Volume 59, Issue 6 Page 1671

GENOMICS AND TOXIC TORTS: DISMANTLING THE RISK-INJURY DIVIDE

Jamie A. Grodsky

GENOMICS AND TOXIC TORTS: DISMANTLING THE RISK-INJURY DIVIDE

Jamie A. Grodsky*

Emerging genetic and molecular technologies are revolutionizing our understanding of the relationship between genes and the environment. This Article develops an innovative framework for understanding the implications of the genomic revolution for the law of toxic torts. Professor Grodsky demonstrates how new technologies are poised to challenge longstanding distinctions between legally inconsequential "risk" and remediable "injury," and how the U.S. legal system will need to adapt to this emerging reality. If the law remains wedded to conventional notions of injury, it will ignore the fruits of a scientific revolution and thus may forego new remedial opportunities as yet unimagined. This is particularly significant given that twenty-first century medicine strives to "go beyond the limitations of biology" and detect, prevent, and treat disease at the molecular level. The transformative and rapidly evolving technologies of the genomic era will present herculean challenges for the legal system. But opportunities to fashion new remedies and create new efficiencies must not be overlooked in the process. Professor Grodsky recommends legal approaches to balance the goals of deterrence and legal restraint in an age of accelerating scientific change.

* Associate Professor of Law, The George Washington University Law School. I am especially grateful to Robin Craig, Michael Dore, and Andrew Klein for their comments at various stages in the preparation of this Article. Thanks also to Michael Abramowicz, John Applegate, Jay Austin, Elizabeth Cabraser, Dan Farber, John Goldberg, Robin Juni, Richard Lewis, Gary Marchant, Tom Mounteer, Robert Rabin, Arnold Reitze, William Rodgers, J.B. Ruhl, Roger Schechter, Chris Schroeder, Dinah Shelton, Dan Solove, and Sonia Suter. I appreciated the opportunity to present this Article to the Vanderbilt Law School faculty. Katherine Strong, Elizabeth Moore, Joe Kakesh, and Maribeth Klein provided excellent research assistance. Beyond the legal community, I would like to thank Dr. David Eaton, Director of the Center for Ecogenetics and Environmental Health at the University of Washington, and Dr. Paul Schulte of the National Institute for Occupational Safety and Health (NIOSH) for reviewing the manuscript.

INTRODUCTION

Advances in molecular biology and genomics are poised to transform current conceptions of "risk" and "injury" in the law of toxic torts.[1] The legal

1. A toxic tort has been defined as "an alleged personal injury and related harm resulting from exposure to a toxic substance—usually a chemical but perhaps a biological or radiological agent." Steve Gold, Note, *Causation in Toxic Torts: Burdens of Proof, Standards of Persuasion, and Statistical Evidence*, 96 YALE L.J. 376, 376 n.1 (1986). Toxic tort cases may result from exposure to radioactive substances, airborne and waterborne releases by chemical manufacturers, and releases from toxic waste dumps and pesticide applications—such events often involve groundwater contamination. Toxic tort cases frequently are included in broader discussions of mass hazardous substances litigation and product liability law, with which they have significant overlap.

system has yet to anticipate or plan for this emerging reality. This Article argues that if the law remains wedded to conventional notions of injury, it will ignore the fruits of a scientific revolution and thus may forego new remedial opportunities that could benefit both plaintiffs and defendants in the end.

An elemental principle of personal injury law is that plaintiffs must demonstrate "harm" in the form of physical injury prior to recovery. The modern world of synthetic chemicals and toxic torts has challenged this bedrock principle. Unlike traditional accidents involving broken bones or other immediate and obvious injuries, toxic exposure may breed diseases whose symptoms take years to manifest. These delayed effects can create intractable barriers for tort plaintiffs, potentially undermining the law's deterrent and corrective justice functions. Thus, toxic torts pose the novel question of whether plaintiffs exposed to toxic hazards and placed at significant risk of disease—yet perhaps not physically "injured"—should nonetheless be entitled to some form of legal remedy.

In recent years, several nontraditional claims have evolved to help toxic tort plaintiffs overcome traditional barriers to recovery, including claims for "mental distress," "enhanced risk," and "medical monitoring." Courts are now struggling with these developments, some of which serve important fairness and deterrence goals, yet arguably may divert resources from the truly impaired and unsettle established tort doctrine. Fueled partly by recent asbestos litigation, in which claims by the allegedly "unimpaired" have overwhelmed court dockets, the "latency problem" has emerged as one of the most critical issues in modern tort law. The genomic revolution promises to make this problem even more salient and controversial in the future.[2]

Remarkably, the debate over the tort system's role in responding to risks of toxic hazards all but overlooks emerging science. While commentators engage in abstract normative discussions of whether the law should remedy for latent "risk" versus concrete "injury," I argue that science may no longer support this conceptual dichotomy. New genomic technologies will strike at the core of the current risk-injury divide.

This is happening because foundational developments in molecular biology, fueled by the application of new genomic technologies since the 1990s, are enabling progressively fine-tuned observation of the effects of toxic

---

2. The terms "genetics" and "genomics" frequently are distinguished but also have overlapping meanings. Genetics is defined most broadly as "the scientific study of heredity and hereditary variations." NEIL A. CAMPBELL ET AL., BIOLOGY: CONCEPTS AND CONNECTIONS G-11 (4th ed. 2003). Genetics has been defined more specifically as "the study of single genes and their effects," while genomics is defined as the study "of the functions and interactions of all the genes in the genome." Alan E. Guttmacher & Francis S. Collins, *Genomic Medicine—A Primer*, 347 NEW ENG. J. MED. 1512, 1512 (2002). However, "genomics" is a relatively recent term, first appearing in 1987 to mark the advent of a new scientific journal. *See* Victor A. McKusick & Frank H. Ruddle, Editorial, *A New Discipline, A New Name, A New Journal*, 1 GENOMICS 1 (1987).

substances on the body and the role of genetic makeup in modifying those toxic effects. The identification of new biological markers or "biomarkers" at the genetic and molecular levels has allowed scientists to characterize a number of previously undetectable, intermediate events between chemical exposure and environmentally induced disease. New high-speed, high-volume technologies, such as DNA "microarrays," are generating new kinds of biomarkers at an unprecedented rate and level of resolution. And as observational techniques evolve, scientists can test for suites of biological changes, providing more information than the genome alone can reveal. As a result, science may detect evidence that bodily integrity has been compromised long before classic clinical symptoms emerge.

Yet despite these developments, I argue that the law clings tenaciously to an older scientific model. Although the case law addressing subcellular damage is limited and has not yet addressed the fruits of "whole genome" research, most courts have treated such damage as benign, de minimis, or otherwise legally inconsequential.[3] Courts greatly prefer to draw bright lines between risk and injury, and continue to place the boundary at proof of classic medical symptoms or overt impairment. And indeed, this was fitting in an earlier era, when research tools were insufficient to identify many intermediate effects or to establish their relationship to ultimate disease—giving birth to the metaphor of disease emerging from an impenetrable "black box."[4] But these traditional legal presumptions about when "risk" translates to "injury" or disease may become less appropriate or desirable in the future.

Challenging this conventional framework, I draw upon the scientific literature to illustrate a growing "middle ground" between de minimis effects and classic medical symptoms. It follows that certain asymptomatic conditions, though perhaps not qualifying as fully developed (and hence fully compensable) "illness" or "disease," may nevertheless constitute risks or injuries that merit some form of legal recognition. New technologies lend support to my claim through their ability to identify damage to the body's repair functions. And so-called "early-stage" disease biomarkers may represent not only risk but the presence of disease itself. Thus, I argue, newly identifiable subclinical events may themselves represent substantially enhanced risk of disease or even a "diseased state."

Not only is the law failing to anticipate emerging science, but it may also be moving at cross purposes. For example, a growing number of jurisdictions require plaintiffs to show separately compensable physical injuries in the form of overt disease prior to recovering for medical monitoring.[5] Yet a defining feature of this cause of action, as it evolved to address the perils of toxic

3. *See infra* Part II.A.
4. *See infra* note 62 and accompanying text.
5. *See infra* Part I.A.4.

hazards,[6] is that exposed plaintiffs need *not* prove physical injury prior to recovery. This principle is grounded in pragmatism, as the very purpose of monitoring is to detect the onset of disease and allow for preventive medical intervention. Indeed, preventing disease progression at earlier stages may reduce treatment costs, limit future personal injury claims, and ultimately reduce health care costs for the nation. By forcing plaintiffs to attain late-stage injury, toxic tort law may actually discourage medical interventions that could benefit defendants and plaintiffs alike. Hence, recent legal developments not only undermine monitoring's preventive and deterrent functions, but run counter to a primary goal of twenty-first century medicine, which is to detect, prevent, and treat disease at the molecular level.[7]

Therefore, I conclude that the judiciary's retreat from medical monitoring may be coming at precisely the time when increased attention to this remedy is necessary. As research opens up new possibilities for ever-earlier medical intervention, society will need to consider whether a legal system whose remedies depend on unclear and perhaps outmoded notions of "physical injury" reflects sound science or appropriate legal policy. Limited relief for monitoring, where plaintiffs can prove the necessary elements, may appropriately balance deterrence and legal restraint in an age of accelerating scientific change.

Part I of this Article provides a snapshot of the remarkably unsettled legal landscape that the molecular-genomic revolution is soon to confront. Illustrating judicial ambivalence toward nontraditional tort theories, I highlight the growing role of a "physical injury" requirement in claims based presumptively on exposure and risk. In particular, I note the irony of requiring separately compensable injuries as predicates to medical monitoring recovery. And while a frequent justification for the injury requirement is to create a principled standard for separating valid from speculative claims, there is no consistency in the courts as to how to define physical injury. This Part also introduces elements of the new science most relevant to the future of toxic tort law. In brief, I suggest that as new molecular biomarkers blur the boundaries between risk and injury, health and disease, the oft-stated presumption that tort law provides remedies for injury but not for risk may prove to be a distinction without a difference. The concept of physical injury, already a tenuous standard for assigning legal rights and remedies, will become even more opaque as science observes the mechanisms of toxicity at the subcellular level. Courts will need to rethink just where in the exposure-disease continuum remediable injury or harm has occurred.

Part II situates future scientific discoveries in the existing doctrinal landscape, examining case law dealing with subcellular damage and other subclinical effects of toxic exposure. I suggest that two competing conceptions

---

6. *See infra* note 57, discussing the characterization of monitoring as a cause of action or, conversely, as a remedy.

7. *See infra* Part III.A.

undergird these decisions and offer these two models as heuristic devices for thinking about the implications of the genomic revolution for the future of toxic tort law. The first model, clearly the dominant view, treats subcellular damage as benign, de minimis, or, at best, legally inconsequential "risk." The alternative model, which I label the "diseased state" model, presumes that although disease may be undetectable using traditional techniques, it may nevertheless be present and ongoing, and thus the latency period may be shorter than classic medical symptoms would suggest. In applying the new science to both models, I argue that although the new data could be used to support either view, over time genomic tools may provide additional ammunition for the "diseased state" view. As a result, certain subcellular events may need to be treated as "injuries" rather than "risks," at least for certain types of claims. Viewed another way, society must decide when indicators of future harm are sufficiently predictive to qualify as harms in themselves—particularly where early intervention could thwart the ultimate disease. In sum, new molecular discoveries may represent risks or injuries that may justify certain measured remedies.

Part III discusses how the law should respond once this new generation of subclinical information inevitably finds its way into the courtroom. As can be expected, evidence of subclinical biological effects and susceptibilities may serve as ingredients for a new generation of tort claims based on exposure, risk, or the earmarks of developing disease. I argue that tailored relief for medical monitoring may be more worthwhile, and less speculative, than a new generation of claims for enhanced risk, mental distress, or "personal injury" based on subcellular damage. In essence, as technology continues to move toward earlier detection and treatment, the law may need to adapt by either: (1) recognizing the reality of "risk" while tailoring the remedy (e.g., monitoring funds), or (2) avoiding "risk" rhetoric altogether and redefining physical injury to include subcellular damage where the monitoring remedy is sought. More globally still, in the genomic age, society may need to rethink physical injury in the context of the requested remedy. For the present—until such time as science can determine just when bodily integrity has been compromised—I suggest that the law should maintain a risk-oriented framework for medical monitoring.

Next, anticipating justifiable concerns about opening the floodgates of litigation, I argue that monitoring claims will be limited by transaction costs, barriers to class certification, and other access barriers rarely discussed in this context. Moreover, while I present asbestos litigation as a useful metaphor for understanding the legal status of non-impairing conditions, I also argue that the asbestos problem is unique and must be distinguished from problems posed by genomics.[8] In this vein, I similarly distinguish the Supreme Court's holding in

8. *See infra* discussion accompanying notes 239-45.

Metro-North Commuter Railroad v. Buckley,[9] which denied a monitoring claim for "lump-sum" damages in an asbestos case arising under the Federal Employers' Liability Act (FELA).[10] The Sixth Circuit's decision in *Rainer v. Union Carbide Corp.*,[11] which denied a personal injury claim based on subcellular damage, provides hints of alternative litigation scenarios for the future.

Finally, I conclude that the blurring of risk and injury in the genomic era ultimately may lead to a convergence of remedies. Where science can not only diagnose but also treat disease at the molecular level, medical monitoring would be converted into the equivalent of a compensatory damage remedy—yet with damages greatly reduced from the damages of today. Indeed, monitoring may prove to be not just an intermediate legal remedy, but a transitional remedy in the law of torts. This future convergence of monitoring and personal injury claims will demand entirely new ways of thinking about tort law's treatment of "latent" harms.

I. THE DANCE OF LEGAL AND PHYSICAL INJURY

This Part sets the stage for thinking about the future of toxic tort law in the genomic age. In so doing, it examines the interplay of concepts of physical injury and legal injury in contemporary tort law. The concept of physical injury, the fulcrum upon which legal rights and remedies traditionally have been balanced, is an elusive one, and will become even more so in the future. This Part illustrates how an unsettled legal landscape is soon to confront a complex, increasingly controversial, yet enormously beneficial area of science, as new and powerful technologies move toward practical application. The genomic revolution will present the courts with formidable challenges, yet opportunities to fashion new remedies and create new efficiencies must not be overlooked in the process. The danger is that the coming inundation of highly complex information could prove so overwhelming and controversial that courts will retreat to the relative clarity and simplicity of traditional medical symptoms, hence reinforcing the conventional risk-injury divide.

A. *The Latency Problem in Toxic Torts*

Toxic tort cases are widely recognized to have features that distinguish them from the traditional "accident" paradigm that the tort system is best equipped to address. Unlike traditional accidents, toxic exposure often results in diseases for which symptoms may not develop for significant periods of

9. Metro-N. Commuter R.R. v. Buckley, 521 U.S. 424 (1997).
10. 45 U.S.C. §§ 51-60 (2007); *see also infra* discussion accompanying notes 183-89.
11. Rainer v. Union Carbide Corp., 402 F.3d 608 (6th Cir. 2005).

time.[12] Hence, among the many features that make toxic injuries problematic is that they are often latent, and such delayed effects aggravate the problem of proving causal relationships.[13] These and other features are widely recognized to create barriers for tort plaintiffs,[14] potentially undercutting the deterrent effect of civil liability.

1. *Emergence of nontraditional tort theories*

In recent decades, a variety of theories have evolved to help plaintiffs overcome traditional barriers to recovery in cases involving latent harm from toxic exposure.[15] Such theories support recovery for "mental distress,"[16]

---

12. *See* Robert L. Rabin, *Environmental Liability and the Tort System*, 24 HOUS. L. REV. 27, 29 (1987) ("Toxics of all sorts—impure water, hazardous chemicals, defective synthetics—often breed disease rather than cause immediate injury. . . . Since diseases do not occur instantaneously, there are serious time-lag issues . . . [as opposed to] accidental injury, the relatively sudden event in which the victim's bodily security or property is violated."); *see also* GERALD W. BOSTON & M. STUART MADDEN, LAW OF ENVIRONMENTAL AND TOXIC TORTS 7 (2d ed. 2001) (distinguishing toxic tort cases from automobile accident cases and other sporadic injuries). Moreover, bodily insults caused by chemicals may be imperceptible in the early stages. Therefore, even the most elemental question of showing injury, generally obvious in the traditional accident case, may be elusive where disease is involved.

13. Ayers v. Twp. of Jackson, 525 A.2d 287, 301 (N.J. 1987) ("By far the most difficult problem for plaintiffs to overcome in toxic tort litigation is the burden of proving causation."); *see also* Glen O. Robinson, *Probabilistic Causation and Compensation for Tortious Risk*, 14 J. LEGAL STUD. 779, 779-80 (1985) ("These lagged effects are important because they exacerbate the more basic problem of proving, even defining, causal relationships in an environment where multiple causation confounds the possibility of isolating one 'responsible' cause as the touchstone of legal liability."). Exacerbating plaintiffs' burdens, statutes of limitation may preclude suit unless jurisdictions have adopted a "discovery rule," whereby the statutory period may be tolled pending discovery of an injury. In addition, the latency problem compounds the challenge of identifying solvent defendants.

14. *See* Rabin, *supra* note 12, at 43 (discussing multiple barriers for plaintiffs in toxic injury litigation); David Rosenberg, *The Causal Connection in Mass Exposure Cases: A "Public Law" Vision of the Tort System*, 97 HARV. L. REV. 849, 892 (1984) (same). Commenting on the latency problem, one judge has noted:

> This issue goes to the very heart of our tort system, and it divides courts and commentators. The tort system evolved to redress the wrongs of a society where injuries were much more direct. The issues of lengthy latency periods and increased risks of cancers are relatively new to our system of laws. The greatest lesson that we can draw from the common law of torts to apply here is that the system must evolve to meet the needs of society.

Barth v. Firestone Tire & Rubber Co., 661 F. Supp. 193, 196 (N.D. Cal. 1987).

15. *See generally* James A. Henderson, Jr. & Aaron D. Twerski, *Asbestos Litigation Gone Mad: Exposure-Based Recovery for Increased Risk, Mental Distress, and Medical Monitoring*, 53 S.C. L. REV. 815 (2002); Andrew R. Klein, *Fear of Disease and the Puzzle of Futures Cases in Tort*, 35 U.C. DAVIS L. REV. 965, 971 (2002); Tamsen Douglass Love, *Deterring Irresponsible Use and Disposal of Toxic Substances: The Case for Legislative Recognition of Increased Risk Causes of Action*, 49 VAND. L. REV. 789, 804 (1996); Kara L. McCall, *Medical Monitoring Plaintiffs and Subsequent Claims for Disease*, 66 U. CHI. L.

"enhanced risk of disease,"[17] and "medical monitoring."[18] These nontraditional (or newly adapted)[19] theories are significant because they redefine what constitutes a legally cognizable injury[20] and hence limit the amount of evidence needed to show causation.[21] For example, for a mental distress claim, the legally cognizable injury is characterized as the plaintiff's current suffering due

---

REV. 969 (1999); Pizzirusso, *supra* note 1; Bill Charles Wells, *The Grin Without the Cat: Claims for Damages from Toxic Exposure Without Present Injury*, 18 WM. & MARY J. ENVTL. L. 285 (1994).

16. *See, e.g.*, Klein, *supra* note 15, at 971.

17. The enhanced risk cause of action seeks compensation for the present risk of future harm. *See generally* Gregory L. Ash, *Toxic Torts and Latent Diseases: The Case for an Increased Risk Cause of Action*, 38 U. KAN. L. REV. 1087 (1990); Geoffrey P. Kirshbaum, *Increased Risk of Disease as an Independent Cause of Action in Toxic Tort Cases*, 43 S. TEX. L. REV. 273 (2001); Andrew R. Klein, *A Model for Enhanced Risk Recovery in Tort*, 56 WASH. & LEE L. REV. 1173 (1999); Deirdre A. McDonnell, *Increased Risk of Disease Damages: Proportional Recovery as an Alternative to the All or Nothing System Exemplified by Asbestos Cases*, 24 B.C. ENVTL. AFF. L. REV. 623 (1997).

18. The medical monitoring claim seeks compensation for the costs of periodic testing to detect disease onset. *See generally* Akim F. Czmus, Comment, *Medical Monitoring of Toxic Torts*, 13 TEMP. ENVTL. L. & TECH. J. 35 (1994); Christopher P. Guzelian et al., *A Quantitative Methodology for Determining the Need for Exposure-Prompted Medical Monitoring*, 79 IND. L.J. 57 (2004); Andrew R. Klein, *Rethinking Medical Monitoring*, 64 BROOK. L. REV. 1 (1998); Arvin Maskin et al., *Medical Monitoring: A Viable Remedy for Deserving Plaintiffs or Tort Law's Most Expensive Consolation Prize?*, 27 WM. MITCHELL L. REV. 521 (2000).

19. Most of these theories emerged from older theoretical roots but have been adapted for the world of chemical exposures, latent diseases, and toxic tort liability. For example, plaintiffs in personal injury cases traditionally have been able to recover provable future medical expenses—including continuing medical surveillance—upon sufficient proof of an existing injury. The more recently adapted cause of action for medical monitoring "is a distinct and new form of liability only for plaintiffs who have as yet suffered no physical harm." Kenneth S. Abraham, *Liability for Medical Monitoring and the Problem of Limits*, 88 VA. L. REV. 1975, 1976 (2002). Likewise, recovery for emotional distress, originating with cases involving traumatic physical injury or death, has since expanded to cases involving distress due to exposure and fear of future disease.

20. This expansion of legally cognizable injury builds on the Restatement's definition of injury as "the invasion of any legally protected interest of another." RESTATEMENT (SECOND) OF TORTS § 7(1) (1979). Regarding the distinction between legal injury and physical injury, an early decision by Kentucky's highest court noted that "legal injury must be a violation of some legal right, and is distinct in meaning from the damage [e.g., physical] that may flow from the injury. . . . [Legal] 'injury' *means a wrong or a tort*." Combs v. Hargis Bank & Trust Co., 27 S.W.2d 955, 956 (Ky. 1930) (emphasis added).

21. As one scientist has noted, one "of the most important issues in toxic tort litigation today" is "the issue of injury and how we define it." Jack W. Snyder, *Environmental (Toxic) Torts*, 34 DUQ. L. REV. 900 (1996). This is because "our definitions of compensable injury ultimately determine what must be brought to the table to prove causation." *Id.* at 906. In *Ayers v. Township of Jackson*, one of the first decisions to recognize an independent tort claim for monitoring, the difficulty of proving causation was cited as evidence of the need for alternative tort theories. 525 A.2d 287, 302-03, 311 (N.J. 1987).

to fear of developing disease in the future.[22] In the enhanced risk cause of action, the legally cognizable injury is the present risk of future disease.[23] In a medical monitoring claim, the legal injury is variously characterized as the imposition of costs of periodic checkups,[24] the exposure and the need for monitoring,[25] or "the invasion of [plaintiff's] interest in being free from the economic burden of extraordinary medical surveillance."[26] Under these alternative theories, the tradeoff for the reduced evidentiary burden of proving injury and causation is a nontraditional remedy or reduction in recoverable damages.[27]

This expansion of the universe of legally cognizable harms has piqued impassioned debate in the courtroom and the academy. Perhaps what makes these claims most intriguing is that the normative arguments for honoring them are as persuasive as the arguments for limiting or even eliminating them.

---

22. *See* Nancy Campbell Brown, Note, *Predicting the Future: Present Mental Anguish for Fear of Developing Cancer in the Future as a Result of Past Asbestos Exposure*, 23 MEMPHIS ST. U. L. REV. 337, 344 (1993).

23. *See* Klein, *supra* note 15, at 968; *see also* Sterling v. Velsicol Chem. Corp., 647 F. Supp. 303, 321 (W.D. Tenn. 1986) (characterizing enhanced risk of disease as a "presently existing condition" in plaintiffs who suffered exposure to various toxic substances), *aff'd in part, rev'd in part*, 855 F.2d 1188 (6th Cir. 1988).

24. *See Ayers*, 525 A.2d at 304.

25. *See, e.g.*, Henry v. Dow Chem. Co., 701 N.W.2d 684, 707 (Mich. 2005) (Cavanagh, J., dissenting) ("In this case, the exposure itself and the need for medical monitoring constitute the injury."); Hansen v. Mountain Fuel Supply Co., 858 P.2d 970, 977 (Utah 1993) ("Although the physical manifestations of an injury may not appear for years, the reality is that many of those exposed have suffered some legal detriment; the exposure itself and the concomitant need for medical testing constitute the injury.").

26. Metro-N. Commuter R.R. v. Buckley, 521 U.S. 424, 450-51 (1997) (Ginsburg, J., concurring in part and dissenting in part); *see also* Redland Soccer Club, Inc. v. Dep't of Army, 55 F.3d 827, 846 (3d Cir. 1995) (defining the injury as "a need for medical monitoring greater than that what is required by all persons"); Friends for All Children, Inc. v. Lockheed Aircraft Corp., 746 F.2d 816, 826 (D.C. Cir. 1984) (describing plaintiff's injury as an invasion of a legally protected interest in avoiding medical monitoring expenses). As Justice Ginsburg noted, dissenting from the majority's denial of a monitoring claim in *Metro-North*, "'[i]t is difficult to dispute that an individual has an interest in avoiding expensive diagnostic examinations just as he or she has an interest in avoiding physical injury.'" *Metro-North*, 521 U.S. at 451 (quoting *Friends for All Children*, 746 F.2d at 826); *see also* Petito v. A.H. Robins Co., 750 So. 2d 103, 105 (Fla. Dist. Ct. App. 1999) (same).

27. *See* Robinson, *supra* note 13, at 782-83 ("[T]he issue of liability for risk creation does not entail changing the standards for defining what activities (risks) are tortious. Instead it redefines compensable 'injury' to make 'tortious' risk a basis of liability—adjusting compensation according to a probabilistic measure of anticipated loss."); *see also* Allan L. Schwartz, Annotation, *Recovery of Damages for Expense of Medical Monitoring to Detect or Prevent Future Disease or Condition*, 17 A.L.R. 5th 327, 340, 342 (1994) (noting that the enhanced risk claim "seeks compensation for the anticipated harm itself, proportionately reduced to reflect the chance that it will not occur"); Wells, *supra* note 15, at 348 ("A major part of this response has been an expansion in the definition of injury. This has in turn modified the damages a plaintiff can recover.").

Supporters of post-exposure, pre-symptom claims argue that the fundamental objectives of modern tort law, both moral (corrective justice and individual fairness)[28] and utilitarian (deterrence and loss spreading),[29] are better served if liability for toxic hazard is based on risk of injury rather than on actual occurrence of physical harm.[30] In the absence of alternative remedies, toxic torts would go seriously underdeterred.[31] Moreover, some have suggested that imposing a significant risk of disease is an affront to personal autonomy or dignitary interests,[32] making the absence of physical injury normatively irrelevant.

Critics counter that these alternative theories represent a major disruption of the longstanding intellectual framework.[33] Whether characterized as new tort theories or adaptations of earlier ones, these theories—at least in concept—may

28. For example, Christopher Schroeder has argued that a system that imposes liability based on risk of harm is better aligned with corrective justice principles than a system that focuses on actual harm. The former approach allows the defendant's behavior to be evaluated based on information available to the defendant at the time the action in question occurred. Christopher H. Schroeder, *Corrective Justice and Liability for Increasing Risks*, 37 UCLA L. REV. 439, 451-69 (1990); *see also* Christopher H. Schroeder, *Rights Against Risks*, 86 COLUM. L. REV. 495 (1986).

29. From a deterrence perspective, "[t]he cumulative result of the judicial system's disinclination to confront and redress [toxic] harms head-on is an informal immunity for those creating the risks of exposure." Lisa Heinzerling & Cameron Powers Hoffman, *Tortious Toxics*, 26 WM. & MARY ENVTL. L. & POL'Y REV. 67, 75 n.40 (2001). "The focus of deterrence policy is properly on the avoidance of unreasonable *risks*, not simply on the avoidance of injury." Robinson, *supra* note 13, at 784. "When liability is imposed for actual injury arising out of unreasonable risk, the supposed effect is to deter the creation of the risk itself. However, the present all-or-nothing character of [physical] injury-based liability rules can distort deterrence." *Id.* Admittedly, the all-or-nothing system can produce overdeterrence, but a more serious concern is the possibility of "underdeterrence where significant but not 'substantial' risks go unpenalized." *Id.*

30. Robinson, *supra* note 13, at 789 ("The argument for recognizing risk as a sufficient basis for liability is as strong from corrective justice norms of fairness as from norms of efficiency."); *see also* Robert J. Rhee, *The Application of Finance Theory to Increased Risk Harms in Toxic Tort Litigation*, 23 VA. ENVTL. L.J. 111 (2004) (asserting, on both moral and utilitarian grounds, that freedom from increased risk should be a legally cognizable interest).

31. *See, e.g.*, Love, *supra* note 15, at 804.

32. *See, e.g.*, Heinzerling & Hoffman, *supra* note 29, at 90; E. Donald Elliott, *The Future of Toxic Torts: Of Chemophobia, Risk as a Compensable Injury and Hybrid Compensation Systems*, 25 HOUS. L. REV. 781, 784-90 (1988); Kirshbaum, *supra* note 17, at 294; *cf.* Albert C. Lin, *The Unifying Role of Harm in Environmental Law*, 2006 WIS. L. REV. 897, 946 (discussing "dread" and fear as setbacks to individuals' welfare interests).

33. *See* Henderson & Twerski, *supra* note 15, at 846-47 ("[T]he plaintiffs are seeking to recover pure economic loss in the absence of either personal injury or property damage. Recognizing these claims represents an important conceptual extension that is obfuscated in the judicial decisions and academic commentary characterizing the recoveries as being merely remedial in nature.").

permit physically "uninjured" plaintiffs to recover.[34] To many, this represents a radical departure from common law tradition, where tort recovery generally required physical contact that caused bodily harm.[35] Moreover, expending judicial resources in the pre-symptom stage can result in awards of speculative damages that may over- or under-compensate for actual loss.[36] Such a program may bring about an inefficient and inequitable distribution of a finite asset pool, diverting judicial resources from plaintiffs with disabling disease.[37] Beyond this, the expansive conception of legally cognizable injury arguably raises enormous practical challenges in limiting the universe of potential claimants, given that most people are exposed to potentially hazardous substances in their daily lives.[38]

2. *The asbestos controversy*

The asbestos problem, while unique on many dimensions,[39] has heightened the debate over post-exposure, pre-symptom claims.[40] A significant proportion

34. Indeed, a critique of medical monitoring by the Washington Legal Foundation refers to monitoring as "the non-injured plaintiff's tort." Steven J. Boranian & Kevin M. Hara, *Medical Monitoring: Innovative New Remedy or Money for Nothing?* 2 (Washington Legal Found. Critical Legal Issues, Working Paper Series No. 136, 2006).

35. *See, e.g.*, RESTATEMENT (THIRD) OF TORTS: LIABILITY FOR PHYSICAL HARM § 4 (Tentative Draft No. 1, 2001) ("Physical Harm means the physical impairment of the human body . . . . The physical impairment of the human body includes physical illness, disease and death."); *see also* Wells, *supra* note 15, at 348.

36. *See* Henderson & Twerski, *supra* note 15, at 823 (arguing that courts should not allow recovery for "guessing" at future results); Robinson, *supra* note 13, at 786 (contending that depending on whether the risk actually materializes in the future, recovery for risk-based claims may either over- or under-compensate).

37. *See* Henderson & Twerski, *supra* note 15, at 834 (noting that permitting risk-based claims may give precedence to the unimpaired over the seriously injured); Robinson, *supra* note 13, at 795-97 (discussing the difficulty of defining the class(es) of cases for which risk-based liability is appropriate and noting that unchecked recognition of risk as a basis for liability would increase the number of claims filed and lead to prosecution of insubstantial and highly speculative claims); *see also* Ash, *supra* note 17, at 1090.

38. Henderson & Twerski, *supra* note 15, at 846 (discussing "the potential for a plague of future litigation" with which the tort system may be "institutionally incapable of dealing"); *see also infra* notes 183, 230 (discussing floodgates concerns in *Metro-North* and decisions following).

39. *See infra* text accompanying notes 239-41 (discussing the signature status of certain asbestos-related diseases, the availability of clear and durable markers of asbestos exposure, and other distinguishing features of asbestos litigation).

40. A recent report by the RAND Institute for Civil Justice noted that "[o]ne of the most hotly debated issues in asbestos litigation concerns whether unimpaired asbestos claimants ought to be compensated." STEPHEN J. CARROLL ET AL., RAND INST. FOR CIVIL JUSTICE, ASBESTOS LITIGATION 7 (2005). For a discussion of recent trends in asbestos litigation, see Deborah R. Hensler, *As Time Goes By: Asbestos Litigation After* Amchem *and* Ortiz, 80 TEX. L. REV. 1899 (2002).

of new asbestos claims are by plaintiffs who may have recognized markers of asbestos exposure but who are not currently considered impaired or diseased.[41] The vast majority are those experiencing non-impairing "pleural plaques," or calcified deposits on the lining of the lung that occur well before development of serious asbestos-related malignancies such as lung cancer or mesothelioma.[42] Indeed, many plaque cases never progress at all.[43] Other plaintiffs may have asbestosis, scarring of the lung tissue that is non-impairing in some forms but "debilitating and even fatal" in others.[44] Although plaques and asbestosis are clear markers of asbestos exposure, enormous controversy has surrounded the question of whether they are "injurious" in their own right or whether they are predictive of future disease. Underscored by the surge in asbestos filings, the problem of latent risk claims has emerged as one of the

41. The recent RAND report on asbestos litigation concluded that "a large and growing proportion of the claims entering the system in recent years" had been submitted by "individuals who had not . . . suffered an injury that had as yet affected their ability to perform the activities of daily living." CARROLL ET AL., *supra* note 40, at 76. The report noted, however, that "the fraction of claimants with nonmalignant diseases that are functionally unimpaired at the time they file a claim is a source of sharp controversy." *Id.* at 75. Statistics are more readily available for plaintiffs with nonmalignant conditions, whether or not such plaintiffs would be characterized as unimpaired. According to the report, claims by individuals with nonmalignant conditions accounted for roughly eighty percent of asbestos claims entering the system through the mid-1980s. "The fraction of claims that asserted nonmalignant conditions grew through the late 1980s and early 1990s, rising to more than 90 percent of annual claims in the late 1990s and early 2000s." *Id.*

42. Peter Schuck, James Henderson, and Aaron Twerski have provided clear descriptions of the progression of effects related to asbestos exposure. *See* Henderson & Twerski, *supra* note 15, at 817 n.2; Peter H. Schuck, *The Worst Should Go First: Deferral Registries in Asbestos Litigation*, 15 HARV. J.L. & PUB. POL'Y 541, 544-49 (1992). The beginning of the progression occurs when asbestos fibers first lodge in the lungs. *See* Henderson & Twerski, *supra* note 15, at 817 n.2. Although the continuing presence of asbestos fibers is considered to be asymptomatic and does not always lead to pulmonary harm, asbestos fibers serve as ongoing markers of asbestos exposure. *Id.* Ten to fifteen years after exposure to asbestos, pleural "plaques," or calcified deposits on the lining of the lung, may become visible. *See* Lester Brickman, *On the Theory Class's Theories of Asbestos Litigation: The Disconnect Between Scholarship and Reality*, 31 PEPP. L. REV. 33, 51 (2004). Although the development of plaques is strongly indicative of asbestos exposure, most scientists believe they are not predictive of future asbestos-related malignancies. *See infra* note 157. Eventually, some individuals exposed to asbestos will develop a more serious condition called asbestosis. This scarring of the lung tissue may be non-impairing in its mildest forms, but can also produce highly debilitating symptoms. Henderson & Twerski, *supra* note 15, at 817 n.2. Further along the exposure-disease timetable, certain exposed individuals will experience lung and other cancers that have been linked to asbestos, but may have causes other than asbestos exposure. The most serious disease that may follow asbestos exposure is mesothelioma, a malignant and incurable form of cancer that usually results in death within fifteen months of onset. *Id.* Asbestosis and mesothelioma are "signature diseases," in that asbestos exposure is generally believed to be the exclusive or predominant cause. *See infra* notes 240-41.

43. *See infra* note 157 (discussing the limited predictive capacity of pleural plaques).

44. CARROLL ET AL., *supra* note 40, at 13; *see also* Brickman, *supra* note 42, at 44-46.

most nettlesome and critical problems in modern tort law.[45] Although problems posed by genomics are readily distinguishable from those posed by asbestos,[46] the genomic revolution promises to make the problem of latent risk claims even more nuanced and compelling in the future.

3. *Jurisprudence of ambivalence: Physical injury as a predicate to "risk-based" recovery*

As a result of the tensions between traditional and emerging tort theories, courts have not reached consensus regarding the requisite elements or even the legitimacy of the newer claims. Perhaps ironically, escalating judicial discomfort with "risk-based" theories has led to renewed efforts to require "present physical injuries" as predicates to recovery. In some cases, as with claims for mental distress, evidence of impact or some physical consequence may help validate plaintiffs' claims.[47] Yet where late-stage injuries are required, such conditions challenge the very premise of these alternative theories by restoring the traditional injury-causation relationship and reconstructing traditional barriers to recovery.[48] Thus, inextricably bound up with the philosophical question of whether the tort system should redefine *legal* injury is the appropriate definition of *physical* injury. And while an oft-stated justification for the injury requirement is to create a principled standard for

45. *See* Geoffrey C. Hazard, Jr., *The Futures Problem*, 148 U. PA. L. REV. 1901, 1901 (2000) (discussing the problem of future claimants as "[p]erhaps the most difficult problem in addressing mass torts").

46. *See infra* text accompanying notes 239-45 (discussing issues of validity, specificity, durability, and reliability posed by subcellular data). *But see infra* Part II.C.2 (discussing the potential predictive value of certain kinds of genetic and molecular markers and the presumptively limited predictive capacity of pleural plaques).

47. To recover for mental distress, a plaintiff generally must prove that he or she has been exposed to a hazardous substance, that the plaintiff has suffered mental distress due to the exposure, and that the fear which causes the distress is "reasonable." To validate this fear and limit potentially unlimited claims in toxic tort cases, courts have required either a physical consequence or other evidence showing that the mental distress is reasonable. *See* Klein, *supra* note 16, at 972. The vast majority of mental distress claims are rejected unless accompanied by physical harm or at least some form of physical manifestation. *See* Brown, *supra* note 22, at 344. Similarly, many courts have required a present physical injury to provide an objective basis for an enhanced risk claim. This requirement may be explicit or implicit. *See* Amendola v. Kan. City S. Ry. Co., 699 F. Supp. 1401, 1405 (W.D. Mo. 1988) ("Dicta found in several opinions further indicates that a claim for increased risk of future disease without accompanying allegations of present physical injury is insufficient to state a claim for relief."); Brafford v. Susquehanna Corp., 586 F. Supp. 14, 17 (D. Colo. 1984) (requiring a showing of present physical injury to validate an enhanced risk claim). Those courts that do not require predicate injuries for enhanced risk recovery generally require plaintiffs to prove that the emergence of disease is "more probable than not"—a barrier that has proved insurmountable for most plaintiffs. *See* Klein, *supra* note 17, at 1180-81.

48. *See infra* Part III.

separating valid from speculative claims,[49] there is no consistency in the courts as to what constitutes physical injury.[50]

4. *Requiring injury to detect injury: The medical monitoring paradox*

Most emblematic of escalating judicial tensions are recent decisions requiring predicate injuries for medical monitoring recovery.[51] Importantly, the *sine qua non* of a cause of action for medical monitoring—arguably the most distinctive feature that sets it apart from a traditional tort claim—is that plaintiffs need not prove physical injury prior to recovery.[52] Instead, plaintiffs generally must prove significant exposure to known hazardous substances, significant risk of serious disease, and a demonstrated need for special testing.[53] This exclusion of a physical injury requirement is logical, given

---

49. As the Michigan Supreme Court noted recently when denying a claim for medical monitoring in *Henry v. Dow Chemical Co.*:

> In allowing recovery only to those who have actually suffered a present physical injury, the fact-finder need not engage in speculations about the extent to which a plaintiff possesses a cognizable legal claim. . . . The present physical injury requirement establishes a clear standard by which judges can determine which plaintiffs have stated a valid claim, and which plaintiffs have not.

701 N.W.2d 684, 690-91 (2005).

50. *See infra* Part II.

51. *See, e.g.*, Paz v. Brush Engineered Materials, Inc., 949 So.2d 1 (Miss. 2007); *Henry*, 701 N.W.2d at 686, 695-97; Wood v. Wyeth-Ayerst Labs., 82 S.W.3d 849, 857-59 (Ky. 2002); Hinton v. Monsanto Co., 813 So. 2d 827, 830-32 (Ala. 2001). For a compilation of state requirements for medical monitoring recovery through 2006, see D. Scott Aberson, Note, *A Fifty-State Survey of Medical Monitoring and the Approach the Minnesota Supreme Court Should Take When Confronted with the Issue*, 32 WM. MITCHELL L. REV. 1095, 1115-16 (2006).

52. The rationale for excluding physical injury was underscored in *Ayers v. Township of Jackson*, 525 A.2d 287 (N.J. 1987), and *In re Paoli Railroad Yard PCB Litigation v. Monsanto Co.* (*Paoli I*), 916 F.2d 829, 852 (3d Cir. 1990) (same), two early and influential cases recognizing monitoring as an independent cause of action.

53. To support a monitoring claim, plaintiffs generally must prove that they were (1) significantly exposed to a proven hazardous substance through the actions of the defendant; (2) as a proximate result of exposure, they suffered a significantly increased risk of contracting a serious latent disease; (3) as a consequence of the exposure, a reasonable physician would prescribe a monitoring regime different from the one that would have been prescribed in the absence of the exposure; and (4) testing procedures exist that make the early detection and treatment of disease possible and beneficial. *See, e.g*, *In re* Paoli R.R. Yard PCB Litig. (*Paoli II*), 35 F.3d 717, 788 (3d Cir. 1994); *Paoli I*, 916 F.2d at 852; *Ayers*, 525 A.2d at 312. Many jurisdictions that recognize monitoring as an independent tort roughly follow the *Ayers* or *Paoli* formulations; however, the required elements vary considerably among jurisdictions. *See infra* notes 218-23. For example, several decisions require the exposure to have been caused by a negligent act. *Paoli I*, 916 F.2d at 852 (requiring proof of exposure "through the negligent actions of the defendant"). While some jurisdictions condition monitoring recovery on the availability of treatment techniques, *see, e.g.*, *Paoli I*, 916 F.2d at 852, others hold that such a requirement overlooks the possibility of scientific advancement, *see, e.g.*, Bower v. Westinghouse Elec. Corp., 522 S.E.2d 424, 433

monitoring's inherently preventive function.[54] Indeed, requiring late-stage injury as a precondition to recovery[55] arguably eviscerates the monitoring claim.[56] Hence, the heightened focus on present physical injury is part of a deeper, foundational struggle over whether this alternative form of recovery should even be recognized.[57]

B. *Genomics and Toxic Tort Law: Two Onrushing Freight Trains*

As if the present legal muddle were not enough, it will soon be transformed by the genomic revolution. In the not-so-distant future, this unsettled and increasingly controversial area of the law will come face-to-face with an unsettled, enormously complex, and increasingly controversial area of science, as emerging molecular and genomic techniques push toward practical application.

(W. Va. Ct. App. 1999) (adopting the *Paoli* test "[w]ith the significant divergence of eliminating the requirement that diagnostic monitoring must be tied to the existence of a proven treatment protocol").

54. *See Ayers*, 525 A.2d at 312 ("The availability of a substantial remedy before the consequences of the plaintiffs' exposure are manifest may . . . have the beneficial effect of preventing or mitigating serious future illnesses and thus reduce the overall costs to the responsible parties.").

55. *See, e.g.*, *Paz*, 949 So.2d 1 (rejecting a monitoring claim in the absence of manifest physical injury and citing recent decisions holding same). Where these threshold requirements rise to the level of fully developed disease or impairment—in other words, separately compensable injuries—such conditions threaten to undermine the preventive and deterrent functions articulated in *Ayers* and *Paoli*.

56. For a recent decision sharing this perspective, see Meyer *ex rel.* Coplin v. Fluor Corp., No. SC 87771, 2007 WL 827762, at *4 (Mo. Mar. 20, 2007) ("[A] physical injury requirement essentially extinguishes the claim and bars the plaintiff from a full recovery."). Notably, the role of physical injury in monitoring recovery is currently at issue in New Jersey, home to the *Ayers* decision. New Jersey's Appellate Division recently reversed a trial court that dismissed a monitoring claim on the ground that recovery pertaining to pharmaceutical products would be denied in the absence of physical injury. *See* Sinclair v. Merck & Co., 913 A.2d 832, 840 (N.J. Super. Ct. App. Div. 2007) ("We . . . hesitate to adopt a bright-line test that would make the availability of medical monitoring dependent on the existence of a manifested disease or condition, alone.").

57. The growing trend to impose physical injury requirements reflects a fundamental disagreement as to whether the need for monitoring forms the basis of an independent cause of action, or whether monitoring costs are merely elements of damages based upon an independently compensable injury—namely, physical harm. *See, e.g.*, Henry v. Dow Chem. Co., 701 N.W.2d 684, 690 (Mich. 2005) ("We therefore reaffirm the principle that a plaintiff must demonstrate a present physical injury . . . *in addition to* economic losses that result from the injury in order to recover under a negligence theory.") (emphasis in original). While some courts are explicit in denying the stand-alone monitoring claim, other courts—by requiring manifest disease or impairment prior to recovery—may be doing so implicitly.

1. *Opening the "black box": New intermediate biomarkers*

First, developments emerging in the 1980s[58] and fueled by the application of new genomic technologies since the 1990s have enabled progressively more nuanced and fine-tuned observation of the effects of toxic substances on the body and the role of genetic makeup in modifying those toxic effects.[59] The identification of new biological markers, or "biomarkers,"[60] at the genetic and molecular levels has allowed scientists to characterize a number of previously undetectable, intermediate events between chemical exposure and environmentally induced disease.

Traditionally, the science of toxic injury has focused on health effects toward the end of the exposure-disease continuum, such as tumor formation, major organ and tissue dysfunction, or other clinical symptoms.[61] Classical toxicological methods were insufficient to identify and characterize many

58. The most significant development consisted of the application of the tools of molecular biology to the disciplines of toxicology and epidemiology, the cornerstones of environmental risk assessment. *See, e.g.*, Robert E. Hurst & Jian Yu Rao, *Molecular Biology in Epidemiology*, *in* MOLECULAR EPIDEMIOLOGY: PRINCIPLES AND PRACTICES, *supra* note 58, at 45; Bernard A. Schwetz, *Toxicology at the Food and Drug Administration: New Century, New Challenges*, 20 INT'L J. TOXICOLOGY 3, 4-5 (2001) (describing the influx of molecular biologists into the field of toxicology during the 1980s). The application of molecular biology techniques to other disciplines during the 1980s provided new molecular-level insights into important biological processes, including cellular responses to drugs and toxic chemicals. Hurst & Rao, *supra*, at 45.

59. *See, e.g.*, Jamie A. Grodsky, *Genetics and Environmental Law: Redefining Public Health*, 93 CAL. L. REV. 171 (2005) (discussing recent technological advances and their implications for environmental regulation).

60. Biomarkers are "clues" or "flags" signaling events in living systems. *See, e.g.*, Frederica P. Perera, *Uncovering New Clues to Cancer Risk*, SCI. AM., May 1996, at 54-55. Formally they are defined as "indicators signaling events in biological systems or samples." Comm. on Biological Markers, Nat'l Research Council, *Biological Markers in Environmental Health Research*, 74 ENVTL. HEALTH PERSP. 3, 3 (1987). Another definition is "any measurement in or from biological material that defines an exposure or response to that exposure." Andrij Holian, *Air Toxics: Biomarkers in Environmental Applications—Overview and Summary of Recommendations*, 104 ENVTL. HEALTH PERSP. 851, 851 (1996). Hence, "[b]iomarkers are indicators of exposure, effect, or susceptibility that are measured in biologic materials," such as tissues or bodily fluids, as opposed to estimates based on levels of foreign compounds in the ambient environment. Am. Thoracic Soc'y, *What Constitutes an Adverse Health Effect of Air Pollution? Official Statement of the American Thoracic Society*, 161 AM. J. RESPIRATORY & CRITICAL CARE MED. 665, 669 (2000) (adopted July 1999). The biomarker concept predated the 1980s. Traditional biomarkers include the presence of lead in the bloodstream and arsenic, lead, or mercury in urine, indicating exposure to these substances. Nat'l Research Council, *supra*, at 6. However, the new biomarkers reveal molecular *interactions* or events within biological systems, thereby providing more information than the physical presence of foreign compounds or their immediate derivatives.

61. Anthony P. DeCaprio, *Biomarkers: Coming of Age for Environmental Health and Risk Assessment*, 31 ENVTL. SCI. & TECH. 1837, 1841 (1997).

intermediate events or to establish their relationship to ultimate disease, giving rise to the concept of a "black box."[62] The tools of molecular biology effectively opened this black box,[63] revealing a continuum of events between chemical exposure and clinical disease.[64] As a result, science may detect evidence of chemically induced changes long before clinical symptoms emerge. In addition, new genetic technologies are revealing gene sequence variations, or "polymorphisms,"[65] that may confer differential human sensitivity to the effects of toxic substances.[66] These susceptibilities may influence an

62. *Id.* at 1838. The black box signified the unknown biological events occurring between exposure to chemicals and the ultimate development of overt disease symptoms.

63. Geoffrey Rose, *Preventive Cardiology: What Lies Ahead?*, 19 PREVENTIVE MED. 97, 100-01 (1990); *see also* Paul A. Schulte, *A Conceptual and Historical Framework for Molecular Epidemiology*, *in* MOLECULAR EPIDEMIOLOGY: PRINCIPLES AND PRACTICES, *supra* note 58, at 3, 13 (discussing the "black box" approach and the new resolving powers of molecular epidemiology); DeCaprio, *supra* note 61, at 1838-40 (discussing the "black box" model and molecular techniques that identify multiple steps between exposure and disease); Perera, *supra* note 60, at 54-55 (using diagram to distinguish molecular epidemiology from the earlier "black box" model).

64. DeCaprio, *supra* note 61, at 1838. Perhaps the most important contribution of the biomarker paradigm is the concept of a continuum of effects between environmental exposure and disease. At one end of the continuum is exposure to a toxic substance, and at the other a manifestation of overt disease, such as a cancerous tumor that may appear years after the initial toxic exposure. The area between the two, once considered a "black box," now includes subcellular biomarkers of exposure and effect. When visualized as points along a horizontal bar, markers will proceed from left to right, with markers of exposure followed by markers of effect. *See* Perera, *supra* note 60, at 54-55. Although these events are most clearly illustrated in the context of cancer, they also may be applied to neurological, immunological, reproductive, developmental, pulmonary, and other environmentally related health effects.

65. Polymorphisms are common sequence variations within genes that may include nucleotide substitutions, deletions, insertions, or gene duplications or deletions. Merrill C. Miller, III et al., *Genetic Variability in Susceptibility and Response to Toxicants*, 120 TOXICOLOGY LETTERS 269, 270 (2001); N.J. Schork et al., *Single Nucleotide Polymorphisms and the Future of Genetic Epidemiology*, 58 CLINICAL GENETICS 250, 251-52 (2000); *see also* Francis S. Collins et al., *Variations on a Theme: Cataloging Human DNA Sequence Variation*, 278 SCI. 1580 (1997).

66. Susceptibility biomarkers have been defined as any identifiable genetic variations in absorption, metabolism, or response to environmental agents. Nat'l Research Council, *supra* note 60, at 6. Importantly, these genetic variations do not act alone to trigger disease, but confer differential sensitivity to the effects of drugs or chemicals. In other words, "susceptibility genes are neither necessary nor sufficient to cause disease. They modify risk." Kenneth Olden & Janet Guthrie, *Genomics: Implications for Toxicology*, 473 MUTATION RES. 3, 5 (2001). The relationship between genes and the environment has been compared to a "loaded gun and its trigger": "A loaded gun by itself causes no harm; it is only when the trigger is pulled that the potential for harm is related or initiated. Likewise, one can inherit a predisposition for a devastating disease, yet never develop the disease unless exposed to the environmental trigger(s)." *Id.* at 3-4. Such genetic variations may increase the rate at which carcinogens or other harmful substances are activated, reduce an individual's ability to detoxify harmful compounds, or disable DNA repair mechanisms, tumor suppressor genes,

individual's rate of progression from one biomarker to the next along the exposure-disease continuum[67]—suggesting that, for a given exposure, certain individuals are at greater risk of future disease. The molecular-level biomarkers have been roughly divided into three categories signifying exposure,[68] effects of exposure,[69] and susceptibility.[70]

2. *Power, speed, and scale: Whole genome research*

Since the mid-1990s, "high-throughput" technologies,[71] including DNA "microarrays" or "gene chips,"[72] have permitted thousands of genes to be

---

or other protective functions. In this manner, one's genetic complement may affect the toxicity or potency of chemicals. *Id.*

67. Schulte, *supra* note 63, at 14.

68. Exposure biomarkers reflect the amount of a foreign compound that is absorbed into the body. Exposure biomarkers may include the parent chemical, metabolic derivatives, or early interactive products of the chemical or drug and the biological system. Nat'l Research Council, *supra* note 60, at 3. Biological measurements performed on human tissues are expanding the range of tools available to classical epidemiology, which has relied primarily on indirect estimates of human exposure derived from chemical levels in the air, water, and other exposure routes. Paul W. Brandt-Rauf & Sherry I. Brandt-Rauf, *Biomarkers—Scientific Advances and Societal Implications*, *in* GENETIC SECRETS—PROTECTING PRIVACY AND CONFIDENTIALITY IN THE GENETIC ERA 186 (Mark A. Rothstein ed., 1997).

69. Biomarkers of effect reflect changes in cells or tissues triggered by chemical exposures, Perera, *supra* note 60, at 54, or changes that are qualitatively or quantitatively predictive of health impairment or potential impairment due to toxic exposure. Nat'l Research Council, *supra* note 60, at 4-5. Biomarkers of effect may include early biochemical or cellular changes, structural or functional changes in affected cells or tissues, or changes formally recognized as health impairments or clinical disease. DeCaprio, *supra* note 61, at 1839. The distinction between biomarkers of exposure and biomarkers of effect is not clear-cut: "These assignments are not mutually exclusive, and the distinctions between adjacent stages are frequently blurred." *Id.* at 1838; *see also* Nat'l Research Council, *supra* note 60, at 3 ("[T]here is a continuum between markers of exposure and markers of health status, with certain events being relatable to both types of markers."). Importantly, these classifications may change as our knowledge increases.

70. For a discussion of susceptibility biomarkers, see *supra* note 66. For a comprehensive discussion of the three categories of biomarkers, see Nat'l Research Council, *supra* note 60, at 3.

71. The term "high throughput" signifies the high volume and rate at which biological information can be assessed using these technologies.

72. Microarray technology blends molecular biology techniques with advanced computer, robotics, and information technologies. In a DNA microarray, each chip is manufactured to contain thousands of target genes or pieces of genes. The technology permits expression patterns in normal cells to be compared to mutant cells, untreated cells to be compared to cells treated with drugs or chemicals, and normal tissues to be compared to diseased tissues. *See* Marilyn J. Aardema & James T. MacGregor, *Toxicology and Genetic Toxicology in the New Era of "Toxicogenomics": Impact of "-Omics" Technologies*, 499 MUTATION RES. 13, 14 (2002); Hisham K. Hamadeh et al., *Discovery in Toxicology: Mediation by Gene Expression Array Technology*, 15 J. BIOCHEMISTRY & MOLECULAR

monitored simultaneously to observe their responses to chemical exposures.[73] Whereas scientists traditionally focused on one or a few genes at a time, these high-speed, high-volume technologies can potentially be used to scan the entire human genome to search for chemically induced changes.[74] The fields of "toxicogenomics"[75] and "toxicogenetics"[76] are devoted exclusively to this research. The former focuses on general mechanisms of toxin-induced disease, while the latter studies how an individual's genetic makeup may affect the response to toxic substances.

TOXICOLOGY 231, 231-32 (2001); Hisham Hamadeh & Cynthia A. Afshari, *Gene Chips and Functional Genomics*, 88 AM. SCI. 508, 509-11 (2000); Sandra Steiner & N. Leigh Anderson, *Expression Profiling in Toxicology—Potentials and Limitations*, 112 TOXICOLOGY LETTERS 467, 468 (2000).

73. *See* Aardema & MacGregor, *supra* note 72, at 14; Hamadeh et al., *supra* note 72, at 231-32; Hamadeh & Afshari, *supra* note 72, at 509-11. The announcement of the completion of the full human genome sequence in 2003 has allowed the focus of the field of genomics to shift from mapping the genome to elucidating the function of genes, including their interaction with toxic substances.

74. Cynthia A. Afshari, *Perspective: Microarray Technology, Seeing More than Spots*, 143 ENDOCRINOLOGY 1983, 1984 (2002); Hamadeh et al., *supra* note 72, at 231; Olden & Guthrie, *supra* note 66, at 7.

75. A major focus of toxicogenomics is the systematic investigation of patterns of gene expression in cells exposed to toxic substances. *See* Khew-Voon Chin & A.-N. Tony Kong, *Application of DNA Microarrays in Pharmacogenomics and Toxicogenomics*, 19 PHARMACEUTICAL RES. 1773 (2002); Spencer Farr & Robert T. Dunn, II, *Gene Expression Applied to Toxicology*, 50 TOXICOLOGICAL SCI. 1, 1 (1999); Hisham K. Hamadeh et al., *An Overview of Toxicogenomics*, 4 CURRENT ISSUES IN MOLECULAR BIOLOGY 45 (2002); G. Orphanides, *Toxicogenomics: Challenges and Opportunities*, 140-41 TOXICOLOGY LETTERS 145 (2003). Toxicogenomics is based on the assumption that toxicity frequently evokes qualitative or quantitative changes in gene expression. *See, e.g.*, Emile F. Nuwaysir et al., *Microarrays and Toxicology: The Advent of Toxicogenomics*, 24 MOLECULAR CARCINOGENESIS 153, 154-55 (1999). As a result, scientists may gain insights into the mechanisms of toxicity by studying gene expression and downstream effects. Situated in the biomarker context, toxicogenomics focuses primarily on biomarkers of exposure and effect by studying molecular precursors to toxin-induced disease. Kenneth Olden et al., *A Bold New Direction for Environmental Health Research*, 91 AM. J. PUB. HEALTH 1964, 1965 (2001).

76. Toxicogenetics is the study of the relationship between innate genetic makeup and susceptibility to the effects of toxic substances. *See, e.g.*, Farr & Dunn, *supra* note 75, at 1-2; Nuwaysir et al., *supra* note 75, at 158. Put another way, toxicogenetics might be viewed as a search for biomarkers of genetic susceptibility and an understanding of their mechanisms of action.

3. *Emerging fields: New molecular tapestries*

As new observational techniques evolve, scientists are testing for suites of interrelated biological changes rather than changes to the genome alone.[77] Beyond genomics, developments in "proteomics" (study of proteins),[78] "metabonomics" (metabolism),[79] "epigenetics" (non-genetic processes that activate genes),[80] and molecular imaging[81] seek to provide holistic portraits of the molecular mechanisms of disease. Studied in combination, these multidimensional suites of biomarkers may reveal new clues as to when inchoate risks transform into disease.[82]

---

77. *See* Mark Gerstein et al., *Integrating Interactomes*, 295 SCI. 284, 285 (2002). This need for linkage of genetic changes with other biological processes has long been recognized and is a major focus of contemporary research.

78. Proteomics is the study of proteins in biological systems. *See* Marc R. Wilkins et al., *Progress with Proteome Projects: Why All Proteins Expressed by a Genome Should be Identified and How To Do It*, 13 BIOTECH. & GENETIC ENGINEERING REV. 19, 20 (1995) (defining the proteome as the "entire PROTein complement expressed by a genOME"); s*ee also* Peter James, *Protein Identification in the Post-Genome Era: The Rapid Rise of Proteomics*, 30 Q. REV. BIOPHYSICS 279, 284 (1997). At present, proteomic methods are less developed than are genomic techniques, but the field is developing rapidly. *See* N. Leigh Anderson et al., *Proteomics: Applications in Basic and Applied Biology*, 11 CURRENT OPINION IN BIOTECH. 408 (2000) (expecting proteomics to follow genomics as the new dominant technology in biology for the next decade).

79. Metabonomics is the study of chemical metabolism, or the biological breakdown of chemicals, using techniques that permit observation of tissue-wide patterns of metabolites. Michael D. Waters et al., *Toxicogenomic Approach for Assessing Toxicant-Related Disease*, 544 MUTATION RES. 415, 418 (2003). Importantly, scientists are recognizing that metabolic changes may reveal more about the presence of disease than patterns of gene expression. As one scientist has noted, "Metabolic changes are real-world end points, whereas gene expression changes are not; [gene expression changes] merely indicate the potential for an end-point change." Jeremy K. Nicholson et al., *Metabonomics: A Platform for Studying Drug Toxicity and Gene Function*, 1 NATURE REVS. DRUG DISCOVERY 153, 153 (2002).

80. So-called "epigenetic" processes are non-genetic, cellular processes that can influence the expression and function of genes. These biological processes, which may "turn on" or "turn off" the "switches" that activate genes, may be heritable even in the absence of any genetic mutation. Alan P. Wolffe & Marjori A. Matzke, *Epigenetics: Regulation Through Repression*, 286 SCI. 481, 481 (1999) ("Epigenetics is the study of heritable changes in gene expression that occur without a change in DNA sequence."); *see also* Judith G. Hall, *Epigenetics Is Here to Stay*, 147 J. PEDIATRICS 427 (2005); Bob Weinhold, *Epigenetics: The Science of Change*, 114 ENVTL. HEALTH PERSP. A160, A163 (2006).

81. Recent developments in molecular imaging technology permit direct visual examination of gene and protein activity in response to drugs and chemicals. *See, e.g.*, Harvey R. Herschman, *Molecular Imaging: Looking at Problems, Seeing Solutions*, 302 SCI. 605, 605-06 (2003); *Molecular Imaging: Diagnosing Diseases Before Symptoms Strike*, *supra* note 81 (describing efforts "to track molecular events in the body to diagnose disease long before symptoms appear and to predict the effectiveness of drug therapies").

82. While this Article uses "genomics" as a shorthand, it is important to recognize that these technologies are moving well beyond the gene, to the protein and cellular metabolism levels.

Indeed, we are entering an age of "molecular epidemiology,"[83] in which individual biological evidence of exposure, risk, and developing disease[84] increasingly will supplement traditional, population-based estimates of exposure and disease risk.[85] The science of epidemiology, historically the study of health effects in populations,[86] must now evaluate and incorporate new kinds of direct biological evidence—and hence individualized evidence—of toxic risk and harm. A crucial step in developing this new evidence is biomarker "validation,"[87] an extensive and rigorous process whereby each biomarker must

---

83. *See generally* MOLECULAR EPIDEMIOLOGY: PRINCIPLES AND PRACTICES, *supra* note 58.

84. As two scientists have noted,

> Epidemiologists, now and in the future, will be asked to use increasingly powerful biologic markers of exposure, disease, or susceptibility. These markers promise to . . . detect disease earlier in its natural history by identifying biological changes on increasingly smaller scales, eventually examining individual molecular perturbations. . . . Molecular epidemiology . . . presents the opportunity to use a new resolving power in the assessment of exposure-disease relationships. [This] resolving power . . . can provide new approaches to research, prevention, and intervention.

Paul A. Schulte & Frederica P. Perera, *Preface to* MOLECULAR EPIDEMIOLOGY: PRINCIPLES AND PRACTICES, *supra* note 58, at xvii, xvii.

85. Traditional epidemiology allows an investigator to calculate and compare rates of disease within exposed and non-exposed populations. A population exposed to a certain dose of a hazardous substance generally will show a greater incidence of disease than that population would have shown absent the exposure. This comparison of rates is the relative risk: the risk in the exposed population relative to the risk in the non-exposed population. Gerald W. Boston, *A Mass-Exposure Model of Toxic Causation: The Content of Scientific Proof and the Regulatory Experience*, 18 COLUM. J. ENVTL. L. 181, 234-35 (1993).

86. Epidemiologists generally are concerned with the causes, prevention, and control of disease in populations. *See id.* at 231 ("Epidemiology is the study of the distribution and determinants of disease in human populations."); Schulte, *supra* note 63, at 10 ("Epidemiology is the study of health effects in groups of people."). The introduction of molecular biology techniques into this field in the 1980s has augmented the traditional focus, as these techniques may permit more direct observation of environmental exposures, effects, and susceptibilities on an individualized basis.

87. Before molecular and genetic biomarkers can be ready for practical application, they must first be "validated." *See generally* Paul A. Schulte, *The Use of Biomarkers in Surveillance, Medical Screening, and Intervention*, 592 MUTATION RES. 155, 157 (2005) ("Validation is a required step in the continuum that brings the biomarker from the laboratory to the field."). There is extensive work to be done in this area. *See, e.g.*, Grodsky, *supra* note 59, at 187-89 (discussing the challenges inherent in validating molecular and genetic biomarkers). The term "validation" can be taken to have different meanings. From an analytical perspective, validation refers to the process of establishing that a given test responds when a biomarker is present but not when it is absent. From a clinical perspective, validation refers to the probability that a particular clinical effect will become manifest in a person with a given biomarker. For purposes of this Article, I am concerned primarily with clinical validation and will use the term "validation" to refer to such. While some biomarkers have been deemed to be validated and ready for use in legal or regulatory settings, the discovery of candidate biomarkers has outpaced the validation process. It is essential to develop standardized protocols for validating biomarkers, and to guard against premature use or misuse of biomarker information. For discussions of the validation process, see, for

be evaluated for its accuracy and reliability as a measure of exposure, risk, or harm.[88]

C. *Implications: Challenging the Risk-Injury Divide*

Over time, new genomic technologies will permit us to identify an expanding progression of biological effects between chemical exposure and fully developed disease. For those effects that can be validated, we may expect new legal claims by plaintiffs who are classified as unimpaired yet show signs of exposure, enhanced risk, or nascent disease.

The concept of physical injury, already a tenuous standard for assigning legal rights and remedies, will become even more unsettled as science gazes into cells to observe the mechanisms of toxicity at the subcellular level. Although there will be scores of molecular signs that do not portend illness, new technologies will expand considerably the number of signs of disease beyond those detectable using traditional diagnostic techniques. Technological advancements will prompt us to reconsider exactly where in the exposure-disease continuum remediable harm has occurred.

At the same time, as new molecular biomarkers supplant the conventional "black box" model, one must ask whether there is any principled way to distinguish those who are "injured" from those who are merely "at risk." Although courts struggle to apply these labels, science will challenge this longstanding legal dichotomy. In the future, as science opens up new possibilities for earlier medical intervention, society will need to reconsider whether a legal system whose remedies depend on unclear and perhaps outmoded notions of "physical injury"—late-stage symptoms and "actual loss" or damage[89]—continues to reflect sound science or appropriate legal policy.

II. EXPLORING THE RISK-INJURY DIVIDE

The tenuous distinction between risk and injury is evident in case law dealing with DNA damage and other presumptively non-impairing conditions,

---

example, Paul A. Schulte & Frederica Perera, *Validation*, *in* MOLECULAR EPIDEMIOLOGY: PRINCIPLES AND PRACTICES, *supra* note 58, at 80; A. Aitio & A. Kallio, *Exposure and Effect Monitoring: A Critical Appraisal of Their Practical Application*, 108 TOXICOLOGY LETTERS 137, 142 (1999); DeCaprio, *supra* note 61, at 1846 (discussing the lengthy process of translating into practical application research findings of a new scientific paradigm); *see also infra* note 245 (providing a general discussion of the role of *Daubert* and *Joiner* in barring premature use of scientific evidence in the courtroom).

88. *See infra* note 157 (discussing the process of confirming associations between biomarkers and disease).

89. *See* discussion *infra* Part III.B.

such as pleural plaques resulting from asbestos exposure.[90] Although the law addressing subcellular damage is scant—and has not yet dealt with the discoveries of "whole genome" research[91]—jurisdictions differ as to whether such damage is injurious. While many courts exclude subcellular evidence as a matter of law,[92] others have ruled otherwise or have left the matter for juries to decide.[93] The U.S. District Court for the District of Minnesota, for example, refused to bar such evidence from the jury:

---

90. For example, courts remain divided as to whether asbestos-related pleural plaques are themselves compensable personal injuries or sufficient predicates for recovery for risk, fear, or monitoring. *See, e.g.*, Parker v. Brush Wellman, Inc., 420 F. Supp. 2d 1355, 1362 (N.D. Ga. 2006) ("The issue of whether such conditions [pleural plaques] constitute an 'injury' . . . remain[s] unsettled in the broader body of tort law."); *see also* Howell v. Celotex Corp., 904 F.2d 3, 5 (3d Cir. 1990) (holding that the question of whether plaques are legally compensable present injuries remained with the trier of fact because medical experts disagreed on the issue); Caterinicchio v. Pittsburgh Corning Corp., 605 A.2d 1092, 1096-97 (N.J. 1992) (same); *see also, e.g.*, Amchem Prods., Inc. v. Windsor, 521 U.S. 591, 631 (1997) (Breyer, J., concurring in part, dissenting in part) (recognizing that the "harmfulness" of "pleural thickening and plaques . . . is apparently controversial"). *Compare* Herber v. Johns-Manville Corp., 785 F.2d 79, 81 (3d Cir. 1986) (treating pleural thickening as a physical injury in the context of claim for medical monitoring); Joyce v. A.C. & S., Inc., 785 F.2d 1200, 1205 (4th Cir. 1986) (finding that asymptomatic pleural thickening constituted injury), *and* Brennan v. Owens-Corning Fiberglas Corp., 10 P.3d 749, 751 (Idaho 2000) (determining that presence of pleural plaques or scarring was evidence of "actual injury"), *with* Bowerman v. United Illuminating, No. X04CV 940115436S, 1998 WL 910271, at *8-12 (Conn. Super. Ct. Dec. 15, 1998) (denying claims for medical monitoring, enhanced risk, and mental distress on the ground that alleged injuries from asbestos exposure, including implantation of asbestos fibers and pleural plaques, did not amount to separately compensable harms).

91. The limited body of case law dealing with cellular and genetic effects of toxic chemicals has not yet addressed the new generation of subcellular data generated by microarrays and other emerging technologies that permit high-speed, high volume, and genome-wide analyses of toxic responses.

92. *See, e.g.*, *Parker*, 377 F. Supp. 2d at 1306 (concluding that Georgia courts would not recognize "sub-clinical, cellular, and sub-cellular" effects as actionable "injuries" as a matter of law); Caputo v. Boston Edison Co., No. 88-2126-Z, 1990 WL 98694, at *4 (D. Mass. July 9, 1990) (excluding cellular-level damage as a matter of law in case involving radiation-induced chromosomal damage and claims for physical injury and emotional distress).

93. *See, e.g.*, Werlein v. United States, 746 F. Supp. 887, 901 (D. Minn. 1990) (leaving question of subcellular injury for the jury in response to claims for mental distress and enhanced risk), *vacated in part on other grounds*, 793 F. Supp. 898 (D. Minn. 1992); *see also* Sterling v. Velsicol Chem. Corp., 855 F.2d 1188, 1206 (6th Cir. 1988) (finding cellular damage sufficient to support a claim for emotional distress); Anderson v. W.R. Grace & Co., 628 F. Supp. 1219, 1228 (D. Mass. 1986) (rejecting defendants' contentions that cellular or subcellular damage could not support parasitic damages for mental distress); Brafford v. Susquehanna Corp., 586 F. Supp. 14, 18 (D. Colo. 1984) (holding, in response to a request for recovery for chromosomal damage and increased risk of cancer due to radiation exposure, that a genuine issue of material fact existed as to whether subcellular damage constituted present injury); Bryson v. Pillsbury Co., 573 N.W.2d 718, 720-21 (Minn. Ct. App. 1998) (requiring, in a case involving chromosomal aberrations from toxic exposure, a

> Based on the record before it, this Court cannot rule as a matter of law that plaintiffs' alleged injuries are not "real" simply because they are subcellular. The effect of volatile organic compounds on the human body is a subtle, complex matter. It is for the trier of fact, aided by expert testimony, to determine whether plaintiffs have suffered present harm.[94]

The status of subcellular damage will gain new urgency as high-throughput technologies identify molecular-level biomarkers at a speed and scale unimaginable in the past. This Part examines principles emerging from case law dealing with DNA and other subclinical damage, providing examples from asbestos cases where relevant. I suggest that two competing paradigms undergird these decisions, and offer these models as alternate conceptual frameworks for thinking about the implications of the genomic revolution for the future of tort law. The first I will call the "de minimis effects" or "reversible effects" model; and the second, the "significantly enhanced risk" or "diseased state" model. These models overlap, in that they both may acknowledge the predictive capacity of certain subclinical markers. But the former would treat risk (or early injury) as legally inconsequential, while the latter would—at the very least—preserve the question for expert debate.[95]

---

present physical injury for medical monitoring but permitting trier of fact to determine whether chromosome breakage constituted present injury).

94. *Werlein*, 746 F. Supp. at 901.

95. It should be noted that courts' or parties' adoption of a "de minimis effects" or "diseased state" conception may implicitly reflect the nature of the claim, but these connections often are left unarticulated. Even more salient, the determination of whether an injury has occurred is often divorced from any consideration of the requested remedy. Put another way, one source of current confusion in the law's treatment of subclinical effects is that many decisions fail to articulate whether or how the nature of the requested remedy should affect the definition of physical injury. For example, some courts treat requests for mental distress or medical monitoring as though they were requests for compensatory damages, requiring harm in the form of "actual loss" or damage prior to recovery. *See, e.g.*, Macy's Cal., Inc. v. Superior Court, 48 Cal. Rptr. 2d 496, 503 (Cal. Ct. App. 1996) (dismissing claim for mental distress on the ground that subcellular evidence does not represent "actual damage," "harm," or "detrimental change to the body"). Others do not equate the predicate physical injury with traditionally compensable injuries. *See, e.g.*, *Werlein*, 746 F. Supp. at 906 (rejecting defendants' assertion that subcellular harm is insufficient to support mental distress damages, although noting that several courts have held that such damage is insufficient); Merry v. Westinghouse Elec. Corp., 684 F. Supp. 852 (M.D. Pa. 1988) (finding subcellular changes sufficient to support mental distress damages). In the monitoring example, requiring physical injury to rise to the level of harm in the form of "actual loss" may signal that the court refuses to treat monitoring as an independent tort; thus, plaintiffs must prosecute separately compensable injuries prior to recovery. Under this view, medical surveillance is part of a traditional tort remedy rather than a stand-alone cause of action. *See, e.g.*, Henry v. Dow Chem. Co., 701 N.W.2d 684, 691 (Mich. 2005) (noting that plaintiff must be prosecuting a separately compensable injury prior to recovering for monitoring). Further complicating the picture, some courts suggest that actionable harm in tort need not have reached its fullest manifestation for a cause of action to accrue. *See, e.g.*, Goodall v. United Illuminating, No. X04CV 950115437S, 1998 WL 914274, at *3 (Conn.

A. *"De Minimis Effects" or "Reversible Effects" Model*

The "de minimis effects" model treats subclinical damage as legally inconsequential. This model presumes that overt clinical symptoms—such as tumor formation or major organ dysfunction—provide an appropriate line of demarcation between risk and injury, health and disease. It follows that the law should not recognize toxic effects as "injurious" or harmful prior to the manifestation of medical symptoms. In cases where subcellular changes or other asymptomatic conditions are recognized as damaging to some degree, such changes are considered insufficiently serious, harmful, or detrimental to trigger legal remedies.[96] Moreover, subclinical damage may be invisible to the naked eye and thus considered subjective and speculative.[97] This model

---

Super. Ct. Dec. 15, 1998). This might suggest that subcellular damage could serve as a predicate to monitoring even under the above formulation.

Other courts appear to require physical injuries as screening devices for separating valid from speculative monitoring claims—hence, something less than an independently compensable injury would suffice. *See, e.g.*, Bocook v. Ashland Oil, Inc., 819 F. Supp. 530, 537 (S.D. W. Va. 1993) (distinguishing a monitoring claim from a personal injury claim, and noting that "[i]t is apparent that the Kentucky Supreme Court requires proof of *some* present physical harm, however slight"). This conception is well suited to the preventive aspect of monitoring, yet adds to the current doctrinal confusion as courts treating monitoring as an independent tort presumptively would not need to discuss physical injury at all. *See supra* Part I. Indeed the *Bocook* court, while generously citing *Paoli I*—which eliminated physical injury as a precondition of monitoring recovery—nevertheless required plaintiffs to show some form of physical injury prior to recovery. *Bocook*, 819 F. Supp. at 537 (stating that "proof of exposure alone" is insufficient) (emphasis removed).

96. This distinction builds on the Second Restatement's definition of "harm" as "loss or detriment to a person, and not a mere change or alteration in some physical person." RESTATEMENT (SECOND) OF TORTS § 7 cmt. b (1979); *see also* RESTATEMENT (THIRD) OF TORTS: LIABILITY FOR PHYSICAL HARM § 4 cmt. c (Proposed Final Draft No. 1, 2005) (drawing upon the Second Restatement and explaining that "physical harm" requires a detrimental change rather than "simply a change in the condition of the plaintiff's body"). For example, as noted, even for recovery of mental distress damages, several courts have treated subclinical impacts as inconsequential on the theory that they do not represent "detrimental changes to the body" or "harm." *See, e.g.*, *Macy's Cal., Inc.*, 48 Cal. Rptr. 2d at 503. This reasoning is also apparent in certain asbestos cases involving microscopic damage to lung tissues or scarring from asbestos exposure. *See, e.g.*, *In re* Haw. Fed. Asbestos Cases, 734 F. Supp. 1563, 1567 (D. Haw. 1990) (refusing to recognize pleural plaques as sufficient predicates for recovery for mental distress damages, instead requiring an underlying compensable "harm," in the form of functional impairment, prior to recovery); Bowerman v. United Illuminating, No. X04CV 940115436S, 1998 WL 910271, at *8-12 (Conn. Super. Ct. Dec. 15, 1998) (denying plaintiffs' claims for medical monitoring, enhanced risk, and mental distress on the ground that alleged injuries from asbestos exposure, including implantation of asbestos fibers and pleural plaques, were not detrimental physical harms and hence failed to present a cause of action).

97. *See, e.g.*, Parker v. Brush Wellman, Inc., 420 F. Supp. 2d 1355, 1358 (N.D. Ga. 2006) (emphasizing that subcellular damage involving beryllium sensitization from beryllium exposure "cannot be observed, and is invisible to the eyes, to x-rays, and even to the microscope; indeed, it exists only at the biochemical level"). The element of subjectivity

emphasizes the myriad possibilities for adaptation and repair following subclinical insults to the body.[98] Accordingly, subclinical effects may be of limited predictive value.[99] As the First Circuit has noted:

> Every disease is presumably preceded by the onset of sub-clinical changes in the body. To state that the disease occurs when these sub-clinical alterations take place, where, as here, the disease does not inevitably or even usually result from the sub-clinical changes, is to subvert the plain meaning of "disease."[100]

Not surprisingly, courts embracing this paradigm often treat plaintiffs alleging subclinical damage as "healthy." The Sixth Circuit's decision in *Rainer v. Union Carbide Corp.*[101] is illustrative. Although plaintiffs suffered

---

was emphasized in *Caputo v. Boston Edison Co.*, No. 88-2126-Z, 1990 WL 98694 (D. Mass. July 9, 1990). Responding to plaintiffs' claims of radiation-induced physical injury at the "chromosomal cellular level" and emotional distress, the court held that "cellular damage does not rise to the level of physical injury as a matter of law because nothing in the record relates them to any *objective* symptoms of illness or disease." *Id.* at *4 (emphasis added).

98. As one commentator has noted, the characterization of the developmental process of a disease as enduring must be weighed against the reality that "the process is also marked by significant discontinuity, the lack of any 'new' injury and the great likelihood that the disease will never develop." Donald T. Ramsey, *The Trigger of Coverage for Cancer: When Does Genetic Mutation Become 'Bodily Injury, Sickness, or Disease'?*, 41 SANTA CLARA L. REV. 293, 299 (2001). In *In re Rezulin Products Liability Litigation*, the court refused to award emotional distress damages on the basis of subcellular damage to the mitochondria—the principal energy sources of cells. 361 F. Supp. 2d 268, 278 (S.D.N.Y. 2005). The court noted that future consequences were speculative because, among other things, there was no evidence that the alleged mitochondrial damage was permanent or irreversible. *Id.*

99. A presumption inherent in certain decisions requiring manifest disease prior to recovery for emotional distress is that subclinical injury is inadequately predictive of future disease to make plaintiff's fear "reasonable." For example, pleural plaques, while recognized as solid evidence of asbestos exposure, are not considered highly predictive of serious asbestos-related malignancies such as lung cancer or mesothelioma. As the federal district court noted in *In re Hawaii Federal Asbestos Cases*, "[a] reasonable person, exercising due diligence, should know that of those exposed to asbestos, only a small percentage suffer from asbestos-related physical impairment and that of the impairment group fewer still eventually develop lung cancer." 734 F. Supp. at 1570.

100. Eagle-Picher Indus., Inc. v. Liberty Mut. Ins. Co., 682 F.2d 12, 19-20 (1st Cir. 1982). In this case, an insurance adjudication involving non-impairing conditions related to asbestos exposure, the court adopted a manifestation approach. *Id.* According to the court, "disease 'results' . . . when it becomes clinically evident, that is, when it becomes reasonably capable of medical diagnosis." *Id.* at 25. Although the court recognized that asbestosis is "an injurious process that begins with the deposition of asbestos fibers in the lung, causing tiny sub-clinical 'insults' to the lung tissue," *id.* at 18, the reversible nature of the process influenced the court's adoption of a "manifestation" rather than an exposure trigger. *See also infra* note 116 and accompanying text (distinguishing definitions of bodily injury for insurance coverage purposes from definitions of compensable injury or harm for tort liability purposes).

101. Rainer v. Union Carbide Corp., 402 F.3d 608 (6th Cir. 2005). In *Rainer*, the Sixth Circuit noted that a plaintiff who had alleged extensive chromosome damage from radiation

permanent and extensive chromosomal damage from radiation exposure, the court underscored the district court's observation that plaintiffs were not "sick."[102] As a matter of policy, subscribing to a contrary view could mean that most human beings are "diseased" in some manner,[103] potentially unleashing a torrent of litigation.[104] Hence, this model clearly distinguishes the domains of latent risk and patent injury, drawing the Maginot Line at classic medical symptoms or overt impairment.

B. *"Significantly Enhanced Risk" or "Diseased State" Model*

The alternative model, which I label the "significantly enhanced risk" or "diseased state" model, presumes that although disease may be invisible to the naked eye or undetectable using traditional techniques, it may nevertheless be present and ongoing.[105] The latency period, viewed as an incubator of developing disease, may be shorter than classic medical symptoms would suggest.[106] It follows that legally relevant injury or harm may occur well prior

---

exposure also had revealed in a recent medical examination that she exhibited no "problems of any kind." *Id.* at 612.

102. *Id.* at 621. The court's emphasis on lack of physical impairment, however, may be attributable in part to the fact that *Rainer* involved a claim for compensatory damages, in the absence of any "risk-based" claims. *See infra* Part III. Courts rarely discuss whether or how the nature of the requested remedy should affect the definition of "present physical injury."

103. One scientist has noted that labeling all phases in the development of cancer as disease would mean that almost half of the population would be considered diseased or injured throughout most of their lives. Injury and disease, under such a definition, would be considered universal and a normal aspect of being alive. Ramsey, *supra* note 98, at 298. An early case involving a longshoreman's alleged subclinical damage from breathing grain dust reflects this view. In *Grain Handling Co. v. Sweeney*, 102 F.2d 464 (2d Cir. 1939), where the court held that an industrial disease under the Longshoreman's Act did not "occur" until manifestation, Judge Learned Hand noted:

> Few adults are not diseased, if by that one means only that the seeds of future trouble are not already planted; and it is a common place that health is a constant warfare between the body and its enemies: an infection mastered, though latent, is no longer a disease, industrially speaking, until the individual's resistance is again so far lowered that he succumbs.

Id. at 466.

104. This is no small matter. The floodgates problem was one of the *Rainer* court's central policy rationales for refusing to treat subcellular damage as a present injury. *Rainer*, 402 F.3d at 621 ("Accepting the plaintiffs' claim would therefore throw open the possibility of litigation by any person experiencing even the most benign subcellular damage."). For a discussion of the floodgates issue in the context of medical monitoring, see *infra* Part III.E.

105. For example, in *Barth v. Firestone Tire & Rubber Co.*, in which plaintiffs were exposed to benzene, other heavy metal compounds, and other industrial toxins, the complaint alleged that serious diseases were present in the plaintiff in their "latency stage." 661 F. Supp. 193, 196 (N.D. Cal. 1987).

106. Consistent with this perspective on latency, two commentators have observed that "[l]atency is, simply, dormancy, and dormancy . . . refers to a condition in which a harmful agent, or the beginning of disease itself, is present but invisible." Heinzerling & Hoffman, *supra* note 29, at 88.

to manifestation of classic symptoms. Certain subclinical changes may represent disease itself, or a substantial probability that full-fledged disease or impairment will develop.

Reflecting this conception, plaintiffs' expert in *Rainer* argued that "[r]adiation damage to chromosomes is the quintessential determinant of altered physiologic function because our chromosomes control each and every bodily function . . . . As such this premorbid state is disease."[107] That expert analogized such damage to infection with HIV: "[P]atients who test positive for the HIV virus may not have any signs or symptoms of clinical disease for many years . . . . [But] even though a person with HIV does not have 'clinical disease' they are clearly in a diseased state."[108]

The diseased state conception would treat as artificial any a priori distinction between cellular and gross harm. Hints of this thinking emerged in *Anderson v. W.R. Grace & Co.*,[109] the well-known toxic tort case chronicled in the best-selling book and movie, *A Civil Action*.[110] Among other claims, residents of Woburn, Massachusetts alleged subcellular damage from ingesting heavily contaminated drinking water.[111] Rejecting defendants' contentions that such damage was insufficient to support a claim for emotional distress,[112] the court emphasized that the law did not distinguish between gross and subcellular harm.[113] Rather, the law distinguished between "harm which is merely speculative or based solely on a plaintiff's unsupported assertions" and harm that could be "objectively evidenced" through expert medical testimony.[114] As plaintiffs could support their allegation of subcellular damage through expert medical testimony, there was sufficient evidence to preclude summary judgment.[115]

---

107. *Rainer*, 402 F.3d at 613.

108. *Id.*

109. 628 F. Supp. 1219 (D. Mass. 1986).

110. JONATHAN HARR, A CIVIL ACTION (1995).

111. *See Anderson*, 628 F. Supp. at 1226-27.

112. *Id.* at 1226.

113. *Id.* at 1227.

114. *Id.* According to the court:

> The phrase "manifested by objective symptomatology" does not indicate that the necessary harm need be immediately apparent but that its existence must be objectively evidenced. Where, as in this case, the harm is not obvious to the layman, its existence may not be demonstrated solely by the complaints of the alleged victim; it must also be "substantiated by expert medical testimony." . . . I cannot say as a matter of law that this standard will not be met at trial.

Id.

115. *Id. Anderson v. W.R. Grace* thus directly challenged the notion that subcellular damage is presumptively subjective or speculative. In a similar vein, a leading appellate decision in the regulatory sphere questioned the logic of distinguishing, a priori, between clinical and subclinical effects. The D.C. Circuit's 1980 decision in *Lead Industries* clarified that subclinical events may be defined as adverse for regulatory purposes:

The diseased state conception finds interesting parallels in the "exposure," "injury-in-fact," and "exposure in residence" theories applied in cases adjudicating insurance coverage for progressive diseases.[116] The "exposure" theory recognizes that disease may be present, and hence coverage may begin, before disease is "discovered."[117] Courts applying this theory do not necessarily presume that exposure constitutes injury in itself, but rather acknowledge that exposure may cause immediate tissue damage—and that such damage may be injurious even if not immediately manifested.[118] Similarly, the "injury-in-fact" trigger posits that some forms of injury to the body or loss may occur prior to the appearance of symptoms.[119] The "exposure in residence" theory conceives of injury or disease as an iterative process. It maintains that subsequent to exposure but prior to appearance of classic symptoms, the body's

---

> [T]he clinical/subclinical distinction has little to do with the question whether a particular effect is properly viewed as adverse to health. Rather, the distinction pertains to the means through which the particular effect may be detected: observation or physical examination in the case of clinical effects, and laboratory tests in the case of subclinical effects. Thus describing a particular effect as a "subclinical" effect in no way implies that it is improper to consider it adverse to health.

Lead Indus. Ass'n v. EPA, 647 F.2d 1130, 1158 (D.C. Cir. 1980) (upholding EPA's standard for airborne lead, which was based on a subclinical effect—elevated levels of a protein in the blood).

116. Litigation between manufacturers and insurers often requires courts to construe provisions of the insurance policy to determine when coverage is triggered. Such cases are instructive because the central question in such litigation is when bodily injury or disease "results." This task is particularly difficult when undertaken in the case of a progressive disease. Courts have advanced a variety of theories for determining when bodily injury can be deemed to have occurred. These include the exposure theory, exposure-in-residence theory, injury-in-fact theory, manifestation theory, and the continuous or multiple trigger theory. *See* AMERICAN LAW OF PRODUCTS LIABILITY § 58:33-58:51 (3d ed. 2007).

It is important to note that the definition of bodily injury for insurance coverage purposes may differ from the definition of compensable injury or harm for tort liability purposes, and the former may vary depending on the insurance policy language. *See, e.g.*, Am. Home Prods. Corp. v. Liberty Mut. Ins. Co., 748 F.2d 760, 765 (2d Cir. 1984) ("[C]ompensability is a legal concept that is not material to the determination of whether an injury has in fact occurred."); *see also* AMERICAN LAW OF PRODUCTS LIABILITY, *supra*, § 58:48 ("Injury-in-fact, however, does not mean injury that is 'diagnosable' or 'compensable' during the policy period. . . . That determination must be made according to the facts of the particular case based on medical evidence as to the product and what type of injury was claimed."). However, the insurance cases are instructive in their elucidation of the mechanisms of disease.

117. *See, e.g.*, Ins. Co. of N. Am. v. Forty-Eight Insulations, Inc., 633 F.2d 1212, 1218-19 (6th Cir. 1980). The court noted that regardless of whether the microscopic tissue damage that occurs upon initial inhalation of asbestos fibers should be defined as a disease or bodily injury, "there is universal medical agreement that the time when asbestosis manifests itself is not the time when the disease occurred. No doctor would say that asbestosis occurred [only] when it was discovered." *Id.* at 1219.

118. AMERICAN LAW OF PRODUCTS LIABILITY, *supra* note 116, § 58:38.

119. This theory recognizes that an injury-in-fact can occur at different points of time along the continuum from initial exposure to manifestation.

efforts to resist, adapt, and accommodate to a foreign matter are, in themselves, part of the disease process.[120] This view assumes that, at some preclinical point, biological processes are set in motion that have a significant probability of resulting in a covered loss.[121]

Hence, implicit in the "diseased state" conception is the recognition that bodily integrity may be compromised prior to the appearance of classic symptoms. The point at which risk becomes injury or disease may be far more nuanced than traditional diagnostic techniques can reveal.

C. *Genomics and the Risk-Injury Divide*

As technology generates new biomarkers at an unprecedented rate, the question of what constitutes a "physical injury" will become even more compelling—and more controversial. Adding fuel to the fire, new genomic data may lend support to *both* the "de minimis effects" and the "diseased state" conceptions, as parties, courts, and experts debate the meaning of these subtle biological effects.

1. *Genomics and "de minimis effects"*

To be sure, many genetic and molecular responses are benign, insufficiently specific, or, at most, evidence of exposure or enhanced risk—in any event, insufficient to constitute legally relevant "injury." And indeed, it is a given that not all exposure-related biological changes imply toxicity. A central question is which biomarkers represent deleterious effects of a poison and which represent benign biological changes or even adaptive responses to fight the poison.[122] In many cases, genetic damage alone will not constitute injury

---

120. *See, e.g.*, Keene Corp. v. Ins. Co. of N. Am., 667 F.2d 1034, 1046 (D.C. Cir. 1981) (adopting a multiple trigger approach that included exposure in residence).

121. When it becomes known that an occurrence has set in motion a process that has a significant probability of resulting in a covered loss, either through direct observation or statistical inference, the insurer must bear the risk. *Id.* at 1046. Otherwise, the insurer could shift a covered loss back to the insured by terminating coverage during the interim period between exposure and manifestation. *Id.* at 1046-47. However, the exposure in residence theory recognizes that there is often no medical certainty as to when diseases "occur":

> This is a case of first impression and, irrespective of how it is resolved, requires a "leap of logic" from existing precedent, for it concerns diseases about which there is no medical certainty as to precisely how or when they "occur." We do know the prerequisite—exposure to asbestos fibers—and the symptoms that manifest themselves, generally too late for effective treatment. What happens in between is still something of a mystery; why does one exposed person fall victim to the diseases while another does not?

Id. at 1057 (Wald, J., concurring in part) (internal citations omitted).

122. *See, e.g.*, Aardema & MacGregor, *supra* note 72, at 18; Mark R. Fielden & Tim R. Zacharewski, *Challenges and Limitations of Gene Expression Profiling in Mechanistic and Predictive Toxicology*, 60 TOXICOLOGICAL SCI. 6, 7 (2001) ("The challenge will be to distinguish the therapeutic affects [sic] from the pathological changes.").

because events beyond the genome—including protein activity and metabolic processes—are critical to the disease process.[123] Put another way, the journey from genetic damage to disease may be attenuated. Even where the genetic role is dominant, disease pathology is a multi-stage process—often requiring a series of mutations in order to progress to dysfunction.[124]

In addition, many subcellular events, including changes in gene expression, may be transient, reversible, or otherwise unstable.[125] Even where genetic

---

123. For example, the author of a study involving genotoxicity in marine invertebrates recognized that, likely for all animals, "genotoxic agents rarely damage only DNA." M.H. Depledge, *The Ecotoxicological Significance of Genotoxicity in Marine Invertebrates*, 399 MUTATION RES. 109, 110 (1997). Because genetic damage may work in tandem with metabolic damage that occurs as a result of exposure to the same chemical, courts would be well advised to focus on metabolic changes as well. Commentators arguing against "genetic reductionism" in environmental research have supported the current integrated approach that incorporates data from proteomics, metabonomics, and physiological studies. *See* David E. Adelman, *The False Promise of the Genomics Revolution for Environmental Law*, 29 HARV. ENVTL. L. REV. 117, 161-68 (2005) (discussing the promise and limitations of contemporary genomics methods, cautioning against genetic reductionism, and supporting current methods that integrate functional analyses of genes, proteins, and their associated biological processes); Samuel M. Cohen, *Risk Assessment in the Genomic Era*, 32 TOXICOLOGIC PATHOLOGY 3, 5-6 (Supp. 1) (2004) (recognizing the significance of toxicogenomic methods for risk assessment, and emphasizing the necessity of complementary biochemical and epidemiological techniques). The importance of integration has long been recognized and is a major focus of contemporary research. *See, e.g.*, Adelman, *supra*, at 160-61 ("These methods may offer another view of biological mechanisms important to toxicology and essential to toxicogenomic methods."); *id.* at 168 ("The great potential of toxicogenomics resides . . . in the suite of methods it will bring to bear on functional analyses of genes, proteins, and their associated biological processes.").

124. For example, scientists have described the development of cancer as "a multi-stage process that begins with the mutation of a gene in a single cell." Ramsey, *supra* note 98, at 293-94. According to Ramsey, four to seven key genes (those that control the cell's growth and repair functions) must mutate before a malignant tumor develops, *id.* at 294, and some of the damage induced through mutation may be repaired:

> Unlike the [continuing] presence of asbestos fibers in the lung, genetic damage induced by mutagens, is not, strictly speaking, "irrevocable." Genetic damage, if minor, may be repaired. . . . [D]epending upon the type of particular cancer, the terms "injury" and "disease" fail properly to describe all phases in the complex etiology of cancer, which, especially in its long "early" phase of development is "a dynamic process that may stop temporarily, or permanently, revert to normality, or progress to frank [disease]."

Id. at 298 (internal citations omitted). "Indeed, the development of cancer is at once both too rare and too common, too normal and too exceptional, for all stages of the entire process comfortably to bear the designation 'bodily injury, sickness, or disease.'" *Id.*

125. *See, e.g.*, Stefano Bonassi & William W. Au, *Biomarkers in Molecular Epidemiology Studies for Health Risk Prediction*, 511 MUTATION RES. 73, 76 (2001) ("For many types of biomarkers the most important consideration is the stability of the biomarkers with respect to time after the exposure."). Current toxicogenomics techniques focus primarily on gene expression rather than gene mutation, and scientists have cautioned against the treatment of all gene expression changes as adverse effects:

> The biological relevance of gene expression changes . . . must be defined with respect to whether the gene(s) is pivotal in the pathway for a toxicity, if the change is reversible, and if

mutations occur, molecular buffering mechanisms may compensate for potential adverse consequences.[126] Because our DNA has significant error-correcting ability, one might emphasize the myriad possibilities for adaptation and repair following subclinical insults to the body and the related conclusion that subcellular markers are of limited predictive value. In addition, although scientists hope to "fingerprint" particular chemical "culprits" by linking them to telltale molecular responses,[127] the discovery of unique genetic fingerprints is proceeding slowly.[128]

In short, the coming inundation of new and highly complex information could prove so incomplete, overwhelming, and controversial that courts further embrace the relative clarity of traditional medical symptoms, hence reinforcing the conventional risk-injury divide.

> the change in gene expression leads to altered cell or tissue function. It is important to guard against the temptation to classify every gene/protein expression change as adverse, as many changes will be . . . reversible.

Aardema & MacGregor, *supra* note 72, at 19. The American Thoracic Society has underscored this problem: "We do not know if elevations of biomarkers during short-term experimental exposures signal risk for ongoing injury and clinical effects or simply indicate transient responses that can provide insights into mechanisms of injury." Am. Thoracic Soc'y, *supra* note 60, at 669.

126. *See, e.g.*, Adelman, *supra* note 123, at 138 (discussing the molecular buffering of effects of certain mutations); Suzanne L. Rutherford, *Between Genotype and Phenotype: Protein Chaperones and Evolvability*, 4 NATURE REV. GENETICS 263, 263-64 (2003) (same).

127. Ongoing research is based on the premise that exposure to a particular class of chemicals may be established by the unique gene expression pattern—known as a "gene signature" or "gene fingerprint"—that the particular class of chemical induces. *See, e.g.*, Gary E. Marchant, *Genomics and Toxic Substances: Part II—Genetic Susceptibility to Environmental Agents*, 33 ENVTL. L. REP. 10641, 10651 (2003); Aardema & MacGregor, *supra* note 72, at 17; Hisham K. Hamadeh et al., *Gene Expression Analysis Reveals Chemical-Specific Profiles*, 67 TOXICOLOGICAL SCI. 219 (2002). The ability to correlate a signature response with exposure to a particular chemical or class of chemicals could suggest a relationship to subsequent disease. The capacity to monitor genes globally is hoped to assist in the identification of signature patterns. Hamadeh et al., *supra*, at 219; s*ee also* Aardema & MacGregor, *supra* note 72, at 16 (discussing the implications of global monitoring of gene expression). Gene expression patterns are not the exclusive form of genetic signature. Traditional biomarkers such as DNA adducts and signature mutations may in some cases reveal the identity of particular chemicals through their effects on individual genes. *See, e.g.*, M. Prakash Hande et al., *Past Exposure to Densely Ionizing Radiation Leaves a Unique Permanent Signature in the Genome*, 72 AM. J. HUM. GENETICS 1162, 1167 (2001) (concluding that certain unique and dense chromosomal aberrations in workers exposed to plutonium represented an "unequivocal biomarker of densely ionizing radiation exposure in a human population exposed many years earlier").

128. *But see infra* note 155 (discussing the fact that "signature" markers are helpful, although not essential, for establishing causal relationships in tort law); *see also infra* note 156 (illustrating how markers of ongoing disease, even if not "chemical-specific" markers, may be useful to tort plaintiffs when combined with traditional evidence of exposure).

2. *Genomics and the "diseased state" conception*

On the other hand, as time passes and technology advances, genomic tools may provide new ammunition for the diseased state view. In certain cases, the simple distinction between clinical and subclinical injuries will cease to hold, as technology enables ever more nuanced and multidimensional insights into the effects of toxic substances on the body. Should the molecular-genetic revolution further uncouple the current risk-injury divide from its scientific foundations, the argument can be made that certain subcellular events should be moved from the "risk" to the "injury" column.[129]

a. Objectivity and permanence

It is a given that many subclinical events, once considered invisible and thus speculative, will become detectable and hence objectively verifiable.[130] Dan Farber has aptly characterized the problem of latent injury as a problem of imperfect information.[131] Following this logic, and that of *Anderson v. W.R. Grace*, the argument can be made that better detection should—at the very least—allow for legal consideration.[132]

Countering the view that subcellular damage is presumptively reversible, gene mutations may be permanent or highly persistent,[133] and long-lasting changes in gene expression, protein synthesis, or metabolic processes may reflect the presence of mutated genes.[134] The paradigmatic example of

129. *See infra* Part III, discussing potential use of earlier markers as predicates to medical monitoring claims.

130. *See* Gary E. Marchant, *Genomics and Toxic Substances: Part I—Toxicogenomics*, 33 ENVTL. L. REP. 10071, 10079 (2003).

131. *See, e.g.*, Daniel A. Farber, *Toxic Causation*, 71 MINN. L. REV. 1219, 1247 (1987) ("The only real difference between the automobile case and the toxics case is that better information is available about the events in the automobile case whereas the relevant biological events in the toxics case are unobservable."). Extending this reasoning to DNA damage, one could argue that, in the past, "the absence of adequate methods to detect" such damage "forced courts to make artificial distinctions" between genetic and related injury and damage to body tissues and organs. Mark S. Ellinger, *DNA Diagnostic Technology: Probing the Problem of Causation in Toxic Torts*, 3 HARV. J.L. & TECH. 31, 65 (1990).

132. Existing case law suggests that, at least in some instances, detectability could lead to legal liability. *See, e.g.*, Jackson v. Johns-Manville Sales Corp., 781 F.2d 394, 412 (5th Cir. 1986) ("[O]nce the injury becomes actionable—once *some* effect appears—then the plaintiff is permitted to recover for all probable future manifestations as well.").

133. Mutations involve changes in the DNA sequence. They are often permanent because when a cell divides, the mutation is copied and hence passed to daughter cells, unless repaired.

134. *See, e.g.*, Marchant, *supra* note 130, at 10075 (quoting Toby G. Rossman, *Cloning Genes Whose Levels of Expression Are Altered by Metals: Implications for Human Health Research*, 38 AM. J. INDUS. MED. 335, 336 (2004) (noting that certain gene expression changes may be permanent)). Likewise, new kinds of proteins may be expressed,

permanent genetic damage is that caused by ionizing radiation. Because ionizing radiation can permanently and irreversibly alter the genome,[135] such damage may represent significantly enhanced risk of disease or perhaps even a diseased state. In *Brafford v. Susquehanna*, for example, one expert linked genetic damage to the genesis of cancer, describing radiation-induced chromosome injury as present "in the sense that the 'damage has been done' and the 'trigger' of a cancer change has been cocked."[136] So-called epigenetic (beyond genetic) events may also represent permanent, heritable effects. These little-explored non-genetic processes, which can "turn on" or "turn off" the "switches" that activate genes, are fascinating in their own right as they may be heritable (and thus permanent or long-lasting) even in the absence of genetic mutation.[137] Moreover, chemical exposures may trigger "gene amplification"[138] or "gene reprogramming"[139]—which are also permanent or persistent changes.

---

or existing proteins may be over-expressed, reflecting the presence of mutated genes. Interview with David L. Eaton, Dir., Ctr. for Ecogenetics and Envtl. Health, Univ. of Wash., in Seattle, Wash. (Apr. 23, 2006) (on file with author).

135. For example, in the Hande study, workers exposed to ionizing radiation were considered to be clinically "healthy," but their genomes contained unique chromosomal changes that had persisted since their exposure five decades earlier. *See* Hande, *supra* note 127, at 1167-68. In *Brafford v. Susquehanna Corp.*, involving exposure to ionizing radiation, plaintiffs asserted their expert would testify that "to a reasonable degree of medical probability . . . plaintiffs have suffered present, permanent, and irreparable genetic and chromosomal damage as a result of their exposure to the radiation emitted from the [uranium] mill tailings." 586 F. Supp. 14, 17 (D. Colo. 1984).

136. *Id.* at 18. Plaintiffs in *Brafford* sued for chromosome damage and increased risk of cancer alleged to have resulted from exposure to radiation that was greatly in excess of regulatory standards. *Id.* The *Brafford* court refused to rule as a matter of law that chromosomal injury would not constitute a "present physical injury" in this context. The court's decision relied heavily on consensus expert opinion that indicated high levels of radiation exposure. *Id.* at 18. As a result of these significant levels, experts could agree "with a reasonable degree of medical probability" that there had been chromosomal damage and that it was caused by the radiation as opposed to other environmental carcinogens. *Id.* at 17-18.

137. *See supra* note 80. Hence, distinctive epigenetic changes may represent significantly enhanced risk of disease or even markers of existing disease.

138. Gene amplification involves multiple copying of genes. Although cells do have DNA repair mechanisms that could theoretically remove extra copies of genes occurring during amplification, so many copies may be generated during this process that they will overwhelm repair capabilities. Gene amplification is believed to play a role in cancer. *See, e.g.*, Donna G. Albertson, *Gene Amplification in Cancer*, 22 TRENDS IN GENETICS 447 (2006).

139. Recent research suggests that some chemical exposures may "reprogram" certain genes, hence affecting their function. *See, e.g.*, *Toxicology: Harmful Chemicals May Reprogram Gene Response to Estrogen*, GENOMICS & GENETICS WEEKLY, June 24, 2005, at 224 (discussing recent findings that exposure to harmful chemicals and drugs, particularly during critical developmental periods, may "reprogram" the way certain genes respond to estrogen). According to the article, "[u]ntil now, scientists thought that exposure to harmful

b. Failure to repair

Most significantly, emerging technologies may lend support to the diseased state conception through their ability to identify damage to the body's own repair functions.[140] For example, for a cancer tumor to grow, tumor suppressor genes must be "turned off." If one can show that a gene mutation or another process[141] has destroyed the function of a tumor suppressor gene, the probability of cancer escalates. If copies of the tumor suppressor gene similarly are lost, the likelihood of disease may increase exponentially.[142]

Further illustrating the centrality of repair functions, so-called "anti-oncogenes" are thought to combat uncontrolled cell growth and thus to protect against cancer.[143] Deletion or mutational inactivation of these biological warriors "could cause cells to proliferate out of control."[144] As such, these discernable subclinical events may represent a significantly enhanced risk of

---

agents in the environment caused damage to the gene. This study, however, indicates that an environmental agent can actually change or reprogram the gene so that it functions differently." *Id.* If such reprogramming can be linked to particular classes of chemicals, these modifications arguably could provide more durable markers of exposure, risk, or disease.

140. As one scientist has observed, if there is an adverse effect, the biological system either repairs itself or it does not. The failure to adapt or repair is then seen as a form of permanent injury. Snyder, *supra* note 21, at 902. Interestingly, molecular imaging technologies can now observe proteins that pump certain anti-cancer drugs out of tumor cells, hence identifying how therapies may fail. *Molecular Imaging: Diagnosing Diseases Before Symptoms Strike*, *supra* note 81.

141. For example, in addition to gene mutations, DNA "adducts" or epigenetic processes may destroy the function of a tumor suppressor gene. Adducts are formed when chemicals bind directly to DNA (creating "chemical-DNA adducts"). These adducts frequently damage DNA and thus may induce changes in genes that promote or suppress tumor growth. Frederica P. Perera, *Molecular Epidemiology: Insights into Cancer Susceptibility, Risk Assessment, and Prevention*, 88 J. NAT'L CANCER INST. 496, 499 (1996).

The role of epigenetic processes in the development of cancer is a subject of ongoing research. *See, e.g.*, Kazuaki Miyamoto & Toshikazu Ushijima, *Diagnostic and Therapeutic Applications of Epigenetics*, 35 JAPAN J. CLINICAL ONCOLOGY 293 (2005). Frequently, the promoter regions (active regions) of tumor suppressor genes will become "hyper-methylated," which causes the tumor suppressor genes to turn off. *Id.* Hence, these genes will not make tumor suppressor proteins and cancer cells may proliferate.

142. Interview with David L. Eaton, *supra* note 134.

143. Robert A. Weinberg, *Finding the Anti-Oncogene*, SCI. AM., Sept. 1988, at 44. "Oncogenes" are genes that have been implicated as causative agents in many kinds of cancers. Robert A. Weinberg, *A Molecular Basis of Cancer*, SCI. AM., Nov. 1983, at 126. Many cancers are believed to develop as a result of defects in various oncogenes. *See* Stuart H. Yuspa & Miriam C. Poirier, *Chemical Carcinogenesis: From Animal Models to Molecular Models in One Decade*, 50 ADVANCES IN CANCER RES. 25, 36, 38 (1988).

144. Ellinger, *supra* note 131, at 40 n.53. "For several of the oncogenes and anti-oncogenes, specific point mutations at specific locations in the genes have been implicated in activation or inactivation." *Id.* (emphasis omitted). The term "anti-oncogene" is frequently subsumed under the broader term "tumor suppressor gene."

disease or evidence of ongoing disease processes. As a plaintiff's expert noted in *Rainer*:

> The physical injuries sustained by the DNA . . . *and the misrepair of those DNA strands* is analogous to a cutting wound of the tissue of the body. . . . The primary difference is that DNA injury and chromosome misrepair have much more ominous consequences for the individual since such an injury is associated with an increased likelihood of the occurrence of cancer.[145]

Such evidence of damage to the body's defense mechanisms will further challenge the presumption that cellular-level damage is uniformly reversible and hence of limited predictive value. A future focus on how toxic substances can undermine the body's defenses may lead to a new paradigm for thinking about how—and when—inchoate risks manifest into disease.

c. "Early-stage disease" biomarkers

In further support of the diseased state conception, molecular technologies are poised to identify ever-earlier signs of disease. A crucial and often overlooked variable in the debate over the application of genomics is the distinction between two kinds of tests: (1) "signature tests," which are designed to detect the presence of a specific *chemical*;[146] and (2) "disease tests," which are designed to detect *disease risk* or *developing disease*.[147]

Importantly, quite apart from finding "chemical-specific" markers, a major focus of medical research is to identify "early-stage disease" biomarkers.[148] The goal, of course, is to find new possibilities for early medical intervention. As an example, more than thirty genes are believed to undergo "aberrant hyper-methylation"[149] in prostate cancer[150]—another way of saying that distinct events beyond the genome are activating or deactivating these genes. Because such events occur early in carcinogenesis, and because they impede the body's

145. Rainer v. Union Carbide Corp., 402 F.3d 608, 613 (6th Cir. 2005) (quoting expert testimony) (emphasis added).

146. Signature tests provide "evidence of whether or not a particular chemical or physical agent has interacted with an individual's DNA." Ellinger, *supra* note 131, at 59.

147. These include tests that provide evidence that a victim's genetic material or other biological attributes possess structural characteristics consistent with disease or increased risk of disease. *See, e.g.*, *id.* at 69-72.

148. In addition to genes, certain changes in protein type, volume, or behavior may serve as early indicators of disease. Moreover, scientists have labeled various epigenetic events as early-stage disease biomarkers. These constellations of markers arguably may reflect an actual, preclinical diseased state rather than mere risk of disease.

149. "Methylation" is a process by which a small chemical group called a "methyl" group is attached to DNA. *See* Weinhold, *supra* note 80, at A163. Such events may alter biological processes by activating or deactivating particular genes. *See* Miyamoto & Ushijima, *supra* note 141, at 293-94.

150. *See* Long-Cheng Li et al., *Epigenetic Changes in Prostate Cancer: Implication for Diagnosis and Treatment*, 97 J. NAT'L CANCER INST. 103 (2005).

defense mechanisms,[151] scientists have variously labeled them as risk factors or as "early indicators of existing disease."[152] When coupled with traditional evidence of chemical exposure, early-stage disease biomarkers—akin to those described above—may assist tort plaintiffs in proving toxic risk or harm.

Indeed, some disease markers may well be linked to particular chemical exposures. For example, a unique mutation of a tumor-suppressor gene is found in patients who are exposed to the chemical trichloroethylene[153]—notably, the chemical responsible for groundwater contamination in *A Civil Action*. Where, as here, the DNA is altered in a distinct, identifiable manner, and where a tumor-suppressor gene is inactivated through mutation, such damage to the body's defenses may reveal more than mere exposure—such markers may represent significantly enhanced risk or early-stage disease. Indeed, researchers have postulated that individuals exhibiting this chemically associated mutation—and consequent inactivation of the tumor-suppressor gene—may be suffering from a "precancerous condition" in which the tumor has not yet become detectable.[154]

Yet even where such "signature" markers are lacking,[155] it is important to note that new subcellular data will not be used in isolation but will supplement traditional evidence of exposure, risk, and harm.[156] Debates over the

---

151. This process is believed to undermine the protective function of tumor suppressor genes.

152. Peter W. Laird, *The Power and the Promise of DNA Methylation Biomarkers*, 3 NATURE REVS. CANCER 253, 257 (2003). As one scientist has noted in the context of cancer, "increase[s] in knowledge about the molecular [and] genetic basis of cancer may soon yield screening techniques that permit scientists . . . to pinpoint when a tumor becomes malignant, or at least to better estimate when cancer becomes a genuine probability . . . ." Ramsey, *supra* note 98, at 299-300.

153. This persistent marker—a mutation of the Von Hippel-Lindau (VHL) tumor suppressor gene—can be found shortly after trichlorethylene exposure as well as in patients with manifest kidney cancer. Hiltrud Brauch et al., *Trichloroethylene Exposure and Specific Somatic Mutations in Patients with Renal Cell Carcinoma*, 91 J. NAT'L CANCER INST. 854, 854 (1999).

154. *Id.* at 859.

155. Signature markers are helpful, though not essential for proving causal relationships in tort cases. *See, e.g.*, Boston, *supra* note 85, at 239.

> This criterion [specificity] refers to the correspondence of exposure [to a particular chemical] to a specific disease. Is the exposure associated with a specific disease and vice versa? . . . The argument for a causal relationship is weakened if the relationship is nonspecific. However, this criterion has been de-emphasized in recent years because many causes have multiple effects and many diseases have multiple causes. . . . In short, the presence of specificity adds to the cogency of the inference of a causal relationship, but its absence does not preclude such an inference.

Id.

156. Traditional evidence could include evidence derived from occupational, site-specific, or product-specific settings where a cohort of exposed individuals may be identified, the defendants are identifiable, and suspect contaminants are known. For example, in the recent *Henry* case, decided by the Michigan Supreme Court, residents living

applicability of genomics methods frequently overlook this broader legal reality. When considered as part of a larger body of evidence, the criticism that many molecular events are insufficiently distinctive to be correlated with particular chemicals loses some of its bite.

The capacity of new suites of biomarkers to predict disease may distinguish them from other non-impairing conditions, such as asbestos-related pleural plaques, that are presently believed to be of limited predictive value.[157] And the role of plaintiffs' genetic susceptibilities adds a new dimension to discussions of risk. Indeed, one's inherent genetic makeup may hasten the rate of progression from exposure to chemically induced disease. We are entering an era in which the characteristics of the individual, or "host factors,"[158] will

---

near Dow Chemical's Midland plant had been exposed to dioxin over eighty times the level deemed safe for residential contact. Henry v. Dow Chem. Co., 701 N.W.2d 684, 707 (Mich. 2005) (Cavanagh, J., dissenting). The Midland plant was identified as the "principal source of dioxin contamination in the Tittabawassee River sediments and the Tittabawassee River flood plain soils." *Id.* at 687 (majority opinion). Biological tests for disease or disease risk, when combined with proof of exposure to a confined set of polluting sources—as in the Midlands example—could be helpful in demonstrating toxic harm. In this manner, markers of disease may be useful to tort plaintiffs even where these biological changes cannot "fingerprint" specific chemicals.

157. Although there is some disagreement among scientists, plaques generally are not considered to be pre-malignant, as existing studies provide no evidence that plaques "independently cause any progression of further asbestos-related conditions." Schuck, *supra* note 42, at 550 (1992); *see* Brickman, *supra* note 42, at 52. However, scientists and jurists have also recognized that simply by virtue of asbestos exposure, plaintiffs face some degree of enhanced risk of disease. *See, e.g.*, Bowerman v. United Illuminating, No. X04CV 940115436S, 1998 WL 910271, at *6 (Conn. Super. Ct. Dec. 15, 1998) (acknowledging that plaques, while not pre-malignant or auguring malignancy, nevertheless "serve as a good marker for previous exposure and/or biologic responsiveness and, therefore, help us select a group which probably has a somewhat higher risk of developing lung cancer and/or mesothelioma than others who appear comparable in other ways").

Notably, the predictive value of the new molecular and genetic biomarkers will be tied to the validation process, a critical aspect of which is to confirm associations between biomarkers and disease. Dr. Paul Schulte of the National Institute of Occupational Safety and Health (NIOSH) has elaborated on the process for establishing associations between biomarkers and disease as follows:

> Once the association is assessed and confirmed by repeated findings in case-control and cohort studies, it is useful to quantify the change in disease incidence in relation to a given proportion of persons who have a particular biomarker. This can be calculated by use of the attributable proportion (AP). The AP is the proportion of cases attributable to the biomarker. It is based on the senstitivity (S) of the test for the marker and the relative risk (R) of the association between the marker and disease found in case-control, cohort studies or clinical trials. The formula for attributable proportion is $AP=S(1-(1/R))$. The AP ranges between 0 and 1.0. The closer to 1.0, the more valid the biomarker as a surrogate for disease. For use in surveillance, screening, and intervention, the underlying assumption is that the biomarker will be a surrogate for disease or disease risk in such a way that it can be counted or used to trigger action (diagnosis, treatment, or other interventions).

Schulte, *supra* note 87, at 157.

158. Host factors are characteristics of the exposed individuals, whether innate or acquired. These factors may affect susceptibility to the effects of environmental agents. *See,*

play an increasingly important role in estimating the predictive capacity, and hence potential legal status, of early biological changes.[159]

D. *Implications: The nebulous concept of physical injury*

In essence, the more sophisticated our understanding of subcellular events, the less settled the boundaries between risk and injury will become.[160] As technology allows us to perceive adverse health effects in much closer temporal proximity to the chemical exposures that caused them, our conception of "latent" harms may change. When harms traditionally viewed as latent arguably become patent, the legal system will need to reevaluate current mechanisms for addressing these effects.

Technological advancements may call for a more nuanced conception of physical injury. In some cases, there may be a discernable "middle ground" between de minimis effects and classic medical symptoms. In the future, it may become increasingly possible to discern the point at which the body loses its ability to adapt or repair following subclinical insults to the body. Indeed, certain new constellations of biological markers—even if not fully compensable harms[161]—may indicate that bodily integrity has been compromised.

For those who maintain that all subcellular changes are "risk," we must turn our attention to predictive capacity. Society must decide when, in the exposure-disease continuum, indicators of future harm are sufficiently predictive to qualify as harms in themselves, particularly where early intervention could prevent the ultimate disease. If certain subclinical effects can be shown to be predictive with some degree of certainty, the argument can be made that plaintiffs with some non-impairing conditions may nevertheless be entitled to certain measured remedies.

As genetic testing becomes more widespread, it will become increasingly possible to diagnose people who have a disease before any clinical symptoms

---

e.g., Wolfgang Hoffmann et al., *"Host Factors"—Evolution of Concepts of Individual Sensitivity and Susceptibility*, 204 INT'L J. HYGIENE & ENVTL. HEALTH 5, 7-8 (2001).

159. As noted, susceptibility factors may influence the rate of progression from one marker to the next on the exposure-disease continuum.

160. To be sure, many newly identified biomarkers could be characterized as representing either enhanced risk or subcellular signs of actual disease. For example, scientists have already characterized various epigenetic events, such as those discovered early in developing prostate cancer, as either risk assessors or early indicators of disease. *See* Li et al., *supra* note 150, at 103; *see also* Laird, *supra* note 152, at 257. Likewise, the unique mutation of the VHL tumor-suppressor gene following exposure to trichloroethylene may represent, at a minimum, an increased risk of developing kidney cancer, or at a maximum, an actual symptom of developing disease. *See* Miyamoto & Ushijima, *supra* note 141.

161. *See infra* Part III (distinguishing actual loss or harm for purposes of a compensatory damage remedy from injury for the purposes of medical monitoring).

appear, and to identify those who do not have a disease but who are at greater risk. Even those individuals currently labeled "healthy" may already be at some stage along the exposure-disease continuum. For the law of torts, there will be growing pressure to broaden the definition of legal injury to include risk, the need for monitoring, or the increasingly "reasonable" fear of harm, as well as pressure to broaden the definition of physical injury to include more subtle effects of toxic exposure. Hence, genomics will highlight the elemental question of just what it means to suffer a tort.

III. TOWARD A NUANCED CONCEPTION OF INJURY AND REMEDY FOR THE GENOMIC AGE

As technology reveals intermediate events along the exposure-disease continuum, the argument for an intermediate legal remedy becomes ever more compelling. Certain asymptomatic conditions, though themselves not qualifying as fully compensable "illness" or "disease," may nevertheless constitute risks or injuries meriting some form of legal recognition. In this Part, I argue that tort law's traditional focus on overt medical symptoms and "actual loss or damage" will become less appropriate in the future as the capacity to detect—and ultimately to treat—disease moves to the subcellular level. As technology advances, relying on the current model could have the untoward effect of enlisting the legal system's resources only after optimal opportunities for cheap and effective treatment have passed. In the end, the present system's paramount focus on fully developed disease may disserve defendants and plaintiffs alike. If the law fails to anticipate emerging scientific realities, it may forego opportunities to promote public health, limit liability awards, and ultimately reduce society's health care costs.

As science continues to blur the distinction between risk and injury, medical monitoring is recommended as an intermediate and immediately available remedy. In essence, as technology moves inexorably toward earlier detection and treatment, the law may need to: (1) recognize the reality of risk but tailor the remedy (e.g., monitoring funds); or (2) redefine "physical injury" where monitoring remedies are sought. More globally still, society may need to rethink physical injury altogether in the context of the requested remedy.[162] Yet until science can determine just when bodily integrity has been compromised, I recommend a risk-oriented framework for monitoring.

162. Case law relating to toxic exposure appears to suggest that the definition of physical injury is frequently divorced from consideration of the nature of the requested remedy. *See supra* note 95.

A. *Reconsidering Intermediate Remedies*

If technological breakthroughs lend support to the "diseased state" model, medical monitoring will require a renewed focus. An improved understanding of the natural history of disease will help elucidate the precise points at which enhanced medical vigilance will be possible and beneficial.

Of the three nontraditional tort theories discussed in Part I (enhanced risk, medical monitoring, and mental distress), medical monitoring has enjoyed the greatest acceptance in the courts,[163] at least until recently. However, a narrowly focused yet widely cited Supreme Court decision, *Metro-North Commuter Railroad Co. v. Buckley*,[164] denied monitoring recovery in an asbestos case and appears to have triggered a general retreat from this nontraditional cause of action. Although *Metro-North* concerned the interpretation of a specific federal statute, the Federal Employers' Liability Act (FELA),[165] and thus has limited direct precedential value,[166] it has proven influential in recent state court decisions that dismiss monitoring claims absent separately compensable injuries.[167] Unfortunately, the judiciary's retreat may be coming just at the time when increased attention is necessary.

Indeed, the primary purpose of monitoring is to detect disease in its earliest phases,[168] allowing for timely medical intervention.[169] Technical challenges

---

163. In contrast, the enhanced risk claim, which is frequently advocated by scholars, has met with tremendous resistance in the courts—to the extent that many courts have fashioned nearly insurmountable barriers to recovery. Plaintiffs generally must quantify their risk and show that some future physical injury is "more probable than not," or has an over fifty percent chance of occurring. This quantification requirement resembles traditional causation hurdles, except that plaintiffs must apply the standard to a future injury rather than an existing one. Love, *supra* note 15, at 809. For court decisions rejecting enhanced risk claims, see, for example, *In re* Haw. Fed. Asbestos Cases, 734 F. Supp. 1563, 1567 & n.8 (D. Haw. 1990); Eagle-Picher Indus., Inc. v. Cox, 481 So. 2d 517 (Fla. Dist. Ct. App. 1985); Mauro v. Raymark Indus., Inc., 561 A.2d 257, 264-67 (N.J. 1989); Simmons v. Pacor, Inc., 674 A.2d 232, 237 (Pa. 1996). For an argument in favor of the enhanced risk claim, see generally Love, *supra* note 15 (arguing that both the environmental regulatory system and the toxic tort system inadequately deter risk creators and thus a novel remedy is needed).

164. 521 U.S. 424 (1997).

165. 45 U.S.C. §§ 51-60 (2007).

166. Because *Metro-North* involved a cause of action based on the Federal Employers' Liability Act, it is not technically binding on state courts. Most medical monitoring cases involve state law.

167. *See, e.g.*, Paz v. Brush Engineered Materials, Inc., 949 So.2d 1, 5-6 (Miss. 2007); Henry v. Dow Chem. Co., 701 N.W.2d 684, 686, 695-97 (Mich. 2005); Wood v. Wyeth-Ayerst Labs., 82 S.W.3d 849, 857 (Ky. 2002); Hinton v. Monsanto Co., 813 So. 2d 827, 830-32 (Ala. 2001).

168. *See, e.g.*, *In re* Paoli R.R. Yard PCB Litig. (*Paoli I*), 916 F.2d 829, 851 (3d Cir. 1990) (defining the proper inquiry as whether monitoring is "necessary in order to diagnose properly the warning signs of a disease"), *aff'd*, *In re* Paoli R.R. Yard PCB Litig. (*Paoli II*), 35 F.3d 717, 785-88 (3d Cir. 1994).

aside, such a remedy would seem fitting as molecular biology and genomics begin to illuminate the epidemiological "black box." Whereas traditional epidemiology is limited in its ability to detect disease at a time in its natural history when intervention would be most effective,[170] new techniques may allow for preventive intervention before disease progresses to an irreversible stage. In the future, at least for some exposures, science may provide courts with new information regarding (1) which exposed individuals have suffered genetic and/or cellular damage; and (2) which of these individuals are likely to progress to symptomatic disease.[171]

The argument for early detection will gain added currency as medical breakthroughs open up new possibilities for cellular-level interventions.[172] For example, molecular imaging technologies can now pinpoint cancer cells growing in the earliest stages,[173] and emerging drugs can fight cancer by "turning off" proteins that cause cells to proliferate out of control.[174] The

---

169. Judge Cavanagh, dissenting in *Henry*, illustrated the preventive aspect of monitoring:

> Say there's a woman of child bearing age and her blood is tested for high levels of dioxin and she is found to have high levels of dioxin, 95th percentile or so in her body. Medical doctors who are familiar with dioxin contamination say well one of the possible results of having high levels of dioxin contamination in your blood is that you may have depressed thyroid function. So they do a very simple test, a standard test for thyroid function and find out that there is depression of thyroid function. She is then treated and birth defects that are linked to depressed thyroid function do not happen to her [child]. She does not have a child with a birth defect because that preventative measure prevented that irreparable harm.

701 N.W.2d at 711 (Cavanagh, J., dissenting) (quoting plaintiff's counsel).

170. *See* Paul A. Schulte, *Contribution of Biological Markers to Occupational Health*, 20 AM. J. INDUS. MED. 435, 437 (1991).

171. *See supra* Part II.

172. For example, scientists believe that epigenetic processes can be reversed by manipulating the behavior of enzymes. *See* Miyamoto & Ushijima, *supra* note 141, at 297-98. In other words, scientists may not be able to modify genetic mutations, but they may be able to alter the consequences of such mutations.

173. Scientists currently are focusing on the use of molecular imaging technologies for cancer detection and treatment. Whereas traditional imaging technologies were limited to detecting later-stage cancer, new technologies can spot the initial growth of cancer cells:

> Molecular imaging is expected to play an important role in [cancer detection], because it will allow sensitive and specific monitoring of key molecular targets and host responses associated with early events in carcinogenesis. In lung cancer, for example, potential molecular targets include activated oncogenes . . . as well as proteins whose expression or activity is consistently altered in tumor cells versus normal cells. An optical probe . . . has been used in mouse models to detect tumors as small as 1 mm in diameter.

Ralph Weissleder, *Molecular Imaging in Cancer*, 312 SCI. 1168, 1168 (2006).

174. For example, a drug recently approved to treat leukemia is the first of its kind to fight cancer by turning off a specific protein that causes cells to become cancerous and proliferate. *See, e.g.*, William Blum & Guido Marcucci, *Targeting Epigenetic Changes in Acute Myeloid Leukemia*, 3 CLINICAL ADVANCES HEMATOLOGY & ONCOLOGY 855 (2005) (discussing the experimental drug imatinib mesylate, commercially marketed as Gleevec); Karl Peggs & Stephen Mackinnon, *Imatinib Mesylate—The New Gold Standard for Treatment of Chronic Myeloid Leukemia*, 348 NEW ENG. J. MED. 1048 (2003); Nat'l Cancer

emerging field of "nanomedicine" aims to translate discoveries arising from genomics and proteomics into techniques to detect, prevent, and treat disease at the molecular level.[175] Indeed, one goal of the inexorable march toward miniaturization—in information technology as well as biotechnology—is to permit scientists to "go beyond the limitations of biology" and alter the course of disease at the molecular level.[176]

Monitoring's inherently pragmatic focus broadens its appeal relative to other nontraditional tort theories. Indeed, reconsideration of the monitoring remedy would reduce pressure on courts to respond to a potentially expanding universe of claims for mental distress or enhanced risk in the genomic age. In addition to serving fairness and deterrence rationales,[177] medical monitoring—in the end—may serve the interests of defendants and plaintiffs alike. Discovery of disease at earlier stages may help prevent disease progression and ultimately reduce treatment costs and limit future personal injury claims.[178] Moreover, monitoring relief arguably is less speculative than awards for mental distress[179] or enhanced risk,[180] as monitoring can be fashioned into a tailored

---

Inst., National Cancer Institute Fact Sheet, Gleevec: Questions and Answers (May 10, 2001), http://www.cancer.gov/newscenter/qandagleevec.

175. As researchers have recently observed:

> The development of a wide spectrum of nanoscale technologies is beginning to change the foundations of disease diagnosis, treatment, and prevention. These technological innovations, referred to as nanomedicines by the National Institutes of Health . . . , have the potential to turn molecular discoveries arising from genomics and proteomics into widespread benefit for patients. . . . [T]here is a vast array of intriguing nanoscale particulate technologies capable of targeting different cells and extracellular elements in the body to deliver drugs, genetic materials, and diagnostic agents specifically to these locations.

S. Moein Moghimi et al., *Nanomedicine: Current Status and Future Prospects*, 19 FASEB J. 311, 311 (2005).

176. Ray Kurzweil, *Reprogramming Biology: Tinkering with Our Genetic Programs Will Extend Longevity*, SCI. AM., June 6, 2006, *available at* http://www.sciam.com/article.cfm?articleID=000AAD56-1B73-148F-9B7383414B7F0000 (discussing medical applications of nanotechnology).

177. *See, e.g.*, Bower v. Westinghouse Elec. Corp., 522 S.E.2d 424, 431 (W. Va. 1999) (citing *Potter v. Firestone Tire & Rubber Co.*, 863 P.2d 795 (Cal. 1993), for the argument that recognizing a monitoring cause of action advances the tort system's deterrence function).

178. Maskin et al., *supra* note 18, at 527; *see also Potter*, 863 P.2d at 824 (noting that monitoring remedies "may also have the beneficial effect of preventing or mitigating serious future illnesses and thus reduce the overall costs to the responsible parties").

179. Contrasting monitoring to a mental distress claim, one jurist has noted that "[m]edical monitoring tests would not be done to placate plaintiffs' fears; they would be done [only] when qualified health professionals using accepted scientific principles order medical testing." Henry v. Dow Chem. Co., 701 N.W.2d 684, 715 (Mich. 2005) (Cavanagh, J., dissenting).

180. Contrasting monitoring to an enhanced risk claim, the Third Circuit has noted: "[R]ecognizing [monitoring] does not require courts to speculate about the probability of future injury. It merely requires courts to ascertain the probability that the far less costly remedy of medical supervision is appropriate." *In re* Paoli R.R. Yard PCB Litig. (*Paoli I*),

injunctive remedy.[181] The typical approach is a court-approved fund that compensates plaintiffs through specific diagnostic tests as they are ordered by medical professionals; the fund can be structured so expenses are deducted only as they are incurred.[182]

Notably, the Supreme Court's arguments against monitoring in *Metro-North*—particularly the Court's floodgates concerns[183]—were largely directed

---

916 F.2d 829, 852 (3d Cir. 1990); *see also* Friends for All Children, Inc. v. Lockheed Aircraft Corp., 746 F.2d 816, 826 (D.C. Cir. 1984):

> The "injury" that stems from having an increased risk of disease is obviously speculative. It is both difficult to quantify the amount of increased risk imposed on an individual who does not yet have a disease and difficult to conceptualize what that risk is worth in money damages. . . . Here, however, the plaintiffs' need for diagnostic examinations can be shown through competent medical testimony.

Likewise, in *Simmons v. Pacor, Inc.*, 674 A.2d 232 (Pa. 1996), the Pennsylvania Supreme Court, while rejecting a claim for enhanced risk as unduly speculative, allowed an accompanying claim for monitoring to proceed:

> [T]he injury in an enhanced risk claim is the anticipated harm itself. The injury in a medical monitoring claim is the cost of the medical care that will, one hopes, detect [a physical] injury. The former is inherently speculative because courts are forced to anticipate the probability of future injury. The latter is much less speculative because the issue for the jury is the less conjectural question of whether the plaintiff needs medical surveillance.

Id. at 240 n.11 (quoting *Paoli I*, 916 F.2d at 850-51).

181. Many courts and commentators have characterized a court-administered program as injunctive relief. *See, e.g.*, *In re* Diet Drugs Prods. Liab. Litig., No. 99-20593, 2000 U.S. Dist. LEXIS 12275, at *165-66 (E.D. Pa. Aug. 28, 2000) (quoting Barnes v. Am. Tobacco Co., 161 F.3d 127, 132 (3d Cir. 1998)) ("Establishment of a court-supervised program through which class members would undergo periodic medical examinations in order to promote early detection of diseases is a 'paradigmatic request for injunctive relief.'"); Katz v. Warner Lambert Co., 9 F. Supp. 2d 363, 364 (S.D.N.Y. 1998) ("A claim for medical monitoring and research fund is injunctive in nature."); *see also In re* NLO, Inc., 5 F.3d 154, 159-60 (6th Cir. 1993) (refusing to reverse as clearly erroneous the district court's determination of medical monitoring relief as injunctive in nature). Regarding characterization of a monitoring fund as injunctive relief for purposes of applying different rules for class certification, *Gibbs v. E.I. Dupont de Nemours & Co.*, 876 F. Supp. 475 (W.D.N.Y. 1995), would require the fund to have broader public health benefits: "[A] court-administered fund which goes beyond payment of the costs of monitoring an individual plaintiff's health to establish pooled resources for the early detection and advances in treatment of disease is injunctive in nature rather than 'predominantly money damages.'" *Id.* at 481. However, the characterization of monitoring funds as injunctive in nature is not universal. *Compare* Barth v. Firestone Tire & Rubber Co., 661 F. Supp. 193, 205 (N.D. Cal. 1987) (authorizing a medical monitoring fund as common-law injunctive relief), *with* Werlein v. United States, 746 F. Supp. 887, 895 (D. Minn. 1990) (refusing to characterize the medical monitoring fund proposed by plaintiffs as an injunctive remedy), *vacated in part*, 793 F. Supp. 898 (D. Minn. 1992).

182. In some instances, defendants may even be provided with the opportunity to respond to the disbursement.

183. Metro-N. Commuter R.R. v. Buckley, 521 U.S. 424, 442 (1997). The Court explained:

> [T]ens of millions of individuals may have suffered exposure to substances that might justify some form of substance-exposure-related medical monitoring . . . . And that fact, along with

to lump-sum damage awards.[184] Indeed, the Court acknowledged that its conclusion was "limited."[185] In contrast to lump sum recovery, relief that is confined to reimbursement for actual checkups will serve as a deterrent to speculative or frivolous litigation.[186]

Moreover, *Metro-North*'s presumption that monitoring is a redundant remedy for those with health insurance[187] will become increasingly difficult to

---

> uncertainty as to the amount of liability, could threaten both a "flood" of less important cases . . . and the systemic harms that can accompany "unlimited and unpredictable liability.

Id.; *see also Henry*, 701 N.W.2d at 695-96; Hinton v. Monsanto Co., 813 So. 2d 827, 831 (Ala. 2001); Wood v. Wyeth-Ayerst Labs., 82 S.W.3d 849, 857 (Ky. 2002).

184. "[W]e do not find sufficient support in the common law for the unqualified rule of lump-sum damages recovery that is, at least arguably, before us here." *Metro-North*, 521 U.S. at 444.

185. "We need not, and do not, express any view here about the extent to which the FELA might, or might not, accommodate medical cost recovery rules more finely tailored than the rule we have considered." *Id.* "We believe that the note of caution, the limitations, and the expressed uneasiness with a traditional lump-sum damages remedy are important, for they suggest a judicial recognition of some of the policy concerns that have been pointed out to us here . . . ." *Id.* at 441. In fact, the limited nature of the majority's holding prompted Justice Ginsburg to observe: "If I comprehend the Court's enigmatic decision correctly, Buckley may replead a claim for relief and recover for medical monitoring, but he must receive that relief in a form other than a lump sum." *Id.* at 455-56 (Ginsburg, J., dissenting in part). Importantly, although subsequent decisions have relied on *Metro-North* when refusing to recognize monitoring as an independent tort, the Supreme Court acknowledged state court decisions that had recognized monitoring as a stand-alone claim but had cautioned against lump-sum awards. As the Court noted:

> We find it sufficient to note . . . that the cases authorizing recovery for medical monitoring in the absence of physical injury do not endorse a full-blown, traditional tort law cause of action for lump-sum damages . . . . Rather, those courts, while recognizing that medical monitoring costs can amount to a harm that justifies a tort remedy, have suggested, or imposed, special limitations on that remedy.

Id. at 440-41. The Court cited the following decisions that recognize monitoring as an independent tort: Ayers v. Twp. of Jackson, 525 A.2d 287, 314 (N.J. 1987) (recommending creation of a "court-supervised fund to administer medical surveillance payments"); Burns v. Jacquays Mining Corp., 752 P.2d 28 (Ariz. Ct. App. 1987) (same); Potter v. Firestone Tire & Rubber Co., 863 P.2d 795, 825 n.28 (Cal. 1993) (same); Hansen v. Mountain Fuel Supply Co., 858 P.2d 970, 982 (Utah 1993) (suggesting an insurance mechanism or court-supervised fund as the proper remedy). *See Metro-North*, 521 U.S. at 441.

186. Only those who calculate expected judgments in excess of litigation costs are likely to seek access to the adjudicative process. A court-supervised fund that pays for expenses only as they arise would clearly affect the plaintiffs' attorneys' analyses of the transaction costs of litigation. As a leading text notes, "the form of relief requested has a dramatic impact on the financial incentive to pursue such claims." BOSTON & MADDEN, *supra* note 12, at 263.

187. *Metro-North*, 521 U.S. at 442 ("[A] traditional, full-blown ordinary tort liability rule would ignore the presence of existing alternative sources of payment."); *see also* Henderson & Twerski, *supra* note 15, at 843-44 (arguing that recovery of medical monitoring expenses may yield only "marginal improvements" in general health because "[a] large majority of Americans (admittedly not all) are covered by one form or another of

defend. In the future, technology may permit lower-cost monitoring for specific chemical exposures and innate genetic susceptibilities as well as for evidence of developing disease—such tests may go well beyond what physicians would ordinarily prescribe.[188] One might argue that the *Metro-North* decision is based at least partly on practical concerns rather than an objection to monitoring in principle; therefore, if such concerns may be addressed in other ways, the case should not be interpreted as expansively as some jurisdictions have done.[189]

Rather than reflexively inveigh against "novel" techniques and remedies, one must also contemplate that if biological evidence can help identify who is among the injured, it can also help identify who is not. Under proper conditions, biomarkers may equally reassure exposed individuals that they face no future adverse health risk.[190] We may know when an individual's condition is static rather than progressive, or when cellular damage is reparable. Hence, for certain exposures or diseases, new biological data may permit individuals to withdraw (or, indeed, to be excluded) from a plaintiff class or to discontinue a monitoring regime. Although the challenges of medical monitoring are well-recognized,[191] this remedy merits a much closer look in this convergent era, where statistical estimates in populations increasingly confront biological evidence in individuals.

---

general health insurance"); Maskin et al., *supra* note 18, at 528 (noting that medical monitoring recovery may create a windfall for insured plaintiffs).

188. As one jurist has noted, quite apart from any discussion of emerging technologies, "doctors do not generally prescribe testing to determine a patient's dioxin level." *Henry*, 701 N.W.2d at 708 (Cavanagh, J., dissenting). Moreover, structuring the monitoring remedy as a court-approved fund—in lieu of lump-sum damage awards—would help resolve concerns that monitoring provides a windfall to plaintiffs where health insurance or other collateral sources of payment are available. Most jurisdictions limit monitoring to unusual procedures that would not be prescribed in the absence of the exposure, and in-kind recovery could help actuate this requirement. *See, e.g.*, *In re* Paoli R.R. Yard PCB Litig. (*Paoli I*), 916 F.2d 829, 851 (3d Cir. 1990) (requiring that, as a consequence of the exposure, a reasonable physician would prescribe a monitoring regime different from the one that would have been prescribed in the absence of the exposure).

189. For examples of recent state court decisions drawing upon *Metro-North*, see *supra* note 167.

190. Schulte, *supra* note 58, at 517 (illustrating how biomarkers of liver injury may be used to assure individuals of no future health risk from environmental exposures). In essence, if, in the future, we can discern the point at which the body loses its ability to adapt or repair following subclinical insults, the converse may also become true. In thinking about the role of biomarkers in signaling improved health status, I benefited from discussions with Robin Craig.

191. *See, e.g.*, Abraham, *supra* note 19; Guzelian et al., *supra* note 18; Laurel J. Harbour & Angela Splittgerber, *Making the Case Against Medical Monitoring: Has the Shine Faded on this Trend?*, 70 DEF. COUNS. J. 315 (2003); Henderson & Twerski, *supra* note 15; Klein, *supra* note 18; Maskin et al., *supra* note 18; Victor E. Schwartz et al., *Medical Monitoring: Should Tort Law Say Yes?*, 34 WAKE FOREST L. REV. 1057 (1999).

B. *Physical Injury in the Context of Remedy: The* Rainer *Decision*

The judiciary's future approach to monitoring must be considered in conjunction with another species of legal claim—the personal injury claim for subcellular damage, without accompanying claims for mental distress, enhanced risk, or monitoring. In light of this eventuality, the tailored monitoring remedy may prove far less nettlesome for the courts than a new generation of personal injury claims. In this light, the Sixth Circuit's decision in *Rainer v. Union Carbide* is significant because the handful of other decisions addressing subcellular damage generally have addressed the issue through the prism of mental distress, medical monitoring, or enhanced risk of disease.[192] In contrast, the *Rainer* plaintiffs omitted these claims, arguing that, standing alone, subcellular injury is a cause of action justifying a compensatory damage remedy.[193] *Rainer*'s holding thus provides a modicum of clarity, at least where compensatory damages are concerned.

Rainer's conclusion—that asymptomatic DNA damage may not stand alone as a cause of action—was premised on several policy grounds, most notably the floodgates problem and the difficulty of calculating damages for subcellular harm.[194] At least at present, I would suggest that *Rainer*'s holding is justified in reserving compensatory damages for claims involving fully developed disease. As currently conceptualized, compensatory damages are designed to compensate for actual loss in the form of pain, impairment, suffering, lost wages, and costs of treatment—damage that has resulted in changed quality of life.[195] Subcellular injury presents none of these actualities,

---

192. For example, *Werlein v. United States*, 793 F. Supp. 898 (D. Minn. 1992), and *Anderson v. W.R. Grace & Co.*, 628 F. Supp. 1219 (D. Mass. 1986), which left the matter of subcellular injury to the jury, involved claims for mental distress. *Brafford v. Susquehanna Corp.*, 586 F. Supp. 14 (D. Colo. 1984), which also left the matter to the jury, involved a claim for enhanced risk. These claims were absent in *Rainer*, leaving the court to confront a claim for the present injury of subcellular damage. *See generally* Maya Sen, Comment, *Defining the Boundaries of "Personal Injury":* Rainer v. Union Carbide Corp., 58 STAN. L. REV. 1251 (2006) (discussing the *Rainer* holding and its policy justifications).

193. Rainer v. Union Carbide Corp., 402 F.3d 608, 618 (6th Cir. 2005) (responding to a Price-Anderson Act claim, the court noted that "[t]he key question before us . . . is whether Kentucky caselaw equates 'subcellular damage' with 'bodily injury'").

194. Regarding the latter, the court highlighted the challenges of calculating damages for chromosomal harm as compared to losses from manifest diseases such as cancer:

> Losses resulting from salient physical diseases such as cancer or asbestosis are at least quantifiable, and courts have familiarized themselves with methods of computing the associated costs of medical care, absences from work, and physical pain. Here, however, the plaintiffs have suggested no mechanisms for calculating losses resulting from subcellular damage. Indeed, the injuries claimed to date have caused no financial losses or impairments.

Rainer, 402 F.3d at 622. Moreover, the court noted that, in jurisdictions following the "single-action" litigation rule, recovery for subcellular damage would foreclose subsequent recovery for plaintiffs who later become diseased. *Id.* at 621-22.

195. Compensatory damages in tort are divided into two categories—general damages and special damages. Special damages compensate the claimant for quantifiable monetary

and hence a personal injury claim is premature. Reserving compensatory damages for symptomatic disease provides clarity to litigants and is normatively appropriate—at least until science permits diagnosis and treatment at the cellular level. At that time, science may demand a rethinking of the compensatory damage remedy.[196]

Yet from a fairness and deterrence perspective, the *Rainer* decision is troubling in the absence of other avenues of relief. The plaintiffs—workers at Union Carbide's Paducah, Kentucky uranium enrichment facility—were negligently exposed to dangerous radioactive substances (uranium-236 and plutonium-235) in quantities "well beyond the amount considered safe."[197] Neither party disputed the toxicity of the substances—known potent carcinogens—which "quickly settle in the bones and liver, posing a risk as they decay."[198] Tests revealed high levels of chromosomal damage attributable to radiation exposure.[199] Yet the Sixth Circuit agreed with the district court that no present harm had been shown, as plaintiffs had yet to display clinical symptoms of disease.[200]

Indeed, one might argue that the very logic of *Rainer* calls for a monitoring remedy. While treating DNA damage as legally inconsequential, the court nevertheless acknowledged the predictive capacity of the subcellular markers, stipulating that plaintiffs had "amply demonstrated that chromosomal damage is directly linked with an increased likelihood of cancer."[201] And the court's allusion to the difficulty of calculating damages for subcellular harm could provide support for an arguably less speculative remedy in the form of a monitoring fund where medical expenses are deducted as they are incurred. Leaving plaintiffs without legal remedy in situations such as *Rainer* and other recent decisions[202] could result in under-deterrence of risk creators and significantly enhanced risk of serious latent disease.

---

losses suffered, such as costs of medical treatment or lost earnings. General damages compensate for the non-monetary aspects of the harm suffered, including pain and suffering. *See* RESTATEMENT (SECOND) OF TORTS § 7 cmt. a (1979).

196. *See infra* Part III.D.

197. *Rainer*, 402 F.3d at 612.

198. *Id.* at 612. Moreover, company records revealed a history of concealment and disregard for worker safety. One company memo noted that analyzing neptunium exposure through urine samples would be too "tedious and expensive." *Id.*

199. *Id.* at 613.

200. *Id.* at 612, 622.

201. *Id.* at 622.

202. *Rainer* is not an isolated example. The Michigan Supreme Court's recent denial of a monitoring claim in *Henry v. Dow Chem. Co.*, 701 N.W.2d 684 (Mich. 2005), although distinguishable from *Rainer* in terms of alleged injuries and claims, similarly involved egregious exposures and demonstrable risks arguably justifying some form of legal response. The *Henry* case involved extensive exposure to dioxin, a known potent carcinogen that persists in the body, and a single, identifiable defendant, Dow Chemical, who admitted to the negligent exposure. *Id.* at 685-87. Plaintiffs residing near the company's Midland plant had

C. *Medical Monitoring in the Genomic Age: Recognizing Risk or Redefining Physical Injury?*

As noted above, medical monitoring stands out as an immediately available, intermediate remedy that will address some concerns on both sides of future debate over subcellular harm. In essence, as technology continues to move toward earlier detection and treatment, the law may need to adapt by: (1) explicitly recognizing risk while tailoring the remedy (e.g., monitoring funds); or (2) redefining physical injury where monitoring is concerned. More generally, in the genomic age, society may need to rethink physical injury in the context of the requested remedy.

1. *Risk as legal injury: Returning to original conceptions of a nontraditional tort*

Where monitoring is the requested remedy, I argue that the difficulty of distinguishing risk from injury may compel reversing the trend to dismiss such claims absent predicate physical injuries. This approach would acknowledge that the physical injury requirement is an imprecise screening device[203] and will become even more so in the future as a new generation of subcellular biomarkers enters the courtroom. Moreover, this approach would recognize that risks of a certain magnitude may be just as concrete as physical injuries,

---

been exposed to dioxin at over eighty times the level deemed safe for direct residential contact. *Id.* at 707 (Cavanagh, J., dissenting). In fact, plaintiffs had been advised against routine activities such as flower gardening that might increase their dioxin exposure, and were counseled to prohibit their children from playing in the soil. *Id.* Plaintiffs sought a court-supervised medical monitoring program administered by qualified health professionals.

From a fairness and deterrence perspective, the facts of *Metro-North* similarly are troubling when contemplated in the absence of any form of legal relief. *Metro-North* involved a railroad pipefitter who helped maintain pipes in the steam tunnels of Grand Central terminal in New York City. As part of their jobs, pipefitters pulled asbestos from the pipes, releasing dust onto skin, clothing, and into the surrounding air. Covered with white dust, these workers were dubbed "the snowmen of Grand Central." Metro-N. Commuter R.R. Co. v. Buckley, 521 U.S. 424, 446 (1997) (Ginsburg, J., dissenting). The case involved negligence, prolonged exposure to a known toxic substance, disregard for worker safety, and a history of concealment by the company. *Id.* at 447. Justice Ginsburg, dissenting, noted that "[o]n all counts—exposure, increased risk of devastating disease, and the necessity of monitoring—Michael Buckley's complaint presents a textbook case." *Id.* at 451. In support of this position, one could argue that although mesothelioma, the most serious asbestos-related disease, is incurable, other diseases associated with asbestos, particularly certain lung cancers, would benefit from early detection. Notably, the majority acknowledged that "Buckley *is* sympathetic and he *has* suffered wrong at the hands of a negligent employer." *Id.* at 443 (emphasis in original).

203. *See, e.g.*, Potter v. Firestone Tire & Rubber Co., 863 P.2d 795, 810 (Cal. 1993) (rejecting the physical injury requirement as a predicate for emotional distress damages, and describing such a requirement as a "hopelessly imprecise screening device").

requiring some form of legal intervention. Certain asymptomatic conditions, though perhaps not qualifying as fully compensable "illness" or "disease,"[204] may nevertheless amount to risks or injuries that merit a limited legal remedy. Predicating the remedy on significant exposure and disease risk[205]—and a demonstrated need for monitoring[206]—returns to the core principles of *Ayers v. Township of Jackson*[207] and *In re Paoli Railroad Yard PCB Litigation*,[208] two of the leading cases to recognize the independent monitoring claim. Such an approach is in line with monitoring's preventive and deterrent functions.[209]

2. *Rethinking physical injury*

An alternate approach would be to avoid the rhetoric of "risk" altogether and instead predicate the monitoring remedy upon a "present physical injury." This approach would allow courts to move from the controversial domain of compensating for risk to the less problematic and more familiar terrain of compensating for actual injury.

Yet in the genomic age, courts increasingly will need to determine whether such injury includes subcellular injury. Beyond mere "impacts" or de minimis trespassory invasions, certain subcellular changes may be "injurious" or harmful, yet ill-suited to a compensatory damage remedy. As science undermines the comfortable distinction between cellular and gross harm, the elemental debate over whether present injury is required will expand to include

---

204. *See infra* Part III.D.

205. This approach would recognize that prior to the time where disease is "more probable than not," some form of legal injury may occur, requiring some type of legal response. In contrast to a compensatory damages remedy, or damages for "enhanced risk of disease," the stand-alone monitoring claim does not require plaintiffs to prove to a "reasonable medical certainty" that an exposure will result in disease. Instead, competent expert testimony must confirm that the exposure has significantly increased the risks of contracting a serious latent disease and that more frequent monitoring is medically necessitated due to the exposure. *In re* Paoli R.R. Yard PCB Litig. (*Paoli I*), 916 F.2d 829, 851 (3d Cir. 1990). Hence, in contrast to a claim for enhanced risk or compensatory damages, the significantly increased risk in a monitoring case need not be quantified.

206. *See, e.g.*, Bocook v. Ashland Oil, Inc., 819 F. Supp. 530, 536 (S.D. W. Va. 1993) ("A plaintiff seeking such medical monitoring costs in a toxic pollution case of course would have to present substantial evidence of the need for such monitoring.").

207. 525 A.2d 287 (N.J. 1987).

208. *Paoli I*, 916 F.2d 829.

209. As the Third Circuit noted in *Paoli I*:

> Medical monitoring claims acknowledge that, in a toxic age, significant harm can be done to an individual by a tortfeasor, notwithstanding latent manifestation of that harm . . . . Allowing plaintiffs to recover the cost of this care deters irresponsible discharge of toxic chemicals by defendants and encourages plaintiffs to detect and treat their injuries as soon as possible. These are conventional goals of the tort system as it has long existed in Pennsylvania.

Id. at 852.

the question of what kind of injury suffices. Recognition of certain subcellular injuries may need to go hand-in-hand with safeguarding a more traditional, injury-oriented framework.[210]

The alternative option within such a framework—requiring fully-developed disease or impairment as a predicate physical injury—is anathema to monitoring's preventive purpose. Yet this is precisely what is occurring in jurisdictions that require classic symptoms or separately compensable injuries prior to recovery.[211] By forcing plaintiffs to reach this stage, toxic tort law may actually discourage medical interventions that could ultimately benefit defendants and plaintiffs alike.

At the same time, recognition of subcellular injury, while grounded in relatively comfortable philosophical terrain, leaves enormous practical challenges in its wake, at least at present.[212] As noted, an oft-stated justification for the physical injury requirement is to create a principled standard for separating valid from speculative claims.[213] As illustrated in Part II, however, there is no consistency in the courts as to how to define "physical injury," and only a handful of courts have even attempted to think about the *level* of injury appropriate for the monitoring remedy.[214] These problems promise to

---

210. In *Rainer*, for example, one might argue that plaintiffs' DNA damage should be sufficient to serve as a predicate injury for medical monitoring in jurisdictions that require physical injury. Plaintiffs suffered distinct, irreversible chromosomal damage from extensive exposure to ionizing radiation, which poses a risk of cancer—a disease for which the benefits of early intervention are widely recognized.

211. *See, e.g.*, Wood v. Wyeth-Ayerst Labs., 82 S.W.3d 849, 859 (Ky. 2002) (noting that "[t]hose who have ingested fenfluramine, but in whom no disease is yet manifest, will be forced to either forego medical evaluations or proceed with them at their own cost").

212. As the U.S. District Court for the Northern District of Georgia noted in 2005, "[t]he issue of whether the presence of subclinical effects constitute a cognizable injury is not one on which the law, from a national perspective, is well-settled." Parker v. Brush Wellman, Inc., 377 F. Supp. 2d 1290, 1298 (N.D. Ga. 2005); *see also* Bocook v. Ashland Oil, Inc., 819 F. Supp. 530, 534-35 (S.D. W. Va. 1993) (noting that among those courts allowing recovery for medical monitoring only upon proof of some physical injury, "the degree of injury required varie[s] greatly among the courts").

213. *See* Henry v. Dow Chem. Co., 701 N.W. 2d 684, 691 (Mich. 2005).

214. *Bocook* provides a useful exception. When confronted with alleged subcellular injuries, the court made an effort to tease out the meaning of injury for the purpose of medical monitoring:

> The issues facing this court are threefold: first, would Kentucky recognize a claim for recovery of the costs of future medical testing to detect possible diseases caused by toxins; second, would Kentucky require proof of a present physical injury from, or merely exposure to, the toxin before entertaining such a claim; and third, would a plaintiff have to be prosecuting a claim for compensation for present injuries in order to recover for future medical monitoring?

Bocook, 819 F. Supp. at 535-36. Curiously, however, the *Bocook* court, while generously citing the *Paoli* decision—which stands for the proposition that physical injury is not required—nevertheless required a predicate physical injury prior to monitoring recovery. *Id.* at 537 ("proof of exposure alone" is insufficient). Yet the court distinguished a monitoring claim from a personal injury claim, permitting proof of "*some* present physical harm,

escalate—particularly in the short term—as new subcellular information enters the courtroom.[215]

Until such time as science permits diagnosis and treatment at the cellular level, I suggest that the law should maintain a risk-oriented framework for monitoring. This view would recognize that as technology muddies the risk-injury divide, the oft-stated presumption that tort law provides remedies only for injury and not for risk may eventually become a distinction without a difference. This formulation would clarify for litigants that monitoring—if fashioned as a narrow, injunctive remedy—is an appropriate, intermediate remedy for addressing significant exposure to selected hazardous substances and serious disease risk, leaving compensatory damage remedies for fully developed disease. Limited relief for monitoring, where plaintiffs can prove the necessary elements,[216] may appropriately balance deterrence and legal restraint in an age of exponential scientific change. In this vein, although monitoring is a

---

however slight." *Id.* While *Bocook* represents a welcome attempt to tailor the physical injury requirement to the nature of the requested remedy, the decision also illustrates the hopelessly confused state of jurisprudence in this area.

215. *Parker v. Brush Wellman, Inc.*, 420 F. Supp. 2d 1355 (N.D. Ga. 2006), provides hints of things to come. The case posed the question whether "beryllium sensitization," caused by beryllium exposure, should be treated as a physical injury in toxic tort law. Beryllium sensitivity is a subclinical, cellular-level reaction that is recognized as a strong precursor to "chronic beryllium disease" (CBD), a serious, impairing lung disease. The court recognized that although persons experiencing beryllium sensitivity will "most likely suffer from CBD at some future date," sensitivity to beryllium is not a condition that, to a "reasonable medical certainty," will result in disease. *Id.* at 1361 (citation omitted). However, illustrating the complexities of this legal terrain, the court acknowledged that some courts might treat even relatively asymptomatic, early-stage CBD as an injury compensable in tort. *Id.* at 1362. Underscoring the complexities of the biology, one commentator has noted, "[t]he relationship between [beryllium] sensitization and development of CBD . . . is not completely understood. Experts disagree on the strength of the relationship between sensitization and CBD, the exact rate at which sensitized people may develop CBD, and even how to define sensitization and CBD." Scott Fields, *Toxic Beryllium: New Solutions for a Chronic Problem*, 109 ENVTL. HEALTH PERSP. A-74, A-77 (2001). Further complicating the picture, and nowhere mentioned in *Parker*, studies have shown that beryllium-exposed individuals possessing certain gene variants are at considerably greater risk than the general population for developing CBD. *Id.* at A-77 to A-79 (discussing glutamic acid GLU-69 as a marker for susceptibility to the effects of beryllium).

216. This is not a blank check for plaintiffs, as they would need to prove the necessary elements of a monitoring claim. Although the elements vary considerably among jurisdictions, the *Paoli* factors provide a helpful starting point. *See supra* note 53 (discussing *Ayers* and *Paoli*). Countering the claim that the monitoring remedy is unduly open-ended, Justice Ginsburg cited the *Paoli* factors in her dissent in *Metro-North*: "It is doubtful that many legions in the universe of individuals ever exposed to toxic material could demonstrate that their employers negligently exposed them to a known hazardous substance, and thereby substantially increased the risk that they would suffer debilitating or deadly disease." Metro-N. Commuter R.R. Co. v. Buckley, 521 U.S. 424, 454 (1997) (Ginsburg, J., dissenting in part) (citation omitted). Notably, various jurisdictions have elaborated upon the *Ayers* and *Paoli* factors. *See infra* notes 218-23 and 228-29.

highly contextual endeavor,[217] it will be important to evaluate recommended and court-implemented approaches designed to help strike this balance. These include mechanisms to: (1) limit the universe of relevant exposures;[218] (2) increase the burden of proving exposure;[219] (3) account for absolute and relative risks;[220] (4) ensure the appropriateness of selected tests;[221] (5) prevent

217. *See, e.g.*, Guzelian et al., *supra* note 18, at 73 (stating that there is no universal protocol for monitoring a population for adverse health effects, thus each disease must be evaluated separately).

218. For example, the Supreme Court of Utah has set out a detailed, eight-part test for monitoring recovery that, among other things, requires exposure to a "toxic" substance, defined as a "poison," that through its chemical action usually "kills, injures, or impairs an organism." Hansen v. Mountain Fuel Supply Co., 858 P.2d 970, 979 (Utah 1993) (quoting WEBSTER'S NEW COLLEGIATE DICTIONARY 881 (1981)). The exposure must be substantial in terms of intensity or duration. *Id.* As another way of confining the universe of exposures, one commentator has recommended limiting monitoring recovery to a specified number of the most hazardous chemicals as designated by a government agency such as the Agency for Toxic Substances and Disease Registry (ATSDR). *See* Troyen A. Brennan, *Environmental Torts*, 46 VAND. L. REV. 1, 69 (1993). In the genomic age, one might envision combining such a threshold approach with development of a scientific consultative organization tasked to disseminate information on serious related diseases for which validated, predictive biomarkers are available and early treatment is technically possible or expected. Such an approach might help the law keep pace with evolving science, and such notice could provide incentives for potential defendants to fund monitoring programs that would obviate the need for litigation and/or reduce future recoveries.

219. Utah's test for monitoring recovery defines "exposure" as "ingesting, inhaling, injecting or otherwise *absorbing* the substance in question in the body." *Hansen*, 858 P.2d at 979 (emphasis added). The New Jersey Supreme Court has limited monitoring recovery to plaintiffs experiencing "direct" exposure. Theer v. Philip Carey Co., 628 A.2d 724, 733 (N.J. 1993) (holding that monitoring is not required for plaintiffs with indirect exposure to toxic substances whose risk of injury cannot be related specifically and tangibly to that exposure).

220. Kenneth Abraham has recommended that plaintiffs be required to show a significant increase in relative risk (change in plaintiff's disease risk level attributable to the exposure) as well as absolute risk (actual chance of contracting a particular disease) prior to recovering for monitoring. *See* Abraham, *supra* note 19, at 1979-80. To illustrate the need for such dual tests, he provides the following example: An exposure that increases a plaintiff's chance of contracting cancer from one in five million to three in five million represents a three hundred percent increase in plaintiff's chance of contracting cancer, representing a significant increase in plaintiff's relative risk. *Id.* at 1980. However, if plaintiff's absolute risk is still very small, the change in risk level should be insufficient to warrant recovery. *Id*. Conversely, plaintiff's pre-exposure risk of contracting cancer may be significant, e.g., one in a thousand. If plaintiff's risk of cancer increases to 1.01 in 1000 due to an exposure, although the risk is significant, the defendant's actions have not significantly increased the plaintiff's risk. *Id.* at 1981. While urging consideration of absolute and relative risks, Abraham resists the establishment of quantitative risk thresholds (such as requiring that the defendant's conduct double plaintiff's likelihood of contracting cancer) "because of the impossibility of precise quantification." *Id.* at 1982.

221. For example, to ensure that recommended tests are appropriate for a given plaintiff, Utah requires "not only that a doctor prescribe the test for this plaintiff, but also that the test is shown by expert testimony to be one a reasonable physician in the area of specialty would order for a plaintiff similarly situated." *Hansen*, 858 P.2d at 980. "This dual

double recoveries;[222] and (6) evaluate the benefits of monitoring in light of risks and costs.[223]

Indeed, in the best of worlds, opening the courthouse door—but just part way—might well encourage defendants to develop monitoring programs of their own, ultimately reducing litigation pressures in the future.

D. *Future Convergence of Remedies*

In the long term, science may better illuminate the point at which "risk" translates to bodily dysfunction or disease. Advances in molecular biology and genetics may sharpen our ability to determine just when injury or disease has

---

requirement prevents recovery for costs of treatment not generally accepted by the medical community." *Id*. One interesting model that might be adaptable to the new age of molecular-level biomarkers is presented in *In re Diet Drugs Products Liability Litigation*, No. 0-99-20593, 2000 U.S. Dist. LEXIS 12275 (E.D. Pa. Aug. 28, 2000). In that case, the judge endorsed the fairness of a medical monitoring class action settlement because it met the following criteria: (1) whether the disease in question progresses asymptomatically following exposure; (2) a diagnostic test with high sensitivity exists; (3) the exposed population has a relatively high prevalence of disease; (4) the diagnostic test therefore has a high predictive value; (5) the test is relatively low-cost; (6) medical monitoring could be integrated into standard clinical follow-up of those with disease; (7) monitoring could lead to early preventive care; and (8) the monitoring allows for the appropriate timing of definitive treatment. *Id.* at *166-67 (discussed in Victor E. Schwartz et al., *Medical Monitoring, The Right Way and the Wrong Way*, 70 MO. L. REV. 349, 379 n.183 (2005)).

222. As noted, most jurisdictions require that a reasonable physician would prescribe for the exposed plaintiff a monitoring regime different from one that would have been prescribed in the absence of the exposure. *See, e.g.*, *In re* Paoli R.R. PCB Litig. (*Paoli II*), 35 F.3d 717, 788 (3d Cir. 1994); Theer v. Carey Co., 628 A.2d 724, 733 (N.J. 1993); Hansen v. Mountain Fuel Supply Co., 858 P.2d 970, 980 (Utah 1993). Adding some specificity to this requirement, one commentator has suggested that if the medical literature recommends monitoring for the population at large, courts should not permit monitoring claims to proceed because such monitoring would not be special or a result of the alleged exposure. Guzelian et al., *supra* note 18, at 74. Similarly, if the medical literature advises monitoring for a special group absent any exposure, medical monitoring costs should not be granted because of the exposure. *Id.* at 75. It has been further suggested that attorneys' fees for monitoring recovery could be tied to plaintiffs' actual *use* of the monitoring remedy. Interview with Michael Dore, Director, Lowenstein Sandler PC, in Roseland, N.J. (Jan. 21, 2007).

223. As one commentator has noted, "[c]ourts have generally allowed physicians to take into account such practical considerations as the burdensome frequency of the testing, its excessive price and the risk of harm to the plaintiff in determining whether or not the testing would be recommended." Maskin et al., *supra* note 18, at 534. Several jurisdictions explicitly limit monitoring to situations where early detection is not only possible, but could have a beneficial effect. *See, e.g.*, *Paoli II*, 35 F.3d at 787; *In re* Paoli R.R. Yard PCB Litig. (*Paoli I*), 916 F.2d 829, 852 (3d Cir. 1990). This arguably would eliminate recovery for medical surveillance where the predictive value of the evidence is insufficient to justify risks of testing procedures. Hence, if the risk of the chemical exposure is relatively small and the monitoring itself entailed side effects or other risks, the monitoring claim presumptively would be denied. For discussions of monitoring-related risks, see, for example, Guzelian et al., *supra* note 18, at 63.

occurred, thereby reducing the time period between exposure and remediable harm. In essence, the gap may close between the cell biologist's definition of injury and the clinician's definition of disease,[224] as genomic tools become more commonplace in the clinical world.[225] By challenging our reliance on traditional clinical symptoms, new genomic data may transform the way we define disease. Ultimately, at least for some exposures and diseases, we may not only be able to diagnose but also to treat disease at the molecular level—thereby arresting the progression to clinical symptoms. Because treatment entails costs, treating subclinical injuries would constitute "actual losses" for plaintiffs and thus would command a compensatory damage remedy[226]—yet one much reduced compared to current damages for lost wages and late-stage therapies.

In essence, the blurring of risk and injury in the new genomic era may lead to a convergence of remedies. Where cellular-level treatment is available, the need for monitoring would recede, calling into question exactly where monitoring leaves off and compensatory remedies begin. At that point *Rainer* itself, however appropriate in current circumstances, would need to be re-thought. In sum—at least for some diseases and exposures—monitoring will not only be an intermediate remedy, but a transitional remedy in the law of torts. This future convergence of monitoring and personal injury claims will demand entirely new ways of thinking about tort law's treatment of "latent" harms. Such a transition will challenge both the underlying assumptions and the

224. Pathologists tend to define injury as "an alteration of structure and function of a cell, tissue or organ," including damage which is detectable only on the subclinical level. Zurich Ins. Co. v. Northbrook Excess & Surplus Ins. Co., 494 N.E.2d 634, 639 (Ill. App. Ct. 1986). "The fact that the cell damage is subclinical and requires medical research to verify does not detract from the fact that a real injury occurs." *Id.* at 643. Clinicians, on the other hand, define injury as requiring some type of noticeable harm, resulting in impaired appearance or function. *Id.* at 639. As genomic tools become more commonplace in the clinical world, there will be growing pressure to find a middle ground between cellular harm and traditional clinical harm.

225. *See, e.g.*, Lisa Belkin, *A Doctor for the Future*, N.Y. TIMES, Nov. 6, 2005, (Magazine), at 68 (discussing the merging of genetic research and patient diagnosis and treatment); Nicholas Wade, *The Quest for the $1,000 Human Genome*, N.Y. TIMES, July 18, 2006, at F1 (discussing the fact that low-cost decoding may "bring the genomic age to the doctor's office"); *see also Molecular Imaging: Diagnosing Diseases Before Symptoms Strike*, SCI. DAILY, *supra* note 81 ("We believe that molecular imaging will one day enable us to diagnose specific molecular events of cancer, neurologic disease or inflammation earlier in the course of disease, and that this will help doctors identify the most effective therapy for individual patients." (quoting the director of the Molecular Imaging Center at Washington University, St. Louis)).

226. *See* RESTATEMENT (SECOND) OF TORTS § 7 cmt. a (1979) (defining remediable "harm" as "loss or detriment to a person, and not a mere change or alteration in some physical person"); *see also* RESTATEMENT (THIRD) OF TORTS: LIABILITY FOR PHYSICAL HARM § 4 cmt. c (Proposed Final Draft No. 1, 2005).

semantics of core debates over the single-action rule,[227] statutes of limitation,[228] and the essential elements of these legal claims.[229]

---

227. The single action rule ordinarily bars a second claim based on the same transaction or occurrence as a previous one. RESTATEMENT (SECOND) OF JUDGMENTS §§ 17-19 (1982). Quite apart from considerations of genomics, the case has been made for relaxing the single-action rule where the monitoring remedy is sought. *See generally* Kara McCall, Comment, *Medical Monitoring Plaintiffs and Subsequent Claims for Disease*, 66 U. CHI. L. REV. 969 (1999). The core argument is that because monitoring recovery incorporates costs of detecting disease and not costs of future treatment, monitoring awards pose a reduced threat of double recovery compared to certain claims for emotional distress or enhanced risk. *Id.* at 987-88; *see also* Brennan, *supra* note 218, at 67 (medical monitoring, if properly structured to reimburse for tests taken, confines the award to costs flowing from the specific harm that occurred, not from future disease risk). Interestingly, as treatment moves to the subcellular level and monitoring and personal injury claims merge, as suggested here, the problem of future claims may diminish considerably—at least where such treatments are successful. Yet where treatments are incomplete or ineffective, the problem of future claims will persist, and will require case-by-case analysis in the courts.

228. Courts have reached a variety of conclusions concerning the factors to be considered in deciding when to commence the running of tort statutes of limitation. The nature of the claim, the age of the plaintiff, the ability to serve process on the defendant, and a variety of other factors have led to very different conclusions as to when particular statutes of limitation should begin to run. *See generally* MICHAEL DORE, LAW OF TOXIC TORTS § 12.1 (2007) ("[T]he key statute of limitation issue involved in these cases is the question of when the statute accrued. The courts of different states have come up with a variety of rules for answering this accrual question."). Even states that have adopted a "discovery rule" have applied a range of complex standards for determining when the requirements of such a rule have been satisfied. *See id.* § 12.4-.7; *see also* Evenson v. Osmose Wood Preserving Co. of Am., 899 F.2d 701, 705 (7th Cir. 1990) (finding that plaintiff's awareness of a "reasonable possibility" that defendant's act or product caused plaintiff's injuries is sufficient to trigger the statute of limitation; "reasonable probability" is not required, but plaintiff's "mere suspicion" is insufficient). The availability of a new generation of biomarkers will raise complex new questions about the level of a plaintiff's knowledge sufficient to trigger a statute of limitation under the discovery rule. Moreover, emerging genomic technologies and biomarkers may pose dilemmas for plaintiffs. To the extent that new diagnostic tools (and screening programs) would make it easier to "discover" an effect from a toxic exposure, that could trigger the running of a statute of limitation for certain claims, yet a rule equating "harm" exclusively with late-stage disease may deny plaintiffs recovery during the window when the claim would be timely. Complicating the picture further, a possible merger of monitoring and personal injury claims, as discussed here, may hobble efforts to characterize the claim for which the limitation period applies. The resolution of these issues is beyond the scope of this Article. It is clear, however, that scientific advancements will add factual and legal complexity to the already difficult judicial (and sometimes legislative) task of determining when statutes of limitation begin to run for particular tort claims.

229. For example, many jurisdictions condition monitoring recovery on the availability of *treatment* techniques. *See, e.g.*, *In re* Paoli R.R. Yard PCB Litig. (*Paoli I*), 916 F.2d 829, 852 (3d Cir. 1990) (requiring the existence of monitoring and testing procedures that make "the early detection *and treatment* of disease possible and beneficial" (emphasis added)). As treatment technologies inexorably move to the subcellular level, and detection and treatment go hand-in-hand, one must ask whether this would continue to be a monitoring as opposed to a personal injury claim.

E. *Genomics and Toxic Tort Law: Unbridled Liability or Risks Without Remedies?*

If the law recognizes new combinations of subclinical markers, or if a growing number of courts recognize monitoring as an independent tort, as suggested here, the question becomes whether such claims can be limited. The most common and strenuous critique of the monitoring cause of action is the floodgates problem.[230] A major motivator of the predicate injury requirement is precisely to avoid this problem.[231]

Indeed, a floodgates scenario is not difficult to envision. As we have seen, evidence of subclinical biological effects and susceptibilities may serve as ingredients for a new generation of tort claims based on exposure, risk, or developing disease. As one contributing factor, the portability of microarrays and other genetic testing methods may expand possibilities for ongoing monitoring in the field.[232] With echoes of asbestos and silicosis, the specter of

---

230. *See, e.g.*, Metro-N. Commuter R.R. Co. v. Buckley, 521 U.S. 424, 442 (1997); Hinton v. Monsanto Co., 813 So. 2d 827, 831 (Ala. 2001); Wood v. Wyeth-Ayerst Labs., 82 S.W.3d 849, 857 (Ky. 2002); Henry v. Dow Chem. Co., 701 N.W.2d 684, 695-96 (Mich. 2005); *see also* Henderson & Twerski, *supra* note 15, at 844-46 (cautioning that rationales for the recognition of medical monitoring claims cannot outweigh the unlimited liability placed on industry); Victor E. Schwartz et al., *Medical Monitoring—Should Tort Law Say Yes?*, 34 WAKE FOREST L. REV. 1057, 1071-72 (1999) (expressing concern that recognition of medical monitoring as an independent tort would place unpredictable and limitless liability on industry and divert resources from the truly impaired).

231. *See, e.g.*, *Hinton*, 813 So. 2d at 829-31.

232. Although it may be several years before microarray-based tests are accepted for routine screening for chemical exposure, effects, and susceptibilities, the groundwork is now being constructed. Genotyping of an individual from a sample of DNA is already possible, Michael F.W. Festing, *Experimental Approaches to the Determination of Genetic Variability*, 120 TOXICOLOGY LETTERS 293, 294 (2001), and microarray technologies may permit large-scale, low-cost screening of individuals or populations in the future, *see, e.g.*, Marchant, *supra* note 130, at 10086-87 (discussing microrarray-based targeted screening programs for purposes of individualized interventions as well as population-wide risk assessment and risk management). Such screening would be best suited to occupational settings or site-specific environmental problems (including hazardous waste sites and attendant groundwater contamination) in which samples from the same individuals can be taken over time. *Id.*; *see also* Nuwaysir et al., *supra* note 75, at 157; Philip M. Iannaccone, *Toxicogenomics: "The Call of the Wild Chip,"* 109 ENVTL. HEALTH PERSP. A8, A10 (2001) (stating that using microarrays, "[i]t may be possible to screen biological samples obtained from workers at Superfund sites for the adverse effects of exposure to compounds present in the site"); M. Vrijheid et al., *Chromosomal Congenital Anomalies and Residence Near Hazardous Waste Landfill Sites*, 359 LANCET 320-22 (2002) (reporting an increased frequency of chromosomal anomalies in residents living near hazardous waste sites). Indeed, screening capabilities could encourage litigation, as many U.S. residents live near hazardous sites. *See* Maskin et al., *supra* note 18, at 528 (noting that nearly twenty percent of the U.S. population lives within four miles of a hazardous waste site placed on the EPA's National Priority List).

mass screenings for litigation purposes is easy to imagine.[233] This possibility gains even more currency as scientists strive to develop ever more accessible biomarkers.[234] Indeed, while this newly accessible data may spawn breakthroughs in disease treatment and prevention, a new generation of genetic and molecular information also could trigger an epidemic of fear, with significant implications for the legal system.

However, I suggest an alternative scenario—little explored but at least as plausible. Claims by the unimpaired may be limited by access barriers, doctrinal constraints, and inherent disincentives—quite apart from any physical injury requirement. First, as discussed above, relief that is limited to reimbursement for actual checkups will serve as a deterrent to litigation.[235]

---

233. In the asbestos example, the surge in claims by the unimpaired has been linked, at least in part, to the phenomenon of mass screenings. Reports have documented the practice of certain labor unions and plaintiff's lawyers who engage in aggressive claim-solicitation campaigns on a mass basis, "multiply[ing] the number of filed cases, thereby increasing the pressure on defendants to settle cases wholesale." Schuck, *supra* note 42, at 564. Mobile X-ray vans have been driven to plant sites to screen industrial workers for pleural plaques or other signs of asbestos exposure. *See, e.g.*, *Asbestos Litigation Crisis in Federal and State Courts: Hearings Before the Subcomm. on Intellectual Prop. and Judicial Admin. of the H. Comm. on the Judiciary*, 102d Cong. 81, 100 (1991) (testimony of Professor Lester Brickman). It would not be difficult to imagine replacement of X-ray vans with genetic testing mobiles.

234. As a dramatic example, one recent study has attempted to identify air pollution-induced pulmonary disease using biomarkers captured from exhaled breath. *See* Sergei A. Kharitonov & Peter J. Barnes, *Biomarkers of Some Pulmonary Diseases in Exhaled Breath*, 7 BIOMARKERS 1 (2002). Importantly, many conventional biomarkers must be obtained through biopsies, blood samples, and other relatively invasive procedures, which makes continuous monitoring difficult. Researchers are now testing for genetic and epigenetic changes in more accessible tissues and body fluids. *See, e.g.*, T. Brüning et al., *Pathological Excretion Patterns of Urinary Proteins in Renal Cell Cancer Patients Exposed to Trichloroethylene*, 49 OCCUPATIONAL MED. 299, 302 (1999) (identifying specific urinary proteins as biomarkers of kidney cancer in individuals exposed to tricholoroethylene). Scientists are also analyzing epigenetic changes in sputum to detect lung cancers, urine to detect prostate and bladder cancers, mammary aspirate to detect breast cancers, and saliva to detect head and neck cancers. *See* Miyamoto & Ushijima, *supra* note 141, at 294.

235. *See supra* note 186 and accompanying text. Interestingly, the Proposed Final Draft for the upcoming Third Restatement of Torts makes a similar point in its comment explaining the definition of physical harm:

> [S]o long as there is physical impairment there is no need to establish any minimum amount of physical harm. A change in the physical condition of a person's body or property must be detrimental for the change to count as a harmful impairment; yet there is no requirement that this detriment be major . . . . Under this Restatement's Sections on Liability, the problem of trivial physical harm often will solve itself: the plaintiff who has incurred only trivial harm may not be inclined to sue so as to secure trivial compensation. Asbestos claims by plaintiff[s] who suffer no clinical symptoms but who have abnormal lung X-rays are an unfortunate and aberrational exception, but exist only because of the massive number of such claimants.

RESTATEMENT (THIRD) OF TORTS: LIABILITY FOR PHYSICAL HARM § 4 cmt. c (Proposed Final Draft No. 1, 2005).

Second, the transaction costs of bringing suit individually in advance of fully developed disease may suggest the need for class treatment. Yet there is a clear trend in the courts to deny certification of monitoring classes,[236] and variations in genetic susceptibility could make broad-based class certification even more difficult in the future.[237] Ironically, in some cases, genetics' increasingly sophisticated individualization of risk could limit future access to legal remedies. Indeed, current battles over predicate physical injury may shift to new battles over certification of monitoring classes—requiring fresh consideration of the balance between individualized risk information and collective legal remedies.[238]

Moreover, while asbestos provides a useful metaphor for understanding the legal status of non-impairing conditions, the asbestos problem is unique and must be distinguished. First, asbestos produces durable and virtually unequivocal markers of exposure,[239] and asbestosis and mesothelioma are "signature diseases" in that asbestos exposure is believed to be the sole or

---

236. *See, e.g.*, *In re* St. Jude Med., Inc., Silzone Heart Valve Prods. Liab. Litig., 425 F.3d 1116, 1122 (8th Cir. 2005) (denying class certification for medical monitoring plaintiffs and noting that such classes suffered from "cohesion difficulties"—that the highly individualized nature of each plaintiff's need for medical monitoring made class certification improper).

Surprisingly, even those courts recognizing monitoring as an independent tort have often resisted class certification. *See, e.g.*, Lockheed Martin Corp. v. Superior Court, 63 P.3d 913, 917-22 (Cal. 2003) (recognizing monitoring as an independent cause of action but denying class certification); Goasdone v. Am. Cyanamid Corp., 808 A.2d 159, 170-71 (N.J. Super. Ct. Law Div. 2002) (same); Askey v. Occidental Chem. Corp., 477 N.Y.S.2d 242, 248 (N.Y. App. Div. 1984) (same).

237. *See* Marchant, *supra* note 127, at 10651.

238. A recent publication by the Washington Legal Foundation noted that "defendants should be encouraged that both state and federal law continue to provide safeguards against the abuse of medical monitoring, including against the inappropriate certification of medical monitoring class actions." BORANIAN & HARA, *supra* note 34, at 11-12. The report concluded that "[c]ontrolling authorities . . . give defendants and absent class members substantial protection against the many problems that medical monitoring class actions present and against the potential abuse of this relatively new area of the law." *Id.* at 23.

239. Asbestos fibers permanently embed in lung tissues, and pleural plaques, which generally develop ten to fifteen years after initial asbestos exposure, serve as ongoing markers through much of the latency period of later-emerging diseases. *See, e.g.*, AGENCY FOR TOXIC SUBSTANCES & DISEASE REGISTRY, DEP'T OF HEALTH & HUMAN SERVS., TOXICOLOGICAL PROFILE FOR ASBESTOS § 3.8.1 (2001), *available at* http://www.atsdr.cdc.gov/toxprofiles/tp61-c3.pdf (discussing the presence of asbestos fibers in the lung and related fluids as "principal biomarkers of exposure to asbestos"); *see also* David L. Faigman et al., *Modern Scientific Evidence, The Law and Science of Expert Testimony*, *in* 4 MOD. SCI. EVIDENCE § 38-2.3.2 (2d ed.) ("[B]ilateral plaques are practically pathognomonic of prior exposure to asbestos . . . ."); Brickman, *supra* note 42, at 60 (stating that pleural plaques are markers of extensive exposure to asbestos). Such strong and durable exposure data helps reduce the "indeterminate plaintiff" problem that frequently hobbles cases involving diffuse environmental hazards.

predominant cause.[240] Hence, plaintiffs with certain asbestos-related markers are significantly liberated from the two greatest barriers facing toxic tort plaintiffs—proving exposure and causation.[241] In contrast, the new generation of subcellular markers will present questions of specificity,[242] durability,[243] validity,[244] and reliability that will engender case-specific challenges to their admissibility and interpretation.[245]

---

240. *See, e.g.*, Margaret A. Berger, *Upsetting the Balance Between Adverse Interests: The Impact of the Supreme Court's Trilogy on Expert Testimony in Toxic Tort Litigation*, 64 LAW & CONTEMP. PROBS. 289, 298 n.66 (2001) (defining a signature disease as "one that nearly always occurs as a result of exposure to a certain substance"). Signature diseases can also be defined in terms of relative risk, as their association with exposure to a particular substance suggests that they rarely occur in the non-exposed population. Hence "[t]he incidence of the background risk for signature diseases is virtually zero." Boston, *supra* note 85, at 203. Such signature diseases are believed to be rare.

241. The signature status of asbestos-related diseases limits the problem of alternate cause. *See, e.g.*, Donald G. Gifford, *The Peculiar Challenges Posed by Latent Diseases Resulting from Mass Products*, 64 MD. L. REV. 613, 688 (2005) (noting that most toxic tort plaintiffs have difficulty proving causation but asbestosis and mesothelioma are signature diseases "in which there is a clearly evident and exclusive causal connection" to asbestos exposure). "Once a signature disease is identified, there is no need for proof of either general causation or specific causation, as the existence of the disease is tied to exposure to the signature agent." Michael D. Green, *Causation in Pharmaceutical Cases*, SL038 ALI-ABA 139, 166 (2005). The process establishing causation is somewhat more complex, as proof of causation in asbestos cases may be broken into two components: medical causation and proof of risk. Daniel J. Penofsky, *Asbestos Injury Litigation*, 60 AM. JUR. TRIALS 73, § 41 (2007). Proof of medical causation, discussed above, requires testimony that the exposure can cause the asbestos-related disease and that the exposure did cause the asbestos-related disease. *Id.* Courts have found the presence of asbestos fibers in tissue and signature disease associated with asbestos to be persuasive evidence of medical causation. *Id.* § 44. Proof of risk requires identifying the defendant(s) and the asbestos-containing product, showing plaintiff's exposure to the product, and establishing the duration of the exposure. *Id.* § 41. Hence, where multiple defendants are involved, additional questions of causation are posed.

242. As noted, the discovery of unique genetic or molecular "fingerprints" identifying specific chemical exposures is proceeding slowly. However, the quest for "signature" markers is still in its infancy and is a subject of intense research. *See* John D. Groopman & Thomas W. Kensler, *The Light at the End of the Tunnel for Chemical-Specific Biomarkers: Daylight or Headlight?*, 20 CARCINOGENESIS 1, 4 (1999).

243. *See, e.g.*, Bonassi & Au, *supra* note 125, at 76 ("For many types of biomarkers the most important consideration is the stability of the biomarkers with respect to time after the exposure.").

244. *See supra* note 87.

245. The use of particular subcellular markers for establishing associations between exposure and subsequent injury will depend on their admissibility in court. *See generally* John C. Childs, *Toxicogenomics: New Chapter in Causation and Exposure in Toxic Tort Litigation*, 69 DEF. COUNS. J. 441 (2002). In the landmark case *Daubert v. Merrell Dow Pharm., Inc.*, 509 U.S. 579 (1993), the Supreme Court cast the federal trial judge in the role of a "gatekeeper" who uses a two-prong test to determine the admissibility of scientific evidence. This test requires the trial judge to determine (1) the reliability of the evidence, such that it is "scientific knowledge," and (2) whether the evidence will assist the understanding of the jury. *Id.* at 589-92. The *Daubert* Court indicated that the main focus

In addition, the new data will be useful to defendants and plaintiffs alike.[246] For example, new molecular technologies may help defendants identify intrinsic or background risks unrelated to the exposures at issue.[247] And even for nontraditional tort claims with reduced causation hurdles, questions of exposure and causation will continue to pose a problem.[248] In sum,

---

should be on the methodologies used rather than the conclusions reached. *Id.* at 595 ("The focus, of course, must be solely on principles and methodology, not on the conclusions that they generate."). The standard under *Daubert* is typically considered to be a liberal one. *See, e.g.*, Patricia E. Lin, *Opening the Gates to Scientific Evidence in Toxic Exposure Cases: Medical Monitoring and* Daubert, 17 REV. LITIG. 551, 569 (1998); *see also* David E. Bernstein & Jeffrey D. Jackson, *The* Daubert *Trilogy in the States*, 44 JURIMETRICS J. 351, 365 (2004) (concluding that only a minority of states have fully embraced *Daubert*).

Where subcellular markers and other toxicogenomic data meet the *Daubert* test, a court could nevertheless exclude such evidence based on uncertainty in linking the results to a specific disease, or other uncertainties surrounding their representation. In *General Electric Co. v. Joiner*, 522 U.S. 136 (1997), the Supreme Court made clear that a trial judge need not focus entirely on methodology, but may also examine the reliability of expert conclusions reached using the evidence. *Id.* at 142-47. Thus the *Joiner* test will likely be instrumental in barring premature use of specific biomarkers. Notably, courts already have some experience with molecular-level biomarkers, which indicates that they have begun establishing templates for addressing and evaluating this type of evidence. *See* Gary E. Marchant, *Genetic Susceptibility and Biomarkers in Toxic Injury Litigation*, 41 JURIMETRICS 67, 108-09 (2000). Of significance for future discussions of the use of genomic data in the courtroom, this evidence generally will not be used in isolation (for example, to establish disease association or causation), but will supplement traditional evidence of exposure, risk, and disease. As noted earlier, this reality is often overlooked in discussions of future applicability of genomics and related methods.

246. For example, where the necessary elements of a case are lacking, defendants may use biomarker data to help show lack of exposure, causation, or other elements. *See* Marchant, *supra* note 130, at 10074.

247. *See, e.g.*, Ellinger, *supra* note 131, at 63-64, 71-72. As one illustration, so-called "disease genes" may predispose individuals to disease quite apart from any chemical exposure. Examples of disease genes include the cancer genes BRCA1 and BRCA2, which may confer up to fifty percent lifetime risk for some cancers in the absence of any environmental influences. *See* Fabio Marroni et al., *Penetrances of Breast and Ovarian Cancer in a Large Series of Families Tested for BRCA1/2 Mutations*, 12 EUR. J. HUM. GENETICS 899 (2004); *see also* Susan R. Poulter, *Genetic Testing in Toxic Injury Litigation: The Path to Scientific Certainty or Blind Alley?*, 41 JURIMETRICS 211, 213-21 (2001); Marchant, *supra* note 245, at 68. As another element of background risk, genetic and molecular biomarkers may provide evidence of an alternative chemical or environmental cause—damage caused by factors unrelated to the defendant's toxic agent. *See id.* at 97-98**.** Moreover, genetic tests may detect polymorphisms that may make some plaintiffs more *resilient* to particular toxic exposures.

248. For instance, even where monitoring is recognized as an independent cause of action, a plaintiff generally would need to prove that the defendant caused a significant exposure to a proven hazardous substance, placing the plaintiff at significant risk of serious disease such that a reasonable physician would prescribe a monitoring regime different from one that would have been prescribed in the absence of the exposure. Moreover, the negligence element, while not required in all jurisdictions, may permit various defenses to causation that would be unavailable under a strict liability theory.

floodgates fears may well prove overstated, and we may find that as science uncovers new risks and injuries, the legal system is ill-equipped to respond. Only in the future will we know whether we will face "unbridled liability," or conversely "risks without remedies." If either proves true, we may then need to consider complementary administrative[249] or regulatory[250] strategies to help strike the balance between deterrence and legal restraint in an age of meteoric scientific change.

CONCLUSION

The genomic revolution will call into question longstanding presumptions about the nature of "risk" and remediable "injury" in the law of toxic torts. Although a new generation of powerful biological evidence is soon to enter the courtroom, the legal system is unprepared for this emerging reality.

As the medical world leaps forward to prevent and treat disease at the subcellular level, the law's traditional focus on overt, symptomatic disease is increasingly out of step with science. New constellations of biological markers may indicate that bodily integrity has been compromised well before the appearance of classic symptoms. By forcing plaintiffs to attain late-stage injury before seeking remedies, current toxic tort law may actually discourage medical interventions that could benefit both defendants and plaintiffs. If the

249. For example, if a particular exposure were to result in a "crisis" situation in the future, an argument could be made for experimenting with certain mechanisms employed in the asbestos context. Such mechanisms include "deferral registries" which allow cases of presently impaired plaintiffs to be processed prior to those of the unimpaired. *See generally* Schuck, *supra* note 42 (discussing use of deferral registries in asbestos litigation). However, in the future, medical monitoring would need to be incorporated into such a scheme, and the criteria for moving plaintiffs from the slow to the fast track would need to account for the emerging availability of early diagnosis and treatment capabilities. For additional discussions of administrative mechanisms for addressing harms from toxic exposure, see, for example, Klein, *supra* note 18, at 33-38 (arguing for a limited administrative system to fund medical monitoring costs for individuals who have been exposed to toxic substances but who cannot avail themselves of the tort system); Albert C. Lin, *Beyond Tort: Compensating Victims of Environmental Toxic Injury*, 78 S. CAL. L. REV. 1439, 1527-28 (2005) (supporting an administrative system for addressing problems of toxic exposure, but cautioning that such a system is premature for the foreseeable future).

250. For example, questions such as the availability of medical monitoring under the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA), 42 U.S.C. § 9607(a)(4)(B) (2007), will become more important, as will the question whether state workers compensation tribunals may order employers to engage in monitoring, *see, e.g.*, Goasdone v. Am. Cyanamid Corp., 808 A.2d 159, 164 (N.J. Super. Ct. Law Div. 2002), or whether the Food and Drug Administration's equitable powers extend to monitoring orders, *see* 21 U.S.C. § 332(a) (2007) ("The district courts of the United States . . . shall have jurisdiction, for cause shown to restrain violations of [the substantive provisions of the Food, Drug, and Cosmetic Act]."). Notably, however, incorporating the new generation of genetic and molecular data into public law frameworks will be no less challenging than for the private law sphere. *See* Grodsky, *supra* note 59, at 212-20, 234-36.

law remains wedded to conventional notions of injury, it will ignore the fruits of a scientific revolution and thus may forego preventive opportunities as yet unimagined.

Indeed, newly identified risks and injuries may call for certain intermediate legal remedies. Medical monitoring, in many ways an elegant solution to the problem of latent harm, enjoys synergies with emerging science and may promote efficiencies that have been overlooked. Despite the fact that the courts, perhaps haunted by the asbestos crisis, have retreated from monitoring and other nontraditional tort claims, the monitoring remedy may be more worthwhile and less speculative than a new generation of claims for mental distress, enhanced risk, or other less pragmatic approaches.

At this juncture, we do not know which suites of molecular markers will prove most useful in the courtroom. And admittedly, these transformative technologies will present herculean challenges for the legal system. As biological evidence moves to the subcellular level, experts, parties, and courts will strenuously debate its meaning. Yet rather than simply retreat from the sheer magnitude and complexity of the challenges presented, each situation must be debated and decided on its own—biomarker by biomarker—within a responsive legal framework. By taking cues from the scientific world, perhaps jurists, scholars, and policymakers can transform the "latency problem" into an opportunity—to promote public health, limit liability awards, and prevent disease, pain, and loss. This transformation is essential if the law is to fully embrace the benefits of the ongoing scientific revolution.