposure to asbestos and the exclusion of other even more rare established causes such as thorium. The work–disease connection is especially likely since these brake workers are exposed to asbestos, a known cause of mesothelioma, in the act of replacing brake linings and breathing the resulting dust.[17–19] Grinding and drilling of brake linings in manufacturing finishing processes is known to cause high exposure. Mechanics working similarly with asbestos-containing brakes and clutches are exposed to high concentrations of asbestos; therefore, the epistemological inquiry into mesothelioma causation ends unless an alternative etiological factor is present. Faced with this conundrum, Wong and others attempt to use epidemiologic studies to refute the indisputable fact that asbestos causes cancer. Wong relies on a variety of epidemiologic studies that contain no information regarding the quantum and duration of exposure to asbestos

as proxies for studies of exposure to asbestos in a specific population.[20] He draws inferences from these studies to explain away the hundreds of reported cases of mesothelioma in brake mechanics.[21]

Wong might assert that these workers are exposed only to chrysotile, since it is the most common fiber type found in brakes and—as others have tried to argue—chrysotile causes lung cancer but not pleural cancer. However, The World Trade Organization found the evidence of mesothelioma risk substantial enough to allow countries to ban the importation of chrysotile.[22] Moreover, recent pathologic studies demonstrate that short, thin chrysotile fibers are more prevalent than amphibole fibers in mesothelioma tumors and pleural tissues, providing more evidence that chrysotile is a cause of mesothelioma.[23] The epistemology of asbestos–mesothelioma causation requires contact between the causative substance and pleural tissue. Regardless of the carcinogenicity of chrysotile, it is also important to note that some brake manufacturers used amosite, and that tremolite—an undisputedly mesotheliogenic fiber—is a universal contaminant of the Canadian chrysotile most often used in brakes and therefore brake workers were exposed to both amphiboles and chrysotile.[24,25]

MISCLASSIFICATION BIAS AND USE OF INAPPROPRIATE (EXPOSED) CONTROLS

All of the studies Wong relies on in his 2001 meta-analysis have results significantly below the null. These results raise a red flag, simply because it is illogical to maintain that asbestos exposure or some unknown brake-work–associated confounder *reduces* the risk of asbestos-related disease. The more likely explanation for this counterintuitive result is systematic misclassification bias. Choice of questions and viewpoint are of particular import when evaluating levels of interview or recall bias. Although some of the asbestos studies analyzed by Wong took steps to minimize interview bias, they could not eliminate it.

Interviews of brake workers can successfully elicit the numbers of brake linings they have changed for various employers, as well as how many they did in years past in after-school jobs and for friends and family. In post-mortem interviews, family members are much less likely to possess such complete histories and to know all possible sources of exposures in the work situation, leading to an underestimation of exposures.[1] Persons not fluent in English are at special risk.

Wong claims that people are likely to overestimate exposures due to their knowledge that exposure to asbestos causes mesothelioma. However, many people are still unaware of their potential sources of exposure and—as reported by Wong—they may feel that their exposures were trivial.[13] The belief that encounters with asbestos products resulted in trivial exposures would lead to underreporting of these exposures; the fact that many people are unaware that brakes presently contain asbestos means that the underestimation may occur in an area of particular interest to Wong's meta-analysis, and explain his counterintuitive findings.[1]

In Wong's discussion of "Misclassification of occupation or exposure," he criticizes the way several of the studies reported in his 2001 meta-analysis combined work categories. He states that grouping auto mechanics with insulators and other end-product users is inappropriate because their exposure levels are different. We agree, and this is another reason we found his use of these studies to be inappropriate. He fails to address the clear misclassifications of occupations and exposures and other problems in the studies he favors.

Wong (2001[20]) ignores Teschke et al.'s statement that "grouping of occupations was likely to result in non-differential misclassification, usually biasing risk estimates to the null value."[26] The Woitowitz and Rodelsperger study, while designed to address auto mechanics in particular, is of limited value because they used lung cancer patients as controls.[27] As asbestos exposure is a well-known risk factor for lung cancer, any mesothelioma case–control study with lung cancer patients as the control group will almost certainly produce results at or below the null. This study demonstrates the effect of using inappropriate controls, since the researchers used "population controls" as well as lung cancer patients as controls. The odds ratio (OR) for "hospital controls" was 0.75, almost half that for population controls, 1.32.[27] Rather than report both results, Wong presented an average OR of 0.87.[20] McDonald and McDonald conducted a case–control study, published in 1980, examining mesothelioma cases from Canada and the United States. Like the Woitowitz and Rodelsperger study, this study relied on an inappropriate control group; control subjects were patients in cases diagnosed by pathologists in which "pulmonary metastases were present from a non-pulmonary malignant tumor."[28] The control group did not exclude patients who had died from asbestos-induced malignancies such as peritoneal mesothelioma, laryngeal cancer, or colon cancer. The choice of such controls biased results towards the null.

In the case of the Hansen (1989[29]) study, Wong omitted the mesothelioma findings from a relevant study that contradicted his findings, and in his original publication, he failed to explain his omission. He retrospectively excuses the omission by dismissing the one case of mesothelioma in 21,800 people over ten years allegedly "because Hansen ignored the background risk."[13] This is incorrect, and in his original paper Wong relies on Hansen and quotes Hansen's SMRs for lung cancer. [20] Table 4 in Hansen's paper presents population SMR data for mesothelioma.[29] Since no cases were expected, Hansen did not present an SMR

for mesothelioma but did conclude that he had found an excess of mesothelioma in brake workers. The age distribution of Hansen's cohort explains the low expected number for mesothelioma. Table 1 shows that 64% of those studied were between 25 and 34 years old at the end of follow-up. Another 21% were 35–44 years old. Accordingly, at the end of the study, 85% of the study population were under the age of 45, 92% were under 55, and 96% were under 65.[29] Wong used the *United States* NCI annual incidence rate of ten per million to suggest that two cases "could be expected" in Hansen's Danish cohort. However, the incidence rate is not synonymous with the background rate, and Denmark is not the United States. The incidence rate includes exposed workers, while the background rate is meant to estimate the combined number of "idiopathic" mesotheliomas and those caused by other known causes, e.g., thorium and exposures to asbestos in ambient air.

The generally accepted "background rate" is only one or two per million, not ten per million, and even this rate includes workers who have actual but undocumented exposures.[22] This is easily shown by comparing "general" female with male mesothelioma rates. Since there is no evidence that gender is a confounder for mesothelioma risk, the true background rate is below the female rate, since some females have direct and indirect asbestos exposures. All the epidemiologic studies Wong cites use this elevated "background" as a comparator. However, even using the upper end of this background rate, the expected rate in Hansen's nearly 22,000 auto mechanics is only 0.44. Thus, Hansen's conclusion that even one case is indicative of possible increased risk is valid and this study provides useful information about possible mesothelioma causation.

MISINTERPRETING THE MEANING OF A RATE RATIO OF 1 OR LESS

Dr. Wong and other automobile consultants misinterpret epidemiologic studies whose results reveal a rate ratio of 1 or less for a particular exposure. They interpret such results to mean that the exposure in question does not and cannot cause the disease in question. However, as Greenland has noted, this is a complete misinterpretation of epidemiologic results.[30] First, they failed to recognize the definition of a "caused" case. A caused case is either a new case that would not have occurred but for the exposure or, equally, a case that would have occurred without the exposure but that occurs sooner than it would have occurred due to the exposure. For example, if a person was destined to get breast cancer at age 80 but an exposure to a toxic substance causes that person to develop breast cancer at age 40 this is a "caused" case. As Greenland has noted, the usual age stratification used in epidemiologic studies does not account for this phenomenon.[30] At the

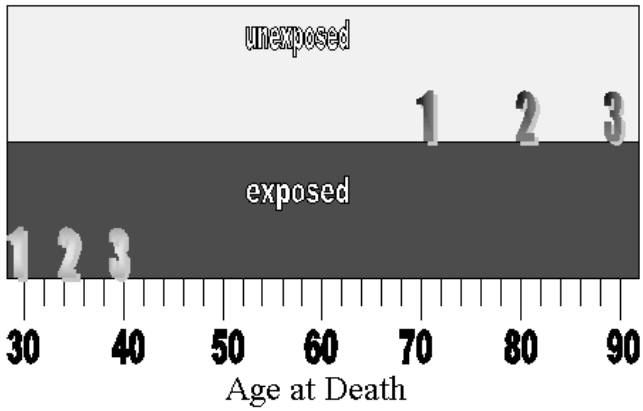

Figure 1—Age at death (exposed vs unexposed). If three people are predisposed to a disease, but exposure to a carcinogen causes it to occur much earlier than it would have otherwise, the exposure is a cause of the disease. Age stratification in epidemiologic studies may detect the extremes such as those in Figure 1, but not instances where the exposure causes disease to occur only a few years earlier than might otherwise have happened (Figure 2).

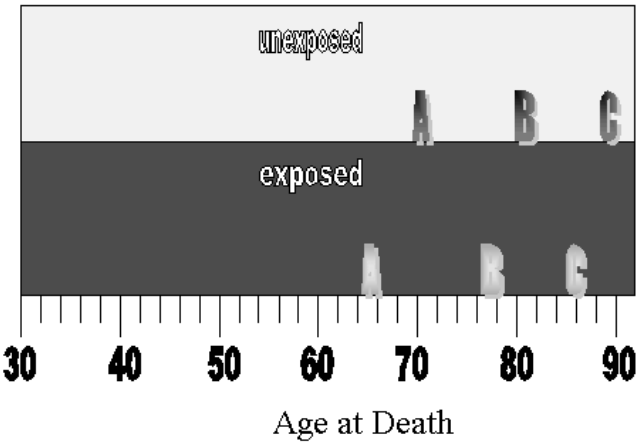

Figure 2—Age at death (exposed vs unexposed) undetected by standard stratification.

extreme, since we are all going to die, all rate ratios that compare total deaths between populations are always 1 after 100 (or even fewer) years of latency. This does not mean that some of the deaths were not caused by occupational or environmental exposures.

Figure 1 illustrates this principle; in a hypothetical population of 100 women, we expect three deaths to occur as a result of breast cancer at ages 70, 80, and 90. If the exposed population also has three deaths from breast cancer but these cases occur at ages 30, 35, and 40, it is reasonable to infer that the exposure in question "caused" all three cases, because they occurred sooner then they otherwise would have. The exposed cases would have lost over 150 years of useful life and their families and friends would have been deprived of

considerable love, learning, and friendship. If the exposure was accompanied by a specific biologic marker, then scientists could provide unequivocal evidence to a jury that the exposure in question was, on a more-likely-than-not basis, a contributing factor for the deaths of these individuals. Therefore, from a compensation perspective, if negligent conduct caused the exposures, all three individuals would deserve recompense, despite the fact that the RR was 1.

Unfortunately, Dr. Wong and other automobile consultants claim that their meta-analyses, which have reported a statistically significant protective effect of work with brakes, "prove" that asbestos brakes have not and cannot cause mesothelioma in any individual.[31–33] This is a misinterpretation of epidemiologic studies, which may result in an injustice to victims. More importantly, since some of the auto industry consultant epidemiologists have used this misinterpretation to lobby the government to remove warnings from brake products and to stop training brake mechanics in how to avoid the hazards of work with asbestos brakes, it may serve an anti-public health purpose.[34]

PETROLEUM WORKER STUDIES—CONTRACTOR BIAS

In the analysis of studies of petroleum workers, Wong's systematic misclassification once again biases his results toward the null hypothesis. Wong relies on company employment records as a surrogate for exposure. Petroleum refineries and chemical plants frequently use contractors to perform the most hazardous jobs, such as asbestos insulation and tank cleaning, during turnarounds. As a result, the most heavily exposed workers are often not official "employees" but are contract workers who are not included in employment records. Ironically, these workers may work for decades at a single plant.[35] They are not subject to exposure monitoring, and they are often not aware of potential exposures because the plant health and safety officers do not provide them with safety training or equipment. These most heavily exposed individuals are included in Wong's studies as unexposed controls (i.e., as part of the general population). Additionally, many plant employees whom Wong classifies as exposed are never subjected to chemicals at work (e.g., lawyers, office personnel, etc.). Wong empties the top left square of the classic 2-by-2 epidemiologic contingency table (the "A box") by moving the exposed–diseased cases that belong there into the "D box" (unexposed–diseased).

Such flaws in method bias Wong's results to the null. In fact, as with his asbestos studies, this systematic reclassification of exposed–diseased to unexposed–diseased often results in the counterintuitive result that exposures to benzene and other known carcinogens reduce the risks of developing cancer. In 1958, Wong's predecessor consultant to the petroleum industry, Robert Kehoe, told the American Petroleum Institute that "turnarounds" (refurbishment of refinery installations) were being subcontracted by many companies and that:

> It was quickly discovered as various industrial situations were studied also that job titles differed greatly from company to company and indicated little if any relationship to the degree and kind of hazard. Men charged with the actual operation of the equipment, for example in the petroleum industry, may have little or no contact with either petroleum or the various refined products, but the samplers and maintenance personnel may have frequent and heavy exposures. At the same time, maintenance and labor classifications may work in all parts of the refinery or, equally common, be assigned for a specific period of time to a single area. . . . Maintenance problems are reduced and even presently widely spaced "turn arounds" are being substantially subcontracted by many companies. It must be admitted that, although this development may diminish the exposure hazard for the refinery worker, it may be concentrating and augmenting the danger to employees of companies specializing in this service-type operation.[35]

In response to the criticism that he has misclassified study subjects, Wong says, "the issue of cohort definition and misclassification bias in occupational studies should be examined in terms of the availability of records for cohort identification and subsequent analysis within the cohort."[13] The petroleum and chemicals companies' cohorts are large, and these companies can distinguish the lawyers from the operators. Thus, Wong could have and should have compared cancer rates between exposed and truly unexposed workers from the same company, in the same location. He has, in fact, done this in the past. In one of his previous studies he noted:

> [T]he provision of an internal comparison group was designed to minimize several problems resulting from comparing an occupational cohort with the general population. Such an internal comparison group is regarded as the most appropriate basis for comparison in occupational mortality studies. In particular, comparisons based on internal groups were found to be more sensitive in detecting the association between leukemia and occupational exposure to benzene, and in demonstrating a suggested increase in lymphoma mortality with increased exposure among refinery workers.[36]

In his earlier studies, Wong even compared the use of internal and population control groups and documented the dramatic lowering of the apparent risk caused by the use of population controls. He wrote:

> The group exposed to benzene in this study did not show any significant mortality excess from all lym-

> phatic and haematopoietic cancers combined when compared with the United States male population, although some of the lymphopoietic cancer SMRs were slightly raised. When the SMRs for the occupationally exposed group (continuous and intermittent) were compared with those for the occupationally unexposed, however, a considerable difference was observed for all lymphopoietic cancer (121.1 v 34.6), lymphosarcoma and reticulosarcoma (112.8 v. 50.8), leukemia (117.4 v 0), and other lymphatic tissue cancer (163.8 v 53.0). . . . Hence, mortality from all non-Hodgkin's lymphopoietic cancers (non-Hodgkin's lymphoma and leukemia) was higher in the benzene exposed group than in the comparison group.[36]

Despite his knowledge that an internal control group led to the most sensitive study, all of his subsequent refinery and chemical plant cohort studies used general U.S. population controls.

Wong asserts that the critique of his systematic misclassification should be ignored because evidence for it came to light during cross-examination in a lawsuit. In this lawsuit, a worker with acute myelogenous leukemia (AML) sought compensation from a petroleum company.[13] At the request of the company, Wong argued that his studies proved that benzene exposure had not contributed to cause this benzene-exposed worker to develop AML. We are unaware of any "methodologic" construct that maintains that the source of a critique invalidates the criticism. "Consider the source" is not a scientific defense—it is a baseless, *ad hominem* affront.

Wong claims he is defending proper methodology in epidemiologic practice. This claim is common among scientists defending corporate interests. For the past decade, the chemical and tobacco industries have funded the promotion of what they refer to as "good epidemiological practices." These "practices" seek to prevent plaintiffs from prevailing in court and thwart government regulations.[37] In Wong's case, his "proper methodology" systematically underestimates disease among exposed workers. Just as following a flawed recipe will yield a foul-tasting cake, no matter how well you follow it, a faulty method will always yield unreliable results. In following his bad recipe to the letter, Wong ends up with a study marred by interview and misclassification bias. His manipulation of the "A Box" is akin to the effect that substituting salt for sugar has on a cake recipe; it makes for a bad-tasting cake. In epidemiology this makes for a very bad study. Although, in both cases the seller can represent that he followed the recipe to the "T."

CONCLUSION

Wong has repeatedly conducted studies on behalf of chemical and manufacturing companies that use these studies to defend against lawsuits and to influence government regulation.[1] Auto-parts manufacturers, including General Motors, Ford, Chrysler, and Bendix, have paid Wong and others tens of millions of dollars to promote views that will protect their profits.† They also have harassed experts whose views conflict with their economic goals.[38]

As Nobel laureate Gunnar Myrdal observed, "There can be no view except from a viewpoint. Prior to answers there must be questions. In the questions raised, the viewpoint has been chosen and the valuations implied." Wong chose his viewpoint before his questions, and his valuations biased his studies. He may follow his method to the letter, but it is a flawed method that leads to flawed results. Those results ultimately help his sponsors pollute the environment and injure workers and consumers.

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