No. 18-0056

In the
SUPREME COURT OF TEXAS

The Goodyear Tire & Rubber Company,

Petitioner,

v.

Vicki Lynn Rogers, Individually and as Representative of the Estate of Carl Rogers, Natalie Rogers, and Courtney Dugas,

Respondents.

On Petition for Review from the Court of Appeals
Fifth District of Texas, Dallas, Texas

RESPONSE TO PETITION FOR REVIEW

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Pursuant to TEX. R. APP. P. 53.2(a), the following is a complete list of all parties to the trial court's judgment and the names and addresses of all trial counsel and appellate counsel.

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STATEMENT OF THE CASE

Nature of the Case:	Carl Rogers died of mesothelioma after many years of exposure to asbestos at Goodyear's tire manufacturing facility in Tyler. His family and estate brought a wrongful death action against Goodyear under the Texas Workers' Compensation Act, alleging that Goodyear was grossly negligent and therefore liable for exemplary damages under the statute. TEX. LABOR CODE § 408.001(b).
Trial Court:	County Court at Law No. 5 of Dallas County, Texas. Honorable Mark Greenberg, Presiding Judge.
Course of Proceedings:	After pretrial proceedings before the MDL judge for asbestos cases (the Honorable Mark Davidson), the case was tried for three weeks before a Dallas County jury. The jury found that asbestos fibers from the Goodyear Tyler facility were a proximate cause of Rogers's death from mesothelioma; that Goodyear was grossly negligent; that Rogers's wife and daughters were entitled to economic and non-economic damages; and that exemplary damages should be assessed against Goodyear. (CR38-50)
Trial Court's Disposition:	Applied the statutory cap on exemplary damages in TEX. CIV. PRAC. & REM. CODE § 41.008(b) and rendered judgment awarding Plaintiffs a total of $2,890,000 plus post-judgment interest. (CR98-99)
Court of Appeals:	Fifth District of Texas, Dallas, Texas. Panel of Justices Bill Whitehill (author of majority opinion), Douglas Lang, and Ada Brown (author of dissenting opinion).
Court of Appeals' Disposition:	Unanimously affirmed the jury's gross-negligence and causation findings, but held in a divided opinion that the entire amount of the economic damages award was not supported by legally sufficient evidence. Majority suggested a remittitur of $1,740,000, which Plaintiffs accepted.

RESPONSE TO STATEMENT OF JURISDICTION: LACK OF IMPORTANCE

As discussed below in the Summary of Argument and the Arguments in Response, the court of appeals decided this case correctly. There is no error to review. And the issues Goodyear presents in its petition do not have an impact beyond the specific facts of this particular case. The bottom line is that Goodyear engaged in extremely dangerous and irresponsible conduct. It violated OSHA's requirements by failing to monitor, warn, and protect its employees from the dangers of asbestos exposure, and as a direct result, Rogers and three other workers at Goodyear's Tyler facility developed mesothelioma and died.

ISSUES IN RESPONSE

Gross Negligence

Did the court of appeals correctly reject Goodyear's challenge to the jury's finding of gross negligence in Question 2, when:

- Goodyear urges an erroneous standard for evaluating whether its acts and omissions in not monitoring, warning, and protecting its employees from exposure to asbestos involved an "extreme degree of risk";

- Goodyear's narrow focus on the mathematical probability of harm is inconsistent with the statutory language and the law, uses erroneous numbers, and is logically flawed; and

- Goodyear ignores other evidence of the extreme degree of risk to Rogers and his co-workers from being exposed to cancer-causing asbestos in the workplace?

Causation

Did the court of appeals correctly reject Goodyear's challenge to causation, when:

- Goodyear does not challenge the jury's finding in Question 1 that asbestos fibers from its Tyler facility were a proximate cause of Rogers's mesothelioma, but instead claims only that Plaintiffs did not "rule out" radiation as a "plausible alternative cause";

- Goodyear failed to preserve its radiation hypothesis by not raising it in any post-verdict motion and by not raising an appellate challenge to the MDL judge's no-evidence summary judgment that had the effect of "ruling out" radiation as a cause; and

- there is no evidence that the therapeutic radiation administered to Rogers's brain, as opposed to his significant exposure to asbestos at Goodyear's facility, was even a "plausible" cause of his mesothelioma, much less a mutually-exclusive "alternative" cause that had to be ruled out?

STATEMENT OF FACTS AND PROCEDURAL HISTORY

The court of appeals' opinion accurately summarizes the facts relating to Carl Rogers's work history at Goodyear's tire manufacturing facility in Tyler, the sources of the asbestos to which he was exposed, Goodyear's decades-long awareness of the dangers of asbestos exposure, and Goodyear's failure to monitor, warn, and protect its employees from exposure to asbestos. (Op. at 2-8) The opinion also accurately describes the pre-trial proceedings before MDL Judge Davidson and the trial and post-trial proceedings before Judge Greenberg. (Op. at 8-9, 17-18) Notably, Goodyear's petition does not challenge these aspects of the opinion; accordingly, this response will not discuss them further.

But Goodyear's statement of facts makes several points that do warrant response because they are irrelevant, misleading, or incomplete:

(1) Goodyear makes a point of emphasizing Rogers's cigarette smoking. (Pet. at 1, 2) But the evidence is uncontroverted that cigarette smoking does not cause mesothelioma. (5RR44; 9RR76; 18RR164) It does not even increase the risk of developing mesothelioma. (9RR76)

(2) Goodyear claims that Rogers received "huge amounts" of radiation when he was successfully treated for lung cancer. (Pet. at 2) But there is no evidence that the type and amount of radiation be received—therapeutic radiation to his brain and diagnostic radiation to his chest and abdominal area—are a cause

of mesothelioma. (3Supp.CR2038-39, 2080-81; 5Supp.CR3208-09) There is no question, however, that asbestos exposure causes mesothelioma, and Goodyear does not deny that long before Rogers was treated for lung cancer, he was exposed to levels of asbestos at the Tyler facility that were more than sufficient to cause the mesothelioma that took his life.

(3) Goodyear mentions that it "started taking asbestos samples in the late 1970s." (Pet. at 1) But it fails to mention that the Tyler facility did *no* monitoring of the asbestos levels of *any* employee until March 1983, nearly *eleven years* after OSHA's monitoring requirements went into effect. (PX872[App. 1]; PCX4 at 11-12, 64, 67) Even when monitoring finally began, the Tyler facility took a total of only 26 personal samples in its entire history (PX872; PCX4 at 11-12)—and none were from the tire builders who operated asbestos-emitting machines and worked under asbestos-emitting pipe insulation (PX872; 12RR62, 76, 78-79; PCX4 at 12; PCX5 at 6-7, 11). And the asbestos removal to which Goodyear refers (Pet. at 1) did not begin until the mid-1980s (15RR60)—far too late to do any good for Rogers and his three co-workers, all of whom died from mesothelioma after being exposed to asbestos at the Tyler facility (15RR 94-97; PCX7).

(4) Goodyear makes a special point of emphasizing that Rogers's family sued 17 other defendants and settled with some of them. (Pet. at 2) But it fails to

disclose that the settlements totaled less than $450,000 (17RR124)—reflecting the reality that Goodyear bears the lion's share of responsibility for causing Rogers's death.

(5) Goodyear claims that Judge Davidson's no-evidence summary judgment relating to its radiation hypothesis focused solely on "the defensive theory of sole cause" and "the need to show risk-doubling." (Pet. at 3-4) That is not so: Plaintiffs' summary-judgment motion and Judge Davidson's order were broader, and focused on the absence of any causal relationship between therapeutic radiation to the brain and the subsequent development of mesothelioma, especially in an individual who previously was exposed to substantial doses of asbestos. (1Supp.CR182-87; 8Supp.CR4826) Judge Davidson's order thus effectively "ruled out" Goodyear's radiation hypothesis before the trial began. And importantly, Goodyear did not mention that order in its appellant's brief, much less challenge it on appeal.

SUMMARY OF THE ARGUMENT: REVIEW IS UNWARRANTED

Gross negligence: In rejecting Goodyear's "1 in 45,000" argument, the court of appeals correctly recognized that the "extreme degree of risk" element of gross negligence does not turn exclusively on the probability of harm, but also on the severity of the act or omission and the magnitude of the harm. The court properly applied these settled principles to the specific evidence in this case, which

showed that Goodyear violated OSHA's requirements by failing to monitor, warn, and protect its employees from the well-known dangers of asbestos exposure, and caused Rogers and three other workers at the Tyler facility to die from mesothelioma. Further review of this fact-intensive determination is not necessary.

Causation: Goodyear's unorthodox twist on causation—not contesting that asbestos exposure caused Rogers's mesothelioma but nonetheless claiming that Plaintiffs did not "rule out" radiation as a "plausible alternative cause"—is also unworthy of review. Goodyear waived its radiation argument in both the trial court and the appellate court. In any event, there is no evidence that the therapeutic radiation administered to Rogers's brain was even a "plausible" cause of his mesothelioma, much less an "alternative" cause that is mutually exclusive of the asbestos to which Rogers indisputably was exposed.

ARGUMENTS IN RESPONSE

I. The Court of Appeals Correctly Held that the Evidence Supports the Jury's Finding of Gross Negligence by Goodyear.

Focusing exclusively on the "probability" consideration under the objective prong of gross negligence, *see* TEX. CIV. PRAC. & REM. CODE § 41.001(11)(A), Goodyear argues that Rogers did not face "an extreme degree of risk" from working at its Tyler facility because:

- the "background risk" of an average person getting mesothelioma is 1 in 1,000,000 (Pet. at 9);

- Rogers's risk of getting mesothelioma was 1 in 45,000 based on the estimated amount of asbestos he was exposed to at the Tyler facility (*id*.); and
- a 1 in 45,000 risk is not "extreme" because it is lower than the risk of "being hit by lighting or drowning in a bathtub" (*id*. at 9-10).

As the court of appeals correctly held, this myopic focus on the mathematical probability of outcomes is inconsistent with the statutory definition of gross negligence, the law, and simple logic. And contrary to Goodyear's contention (Pet. at 6), it is Goodyear's own argument, not the court of appeals' opinion, that ignores the evidence—particularly, of the extreme degree of risk facing workers in general, and Rogers and his co-workers in particular, from being exposed to cancer-causing asbestos in the workplace.

A. Goodyear's Emphasis on Mathematical Probabilities Is Inconsistent With the Statutory Language, the Law, the Facts, and Common Sense.

The statute and the law: The first of many deficiencies in Goodyear's analysis of the "extreme degree of risk" element is that it focuses exclusively on the probabilistic "likelihood" of injury, but ignores the statutory language requiring consideration of both the "act or omission" that created the risk and the "magnitude" of the potential harm. Each of these statutory components is equally important; they operate in tandem to determine whether a given risk from certain conduct is sufficiently "extreme" to warrant a finding of gross negligence. Thus,

courts are more likely to find gross negligence when the defendant's acts and omissions are severe and the magnitude of potential harm is great, even though the probability of the injury might be low or simply unascertainable.[1]

The relationship between these three statutory components is reflected in a trio of cases from this Court affirming jury findings of gross negligence. In *Mobil Oil Corp. v. Ellender*, for example, the Court examined Mobil's conduct—"not monitoring contract workers for benzene exposure, not warning them of the danger of such exposure, and not providing them with protective gear"—and held that the conduct "involved an extreme degree of risk." 968 S.W.2d 917, 923 (Tex. 1998). Notably, the Court did not discuss the probability of injury to the plaintiff or his co-workers from the benzene exposure. The Court reached a similar result in *Lee Lewis Construction, Inc. v. Harrison,* holding that a contractor's failure to provide lifelines to subcontractors working on the outside of a multi-story building "created an extreme risk of a fatal fall," even though the probability of such a fall was not discussed. 70 S.W.3d 778, 785-86 (Tex. 2001). And more recently, the Court held

[1] That, of course, explains why the court of appeals' discussion of the objective prong properly focused on Goodyear's failure to do any sampling or to provide any warnings at its Tyler facility, and the fact that four of its workers out of 3000 died from mesothelioma. (Op. at 11-12) Far from a "*non sequitur*" (Pet. at 7), these facts are highly relevant to the objective prong's focus on both the defendant's "act or omission" and the "magnitude of the potential harm." Without consideration of these facts, the "probability" component would improperly be "viewed in a vacuum." (Op. at 10)

in *Columbia Medical Center of Las Colinas v. Hogue* that a hospital's failure to provide "stat" echocardiogram services to its physicians created an extreme degree of risk that a patient could die, despite testimony that the need for echocardiograms on a stat basis "is uncommon." 271 S.W.3d 238, 252 (Tex. 2008).

By emphasizing the severity of the conduct at issue and the magnitude of the potential harm, these cases also expose the flaw in Goodyear's argument that "tiny risks" are an unavoidable part of daily life. (Pet. at 9-10) In contrast to the risks Goodyear describes—"being hit by lighting, drowning in a bathtub, or getting cancer from eating charbroiled steak every week" (Pet. at 9-10), the risks of dying from mesothelioma caused by asbestos exposure at a workplace are extreme because they are entirely avoidable *if the employer simply obeys the law*. Thus, if any hypothetical illustrates when a risk can be extreme even though the probability of injury might be low, it is the one posed to Goodyear's expert on cross examination:

> Q. [I]f you were . . . planning to go to a NASCAR stadium to watch . . . a race, and you knew that out of those 250,000 people, someone was going to randomly shoot one bullet into the stadium . . . and hit one person, would you still go?
>
> . . .
>
> A. No, I wouldn't. I tend to be a little conservative in my choice of risks.

(16RR37) As this answer confirms, a risk can be extreme as long as the act is sufficiently egregious and the consequences sufficiently severe, even though the probability of the harm occurring might be low or even unascertainable.

And contrary to Goodyear's suggestion, this Court has never reversed a finding of gross negligence based solely upon a mathematical calculation of a probabilistic outcome. Goodyear's reliance on *Wal-Mart Stores, Inc. v. Alexander* is misplaced because the outcome there turned not just on the unlikelihood of serious injury from tripping on a small "ridge" in a parking lot, but also on the absence of any evidence that the ridge was "highly dangerous." 868 S.W.2d 322, 327 (Tex. 1993). And not only is Goodyear's "West Nile virus case" *not* a gross-negligence case, but it also involved a much lower probability and magnitude of harm, and a risk of injury that was beyond the employer's control. *Union Pac. R. Co. v. Nami*, 498 S.W.3d 890, 892-93, 898-99 (Tex. 2016). Neither of these cases—nor any other Goodyear cites (Pet. at 10)—suggests any categorical rule about the probability of a particular risk, especially in an asbestos-mesothelioma case in which the conduct is highly dangerous, the link between exposure and the disease is undisputed, and the magnitude of harm is catastrophic.

The facts: The legal flaws in Goodyear's probabilistic-outcome approach are compounded by its use of contrived numbers. For one thing, Goodyear's "1 in 45,000" figure is not based on any evidence relating to the "extreme degree of

risk" component of gross negligence. Rather, it comes from Plaintiffs' proof of substantial-factor causation, in which they quantified the approximate dose of asbestos fibers to which Rogers was exposed at the Goodyear facility, and then established that this exposure more than doubled his risk of developing mesothelioma. *See Bostic v. Georgia-Pacific Corp.*, 439 S.W.3d 332, 353 (Tex. 2014). Not only is this exposure evidence irrelevant to the "extreme degree of risk" component of gross negligence, but it is also a highly conservative measure of causation because it focused on only 10 of the 30 years that Rogers worked for Goodyear and assumed that he was exposed to asbestos fibers from only two of the tire-building machines around him instead of the actual number of eight. (5RR83-84, 88; 7RR21, 96)

Moreover, by focusing solely on Plaintiffs' causation proof, Goodyear has consistently ignored some of the most compelling evidence in this case of how its acts and omissions created an *actual and unacceptably high probability* that workers at the Tyler facility would die from asbestos-caused mesothelioma. This evidence—a 2007 study of workers at Goodyear's Tyler facility, co-authored by one of Goodyear's experts in this case—revealed that *three other employees* at the facility also died of mesothelioma, thus increasing by *9.6 times* the risk that a worker at the facility would develop mesothelioma. (15RR95-97; PCX7[App. 2]) And with four mesothelioma victims out of roughly 3,000 employees over the

years at issue (PCX7; 8RR92), the odds a worker at Goodyear's Tyler facility will develop mesothelioma are *1 in 750*—the true "probability" of potential harm from Goodyear's failure to monitor, warn, and protect its workers from asbestos exposure.[2]

Lack of logic: A simple example illustrates the illogic in Goodyear's myopic reliance on statistical probabilities to prove (or disprove) the extreme degree of risk created by certain conduct. When a person is killed or injured by a drunken driver, no one would dispute that the driver's act of becoming intoxicated and operating a vehicle involves an extreme degree of risk. But under Goodyear's singular focus on statistical probabilities, the driver would be able to claim that the odds of a pedestrian or someone in another vehicle being killed by a drunken driver are just 1 in approximately 60,000. (*See* App. 3, 4) In light of these odds—roughly the same odds Goodyear relies on here—would any court hold that driving while intoxicated does not involve an extreme degree of risk merely because the "probability" of someone dying is statistically low? Of course not.

[2] Predictably, Goodyear argues that these odds are skewed because two of the four victims previously worked at a local factory that manufactured asbestos insulation. (15RR97; 18RR99-105; PCX7) Putting aside the fact that one victim worked there only four months *(id.*), Goodyear's experts admitted that even two mesothelioma victims would indicate an increased risk in a workplace the size of the Tyler facility (16RR34; 17RR54; 18RR154).

B. Goodyear Ignores the Evidence that Its Acts and Omissions Involved an Extreme Degree of Risk that Employees Exposed to Asbestos Would Die from Mesothelioma.

Goodyear has also consistently failed to acknowledge other indisputable facts establishing the extreme degree of risk from not monitoring, warning, or protecting workers from asbestos exposure:

- the link between asbestos exposure and mesothelioma cannot be questioned—"it's complete," as one expert testified (9RR54);
- people develop mesothelioma from low levels of asbestos exposure (PX816, 822, 823, 839, 846; 15RR112; 16RR104-05, 138-39);
- mesothelioma always results in death, and the time between diagnosis and death is very short (5RR41; 11RR37); and
- OSHA and the scientific and medical community do not recognize any "safe" level of asbestos exposure below which there is no risk of developing mesothelioma (9RR55, 78, 12RR67, 152; 15RR113-14; 18RR162).

Given these indisputable facts, even Goodyear's corporate representative had to admit that asbestos exposure—at least at "certain levels"—involves an extreme degree of risk. (12RR80)

But there is much more. As long ago as the 1930s, industrialists were warned about the risk of illness and death from asbestos exposure, and were told to use substitute products, to isolate workers, to educate and train people about the hazard, to install exhaust ventilation, and to provide protective equipment. (5RR107-12) The concern about the link between asbestos exposure and

mesothelioma became especially heightened in the 1960s. (15RR115-16, 118) For example, a digest that Goodyear received summarized a 1966 study showing that "[t]he rapidly increasing number of cases of fatal mesothelioma . . . have been definitely traced to exposure in asbestos dust. The degree of exposure in many cases is slight . . . . Preventive measures recommended are restriction of use and greater precaution in the handling of all types of asbestos." (PX816 at p. 19 [App. 5])

Recognizing these serious risks of harm, the federal government stepped in. On June 7, 1972, OSHA published detailed regulations establishing a "Standard for Exposure to Asbestos Dust." (PX839 [App. 6]) In the preamble, OSHA stated in no uncertain terms:

> *In view of the undisputed grave consequences from exposure to asbestos fibers, it is essential that the exposure be regulated now* on the basis of the best evidence available now, even though it may not be as good as scientifically desirable. An asbestos standard can be reevaluated in the light of the results of ongoing studies, and future studies, *but cannot wait for them. Lives of employees are at stake*.

(PX839, emphasis added) This directive confirms the extreme degree of risk in this case. And coupled with the other evidence discussed above, it supports a firm belief or conviction that Goodyear's acts and omissions, when viewed objectively, created an extreme degree of risk that employees at the Tyler facility would die from asbestos-caused mesothelioma. The court of appeals was correct in so holding, and further review of its fact-specific conclusion is unwarranted.

II. The Court of Appeals Correctly Held that Goodyear's Challenge to Causation Based on Its Radiation Hypothesis Is Unpreserved and Unmeritorious.

Though couched as a challenge to Plaintiffs' proof of "causation" (Pet. at x), Goodyear's second issue does not actually challenge the sufficiency of the evidence supporting the jury's finding in Question 1 that "asbestos fibers from the Goodyear Tyler facility were a proximate cause of Carl Rogers' mesothelioma that resulted in his death." (CR42) Nor could it, because the evidence clearly and convincingly shows that Rogers's only exposure to asbestos was at the Goodyear facility, and the dose of asbestos fibers to which he was exposed more than doubled the risk of developing mesothelioma. Goodyear's "causation" argument thus has a different twist—that Plaintiffs purportedly failed to "rule out" the radiation treatment Rogers underwent for lung cancer as a "plausible alternative cause" of the mesothelioma he developed ten years later. (Pet. at 16-19) But this twist on the concept of causation has fatal flaws, both procedural and substantive.

A. Goodyear's Post-Verdict Motions Failed to Preserve a No-Evidence Challenge to Causation Based on Its Radiation Hypothesis.

Goodyear spends several paragraphs making the unremarkable point that a party can preserve a no-evidence challenge with a "simple statement that no evidence supports the finding" at issue. (Pet. at 14-15) Thus, Goodyear claims that it "preserved its sufficiency complaint about causation" by stating in its JNOV

motion that "[t]he jury's finding regarding Question 1 was unsupported by legally sufficient evidence and is contrary to the conclusive evidence." (Pet. at 13, quoting CR1171)[3] But this discussion raises a false issue because it misconstrues the reason why the court of appeals determined that Goodyear failed to preserve its no-evidence challenge based on its radiation hypothesis—namely, that argument was *different* from the one it raised in the trial court.

As Goodyear acknowledges, Question 1 asked whether "asbestos fibers from the Goodyear Tyler facility were a proximate cause of Rogers' mesothelioma." (Pet. at 13) There is no question that Goodyear's post-trial motions challenged the sufficiency of the evidence supporting the jury's affirmative answer to that question—arguing that Plaintiffs failed to prove a level of asbestos exposure that was sufficient to cause Rogers's mesothelioma. (*See* CR103-05, 1158-64, 1171) But none of these motions asserted the *different* challenge Goodyear chose to raise on appeal—that Plaintiffs' evidence was insufficient to prove causation, *not* because of a failure to establish a certain level of asbestos exposure, but because of their failure to "rule out" the radiation treatment Rogers received as a "plausible

[3] Goodyear also suggests that it preserved a no-evidence complaint by "getting a definitive ruling" from the MDL judge on its motion to exclude the testimony of Plaintiffs' experts. (Pet. at 11) But a pretrial motion to exclude evidence is not one of the four ways in which a no-evidence challenge to a jury finding is preserved. *See T.O. Stanley Boot Co., Inc. v. Bank of El Paso*, 847 S.W.2d 218, 220 (Tex. 1992).

alternative cause" of the mesothelioma. Goodyear thus violated a cardinal rule for preserving error: "[T]he objection to the trial court must comport with the argument made on appeal," and "[a]n objection on appeal that is not the same as that urged at trial presents nothing for review." *Basic Energy Service, Inc. v. D-S-B Properties, Inc.*, 367 S.W.3d 254, 264 (Tex. App.—Tyler 2011, no pet.) (citing cases). This preservation principle is well-established and unremarkable.

Nor can Goodyear avoid its preservation problem by invoking footnote 2 of its JNOV motion, which merely incorporated by reference "the arguments it has otherwise preserved" through "prior briefs and motions." (Pet. at 13, citing CR1159 n.2) This footnote fails to satisfy another "cardinal rule for preserving error"—"that an objection must be clear enough to give the trial court an opportunity to correct it." *Arkoma Basin Expl. Co., Inc. v. FMF Assocs. 1990-A, Ltd.*, 249 S.W.3d 380, 387 (Tex. 2008). Given the voluminous "prior briefs and motions" that were filed in this case—and handled by the MDL judge—the vague and uninformative footnote in Goodyear's JNOV motion was in no way "clear enough" to give the different judge who presided at trial an opportunity to consider the specific failure-to-rule-out radiation argument that Goodyear later raised on appeal. *Arkoma Basin*, 249 S.W.3d at 387.

B. Goodyear Also Failed to Challenge on Appeal the MDL Judge's Summary Judgment that Ruled Out Radiation as a Cause.

Although the court of appeals could have overruled Goodyear's radiation-causation argument based solely on its failure to preserve error at the trial level, the court went on to "assum[e] Goodyear preserved its legal insufficiency complaint" but to "overrule it" on an independent basis. (Op. at 17) Specifically, in addressing Goodyear's contention that Plaintiffs' experts "did not rule out radiation," the court held: "In making this argument, Goodyear ignores the fact that [Plaintiffs] got a summary judgment ruling from the MDL judge that there was no scientifically valid epidemiology to create a causal relationship between therapeutic radiation for lung cancer and mesothelioma. *Goodyear has not challenged the summary judgment ruling in this appeal and did not mention it in its opening brief."* (Op. at 18, emphasis added) Notably, Goodyear's petition does not complain about this finding of appellate waiver, and its failure to do so provides yet another reason for denying review.

At most, Goodyear's petition rehashes an argument it raised for the first time in its reply brief below—that the MDL judge's summary-judgment order has no bearing on Goodyear's legal-insufficiency challenge because the order focused on whether radiation was a "sole cause" of mesothelioma (requiring "risk-doubling" according to Goodyear) rather than a "plausible cause" (requiring a standard less than 2.0 according to Goodyear). (Pet. at 17) Goodyear offers no support for these

purported standards, but more importantly, it mischaracterizes the summary-judgment proceeding. Neither Plaintiffs' summary-judgment motion nor the MDL judge's order was limited to Goodyear's "sole cause" defense or the failure to prove "risk doubling"; instead, they were based on the absence of *any* causal relationship between therapeutic radiation to the brain and the subsequent development of mesothelioma, especially in an individual who previously was exposed to substantial doses of asbestos. (1Supp.CR182-87; 8Supp.CR4826) The order thus had the purpose and effect of "ruling out" Goodyear's radiation hypothesis before the trial began. And Goodyear did not even mention that order in its appellant's brief, much less challenge it on appeal.

C. There Is No Evidence that the Therapeutic Radiation to Rogers's Brain Was Even a "Plausible" Cause of His Mesothelioma, Much Less an "Alternative" Cause.

Finally, even if Goodyear had preserved its radiation hypothesis in the trial and appellate courts, it is wrong in claiming that "Rogers' massive radiation exposure is a plausible alternative cause that plaintiffs failed to exclude." (Pet. at 18) In fact, there is no evidence that radiation therapy of the type, frequency, and duration Rogers received was even a "plausible" cause of his mesothelioma. And there is certainly no evidence that it was an "alternative" cause—*i.e.*, something that would negate any causal connection between the asbestos to which Rogers was exposed and the mesothelioma he developed. Indeed, because Goodyear has

not disputed that Plaintiffs satisfied the *Bostic* requirements for proving that Rogers's asbestos exposure was a substantial factor in causing his mesothelioma, then the radiation at most could only be a *contributing* or *additional* cause—not a mutually-exclusive alternative cause—and therefore did not have to be "ruled out." *Bostic*, 439 S.W.3d at 345 (citing doctrine of "multiple causes" in RESTATEMENT (SECOND) OF TORTS § 432(2) (1965)).

But no matter how Goodyear's radiation hypothesis is characterized—as a "plausible," "alternative," "contributing," or "additional" cause—the fatal flaw in the argument is that it rests on a mere generality that "radiation can cause mesothelioma" (Pet. at 18), but ignores three specific and critical facts *in this case*:

- Rogers's therapeutic radiation was to his brain, not his chest (where mesothelioma develops) or even his abdomen;
- the only radiation he received in the area of his chest and abdomen was diagnostic, not therapeutic, consisting of x-rays and CT scans—none of which has a proven link to mesothelioma; and
- long before Rogers received therapeutic radiation to his brain, he was exposed to significant doses of asbestos at Goodyear's Tyler facility.

Consistent with these facts, none of Plaintiffs' experts testified that therapeutic radiation to the brain and x-rays to the chest can be the cause of mesothelioma in people who were previously exposed to asbestos. (4Supp.CR2705-17) Similarly, Goodyear's experts could not say that the dose of radiation Rogers purportedly received—as opposed to the dose of asbestos to

which he was exposed—was the cause of his mesothelioma. (3Supp.CR2038-39, 2080-81; 5Supp.CR3208-09) And none of the studies Goodyear cites (Pet. at 18-19) draw any link between mesothelioma and therapeutic radiation to the brain; instead, all of them deal with radiation to or near the same area of the body where mesothelioma develops as a treatment for diseases like lymphoma and breast cancer. Even more importantly, none of these studies rule out asbestos as a contributing cause of mesothelioma in cases where the decedents were exposed to both asbestos and radiation; in fact, they identify asbestos exposure as a co-factor. (*See, e.g.*, 4Supp.CR2576, 2585, 2596)

In short, Goodyear failed to show that radiation to the brain is even a "plausible" cause of mesothelioma, let alone an "alternative" cause that is mutually exclusive of the asbestos to which Rogers was exposed. Further review of Goodyear's twist on causation is unwarranted.

PRAYER

Respondents respectfully request that Goodyear's petition for review be denied.

Respectfully submitted,

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CERTIFICATE OF COMPLIANCE

1. This response complies with the type-volume limitation of TEX. R. APP. P. 9.4(i)(2)(D) because it contains 4,476 words, excluding the parts of the response exempted by TEX. R. APP. P. 9.4(i)(1).

2. This response complies with the typeface requirements of TEX. R. APP. P. 9.4(e) because it has been prepared in a proportionally spaced typeface using Microsoft Word 2010 in 14-point Times New Roman font (and 13-point for footnotes).

/s/ Jeffrey S. Levinger

Jeffrey S. Levinger

CERTIFICATE OF SERVICE

The undersigned certifies that a copy of this Response to Petition for Review was served on all counsel of record via the Court's electronic filing system on this 6th day of September, 2018.

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APPENDIX

TAB 1

Plaintiffs' Exhibit
GTR-KS 43

CHAT Data - Tyler, Tx

Compound	Location	Conc		Date
AMMONIA	BEAD STRIP MILL	0.01	PPM	02/27/78
AMMONIA	TRAIN CAL 1 CAL RO	0.01	PPM	02/27/78
AMMONIA	TRAIN CAL INTER MI	0.01	PPM	02/27/78
AMMONIA	TRAIN CAL 2 CAL RO	0.01	PPM	02/28/78
AMMONIA	TRAIN CAL CENT ROL	0.01	PPM	02/28/78
AMMONIA	TRAIN CAL FEED MIL	0.01	PPM	02/28/78
AMMONIA	TRAIN CAL INTER MI	0.01	PPM	02/28/78
AMMONIA	TRAIN CAL INTER MI	0.01	PPM	02/28/78
AMMONIA	TRAIN CAL MILL 1	0.01	PPM	02/28/78
AMMONIA	TRAIN CAL MILL 2	0.01	PPM	02/28/78
AMMONIA	#1 FAB CAL MILL	25.00	PPM	07/27/82
AMMONIA	FAB CAL FEED MILL	5.00	PPM	07/27/82
AMMONIA	FAB CAL HOT DRUMS	5.00	PPM	07/27/82
ASBESTOS	CONTRACTOR	3.05	F/CC	07/29/78
ASBESTOS	CONTRACTOR	0.23	F/CC	07/29/78
ASBESTOS	CONTRACTOR	0.58	F/CC	07/29/78
ASBESTOS	PIG-310 THOMS STON	1.00	PRCNT	01/17/80
ASBESTOS	PIG-433A THIELE	1.00	PRCNT	01/17/80
ASBESTOS	PIG-448 DIAMD MICA	1.00	PRCNT	01/17/80
ASBESTOS	PIGMENT 471 PPG	1.00	PRCNT	01/17/80
ASBESTOS	PIG 117 MILWHIT1443	0.20	PRCNT	10/28/80
ASBESTOS	MAINT ELECTRICIAN	0.01	F/CC	03/22/83
ASBESTOS	MAINT ELECTRICIAN	0.12	F/CC	03/23/83
ASBESTOS	MAINT ELECTRICIAN	0.06	F/CC	03/23/83
ASBESTOS	MAINT ELECTRICIAN	0.26	F/CC	03/23/83
ASBESTOS	MAINT ELECTRICIAN	0.20	F/CC	03/23/83
ASBESTOS	MAINT ELECTRICIAN	0.01	F/CC	06/29/83
ASBESTOS	AISLE #48 TIRE MACH	0.01	F/CC	08/21/84
ASBESTOS	AISLE #55 TIRE MACH	0.01	F/CC	08/21/84
ASBESTOS	RENOVATION CURING	0.03	F/CC	08/21/84
ASBESTOS	S G/H PRESSES	0.01	F/CC	08/21/84
ASBESTOS	CURE TIRES A-LINE	0.06	F/CC	02/22/85
ASBESTOS	CURE TIRES B-LINE	0.06	F/CC	02/22/85
ASBESTOS	CURE TIRES B-LINE	0.03	F/CC	02/23/85
ASBESTOS	CURE TIRES C-ROW	0.04	F/CC	02/23/85
ASBESTOS	CURE TIRES A-ROW	0.09	F/CC	02/24/85
ASBESTOS	CURE TIRES A-ROW	0.14	F/CC	02/24/85
ASBESTOS	CURE TIRES A-ROW	0.33	F/CC	02/24/85
ASBESTOS	CURE TIRES A-ROW	0.47	F/CC	02/24/85
ASBESTOS	CURE TIRES A-ROW	0.24	F/CC	02/24/85
ASBESTOS	CURE TIRES A-ROW	0.09	F/CC	02/24/85
ASBESTOS	CURE TIRES A-ROW	0.47	F/CC	02/24/85
ASBESTOS	CURE TIRES A-ROW	0.14	F/CC	02/24/85
ASBESTOS	CURE TIRES A-ROW	0.03	F/CC	02/25/85
ASBESTOS	REMOVE PRESS DOMES	0.12	F/CC	02/25/85
ASBESTOS	REMOVE PRESS DOMES	0.08	F/CC	02/25/85
ASBESTOS	TIRE MACHINE #21	0.01	F/CC	02/25/85
ASBESTOS	REMOVE PRESS DOMES	0.04	F/CC	02/26/85
ASBESTOS	REMOVE PRESS DOMES	0.05	F/CC	02/26/85
BENZENE	CEMENT HOUSE	0.20	PRCNT	05/12/77

TT 472

CHAT Data - Tyler, Tx

Compound	Location	Conc		Date
AMMONIA	TRAIN CAL INTER MI	0.01	PPM	02/27/78
AMMONIA	BEAD STRIP MILL	0.01	PPM	02/27/78
AMMONIA	TRAIN CAL 1 CAL RO	0.01	PPM	02/27/78
AMMONIA	TRAIN CAL FEED MIL	0.01	PPM	02/28/78
AMMONIA	TRAIN CAL 2 CAL RO	0.01	PPM	02/28/78
AMMONIA	TRAIN CAL INTER MI	0.01	PPM	02/28/78
AMMONIA	TRAIN CAL MILL 2	0.01	PPM	02/28/78
AMMONIA	TRAIN CAL CENT ROL	0.01	PPM	02/28/78
AMMONIA	TRAIN CAL MILL 1	0.01	PPM	02/28/78
AMMONIA	TRAIN CAL INTER MI	0.01	PPM	02/28/78
AMMONIA	FAB CAL HOT DRUMS	5.00	PPM	07/27/82
AMMONIA	FAB CAL FEED MILL	5.00	PPM	07/27/82
AMMONIA	#1 FAB CAL MILL	25.00	PPM	07/27/82
ASBESTOS	CONTRACTOR	0.23	F/CC	07/29/78
ASBESTOS	CONTRACTOR	3.05	F/CC	07/29/78
ASBESTOS	CONTRACTOR	0.58	F/CC	07/29/78
ASBESTOS	PIG-433A THIELE	1.00	PRCNT	01/17/80
ASBESTOS	PIGMENT 471 PPG	1.00	PRCNT	01/17/80
ASBESTOS	PIG-310 THOMS STON	1.00	PRCNT	01/17/80
ASBESTOS	PIG-448 DIAMD MICA	1.00	PRCNT	01/17/80
ASBESTOS	PIG 117 MILWHITI443	0.20	PRCNT	10/28/80
ASBESTOS	MAINT ELECTRICIAN	0.01	F/CC	03/22/83
ASBESTOS	MAINT ELECTRICIAN	0.06	F/CC	03/23/83
ASBESTOS	MAINT ELECTRICIAN	0.26	F/CC	03/23/83
ASBESTOS	MAINT ELECTRICIAN	0.12	F/CC	03/23/83
ASBESTOS	MAINT ELECTRICIAN	0.20	F/CC	03/23/83
ASBESTOS	MAINT ELECTRICIAN	0.01	F/CC	06/29/83
ASBESTOS	S G/H PRESSES	0.01	F/CC	08/21/84
ASBESTOS	AISLE #55 TIRE MACH	0.01	F/CC	08/21/84
ASBESTOS	AISLE #48 TIRE MACH	0.01	F/CC	08/21/84
ASBESTOS	RENOVATION CURING	0.03	F/CC	08/21/84
ASBESTOS	CURE TIRES B-LINE	0.06	F/CC	02/22/85
ASBESTOS	CURE TIRES A-LINE	0.06	F/CC	02/22/85
ASBESTOS	CURE TIRES B-LINE	0.03	F/CC	02/23/85
ASBESTOS	CURE TIRES C-ROW	0.04	F/CC	02/23/85
ASBESTOS	CURE TIRES A-ROW	0.09	F/CC	02/24/85
ASBESTOS	CURE TIRES A-ROW	0.09	F/CC	02/24/85
ASBESTOS	CURE TIRES A-ROW	0.47	F/CC	02/24/85
ASBESTOS	CURE TIRES A-ROW	0.14	F/CC	02/24/85
ASBESTOS	CURE TIRES A-ROW	0.33	F/CC	02/24/85
ASBESTOS	CURE TIRES A-ROW	0.47	F/CC	02/24/85
ASBESTOS	CURE TIRES A-ROW	0.24	F/CC	02/24/85
ASBESTOS	CURE TIRES A-ROW	0.14	F/CC	02/24/85
ASBESTOS	REMOVE PRESS DOMES	0.08	F/CC	02/25/85
ASBESTOS	REMOVE PRESS DOMES	0.12	F/CC	02/25/85
ASBESTOS	CURE TIRES A-ROW	0.03	F/CC	02/25/85
ASBESTOS	TIRE MACHINE #21	0.01	F/CC	02/25/85
ASBESTOS	REMOVE PRESS DOMES	0.05	F/CC	02/26/85
ASBESTOS	REMOVE PRESS DOMES	0.04	F/CC	02/26/85
BENZENE	CEMENT HOUSE	0.20	PRCNT	05/12/77

Tyler Database Department 511 (02/06/90)

Compound	Location	Concentration	Date	TLV
ASBESTOS	AISLE #55 TIRE MACH	0.01 F/CC	08/21/84	0.2 fib/cc *
	AISLE #48 TIRE MACH	0.01 F/CC	08/21/84	0.2 fib/cc *
	TIRE MACHINE #21	0.01 F/CC	02/25/85	0.2 fib/cc *
BENZENE	GREEN TIRE REPAIR 2	0.10 PPM	07/01/77	10 ppm *
	GREEN TIRE REPAIR	0.16 PPM	02/26/78	10 ppm *
	TIRE BUILDER CONV	0.05 PPM	04/07/78	
	GREEN TIRE REPAIR	0.45 PPM	04/07/78	
	GREEN TIRE INSPECT	0.15 PPM	01/18/79	10 ppm *
	GREEN TIRE INSPECT	0.13 PPM	01/18/79	
	GREEN TIRE REPAIR	0.16 PPM	02/26/79	
	INSP/REPR GRN TIRES	0.03 PPM	01/20/83	10 ppm *
	INSP/REP GREEN TIRE	0.03 PPM	01/20/83	
	INSP/REPR GRN TIRES	0.03 PPM	01/20/83	
		0.05 PPM	01/20/83	
	INSP/REP GREEN TIRE	0.03 PPM	01/20/83	
	INSP/REPR GRN TIRES	0.03 PPM	11/15/83	
	INSP/REPR GRN TIRES	0.03 PPM	11/15/83	
	INSP/REPR GRN TIRES	0.03 PPM	11/16/83	
	INSP/REPR GRN TIRES	0.03 PPM	11/16/83	
	INSP/REPR GRN TIRES	0.03 PPM	11/16/83	
	INSP/REPR GRN TIRES	0.11 PPM	11/16/83	
	INSP/REPR GRN TIRES	0.22 PPM	11/17/83	
	INSP/REPR GRN TIRES	0.03 PPM	11/17/83	
	INSP&REPGGRN TIRES	0.03 PPM	11/13/84	10 ppm *
1,3-BUTADIENE	TIRE BLDR MACH#12	0.03 PPM	07/17/84	(1000 ppm) **
	TIRE BLDR MACH #12	0.04 PPM	07/18/84	
	TIRE BLDR MACH #12	0.04 PPM	07/19/84	
HEPTANE	INSP/REPR GRN TIRES	0.23 PPM	11/15/83	400 ppm
	INSP/REPR GRN TIRES	0.64 PPM	11/15/83	
	INSP/REPR GRN TIRES	0.18 PPM	11/16/83	
	INSP/REPR GRN TIRES	0.29 PPM	11/16/83	
HEXANE	GREEN TIRE REPAIR	10.78 PPM	02/26/78	100 ppm ***
	GREEN TIRE INSPECT	9.22 PPM	01/18/79	(100 ppm)
	GREEN TIRE INSPECT	9.07 PPM	01/18/79	
	GREEN TIRE REPAIR	10.78 PPM	02/26/79	
	INSP/REPR GRN TIRES	0.68 PPM	11/15/83	50 ppm
	INSP/REPR GRN TIRES	1.20 PPM	11/15/83	
	INSP/REPR GRN TIRES	0.38 PPM	11/16/83	
	INSP/REPR GRN TIRES	0.34 PPM	11/16/83	
ISOPROPYL ALCOHOL	INSP/REPR GRN TIRES	0.78 PPM	01/20/83	400 ppm
	INSP/REPR GRN TIRES	1.62 PPM	01/20/83	
NUISANCE DUST	#1 INSPECTION STATION	0.52 MG/M3	11/12/74	10 mg/m^3 total dust
RUBBER SOLVENT NAPHTHA	INSP/REPR GRN TIRES	29.20 MG/M3	01/20/83	1600 mg/m^3
	INSP/REPR GRN TIRES	43.80 MG/M3	01/20/83	
	INSP&REPGGRN TIRES	14.64 MG/M3	11/13/84	1600 mg/m^3

* TLV for Asbestos depends on mineral type ie Amosite = 0.5 fib/cc, Chrysotile = 2 fib/cc, Crocidolite = 0.2 fib/cc and all other forms = 2 fib/cc (all > 5 um in length)

** Notice of Intended Changes (for 1984-85) 1, 3-Butadiene - 10 ppm

*** Notice of Intended Changes (for 1979) Hexane - 25 ppm

TAB 2

Mortality and Cancer Incidence Among Tire Manufacturing Workers Hired in or After 1962

Colleen Beall, DrPH
Morton Corn, PhD
Hong Cheng, PhD
Robert Matthews, BS
Elizabeth Delzell, SD

Objective: This study evaluated mortality during 1962 through 2003 and cancer incidence during 1995 through 2003 at a tire manufacturing plant. ***Methods:*** *The mortality study included 3425 men and women, employed for at least one year. Of these, 3069 were eligible for the cancer incidence study.* ***Results:*** *Employees experienced 390 deaths compared with 608 expected (standardized mortality ratio (SMR) = 64; 95% confidence interval (CI) = 58–71). Total cancer mortality (123 observed, SMR = 75, CI = 62–89) and lung cancer mortality (47 observed, SMR = 72, CI = 53–96) were lower than expected. Hourly white men had small increases in stomach cancer, bladder cancer, and leukemia deaths. During 1995 through 2003, 169 incident cancers were observed compared with 197 expected (SIR = 86, 95% CI = 74–100). Three mesothelioma cases occurred among hourly white men (SIR = 653, CI = 135–1907); all were exposed potentially to asbestos before starting at the rubber plant.* ***Conclusions:*** *Small numbers and limited information on jobs, occupational agents, and lifestyle preclude attribution of observed increases to workplace exposures.* (J Occup Environ Med. *2007;49:680–690*)

The International Agency for Research on Cancer (IARC) has determined that work in the rubber industry is carcinogenic to humans.[1,2] The evidence of a causal relation is strongest for bladder cancer and is at least suggestive for leukemia, stomach cancer, and lung cancer.[1–4] Epidemiologic assessment of specific agents or work activities within the industry have indicated that bladder cancer was associated with potential exposure to antioxidants contaminated with aromatic amines and with jobs in materials preparation, tire building, maintenance or storage operations, and leukemia, with exposure to solvents and work in materials preparation, tire curing, and storage.[4,5] Although the specific aromatic amine (β-naphthylamine) possibly responsible for the bladder cancer excess was removed from the manufacturing environment by 1960,[4,6] some studies of rubber workers hired after 1960 reported an excess of this cancer.[5,7] Also, rubber workers hired in or after 1950 may have increased risks of leukemia, lung cancer, and stomach cancer,[5,8–10] although the evidence is not consistent or conclusive.[7]

Carlo et al[11] previously reported the mortality of 2306 men employed for a year or more at the Goodyear Tire and Rubber Company's tire manufacturing plant in Tyler, Texas. The plant opened in 1962. During a period of follow-up through 1989, Carlo et al observed 102 deaths from all causes combined, compared with 192 deaths expected based on comparisons with US mortality rates, and reported 24 observed, compared with

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DOI: 10.1097/JOM.0b013e318074bb39

37 expected, cancer deaths. The present study expanded the original cohort to include 3425 men and women who worked at the plant for at least one year between 1962 and the end of 2003. The study also evaluated cancer incidence among 3069 employees who lived in Texas in or after 1995, the earliest year for which statewide incidence data were available.

Materials and Methods

The Institutional Review Board at the University of Alabama at Birmingham approved the research and monitored process.

Plant Description and Exposure Monitoring Data

The plant includes operations of compounding and mixing, processing (extrusion for tread formation, cutting/winding, beading), assembly, curing, and inspection.[12] Extensive local exhaust and general dilution ventilation was part of the original design and was built into the facility in 1962. Ventilation has been continuously improved with process modifications. Major process changes and upgrading and expansion occurred in the mid 1980s. Currently, approximately 25,000 truck and passenger vehicle tires per day are produced.

Industrial hygiene considerations and oversight have been present since plant inception. Early approaches to exposure monitoring at the plant used departments, or areas of particular interest, as the nucleus of worker selection for sampling. In recent years, homogenous exposure groups and random sampling of employees for scheduled sampling has been used in a more sophisticated, defensible approach to scientifically based air sampling.[11]

Air sampling records for the years 1972 to 1988 were previously analyzed as part of the study by Carlo et al.[11] We updated the data with personal air sampling results collected since 1988. Both the original and the updated data included area samples, as well as personal air samples, ie, samples collected in the breathing zone of the worker. The personal samples were collected for full eight-hour shifts. A sample of completed air sampling, chain of custody, and analytical laboratory forms was examined on-site and found to be complete. We compared air sampling results for 45 analytes in 1972 to 1988 and for 43 in 1988 to 2006, exclusive of noise, to threshold limit values (TLVs) or permissible exposure limits (PELs). A total of 3503 eight-hour time-weighted average personal samples for analytes (exclusive of noise) are included in the two Goodyear databases: 1972 to 1988 (1192 samples) and 1988 to 2006 (2311 samples). Additional, non-eight-hour personal samples and area samples existed but were not included in the results. The criterion for using a TLV or PEL was to select whichever was lower, ie, more stringent. In instances where a PEL or TLV did not exist for an analyte, professionals at the plant adopted a criterion. For example, for nitrosamines, Goodyear used 2.5 μg/M^3. Germany's Technical Rule for Dangerous Materials 552, as an internal guideline for processes such as curing.

Mortality Study Subjects, Follow-Up, and Analysis

Subjects for the retrospective follow-up study were employees of the Tyler tire plant who worked for at least one year during the period 1962 through 2003 and who had records containing information on birth date, gender, social security number (SSN), and plant hire date. Follow-up was from 1963 through 2003. Follow-up ended in 2003 because this was the most recent year for which mortality data were available from national sources when we conducted record linkages to determine vital status and causes of death.

To identify subjects, we compiled an employee list using data from the study by Carlo et al[11] and from electronic data in the Goodyear Employee Management System (GEMS) and cross-checked the list with hardcopy employment records stored at the Tyler tire plant. For each subject, we developed information on name, SSN, birth date, gender, race, plant hire and separation dates, and, where applicable, death date and cause of death. Data from these sources identified 4879 employees who were hired at Tyler before 2003 and who had valid SSNs and hire and separation dates. Detailed data on all jobs held while working at the plant were available for 2851 (58%) of these employees. We did not develop detailed work histories on all employees because this information was not available from the previous study, because GEMS contained incomplete job histories for many employees and because we lacked resources to obtain and abstract all plant employee records. We classified workers' pay status as always salaried if their available work history was comprised entirely of salaried jobs, or if they had no work history and Carlo et al classified them as salaried. Employees with any hourly job or with an hourly pay status in the Carlo et al study were classified as ever hourly.

For the employees with detailed work histories, we calculated duration of employment as the sum of the time spent working in each job. For the remaining employees, we estimated duration of employment as the difference between their first hire and separation dates. Using these data, we identified 3425 employees, who worked for at least 1 year by the end of 2003, as eligible for the mortality study. Of these, detailed data on all jobs were available for 1704 (50%).

Information on race was not available for 252 eligible employees. For 117 of these, we assigned race based on the school they had attended (N = 51) (indicated on employment applications) or based on the personal recollection of long-term active employees at the plant (N = 66). For race assignments based on school,

we contacted the applicable school or Board of Education to ascertain if the school of interest was segregated during the years an employee attended and, if so, to what race the student belonged. We assumed that the remaining employees with unknown race were white because the majority (76%) of employees with known race were white, and we included these employees in all analyses. We were unable to determine pay status for a small number of employees and excluded these employees from separate analyses of hourly and salaried subgroups.

Information on vital status as of December 31, 2003, came from Goodyear sources, from the previous study,[11] and from linkages with the Social Security Administration and the National Death Index (NDI). In all, we confirmed the vital status as alive for 2887 (84%), deceased for 390 (12%), and unknown for 148 (4%). For employees who died before 1979, we retrieved death certificates and coded the underlying cause of death according to the International Classification of Diseases (ICD) code in effect at the time of death. For subjects who died in or after 1979, NDI furnished causes of death, coded using the ICD revision in effect on the death date. We were unable to determine the cause of death of 3 (1%) of the 390 decedents.

Analyses considered all employees and subgroups specified by gender, race, years since hire, years worked, and pay status. Using the standardized mortality ratio (SMR) as the measure of association, we compared employees' overall and cause-specific mortality rates with the mortality rates of the general population of the 15 counties within a 50-mile radius of the plant; including Anderson, Camp, Cherokee, Franklin, Gregg, Harrison, Henderson, Hopkins, Kaufman, Rains, Rusk, Smith, Upshur, Van Zandt, and Wood. To compute the expected numbers of deaths, we accumulated person-years of observation for each subject into race- and gender-specific 5-year age and calendar time categories beginning when the employee had worked for 1 year. Person-year accumulation ended on the earlier of the study closing date (December 31, 2003) or the date of death. We assumed that employees with unknown vital status were alive at the end of 2003. When there were at least three observed or expected deaths, we computed SMRs and their exact 95% confidence intervals (CIs). To reduce the dilution of any true association between occupational factors and cancer resulting from the inclusion of relatively short-term or recently hired employees, we restricted some analyses to employee subgroups with 20 or more years since hire and 10 or more years worked.

We also carried out comparisons of employees' mortality rates with those of the general population of the entire state of Texas or the entire United States. We mention the results of these analyses only briefly, as the population of the region is likely to be a more appropriate general population comparison group.

Cancer Incidence Study Subjects, Follow-Up, and Analysis

The cancer incidence study was limited to 3069 employees who lived in Texas in or after 1995, the earliest year for which state-wide cancer incidence data were available. Assessment of eligibility for the cancer incidence study required the development of residential histories.[14] GEMS provided residential history for employees who were actively working, and for other employees we obtained their post-employment residential history from LexisNexis, a private vendor.

We identified incident cancer cases through record linkage with the Texas Cancer Registry (TCR). We counted as cases all invasive cancers identified among eligible employees between 1995 and the end of 2003, if the diagnosis date occurred after accruing 1 year of employment and occurred when the employees were living in Texas.

Analyses compared employees' cancer incidence rates during 1995 through 2003 with those of the general population of the 15-county region surrounding the plant, using the standardized incidence ratio (SIR) as the measure of association. Person-year accumulation began on the later of the TCR inception date (January 1, 1995) or the date on which an employee accrued 1 year of employment, and it ended on the earliest of the study closing date, the date of last residence in Texas, or the death date. The TCR provided general population rates for the 15-county region surrounding the plant.

Results

Exposure Data

Exposure data included a total of 3503 personal samples for analytes, exclusive of noise. During 1972 to 1988, 10 (0.84%) of 1192 samples exceeded the TLV or PEL, and during 1989 to 2006, 11 (0.48%) of 2311 personal samples exceeded the TLV or PEL (Table 1). Measured concentrations were predominantly at least 96% lower than the TLV or PEL.

Mortality

The total group of 3425 employees eligible for the mortality study included 2488 white men (73%), 691 nonwhite men (20%), 150 white women (4%), and 96 nonwhite women (3%) (Table 2). The pay status was hourly for 2806 (82%), salaried for 589 (17%), and unknown for 30 (<1%). Employees had 79,281 person-years of follow-up, and median values were 1973 for year of hire, 29 years for age at hire, 11 for years worked, and 28 for years since hire.

Overall, employees at the plant had 390 observed, compared with 608 expected, deaths (SMR = 64, CI = 58–71), indicating that their overall mortality rate was 36% lower

TABLE 1

Agent Measured in the Goodyear Plant, Permissible Exposure Limit (PEL) or Threshold Limit Value (TLV) Used for Comparison, Total Number of Samples, and Number of Samples Above the PEL/TLV, by Time Period*

	1972–1988			1988–2006		
		Number of samples			Number of samples	
Agent	PEL/TLV†	Total‡	>PEL/TLV§	PEL/TLV	Total‡	>PEL/TLV§
1,3-Butadiene	1000 ppm	27		1 ppm		
Acetaldehyde	200 ppm			200 ppm	13	
Acrylonitrile	2 ppm	1				
Acetone	750 ppm			500 ppm	113	
Ammonia	25 ppm					
Aniline	2 ppm					
Asbestos	2 fibc	7				
BD dibenzo chrysene	None			1 ppm	25	
Benzene	10 ppm	186		0.5 ppm	116	
Branched hexanes	500 ppm	125				
Butyl cellosolve	25 ppm	1				
Calcium carbonate	15 mg/M³	1				
Carbon black	3.5 mg/M³	4		3.5 mg/M³	11	
Carbon disulfide				4 ppm	47	
Carbon monoxide	50 ppm			25 ppm (1992)	48	7
Carbon tetrachloride	10 ppb/5 ppb in 1990					
Cellosolve				5 ppm	2	
Cellosolve acetate	100 ppm	4		5 ppm	7	
Cyclohexanone	50 ppm	2				
Diacetone alcohol				50 ppm	6	
Ethanol	1000 ppm			1000 ppm	110	
Formaldehyde	0.75 ppm	29		0.75 ppm	104	
Hawkax				No regulation	5	
Hexylene glycol	None					
Hydrochloric acid				No regulation	5	
Iron oxide				5 mg/M³	1	
ISOL				200 ppm	115	
Isoprene				No regulation		
Isopropanol	400 ppm	54	1			
Lead				0.5 mg/M³		
MEK (methyl ethyl ketone) (butanone)	200 ppm	33		200 ppm	110	
Methyl cellosolve	25 ppm	6				
Methyl chloride	50 ppm					
Methylcyclohexane	400 ppm			400 ppm	115	
MIBK (Hexone) (methyl isobutyl ketone)	50 ppm	5		50 ppm	110	
Mineral spirits	350 ppm	17	1			
N-Butyl acetate	150 ppm	1				
N-Heptane	400 ppm	87		400 ppm	119	
N-Hexane	50 ppm	7	1	50 ppm	117	
Nitrobenzene	1 ppm					
Nitrosamines	2.5 µg/M³‖	159		2.5 µg/M³‖	311	
Nuisance particulates (total nuisance dust)	15 mg/M³, 10 mg/M³	41	4	10 mg/M³	24	2
Ortho-Toluidine	2 ppm					
Ozone	0.1 ppm			0.05 ppm	4	
Petroleum naphtha	GY used 1350 mg/M³	39	1			
Phenol	5 ppm	2		5 ppm	25	
Resorcinol	10 ppm	4		10 ppm	30	
Respirable Particulates	10 mg/M³ (1978–80); 5 mg/M³ (1980–87)	35	2	3 mg/M³	64	2
Silica (quartz)	10 mg/M³, 0.1 mg/M³	7				
Styrene	50 ppm	28				
Thiram				1 mg/M³	3	
Toluene	50 ppm	131		50 ppm	122	

(Continued)

TABLE 1
Continued

Agent	1972–1988 PEL/TLV†	1972–1988 Number of samples Total‡	1972–1988 Number of samples >PEL/TLV§	1988–2006 PEL/TLV	1988–2006 Number of samples Total‡	1988–2006 Number of samples >PEL/TLV§
Total hydrocarbons	1350 mg/M³¶	27		1350 mg/M³¶	118	
1,1,1-TCE (Trichloroethane)	350 ppm	8		350 ppm	110	
TRRF (total respirable rubber fumes)¶				0.75 mg/M³¶	91	
Welding fumes				5 mg/M³	1	
Xylenes	100 ppm	116		100 ppm	116	

mg/M³, milligrams per cubic meter of air; f/cc, fibers > 5 μm length per cubic centimeter of air; ppm, parts per million parts of air; μg/M³, micrograms per cubic meter of air; GY, Goodyear.

†A blank cell in the PEL/TLV column means that samples for that analyte were not taken during that period or that a PEL/TLV did not exist.

‡A blank cell in the total number of samples column indicates that personal eight-hour time-weighted average samples were not obtained. Area, ceiling or less than eight-hour samples may have been taken.

§A blank cell in the >PEL/TLV column means that all samples were below the PEL/TLV.

¶A PEL/TLV used by GY and having the following sources: for nitrosamines, Technical Rule for Dangerous Materials 532-Germany; for total hydrocarbons, an internal GY standard; for TRRF, an internal GY guideline derived from an internal project comparing the UK standard for cyclohexane soluble materials (0.6 mg/M³) to TRRF collected with a cyclone.

TABLE 2
Characteristics of Employees at the Tyler Plant Mortality Study Group

Characteristic	White men* N	White men* (%)	Nonwhite men N	Nonwhite men (%)	White women† N	White women† (%)	Nonwhite women N	Nonwhite women (%)	Total‡ N	Total‡ (%)
All employees	2488	(73)	691	(20)	150	(4)	96	(3)	3425	(100)
Hourly	2017	(72)	627	(22)	77	(3)	85	(3)	2806	(100)
Salaried	448	(76)	58	(10)	69	(12)	11	(2)	589	(100)
Unknown	25	(84)	1	(3)	4	(13)	0	(0)	30	(100)
Deaths	316	(81)	58	(15)	11	(3)	4	(1)	390	(100)
Person-yr, 1952–2003	61,024	(77)	14,162	(18)	2685	(4)	1210	(1)	79,284	(100)
Median year of hire	1972		1976		1981		1980		1972	
Median age at hire (yrs)	28		29		31		32		29	
Median yr worked at Tyler	11		16		6		11		11	
Median yr since hire	30		22		21		14		28	

*Includes 115 men with unknown race.

†Includes 20 women with unknown race.

‡Includes 135 employees with unknown race.

than that of the regional general population (Table 3). Cancer mortality was lower than expected overall (123 deaths, SMR = 75, CI = 62–89). Employees had a statistically significant deficit of deaths from colorectal cancer (4 observed, SMR = 28, CI = 8–73) and from lung cancer (47 deaths, SMR = 72, CI = 53–96). Results for other specific forms of cancer were unremarkable and included totals of four observed stomach cancer deaths (SMR = 97, CI = 26–248), three bladder cancer deaths (SMR = 132, CI = 27–384), and five leukemia deaths (SMR = 95, CI = 31–233). Of the observed deaths, 375 (96%) were in men (SMR = 63, CI = 57–70), and 15 were in women (SMR = 90, 50–148). Women had eight cancer deaths overall compared with 5.5 expected (SMR = 146, CI = 63–288), and fewer than three deaths from any specific cancer. All further results pertain to male employees.

SMRs for all causes of death and for all cancers were considerably lower for nonwhite men (all causes: SMR = 42, CI = 32–54; all cancers: SMR = 28, CI = 14–52) than for white men (all causes: SMR = 70, CI = 63–78; all cancers, SMR = 85, CI = 70–103). Separate analyses for white and nonwhite men did not find a statistically significant excess of any cause of death. Also, SMRs tended to be lower for salaried (all causes: 49 observed, SMR = 47, CI = 35–63; all cancer: 15 observed, SMR = 50, CI = 28–83) than for hourly (all causes: 321

TABLE 3
Observed Number of Deaths, SMR* and 95% Confidence Interval (CI) for Men and Total Employees at the Tyler Manufacturing Facility With Follow-Up Through 2003, Using Regional† Comparison Rates

	Men			Total		
Cause of Death‡	Obs	SMR	95% CI	Obs	SMR	95% CI
All causes	375	69	57–70	390	64	58–71
All cancer	115	72	60–87	123	75	62–89
Esophagus	5	105	34–246	5	105	34–244
Stomach	4	99	27–253	4	97	26–248
Colorectum	4	29	8–75	4	20	6–73
Liver	1	21	1–118	2	42	5–150
Pancreas	8	98	42–193	8	95	41–187
Larynx	2	[2.0]	-	2	[2.0]	—
Lung	45	70	51–94	47	72	53–96
Prostate	6	84	31–183	6	84	31–183
Melanoma of skin	4	127	35–324	5	165	50–381
Bladder	3	134	28–391	3	132	27–384
Kidney	3	62	13–182	3	61	13–179
Central nervous system	4	74	20–190	4	72	20–185
Non-Hodgkin lymphoma	5	85	28–199	5	83	27–194
Leukemia	5	99	32–230	5	95	31–223
Multiple myeloma	3	136	28–396	3	132	27–385
Other cancer§	13	60	32–103	17	71	41–113
Diabetes	6	42	15–90	6	40	15–87
Cerebrovascular disease	13	55	30–96	14	58	32–99
Heart disease	103	61	50–74	106	61	50–74
NMRD	16	52	31–83	16	51	30–80
Cirrhosis of liver	9	71	33–135	9	70	32–133
Nephritis & nephrosis	1	29	1–163	1	28	1–157
AIDS	4	68	18–174	5	84	27–196
External causes	67	66	51–84	68	66	51–84
Other known causes	36	53	37–74	36	52	36–72
Unknown	3			3		

*Expected number is provided in brackets, without the SMR and the 95% confidence interval, when the observed number and the expected number of deaths were both <3.

†Regional rates include rates of 15 counties within a 50-mile radius of Tyler, TX.

‡NMRD, non-malignant respiratory disease; AIDS, Acquired Immunodeficiency Syndrome.

§Other cancer sites included mouth (1), pharynx (1), peritoneum, (1), retroperitoneum, (1), small intestine (1), mesothelioma, (2), soft tissue (1), skin (1), male breast (1), female breast (2); uterus (1); unspecified (4)

observed, SMR = 67, CI = 60–75; all cancer: 99 observed, SMR = 78, CI = 63–95) male employees. Neither group had a large or statistically significant increase in observed, compared with expected, deaths from any cause.

Because most deaths occurred among hourly male employees and because SMRs differed by race and pay status, we restricted further analyses to hourly male employees and present results separately for all hourly men and for hourly white men (Table 4). Small increases in observed, compared with expected, numbers of deaths from stomach cancer, bladder cancer, and leukemia occurred among hourly men and were restricted to hourly white men. These increases were concentrated in the subgroup of hourly white men, who had 20 or more years since hire and 10 or more years worked, for stomach cancer (3 observed, SMR = 347, CI = 72–1015) and leukemia (4 observed, SMR = 256, CI = 70–656), but not for bladder cancer (1 observed vs 0.9 expected). All results were based on fewer than five observed and expected deaths, and none was statistically significant. Results for lung cancer mortality were unremarkable.

SMRs computed using expected numbers based on Texas or US general population rates were higher than SMRs based on regional rates for most causes of death. However, analyses using Texas and US comparison groups, like those using the regional comparison group, did not identify a statistically significant excess of any cause of death. For the entire cohort, the SMR computed using Texas mortality rates was 76 (CI = 68–83) for all causes of death combined, 95 (CI = 79–113) for all cancers, 99 (CI = 27–255) for stomach cancer, 100 (CI = 73–134) for lung cancer, 164 (CI = 34–481) for bladder cancer, and 105 (CI = 34–245) for leukemia. Hourly white male employees with 20 or more years since hire and 10 or more years of work had Texas-based SMRs of 301 (CI = 62–881) for stomach

TABLE 4
Observed Number of Deaths, SMR* and 95% Confidence Interval (CI) for Selected Causes of Death, for Hourly Men and for Hourly White Men at the Tyler Manufacturing Facility, Overall and for the Subgroups With ≥20 Yr Since Hire (YSH) and ≥10 Yr Worked (YRS), Using Regional Comparison Rates

Cause of Death	All Hourly Men			Hourly Men With ≥20 YSH, ≥10 YRS			Hourly White Men			Hourly White Men With ≥20 YSH, ≥10 YRS		
	Obs	SMR	95% CI	Obs	SMR	95% CI	Obs	SMR	95% CI	Obs	SMR	95% CI
All causes	321	67	60–75	142	64	54–76	267	76	67–86	111	73	60–87
All cancer	89	78	63–95	52	74	55–97	69	94	75–118	46	95	69–127
Esophagus	4	100	26–255	1	[2.1]	—	4	154	42–395	1	[1.3]	—
Stomach	4	190	33–306	3	168	35–492	4	213	58–545	3	347	72–1015
Colorectum	3	27	6–80	2	33	4–119	3	38	8–110	2	49	6–178
Liver	1	28	1–144	1	[2.2]	—	0	[2.6]	—	0	[1.3]	—
Pancreas	5	77	25–179	2	55	7–199	5	104	34–242	2	[2.4]	—
Larynx	2	[1.6]	—	1	[0.9]	—	2	[1.9]	—	1	[0.5]	—
Lung	38	75	53–103	20	69	42–106	36	91	63–126	18	87	51–137
Prostate	6	107	39–233	5	124	40–289	5	140	45–328	4	171	47–439
Melanoma of skin	2	[2.5]	—	2	[1.9]	—	2	[2.5]	—	2	[0.9]	—
Bladder	3	175	36–511	1	[1.1]	—	3	206	42–601	1	[0.3]	—
Kidney	2	53	6–190	1	[2.1]	—	2	64	8–232	1	[1.6]	—
Central nervous system	4	92	25–235	1	[1.9]	—	4	107	29–274	1	[1.8]	—
Non-Hodgkin lymphoma	5	107	35–249	4	158	46–430	3	79	16–231	2	[1.8]	—
Leukemia	5	124	40–289	4	208	57–532	5	148	48–345	4	258	70–658
Multiple myeloma	3	268	35–497	1	[1.1]	—	2	[1.2]	—	1	[0.7]	—
Other cancer	12	89	25–104	3	34	7–100	10	81	39–148	3	52	11–151

*Expected number is provided in brackets, without the SMR and the 95% confidence interval, when the observed number and the expected number of cancers were both <3.

cancer and 308 (CI = 84–790) for leukemia and had one observed, compared with 0.67 expected, death from bladder cancer. This subgroup also had more than the expected number of deaths from lung cancer when compared with the Texas general population (SMR = 131, CI = 78–207), but the Texas-based SMR also was elevated for hourly white male employees with less than 20 years since hire and less than 10 years of work (7 observed, Texas SMR = 222, CI = 89–458) and for those with 20 or more years since hire and less than 10 years of work (7 observed, Texas SMR = 121, CI = 49–250).

The death certificates of two white male hourly employees indicated "mesotheliomas" as the cause of death. Comparison mortality rates were not available to estimate the expected number of mesotheliomas deaths. Data pertaining to the incidence of mesotheliomas are presented later in the paper.

Cancer Incidence

Of the 3425 employees in the mortality study, 3069 (90%) were eligible for the cancer incidence study. Of those eligible, 73% were white men, 20% were nonwhite men, 4% were white women, and 3% were nonwhite women. The pay status was hourly for 2576 (84%), salaried for 474 (15%), and unknown for 19 (<1%). Median values were 1974 for year of hire, 28 for years of age at hire, 12 for years worked, and 28 for years since hire at the plant. Overall, employees in the incidence study had 24,044 person-years of follow-up, or 30% of the person-time included in the mortality study. The proportion of mortality study person-time included in the incidence study was higher (58%) for those with 20 or more years since hire and 10 or more years worked.

Employees had 169 observed and 196 expected incident cancers (SIR = 86, CI = 74–100) (Table 5). Of the observed cases, 161 (95%) occurred among men (SIR = 86, CI = 73–100), and 8 occurred among women (SIR = 99, CI = 43–194). We observed fewer than expected cases of stomach cancer, lung cancer, bladder cancer, and leukemia, overall and among men; none of these deficits was statistically significant. Nonstatistically significant increases in observed, compared with expected, cases were seen among men for cancer of the pancreas (6 cases, SIR = 122, CI = 45–267), mesothelioma (3 cases, SIR = 475, CI = 98–1389), kidney cancer (9 cases, SIR = 118, CI = 54–225), non-Hodgkin lymphoma (10 cases, SIR = 144, CI = 69–264), and multiple myeloma (5 cases, SIR = 191, CI = 62–446).

Separate analyses of cancer incidence among white men and nonwhite men indicated that 81% of the male cases occurred among the former. The SIR for all cancer was slightly lower for nonwhite (31 observed, SIR = 78, CI = 53–111)

TABLE 5
Observed Number of Cases, SIR* and 95% Confidence Interval (CI) for Men and Total Employees at the Tyler Manufacturing Facility Using Regional Comparison Rates†

	Men			Total		
Type of Cancer	Obs	SIR	95% CI	Obs	SIR	95% CI
All cancer sites combined	161	86	73–100	189	86	74–100
Oral cavity and pharynx	5	66	21–153	5	65	21–151
Esophagus	2	60	7–217	2	59	7–214
Stomach	2	[2.5]	—	2	[2.8]	—
Colorectum	19	88	53–138	20	89	54–138
Liver and biliary	2	[2.7]	—	3	109	23–318
Pancreas	6	120	45–257	6	119	44–258
Larynx	1	24	1–133	1	24	1–132
Lung	32	73	50–104	34	76	53–106
Mesothelioma	3	475	98–1389	3	468	97–1368
Prostate	45	88	63–116	45	86	63–116
Melanoma of skin	4	102	28–262	4	89	27–252
Bladder	7	73	30–151	7	73	29–149
Kidney	9	118	54–225	9	115	53–219
Central nervous system	2	[2.5]	—	2	77	9–278
Non-Hodgkin lymphoma	10	144	69–264	10	138	66–253
Leukemia	3	70	15–205	3	68	14–198
Multiple myeloma	5	191	62–446	5	183	60–428
Other specified cancer‡	1	12	0–68	5	39	13–97
Unknown cancer site	3			3		

*Expected number is provided in brackets, without the SIR and the 95% confidence interval, when the observed number and the expected number of deaths were both <3.
†Regional rates include rates of 15 counties within a 50-mile radius of Tyler, TX.
‡Other cancer sites included soft tissue (1); female breast (3); endometrium (1).

than for white (130 observed, SIR = 87, CI = 73–104) men. The excess of mesothelioma was restricted to white men (3 cases, SIR = 521, CI = 107–1521). Neither white nor nonwhite men had a large or statistically significant increase in any other form of cancer.

Analyses by pay status indicated that hourly male employees had 138 observed and 155 expected cancer cases (SIR = 89, CI = 75–105), whereas the salaried group had 19 observed and 31 expected cases (SIR = 61, CI = 36–95). The increases in pancreatic cancer, mesothelioma, kidney cancer, non-Hodgkin lymphoma, and multiple myeloma among all male employees were limited mainly or entirely to hourly men; the only statistically significant excess in this group was for mesothelioma (3 cases, SIR = 591, CI = 122–1726) (Table 6). All cases of mesothelioma occurred among hourly white men with 20 or more years since hire and 10 or more years worked at the Tyler tire plant (SIR = 966, CI = 199–2823).

The comparison of employees' cancer incidence rates with Texas, rather than regional, rates yielded a Texas SIR of 93 (CI = 80–108) for all cancers combined, and Texas SIRs were below 100 for stomach cancer, bladder cancer, and leukemia. For lung cancer, the Texas SIR was 102 (CI = 71–142). Hourly white male employees with 20 or more years since hire and 10 or more years of work had a Texas SIR of 94 (CI = 73–120) for all cancers, 2 observed, compared with 1.2 expected, stomach cancers; 3 observed, compared with 3.8 expected, bladder cancers; and 2 observed, compared with 1.7 expected, leukemia cases. For lung cancer, the Texas SIR was 124 (CI = 71–201) in this subgroup, based on 16 observed and about 13 expected. There also were slightly more than expected lung cancer cases among hourly white men with less than 20 years since hire and less than 10 years of work (2 observed, 0.7 expected), among those with less than 20 years since hire and 10 or more years worked (1 observed, 0.8 expected) and among those with 20 or more years since hire and less than 10 years of work (6 observed, 5.3 expected).

Discussion

Employees at the Tyler tire plant had mortality rates that, overall, were 36% lower than those of the general regional population. These results were not unexpected and may reflect the possibility that the employees studied were relatively healthy at hire and had socioeconomic advantages over the general population during and after employment. Some other studies of rubber products workers employed during similar time periods have reported similar mortality patterns,[7,15,16] although one study reported somewhat higher mortality rates from all causes and

TABLE 6

Observed Number of Cancers, SIR* and 95% Confidence Interval (CI) for Selected Types of Cancer, for Hourly Men and for Hourly White Men at the Tyler Manufacturing Facility, Overall and for the Subgroups With >20 yr Since Hire (YSH) and ≥10 yr Worked (YRS), Using Regional Comparison Rates

	All Hourly Men			Hourly Men With ≥20 YSH, ≥10 YRS			Hourly White Men			Hourly White Men With ≥20 YSH, ≥10 YRS		
Type of Cancer	Obs	SIR	95% CI	Obs	SIR	95% CI	Obs	SIR	95% CI	Obs	SIR	95% CI
All cancer sites combined	138	89	76–105	90	85	69–105	111	94	77–113	65	87	67–111
Oral cavity and pharynx	4	53	17–162	3	74	15–216	4	82	22–210	3	103	21–302
Esophagus	1	[2.8]	—	0	[1.9]	—	1	[2.1]	—	0	[1.2]	—
Stomach	2	[2.1]	—	2	[1.5]	—	2	[1.4]	—	2	[0.8]	—
Colorectum	17	95	56–153	8	67	29–132	14	102	56–172	6	70	26–153
Liver and biliary	2	[2.3]	—	1	[1.5]	—	1	[1.6]	—	0	[0.9]	—
Pancreas	5	123	40–287	2	[2.8]	—	5	168	55–392	2	[1.8]	—
Larynx	1	29	1–160	0	[2.4]	—	1	[2.4]	—	0	[1.5]	—
Lung	28	81	54–116	18	76	46–118	25	92	60–136	16	90	52–147
Mesothelioma	3	591	122–1726	3	872	180–2549	3	656	135–1916	3	966	199–2823
Prostate	38	91	65–125	27	87	57–126	24	77	49–114	12	58	30–101
Melanoma of skin	2	63	8–227	2	[1.9]	—	2	66	8–239	2	[1.7]	—
Bladder	5	65	21–151	4	78	21–199	4	58	16–149	3	67	14–196
Kidney	8	128	55–251	5	124	40–290	7	140	56–289	4	133	36–341
Central nervous system	2	[2.1]	—	1	[1.2]	—	2	[1.8]	—	1	[1.0]	—
Non-Hodgkin lymphoma	7	122	49–252	5	142	46–331	6	125	46–273	4	141	38–361
Leukemia	2	56	7–203	2	[2.3]	—	2	[2.7]	—	2	[1.6]	—
Multiple myeloma	5	229	74–535	3	190	39–556	4	279	76–714	2	[0.9]	—
Other cancer	1	16	0–88	1	26	1–157	1	18	0–98	1	[2.9]	—
Unknown cancer site	3			2			3			2		

*Expected number is provided in brackets, without the SIR and the 95% confidence interval, when the observed number and the expected number of cancers were both <3.

all cancers among employees hired in or after 1960.[8,9]

In the present study, results for several specific forms of cancer, including bladder cancer, leukemia, stomach cancer, and lung cancer, were of a priori interest, because of previously reported excesses among workers at other rubber industry facilities.[1,2,4] In analyses based on comparisons with the regional general population, we did not observe any positive association between employment at the Tyler tire plant and lung cancer mortality or incidence. Analyses using the Texas or US general population as the comparison group found an increase in lung cancer mortality among hourly white men, but the increase was not concentrated in long-term employees with many years since hire, and it was not statistically significant. Hourly white male employees had slightly increased mortality from stomach cancer, bladder cancer, and leukemia. These results were based on small numbers, they were not clearly supported by cancer incidence results, and they were compatible with chance.

Our cancer incidence results indicated that white male employees had slightly increased rates of pancreatic cancer, kidney cancer, non-Hodgkin lymphoma, and multiple myeloma. Analyses by years since hire and duration of employment were hampered by small numbers but did not suggest that the increases resulted from occupational exposures, and the results for these cancers may be due to chance. Increases in lymphoma deaths have been noted among workers exposed to solvents in the manufacture of rubber footwear and tires,[2] although in one study the association was limited to Hodgkin lymphoma.[17] Pancreatic cancer, kidney cancer, and multiple myeloma have not been consistently associated with work in the rubber industry.[4]

The excess incidence of mesothelioma, a disease caused by exposure to asbestos,[18] among hourly white men in the present study was unexpected. Three cases were observed among long-term employees with many years since hire at the plant. Two of these employees, one with pleural mesothelioma and one with peritoneal mesothelioma, worked for a plant that manufactured asbestos insulation for pipe before starting at the Tyler tire plant. Employees at the pipe manufacturing plant were potentially exposed to high levels of asbestos, and they have sustained a high rate of mesothelioma.[19] The third employee with mesothelioma began working at the Tyler tire plant at age 39 after spending 20 years repairing communications equipment in the Air Force and spending a few months as a welder for pipeline contractors. It is plausible that the three mesothelioma cases were exposed to asbestos before they started working at the Tyler tire plant. Thus, the observed excess of this cancer

among employees of the tire plant may not be causal.

Compared with the original investigation of the Tyler tire plant workers,[13] the present study included approximately 1.8 times more person-years of follow-up and four times as many deaths. Comparisons of the study group's mortality rates with regional and Texas general population rates permitted control for confounding by correlates of geographic region, such as socioeconomic status and smoking patterns.[20] The present study also examined the possible effect of time since hire, a surrogate for potential induction time.

Limitations of the study included the lack of detailed work histories for many employees, absence of information on exposure to specific agents and lack of data on occupational and lifestyle exposures outside of the Tyler tire plant for most employees. Without detailed work histories, we were unable to analyze mortality patterns by job or work area, and we probably misclassified as salaried some employees whose early jobs were hourly. Lack of data on lifestyle exposures impedes the interpretation of patterns observed for certain diseases, such as lung cancer. Our results for lung cancer, particularly the observation of small excesses regardless of duration of employment or time since hire, suggest confounding by smoking, but without appropriate data we cannot confirm this possible explanation.

Due to the small size of the study group, information on the occurrences of rare diseases was sparse, particularly for women and for the subgroup of employees with long potential induction time and long duration of employment. With regard to these considerations, the cancer incidence analyses were more severely limited than were the mortality analyses because the time period for observing cancer incidence patterns was brief. Discrepancies between results from the mortality study and the cancer incidence study could be attributed to temporal restrictions on follow-up for the incidence study that resulted in the loss of cases and person-years accrued before the registry inception date or after a person left the state.[14] The temporal and geographic restrictions imposed by using the TCR as the sole source of information on cancer incidence also resulted in the loss of cancer cases. On the other hand, inclusion of TCR incidence data identified a number of employees with cancer who were alive as of the end of the study or who died of cancer in states other than Texas. For example, 30 employees were counted as lung cancer deaths but not as cases. 17 were identified both as lung cancer decedents and as lung cancer cases, and 17 counted as incident lung cancer cases but not as deaths.

Most measured concentrations were at least 96% below the TLV or PEL during the working life of the plant from 1972 to 2006. Area sampling results were consistent with personal sample results, suggesting that contaminants were effectively controlled both in worker breathing zones and in general plant air. During the years 2000 and 2002, the facility's ventilation capacity, exhaust, and supply, was approximately 2 million cubic feet per minute of air. The extensive ventilation systems were largely responsible for the excellent control of airborne contaminants. Although some of the 3058 area and personal air samples for 1972 to 1988 analyzed in the 1992 mortality study were no longer available, the conclusion from the earlier analysis that, "overall, specific levels of workplace chemicals in this facility were well below established TLVs,"[11] is consistent with present findings.

In summary, employees had a favorable mortality experience overall in comparison to regional, Texas, and US general populations. Hourly white male employees had elevated mortality rates for several cancers previously associated with employment before 1960 in the rubber products manufacturing industry, including stomach cancer, bladder cancer, and leukemia. Results for these cancers were based on small numbers, and detailed data on jobs, occupational exposures, and lifestyle factors were lacking. These limitations preclude attribution of the observed increases to occupational exposure at the tire plant. Analyses of cancer incidence among Tyler tire plant employees were limited by a restricted time period of observation, but results did not indicate any cancer increases likely to be attributable to occupational exposures at the plant. An observed excess of mesothelioma cases may be due to employment outside the Tyler tire plant.

Acknowledgment

Cancer incidence data were provided by the Texas Cancer Registry, Cancer Epidemiology and Surveillance Branch, Texas Department of State Health Services, 1100 West 49th Street, Austin, TX 78756, http://www.dshs.state.tx.us/tcr/default.shtm, or (512) 458-7523.

The Goodyear Tire and Rubber Company supported this research through a contract with the University of Alabama at Birmingham and through funds paid to Monroe Com & Associates, Inc. The authors thank Barbara Toeppen-Spigel, Global Medical Director of The Goodyear Tire and Rubber Company, and all Tyler employees who facilitated access to data for this project.

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TAB 3

Centers for Disease Control and Prevention
CDC 24/7: Saving Lives, Protecting People™

Impaired Driving: Get the Facts

Every day, almost 30 people in the United States die in motor vehicle crashes that involve an alcohol-impaired driver. This amounts to one death every 51 minutes.[1] The annual cost of alcohol-related crashes totals more than $59 billion.[2]

Thankfully, there are effective measures that can help prevent injuries and deaths from alcohol-impaired driving.

On this Page

How big is the problem?

- In 2013, 10,076 people were killed in alcohol-impaired driving crashes, accounting for nearly one-third (31%) of all traffic-related deaths in the United States.[1]
- Of the 1,149 traffic deaths among children ages 0 to 14 years in 2013, 200 (17%) involved an alcohol-impaired driver.[1]
- Of the 200 child passengers ages 14 and younger who died in alcohol-impaired driving crashes in 2013, over half (121) were riding in the vehicle with the alcohol-impaired driver.[1]
- In 2012, over 1.3 million drivers were arrested for driving under the influence of alcohol or narcotics.[3] That's one percent of the 121 million self-reported episodes of alcohol-impaired driving among U.S. adults each year.[4]
- Drugs other than alcohol (e.g., marijuana and cocaine) are involved in about 18% of motor vehicle driver deaths. These other drugs are often used in combination with alcohol.[5]

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Who is most at risk?

Young people:

- At all levels of blood alcohol concentration (BAC), the risk of being involved in a crash is greater for young people than for older people.[6]
- Among drivers with BAC levels of 0.08 % or higher involved in fatal crashes in 2013, one out of every 3 were between 21 and 24 years of age (33%). The next two largest groups were ages 25 to 34 (29%) and 35 to 44 (24%).[1]

Motorcyclists:

- Among motorcyclists killed in fatal crashes in 2013, 27% had BACs of 0.08% or greater.[1]
- Nearly half of the alcohol-impaired motorcyclists killed each year are age 40 or older, and motorcyclists ages 40-44 have the highest percentage of deaths with BACs of 0.08% or greater (44%).[7]

Drivers with prior driving while impaired (DWI) convictions:

- Drivers with a BAC of 0.08% or higher involved in fatal crashes were six times more likely to have a prior conviction for DWI than were drivers with no alcohol in their system. (6% and 1%, respectively).[1]

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A Closer Look - Terminology

Sobriety checkpoints:

Traffic stops where law enforcement officers assess drivers' level of alcohol impairment. These checkpoints consistently reduce alcohol-related crashes, typically by 9%.

Ignition interlocks:

Devices that are installed in the vehicles of people who have been convicted of driving while impaired. They prevent operation of the vehicle by anyone with a blood alcohol concentration (BAC) above a specified safe level (usually 0.02% – 0.04%). When installed, interlocks are associated with about a 70% reduction in arrest rates for impaired driving.

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How can deaths and injuries from impaired driving be prevented?

Effective measures include:

- Actively enforcing existing 0.08% BAC laws, minimum legal drinking age laws, and zero tolerance laws for drivers younger than 21 years old in all states.[3,8,9]
- Promptly taking away the driver's licenses of people who drive while intoxicated.[10]
- Using sobriety checkpoints.[11]
- Putting health promotion efforts into practice that influence economic, organizational, policy, and school/community action.[12,13]
- Using community-based approaches to alcohol control and DWI prevention.[10,14,15]
- Requiring mandatory substance abuse assessment and treatment, if needed, for DWI offenders.[16]
- Raising the unit price of alcohol by increasing taxes.[17,18]

Areas for continued research:

- Reducing the illegal BAC threshold to 0.05%.[17,19,20]
- Mandatory blood alcohol testing when traffic crashes result in injury.[17]

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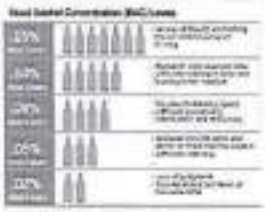
Effects of Blood Alcohol Concentration (BAC)



The more alcohol you consume, the more impaired you become.

Learn how your blood alcohol concentration (BAC) affects your ability to drive.

More >

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What safety steps can individuals take?

Whenever your social plans involve alcohol, make plans so that you don't have to drive after drinking. For example:

- Prior to any drinking, designate a non-drinking driver when with a group.
- Don't let your friends drive impaired. Take their keys away.
- If you have been drinking, get a ride home or call a taxi.
- If you're hosting a party where alcohol will be served, remind your guests to plan ahead and designate their sober driver; offer alcohol-free beverages; and make sure all guests leave with a sober driver.

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Social Media Resources for Sharing (http://www.pinterest.com/cdcgov/safe-driving/)

Even one drink impairs driving ability and increases the risk of a crash.

Help spread the word about the dangers of drunk driving. Visit the CDC Safe Driving Pinterest board (http://www.pinterest.com/cdcgov/safe-driving/) for more ready-to-share graphics.

More (http://www.pinterest.com/cdcgov/safe-driving/) >

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CDC Vital Signs: Teen Drinking and Driving

Teen Drinking and Driving - A Dangerous Mix

October 2012

The percentage of teens in high school who drink and drive has decreased by more than half since 1991, but more can be done.

More >

CDC Vital Signs: Drinking and Driving

Drinking and Driving - A Threat to Everyone

October 2011

US adults drank too much and got behind the wheel about 112 million times in 2010.

More >

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Related Pages	Additional Data
Sobering Facts: Drunk Driving State Fact Sheets	Drunk Driving State Data
What Works: Strategies to Reduce or Prevent Drunk Driving	Drunk Driving Death Rates US Map

Increasing Alcohol Ignition Interlock Use: Successful Practices for States

Effects of Blood Alcohol Concentration (BAC)

State-Based Motor Vehicle Data & Information

CDC's Safe Driving Board on Pinterest (http://www.pinterest.com/cdcgov/safe-driving/)
Help spread the word about the dangers of drunk driving. Visit the CDC Safe Driving Pinterest board for ready-to-share graphics and social media content.

Drunk Driving US Map

Motor Vehicle Prioritizing Interventions and Cost Calculator for States (MV PICCS)
CDC offers a new interactive calculator to help state decision makers prioritize and select from a suite of 12 effective motor vehicle injury prevention interventions. MV PICCS is designed to calculate the expected number of injuries prevented and lives saved at the state level and the costs of implementation, while taking into account available resources.

data.cdc.gov (https://data.cdc.gov/browse?category=Motor+Vehicle&utf8=%E2%9C%93)
View and download dozens of motor vehicle datasets and visualizations, including charts and maps, on data.cdc.gov.

Connect with the CDC Injury Center

(http://www.twitter.com/CDCInjury) (http://www.pinterest.com/cdcgov/safe-driving/)

File Formats Help:

How do I view different file formats (PDF, DOC, PPT, MPEG) on this site? (http://www.cdc.gov/Other/plugins/)

(http://www.cdc.gov/Other/plugins/#pdf)

Page last reviewed: November 24, 2015
Page last updated: November 24, 2015
Content source: Centers for Disease Control and Prevention (http://www.cdc.gov/), National Center for Injury Prevention and Control (http://www.cdc.gov/injury), Division of Unintentional Prevention

TAB 4

Traffic Safety Facts

2013 Data

December 2014

DOT HS 812 102

NHTSA
www.nhtsa.gov

Key Findings

- There were 10,076 fatalities in 2013 in crashes involving a driver with a BAC of .08 or higher; this was 31 percent of total traffic fatalities for the year.
- An average of one alcohol-impaired-driving fatality occurred every 52 minutes in 2013.
- The estimated economic cost of alcohol-impaired-driving crashes in the United States in 2010 (the most recent year for which cost data is available) was $49.8 billion.
- Of the traffic fatalities among children 14 and younger in 2013, about 17 percent occurred in alcohol-impaired-driving crashes.
- In 2013, the 21- to 24-year-old age group had the highest percentage of drivers in fatal crashes, with BAC levels of .08 or higher (33%).
- The percentage of drivers with BACs of .08 or above in fatal crashes in 2013 was highest for motorcycle riders (27%).
- The rate of alcohol impairment among drivers involved in fatal crashes in 2013 was nearly four times higher at night than during the day.
- Among the 10,076 alcohol-impaired-driving fatalities in 2013, 68 percent (6,860) were in crashes in which at least one driver in the crash had a BAC of .15 g/dL or higher.

U.S. Department of Transportation
National Highway Traffic Safety Administration
1200 New Jersey Avenue SE.
Washington, DC 20590

Alcohol-Impaired Driving

Drivers are considered to be alcohol-impaired when their blood alcohol concentrations (BACs) are .08 grams per deciliter (g/dL) or higher. Thus, any fatal crash involving a driver with a BAC of .08 or higher is considered to be an alcohol-impaired-driving crash, and fatalities occurring in those crashes are considered to be alcohol-impaired-driving fatalities. The term "driver" refers to the operator of any motor vehicle, including a motorcycle.

Estimates of alcohol-impaired driving are generated using BAC values reported to the Fatality Analysis Reporting System (FARS) and BAC values imputed when they are not reported. The term "alcohol-impaired" does not indicate that a crash or a fatality was *caused* by alcohol impairment, only that an alcohol-impaired driver was involved in the crash.

In this fact sheet, the 2013 alcohol-impaired-driving information is presented in the following order:

- Overview
- Economic Cost
- Children
- Time of Day and Day of Week
- Drivers
- Fatalities by State

Overview

All 50 States, the District of Columbia, and Puerto Rico have by law set a threshold making it illegal per se to drive with a BAC of .08 or higher. In 2013, 10,076 people were killed in alcohol-impaired-driving crashes, an average of one alcohol-impaired-driving fatality occurred every 52 minutes. These alcohol-impaired-driving fatalities accounted for 31 percent of the total motor vehicle traffic fatalities in the United States.

Of the 10,076 people who died in alcohol-impaired-driving crashes in 2013, 6,515 (65%) were drivers with BACs of .08 or higher. The remaining fatalities consisted of 2,724 motor vehicle occupants (27%) and 837 nonoccupants (8%). The distribution of fatalities in these crashes by role is shown in Table 1.

Table 1

Fatalities, by Role, in Crashes Involving at Least One Driver With a BAC of .08 or Higher, 2013

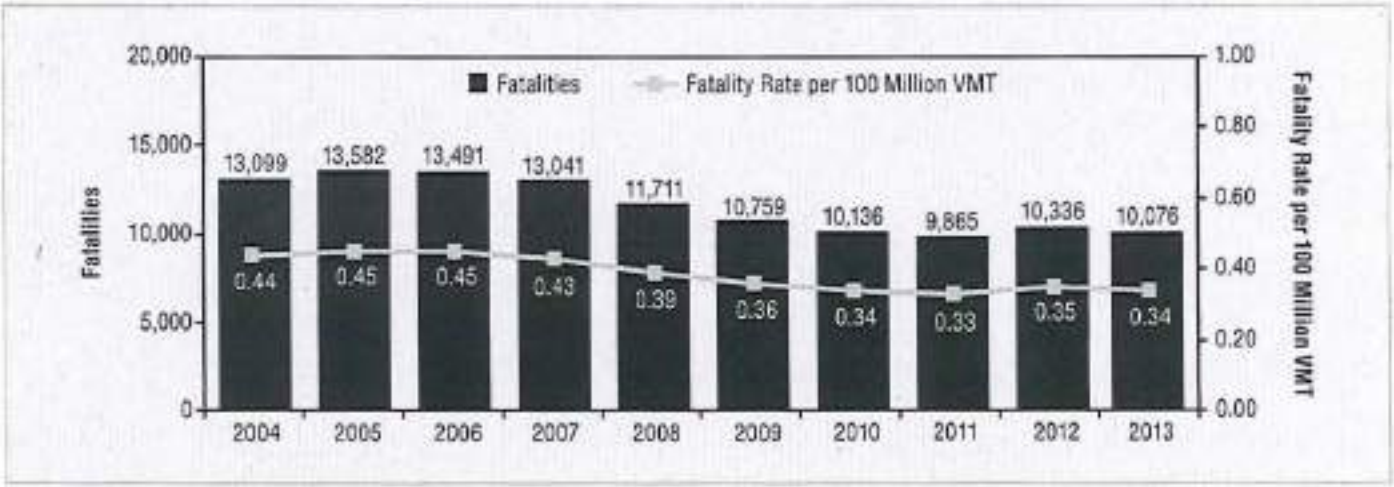
Role	Number	Percent of Total Fatalities
Driver With BAC=.08+	6,515	65%
Passenger Riding w/Driver With BAC=.08+	1,567	16%
Subtotal	**8,082**	**80%**
Occupants of Other Vehicles	1,157	11%
Nonoccupants	837	8%
Total Fatalities	**10,076**	**100%**

Source: Fatality Analysis Reporting System 2013 Annual Report File (ARF).

From 2012 to 2013, fatalities in alcohol-impaired-driving crashes decreased by 2.5 percent (10,336 to 10,076 fatalities). Alcohol-impaired-driving fatalities in the past 10 years have declined by 23 percent, from 13,099 in 2004 to 10,076 in 2013. The national rate of alcohol-impaired-driving fatalities in motor vehicle crashes in 2013 was 0.34 per 100 million vehicle miles traveled (VMT), a decline from 0.35 in 2012. The alcohol-impaired-driving fatality rate in the past 10 years has declined by 23 percent, from 0.44 in 2004 to 0.34 in 2013. Figure 1 presents the fatality numbers and rates for the past decade.

Figure 1

Fatalities and Fatality Rate per 100 Million VMT in Alcohol-Impaired-Driving Crashes, 2004–2013



Source: Fatalities – FARS 2004–2012 (Final File) and 2013 (ARF); 2004–2012 VMT – Federal Highway Administration's (FHWA) Annual Highway Statistics; 2013 VMT – FHWA's Traffic Volume Trends (September 2014)

Economic Cost

The estimated economic cost of all motor vehicle traffic crashes in the United States in 2010 (the most recent year for which cost data is available) was $277 billion, of which $49.8 billion resulted from alcohol-impaired-driving crashes. Included in the economic costs are:

- lost productivity
- workplace losses
- legal and court expenses
- medical costs
- emergency medical services (EMS)
- insurance administration
- congestion
- property damage

These costs represent the tangible losses that result from motor vehicle crashes. However, in cases of serious injury or death, such costs fail to capture the relatively intangible value of lost quality-of-life that results from these injuries. When quality of life valuations are considered, the total value of societal harm from motor vehicle crashes in the United States in 2010 was an estimated $870.8 billion, of which $206.9 billion resulted from alcohol-impaired-driving crashes. For further information on cost estimates, see *The Economic and Societal Impact of Motor Vehicle Crashes, 2010.*[1]

Children

In 2013, a total of 1,149 children 14 and younger were killed in motor vehicle traffic crashes. Of those 1,149 fatalities, 200 (17%) occurred in alcohol-impaired-driving crashes. Out of those 200 deaths, 121 (61%) were occupants of vehicles with drivers who had BACs of .08 or higher, and another 29 children (15%) were pedestrians or pedalcyclists struck by drivers with BACs of .08 or higher.

Time of Day and Day of Week

The rate of alcohol impairment among drivers involved in fatal crashes in 2013 was nearly 4 times higher at night than during the day (35% versus 9%). In 2013, 15 percent of all drivers involved in fatal crashes during the week were alcohol-impaired, compared to 30 percent on weekends. Table 2 presents information on drivers involved in fatal crashes in 2004 and 2013 by time of day and day of week, as well as single-vehicle and multi-vehicle crash data.

[1] Blincoe, L. J., Miller, T. R., Zaloshnja, E., & Lawrence, B. A. (2014, May). *The economic and societal impact of motor vehicle crashes, 2010.* (DOT HS 812 013). Washington, DC: National Highway Traffic Safety Administration. Available at www-nrd.nhtsa.dot.gov/pubs/812013.pdf

Table 2

Drivers Involved in Fatal Crashes With BACs of .08 or Higher, by Crash Type, Time of Day and Day of Week, 2004 and 2013

Drivers Involved in Fatal Crashes	2004			2013			Change in Percentage With BAC=.08+ 2004-2013
	Total Number of Drivers	BAC=.08+		Total Number of Drivers	BAC=.08+		
		Number	Percent of Total		Number	Percent of Total	
Total	*58,395*	*12,057*	*21%*	*44,574*	*9,461*	*21%*	*0*
Drivers by Crash Type and Time of Day							
Single-Vehicle Crash							
*Total**	*21,744*	*7,878*	*36%*	*17,983*	*6,296*	*35%*	*-1*
Daytime	8,553	1,427	17%	7,186	1,229	17%	0
Nighttime	12,862	6,273	49%	10,593	4,962	47%	-2
Multiple-Vehicle Crash							
*Total**	*36,651*	*4,179*	*11%*	*26,591*	*3,164*	*12%*	*+1*
Daytime	23,133	1,173	5%	16,591	878	5%	0
Nighttime	13,498	3,004	22%	9,973	2,280	23%	+1
Drivers by Time of Day							
Daytime	31,686	2,600	8%	23,777	2,107	9%	+1
Nighttime	26,360	9,277	35%	20,566	7,242	35%	0
Drivers by Day of Week and Time of Day							
*Weekday**	*35,159*	*5,205*	*15%*	*27,126*	*4,142*	*15%*	*0*
Daytime	23,014	1,487	6%	17,337	1,242	7%	+1
Nighttime	12,039	3,677	31%	9,715	2,875	30%	-1
*Weekend**	*23,136*	*6,801*	*29%*	*17,388*	*5,284*	*30%*	*+1*
Daytime	8,672	1,113	13%	6,440	865	13%	0
Nighttime	14,321	5,600	39%	10,851	4,366	40%	+1

Source: FARS 2004 Final File and 2013 ARF.
Daytime – 6 a.m. to 5:59 p.m. Weekday – Monday 6 a.m. to Friday 5:59 p.m.
Nighttime – 6 p.m. to 5:59 a.m. Weekend – Friday 6 p.m. to Monday 5:59 a.m.
*Includes drivers involved in fatal crashes when time of day was unknown.

Drivers

In fatal crashes in 2013 the highest percentage of drivers with BACs of .08 or higher was for drivers 21 to 24 years old (33%), followed by ages 25 to 34 (29%). The proportion of drivers involved in fatal crashes with BACs of .08 or higher was 23 percent among males and 15 percent among females. Table 3 provides information on impaired-driving crashes by the age of the driver as well as gender and vehicle type.

Table 3

Drivers With BACs of .08 or Higher Involved in Fatal Crashes, by Age, Gender, and Vehicle Type, 2004 and 2013

Drivers Involved in Fatal Crashes	2004			2013			Change in Percentage With BAC=.08+ 2004-2013
	Total Number of Drivers	BAC=.08+		Total Number of Drivers	BAC=.08+		
		Number	Percent of Total		Number	Percent of Total	
Total	*58,395*	*12,057*	*21%*	*44,574*	*9,461*	*21%*	*0*
Drivers by Age Group (Years)							
16-20	7,755	1,397	18%	3,883	666	17%	-1
21-24	6,413	2,116	33%	4,609	1,500	33%	0
25-34	11,242	3,055	27%	8,762	2,583	29%	+2
35-44	10,743	2,500	23%	7,183	1,733	24%	+1
45-54	9,148	1,704	19%	7,343	1,501	20%	+1
55-64	5,612	701	12%	5,911	827	14%	+2
65-74	3,070	233	8%	3,357	278	8%	0
75+	3,169	151	5%	2,567	128	5%	0
Drivers by Gender							
Male	42,250	10,049	24%	32,442	7,583	23%	-1
Female	15,384	1,675	12%	11,364	1,657	15%	+3
Drivers by Vehicle Type							
Passenger Cars	25,568	5,852	23%	17,731	4,062	23%	0
Light Trucks	22,367	4,808	21%	16,738	3,584	21%	0
Large Trucks	4,837	53	1%	3,858	92	2%	+1
Motorcycles	4,116	1,116	27%	4,769	1,295	27%	0

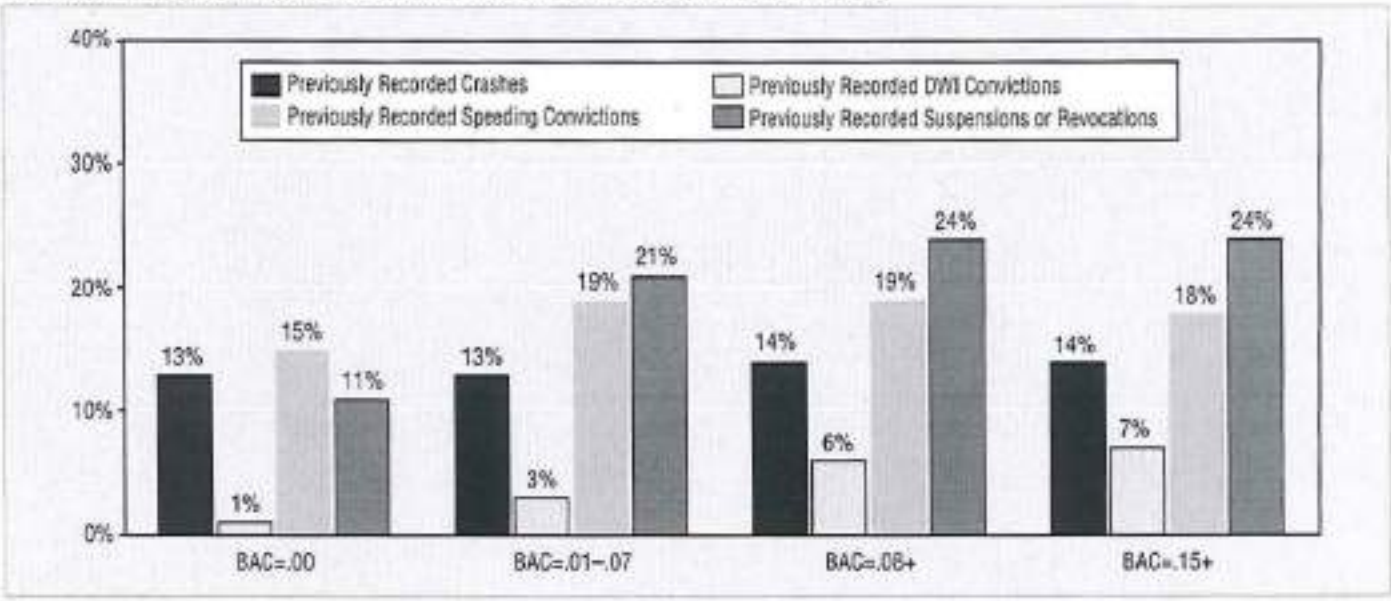
Source: FARS 2004 Final File and 2013 ARF.
Numbers shown for groups of drivers do not add to the total number of drivers due to unknowns/not reported or other data not included.

The percentages of drivers involved in fatal crashes with BACs of .08 or higher in 2013 by vehicle type were 27 percent for motorcycles, 23 percent for passenger cars, and 21 percent for light trucks. The percentage of drivers with BACs of .08 or higher in fatal crashes was the lowest for drivers of large trucks (2%).

In 2013, 5,080 passenger vehicle drivers killed had BACs of .08 or higher ("passenger vehicles" includes cars as well as light trucks, vans, SUVs, and pickups). Out of those driver fatalities for which restraint use was known, 68 percent were unrestrained. Among passenger vehicle drivers killed who had BACs of .01 to .07 g/dL the percentage of unrestrained was 53 percent, and for passenger vehicle drivers killed who had no alcohol (BAC=.00) the percentage of unrestrained was 39 percent.

Figure 2 shows information on the driving record for drivers in fatal crashes in 2013, at different alcohol levels. There was little difference by alcohol level in the percentage of drivers with previously recorded crashes. Drivers with BACs of .08 or higher involved in fatal crashes were six times more likely to have prior convictions for driving while impaired (DWI) than were drivers with no alcohol (6% and 1%, respectively). Note: FARS records drivers' previous crashes, suspensions/revocations, and convictions that occurred up to three years prior to the date of the crash.

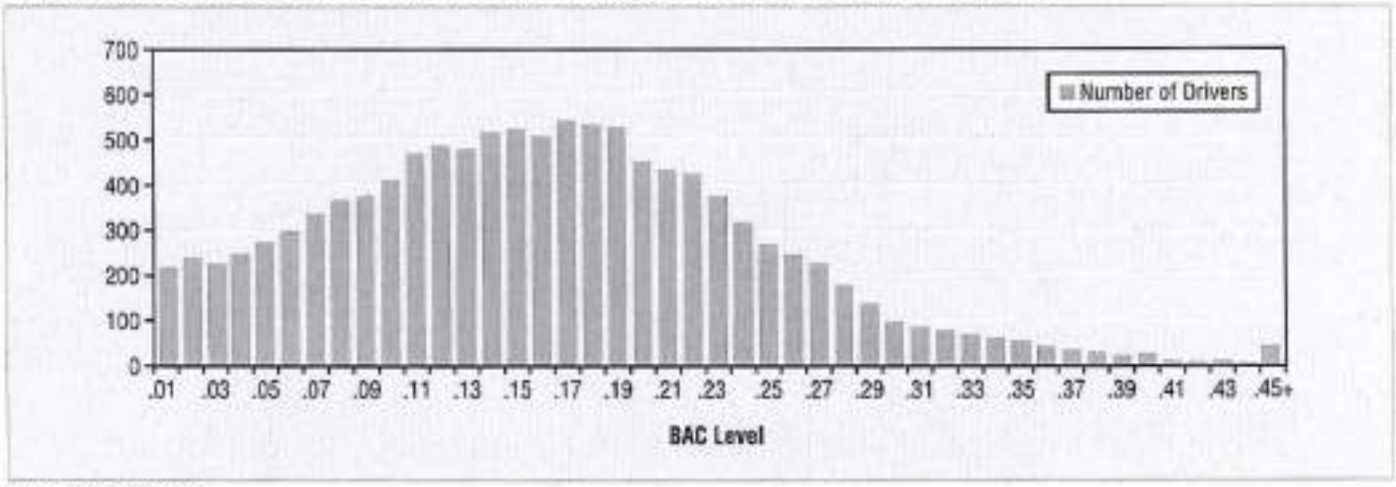
Figure 2
Previous Driving Records of Drivers Involved in Fatal Crashes, by BAC, 2013



Source: FARS 2013 ARF.

While .08 BAC is considered to be impaired in all States, the large majority of drivers in fatal crashes with any measurable alcohol had levels far higher. In 2013, 84 percent (9,461) of the 11,307 drivers with BACs of .01 or higher who were involved in fatal crashes had BAC levels at or above .08, and 56 percent (6,341) had BACs at or above .15. Among the 10,076 alcohol-impaired-driving fatalities in 2013, 68 percent (6,860) were in crashes in which at least one driver in the crash had a BAC of .15 g/dL or higher. The most frequently recorded BAC among drinking drivers in fatal crashes was .17 (see Figure 3). Figure 3 presents the distribution of BACs for those drivers with any alcohol in their systems.

Figure 3
Distribution of BACs for Drivers With BACs of .01 or Higher Involved in Fatal Crashes, 2013



Source: FARS 2013 ARF.

Fatalities by State

Table 4 shows traffic fatalities by State and the highest driver BAC in the crashes in 2013. Among all States, fatalities in motor vehicle traffic crashes in 2013 ranged from 20 to 3,382, depending on the size and population of the State. Alcohol-impaired-driving fatalities were highest in Texas (1,337), followed by California (867), and Florida (676), and lowest in the District of Columbia (6). The proportion of alcohol-impaired-driving fatalities among total fatalities in States ranged from a high of 44 percent (South Carolina) to a low of 17 percent (Utah). The proportion of fatalities in crashes involving a driver with a BAC of .15 g/dL or higher ranged from a high of 36 percent (North Dakota) to a low of 12 percent (Utah).

The suggested APA formation citation for this document is:

National Center for Statistics and Analysis. (2014, December). *Alcohol-impaired driving: 2013 data.* (Traffic Safety Facts. DOT HS 812 102). Washington, DC: National Highway Traffic Safety Administration.

For more information:

Information on traffic fatalities is available from the National Center for Statistics and Analysis (NCSA), NVS-424, 1200 New Jersey Avenue SE., Washington, DC 20590. NCSA can be contacted at 800-934-8517 or by e-mail at ncsaweb@dot.gov. General information on highway traffic safety can be found at www.nhtsa.gov/NCSA. To report a safety-related problem or to inquire about motor vehicle safety information, contact the Vehicle Safety Hotline at 888-327-4236.

Other fact sheets available from the National Center for Statistics and Analysis are *Bicyclists and Other Cyclists, Children, Large Trucks, Motorcycles, Occupant Protection, Older Population, Overview, Passenger Vehicles, Pedestrians, Race and Ethnicity, Rural/Urban Comparisons, School Transportation-Related Crashes, Speeding, State Alcohol Estimates, State Traffic Data,* and *Young Drivers.* Detailed data on motor vehicle traffic crashes are published annually in *Traffic Safety Facts: A Compilation of Motor Vehicle Crash Data from the Fatality Analysis Reporting System and the General Estimates System.* The fact sheets and annual Traffic Safety Facts report can be accessed online at www-nrd.nhtsa.dot.gov/CATS/index.aspx.

Table 4

Traffic Fatalities by State and Highest Driver BAC in the Crash, 2013

State	Total Fatalities*	BAC=.00		BAC=.01-.07		BAC=.08+		BAC=0.15+		BAC=.01+	
	Number	Number	Percent	Number	Percent	Number	Percent	Number	Percent	Number	Percent
Alabama	852	543	64%	48	6%	260	31%	175	21%	308	36%
Alaska	51	34	66%	1	3%	15	30%	12	24%	16	32%
Arizona	849	574	68%	43	5%	219	26%	158	19%	262	31%
Arkansas	483	324	67%	34	7%	123	25%	89	18%	156	32%
California	3,000	1,963	65%	158	5%	867	29%	583	19%	1,025	34%
Colorado	481	309	64%	28	6%	142	30%	105	22%	170	35%
Connecticut	276	145	52%	17	6%	114	41%	74	27%	132	48%
Delaware	99	57	57%	4	4%	38	39%	27	27%	43	43%
Dist of Columbia	20	13	67%	0	2%	6	31%	4	19%	7	33%
Florida	2,407	1,607	67%	115	5%	676	28%	480	20%	790	33%
Georgia	1,179	824	70%	52	4%	297	25%	182	15%	349	30%
Hawaii	102	57	56%	12	12%	33	33%	21	20%	45	44%
Idaho	214	136	64%	15	7%	58	27%	41	19%	73	34%
Illinois	991	601	61%	67	7%	322	32%	227	23%	389	39%
Indiana	783	541	69%	43	6%	198	25%	143	18%	241	31%
Iowa	317	204	64%	10	3%	103	32%	75	24%	113	36%
Kansas	350	230	66%	18	5%	102	29%	74	21%	119	34%
Kentucky	638	444	70%	26	4%	167	26%	113	18%	193	30%
Louisiana	703	427	61%	39	5%	234	33%	148	21%	272	39%
Maine	145	91	63%	12	8%	42	29%	25	17%	54	37%
Maryland	465	289	62%	34	7%	141	30%	95	20%	175	38%
Massachusetts	326	179	55%	24	7%	118	36%	67	20%	142	44%
Michigan	947	638	67%	54	6%	255	27%	168	18%	309	33%
Minnesota	387	272	70%	20	5%	95	25%	74	19%	115	30%
Mississippi	613	372	61%	30	5%	210	34%	129	21%	240	39%
Missouri	757	468	62%	39	5%	248	33%	169	22%	287	38%
Montana	229	125	55%	12	5%	92	40%	67	29%	104	45%
Nebraska	211	136	65%	10	5%	60	28%	44	21%	70	33%
Nevada	262	168	64%	15	6%	79	30%	57	22%	94	36%
New Hampshire	135	83	61%	7	5%	46	34%	34	25%	52	39%
New Jersey	542	358	66%	38	7%	146	27%	93	17%	184	34%
New Mexico	310	192	62%	25	8%	93	30%	65	21%	118	38%
New York	1,199	756	63%	78	6%	364	30%	235	20%	442	37%
North Carolina	1,289	858	67%	57	4%	371	29%	247	19%	428	33%
North Dakota	148	73	49%	12	8%	62	42%	54	36%	73	49%
Ohio	989	664	67%	51	5%	271	27%	170	17%	322	33%
Oklahoma	678	472	70%	37	5%	170	25%	123	18%	206	30%
Oregon	313	189	61%	17	5%	105	33%	80	25%	122	39%
Pennsylvania	1,208	774	64%	64	5%	368	30%	258	21%	431	36%
Rhode Island	65	37	57%	4	6%	24	38%	18	28%	28	43%
South Carolina	767	379	49%	49	6%	335	44%	225	29%	384	50%
South Dakota	135	85	63%	7	5%	41	31%	34	25%	48	36%
Tennessee	995	666	67%	51	5%	277	28%	190	19%	327	33%
Texas	3,382	1,829	54%	213	6%	1,337	40%	896	26%	1,550	46%
Utah	220	175	79%	6	3%	38	17%	25	12%	44	20%
Vermont	69	45	66%	5	8%	18	27%	14	20%	24	34%
Virginia	740	435	59%	48	6%	254	34%	177	24%	302	41%
Washington	436	267	61%	20	4%	149	34%	94	21%	169	39%
West Virginia	332	220	66%	21	6%	91	27%	62	19%	112	34%
Wisconsin	543	329	61%	32	6%	178	33%	129	24%	210	39%
Wyoming	87	58	67%	4	5%	25	29%	18	21%	29	33%
National	**32,719**	**20,713**	**63%**	**1,820**	**6%**	**10,076**	**31%**	**6,860**	**21%**	**11,896**	**36%**
Puerto Rico	344	185	54%	31	9%	127	37%	75	22%	158	46%

Source: FARS 2013 ARF.

*Total includes fatalities in crashes in which there was no driver present.

TAB 5

Stacks-After Current Table

Industrial Hygiene Digest

INDUSTRIAL HEALTH NEWS

LITERATURE ABSTRACTS

MEDICAL

ENGINEERING

CHEMICAL

TOXICOLOGICAL

LEGAL

JUNE, 1968
(Vol. 32, No. 6)

INDUSTRIAL HYGIENE FOUNDATION
4400 FIFTH AVENUE — PITTSBURGH, PA. 15213

551 Pulmonary Function in Bagasse Worker's Lung Disease. A. B. Pierce et al. Am. Rev. Resp. Dis. 97. 561-570 (April, 1968).

Seven previously well men working in a fiberboard plant developed signs and symptoms of acute bagasse worker's lung during a ten-week period. Two of these patients had open lung biopsies to confirm the diagnosis. Pulmonary functions were measured from 2.5 weeks to 26 months after the onset of symptoms. All patients had a restrictive lung defect due to a reduced inspiratory capacity; partial airway obstruction was not a feature of the illness. All patients had a severe reduction in the transfer coefficient of the lung for carbon monoxide at the outset; this was primarily due to a reduction in membrane diffusing capacity, whereas the volume of blood in the pulmonary capillaries was more nearly normal. Although pulmonary functions returned toward normal, the total lung capacity, inspiratory capacity, vital capacity, forced expiratory volume, transfer coefficient of the lung for CO and membrane diffusing capacity remained significantly abnormal more than one year after the onset. Three patients were treated with adrenocorticosteroids and 4 were not. There was no significant difference between the treated and the untreated patients in the rate of objective improvement or in the extent of residual impairment of pulmonary functions. These data suggest that steroids do not hasten the resolution of a granulomatous infiltrate nor do they make resolution more complete. There are 28 references. -- Authors' summary

552 Metal and Mineral Concentrations in Lungs of Bituminous Coal Miners. J. V. Crable, et al. Am. Ind. Hyg. Assn. J. 29. 106-110 (March-April, 1968).

Lung and pulmonary lymph node samples from 26 West Virginia bituminous coal miners have analyzed for total dust, coal, and free silica. In addition, beryllium, cobalt, manganese, nickel, titanium, and vanadium were determined by the quantitative spectrographic methods; lead was determined by the CSPHS dithizone method; and copper, iron, magnesium, and zinc were determined by atomic absorption spectrophotometric methods. The summarized results of the mineral analyses from this study and the authors' previous study are presented. The concentration ranges and mean values for eleven elemental constituents are compared with normal values reported by Tipton and Shafer and Cholak. There are 9 references. -- Authors' summary

553 Tissue Response to Intraperitoneal Asbestos With Preliminary Report of Acute Toxicity of Heat Treated Asbestos in Mice. J. Jagatic, et al. Environ. Research 1. 217-230 (Nov., 1967).

Intraperitoneal asbestos not only produced fibrosis, but a special and peculiar type of fibrosis which was proliferative, granulomatous and invasive, and histologically similar to mesothelioma although morphologic evidence of actual malignancy was not obtained. Further, asbestos heated and exposed to high temperature produced a high degree of toxicity in experimental animals; this resulted in a 60% mortality rate. -- Authors' abstract

Editor's Note: Although the authors of this 14-page article insist that the fibrous inflammatory tissue resulting from intraperitoneally injected asbestos is histologically similar to mesothelioma and that it is "invasive" they gave no evidence in support of these opinions. The 22 illustrations that are included in this report, support neither the claim of similarity to mesothelioma nor of "invasiveness" of the inflammatory tissue. -- P. Gr...

554 Asbestos—A Hazard to the Community. C. Gold and J. Cuthbert. Public Health(London) 80. 261-270 (Sept., 1966).

During a recent study of pulmonary fibrosis in a Southeast Glasgow clinic, 21 proved cases and 3 possible cases of lung pathology associated with exposure to asbestos have come to light. A patient with suspected asbestosis should have, in addition to x-rays, an examination for asbestos bodies in the sputum, lung function tests, and open lung biopsy, since asbestosis has been found in patients with normal or borderline x-rays by the lung function test and most reliable by biopsy. It is also important to take a complete occupational history in cases of obscure pulmonary fibrosis, since asbestosis or pleural tumors may occur many years after a short exposure to the asbestos dust. Also, persons in occupations usually not associated with health hazards may be endangered by working in close proximity with people who use asbestos, especially in confined spaces. Case histories are given and a table is presented showing the type of asbestos, occupation, length of exposure, age, and period elapsed since exposure of the 24 patients studied. Since the use of asbestos has become more widespread in recent years, it is recommended...

possible alternative materials to asbestos should be used whenever possible. A proposal to mix asbestos with tarmacadam for road use is viewed with concern. -- APCA Abstr.

Editor's Note: Reviewing 554-568. Interested readers are referred to the Editorial in the May issue of IHD and ISF Medical Series Bulletins No. 11 and 12 which report progress and planning of ISF asbestos bioeffects research. -- R. T. P. deT.

The Epidemiology and Clinical Features of Asbestosis and Related Diseases. P. C. Elmes. Postgrad. Med. J. 42, 623-635 (Oct. 1966).

Pulmonary fibrosis caused by asbestos dust has become less common since the introduction of dust suppression measures in 1932-33. However, in occupations where these safety measures are not observed, asbestosis is still a threat. Symptoms of asbestosis are breathlessness on exertion, productive cough, tightness in the chest, clubbing of fingers, warts, cyanosis, restricted chest movement, kyphosis, and fine rales heard over the lung bases. Changes in x-rays are not conclusive, since they also occur with other forms of chronic diffuse fibrosis involving the pleura. No laboratory tests except lung biopsy are diagnostic of asbestosis but sputum examination and respiratory function tests help to support the diagnosis. Carcinoma of the lung resulting from exposure to asbestos appears to be common in patients whose exposure to the dust was insufficiently long or intense to cause crippling fibrosis. The rapidly increasing number of cases of fatal mesotheliomas of the pleura and peritoneum have been definitely traced to exposure to asbestos dust. The degree of exposure in many cases is slight and there can be a long interval between exposure to the pollutant and the development of the tumor. Preventive measures recommended are restriction of use and greater precaution in the handling of all types of asbestos. Crocidolite fiber, in particular, should be eliminated from commercial use. -- APCA Abstr.

Asbestos—An Environmental Health Hazard. W. D. Norwood, P. A. Fuqua and T. F. Manousso. Caribbean Med. 66, 821 (Sept., 1967).

Four Case Reports demonstrate asbestosis with pulmonary emphysema and cor pulmonale, asbestosis with lung cancer, mesothelioma, and rectal cancer. The occupational histories of workers as plumbers, boiler makers, carpenters, pipe fitters, steam fitters, mechanics, stationary engineers, undercoaters, foremen, maintenance electricians, builders, rubber workers, construction workers, cement workers, boiler repairers, sheet metal workers and laborers must all be elicited if diagnosticians hope to record the more than 3,000 different exposures to asbestos. For the sake of workers and their dependents with industrial health compensation rights, the diagnostician must not be misled by recent occupational history nor overlook other work exposures within the lifetime of the patients. The obvious need for decreasing the exposure to asbestos by better occupational hygiene programs is recognized but no detailed criteria were presented. Considering the growing uses of asbestos, this occupational and environmental disease may get worse before it gets better. -- J. Occ. Med. Abstr.

Talc in Atmospheric Dusts. H. Windom, J. Griffin, and E. D. Goldberg. Environ. Sci. Tech. 1, 923-926 (Nov. 1967).

The mineral talc has been observed in dusts recovered directly from the atmosphere and in the solid mineral phases of rain and snow. The talc, where detected, attains levels of the order of a per cent by weight in the solid phases. This talc probably arises from agricultural activity where the mineral is used as a carrier and diluent for pesticides. The amounts of talc found in atmospheric dusts appear to reflect a local introduction rather than a generalized global fallout. -- APCA Abstr.

Measurement of Asbestos Exposure. J. R. Lynch and H. E. Ayer. J. Occ. Med. 10, 21-24 (Jan., 1968).

As an outcome of an epidemiologic study, a safe level of exposure should be set in terms of a method of exposure measurement which meets the following criteria in the order given: (1) The environmental factor measured should be sufficiently relevant to the disease mechanism to correlate with health status even in environments where exposures to mixed dust occur. (2) The sensitivity of the method should be such as to measure levels well below the Threshold Limit Values (TLV). (3) The method should lend itself to an appropriate sampling strategy: e.g., long-period personal samples in the case of asbestos. (4) The expense of conversion from existing methods should be reasonable. The impinger method of measuring asbestos exposure does not meet any of the criteria well with the exception of expense. Weight methods would be preferred if there were an analytical breakthrough. At present fiber counts on membrane filters are the best method of measuring exposure as related to the risk of asbestosis and the airborne fiber concentration as a factor in the risk of lung cancer. There are 7 references.

-- Cond. from text.

559 Industrial Hygiene for Insulation Workers. J. L. Balzer.
J. Occ. Med. 10, 25-31 (Jan., 1968)

The asbestos worker is exposed to amosite and chrysotile asbestos-containing materials, fiberglass, cork, plastics, and adhesives. Working in industrial and commercial building projects and marine construction and repair, he is exposed to many other environmental hazards not created by his own trade. Although he works with asbestos-containing products 45% of the time, the over-all time-weighted average exposure to asbestos-containing materials is below the presently recognized Threshold Limit Values (TLV) of 5 mppcf. The three work areas in which dust levels were above the TLV (prefabrication, tearing out, and mixing) account for less than 20% of his total work time. However, the incidence of radiographic changes indicative of asbestosis is over 25% in our men with 20 years or more of work. This and other evidence suggest that the present TLV is too high. Incidence of asbestosis and the risk of malignancies in the asbestos worker can be reduced by local and general ventilation, substitution of materials, changes in work habits and personal respiratory protection combined with education of the men to reduce their own exposures to asbestos-containing dusts. There are 17 references.

-- Author's summary.

560 Asbestos as an Environmental Hazard. I. R. Tabershaw.
J. Occ. Med. 10, 32-37 (Jan., 1968).

In summary, if one accepts the assumptions (1) that asbestos minerals increase the risk of lung cancer in occupational groups, (2) that they lead to an unusual risk of mesothelioma of the pleura and peritoneum in occupational groups and those living near asbestos plants, (3) that such malignancies usually result from exposures 30 to 50 years earlier, (4) that the "asbestos bodies" found in from 25 to 50% of lung smears from routine autopsies are probably due to asbestos in most cases, (5) that these "asbestos bodies" may result from recent exposures as well as those many years earlier, (6) that world production and use of asbestos has increased from 500,000 tons to 3,500,000 tons in 30 years, it is important to consider whether or not asbestos is a major threat to public health. One is not yet justified in such a conclusion. In view of the fact that most of the world tonnage goes into uses that do not lead to air contamination; that asbestos is not actually indestructible; that the effects are dose-dependent; and that low doses probably lead to lower rates and longer latency. Nevertheless, there is need for epidemiologic studies directed to those with intermediate exposures and for evaluation of the beneficial effects of cessation of exposure. Another need is for experimental and industrial hygiene studies to determine the nature and degree of exposure. Biologic studies centered on the meaning of the "asbestos body" are crucial to understanding the clinicopathologic response. Strict control of industrial and neighborhood environments is essential, but it is premature to extrapolate from the effects of heavy exposure to minor and low level exposures. There are 15 references.

-- Author's summary

561 Research on Health Effects of Asbestos. L. J. Cralley, et al.
J. Occ. Med. 10, 38-41 (Jan., 1968).

Asbestos is a general term applied to a group of fibrous crystalline hydrated silicate minerals. Although a number of different types of asbestos minerals exist, only four or five are of commercial importance. Each differs somewhat from the others in chemical composition, physical properties such as ability to withstand heat and chemical erosion, crystalline structure, fiber dimension, and degree of fiber harshness and brittleness. It has been known since the early nineteen hundred's that excessive exposure to asbestos gives rise to the disabling pulmonary disease "asbestosis." More recently, evidence has been developed that the incidence of respiratory tract and other malignancies in asbestos workers is excessive. A major problem in studying the health effects of asbestos is the long latent period of 20 or more years from exposure to the onset of disease. Also in the mid-nineteen thirties and earlier, there was little dust control and the workers were often exposed to massive levels of dust from asbestos and other associated materials in the manufacture of asbestos products. Thus, the causative agents of the resultant disease are essentially unknown. Included in these potential sources of causative agents, either alone or in combination, are asbestos fibers themselves, materials associated with the fibers in the ores such as trace minerals and polycyclic aromatic hydrocarbons, materials added during processing such as metals, tars, and pitches, and concomitant exposures to tobacco smoke or other air pollutants. A strong supported long-term program is needed to provide the information regarding the pathogenesis of asbestosis and the other diseases that appear associated with the inhalation of asbestos dust. More precise data on the

...chemical characterization of the asbestos minerals which lead to their being inhaled and ...ained in the body, in their carrying with them other chemicals, in their migration, may open ...unsuspected avenues of biological research -- Cond. from text.

...Clinical and Pathological Studies of Pulmonary Asbestosis. I. Nakamura. ...Osaka Med. Assn. 19, 415-465 (Aug., 1967).

...e environmental conditions in asbestos factories in Osaka and Nara were investigated. The ...ature of the dust in the factories was studied by electron microscopy. The concentration of ...spended particles is calculated for different plant operations in a Nara factory. The occurrence ...f asbestosis among the employees of these factories was studied by periodic x-ray examinations. ...though definite asbestosis could not be found in employees working less than 5 years, the ...requency of detection of definite asbestosis increased in those working more than 5 years as ...ollows: there was 21% in those employed between 5 and 10 years, 54% between 10 and 15 years, ...0% between 15 and 20 years, and 100% for those employed for over 20 years. The classification ...f x-ray findings and the development of asbestosis are discussed. Case histories and photo-...raphs of x-rays and lung sections are presented. -- APCA Abstr.

Mesothelioma and Its Association With Asbestosis. M. Borow, et al. J. Am. Med. Assn. 201, 587-591 (Aug. 21, 1967).

...ithin a three-year period 17 cases of mesothelioma (9 of the pleura and 8 of the peritoneum) ...ere seen at a community hospital in New Jersey located near a major asbestos mill. The ...ncidence of mesothelioma as well as asbestosis appears to be increasing.
-- Am. Rev. Resp. Dis. Abstr.

Pulmonary Function Tests in Asbestos Workers. G. L. Leathart. Trans. Soc. Occ. Med. 18, 49-59 (April, 1968).

There are three ways in which pulmonary function tests may contribute to the management of workers exposed to asbestos; they may be used to confirm a diagnosis of asbestosis, to follow the progress of the disease, or to monitor unaffected workers at risk. The object of this paper is to consider how successfully these three parts are played. The typical physiological abnormalities are described, serial studies in cases of asbestosis are presented, and tentative conclusions are drawn from a small prospective study of asbestos workers who were free of disease when first seen. The findings from this investigation are summarized as follows: (1) Established asbestosis causes a loss of diffusing capacity, vital capacity and compliance. Pleural calcification by itself does not. (2) Progressive loss of function is seldom arrested when the patient is transferred to other work and most cases deteriorate. (3) Diffusing capacity appears to be the best measurement for making an early diagnosis, but the diagnosis has been subsequently confirmed by other means in only four of the cases reported in the literature. (4) The development of crepitations at the lung bases sometimes precedes a significant drop in diffusing capacity and this may be a useful observation. (5) Finally, as a general conclusion, the function tests should not be interpreted in isolation nor be given undue respect. They should always be considered in conjunction with clinical and radiographic examination.
-- Cond. from text.

Source and Identification of Respirable Fibers. L. J. Cralley, et al. Am. Ind. Hyg. Assn. J. 29, 129-135 (March-April, 1968).

Fibrous bodies with an iron-containing coating have been found in the lungs of persons coming to autopsy in a number of urban hospitals, the number of fibrous bodies varying greatly with approximately 4 to 67% of the persons examined showing numerous bodies. Because these findings raise questions with regard to the possibility that asbestos is a factor in increased lung cancer, questions concerning the nature, source, and significance of these bodies are discussed in the light of research needed to find answers. There are 17 references.-- Authors' Abst.

Pulmonary Ferruginous Bodies. Development in Response to Filamentous Dusts and a Method of Isolation and Concentration. P. Gross, et al. Arch. Pathol. 85, 539-546 (May, 1968). Reprints available from Industrial Hygiene Foundation, 4400 Fifth Ave., Pittsburgh, Pa. 15213.

Formation of ferruginous bodies should not be confused with pathogenicity. Failure to understand this differentiation may result in the erroneous generalization that all fibrous dusts share the

TAB 6

Plaintiffs' Exhibit
GTR-KS 55

K-S - Tyler, Tex

July 25, 1972

To: Plant Personnel Manager
Plant Medical Department

Subject: Asbestos

Attached is a copy of the instructions regarding asbestos and a copy of the final OSHA Standard for asbestos.

This information is to be inserted in the Medical-Industrial Hygiene Manual and in those facilities where asbestos or asbestos-containing materials are used, the procedures for work practices, personal protective equipment, monitoring, labeling and medical surveillance must be started immediately.

Each facility must carefully survey their operations for their use of asbestos and asbestos-containing materials. If there is no potential asbestos exposure in the facility operations then these procedures need not be initiated.

Director
Occupational Health Services

C A Johnson, M.D.
jap

Attn.

T839

GY 10132

(2465)

ASBESTOS

General

Asbestos is a term used to describe several fibrous mineral silicates. The most important types of asbestos are chrysotile, a simple magnesium silicate; amosite and anthophyllite, complex magnesium silicates; and crocidolite, a complex sodium iron silicate. Chrysotile fiber is of the greatest importance because of its qualities and extensive usage.

Toxicity

Systemic

Long continued inhalation of asbestos fibers may result in the development of asbestosis (a chronic lung disease) accompanied by severe respiratory symptoms, changes in pulmonary function and changes in the appearance of an x-ray of the lungs.

Asbestos fiber inhalation has also been implicated as a possible causative factor in lung cancer, particularly bronchogenic carcinoma, and another form of cancer called a mesothelioma.

Local

There are no specific local toxic effects.

Surveillance

The following medical surveillance procedures shall be followed on all applicants for hire, employes transferring to, employes presently working and terminating employment on job assignments involving the use of asbestos or materials containing asbestos.

Pre-placement

Routine pre-placement medical history and examination
Chest x-ray (PA view) 14x17
Pulmonary function test - FEV_1 (one second forced expiratory volume) using McKesson and Robins "Vitalor" or similar equipment.

Transfer

Medical history - with particular reference to symptoms of respiratory disease
Chest x-ray (PA view) 14x17 to be done only if no record of chest x-ray within previous one-year period in medical file
Pulmonary function test - FEV_1 using "Vitalor" or similar equipment.

Periodic

The following procedures shall be done annually:

Review of medical history - with special emphasis on symptoms of respiratory disease - cough, dyspnea, chest pain, etc.
Physical examination - with emphasis on auscultation, percussion, etc of the chest
Chest x-ray (Pa view) 14x17
Pulmonary function test - FEV_1 using "Vitalor" or similar equipment

Termination -

Must be done within thirty (30) days before or after termination of employment with the company.

Review of medical history - with particular reference to symptoms of respiratory disease
Physical examination - with emphasis on auscultation, percussion, etc of the chest
Chest x-ray (Pa view) 14x17
Pulmonary function test - FEV_1 using "Vitalor" or similar equipment

NOTE: No medical examination is required of any employe, if adequate medical records show that the employe has been examined in accordance with any of the above examination requirements within the past one-year period.

Interpretation

Any person with a history, symptoms, or physical findings of chronic lung disease or with an abnormal chest x-ray or FEV_1 shall not be hired for a work assignment involving the use of asbestos or materials containing asbestos.

Any employe with a history, symptoms or physical findings of chronic lung disease, or with an abnormal chest x-ray or FEV_1 shall not be permitted to transfer into a work assignment involving the use of asbestos or materials containing asbestos.

Any employe who while working with asbestos or materials containing asbestos develops symptoms or physical findings suggesting lung disease or who is found to have changes in the chest x-ray or FEV_1 shall be removed from the particular work assignment. The employe shall be referred to the plant physician for further medical evaluation.

It is in the best interest of the employe who develops any of the symptoms or findings of chronic lung disease to be permanently restricted from further work with asbestos or materials containing asbestos.

The normal range of the forced expiratory volume (FEV) is 70-85% of the vital capacity.

FEV_1 of 70% or less is not acceptable for employment or transfer

FEV_1 of decreasing value on periodic surveillance may be indicative of onset of lung disease

Chest x-ray - appearance of infiltrates in the lung bases may be indicative of the early stages of asbestosis. The increasing density of the infiltrates and the area of the lung involved indicates progress of the disease.

Medical Records

The records of the above medical examinations shall be retained in the Medical Department files for a minimum of twenty (20) years.

First Aid

There are no special first aid measures.

ASBESTOS

Threshold Limit Value

The 8-hour time-weighed average airborne concentration of dust asbestos to which employes are exposed shall not exceed 5 fibers per milliliter greater than 5 microns in length, as determined by the membrane filter method at 400-450 x magnification (4 millimeter objective) phase contrast illumination. Concentrations above 5 fibers per milliliter, but not to exceed 10 fibers per milliliter, may be permitted up to a total of 15 minutes in an hour for up to 5 hours in an 8-hour day. (OSHA as published in the Federal Register, Dec. 7, 1971).

Monitoring

OSHA limits are based on the membrane filter method at 400-450 x magnification (4 millimeter objective) phase contrast illumination. Initial routine air sampling shall be done weekly until sufficient data is obtained to assure complete coverage of all phases of the work. After the base data is complete, routine sampling shall be done monthly.

Personal Protection

Where engineering control methods are not feasible or where airborne concentrations exceed the Threshold Limit Values, employes shall be provided with and required to use respiratory protective devices. Each employe shall test his respirator before each use in order to insure a proper fit according to the manufacturer's instructions. Employes and supervision must be trained in these techniques.

(a). For an atmosphere containing not more than 25 fibers per milliliter greater than 5 microns in length over an 8-hour average, or more than 50 fibers per milliliter over any period of 15 minutes, a reusable or single-use filter type respirator, operating with negative pressure during the inhalation phase of breathing approved by the U.S. Bureau of Mines (Schedule 21-B), or a valveless respirator providing equivalent protection shall be used. (Mine Safety Appliances Dustfoe or equal).

(b). For an atmosphere containing not more than 250 fibers per milliliter greater than 5 microns in length over an 8-hour average, or more than 500 fibers per milliliter over any period of 15 minutes, a powered filter positive pressure respirator approved by the U.S. Bureau of Mines (Schedule 21-B) shall be used.

(c). Per an atmosphere containing more than 250 fibers per milliliter greater than 5 microns in length over an 8-hour average a type C positive pressure supplied air respirator approved by the U.S. Bureau of Mines (Schedule 19-B) shall be used.

All respirators provided employes shall be properly inspected, cleaned, repaired and stored. A respirator program in accordance with American National Standard Z 88.2-1969 shall be established.

Employees exposed to the spraying of asbestos or the demolition of pipes, structures, or equipment covered or insulated with asbestos shall be provided with respiratory protective devices as described in (C) above.

Special Handling

Asbestos cement, mortar, coatings, grout, and plaster shall be mixed in closed bags or other closed containers.

All hand- or power-operated tools, which produce asbestos dust such as saws, scorers, abrasive wheels, and drills shall be provided with local exhaust ventilation and dust collectors in accordance with American National Standard Z 9.2-1971.

Waste Disposal

All clean-up of asbestos dust and blowing shall be performed by vacuum cleaners. No dry sweeping shall be performed.

Asbestos waste and scrap shall be collected and disposed of in bags or other sealed containers.

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PART 10—ARTICLES CONDITIONALLY FREE, SUBJECT TO A REDUCED RATE, ETC.

Free Withdrawal of Supplies and Equipment for Aircraft

In accordance with section 309(c), Tariff Act of 1930, as amended (19 U.S.C. 1309(c)), the Department of Commerce has failed and under date of April 20, 1972, has advised the Treasury Department that Poland allows privileges to aircraft registered in the United States and engaged in foreign trade substantially reciprocal to those provided for in subparagraph (5) and (17) of the Tariff Act of 1930, as amended (19 U.S.C. 1309, 1317). The same privileges are therefore hereby extended to aircraft registered in Poland and engaged in foreign trade effective as of the date of such notification.

Accordingly, paragraph (f) of § 10.83, customs regulations, is amended by the insertion of Poland in appropriate alphabetical order and the number of this Treasury decision in the "appeals column headed "Treasury Decisions)" in the list of nations in that paragraph.

(Secs. 309, 317, 624, 46 Stat. 690, as amended, 697, as amended, 705; 19 U.S.C. 1309, 1317, 1624)

[SEAL] Edwin F. Klein,
Acting Commissioner of Customs.

Approved: May 23, 1972.

Eugene T. Rossides,
Assistant Secretary of the Treasury.

[FR Doc.72-8319 Filed 6-6-72;8:50 am]

Title 29—LABOR

Chapter XVII—Occupational Safety and Health Administration, Department of Labor

PART 1910—OCCUPATIONAL SAFETY AND HEALTH STANDARDS

Standard for Exposure to Asbestos Dust

On December 7, 1971, an emergency temporary standard concerning exposure to asbestos fibers was published in the FEDERAL REGISTER (36 F.R. 23207). In accordance with section 6(c)(2) of the Williams-Steiger Occupational Safety and Health Act of 1970, a notice of proposed rulemaking regarding a permanent standard for exposure to asbestos fibers was published in the FEDERAL REGISTER on January 13, 1972 (37 F.R. 467). The notice invited interested persons to submit both orally and in writing, data, views, and arguments concerning the proposal.

On or about January 24, 1972, the Advisory Committee on Asbestos Dust was established and requested to make written recommendations with regard to the proposed standard on asbestos. On or about February 1, 1972, the Department of Health, Education, and Welfare transmitted to the Secretary of Labor a criteria document containing Recommendations for an Occupational Exposure Standard for Asbestos by the National Institute for Occupational Safety and Health (NIOSH). Public notice was given of the receipt of the recommendations and their availability for inspection and copying. On or about February 21, 1972, the Advisory Committee on Asbestos Dust submitted its written recommendations to the Assistant Secretary of Labor for Occupational Safety and Health.

Pursuant to the notice of rule making, a hearing was held on March 14 through 17, 1972, for the purpose of receiving oral data, views, and arguments concerning the proposed standard. On or about March 31, 1972, the presiding hearing examiner certified to the Assistant Secretary of Labor for Occupational Safety and Health the record of the proceeding. The record includes prehearing written comments, a transcript of the oral presentations made at the hearing, and numerous exhibits received during the course of the hearing or within the period allowed after the close of the hearing.

The proposed standard dealt with (1) permissible concentrations of asbestos fibers; (2) methods of compliance; (3) warning signs; (4) monitoring; (5) medical examinations; and (6) recordkeeping. Each of these major proposals elicited comments, arguments, objections, and counterproposals. They all have been examined and considered.

1. *Acceptable concentrations of asbestos dust.* The proposed standard would limit occupational exposure to 8-hour time-weighted average (TWA) airborne concentrations of asbestos dust not exceeding five fibers longer than five micrometers per milliliter. Concentrations above five fibers but not to exceed 10 fibers (ceiling concentration) would be permitted up to 15 minutes in an hour, but for not more than 5 hours in any one 8-hour day.

NIOSH in effect has recommended that the five-fiber TWA and 10-fiber peak concentrations be permitted only for 3 years; thereafter, TWA concentrations should be not more than 2 fibers per cubic centimeter (cm.³) of air, and peak concentrations should not exceed 10 fibers/cm.³, with no time restriction. Numerous objections and counterproposals have been made, with regard to both the limits of asbestos fiber concentrations and the time periods to comply with them. Some, for example, have recommended return to a 12-fiber standard of an earlier days; i.e., a level adopted under the Walsh-Healey Public Contracts Act in 1949. Others have recommended a two-fiber standard to become effective in 6 months, then a one-fiber standard for 2 years, and finally a zero-fiber standard after 3 years. These recommendations give a fair indication of the wide spread of the counterproposals.

No one has disputed that exposure to asbestos of high enough intensity and long enough duration is causally related to asbestosis and cancers. The dispute is as to the determination of a specific level below which exposure is safe. Various studies attempting to establish quantitative relations between specific levels of exposure to asbestos fibers and the appearance of adverse biological manifestations, such as asbestosis, lung cancer, and mesothelioma, have given rise to controversy as to the validity of the measuring techniques used and the reliability of the relations attempted to be established. Because of the long lapse of time between onset of exposure and biological manifestations, we have new evidence of the consequences of exposure, but we do not have, in general, adequate measures of the levels of exposure occurring 20 or 30 years ago, which have given rise to these consequences. There are also controversies concerning the relative toxicity of the various kinds of asbestos, and varying hazards in different workplaces.

It is fair to say that the controversy has centered in the area between a two-fiber TWA concentration and five-fiber TWA concentration, with variations on the time needed for compliance. Many employers support a 5-, five-fiber TWA. Much medical opinion is divided between a two-fiber standard and a five-fiber standard.

In view of the undisputed grave consequences from exposure to asbestos fibers, it is essential that the exposure be regulated now on the basis of the best evidence available now, even though it may not be as good as scientifically desirable. An asbestos standard can be re-evaluated in the light of the results of ongoing studies, and future studies, but cannot wait for them. Lives of employees are at stake.

It is concluded that there should be one minimum standard of exposure to asbestos applicable to all workplaces exposed to any kind, or mixture of kinds, of asbestos. Reasons of practical administration preclude a variety of standards for different kinds of asbestos and of workplaces. Also, while the evidence tends to show that crocidolite, for instance, is more harmful than chrysotile, the evidence is not sufficient to establish separate standards for varieties of asbestos.

Because there must be one standard governing exposure to all varieties of asbestos, and in workplaces apparently more hazardous than others; because some present employees with regular exposure to asbestos have probably already accumulated great doses of asbestos fibers, due to higher levels of exposure in the past; because it appears that levels of exposure which may be safe with regard to asbestosis are not safe with regard to mesothelioma; because the statute requires the protection of every employee, even of one who may have regular exposure to asbestos during a working life which may reach, or even exceed, 40 years; and because of several other considerations which have been urged and are reflected in the record of the proceeding, the conflict in the medical evidence is resolved in favor of the health of employees. As of July 1, 1972, TWA concentrations of asbestos fibers longer than 5 micrometers will not be allowed to exceed two fibers/cc., with a ceiling value of 10 fibers/cc. The current TWA concentrations of five fibers, and

(E)

§ 1910.93 Air contaminants.

* * * * *

Table G-3—Mineral Dusts

Substance	mppcf[a]	mg/m³
Silica:		
Crystalline:		
Quartz (respirable)	250[b]	10 mg/m³ / %SiO₂+2
	%SiO₂+5	
Quartz (total dust)		30 mg/m³ / %SiO₂+2
Cristobalite: Use ½ the value calculated from the count or mass formulae for quartz.		
Tridymite: Use ½ the value calculated from the formulae for quartz.		
Amorphous, including natural diatomaceous earth	20	80 mg/m³ / %SiO₂
Silicates (less than 1% crystalline silica):		
Mica	20	
Soapstone	20	
Talc	20	
Portland cement	50	
Graphite (natural)	15	
Coal dust (respirable fraction less than 5% SiO₂)		2.4 mg/m³ or
For more than 5% SiO₂		10 mg/m³ / %SiO₂+2
Inert or Nuisance Dust:		
Respirable fraction	15	5 mg/m³
Total dust	50	15 mg/m³

Note: Conversion factors—
mppcf × 35.3 = million particles per cubic meter = particles per c.c.

a Millions of particles per cubic foot of air, based on impinger samples counted by light-field technics.

b The percentage of crystalline silica in the formula is the amount determined from airborne samples, except in those instances in which other methods have been shown to be applicable.

c As determined by the membrane filter method at 430 X phase contrast magnification.

d Both concentration and percent quartz for the application of this limit are to be determined from the fraction passing a size-selector with the following characteristics:

Aerodynamic diameter (unit density sphere)	Percent passing selector
2	90
2.5	75
3.5	50
5.0	25
10	0

The measurements under this note refer to the use of an AEC instrument. If the respirable fraction of coal dust is determined with a MRE the figure corresponding to that of 2.4 mg/m³ in the table for coal dust is 4.5 mg/m³.

2. A new § 1910.93a is added to Part 1910, reading as follows:

§ 1910.93a Asbestos.

(a) *Definitions.* For the purpose of this section, (1) "Asbestos" includes chrysotile, amosite, crocidolite, tremolite, anthophyllite, and actinolite.

(2) "Asbestos fibers" means asbestos fibers longer than 5 micrometers.

(b) *Permissible exposure to airborne concentrations of asbestos fibers*—(1) *Standard effective July 7, 1972.* The 8-hour time-weighted average airborne concentrations of asbestos fibers to which any employee may be exposed shall not exceed five fibers, longer than 5 micrometers, per cubic centimeter of air, as determined by the method prescribed in paragraph (e) of this section.

(2) *Standard effective July 1, 1976.* The 8-hour time-weighted average airborne concentrations of asbestos fibers to which any employee may be exposed shall not exceed two fibers, longer than 5 micrometers, per cubic centimeter of air, as determined by the method prescribed in paragraph (e) of this section.

(3) *Ceiling concentration.* No employee shall be exposed at any time to airborne concentrations of asbestos fibers in excess of 10 fibers, longer than 5 micrometers, per cubic centimeter of air, as determined by the method prescribed in paragraph (e) of this section.

(c) *Methods of compliance*—(1) *Engineering methods.* (i) *Engineering controls.* Engineering controls, such as, but not limited to, isolation, enclosure, exhaust ventilation, and dust collection, shall be used to meet the exposure limits prescribed in paragraph (b) of this section.

(ii) *Local exhaust ventilation.* (a) Local exhaust ventilation and dust collection systems shall be designed, constructed, installed, and maintained in accordance with the American National Standard Fundamentals Governing the Design and Operation of Local Exhaust Systems, ANSI Z9.2-1971, which is incorporated by reference herein.

(b) See § 1910.6 concerning the availability of ANSI Z9.2-1971, and the maintenance of a historic file in connection therewith. The address of the American National Standards Institute is given in § 1910.100.

(iii) *Particular tools.* All hand-operated and power-operated tools which may produce or release asbestos fibers in excess of the exposure limits prescribed in paragraph (b) of this section, such as, but not limited to, saws, scorers, abrasive wheels, and drills, shall be provided with local exhaust ventilation systems in accordance with subdivision (ii) of this subparagraph.

(2) *Work practices*—(i) *Wet methods.* Insofar as practicable, asbestos shall be handled, mixed, applied, removed, cut, scored, or otherwise worked in a wet state sufficient to prevent the emission of airborne fibers in excess of the exposure limits prescribed in paragraph (b) of this section, unless the usefulness of the product would be diminished thereby.

(ii) *Particular products and operations.* No asbestos cement, mortar, coating, grout, plaster, or similar material containing asbestos shall be removed from bags, cartons, or other containers in which they are shipped, without being either wetted, or enclosed, or ventilated so as to prevent effectively the release of airborne asbestos fibers in excess of the limits prescribed in paragraph (b) of this section.

(iii) *Spraying, demolition, or removal.* Employees engaged in the spraying of asbestos, the removal, or demolition of pipes, structures, or equipment covered or insulated with asbestos, and in the removal or demolition of asbestos insulation or coverings shall be provided with respiratory equipment in accordance with paragraph (d)(2)(iii) of this section and with special clothing in accordance with paragraph (d)(3) of this section.

(d) *Personal protective equipment*—(1) Compliance with the exposure limits prescribed by paragraph (b) of this section may not be achieved by the use of respirators or shift rotation of employees, except:

(i) During the time period necessary to install the engineering controls and to institute the work practices required by paragraph (c) of this section;

(ii) In work situations in which the methods prescribed in paragraph (c) of this section are either technically not feasible or feasible to an extent insufficient to reduce the airborne concentrations of asbestos fibers below the limits prescribed by paragraph (b) of this section; or

(iii) In emergencies.

(iv) Where both respirators and personnel rotation are allowed by subdivisions (i), (ii), or (iii) of this subparagraph, and both are practicable, personnel rotation shall be preferred and used.

(2) Where a respirator is permitted by subparagraph (1) of this paragraph, it shall be selected from among those approved by the Bureau of Mines, Department of the Interior, or the National Institute for Occupational Safety and Health, Department of Health, Education, and Welfare, under the provisions of 30 CFR Part 11 (37 F.R. 6244, Mar. 25, 1972), and shall be used in accordance with subdivisions (i), (ii), (iii), and (iv) of this subparagraph.

(i) *Air purifying respirators.* A reusable or single use air purifying respirator, or a respirator described in subdivision (ii) or (iii) of this subparagraph, shall be used to reduce the concentrations of airborne asbestos fibers in the respirator below the exposure limits prescribed in paragraph (b) of this section, when the ceiling or the 8-hour time-weighted average airborne concentrations of asbestos fibers are reasonably expected to exceed no more than 10 times those limits.

(ii) *Powered air purifying respirators.* A full facepiece powered air purifying respirator, or a powered air purifying respirator, or a respirator described in subdivision (iii) of this subparagraph, shall be used to reduce the concentrations of airborne asbestos fibers in the respirator below the exposure limits prescribed in paragraph (b) of this section, when the ceiling or the 8-hour time-weighted average concentrations of asbestos fibers are reasonably expected to exceed 10 times, but not 100 times, those limits.

(iii) *Type "C" supplied-air respirators, continuous flow or pressure-demand class.* A type "C" continuous flow or pressure-demand, supplied-air respirator shall be used to reduce the concentrations of airborne asbestos fibers in the respirator below the exposure limits prescribed in paragraph (b) of this section, when the ceiling or the 8-hour time-weighted average airborne concentrations of asbestos fibers are reasonably expected to exceed 100 times those limits.

(iv) *Establishment of a respirator program.* (a) The employer shall establish a respirator program in accordance with

ceiling concentrations of 10 fibers/cc. will be permitted until July 1, 1976, during what will be a transitional period deemed necessary to allow employers to make the needed changes for coming into compliance with the more stringent standard.

The record shows that the many work operations subject to the single asbestos standard (textile manufacturing, fabricating, and marine installation, etc.) will meet varying degrees of difficulty in complying with the standard. In some plants, extensive redesign and relocation of equipment may be needed. It appears, however, the delay in the effective date of the two-fiber standard will provide all employers a reasonable time to comply. At the same time, so long as the ceiling limit is complied with, no harm is reasonably expected to result from exposures during the transitional period.

2. Methods of compliance. It has been pointed out by many persons, that protection against asbestos fibers is best obtained by controlling the generation of fibers first, and secondly, by controlling the dispersion of released fibers into the ambient air of the workplaces. Therefore, the standard requires feasible technological controls and appropriate work practices as the primary means of compliance. Rotation of employees as a way of meeting the TWA concentration requirement is allowed only in stated exceptional circumstances, because, as a general rule, it would be difficult to implement. Personal protective equipment, such as respirators, cannot be relied upon because, among other reasons, they may be so uncomfortable as to be burdensome, except for short periods of time. Therefore, it is expected that respirators and shift rotation will be used during the period necessary to install engineering controls and to train employees in sound work practices, but, after technological compliance has been achieved, their use must be limited to special work situations and emergencies. Where both are practicable, shift rotation is required.

3. Labeling. The proposed standard stopped short of requiring labeling asbestos and asbestos-containing products. The proposed standard would have required only warning signs at locations where asbestos hazards are present. However, labeling, rather than warning signs, has proved to be a point of controversy. Both NIOSH and the Advisory Committee on Asbestos Dust recommended labels for asbestos products and containers, and these recommendations became very controversial in the course of the proceeding. Many counterproposals have been made as to the language of the warning as well as to the products to be subject to the labeling requirements. Employers, in general, strongly contend that (1) finished products which effectively entrap asbestos fibers, so that these would not be released in the normal use of the products, should not be required to be labeled; and (2) words such as "danger" and "cancer" are unwarrantedly alarming.

Both contentions have merit, and the standard has been changed accordingly.

4. Monitoring. The proposed standard would have required personal monitoring and environmental monitoring. Many issues have been raised concerning the availability and reliability of measuring instruments, frequency of monitoring, and conditions in which monitoring should be required. The adopted standard takes the objections into consideration. It requires periodic monitoring at intervals no longer than 6 months, thus allowing considerable time and discretion, and prescribes the use of the membrane filter method, which is an acceptable method for determination of asbestos fibers.

It has also been recommended that employees or their representatives should have an opportunity to observe the monitoring. The recommendation has been accepted.

5. Medical examinations. The proposed standard would only require an appropriate medical examination on a periodic basis. The generality of the proposal has attracted many objections and also many helpful comments. The recommendations of NIOSH and of the Advisory Committee on Asbestos Dust were much more specific with respect to both frequency and type of medical examinations to be required. The comments vary as to the class of employees to be examined and as to the frequency of the examinations.

The adopted standard requires medical examinations both at the beginning and the termination of employments exposed to concentrations of asbestos fibers, and also requires annual medical examinations of every employee exposed to airborne concentrations of asbestos. It has been pointed out that in certain industries, such as construction, an employee may work for several employers during the same year. Accordingly, the standard does not require either preemployment, or termination, or periodic examination of any employee who has been examined in accordance with the standard within the past year.

One question which has been raised goes to whether the employer or the employee should be allowed to choose the examining physician. The standard gives the option to the employer. Since some employers already have a medical examination program in operation, and, also, have medical departments with some expertise in the diagnosis of asbestos-related diseases, it seems more reasonable to permit them to utilize the present programs and expertise, than to permit an employee to choose a private general practitioner.

6. Records. The standard, as proposed and as adopted, requires maintenance of records of monitoring and of medical examinations. Most of the controversy in this area has revolved around the question whether an employer should be allowed to have access to the results of the required medical examinations. The apprehension of those who have argued against employer access is based on the expectation that some employers will use the medical examinations as a means of screening employment applicants, and worse, as grounds for discharging current employees, who show signs of being affected by exposure to asbestos. Since the purpose of the medical examinations is to monitor the health of employees exposed to the hazards of asbestos, employers cannot in reason be granted the privilege of refusing to disclose to their employees results of occupational exposure. It does not make sense to require employers to provide medical examinations if they cannot know and use the results of the examinations. For these reasons the standard provides that employers may have a restricted access to some medical information.

On the other hand, there is no intention to allow employers to abuse medical information obtained pursuant to the Act, to the detriment of employees. Therefore, the administration of the medical records requirement will be closely watched, and, in cases of abuse, appropriate action will be considered.

The issues discussed above are believed to be the major ones. Numerous other issues have been raised in the rulemaking proceedings. Some have been referred to incidentally. Many recommendations, for instance, about work practices, are so obviously meritorious that their adoption needs no exposition here. Other recommendations and many objections have not been adopted for a variety of reasons which should be manifest. Several, for instance, have recommended the use of respirators only pursuant to a variance, or in cases of emergency and occasional short-term exposure. The recommendation with respect to variances undoubtedly has many merits, but is considered administratively impractical.

Accordingly, after consideration of the whole record of the proceeding, and pursuant to sections 6 (b) and (c) and 8(c) of the Williams-Steiger Occupational Safety and Health Act of 1970 (84 Stat. 1593, 1596, 1599; 29 U.S.C. 655, 657), 29 CFR 1910.4, and the Secretary of Labor's Order No. 12-71 (36 F.R. 8754), Part 1910 of Title 29 of the Code of Federal Regulations is amended as set forth below.

(1) Section 1910.93 is amended by revising Table G-2 to read as follows:

the requirements of the American National Standards Practice for Respiratory Protection, ANSI Z88.2-1969, which is incorporated by reference herein.

h. See § 1910.5 concerning the availability of ANSI Z88.2-1969 and the maintenance of an historic file in connection therewith. The address of the American National Standards Institute is given in § 1910.800.

(c) No employee shall be assigned to tasks requiring the use of respirators if, based upon his most recent examination, an examining physician determines that the employee will be unable to function normally wearing a respirator, or that the safety or health of the employee or other employees will be impaired by his use of a respirator. Such employee shall be rotated to another job or given the opportunity to transfer to a different position whose duties he is able to perform with the same employer, in the same geographical area and with the same seniority, status, and rate of pay he had just prior to such transfer, if such a different position is available.

(2) *Special clothing:* The employer shall provide, and require the use of, special clothing, such as coveralls or similar whole body clothing, head coverings, gloves, and foot coverings for any employee exposed to airborne concentrations of asbestos fibers, which exceed the ceiling level prescribed in paragraph (b) of this section.

(d) *Change rooms:* (1) At any fixed place of employment exposed to airborne concentrations of asbestos fibers in excess of the exposure limits prescribed in paragraph (b) of this section, the employer shall provide change rooms for employees working regularly at the place.

(ii) *Clothes lockers:* The employer shall provide two separate lockers or containers for each employee, so separated or isolated as to prevent contamination of the employee's street clothes from his work clothes.

(iii) *Laundering:* (a) Laundering of asbestos contaminated clothing shall be done so as to prevent the release of airborne asbestos fibers in excess of the exposure limits prescribed in paragraph (b) of this section.

(b) Any employer who gives asbestos-contaminated clothing to another person for laundering shall inform such person of the requirement in (a) of this subdivision to effectively prevent the release of airborne asbestos fibers in excess of the exposure limits prescribed in paragraph (b) of this section.

(c) Contaminated clothing shall be transported in sealed impermeable bags, or other closed, impermeable containers, and labeled in accordance with paragraph (g) of this section.

(e) *Method of measurement.* All determinations of airborne concentrations of asbestos fibers shall be made by the membrane filter method at 400–450 × (magnification) (4 millimeter objective) with phase contrast illumination.

(f) *Monitoring.*—(1) *Initial determinations.* Within 6 months of the publication of this section, every employer shall cause every place of employment [illegible] monitored in such a way as to determine whether every employee's exposure to asbestos fibers is below the limits prescribed in paragraph (b) of this section: If the limits are exceeded, the employer shall immediately undertake a compliance program in accordance with paragraph (a) of this section.

(2) *Personal monitoring.*—(i) Samples shall be collected from within the breathing zone of the employee, on membrane filters of 0.5 micrometer porosity mounted in an open-face filter holder. Samples shall be taken for the determination of the 8-hour time-weighted average airborne concentrations and of the ceiling concentrations of asbestos fibers.

(ii) *Sampling frequency and patterns.* After the initial determinations required by subparagraph (1) of this paragraph, samples shall be of such frequency and pattern as to represent with reasonable accuracy the levels of exposure of employees. In no case shall the sampling be done at intervals greater than 6 months for employees whose exposure to asbestos may reasonably be foreseen to exceed the limits prescribed by paragraph (b) of this section.

(3) *Environmental monitoring.*—(i) Samples shall be collected from areas of a work environment which are representative of the airborne concentrations of asbestos fibers which may reach the breathing zone of employees. Samples shall be collected on a membrane filter of 0.5 micrometer porosity mounted in an open-face filter holder. Samples shall be taken for the determination of the 8-hour time-weighted average airborne concentrations and of the ceiling concentrations of asbestos fibers.

(ii) *Sampling frequency and patterns.* After the initial determinations required by subparagraph (1) of this paragraph, samples shall be of such frequency and pattern as to represent with reasonable accuracy the levels of exposure of the employees. In no case shall sampling be at intervals greater than 6 months for employees whose exposure to asbestos may reasonably be foreseen to exceed the exposure limits prescribed in paragraph (b) of this section.

(4) *Employee observation of monitoring.* Affected employees, or their representatives, shall be given a reasonable opportunity to observe any monitoring required by this paragraph and shall have access to the records thereof.

(g) *Caution signs and labels.* (1) *Caution signs.* (i) *Posting.* Caution signs shall be provided and displayed at each location where airborne concentrations of asbestos fibers may be in excess of the exposure limits prescribed in paragraph (b) of this section. Signs shall be posted at such a distance from such a location so that an employee may read the signs and take necessary protective steps before entering the area marked by the signs. Signs shall be posted at all approaches to areas containing excessive concentrations of airborne asbestos fibers.

(ii) *Sign specifications.* The warning signs required by subdivision (i) of this subparagraph shall conform to the requirements of 20" × 14" vertical format signs specified in § 1910.145(d)(4), and to this subdivision. The signs shall display the following legend in the lower panel, with letter sizes and styles of a visibility at least equal to that specified in this subdivision.

Legend	Minimum
Asbestos	1" Sans-Serif, Gothic or Block.
Dust Hazard	½" Sans Serif, Gothic or Block.
Avoid Breathing Dust	½" Gothic.
Wear Assigned Protective Equipment.	½" Gothic.
Do Not Remain In Area Unless Your Work Requires It.	½" Gothic.
Breathing Asbestos Dust May Be Hazardous To Your Health.	14 point Gothic.

Spacing between lines shall be at least equal to the height of the upper of any two lines.

(2) *Caution labels.*—(i) *Labeling.* Caution labels shall be affixed to all raw materials, mixtures, scrap, waste, debris, and other products containing asbestos fibers, or to their containers, except that no label is required where asbestos fibers have been modified by a bonding agent, coating, binder, or other material so that during any reasonably foreseeable use, handling, storage, disposal, processing, or transportation, no airborne concentrations of asbestos fibers in excess of the exposure limits prescribed in paragraph (b) of this section will be released.

(ii) *Label specifications.* The caution labels required by subdivision (i) of this subparagraph shall be printed in letters of sufficient size and contrast as to be readily visible and legible. The label shall state:

CAUTION
Contains Asbestos Fibers
Avoid Creating Dust
Breathing Asbestos Dust May Cause Serious Bodily Harm

(h) *Housekeeping.*—(1) *Cleaning.* All external surfaces in any place of employment shall be maintained free of accumulations of asbestos fibers if, with their dispersion, there would be an excessive concentration.

(2) *Waste disposal.* Asbestos waste, scrap, debris, bags, containers, equipment, and asbestos-contaminated clothing, consigned for disposal, which may produce in any reasonably foreseeable use, handling, storage, processing, disposal, or transportation airborne concentrations of asbestos fibers in excess of the exposure limits prescribed in paragraph (b) of this section shall be collected and disposed of in sealed impermeable bags, or other closed, impermeable containers.

(i) *Recordkeeping.*—(1) *Exposure records.* Every employer shall maintain records of any personal or environmental monitoring required by this section. Records shall be maintained for a period of at least 3 years and shall be made available upon request to the Assistant Secretary of Labor for Occupational Safety and Health, the Director of the National

Institute for Occupational Safety and Health, and to authorized representatives of either.

(3) Employee access. Every employee and former employee shall have reasonable access to any record required to be maintained by subparagraph (2) of this paragraph, which indicates the employee's own exposure to asbestos fibers.

(4) Employee notification. Any employee found to have been exposed at any time to airborne concentrations of asbestos fibers in excess of the limits prescribed in paragraph (b) of this section shall be notified in writing of the exposure as soon as practicable but not later than 5 days of the finding. The employee shall also be timely notified of the corrective action being taken.

(5) Medical examinations.—(i) General. The employer shall provide or make available at his cost, medical examinations relative to exposure to asbestos required by this paragraph.

(ii) Preplacement. The employer shall provide or make available to each of his employees, within 30 calendar days following his first employment in an occupation exposed to airborne concentrations of asbestos fibers, a comprehensive medical examination, which shall include, as a minimum, a chest roentgenogram (posterior-anterior 14 x 17 inches), a history to elicit symptomatology of respiratory disease, and pulmonary function tests to include forced vital capacity (FVC) and forced expiratory volume at 1 second (FEV$_{1.0}$).

(iii) Annual examinations. On or before January 31, 1973, and at least annually thereafter, every employer shall provide, or make available, comprehensive medical examinations to each of his employees engaged in occupations exposed to airborne concentrations of asbestos fibers. Such annual examination shall include, as a minimum, a chest roentgenogram (posterior-anterior 14 x 17 inches), a history to elicit symptomatology of respiratory disease, and pulmonary function tests to include forced vital capacity (FVC) and forced expiratory volume at 1 second (FEV$_{1.0}$).

(iv) Termination of employment. The employer shall provide, or make available, within 30 calendar days before or after the termination of employment of any employee engaged in an occupation exposed to airborne concentrations of asbestos fibers, a comprehensive medical examination which shall include, as a minimum, a chest roentgenogram (posterior-anterior 14 x 17 inches), a history to elicit symptomatology of respiratory disease, and pulmonary function tests to include forced vital capacity (FVC) and forced expiratory volume at 1 second (FEV$_{1.0}$).

(v) Recent examinations. No medical examination is required of any employee, if adequate records show that the employee has been examined in accordance with this paragraph within the past 1-year period.

(6) Medical records.—(i) Maintenance. Employers of employees examined pursuant to this paragraph shall cause to be maintained complete and accurate records of all such medical examinations. Records shall be retained by employers for at least 20 years.

(ii) Access. The contents of the records of the medical examinations required by this paragraph shall be made available, for inspection and copying, to the Assistant Secretary of Labor for Occupational Safety and Health, the Director of NIOSH, to authorized physicians and medical consultants of either of them, and, upon the request of an employee or former employee, to his physician. Any physician who conducts a medical examination required by this paragraph shall furnish to the employer of the examined employee all the information specifically required by this paragraph, and any other medical information related to occupational exposure to asbestos fibers.

E. A new § 1910.19 is added to Subpart B of Part 1910, reading as follows:

§ 1910.19 Asbestos dust.

Section 1910.93a shall apply to the exposure of every employee to asbestos dust in every employment and place of employment covered by § 1910.12, § 1910.13, § 1910.14, § 1910.15, or § 1910.16, to any of any different standard on exposure to asbestos dust which would otherwise be applicable by virtue of any of these sections.

Effective date. Paragraph (b)(2) of § 1910.93a shall become effective July 1, 1972. All other provisions of §§ 1910.93a, 1910.93, and 1910.19 shall become effective July 7, 1972. The current emergency temporary standard remains in effect until July 7, 1972.

(Secs. 6, 8, 84 Stat. 1593, 1600; 29 U.S.C. 655, 657; 29 CFR 1910.6; Secretary of Labor's Order No. 12-71, 36 FR. 8754)

Signed at Washington, D.C., this 2d day of June 1972.

G. C. Guenther,
Assistant Secretary of Labor.

[FR Doc.72-8476 Filed 6-6-72;8:45 am]

Title 41—PUBLIC CONTRACTS AND PROPERTY MANAGEMENT

Chapter 9—Atomic Energy Commission

PART 9-1—GENERAL

Subpart 9-1.1—Procurement Regulations

ATOMIC ENERGY ACQUISITION REGULATIONS

The changes made to AECPR, Subpart 9-1.1, Procurement Regulations, have been made in order to establish the AECPR Temporary Regulations which are a part of the AEC Procurement Regulations and the Federal Procurement Regulations System. The AECPR Temporary Regulations implement and supplement the FPR Temporary Regulations. They also contain policies and procedures initiated by the AEC which are to be effective for a period of 6 months or less. The AEC Procurement Regulation section has been revised accordingly. Minor editorial changes have also been made.

1. Section 9-1.101, Scope of subpart, is revised to read as follows:

§ 9-1.101 Scope of subpart.

This subpart describes the Atomic Energy Commission Procurement Regulations and the AECPR Temporary Regulations. It also contains exemptions from the AECPR as contained in the AEC Procurement Instructions.

2. Section 9-1.102 Establishment of AEC Procurement Regulations, is revised to read as follows:

§ 9-1.102 Establishment of the AEC Procurement Regulations and the AECPR Temporary Regulations.

§ 9-1.102-1 AEC Procurement Regulations.

(a) The AEC Procurement Regulations (AECPR) are hereby established.

(b) These regulations implement and supplement the Federal Procurement Regulations (FPR) and are a part of the Federal Procurement Regulations System.

(c) The effective date of FPR issuances throughout AEC will be the date indicated in the respective issuances unless otherwise provided in the AEC Procurement Regulations.

(d) The effective date of AECPR issuances throughout AEC will be the date indicated in the respective issuances.

§ 9-1.102-2 AECPR Temporary Regulations.

(a) The AECPR Temporary Regulations are hereby established.

(b) These regulations implement and supplement the Federal Procurement Regulations Temporary Regulations. They also contain policies and procedures initiated by the AEC which are expected to be effective for a period of 6 months or less.

(c) The effective date of the FPR Temporary Regulations issuances throughout AEC will be the date indicated in the respective issuances unless otherwise provided in the AECPR Temporary Regulations.

(d) The effective date of the AECPR Temporary Regulations issuances throughout AEC will be the date indicated in the respective issuances.

(e) The AECPR Temporary Regulations are a part of the AEC Procurement Regulations and the Federal Procurement Regulations System. All references to the AEC Procurement Regulations or AECPR in §§ 9-1.100 through 9-1.109 of this subpart shall be deemed to include the AECPR temporary regulations.

3. Section 9-1.103 Authority is revised to read as follows:

§ 9-1.103 Authority.

The AEC Procurement Regulations are prescribed by the General Manager, Assistant General Manager for Administration, or the Director, Division of Contracts of the AEC pursuant to the authority of the Atomic Energy Act of 1954, and the Federal Property and Administrative Services Act of 1949.