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DNA DIAGNOSTIC TECHNOLOGY: PROBING THE PROBLEM OF CAUSATION IN TOXIC TORTS

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INTRODUCTION

Courts in the United States are being confronted with an increasing number of tort actions in which claimants allege present harm, or risk of future harm, from exposure to toxic substances.[1] The unique challenges of toxic tort litigation,[2] in particular the seemingly intractable problem of

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1. Huber, *Environmental Hazards and Liability Law*, in LIABILITY: PERSPECTIVES AND POLICY 136 (R. Litan & C. Winston eds. 1988) (list of 14 recent toxic tort cases with brief descriptions of each). *See generally* Black, Zimmerman, Bailey & Westendorf, *Toxic and Hazardous Substances and Environmental Law: 1987 Survey*, 23 TORT & INS. L.J. 455 (1988).

2. *See* Gold, *Causation in Toxic Torts: Burdens of Proof, Standards of Persuasion, and Statistical Evidence*, 96 YALE L.J. 376 n. 1 (1986); Note, *Developments in the Law: Toxic Waste Litigation*, 99 HARV. L. REV. 1458, 1603 (1986); Kanner, *Emerging Conceptions of Latent Personal Injuries in Toxic Tort Litigation*, 18 RUTGERS L.J. 343, 343–46 (1987). A relatively comprehensive list of characteristics of toxic tort actions is given in M. DORE, LAW OF TOXIC TORTS § 2.02 (1987) (quoting selectively):

1. The injuries involved allegedly arose from exposure to a harmful substance.
2. The nature of the exposure was such that there is a significant risk that a large number of people suffered comparable injuries.
3. The full consequences of the exposure may not be immediately apparent (long latency periods).
4. The connection between the exposure and the injuries suffered is open to dispute, either because of questions about the nature of the substance (was it harmful), the nature of the exposure (was it significant) or the nature of the affliction (was it one that can derive from multiple causes).
5. The identity of the particular party responsible for the agent allegedly causing injuries is an open question.
6. The evidence used to establish causation is on the frontiers of science.
7. The injuries suffered are so serious and/or the claimant's situation so sympathetic that traditional legal defenses such as contributory negligence, statute of limitations, etc., are evaluated extremely critically by the court.
8. The actions . . . raise serious administrative and legislative problems for the judiciary. . . .
9. Insurance coverage disputes are or will be present
10. The facts involved give rise to additional potential liability exposure such as possible application of the criminal law or imposition of individual responsibility upon corporate officials.

relating cause and effect,[3] have placed the legal system under considerable stress.[4] New technologies have created many of the problems facing courts in toxic tort litigation, and the judicial system (and appropriate administrative agencies) increasingly will need to embrace technology in order to deal effectively with such problems. Frequently, technological solutions to technology-generated problems appear only after frustrating lag periods, but this should not deter courts from embracing such technologies when they do appear.

DNA diagnostic testing may represent one such technology. Although so far courts have dealt with only one relatively narrow application of DNA diagnostic technology, "DNA fingerprinting,"[5] a much broader array of applications inevitably will need to be addressed. DNA diagnostic technology has potential relevance to any injury with a genetic component,[6] and it is likely that uncertainty about causation of many diseases, such as cancer and birth defects, that are encountered in toxic tort litigation can be reduced through application of DNA diagnostic technology. Widespread use of DNA testing in toxic tort litigation is perhaps several years in the future, but it is not too early to begin examining some of the general technical and legal issues likely to confront the courts.

This article describes the new DNA diagnostic technology, its admissibility in court, and the prospects and problems associated with its use in toxic tort litigation. In Section I of the Article, I examine the biological principles underlying DNA diagnostic technology. Although the

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3. *See infra* notes 173 & 175 and accompanying text.

4. Some commentators have argued that a legal system constrained by traditional tort doctrines is ill-equipped to deal with toxic tort litigation. *See* Ayers v. Jackson Township, 525 A.2d 287, 299 (1987) ("The overwhelming conclusion of the commentators" is that the legal system has not adapted to the problems of toxic tort litigation). *See generally* Brennan, *Causal Chains and Statistical Links: The Role of Scientific Uncertainty in Hazardous-Substance Litigation*, 73 CORNELL L. REV. 469 (1988); Rosenberg, *The Causal Connection in Mass Exposure Cases: A "Public Law" Vision of the Tort System*, 97 HARV. L. REV. 849, 854 (1984); Trauberman, *Statutory Reform of "Toxic Torts": Relieving Legal, Scientific, and Economic Burdens on the Chemical Victim*, 7 HARV. ENVTL. L. REV. 177, 188–89 (1983).

Some commentators have suggested that regulatory agencies might be better suited for resolution of toxic injury claims. Trauberman, *supra*, at 215; Brennan, *supra*, at 523–33 (Brennan's proposal for an administrative "Science Panel" is structured such that the Panel could function either as a replacement for, or as a supplement to, the tort system). For purposes of this Article I will assume that the tort litigation system will remain as the primary mechanism for compensation of individuals injured by toxic agents. However, even an administrative compensation apparatus would face difficult problems in establishing causation in toxic injury cases.

5. *See infra* notes 122–52 and accompanying text.

6. *See infra* notes 48–53 and accompanying text.

various forms and applications of this technology may appear complex, an understanding of a limited number of biological facts can provide non-scientists with the requisite foundation upon which to evaluate the uses and limitations of DNA diagnostic technology in tort litigation. After reviewing these background biological principles, I discuss two related but distinct forms of DNA diagnostic technology: those DNA tests that provide evidence that a victim's genetic material possesses structural characteristics consistent with disease or increased risk of disease, and those tests that provide evidence that a particular chemical or physical agent has, in fact, interacted with the victim's genetic material in some manner.

Section II deals with the threshold issue of admissibility of DNA diagnostic test results as evidence in litigation. I review the legal standards for admissibility of scientific evidence, comparing standards in which admissibility turns on general acceptance of the technology in the scientific community with standards in which courts delve more deeply into the reliability and probative value of particular technologies and in which general acceptance is only one among several factors to be considered for admissibility. I then evaluate the ability of courts to grasp the relevant principles of complex technologies and conclude that courts need not be constrained by the general acceptance standard when evaluating the admissibility of DNA diagnostic test results.

In Section III, I assess the potential utility of DNA diagnostic technology in toxic tort litigation. I suggest that DNA testing should decrease the uncertainty inherent in decisions regarding legal causation when such decisions must be based on probabilistic evidence. Likewise, DNA testing may enable courts to focus on "actual" injury to a plaintiff's genetic material, rather than the troublesome concept of compensation for "latent" injury. Finally, I discuss a problem that may confront many toxic tort litigants subjected to DNA diagnostic testing. Many DNA tests have the potential to reveal not only information of direct relevance to the litigation, but additional information of profoundly disturbing personal significance, information that may be only peripherally related or even unrelated to the physical harm that has been placed at issue in litigation. I suggest several mechanisms of judicial management that might help courts to avoid the ethically troublesome imposition of unwanted knowledge on such litigants.

I. DNA DIAGNOSTIC TECHNOLOGY

A. Background

1. Biological and Physical Characteristics of DNA

DNA molecules encode instructions for the form and function of all living organisms on earth.[7] Each cell in the human body houses approximately five linear feet of DNA, organized as forty-six separate complexes (chromosomes) and localized within a defined subcellular structure (the nucleus).[8] The genetic information represented by the DNA within a given cell in an individual human being represents a relatively accurate copy of the DNA located within every other cell of that same individual.[9] Thus, each cell, though it is only one among the many trillions of cells comprising the human body, houses the totality of genetic information (the genome) directing the form and function of that human being.[10]

DNA in its native configuration in the cell nucleus is double stranded, that is, composed of two single stranded DNA molecules wrapped

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7. *See generally* 1 J. WATSON, N. HOPKINS, J. ROBERTS, J. STEITZ & A. WEINER, MOLECULAR BIOLOGY OF THE GENE 65–94 (4th ed. 1987) [hereinafter WATSON I]. The structure of DNA was reported in a seminal paper by James Watson and Francis Crick in 1953. Watson & Crick, *Molecular Structure of Nucleic Acids: A Structure for Deoxyribose Nucleic Acid*, 171 NATURE 737 (1953). Since an understanding of the structure of DNA led almost immediately to testable hypotheses concerning the mechanisms of replication (necessary for cell division) and function of DNA, the Watson & Crick paper can be viewed as the beginning of modern molecular biology. *See* WATSON I, *supra*, at 91; Watson & Crick, *Genetical Implications of the Structure of Deoxyribonucleic Acid*, 171 NATURE 964 (1953).

8. *See generally* E. D. DE ROBERTIS & E. M. DE ROBERTIS, CELL AND MOLECULAR BIOLOGY 355–58, 378 (8th ed. 1987); B. LEWIN, GENES 641 (3d ed. 1987). Circulating red blood cells, having lost their nuclei during the process of red blood cell maturation, do not contain DNA. *Id.* However, other cells in the blood ("white cells") do possess nuclei and are therefore capable of providing a ready source of DNA for diagnostic studies.

9. 2 J. WATSON, N. HOPKINS, J. ROBERTS, J. STEITZ & A. WEINER, MOLECULAR BIOLOGY OF THE GENE 758 (4th ed. 1987) [hereinafter WATSON II]. *See also infra* note 70 and accompanying text. In reality, some limited rearrangements and modifications of DNA are known to accompany the differentiation and maturation of certain cell lineages. *Id.* at 758, 853–67. Also, small differences between cells occur as mistakes are made in the replication or repair of DNA. WATSON I, *supra* note 7, at 17–19, 339–54. However, for purposes of DNA diagnostic technology, a given sample of human cells (e.g., a tumor biopsy or a blood sample) may be considered to provide a homogeneous set of DNA "carbon copies" for analysis. *Id.* at 17–19. *See also infra* note 225.

10. For informative "popularized" summaries of the biology of heredity and the scientific principles underlying DNA diagnostic technology, see Thompson & Ford, *DNA Typing*, TRIAL, Sept. 1988, at 56; Jaroff, *The Gene Hunt*, TIME, March 20, 1989, at 62.

together in a double helix that may be analogized to a twisted ladder.[11] Each side of the ladder is composed of alternating sugar and phosphate molecules, and extending from each sugar molecule toward the center of the ladder is a so-called nitrogenous base.[12] Each nitrogenous base extending inward from the left side of the ladder is weakly bonded to a corresponding nitrogenous base extending inward from the right side of the ladder.[13] Thus, the rungs of the DNA ladder are composed of pairs of nitrogenous bases, and the weak bonds between the nitrogenous bases provide the necessary force holding together the two strands of DNA in the double helix.[14] There are four different nitrogenous bases in DNA: A, T, G, and C.[15] The bonding characteristics of the four bases represent the basis for much of DNA diagnostic technology. Thus, A and T bond readily to each other, but neither bonds readily to either G or C; likewise, G and C bond readily to each other, but neither bonds readily to A or T.[16] Any given rung in the DNA ladder therefore will be represented by the complementary base pair A-T or G-C.[17] The linear sequence of bases along one DNA strand in the double helix must of necessity be represented by a corresponding complementary sequence of bases in the opposite strand; a significant degree of mismatch would prevent the two strands of the double helix from annealing or "hybridizing" to each other under prevailing conditions in the cell nucleus.[18]

Proteins are a major structural component of cells, and are the major players in the assembly and maintenance of cells, tissues, organs and organisms.[19] Thus, biological form and function depend to a large degree on where, when, and how many proteins of a given type are synthesized in particular populations of cells.[20] The structure and function of each protein is in turn coded in a specific sequence of bases in a defined length of DNA. Such a protein-coding stretch of DNA is termed a gene.[21] The cellular machinery required to transcribe and translate a sequence of several hundred to several thousand DNA bases into a func-

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11. WATSON I, *supra* note 7, at 240–41.
12. *Id.* at 241.
13. *Id.* at 241–44.
14. *Id.*
15. Adenine (A), thymine (T), guanine (G), and cytosine (C). *Id.* at 241.
16. *Id.* at 241–44.
17. *Id.* at 241.
18. B. LEWIN, *supra* note 8, at 57–60.
19. *Id.* at 4–13; K. DRLICA, UNDERSTANDING DNA AND GENE CLONING: A GUIDE FOR THE CURIOUS 194 (1984).
20. *See* E. D. DE ROBERTIS & E. M. DE ROBERTIS, *supra* note 8, at 591, 594; Ross, *The Turnover of Messenger RNA*, SCI. AM., April 1989, at 48.
21. *See, e.g.*, E. D. DE ROBERTIS & E. M. DE ROBERTIS, *supra* note 8, at 506–14.

tional protein is complex and accurate.[22] A change in even one base in the DNA can lead to drastic changes in protein shape and function.[23]

DNA is generally represented in print as a sequence of bases in one of the two strands of the double helix; the complementary strand is of course readily determinable using the complementarity rule that A always binds to T and G always binds to C. Thus, a short region within a hypothetical gene might be represented: CATACTTAGGAG. For purposes of illustration, the sequence is made up of four English words—"cat," "act," "tag," and "gag," in that order. The complementary sequence can be shown thus:

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CATACTTAGGAG
||||||||||||
GTATGAATCCTC
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Since this sequence of bases is quite short, even one mismatch, caused by the depletion or replacement of a single base, would prevent the two strands from hybridizing under appropriate laboratory conditions.[24] Hybridization of progressively longer sequences can accommodate progressively greater numbers of mismatches, although the strength of bonding between the strands would be less than that for perfectly matched sequences of the same length.[25] However, even a perfectly matched and very long (thousands of base pairs) DNA double helix can be separated or "denatured" into two single strands by application of heat or other relatively harsh conditions in the laboratory.[26]

DNA diagnostic technology is concerned with detection of mutations that result in specific changes in sequences of bases in the human genome. Frequently the goal is detection of specific changes in sequences that have been associated with disease or increased risk of disease.[27] The scope of mutational change can range from alterations of single bases—substitutions, deletions, and insertions—to large-scale

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22. *Id.* at 527–93.

23. For example, a single base change in the gene coding for hemoglobin may lead to a single amino acid change in the hemoglobin protein, leading to sickle cell anemia. *Id.* at 47. Similarly, single base changes in some normal human genes can cause such genes to become significant contributors to formation of cancers. *See* WATSON II, *supra* note 9, at 1067–69.

24. T. MANIATIS, E. FRITSCH & J. SAMBROOK, MOLECULAR CLONING: A LABORATORY MANUAL 227 (1982).

25. B. LEWIN, *supra* note 8, at 369–77.

26. E. D. DE ROBERTIS & E. M. DE ROBERTIS, *supra* note 8, at 35.

27. *See infra* notes 48–53 and accompanying text.

changes involving thousands of bases or even entire regions of chromosomes.[28]

2. *Manipulation of DNA*

The advent of DNA diagnostic technology followed from the development of the means to cut and splice, or "recombine," chosen pieces of DNA from any source and to multiply a single piece of DNA into vast numbers of identical copies. This procedure is called DNA cloning.[29] Recombinant DNA ("rDNA") technology, including DNA cloning, made possible for the first time a detailed analysis of genes at the base sequence level.[30]

a) Sequence-specific cutting of DNA

DNA isolated from blood or other sample material is composed of exceptionally long fragments, many thousands or even millions of base pairs long, which are not useful for many types of rDNA manipulations such as DNA cloning.[31] To create fragments of workable size, the DNA is digested with restriction enzymes, which are proteins capable of cutting DNA into fragments at specific points. The restriction enzyme *EcoRI*, for example, recognizes the sequence GAATTC and cuts the double helix wherever this sequence occurs.[32] Hundreds of restriction enzymes, each recognizing a specific sequence, have been isolated from

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28. *See generally* WATSON I, *supra* note 7, at 339–57. With respect to the hypothetical sequence shown in the text, single-base changes ("point mutations") could be represented as follows (focusing on the word "TAG" in the four-word sequence):

CATACTTAGGAG	(normal sequence)
CATACTTTGGAG	(substitution: TAG to TTG)
CATACTTGGAG	(deletion: TAG to TG)
CATACTTAAGGAG	(insertion: TAG to TAAG)

Mutations involving more than one base might appear thus:

CATGAG	(deletion: ACTTAG deleted)
CATACTGGGTAGGAG	(insertion: GGG inserted between ACT and TAG)
CATGATTCAGAG	(rearrangement: inversion of the sequence ACTTAG)

In addition, the 12-base sequence shown above might be part of a much larger sequence that is itself deleted, inserted elsewhere in the genome, or otherwise rearranged in some fashion.

29. *Id.* at 88–89, 208–09.

30. *Id.*

31. *Id.* at 88.

32. *Id.* at 89. *See also id.* at 266–69.

bacteria and other organisms, and are available as tools for rDNA technology.[33]

b) Separation of DNA fragments on the basis of size by gel electrophoresis

A DNA diagnosis may require identification and characterization of one or several specific fragments out of the millions of fragments generated through digestion of sample DNA with a restriction enzyme.[34] To accomplish this, it is usually necessary to sort the fragments according to their various lengths. In one of the most common techniques, DNA is inserted into one end, designated the top, of gel-like material.[35] The gel is then placed in an electrical field with the positive pole at the bottom end of the gel.[36] The negatively charged DNA fragments travel toward the bottom or positively charged end of the gel. The gel functions as a molecular sieve; smaller fragments are able to travel faster through the irregular gel spaces than are the larger fragments.[37] After a given interval of time, the DNA fragments will have been arranged into a continuous size distribution, with the smallest fragments at the bottom and the largest fragments at the top of the gel.[38]

c) Detection of specific fragments

Detection of specific fragments is facilitated by transfer of the size-fractionated DNA fragments out of the gel to a more accessible medium.[39] In one commonly used method, Southern blotting,[40] the DNA is denatured (double-stranded fragments are converted to single-stranded fragments) and then driven out of the gel by capillary action onto the

33. *Id.* at 88. Recently, scientists have developed the means to engineer some types of restriction enzymes to cut at *any* desired sequence. *See* Corey & Schultz, *Generation of a Hybrid Sequence-Specific Single-Stranded Deoxyribonuclease*, 238 SCIENCE 1401 (1987).

34. *See infra* notes 54–74 and accompanying text.

35. B. LEWIN, *supra* note 8, at 75. The gel is often prepared from agarose (an extract of seaweed) in a manner similar to that used for preparation of common household gelatin. That is, the powdered form of agarose is dissolved in hot water, then cooled to room temperature, whereupon the solution solidifies to form a gel.

36. *Id.*

37. *Id.*

38. *Id.*

39. *Id.* at 360–61.

40. *See* D. SUZUKI, A. GRIFFITHS, J. MILLER & R. LEWONTIN, AN INTRODUCTION TO GENETIC ANALYSIS 312 (3d ed. 1986) (includes a brief description of the technique). *See also* Southern, *Detection of Specific Sequences Among DNA Fragments Separated by Gel Electrophoresis*. 98 J. MOLECULAR BIOL. 503 (1975). This is the original article describing the Southern blot method.

surface of a solid support, usually a specially prepared paper or nylon membrane.[41] The relative positions of the fragments in the gel are retained following transfer to the paper or nylon membrane.[42]

At this point, specific DNA fragments can be detected with a DNA probe.[43] A probe is any cloned DNA sequence that has been "tagged" in some fashion, allowing subsequent visualization of any location on the solid support to which the probe has become bound.[44] The probe will bind to any fragment in the size-fractionated DNA that contains a base sequence complementary to all or a portion of the base sequence in the probe.[45] Some DNA diagnostic tests are designed to detect specific size patterns of fragments to which the probe has hybridized,[46] while other tests are designed to distinguish the presence or absence of hybridization.[47]

B. DNA Diagnostic Tests

1. Detection of Disease or Increased Risk of Disease

The list of diseases associated or partially associated with specific variants of DNA sequences is long and rapidly growing longer.[48] Any disease with a genetic basis, where the relevant gene or genes have been identified and cloned, is a candidate for application of DNA diagnostic technology. Even when the genetic basis for a disease remains unknown, DNA sequences often found in persons afflicted with the disease can be identified.[49]

Numerous genetic diseases are linked to defects in a single gene.[50] On the other hand, diseases associated with the circulatory system (e.g., heart disease, strokes) and many cancers are under the influence of

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41. WATSON I, *supra* note 7, at 608–09.
42. *Id.* at 609.
43. B. LEWIN, *supra* note 8, at 359–61.
44. *Id.*
45. *Id.*
46. *See infra* notes 54–61 and accompanying text.
47. *See infra* notes 62–74 and accompanying text.
48. *See generally* Caskey, *Disease Diagnosis by Recombinant DNA Methods*, 236 SCIENCE 1223 (1987); Landegren, Kaiser, Caskey & Hood, *DNA Diagnostics—Molecular Techniques and Automation*, 242 SCIENCE 229 (1988) [hereinafter Landegren]; Watkins, *Restriction Fragment Length Polymorphism (RFLP): Applications in Human Chromosome Mapping and Genetic Disease Research*, 6 BIOTECHNIQUES 310 (1988); White & Lalouel, *Chromosome Mapping with DNA Markers*, SCI. AM., Feb. 1988, at 40. For purposes of this paper, I include genetically based birth defects as "diseases."
49. B. LEWIN, *supra* note 8, at 80–82.
50. *See* Caskey, *supra* note 48, at 1223–24; Landegren, *supra* note 48, at 232–33.

multiple genetic factors.[51] For example, the genetic bases for predisposition to cardiovascular disease might include genes involved in such diverse activites as cholesterol metabolism, regulation of blood pressure, and maintenance of blood vessel integrity.[52] Similarly, over thirty genes have been implicated in the development of various forms of cancer.[53] Thus, a wide array of DNA sequences has been associated with human disease and some of these diseases, such as various forms of cancer, figure prominently in toxic tort litigation. Below I review the major types of DNA diagnostic tests that are relevant for inquiries into disease causation in toxic tort litigation.

a) Restriction fragment length polymorphisms

Restriction enzymes are well-suited to detection of altered base sequences in DNA. This is due to the sequence specificity of restriction enzymes; for example, a change of even one base in a sequence of bases recognized by *EcoRI* will prevent the enzyme from cutting the DNA at that location.[54] Even a point mutation, consisting of an alteration, deletion, or insertion of a single base,[55] is capable of creating or destroying a

51. Landegren, *supra* note 48, at 233, 234–35.

52. *Id.* at 233.

53. WATSON II, *supra* note 9, at 1045. "Oncogenes" are genes that have been implicated as causative agents in one or more types of cancer. *See id.* at 1059–60, 1072–74; Weinberg, *A Molecular Basis of Cancer*, SCI. AM., Nov. 1983, at 126 [hereinafter Weinberg I]. Although not all oncogenes are dysfunctional in any given tumor, it is probable that some and perhaps most cancers develop as a result of defects in more than one oncogene in a single cell. *See* Yuspa & Poirier, *Chemical Carcinogenesis: From Animal Models to Molecular Models in One Decade*, 50 ADVANCES CANCER RES. 25, 36, 38 (1988); Marx, *Many Gene Changes Found in Cancer*, 246 SCIENCE 1386 (1989). Many oncogenes are thought to represent mutationally altered ("activated") versions of normal genes that are involved in the control of cell growth and proliferation; these altered versions actively promote excessive cell division and other malignant characteristics. *See generally* B. LEWIN, *supra* note 8, at 698–715; Weinberg I, *supra*. More recently, "anti-oncogenes," thought to constrain uncontrolled growth under normal conditions, have been discovered. Weinberg, *Finding the Anti-Oncogene*, SCI. AM., Sept. 1988, at 44 [hereinafter Weinberg II]. Deletion or mutational *in*activation of such genes could cause cells to proliferate out of control. *Id.* For several of the oncogenes and anti-oncogenes, *specific* point mutations at *specific* locations in the genes have been implicated in activation or inactivation. *See* Weinberg I, *supra*; Weinberg II, *supra*. This knowledge allows the design of highly specific and discriminating DNA probes for diagnostic purposes. Of course, even probes for large portions of a gene would be highly diagnostic if the disease state (or elevated risk of disease) were due to gene *deletion*, since absence of hybridization would then be diagnostic in the same way that absence of hybridization with short probes can be diagnostic for point mutations. *See infra* notes 62–74 and accompanying text.

54. WATSON I, *supra* note 7, at 266–69.

55. *Id.* at 444. *See also supra* note 28 and accompanying text.

restriction enzyme cutting-site in DNA.[56]

If a sample of human DNA is digested with the restriction enzyme *EcoRI*, thousands of fragments will be generated. *EcoRI* recognizes the six base sequence GAATTC, which occurs thousands of times in the three billion base pairs comprising the human genome.[57] To determine whether one or even several specific *EcoRI* sites have been altered, the digested DNA would be size-fractionated by electrophoresis, transfered to a solid support (such as a sheet of nylon membrane), and hybridized to a selected DNA probe.[58] For example, a DNA probe complementary to the DNA base sequence coding for insulin could be selected. Depending on the number of *EcoRI* sites within or adjacent to the insulin gene, the probe would normally bind to one or several fragments of specific size. If any of these *EcoRI* sites have been altered (or new *EcoRI* sites created) by changes in base sequence, or if stretches of DNA sequences were added or deleted between *EcoRI* sites, the pattern of fragments identified by the insulin probe would be altered. Differences between individuals in the pattern of fragments detected by a particular DNA probe are termed "restriction fragment length polymorphisms" ("RFLPs").[59]

RFLPs have been associated with over twenty-five genetic diseases, including Huntington's disease, cystic fibrosis, familial Alzheimer's disease, three types of muscular dystrophy, manic depressive illness, and several forms of cancer.[60] This list will undoubtedly lengthen with the

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56. *See* Watkins, *supra* note 48, at 312.

57. WATSON I, *supra* note 7, at 266–69; Landegren, *supra* note 48, at 229.

58. *See supra* notes 34–47 and accompanying text.

59. Watkins, *supra* note 48, at 310.

60. *Id.* at 313. It should be noted that the probes used for RFLP diagnosis need not be complementary to known or identified genes, although the strong association of a particular RFLP with a disease suggests the chosen probe is complementary to a DNA sequence near to, if not within, a gene at least partly responsible for the disease. *See generally* White & Lalouel, *supra* note 48.

Not all DNA consists of base sequences coding for protein. Many DNA sequences, for example, are known to exist as multiple copies scattered throughout the human genome and to have no known function. WATSON I, *supra* note 7, at 668–72. DNA probes specific for such sequences frequently identify complex patterns of size-fractionated fragments that have been compared to the "bar codes" utilized by retail merchants. Marx, *DNA Fingerprinting Takes the Witness Stand*, 240 SCIENCE 1616 (1988). These complex RFLP patterns have been found to be highly specific for individual human beings, and represent one of the bases for the so-called "DNA fingerprinting" technology that has been utilized for identification of individuals in a variety of criminal and paternity suits. *Id.* at 1616–19.

Although this Article covers some of the technical and legal issues raised by DNA fingerprinting techniques, this is done only for purposes of illustration. DNA fingerprinting is concerned with identification of *individuals*. The focus of the present Article is on the broader forms of DNA diagnostic technology concerned with identification of *genetic-based diseases* and their causative agents. For general reviews of the technical aspects and legal implications of DNA fingerprinting, see *infra* note 122.

anticipated rapid growth in the number of DNA probes assigned to specific locations in the human genome.[61]

b) Direct detection of base sequence changes

Although it has many useful applications, RFLP analysis depends on interpretation of patterns of fragment sizes and requires relatively extensive manipulation of DNA, including restriction enzyme digestion, gel electrophoresis, and transfer of DNA to solid supports.[62] Other techniques have been designed to detect base sequence changes, including point mutations, based on the presence or absence of hybridization of specific DNA probes to DNA sequences of interest. DNA probes can be designed that are long enough to represent unique sequences in the human genome, but short enough (about twenty bases in length) that a single base mismatch will prevent hybridization of the probe to the target sequence.[63] A variety of methods have been designed to detect the presence or absence of hybridization of such probes to target sequences in human DNA.[64] For example, a recently developed method utilizes two short probes complementary to sequences immediately adjacent to each other.[65] The probes are each tagged with a different fluorescent dye, and the two dyes cooperate to emit a particular wavelength of light only when they are in close proximity.[66] Any change in base sequence preventing one or both of the probes from hybridizing will result in an easily detectable absence of the particular wavelength.[67] This technique avoids the cumbersome radioactive tags and elaborate DNA manipulations that have been required in many of the other procedures.[68]

For many purposes, five to ten micrograms of DNA, such as would be present in a one milliliter blood sample, are sufficient for RFLP analysis

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61. Watkins, *supra* note 48, at 310.

62. *See supra* notes 54–59 and accompanying text.

63. WATSON I, *supra* note 7, at 604–05; Caskey, *supra* note 48, at 1223–24. Such short DNA probes ("oligonucleotide probes") for any particular base sequence can be synthesized readily in the laboratory.

64. Landegren, *supra* note 48, at 229–30. Several of these methods are applicable to DNA in solution, and therefore, they do not require the DNA to be subjected to electrophoresis and transfer to solid supports. *Id.*

65. Greenberg, *Scientists Detect DNA Using New Fluorescent Probe Method*, GENETIC ENGINEERING NEWS, Feb. 1989, at 1, 27.

66. *Id.* A more extensive description of this approach (using, however, more traditional methods to tag the DNA probes) can be found in Landegren, Kaiser, Sanders & Hood, *A Ligase-Mediated Gene Detection Technique*, 241 SCIENCE 1077 (1988).

67. Greenberg, *supra* note 65.

68. *Id.*

or for direct detection of base sequence changes.[69] However, there may be many situations in which only miniscule amounts of DNA will be available. For example, the mutation of interest may be present in only a small fraction of the individual's cells.[70] Or, the cells to be tested may be located in tumors or in other locations in the body where it may not be practical or feasible to obtain amounts of DNA comparable to that contained in a one milliliter blood sample.[71] In such cases, DNA sequences of interest may be present in insufficient quantities to generate detectable signals with traditional techniques. However, the recently developed polymerase chain reaction ("PCR") technique allows defined DNA sequences from even a single gene to be amplified several million-fold.[72] For example, even small needle biopsies of tumors, or blood samples containing only one or several cells possessing diagnostic mutations,[73] may provide sufficient material for DNA diagnostic purposes.[74]

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69. Landegren, *supra* note 48, at 231.

70. If a mutational change responsible for disease or increased risk of disease is present in the nucleus of a fertilized human egg, that mutation will be duplicated in all cells of the resulting individual. This is due to the fact that each cell in the human body represents the end-point in a lineage of cell divisions that can be traced back to the original "cell" (fertilized egg). *See generally* L. BROWDER, DEVELOPMENTAL BIOLOGY 31–36, 41 (2d ed. 1984). Each time a cell ("parent") divides to give rise to two new cells ("daughters"), the DNA in the parent cell nucleus undergoes a high-fidelity doubling and is allocated to each daughter nucleus in such a way that the DNA in each daughter nucleus represents, for practical purposes, an exact copy of the DNA that existed in the parent cell nucleus. *See* B. LEWIN, *supra* note 8, at 22–24, 312–34. On the other hand, exposure of an adult employee to a toxic chemical in the workplace may result in mutational damage to only a small number of cells. Thus, although a one milliliter blood sample from such an individual may provide several thousand nucleated cells, only a small fraction of these cells may carry the diagnostic mutation.

71. The "at risk" cells may be located not in the blood, but in the skin, lungs, digestive system, or other locations depending on the nature of the exposure. Many, and perhaps most, malignant tumors arise from single cells that have undergone mutational changes leading to uncontrolled cell division (and other abnormal behavior depending on the tumor type). WATSON II, *supra* note 9, at 1058–61; Marx, *supra* note 53, at 1386. In the same way that each cell in the human body would carry a mutation present in the fertilized egg, each malignant cell in a tumor would carry the mutations responsible for malignant transformation of the founder cell. *See supra* note 70.

72. Polymerase enzymes are used to generate millions of copies of the DNA sequence of interest, leading to a corresponding increase in the strength of the detection signal. *See* Landegren, *supra* note 48, at 231; Marx, *Multiplying Genes by Leaps and Bounds*, 240 SCIENCE 1408 (1988); Appenzeller, *Democratizing the DNA Sequence*, 247 SCIENCE 1030 (1990).

73. *See supra* note 70.

74. Landegren, *supra* note 48, at 231; Marx, *supra* note 72.

2. "Signature" Tests

Evaluation of changes in DNA base sequence by analysis of RFLPs or by direct detection methods can help alleviate some of the causation problems in toxic tort litigation.[75] However, it is unlikely that these methods will provide "smoking guns" for plaintiffs trying to prove association of injury with specific chemical or physical agents, nor are these methods likely to provide complete exculpation for defendants seeking to disprove such association. DNA diagnostic tests that could indicate unequivocally whether or not an individual's DNA had been in contact with a specific agent, or provide clear association between specific types of mutational change and exposure to specific agents, would be of immense value for the toxic tort litigation system. I will refer to such tests as "signature" tests, since they would, in a sense, read a chemical or physical agent's distinctive "signature" or "fingerprint"[76] in the DNA. The tests can be indirect by defining and measuring specific mutations associated with specific agents, or direct by demonstrating an actual physical association of the agent with the DNA. These two types of signature tests are discussed below.

a) Association of specific patterns of mutational change with specific agents

Recently, scientists have been using the "HPRT"[77] gene as an indicator of mutational damage caused by specific agents.[78] Over 1200 sequence variants of the HPRT gene resulting from exposure to various types of chemicals and radiation have been determined. "The bottom line is that in bacterial and mammalian systems each agent gives its own fingerprint of changes. In other words, when we see the changes we know what the agent was."[79] This method of analysis relies on actual sequencing of the bases in the HPRT gene. DNA sequencing, as practiced presently in most laboratories, is a relatively laborious and

75. *See infra* notes 187–211 and accompanying text.

76. The term "fingerprint" as used here should not be confused with "DNA fingerprinting." The latter term is a type of RFLP analysis used to distinguish one human being from another. *See supra* notes 54–61 and accompanying text. "Fingerprint," as used in the text here, denotes characteristic changes in the DNA that could be identified to implicate specific chemical or physical agents as responsible for mutational damage. I have chosen the term "signature" to represent this class of DNA diagnostic tests in order to avoid confusion with "DNA fingerprinting."

77. Hypoxanthine phosphoribosyltransferase.

78. Marx, *Detecting Mutations in Human Genes*, 243 SCIENCE 737 (1989).

79. Statement of Barry Glickman, York University, Toronto, *quoted in id.* at 738.

expensive procedure.[80] Thus, the great theoretical value of this technique is somewhat limited in practice, although recent advances in automation of DNA sequencing technology hold promise for widespread application in the not-too-distant future.[81]

Another approach for detecting specific patterns of mutational changes induced by specific chemical or physical agents bypasses the need for sequencing. This method relies on a special method of electrophoresis that causes HPRT gene fragments carrying specific patterns of mutational changes (not necessarily differing in fragment length) to migrate to specific locations in a gel.[82] Patterns of mutational changes induced by particular agents can be visualized without the necessity for isolating, cloning, and sequencing individual genes.[83]

Neither of these approaches has been proven effective for cells other than those grown in laboratory dishes.[84] However, additional experience with and refinement of these and other such technologies[85] should yield practical methods for implicating specific chemical or physical agents with specific genetic injury.[86]

b) Direct evidence of chemical interaction with DNA

Many DNA-damaging chemicals form temporary or permanent associations with the DNA molecule itself.[87] Recently, scientists have developed sensitive methods to detect the presence of specific chemical additions or "adducts" to the DNA in human cells.[88] These methods can be used to detect as few as one to ten molecules of a specific chemical per billion base pairs of DNA.[89] In one study, radioactive labelling and immunologic assays were used to detect seven different types of adducts

80. *See* Prober, Trainor, Dam, Hobbs, Robertson, Zagursky, Cocuzza, Jensen & Baumeister, *A System for Rapid DNA Sequencing with Fluorescent Chain-Terminating Dideoxynucleotides*, 238 SCIENCE 336 (1987).

81. *See* Roberts, *New Sequencers to Take on the Genome*, 238 SCIENCE 271 (1987). *See also* Landegren, *supra* note 48, at 232.

82. Marx, *supra* note 78, at 738. *See also* Cariello & Thilly, *Use of Gradient Denaturing Gels to Determine Mutational Spectrum in Human Cells*, 38 BASIC LIFE SCI. 439 (1986); Johnson, *Biological Markers in Tort Litigation*, 3 STATISTICAL SCI. 367, 368–69 (1988).

83. Marx, *supra* note 78, at 738.

84. *Id.* at 737–38.

85. Several other examples are given in *id.*

86. *Id.*

87. WATSON I, *supra* note 7, at 343.

88. Weinstein, *Cigarette Smoking and its Fingerprint in DNA*, 80 J. NAT'L CANCER INST. 548 (1988). *See also* Yuspa & Poirier, *supra* note 53, at 41–45.

89. Weinstein, *supra* note 88, at 548.

in human placental tissue.[90] Three of these types of adducts were strongly correlated with smoking. Levels of smoking-related adducts were inversely associated with birth weight.[91] This and other studies suggest that these methods represent potentially powerful approaches to correlating environmental factors with specific genetic-based diseases.[92]

II. ADMISSIBILITY OF DNA DIAGNOSTIC TEST RESULTS

Before DNA diagnostic technology can be used in toxic tort litigation, the threshold issue of evidentiary admissibility must be addressed. Even if the technology were valuable for clarifying issues of causation, little benefit would accrue if the courts were unwilling to allow test results to be admitted as evidence. The following Section summarizes the evolving legal doctrines governing admissibility of scientific evidence. This summary is followed by a discussion of how these doctrines could (and should) be applied to DNA diagnostic technology.

A. Legal Doctrines Governing Admissibility of Scientific Evidence

As scientific evidence becomes increasingly important for resolution of legal issues, the courts face an increasingly palpable dilemma. Scientific evidence is necessary for resolving issues that judges and juries lacking scientific backgrounds cannot understand easily. Yet these same judges must evaluate the reliability of scientific evidence in order to determine its admissibility at trial.[93] To resolve this dilemma, courts frequently rely on the "general acceptance" standard first enunciated in *Frye v. United States*[94] (the "*Frye* test").[95] The primary alternative to *Frye*, which is to treat scientific evidence in the same fashion as other

90. Everson, Randerath, Santella, Avitts, Weinstein & Randerath, *Quantitative Associations Between DNA Damage in Human Placenta and Maternal Smoking and Birth Weight*, 80 J. NAT'L CANCER INST. 567 (1988).

91. *Id.* at 572–75.

92. Weinstein, *supra* note 88, at 548.

93. *See* Giannelli, *The Admissibility of Novel Scientific Evidence:* Frye v. United States, *a Half-Century Later*, 80 COLUM. L. REV. 1197 (1980); Black, *Evolving Legal Standards for the Admissibility of Scientific Evidence*, 239 SCIENCE 1508 (1988) [hereinafter Black I]; Black, *A Unified Theory of Scientific Evidence*, 56 FORDHAM L. REV. 595 (1988) [hereinafter Black II].

94. 293 F. 1013 (D.C. Cir. 1923).

95. *See* P. GIANNELLI & E. IMWINKELRIED, SCIENTIFIC EVIDENCE 1-14 (1986 & Supp. 1988). Most jurisdictions continue to follow the Frye test. *See* Note, *The Frye Doctrine and Relevancy Approach Controversy: An Empirical Evaluation*, 74 GEO. L. J. 1769 (1986).

evidence, has been invoked by an increasing number of courts willing to examine the validity of the reasoning underlying scientific testimony.[96] The following sections examine in greater detail these two major approaches to the question of admissibility of scientific evidence.

1. The Frye *Test*

Upholding a trial court's refusal to admit the results of an early form of polygraph lie detector test into evidence, the Court of Appeals for the District of Columbia stated in 1923:

> Just when a scientific principle or discovery crosses the line between the experimental and demonstrable stages is difficult to define. Somewhere in this twilight zone the evidential force of the principle must be recognized, and while courts will go a long way in admitting expert testimony deduced from a well recognized scientific principle or discovery, the thing from which the deduction is made must be sufficiently established to have gained general acceptance in the particular field in which it belongs.[97]

The test thus places primary emphasis on general acceptance by the relevant scientific community. The test has been defended on several grounds. General acceptance is said to lower the probability that unreliable scientific evidence will be admitted: "The requirement of general acceptance in the scientific community assures that those most qualified to assess the general validity of a scientific method will have a determinative voice."[98] Furthermore, the test is said to promote uniformity of judicial decisions concerning admissibility,[99] and to prevent inefficiency

96. *See* FED. R. EVID. 401, 403, 702 & 703. The Federal Rules test is labelled the "relevancy test" by some commentators; Black I, *supra* note 93, at 1508. *See, e.g.*, E. CLEARY, MCCORMICK ON EVIDENCE § 203, at 605 (1984 & Supp. 1987); Giannelli, *supra* note 93, at 1203. *See also* Rossi, *Modern Evidence and the Expert Witness*, 12 LITIGATION 18, 20 (1985) ("[w]ithin the last decade, courts in more than 15 jurisdictions have rejected *Frye*").

97. *Frye*, 293 F. at 1014.

98. United States v. Addison, 498 F.2d 741, 743–44 (D.C. Cir. 1974). Additionally, the test is supposed to ensure that "a minimal reserve of experts exists who can critically examine the validity of [the scientific evidence]." *Id.* at 744.

99. *See* People v. Kelly, 17 Cal. 3d 24, 130 Cal. Rptr. 144, 549 P.2d 1240, 1244–45 (1976) ("Individual judges, whose particular conclusions may differ regarding the reliability of particular scientific evidence, may discover substantial agreement and consensus in the scientific community."). *But see infra* note 103.

at trial. [100]

The *Frye* test has been criticized, however, on numerous grounds. First, there is no consensus on what degree of acceptance constitutes "general" acceptance by the scientific community.[101] Second, selection of the proper scientific field in which to examine acceptance may be problematic. Many scientific techniques span two or more scientific disciplines. DNA diagnostic technology could be evaluated at various levels by, among others, chemists, biochemists, molecular biologists, physicists, population geneticists, and medical pathologists. The selection of differing "appropriate" scientific disciplines in which to determine general acceptance could lead to inconsistent results.[102] Third, the *Frye* test has been criticized as overly conservative; it may tend to quash admission of otherwise reliable scientific evidence only because it has not yet become widely known and accepted in scientific circles.[103] Fourth, some have suggested that attempting to discern scientific "voting" patterns represents an abrogation of judicial responsibility in favor of the scientific community.[104] Finally, some commentators believe that *Frye* leads courts to focus on techniques and scientific equipment, the "*thing*[*s*] from which the deduction is made,"[105] rather than on theories or reasoning, the *manner* in which the deduction is made.[106]

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100. *See* Reed v. State, 283 Md.2d. 274, 391 A.2d 364, 371–72 (1978) ("Again and again, the examination and cross-examination of expert witnesses will be protracted and time consuming. . . .").

101. P. GIANNELLI & E. IMWINKELRIED, *supra* note 95, at 18–19. The California Supreme Court requires that a "clear majority" of the scientists in a particular field have accepted the validity of the technique. People v. Guerra, 37 Cal. 3d 385, 208 Cal. Rptr. 162, 690 P.2d 635, 656 (1984).

102. United States v. Williams, 583 F.2d 1194, 1198 (2d Cir. 1978), *cert. denied*, 439 U.S. 1117 (1979) ("Selection of the 'relevant scientific community' appears to influence the result.").

103. *See* P. GIANNELLI & E. IMWINKELRIED, *supra* note 95, at 27. *See also* Coppolino v. State, 223 So. 2d 68, 75 (Fla. Dist. Ct. App. 1968) (concurring opinion) ("Society need not tolerate homicide until there develops a body of medical literature about some particular lethal agent"), *appeal dismissed*, 234 So. 2d 120 (Fla. 1969), *cert. denied*, 399 U.S. 927 (1970). *See also Williams*, 583 F.2d at 1198 ("[U]nanimity of opinion in the scientific community, on virtually any scientific question, is extremely rare. Only slightly less rare is a strong majority.").

104. In response to this fourth criticism, however, other commentators have argued that "courts have not surrendered responsibility but rather have exercised that responsibility prudently by deferring to those best capable of judging the validity of scientific evidence." P. GIANNELLI & E. IMWINKELRIED, *supra* note 95, at 28.

105. *See supra* note 97 and accompanying text (quotation from the *Frye* opinion) (emphasis added).

106. *See, e.g.*, Black I, *supra* note 93, at 1508; Black II, *supra* note 93, at 629–30.

2. The Federal Rules of Evidence Approach

The Federal Rules of Evidence treat scientific evidence in the same manner as any other evidence: probative value is balanced against the potential dangers of misleading, prejudicing, or confusing the jury.[107] Most commentators consider the Federal Rules approach to be the principal alternative to the *Frye* test.[108] Indeed, many believe that the Federal Rules of Evidence specifically abolish the use of the *Frye* test by federal courts and by state courts in states that have adopted the Federal Rules.[109]

The Federal Rules test requires an assessment of the probative value of the evidence (including an assessment of reliability), an assessment of any countervailing dangers, and, finally, a balancing of probity against dangers.[110] In practice, general acceptance is frequently only one of a number of factors considered by courts operating under the Federal Rules of Evidence.[111] It is possible for reliable evidence to be admitted under the Federal Rules test even if knowledge and, thereby, acceptance of the technique has not thoroughly permeated the relevant scientific community.[112] Although judicial decisions regarding admissibility under

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107. *See supra* note 96.

108. *See, e.g.*, P. GIANNELLI & E. IMWINKELRIED, *supra* note 95, at 31; Black I, *supra* note 93, at 1509; Black II, *supra* note 93, at 627–28; Imwinkelried, *The "Bases" of Expert Testimony: The Syllogistic Structure of Scientific Testimony*, 67 N.C.L. REV. 1 (1988) [hereinafter Imwinkelried I]; Imwinkelried, *Federal Rule of Evidence 402: The Second Revolution*, 6 REV. LITIGATION 129, 140–41, 172–74 [hereinafter Imwinkelried II].

109. For a general overview of this issue, see P. GIANNELLI & E. IMWINKELRIED, *supra* note 95, at 28–31. *See also* Imwinkelried II, *supra*, note 108, at 129 (1987) (advocating the position that the Federal Rules of Evidence abolish the *Frye* test).

110. For an overview of application of the relevancy approach, see P. GIANNELLI & E. IMWINKELRIED, *supra* note 95, at 31–34.

111. Giannelli, in *Symposium on Science and the Rules of Evidence*, 99 F.R.D. 187, 189 (1983); *see also* McCormick, *Scientific Evidence: Defining a New Approach to Admissibility*, 76 IOWA L. REV. 879, 911–12 (1982) (admissibility to be judged by reference to a list of 11 "factors").

112. E. CLEARY, *supra* note 96, § 203, at 608–09. With increasing access to scientific data bases and other methods for rapid dissemination of information, it might seem that the "lag" time for general acceptance is negligible for purposes of evidentiary standards. However, this ignores the increasing specialization inherent in modern science. For example, a polymer chemist specializing in the molecular configurations of hydrated gels likely would have little or no reason to access information on the physical chemistry of DNA molecules in solution. Yet both of these fields of knowledge could be critical for correctly interpreting banding patterns of DNA fragments following gel electrophoresis. Similarly, neither polymer chemists nor physical chemists are likely to be fully informed of the most recent advances in population genetics, another branch of science crucial for interpretation of many DNA diagnostic tests. The "permeation" of knowledge throughout the "relevant" scientific community becomes increasingly problematic as members of the scientific community become increasingly specialized.

the Federal Rules of Evidence frequently appear to track the results that would have been obtained under the *Frye* test,[113] the Federal Rules test should lower the barriers to admissibility and result in greater reliance on the adversary system to expose defects in scientific evidence.[114]

B. Admissibility of DNA Diagnostic Tests

How should the courts examine DNA diagnostic technology with regard to admissibility? I propose that the *Frye* general acceptance test is inappropriate for resolving questions of admissibility. DNA diagnostic technology and its potential range of legal applications occupies too broad a spectrum of scientific disciplines to be constrained by general acceptance. Of course, it is likely that in some situations application of a particular DNA diagnostic test to a relatively narrow legal issue would be judged sufficiently reliable by the scientific and legal communities such that admissibility would not be contested. But the *Frye* general acceptance standard may bar the admission of many otherwise reliable DNA test results. The legal system will lose access to reliable and relevant evidence if it is reluctant to abandon the general acceptance approach and allow expert testimony about the applicability of a novel technique to problems of causation.

Adoption of the Federal Rules test will require that judges and lawyers familiarize themselves to some extent with the technology of DNA testing. They must be able to identify the relevant scientific fields occupied by the DNA test at issue, to hold experts to the standards of these fields, and to challenge, if necessary, the validity of their reasoning.[115] Although some commentators have expressed scepticism that the legal system is capable of looking behind the "scientific" assertions of expert witnesses,[116] recent trends suggest an increasing willingness on

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113. Saltzburg, in *Symposium on Science and the Rules of Evidence*, 99 F.R.D. 187, 209 (1983).

114. *See* P. GIANNELLI & E. IMWINKELRIED, *supra* note 95, at 34–35; Giannelli, *supra* note 111, at 195.

115. *See generally* Black I, *supra* note 93; Black II, *supra* note 93. *See also* Imwinkelried I, *supra* note 108 (arguing that a proper application of Federal Rules 702 and 703 to scientific evidence requires courts to examine the expert's "scientific, technical, and other specialized knowledge" as well as the expert's inferences as drawn from case-specific information).

116. Several participants in the *Symposium on Science and the Rules of Evidence*, 99 F.R.D. 187 (1983) expressed such a scepticism. One observer remarked, "[a]n underlying problem is that lawyers do not understand science, including the fundamentals of the scientific method and the techniques by which scientific evidence is generated. . . . Unfortunately, many of the lawyers who might benefit most by overcoming that deficiency exhibit a reluctance to try." *Id.* at 232. Another participant observed, "[t]he sad truth is that those attorneys simply are incapable by education, and all too often by inclination, to become sufficiently familiar with scientific evidence to discharge their responsibilities

the part of the legal profession to assess the reliability of scientific evidence.[117] Particularly in the fields of patent law,[118] environmental law,[119] toxic torts,[120] and recently, DNA "fingerprinting,"[121] courts have closely analyzed the reasoning behind expert testimony and have required conformity with the methodology and norms of science.

The recent DNA fingerprinting cases provide examples of the legal system's inclination and capacity to deal with scientific evidence.[122] In *Andrews v. State*,[123] a Florida District Court of Appeal specifically rejected the *Frye* test of general acceptance in favor of the "relevancy/reliability" approach that is equivalent to the Federal Rules test. The *Andrews* court undertook a relatively thorough analysis of the technique and testimony relating to DNA fingerprinting. It made use of a set of factors adopted by the federal Third Circuit in *United States v. Downing*[124] for establishing reliability when a scientific technique has no track record in litigation: "[t]hese include the novelty of the new technique, i.e., its relationship to more established modes of scientific analysis, the existence of a specialized literature dealing with the technique, the qualifications and professional stature of expert witnesses, and the nonjudicial uses to which the scientific techniques are put."[125]

The *Andrews* opinion contains a brief (and accurate) summary of the theory behind DNA fingerprinting as well as an account of the

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toward the administration of justice. The scientific illiteracy of nearly all lawyers is a disgrace to their profession." *Id.* at 221.

117. *See* Black I, *supra* note 93, at 1511. Another commentator has stated: "It would be a mistake to believe, however, that these differences between law and science prevent members of these professions from understanding each other. There is no reason why lawyers and scientists cannot comprehend the different nature of the other's work and appreciate when it is being done well." Goldberg, *The Reluctant Embrace: Law and Science in America*, 75 GEO. L. J. 1341, 1350 (1987).

118. Courts have had to delve into the intricacies of recombinant DNA technologies with respect to patent litigation for over a decade. *See, e.g.*, Amgen v. Chugai Pharmaceutical Co., 706 F. Supp. 94 (D. Mass. 1989).

119. Courts have had to deal with the scientific issues relating to release of recombinant DNA-containing organisms into the environment. *See, e.g.*, Foundation on Economic Trends v. Heckler, 756 F.2d 143 (D.C. Cir. 1985).

120. *See* Black I, *supra* note 93, at 1511, for examples of how courts have examined (and rejected, where necessary) the reasoning of experts in toxic tort litigation (Agent Orange, Bendectin, and low-level radiation).

121. *See infra* notes 122–45 and accompanying text.

122. *See* Beeler & Wiebe, *DNA Identification Tests and the Courts*, 63 WASH. L. REV. 903 (1988); Burk, *DNA Fingerprinting: Possibilities and Pitfalls of a New Technique*, 28 JURIMETRICS 455 (1988); Thompson & Ford, *DNA Typing: Acceptance and Weight of the New Genetic Identification Tests*, 75 VA. L. REV. 45 (1989).

123. 533 So. 2d 841, 846–47 (Fla. Dist. Ct. App. 1988).

124. 753 F.2d 1224 (3d Cir. 1984).

125. *Id.* at 1238–39 (citing 3 J. WEINSTEIN & M. BERGER, WEINSTEIN'S EVIDENCE § 702[03]).

procedures used in the specific test before the court.[126] In its analysis of admissibility, the court performed a relatively detailed inquiry into the test's reliability. First, the court noted the proper use of quality control procedures for the test reagents as well as the appropriate use of control DNA samples containing DNA fragments of known sizes.[127] Second, the court remarked on the fact that for ten years DNA probes had been used in laboratories around the world to identify organisms as well as to investigate genetic diseases; this extensive nonjudicial use of the procedure, and the abundant scientific literature on the technology, were cited as further evidence of reliability.[128] Third, the court correctly perceived that a required match between two complex patterns of fragments in a gel provides an inherent bias toward false negative results (exoneration for the defendant) rather than toward erroneous positive identifications.[129] Finally, the court attempted to assess the appropriateness of the statistical analysis used to estimate the frequencies with which particular DNA fragments, as identified by the DNA probes used in this test, appear in the population.[130]

In *People v. Wesley*,[131] a county court undertook an even more thorough inquiry into the admissibility of DNA fingerprint test results. The opinion, providing judicial commentary on an extensive evidentiary hearing on DNA fingerprinting, contains a useful overview of genetics and cell biology, complete with diagrams of DNA and cell structure.[132] This is followed by a summary of the theory and techniques relating to DNA fingerprinting.[133] The court critically reviewed the credentials of each of the testifying expert witnesses, and then proceeded to analyze the same indicia of reliability as were analyzed by the *Andrews* court, albeit in somewhat greater detail.[134]

The defense in *Wesley* challenged (1) the adequacy of the laboratory procedures and quality controls, and (2) the adequacy of the population studies upon which were based the claimed powers of identity (probabil-

126. 533 So. 2d at 847–49.
127. *Id.* at 849.
128. *Id.* at 849–50.
129. *Id.*
130. *Id.* at 850.
131. 533 N.Y.S.2d 643 (Co. Ct. 1988).
132. *Id.* at 645–49.
133. *Id.* at 649–50. The opinion provides accurate summaries of restriction enzyme digestion, gel electrophoresis, transfer of DNA fragments to a solid support ("Southern blotting" in this case), hybridization of DNA probes, and detection of hybridization (by autoradiographs in this case).
134. *Id.* at 651–59.

ity of a false match) for the results of the tests.[135] To evaluate the merits of these challenges, the court obviously needed to delve deeply into the science of DNA fingerprinting. As in *Andrews*, the court noted the appropriate use of known DNA fragments as well as other methods of quality control, and evaluated testimony relating to the general reliability of the detailed and specific laboratory procedures.[136] The following portion of the opinion is illustrative of the court's inquiry into the scientific principles and procedures:

> [The] quality control program analyzes the quality of the DNA isolated from a piece of submitted evidence to make sure the DNA is of appropriate quality to do the test; another part of the quality control program looks at the enzyme digestion to assure that correct digestion or fragmentation has taken place; the quality control program includes controls for the DNA fragment separation, the DNA probe, and even the data analysis; other quality control programs are in place to monitor equipment maintenance throughout the test, and reagent preparation. Additional credibility is derived from the use in every test of a control DNA that is processed at the same time as the unknown DNA. The pattern obtained from the control DNA has been seen many times before; thus one knows that the test on the unknown DNA has worked correctly because the pattern seen with respect to the control DNA is what is to be expected.[137]

In addition, the court noted that Lifecodes (the company performing the DNA fingerprint tests) had submitted to external blind trials, and had investigated the effects of heat, humidity, ultraviolet light, and the carpet surface from which the DNA sample was taken on the integrity of the DNA.[138] Again, as in *Andrews*, the court correctly noted the "extreme significance" of the difference between likelihoods of false positive and false negative results in DNA fingerprinting.[139] Finally, after reviewing accepted modes of statistical analysis in the field of population genetics, the court reduced by a factor of ten the claimed mean power of identity

135. *Id.* at 650.
136. *Id.* at 652–56.
137. *Id.* at 655.
138. *Id.*
139. *Id.* at 652, 655.

associated with the test.[140]

Although the *Wesley* court was purporting to follow the *Frye* general acceptance test,[141] it is apparent that general acceptance was only one of several indicia of reliability examined by the court. In fact, the conclusion to the opinion states specifically that DNA fingerprinting is reliable *in addition* to having gained general acceptance in the scientific community.[142] Thus, the court's analysis is much closer to what would have been expected under the Federal Rules of Evidence than what would have been expected under the more narrow *Frye* test. Other courts have made equally searching inquiries into admissibility of DNA fingerprinting test results.[143]

The DNA fingerprinting cases support the thesis that courts are capable of familiarizing themselves with the "science" of DNA diagnostic technology and of reaching informed decisions regarding the admissibility of test results. Several commentators have noted the possibilities for significant error in DNA fingerprint tests,[144] and courts have grappled

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140. Lifecodes, the company that performed the DNA fingerprint tests, claimed a power of identity of one in 1.4 billion for American blacks and one in 840 million for American whites. *Id.* at 656. The court reduced the figures to one in 140 million and one in 84 million, respectively. *Id.* at 658–59.

141. *Id.* at 644.

142. *Id.* at 659.

143. *See, e.g.*, People v. Castro, 545 N.Y.S.2d 985 (Sup. Ct. 1989) (providing judicial commentary on the most extensive evidentiary hearing on DNA fingerprinting to date. The court adopted the *Frye* test, but moved far beyond blind acquiescence to "general acceptance" to make a searching inquiry into the theory, interpretation, and reliability of the test procedures). *See also* Patton, *DNA Fingerprinting: The* Castro *Case*, 3 HARV. J. L. & TECH. 223 (1990). The Supreme Court of Minnesota likewise adopted the *Frye* standard, but as in *Castro, supra*, proceeded to analyze in some detail the reliability of the test procedures. State v. Schwartz, 447 N.W.2d 422 (Minn. 1989). The court made reference to standards promulgated by The Technical Working Group on DNA Analysis Methods, a group of 31 United States and Canadian scientists coordinated by the FBI to establish laboratory procedures and quality control guidelines for forensic DNA testing. These standards may prove useful as general guidelines for judicial evaluation of other forms of DNA testing. It should be noted that the *Schwartz* opinion indicates Minnesota, by legislative enactment, has now adopted the relevancy approach for admissibility of DNA typing evidence. *Id.* at 425. *See also* Cobey v. State, 80 Md. App. 31, 559 A.2d 391 (1989) (providing useful diagrams on DNA manipulations and autoradiography); People v. Shi Fu Huang, 546 N.Y.S.2d 920 (Co. Ct. 1989); State v. Woodall, 385 S.E.2d 253 (W. Va. 1989); Spencer v. Commonwealth, 384 S.E.2d 775 (Va. 1989) (providing a useful "zipper" analogy to explain the structure of DNA and hybridization of DNA probes).

144. *See, e.g.*, Beeler & Wiebe, *supra* note 122 (potential for cross-contamination of DNA samples with DNA from other sources; competency of testing facility personnel; conflicts of interest associated with testimony of experts with a personal or financial stake in the test results); Burk, *supra* note 122 (cross-contamination; faulty estimates of power of identity; and the possibility for false positive results); Thompson & Ford, *supra* note 122 (spurious restriction enzyme activity; sloppy laboratory procedure; distinguishing closely situated bands in autoradiographs).

with these issues when working through their own analyses of reliability.[145] Indeed, Lifecodes has been criticized in several recent cases for failing to control adequately for potential technical artifacts in its performance of DNA fingerprint analyses. Questions have arisen over methods used to control for "band shifting," that is, changes in the speed with which DNA fragments migrate through a gel due to degradation and contaminants, occasionally seen with forensic samples.[146] These problems should not be as acute in the medical diagnostic arena, where fresh and relatively uncontaminated samples should be available in most situations. Nevertheless, the recent criticisms of Lifecodes highlight the need for careful review of quality assurance standards by the courts.[147] It should be noted that lawyers have provided the impetus for critical scrutiny of several of Lifecodes' DNA test results.[148] Blind adherence to the *Frye* standard when application of DNA fingerprinting to forensic samples appeared to many to have widespread support in the scientific community, might have delayed this critical scrutiny of Lifecodes' laboratory procedures.

The willingness of courts to probe the theory and application of DNA fingerprinting is consistent with the primary motivation behind the Federal Rules: to remove arbitrary admissiblity standards in conformity with the prediction that "[t]he manifest destiny of evidence law is a progressive lowering of the barriers to truth."[149]

DNA fingerprinting represents a focused application of DNA diagnostic technology to a narrow legal issue: comparison of specific types

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145. Thompson & Ford, *supra* note 122, are somewhat critical of the *Wesley* court's acceptance of some of the prosecution's expert witness testimony. *Id.* at 102–06. However, I believe the court took appropriate note of the qualifications of the various prosecution witnesses (*e.g.*, Dr. Richard J. Roberts, Assistant Director for Research at the Cold Spring Harbor Laboratory and a leading expert on restriciton enzymes, most of which were discovered at the Cold Spring Harbor Laboratory; Dr. Kenneth K. Kidd, Professor of Human Genetics, Psychiatry, and Biology at the Yale University School of Medicine and Chairman of the DNA Committee of the Human Gene Mapping Conference, an international organization of scientists with responsibility for mapping the human genome. *Wesley*, 533 N.Y.S.2d. at 651, 653). Certainly these eminent scientists would disagree with the reservations expressed by Thompson and Ford, but the point is that, regardless of the ultimate consensus of the legal and scientific communities regarding DNA fingerprinting, the *Wesley* court made a searching inquiry into the merits of this technology, and made a reasoned, and scientifically reasonable, decision concerning admissibility.

146. *See, e.g.*, Anderson, *DNA Fingerprinting on Trial*, 342 NATURE 844 (1989); Norman, *Maine Case Deals Blow to DNA Fingerprinting*, 246 SCIENCE 1556 (1989).

147. Actually, solutions to the problems encountered to date with DNA fingerprinting are readily available with current technologies. *See* Letters to the Editor by Winkler, Sarkar, Brown, and Kumar, 247 SCIENCE 1018–19 (1990).

148. *See* NAT'L L.J., Dec. 18, 1989, at 1, col. 1.

149. *See* Imwinkelried II, *supra* note 108, at 174.

of restriction fragment length polymorphisms to identification of individuals. Several commercial companies now have substantial experience in identification of individuals using this technology.[150] The techniques have received sufficient publicity to have been scrutinized by many members of the relevant scientific communities,[151] and several courts have made inquiries into the reliability and admissibility of the technique with respect to specific fact settings.[152] In these circumstances, courts dealing with future cases in which substantially the same tests are performed in the same manner by the same or similarly situated companies might be justified in relying on the *Frye* general acceptance test.

Although there is now precedent for admissibility of DNA fingerprint test results under certain circumstances, what of the large array of DNA diagnostic technologies whose potential use in litigation ranges far beyond the simple identification of individuals? These tests may prove to be valuable in dealing with problems of causation in toxic tort litigation,[153] and I propose that courts should not reject this evidence for failure to meet the rigid criteria of general acceptance. On the other hand, by not relying on general acceptance, courts will need to make thorough inquiries into the reliability and probative value of the techniques.

Given the broad array of DNA diagnostic technologies, it is impossible to anticipate all of the inquiries required to reach appropriate decisions regarding admissibility. However, by examining the major scientific principles and techniques utilized in the emerging field of DNA diagnostics, it is possible to identify issues of likely relevance for several of the major categories of tests.

1. Detection of Disease or Increased Risk of Disease

a) New technologies

i) Detection of DNA probe hybridization: Traditionally, DNA probes have been "tagged" with radioactive isotopes, which can be detected through the ability of such radioactive molecules to expose or darken an

150. *See* Beeler & Wiebe, *supra* note 122, at 922–26.

151. See generally the discussion of the *Andrews* and *Wesley* opinions, *supra* notes 123–142 and accompanying text.

152. *Id.*

153. *See infra* Section III.

X-ray film.[154] Newer methods utilize a variety of non-radioactive tags in a variety of novel hybridization protocols.[155] The reliability of each of these methods of detection will need to be assessed individually.

ii) Amplification of the hybridization signal: The PCR technique allows DNA sequences of interest to be amplified several million-fold in order to detect rare or underrepresented DNA base changes that otherwise would remain undetected with present levels of detection sensitivity.[156] Courts should insist that any signal amplification technique that relies on DNA strand copying[157] be demonstrated to be accurate. High fidelity of copying is required to avoid spurious hybridization or generation of spurious DNA restriction fragments. In addition, the problem of cross-contamination of DNA samples with DNA from other sources may become extremely important with the PCR technique.

iii) Presence/Absence detection methods: Some of the newer DNA diagnostic tests will rely not on the generation of distinct patterns of fragment sizes, but on the simple presence or absence of hybridization.[158] This "yes or no" type of result is relatively easy to interpret, but courts should be aware that the relationship between false positive and false negative results in such tests is different from that in tests that rely on a comparison of complex patterns of fragment sizes between two DNA samples.[159] Courts should insist on multiple and independent replications and high reproducibility for any tests based on simple presence or absence of hybridization.

b) Disease association

Many genetic diseases can be attributed to defects in a single identified gene.[160] Tests that directly detect such genetic defects should be admissible in court as relevant, probative, and unlikely to mislead the jury. However, many of the diseases often encountered in toxic tort litigation can be "caused" by any one of a number of genetic defects, or

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154. This method of detection, termed autoradiography, was the method validated in the *Andrews* and *Wesley* cases.

155. *See supra* notes 64–68 and accompanying text.

156. *See supra* note 72 and accompanying text.

157. *See supra* note 72.

158. *See supra* notes 63–68 and accompanying text.

159. *See supra* note 129 and accompanying text. In fact, one of the two companies that has been involved in performing DNA fingerprint tests for litigants (Lifecodes) utilizes DNA probes that *do not* generate complex patterns of fragments. Rather, each probe produces only one or two bands, and multiple probes are needed to obtain the reported high powers of identity. Beeler & Wiebe, *supra* note 122, at 923.

160. *See supra* note 50 and accompanying text.

indeed by a combination of such defects.[161] In addition, specific genetic defects may be linked to particular disease states as a result of animal or other laboratory studies, yet remain only tentatively associated with human disease because experiments on humans are impossible or because appropriate epidemiological investigations have not been undertaken. In these cases, should results of DNA diagnostic tests relating to such genetic defects be admissible at trial?

As an illustration of this problem, consider the following hypothetical case: Plaintiff smoker sues defendant cigarette company claiming that defendant's cigarettes caused her lung cancer.[162] Defendant introduces results of a DNA diagnostic test indicating that plaintiff inherited from her parents an oncogene[163] mutation that defendant claims predisposes plaintiff to lung cancer regardless of her smoking habits. Defendant's claim is based on the fact that when this oncogene, carrying the same DNA base sequence change as has been detected in plaintiff, is introduced into normal human cells in a laboratory culture dish, such cells become cancerous.[164] However, no clinical or epidemiological studies have yet demonstrated that such individuals in fact are predisposed to lung cancer.[165]

Should the results of this DNA diagnostic test be admissible? Although courts will need to make fact-based decisions on a case-by-case basis, such test results should not be precluded from introduction at trial. Certainly this is information the jury would want to know in deciding the case. But, would the lack of clinical confirmation of cancer risk mislead the jury?[166] Such evidence should go before the jury, under the

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161. *See supra* notes 51–53 and accompanying text.

162. *See, e.g.*, Latigue v. R.J. Reynolds Tobacco Co., 317 F.2d 19 (5th Cir. 1962), *cert. denied*, 375 U.S. 865 (1963); Cipollone v. Liggett Group, Inc., 644 F. Supp. 283 (D. N.J. 1986), *aff'd in part and rev'd in part*, 893 F.2d 541 (3d Cir. 1990).

163. *See supra* note 53.

164. Numerous such experiments have been performed with a variety of oncogenes. WATSON II, *supra* note 9, at 1061–67. In reality, the presence of two or more mutationally altered oncogenes may be required to cause malignant transformation. *See supra* note 53. However, individuals carrying *one* such mutation in *all* of their cells theoretically would be at greater than average risk for developing cancer. *Id.*

165. Note that in this hypothetical suit, the *defendant* is using the test results as a *shield*. In other situations the *plaintiff* might be using the test results as a *sword*, to establish that defendant has caused genetic harm consistent with a particular disease state, for example. *See* text accompanying *infra* note 190.

166. In one sense the necessary extrapolation between animal or laboratory studies and actual human disease might be considered to be "trans-scientific." That is, the question of extrapolability can be stated in scientific terms, but science is not, as yet, capable of answering the question. *See* Wagner, *Trans-Science in Torts*, 96 YALE L. J. 428, 431, 433 (1986); Brennan, *supra* note 4, at 509–10. In such a case, one might argue that "answers" to such questions are misleading if presented to a jury without adequate explanation. Others might argue that adequate explanations are likely to be forthcoming when the adversary system is functioning as it should. At another level, not all trans-scientific issues are

assumption that both sides in the litigation would have an opportunity to advocate what they consider to be an appropriate interpretation of such evidence. This would be consistent with the policy rationale behind the Federal Rules of Evidence: One relies on the adversary system, not standards that might preclude introduction of otherwise relevant and probative evidence, to clarify the correct interpretation of scientific evidence.[167]

2. Signature Tests

Tests designed to provide evidence of whether or not a particular chemical or physical agent has interacted with an individual's DNA would be of great value in toxic tort litigation.[168] As with tests relating specifically to disease or disease risk, it is possible to identify several general issues relevant to admissibility of signature tests.

a) What is the reliability of any new technology? For detection of DNA adducts[169] or detection of agent-specific spectra of mutational changes,[170] what is the frequency and nature of potential error (false positives, false negatives, quantitative error)?

b) Have the test results been compared to the baseline results of an appropriate control group of individuals? When a specific agent is at issue, as is often the case in most signature tests, selection of control group populations will depend on such factors as: (1) potential sources of exposure (e.g., does the agent come from a "point" source such as a factory, or could geographically distant individuals be exposed to the same agent, such as would be the case with a toxic chemical in home insulation sold throughout the country); (2) impact of lifestyle on exposure (e.g., an "indoor" person living in the vicinity of a factory may have significantly less exposure to an agent than a next-door neighbor who

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equally "trans-scientific." Wagner, *supra*, at 433. And, as scientific knowledge advances, previously trans-scientific questions may become answerable in scientific terms. Thus, it is one thing to label as trans-scientific the question of whether chemical X, which is known to cause cancer in rats at dose Y, causes cancer in human beings at dose Z. However, when DNA testing demonstrates that chemical X causes cancer in rats *because* it induces a particular mutation in oncogene A, when it has been shown that chemical X induces cancerous transformation of human cells in the laboratory *because* it induces the same mutation in oncogene A, and when the same oncogene mutation has been observed after-the-fact in many human tumors, the question of whether a plaintiff carrying the oncogene A mutation is predisposed to cancer is very close to the trans-science/science border even in the absence of epidemiological studies. *See supra* notes 53 & 164 and *infra* note 207.

167. *See supra* notes 107–09 and accompanying text.

168. *See supra* notes 75–92 and accompanying text.

169. *See supra* notes 87–92 and accompanying text.

170. *See supra* notes 77–86 and accompanying text.

exercises outdoors); and (3) individual variation in ability to repair DNA or to remove chemical adducts from DNA.[171]

III. DNA DIAGNOSTIC TECHNOLOGY AND TOXIC TORTS

A. Causation in Toxic Torts

Traditional tort doctrines of negligence require an individual seeking compensation for injury resulting from exposure to hazardous substances to demonstrate that: (1) she suffered a harm or loss; (2) the defendant's act or omission caused the harm or loss; and (3) the defendant was at fault for so acting or failing to act.[172] Although changing conceptions of causation[173] and fault[174] have modified application of traditional negligence standards, causation continues to be a central and difficult issue in toxic tort litigation.[175]

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171. One of the new signature tests, for example, supposedly is capable of correcting for individual variation in the ability to repair damaged genes. Marx, *supra* note 78, at 738.

172. Thomson, *Remarks on Causation and Liability*, 13 PHIL. & PUB. AFF. 101 (1984).

173. *See* Sindell v. Abbott Laboratories, 26 Cal. 3d. 588, 163 Cal. Rptr. 132, 607 P.2d 924, *cert. denied*, 449 U.S. 912 (1980) (where harm possibly could have emanated from two or more product manufacturers, plaintiff's cause of action was allowed to go forward regardless of the fact that she could not show that a *particular* defendant caused her harm). The *Sindell* result has been criticized. *See* Epstein, *Two Fallacies in the Law of Joint Torts*, 73 GEO. L. J. 1377, 1378–82 (1985); Wright, *Causation in Tort Law*, 73 CALIF. L. REV. 1735, 1819–21 (1985). "[Another] means that has been used to undermine causation—increasingly common in toxic tort cases—is the use of presumptions or burden-shifting techniques to force the defendant to prove lack of causation in order to avoid liability. Frequently, this amounts to asking the defendant to meet an impossible burden of proving the negative." U.S. DEPARTMENT OF JUSTICE, REPORT OF THE TORT POLICY WORKING GROUP ON THE CAUSES, EXTENT AND POLICY IMPLICATIONS OF THE CURRENT CRISIS IN INSURANCE AVAILABILITY AND AFFORDABILITY 34–35 (1986).

174. Changing conceptions of strict products liability, as well as strict liability in general, have changed the ways in which courts have approached issues of fault. *See generally* LIABILITY: PERSPECTIVES AND POLICY, *supra* note 1. Causation, it should be noted, still must be established in strict products liability. RESTATEMENT (SECOND) OF TORTS § 402A(1) (1977).

175. *See, e.g.*, Ayers v. Township of Jackson, 525 A.2d 287, 301 (N.J. 1987) (long latency periods of illnesses caused by chemical exposure make proof of causation difficult); Allen v. United States, 588 F. Supp. 247 (D. Utah 1984) (illustrating difficulty of establishing causal relationship between radiation exposure and human cancer). *See also* G. NOTHSTEIN, TOXIC TORTS: LITIGATION OF TOXIC SUBSTANCES CASES 454 (1984) ("In short, the potential problems of proving causation are enormously varied and frequently complex as a scientific and factual matter."); Brennan, *supra* note 4, at 469 (1988) (citing proof of causation as the "paramount obstacle" to appropriate disposition of toxic tort cases); Farber, *Toxic Causation*, 71 MINN L. REV. 1219, 1219–20 (1987) (scientific uncertainty creates "serious problems" for establishing causation); Gold, *Causation in Toxic Torts: Burdens of Proof, Standards of Persuasion, and Statistical Evidence*,

An examination of the peculiar properties of toxic torts[176] suggests that two characteristics represent major impediments to judicial resolution of causation problems. First, scientifically valid associations between hazardous substance exposure and harm are frequently based on probabilistic evidence derived from epidemiology or other fields of investigation.[177] Although courts are most comfortable with "mechanistic, deductively-derived" chains of causal evidence,[178] the courts' struggle with probabilistic evidence is apparent in the contrasting approaches taken in two widely recognized toxic injury cases. In the consolidated litigation over the health effects of Agent Orange, the court attached great significance to epidemiological studies showing no statistical link between exposure to Agent Orange and subsequent health effects.[179] As a result, the court approved a settlement considered to be favorable to the defendants.[180] In contrast, the court in *Ferebee v. Chevron Chemical Co.*,[181] stated: "[T]hus, a cause effect relationship need not be clearly established by animal or epidemiological studies before a doctor can testify that, in his opinion, such a relationship exists."[182]

Second, traditional causation doctrines arose in the context of evident or "actual" injury. In contrast, toxic tort litigation frequently presents courts with claims of "latent" injury.[183] The courts' struggle with

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96 YALE L. J. 376, 376–77 (1986) ("Proving the cause of injuries that remain latent for years, are associated with diverse risk factors, and occur at background levels even without any apparent cause, is the 'central problem' for toxic tort plaintiffs.") (citations omitted); M. DORE, *supra* note 2, § 24–1 (1987) ("No issue in toxic torts presents more complex and difficult problems than causation."); Note, *supra* note 2, at 1617 (largest barrier to recovery is proof of causation); Note, *An Analysis of the Enhanced Risk Cause of Action (Or How I Learned to Stop Worrying and Love Toxic Waste)*, 33 VILL. L. R. 437, 442 (1988) ("[p]roving causation is one of the main impediments to recovery").

176. *See supra* note 2.

177. *See* Brennan, *supra* note 4, at 483–91. *See also* Dant, *Gambling on the Truth: The Use of Purely Statistical Evidence as a Basis for Civil Liability*, 22 COLUM. J.L. & SOC. PROBS. 231 (1988).

178. Brennan, *supra* note 4, at 491.

179. *In re* "Agent Orange" Prods. Liab. Litig., 597 F. Supp. 740, 787–94 (E.D. N.Y. 1984). *See also* P. SCHUCK, AGENT ORANGE ON TRIAL: MASS TOXIC DISASTERS IN THE COURTS (1987).

180. *See* Farber, *supra* note 175, at 1234–35.

181. 736 F.2d 1529 (D.C. Cir.), *cert. denied*, 469 U.S. 1062 (1984).

182. *Id.* at 1535. Although these two opinions are not directly comparable to each other (an epidemiological study was not available in *Ferebee*, and Judge Weinstein in the Agent Orange litigation was attempting to preserve a hard-fought settlement), they do demonstrate contrasting treatments of probabilistic evidence.

183. *See* Kanner, *Emerging Conceptions of Latent Personal Injuries in Toxic Tort Litigation*, 18 RUTGERS L. J. 343 (1987). G. NOTHSTEIN, *supra* note 175, at 457–63, distinguishes *delayed* injury (present injury manifesting itself only after relatively long latent periods) and *future* injury (possible injury manifesting itself in the future due to present or past exposure to a hazardous agent). The use of "latent" injury in this Article will encompass both types of injury.

Although "injury" traditionally has been separated from the issue of "causation," *see*

latency, as with probabilistic evidence, results in divergent approaches. For example, the plaintiffs in *Brafford v. Susquehanna Corp.*[184] claimed no actual injury beyond undetected genetic damage caused by radiation. The court subsequently allowed the finder of fact to determine whether such subcellular damage satisfied the requirement of actual injury.[185] In contrast, the United States Court of Appeals for the Third Circuit has stated that "there is generally no cause of action in tort until a plaintiff has suffered identifiable, compensable injury."[186] The court feared that any other holding would lead to unwarranted speculation and inequitable results. The difficult task of proving the existence of latent injuries is preventing the courts from reaching an acceptable and consistent approach to the problem of toxic tort injuries.

Although DNA diagnostic technology will not be completely dispositive in many fact settings, it may provide courts with valuable assistance in dealing with probabilistic evidence and latent injury, as discussed below.

1. DNA Diagnostic Technology and Questions of Probability

Although the answers to questions of causation often must be framed in probabilities, courts have allowed causation to be presumed when those probabilities have been sufficiently high.[187] For example, the probability of contracting mesothelioma, a rare and deadly form of cancer, is approximately seventy times greater among asbestos workers than among members of the general population.[188] Similarly, clear cell adenocarcinoma, another very rare form of cancer, has a far higher probability of striking daughters of women who took diethylstilbestrol ("DES") during preganancy than women who were not exposed to DES *in utero*.[189] Although DNA diagnostic technology will not always provide such powerful probabilistic evidence, its role in increasing the certainty of causation should be welcomed by judges and juries struggling

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supra note 172 and accompanying text, in the context of latent injury in toxic torts, where frequently all that can be said is that defendants have *caused* an increased *risk of injury*, the two concepts are so closely intertwined that I have chosen to treat latent injury under the rubric of causation.

184. 586 F. Supp. 14 (D. Colo. 1984).

185. *Id.* at 18.

186. Schweitzer v. Consolidated Rail Corp., 758 F.2d 936, 942 (3d Cir.), *cert. denied*, 474 U.S. 864 (1985).

187. *See* Farber, *supra* note 175, at 1251–52.

188. Black & Lilienfeld, *Epidemiologic Proof in Toxic Tort Litigation*, 52 FORDHAM L. REV. 732, 758 (1984).

189. Bohrer, *Fear and Trembling in the Twentieth Century: Technological Risk, Uncertainty and Emotional Distress*, 1984 WIS. L. REV. 83, 97 n. 48 (1984).

with causation issues.

Signature tests demonstrate the presence or absence of the defendant's chemical as an adduct in the plaintiff's DNA, or the presence or absence of a spectrum of mutational changes in the plaintiff's DNA consistent with exposure to the defendant's chemical or physical agent.[190] Consequently, such tests could provide significant shifts in the degree of certainty as to whether the defendant's agent is implicated in the plaintiff's injury. Indeed, where a chemical is produced by only one identified manufacturer, and where the potential health effects are sufficiently rare or otherwise tightly associated with the chemical in question, a positive signature test might allow the court to fashion remedies under what would be, in essence, an actual causation standard.

Less inherently dispositive DNA diagnostic procedures could also clarify probable causation. RFLP analysis[191] or direct detection of base sequence changes[192] could differentiate gross chromosomal damage or relatively large scale DNA rearrangements from point mutations.[193] This information may be relevant because some DNA-damaging agents tend to cause DNA strand breaks and rearrangements, while others tend to cause point mutations. Similar analyses could distinguish virally from chemically induced disease.[194]

The question of whether the plaintiff's injury results from intrinsic or background risk not attributable to the defendant's agent(s) presents a related issue on which DNA diagnostic technology may shed some light. For example, a defendant might desire to show that all of the plaintiff's cells carry an oncogene mutation (indicating the mutation was inherited),[195] or demonstrate the presence of DNA adducts or mutational changes specific for agents other than those produced by the defendant.[196] A defendant might argue that it is much less probable that its

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190. *See supra* text accompanying notes 75–86.

191. *See supra* text accompanying notes 54–61.

192. *See supra* text accompanying notes 62–74.

193. *See* S. OPPENHEIMER, CANCER: A BIOLOGICAL AND CLINICAL INTRODUCTION 9–10, 57 (1982); WATSON I, *supra* note 7, at 339–45.

194. Viral DNAs are known to cause malignant transformation in some cell types, by supplying viral oncogenes, by "activating" the cell's own oncogenes, or by "inactivating" anti-oncogenes, though the exact role of viruses in the complete range of human cancers is unknown. WATSON II, *supra* note 9, at 1010–33. Appropriate DNA probes could readily detect the presence or absence of culprit viral DNA sequences in a diseased tissue.

195. *See supra* note 70. Recently, genetic characteristics other than those associated with oncogenes also have been implicated in susceptibility to cancer. *See, e.g.*, Hein, *Genetic Polymorphism and Cancer Susceptibility: Evidence Concerning Acetyltransferases and Cancer of the Urinary Bladder*, 9 BIOESSAYS 200 (1988).

196. The question of whether a plaintiff could be compelled by the defendant to submit to such testing procedures is dealt with in *infra* Section III B.

chemical caused plaintiff's tumor, if, for example, plaintiff has inherited an oncogene mutation creating a predisposition to cancer.[197] Conversely, a plaintiff might want to come forward with DNA test results demonstrating the absence of such intrinsic or background risk. On the other hand, the plaintiff's increased susceptibility arguably means that the defendant's agent was more likely to have harmed her than other members of the population.[198] Other DNA diagnostic tests, such as positive or negative signature tests, might help to resolve this issue, but in any case the shift in perceived probabilities engendered by the DNA diagnostic technology can only help finders of fact and judges to arrive at more informed decisions.[199]

When estimates of probability are unavoidable in resolving issues of causation, a majority of commentators favor making recoveries proportional, in some sense, to the estimates of probability.[200] To the extent that such proposals are adopted by the courts, the increased reliability of probability estimates based on DNA diagnostic technology should lead to greater justice in allocation of recovery funds.

2. *DNA Diagnostic Technology and Latent Injury*

Latent injury, in particular elevated risk of future injury due to present or past exposure to a hazardous agent, has presented courts with difficult factual and legal issues.[201] However, the difficulty is really one of imperfect information. Courts likely would not entertain a suit against a negligent driver for an accident that had not yet occurred, even though other drivers were at greater risk of harm due to the continuing danger presented by the negligent driver. On the other hand, courts might (but often do not) entertain suits to compensate for increased risk of disease

197. *See supra* notes 163–64 and *infra* note 207 and accompanying text.

198. An analogy can be seen in the asbestos cases. In Dartez v Fibreboard Corp., 765 F.2d 456, 466–67 (5th Cir. 1985), the court refused recovery for increased risk of cancer from asbestos, due to the plaintiff's smoking habits. The plaintiff also smoked in Gideon v. Johns-Manville Sales Corp., 761 F.2d 1129, 1139 (5th Cir. 1985), but was allowed to recover damages despite defendant's assertion that smoking caused the disease.

199. As an example, see the hypothetical scenario presented in *infra* note 226.

200. *See* Farber, *supra* note 175, at 1220–21, 1240. Some commentators favor strict proportional recovery; that is, plaintiff would receive 20% of her total damages if the court sanctioned a 20% probability that defendant caused her injury. Farber favors a "most likely victim" approach. Those victims most likely to have been harmed by the defendant would receive full recovery, while those victims least likely to have been harmed by the defendant would receive nothing. *Id.* at 1221.

201. *See supra* notes 183–86 and accompanying text.

following exposure to hazardous agents.[202] As one commentator has stated:

> 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. If some method did exist of determining the cause of a particular plaintiff's cancer, courts would presumably follow the normal rules of tort law and award damages only to plaintiffs who could show actual causation. Imperfect information prevents us from implementing this rule, but the compensation scheme should attempt to approximate the result as much as possible.[203]

The absence of adequate methods to detect damage to DNA has forced courts to make artificial distinctions between genetic injury (damage to the genome) and somatic injury (damage to body tissues and organs).[204] There is no a priori reason to preclude recovery for injury to DNA that increases the risk of future somatic injury. DNA damage is conceptually no different than a physical blow to the head that results in subclinical tissue damage[205] that increases the risk of future seizures.[206]

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202. *See* G. NOTHSTEIN, *supra* note 175, at 461–65; Farber, *supra* note 175, at 1246–47. *See also* Schwartzbauer & Shindell, *Cancer and the Adjudicative Process: The Interface of Environmental Protection and Toxic Tort Law*, 14 AM. J. LAW & MED. 1, 26–27 (1988); Note, *"Cancerphobia" and Increased Risk of Developing Cancer Due to Toxic Exposure: Will It Spread to Missouri?*, 53 MO. L. REV. 325, 342–54 (1988).

203. Farber, *supra* note 175, at 1247. Though this commentator's argument is somewhat less focused than it might have been (he provides the above language in relation to *risk*, but speaks of the "plaintiff's *cancer*" rather than the plaintiff's cancer *risk*), the point about imperfect information is well taken.

204. Though not stated precisely in these terms, the distinction between "genetic" and "somatic" injury is implicit in many of the cases dealing with cancer "risk." *See, e.g.*, Schweitzer v. Consolidated Rail Corp., 758 F.2d 936, 942 (3d Cir.), *cert. denied*, 474 U.S. 864 (1985). ("[s]ubclinical injury resulting from exposure to asbestos is insufficient to constitute the actual loss or damage to a plaintiff's interest required to sustain a cause of action."); Jackson v. Johns-Manville Sales Corp., 781 F.2d 394, 412–13 (5th Cir. 1986) ("Once the injury becomes actionable—once *some* effect appears—then the plaintiff is permitted to recover for all probable future manifestations as well."); Brafford v. Susquehanna Corp., 586 F. Supp. 14, 17–18 (D. Colo. 1984) (cause of action for increased cancer risk requires proof of present physical injury; here, however, plaintiff was allowed to offer proof of present chromosomal damage). *See also* Allen v. United States, 588 F. Supp. 247 (D. Utah 1984); Ayers v. Township of Jackson, 525 A.2d 287 (N.J. 1987).

205. That is, damage might be detectable with X-rays or other brain imaging technology, but may not be visible externally and does not cause present clinical symptoms.

206. It seems possible that courts would be inclined to fashion a remedy to encompass the total harm to such a victim, including the heightened risk of seizure (and, possibly, emotional distress from the heightened risk of seizure). On the other hand, a demonstrable absence of subclinical damage in such a situation might relieve the defendant from having

If a plaintiff's signature tests regarding defendant's chemical were positive and RFLP analysis or other types of DNA diagnostic tests demonstrated some fraction of plaintiff's cells carried an oncogene mutation only rarely seen in the general population, a court reasonably could conclude that such an individual had suffered bodily harm equally as harmful as the subclinical tissue damage resulting from a severe blow to the head.[207] Conversely, negative results from the DNA diagnostic test results might preclude recovery for claims of elevated risk of future disease, as well as for fear of future disease.[208]

Equation of increased risk of future injury with present injury actually was presaged in a rather remarkable judicial opinion from the 1930s. In *Coover v. Painless Parker, Dentist*,[209] the plaintiff sued for injury caused by overexposure to dental X-rays. The plaintiff claimed damages for

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to compensate for risk of seizure. This point was argued forcefully by Judge Posner in his dissent in DePass v. United States, 721 F.2d 203, 210 (7th Cir. 1983). There, plaintiff had suffered traumatic amputation of his leg below the knee, and the question was whether he could be compensated for alleged increased risk of cardiovascular disease and resultant diminishing of life expectancy. Judge Posner stated that "[t]he goal of awarding damages in tort law is to put the victim as nearly as possible in the position he would have occupied if the tort had not been committed. This goal cannot be attained or even approached if judges shut their eyes to consequences that scientists have found are likely to follow from particular types of accident, merely because the scientists' evidence is statistical." *Id.*

207. That a chemically induced mutation in an oncogene can give rise to an elevated risk of cancer can be illustrated as follows. Many cells likely require mutational damage to two or more oncogenes in order to become malignant. *See supra* note 53. If hypothetical cell A requires mutations in both oncogenes X and Y in order to become malignant, and there is a one in 10^7 chance that either X or Y would acquire the requisite mutation through intrinsic error unrelated to toxic exposure, then cell A has a one in 10^{14} chance ($10^7 \times 10^7 = 10^{14}$, assuming that a mutation in one oncogene has no influence on the intrinsic error mutation rate in the other oncogene) of becoming malignant in the absence of external influence. If defendant's chemical induces a mutation in X, the probability that cell A will become malignant is raised to one in 10^7. The probability that cell A will give rise to a malignant cell is even higher if plaintiff continues to be exposed to defendant's chemical, or if the mutation in X confers a slight growth advantage on cell A, such that cell A proliferates into a population of several thousand or several million cells, each carrying a mutation in X. (This fact situation and these numbers were chosen for illustrative purposes only.)

208. *See, e.g.*, Hagerty v. L & L Marine Serv., 788 F.2d 315 (5th Cir. 1986) (cause of action for "cancerphobia" allowed, but with requirement for establishing causal relationship to defendant's negligence and reasonableness of fear and anxiety due to possibility of contracting cancer). It might be noted here that negative results from DNA diagnostic tests, while possibly precluding suits for fear of future injury, might also help to allay community-wide anxiety that develops in response to publicity surrounding toxic tort litigation. For example, the carcinogenic risk from contaminated well water in Woburn, Massachusetts (containing trichloroethylene, chloroform, and tetrachloroethylene) was less than half the risk presented by ordinary chlorinated tap water. Ames, Magaw & Gold, *Ranking Possible Carcinogenic Hazards*, 236 SCIENCE 271, 272–73 (1987). Negative results from DNA tests for a representative sample of plaintiffs might reassure a community that it is not about to experience an epidemic of hazardous substance-related illnesses.

209. 105 Cal. App. 110, 286 P. 1048 (1930).

severe facial burns resulting from the overexposure and an increased risk of cancer. In allowing the cause of action for increased risk of cancer, the appellate court stated:

> Appellant argues that the evidence as to the possibility of cancer is wholly conjectural and uncertain, and that that element could not have rightfully been considered by the jury. The court instructed the jury that they were to consider as elements of damage only such physical injury as they may find the plaintiff is certain to suffer in the future. If we assume that respondent's skin condition was considered by the jury, it by no means follows that this was improper. While the actual condition of cancer may have been conjectural and uncertain, the record contains positive evidence that a condition actually exists which makes this dread disease much more likely. We think this predisposition *in itself is some damage*, and, when caused by the wrong of another, it is an interference with the normal and natural conditions and rights of the other, which must be held to be a *real and not a fanciful element of damage*.[210]

It appears that the court's reasoning and conclusions were thoroughly dependent on the tangible evidence of present bodily injury (facial skin burns) directly related to the risk of future disease. It is doubtful whether the *Coover* court would have allowed recovery for increased risk of cancer if the plaintiff had received an X-ray overexposure but had not also received facial skin burns. The skin burns provided the necessary connection between the defendant's wrongful behavior and the elevated risk of future injury.[211]

Appropriately dispositive DNA diagnostic test results could function as "facial skin burns" in toxic tort litigation. Thus, DNA diagnostic technology could allow courts, in at least some cases, to step back from the difficult frontiers of compensation for risk into the less troublesome territory of compensation for actual injury.

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210. *Id.* at 1050 (emphasis added).

211. The same sort of connection was of obvious importance to Judge Posner's reasoning in Depass v. United States, 721 F.2d 203, 207–11 (7th Cir. 1983), where observable physical damage to a body part provided one of the necessary justifications for Judge Posner's proposed compensation for reduced life expectancy due to heightened risk of cardiovascular disease.

B. DNA Diagnostic Technology, Toxic Torts, and Unwanted Knowledge

Huntington's disease ("HD") is an inherited neurodegenerative disorder, the symptoms of which generally do not become apparent until the victim is well into adulthood.[212] HD is untreatable and clinically undetectable until symptoms appear.[213] Children of a parent afflicted with HD have a fifty percent chance of being stricken with HD later in life. A DNA diagnostic test of the RFLP type for HD became available in the early 1980s, and can inform presymptomatic at-risk individuals whether they will almost certainly develop HD.[214] However, one study found that over two-thirds of at-risk persons expected they would become depressed if DNA tests were positive, and another study found that twenty-one percent of at-risk individuals might commit suicide if the tests were positive.[215]

The results of the HD psychological studies bring the profound personal consequences of presymptomatic DNA diagnostic tests for serious diseases into sharp focus. Individuals confronted with the results of some DNA diagnostic tests will need to make profound decisions regarding lifestyle, jobs, marriage, and reproduction. They may also become more susceptible to psychological disturbances, including propensity to commit suicide.

Rule 35 of the Federal Rules of Civil Procedure ("FRCP") states that courts, upon a showing of good cause by the opposing party, may require that a party to litigation submit to a physical or mental examination.[216] Nearly all state jurisdictions have adopted similar provisions giving courts discretion to require such examinations.[217] The Supreme Court in

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212. Mastromauro, Myers & Berkman, *Attitudes Toward Presymptomatic Testing in Huntington Disease*, 26 AM. J. MED. GENETICS 271 (1987).

213. Markel, Young & Penney, *At-Risk Person's Attitudes Toward Presymptomatic Testing and Prenatal Testing of Huntington Disease in Michigan*, 26 AM. J. MED. GENETICS 295 (1987).

214. Gusella, Wexler, Conneally, Naylor, Anderson, Tanzi, Watkins, Ottina, Wallace, Sakaguchi, Young, Shoulson, Bonilla & Martin, *A Polymorphic DNA Marker Genetically Linked to Huntington's Disease*, 306 NATURE 234 (1983).

215. *See* Kessler, *Letter to the Editor: The Dilemma of Suicide and Huntington Disease*, 26 AM. J. MED. GENETICS 315 (1987) (summarizing data from several studies presented in Volume 26 of the AMERICAN JOURNAL OF MEDICAL GENETICS).

216. *See* M. DOMBROFF, DISCOVERY 280 (1986). Rule 35 states, in part: "When the mental or physical condition (including the blood group) of a party . . . is in controversy, the court . . . may order the party to submit to a physical or mental examination. . . . The order may be made only on motion for a good cause." FED. R. CIV. P. 35.

217. 6 C. WRIGHT & A. MILLER, FEDERAL PRACTICE AND PROCEDURE §§ 2231, 2234 (West 1970 & Supp. 1988).

Schlagenhauf v. Holder[218] stated: "A plaintiff . . . who asserts mental or physical injury . . . places that mental or physical injury clearly in controversy and provides the defendant with good cause for an examination to determine the existence and extent of such asserted injury."[219] When, on the other hand, the plaintiff's condition is put at issue by the defendant, courts should be more discriminating regarding the "in controversy" and "good cause" requirements.[220] Clearly, the potentially serious personal impact of compelled DNA diagnostic tests mandates that courts consider carefully the moral and ethical boundaries of the discretion granted by FRCP 35 and related state rules. A framework within which questions of this nature might be analyzed is presented below.[221]

1) Tests Related Directly to the Alleged Harm

a) Signature tests

It is difficult to imagine that courts would not require plaintiffs to submit to tests determining the presence or absence of defendant's chemical adducts in plaintiff's DNA.[222] Since such tests prove nothing more than exposure of plaintiff's DNA to the chemical(s) at issue, they would directly relate to plaintiff's allegations of subsequent harm.

A more difficult question arises over use of tests that determine presence or absence of "signature" spectra of mutational changes in plaintiff's DNA. Such tests may reveal evidence of mutational damage from chemical or physical agents other than those at issue in the litigation.[223] In response, courts might consider requiring such tests to be performed by neutral third parties, allowing admission into evidence of only those results that provide answers relating to the issue in litigation, and instituting mechanisms to ensure that test results unrelated to the

218. 379 U.S. 104 (1964).

219. *Id.* at 119.

220. C. WRIGHT & A. MILLER, *supra* note 217, § 2234, at 672.

221. The following framework is given under the assumption that the physical intrusiveness (discomfort or risk associated with obtaining an appropriate DNA sample) will be minimal. If such were not the case, however, the court would need to balance the freedom from pain and the safety of the party to be examined with the need for just resolution of the litigation. *See* C. WRIGHT & A. MILLER, *supra* note 217, § 2235. For example, courts have refused to permit barium meal X-rays, Bartolotta v. Delco Appliance Corp., 254 A.D. 809, 4 N.Y.S.2d 744 (1938), and spinal punctures, Roskovics v. Ashtabula Water Works Co., 174 N.E.2d 295 (Ohio Ct. of Common Pleas 1961).

222. *See supra* notes 87–92 and accompanying text.

223. Examination of the HPRT gene, for example, potentially is capable of revealing the mutational signatures of hundreds of different chemicals. *See supra* notes 77–86 and accompanying text.

litigation are destroyed or otherwise prevented from reaching the parties who do not wish to receive such information. Thus, for example, a court might certify the question of whether or not plaintiff's DNA contains mutational damage consistent with exposure to chemical X. If the test results provided evidence only of damage from chemical Y, the answer provided to the court would be a simple "no," and only this answer would be admitted into evidence. Thus, the information about chemical Y would be kept from the parties. On the other hand, if the information about chemical Y were relevant to a defense based on intrinsic or background risk, then there is little doubt that a judge would allow this information into evidence.

b) Tests relating to disease or disease risk

A defendant might request RFLP or other types of analyses[224] of the plaintiff's DNA in order to demonstrate presence or absence of specific changes relating to specific genes of relevance to the litigation, such as rearrangements or point mutations involving particular oncogenes in particular types of cancers, for example. This request would present little difficulty if the DNA sample were to be taken from a diseased tissue or organ and the plaintiff had already dealt psychologically with the presence of the disease. The DNA test would only clarify causation as it relates to that disease.[225] More problematic is the situation where the plaintiff is disease-free but is claiming heightened risk of disease. Generally it would be to the plaintiff's interests to come forward with RFLP or other test results that demonstrate a present genetic injury. But, if for some reason the defendant wished to expand the scope of the analysis or to request DNA diagnostic testing where the plaintiff had not presented test results, courts again should strive to limit disclosure to those results of relevance to the litigation.[226]

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224. *See supra* notes 48–74 and accompanying text.

225. It should be noted that most tumors contain mixtures of normal and abnormal cells. For example, many solid tumors are infiltrated with non-malignant blood vessels as well as non-malignant connective tissues. Likewise, blood samples from patients with cancers of the blood-forming organs will contain normal and cancerous cells. *See generally* B. ALBERTS, D. BRAY, J. LEWIS, M. RAFF, K. ROBERTS & J. WATSON, MOLECULAR BIOLOGY OF THE CELL 626, 911 (1983). Tests on DNA from such samples might therefore reveal information about intrinsic or background risk (*see* next Section) or disease conditions unrelated to the litigation. Generally the results obtained from normal cells can be discounted in such situations, but courts should be aware of the problem and should exclude test results not relevant to the litigation where necessary.

226. However, this may not always be possible. As a hypothetical example, consider a 25-year-old employee who sues her employer for allegedly exposing her to chemical X, which is known to cause a rare lung disorder the employee has developed. The employer wishes to test the employee's DNA for an inherited defect in a gene coding for an enzyme Y. It is well established in the medical literature that defects in enzyme Y lead invariably

2) Tests Related to Intrinsic or Background Risk

Defendants frequently will wish to establish that plaintiff has inherited genetic characteristics predisposing to the disease or disease risk in question (intrinsic risk), or that plaintiff's lifestyle characteristics, such as smoking cigarettes or residing near sources of industrial pollution, has created genetic injury unrelated to defendant's agent (background risk). DNA diagnostic testing for intrinsic and background risk may raise deeply troubling issues for plaintiffs, and some test results will have only an indirect connection to the nature of the complaint. Yet, intrinsic and background risks are of obvious relevance to a defendant faced with substantial liability.

Courts will need to weigh potential harms to the plaintiff against the need for facts in the interest of justice, and proceed on a case-by-case basis. As part of this analysis, courts should consider the contribution of a particular DNA diagnostic test to resolution of the litigated issue. As with other evidentiary issues, this requires that courts familiarize themselves with the scientific bases for the test and with the rationale for its use in clarifying the alleged harm. Thus, courts must understand precisely what question the test is designed to answer and must judge the reliability of the test itself, including its potential for false positive or false negative results. In addition, a reliable test must be sufficiently dispositive of the issue in litigation to warrant potential disclosure of unwanted information to the plaintiff. Just resolution of the tension between harm to the plaintiff and need for facts may be extremely challenging.[227]

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to the rare lung disorder, but it is also well established that individuals carrying this gene mutation generally develop fatal and untreatable neurological problems around age 30. There is evidence that the employer *may* have been negligent in its use of chemical X, but even when exposed to chemical X, only five percent of individuals so exposed develop the lung disorder. On the other hand, if the employee does *not* possess the gene defect, her lung disorder was almost certainly caused by chemical X, since the incidence of the lung disorder in the general population is less than one in ten million. Here the DNA diagnosis relates directly to the cause of action, but has the potential to reveal profoundly disturbing additional information to the plaintiff.

227. As an example, consider that the court may be aware that plaintiff (or some fraction of the members of a class of plaintiffs) is likely to commit suicide if faced with positive test results. The court also may be faced with the real possibility that defendant's actions are not responsible for the disease or risk in question, that positive test results (demonstrating, for example, that plaintiff is predisposed to the harm in question) would significantly increase the probability of defendant's innocence, that a large judgment against defendant might lead to substantial unemployment in the industry, and that studies have indicated increased incidences of substance abuse, spousal and child abuse, and suicide among unemployed workers' families in this industry.

In such situations,[228] it might be useful for courts to consider trial management that would allow the plaintiffs some discretion to choose the future course of their participation in the litigation. For litigation involving individual or small numbers of plaintiffs, such mechanisms might involve judicial coordination of the extent of plaintiff's participation in DNA diagnostic testing with plaintiff's potential recovery. Reasonable compromises might be obtained, for example, through judicially supervised pre-trial negotiation between the parties.[229] Since toxic tort litigation frequently involves class action suits, courts also may be able to fashion class subdivisions to deal with these issues.[230] For example, plaintiffs opting against DNA diagnostic testing might be placed in a subclass that would receive only a designated fraction of full recovery (assuming the defendant is held liable for harm to the class as a whole). Plaintiffs opting for DNA diagnostic testing might be placed in a subclass whose members would receive full recovery if the test results supported causation by the defendant. Conversely, these plaintiffs might receive little or no recovery if the test results substantially weakened their causation arguments. In any case, creative judicial management of classes and remedies may provide one approach to resolving some of the dilemmas created by the wide-ranging ramifications of some DNA diagnostic tests.

CONCLUSION

Issues of causation will continue to present some of the most difficult obstacles to just resolution of the expanding number of toxic tort claims. Statistical proof and latency of injury pose difficult evidentiary questions for courts seeking to resolve toxic tort causation issues. Consequently, the potential of DNA diagnostic technology to establish the genetic bases for many diseases and disease risks associated with exposure to hazard-

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228. Such situations may include not only issues of intrinsic and background risk, but also DNA tests related directly to the disease or disease risk when such tests have wide-ranging implications that would be difficult or impractical to keep from the parties. *See supra* note 226.

229. I propose this solution with full realization that it can only represent a possible lesser of evils for courts faced with difficult moral and ethical dilemmas. Individual variation in access to information, risk aversion, ability to deal with uncertainty, and even access to professional counseling pose problems which loom large in this proposal. In addition, there may be public policy implications regarding inclusion of punitive damages in such a proposal. These considerations are beyond the scope of the present Article.

230. For a summary of approaches to management of classes in toxic tort litigation, see generally 3 H. NEWBERG, NEWBERG ON CLASS ACTIONS ch. 17 (1985 & Supp. 1988).

ous agents may provide the toxic tort system with a valuable analytic tool.

Before the legal system can apply these new DNA technologies in toxic tort litigation, parties must be able to bring the results of DNA testing into evidence. The *Frye* test, which emphasizes general acceptance by an appropriate scientific community, may be appropriate for applications such as "DNA fingerprinting," where a focused application of the technology in specified fact settings is applied to a narrow and recurrent legal issue. However, this test may prove too restrictive to exploit fully the potential of DNA diagnostic technology. For the broader range of DNA testing procedures likely to be encountered in toxic tort litigation, a more productive approach would be to treat DNA diagnostic evidence like other traditional forms of evidence. The probative value of the evidence would be balanced against the possibility that the jury would be misled or prejudiced. This approach, embodied in the Federal Rules of Evidence, would evaluate general acceptance as only one among several factors relating to admissibility. However, courts would also be required to make relatively searching inquiries into the reliability and probative value of the DNA test.

Once admitted into evidence, results of DNA tests should lead to greater accuracy in resolving causation questions, including the extent of the association between the plaintiff's actual or alleged harm and the defendant's hazardous agent, and the extent of contribution by the plaintiff's intrinsic and background risks. Additionally, DNA technology should allow courts to shift focus from the troublesome issue of compensation for risk of future injury to compensation for actual genetic injury. However, courts will need to explore a variety of mechanisms to manage the ethical issues attendant on disclosure of genetic information to litigants unprepared psychologically to deal with such information.

Use of increasingly powerful DNA diagnostic technologies cannot answer all questions relating to probability and latent injury in toxic torts. Nevertheless, DNA testing can provide solid evidence regarding a central issue in many toxic tort cases—the structural integrity of the plaintiff's DNA. Appropriate use of such information can only represent a significant step forward in the search for truth.