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# Asbestos

## **Getting Back To Basics: Evidence-Based Science In The Courtroom**

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# Commentary

## Getting Back To Basics: Evidence-Based Science In The Courtroom

By  
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Almost a decade ago, the California Supreme Court held in *Sargon Enterprises, Inc. v. University of Southern California* that trial court judges have “the duty to act as a ‘gatekeeper’ to exclude speculative expert testimony.”<sup>1</sup> Despite the court’s decision, juries in asbestos-related personal injury cases are frequently presented with expert opinions based only on scientific possibilities, not actual science.<sup>2</sup> In particular, as the quantity of asbestos fibers potentially released from a product on trial (*a.k.a.*, dose) decreases, the quantity of science supporting medical causation opinions about that product also decreases. The California Second District Court of Appeal’s 2016 decision in *Davis v. Honeywell International Inc.* illustrates how this has played out in California courts.<sup>3</sup>

The observed relationship between the dose and science at trial presented by asbestos plaintiff experts mirrors the linear no-threshold risk assessment model (“LNT model”) utilized by public health entities that set polices which err on the side of overprotec-

tion. *Spoiler-alert:* Dr. Irving J. Selikoff, commonly described as the pioneer of asbestos-medicine, wrote that this type of model is scientifically unreliable.<sup>4</sup>

At trial in low-dose cases, science is replaced by public health policy statements (*e.g.*, no “safe” dose) and unproven hypotheses (*e.g.*, every exposure above “background” increases risk) – all based on the LNT model.<sup>5</sup> Relatedly, in cases that raise asbestos-contamination claims (*e.g.*, cosmetic talc, vermiculite) where the product is not designed to contain asbestos, new techniques for product testing, newly-coined definitions, and new methods for quantification of asbestos content in a non-homogenous product are also showing up at trials despite California’s “general acceptance” *Kelly* rule.<sup>6</sup>

The root of the missing-science problem in California courtrooms is diffuse and involves misconceptions about law, science, and the historical development of both. Unfortunately, *Davis* was decided against a backdrop of these types of misconceptions.<sup>7</sup> This article addresses a few of the protections available under California law to ensure proper gatekeeping against expert opinion testimony unsupported by science in the asbestos context. *Davis* will be used an exemplar case. Knowing the past and understanding the present are key components to bringing science back into the courtroom.

### A. The Science Problem: Understanding the Legal Limits

“Dose makes the poison” is a central tenant of toxicology.<sup>8</sup> The California Supreme Court’s landmark

decision in *Rutherford v. Owens-Illinois, Inc.*<sup>9</sup> mandates that California courts employ a qualitative threshold to limit the liability of a defendant that may have contributed an insignificant or modest dose (*i.e.*, quantity of asbestos fibers present in one's breathing zone).<sup>10</sup> Pursuant to *Rutherford* (and the field of toxicology), not any, each, or every exposure (*a.k.a.*, dose) to a defendant's product is sufficient to establish legal causation—more is required. That “more” is the establishment, in reasonable medical probability, that a particular exposure or series of exposures was a substantial factor contributing to the risk of developing cancer.

Based on *Rutherford*, a jury in an asbestos case is instructed on causation as follows:

A substantial factor in causing harm is a factor that a reasonable person would consider to have contributed to the harm. It does not have to be the only cause of the harm.

[Plaintiff] may prove that exposure to asbestos from [Defendant]'s product was a substantial factor causing [Plaintiff's] illness by showing, through expert testimony, that there is a **reasonable medical probability** that the exposure was a substantial factor contributing to [Plaintiff's] risk of developing cancer.<sup>11</sup>

The reasonable medical probability standard of proof raises a significant gatekeeping issue in an asbestos personal injury case: expert testimony regarding a product's contribution to the risk of cancer is only relevant (and thus only admissible) if it satisfies the “reasonable medical probability” standard of proof. Expert testimony that purports to satisfy this standard should be excluded if it is invalid and unreliable. Additionally, the jury needs to be instructed on how to determine if a plaintiff's evidence satisfies the “reasonable medical probability” standard of proof included in the jury instruction they are given.

### **B. “Reasonable Medical Probability” is More Than a Possibility**

Prior to *Rutherford*, the California Supreme Court had not addressed the meaning of the “reasonable medical probability” standard of proof. Although it does not define the standard, *Rutherford* holds that the

“reasonable medical probability” standard articulated in *Lineaweaver v. Plant Insulation Co.*<sup>12</sup> is applicable to asbestos personal injury cases.<sup>13</sup> *Lineaweaver* defines the standard by citing language from *Jones v. Ortho Pharmaceutical Corp.*, a carcinogenic pharmaceutical personal injury case:

While there are many possible causes of any injury, ‘[a] possible cause only becomes ‘probable’ when, in the absence of other reasonable causal explanations, it becomes more likely than not that the injury was a result of its action. This is the outer limit of inference upon which an issue may be submitted to the jury.’<sup>14</sup>

In defining this standard, *Jones* finds the following discussion taken from a Texas case persuasive:

[O]nce the theory of causation leaves the realm of lay knowledge for esoteric scientific theories, the scientific theory must be more than a possibility to the scientists who created it. For to the scientific mind, all things are possible. And with all things possible, citizens would have no reasoned protection from the speculations of courts and juries.<sup>15</sup>

Based on above, the reasonable medical probability standard requires a showing of something more than proof that a theory is possible. In this regard, *Davis* got it wrong in deciding a court's gatekeeping obligations in an asbestos case. *Davis* ignores the reasonable medical probability standard altogether. Instead, it incorrectly, and without precedent, determined admissibility of the “each and every” exposure theory based on a “not illogical to conclude” standard.<sup>16</sup> The fact that something is not illogical does not equate to it being probable.

Notably, of the seven California Supreme Court cases that even contain the phrase “reasonable medical probability,” only *Cottle v. Superior Court*,<sup>17</sup> a toxic tort case, defines the standard, and it does so by citing to the same *Jones* language reiterated by *Lineaweaver*.

In assessing the medical probability that an exposure contributed to a plaintiff's asbestos disease (*i.e.*, was a substantial factor), *Lineaweaver* identifies many fac-

tors that are relevant: frequency of exposure, regularity of exposure, and proximity of the asbestos product to plaintiff, type of asbestos product to which plaintiff was exposed, the type of injury suffered by plaintiff, and other possible sources of plaintiff's injury.<sup>18</sup>

Similarly, *Rutherford*, referring to *Lineaweaver*, states that the question of whether or not the risk of cancer created by a particular product is significant enough to be considered a legal cause of disease (*i.e.*, a substantial factor) requires an accounting of “the length, frequency, proximity and intensity of exposure, the peculiar properties of the individual product, any other potential causes to which the disease could be attributed (*e.g.*, other asbestos products, cigarette smoking), and perhaps other factors affecting the assessment of comparative risk.”<sup>19</sup>

Based on the above discussion, several observations are to be made:

1. The phrase “reasonable medical probability” defines the outer limit of inference upon which an issue may even be submitted to the jury. In other words, if the evidence shows only a possibility of contribution to risk instead of a reasonable medical probability of contribution, then the judge should not pass the issue to the jury.
2. Because the “reasonable medical probability” standard of proof is determinative of whether an issue may be submitted to the jury, the judge must decide if the evidence shows that a product, in the absence of other reasonable causal explanations (*e.g.*, other more significant exposures), more likely than not contributed to a person's risk of cancer.
3. In deciding whether the evidence shows that a product more likely than not contributed to a person's risk of cancer, the court should consider whether an expert considered the many relevant factors identified in both *Rutherford* and *Lineaweaver*, including length, frequency, proximity, and intensity of exposure, the peculiar properties of the individual product, and any other potential causes to which the disease could be attributed (*e.g.*, other asbestos products, cigarette smoking).
4. If the court finds that the evidence is sufficient for a jury to find that a that a product more

likely than not contributed to a person's risk of cancer, the jury needs to be instructed on those same factors identified in *Rutherford* and *Lineaweaver* so that it can decide if the evidence satisfies the “reasonable medical probability” standard of proof.

On this last point, based on the discussion above, *Davis* incorrectly states that the factors identified in *Rutherford* and *Lineaweaver* are applicable only to an expert rendering her medical opinion.<sup>20</sup> In accordance with *Rutherford*, CACI 435 instructs the jury to decide if a plaintiff's expert's opinion satisfies the “reasonable medical probability” standard of proof. The jury cannot decide this if it is not instructed on how to do it. Thus, this type of instruction, which the *Davis* court found to be unnecessary, is necessary for the jury to do its job in accordance with the causation standard set forth in *Rutherford*.

### C. LNT Model Does Not Rise to the Level of a Reasonable Medical Probability

A plaintiff's problem in a low-dose product case is that there are no scientific studies that prove that low doses of asbestos contribute to a person's risk of cancer. Plaintiffs' experts have attempted to bridge the gap between the scientific data available regarding high-dose exposures (*e.g.*, insulation, cement, textile manufacturing) and a lack of scientific data available regarding low-dose exposures by reliance on the LNT model. This is the same model utilized by OSHA, EPA, and other public health agencies for risk assessments.

There is a growing list of LNT model derivative theories offered at trial:

- “There has never been established a level of exposure to asbestos that does not increase one's risk of the cancer.”
- “Look at the CPSC, look at IARC, look at OSHA, look at the CDC, look at the American Cancer Society. Look at the people who nobody is paying who give you an opinion in this case, and when you look at those it's a unanimous answer. Unanimous. There is no safe level of exposure when it comes to asbestos products and cancer.”
- “Every exposure above background increases a person's risk of cancer.”

- “You could not as a matter of fundamental biology parse that dose out and say that this dose, did not contribute to the risk.”
- “In somebody who actually gets the disease, all of the exposures given a sufficient minimum latency acted cumulatively together to cause in that individual the disease that he got.”

But an expert opinion based on any of these types of theories is not allowed under California law.

For background, the LNT model was never designed to be used for a medicolegal causal analysis. Instead, a LNT model assumes that every dose increment, no matter how small, constitutes an increased cancer risk. The LNT model draws a straight line between the point of departure from observed data and the origin (*i.e.*, zero). The linear default provides an upper-bound calculation of potential risk at low doses.<sup>21</sup> In other words, it overestimates risk on purpose.

An overestimation of risk may be useful for achieving public policies, but is not determinative of actual risk, the relevant inquiry in any personal injury case. LNT model assumptions include: (1) cancer risk is linearly proportionate to dose; (2) there is no threshold (*i.e.*, any exposure level causes risk) and risk is additive; and (3) dosage outweighs any biological variables.<sup>22</sup> As *Sargon* itself states: an expert’s opinion may not be based on assumptions of fact without evidentiary support, or on speculative or conjectural factors.<sup>23</sup>

EPA uses the LNT model when: (1) there is an absence of sufficient information on modes of action or (2) the mode of action (“MOA”) information indicates that the dose response curve at low dose is or is expected to be linear.<sup>24</sup> EPA as recently as December 2020 (Final Draft Evaluation for Asbestos Part I: Chrysotile Asbestos) confirmed that “there is currently insufficient information to determine the MOA for either chrysotile lung carcinogenicity or mesothelioma.”<sup>25</sup> Accordingly, “because MOA for chrysotile asbestos is uncertain, following the recommendations of the Guidelines for Carcinogen Risk Assessment (U.S. EPA, 2005) a linear extrapolation to low doses was used” to determine whether chrysotile presents an unreasonable hazard under certain conditions of use.<sup>26</sup> Notably, three of the peer reviewers of the December 2020 chrysotile risk evaluation are

testifying experts for asbestos plaintiffs who offer opinions at trial based on the assumption that there is no threshold for safety for asbestos: Drs. Henry Anderson, Steven Markowitz, and Marty Kanarek.<sup>27</sup>

OSHA likewise assumes a LNT model.<sup>28</sup> However, Dr. Irving J. Selikoff, in a February 11, 1972, letter to OSHA wrote that the development of any numerical threshold value limit for asbestos exposures in the workplace “must rely on extrapolations that would hardly be countenanced in many other scientific circumstances.”<sup>29</sup>

“The validity of the LNT risk model has increasingly been questioned because of the recurring observation that an organism’s response to high stressor doses differs from that to low doses.”<sup>30</sup> There is no shortage of scientific articles critiquing the use of this model. In March 2019, the Chemico-Biological Interactions journal released a special edition titled *Assessing the Scientific Basis of the Linear No Threshold (LNT) Model with Threshold Models for Cancer Risk Assessment of Radiation and Chemicals*.<sup>31</sup> The issue contains 10 articles addressing all aspects of the LNT, including its history, applications, science, debunking, and its use by governmental regulatory agencies.

#### **D. The Rutherford Standard Was Defined in the Context of a High-Dose Case**

California’s case law identifying the minimal threshold for evidence necessary to prove causation in an asbestos case was decided in the context of what we now consider high-dose exposures. The product at issue in *Rutherford* was Kaylo, an asbestos-containing insulation product manufactured by Owens-Illinois from 1948 to 1958.<sup>32</sup> Kaylo contained both amosite (12% per volume) and chrysotile (3% per volume) asbestos fibers.<sup>33</sup> Kaylo work practice studies have measured airborne concentrations of asbestos fibers equal to or greater than 5 microns ranging from 20 f/cc (hand sawing) to 70 f/cc (clean up).<sup>34</sup> Airborne concentrations for these same activities measured in the field have been reported to be much higher: 158 f/cc (hand sawing) and 1191 f/cc (clean up).<sup>35</sup> Several studies<sup>36</sup> have concluded that airborne concentrations of asbestos fibers associated with insulation products like Kaylo increase incidence of cancer. The evidence in *Rutherford* was that work with insulation products like Kaylo created areas of asbestos dust that looked like a “Texas dust storm.”<sup>37</sup>

Nowadays, no person in the litigation would consider asbestos insulation exposures like those described in *Rutherford* as low-dose. Yet, throughout the 1960s and 1970s, Selikoff repeatedly described insulation exposures as limited and intermittent.<sup>38</sup> *Rutherford* likewise describes Kaylo exposures as “relatively small.”<sup>39</sup> Notably, the *Lineaweaver* opinion also related to asbestos insulation products. So did the cases relied on by *Lineaweaver*, including *Lockwood v. A C & S*,<sup>40</sup> a shipyard insulation case, and *Lohrmann v. Pittsburgh Corning Corp.*,<sup>41</sup> an insulating materials case.

At the other end of the asbestos exposure spectrum from Kaylo is the product at issue in *Davis*: automotive brakes, a non-friable, low-dose, chrysotile product. The plaintiff's experts in *Davis* were forced to resort to an “each and every” exposure theory based on an LNT model about asbestos-disease specific causation.

Contrary to *dicta* in *Davis*, *Rutherford* makes no comment on the appropriateness or legality of the “each and every exposure” opinion expressed in *Davis* based on an LNT model.<sup>42</sup> As a starting point, the “each and every” exposure theory was not an issue raised by the parties on appeal in *Rutherford*.<sup>43</sup> The application of that theory in *Rutherford* was unimportant because of the nature of the product at issue in that case (*i.e.*, friable amosite insulation).

The holding in *Rutherford* itself is a disapproval of “each and every exposure” opinions based on the LNT model. Thus, *Davis* got it wrong here again.

In *Rutherford*, the court struck down a jury instruction that basically provided that if plaintiff proved (1) defendant's product was “defective”; (2) plaintiff's injuries were legally caused by asbestos exposure *generally*; and (3) plaintiff was exposed to asbestos fibers from defendant's products, then the burden shifted to defendant to prove that its product was not a legal cause of the plaintiff's injuries or death (*i.e.*, specific causation).<sup>44</sup> In short, *Rutherford* rejected a jury instruction that allowed the plaintiff to merely meet his burden of proof on the issue of specific causation by presenting evidence of any exposure to the defendant's defective product.

Additionally, *Rutherford* embraces the fact that liability should be factually premised on a “sufficiently lengthy, intense and frequent exposure as to render

the defendant's product a substantial factor contributing to the risk of cancer.”<sup>45</sup> It does so in the context of a discussion about a potential jury instruction. The dissent in *Rutherford* even points out that that the majority requires that plaintiffs must establish legal cause through factors including frequency of exposure, regularity of exposure, proximity of the asbestos product to plaintiffs, and other possible sources of plaintiffs' injury.<sup>46</sup>

### E. Sargon Requires Exclusion of Opinions Based on the LNT Model

The goal of trial court gatekeeping is simply to exclude ‘clearly invalid and unreliable’ expert opinion.”<sup>47</sup> To be admissible, an expert's opinion must:

1. Rise to the level of a reasonable medical probability (see section B, *supra*);
2. Not be based on irrelevant or speculative matters;<sup>48</sup>
3. Not be based on assumptions of fact without evidentiary support;<sup>49</sup> and
4. Not be based on reasons or matter precluded “by law.”<sup>50</sup>

First, an expert opinion that a low-dose product contributes to one's risk of disease based on the LNT and its derivative theories does not satisfy the reasonable medical probability standard of proof. As noted above, the LNT model by definition provides only an upper-bound calculation of potential risk at low doses. As noted earlier, California courts (with the exception of *Davis*) draw a line between a potential/possible risk and a probable risk. Experts should be challenged on whether the LNT model more likely than not is an accurate dose curve for asbestos based on available science.

Second, the LNT model constitutes irrelevant and speculative matters because it is merely a default model used to calculate potential risks. Experts should be challenged on whether asbestos exhibits a linear dose response relationship rather than S-shaped. As noted above, EPA as recently as December 2020 was unable to conclude that it does.

Third, the LNT model and its derivatives are based on assumptions of fact lacking evidentiary support. Specifically, the LNT model both assumes without

scientific support that cancer risk is linearly proportionate to dose and dosage outweighs any biological variables. Experts should be challenged on whether these assumptions are more probable than not. However, most experts will agree that “background” doses of asbestos do not cause disease (or at least there is no evidence it causes disease, like opined in *Davis*), and most substances exhibit S-shaped dose-response curves at low doses.

Fourth, the California Supreme Court has not yet determined whether the LNT-derivative “every exposure” theory of liability is consistent with the substantial factor test that applies in asbestos cases. The Ninth Circuit in *McIndoe v. Huntington Ingalls Inc.*<sup>51</sup> explained that allowing such a theory would permit imposition of liability on the manufacturer of any asbestos-containing product with which a worker had the briefest of encounters on a single occasion. “This is precisely the sort of unbounded liability that the substantial factor test was developed to limit.” More than thirty other federal courts and state courts have held that this cumulative “any exposure” theory is not reliable.<sup>52</sup>

At least some California federal courts believe that the California Supreme Court would likely agree that the LNT-derivative “each and every exposure” theory is inconsistent with the substantial factor test.<sup>53</sup> If that were to happen, experts would not be allowed to rely on the theory at trial for their opinions. In the meantime, litigants should continue to remind courts of their substantial gatekeeping duty, especially in light of *Rutherford’s* express reasonable medical probability standard of proof bar on issues submitted to the jury when it comes to the issue of causation.

## Endnotes

1. 55 Cal.4th 747, 753 (2012).
2. See *Davis v. Honeywell Internat. Inc.*, 245 Cal. App.4th 477 (2016); see also *Phillips v. Honeywell Int’l Inc.*, 9 Cal.App.5th 1061 (2017); *Izell v. Union Carbide Corp.*, 231 Cal.App.4th 962 (2014); see generally Mark A. Behrens & Andrew J. Trask, *The Rule of Science and the Rule of Law*, 49 Sw. U. L. Rev. 436 (2021).
3. 245 Cal. App. 4th 477 (2016).
4. Letter from Irving J. Selikoff to Office of Safety and Health Standards, Feb. 11, 1972, enclosing comment titled “Epidemiological Constraints in Development of a Standard for Asbestos Exposure.”
5. The linear no-threshold problem in low-dose cases is not new and has been addressed thoroughly by others. See William L. Anderson & Kieran Tuckley, *How Much is Enough? A Judicial Roadmap to Low Dose Causation Testimony in Asbestos and Tort Litigation*, 42 Am. J. Trial Advoc. 39 (2018); William L. Anderson et al., *The “Any Exposure” Theory Round II – Court Review of Minimal Exposure Expert Testimony in Asbestos and Toxic Tort Litigation Since 2008*, 22 Kan. J.L. & Pub. Pol’y 1 (2012); Mark Behrens & William L. Anderson, *The “Any Exposure” Theory: An Unsound Basis for Asbestos Causation and Expert Testimony*, 37 Sw. U. L. Rev. 479 (2008); Jason Litt et al., *Returning to Rutherford: A Call to California Courts to Rejoin the Legal Mainstream and Require Causation Be Proved in Asbestos Cases Under Traditional Torts Principles*, 45 Sw. L. Rev. 989 (2016); Steven D. Wasserman et al., *Asbestos Litigation in California: Can it Change for the Better?*, 34 Pepp. L. Rev. 883 (2007).
6. See *People v. Leahy*, 8 Cal.4th 587, 591 (1994).
7. Notably, at least one federal court has said that *Davis* embraced unreliable science. See *Barabin v. Scapa Dryer Fabrics, Inc.*, No. C07-1454JLR, 2018 U.S. Dist. LEXIS 22725, at \*35 (W.D. Wash. Feb. 12, 2018).
8. FEDERAL JUDICIAL CENTER REFERENCE MANUAL ON SCIENTIFIC EVIDENCE (3d ed. 2011), at 636 (“the dose makes the poison”; this implies that all chemical agents are intrinsically hazardous — whether they cause harm is only a question of dose.”)
9. 16 Cal.4th 953, 982-983 (1997).
10. See Restatement (Third) of Torts: Liab. for Physical & Emotional Harm § 36 reporter’s note, cmt. b (Am. L. Inst. 2010) (referencing *Rutherford*, 16 Cal.4th at 978) (substantial-factor standard eliminates “negligible or theoretical” contributions); see also *McIndoe v. Huntington Ingalls Inc.*, 817 F.3d 1170, 1172 (9th Cir. 2016) (citing to Restatement (Third) of Torts: Liab. for Physical & Emotional Harm § 36).



11. Jud. Council of Cal. Civ. Jury Instruction (“CACI”) No. 435 (emphasis added).
12. 31 Cal.App.4th 1409, 1416 n.2 (1995).
13. *Rutherford*, 16 Cal.4th at 976 n.11.
14. *Lineaweaver*, 31 Cal.App.4th at 1416, quoting *Bromme v. Pavitt*, 5 Cal.App.4th 1487, 1498 (1992), quoting *Jones v. Ortho Pharm. Corp.*, 163 Cal.App.3d 396, 402-403 (1985).
15. 163 Cal.App.3d 396, 403-404 (1985), quoting *Parker v. Employers Mut. Liab. Ins. Co.*, 440 S.W.2d 43, 49 (Tex. 1969).
16. *Davis*, 245 Cal.App.4th at 487 and 494, finding it is “not illogical to conclude” that each exposure when added to other exposures, can result in a cumulative exposure sufficient to cause mesothelioma.
17. 3 Cal.App.4th 1367, 1384 (1992).
18. *Lineaweaver*, 31 Cal.App.4th at 1416-1417.
19. *Rutherford*, 16 Cal.4th at 975; *see also id.* at 988 (“Majority’s requirement that plaintiffs must bear the formidable burden of establishing legal cause through factors including frequency of exposure, regularity of exposure, proximity of the asbestos product to plaintiffs, and other possible sources of plaintiffs’ injury.”)
20. *Davis*, 245 Cal.App.4th at 495-497.
21. U.S. EPA Guidelines for Carcinogen Risk Assessment, Mar. 2005, at A-9.
22. Rebecca A. Clewell et al. *Dose-Dependence of Chemical Carcinogenicity: Biological Mechanisms for Thresholds and Implications for Risk Assessment*, 301 CHEMICO-BIOLOGICAL INTERACTIONS 112 (Mar. 1, 2019), <https://www.sciencedirect.com/science/article/pii/S0009279718314467>.
23. *Sargon*, 55 Cal.4th 747, 770.
24. U.S. EPA Guidelines for Carcinogen Risk Assessment, *supra* note 21, at 1-15.
25. Risk Evaluation for Asbestos Part I: Chrysotile Asbestos, Dec. 2020, at 152.
26. *Id.* at 160. Note: “EPA generally takes public health protective, default positions regarding the interpretation of toxicologic and epidemiologic data: ... cancer risks are assumed to conform with low dose linearity.” U.S. EPA Guidelines for Carcinogen Risk Assessment, *supra* note 21, at 1-10-11, [https://www3.epa.gov/airtoxics/cancer\\_guidelines\\_final\\_3-25-05.pdf](https://www3.epa.gov/airtoxics/cancer_guidelines_final_3-25-05.pdf).
27. For an explanation of the lead up to the finalization of the 2020 Final Draft Evaluation for Asbestos Part I: Chrysotile Asbestos, see Claire Weglarz et al., *The EPA’s March to Ban Asbestos: 2020 Draft Risk Evaluation*, 87 Def. Couns. J. (Oct. 2020), <https://www.iadclaw.org/defensecounseljournal/the-epas-march-to-ban-asbestos-2020-draft-risk-evaluation/>.
28. Fed. Reg., vol. 59, no. 153: 41037 (Aug. 10, 1994); Fed. Reg., vol. 48, no. 215:51122, 51123 (Nov. 4, 1983).
29. Letter from Irving J. Selikoff, *supra* note 4.
30. David Costantini & Bennie Borremans, *The Linear No-Threshold Model is Less Realistic Than Threshold or Hormesis-Based Models: An Evolutionary Perspective*, 301 CHEMICO-BIOLOGICAL INTERACTIONS 26 (Mar. 1, 2019), <https://www.sciencedirect.com/science/article/pii/S0009279718310949>.
31. Symposium, 301 CHEMICO-BIOLOGICAL INTERACTIONS 1-146 (Mar. 1, 2019), <https://www.sciencedirect.com/journal/chemico-biological-interactions/vol/301/suppl/C>.
32. *Rutherford*, 16 Cal.4th at 961.
33. William E. Longo et al., *MAS Work Practice Study: Kaylo III* (Oct. 2006).
34. *Id.* (only asbestos fibers measuring equal to or greater than 5 microns were counted; air samples collected during 30 minute time periods.)
35. P.G. Harries, *Asbestos Dust Concentrations in Ship Repairing: A Practical Approach to Improving Asbestos Hygiene in Naval Dockyards*, 14 Ann. OCCUP. HYGIENE 241 (1971), <https://academic.oup.com/annweh/article-abstract/14/3/241/1448866?redirectedFrom=fulltext>.

36. Irving J. Selikoff et al., *Mortality Experience of Insulation Workers in the US and Canada, 1943-1976*, 1 ANN. J. INDUS. MED. 8 (1979), <https://pubmed.ncbi.nlm.nih.gov/294225/>; Julian Peto et al.; *Mesothelioma Incidence in Asbestos Workers: Implication for Models of Carcinogenesis and Risk Assessment*, 45 Br. J. Cancer 124 (1982), <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2010947/pdf/brjcancer00436-0137.pdf>.
37. *Rutherford*, 16 Cal. 4th at 961.
38. E.g., Selikoff et al., *supra* note 4 Irving J. Selikoff et al., *Relation Between Exposure to Asbestos and Mesothelioma*, 272 NEW ENG. J. OF MED. 560 (1965), <https://pubmed.ncbi.nlm.nih.gov/14248731/>.
39. *Rutherford*, 16 Cal. 4th at 985.
40. 109 Wn.2d 235, 238 (1987).
41. 782 F.2d 1156, 1158 (4th Cir. 1986).
42. See *Davis*, 245 Cal.App.4th at 492-493.
43. *Id.* at 480; *Rutherford*, 16 Cal.4th at 962-963, n.6.
44. *Rutherford*, 16 Cal.4th at 960-961.
45. *Id.* at 979.
46. *Id.* at 988 (Mosk, J., dissenting).
47. *Sargon*, 55 Cal.4th at 772 (citation omitted).
48. *Id.* at 770; see also Cal. Evid. Code § 801.
49. *Sargon*, 55 Cal.4th at 770.
50. *Id.* at 771; see also, Cal. Evid. Code § 802.
51. *McIndoe*, 817 F.3d at 1177.
52. See *Krik v. Exxon Mobil Corp.*, 870 F.3d 669, 677-678 (7th Cir. 2017).
53. *In re Toy Asbestos*, No. 19-cv-00325-HSG, 2021 U.S. Dist. LEXIS 64440, at \*16, n.2 (N.D. Cal. Mar. 30, 2021). ■



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