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Features

DECONSTRUCTING A STATE-OF-THE-ART REVIEW OF THE ASBESTOS BRAKE INDUSTRY

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ABSTRACT

State of the art is a legal concept that describes what was known as knowable by experts including manufacturer's state of knowledge about the potential hazards of their product(s) at a point in time. In 2004, Paustenbach et al. published a state-of-the-art review that describes the development of knowledge about asbestos hazards to brake mechanics performing asbestos brake installation and maintenance. Paustenbach et al.'s review, however, omits important pieces of corporate knowledge, dismisses several historical scientific conclusions and ignores the way experts have applied the results of scientific research to protect workers and consumers handling asbestos brakes. By taking their state-of-the-art review out of the legal liability context, Paustenbach et al. create a misleading version of events that fails to properly address the question of what asbestos brake manufacturers knew or should have known about the potential hazards of their brakes to mechanics over time. Without proper presentation of this information, judges and juries cannot adequately assess whether these companies had a duty to warn or take other action to prevent injury to those exposed to their asbestos brakes.

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Over the past 20 to 30 years, entire corporations and private consultancies with hundreds of millions of dollars in annual revenue have been established to produce scientific knowledge that corporations can use to avoid regulation and defend against worker and consumer lawsuits. These lawsuits seek compensation for injuries and pollution resulting from corporate product manufacture and use. Most of the corporate-sponsored reviews have been designed to cast doubt on the relationship between exposures and disease (e.g., smoking or asbestos and lung cancer) [1-5]. However, in most states, the injured party must show that a company "should have known" about a risk before the company can be held responsible for having produced injuries. Thus lawsuits hinge on a "state-of-the-art" analysis answering the question: "Who knew what when?" In response over the past decade, companies have begun to fund these same consultants and historians to create revisionist histories of what companies should have known about a particular risk at a particular time [6]. An accurate "state-of-the-art analysis" is not revisionist but should contextualize the information within the relevant *standards of the times* in which a product was produced [7].

To highlight several of the typical methods experts have used to generate results that meet the litigation needs of their corporate clients, we critically examined a corporate-funded expert medico-legal state-of-the-art review, "Environmental and occupational health hazards associated with the presence of asbestos in brake linings and pads (1900 to present)," published by Paustenbach, Finley, Lu, Brorby, and Sheehan in 2004[1] [8]. The authors guide readers through three time periods—1900 through 1959, 1960 through 1974, and 1975 through 2002—discussing the published literature regarding asbestos dust and disease and characterizing the development of knowledge about pulmonary fibrosis, lung cancer, and mesothelioma in asbestos workers. They conclude that the published literature regarding asbestos hazards and brake work failed to document asbestos exposures sufficient to cause asbestos-induced diseases. Paustenbach et al. overlook several key components of a state-of-the-art review and thus inappropriately conclude that brake manufacturers did not have a duty to warn consumers of the asbestos-related hazards of brake work. As we will illustrate, historical standards stated that manufacturers had a duty to test products and warn workers, as well as end product users, of potential hazards, and there was sufficient evidence to require that greater action be taken to protect workers from asbestos exposure.

Paustenbach et al. limit their review to published studies, omitting information privately held by the corporations that manufactured asbestos products, as well as

[1] This stands in contrast to a medical state-of-the-art review, which provides the most current information on a disease and its treatment and generally does not include a historical analysis of the medical literature, as with a medico-legal state-of-the-art review.

international literature about the dangers of asbestos. They insist—contrary to historical and scientific evidence—that dose-response curves, fiber type data, and "quantitative" epidemiologic evaluations are necessary to establish any basis for understanding the health effects of asbestos exposure in brake mechanics. Their set of criteria does not account for the way industrial hygienists and occupational physicians used scientific information to protect workers during the periods in question, including both friction product manufacturers and brake mechanics. Further, the authors mistake the premise of a state-of-the-art review by focusing on "causation" instead of "cause for concern," which is the appropriate consideration for triggering a warning.

For public safety, all substances or products that trigger cause for concern generate a "duty to warn" [9]. Current proponents of public health must be aware of corporate production of inaccurate science and be prepared to deconstruct and reject these arguments in order to facilitate the implementation of policies that protect the public, workers, and the environment rather than corporate economic interests.

In the first half of this article, we introduce the studies that Paustenbach et al. dismiss and present data they omit in order to evaluate the validity of their "state-of-the-art" review. We compare the contemporaneous types of knowledge that physicians and others accepted to the available information, illustrating what was known about the association between asbestos brake work and disease over the past 100 years [10]. We subsequently examine state-of-the-art through the perspective of what *corporations* actually knew at certain points in the past and what duties corporations held according to community standards at the time. Our review includes corporate documents uncovered through toxic tort litigation, international knowledge available within the United States, and a re-analysis of several of the articles cited by Paustenbach et al.

EXCLUSION OF UNPUBLISHED STUDIES, CORPORATE DOCUMENTS, AND THE INTERNATIONAL CONSENSUS ON THE DANGERS OF ASBESTOS

Paustenbach et al. restrict their state-of-the-art review to published literature while excluding unpublished research conducted by corporations as well as internal documents and communications among company executives. In other words, the "who" in Paustenbach et al.'s answer to "who knew what when?" is limited to regulatory officials and the physicians in the public domain, who generally had access only to the published medical literature. Asbestos product manufacturers, however, commissioned a variety of confidential studies—including epidemiologic studies, animal studies and dose measurements—and had access to data from their own employees who incurred exposures, including those related to brake maintenance. The corporations took concerted action to keep study results linking asbestos exposure with pulmonary fibrosis and

cancer out of the public domain and filled the literature with adulterated animal and human studies asserting that asbestos was not a carcinogen—a course of action that helped delay effective regulation on asbestos use and warnings [3, 5, 11-13]. This privately held information has become available through document production and depositions related to litigation. Dr. Paustenbach and his co-authors omit this information despite having access to these discovery materials.

Limiting an analysis to "published information" is particularly inappropriate in this case since the brake manufacturers concealed knowledge and published misinformation about the risks of asbestos exposure. Corporate documents reveal that the brake companies knew more about the risks to brake mechanics than did the medical and regulatory sectors. This information includes speeches and minutes from meetings of the Friction Manufacturing Standards Institute (FMSI), and personal correspondence among company executives, industrial hygiene consultants, and representatives from industry-sponsored asbestos research institutes. Appendix 1 lists some of the documents omitted from Paustenbach et al.'s review.

International Knowledge

In addition to omitting unpublished information, Paustenbach et al. largely ignore the importance of international consensus and regulations on asbestos. Efforts to research and identify the cause of pneumoconiosis in industrial workers originated in England in the first decade of the 20th century. Regulation of asbestos exposures in the workplace and inclusion of work-related asbestos diseases in insurance programs occurred early in the 20th century in Germany and England. By 1930, representatives in the English Parliament had proposed to include asbestosis in worker's compensation programs, and in 1931 the country had adopted the British Asbestos Regulations. Paustenbach et al. make note of regulations that "required monitoring and dust control in industries where workers were exposed to asbestos," but assign little importance to them as indicators of what was known about asbestos hazards in 1931 [2, p. 45]. It should be noted that Ford and General Motors, two of the funders of Paustenbach et al.'s paper, had major operations in Europe beginning in the 1930s.

Castleman details the development of knowledge about asbestos hazards in England, Germany and other European countries, citing various case reports and studies—many of which were translated into English and published in important medical journals—as well as regulations and workers' compensation and insurance policies that recognized asbestosis and lung cancer as industrial diseases [12]. Instead of discussing the implications of these regulations and medical consensus, Paustenbach et al. highlight the fact that there was no national guideline for asbestos regulation in the United States until 1946 and no official national regulation of asbestos until 1971, while diminishing the fact

that some states (Illinois, Oregon, and California) had issued asbestos recommendations prior to 1946 and all states (including Michigan) had adopted the ACGIH asbestos Threshold Limit Values (TLVs) by the mid-1960s. They also discount the 1943 Federal Maritime Commission's Minimum Safety Standards for shipbuilding, which required "periodic medical evaluation" and respirators for workers exposed to asbestos dust, and the 1960 Walsh-Healy regulations, which included TLV requirements for asbestos that applied to all federal contracts above $10,000 [14, 15].

A PROBLEMATIC APPROACH TO A REVIEW OF ASBESTOS LITERATURE

Experts have always relied on multiple sources of information in order to solve medical and scientific mysteries [16]. However, Paustenbach et al. privilege a limited and fixed set of evidence that they claim may be used to establish a cause-effect relationship between asbestos and disease. They restrict their evidence to a subset of epidemiologic studies conducted on specific worker populations that include dose measurements and fiber type. Unfortunately (conveniently for them), these data do not exist. Though they concede that a large body of literature existed to support a link between asbestos and pulmonary fibrosis, mesothelioma, and lung cancer, they diminish the importance of many of these studies and case series for one or more of the following reasons: 1) case studies are not sufficient to establish a causal link between asbestos and disease; 2) the study lacks dose-response data; 3) the study lacks fiber type information; 4) the study fails to explicitly state criteria used to diagnose asbestosis; and 5) epidemiologic studies that follow cohorts in a particular occupation (e.g., textile, insulation, brake manufacturing) cannot be used to analogize risk to workers in another occupation (i.e., brake maintenance). The authors do not adequately explain these assertions nor do they cite any scientific literature—historical or current—that supports their criteria. Paustenbach et al. acknowledge that no early studies documenting the existence of pulmonary fibrosis (asbestosis) in asbestos workers relied on dose-response, fiber type, or quantitative exposure data. Despite arguing that there was insufficient data in these studies to quantify the extent of exposures, they assert that these same studies established risk to "highly exposed" asbestos manufacturing workers including friction product manufacturing workers [2, p. 49].

Case Studies

Case reports of lung disease in asbestos workers appeared in the literature in the first decade of the 1900s [17, 18]. In 1924, W.E. Cooke published the groundbreaking autopsy findings of Nellie Kershaw, a 33-year-old textile manufacturing worker, describing "fibrosis of the lungs due to the inhalation of

asbestos dust" [19]. In the following years, in both the United States and England, major medical journals published a flurry of similar case reports documenting pulmonary fibrosis and definitively attributing it to asbestos inhalation [12, 20].

Paustenbach et al. would have readers believe that the early case studies describing asbestosis (and later, lung cancer and mesothelioma) did not convince experts of asbestos dangers and that epidemiologic studies are and have always been a prerequisite to establishing a consensus that a certain exposure caused a disease. However, case studies have been reliably used by doctors throughout the 20th and into the 21st century in order to make causal determinations about occupational exposures. In the first half of the 20th century, it was accepted practice for doctors to attribute causation to occupational exposures when the diseases were unusual or unique [21]. Case comparisons are epistemologically identical to formal epidemiologic studies: a doctor publishes an "unusual" case as compared to the "control group" consisting of all other patients the clinician has seen or read about. If a patient presented with the unique signs and symptoms of a disease that rarely occurred in the general population and the patient had a history of exposure to an agent associated with the disease, a physician could find to a reasonable degree of medical certainty that exposure to that agent is what caused the disease. Percival Pott did this as early as 1775 when he determined that a high incidence of scrotal cancer in chimney sweeps—a disease uncommon in the general population—was due to the soot they encountered at work [22]. Similar causal inferences of associations between industrial agents and occupational diseases were made through cluster observations for a variety of occupational hazards including exposure to benzene, radium, vinyl chloride, and chromium. Fleming et al. conclude that even in modern-day medicine, where formal epidemiology has increased in importance, case reports and cluster observations continue to generate important conclusions about cause-effect relationships [21].

In addition to case studies, pathology and clinical findings provided evidence of causation, particularly when a disease agent left a unique marker of exposure. Asbestos leaves specific markers such as asbestos bodies (coated asbestos fibers) and pleural plaques in the lungs and pleura of patients with asbestosis, asbestos-induced lung cancer, and mesothelioma. Asbestosis manifests a characteristic x-ray appearance, affecting the lower lobes of the lungs and pleura, which distinguishes it from other lung diseases such as tuberculosis and also from other types of fibrosis such as silicosis [9, 12]. By 1930, lung x-rays had become a tool for identifying asbestos-induced disease, and medical practitioners who observed these markers in fibrotic tissue linked asbestos with fibrosis [12, 13].

Merewether, Dose-Response Data, and Fiber Type Information

The number of case reports that confirmed, both clinically and radiologically, the existence of pulmonary fibrosis in asbestos workers spurred an in-depth

investigation by the British Factory Inspectorate. In 1930, Merewether and Price published the results of their investigation—a cross-sectional study of currently employed workers which had no control group—in an official government report. In the same year, Merewether published another lengthy report in the *Journal of Industrial Hygiene* (United States) that detailed the disease progression and included safety recommendations for workers in the asbestos industry [9, 24]. The Merewether and Price study did not include specific dose measurements for the various processes, fiber-type analysis, or a control group for comparison. Nevertheless, the authors were able to conclude that a dose-response relationship existed.

Furthermore, the 1930 paper described various dust-producing processes found in the asbestos industry, including manufacturing and finishing work done on asbestos brakes and clutches, and clearly outlined guidelines for reducing hazardous dust exposures in the workplace. Although Merewether studied only textile workers, these recommendations were intended to apply to all workers involved in dusty processes, including end users of asbestos products and those who worked in manufacturing facilities not yet studied. The report noted, for example, that working with insulation materials (which were often only 15% asbestos) was a "dangerous trade" [9].

Merewether listed among the hazardous, dust-producing processes "[s]awing, grinding, trimming, polishing and other abrading machines, used on dry asbestos products" (all common to many end uses of asbestos products including insulating, pipe fitting and brake maintenance) and recommended the "application of Efficient Localized Exhaust Ventilation at dust-producing points" [9]. Merewether also specifically addressed the need for ventilation during work on brakes and clutches and told employers (manufacturers) that protective measures "include the education of the individual, as in other dangerous trades, to a *sane appreciation* of the risk, and to his personal responsibility in the prevention and suppression of dust," thus charging them with the responsibility to warn at-risk workers of the hazard [17]. By 1930, Merewether had established the need to warn about asbestos hazards absent any dose measurement, fiber type discussion, or specific dose-response information [17].

Paustenbach et al. acknowledge the importance of Merewether and Price's study in that it offered confirmation that "there was a clear risk of developing asbestosis from inhalation of asbestos dust, and that duration of exposure and amount of dust inhaled were key factors for predicting disease" [2, p. 45]. However, they criticize the study because it did not provide data on the dose-response relationship between asbestos exposure and disease. This is exactly our point: contemporaneous experts did not require dose or fiber type information to establish cause-effect relationships and implement public health interventions. Merewether and Price included copious warnings in their report, noted the general applicability of the study's findings across various occupations, and recommended warnings and workplace controls. Paustenbach et al. fail to

mention these aspects of the report and regard with a certain degree of surprise the fact that Merewether and Price's study "appears to have resulted in the British Asbestos Regulations of 1931 (His Majesty's Stationery Office, 1931), which required monitoring and dust control in industries where workers were exposed to asbestos" [2, p. 45].

Studies on Friction Product Manufacturers' Workers and Their Application to Brake Mechanics

Paustenbach et al.'s discussion of the surge in asbestos health effects studies following the publication of Merewether and Price's 1930 report mitigates the relevance of these studies to the plight of brake mechanics. The authors identify eight other publications they claim include data specific to workers in brake manufacturing plants [24-31]. The authors thoroughly discuss their findings and credit the studies with making clear links between asbestos exposure from friction products and disease; however, they fault the studies because most were carried out in manufacturing settings as opposed to garages, did not identify asbestos fiber type, and failed to quantify the concentration of asbestos fibers to which workers were exposed.

They argue that the data from the studies of friction product manufacturing workers cannot apply to brake mechanics using friction products because their exposures are not comparable. Their assertion is uncited and unfounded, and it conflicts with information that brake manufacturing companies had at their disposal. Castrop, GM's industrial hygienist, for example, describes the risk of working with friction products in manufacturing plants: "dust exposures to asbestos and other ingredients of the brake lining exist when the dry materials are handled and emptied into the mixers *and in the subsequent operations of slitting, grinding, or surfacing*" (emphasis added) [32]. Brake mechanics slit, ground, and surfaced these same brakes in auto body shops and cleaned out asbestos dust by use of pressured air and sweeping. These processes produced visible dust in the breathing zone of workers wherever they performed this work. It is clear that early research, such as Merewether and Price's study, should have applied to all workers in the asbestos industry and also to end users of asbestos products as the paper included precautions for molding, cutting, sawing, grinding, finishing and lathing tasks, several of which are involved in the installation of brakes and clutches. The authors specifically mention "brake and clutch linings" as a source of asbestos-containing materials, and aptly note that "[a]part from manufacture, certain work is carried on . . . which involves use or manipulation of asbestos or products containing it" [24].

Paustenbach et al. also diminish the value of these studies for end users by insisting that "quantitative" data is necessary for making the link in the absence of studies done on mechanics. In 1934, Osborn reported the "quantitative" brake work exposure data the other studies lacked, reporting some exposure levels that

were three times higher than the safe threshold that was proposed to protect against asbestosis just four years later [33]. However, the authors fault this study as well by pointing out that the report did not include an analysis of health effects. However, Osborn did express concern about the levels of exposure he uncovered, calling for a "very much more effective" exhaust hood when a dust count was too high during grinding [33], clearly because higher exposures endangered workers' health. Osborn noted the marked difference between processes that included dust control measures and those that did not. For example, the exposures incurred by a worker operating an internal brake grinder with a good dust removal system were found to be 4 mppcf (million particles per cubic foot), whereas a worker operating an external brake grinder with a poor ventilation system was exposed to 17 mppcf. Brake mechanics grinding or finishing brakes before installing them were not warned of asbestos hazards or the need to ventilate their workspaces until the mid-1970s, and were, therefore, more likely exposed to these higher concentrations. Moreover, Osborn expressed concern about high exposures during intermittent operations, a type of exposure typical of those incurred by brake mechanics during compressed air blowout of brake dust from drums [33]. Paustenbach et al. do not discuss these aspects of the study in their analysis.

Furthermore, the principle of the maximum allowable concentration (later the threshold limit value), designed to reduce nonmalignant disease, applied to all asbestos exposures, regardless of occupation, and, therefore, epidemiology studies that included dose-response data for specific occupations were not necessary to determine if a job created a risk of contracting asbestos-related disease [34].

Brake Mechanics and Mesothelioma

Paustenbach et al. regard the time period from 1960 through 1974 as one in which "the focus of health effects studies expanded to include manufacturing environments and end users of asbestos-containing products, including brake linings and pads" [2, p. 54]. They argue that the preponderance of medical literature suggesting that asbestos exposures could result in mesothelioma appeared during these years, noting that this was when industrial hygienists began to measure the dust exposures of brake mechanics.

Indeed, the 1960s and 1970s saw the publication of several studies that documented mesothelioma in the end users of asbestos products, including brake mechanics [35-41]. Paustenbach et al., however, dismiss these studies, claiming that "the health effects studies of primary interest during this and subsequent eras are those epidemiological studies that quantitatively evaluated the association of work as a garage mechanic and asbestos-related diseases." This uncited assertion is another instance of Paustenbach et al.'s application of a temporally inappropriate selection of evidence. The first studies that

quantitatively compare asbestos disease rates for different occupations did not appear until the late 1980s and, therefore, could not have been of "primary interest" during the 1960s and 1970s. In fact, during this "era" no author of asbestos studies suggested that "quantitative evaluation" of workers performing any asbestos-related job was required to establish that these workers were at risk. The medical literature of the time was made up of case series and reports from medical registries, the same types of evidence that Paustenbach et al. dismiss.

These papers did not merely "include a reference to occupations such as garage mechanics or service station operators," as Paustenbach et al. state, but concluded that their cancers were caused by asbestos exposure when the reported jobs involved exposure to asbestos products, including friction products. This conclusion fits within a larger medical/scientific model that attributes a rare or unique disease to an occupational exposure to a substance (such as asbestos and mesothelioma) if the medical community has accepted a cause-effect relationship between that substance and the disease. These analyses, therefore, were not intended to establish whether asbestos exposures from end product use were significant contributing causes of mesothelioma but rather to alert the wider medical community to the variety of jobs that presented a risk of causing asbestos-related diseases.

Paustenbach et al. again misinterpret expert consensus on asbestos hazards for brake mechanics when considering publications that called for implementation of exposure controls during brake work or substitution of non-asbestos friction products. For example, the authors cite a 1970 paper by Hatch, a consultant for automobile manufacturer Ferodo, and another 1970 paper by Lee, a consultant for the automobile manufacturer British Leland, as studies that measure the amount of asbestos fibers released during brake servicing [42, 43]. While it is true that these studies do include these measurements, it is misleading to suggest that their intention was to determine whether asbestos was a risk for brake mechanics. The title of Hatch's paper was "Possible Alternatives to Asbestos as a Friction Material" and Lee's paper evaluated the efficacy of dust reduction of various brake cleaning methods. Risk to brake mechanics was already assumed in these papers; it was not an issue the authors sought to determine.

In 1970, Hickish and Knight, consultants for Ford Motor Company, published the first and only study of brake mechanics that measured dust exposures over the course of an entire 8-hour day. Paustenbach et al. selectively report some of the data from this study that appears to support their argument: "exposure to asbestos during brake maintenance [was] not as severe as was anticipated"; however, they omit important conclusions including the fact that "the [asbestos] standard may be exceeded in the dust cloud during actual brake cleaning." They also omit the conclusion that during truck servicing "personal exposures in the vicinity do exceed the standard. On a daily basis, the personal exposure levels are below the standard, although that of the brake cleaner approaches it."

This standard, however, did not protect against the risk of cancer (see below: *TLV and Cancer*). Moreover, the authors omit Hickish and Knight's recommendation that "care should be exercised during brake cleaning to avoid inhalation of the dust produced" as well as their caveat that their "studies have not included maintenance procedures which involve the filing or grinding of brake lining material, and [they] would envisage that these would give rise to considerably increased air contamination by chrysotile asbestos, with the attendant need for strict precautions to prevent the inhalation of fibres" [44].

Criteria to Diagnose Asbestosis

Paustenbach et al. discount the studies by Stone and Hawes (1937) and George and Leonard (1939) because they do not provide quantitative information, indicate whether workers were handling raw asbestos or fabricating molded brake linings, or provide "the criteria used to diagnose asbestosis" [2, p. 49]. (Interestingly, Paustenbach et al. acknowledge that no studies between 1930 and 1950 provided "diagnostic criteria" for the diagnosis of clinical asbestosis.) Stone and Hawes examined 148 workers at a Dewey's Multibestos brake lining manufacturing facility and found that 82 percent of them had asbestosis [30, 45]. George and Leonard, consultants for the company, reexamined the workers and found that 13 percent of the workers had developed "clear and advanced" asbestosis through the manufacture and finishing of brake pads and linings [28]. Despite the difference in the two results, the 13 percent rate was enough to alert industry to a problem. Hawes stated that asbestos was "the most dangerous of all dusts" and exposure was often "fatal" [45]. Osborn had already shown that exposures during finishing operations exceeded the TLV. In combination, these two studies provided exposure and disease information.

HISTORICAL SIGNIFICANCE OF OMITTED INFORMATION AND DATA

Experts Issued Warnings Even with "Imperfect" Data

In their dismissal of many studies, Paustenbach, et al. demonstrate a fundamental misunderstanding of how research in industrial hygiene was interpreted and used to implement worker protection during the first six decades of the 20th century. They confuse "cause for warning or action" with "causation," asserting that the duty to test and/or warn is not triggered until there is consensus that asbestos caused an increased rate of disease in a particular group of exposed workers or bystanders. Causal consensus is not the marker that determines when manufacturers had a duty to take action to prevent injury to those handling their products. Companies have a duty to warn "wherever a

reasonable man would want to be informed of the risk in order to decide whether to expose himself to it" [46]. This is an especially important distinction when an industry's financial interests delay causal consensus as they did in the case of asbestos products.

Paustenbach et al. are correct in noting that the "field of industrial hygiene was just emerging" during the first half of the 20th century. Early industrial hygienists knowingly worked with imperfect measurement techniques and often focused on specific occupations. However, despite the imperfection of the techniques of industrial hygiene research or their divergence from today's industrial hygiene practices, the research conducted led experts to reasonably conclude that asbestos exposure was dangerous and manufacturers in fact had a duty to warn workers, end users, *and* workers in other occupations.

It is vital to note that before the 1970s there was no standard technique for counting asbestos fibers, and there was no way to accurately convert total dust counts to fiber counts [47]. Industrial hygienists could measure only the total number of dust particles in the air, and dust counts could not distinguish asbestos fibers from rock or other dust. Research funded by the Quebec Asbestos Mining Association showed that there was no way to correlate particle and fiber counts, making the acquisition of historical dose estimates essentially impossible [48]. Dr. Anthony Lanza, the associate medical director of Metropolitan Life Insurance and a consultant for General Motors, proposed the earliest guidelines for maximum allowable concentrations of asbestos-containing dust in 1935 based on analogy to preexisting guidelines for silica [27]. In 1938, a study by Dresseen et al. of the U.S. Public Health Service formed the basis of a 5 mppcf guideline [49]. This study and others, utilizing the best measurement techniques available at the time, based the guidelines on measurements of total dust particles. The authors of this recommendation based it on imperfect data derived from exposure to dust that contained low percentages of asbestos fibers [47]. Despite this 50 percent of workers studied who had been exposed to dust at the proposed threshold developed asbestosis after 20 years of exposure.

The Duty to Warn Brake Mechanics: 1970s

Corporate documents that were available to Paustenbach et al. directly contradict their assertion that brake installation and maintenance produce less asbestos exposures from work with brakes did not exceed the TVL. These documents show that the brake manufacturing companies were indeed aware of the risk to mechanics in the 1970s. In 1972, Ed Drislane, the president of the Friction Materials Standards Institute, wrote a letter to J. H. Kelly of Bendix Corporation noting that brake installation exposed mechanics to dangerous levels of asbestos; "[w]hen customers of yours drill linings, chamber linings, cut linings, or grind linings, they may very well raise the asbestos concentration in the atmosphere to above the OSHA standard [of 5 fibers per cubic centimeter (f/cc)]." He went on to directly compare factory and user exposures.

> Some members [of FMSI] have indicated that the drilling and grinding operations are problem areas in brake lining factories with existing exhaust systems. Therefore, if a customer of yours started drilling or grinding without having proper dust collectors, he would probably be in violation of the OSHA standard. It therefore becomes your responsibility, as a supplier of the brake lining, to warn the customer of this possibility [50].

I. H. Weaver, president of the major brake manufacturer Raybestos Manhattan, confirmed Drislane's assessment of dangerous exposures in a 1973 speech to FMSI:

> If this kind of thing [grinding, drilling, and cutting operations] occurs in fabrication operations of major OE customers, it appears to me there can be no argument about the need for educational measures to reduce chances of unnecessary exposure during grinding, drilling, or cutting operations. To those who argue that labeling or other types of warning need not apply to replacement materials because fabricators or appliers handling replacement quantities are exposed relatively intermittently, I say emphatically this just ain't necessarily so! Large volume replacement users present major potential hazards, and even small job shops can needlessly expose people to high fibre concentrations if operations are performed without controls [51].

Furthermore, Paustenbach et al. summarize a 1975 bulletin issued by the National Institute for Occupational Safety and Health (NIOSH) entitled "Current Intelligence Bulletin 5 Asbestos: Exposure During Servicing of Motor Vehicle Brake and Clutch Assemblies" by stating that the NIOSH document stood for the proposition that "the full extent of asbestos-related disease in brake servicing personnel is not known at present because this particular occupational group has not been studied systematically up to now" [52]. The authors mischaracterize by omitting the bulletin's determination that servicing motor vehicle brake and clutch assemblies resulted in dangerous exposures and the copious recommendations for controlling exposures to brake mechanics. The bulletin also included recommendations for designated areas for brake maintenance work, use of respirators and high-efficiency vacuums for cleaning work areas and ventilation systems for brake and clutch installation and servicing operations, which included grinding, arcing and compressed air blowout [52]. In Appendix 2 we have reproduced some excerpts from the 1975 bulletin that Paustenbach et al. omitted from their analysis.

Furthermore, a 1980 Italian study of a friction materials manufacturing plant, conducted by Scansetti et al. and published in English, revealed exposure measurements higher than the data cited by Paustenbach et al. during the same period. They found that the mean concentration of fibers (without full control measures) was approximately 1.5 f/cc for clutch facings, 1 f/cc for brake linings, and 1.5 f/cc for pads [53]. This is much higher than the mean exposures that NIOSH reported (0.05 f/cc) while using a variety of controls, which were not generally implemented in commercial use. Paustenbach et al. cite the NIOSH data

and several other international studies, which they note all fall within the NIOSH data range [2, p. 76], but fail to cite this study.

Historical Context of Fiber Type Defense

Paustenbach et al. argue that many of the studies used to make causal determinations about the danger to brake workers were inadequate because they did not consider fiber type as a factor in the potential for asbestos to cause disease [2, pp. 39, 46]. This assertion is anachronistic, as fiber type was not a consideration of early investigations into asbestos hazards. Questions concerning the difference in potency of various fiber types did not arise until the mid-1960s and were primarily an industry tactic to create doubt about the capacity of chrysotile asbestos to cause mesothelioma in end users such as brake mechanics [54, 55]. Most pre-1960 literature never mentioned fiber type, and all studies, including Wagner's mesothelioma case series, involved workers who probably had exposure to multiple fiber types [56]. Prior to 1960, country-specific asbestos types, such as Canadian, South African, or British asbestos, were mentioned in some documents. In 1937, A. J. Lanza explained the origin and purpose of the fiber type defense in response to a question posed to him by the owner of the Multibestos asbestos brake manufacturing facility, Bradley Dewey:

> Of course, the asbestos people in Canada have advanced that idea for some time as an explanation of why asbestos seems to be more clinically severe in England than in this country but I have always had the feeling that their argument was motivated by self-interest rather than to make a scientific contribution [57].

Corporations understood that the differentiation between the risks of different "types" of asbestos was a public relations strategy that lacked a scientific foundation.

Quantifying Exposure: TLV Irrelevant After Introduction of Asbestos as a Carcinogen

Before addressing the TLV, it is first important to note that the development of knowledge about the carcinogenic effects of asbestos happened much earlier than 1955, when Paustenbach et al. claim. Recognition that asbestos caused cancer occurred nearly 20 years earlier, with the first case reports of pleural carcinomas in autopsied asbestosis patients [58-63]. In 1938 Nordmann was the first to definitively link asbestos and cancer in a published review of the case literature on the issue of carcinoma and asbestosis [64]. He based this determination on distinguishing characteristics of carcinomas among asbestotics, including 1) an unusually large number of carcinomas among asbestotics compared to workers suffering from silicosis, 2) the young age of a large proportion of lung cancer deaths among asbestotics compared to the general population, 3) a consistent lapse of time (15-21 years) between first exposure to asbestos and

death from lung cancer, and 4) characteristic tumors in the lower lobes of the lung. Nordmann's determination of asbestos cancer causality reflected contemporaneous understanding of scientific knowledge production.

Notably, the cause-effect relationship between asbestos and cancer was based on pathological evidence and included none of Paustenbach et al.'s criteria for state-of-the-art: formal epidemiology, fiber type, and dose measurements or dose-response data. Before 1950, pathologists compared the physical findings of microscopic cancers in asbestotics to the lack of such findings in the lung tissue of silicotics to conclude that asbestos was a carcinogen [12]. Autopsy studies of silicotics served as the control group for these studies. Castleman outlines a large number of studies that supported the asbestos-cancer relationship prior to 1955 [12].

Despite all this evidence, Paustenbach et al. do not consider the causal relationship between asbestos and cancer to be confirmed until Doll's 1955 epidemiology study, which compared lung cancer rates found in asbestos worker autopsies with the lung cancer rates in the general population of England and Wales [65]. Doll concluded that asbestos caused lung cancer despite the fact that he did not have any exposure information, a specific control group, or smoking histories.

With the development of cancer addressed, we now turn to the concern over the TLV. Paustenbach et al. rely heavily on the fact that measurements of average dust exposures to brake mechanics taken after 1970 using workplace exposure controls were consistently below the pre-1970 asbestos TLV. However, TLVs were designed only to protect against the noncarginogenic effects of asbestos, not lung cancer or mesothelioma.

The development of knowledge about another inhaled lung carcinogen, nickel carbonyl, illustrates this point, showing the type, quantity and quality of information that was relied on during the 1950s to establish cause-effect relationships. With the publication of several case reports linking nickel carbonyl with lung cancer, health experts treated nickel carbonyl as though it were a carcinogen even though they only "suspected" but had no definitive "proof" of its carcinogenicity [66]. Because nickel carbonyl was not a major product or profit center for any manufacturer, the corporations that manufactured it did not try to influence the medical literature or standard-setting process for its control. Without the influence of industry, public health authorities took action based on scientific, medical, and public health principles [67].

In 1956, Smyth of the Mellon Institute and Union Carbide Corporation explained the public health response to carcinogenic substances in a re-publication of a lecture presented at the 17th Annual Meeting of the American Industrial Hygiene Association:

> At this time, it is prudent to set the standard for a cancergenic substance substantially at zero as has already been done for nickel carbonyl and no considerations can justify allowing the inhalation of any concentration which is avoidable [68].

Therefore, even suspected carcinogens were treated with extreme caution by responsible industries, government, and scientists. Public health actions were taken to protect and safeguard the public's health before it was compromised. Stokinger, the chair of the American Council of Governmental Industrial Hygienists (ACGIH) Threshold Limit Value Committee, commented that levels should be adjusted by a safety factor from 100 to 500 for carcinogens [69]. This would have meant that once the carcinogenicity of asbestos was established, the TLV to protect from cancer due to asbestos ought to have been at least 100 times less than the 5-mppcf TLV intended to lower the incidence of asbestosis. Asbestos was suspected as a carcinogen as early as the 1930s and, even according to Paustenbach et al.'s narrow standard, its link to lung cancer was confirmed no later than the mid-1950s. However, regulatory action was slow to develop after this initial recognition because asbestos product manufacturers engaged in a successful campaign to forestall ACGIH classification of asbestos as a carcinogen [47]. As Stokinger puts it:

> The TLV's are industry's values. But industry generally does not develop anywhere near enough of kinds and amounts of data that can be used for establishing a TLV of a new substance. But industry has the sole responsibility to develop data on its own products; government is not in a position to develop enough facilities to handle the problem in total, nor should it, when reliable toxicologic consultants are now available [69].

Although Dr. Paustenbach has testified in litigation that "[he did not] think anybody from a scientific body standpoint put forward an occupational exposure limit [for asbestos] that they thought would eliminate the increased risk of lung cancer or mesothelioma," Paustenbach et al. fail to disclose the fact that asbestos TLVs did not apply to carcinogens in their review [70]. As John Wells, the medical director of Uniroyal, noted at the 1964 Selikoff conference:

> But as far as a safe level of asbestos dust is concerned, our own conclusion in Hogansville, Ga., is that there is no safe level. The safe level is nil and anything above the safe level represents certain risk [71].

Therefore, Paustenbach et al.'s arguments that are based on a lack of exposure measurements, dose-response, or fiber type are spurious after 1955, when they acknowledge that the relevant scientific community determined that asbestos was a carcinogen. By 1965, the fact that there was no "safe" level of exposure to asbestos became painfully obvious as the medical literature was inundated with mesothelioma cases not only in miners and manufacturing workers but in end users such as brake mechanics and insulators and also in workers' wives, children, siblings, and others who lived in the vicinity of asbestos factories [12].

CONCLUSION

Dennis Paustenbach is the President and Founder of ChemRisk, a "scientific consulting firm that specializes in using risk assessment methods to characterize and provide improved understanding of complex exposures" [72]. Paustenbach has outlined and described the business model for his company and consulting: "I've followed a model of trying to learn about the substance, share what I've learned in the published literature, and if they ask, testify about it" [73]. Paustenbach marketed this model in a speech to the Asbestos Information Association: "These kinds of $250,000, $500,000 investments [in ChemRisk research] go a long way. If you've got 100 cases and it takes $4 million for the lawyers and consultants to get ready for the case and to take it to trial, you can see this is a drop in the bucket" [74]. Thus corporate investments in research will generate "scientific papers" that companies can use to avoid regulation and compensation. This is a perversion of the scientific method, which, if used correctly, does not promise particular results. He also explained the magnitude of the benefits:

> There are over 60,000 cases in the United States . . . involving brake dust. It's a serious litigation matter . . . recently, most of the time the cases are not lost, but last December, just three months ago there was a 30 million dollar verdict on a meso with a brake mechanic. $30 million. $30 million. A single meso case. Even though there was other exposures in his life. So you can lose a brake dust case. And I think the more data you have the better off you are, and it's best to get the approval of your colleagues in the scientific community [74].

Awareness of knowledge production that is designed to serve corporate interests has increased over the past decade, leading courts and the public to place greater scrutiny on corporate-funded science [5, 75]. As a result some consultants have modified their business plans to avoid this critique. Paustenbach now partially or completely funds his own research; he then markets this research (through letters) to companies engaged in lawsuits and solicits consulting arrangements to use the resultant knowledge production to defend law suits [76].

During a 2008 cross-examination Paustenbach stated:

> I've always, like so many Americans, I felt sorry for the underdog, so I felt there were times in the environmental movement when there was a redistribution of wealth that was inappropriate . . . So I thought scientists had a responsibility to talk about these matters, and that's what I've chosen to do [73].

It is important to note that Paustenbach conducted research on hundreds of toxins for over a hundred companies (see Appendix 3).

The production of knowledge designed to serve corporate interests can stifle regulatory efforts and affect public health policy by creating doubt about the association between exposures to toxic substances and disease. Corporate experts call for a false epistemology that requires a continually changing, arbitrary set of criteria to establish a cause-effect relationship, which is always one set of information beyond the available information. When animal studies are missing the consultants call them *a key data gap* that precludes public health intervention and, of course, compensation. On the other hand, when this type of information is available the corporate consultants demand epidemiologic studies *reducto ad absurdum*; as in this case, Paustenbach et al. call for epidemiologic studies that use exact measurements of historical exposures that were never measured. Proponents of public health must be aware of the revisionist techniques of corporations and companies hired to generate scientific findings that serve their economic interests. These findings are presented to create the appearance of sound science to persuade the courts and regulators to deny claims of harm and/or the need for regulatory action. Public health advocates need to be well prepared to invalidate such purported scientific findings. This report has provided a framework for deconstructing revisionist scientific reports prepared on behalf of corporate interests.

Editor's Note: Dr. Egilman serves as an expert witness to injured workers and to asbestos product manufacturing companies in asbestos tort cases.

Appendix 1 follows

APPENDIX 1: Information Omitted by Paustenbach et al.

Source	Year	Information
American Industrial Hygiene Association [77]	1958	*Guidelines*. Cited brake linings as source of asbestos exposure.
Ford Motor Company "Fibrous Asbestos Emissions" [78]	1971	*Report prepared for the State of Illinois*. Part of a deliberation on banning asbestos. In the summary, Ford stated that "[i]nhalation of fibrous asbestos has been considered the source of asbestosis and mesothelioma. . . ." Ford also notes: "Semi-met linings are superior in performance to traditional brake-linings (about equivalent in terms of cold-wear and noise), but the cost penalty is severe, $1.25/car just for front-end brakes."
B. Newman "Perilous Particles," *Wall Street Journal* [79]	1972	The article begins with a description of mechanics blowing dust out of brake parts and states, "What the mechanics don't seem to know—like thousands of other mechanics—is that the brown dust is dangerous. And if a mechanic breathes in enough dust from worn out linings it could eventually kill him."
Ed Drislane Executive Director Friction Manufacturers' Standards Institute [50]	1972	*Internal document*. In a letter to J. H. Kelly of Bendix Corporation, Ed Drislane stated, "[w]hen customers of yours drill linings, chamber linings, cut linings, or grind linings, they may very well raise the asbestos concentration in the atmosphere to above the OSHA standard [of 5 f/cc]." He went on to state that "if a customer of yours started drilling or grinding without having proper dust collectors, he would probably be in violation of the OSHA standard. It therefore becomes your resonsibility, as a supplier of the brake lining, to warn the customer of this possibility."

APPENDIX 1: (Cont'd.)

Source	Year	Information
I. H. Weaver Raybestos-Manhattan Chairman of FMSI Asbestos Study Commission [51]	1973	*Address Delivered at the Annual Meeting of the Friction Materials Standards Institute*. " Many other elements of the OSHA Regulations came out more favorably toward industry than the recommendations that were submitted by NIOSH and the Advisory Committee, and stiff resistance by industry will be needed to prevent OSHA from strengthening the regulations in months to come. . . . I was exceedingly surprised when the final OSHA Standard came out in favor of considerably milder wording. [The OSHA Advisory Committee had recommended using the word "danger" on asbestos labels, which was then downgraded to "caution."] Now I am perplexed that industry resists the OSHA labeling requirement as vigorously as it does."
Daniel Stone Bendix Corporation [80]	1976	*Ballot for Publication in FMSI Catalogs of Recommended Procedures for Brake Service Shops*. Representative of the Bendix Corporation voted in favor of the "Asbestos Study Committee recommending to the Board of Directors the printing of a page in FMSI Catalogs recommending procedures for reducing asbestos dust during brake servicing." However, the representative noted that "the drawback of course is several pennies cost."

APPENDIX 2:
Excerpted from 1975 NIOSH Bulletin: "Current Intelligence Bulletin 5 Asbestos: Exposure During Servicing of Motor Vehicle Brake and Clutch Assemblies" [52]

. . . Data was presented by investigators from the Mount Sinai School of Medicine in New York City indicating that workers engaged in the maintenance and repair of automobile and truck brake linings are exposed to potentially hazardous levels of airborne asbestos dust.

The present findings indicate that enough asbestos is preserved to produce significant exposures during certain brake servicing procedures.

The full extent of asbestos-related disease in brake servicing personnel is not known at present because this particular occupational group has not been studied systematically up to now. *However, a review of the scientific literature on the association between asbestos exposure and mesothelial tumors of the pleura peritoneum has revealed at least four cases of these rare tumors in persons who were employed in jobs involving automobile brake servicing.* (Italics added)

The environmental studies of brake lining servicing operations outlined above together with observations of mesothelial tumors in persons so employed affirms the necessity for instituting and maintaining recommended control measures in this industry so that the health hazards of asbestos are minimized. . .

Recommended (Interim) Procedures for Asbestos Brake and Clutch Servicing

1. If possible, an area shall be designated for all brake and clutch repairs. Entrances into this area shall be posted with an asbestos exposure warning sign as follows:

Asbestos
Dust Hazard
Avoid Breathing Dust
Wear Assigned Protective Equipment
Do Not Remain in Area Unless Your Work Requires It
Breathing Asbestos Dust May Cause Asbestosis and Cancer

APPENDIX 3:
Paustenbach's "Underdogs" and Toxic Substances

Paustenbach's "Underdogs" [81]

3M, Abbott Laboratories, Aerojet, Air Products, Alcoa, Allergan, Allied Signal, Amchem, American Petroleum Institute, American Paper Institute, Amoco, Apple Computers, ARCO, Beazer Corporation, Boeing, Borg Warner, Bucyrus,

Buffalo Pumps, Caterpillar, Chemtura, Champion Paper, Cheese Institute, Chemical Manufacturers Association, Chevron Chemical Company, Chevron Land & Development, Chrysler, Coltec Industries (Garlock), Dow Chemical, Eli Lilly (chemicals and asbestos), Exxon, Fermenta, First Federal, Fischer Scientific, FMC, Food Trade Association, Ford Land & Development, Ford Motor Company, Formaldehyde Institute, GTX, General Dynamics, General Electric, General Telephone, Georgia Pacific, Gould Ind., GSF Industries, GSX Inc., Halogenated Solvents Industry Alliance (HSIA), Hawker (n/k/a Hawker Beachcraft), Hercules, Hewlett Packard Company, Holly Sugar, Honeywell, Hughes Aircraft, IBM, ICI Americas, Industrial Health Foundation, James River Paper, Johnson & Johnson, John Crane, Joslyn, Koppers, Kaiser Gypsum, Leslie Controls, Lockheed Missiles & Space, Los Alamos, Lucent, Maxus Energy Corporation, McKesson, Mead Paper, Mercedes Benz, Merck, Miles, Mobay, Mobil Oil Corporation, Monsanto, Montrose, NACASSI, Northrop, Northwest Paper and Pulp Association, Occidental Petroleum, Pacific Treatment, Pacific Gas & Electric, Pfizer, Plantronics, PPG Incorporated, Procter & Gamble, Questar, RCA, Reichhold, Rhône Poulenc, R.J. Reynolds, Roche, Rocketdyne, Rockwell, Rogers Seeds, Sandoz Pharmaceutical, Semiconductor Industry Association, Shell, Sidney Regional Transit Authority, Simpson Paper Company, Southern Wood Piedmont, Stauffer Chemicals, Syntex Agribusiness, Texas Instruments, U.S. Steel, Union Carbide, Volkswagen, Vulcan Chemicals, Warren Pumps, Westvaco, Weyerhauser, Yarway, Zeneca.

Toxic Substances on Which Paustenbach has Conducted Research [81]

Asbestos, Asphalt Fumes, BEHP, Benzenes, Beryllium, 1, 3 – Butadiene, Carbon Tetrachloride, Chlorinated Hydrocarbons, Chlorine, Chloroform, Chromium, CIN, Copper Chromium Arsenate, Creosote, DDT, Decabromodiphenyl Oxide, DES, Dicyclopentadinolyron (ferrocene), Diesel Exhaust, Dioxins, Ethylcyanoacrylate, Ethylene Oxide, Fly Ash, Formaldehyde, Furans, Glycol Ethers, Hydrogen Sulfide (Selexol), Lead, Mercury, Methyl Alcohol, Methyl bromide, Methyltertiary-butyl ether (MTBE), Methylene Chloride, Nitroglycerine, Nitrosamines, PBDE, Perfluorooctonoic Acid (PFOA), Pesticides, Petroleum Products, Phosphorus (radionucleotides), PFOS, Polychlorinated biphenyls (PCBs), Polycyclic Aromatic Hydrocarbons (PAHs), Perfluorooctonoic Acid, Silica, Silver, Styrene, Tetrachloroethylene (PCE), Tobacco, Toulene, Trichloroethylene, Trihalomethane, Vinyl Chloride, VPA, Welding Rods.

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