

Navigating Uncertainty: Gatekeeping in the Absence of Hard Science

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## NAVIGATING UNCERTAINTY: GATEKEEPING IN THE ABSENCE OF HARD SCIENCE

*Once upon a time a young rabbinical student went to hear three lectures by a famous rabbi. Afterwards he told his friends: "The first talk was brilliant, clear and simple. I understood every] word. The second was even better, deep and subtle. I didn't understand much, but the rabbi understood all of it. The third was by far the finest, a great and unforgettable experience. I understood nothing and the rabbi didn't understand much either."<sup>1</sup>*

Groping, as always, for both truth and the proper words to convey it,<sup>2</sup> Niels Bohr used this fictional story to introduce his more philosophical speculations.<sup>3</sup> The formal presentation of such thinking invited tension among Bohr's scientific colleagues: there was cause for concern that Bohr might be exceeding the grasp of his expertise and diverting attention from the real affairs of his godchild — quantum mechanics. Bohr's use of humor helped to defuse this tension, giving his colleagues an opportunity to choose merely to play along — to laugh while listening, and to take the great man's words with a dash of salt.

The modern federal trial judge lacks the luxury enjoyed by Bohr's colleagues. In determining whether to admit an expert witness's testimony, a federal judge cannot choose simply to "play along." Pursuant to the Federal Rules of Evidence, the judge must make a sober determination whether the proffered testimony is "relevant and reliable" — that is, whether it indeed brings before the court "specialized knowledge" that will facilitate accurate fact-finding.<sup>4</sup> Consequently,

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<sup>1</sup> ABRAHAM PAIS, *NIELS BOHR'S TIMES, IN PHYSICS, PHILOSOPHY, AND POLITY* 439 (1991).

<sup>2</sup> *See id.* at 435 (Albert Einstein's description of Bohr).

<sup>3</sup> *See id.* at 439.

<sup>4</sup> *See* *Daubert v. Merrell Dow Pharms., Inc.*, 509 U.S. 579, 589–95 (1993). The federal standard for admitting expert testimony derives primarily from Rule 702 of the Federal Rules of Evidence, which states: "If scientific, technical, or other specialized knowledge will assist the trier of fact to understand the evidence or to determine a fact in issue, a witness qualified as an expert by knowledge, skill, experience, training, or education, may testify thereto in the form of an opinion or otherwise." FED. R. EVID. 702; *see also* *Daubert*, 509 U.S. at 589–92 (identifying the "primary locus of [the gatekeeping] obligation" as Rule 702).

A troika of recent Supreme Court decisions has attempted to provide an intelligible framework for rulings on the admissibility of expert testimony. In the first decision, *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, the Court construed Rule 702 to mean that expert scientific testimony is admissible only if it is both relevant and reliable, *see Daubert*, 509 U.S. at 589, and listed factors that lower courts might consider in determining the reliability of expert testimony, *see id.* at 593–95; *see also infra* note 26. Four years later, the Court revisited the issue of admissibility in *General Electric Co. v. Joiner*, 118 S. Ct. 512 (1997), in which the Court made clear that a trial judge's decision to admit or exclude scientific testimony can be reversed only for abuse of discretion. *See id.* at 519. Finally, in the third decision, *Kumho Tire Co. v. Carmichael*, 119 S. Ct. 1167 (1999), the Court established that *Daubert's* requirement of relevance and reliability applies whether ex-

judicial hackles are rightly raised in situations of *strong scientific uncertainty*<sup>5</sup> — a phrase used in this Note to denote situations in which a qualified expert proposes to testify on an issue that hard science<sup>6</sup> can resolve, but upon which substantial scientific study has yet to be done and (given cost or time constraints) could not reasonably have been done in preparation for trial. In such situations, a judge might rightly wonder whether the expert's opinion is grounded more in speculation than in "specialized knowledge."<sup>7</sup>

Although the Supreme Court has required that lower federal judges try to make such a distinction, it has not provided detailed guidance on how the distinction should be made.<sup>8</sup> With only limited instruction from above, lower courts have differed substantially regarding the admissibility standards that they have enforced.<sup>9</sup> The result is a juris-

pert testimony is "scientific" or "non-scientific." See *id.* at 1171. The Court also asserted that *Joiner's* abuse of discretion standard applies not only to a trial court's determination of relevance and reliability, but also to a trial court's decision of how to make that determination. See *id.*

<sup>5</sup> The phrase "strong uncertainty" comes from Heidi Li Feldman's article, *Science and Uncertainty in Mass Exposure Litigation*, 74 TEX. L. REV. 1, 2 (1995). However, this Note uses "strong uncertainty" in a different, more functionally oriented sense. Whereas Professor Feldman describes "strong uncertainty" as a state in which a factfinder is "unable to conclude" that a substance is safe or unsafe, *id.*, this Note characterizes "strong uncertainty" as a state in which hard science does not provide an answer, although the question at hand is scientific. The difference in definition is significant. Professor Feldman's definition presents situations of strong uncertainty as ones of adjudicatory breakdown, and therefore her definition naturally leads to a conclusion that the current tort system needs to undergo fundamental reform. See *id.* at 43–48. By not incorporating a presumption about the capacities of the factfinder, this Note's definition leaves the issue of substantive legal reform in reserve and investigates how the current system can deal with scientific uncertainty most effectively.

<sup>6</sup> This Note uses "hard science" to describe scientific methodologies characterized by careful quantification and rigorous testability. In the context of medical causation, the reference is primarily to population studies and laboratory experimentation, the methodologies associated with epidemiology and toxicology, respectively.

<sup>7</sup> The judge might even wonder how to tell the difference. In *Daubert* on remand, Judge Kozinski described federal judges as having the "heady task" of "resolv[ing] disputes among respected, well-credentialed scientists about matters squarely within [the scientists'] expertise, in areas where there is no scientific consensus as to what is and what is not 'good science.'" *Daubert v. Merrell Dow Pharms., Inc.*, 43 F.3d 1311, 1316 (9th Cir. 1995); see also *Daubert*, 509 U.S. at 600 (Rehnquist, C.J., concurring in part and dissenting in part) ("I am at a loss to know what is meant when it is said that the scientific status of a theory depends on its 'falsifiability,' and I suspect some of [my fellow federal judges] will be, too.").

<sup>8</sup> Both before and after the Supreme Court's recent decision in *Kumho*, commentators have remarked on the need for "guideposts" to direct judges in deciding questions of admissibility. See, e.g., Daniel J. Capra, *The Daubert Puzzle*, 32 GA. L. REV. 699, 703, 714 (1998) (observing that the district judge in *Joiner* would not have abused his discretion either by admitting or by excluding the expert testimony at issue); K. Isaac DeVyver, *Opening the Door but Keeping the Lights Off: Kumho Tire Co. v. Carmichael and the Applicability of the Daubert Test to Nonscientific Evidence*, 50 CASE W. RES. L. REV. 177, 200–02 (1999) (asserting that *Kumho* "left judges without a consistent or dependable standard for determining the reliability of nonscientific expert testimony").

<sup>9</sup> See 1 DAVID L. FAIGMAN, DAVID H. KAYE, MICHAEL J. SAKS & JOSEPH SANDERS, MODERN SCIENTIFIC EVIDENCE: THE LAW AND SCIENCE OF EXPERT TESTIMONY § 1-3.4

prudence that has at times been unpredictable and inconsistent, and, from some perspectives, borderline chaotic.<sup>10</sup>

This Note seeks to show how the lower courts might escape the threatened chaos. In particular, it provides guidance for how judges can handle a particularly prominent class of “strong uncertainty” scenarios — those in which a clinical physician offers testimony on medical causation but cannot, or does not, refer to rigorous scientific evidence that a specific set of circumstances could have caused the plaintiff’s injury.

The paradigmatic situation involves a plaintiff who has suffered injury shortly after exposure to a toxic substance. The plaintiff alleges that the exposure caused the injury, even though the precise level of exposure is unknown and the physiological consequences of exposure are largely untested.<sup>11</sup> In an effort to show that the exposure “more likely than not” caused the injury,<sup>12</sup> the plaintiff proffers the expert testimony of a clinical physician. This physician plans to testify that the toxic substance could have caused — perhaps even that it did cause — the plaintiff’s injury.<sup>13</sup> In legal terms, the physician’s testimony seeks to establish one or both of two propositions: general causation (“whether a substance is capable of causing a particular injury or condition in the general population”) and specific causation (“whether a substance caused a particular individual’s injury”).<sup>14</sup> The physician’s conclusion derives from application of accepted tools of the medical

(1997 & Supp. 1999) (describing the struggle to develop appropriate admissibility standards); 2 MICHAEL H. GRAHAM, HANDBOOK OF FEDERAL EVIDENCE § 702.5 (4th ed. 1996 & Supp. 1998) (noting variations in lower courts’ approaches); DeVyver, *supra* note 8, at 185 (noting significant pre-*Kumho* divisions regarding the admissibility standards for nonscientific evidence).

<sup>10</sup> See Marilee M. Kapsa & Carl B. Meyer, *Scientific Experts: Making Their Testimony More Reliable*, 35 CAL. W. L. REV. 313, 331 (1999) (complaining that the “lack of an objective standard” makes admissibility determinations unpredictable); James E. Starrs, *There’s Something About Novel Scientific Evidence*, 28 SW. U. L. REV. 417, 437–38 (1999) (stating that, by extending *Daubert’s* principles to all expert testimony, *Kumho* created “the potential for considerable chaos in federal trial courts”).

<sup>11</sup> Such a situation of “strong uncertainty” is not unusual because “[s]cientific research into causation is a slow, arduous process, often barely begun when litigation commences.” Feldman, *supra* note 5, at 17.

<sup>12</sup> The plaintiff is assumed to bear the traditional burden of proving his or her case by the preponderance of the evidence. See W. PAGE KEETON, DAN B. DOBBS, ROBERT E. KEETON & DAVID G. OWEN, PROSSER AND KEETON ON THE LAW OF TORTS § 41, at 269 (5th ed. 1984).

<sup>13</sup> Similar issues arise in situations in which the defendant proffers a clinical physician to testify *against* the plaintiff’s theory of causation. See, e.g., *Baker v. Dalkon Shield Claimants Trust*, 156 F.3d 248, 253 (1st Cir. 1998) (analyzing a defense expert’s theory of alternative causation). Because the standard for admissibility does not depend on the party for which an expert testifies, the admissibility analysis in such situations parallels that discussed in this Note.

<sup>14</sup> *Merrell Dow Pharms., Inc. v. Havner*, 953 S.W.2d 706, 714–16 (Tex. 1997); see also 2 FAIGMAN, KAYE, SAKS & SANDERS, *supra* note 9, §§ 31-1.2.1 to -1.2.2 (1997).

trade.<sup>15</sup> Nonetheless, this conclusion lacks a substantial basis in hard science: the expert has, for example, failed to cite any toxicological or epidemiological studies that show that the specific substance in question could have caused the plaintiff's injury. After considering both what the expert's proposed testimony offers and what it lacks, the trial judge must decide how much of the testimony to admit.<sup>16</sup>

In deciding this question of admissibility, the trial judge must resort to some combination of decision rules<sup>17</sup> and intuition. This Note shows how the trial judge's approach to the problem might be organized. In particular, this Note investigates how a trial judge might weigh the results of differential diagnosis,<sup>18</sup> as well as the probative value of the temporal proximity between alleged cause and effect.<sup>19</sup> Part I presents two paradigmatic sets of decision rules for analyzing such data — the "generic toxic tort" rules that the Supreme Court articulated in 1993,<sup>20</sup> and the "slip and fall" rules of "reasonable medical certainty" that courts have continued to apply in more traditional cases of personal injury.<sup>21</sup> Part I suggests that neither set of rules provides a general resolution to the problem of "strong uncertainty." Instead, a judge must choose which set of rules to apply based on the context of the particular case.

The devil being in the details, Part II investigates how context determines the evidentiary significance of differential diagnosis and temporal proximity. Sections II.A and II.B show that the reliability of differential diagnosis substantially depends on base probabilities of

<sup>15</sup> These tools include personal examination of the plaintiff, analysis of the plaintiff's medical history, reference to professional training and experience, review of existing knowledge concerning the toxic substance, and testing to eliminate alternative potential causes of the plaintiff's symptoms. See, e.g., *McCulloch v. H.B. Fuller Co.*, 61 F.3d 1038, 1044 (2d Cir. 1995) (cataloging the standard medical techniques that the plaintiff's expert used to find that glue fumes caused the plaintiff's throat polyps), quoted in *Zuchowicz v. United States*, 140 F.3d 381, 387 (2d Cir. 1998).

<sup>16</sup> A judge who excluded the physician's testimony on causation could allow the physician to testify regarding other issues, such as the plaintiff's physical condition and medical history. See, e.g., *Moore v. Ashland Chem. Inc.*, 151 F.3d 269, 273 (5th Cir. 1998) (en banc) (observing that, although the trial court did not allow a physician to testify on causation, it did allow him to present his conclusions concerning the nature of the plaintiff's illness), cert. denied, 119 S. Ct. 1454 (1999).

<sup>17</sup> See Vern R. Walker, *Risk Regulation and the "Faces" of Uncertainty*, 9 RISK: HEALTH, SAFETY & ENV'T 27, 37-38 (1998).

<sup>18</sup> Differential diagnosis, often called differential etiology, is a process in which a physician rules out possible causes of a patient's symptoms by examining the patient, as well as the patient's personal and family medical history. See FEDERAL JUDICIAL CTR., REFERENCE MANUAL ON SCIENTIFIC EVIDENCE 214 (1994); see also Lars Noah, *Pigeonholing Illness: Medical Diagnosis as a Legal Construct*, 50 HASTINGS L.J. 241, 245-47 (1999) (describing differential diagnosis and its limitations).

<sup>19</sup> A plaintiff's expert may argue that the fact that the plaintiff's symptoms appeared soon after the plaintiff's exposure provides evidence that the exposure caused the injury. See, e.g., *Heller v. Shaw Indus., Inc.*, 167 F.3d 146, 151 (3d Cir. 1999); *Moore*, 151 F.3d at 273.

<sup>20</sup> See *infra* note 26.

<sup>21</sup> See *infra* p. 1473.

causation<sup>22</sup> that are often ill-defined, but that have some relation to whether the case better fits the paradigm of the generic toxic tort or the “slip and fall.” Indeed, by analyzing two recent cases, section II.C shows that the proposed approach to differential diagnosis and temporal proximity provides a sensible formula for “gatekeeping” — one that, as the Supreme Court desires, allows broad possibilities for admissibility while keeping much specific testimony away from the finder of fact.<sup>23</sup>

## I. THE TOXIC TORT AND SLIP-AND-FALL PARADIGMS

As observed above, the crucial thing about the gatekeeper’s role is that he or she must make a decision.<sup>24</sup> There are a variety of objective rules that a court could use to structure its analysis. One prominent set of rules, which this Note calls *toxic tort rules*, primarily requires that expert testimony satisfy a multifactor test suggested by the Supreme Court in *Daubert v. Merrell Dow Pharmaceuticals, Inc.*<sup>25</sup> The *Daubert* test demands that expert testimony derive from a methodology that meets criteria of testability, peer review and publication, precision and control, and general acceptance.<sup>26</sup>

As the Supreme Court’s recent decision in *Kumho Tire Co. v. Carmichael*<sup>27</sup> indicated, the propriety of applying these *Daubert* criteria is “situational”: the rules may be appropriate for some situations but less

<sup>22</sup> These probabilities are established or intuited through other evidence before the results of differential diagnosis are considered.

<sup>23</sup> See *Kumho Tire Co. v. Carmichael*, 119 S. Ct. 1167, 1174–76 (1999) (affirming the judge’s absolute duty to act as a “gatekeeper,” with the qualification that the admissibility inquiry is “a flexible one” (quoting *Daubert v. Merrell Dow Pharms., Inc.*, 509 U.S. 579, 594 (1993))).

<sup>24</sup> See *id.* at 1179 (Scalia, J., concurring) (affirming that the trial court lacks “discretion to abandon the gatekeeping function”).

<sup>25</sup> 509 U.S. 579 (1993).

<sup>26</sup> See *id.* at 593–95. The four *Daubert* factors are:

- (1) whether the methodology upon which the testimony is based has been, or can be, tested;
- (2) whether the methodology “has been subjected to peer review and publication”;
- (3) the methodology’s “known or potential rate of error” and the availability and use of standards to control the methodology’s operation; and
- (4) the extent to which the methodology is generally accepted in the relevant scientific community.

*Id.*; see also *Kumho*, 119 S. Ct. at 1175. The *Kumho* Court made clear that these factors are non-exclusive, as well as nondispositive. See *Kumho*, 119 S. Ct. at 1175. Supplemental considerations may include whether an expert arrived at his or her conclusions independently or only in preparation for litigation, see *Daubert v. Merrell Dow Pharms., Inc.*, 43 F.3d 1311, 1317 (9th Cir. 1995), and whether the question at issue is one that the expert is expected to answer reliably in his or her normal professional life, cf. FED. R. EVID. 703 & advisory committee’s note (speaking of the information upon which a physician bases his or her diagnosis as being “of a type reasonably relied upon by experts” in the physician’s field).

<sup>27</sup> 119 S. Ct. 1167 (1999).

than ideal for others.<sup>28</sup> Strict and universal application of the *Daubert* criteria would lead to a Draconian result: the exclusion of causation testimony in all cases of strong scientific uncertainty — cases in which an expert's opinion by definition lacks the imprimatur of "hard science," and perforce has not passed peer review.<sup>29</sup> Such a rule of exclusion might be proper for the subset of cases that fit the model of the generic toxic tort — a case in which a complicated causal chain, a long latency period, or low levels of exposure render the argument for causation inherently weak.<sup>30</sup> *Daubert* itself was such a case: the plaintiff's central allegation was that an anti-nausea drug caused birth defects.<sup>31</sup> In a case of this type, reliable testimony on causation can only derive from extremely accurate data and methods.<sup>32</sup> Therefore, there is often good reason to exclude testimony that lacks the sanction of peer review and does not possess a small and controllable rate of error.<sup>33</sup>

Such a rule of systematic exclusion might, however, be improper in cases in which the inherent probabilities of causation are far more substantial. The paradigmatic example of such a case is the "slip and fall"

<sup>28</sup> See *id.* at 1175 ("[W]e can neither rule out, nor rule in, for all cases and for all time the applicability of the factors mentioned in *Daubert*, nor can we now do so for subsets of cases categorized by category of expert or by kind of evidence.")

<sup>29</sup> See Bert Black, *A Unified Theory of Scientific Evidence*, 56 *FORDHAM L. REV.* 595, 678 (1988) ("Normally, no [scientific] answer would mean no proof, and plaintiffs would lose."). In a recent article, Michael Kent Jr. uses the distinction between general and specific causation, see *supra* p. 1469, to argue for just such a Draconian rule. See Michael B. Kent Jr., *Daubert, Doctors and Differential Diagnosis: Treating Medical Causation Testimony as Evidence*, 66 *DEF. COUNS. J.* 525, 536 (1999). Although Kent would admit testimony based on differential diagnosis for the purpose of showing specific causation, he would not admit it for proving general causation — an issue for hard science that in his view demands testimony based on epidemiological or toxicological techniques. See *id.*

<sup>30</sup> See Recent Case, 112 *HARV. L. REV.* 719, 722 & n.36 (1999) (citing PETER H. SCHUCK, *AGENT ORANGE ON TRIAL: MASS TOXIC DISASTERS IN THE COURTS* 3–12 (1987)); see also Susan R. Poulter, *Science and Toxic Torts: Is There a Rational Solution to the Problem of Causation?*, 7 *HIGH TECH. L.J.* 189, 199–200, 216 (1992) (discussing the characteristics of a typical toxic tort).

<sup>31</sup> See *Daubert*, 509 U.S. at 582. In *General Electric Co. v. Joiner*, 118 S. Ct. 512 (1997), the plaintiff likewise alleged causation through complex metabolic processes. See *id.* at 515–16 (reciting the claim that chemicals caused the development of lung cancer years after exposure).

<sup>32</sup> A basic insight of Bayesian statistics is that the accuracy of a given scientific "test" depends on the background probability of its result. See KENNETH R. FOSTER & PETER W. HUBER, *JUDGING SCIENCE: SCIENTIFIC KNOWLEDGE AND THE FEDERAL COURTS* 113–21 (1997). Extremely precise tests are needed to "prove" the presence of highly unlikely results, whereas crude tests can accurately predict substantially probable results. See *id.* at 117–19.

<sup>33</sup> These reasons for exclusion are drawn from the second and third factors in *Daubert*'s list, summarized above in note 26. They may not provide strict grounds for exclusion even in the context of the generic toxic tort. Lack of peer review could merely reflect a normal time lag between research and publication, or lack of prior editorial interest in the subject matter. In *Kumho*, the Court itself noted, "It might not be surprising in a particular case, for example, that a claim made by a scientific witness has never been the subject of peer review, for the particular application at issue may never previously have interested any scientist." *Kumho*, 119 S. Ct. at 1175.

— a phrase used in this Note to denote a case of abrupt physical injury that nearly coincides with a discrete and dramatic external event.<sup>34</sup> As the name suggests, the classic slip-and-fall scenario is one in which a plaintiff alleges that his or her injury resulted from a fall on the defendant's overly dangerous premises.<sup>35</sup> In such cases, courts have customarily (and often justifiably) felt comfortable admitting causation testimony that lacks true scientific rigor but that is supported by a combination of "reasonable medical certainty," physician expertise, and adherence to standard diagnostic techniques.<sup>36</sup> The courts' relaxed *slip-and-fall* rules seem most appropriate where issues of general causation are subject to broad societal consensus and where issues of specific causation are not remote from common intuition and experience.

Unfortunately, the existence of two paradigms for decision-making — toxic tort rules and slip-and-fall rules — only complicates matters for a trial judge confronted with strong scientific uncertainty. In such a context, the issue of general causation, although properly an object for study by hard science, has not yet been substantially investigated by epidemiology, toxicology, or even peer-reviewed case studies.<sup>37</sup> Nonetheless, because *Kumho* rules out mechanical application of toxic tort rules,<sup>38</sup> the trial judge cannot automatically exclude expert testimony on causation. Under certain circumstances, toxic tort rules, and the resulting exclusion of testimony, will be appropriate. Under others, slip-and-fall rules will be best.<sup>39</sup> There is no single definite formula for determining admissibility. However, in all cases, there are more and

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<sup>34</sup> See Recent Case, *supra* note 30, at 723 & n.44 (citing Ellen Relkin, *Some Implications of Daubert and Its Potential for Misuse: Misapplication to Environmental Tort Cases and Abuse of Rule 706(a) Court-Appointed Experts*, 15 CARDOZO L. REV. 2255, 2256 (1994)).

<sup>35</sup> See, e.g., Huff v. Wal-Mart Stores, Inc., No. 99-35024, 1999 WL 1206845, at \*2 (9th Cir. Dec. 15, 1999) (admitting a physician's testimony that "causally relat[ed]" the plaintiff's claimed leg pain to his fall).

<sup>36</sup> See Black, *supra* note 29, at 659-69; see also Licciardi v. TIG Ins. Group, 140 F.3d 357, 360-61 (1st Cir. 1998) (assuming a physician's general capacity to testify on the causal relation between an unexpected jolt and the plaintiff's leg and back pain).

<sup>37</sup> Although a champion of "hard science," Bert Black has acknowledged that careful case studies can provide a reliable basis for expert testimony on general causation. See Black, *supra* note 29, at 688-89 (stating that two peer-reviewed case studies provided a sufficient basis for admitting testimony on the relation between nitroglycerin and heart disease).

<sup>38</sup> See *supra* pp. 1471-72.

<sup>39</sup> The Fourth Circuit has declared that epidemiological evidence is not needed to admit expert testimony derived from differential diagnosis, see *Benedi v. McNeil-P.P.C., Inc.*, 66 F.3d 1378, 1384 (4th Cir. 1995), and has endorsed the application of slip-and-fall rules in at least two post-*Kumho* cases, see *Anderson v. Quality Stores, Inc.*, No. 98-2240, 1999 WL 387827, at \*2-\*3 (4th Cir. June 14, 1999) (finding that the district court abused its discretion in excluding causation testimony "based on a reliable differential diagnosis and a strong temporal relationship"); *Westberry v. Gislaved Gummi AB*, 178 F.3d 257, 262-63 (4th Cir. 1999) (upholding the admission of a physician's testimony based on differential diagnosis).

less satisfactory thought processes that a judge may follow. Part II shows what some “more satisfactory” thought processes might be.

## II. FRAMEWORKS FOR EVALUATING DIFFERENTIAL DIAGNOSIS

### A. *The Conditional Utility of Differential Diagnosis*

A common basis for a clinical physician’s opinion on causation is differential diagnosis.<sup>40</sup> This technique is conceptually simple, consisting of two basic steps: identification of the potential causes of a patient’s symptoms, and elimination of a subset of those causes by finding them inconsistent with the patient’s current condition, or personal and family history.<sup>41</sup> Despite this conceptual simplicity, the relevance and reliability of differential diagnosis can be difficult to determine. Courts generally agree that testimony based on differential diagnosis may be admitted even when the technique has not eliminated *all* alternative causes.<sup>42</sup> The question for courts, therefore, is when differential diagnosis has eliminated *enough* alternative causes to produce relevant and reliable conclusions.<sup>43</sup>

One established requirement is that the alleged path of causation be scientifically possible. To establish this possibility, there must be a preliminary showing of a likelihood of general causation — that is, a showing that the toxic exposure at issue *could* have caused the plaintiff’s injury.<sup>44</sup> As this section will demonstrate, how substantial such a

<sup>40</sup> See, e.g., *In re Paoli R.R. Yard PCB Litig.*, 35 F.3d 717, 756 (3d Cir. 1994) (repeating the district court’s finding that differential diagnosis is the “hallmark” of internal medicine).

<sup>41</sup> See *supra* note 18. The presence of multiple risk factors, such as the plaintiff’s exposure to the toxic substance and history of smoking, can lead to a probability of injury substantially greater than the probability of injury if the individual risk factors acted separately. When properly accounted for under the rubric of “proximate causation,” such “risk factor synergies” do not destroy the basic conceptual simplicity of differential diagnosis. See Poulter, *supra* note 30, at 233–34. They can, however, add to uncertainty in estimating probabilities of causation.

<sup>42</sup> See, e.g., *Ambrosini v. Labarraque*, 101 F.3d 129, 140–41 (D.C. Cir. 1996) (finding a doctor’s testimony admissible despite its failure to exclude all alternative causes); *Cavallo v. Star Enter.*, 892 F. Supp. 756, 771 (E.D. Va. 1995) (indicating that minimization of the probability of other causes could suffice to meet the “more likely than not” threshold), *aff’d in part and rev’d in part on other grounds*, 100 F.3d 1150 (4th Cir. 1996).

<sup>43</sup> A further question might be what methodology a physician must follow to be recognized as having performed “differential diagnosis.” This question lay at the heart of the debate in *In re Paoli*, in which the Third Circuit found the required contents of “differential diagnosis” to depend on individual circumstances. See *In re Paoli*, 35 F.3d at 758–59. For the purposes of this Note, such “questions of methodology” form part of the concern about the accuracy of differential diagnosis. Techniques less worthy of the title “differential diagnosis” may be presumed to have higher error rates and lower reliability.

<sup>44</sup> See *Cavallo*, 892 F. Supp. at 771. A panel of the D.C. Circuit has stated that testimony based upon differential diagnosis “ha[s] legitimacy only as a follow-up to admissible evidence” of general causation. *Raynor v. Merrell Pharms. Inc.*, 104 F.3d 1371, 1376 (D.C. Cir. 1997).

showing must be depends on the accuracy and comprehensiveness of the physician's diagnostic techniques. At a minimum, however, a trial court should demand a "biologically plausible" theory of general causation.<sup>45</sup>

Once a plaintiff has shown general causation, the trial court faces the issue of specific causation: whether the alleged cause did in fact produce the plaintiff's injury. In addressing this issue, the court almost inevitably confronts a scenario in which there are three sets of possible causes: the alleged cause (set *A*); the set of alternative causes that can be ruled out by differential diagnosis (set *B*); and the set of alternative causes that cannot be ruled out by differential diagnosis (set *C*). Each of these sets is characterized by an "initial probability of causation" — that is, a causation probability based on the information known prior to differential diagnosis.

For the moment, assume that the trial court knows these initial probabilities. In particular, assume that, in the absence of the information from differential diagnosis, the trial court knows that the probability that the real cause of the plaintiff's injury lies in set *A* has the fractional value *a*; the probability that the real cause lies in set *B* has the fractional value *b*; and the probability that the real cause lies in set *C* has the fractional value *c*.<sup>46</sup> Differential diagnosis shifts these probabilities.<sup>47</sup> A finding of not-*B* — a finding that rules out the potential causes in set *B* — increases the probabilities that the real cause lies in sets *A* and *C*. By increasing the probability that the real cause lies in set *A*, differential diagnosis helps advance the plaintiff's case. However, for a physician to be able to give *relevant and reliable* testimony that *A* caused the plaintiff's injury, the increase in the probability *a* must be sizable enough to give a solid basis for arguing that *A* was *more likely than not* the cause of the plaintiff's injury.<sup>48</sup> Otherwise,

<sup>45</sup> FEDERAL JUDICIAL CTR., *supra* note 18, at 163, 204 (stating that biological plausibility should be an important factor in determining admissibility).

<sup>46</sup> Because one can readily convert between fractional and percentage probabilities, this Note does not consistently distinguish between the two. A percentage probability of *X*% is equivalent to a fractional probability of *X* divided by 100: 50% is equivalent to 0.5.

<sup>47</sup> This shift accords with standard Bayesian analysis, which has recently gained increased favor in academic circles, see David Malakoff, *Bayes Offers a 'New' Way to Make Sense of Numbers*, 286 SCIENCE 1460, 1460 (1999), although its value in legal reasoning remains contested, see Richard Goldberg, *The Role of Scientific Evidence in the Assessment of Causation in Medicinal Product Liability Litigation: A Probabilistic and Economic Analysis*, in LAW AND SCIENCE 55, 61–65 (Helen Reece ed., 1998) (arguing, in the face of acknowledged resistance, that Bayes's Theorem helps in applying statistical evidence to the individual case).

<sup>48</sup> This formulation of the admissibility standard is consistent with Rule 702's requirement that the expert's testimony be capable of "assist[ing] the trier of fact" through the presentation of specialized knowledge. FED. R. EVID. 702. Because the formulation does not require that the expert's testimony be *sufficient* for a favorable verdict, it conforms with *Daubert's* statement that sufficiency is not necessary for admissibility, see *Daubert v. Merrell Dow Pharms., Inc.*, 509 U.S. 579, 596 (1993).

the physician should be restricted to reporting the intermediate findings from his or her diagnosis, without stating a final opinion on causation.<sup>49</sup>

To see how a determination of relevance and reliability might work in practice, consider a simple idealized scenario in which the initial probabilities  $a$ ,  $b$ , and  $c$  are precisely known, and the techniques of differential diagnosis are error-free — that is, 100% reliable. Because of the techniques' 100% reliability, a not- $B$  test result reduces to zero the probability that the real cause lies in set  $B$ . Following a not- $B$  test result, the post-diagnosis causation probabilities are therefore given by the identities  $a_{new} = a/(a + c)$ ;  $b_{new} = 0$ ; and  $c_{new} = c/(a + c)$ .<sup>50</sup> The post-diagnosis probabilities  $a_{new}$  and  $c_{new}$  have the same relative proportions as  $a$  and  $c$ .<sup>51</sup> Because of this conservation of proportions, the relevance and reliability of differential diagnosis depends on the relative magnitudes of the initial causation probabilities  $a$  and  $c$ . If it is initially much more likely that the real cause lies in the set of non-excludable causes than in the set of alleged causes, differential diagnosis does very little to advance the argument for causation.

Consider, for example, Situation 1 of Table 1, in which the initial probabilities are  $a = 5\%$ ,  $b = 50\%$ , and  $c = 45\%$ . Prior to differential diagnosis, it is nine times more probable that the real cause is one of the non-excludable causes  $C$  than that it is the alleged cause  $A$ . If differential diagnosis yields a not- $B$  test result, the post-diagnosis probabilities are  $a_{new} = 10\%$ ,  $b_{new} = 0$ , and  $c_{new} = 90\%$ . It is still nine times more probable that the non-excludable causes produced the harm than that the alleged cause did. Differential diagnosis has only allowed the doctor to say that the alleged path of causation is still extremely unlikely, a statement very far from an assertion that the alleged path of causation is "more likely than not." Under these circumstances, a trial court would be justified in excluding testimony that purported to show causation through differential diagnosis.<sup>52</sup> In this situation, the question that differential diagnosis answers reliably, "Did one of the ex-

<sup>49</sup> A judge might even exclude such restricted testimony under Federal Rule of Evidence 403 if the judge believed the testimony to be substantially more prejudicial than probative. See, e.g., *United States v. Shay*, 57 F.3d 126, 134–35 & n.6 (1st Cir. 1995); *United States v. Dorsey*, 45 F.3d 809, 815 (4th Cir. 1995).

<sup>50</sup> Various books describe how Bayesian analysis updates probabilities. See, e.g., FOSTER & HUBER, *supra* note 32, at 113–21; D.H. KAYE, *SCIENCE IN EVIDENCE* 28–29 (1997).

<sup>51</sup> If, before differential diagnosis,  $A$  was  $x$  times as likely as  $C$  to be the cause of the plaintiff's injury,  $A$  is still  $x$  times as likely as  $C$  to be the cause after differential diagnosis.

<sup>52</sup> In *Whiting v. Boston Edison Co.*, 891 F. Supp. 12 (D. Mass. 1995), a trial court faced circumstances similar to Situation 1 and correctly reasoned that the proffered testimony was inadmissible. See *id.* at 21 & n.41 (stating that there was an initial 90% probability that plaintiff's injury derived from unknown causes).

cludable causes produce the plaintiff's injury?" does not "fit" the pertinent legal question, "Is causation by *A* more probable than not?"<sup>53</sup>

TABLE 1

	Initial Probability Distribution			Post-Diagnosis Probability Distribution		
	<i>a</i>	<i>b</i>	<i>c</i>	<i>a<sub>new</sub></i>	<i>b<sub>new</sub></i>	<i>c<sub>new</sub></i>
SIT. 1	0.05	0.50	0.45	0.10	0	0.90
SIT. 2	0.35	0.50	0.15	0.70	0	0.30

Suppose, however, that the initial distribution of probabilities is more favorable. For example, suppose that the initial probability distribution corresponds to Situation 2 of Table 1, in which  $a = 35\%$ ,  $b = 50\%$ , and  $c = 15\%$ . It is more than twice as likely that the real cause is the alleged cause than that the real cause is a non-excludable cause. If differential diagnosis yields a negative result, the updated probability that *A* is the cause is 70%. Differential diagnosis is not only a relevant and reliable basis for testimony; if unimpeached, it may afford sufficient grounds for a factual finding of causation. Testimony founded upon such a basis should be admitted.<sup>54</sup>

The above hypotheticals show that the intuition of most courts is correct. If the initial probability of causation is substantial and if most other substantially probable causes can be ruled out, differential diagnosis *can* provide a basis for relevant and reliable testimony. The conditionality of the word "can" reflects at least two caveats: uncertainty in the initial probabilities (*probability estimation uncertainty*) and uncertainty in the accuracy of differential diagnosis (*measurement uncertainty*). When faced with a specific case, a trial court should consider both sources of uncertainty before it replaces "can" with "does" or "does not."

By showing the sensitive dependence of relevance and reliability on initial probability distributions, the examples in Table 1 illustrate the significance of probability estimation uncertainty. To understand the significance of the second type of uncertainty, measurement uncertainty, consider a scenario in which there are only two potential causes: *A*, the alleged cause, and *B*, a cause that can be excluded through dif-

<sup>53</sup> In *Daubert*, the Supreme Court noted that testimony that is reliable for one purpose is not necessarily reliable for the relevant legal purpose. See *Daubert*, 509 U.S. at 591.

<sup>54</sup> Thus, this Note has posited that, given the initial probability distribution of our idealized scenarios, testimony that "*A* caused the plaintiff's injury" should not be admitted if differential diagnosis only shifts the causation probability from 5% to 10%, but should be admitted if it increases the probability from 35% to 70%. In principle, however, there is no bright line rule on the magnitude of the probability shift required for admissibility.

ferential diagnosis. If the diagnostic techniques are 100% reliable, a not-*B* test result means that the probability of causation by *A* is 100%. If the diagnostic techniques are less than 100% reliable, the post-diagnosis probability of causation by *A* depends upon both the initial probability distribution and the accuracy of the diagnostic techniques. Suppose, for example, that the not-*B* test has an accuracy rate *r* of 90%: when *A* is the real cause, the test erroneously reports that *B* is the cause 10% of the time; when *B* is the real cause, the test erroneously reports that *B* is not the cause 10% of the time.<sup>55</sup> If the pre-diagnostic probability that *A* is the cause is *a* and the pre-diagnostic probability that *B* is the cause is *b*, a not-*B* test result yields the following post-diagnostic probability that *A* is the real cause:  $a_{new} = ra / (ra + (1-r)b)$ .<sup>56</sup> Assuming the initial probabilities are those of Situation 1 in Table 2, in which *a* = 20% and *b* = 80%, a not-*B* test result produces a 69% post-diagnosis probability that *A* is the real cause.

TABLE 2

	<i>r</i>	Initial Probability Distribution		Post-Diagnosis Probability Distribution	
		<i>a</i>	<i>b</i>	$a_{new}$	$b_{new}$
SIT. 1	0.90	0.20	0.80	0.69	0.31
SIT. 2	0.55	0.20	0.80	0.23	0.77

Contrast the result of differential diagnosis in Situation 2, in which the test for *B* is only 55% reliable. Despite an initial probability distribution identical to that of Situation 1, a not-*B* test result yields only a 23% probability that *A* caused the plaintiff's injury. When an expert's techniques cannot even demonstrate a twenty-five percent probability of causation, a court is presumably justified in barring the expert from testifying that more probably than not, *A* caused the plaintiff's injury.<sup>57</sup>

### B. The Problem of Initial Probability Estimates

Section II.A shows that even inherently accurate diagnostic techniques may not justify testimony on causation if pre-diagnostic prob-

<sup>55</sup> The error rates for "false positives" and "false negatives" need not be equal, but accounting for differential error rates would add calculational complexity without analytical substance. Cf. FOSTER & HUBER, *supra* note 32, at 113-21 (showing how to account for differential error rates).

<sup>56</sup> The formula follows from the fact that a not-*B* test result has a probability  $(1-r)$  of being wrong. See *id.* at 115-17.

<sup>57</sup> *Kumho*, a case in which an expert sought to explain the cause of a tire explosion, might be viewed as a case in which the Court upheld the exclusion of testimony based on an unreliable "diagnostic" technique akin to the 55%-reliable technique of Situation 2. See *Kumho Tire Co. v. Carmichael*, 119 S. Ct. 1167, 1176-77 (1999).

abilities do not favor an expert's conclusions. Unfortunately, in a situation of strong scientific uncertainty, the pre-diagnostic probability distribution often reflects no more than an informed guess.<sup>58</sup> Frequently, the expert and judge must rest their initial probability estimates on such uncertain grounds as temporal proximity between exposure and injury,<sup>59</sup> analogies to the effects of other toxic substances, and past experience with cases involving comparable levels of exposure.<sup>60</sup> The resulting uncertainties in probability estimation will be high and largely unquantifiable.<sup>61</sup>

<sup>58</sup> An additional complication to probability estimation is that unguided perceptions of risk tend systematically to overestimate the dangers of the "novel risks" that are likely to be at issue in situations of strong uncertainty. W. Kip Viscusi, *How Do Judges Think About Risk?*, 1 AM. L. & ECON. REV. 26, 56-57 (1999).

<sup>59</sup> Although temporal proximity often may reflect coincidence more than causation, cf. *Rosen v. Ciba-Geigy Corp.*, 78 F.3d 316, 318 (7th Cir. 1996) (affirming that "lay speculations" inspired by temporal proximity "are a perilous basis for inferring causality"), courts and commentators agree that it can sometimes provide a basis for relevant and reliable testimony, see *Capra, supra* note 8, at 723-25. The canonical case in which temporal proximity might be "compelling" — one in which a plaintiff doused with a toxic substance develops symptoms shortly thereafter, see *Cavallo v. Star Enter.*, 892 F. Supp. 756 (E.D. Va. 1995), *aff'd in part and rev'd in part on other grounds*, 100 F.3d 1150 (4th Cir. 1996) — does occur in the real world. See, e.g., *Goewey v. United States*, No. 95-2257, 1997 WL 35348, at \*1-\*2 (4th Cir. Jan. 30, 1997) (per curiam) (assuming admission of a physician's causation testimony in a case in which a one-year-old child developed a neurological disorder after being found sitting in a puddle of roof sealant).

In trying to determine whether temporal proximity significantly supports an expert's testimony, a trial judge should ask a number of questions. First, as a check on mere coincidence, the judge should demand a plausible biological explanation for the proposed path of causation. See *supra* pp. 1474-75. The expert does not necessarily have to identify "precisely how the damage occurred," but the expert should give a sensible explanation "based on objective, verifiable evidence" and a reasonable methodology. *Kennedy v. Collagen Corp.*, 161 F.3d 1226, 1229-30 (9th Cir. 1998) (emphasis omitted) (quoting *Daubert v. Merrell Dow Pharms., Inc.*, 43 F.3d 1311, 1314 (9th Cir. 1995)) (reversing the trial court's determination that testimony based upon analogical reasoning was inadmissible), *cert. denied*, 119 S. Ct. 1577 (1999).

Likewise, there should be some assurance that the plaintiff's injury did not occur before or after the time in which the alleged cause could have produced it. See FEDERAL JUDICIAL CTR., *supra* note 18, at 207-08. A judge should therefore request an estimate of the "time window" during which the alleged cause would be expected to produce the observed symptoms.

Finally, for purposes of comparison, a judge should seek to determine the relative probability of alternative causes. Cf. *Rosen*, 78 F.3d at 319 (describing the plaintiff's period of exposure as "a blink of an eye" in comparison to "the long, gradual progression of [his] coronary artery disease"). With regard to each likely alternative cause, the judge should inquire about the "time window" in which that potential cause might have produced the plaintiff's symptoms, as well as the likelihood of that potential cause being the cause in fact.

<sup>60</sup> See *Capra, supra* note 8, at 714-25 (characterizing reliance on chemical analogies, anecdotal evidence, and temporal proximity as "red flags" that "cut] against admissibility"); *Poulter, supra* note 30, at 217-26 (similar); see also *Kennedy*, 161 F.3d at 1230 (finding that a physician's "analogical reasoning was based on objective, verifiable evidence and scientific methodology of the kind traditionally used by rheumatologists").

<sup>61</sup> The characteristics of strong uncertainty are somewhat akin to those of the "model uncertainty" for which Clark Carrington proposes several alternative administrative solutions. See Clark D. Carrington, *An Administrative View of Model Uncertainty in Public Health*, 8 RISK: HEALTH, SAFETY & ENV'T 273, 273-78 (1997).

In such a situation, a trial judge might most usefully proceed by asking an expert to provide rough estimates, including reasonable upper and lower bounds, of the expected probabilities of causation.<sup>62</sup> The goal would be to determine whether there are objective reasons to believe that the alleged cause's initial probability is fairly substantial (in the vicinity of twenty percent, for example), and whether other likely causes of injury are known and excludable. Consistent with *Daubert's* insistence on proper reasoning,<sup>63</sup> the trial judge should demand the expert's best rational explanation for probability estimates, including an account of any "analytical gaps" in the expert's reasoning.<sup>64</sup> The judge can use this information to assess, within the framework of the differential diagnosis calculus, whether the expert's testimony is relevant and reliable.

The ultimate admissibility determination will often closely track the judge's impression of where the case falls on the spectrum between the generic toxic tort and the classic "slip and fall." Because of an absence of signature symptoms,<sup>65</sup> the injury in a case of generic toxic tort may have many possible causes. Furthermore, other characteristics of the generic toxic tort — a long latency period, complicated biological explanation, and lack of a single sharp exposure event — are likely to make alternative causes seem both substantially probable and difficult to exclude.<sup>66</sup> In the symbolic language of section II.A, the initial probability  $c$  of non-excludable causes is likely to be large and the diagnostic accuracy  $r$  is likely to be low. In sum, the generic toxic tort embodies a scenario that recalls the least favorable (from the plaintiff's viewpoint) aspects of Table 1's Situation 1 and Table 2's Situation 2.

In contrast, the "slip and fall" — with its single sharp "exposure" event, straightforward physiological explanation, near coincidence of "exposure" and injury, and signature-like symptoms — is likely to have a high initial probability for the alleged path of causation, and relatively few and frequently excludable alternative causes. Thus, for the

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<sup>62</sup> The provision of upper and lower bounds is modeled on the common scientific practice of providing "confidence intervals" that mark the limits within which a value is expected to lie with a specified level of probability. See FOSTER & HUBER, *supra* note 32, at 77. For example, in an ideal world, a physician might estimate the probability of causation as 30%, with a 90% chance that the real probability is between 20% and 50%.

<sup>63</sup> See *Daubert v. Merrell Dow Pharms., Inc.*, 509 U.S. 579, 592–93 (1993) (requiring that the judge assess whether the expert's reasoning is valid).

<sup>64</sup> Even "hard science hawks" tend to believe that such inquiries can eliminate much unfounded testimony. See, e.g., Black, *supra* note 29, at 685–86 ("Courts that have effectively controlled medical and forensic scientific evidence generally have done so because they identify and examine the reasoning behind conclusions."); Poulter, *supra* note 30, at 231–32 (asserting that medical speculation can "easily be unmasked by inquiring into the reasoning behind the witness's opinion").

<sup>65</sup> See Poulter, *supra* note 30, at 199–200, 216.

<sup>66</sup> See *id.*

“slip and fall,” a large initial probability  $a$  for the alleged cause and a small initial probability  $c$  for the non-excludable causes may mitigate deficiencies in the diagnostic accuracy  $r$ : the “slip and fall” likely corresponds to a scenario resembling Table 2’s Situation 1. In sum, this Note has both confirmed the general judicial intuition and supplemented it with a useful analytic structure: straightforward number-crunching shows that a physician’s testimony on causation is far more likely to be helpful in a case involving a “slip and fall” than in one involving a generic toxic tort.

### C. Application of the Framework

Having developed guidelines for assessing the relevance and reliability of differential diagnosis, this Note now explores how these guidelines might have applied to two recently decided cases — *Moore v. Ashland Chemical Inc.*<sup>67</sup> and *Heller v. Shaw Industries, Inc.*<sup>68</sup> The proposed applications show this Note’s approach to have the desired combination of flexibility and substance,<sup>69</sup> admitting more testimony than toxic tort rules,<sup>70</sup> while retaining significant hurdles to admissibility.

In *Moore*, the Fifth Circuit confronted the case of a truck driver, Bob Moore, who began suffering respiratory problems within approximately one hour of completing a cleanup of a serious chemical spill in his trailer.<sup>71</sup> In the weeks following his exposure, Moore continued to experience difficulty breathing, and within a few months he left his job.<sup>72</sup> Dr. Daniel Jenkins, a pulmonary specialist, diagnosed Moore as having reactive airways dysfunction syndrome (RADS), an asthma-like condition.<sup>73</sup> Dr. Jenkins supported his opinion with a rough estimate that Moore was exposed to contaminant levels of at least 200 parts per million,<sup>74</sup> and based his diagnosis on the normal sources of a clinical physician’s opinion, including differential diagno-

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<sup>67</sup> 151 F.3d 269 (5th Cir. 1998) (en banc), cert. denied, 119 S. Ct. 1454 (1999).

<sup>68</sup> 167 F.3d 146 (3d Cir. 1999).

<sup>69</sup> See *supra* p. 1471 and note 23.

<sup>70</sup> See *supra* p. 1472.

<sup>71</sup> See *Moore*, 151 F.3d at 271–72. Moore was the epitome of the “eggshell plaintiff”: he had just returned to work after having pneumonia, had been a pack-a-day smoker for approximately twenty years, and had a history of childhood asthma. See *id.* at 272.

<sup>72</sup> See *id.*

<sup>73</sup> See *id.*

<sup>74</sup> See *Moore v. Ashland Chem., Inc.*, 126 F.3d 679, 695 (5th Cir. 1997), *rev’d en banc*, 151 F.3d 269 (5th Cir. 1998), cert. denied, 119 S. Ct. 1454 (1999). There was no record of the actual contaminant level because Ashland Chemical’s plant manager had neglected to test the air in the trailer, despite “ha[ving] access to a meter provided by Ashland for this purpose.” *Id.* at 693.

sis.<sup>75</sup> Nonetheless, the trial court refused to admit Dr. Jenkins's testimony that Moore's exposure caused his RADS.<sup>76</sup>

The clinical physician in *Heller* fared no better. The physician's patients, Thomas and Carol Heller, experienced respiratory problems after moving into a nine-year-old house.<sup>77</sup> Thomas Heller's problems began shortly after the move,<sup>78</sup> and Dr. Joseph Papano, an allergist, attributed them to cat hair in the house's old carpets.<sup>79</sup> Some time after the Hellers installed a new carpet, Carol Heller began to experience respiratory difficulties.<sup>80</sup> The Hellers moved out of the house.<sup>81</sup> In their absence, a test of the air in a closet containing the new carpet revealed the presence of fourteen volatile organic compounds (VOCs).<sup>82</sup> The Hellers then had the new carpet removed.<sup>83</sup> Nonetheless, when the Hellers visited the house six days after the carpet's removal, both their breathing problems returned — even though the number of detectable VOCs had dropped from fourteen to five and the total concentration of VOCs had dropped to 6.85 parts per billion, a decrease of two thirds from the level previously measured.<sup>84</sup> In the Hellers' subsequent suit against the carpet manufacturer, Dr. Papano offered to testify that the new carpet had caused Carol Heller's problems, the basis for his testimony being differential diagnosis and "the temporal relationship between her symptoms and the installation of the [new] carpet."<sup>85</sup> The trial court excluded Dr. Papano's testimony.<sup>86</sup>

Even with these skeletal fact patterns, it is not hard, within the scheme of sections II.A and II.B, to conclude that Dr. Jenkins's testimony in *Moore* might have been properly admitted, but that Dr. Pa-

<sup>75</sup> Among the bases for Dr. Jenkins's opinion were personal examination of Moore, study of Moore's medical history, analysis of the results of laboratory tests designed to rule out possible allergic or immunologic causes, review of reports by other doctors, review of the manufacturer's warning about the hazardous nature of the chemical solution at issue, reference to medical literature that documented the triggering of RADS by similar compounds, and reference to his own extensive training and experience. *See id.* at 696–97.

<sup>76</sup> *See Moore*, 151 F.3d at 273. Somewhat oddly, "[t]he district court decided to admit [another doctor's] causation opinion even though it was essentially identical to Dr. Jenkins's proffered opinion." *Id.* at 274.

<sup>77</sup> *See Heller v. Shaw Indus.*, 167 F.3d 146, 150 (3d Cir. 1999).

<sup>78</sup> *See id.*

<sup>79</sup> *See Heller v. Shaw Indus.*, No. Civ. A. 95-7657, 1997 WL 535163, at \*1 (E.D. Pa. Aug. 18, 1997), *aff'd*, 167 F.3d 146 (3d Cir. 1999).

<sup>80</sup> *See Heller*, 167 F.3d at 150.

<sup>81</sup> *See id.* According to Carol, her condition improved after the Hellers moved. *See id.*

<sup>82</sup> *See id.* at 150.

<sup>83</sup> *See id.* at 151.

<sup>84</sup> *See Heller*, 1997 WL 535163, at \*3–\*4. Whereas VOC concentrations of several parts per million often exceed recommended safety limits, concentrations of less than ten parts per billion are not far from naturally occurring levels and are "substantially lower than any amounts ever known or believed to cause illnesses in humans." *Heller*, 167 F.3d at 161 & n.13.

<sup>85</sup> *Heller*, 167 F.3d at 151.

<sup>86</sup> *See id.*

pano's testimony in *Heller* was rightly excluded. Both experts offered biologically plausible theories of causation, and both invoked temporal proximity. Nevertheless, the argument of temporal proximity was much more compelling in *Moore* than in *Heller*. In *Moore*, there was a discrete event of sharp exposure, followed almost immediately by the onset of respiratory problems. In *Heller*, the intensity of exposure was comparatively small,<sup>87</sup> and the "late December" time period in which Carol's symptoms developed was much more diffuse.<sup>88</sup> On the spectrum stretching from the classic "slip and fall" to the generic toxic tort, *Moore* stood far closer to the "slip and fall" than did *Heller*, which was plagued by more of the "low-*a*" vagaries of the generic toxic tort.

Further consideration of the time scales relating exposure and injury reinforces the sense that Moore's case for causation was inherently stronger than that of the Hellers. With his history of asthma, pneumonia, and smoking, Bob Moore appears to have been a respiratory time bomb. However, his background health risks operated at a significantly longer time scale than his exposure in the trailer. There seems no particular reason to believe that these other potential causes of respiratory disease triggered RADS on the day, the week, or perhaps even the month that Moore's symptoms developed — never mind the hour. Meanwhile, the expected time scale for an adverse reaction to his sharp exposure event was probably much shorter — much closer to the one hour that occurred. In sum, the severity of Moore's exposure, the near coincidence of his developing RADS, and the relative improbability of alternative causes suggest that Moore's situation was more like Table 1's Situation 2 than Table 1's Situation 1 — that is, more likely a situation in which differential diagnosis could be a reliable basis for testimony.<sup>89</sup>

Carol Heller's situation differed substantially from Bob Moore's. Carol Heller had just moved into a new house filled with a variety of potential triggers for an allergic reaction. Most of the possible alternative causes for her symptoms — such as the residual cat hair to which Dr. Papano had attributed her husband's symptoms — were likely to operate on a time scale (and to be present with an intensity) quite close to that associated with the risks presented by the new carpet. Furthermore, the sequence of events in *Heller* itself cast doubt on Dr. Pa-

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<sup>87</sup> Although the Hellers offered expert testimony that the total VOC concentration in December was approximately thirteen parts per million, *see id.* at 159–60, the district court justifiably found this testimony based on backward extrapolation to be inadmissible because of its reliance on "speculation and estimation that was subject to gross error," *id.* at 162.

<sup>88</sup> *Id.* at 150.

<sup>89</sup> Differential diagnosis might still be an unreliable basis for testimony if the techniques of diagnosis are themselves unreliable. Recall the contrast between Table 2's Situation 1, in which highly reliable techniques provide a solid basis for admissible testimony, and Table 2's Situation 2, in which unreliable techniques do not provide such a basis. *See supra* p. 1478.

pano's theory: Thomas Heller experienced respiratory problems even before the new carpet was installed, and both Hellers experienced allergic reactions after the new carpet had been removed.

Thus, in contrast to Dr. Jenkins in *Moore*, Dr. Papano applied his techniques of differential diagnosis to a situation unfavorable to his approach — one resembling Table 1's Situation 1 more than Table 1's Situation 2. The Third Circuit correctly concluded that the trial court did not abuse its discretion in excluding Dr. Papano's testimony. Dr. Papano's diagnostic techniques do not appear to have been sufficiently comprehensive and accurate to overcome the febrility of the initial argument for causation. In contrast, *Moore's* case came to Dr. Jenkins with real momentum: the pre-diagnostic probability of causation seemed relatively high. Careful judicial inquiry might have been necessary to decide that Dr. Jenkins did not proffer relevant and reliable testimony.

### CONCLUSION

The examples of *Moore* and *Heller* show that this Note's proposed mode of analysis can aid courts in developing reasonable responses to situations of strong scientific uncertainty. In such situations, the relevance and reliability of expert testimony has much to do with whether a particular case bears a greater resemblance to the generic toxic tort or to the classic "slip and fall." The significance of differential diagnosis and temporal proximity is inherently contextual: it depends on the sharpness of the plaintiff's exposure event, the characteristic time scales upon which the alleged cause and other potential causes are expected to act, and the comprehensiveness and accuracy of the physician's diagnostic techniques. In deciding whether such evidentiary bases are enough to let testimony through Rule 702's gate, judges should not expect easy answers. They can, however, look for reasoned justifications.<sup>90</sup> This Note's prudential approach cannot guarantee the right result, but it can consistently produce a sensible one. In a world of both imperfect knowledge and imperfect knowledge filters, one can ask for little more.<sup>91</sup>

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<sup>90</sup> See Henry Berry, *Logical Analysis: A Method of Examination of Expert Medical Opinion Through the Basic Logic of Medical Reasoning*, 34 TORT & INS. L.J. 949, 962 (1999) ("[A]n explicit and detailed analysis of differential diagnosis should assist counsel and the courts in determining the logical basis, scientific merits, level of certainty, and truth value of a final diagnosis.").

<sup>91</sup> Niels Bohr expected no more from science itself. See PAIS, *supra* note 1, at 446 ("Our task is not to penetrate into the essence of things, the meaning of which we don't know anyway, but rather to develop concepts which allow us to talk in a productive way about phenomena in nature.").