The Bendectin Litigation: A Case Study in the Life Cycle of Mass Torts

Joseph Sanders

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The Bendectin Litigation: A Case Study in the Life Cycle of Mass Torts

by

JOSEPH SANDERS*

Table of Contents

I. Introduction ............................................ 303
II. Case Congregations ..................................... 305
   A. Beyond the Individual Case ......................... 305
   B. Case Congregations and Mass Torts ................. 307
   C. A Note on Case Studies ............................. 310
III. Bendectin Background .................................. 311
   A. The Firm .......................................... 311
   B. The Drug .......................................... 312
      (1) Thalidomide ................................... 313
      (2) MER/29 ....................................... 315
      (3) Bendectin ...................................... 317
IV. The Science ............................................. 321
   A. Background ........................................ 321
      (1) In Vitro Studies ................................ 322
      (2) In Vivo Studies ................................ 322
      (3) Epidemiological Studies ....................... 326
         a. The Primacy of Epidemiology? ............... 328
         b. Causal Inference ............................. 329
   B. The Bendectin Studies .............................. 331

* Professor of Law, University of Houston Law Center; B.A. 1966; J.D. 1969; Ph.D. 1974, Northwestern University. This Article is a revised version of a paper originally given at the Law and Society Meetings in Berkeley, California in June 1990. The research for the Article was supported by the University of Houston Environmental Liability Law Program. The Article would not have been possible without the assistance of the following University of Houston law students who work for the Environmental Law Liability Program: Michael Brown, Bill Robbins, Michael Rose, Lisa Salzman, and especially Terry Salem and Randall Terrell. The Environmental Law Liability Program is indebted to Marion Merrell Dow for its willingness to provide transcripts and other materials from the Bendectin cases. Herbert Kritzer and Michael Rustad also provided valuable materials. I wish to thank W. Glenn Forrester, Marc Galanter, Richard Lempert, Barry Nace, Al Schretter, the student editors of the Hastings Law Journal, and especially Michael Green for helpful comments on earlier drafts.
(1) In Vitro Studies ................................ 331
(2) In Vivo Studies ................................ 333
(3) Epidemiological Studies ........................ 339
a. Background ........................................ 339
b. The Evolution of Bendectin’s Epidemiological
   Research ........................................... 341
c. The Increase in Statistical Sophistication and
   Power .............................................. 342
d. The End of Research .............................. 345
(4) The Life Cycle of the Bendectin Science ...... 347

V. The Law .............................................. 348
A. Mobilization ....................................... 349
   (1) The “First Plaintiff” Problem ................. 349
   (2) Merrell’s Preventive Efforts ..................... 354
B. The Caseload and Trial Load ..................... 359
C. Rationing Law ...................................... 362
   (1) Procedural Rationing .......................... 362
   (2) Substantive Rationing ........................ 372
      a. Trial Court Opinions ......................... 372
      b. Appellate Court Opinions ................... 375
         (i) Oxendine ................................ 379
         (ii) Richardson ............................ 380
         (iii) Brock ................................ 382
         (iv) Ealy ................................. 383
c. A Defense Bias? .................................. 384

VI. The Larger Context ................................ 386
A. Substantive Rationing and the Judge-Jury
   Relationship ....................................... 386
B. Substantive Rationing in the School Desegregation
   Cases ............................................. 390

VII. Conclusion: Case Congregations and the Interaction of
     Macro and Micro Processes ..................... 391

VIII. Appendices ....................................... 394
One precedent creates another. They soon accumulate and constitute law. What yesterday was fact, today is doctrine.

Junius

I. Introduction

In an important recent article on prodefendant developments in products liability during the late 1980s, James Henderson and Theodore Eisenberg discuss areas of products liability law in which, during the early 1980s, new plaintiff breakthroughs had seemed imminent. One of these areas involved "[t]he imposition of liability on manufacturers of prescription drugs and other toxic substances based solely on epidemiological evidence of causal links between those products and plaintiffs' injuries ...." Among the products believed most likely to lead to such a breakthrough was the drug Bendectin, a widely prescribed morning sickness remedy alleged to cause birth defects in the children of mothers who had taken it. Given the sympathetic early reaction of several courts to plaintiffs' presentation of epidemiological proof of causation, the expectation that Bendectin cases would produce the next breakthrough for plaintiffs was more than reasonable. Yet, as Henderson and Eisenberg note, by the end of the decade early plaintiff victories were replaced by general defeat, "now mak[ing] it unlikely that Bendectin-related claims have a promising future."

What occurred in the intervening years to lead to this altered assessment? This Article addresses the question through an in-depth examination of the legal and scientific history of Bendectin. While Henderson and Eisenberg use the Bendectin litigation as an example of general trends in products liability law, the cases can be understood at a micro level as well. The failure of the Bendectin cases to usher in a new era of causal proof based solely on statistical data is due as much to factors specific to the drug as it is to large-scale changes in tort law. The Bendectin cases are not simply a random group of otherwise independent

3. Id. at 486.
4. See, e.g., Oxendine v. Merrell Dow Pharmaceuticals, 506 A.2d 1100, 1107-14 (D.C. 1986) (testimony of expert witness that drug used during pregnancy caused child's birth defects sufficient to carry case to jury where testimony was based on four different types of scientific studies).
5. Henderson & Eisenberg, supra note 2, at 491.
6. Id.
products liability cases that happen to involve the same drug. Rather, they are a collection of cases that, over time, have interacted in varied ways. Robert Emerson and, more recently, Marc Galanter have discussed the nature of these interactions. Like other mass torts, the cases concerned with Bendectin are usefully thought of as a unit—the Bendectin Cases. This "congregation" of cases, to use Galanter's term, has a beginning, a middle, and apparently a rapidly approaching end.

The topic of this Article is the life cycle of the Bendectin Cases. In Part II, I review Emerson's and Galanter's thoughts as to how the existence of a congregation affects the way individual cases are processed and resolved. Part III provides some background on Bendectin and its manufacturer, Merrell Dow. The Bendectin litigation, like that of most mass torts, revolves around a body of scientific evidence employed to demonstrate that the product does or does not cause the harm alleged. Part IV surveys the science on Bendectin, examining it from a congregation perspective as well. In Part V the attention turns to the law and legal process surrounding Bendectin. I examine plaintiff and defense efforts to mobilize resources and fight for position in the early stages of the congregation. After presenting an overview of the Bendectin litigation's life cycle, this Part concludes by discussing how the judiciary procedurally and substantively rations adjudicatory resources to large congregations such as the Bendectin Cases.

The Article concludes with some general thoughts about the relationship between judicial rationing and case congregations and the ways in which case studies such as this one help us understand the larger trends in tort law uncovered by Henderson and Eisenberg. In the Bendectin Cases, the judiciary has played a much more active role in rationing its resources than anticipated by Galanter. I discuss the factors that compel the judiciary in this direction when it is confronted with congregations of mass tort cases. Efficiency, stability, and consistency become relatively more important as a congregation matures, and the judiciary becomes more willing to intervene in the fact-finding process to achieve these goals. Case congregation analysis also helps explain the trend in products liability cases observed by Henderson and Eisenberg. The micro processes that create incentives for procedural and substantive rationing cumulatively create a macro effect of greater judicial willing-

8. See Marc Galanter, Case Congregations and Their Careers, 24 LAW & Soc'y REV. 371 (1990) [hereinafter Galanter, Case Congregations].
9. Id. at 371.
ness to intervene in the trial process, and this in turn tends to generate prodefendant outcomes.\textsuperscript{10}

\section*{II. Case Congregations}

In a common-law system the individual case is usually the primary unit of analysis. In keeping with the doctrine of stare decisis, lawyers strive to differentiate the present case from adverse precedents and align it with favorable ones. The tendency is to view each prior case as standing on its own—a unique combination of law and fact. Likewise, the disposition of each current case is thought to be governed by the idiosyncratic combination of lawyers, jurors, and judges who develop and decide it.

Even when cases are aggregated, it is most frequently according to legal principle or doctrine. Cases are viewed as links in a chain of conceptual development, as pieces in the precedent puzzle. From this perspective, the individual case remains the basic unit of analysis. The case is put into a larger picture as a brick is put into a wall; one discrete entity is combined with others to construct a larger edifice. Because the case is the law's basic unit of analysis and because each case is factually and procedurally distinct, we are easily led to adopt this narrow perspective.

More often than we might suppose, however, the study of the legal process will benefit from a different perspective. As Emerson has observed, this is most surely true of those circumstances in which the parties themselves do not "examine and dispose of cases as discrete units, treating each on its own merits independently of the properties and organizational implications of other cases."\textsuperscript{11}

\subsection*{A. Beyond the Individual Case}

Emerson describes three "holistic" frameworks that shape the way legal actors approach each individual case.\textsuperscript{12} First, there are "case streams," against which individual cases often are assessed by reference to what is "normal" for the stream.\textsuperscript{13} Those who must routinely process similar cases develop typification procedures that help them to channel normal cases into the proper stream and allow them to devote extra attention to abnormal cases.\textsuperscript{14}

\begin{flushright}
\textsuperscript{10} See Henderson & Eisenberg, supra note 2, at 491.
\textsuperscript{11} Emerson, supra note 7, at 425.
\textsuperscript{12} Id. at 427.
\textsuperscript{13} Id. at 426.
\textsuperscript{14} See generally Richard Lempert & Joseph Sanders, An Invitation to Law and Social Science 43-58 (1986) (explaining components of typification process); H. Lau-
Second, there are "caseloads." Legal actors are influenced by the number of cases they are required to process. When, as frequently occurs, caseloads overwhelm resources, decisions must be made regarding the time and energy to devote to each case. The focus is not on the individual case, but on the actor's responsibility for an entire caseload. Caseload effects are most apparent when cases that admittedly deserve attention are nevertheless given little or none because other cases are considered more serious.

When legal actors on both sides of a case face like caseloads and case streams, as in the criminal courts, they may well move beyond a process of comparison, into active horse-trading of case outcomes. Emerson gives as an example a typical multiple case bargaining session between prosecutor and defense attorney (reported by Feeley):

After they have discussed a few, the prosecutor might say rhetorically, "Jesus, I've already given you three nolles today, do you want me to go out of business?". Conversely, the prosecutor might hear a defense attorney plead, "You've put me through the wringer this morning; give me a break on this one!"

Third, Emerson notes the holistic effects of sequence and precedent. Cases that are recognized as "first cases" will be treated differently from later ones. For example, the first offense a teacher encounters in a school year may be treated more harshly than later infractions because the teacher wants to "set a precedent" by making an example of the offender. In this situation the first case is the most important, but this will not always be true. For instance, the legal actors might simply be unaware that a case is first or that the sequence of cases to come will prove important. At some point, however, the parties may recognize

15. Emerson, supra note 7, at 426.
16. Id.
17. Id. at 445 (quoting Malcolm Feeley, The Process is the Punishment: Handling Cases in a Lower Criminal Court 192-93 (1979)).
18. Id. at 427 (citing Gerald Levy, Ghetto School: Class Warfare in an Elementary School (1970)).
19. There are situations where only one party may view a case as part of a congregation. A defendant searching for the best case to take up on appeal may view a group of cases as a congregation, while each plaintiff sees its case in isolation. This broader perspective allows the defendant to "play for rules" in the appellate process (that is, to obtain a favorable appellate opinion, allowing the party to win future cases). This is one of the advantages Galanter attributes to parties who repeatedly litigate an issue. Marc Galanter, Why the "Haves" Come Out
they are involved in a sequence of cases and that earlier trial verdicts and settlements have important consequences for those coming later.\(^{20}\)

Each of these case aggregations reflects Emerson's basic point that since the work of legal actors sometimes focuses on a larger whole, the individual case is not always the most appropriate unit of analysis.\(^{21}\) In most of the examples discussed by Emerson, each aggregation's individual cases are related to each other only because of the organizational structure of the legal system. They are connected procedurally and structurally, but are factually discrete. In some situations, the connection goes beyond procedure and organization to substance. When this occurs, substance and legal organization interact, creating even stronger relationships among cases.

**B. Case Congregations and Mass Torts**

One area in which substantive interrelationships occur involves the mass tort. "Mass tort" is a slippery phrase. Here I mean injury to multiple parties allegedly caused by a single product or event. One frequent type of mass tort involves an event such as an airplane crash.\(^{22}\) Perhaps less frequent in number, but often involving many more actual and potential plaintiffs are mass torts involving toxic substances or defective products. These product-caused mass torts include injuries resulting from exposure to substances such as asbestos and Agent Orange,\(^{23}\) as well as injuries caused by products such as the Ford Pinto, Firestone 500 tires, or the Dalkon Shield.\(^{24}\) A third group of product-related mass tort

_Ahead: Speculations on the Limits of Legal Change, 9 LAW & SOC'Y REV. 95, 98-103 (1974) [hereinafter Galanter, Limits of Legal Change].

- A failure to recognize early on that a case or set of cases is the first in a large congregation can prove expensive for defendants if they settle the early cases for substantial sums or if they lose at trial; jury verdicts and settlement values of first cases are likely to become the baseline from which all future bargaining begins. This occurred with respect to the asbestos cases. See Francis McGovern, Toward a Functional Approach for Managing Complex Litigation, 53 U. CHI. L. REV. 440, 488 (1986). Past judgments are also playing a role in the Dalkon Shield bankruptcy proceedings. Under the Dalkon Shield Claimants' Trust Plan, claimants who elect Option III and show that the Shield caused their injury will be offered settlement amounts based on historic settlement values. 4 Toxics L. Rep. (BNA) No. 36, at 1034 (Feb. 14, 1990). For a general discussion of this and other ways of establishing the monetary value of cases in mass tort situations see Mark Peterson, New Tools for Reducing Civil Litigation Expenses (1983).

- Emerson, supra note 7, at 454.


- Id.

cases arises from exposure to drugs such as Thalidomide, DES, and Bendectin.\textsuperscript{25} While there are many differences between these various types of mass torts,\textsuperscript{26} all are characterized by the fact that their component cases share a single underlying type of harm; further, all the parties to the litigation recognize this fact.

A mass tort is a physical event, but it is also a social construct. The cases are quickly recognized as a group by the parties, the courts, and frequently by the public at large. The term "asbestos cases" or "Dalkon Shield cases" comes to have meaning outside the courthouse as well as within it. Not only are the cases arising from a mass tort connected substantively and procedurally, they have this important additional property: ultimately they share a common legal fate.\textsuperscript{27}

In this respect, mass tort cases are an example of what Galanter calls a case congregation; that is, "a group of cases that . . . share common features, that are shaped by a common history, that are subject to shared contingencies, and that lean into a common future."\textsuperscript{28} The central observation about case congregations is that they have careers; that over time, the volume and nature of cases change in an orderly fashion according to particular qualities of the congregation.\textsuperscript{29}

Galanter discusses several general factors that influence case careers. First, there is the volume of underlying activity that gives rise to certain types of cases. For example, the number of selective service cases filed in the federal courts mirrored the progress of the Viet Nam War. As the War ended so did the "draft" cases.\textsuperscript{30}

Second, there are several ways in which the legal system itself influences the career of a case congregation, at both an individual and a more holistic level. At the individual case level, early cases can generate mobilization efforts by potential claimants and preventive measures by potential defendants that ultimately alter the volume and the outcome of further suits (sometimes by altering the level of the underlying activity).\textsuperscript{31} Thus, as Galanter points out, confronted with the ruling in

\textsuperscript{25} Trangsrud, \textit{Joiner Alternatives, supra} note 22, at 780-81.
\textsuperscript{26} For example, toxic tort claims usually seek compensation for chronic injuries and diseases, whereas product claims usually seek recompense for traumatic injuries.
\textsuperscript{27} This does not mean, of course, that every case in the congregation has the same outcome. The outcome of an individual case is influenced by, among other factors, where it occurs in the life-cycle and whether it is swept into procedural rationing devices such as class actions.
\textsuperscript{28} Galanter, \textit{Case Congregations, supra} note 8, at 372.
\textsuperscript{29} \textit{Id.} at 373.
\textsuperscript{30} \textit{Id.} at 374 fig. 1.
\textsuperscript{31} \textit{Id.} at 379-84.
that under certain circumstances therapists have a duty to protect third parties from violence at the hands of their patients, therapists have responded by "giving more warnings, initiating more involuntary hospitalizations and taking more notes."  

On a more holistic level, the cases begin to interact with and affect each other. When cases become known as a collection, parties begin to share information and to coordinate efforts. These coordination networks are qualitatively different from the mobilization and prevention activities discussed above because they specifically involve intercase activities. Occasionally, coordination becomes institutionalized. For example, information on school desegregation efforts was shared among several special organizations, including the NAACP, the Legal Defense Fund, the Lawyer's Committee for Civil Rights Under Law, and the Harvard Center for Law and Education.

Coordination networks represent one way in which parties may respond to a case congregation. Courts also respond to groups of cases. A fundamental holistic response of the courts is rationing. The most extreme type of rationing is exemplified by worker's compensation statutes, which attempt to close the courts to most work-related injury claims. Less dramatic rationing efforts involve class actions, special masters, and similar devices.

A third holistic effect is depletion. As the easy cases are resolved, a smaller and smaller group of cases, in which it becomes increasingly difficult for plaintiffs to prevail, is left behind. Depletion is most obvious in disasters such as airplane crashes, in which there is a known finite number of victims; but it occurs in other situations as well, especially

35. Galanter, Case Congregations, supra note 8, at 387.
37. Galanter, Case Congregations, supra note 8, at 385.
39. Galanter, Case Congregations, supra note 8, at 388.
where defendants begin to take preventive measures to thwart future claims.\footnote{40}{Id.}

Finally, there are what Galanter calls \textit{outcome-stabilization} effects.\footnote{41}{Id. at 389.} Liability determinations and damage awards become more predictable as more and more cases are tried. In the area of mass torts, perhaps the most developed theory of outcome stabilization is Francis McGovern's "cyclical theory of mass torts."\footnote{42}{See McGovern, \textit{supra} note 20, at 482.} According to McGovern:

\begin{quote}
In the early stages of the cycle, defendants tend to win more cases than plaintiffs because of strategic and informational superiority. If the litigation has any merit, however, plaintiffs will eventually develop successful information and strategies and win an extremely high percentage of the cases tried. Next, the plaintiffs will bring cases for trial that stretch the envelope of viable plaintiffs too far, and defendants will create more effective counterstrategies, resulting in a reduced percentage of plaintiff victories. Eventually, after full aggregation and dissemination of information, crystallization of the law, and thorough development of strategies, there will be a rough equilibrium of trial results. Remaining variations will then be due to jury demographics, attorney caliber, and random events during trials. Although perhaps it is counter-intuitive, settlements will also reflect this equilibrium: the average settlement amount will be virtually identical to the average jury verdict. The variance, however, will be substantially different. Settlements for similarly situated plaintiffs will be extremely similar; verdicts will vary in accordance with idiosyncracies of the trial process.\footnote{43}{Id. (footnotes omitted).}
\end{quote}

As this theory suggests, the careers of some case congregations resemble a life cycle. Early on there are a few immature cases. Slowly, a mature and substantial body of litigation develops and a period of relative stability ensues. Finally, there is a period of decline, as depletion and rationing cause the cases to move out of the legal system. This Article examines the life cycle of a particular case congregation: the Bendectin Cases.

\section*{C. A Note on Case Studies}

Because this Article explores hypotheses about the careers of case populations through a detailed examination of one particular congregation of mass tort cases, some initial observations about such an enterprise are in order. From one perspective, the case study is an inherently weak form of hypothesis testing. Many unique features of the Bendectin Cases have influenced the way the congregation has developed. It is difficult to
disentangle these idiosyncratic features from the underlying processes. Thus, the analysis of a single congregation of cases, no matter how detailed, cannot alone support acceptance or rejection of general hypotheses about case careers.

From another perspective, however, the case study is a particularly useful methodology for examining the careers of case congregations. It forces us to remain close to the evidence, and more importantly, it allows us to adopt the perspective the parties had as they worked toward an understanding of what they had on their hands.\textsuperscript{44} Case congregations are "cultural categories created by an act of labeling."\textsuperscript{45} Unlike the first disciplinary problem of the school year, in which scenario the teacher knows a priori that this is the first of a relatively well defined population of cases, some first mass tort cases arise while yet unlabeled. The process by which the contours of a congregation of cases are defined is crucial to the ultimate development of the congregation. This definitional work cannot be studied easily from a more general point of view. The case study approach offers the opportunity to observe the micro processes through which macro changes in litigation rates occur.\textsuperscript{46}

III. Bendectin Background

A. The Firm\textsuperscript{47}

The William S. Merrell Company, based in Cincinnati, Ohio, was one of the oldest "ethical" (prescription) pharmaceutical companies in the United States. In 1938 it was acquired as a wholly-owned subsidiary by Vick Chemical, a recently formed Delaware Corporation. Vick's product line was aimed at the symptomatic relief of the common cold (Vicks Vapo-rub). Fearing a cure for the cold was at hand, it acquired William S. Merrell for its prescription drug line. Bendectin was developed during the time William S. Merrell was a subsidiary of Vicks. In 1960 the Vick Chemical Company changed its name to Richardson-Merrell, Inc. (Vick had been founded by the Richardson family). A few

\textsuperscript{44} See generally Barney G. Glaser & Anselm L. Strauss, The Discovery of Grounded Theory: Strategies for Qualitative Research (1967) (arguing for theories "grounded" in the details of particular events).

\textsuperscript{45} Galanter, Case Congregations, supra note 8, at 373.


\textsuperscript{47} This history comes from personal correspondence from W. Glenn Forrester, attorney for Marion Merrell Dow, to Joseph Sanders, (Aug. 23, 1990) (on file with author) and from Carol Loomis, Richardson-Merrell Unswallows a Pill, FORTUNE, Jan. 12, 1981, at 54-56.
years later, the William S. Merrell Company was merged into Richardson-Merrell and ceased to exist as a separate entity.

In 1981 a new company, Richardson-Vicks, became an independent, publicly held corporation. It received all of Richardson-Merrell's business and assets except its ethical pharmaceutical business. Richardson-Vicks kept such products as Clearasil, NyQuil, Lavoris, and Oil of Olay. All shares of what remained of Richardson-Merrell, including Bendectin, were sold to Dow Chemical Company for $260 million. Dow was apparently the high bidder among a number of potential buyers, including G.D. Searle. At this time, Richardson-Merrell's name was changed to Merrell Dow Pharmaceuticals. In a sense, the 1981 transaction undid Vick's 1938 acquisition of Merrell, as it once again separated the prescription and non-prescription parts of the company. One result of these transactions was to spin off the parts of Richardson-Merrell that were least threatened by litigation, and sell the rest to Dow.

Dow Chemical became the parent of a subsidiary with outstanding claims involving a number of products, including Bendectin, Thalidomide, MER/29, DES, and the DPT vaccine. It was Bendectin that presented the most serious threat to the new Merrell Dow. Paul Oreifice, Dow Chemical's chief executive, said the Bendectin suits were a "sobering negative," but were adjusted for in Dow's bid.48 (At the time, Dow itself was embroiled in major products liability litigation involving Agent Orange.)

Most recently, in 1989 Merrell Dow merged with Marion Laboratories and became a wholly-owned subsidiary of Marion. Marion's name was changed to Marion Merrell Dow. Currently, Dow Chemical owns almost 67% of Marion Merrell Dow's stock.

The checkered corporate history of Merrell has created several different named defendants in the Bendectin litigation. For simplicity, I refer to the defendant as Merrell, the one word that has always been a part of the corporate name of Bendectin's manufacturer.

B. The Drug

Bendectin and the Bendectin litigation must be understood in light of two other drugs with which the Merrell Corporation was associated during the time Bendectin was being developed and sold: Thalidomide and MER/29. The obvious harm caused by these two drugs set the stage for allegations that Bendectin, too, was a product that never should have been marketed.

48. Loomis, supra note 47, at 55.
(1) Thalidomide

Thalidomide was first synthesized in West Germany in 1953 by Ciba. Because the drug had no pharmacologic effect on laboratory animals, the company discarded it. Another West German firm, Chemie Grünenthal, then began to explore its uses. Because the drug's molecular structure suggested it might be useful as a sedative, Grünenthal experimented with it as an anticonvulsant for epileptics. The drug failed to prevent convulsions, but did work as a hypnotic, providing a deep, natural sleep without a hangover. In 1960 the drug was given the trade name Contergan and became the favorite sleeping pill in West Germany, available over the counter to anyone. The drug was combined with other ingredients, including aspirin, to form compounds for colds, coughs, flu, headaches, and asthma. In liquid form it was widely used to sedate children. And as an antiemetic it was used for nausea during pregnancy.

Thalidomide was marketed around the world, and was usually sold without prescription. Merrell's Canadian branch sold the drug as Kevadon. Merrell obtained rights to market it in the United States under the same trade name and applied for FDA approval in September 1960. Shortly thereafter, the first reports of trouble began to surface in Germany; patients complained of tingling hands, atrophy of the thumb, and other forms of peripheral neuropathy. As a consequence, the drug was made available only by prescription. In the summer of 1961 German doctors first observed that an increasing number of babies were being born with arm and leg deformities. The full extent of the disaster began to unfold. Ultimately, Thalidomide proved to be one of the most potent human teratogens ever found.

In the United States, large-scale disaster was narrowly averted. The Food Drug and Cosmetic Act of 1938 required the seller of a new drug

50. Id. at 460.
51. Id.
52. Id.; Helen B. Taussig, A Study of the German Outbreak of Phocomelia, 1180 JAMA 1106 (1962).
53. Sherman & Strauss, supra note 49, at 461. A teratogen is "[a] substance that produces abnormalities in the embryo or fetus by disturbing maternal homeostasis or by acting directly on the fetus in utero." A Dictionary of Epidemiology (John M. Last ed., 2d ed. 1988)). Sometimes the term is used in a narrower sense to describe substances that produce a disruption during gestation that results in malformation of the fetus. Jennie Kline & Zena Stein, Circumstances of Exposure and Reproductive Consequences, in Epidemiology and Health Risk Assessment 256, 262 (Leon Gordis ed., 1988).
to present convincing scientific evidence of the drug's safety. Merrell presented the existing data and was allowed to begin clinical investigation, but not widespread sale of Thalidomide. Approval might quickly have followed had it not been for the tenacity of Dr. Frances Kelsey, a physician and pharmacologist at the FDA who took notice of the earlier German reports of adverse neuropathic effects. She also noted that the drug was prescribed for morning sickness; from her earlier work with quinine on a malaria project in World War II, she was acutely aware of the pharmacological differences between fetuses and adults. She therefore requested more information from Merrell and withheld FDA approval.

Circumstances and the FDA saved Merrell from disaster, but just barely. Moreover, Merrell did not come away with completely clean hands. While awaiting FDA approval, Merrell had distributed the drug for investigational use. The company engaged in what might charitably be called extremely lax behavior. While Thalidomide was still under review by the FDA, 2,500,000 tablets were distributed and given to nearly 20,000 individuals, including 624 pregnant women. Ultimately, at least ten American babies were born with defects attributed to Thalidomide.

55. Id. § 355.
57. Id. Ironically, the FDA's primary concern was that Thalidomide might cause peripheral neuropathy in adults, not birth defects in infants. One of the most active human teratogens known had produced no teratogenic effects in mice, rats, or hamsters. Id. Teratogenic effects of Thalidomide are observable in monkeys and rabbits, but finding an appropriate animal model is not always an easy task. See id. at 464.
58. Richard E. McFadyen, Thalidomide in America: A Brush with Tragedy, 11 CLIOMEDICA 79 (1976). Sherman and Strauss note that even today a product like Thalidomide might slip through the FDA's safety net. Sherman & Strauss, supra note 49, at 464-65. If a suitable animal model could not be found to indicate the potential for fetal injury, tests on human subjects would not uncover the danger because current regulatory policy excludes women in the first trimester of pregnancy from randomized trials of new drugs. They conclude that "[u]nder current FDA policy the only way to prevent another thalidomide disaster is to forbid pregnant women from taking any but the most essential medications—a mode of prevention well known and readily applicable before the 1962 regulations were instituted." Id. at 465.
59. McFadyen, supra note 58, at 86.
60. Today, the exact way in which Thalidomide affects humans is still not entirely clear. Sherman & Strauss, supra note 49, at 462. Moreover, the birth defects associated with the drug are largely indistinguishable from birth defects arising from other causes. Id.; Janet McCredie, Mechanism of the Teratogenic Effect of Thalidomide, 2 MED. HYPOTHESES 63, 63 (1976). The conclusion that Thalidomide causes these defects rests on the clinical evidence showing an overwhelmingly greater likelihood of defects among children of women who ingested the product in the early weeks after conception. The best estimates of what now is largely an unknowable figure is that if Thalidomide is taken between 35 and 50 days after menstruation, the risk of malformation is over 50%. W. Lenz, Malformations Caused by Drugs in Pregnancy, 112 AM. J. DISEASES CHILDREN 99, 105 (1966); Sherman & Strauss, supra note 49, at 462.
Merrell reached settlements with victims in the United States and Canada ranging from $100,000 to $999,000.61

(2) MER/29

The second drug that is important to the Bendectin story is MER/29. Merrell designed MER/29 to reduce the level of cholesterol in the bloodstream. Between 1956 and 1959, tests were conducted on animals (rats, dogs, and monkeys) and humans to explore its therapeutic and toxicologic effects. In June 1959 Merrell applied to the FDA for permission to market the drug. The application was approved in April 1960, and Merrell subsequently promoted MER/29 as the first drug to lower cholesterol safely. In fact, the drug was anything but safe. In December 1961, when the drug had been used by approximately 400,000 individuals, Merrell mailed a letter approved by the FDA to all practicing physicians. This so-called "Dear Doctor" letter warned for the first time that MER/29's potential side effects included cataracts, baldness, and severe dermatitis. In April 1962 Merrell voluntarily withdrew the drug from the market. The withdrawal followed an FDA inspection of Merrell's records that revealed the FDA had received incorrect animal data from the firm. The following month, the FDA suspended the new drug application for MER/29 based upon the clinical experience showing its danger to humans.62

The following year Merrell, its parent Richardson-Merrell, and three of Merrell's scientists were indicted under the federal False Writing Statute63 for withholding data from the FDA.64 For example: the FDA had not been told that two laboratory dogs had developed cataracts, even though this fact was noted on their autopsy sheets;65 results of tests on monkeys had been altered; and falsified data had been presented to the FDA.66 The defendants, including Dr. Evert van Maanen, Merrell's director of biological sciences, entered nolo contendere pleas. The firms

64. Rheingold, The MER/29 Story, supra note 62, at 120-21.
65. Id. at 119.
(Merrell and Richardson-Merrell) were fined a statutory maximum of $80,000, and the scientists received suspended sentences.\textsuperscript{67}

In part, the defendants' pleas may have been an attempt to limit the use of the criminal trial record in civil suits. Nonetheless, the first civil suits were filed in 1961 and filings continued throughout the decade, peaking in 1964. While MER/29 gave rise to many suits, each alleged injury was relatively minor in comparison to those allegedly caused by Thalidomide and Bendectin. According to one estimate, at least 5000 people were harmed by MER/29, and over 1500 suits eventually were filed.\textsuperscript{68} Merrell won most of the early cases, in 1964 and 1965, but plaintiffs prevailed in several subsequent cases in 1966 and 1967.\textsuperscript{69} These plaintiff victories increased the value of untried cases—it is estimated that Merrell ultimately paid over $200 million in damages.\textsuperscript{70}

The MER/29 episode gained widespread publicity. Numerous books and articles were written about both the product and the company that developed it. None was complimentary, and many vilified Merrell.\textsuperscript{71} The company became one of the principal bad guys in an industry of bad guys.

Merrell's experience with these two earlier drugs set the stage for the Bendectin episode. While the litigation surrounding Thalidomide and MER/29 is not legally relevant to the Bendectin litigation, it did make Merrell a likely target for litigation. To employ a criminal law metaphor, Merrell had one prior and on another occasion had been found lurking around the scene of a crime. The MER/29 episode in particular raised questions concerning Merrell's testing program.\textsuperscript{72} When hints of trouble emerged concerning Bendectin, many in the plaintiff's bar must have concluded that, with respect to this firm, where there was smoke there must be fire.

\begin{footnotes}
\item 67. Rheingold, \textit{supra} note 62, at 121.
\item 68. \textit{Id.}
\item 69. \textit{Id.}
\item 70. \textit{See} Kolata, \textit{supra} note 61, at 519.
\item 71. \textit{E.g.}, \textsc{Ralph A. Fine}, \textsc{The Great Drug Deception: The Shocking Story of MER/29 and the Folks Who Gave You Thalidomide} (1972); \textsc{John Fuller}, \textsc{200,000,000 Guinea Pigs} (1972); \textsc{Philip Knightley et al.}, \textsc{Suffer the Children: The Story of Thalidomide} (1979); \textsc{Milton Silverman}, \textsc{The Drugging of the Americas} (1976); Sanford Ungar, \textit{Get Away With What You Can}, \textit{in} \textsc{In the Name of Profit} (Robert Heilbroner ed., 1973).
\item 72. \textsc{Braithwaite}, \textit{supra} note 66, at 60-65.
\end{footnotes}
In 1953 Dr. Raymond Charles Pogge, director of medical research at Merrell, came up with the idea of using pyridoxine (Vitamin B-6) to create an antinausea medicine to combat morning sickness among pregnant women. After preliminary discussions with others at Merrell, including Dr. Margaret Higgins Morson and D.B. Bowles, Dr. Pogge proposed a drug made up of three ingredients, each of which had been marketed separately: dicyclomine hydrochloride (an antispasmodic); doxylamine succinate (an antihistamine acting as an antinauseant); and pyridoxine hydrochloride. The new drug was named Bendectin. Prior to the Bendectin formulation, each ingredient had been prescribed singly, and each alone had no recorded adverse effect on humans.

Bendectin was first marketed in 1956. In the United States, it was available only by prescription. In 1976, the drug was reformulated to eliminate the first ingredient, dicyclomine hydrochloride, because it was found not to add anything to the efficacy of the formulation. The possibility that Bendectin might be a teratogen apparently was first brought to


76. Id. Bendectin is also known by the trade names Debendox, Lenotan, and Merbental, and is available in many other countries. Id. In both Canada and Britain the drug was available over the counter, although usually dispensed by prescription. Id.

77. U.S. DEP'T HEALTH, EDUC. & WELFARE, ADVISORY COMM., THE BENDECTIN PEER GROUP REPORT (1975). This finding was based primarily on two studies. The first was a four-way comparison of dicyclomine and doxylamine singly and in combination, versus a placebo in 716 patients. The main effect of dicyclomine alone was greater than the placebo, but not statistically significant. The effect of dicyclomine in the presence of doxylamine showed similar results—a marginal but not statistically significant increase in efficacy when compared to doxylamine alone.

The second study was a double-blind eight-way comparison of all three ingredients and their combinations with a placebo in 2300 patients. While the study demonstrated the efficacy of three ingredient Bendectin when compared to the placebo, it failed to demonstrate any dicyclomine contribution. Doxylamine again was the major component, demonstrating a significant effect on every response variable including physician assessments of patient nausea, patient self-reports of nausea, eating habits, and daily activities. The study also demonstrated for the first time in double-blind experimental conditions that pyridoxine did have a significant effect on maternal self-reports of nausea when compared with all combinations lacking this.
public attention in 1969. Dr. Donald Paterson, a physician in British Columbia filed a drug evaluation report ("DER") with the Canadian Food and Drug Directorate and wrote a letter to the editor of the Canadian Medical Association Journal questioning Bendectin's safety. The same year, Merrell forwarded to the FDA physician reports of limb reductions associated with the use of Bendectin, and physician letters questioning the drug's safety appeared in the British Medical Journal. Questions concerning the safety of Bendectin and whether it, like Thalidomide, was a teratogen continued to accumulate; by the mid-1970s, the FDA had on file over ninety physician reports noting defects among children whose mothers had taken Bendectin. By the end of the decade, personal injury suits had been filed, the first going to trial in January 1980. In 1980, following a number of reports in the popular press that Bendectin was unsafe and under pressure from Congress to investigate the drug, the FDA conducted a two-day hearing as to its teratogenic effect. The FDA's conclusion, based on the report of its Fertility and Maternal Health Drug Advisory Committee, was that "available data do not demonstrate an association between birth defects and drug. Pyridoxine also had a marginally significant effect on physician evaluations of nausea (p=.08). See also Korok, supra note 75, at 923.

79. Id.
81. See Mekdeci v. Merrell Nat'l Lab., 711 F.2d 1510, 1516 (11th Cir. 1983). Mekdeci was originally filed in June 1977, well before any other cases. The plaintiff’s mother had actively sought a reason for her son’s defect and, on the basis of her own research, determined that it was caused by Bendectin. Id. Mekdeci is discussed in detail infra notes 204-22 and accompanying text.
83. See, for example, a letter from California Representative Don Edwards to Jere Goyan, FDA commissioner, expressing his “strongest concern over the FDA’s inaction regarding the drug Bendectin.” The congressman concluded, “I believe that . . . the substantial amount of new evidence linking Bendectin to fetal malformations mandate [sic] the immediate removal of this harmful drug from the marketplace.” Letter from Representative Edwards to FDA Commissioner Jere Goyan (May 7, 1980) (on file in the offices of the Environmental Law Liability Program, University of Houston Law Center).
Bendectin, . . . but [the drug] should be used only when conservative treatment fails." The drug was allowed to remain on the market. Nevertheless, in response to the increasing number of lawsuits and the decline in sales caused by negative publicity, Merrell voluntarily withdrew Bendectin from the market in 1983. The litigation has continued to the present. Thus far, over 2100 Bendectin claims have been made against Merrell. This figure includes almost 1700 suits brought by children who suffer from a wide variety of birth defects, allegedly because their

84. DEP'T HEALTH & HUMAN SVCS. NEWS, Oct. 7, 1980. In response to the question, "[d]o the animal and human data reviewed by you and presented to you support the conclusion that Bendectin is associated with an increased risk for human birth defects?" the Advisory Committee answered: "No, the data presented to date do not demonstrate an association of increased risk for human birth defects with the use of Bendectin. The committee notices with concern two studies that suggest an association between Bendectin and certain anomalies. Therefore, a residual uncertainty regarding a possible relationship does exist." FDA, UNEDITED ANSWERS TO QUESTIONS SUBMITTED TO THE FERTILITY AND MATERNAL HEALTH ADVISORY COMMITTEE (Sept. 15, 1980).

85. For a brief history of the circumstances leading up to the drug's withdrawal, from one critical of this result, see David Williams, How Nader Campaign Killed a Beneficial Drug, HUM. EVENTS, January 14, 1984, at 10-11. At the time, the withdrawal met with widespread criticism. An editorial in the Wall Street Journal concluded:

[T]here's something terribly askew with a legal system that cannot distinguish between a thalidomide and a Bendectin. Let us hope that the withdrawal of Bendectin is not a harbinger of other products being driven from the market by legal costs, even when the weight of medical evidence suggests they haven't been at fault.

WALL ST. J., June 15, 1983, at 34. Bendectin's fate has influenced the development of case law in the products liability area. For example, the California Supreme Court cited the withdrawal of Bendectin as one reason why it should reject a design defect analysis in prescription drug cases, declaring instead that all prescription drugs are unavoidably unsafe products as described in comment k of section 402A of the Second Restatement of Torts. Brown v. Superior Ct., 44 Cal. 3d 1049, 1064, 751 P.2d 470, 479, 245 Cal. Rptr. 412, 421 (1988). The court commented:

The possibility that the cost of insurance and of defending against lawsuits will diminish the availability and increase the price of pharmaceuticals is far from theoretical . . . . For example, according to defendant E.R. Squibb & Sons, Inc., Bendectin, the only antinauseant drug available for pregnant women, was withdrawn from sale in 1983 because the cost of insurance almost equalled the entire income from sale of the drug. Before it was withdrawn, the price of Bendectin increased by over 300 percent.

Id. at 1064, 751 P.2d at 479, 245 Cal. Rptr. at 421 (citations omitted).

86. Over 800 claims were disposed of at a single common issues trial in Ohio in 1985 (the Common Issues Trial). The jury in the Common Issues Trial was asked to consider whether Bendectin causes eight different categories of birth defects:

1. Musculoskeletal Defects: Defects in the muscles, bones and cartilage of a child, including limb defects.
2. Central Nervous System Defects: Defects in the brain, spinal cord and nerves.
3. Heart Defects: Defects in the heart and circulatory system.
4. Head Defects: Defects in the head and face.
5. Respiratory Defects: Defects in the larynx, trachea, bronchi, and lungs.
mothers used the drug during pregnancy. Because every state presumably has childhood disability provisions in its statute of limitations, new suits may be brought into the next century.  


As the report indicates, although claims have been based upon many types of injuries, the majority involved musculoskeletal defects—specifically limb reductions. As I discuss below, this tendency is much more pronounced when only those individual cases that have gone to trial are examined. See infra notes 318-20 and accompanying text.

87. Letter from Alfred Schretter, attorney for Merrell-Dow, to Sanford Gaines (August 9, 1989) (on file in the offices of the Environmental Law Liability Program, University of Houston Law Center). For nearly identical figures as to the child-plaintiff cases see U.S. GEN. ACCT. OFF., PRODUCT LIABILITY: EXTENT OF “LITIGATION EXPLOSION” IN FEDERAL COURTS QUESTIONED 35 (January 1988) [hereinafter GAO, “LITIGATION EXPLOSION” QUESTIONED].


89. Because of these disability provisions, the Bendectin Cases have a “long tail”: a long period between the time when a product is distributed in the market and the time after which a suit may not be brought. Some have argued that the long tails of many products liability claims have led to an unravelling of insurance markets. E.g., George L. Priest, The Current Insurance Crisis and Modern Tort Law, 96 YALE L.J. 1521, 1582-90 (1987). Insurance companies have responded to long-tailed risks by being less willing to write “occurrence” policies covering all injuries that occur during a time period. Instead, they have turned to “claims-made” policies that cover only claims made while the policy is in effect. This, in turn, has led the attorneys general of several states to sue a number of insurance companies. See Joseph Sanders & Craig Joyce, “Off to the Races”: The 1980s Tort Crisis and the Law Reform Process, 27 HOUS. L. REV. 207, 215 n. 33 (1990).
With this background in place, the next two Parts of this Article examine the science and the law surrounding the Bendectin litigation. In each Part, the primary objective is to take a “life cycle” perspective. Neither the science nor the law is analyzed as a collection of autonomous studies or cases, but rather as part of a larger, interactive whole. As shall become clear, both the science and the law have been fundamentally altered by their interaction with each other.

IV. The Science

Science is discussed first because it was, in a sense, a leading indicator of what was to happen in the Bendectin trials. To know the science was, to some extent, to be able to predict what would come. On the other hand, the law, especially as reflected in appellate opinions, was a lagging indicator—a reflection of what already had occurred.

A. Background

Bendectin was already in the marketplace when Merrell experienced problems with Thalidomide and later with MER/29. In part because Bendectin was comprised of existing drugs, the compound had not undergone substantial testing when introduced. At that time, FDA standards did not require testing for teratogenicity, and Merrell did not test Bendectin for teratogenicity until after the Thalidomide disaster in 1961. Indeed, the Thalidomide disaster acted as an important catalyst for reformation of the Food and Drug Act.

In the aftermath of that disaster, Merrell began investigating the safety of Bendectin. As is typical, early research involved in vitro and in vivo studies. Later, when questions arose regarding Bendectin’s safety, new in vivo studies were undertaken, and researchers conducted a series of epidemiological studies assessing Bendectin’s effect on human fetuses. Because the relative probative value of various types of data became a determining factor in the Bendectin cases, the next sections provide an overview of each type of research: in vitro, in vivo, and epidemiological.

91. Id. at 42.
92. Id. at 56-57; Sherman & Strauss, supra note 49, at 463-64.
93. See supra notes 78-83 and accompanying text.
94. In addition to using these three types of studies, toxicologists also draw inferences regarding a drug’s effects from its structure. If the molecular structure of a suspect compound is similar to that of a known teratogen, there is some reason to suspect that the compound will have similar effects. C.T.G. King et al., Antihistamines and Teratogenicity in the Rat, 147 J. PHARMACOLOGY & EXPERIMENTAL THERAPEUTICS 391-98 (1965); Michael Green, Expert
Following this, I discuss the scientific findings and how they changed as the Bendectin litigation matured.

(1) In Vitro Studies

In vitro studies expose single cells, organs, or even whole embryos maintained in a culture to some substance and examine the biochemical events. In vitro studies testing for teratogenicity. Organ cultures of embryonic limb buds are the most frequent subjects of in vitro studies. Experiments indicate that embryonic mouse limbs developing in a culture exhibit pathological responses to several teratogens similar to those developing in vivo. An important advantage of in vitro studies is cost. There also is an as yet unrealized longer term advantage: by using animal and human cell cultures, in vitro studies allow direct comparisons across species of the effects of cell and organ exposure to drugs. Ultimately, such comparisons may enable us to make better cross-species extrapolations of in vivo study results. There are, however, many aspects of teratogenicity that cannot be investigated through in vitro studies, and extrapolation to live animals, not to mention humans, is difficult at best.

(2) In Vivo Studies

In vivo studies examine the effects of a drug on various animal species thought to be similar to humans in their response to certain drugs. Different protocols are employed depending on the type(s) of injuries for
which one is testing. Today the protocols for teratogenic effects testing are fairly well standardized and call for tests on at least two species at two dose levels. For each species a minimum \( N \) consists of two test groups and one control group, each containing twenty rodents or ten rabbits. Because of the nonnormal distribution of teratological events, nonparametric tests are often employed, with the litter, not the fetus, as the unit of analysis. The percentage of fetuses affected within a litter is the basic estimate of statistical effect. By the mid-1970s, around the time Bendectin’s safety came into question, approximately 2000 chemicals (mostly drugs) had been tested for teratogenic effects. Of those tested, approximately one third had shown some teratogenic effect.

Perhaps the central issue with respect to \textit{in vivo} teratology studies is the degree to which the results can be extrapolated from animals to humans. Because many substances produce an adverse effect in only a small percentage of organisms when ingested at a rate similar to that encountered in the environment or prescribed by a physician, it takes a large number of animal subjects to detect a given substance's effect with any reliability. Smaller samples would generate an unacceptably large number of false negatives (failure to detect an effect when it exists). Thus, animals are generally administered high doses of the drug in hopes of maximizing the incidence of effects. If there is a positive result,
toxicologists must then extrapolate a predicted incidence at a more realistic lower rate. Unfortunately, there is no single agreed upon extrapolation model for dose-rate effects, and competing models generate widely differing predictions at low response rates. Extrapolation is particularly difficult in the case of teratogenic effects.

Once a dose rate extrapolation has been made, a second extrapolation is necessary to adjust across species. Again, unfortunately, there is no uniformly accepted formula for the adjustment. Two common formulas respectively compare either the body weight or the surface area of the animal with the corresponding human measurement. The use of these different scaling factors can lead to widely divergent estimates of effects on humans.

Finally, it is important to emphasize the different role animal studies play when they are used to regulate risk versus when they are used to

defects. Moreover, a drug's effects may vary depending on when it is given during organogenesis. Thus, dose variations over the entire period of organogenesis may produce different results than dose variations on a single day of gestation. Manson et al., supra note 96, at 149-51.

Howard Latin, Good Science, Bad Regulation, and Toxic Risk Assessment, 5 YALE J. ON REG. 89, 98 (1988). For example, carcinogenic effects often are measured on a dose scale that defines a base dose rate as the dose necessary to produce cancer in 50% of a sample of test animals. David S. Salsburg, Statistics and Toxicology: An Overview, in Scientific Considerations in Monitoring and Evaluating Toxicological Research 123, 127-29 (Edward J. Gralla ed., 1981) [hereinafter Scientific Considerations]. Several statistical models, including log-normal, log-logistic, and single-hit models are used to extrapolate to lower dose rate effects. All of the extrapolation models produce similar estimates when the dose rate is relatively close to 50%, but they produce widely divergent results at low doses. For instance, at a dose that is 1/1000 of the 50% dose, the single-hit model gives an estimated response rate 200 times greater than the log-normal model. OSHA Generic Cancer Policy, 45 Fed. Reg. 5001, 5184-5185 (1980).

Hogan and Hoel note that when compared to carcinogenic or mutagenic effects, teratogenic effects are “perhaps the most difficult to address in terms of species extrapolation and quantitative risk assessment.” Michael D. Hogan & David G. Hoel, Extrapolation to Man, in Principles and Methods of Toxicology, supra note 96, at 711, 724. The biologic mechanisms underlying teratogenic responses are poorly understood, partly because the basic unit of the experiment is not really one organism, but two—the mother and her offspring. Id. This greatly increases the difficulty of extrapolating from high to low dose rates.

James P. Leape, Quantitative Risk Assessment in Regulation of Environmental Carcinogens, 4 HARV. ENVTL. L. REV. 86, 98-99 (1980). Implicit in these formulas is an assumption that animals and humans respond similarly to the substance.

For example, the National Academy of Sciences estimated the incidence of cancer in humans exposed to Saccharin using different formulas to extrapolate from animal data. A body-weight scale predicted 210 cases per million people exposed. A surface-area scale predicted 1200 cases. A third formula using a total lifetime dose conversion scale predicted 5200 cases per million. Id. In general, comparisons between cancer risk estimates based on extrapolations from animal data and actual human epidemiological data indicate that estimates have been accurate for only half the substances examined. See Jack L. Landau & W. Hugh O'Riordan, Of Mice and Men: The Admissibility of Animal Studies to Prove Causation in Toxic Tort Litigation, 25 IDAHO L. REV. 521, 547-548 (1988-89).
prove causation in a tort case. When testing a new drug, the critical question is whether there might be a teratogenic effect in humans even though no effect is detected in test animals.\textsuperscript{112} (Recall that Thalidomide did not produce effects in rats or hamsters.)\textsuperscript{113} When we move from the laboratory to the courtroom, however, the question changes subtly. In the courtroom the crucial issue becomes whether a known effect in a test animal is probative of causation in humans. On the first question, a 1980 FDA report indicated that of thirty-eight compounds known to produce human birth defects, all but one have produced a positive result in at least one animal species and 80% are positive in more than one species.\textsuperscript{114} Thus, the Thalidomide experience notwithstanding, negative \textit{in vivo} tests on two species are relatively strong evidence that a drug will be safe for humans.

As to the second question, the answer appears to be that the relationship is weaker and somewhat dependent on the species used to prove causation.\textsuperscript{115} According to the FDA study, of 165 compounds for which no human teratologic effects had been reported only 28% appeared negative in all animal species tested, and 50% appeared negative in multiple species.\textsuperscript{116} On the other hand, over 41% of the compounds appeared to be positive in more than one animal species.\textsuperscript{117} In a species-by-species analysis, the FDA found that a negative response to substances for which human teratologic effects have not been reported was observed only 35% of the time in mice and hamsters, 50% in rats, 70% in rabbits, and 80% in monkeys.\textsuperscript{118} Compounding the problem of what to make of positive

\textsuperscript{112} Because there are no commonly accepted models for extrapolation, one technique is to employ some arbitrary safety factor when scaling up to humans. Hogan \& Hoel, \textit{supra} note 109, at 711-12. For example, one technique for establishing safe doses for humans is to find the maximum dose level that may be administered without causing an observed effect in animals and then multiply this by a safety factor of 100 to determine a "safe level" for humans. \textit{Id.;} Landau \& O'Riordan, \textit{supra} note 111, at 536. There appears to be no theoretical basis for choosing a factor of 100. Landau \& O'Riordan, \textit{supra} note 111, at 536 n.66.

\textsuperscript{113} \textit{See supra} note 57.

\textsuperscript{114} \textsc{Nisbet \& Karch}, \textit{supra} note 101, at 98 (citing 45 Fed. Reg. 69, 823). The FDA reports that known or suspected human teratogens produced positive teratologic responses 85% of the time in mice, 80% in rats, 60% in rabbits, 45% in hamsters, and only 30% in monkeys. \textit{Id.}

\textsuperscript{115} \textit{Id.} at 105.

\textsuperscript{116} \textit{Id.} (citing 45 Fed. Reg. 69, 823)

\textsuperscript{117} \textit{Id.}

\textsuperscript{118} \textit{Id.} For similar results, see \textsc{Edward J. Calabrese}, \textsc{Principles of Animal Extrapolation} 237-38 (1983); Gary P. Carlson, \textit{Factors Modifying Toxicity, in Toxic Substances and Human Risk}, \textit{supra} note 100, at 47, 49. These results are subject to an important qualification. It is impossible to be certain that any of the compounds that appear not to be teratogenic or carcinogenic in humans are, in fact, completely without teratogenic or carcinogenic effect. Leape, \textit{supra} note 110, at 92. A relatively rare human effect may not have
animal findings is, again, the problem of dosage. At sufficiently high
dose levels most, perhaps all, substances are teratogenic in some ani-

mals. If the question is whether a finding that a substance is terato-
genic in mice constitutes strong evidence it is teratogenic in humans, the
answer is no.

These limitations on our ability to extrapolate from animal research
to humans create what Weinberg has called “trans-scientific” uncer-
tainty: uncertainty concerning an issue that can be put in scientific terms
but for which scientific proof is unavailable. As Brennan notes, “we
accept that animal carcinogens are human carcinogens as a matter of
policy; this proposition cannot be proven given our present understand-
ing of carcinogenesis.” A similar statement could be made about in
vivo studies showing teratogenesis.

(3) Epidemiological Studies

A third type of scientific research involves epidemiological studies of
human subjects. Epidiological studies compare the incidence of
birth defects among those exposed and those not exposed to a drug.
There are two general ways of making such comparisons: cohort studies
and case-control studies. Cohort studies compare the incidence of de-

been detected in studies using relatively small sample sizes. Thus, even in a study of 1000
individuals, a negative finding means only that the excess incidence of adverse effects due to
the drug under examination is not more than 1%. NIBET & KARCH, supra note 101, at 92.

119. Bernard A. Schwetz, Monitoring Problems in Teratology, in SCIENTIFIC CONSIDERA-
TIONS, supra note 108, at 180. As one author notes: “Most teratologists accept the principle
that any agent can be shown to be teratogenic in an animal providing enough is given at the
right time. For instance, both sodium chloride and sucrose have been shown to produce
animal teratogenicity.” Thomas H. Shepard, Human Teratogenicity, 33 ADVANCES IN PEDI-
ATRICS 225, 227 (1986).

120. Nevertheless, rodents are particularly attractive candidates for in vivo testing, in part
because of their relatively low cost and ease of handling. Bernard L. Oser, The Rat as a Model


122. Troyen Brennan, Helping Courts With Toxic Torts: Some Proposals Regarding Alter-
native Methods for Presenting and Assessing Scientific Evidence in Common Law Courts, 51 U.

123. One commentator notes that there is often a period between the time when animal
studies indicate a substance may be dangerous and the time when epidemiological studies indi-
cate whether it is, in fact, dangerous to humans. Wendy E. Wagner, Note, Trans-Science in
Torts, 96 YALE L.J. 428, 432-34 (1986). She proposes that during the interim period liability
standards be revised so as to facilitate plaintiff recovery. Id. at 442-49.

124. Still another source of information about a drug’s effects are the previously men-
tioned DERs filed by physicians when they observe some adverse effect associated with the
drug. Though DERs are helpful in bringing potential problems to the attention of the manu-
facturer and regulators, their usefulness as a source of systematic data on the effects of a drug
is severely limited by their anecdotal nature.
fects in groups of persons exposed to the drug to the incidence in groups of persons not exposed.\textsuperscript{125} Case-control studies match a group of persons who have the defect in question ("cases") against another group that does not have it ("controls"). The two groups then are compared with regard to the frequency of exposure to the drug.\textsuperscript{126} Epidemiological studies typically measure risk by using the concepts of \textit{relative risk}, \textsuperscript{127} \textit{odds ratio}, \textsuperscript{128} and \textit{attributable risk}.\textsuperscript{129}

Epidemiological studies also vary in the way they collect their data—prospectively or retrospectively. All case-control studies are ret-

\begin{footnotesize}
\textsuperscript{125} Leon Gordis, \textit{Estimating Risk and Inferring Causality in Epidemiology, in Epidemiology and Health Risk Assessment}, supra note 53, at 51, 52.

\textsuperscript{126} \textit{Id.}; Michael Dore, \textit{A Commentary on the Use of Epidemiological Evidence in Demonstrating Cause-In-Fact}, 7 HARV. ENVTL. L. REV. 429, 432 & n.22 (1983).

\textsuperscript{127} The relative risk is the risk of defect in exposed individuals divided by the risk of defect in those not exposed. Gordis, supra note 125, at 52. For example, imagine that 20\% of mothers in a particular area ingested Bendectin during pregnancy. If we drew a sample and found that of 1000 children whose mothers were exposed to Bendectin, 60 have a defect, while of 4000 children whose mothers were not exposed to Bendectin, 160 have a defect, the relative risk associated with Bendectin ingestion would be \((a/(a + b)) / (c/(c + d)) = (60/(60 + 940)) / (160/(160 + 3840)) = .06/.04 = 1.5.\textsuperscript{128}

\textsuperscript{128} The odds ratio is the cross-product in a 2 \times 2 table. In a cohort study examining exposed and unexposed individuals, it equals the ratio of the odds of injury if the person was exposed, to the odds of injury if the person was not exposed. \textit{Id.}

\textit{Odds Ratio in a Cohort Study}

<table>
<thead>
<tr>
<th>Injured</th>
<th>Not Injured</th>
</tr>
</thead>
<tbody>
<tr>
<td>Exposed</td>
<td>a</td>
</tr>
<tr>
<td>Not Exposed</td>
<td>c</td>
</tr>
</tbody>
</table>

\textit{Id.} at 53 fig. 5-1. For example, using the data from the preceding note, the odds ratio would be \(ad/bc = (60 \times 3840)/(940 \times 160) = 1.53.\textsuperscript{129}

\textsuperscript{129} In a case-control study comparing a group of cases (people with the injury) to a group of controls (people without the injury), the odds ratio is the ratio of the odds that the cases were exposed to the odds that the controls were exposed. \textit{Id.} at 52.

\textit{Odds Ratio in a Case-Control Study}

<table>
<thead>
<tr>
<th>CASES</th>
<th>CONTROLS</th>
</tr>
</thead>
<tbody>
<tr>
<td>(Injury)</td>
<td>(No Injury)</td>
</tr>
<tr>
<td>History of Exposure</td>
<td>a</td>
</tr>
<tr>
<td>No History of Exposure</td>
<td>c</td>
</tr>
</tbody>
</table>

\textit{See id.} at 53 fig. 5-2. Note that cohort and case-control studies differ in how they are conducted, but not in how the odds ratio is computed.

With case-control studies we know the risks of exposure, but we usually do not know the risk of injury. The odds ratio is a good approximation of relative risk when, with respect to their history of exposure, both the cases and the controls are representative of the population from which they are drawn, and when the disease or defect is rare in the population. \textit{Id.} at 52, 54 fig. 5-3; see HAROLD KAHN, \textit{AN INTRODUCTION TO EPIDEMIOLOGIC METHODS} 46 (1983).

\textsuperscript{129} Attributable risk is an assessment of the differences in the risks of exposed and unexposed populations. It is calculated by subtracting the level of risk of the unexposed population (the background risk) from the level of risk of the exposed population. Gordis, supra note 125, at 53-54. Thus, if the background risk of contracting cancer is 25\% while the risk for those who spend a lifetime employed at a commercial nuclear reactor is 28\%, the attributable risk of working at the site is 3\%.
rospective; indeed, the term "retrospective study" is sometimes used as a synonym for case-control study. Similarly, the term "prospective study" is sometimes used as a synonym for cohort study, even though some cohort studies do collect data retrospectively. An important limitation of all studies using retrospectively collected data is that the data may be affected by recall bias. Mothers who have children with birth defects, for example, are likely to search their pasts carefully, trying to find the cause of their child's injury. Consequently, they are more likely to recall the drugs ingested during pregnancy than are mothers of healthy children. The dangers of bias presumably increase with the passage of time. Researchers employ a number of strategies in an attempt to overcome these dangers.

a. The Primacy of Epidemiology?

Because epidemiological studies are done on human subjects, they are sometimes thought to be the preferred evidence of environmental risk. As Erdreich notes, at least in situations where the proposed effect of a drug does not have a long latency period, "descriptive epidemiologic data . . . will settle the uncertainty associated with even the best animal data." Occasionally, epidemiological evidence has been said to trump all other types of information. Thus, Judge Weinstein, in his opinion granting summary judgment against opt-out plaintiffs in the Agent Orange Case, said the epidemiological studies "are the only useful studies having any bearing on causation. All the other data supplied by the parties rests on surmise and inapposite extrapolations from animal studies and industrial accidents."

130. KAHN, supra note 128, at 39-40.

131. Recall bias is but one of many sources of potential bias in epidemiological research. For a useful discussion, see David L. Sackett, Bias in Analytic Research, 32 J. CHRONIC DISEASES 51, 56 (1979).

132. In one study of Bendectin, for example, the researchers conducted an in-depth interview with each mother and in most cases corroborated the interview results with exposure information in the mother's obstetric record. Sally Zierler & Kenneth Rothman, Congenital Heart Disease in Relation to Maternal Use of Bendectin and Other Drugs in Early Pregnancy, 313 NEW ENG. J. MED. 347, 347-48 (1985).


134. Linda Erdreich, Combining Animal and Human Data: Resolving Conflicts, Summarizing the Evidence, in EPIDEMIOLOGY AND HEALTH RISK ASSESSMENT, supra note 53, at 197, 198.

On the other hand, epidemiological studies do have limitations. Not infrequently, they contain methodological or data collection shortcomings that undermine their findings. Thus, some experts would disagree with Judge Weinstein’s conclusion that he could disregard in vivo studies and other types of evidence that contradict epidemiological findings. Moreover, when epidemiological findings are inconsistent or unclear, the animal studies take on greater importance. Even if an epidemiological study indicates that there is a correlation between a product and a defect, the question remains whether the product caused the problem.

b. Causal Inference

Because we often may not understand the mechanisms by which a drug causes human injury, epidemiological correlation alone does not constitute proof of causation. Epidemiologists have developed a set of additional criteria to assist in determining whether an injury is the result of exposure to a particular product.

The first criterion is temporal order. Cause precedes effect. If the effect came before the cause, there can be no causal relationship. Ambiguity as to causal order undermines the ability to make causal arguments.

Second, there is the strength of association. The stronger the statistical association (usually measured by relative risk or the odds ratio) and the greater the level of statistical significance (usually measured by a chi square test) the more willing epidemiologists are to attribute a causal relationship.


138. Herein lies the complex question of when it is appropriate to reject the null hypothesis that there is no relationship between the treatment and the effect. For a general statistical discussion of calculating relative risks, chi squares, and confidence intervals see Kahn, supra note 128, at 3-10. The literature on the role statistical evidence and statistical significance should play in legal assessments of liability is voluminous. Among the more interesting articles are: Bert Black, A Unified Theory of Scientific Evidence, 50 Fordham L. Rev. 595 (1988); Black & Lilienfeld, supra note 136; Harold Ginzberg, Use and Misuse of Epidemiologic Data in the Courtroom: Defining the Limits of Inferential and Particularistic Evidence in Mass Tort Litigation, 12 Am. J. L. & Med. 423 (1986); Steve Gold, Causation in Toxic Torts: Burdens of Proof, Standards of Persuasion, and Statistical Evidence, 96 Yale L.J. 376 (1986); Ora Fred Harris, Jr., Toxic Tort Litigation and the Causation Element: Is There Any Hope of
The third criterion is the specificity of the relationship. There can be specificity in the causes and in the effects. Perfect specificity is achieved when a cause is a necessary and sufficient condition for one sole effect. If exposure to a certain product leads to a “signature disease,” as is the case with asbestosis, causal inferences are easier. But as causes come to have multiple results and as effects come to have multiple sources, causal arguments become indeterminate. Not infrequently, causal hypotheses have been attacked because a cause has multiple effects. Almost all epidemiologists agree, however, that while lack of specificity in the effects of a cause may make a causal relationship indeterminate, it does not disprove the existence of such a relationship. Similarly, while specificity in the causes of a given effect strengthens the plausibility of a causal relationship the absence of specificity alone does not refute the existence of a causal relationship.

Fourth, there is consistency on replication. Multiple replications, especially when they use a variety of methods and are done in a variety of circumstances, support causal arguments by diminishing the probability that results are spurious or the product of methodological flaws. Whenever it is impossible to implement an experimental design fully, unmeasured confounding factors threaten the accuracy of the results.

Finally, there is the coherence of the evidence. A finding is more likely to support a causal interpretation when it fits within a known body of facts or an existing theoretical framework. One type of coherence is biological plausibility; that is, an understanding of the mechanisms through which a cause may lead to an effect. Another type of coherence that increases the plausibility of a causal relationship is a finding of a dose-response relationship; that is the higher the dose, the greater the response.
The history of the Bendectin litigation has been marked by questions concerning the relative probative value of each type of evidence and the causal inferences that can be drawn from statistical correlations.

B. The Bendectin Studies

Against this background, the next sections present the Bendectin research. The purpose here is not to determine whether Bendectin is a teratogen; rather, it is to examine the flow of science over the life cycle of the Bendectin litigation. In mass tort cases, the importance of the science cannot be overemphasized. Without \textit{in vitro}, \textit{in vivo} and epidemiological findings, and experts prepared to present them, the plaintiff has no case. In order to understand the litigation, something first must be known about the science. Further, there are two other issues suggested by the idea of a congregation of cases. First, the science, like the law, of a case congregation develops and matures; the science also experiences a life cycle.\footnote{We should expect that the science will be relatively poorly developed in the early stages of litigation, but that over time the mobilization efforts of the parties will produce a richer body of scientific evidence.} We should expect that the science will be relatively poorly developed in the early stages of litigation, but that over time the mobilization efforts of the parties will produce a richer body of scientific evidence.

This leads to the second point: We should anticipate that the science itself is influenced by the legal process. As the congregation of cases grows and matures, it creates its own gravity field, attracting and distorting the science that comes near it.\footnote{In turn the science affects the law. Ultimately, science and law interact in complex ways to produce unique patterns of development in various case congregations. In the following three sections, I show how the science developed in the Bendectin Cases.} We should anticipate that the science itself is influenced by the legal process. As the congregation of cases grows and matures, it creates its own gravity field, attracting and distorting the science that comes near it.\footnote{We should expect that the science will be relatively poorly developed in the early stages of litigation, but that over time the mobilization efforts of the parties will produce a richer body of scientific evidence.}

(1) \textit{In Vitro Studies}

While probably all Bendectin cases have contained some testimony on \textit{in vitro} research, the number of studies on point is limited. In a recent letter published in the \textit{Journal of the American Medical Association}, Dr. does not necessarily detract from an analysis, since sometimes the cause may not operate in a linear fashion. \textit{Id.}

\footnote{144. See Michael Green, \textit{The Paradox of Statutes of Limitations in Toxic Substances Litigation}, 76 CAL. L. REV. 965, 975 (1988) (arguing that knowledge of the dangerousness of toxic substances generally improves with time).}

\footnote{145. In this regard, Sackett notes that research may be susceptible to what he calls the "hot stuff bias": "When a topic is hot, neither investigators nor editors may be able to resist the temptation to publish additional results, no matter how preliminary or shaky. . . ." Sackett, supra note 131, at 61. For a period of time in the 1980s, Bendectin was hot stuff. \textit{See infra} text accompanying notes 198-99.}
Stuart Newman cites six \textit{in vitro} studies that examine Bendectin or its antihistamine component, two of which were published in 1989.\textsuperscript{146} Compared to \textit{in vivo} or epidemiological research, \textit{in vitro} studies are the least accessible to the lay person. Summarizing the findings is difficult. One study that is damaging with respect to Bendectin's safety is by Budroe, Shaddock, and Casciano, who conclude that Doxylamine may be a weak DNA-damaging agent.\textsuperscript{147} Hassell and Horigan report that Bendectin inhibits limb bud mesenchyme cell differentiation.\textsuperscript{148} Dr. Newman interprets the other studies as indicating doxylamine also curtails the formation of embryonic cartilage.\textsuperscript{149} The authors of these studies, however, apparently do not conclude that their research indicates that Bendectin or doxylamine is teratogenic.\textsuperscript{150} Cumulatively, the evidence suggests that Bendectin's antihistamine component may have some adverse effects on embryonic cell development, but there is relatively little research, and the findings are not clear cut.

This circumstance is not surprising since the purpose of most of these studies is not to prove or disprove that Bendectin causes specific types of developmental defects in animals or children. Instead, most appear to have a different purpose: to study a group of known teratogens alongside a group of known nonteratogens and determine whether the

\textsuperscript{146} Stewart A. Newman, \textit{Bendectin—Birth Defects Controversy}, 264 JAMA 569, 569 (1990) (letter to the editor). As the journal editor noted, Dr. Newman has provided expert advice and testimony (for plaintiffs) in Bendectin cases. For a listing of the \textit{in vitro} studies, see Appendix B.


\textsuperscript{148} John R. Hassell & Elizabeth A. Horigan, \textit{Chondrogenesis: A Model Developmental System for Measuring Teratogenic Potential of Compounds}, 2 \textit{TERATOGENESIS, CARCINOGENESIS, AND MUTAGENESIS} 325, 331 (1982). Using a unit of measure called the "teratogenic potential," they found Bendectin inhibited cell differentiation at a dose of .05 mg/ml. This dose was approximately 1.5 orders of magnitude smaller than the amount of caffeine necessary to produce a similar effect (2.3 mg/ml). On the other hand, the Bendectin dose was 3.5 orders of magnitude larger than the amount of vitamin A required to produce a similar effect (.000013 mg/ml). \textit{Id.} at 330. Hassell and Horigan do not express an opinion in the article as to whether Bendectin is dangerous to humans when taken at normal therapeutic doses.

\textsuperscript{149} Newman, \textit{supra} note 146, at 569.

\textsuperscript{150} See, e.g., M. Gunatathata et al., \textit{Development of a Mouse Embryo Limb Bud Cell Culture System for the Estimation of Chemical Teratogenic Potential}, 4 \textit{TERATOGENESIS, CARCINOGENESIS, AND MUTAGENESIS} 349, 362 (1984) ("responses observed for each of these chemicals . . . were statistically insignificant"); J.Y. Renault et al., \textit{Limb Bud Cell Culture for In Vitro Teratogen Screening: Validation of an Improved Assessment Method Using 51 Compounds}, 9 \textit{TERATOGENESIS, CARCINOGENESIS, AND MUTAGENESIS} 83, 88-89 (1989) ("[i]n contrast with . . . patterns of specific inhibitory activity [produced by two known teratogens], treatment with Doxylamine (succinate), a known nonteratogen, resulted in a concomitant inhibition of proliferation and differentiation . . . signifying nonspecific toxicity").
procedure used in the study is in fact able to differentiate the two groups of drugs. If a procedure is able to distinguish known teratogens from known nonteratogens then it will be a better predictor as to whether a new compound will in fact be teratogenic in humans. To return to the distinction developed earlier between studies used to regulate risk versus studies used to prove causation in a tort case, the primary goal of most of these in vitro studies appears to have been the development of techniques that will be most helpful in regulating risk.

Perhaps this is why, in comparison to the in vivo and epidemiological research discussed below, relatively little Bendectin-specific in vitro research has occurred. As a consequence, the in vitro data is second best evidence when used in the courtroom to demonstrate that Bendectin is a teratogen.

(2) In Vivo Studies

In the aftermath of the Thalidomide disaster, the first in vivo tests of Bendectin were conducted by Merrell employees in 1963, using rabbits and rats. The principal investigator, Dr. Robert Staples, concluded that the study did not reveal teratogenicity. However, he noted some malformations in rabbits given the highest doses, and therefore recommended further research be conducted at higher dose rates. In 1966 and 1967 further tests were done on the individual components of Bendectin by two other Merrell employees, Drs. James Newberne and John Gibson. These results were published in 1968. Since that time there have been surprisingly few published in vivo studies designed to explore the teratogenic effect of Bendectin or its constituent ingredients. Table 1

151. See supra notes 112-20 and accompanying text.
152. Hagan v. Richardson-Merrell, Inc., 697 F. Supp. 334, 338 (N.D. Ill. 1988). Merrell did not immediately follow up on this recommendation, nor did it submit the findings to the FDA until three years later. This type of behavior on Merrell's part helped fuel speculation by plaintiffs that if they only dug deeply enough, they would find a cover-up. Id.
154. Table 1 is found in appendix A. For a listing of the in vivo studies, see Appendix C.
presents a summary of the published studies, plus two unpublished ones, in chronological order.

The table is useful for indicating the development of the literature. The first thing to note is the erratic nature of the research—bursts of activity separated by several years during which no research occurred. In fact, the pattern reflects the regulatory purpose of most in vivo studies and, later, the demands of the Bendectin litigation. The early 1967 research represents Merrell's response to Thalidomide. It was followed by a long period of quiet. The mid-1970s work reflects the impact of the epidemiological effectiveness study that found two ingredient Bendectin to be as effective as the three ingredient version. Subsequently, Merrell conducted in vivo studies to test the safety of the new formulation on animals. The final and largest wave of studies, from the early 1980s to date, reflects the need for new evidence regarding the drug's safety, driven largely by the interest in and demand for information resulting from the Bendectin litigation.

The table is somewhat misleading as an indicator of the total volume of animal research. Gibson's 1968 study reported on six separate experiments that had examined the effects of doxylamine succinate, dicyclomine hydrochloride, and three ingredient Bendectin on rats and rabbits. Because these all were done in the same laboratory by Merrell employees, however, they are inherently suspect.

Studies 3 and 4 were also done in Merrell's laboratories. They were designed to examine the

155. With regard to the number of mothers in experimental and control groups (column 3), it should be noted that the total number of fetuses examined may be much larger depending on the species. For example, in the rat study by Tyl et al., the 116 experimental animals and 53 controls produced a total of 2007 fetuses. Rochelle W. Tyl et al., Developmental Toxicity Evaluation of Bendectin in CD Rats, 37 TERATOLOGY 539, 546 (1988). Recall from the earlier discussion that the preferred analysis focuses on the percentage of fetuses within litters that have a defect. See supra text accompanying notes 103-104. The relatively greater cost of rabbit and monkey studies is in part a function of the smaller litter sizes of these species.

156. Several additional unpublished studies should be mentioned as well. Merrell conducted a number of studies, most, but not all of which are summarized in Gibson's 1968 article. See Gibson, supra note 153, at 440. In addition, there are studies by Roll & Matthiaschk (1981), Roll (1982) (testing Bendectin on rats), and McClure (1982) (a small monkey study showing no effect). Tyl et al. supra note 155, at 540. Table 1 does not include these studies.

157. See supra note 77 and accompanying text.

158. In all these experiments the authors interpreted the results as indicating no effect, although there does appear to be both a higher incidence of contracted tendons in rabbits receiving high doses of Bendectin and a higher incidence of focal hematomas in rats receiving high doses of dicyclomine hydrochloride. Additionally, no statistical analyses are presented for any of this study's findings. Plaintiff's experts have challenged the way those who conducted the study coded certain animals and have claimed that if the data had been coded correctly, these studies would indicate that Bendectin causes birth defects.
teratogenic effects of the two ingredient Bendectin formulation. In addition, studies 6, 7, 9, and 10 were all conducted in a single laboratory at the University of California, Davis by Hendrickx and colleagues. Papers 6 and 7 are, in fact, only abstracts of work in progress that is fully reported in 9 and 10. If Hendrickx's studies are counted as one piece of research, there turn out to be only six separate research projects in the published literature, and one of these (study 5, involving chicken eggs) is only marginally an in vivo study.

Several of the studies report some relationship between the drug and a teratogenic effect, although the authors are generally cautious about attributing causation. The research on monkeys concludes that two ingredient Bendectin causes heart defects. One rabbit study (study 8) concludes that doxylamine succinate causes limb and skeletal defects. The rat studies find, at most, minor teratogenic effects. Several studies

159. See infra app. A, tbl.1, col. 5.
160. See infra app. A, tbl. 1, col. 9. Their caution is explained by a variety of factors: the relationship is not statistically significant; the defect is one frequently found in the particular strain of animal used; the effect is produced because of the drug's maternal toxicity; or there are other, fortuitous explanations for the result.
161. The first epidemiological study to show a teratogenic effect in humans also involved heart defects. See infra note 183. The defects exhibited by the monkeys (ventricular septal defects) apparently are temporary. Bendectin causes a delay in the closure of the ventricular septa, but it closes spontaneously by full term. The defect is not detectable in newborn monkeys. A.G. Hendrickx et al., Evaluation of Bendectin Embryotoxicity in Non-Human Primates: Double-Blind Study in Term Cynomolgus Monkeys. 32 TERA TOLOGY 191, 194 (1985).
162. Because the most seriously injured plaintiffs have suffered limb defects, the rabbit studies are particularly relevant to the Bendectin litigation. Dr. McBride, the researcher who found these defects, has been a central expert witness for plaintiffs in these cases. He has also been at the center of considerable controversy, including serious charges of misconduct surrounding research on a different drug, scopalamine. See Andrew Skolnick, Key Witness Against Morning Sickness Drug Faces Scientific Fraud Charges, 26 JAMA 1468, 1468 (1990).
163. While the Tyl study did show a dose-related skeletal defect (reduced number of ossified caudal vertebral centra), on the basis of an analysis of covariance ("ANCOVA"), the authors concluded that this and other fetal defects are best understood as the consequence of reduced fetal body weight. Tyl, supra note 154, at 550-51. In general, the authors interpret the defects detected in this study as the byproduct of maternal and developmental toxicity resulting from the very high doses of Bendectin given to the experimental rats. The lowest dose rate in the study (200 mg/kg/day) is approximately 300 to 600 times the therapeutic dose taken by pregnant women. The highest dose rate was 800mg/kg/day. At this rate, Bendectin caused profound maternal toxicity.
Tyl also reports that Roll and Matthiaschk conducted an experiment in 1981 finding teratogenic effects (diaphragmatic hernia) in rats; but that Roll was unable to replicate the results in the same laboratory. Id. at 540.
Gibson's 1975 study indicates a relationship between Bendectin and dilated kidney pelvis and subcutaneous hematomas. Both are said to be common to the strain of rat used in the study, and the latter defects generally disappear within a few days in newborn rats. John Gibson, Teratology Study With A New Antinauseant Formulation in Rabbits 3 (1975) (unpublished study, on file with author).
find that high dose rates of Bendectin do produce maternal toxicity, and this in turn injures the fetus.\footnote{164}{See Tyl et al., \textit{supra} note 155, at 550.}

One would expect that as the Bendectin litigation matured, so too should the quality of the \textit{in vivo} evidence have improved, allowing the parties and the courts to reach a consensus regarding the drug's teratogenicity. And there is some evidence that the quality of the research did improve over time. Early studies rarely utilized any statistical analysis of experimental results, while later ones employed some rather sophisticated analytic tools.\footnote{165}{See id.} Additionally, the sample sizes of the studies have grown, and dose rates have been increased.\footnote{166}{Tyl's 1988 rat study involved over 2000 fetuses, some of whose mothers had been exposed to Bendectin at levels nearly three orders of magnitude greater than human therapeutic doses.} Both of these methodological changes increase a study's ability to detect minor defects. On the other hand, the limited number of \textit{in vivo} studies, which employed a variety of test animals and tested a variety of component drugs, makes it difficult to draw any conclusions as to whether Bendectin has a specific teratogenic effect on humans.

How to account for the relatively limited number of \textit{in vivo} studies, as well as their relatively late appearance? The small number of studies cannot be explained by a direct depletion effect. While Bendectin is no longer available as a prescription drug, it remains available for laboratory studies. Indeed, the most recent research occurred subsequent to Merrell's withdrawal of Bendectin from the market. Nevertheless, the volume of published work is thin. Why?

In part, the answer is found in the relationship of this research to all Bendectin research and to the ongoing litigation surrounding the product. While \textit{in vivo} studies in general are cheaper to conduct than epidemiological studies, with respect to a particular drug the relative advantage is lessened. Large scale epidemiological studies can be used to investigate a wide variety of drugs simultaneously. Most of the epidemiological research on Bendectin, for instance, uses data sets collected for more general purposes. Animal studies, by contrast, must focus on the drug in question. Consequently, there has been less interest in mobilizing the resources necessary to conduct Bendectin-specific \textit{in vivo} experiments.

This has proven especially true since Bendectin was removed from the market. Further study of the drug is of limited interest to people who are not in some way involved with Bendectin as a legal, as well as sci-
tific, issue. In other words, Merrell's decision to remove Bendectin from the market did lessen demand for animal research. This fact is reflected in the sponsorship of the published studies that do exist. Five separate research projects are listed in Table 1 (Gibson, Khera, McBride, Hendrickx, and Tyl). The first (Gibson) was sponsored and conducted by Merrell. The second (Khera) was done to explore the teratogenicity of pyridoxine; it did not directly involve Bendectin. The third (McBride) was independent. Hendrickx's work began independently, but the research reported in the second 1985 article was funded by Merrell.167 Tyl's study, the largest and most sophisticated in vivo study to date, was undertaken by the National Toxicology Program at the specific request of the FDA's Bureau of Drugs.168 Given the fact that Merrell was, by far, more interested in such research than any other entity, its decision to drop Bendectin naturally led to a decrease in animal studies of the drug's teratogenicity.

If those not involved in the litigation had little interest in new research, neither did the parties themselves have strong incentives to conduct new studies. Once litigation began, Merrell's incentive to continue its own research was substantially diminished. New studies done by Merrell scientists in Merrell laboratories were, from the company's point of view, a lose-lose proposition. If they showed an effect, the studies would be used against the company in subsequent litigation. If they failed to show an effect, their persuasiveness would be seriously limited by the fact that Merrell had conducted them. Any slight technical flaw in the design or execution of the experiment would be exploited by plaintiffs to undermine Merrell's findings.169 A similar analysis would apply to research funded by Merrell, especially if it was conducted by someone who had previously testified on the firm's behalf.170

167. Hendrickx, supra note 161, at 194.
168. Tyl, supra note 155, at 540.
169. Typically, this has happened with earlier Merrell animal studies. See In Re Bendectin Litig., 857 F.2d 290, 318 (6th Cir. 1988), cert. denied sub nom. Hoffman v. Merrell Dow Pharmaceuticals, 488 U.S. 1006 (1989). In more recent trials, Merrell has relied much less on its own animal or epidemiological studies, in part because it then does not have to defend them. For example, in Hagaman v. Merrell Dow Pharmaceuticals, No. 84-2202-5, 1987 U.S. Dist. LEXIS 6124 (D. Kan. Jun. 26, 1987), Merrell moved to exclude the videotape deposition of Isalene Smock, whose testimony cast doubt on an early Merrell epidemiological study. The court granted the motion, stating: "In their brief, plaintiffs indicate that this deposition will be offered because defendant relies heavily upon the Bunde-Bowles study. Defendant has indicated that it does not intend to rely upon the study at trial. Accordingly, the court cannot find any relevance in showing the videotape to the jury." Id. at *28.
170. The most noteworthy example of this problem in the Bendectin Cases arose with respect to the epidemiological research done by Professor Richard Smithells for Merrell. Plaintiffs were able to obtain a letter from Professor Smithells to Dr. Mark Hoekenga, an
In this respect it is interesting to note that while the Hendrickx study, which was funded by Merrell, did involve some risk of an adverse finding, from the firm's perspective it had the benefit that it was conducted by a non-Merrell employee who had previously concluded Bendectin causes heart defects in monkeys. Merrell thus could hope to make effective use of a negative finding in court. Even so, the researchers used a double-blind design so as to minimize the potential that the study might appear biased.\(^{171}\) Even this strategy, however, did not shield Merrell from the suggestion that because they funded the study its results were suspect.\(^{172}\)

employee of Merrell, in which Smithells reports that he is attempting to publish findings favorable to Merrell and asks Merrell to support his research with sums up to £15,000. He concludes his request by stating:

> Much clearly depends on the value of publication to Merrell-National Labs. If it may save the company large sums in Californian Court [sic] (which is rather what I thought when we undertook the study) [Merrell] may feel magnanimous. If with passage of time the study is of no great significance I can only regard the figure you suggest as generous and welcome.


\(^{171}\) Hendrickx, supra note 161, at 191.

\(^{172}\) The first Hendrickx study had found that when fetuses from three species were sacrificed after approximately two-thirds of their gestation period, those whose mothers had taken Bendectin were more likely than those whose mothers had not to exhibit interventricular septal defect (the membrane separating the two sides of the heart, the septum, is not completely closed). The second study, funded by Merrell, replicated the first study on one species but did not sacrifice the fetuses until normal term. By then there was no apparent effect of Bendectin exposure; apparently the membrane spontaneously closes by term. \textit{Id.} at 194.

The following exchange took place between plaintiff's lawyer and plaintiff's expert, Adrian Gross, with respect to the Hendrickx studies:

Q: All right. Let's talk about who pays for some of these things, then, Doctor. Let's look at the Hendrickx study.
A: The first one was done under the sponsorship of the government with a grant from the NIH.
Q: And the second one?
A: That was paid for by Merrell.
Q: Let me show you what's been marked as Exhibit 231. Have you seen this before?
A: I think I have.
Q: Can you tell us what—whose letterhead it's on?
A: Merrell Research Center.
Q: Turn to the second page of that.
A: Yes, I have it.
Q: Is there a reference there to the Hendrickx study?
A: Yes.
Q: Hendrickx monkey study?
A: Right.
Q: The category is what?
A: Defense.
It is not only the existence of litigation that alters the incentives to conduct in vivo research; so too does the existence of a large body of epidemiological data diminish the market for animal research. Inevitably, the direct evidence on human effects provided by the epidemiological evidence diminishes the demand for in vivo studies insofar as they are designed to answer the question of whether a drug causes harm to humans. Although there is considerable disagreement about the proper role of in vivo studies in answering that question, many would agree they are less probative than epidemiological evidence. As the following discussion shows, the Bendectin litigation and epidemiology eventually interacted in a way that seriously undermined the worth of animal studies. Increasingly, courts simply refused to rely on or even to admit in vivo evidence. Considering the declining demand for in vivo research, it is not surprising that the supply has been limited.

(3) Epidemiological Studies

The epidemiological evidence stands at the center of the Bendectin cases. As noted earlier, a number of judges and scientists have argued that a coherent body of epidemiological evidence trumps other information.173

a. Background

I have found and examined thirty-nine published epidemiological studies that discuss Bendectin.174 Table 2175 summarizes these studies.176

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Q: Defense. How much in thousands—
A: How much money?
Q: Is it listed there? Would you read that?
A: 200 and—excuse me. Let me get my eye helper here. 248.5.
Q: In thousands?
A: Yes.
Q: That's just phase one, though, isn't it, Doctor?
A: I'm sorry.
Q: That's just phase one of the study, isn't it?
A: Yes. Phase two was—
Q: What about phase two?
A: $60,000.
Q: So over $300,000 for defense?
A: Yes, that's true.

173. See supra notes 133-36 and accompanying text.
174. These studies were found by reading Bendectin trial transcripts and exhibits, searching for articles on Bendectin in MEDLINE, a CD/ROM index to medical publications and periodicals, and collecting the references cited in each discovered article.
175. Table 2 is found in appendix A.
In this section, I briefly discuss the epidemiological findings on Bendectin and then examine how the research changed over time.

What do these studies show concerning the teratogenic effects of Bendectin? Needless to say, much of the litigation in this and other drug cases centers on varying interpretations of the epidemiological findings. However, as with the in vivo studies, two summary facts are worth reporting: (1) the authors’ conclusions about the drug; and (2) whether the study contains any objective indication that Bendectin has adverse effects.

In no study did the authors clearly conclude that Bendectin does have teratogenic effects. In six studies, the authors report at least one significant correlation between Bendectin and some adverse effect and conclude that while their findings alone are insufficient to support an attribution of causation, one may exist. In the remaining thirty-three studies, the authors either draw no conclusion or conclude that there is no statistical relationship.

Apart from the author’s own conclusions, a second measure of Bendectin's effect is the odds ratio or the relative risk reported in the study. For each study, I have attempted to extract the “most important” odds ratio or relative risk. Either by using the statistic reported

176. Three known studies have been excluded from Table 2. One, M.M. Adams & J. Mulinare, A Case-Control Study of Bendectin and Ventricular Special Defects (Abstract), 31 TERATOLOGY 61A (1985), is a brief abstract, and lacks sufficient information to be coded. The authors report that they found three ingredient Bendectin to be correlated with a nonsignificant increased incidence of birth defects, but that the two ingredient version was significantly related to a decrease in defects. No explanation is offered for this seemingly contradictory finding. Another study, Robert L. Brent, Bendectin and Interventricular Septal Defects, 32 TERATOLOGY 317 (1985), does report some data comparing Bendectin sales over time with the number of reported ventricular septal defects. However, this publication is primarily an editorial discussing Hendrickx's monkey studies. A third study, Leslie L. Robinson et al., Maternal Drug Use and Risk of Childhood Nonlymphoblastic Leukemia Among Offspring, 63 CANCER 1904 (1989), is excluded because it does not concern birth defects. It does report a relative risk of 1.75 associated with the use of Bendectin or other morning sickness medication (P = 0.06), and also indicates a significant elevation in risk associated with increased duration of usage. Id. at 1906.

177. In a later paper, I will discuss the ways in which attorneys and experts for both Merrell and plaintiffs interpreted the data.

178. The six studies are numbered 16, 23, 25, 26, 29, and 33 in Table 2. (A list relating the number to a particular database appears in Appendix E.) Eskenazi and Bracken come closest to saying Bendectin is a teratogen. They conclude that “these data suggest that the material use of Bendectin is strongly associated with the occurrence of pyloric stenosis in the infant. Whether this is a direct causal relationship is unclear.” Brenda Eskenazi & Michael Bracken, Bendectin (Debendox) as a Risk Factor for Pyloric Stenosis, 144 AM. J. OBSTETRICS & GYNECOLOGY 919, 924 (1982).

179. For a description of these two measurements, see supra notes 127-28.

180. “Most important” is a slippery term. Where the study reports a significant correla-
or by calculating an odds ratio based on the raw data published, it was possible to extract one or both measures in twenty-six of the thirty-nine studies. In general, odds ratios and relative risks are not strictly comparable—either with each other or across studies. But one crude type of comparison that can be made is whether the statistic is greater or less than 1. An odds ratio or relative risk greater than one indicates that children whose mothers took Bendectin are more likely to have suffered a defect than those whose mothers did not. An odds ratio or relative risk of less than one indicates the opposite. If Bendectin truly has no effect, given a large number of equally valid studies, half should produce statistics greater than one and half should result in measures of less than one. With regard to the twenty-six studies for which I was able to extract a value, in thirteen the most important measure is greater than one and in twelve it is less than one. One study reports a relative risk of exactly one.

b. The Evolution of Bendectin's Epidemiological Research

While the aggregate findings of the studies are critical to the ultimate disposition of the Bendectin Cases as a group, the key point for this analysis is that the studies, like the case law, evolved over time. In the early years, the epidemiological research tended to parallel the in vivo research. A number of investigations were conducted in the early 1960s, partly in response to the Thalidomide disaster. Then the epidemiological research virtually ceased for a number of years. In the late 1970s, a
number of new studies were published, although it is important to note that most of them did not target Bendectin specifically. Rather, they reported the results of broad epidemiological studies of drugs taken during pregnancy, including Bendectin and its constituent ingredients.

A turning point in the epidemiological research occurred in 1979 with the publication of the first article suggesting an association of Bendectin with birth defects. The study was important for several reasons: It was published in one of the leading American journals on epidemiology; the analysis was relatively sophisticated; and the senior author, Kenneth Rothman, was a professor in the Department of Epidemiology in the Harvard School of Public Health. With the publication of the Rothman study, Bendectin, like Thalidomide and MER/29 before it, became epidemiologically suspect.

To demonstrate the importance of the Rothman study, I have coded the thirty-nine studies as either pre- or post-1979; the results are contained in Table 3. As the table indicates, prior to 1979 most of the studies focused on teratogenicity in general, not Bendectin in particular. The Rothman finding, plus the emerging litigation concerning Bendectin, generated study after study of this particular drug. With this increased focus came an increase in the sophistication and the power of the studies.

c. The Increase in Statistical Sophistication and Power

Table 4 indicates that studies rarely reported more than a basic Chi-square statistic prior to 1980, but uniformly began to report more after that date. Most importantly, they began to report confidence intervals for estimates and started to control for other factors through the use of multiple regression techniques, most frequently logistic regression.

As time passed, the power of individual studies also increased. The power of a study is its ability to detect a difference of some given magnitude.

Epidemiological studies, like all statistical analyses, are vulnerable to two types of errors. Type I errors occur if we reject the null hypothesis when in fact it is true. In terms of this discussion, we would conclude Bendectin causes birth defects when in fact it does not. Scientists, being conservative, generally try to minimize Type I errors; that is, they de-

183. See Kenneth Rothman et al., Exogenous Hormones and Other Drug Exposures of Children with Congenital Heart Disease, 109 AM. J. EPIDEMIOLOGY 433, 435 (1979).
184. Table 3 is found in appendix A.
185. Table 4 is found in appendix A.
cline to find a causal relationship unless it is unlikely that the observed results occurred by chance.

Traditionally and typically, the null hypothesis will not be rejected unless the probability that a result occurred by chance is less than one in twenty, or sometimes less than one in one-hundred. The Greek letter Alpha is by custom used to designate this criterion. In other words, if the probability of a result occurring by chance must be less than one in twenty, Alpha must be less than .05.

Type II errors occur if we fail to reject the null hypothesis when in fact it is false. In terms of this discussion, we would conclude that Bendectin does not have a teratogenic effect when in fact it does. Science has traditionally been less concerned with this type of error. Nevertheless, we would like to minimize the probability of this error (by custom designated as Beta) as well. From the plaintiff's point of view, reducing Type II errors is at least as important as reducing Type I errors. The probability that we will reject the null hypothesis when some specific alternative hypothesis is true is called the power of the test, and is equal to 1 minus Beta.

Given some level of Alpha, Beta is a function of several factors, one of the most important being the sample size of treatment and control groups. Ceteris paribus, the larger the sample the lower the Beta value. It is thus worth investigating whether sample sizes increased substantially before or after the Rothman study. However, in examining relative sample size it is necessary to distinguish between cohort and case-control studies. Because the power of a given test is contingent upon the sample size of both the treatment and control groups, case-control studies can achieve low Betas with much smaller samples than can cohort studies. This is the case because birth defects are relatively rare in the population. Thus, cohort studies exploring rare events require a large number of total cases in order to obtain a significant number of individuals who have experienced an injury. Case-control studies begin with a large number of injuries, and thus need far fewer cases.

For example, assume that some birth defect occurs in 1 out of 1000 cases among children who were not exposed to Bendectin, and that 50% of all prospective mothers take Bendectin during their pregnancy. If we set Alpha at .05, in order to be able to detect a relative risk of 2.0 among the children of Bendectin takers 90% of the time (Beta = .10) we would need an N of 177 in a case-control study, but we would need an N of 31,444 in a cohort study. If we wished to detect a relative risk of 1.2 90% of the time, the case-control N would have to be 2535, while the
Cohort study N would be an enormous 576,732 cases. Consequently, in examining sample sizes before and after 1979, it is important to distinguish between cohort and case-control studies. Within the Bendectin epidemiological research, was there an increase in Ns in either type of study or a shift from cohort to case-control studies?

The answer is that, while sample sizes did not change, the types of studies did. There has been a trend toward relatively more case-control studies after 1979 (38% before, 48% after). Moreover, the nature of the case-control studies has changed substantially. I have coded each case-control study for the nature of the cases, that is the type of defect they represented. Early studies adopted a shotgun approach, in which the cases were any type of birth defect. Three of the five case-control studies conducted before 1979 were of this type. After 1979, however, the case-control studies tended to test for specific types of birth defects. Of the ten post-Rothman case-control studies, seven tested whether Bendectin caused one or more specific defects. Two looked for congenital heart defects—the type of defect correlated with Bendectin in the Rothman study. One looked for pyloric stenosis. Four studies examined the

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187. On the other hand, if a phenomenon occurs frequently in the population, the relative advantage of case-control studies is essentially eliminated. If a defect occurs in one out of ten cases in the population, using the same assumptions as in the text, we would still need an N of 177 to detect a two-fold increase in relative risk 90% of the time in a case-control study. On the other hand, to have the same probability of detecting this effect in a cohort study we would need an N of only 266, not 31,433. KAHN, supra note 128, at 54.


The most recent case control study, David J. Erickson, Risk Factors for Birth Defects: Data From the Atlanta Birth Defects Case-Control Study, 43 TERATOLOGY 41, 43-44 (1991), does not report data for specific defects and, therefore, was not counted among the seven that report on specific defects. It should be noted, however, that the author of the study offers to send cross-tabulations reporting the relationship of Bendectin to 92 types of birth defects to anyone who requests the information. Id. at 41.

189. See Mitchell et al., Bendectin I, supra note 188; Zierler & Rothman, supra note 188.

190. This study, by Mitchell et al., failed to replicate the results of the study by Eskenazi et al., in which the authors found a significant correlation between Bendectin and pyloric stenosis. See Mitchell et al., Bendectin II, supra note 188, at 741 (comparing the two studies).
effect of Bendectin use on oral clefts, and two looked for limb defects.\textsuperscript{191} This trend toward focused case-control studies makes sense. Known teratogens tend to produce certain types of defects.\textsuperscript{193} Thus, if Bendectin were a teratogen, it would be unlikely to produce a similar increase in all types of defects. Moreover, these focused studies also allowed for relatively powerful analyses. With case sample sizes ranging from the 90s to over 300\textsuperscript{194} they were unlikely to miss an effect if exposure created a relative risk of 2 or greater.\textsuperscript{195}

In sum, from the mid-1970s to the mid-1980s the quantity and quality of the epidemiological evidence on Bendectin improved dramatically. The research focused on the drug and used increasingly sophisticated methods to control for confounding factors. Specifically, it used a combination of increased sample size, a shift to case-control studies, and a focus on particular types of defects to greatly increase the power of the research. During this period, the probability of making either Type I or Type II errors was significantly reduced.

d. The End of Research

Suddenly, in 1985 with nearly three dozen studies in print, epidemiological research on Bendectin came to a virtual halt.\textsuperscript{196} As noted earlier, \textit{in vivo} research experienced a similar, contemporaneous decline.\textsuperscript{197} Figure 1 shows the number of \textit{in vivo} and epidemiological studies published each year from the early 1960s to the present.

\textsuperscript{191} See Cordero et al., supra note 188; Elbourne et al., supra note 188; Golding & Baldwin, supra note 188; Mitchell et al., \textit{Bendectin I}, supra note 188.

\textsuperscript{192} See Cordero et al., supra note 188; McCredie et al., supra note 188.


\textsuperscript{194} The number of cases—as distinguished from the number of controls—ranged from a low of 93 in Elbourne et al., supra note 188, at 780, to a high of 325 in Mitchell et al., \textit{Bendectin I}, supra note 188, at 737. The number of controls was usually larger than the number of cases, sometimes substantially so. For example, there were over 3000 controls in the first study by Mitchell et al. \textit{Id}.

\textsuperscript{195} The advantage case-control studies have in detecting rare effects is offset, however, by the fact that these studies must match cases and controls based on some criteria. Inevitably there is a question whether the matching failed to control for some confounding factor that explains observed differences between the groups.

\textsuperscript{196} As far as I know, there is only one post-1985 epidemiologic study (Erickson) specifically focused on Bendectin. That study includes Bendectin among 105 exposure-risk factor variables that have been thought to cause birth defects. Aggregating across all defects, the odds ratio associated with Bendectin is 0.87. Erickson, \textit{supra} note 188, at 46.

\textsuperscript{197} \textit{See infra} app. A, tbl. 1.
The figure illustrates two phenomena. First, and most important, it clearly shows that the science was driven by the law. The study of Bendectin became a hot topic and substantial resources were mobilized to study it. This mobilization can be understood at several levels. Because Bendectin was a hot topic, articles on the subject were relatively likely to find their way into print. Careerist concerns may have caused academics to select topics that would lead to publication in prestigious journals. Moreover, the federal government, through the FDA, encouraged research by offering grants to fund the study of Bendectin's effects. Finally, the Bendectin litigation itself generated research, as parties encouraged and even funded work on Bendectin. Legal needs gave shape and direction to the epidemiological study of teratogenic effects. The volume and sophistication of studies focusing specifically on Bendectin was, in large part, the result of the litigation.

198. See Sackett, supra note 131, at 51.
Second, Figure 1 demonstrates the relatively sudden cessation of studies in the mid-1980s. Galanter's hypotheses about holistic effects can be extended to explain the decline of scientific investigations as well as case congregations. Because Bendectin was removed from the market in 1983, and because its sales had plummeted in the preceding year or two, there eventually were no new data bases to exploit. By withdrawing the product from the market Merrell created an epidemiological depletion effect as well as the intended case depletion effect.

It would be a mistake, however, to attribute the decline of Bendectin studies entirely to depletion effects. Two other factors were also at work. The scientific community seems to have reached something close to a consensus concerning the drug. While no study can remove all residual uncertainty regarding Bendectin's safety, if the drug is a teratogen, it is a relatively mild one (having effects too subtle to be measured reliably with existing techniques). As a result, many felt that Bendectin had been overstudied. There arose the desire to ration Bendectin studies and save limited resources to study other drugs. As Lewis Holmes notes in an influential essay:

While we can always wish for more and better studies, two issues must be borne in mind. First, well-designed and extensive epidemiologic studies are expensive. Where will the funds to support these studies come from in this period of limited funding? Second, in view of the extensive data cited above on Bendectin and the limited data available on many other commonly used drugs one can argue that well-designed studies of other drugs would be of greater value to the public at this time.200

(4) The Life Cycle of the Bendectin Science

The preceding analysis of the research regarding Bendectin demonstrates that the science has followed a life cycle of its own. There was a substantial mobilization of resources devoted to the study of Bendectin, much of it apparently in response to the litigation and concomitant political pressure. The quantity and quality of epidemiological research increased dramatically in the 1980s, to the point where people like Holmes concluded that we knew more about the teratogenic potential of this drug

200. Lewis B. Holmes, Teratogen Update: Bendectin, 27 TERATOLOGY 277, 280-81 (1983). Holmes, however, cannot be called a completely disinterested observer. In January 1983 Merrell Research Center provided Dr. Holmes with a grant of $147,318 to fund proposed research on "The Use of Major and Minor Malformations to Evaluate the Putative Teratogenic Effects of Bendectin." Letter from J.W. Newberne, Vice President, Drug Safety Assessment at Merrell Research Center, to Ronald W. Lamont-Havers, Deputy to the General Director for Research Policy and Administration, Massachusetts General Hospital (Jan. 27, 1983) (on file with the Houston Environmental Law Liability Program).
than nearly any other. The extent of the epidemiological literature stands in contrast to the paucity of in vivo studies. In part, this may be understood as a consequence of the fact that there were few if any basic science questions to be answered through the study of Bendectin, and as a legal resource the in vivo studies were of increasingly little value in the face of a mounting body of epidemiological evidence. In a sense, epidemiology drove out animal studies.

Finally, in the mid-1980s came the end of new epidemiological studies as well.201 The combination of depletion effects and an emerging scientific consensus that if Bendectin has any teratogenic effects they are virtually undetectable by existing methods together led to the cessation of research. The leading indicator, published scientific articles, had stabilized at an essentially prodefendant conclusion. But while the science was winding down, the law was just getting into full swing. It is to the development of the law in this congregation of cases that this Article now turns.

V. The Law

In this Part, I focus upon the legal aspects of the Bendectin congregation. The first section examines the early stages of the life cycle, describing plaintiffs' efforts to mobilize resources and the problems the "first plaintiff" faces in a mass tort case. It also examines the defendant's efforts to avoid early adverse precedents that would facilitate other plaintiffs' efforts in future cases. Finally, it notes the importance of the battle in the early cases to establish the "going rate" for injuries.

The second section examines the flow of Bendectin filings and trials over time. As we shall see, the number of filings grew through 1985, but then collapsed in the aftermath of a defense victory in the Common Issues Trial in Ohio.202 The number of trials, however, continued to grow over the next two years, but then quickly began to diminish, and today most litigation seems to be behind us. A depletion effect has in fact occurred. As Galanter notes, however, depletion is not simply a product of the underlying rate of injury. It is also a product of the legal experience of a congregation of cases in court.203

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201. Only one animal study and two epidemiological studies were published after 1985. See infra app. A, tbs. 1, 2.
203. Galanter, Case Congregations, supra note 8, at 388.
The third section examines this experience. It turns away from the behavior of the parties and concentrates on the behavior of the judiciary. Of primary interest is the judiciary's efforts to ration legal resources. In the Bendectin Cases, the judiciary's efforts have followed a two-part process: first, the use of traditional consolidation devices; and second, the willingness to make and act upon independent determinations regarding the scientific evidence. As we shall see, in attempting to ration resources, the courts are confronted with a dilemma: how are they to maximize judicial efficiency without unduly subverting the interest of individual plaintiffs in a full and fair trial of their lawsuits? Before addressing this thorny problem, I turn to the early stages of Bendectin's legal life cycle.

A. Mobilization

When parties begin litigation concerning a new product, they start without scientific or legal resources. The first task is to begin to mobilize these resources. This difficult task falls disproportionately on the "first plaintiff" who must connect an injury with a product, find a lawyer who will represent her and then amass sufficient scientific evidence to prove the product causes the injury in question.

(1) The "First Plaintiff" Problem

David Mekdeci, like many plaintiffs to follow, was born with a limb defect; his consists of malformed and missing fingers and a missing pectoral muscle. From the beginning, David and his parents faced many aspects of the first plaintiff problem. No lawyer came knocking on the Mekdeci's door asking to take their case. Instead, David's mother, Elizabeth Mekdeci, made extensive efforts to discover possible causes of his defects. After several years of talking to medical experts and reading documents and government studies, she became convinced that one of the drugs she had taken during pregnancy had caused David's injury. Ultimately, she contacted Melvin Belli, a famous San Francisco plaintiff's personal injury lawyer, and persuaded him to take the case. He also referred the Mekdecis to Florida counsel. As the case developed, based on the time of ingestion and other evidence, the Mekdecis and their lawyers focused most of their attention on Bendectin. A complaint naming Merrell as defendant was filed on behalf of David and his parents in

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204. This defect is commonly called Poland's Syndrome although plaintiffs' experts have disagreed with this diagnosis.

205. Mekdeci v. Merrell Nat'l Lab., 711 F.2d 1510, 1516 (11th Cir. 1983). Shortly before the trial began, Mr. Belli informed the other counsel that he would not personally appear, and left the local attorneys to try the lawsuit. Id. at 1516.

The trial lasted two months, and after three days of deliberation, the jury reported itself to be “hopelessly deadlocked.” After receiving further instructions from the court, it returned a verdict awarding the “plaintiff” $20,000, the amount the parties had stipulated to as the parent’s medical expenses. The jury wrote “nothing” as the amount of compensatory damages due David individually. The plaintiffs sought a new trial on the damages question only, but the trial judge declared the jury’s award to be a compromise verdict and ordered a new trial. The jury’s uncertainty, reflected in the apparent compromise verdict, also reflects another aspect of the first plaintiff problem: the difficulty of amassing sufficient scientific evidence to prove the case.

Compared to plaintiffs, defendants generally have a substantial early advantage. Their financial resources are much greater. They can hire counsel who are experienced in the defense of defective pharmaceuticals against personal injury claims and who are well connected to governmental agencies. They have ready access to nearly all the safety data concerning their product. All these advantages inured to Merrell’s benefit in the *Mekdeci* case. The company committed unlimited resources to Bendectin’s defense, hiring Lawrence E. Walsh and the firm of Davis Polk & Wardwell as defense counsel. Moreover, after its experiences with Thalidomide and MER/29, Merrell probably had as much experience as any firm in defending prescription drugs in court.

Notwithstanding these advantages, defendants may be confronted with some first case problems similar to those of the first plaintiff. The defendant, too, must confront the relative lack of evidence and the considerable uncertainty that permeates such early litigation. The defendant’s legal staff most likely is not yet organized to mount a defense of the particular product. And if the firm is insured, the case might be turned over to the insurer’s counsel who may lack the familiarity with pharma-

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206. *Id.* at 1515.
207. *Id.* at 1514.
208. The Eleventh Circuit Court of Appeals held that the trial court had not abused its discretion in ordering a new trial. *Id.* at 1515. Comments by jurors after the case seem to support this position. The London Times quoted two of the *Mekdeci* jurors as to their feelings about Judge Hoffman’s new trial order. One juror, Grover Ashcraft, said “I resent him (Hoffman) saying that we didn’t know what we were doing. He’s giving us a black eye.” Davin Light, *US Judge Orders Debendox Retrial*, THE SUNDAY TIMES (London), May 18, 1980, at 8. But another juror, Cora Newtz, agreed with the order. She did not believe the evidence against Bendectin was sufficient: “All my daughters took Bendectin all the time they were pregnant, and all their children are perfect.” *Id.*
ceutical defense and ease of access to the safety data that the defendant presumably enjoys. In sum, like plaintiffs, defendants do not begin with any particular expertise in litigating the product’s safety. Indeed, in the earliest stages of mass tort litigation, the defendants may not even appreciate that they have something out of the ordinary on their hands. The first suit is not necessarily like the teacher’s first occasion for sanctioning a wayward student at the beginning of the school year. The parties may not know that this is the first of a large congregation of cases. Bendectin had been on the market for nearly twenty years when litigation began, and at the time of the Mekdeci suit it had been nearly a decade since the Thalidomide disaster. It might well not have been immediately obvious to Merrell, upon the initial filing of the Mekdeci case, that this was the first of a series of cases.

In the case of Bendectin, however, Merrell’s appreciation that it faced a potential tidal wave of litigation must have come very early on. It recently had experienced the Thalidomide and MER/29 episodes and must have known it was a suspect firm in the eyes of many, especially the plaintiffs’ personal injury bar. Perhaps the first clear signal that Mekdeci would be the first of many was the hiring of Melvin Belli. Mr. Belli’s stock-in-trade is widespread publicity. Even before Mekdeci came to trial, he was searching out additional potential clients. By the time of the trial, the stakes were known to be high. Like the plaintiffs, however, the defendant was hampered by a relative lack of hard evidence on the teratogenicity of Bendectin.

The trial court’s ruling that the first Mekdeci verdict was the result of compromise led to a new trial in the Winter of 1981. This time, the result was a jury verdict for the defendant. The plaintiffs appealed, claiming, in part, that they had been afforded inadequate representation by their counsel. The Mekdeci’s attorneys had repeatedly attempted to withdraw, apparently for a combination of reasons, including their belief that the case was not strong on the facts, their lack of financial resources, and the difficulty of working with Mrs. Mekdeci.

As this history suggests, part of the Mekdecis’ problem was that their counsel were poorly prepared and underfinanced. The local attorneys were relatively inexperienced in this type of litigation and knew little about Bendectin. The solution to the Medkecis’ first plaintiff problems would have been to invest resources in discovery and experts in order to develop the case. But the Mekdecis’ counsel were always

209. See infra note 217.
210. Mekdeci, 711 F.2d at 1516-19.
211. Because of the inevitable costs of discovery and expert witnesses, substantial re-
badly undercapitalized. Indeed, funds became so tight that much of the second trial was tried on the record of the first trial, without the benefit of live expert witnesses. Only a last minute infusion of $25,000 from Mr. Belli kept the second trial going.\textsuperscript{212}

The Mekdecis repeatedly stated to the trial judge that in spite of these difficulties, they wanted the second trial to go forward with existing counsel. On appeal, however, they argued that the trial court had erred by refusing to grant requests for continuance. Nevertheless, the Fifth Circuit Court of Appeals affirmed the judgment below, leaving the Mekdecis with the cold comfort that if they felt they had been inadequately represented, their remedy was a malpractice suit against their attorneys, not a new Bendectin trial.\textsuperscript{213}

Counsels' desire to withdraw from the Mekdeci case was not motivated entirely by funding problems and the difficulty of working with Mrs. Mekdeci. They were also drawn by the prospect of "better" claims,\textsuperscript{214} especially claims where the specific causation question\textsuperscript{215} sources are required to develop any products liability case. Drug cases typically are even more expensive to litigate because of the volume and variety of information that must be assimilated, including the substantial amount of material generated during the process of regulatory approval of the drug.

\begin{itemize}
\item \textsuperscript{212} Mekdeci, 711 F.2d at 1519. The inability of an initial set of plaintiffs' attorneys to fund mass tort litigation has occurred in other cases. In the Agent Orange litigation, the original team of plaintiffs' counsel ran out of funds, and after brief negotiations, a new set of lawyers took control of the plaintiff steering committee by investing $250,000 each in litigation expenses. In total, the second set of attorneys put up almost $1.5 million. John C. Coffee, Jr., The Regulation of Entrepreneurial Litigation: Balancing Fairness and Efficiency in the Large Class Action, 54 U. Chi. L. Rev. 877, 901 (1987).
\item \textsuperscript{213} Mekdeci, 711 F.2d at 1523.
\item \textsuperscript{214} By looking for a "better" case than Mekdeci, the lawyers were behaving like "repeat players," thinking of this particular case as only one of many. See Galanter, Limits of Legal Change, supra note 19, at 100. In hindsight, their judgment that this was not the best case to try first appears to have been correct.
\item \textsuperscript{215} The cause-in-fact question in many such mass tort situations can be divided into two parts, general and specific causation. General causation addresses the question of whether the substance is capable of causing any injury. Specific causation addresses the question of whether a particular plaintiff's injury was caused by exposure to the drug or product. A substantial number of Bendectin plaintiffs have lost on the specific causation issue because they have been unable to show that the mother took Bendectin during the critical period of fetal development.

There is, however, a more fundamental specific causation question in the Bendectin cases. If the relative risk of a Bendectin-induced birth defect is shown to be less than 2, then a plaintiff presenting no other causal evidence than the ingestion of Bendectin could be said to have failed to show by a preponderance of the evidence that Bendectin caused the specific birth defect. The specific causation problems posed by toxic tort and mass tort cases have produced a large literature on how the courts ought to deal with this question. See Richard Delgado, Beyond Sindell: Relaxation of Cause-In-Fact Rules for Indeterminate Plaintiffs, 70 Cal. L. Rev. 881 (1982); Harris, supra note 138, at 909; Glen O. Robinson, Probabilistic Causation
would be less troublesome—apparently Mrs. Mekdeci had taken a large number of drugs during pregnancy.216 The lawyers were also drawn to the prospect of more claims and the possibility that they could mobilize a large number of Bendectin cases.217 In this vein, the appellate court in Mekdeci noted:

In a statement that perhaps shed more light than intended on the attorneys' motivations in the Mekdeci case, [plaintiff's attorney Allen] Eaton recounted numerous problems in the Mekdeci lawsuit and said, "that being the case, we have a group of people—a group of attorneys who have agreed that, in order to start afresh and develop the issues


216. Belli was quoted by the London Sunday Times as saying, "Our client is too demanding and too hard to work with. Her's wasn't that good a case, and we want to get on with the 200 other Debendox cases we are preparing." Edward Burke, The Brawl Over Bendectin, NAT'L L.J., Apr. 6, 1981, at 1, 12.

217. An advertisement placed in the San Francisco Chronicle by Melvin Belli in January and February 1980 reads:

BENDECTIN TAKEN
For Morning Sickness
Causing Malformed Children
We have a number of these cases and we are trying one now in Federal Court, Florida. We need your help for epidemiological and statistical purposes. If you have any information, please call or write:

Melvin M. Belli, Belli Building
San Francisco, CA. 94111 (415) 981-1849

Id. at 1. The same article in the National Law Journal quotes Douglas Peters, a lawyer retained by Mrs. Mekdeci to represent her against her previous lawyers, as saying:

Here you had a bunch of lawyers sensing a very big case and sitting down and dividing up the pie before there was any pie to divide. Then after trumpeting the case and gathering in excess of 100 other Bendectin cases, things began to go wrong and everybody scrambled to cut their losses.

Id. at 12.

The rush to mobilize clients is a recurring feature of many mass tort cases. Similar efforts have been noted in the Agent Orange, Bhopal, Dalkon Shield, and asbestos litigation. Coffee, supra note 212, at 886. As Coffee notes, in each of these situations the normal relationship between client and attorney is reversed. The attorney is no longer the agent of the client. The mass tort plaintiff, even more than the ordinary tort plaintiff, confronts substantial "agency costs" that make it very difficult to control plaintiffs' counsel. Id. at 885; John C. Coffee, Jr. Rethinking the Class Action: A Policy Primer on Reform, 62 IND. L.J. 624, 628 (1980). As Mekdeci suggests, this problem is especially acute in the early stages of a mass tort when counsel have particularly strong incentives to behave opportunistically, pursuing their own agendas rather than that of the client.
properly, that the Koller case in our opinion represents perhaps the clearest case of all the cases and the clearest one. We have a nurse here who ingested Bendectin, and only Bendectin. She ingested it during the critical period. And little Anne Koller has no arms and she has only a left leg which has a club foot on it. We figure this was a clear case in order to litigate the issues properly.\textsuperscript{218}

As the preceding discussion indicates, when a potential mass tort arises, plaintiffs' lawyers quickly organize. Plaintiff litigation networks have played an increasingly important role in mass tort cases.\textsuperscript{219} Rheingold notes there have been networks in the following areas: Swine Flu; Dalkon Shield; Agent Orange; MER/29; Birth Control Pills; Asbestos; DES; Ford Transmission; and of course, Bendectin.\textsuperscript{220} Moreover, there is a substantial benefit to being a leader of the litigation network; an advantage that apparently was recognized by counsel for the Mekdecis.\textsuperscript{221} As Rheingold notes, "A mere 5% of the recovery in the thousands of cases filed, and to be filed, dance like sugar plums in the heads of lead counsel-to-be."\textsuperscript{222} The Mekdeci case, then, was plagued not only by a lack of prior mobilization, but also by concurrent efforts to mobilize other cases.

Following Mekdeci, the next case did not go to trial until three years later, in the spring of 1983.\textsuperscript{223} Then there was a twenty-one month hiatus before the Common Issues Trial, about which I shall say more below. Over that period of time, lawyers representing plaintiffs made progress toward mobilizing resources and coordinating activities, driven in large part by the coordination of discovery imposed by the federal judiciary under the Multi-District Litigation Act.\textsuperscript{224}

(2) Merrell's Preventive Efforts

After Mekdeci, only one additional case came to trial on its own without being either swept into the unified multidistrict discovery in


\textsuperscript{219} See Rheingold, The MER/29 Story, supra note 62, at 123; see generally Paul D. Rheingold, The Development of Litigation Groups, 6 AM. J. TRIAL ADVOC. 1 (1982) [hereinafter Rheingold, Litigation Groups].

\textsuperscript{220} Rheingold, Litigation Groups, supra note 219, at 14.

\textsuperscript{221} See the court's comments in Mekdeci, 711 F.2d at 1516.

\textsuperscript{222} Rheingold, Litigation Groups, supra note 219, at 3.

\textsuperscript{223} Oxendine v. Merrell Dow Pharmaceuticals, 506 A.2d 1100 (D.C. App. 1986).

Ohio or delayed while the Ohio case proceeded. The one other case, *Oxendine v. Merrell Dow Pharmaceuticals*, went to trial in May 1983. The one month *Oxendine* trial resulted in a plaintiff's verdict for $750,000. The trial judge granted judgment n.o.v. (or judgment notwithstanding the verdict) in favor of Merrell, finding the verdict to be against the great weight of the evidence. The District of Columbia Court of Appeals reversed, saying this was an abuse of discretion, and reinstated the verdict. On remand, Merrell filed a motion for a new trial on the ground that one of the plaintiff’s experts, Dr. Done, had testified falsely at trial. After an evidentiary hearing, the trial judge granted the motion in February 1988. In August 1989 the District of Columbia Court of Appeals again reversed, ruling that the trial judge erred in granting a new trial and reinstated the original verdict. Defendant’s petition for a writ of certiorari was denied in 1990, presumably drawing the case to a close seven years after it was tried. Currently, *Oxendine*


226. *Id.* at 113. As the comments of one of the Mekdecis’ lawyers indicate, see *supra* text accompanying note 218, in the early years of litigation many plaintiffs’ lawyers looked to *Koller* as the best test case, in part because, unlike *Mekdec*, *Koller* presented no uncertainty as to whether some other drug taken during pregnancy could have caused the injury. But as previously noted, *Mekdec* was tried first. And in the meantime, *Oxendine* arose quickly. It was filed and resolved within a matter of months. *Koller*, on the other hand, became completely bogged down after the trial judge removed plaintiff’s counsel because of allegations (later retracted) by plaintiff’s counsel’s secretary that the plaintiff had lied in stating she had taken Bendectin, and because counsel had shared with the press several DERs the judge had ruled were inadmissible at trial. *Koller v. Richardson-Merrell Inc.*, 737 F.2d 1038 (D.C. Cir. 1984), *vacated*, 472 U.S. 424 (1985). Subsequent appeals of the actions took several years, ending in the United States Supreme Court. See *id.* By the time the issue was resolved, the Bendectin Cases had matured and *Koller* was but a footnote. For a description of these events see David Lauter, *Plaintiffs’ Firm Removed From Bendectin Lawsuit*, NAT’L L.J., Jan. 24, 1984, at 4; David Lauter, *Bendectin Trial Disintegrates: Allegations of Misconduct Mar 'Perfect Case,'* NAT’L L.J., Feb. 21, 1983, at 1. In August 1990 the United States Court of Appeals for the District of Columbia affirmed a dismissal of the Kollers’ case against Merrell. *Koller v. Richardson-Merrell, Inc.*, 5 Toxics L. Rep. (BNA) No. 16, at 528 (D.C. Cir. Sept. 19, 1990), *cert denied*, 111 S. Ct. 1391 (1991).


229. *Id.*

230. As of midsummer 1991, however, the plaintiff had still not been able to collect on the judgment. The District of Columbia Court of Appeals recently held that the trial court may not enter an enforceable and appealable “final judgment” on the compensatory damages claim while the question of punitive damages still remains to be tried. *Merrell Dow Pharmaceuticals, Inc. v. Oxendine*, 593 A.2d 1023, 1023 (D.C. 1991).
is one of only three verdicts against Merrell that has not been overturned by a trial or appellate court.\textsuperscript{231}

Merrell’s relentless efforts to overturn the Oxendine verdict were driven in part by its desire to avoid an adverse judgment. But the efforts also reflect the fact that Oxendine does not stand alone, but rather as one of the Bendectin cases. The stakes in these appeals reach far beyond Oxendine itself. Merrell is a “repeat player” playing for rules and outcomes in future cases.\textsuperscript{232} Oxendine blemishes a near-perfect record of defense outcomes. It singlehandedly serves as an incentive for plaintiffs to continue to litigate while acting as an important barrier to Merrell’s efforts to obtain summary judgment, directed verdict, or judgment n.o.v. based on collateral estoppel-type arguments in future cases.\textsuperscript{233}

\textsuperscript{231} The second is Raynor v. Richardson Merrell, No. 83-3506, 1987 WL 8518, at *1 (D.D.C. Mar. 5, 1987). In Raynor, a jury returned a verdict of $300,000 against Merrell in May 1987. As of November 1991, Merrell’s post-trial motions for judgment n.o.v. and a new trial were still pending before the trial judge. Letter from Glenn Forrester to Joseph Sanders (Oct. 19, 1990) (on file with author); Telephone Interview with Barry Nace, Plaintiffs’ Attorney in Raynor (Dec. 9, 1991). Given the D.C. Circuit’s opinions in Richardson v. Richardson-Merrell, Inc., 857 F.2d 823 (D.C. Cir. 1988) (discussed infra notes 331-39), and Ealy v. Richardson-Merrell, Inc., 897 F.2d 1159 (D.C. Cir.), cert. denied, 111 S. Ct. 370 (1990), it is difficult to imagine that, absent new evidence as to the teratogenicity of Bendectin, the Raynor verdict can survive an appeal. (Ealy is discussed infra notes 343-45 and accompanying text.)


In addition to these three cases, there is a plaintiff’s verdict in Blum v. Merrell Dow Pharmaceuticals, Inc., 560 A.2d 212 (1989), that was overturned by a middle level appellate court in Pennsylvania on the ground that the verdict was returned by an 11 person jury in violation of the Pennsylvania constitution. Id. The Pennsylvania Supreme Court has granted an appeal, 590 A.2d 755 (1991), but as yet, there is no opinion.

\textsuperscript{232} See Galanter, supra note 19, at 99-100.

\textsuperscript{233} Collateral estoppel (or issue preclusion) prevents a party from relitigating in a subsequent action one or more issues that have already been adjudicated. For collateral estoppel to apply, an issue practically identical to the issue in the current action must have been fully and fairly litigated in the original action, it must have been actually decided, and it must have been necessary to the outcome of the litigation. 5 CHARLES A. WRIGHT & ARTHUR R. MILLER, FEDERAL PRACTICE & PROCEDURE § 1225 (2d ed. 1990). All of these criteria are typically met with respect to the general causation issue in the Bendectin cases.

Traditionally, mutuality had to exist for collateral estoppel to apply; that is, both parties had to be bound by the prior decision. However, mutuality is no longer always necessary. In Parklane Hosiery Co. v. Shore, 439 U.S. 322 (1979), the Supreme Court gave qualified approval to offensive, nonmutual estoppel. Id. at 322. Offensive collateral estoppel is used by plaintiffs who attempt to prevent a defendant from denying liability. In at least two cases, In re Bendectin Products Liab. Litig., 749 F.2d 300, 305 (6th Cir. 1984), and Raynor v. Richardson-Merrell, Inc., 643 F. Supp. 238 (D.D.C. 1986), Bendectin plaintiffs have attempted to use offensive collateral estoppel (or summary judgment under a collateral estoppel-like analysis) based on Oxendine. In each case the court refused, noting in Raynor that using offensive
most cases are successfully defended, an occasional plaintiff success will encourage other suits. Even a high batting average may spell doom for the defendant. The General Counsel for one pharmaceutical company is quoted as saying:

Even if we win almost every case against us, the few verdicts we lose engender more suits, and make all the other suits more expensive and more difficult to settle . . . . There has to come a point with a particular product, even a good product, where you say, that's enough, and you get out of the market.234

As the quote suggests, because Oxendine offers some hope for future plaintiff victories, it also influences the settlement value of other cases.235 It is clearly in the interest of the defendant to suppress settlement value as much as possible. This is especially true in dealing with a congregation of cases. Evidence from the MER/29236 and asbestos cases237 suggests that as a congregation matures, the settlement value can steadily increase unless the defendant is uniformly successful. Rheingold reports that for a "typical MER/29 case" settlements rose from approximately $25,000 in the early period, to $75,000 in the mid-period, to $125,700 near the end.238 Adverse verdicts played an important role in this increase. Merrell won most of the early cases in 1964 and 1965, but plain-

collateral estoppel would be particularly unfair to the defendant when there were inconsistent jury verdicts outstanding. Raynor, 643 F. Supp. at 246.

Merrell, of course, has tried to invoke defensive collateral estoppel in numerous cases. Parklane, however, says that collateral estoppel is inappropriate when there is "a series of cases with inconsistent judgments." Parklane, 439 U.S. at 330. On the basis of this language, Judge Rubin rejected Merrell's invocation of collateral estoppel in the Michigan Consolidated Trial partly because of the Oxendine verdict. In re Bendectin Products, No. 85-0996, 1986 WL 20466, at *3 (E.D. Mich. May 2, 1986).

Even if Oxendine did not exist, Merrell's use of defensive collateral estoppel would still face formidable barriers, as most courts would probably conclude that the plaintiff had not been given a full and fair chance to litigate the issue. See In re Bendectin Products Liab. Litig., 732 F. Supp. 744, 746 (E.D. Mich. 1990). Still, Merrell's chances of obtaining summary judgment based on arguments that echo collateral estoppel would be better were it not for Oxendine. See Lynch v. Merrell-Nat'l Lab., 830 F.2d 1190, 1193 (1st Cir. 1987) (defendant not allowed to invoke defensive collateral estoppel where plaintiffs in multidistrict litigation had been allowed to withdraw without forfeiting any rights after discovery and before trial).

234. Tamir Lewin, Pharmaceutical Companies Are Hardest Hit, N.Y. TIMES, Mar. 10, 1985, § 3, at 1, quoted in Galanter, supra note 8, at 381.

235. The Oxendine verdict must have influenced Merrell's settlement offer in the consolidated multidistrict action in Ohio, discussed infra notes 255-300 and accompanying text.

236. See Rheingold, supra note 62, at 137.


238. He defines a "typical case" as involving a man under 60 with no earnings loss, only slight medical expenses, and strong medical proof of cataracts and hair and skin change. Rheingold, The MER/29 Story, supra note 62, at 137.
tiffs won several in 1966 and 1967. It seems apparent that these victories increased the value of untried cases.

There is a final reason Merrell continues to fight the Oxendine verdict. They may have come to believe, based on the outcome of more recent cases, that a new trial based on the evidence available at the beginning of the 1990s would result in a judgment for the defense. This circumstance reflects how Oxendine, like Mekdeci, presented Merrell with a "first case" problem. Inevitably, if mass tort cases are tried while science is still developing, early cases will be tried on different, less well developed facts than later ones. Nearly as inevitably, this will benefit one party or the other. In the Bendectin cases, the plaintiffs appear to have had the early advantage. By using every available means to obtain a new trial in Oxendine, the defendant has sought to erase this advantage.

239. As of 1967, 11 cases had gone to trial. Id. at 133. A Westlaw search in Spring 1990, using MER/29 as the search criterion, failed to discover any additional trials that resulted in appellate opinions. The most recent case for which there is an appellate opinion is Martinez-Ferrer v. Richardson-Merrell, Inc., 105 Cal. App. 3d 316, 164 Cal. Rptr. 591 (1980). Not surprisingly, the appeal concerned the statute of limitations. Id. at 318, 164 Cal. Rptr. at 591.

240. Punitive damages also played a role in the settlement value of the MER/29 cases. Because compensatory damages tended to be relatively small and because of Merrell's failure to disclose information to the FDA, the litigation efforts were driven by the desire to obtain punitive damages. While the plaintiffs were awarded punitives in only three cases (all three of which were reduced or overturned on appeal), these awards drove up the settlement value of other cases. Rheingold, The MER/29 Story, supra note 62, at 138. Rheingold cites two other factors as having influenced the size of MER/29 settlements. First, the earliest cases that went to trial or were settled were those in rural areas and smaller states where the expected value of cases was lower. Id. at 132. Second, there were differences in the quality of plaintiffs' counsel. Early in the litigation 33 attorneys representing MER/29 plaintiffs formed the MER/29 Group, whose purpose it was to disseminate information about ongoing litigation, to hire expert consultants and to conduct joint preparation of cases, primarily through a shared discovery effort. Eventually 288 attorneys and firms joined the Group. Among the packages of materials distributed by the Group were transcripts of previous trials, key documents that had been discovered from Merrell files, depositions, and a "trial package" with suggested examination questions and examples of motions and briefs used in earlier trials. Id. at 123-24. According to Rheingold, attorneys who were not members of the Group made worse settlements than attorneys who had the advantages of the Group's resources. Id. at 138. It is worth noting that this effect carries over to the litigated cases; plaintiffs who lost earlier cases were disproportionately represented by attorneys who were not members of the MER/29 Group.

The MER/29 settlement experience supports McGovern's "cyclical theory," see supra note 42, at least in its early stages. Plaintiffs' counsel slowly developed information and strategies that eventually allowed them to win most litigated cases. Coincidentally, this point was reached at approximately the same time that Merrell's insurance was exhausted. This caused Merrell to take over its own defense and actively seek settlement of all pending cases. Id. at 140. The number of cases rather quickly declined as depletion effects, accelerated by the statute of limitations, exhausted the number of potential cases. As we shall see, the Bendectin cases had a different fate.
B. The Caseload and Trial Load

Mekdeci and Oxendine marked the beginning of over a decade of litigation concerning Bendectin. For a while, they appeared to be just the beginning. Filings rose dramatically in the early 1980s, but then collapsed. After the Common Issues Trial. Table 5241 presents the Bendectin-related case filings in state and federal courts from 1977 to 1988.242

Bendectin trials have followed a pattern similar to case filings. Through December 1991 there had been approximately thirty trials, including two German cases, one Italian case, and several consolidated trials—by far the most important being the Common Issues Trial in the Southern District of Ohio which consolidated 818 cases, some involving multiple plaintiffs.243 All but a handful of the cases have been tried in the federal courts, most removed there by the defendant under diversity jurisdiction. Table 6244 reports on trials by year.245

There were two trials in 1985, eight in 1986, and eleven in 1987; then the number of trials collapsed. While a few cases are still pending, the stock of active potential cases is disappearing, due, in part, to Merrell's aggressive efforts to bring litigation to a close.246

241. Table 5 is found in Appendix A.
242. GAO, "LITIGATION EXPLOSION" QUESTIONED, supra note 87, at 35. In the table, any case originally filed in state court, but removed to federal court is counted twice. Between 1977 and 1986 there were 261 such cases. If the double counting is eliminated, there were 1648 filings between 1977 and 1986. Merrell reports that as of mid-1989 there were a total of 1696 child plaintiff cases. See supra text accompanying note 86. Between 1974 and 1986 the Bendectin cases constituted approximately 1.7% of the total products liability filings in the federal courts. Marc Galanter, The Life and Times of the Big Six: The Federal Courts Since the Good Old Days, 1988 Wis. L. Rev. 921, 941 [hereinafter Galanter, Life and Times of the Big Six]. See TERRY DUNGWORTH, PRODUCT LIABILITY AND THE BUSINESS SECTOR: LITIGATION TRENDS IN FEDERAL COURTS 41 (1988). By way of comparison, the enormous Asbestos congregation comprised approximately 6.4% of all civil filings in federal courts in 1990. 19 Prod. Safety & Liab. Rep. (BNA) No. 11, at 287 (Mar. 15, 1991).
244. Table 6 is found in Appendix A.
245. The table reports every trial, including retrials of the same case, such as occurred in Mekdeci. Because some cases are counted twice, and because some trials are consolidated trials of a number of individual cases, the number does not accurately reflect the number of cases that have actually gone to trial.
246. For instance, one of the most important recently-outstanding cases was a multi-plaintiff case in Michigan before Judge Rubin (who had presided over the Ohio Common Issues Trial). In October 1989 Merrell filed a motion for summary judgment on the causation question and also filed case-specific motions based on statute of limitations, lack of ingestion, or timing of ingestion defenses in approximately 20 cases. Merrell also moved for discovery sanctions because over half the plaintiffs had failed to respond to discovery requests. While many of these motions were granted, Judge Rubin denied Merrell's motion for a summary judgment.
The key events in Bendectin's trial history are not difficult to spot. The partial success in *Mekdeci* and the plaintiff's verdict in *Oxendine* were followed by a dramatic rise in filings over the next several years. Due to the common discovery procedures discussed below, however, there were no further trials until 1985. The next trial after *Oxendine*, the *Common Issues Trial* in Winter 1985 in the Southern District of Ohio, was the single most important trial in the Bendectin litigation. The case ultimately involved over 800 plaintiffs, and resulted in a defense verdict on the question of whether Bendectin was a teratogen. As a consequence of this verdict, new filings collapsed. However, the number of trials, functioning as a lagging indicator of events, continued to rise for two years before trials also began to decline in frequency. The relationship of filings and trials through the 1980s is presented in Figure 2.

**Figure 2**

*Total Filings and Trials, by Year*

![Graph showing total filings and trials by year](image.png)

in January 1990. Merrell moved for reconsideration of the motion and a hearing was held in March 1990. At that time, Judge Rubin proposed a mini-trial to ascertain whether the testimony proffered by plaintiffs in opposition to the summary judgment motion would be admissible. Transcript of Proceedings at 22-23, *In Re Bendectin Litig.*, No. 85-0996, 1986 WL 20466 (E.D. Mich. May 2, 1986) (Hearing on Motion). One possible interpretation of the judge's statements at the hearing is that he was likely to hold the animal studies inadmissible. See id. at 3. Within a few weeks of the hearing the remaining plaintiffs agreed to dismiss their cases without prejudice. They have three years to refile. Letter from W. Glenn Forrester, attorney for Marion Merrell Dow, Inc., to Joseph Sanders (Oct. 18, 1990) (on file with author).
The full extent to which trials can be viewed as lagging indicators in the Bendectin cases is further demonstrated in Figure 3. It presents the number of in vivo and epidemiological studies published and the number of trials begun in different time periods from 1962 to 1991. For the period covered in this figure, 94% (47/50) of all in vivo and epidemiological studies were published before 80% (24/30) of the trials had begun.

Figure 3
Bendectin Scientific Articles and Trials, by Years

Importantly, in the Bendectin Cases the declining number of filings and trials has not been primarily a result of the exhaustion of potential claims. A number of leading plaintiffs' attorneys, including Barry Nace and Thomas Bleakley, took their cases out of the Common Issues Trial and were prepared to proceed separately.247 In addition, there are many potential plaintiffs who have not entered the fray. Estimates made at the time of the Common Issues Trial were that the total number of Bendectin

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247. In fact, most of the subsequent Bendectin litigation involved these cases. For these cases, the impact of the outcome in the Common Issues Trial was indirect. As I shall discuss below, the defense verdict in the Common Issues Trial was an important factor in the judicial willingness to direct verdicts and enter judgment n.o.v. in later cases. See infra text accompanying note 309-10.
filings ultimately would be in the 15,000 range. These estimates were based on relatively conservative estimates as to what percentage of potential plaintiffs (children with birth defects whose mothers had taken Bendectin during a critical period of their pregnancy) would in fact raise claims. The fact that far fewer cases have been filed is a consequence of the lack of plaintiff success in the cases that have been tried. Fundamentally, the process has been one of judicial rationing of access to the courts, beginning with the discovery and trial in Ohio.

C. Rationing Law

In the Bendectin Cases, the rationing process has occurred in two stages: first, through the use of traditional procedural techniques to conserve judicial resources; second, through increasingly aggressive judicial management of cases. In the following sections, I discuss these strategies in turn.

(1) Procedural Rationing

Faced with the stunning volume of cases presented by mass torts and the time it would take to try each case separately, commentators have suggested various ways to expedite proceedings through the increased use of mass discovery and mass trials. Several devices, including the Multi-District Litigation Act, class actions under Rule 23 of the Federal Rules of Civil Procedure ("Rule 23"), and consolidation of cases for trial under Rule 42 of the Federal Rules of Civil Procedure ("Rule 42") have been used in the Bendectin Cases. All of these propos-

251. See Mullenix, supra note 249, at 1045-46.
als and devices are justified by their proponents on the basis of the inefficiency and costs involved in single plaintiff litigation.\textsuperscript{252} Whether or not there in fact are large savings to be gained by using these devices is, to some extent, an open question.\textsuperscript{253} What is clear is that these devices can save a substantial amount of judicial time, and that judges will explore various procedural devices that allow them to avoid spending months and even years litigating a group of mass tort cases one at a time.\textsuperscript{254} For example, Judge Rubin, the trial judge in the \textit{Common Issues Trial}, calculated that the trial of all 1100 then existing Bendectin cases would take 182 judge years.\textsuperscript{255} Trying even 5\% of the cases would consume over nine judge years. It is not surprising, therefore, that the judiciary, especially at the trial court level, searches for ways to ration judicial resources. The Bendectin Cases reflect this phenomenon.

Because the \textit{Common Issues Trial} so aptly demonstrates coordination and rationing in mass torts, it is worth discussing in some depth. The trial was the direct result of the consolidation of cases under Rule 42 and the transfer of cases to Ohio under the Multi-District Litigation Act ("Act" or "MDL statute").

The Act is a fundamental tool in the federal judiciary’s efforts to force coordination on parties. As a byproduct, it facilitates the mobilization of plaintiffs’ groups. The MDL statute was enacted in 1968. It calls for the creation of a seven member Judicial Panel on Multi-District Litigation ("Panel"). The members are United States District Court or

\textsuperscript{252} It has been said that mass tort cases lead to an exponential growth in transaction costs as the number of parties increases. Richard Epstein, \textit{The Legal and Insurance Dynamics of Mass Tort Litigation}, 13 J. LEGAL STUD. 475, 477 (1984). Whether this is in fact true for all mass torts is debatable. It is distinct from the question whether savings can be realized by consolidating individual cases into larger groupings by way of common discovery or class actions.

\textsuperscript{253} For an argument that the savings are not always as large as advertised, see Trangsrud, \textit{Mass Trials}, supra note 249, at 79. For an effort to set forth a method for assessing the effects of mass procedures, see \textsc{Mark A. Peterson \& Molly Selvin, Resolution of Mass Torts: Toward a Framework for Evaluation of Aggregative Procedures} (1988).

\textsuperscript{254} Judge Parker’s efforts in the Eastern District of Texas to use collateral estoppel and class actions to manage the very large asbestos caseload in that district serve as an example. See Jenkins v. Raymark Indus., 782 F.2d 468, 469 (5th Cir. 1986) (class action); Hardy v. Johns-Manville Sales Corp., 681 F.2d 334, 338 (5th Cir. 1982) (collateral estoppel). For a discussion by a commentator who has been intimately involved with managing mass tort cases, see McGovern, \textit{supra} note 20, at 442.

\textsuperscript{255} In \textit{re} Richardson-Merrell, Inc. “Bendectin” Prods. Litig., 624 F. Supp. 1212, 1221 n.6. (S.D. Ohio 1985). Judge Rubin’s calculation was based on his estimate that the average trial would take 38 days. This figure in turn was based on the average time it had taken to try the four cases (other than the \textit{Common Issues Trial}) that had been tried at the time Rubin wrote his opinion. \textit{Id.} at 1221 n.5.
Court of Appeals judges, no two from the same district. The Panel maintains a permanent staff in Washington and meets monthly. It monitors district court cases that appear to be likely candidates for consolidation. Claims may be consolidated by motion of the Panel itself or by approved motion of a party to a claim. The Panel may issue a consolidation order over the objections of all parties. For the Panel to take action, four of the seven members must concur. Civil actions may be consolidated when: (1) they involve one or more common questions of fact, (2) they are pending in different districts, (3) the transfer will be for the convenience of parties and witnesses, and (4) the transfer promotes the just and efficient conduct of the actions. Factors to be considered in determining whether consolidation would promote just and efficient conduct of the actions include whether it will prevent duplication of discovery and pretrial conferences and whether it will economize judicial efforts.

As of July 1984, the Panel had used the MDL statute to transfer seventy-eight air disaster cases and seventeen other mass disaster cases. It had also transferred nine mass product defect cases, including those involving Bendectin. The Act, on its face, provides for the consolidation of pretrial proceedings only. At the end of these proceedings “shall be remanded by the panel . . . to the district from which [they] were transferred” for trial. Notwithstanding this language, both courts and commentators have asserted that transferee courts may conduct a common trial in cases consolidated under the Act.

Even as Mekdeci was being tried, the landslide of lawsuits was beginning. In early 1982 the Panel began to assign cases to the Southern District of Ohio. It selected Judge Carl A. Rubin to conduct the consolidated pretrial discovery. In May, a central component of the coordi-

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260. *Id.* at 803-04 n.139. Other products involved in transfer orders include an aortic heart valve, multipiece rims, an antibiotic called Cleocin, the Swine Flu vaccine, the Dalkon Shield, plastic insulation, and helicopter engines. *Id.*
nation objective was realized with the appointment by Judge Rubin of five attorneys who would act as lead counsel and coordinate plaintiffs' discovery efforts.264

Within the context of the Act, another word for coordination is centralization. In ordinary tort litigation, unaffected by multidistrict consolidation, the very fact that lawsuits are scattered throughout the land is simultaneously one of the main weaknesses and strengths of private litigation. It is a weakness because coordination is difficult, and because some plaintiffs will not have the resources to pursue their claims.265 On the other hand, this diversity is a strength. If it spreads plaintiffs thin, it does the same to the defendants, who must fight on many fronts. Moreover, it spreads the risk. Prosecuting 800 single plaintiff cases instead of one 800 plaintiff case is a type of insurance for plaintiffs as a group. One large case places all of the plaintiffs' eggs in one basket.266

In November 1983 Judge Rubin took a large step toward centralization. Proceeding under Rule 42, the judge consolidated all cases that had been filed in Ohio for a common issues trial. Rule 42 is a particularly potent weapon for forcing consolidation because plaintiffs do not have a right to opt out of a common trial as they do under the MDL statute.267 Judge Rubin set a date of March 1, 1985 for all those who did not bring their action in Ohio to choose whether to opt in or opt out of the Common Issues Trial.268 And on June 11, 1984 a jury was empaneled.269


265. The contingent fee can relieve the individual plaintiff of the burden of direct litigation expenses, but the system will operate only when the expected value of a case warrants the costs that must be incurred in pursuing it. For the plaintiff with a less serious injury, such as a minor cleft, it may be impossible to find an attorney willing to pursue the claim if that plaintiff alone has to bear the entire cost of developing the case. Consolidation of discovery proceedings under the Act makes it possible for many plaintiffs to pursue claims that they otherwise could not afford.

266. In August 1982 Judge Rubin removed the foreign eggs. He ruled that claims by British plaintiffs were barred under the doctrine of forum non conveniens. In re Richardson-Merrell, Inc., 545 F. Supp. 1130, 1133-36 (S.D. Ohio 1982) (where United Kingdom was alternative forum and prospect of numerous additional actions on behalf of foreign plaintiffs was likely absent dismissal, action was dismissed on forum non-conveniens ground). British plaintiffs had survived an earlier motion by defendant to dismiss on forum non conveniens grounds. See Alexander v. Richardson-Merrell Inc., 541 F. Supp. 93, 98-99 (S.D.N.Y. 1982). Now, however, no foreign national would be able to pursue a Bendectin case in Federal court.


269. Id.
One week later, however, with an eye toward settlement, Judge Rubin issued an order certifying the plaintiffs as a class under Rule 23. Merrell made a settlement offer of $120 million and the jury was dismissed. Taking into consideration potential future suits, Judge Rubin divided the settlement class into two subclasses, those who had already filed suit by June 18, 1984 and those who had not. The class was a "non-opt-out" class. No individual plaintiff would be permitted to opt out and continue private settlement negotiations or proceed to trial. Every injured child and parent of a stillborn child exposed to Bendectin would be bound by the settlement agreement.

Judge Rubin's action did not meet with universal acceptance. Many attorneys, including two members of the five-person lead counsel committee, opposed the settlement and therefore objected to the formation of the class. After failing to obtain a reversal of the class certification order on direct appeal, they sought a writ of mandamus from the Sixth Circuit to vacate the order establishing the class. The court recognized that mandamus is an extreme remedy, but nevertheless granted the writ. This ended any hope of settlement.

270. In re "Bendectin" Prods. Liab. Litig., 102 F.R.D. 239, 242 (S.D. Ohio 1984). Rubin found that the class met the four criteria of commonality, typicality, numerosity, and adequacy of representation required by Rule 23. Id. at 241. For the argument that Rule 23 should be used this way in mass tort cases, see Note, Class Certification in Mass Accident Cases under Rule 23(b)(1), 96 Harv. L. Rev. 1143 (1983).

271. In re Bendectin Prods. Liab. Litig., 749 F.2d 300, 306 (6th Cir. 1984). At the time of the settlement offer, pending before Judge Rubin were 253 cases filed in the Southern District of Ohio and 315 transferred by the Panel (in addition roughly 149 actions were pending in state courts). Id. Had the settlement been consummated and distributed pro rata among the existing plaintiffs under Rubin’s jurisdiction, the per case settlement would have been slightly in excess of $200,000.


273. In re Bendectin Prods. Liab. Litig., 749 F.2d at 302. In addition, the certification order purported to stay all state court discovery proceedings. Id. at 304.

274. These extreme measures reflect the frustration judges feel in trying to work out a settlement in mass tort cases. Some have argued that Judge Weinstein's summary judgment for defendants in the Agent Orange opt-out opinion reflects similar frustration and was motivated in substantial part by his desire to protect the perceived reasonableness of the settlement agreement between the defendants and the plaintiffs who did not opt out of the class action. See Green, supra note 94, at 65.


277. In re "Bendectin" Products Liab. Litig., 749 F.2d at 303, 307. The court found that Judge Rubin had failed to establish any grounds for certification under either Rule 23(b)(1)(A) (requiring a showing that separate actions would create a risk of inconsistent or varying adjudications) or Rule 23(b)(1)(B) (requiring a showing that there is a risk that a limited fund may exist from which judgments can be satisfied). Id. at 305-06.
Actions taken in the early years of a mass tort have the secondary consequence of encouraging or discouraging the filing of new cases. With a product such as Bendectin, for which over thirty million prescriptions had been filled world-wide, it is extraordinarily difficult to estimate the number of potential cases. Indeed, in the Common Issues Trial, Judge Rubin solicited the assistance of several experts in order to obtain a reasonable estimate of the number of cases he could ultimately anticipate. The experts found it difficult to make a firm estimate, in part because there was no effective statute of limitations bar to future suits. The experts' best guess was that future filings would be in the range of 5700 to 16,800 claims. Thus, a settlement with only the existing plaintiffs seemed particularly risky for Merrell. In the asbestos, Dalkon Shield, and DES cases, initial settlements and jury verdicts set the going

278. Attempts to estimate the number of potential plaintiffs who would meet the two criteria of a birth defect and a mother who ingested Bendectin during pregnancy have produced very large numbers. Judge Rubin noted in the Common Issues opinion that if 30 million pregnant women had taken the drug and the birth defect rate is between 2% and 5% of all births, then by chance (i.e., by causes other than Bendectin), there would be between 600,000 and 1,500,000 potential claimants. In re Richardson-Merrell, Inc. “Bendectin” Prods. Liab. Litig., 624 F. Supp. 1212, 1229 (S.D. Ohio 1985).

279. For a discussion of why this is so, see supra notes 88-89 and accompanying text.

280. The experts estimated that there were between 115,000 and 168,000 potential claimants and that 5% to 10% would actually raise claims, creating a range of 5750 to 16,800. See Transcript of Proceedings, at 88-90 (Oct. 31, 1984), In re Richardson-Merrell, Inc. “Bendectin” Prods. Liab. Litig., 624 F. Supp. 1212 (S.D. Ohio 1985) (MDL No. 486). Among the factors involved in a calculation are estimates of the number of injuries and estimates of the number of injured people who will advance claims. The percentage advancing claims is influenced by public knowledge that there is a potential claim, which in turn is influenced by the steps courts and others take in advertising the right to claim.

In the Dalkon Shield case, for instance, the bankruptcy court made significant efforts to publicize the right to claim. A survey of almost 1000 individuals conducted in 1984 asked whether they had heard of a variety of products (including the Dalkon Shield, Bendectin, asbestos, DES, and super-absorbency tampons), and if so whether they thought they the product was hazardous to health. Of those surveyed, 80% thought asbestos was hazardous; 43% thought the Dalkon Shield was. Only 10% or so thought Bendectin was a health hazard, approximately 10% thought it was not a hazard, and 80% either had never heard of Bendectin or did not know if it was hazardous. Herbert M. Kritzer, Public Notification Campaigns in Mass Litigation: The Dalkon Shield Case, 13 J. Sys. J. 220, 231 (1988-89). Clearly, in 1984 public awareness of Bendectin was relatively low.

After the Common Issues Trial, plaintiffs' lead counsel requested reimbursement for out-of-pocket discovery expenses, which tallied $750,000. Judge Rubin charged two thirds of the costs to the Southern District cases, but also had to set a price for counsel in future cases who might wish to acquire the materials. He set a fee of $600, by estimating that there would be 400 future cases where counsel would use the materials. To date, however, there have not been nearly that many cases, presumably in part because the outcome of the Common Issues Trial had a chilling effect on future litigation. See infra app. A., tbl. 5. Had the outcome been different, there is reason to suspect the expert's estimates would, if anything, have been too low and the number of claims would have run in the thousands, if not tens of thousands.
rate for later cases. It usually does not avail the defendant to argue that it never would have offered so much in earlier cases had it known there would be so many later ones. Nor are existing plaintiffs in any position to offer reliable indemnity against future claims or even unsettled existing claims. By forming a non-opt-out class, and establishing a subclass of future suits, for whom some percentage of the $120 million settlement offer would be earmarked, Judge Rubin's proposed settlement insured Merrell against the risk that by settling, it would encourage even more new plaintiffs, who would tend to begin negotiations from the basis of the earlier settlement.

The Sixth Circuit's issuance of the writ of mandamus reflects the constant tension that exists in mass tort cases between the desire to provide individualized justice and the desire to reach for aggregate resolutions. Apparently, the Sixth Circuit felt that this settlement plan leaned too far in favor of efficient, aggregate solutions. The consequence of the Sixth Circuit's decision was that Judge Rubin resumed preparations for a common issues trial on the question of general causation. A second jury was empaneled in February 1985, and a new opt-in deadline was set. A total of 818 cases were consolidated: 557 cases originally filed in the Southern District of Ohio, and 261 opt-in cases. Judge Rubin took the unusual step of trifurcating the case. First, he would try the issue of general causation: does Bendectin cause any defects? He then would try liability issues and, finally, damages issues. This strategy

281. Trangsrud, Joiner Alternatives, supra note 22, at 835.
282. Frank Woodside III, chief defense counsel in the Common Issues case noted:

One thing that we have attempted to obtain in settling the cases the way we have settled them is to achieve a degree of finality so that the litigation does not pend for 20 years. Because you involve children, you have litigation that can go on for a long time because you really don't have a statute of limitations.

Bendectin Pact Creating Furor, supra note 275, at 31.
285. In re Richardson-Merrell, Inc. "Bendectin" Prods. Liab. Litig., 624 F. Supp. at 1216 n.1. Judge Rubin reports in his later opinion, upholding the jury verdict in this case, that there were an additional 368 cases assigned by the panel that did not opt in or were otherwise disposed of. In total, then, he had jurisdiction over 1186 cases. Id.
286. The exact nature of the trifurcation and the forum in which each issue would be tried was not clear when the trial began. Apparently the parties never fully considered how to resolve the difficult question of specific causation that might arise if the jury were to find a significant correlation between Bendectin and some birth defect, but a relative risk of injury less than two. In this situation, based on the bare statistical evidence, no plaintiff would be able to show by a preponderance of the evidence that his or her defect had been caused by Bendectin. See supra note 215.

In the event that the jury was to find for the plaintiffs on the causal and liability questions,
had the potential for greatly streamlining the case, for if the defendants won on the general causation issue there would be no need to continue the trial.

Moreover, the strategy had the effect of preventing the plaintiffs from trying their entire cause of action at one time, thus denying them the opportunity that exists in unitary trials to bolster a weak case on liability or causation with a strong case on damages. The only witnesses permitted to testify at the trial were experts on the chemical composition, toxicology, and epidemiology of Bendectin (ten experts on behalf of the plaintiffs and nine on behalf of the defendants). Testimony and evidence pertaining to individual plaintiffs was not permitted. Indeed, Judge Rubin would not even allow individual malformed plaintiffs in the courtroom during the trial, on the theory that to do so would inevitably bring the question of damages into the trial as the jury observed the plaintiffs' serious injuries.

Judge Rubin proposed a second innovation for this trial. Because of concerns that the complexity of the scientific evidence might be too difficult for the average juror to understand he offered to use either a "blue ribbon jury" (persons having the most formal education available in the jury panel) or a "blue-blue ribbon jury" (persons knowledgeable in the field). The offer was rejected by plaintiffs, and the case was tried to a jury from the regular jury pool.

Judge Rubin planned to return all cases referred under the MDL statute to the originating district for the purpose of determining damages. He left open the procedures to be employed with respect to cases originally filed in the Southern District of Ohio. They would be handled either by impaneling separate juries for each plaintiff, or by determination of the court or a special master. Pre-trial Order No. 1414, Mar. 5, 1984, In re Bendectin Prods. Liab. Litig., 102 F.R.D. 239 (S.D. Ohio 1984) (MDL No. 486).

287. It is partly on this ground that Trangsrud objects to the practice of splitting mass trials:

[Trifurcation of issues] is not fair because it robs the jury of its traditional flexibility in tort cases to balance uncertainties in the plaintiff's case on liability against strengths in the plaintiff's case on damages. Trifurcation of issues also inevitably leads to the sterile trial of technical issues related to causation divorced from the fact of the plaintiff's injury and a full account of the defendant's role in the tragedy. Trangsrud, Joiner Alternatives, supra note 22, at 80. Some would disagree with this analysis and argue that all trials should be bifurcated so that the damage question does not contaminate the logically prior liability and causation questions. Recall that the first Mekdeci jury verdict was overturned precisely because it appeared to be a compromise verdict.


289. Id. at 1228.

290. Id. at 1222-24. On similar grounds, Judge Rubin restricted pretrial discovery to Bendectin and at trial excluded all evidence concerning Thalidomide and MER/29. While Merrell's history with these drugs might go to the issue of liability, it did not go to the question of whether Bendectin caused birth defects. Id. at 1236, 1241, 1249.

291. Id. at 1217.
Perhaps because of the trifurcation of the trial, the jury returned a verdict for the defendants. In a lengthy opinion Judge Rubin denied plaintiffs' motion for judgment n.o.v. or a new trial. The Sixth Circuit ultimately upheld the verdict over objections that Judge Rubin abused his discretion in, among other things, trifurcating the trial, excluding the plaintiffs from the courtroom, and excluding references to Thalidomide and MER/29. In April 1985, in light of the jury verdict and Judge Rubin's declaration that he had completed his duty, the Panel remanded the claims of eighty-three plaintiffs who had not opted into the Common Issues Trial.

At a theoretical level, the verdict in the Common Issues case is just another jury verdict, binding on the parties to the case but of no particu-

292. In a laboratory experiment, using a toxic tort trial stimulus, Horowitz and Bordens found that juries hearing a unitary trial were significantly more likely to find for the plaintiff (85%) than were juries that heard bifurcated trials (68%). Irwin A. Horowitz & Kenneth S. Bordens, An Experimental Investigation of Procedural Issues in Complex Tort Trials, 14 LAW & HUM. BEHAV. 269, 277-78 (1990). This tendency was strongest when the bifurcated trial juries heard the general causation testimony first. If these juries did find for the plaintiff, however, their compensatory damages awards were significantly larger. Id.


294. The plaintiffs may have been somewhat prejudiced by the fact that one of the lead counsel had suggested a bifurcated proceeding early in the case. Id. at 1249.

295. In re Bendectin Litig., 857 F.2d 290, 314-17, 321-25 (6th Cir. 1988). The appellate process was delayed by litigation concerning whether Judge Rubin had jurisdiction over claims by two foreign nationals who had originally filed in Ohio state court. These plaintiffs alleged that Merrell had violated the Food, Drug and Cosmetic Act, and that this violation constituted a “rebuttable presumption of negligence.” Merrell argued that removal to federal court was proper under 28 U.S.C. § 1441 because the allegation raised a federal question. The trial court agreed, but was reversed in Thompson v. Merrell Dow Pharmaceuticals, 766 F.2d 1005, 1006 (6th Cir. 1985). The Sixth Circuit's decision was affirmed by the Supreme Court in a 5-4 opinion. Merrell Dow Pharmaceuticals v. Thompson, 478 U.S. 804, 817 (1986).

This ruling ultimately affected all Ohio resident plaintiffs. Merrell was a Delaware corporation, but had its principal place of business in Ohio. Therefore it was, for purposes of diversity jurisdiction, a citizen of both states. 28 U.S.C. § 1332(c) (1988). On the basis of the Supreme Court's Thompson decision, Judge Rubin proceeded to remand all cases brought by Ohio residents in Ohio state court that had been removed to his court under 28 U.S.C. § 1441 on the ground that they, too, had stated causes of action based on violations of the FDCA. Bendectin Litig., 857 F.2d at 297. For the same reason, he also dismissed without prejudice all claims brought by Ohio plaintiffs who had originally filed in Ohio federal court. Id.

On appeal, however, the Sixth Circuit held that only those Ohio plaintiffs (13 cases) who had not attempted to establish federal question jurisdiction through a cause of action based on the violation of the FDCA were entitled to have their claims dismissed without prejudice. Id. at 297-98. The remainder, who had claimed federal question jurisdiction under 28 U.S.C. § 1331, were properly in federal court and, therefore, bound by the Common Issues verdict. Id.

lar precedential consequence for other parties, not even those plaintiffs who were part of the common pretrial proceedings and then opted out. This theoretical level perceives each case as an isolated event, legally uncontaminated by other suits, and it completely misses the importance of the *Common Issues Trial*. The verdict produced a complete reversal of fortunes for the parties. The claims of over 800 plaintiffs had been dealt a death blow, and remaining plaintiffs now faced what amounted to an adverse finding of fact—subsequent trial and appellate judges referred to the *Common Issues Trial*’s outcome when discussing whether the plaintiffs could prove that Bendectin causes birth defects.\(^2\)

The defendant enjoyed an equally profound reversal of fortunes. From an offer of $120 million to settle the case, it now moved to stay Judge Rubin’s dismissal and remand order. The defendant hoped to sweep the board clean by using the *Common Issues Trial* to prevent future plaintiffs from arguing that Bendectin was a teratogen.\(^2\)

Nevertheless, trial of the Bendectin Cases had barely begun.\(^2\) Many of the most seriously injured plaintiffs had opted out of the *Common Issues Trial*.\(^3\) The existence of Oxendine made it unlikely that the defendant would succeed with any collateral estoppel arguments and also

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300. Class actions are, in part, a public good. Benefits accrue to members of the group (all plaintiffs) regardless of their individual efforts. Olson notes that in such situations, because those with the most at stake will invest the most in the common good, there is a tendency for the “exploitation’ of the great by the small.” MANCUR OLSON, THE LOGIC OF COLLECTIVE ACTION: PUBLIC GOODS AND THE THEORY OF GROUPS 29 (1968). Members with greater stakes in the outcome (the great) will make larger investments (in, for instance, pretrial discovery) than those with smaller stakes (the small). Those with smaller claims are able to free ride on the investment of those with larger claims.

This exploitation may occur even if there is no bargaining among plaintiffs. Bargaining may exacerbate this problem. To the degree there are limited assets to be shared, the plaintiffs will compete for shares of the defendant’s assets. Here, the large stakes plaintiffs almost certainly will be in a minority, and may well be subjected to efforts by the majority to squeeze the range of recoveries. There will be pressure to hold down the maximum recoveries to raise the minimum recoveries. Coffee, *supra* note 212, at 916.

As a consequence of these processes, it is not surprising that many of the opt outs in mass tort cases are the large stakes players. These include plaintiffs who have the more serious injuries and who have claims that are large enough to justify separate prosecution. They also include plaintiffs’ attorneys representing a number of clients who, aggregating across all of their claims, have a large enough pool of claims to justify trying individual cases. Such a division apparently occurred in the Bendectin Cases. As this discussion indicates, mass tort cases present substantial issues of distributive justice. For a set of proposals as to how to manage mass tort cases so as to minimize distributional problems, see *id.* at 918-30.
indicated that plaintiffs might well succeed in a unitary trial. The judiciary, however, was not to remain passive. As the Bendectin Cases matured, a new type of rationing was about to begin.

(2) Substantive Rationing

Throughout the life cycle of a congregation of cases the judiciary responds to the experts and their evidence. Trial judges are the most closely involved. The majority of their responses occur at the micro level of evidentiary and procedural rulings, permitting or prohibiting various initiatives by the parties. Usually, however, the trial judge also must make larger decisions concerning who to permit to testify, what evidence and instructions to give the jury, whether the case ultimately should go to the jury, and if it does, whether the verdict should stand. These more macro judgments are especially likely to be appealed. In the process of justifying or rejecting what has been done below, appellate opinions shape the future direction of a congregation of cases. Like all judicial, and other, decisions within the congregation, appellate opinions are not isolated, atomistic events. They, too, exhibit stages of development and are best understood within the context of the collection of cases. In this section I briefly discuss the trial court opinions and then turn to an analysis of the Bendectin appellate opinions. Here, we examine a different type of rationing, not through the use of procedures to streamline but through the use of substantive decisions to dispose of cases on their merits. As the congregation matures, the courts move from procedural to substantive rationing.

a. Trial Court Opinions

Using both Westlaw and Lexis searches, I have uncovered a total of fifty-eight trial court opinions concerning Bendectin, almost all of which

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301. I hope to explore these micro decisions in a subsequent paper through an analysis of Bendectin transcripts.

302. This shift suggests an interesting question: Under what circumstances will courts employ substantive rationing? We might hypothesize that courts will resort to substantive rationing only when procedural rationing has failed to achieve the desired result.

For example, in the mass torts that are precipitated by a single cataclysmic event such as an airplane crash, multidistrict discovery and the consolidation of cases for trial are sufficient to assure reasonably prompt and consistent outcomes within the congregation of cases. In such a scenario, substantive rationing is unnecessary even for the occasional case that escapes the procedural net.

If this hypothesis is correct, we should expect substantive rationing in relatively few instances. In the final section of this Article, I elaborate on the circumstances in which we should anticipate substantive rationing.
are from federal district courts. Table 7 summarizes the district court opinions.

In the table, I have coded the subject matter of each opinion (column 4) and designated it substantive or nonsubstantive (column 5). The key to the codes is found at the bottom of the table. Nonsubstantive opinions involve: discovery motions; motions to bifurcate or trifurcate a trial; questions of federal jurisdiction, statute of limitations, and forum non conveniens; and decisions concerning inclusion or exclusion from the multidistrict proceedings. Substantive opinions involve: questions concerning the admissibility of evidence; motions for summary judgment, directed verdict, or judgment n.o.v.; motions to exclude physicians or pharmacists as defendants; and findings concerning the merits of the plaintiff's case.

Drawn on the basis of information from Table 7, Figure 4 presents two indicators that illustrate how the Bendectin litigation matured over time. For each year between 1982 and 1990 it indicates the percentage of trial court opinions dealing with substantive issues and the percentage of opinions that were "final" in the sense that, unless overturned on appeal, the case was to end with that decision. In the early years of litigation, nonsubstantive issues tend to dominate case congregations. In the Bendectin cases, a substantial number of procedural issues arose because of the consolidation of cases in the Common Issues Trial. Additionally, several courts faced venue issues raised by claims of foreign nationals. Presumably, the foreigners and their attorneys preferred to litigate in American courts because they assumed their damage recoveries would be larger here than abroad. After some earlier opinions allowing them a cause of action, the foreign claimants eventually were barred under the doctrine of forum non conveniens. Finally, there was the recurring

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303. The opinions are from thirty-six different cases and multidistrict rulings of the Joint Panel on Multi-District Litigation (labeled MDL-JPML). Note that two of the cases are consolidated actions (labeled MDL-Ohio and MDL-Michigan).

304. Table 7 is found in Appendix A.

305. Not every Bendectin opinion is included in Table 7. Memorandum opinions, denials or grants of certiorari, and the like are excluded. They are listed in appendices H-I. The table does not include opinions that are neither published nor reported on Westlaw or Lexis.

306. Note that, in the case of summary judgments, directed verdicts, and judgments n.o.v. (codes 4, 5, and 6), where possible I have given the basis of the motion as well. For example, in the fifth case, Koller, the motion for a summary judgment was based on a claim that the plaintiff could not show general causation.

307. A key opinion is Judge Rubin's decision that foreign nationals would be barred from the multidistrict litigation, but that, in return, Merrell would have to consent to being sued in Great Britain. In re Richardson-Merrell, Inc., 545 F. Supp. 1130, 1135 (S.D. Ohio 1982). Judge Rubin was particularly concerned that allowing foreign nationals to litigate in Ohio would flood the courts with litigants:
issue of federal jurisdiction. Merrell occasionally asked to have a court rule that the joinder of a local physician or pharmacy was done fraudulently solely in order to defeat diversity jurisdiction.308

In more recent years an increasing percentage of opinions have dealt with the cases on their merits.309 This progression from nonsubstantive
to substantive opinions is a key element of the maturation of the congregation of Bendectin cases. By 1987, most nonsubstantive legal questions had been resolved, and cases were being decided on the merits. The fact that fewer and fewer cases addressed procedural questions also signifies that relatively few new cases were entering the stream and that deple- 

tion was underway. Figure 4 indicates that as the percentage of substantive opinions rises in a case congregation, so too does the percentage of final dispositions.

Finally, an examination of the last three or four years of trial court opinions indicates that Merrell's motions for summary judgement, directed verdict, and judgment n.o.v. have increasingly been granted. This trend is, in large part, a response to appellate court opinions concerning Bendectin. It is to those appellate opinions that we now turn.

b. Appellate Court Opinions

I have found a total of thirty-six appellate opinions concerning Bendectin. This figure includes both published opinions and unpublished opinions available on the legal databases. The thirty-six opinions represent twenty-eight cases. Table 8 lists the opinions in chronological order. As one would expect, the volume of appellate opinions has lagged behind the trial opinions, peaking in 1988.

Most of these opinions did not follow a trial on the merits below, and those that did tend to have come relatively later in the series, again indicating the maturation of the Bendectin congregation. The early nontrial cases involved one of two issues: jurisdictional questions, or the inclusion of defendants other than Merrell. The jurisdictional questions usually involved foreign national plaintiffs hoping to find an American forum. As noted earlier, all eventually were excluded on forum non conveniens grounds. The non-Merrell defendant cases involved pharmacists or treating physicians against whom there was no specific claim except that they were in the chain of distribution.

The nontrial opinions of the last two or three years have been much more likely to involve substantive questions. They address questions

Ohio for discovery but then opted out of the Common Issues Trial, had been on hold awaiting the outcome of that case. They now were transferred back to their original jurisdictions and allowed to proceed.

310. From 1988 to the present, only two opinions have not involved motions for summary judgment, directed verdict, or judgment n.o.v.

311. Table 8 is found in Appendix A.

312. Again, memorandum opinions and the like have been excluded from the table.

313. See infra app. A, tbl. 8, col. 3.
such as whether the plaintiffs have shown specific causation,\textsuperscript{314} whether they have shown by a preponderance of the evidence that Bendectin causes defects, and whether they have presented a qualified expert on the causation issue. Since 1987, all but three\textsuperscript{315} of the opinions have been final dispositions, yet another sign of the maturation and impending old age of the Bendectin congregation.

Most of the nontrial opinions are relatively short and deal only with the specific issue under review. The opinions that followed full trials are substantially longer and often review the evidence presented at trial. These opinions reveal more fully the appellate courts’ reactions to the body of fact and law that has developed as the congregation of cases has matured. There are nine such opinions.\textsuperscript{316} Table \textsuperscript{9}\textsuperscript{317} presents information about these nine cases. One notable feature of these cases is that all except the Common Issues Trial involved a limb defect injury.\textsuperscript{318} While there are epidemiological studies that have found significant correlations between Bendectin and congenital heart defects, pyloric stenosis, and oral clefts, there appears to be no published epidemiological study that has found a statistically significant correlation between Bendectin ingestion and limb defects. Nevertheless, these cases have gone farthest through the appellate process, presumably because they involve the most serious injuries and the largest potential damage awards. Physically, psychologically, and perhaps financially, these tend to be the most devastating injuries for the child and his or her parents.\textsuperscript{319} For a contingency fee lawyer, they are the cases most worth pursuing, the cases whose expected value justifies the high costs involved in pursuing recovery.\textsuperscript{320}

\textsuperscript{314} A common example involved plaintiffs with limb deformities who could not show that Bendectin caused their particular injuries because the mother did not ingest the product during the critical period when limb buds were forming.

\textsuperscript{315} One of these three opinions, Merrell Dow Pharmaceuticals v. Oxendine, 593 A.2d 1023 (D.C. 1991), is not final in the sense that the appellate court has refused to allow the plaintiff to collect compensatory damages until her punitive damages claim has been settled. \textit{Id.} at 1030.

\textsuperscript{316} Most trials that have gone to verdict have produced at least one appellate opinion. As far as I can determine, only three of the American trials have not led to an appeal, each having ended in a defense judgment, by either jury verdict, directed verdict, or judgment n.o.v.

\textsuperscript{317} Table 9 is found in Appendix A.

\textsuperscript{318} \textit{See infra} app. A, tbl. 9, at col. 2.

\textsuperscript{319} There is something especially horrific about a visible limb reduction defect that in some ways sets it apart from other defects. The financial burden created by such defects varies depending on the seriousness of the injury. For seriously deformed children the cost of care can be quite high. For those less seriously injured, however, the out of pocket financial cost may be less than it is for some other injuries, such as heart defects.

\textsuperscript{320} \textit{See} Coffee, \textit{supra} note 212, at 881.
The more recent trials, those that occurred in 1987-1988, resulted in plaintiffs' verdicts. As noted earlier, however, as of this writing only the Oxendine verdict has survived the entire appellate process. In several cases, the trial judge directed the verdict or entered judgment n.o.v. Most remarkably, in recent cases appellate courts have reversed jury verdicts on which the trial judge has entered judgment. Over time, the appellate courts have taken a very aggressive posture toward the Bendectin Cases.

The following discussion examines this trend within the context of a case congregation analysis.

All Bendectin cases pose the same general causation issue. Viewing the litigation from the congregation perspective, one would expect that over time the courts would come to a more complete understanding of the facts of the cases. A question to be asked about case congregations is whether, at some point, courts are prepared to act on that understanding and make substantive determinations in individual cases based upon knowledge drawn from the congregation as a whole. With respect to Bendectin, the answer is yes.

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321. See supra note 230 and accompanying text.

322. This assertion, especially as it refers to substantive rationing, applies most clearly to appellate opinions following jury verdicts. When ruling on summary judgment motions, appellate courts have exhibited much greater reluctance to cut off plaintiffs' claims based merely on the courts' understanding of the Bendectin science. For example, in DeLuca v. Merrell Dow Pharmaceuticals, Inc., 911 F.2d 941 (3d Cir. 1990), a scientifically sophisticated opinion, the court reversed a summary judgment in favor of Merrell that was based on the trial court's conclusion that the evidence offered by plaintiff's only expert was inadmissible under Federal Rule of Evidence 703 because it was not reasonably relied on by experts in the relevant field. The appellate court objected to the sketchy record upon which this ruling was based:

Following Merrell Dow's lead, the district court did not point to specific deficiencies in the data utilized by Dr. Done and while it cited Rule 703, it made no record-supported, factual finding that Dr. Done had relied upon data experts in the field would have considered unreliable . . . . In only two brief sentences of its opinion did the district court address Dr. Done's statistical analysis of the available epidemiological evidence . . . .

. . . . [I]ts cursory ruling that Done's testimony was inadequate under Rule 703 does not comply with the standard set forth in Japanese [Electronics] Products Litigation [723 F.2d 238 (3d Cir. 1983)], as it was not predicated upon a record-supported, factual finding that Done relied upon identified data not regarded as reliable by experts in the field.

Id. at 944, 953.

Nonetheless, the court noted in conclusion:

[Even if Dr. Done's epidemiological analysis is found to be admissible, the DeLucas are entitled to get to trial only if the district court is satisfied that this analysis together with any other evidence relevant to the causation issue would permit a jury finding that Amy's birth defects were, when measured against the appropriate burden of proof, caused by her mother's exposure to Bendectin.

Id. at 959.
I have analyzed the way each appellate opinion approaches the evidence on Bendectin. In Table 9 each opinion is coded as exhibiting a legalistic, scientific, or legal-scientific approach to the pharmacological and epidemiological evidence presented at trial. An opinion is coded legalistic if it: (1) reviews the jury verdict and trial judge determinations using a set of legal formulas concerning admissible evidence, the burden of proof, judicial discretion, and the allocation of tasks between judge and jury; and (2) does not look behind the statements of qualified experts to assess the merits of the experts' opinions. An opinion is coded scientific when a court adopts an aggressive, proactive posture toward the evidence and the experts who presented it. These scientific opinions exhibit, to varying degrees, three characteristics: (1) the court places weights on the various types of evidence \textit{(in vitro, in vivo, or epidemiological)}; (2) the court examines and makes a substantive assessment of the evidence upon which an expert or set of experts have based their opinions; and (3) as a part of this assessment the court emphasizes formal scientific criteria of probative value, especially peer review and publication in refereed journals.\footnote{323} 

As the Bendectin congregation has worked its way through the legal system, opinions have become more scientific,\footnote{324} and the courts have

\footnote{323. There is a deeper sense in which an opinion can be "scientific." There is a fundamental difference between the process of explanation engaged in by scientists and the process of attribution. \textit{See} H.L.A. HART \& TONY HONORE, \textit{CAUSATION IN THE LAW} 68-83 (2d ed. 1985). As Hamilton notes, the search for a causal explanation involves an inquiry into the cause of an unexpected or puzzling effect. The outcome is a causal inference, and the key processing rule is a covariation principle. The process of attribution, on the other hand, involves a determination as to whether some effect can be assigned to a cause such that the cause is liable for sanctions. The outcome is a responsibility judgment and the key processing rule is whether someone "could have done otherwise." V. Lee Hamilton, \textit{Intuitive Psychologist or Intuitive Lawyer? Alternative Models of the Attribution Process}, 39 J. PERSONALITY \& SOC. PSYCHOL. 767, 768 (1980). The opinions I have coded "scientific" are not fully scientific in this deeper sense. They still are about the business of attributing responsibility and they sometimes blur the explanation-attribution distinction. It is fair to say, however, that the opinions I have coded "scientific" are in fact more scientific in the explanation sense than are most appellate opinions. Scientists, presumably, will feel comfortable with these opinions. Within the legal community, however, they seem to create a sense of unease.}

\footnote{324. More specifically, appellate opinions have become more scientific when they involve a jury verdict for the \textit{plaintiff}. In cases where the jury found for the defense, the courts have issued relatively legalistic opinions rejecting a number of plaintiffs arguments as to reversible error at trial. One such case is Wilson v. Merrell Dow Pharmaceuticals, 893 F.2d 1149 (10th Cir. 1990). Even in Wilson, however, the court's position was not purely legalistic. In responding to the plaintiff's contention that the trial court erred in denying their motion for judgment n.o.v. or a new trial, the court said: Merrell Dow presented expert testimony, which was not contradicted by the Wilsons' experts, that of the approximately forty epidemiological studies of Bendectin, none has shown a statistically significant association between ingestion of the drug}
shown less willingness to leave factual determinations to the jury. Finally, with the development of a body of cases assessing the merits of the evidence, at least one appellate court (and two trial courts ruling on summary judgment motions) have reverted to a legalistic analysis, but a legalistic analysis with a twist. In these cases, the courts did not review or assess the evidence. They simply accepted the earlier appellate assessment of the evidence as a matter of law. I have coded these opinions legal-scientific. The following passages demonstrate each of the three types of opinion.

(i) Oxendine

The clearest example of a legalistic appellate opinion is Oxendine. The court in Oxendine reversed the trial judge, who had granted judgment n.o.v. and ordered a new trial on the ground that the jury’s verdict for the plaintiff was against the great weight of the evidence. The appellate court relied heavily on Ferebee v. Chevron Chemical Co., a case involving the question whether paraquat exposure causes pulmonary fibrosis. It favorably quoted the following passage from that opinion:

Judges, both trial and appellate, have no special competence to resolve the complex and refractory causal issues raised by the attempt to link low level exposure to toxic chemicals with human disease. On questions such as these, which stand at the frontier of current medical and epidemiological inquiry, if experts are willing to testify that such a link exists, it is for the jury to decide whether to credit such testimony.

and incidence of birth defects generally or limb defects in particular. This lack of epidemiological proof for the Wilsons’ claims is particularly significant in light of recent decisions of federal courts of appeals granting judgment n.o.v. for Merrell Dow based upon the absence of epidemiological evidence showing a causal relationship between Bendectin use and birth defects . . . .

Although the Wilsons called several experts who testified in support of their claims, Merrell Dow presented at least sufficient expert testimony to create a conflict in the evidence, and perhaps even enough to sustain a directed verdict under the reasoning of Brock, Richardson, and Lynch.

Id. at 1154-55 (citations omitted).

325. Randall Terrell originally drew this pattern to my attention, and I am strongly indebted to his insight.


328. 736 F.2d 1529 (D.C. Cir. 1984).

329. Id. at 1534, quoted in Oxendine, 506 A.2d at 1104.
The Oxendine court concluded its review of the trial with the following observation:

Although the trial in this case was long and the evidence complex, the issue before the jury was a straightforward one: did Bendectin cause appellant's birth defects? Expert witnesses testified at length on both sides of that issue. Not surprisingly, their testimony revealed a disagreement as to how the epidemiological and other data should be interpreted. "The case was thus a classic battle of the experts, a battle in which the jury must decide the victor."

The Oxendine opinion, standing as it does at the beginning of the Bendectin congregation, is a clear example of a legalistic opinion. As the congregation matured, more scientific opinions began to emerge.

(ii) Richardson

If the Common Issues Trial is the most important trial in the Bendectin Cases, the most important appellate opinion is Richardson v. Richardson-Merrell. Unlike Oxendine, in Richardson the court affirmed a trial court's grant of judgment n.o.v. As in all the opinions coded "scientific," the Richardson court exhibited an unwillingness to accept expert opinion without examining its scientific underpinnings.

330. Oxendine, 506 A.2d at 1110 (quoting Ferebee, 736 F.2d at 1535).
331. 857 F.2d 823 (D.C. Cir. 1988).
332. Judge Jackson granted judgment n.o.v. on the basis of Merrell's epidemiological evidence. He concluded, "the literature on Bendectin, individually and in the aggregate, fails to demonstrate Bendectin's teratogenicity to a scientifically acceptable degree of accuracy." Richardson v. Richardson-Merrell, 649 F. Supp. 799, 802 (D. Mass. 1986).
333. An early appellate opinion to take a scientific approach to the data is Lynch v. Merrell-Nat'l Lab., 830 F.2d 1190 (1st Cir. 1987). Although Lynch did not follow a trial—the trial judge had granted Merrell's motion for summary judgment—it did contain a lengthy discussion of the data. Typical of the opinion was the court's discussion of the proffered testimony of Dr. Shanna Swan:

The plaintiffs also offered the opinion evidence of Shanna Helen Swan, a holder of a 1963 doctorate in statistics from the University of California at Berkeley. Swan had served from 1969 to 1975 as senior biostatistician in a Kaiser Health contraceptive drug study; been associate professor of mathematics at California State University, Sonoma, from 1974 to 1979; directed between 1979 and 1981 the training program in biostatistics and epidemiology at the School of Public Health of the University of California, Berkeley; and, while remaining in this school, has been since 1981 the chief of the Methodology and Analysis Unit, Epidemiology and Statistics, Department of Health Services of the State of California.

Swan's opinion as to causation was based upon her reanalysis of data collected between 1970 and 1978 by the Metropolitan Atlanta Congenital Defects Program. This data had been previously analyzed by four members of the Center for Disease Control . . . . [The researchers found "weak" but not causal relationship between the use of Bendectin and amniotic bands and concluded that the study "excluded" a risk of limb reduction from the use of Bendectin.

Swan testified in the multi-district litigation in Cincinnati, and the district court here took note of her testimony as it was excerpted by the defendant . . . . Compar-
The question whether Bendectin causes limb reduction defects is scientific in nature, and it is to the scientific community that the law must look for the answer. For this reason, expert witnesses are indispensable in a case such as this. But that is not to say that the court’s hands are inextricably tied, or that it must accept uncritically any sort of opinion espoused by an expert merely because his credentials render him qualified to testify ....

Next, the court distinguished among various types of evidence. In vitro and in vivo evidence was relegated to no more than a secondary role when compared to epidemiological studies:

These three types of studies then—chemical, in vitro, and in vivo—cannot furnish a sufficient foundation for a conclusion that Bendectin caused the birth defects at issue in this case. Studies of this kind, singly or in combination, are not capable of proving causation in human beings in the face of the overwhelming body of contradictory epidemiological evidence. Perhaps mindful of this, the last type of evidence considered by Dr. Done consisted of the epidemiological studies. When such studies are available and relevant, and particularly when they are numerous and span a significant period of time, they assume a very important role in determinations of questions of causation.

Third, the court judged the probative value of evidence, in part by the rules science would use to make such a judgment—whether the results have been published in a refereed journal and whether they have been subjected to peer review: “Only by recalculating the data was Dr. Done able to obtain what he deems a statistically significant result. Moreover, the studies rejected by Dr. Done had been published in peer-reviewed scientific journals, while Dr. Done has neither published his recalculations nor offered them for peer review.”

Informing the court’s approach to the evidence was its own recognition that Richardson did not stand alone, but rather was part of a congregation of cases. On this basis, the court rejected its own previous analysis in Ferebee of the proper posture of courts to conflicting scientific evidence:

... Swan’s study has never been refereed or published in a scientific journal or elsewhere. We are informed of it only by the defendant’s excerpts. On the basis of what we have, it could not form the foundation for an expert opinion challenging the scientific consensus and making the issue of causation a factual question to be decided by the jury.

Id. at 1194-95 (citation omitted).

334. Richardson, 857 F.2d at 829.
335. Id. at 830.
336. Id. at 831.
The Richardsons, however, direct attention to our decision in Ferebee v. Chevron Chemical Co. We think reliance thereon is misplaced here. Ferebee stands for the proposition that courts should be very reluctant to alter a jury's verdict when the causation issue is novel and "stand[s] at the frontier of current medical and epidemiological inquiry." If experts are willing to testify to causation in such situations and their methodology is sound, the jury's verdict should not be disturbed.

The case before us, however, is not like Ferebee. Indeed, we are at the other end of the spectrum, a great distance from the "frontier of current medical and epidemiological inquiry." And far from a paucity of scientific information on the oft-asserted claim of causal relationship of Bendectin and birth defects, the drug has been extensively studied and a wealth of published epidemiological data has been amassed, none of which has concluded that the drug is teratogenic. Uniquely to this case, the law now has the benefit of twenty years of scientific study, and the published results must be given their just due.

In spite of this language, Richardson might be thought of as an early scientific opinion. The court was not fully confident of its separate analysis of the evidence. It presented a significant excerpt from the testimony of Dr. Done, one of the plaintiffs' key experts, designed to demonstrate that even if the court were to accept plaintiffs' expert testimony at face value, the plaintiffs' experts themselves were unable to conclude that Bendectin more likely than not causes birth defects:

Dr. Done himself conceded the import of this literature when he agreed on cross-examination that "in connection with the animal studies... because so many substances are teratogenic in animals, before you can make a conclusion that a substance is teratogenic in humans, you must look to the human data." Dr. Done also acknowledged the necessity of a statistically significant association between the drug and its effect in human populations.

Dr. Done further admitted that no one who has published work on Bendectin has concluded that there is a statistically significant association between Bendectin and limb reduction defects of the type at issue in this case.

Based on this analysis of Dr. Done's testimony the court of appeals concluded that his opinion concerning the teratogenicity of Bendectin lacked an adequate foundation and was therefore inadmissable.

(iii) Brock

The court in Brock v. Merrell-Dow Pharmaceuticals exhibited no such uncertainty about the reallocation of tasks between judge and jury:

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337. Id. at 831-32 (footnotes omitted) (emphasis in original).
338. Id. at 830-31.
339. Id. at 831-32.
340. 874 F.2d 307 (5th Cir.), modified, 884 F.2d 166 (5th Cir. 1989).
This case is one of a series of many cases filed against Merrell-Dow by parents of children with birth defects allegedly caused by the ingestion of Bendectin during pregnancy. Academic commentators have dubbed this case and others like it "mass toxic torts." This represents a growing realization among academics, lawyers, and judges that cases such as this present special problems and challenges to traditional ideas regarding the role of the jury as a decision maker.

Under the traditional approach to scientific evidence, courts would not peer beneath the reasoning of medical experts to question their reasoning. Confronted, as we now are, with difficult medical questions, courts must critically evaluate the reasoning process by which the experts connect data to their conclusions in order for courts to consistently and rationally resolve the disputes before them.\[341\]

As in Richardson, the court was particularly concerned with the fact that Brock was one of a long string of cases. The Brock court, however, added a further justification for a scientific approach to the data. It argued that courts should be more willing to intervene because inconsistent jury verdicts are particularly undesirable in situations where the same facts are tried over and over:

Moreover, in mass torts the same issue is often presented over and over to juries in different cases, and the juries often split both ways on the issue. The effect of this is to create a state of uncertainty among manufacturers contemplating the research and development of new, and potentially lifesaving drugs. Appellate courts, if they take the lead in resolving those questions upon which juries will go both ways, can reduce some of the uncertainty which can tend to produce a sub-optimal amount of new drug development.\[342\]

(iv) Ealy

Once a body of cases assessing the merits of the evidence has developed, courts may adopt a legalistic analysis that no longer reviews evidence, but accepts earlier appellate assessments of the evidence. The quotation from Junius at the beginning of this essay captures the essence of this phenomenon: "What yesterday was fact, today is doctrine."\[343\]

Ealy v. Richardson-Merrell,\[344\] is a case in this tradition. In Ealy, as in Brock, the appellate court took the particularly aggressive step of reversing a trial court judgment entered on a plaintiff's verdict. Unlike Brock, however, the Ealy court justified this action not by directly attacking the

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341. Id. at 309-10 (citations omitted).
342. Id. at 310.
343. DEDICATION TO THE ENGLISH NATION, supra note 1, reprinted in FAMILIAR QUOTATIONS at 1001.
plaintiff's experts or their evidence but by legalistically arguing that it was following the earlier *Richardson* opinion:

We find that this case is squarely within the binding rule articulated in *Richardson*: an expert opinion that Bendectin is a human teratogen which caused the plaintiff's birth defects is without scientific foundation under Federal Rule of Evidence 703 in the face of "a wealth of published epidemiological data" to the contrary. Accordingly such expert opinion is inadmissible. Because we discern no material difference between the evidence presented in *Richardson* and that presented in this case, we reverse the trial court's denial of the motion for judgment n.o.v.

Because *Richardson* provides a binding legal precedent governing the admissibility of expert opinion on the ability of Bendectin to cause human birth defects, the Ealys can only avoid that decision by showing that the record here is materially different from that in *Richardson*. We find no such difference.345

Although the language of the opinion appears to hold out the potential that the plaintiffs might present new or additional data to avoid the precedential effect of *Richardson*, as a practical matter this is impossible. No *in vitro* or *in vivo* study will overcome the body of existing epidemiological evidence. And since Bendectin has not been on the market since 1983, there appear to be no significant data sets remaining that could cast new light on its teratogenic effects. As a matter of law, Bendectin does not cause birth defects.

c. A Defense Bias?

These Bendectin opinions followed in the wake of and were clearly influenced by Judge Weinstein's similarly "scientific" finding for the defense in the *Agent Orange* opt-out opinion.346 The *Brock* case divided the Fifth Circuit along apparently political lines.347 In light of these two circumstances, a natural question is whether what I have called substan-
tive rationing is part of a general drift within the federal judiciary toward a more conservative, prodefendant position. Such a trend may of course play a role in these opinions, but there is reason to believe that the use of substantive rationing is more than a partisan phenomenon. Within the Bendectin Cases, not all the judges issuing scientific or legal-scientific opinions are known conservatives or Republican appointees. The Ealy opinion, for example, was authored by Judge Mikva, who at one time was a liberal Democratic congressman from Illinois.

More to the point, perhaps, is the question whether similar decisions will be issued, but favoring plaintiffs rather than defendants. There are reasons to think that such an outcome is unlikely. From the plaintiffs' point of view, the Bendectin Cases are an example of an unsuccessful mass tort congregation. The plaintiffs have routinely met with defeat, but because of the large number of separate actions the litigation has continued for several years. "Successful" mass tort cases will usually have a different career. In such cases it is frequently the case that by the time the courts have come to a conclusion as to the proper substantive outcome of the congregation, the defendant has been overwhelmed by the litigation. Bankruptcy is a different form of legal rationing—one that supersedes both procedural and substantive judicial rationing. Nevertheless, if substantive rationing is the product of the forces described in this Article, we should expect that on occasion it may be used to the plaintiffs' advantage.

In this regard it is worth noting a recent effort on the part of two federal district court judges from Ohio and Texas to consolidate all asbestos cases. Among other actions taken, the Ohio judge issued an order setting a hearing date for September 1990 in Cleveland to allow the parties to nearly 60,000 outstanding asbestos claims to address the proposed

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348. See generally Henderson & Eisenberg, supra note 2.

349. One example, perhaps, is Beshada v. Johns-Manville Prods. Corp., 90 N.J. 191, 447 A.2d 539 (1982), in which the New Jersey Supreme Court ruled that the defendant could not raise a state-of-the-art defense in an asbestos products liability case. The New Jersey court was quickly forced to retreat from this position. See Feldman v. Lederle Lab., 97 N.J. 429, 479 A.2d 374 (1984). In distinguishing Beshada, the court in Feldman stated that, "[t]he rationale of Beshada is not applicable to this case. We do not overrule Beshada, but restrict Beshada to the circumstances giving rise to its holding." Id. at 455, 479 A.2d at 388. One possible interpretation of this statement is that Beshada came after a substantial amount of asbestos litigation in which the defendants had tried and failed to make a state-of-the-art defense. By denying the defendants this line of defense in asbestos cases, the court was engaged in substantive rationing. Such rationing was inappropriate in cases such as Feldman that raised a relatively new factual question: whether defendants could have known that large doses of tetracycline could discolor the teeth of infants and young children. I wish to thank Michael Green for pointing out the relevance of Beshada to the argument in the text.
certification of a class in the United States District Court for the Eastern District of Texas, which would enable the court to:

Declare and apply federal common law that establishes as a matter of law that asbestos-containing insulation products capable of producing dust on use, application, or removal are inherently dangerous in accordance with the governmental ban and the findings of all federal agencies that have addressed the question; and that such products were marketed without an adequate warning and were therefore, defective and unreasonably dangerous.\(^{350}\)

The order, issued after a meeting in August 1990 at the Federal Judicial Center, was reviewed and approved by eight other federal judges, including Judge Weinstein who is overseeing the Manville Personal Injury Settlement Trust.\(^{351}\) This effort would have settled all but the specific causation and damages questions in almost all asbestos litigation. This is additional evidence that the pressures created by a congregation of cases, and not party bias, are the primary forces moving courts toward substantive rationing.

VI. The Larger Context

Thus far, the argument has focused on the specifics of the Bendectin Cases. In the previous section I discussed a particularly interesting judicial reaction to a case congregation: substantive rationing. In this last section I move beyond the Bendectin Cases to discuss more general circumstances that produce this type of judicial behavior. I conclude with some thoughts on how the judicial response to case congregations may, in turn, affect all tort law.

A. Substantive Rationing and the Judge-Jury Relationship

Substantive rationing as evidenced in the Bendectin Cases, and perhaps soon to be seen in the asbestos cases, is the ultimate form of what Galanter calls an outcome stabilization effect.\(^{352}\) The effect, however, occurs in a way unanticipated by either Galanter's congregation theory or McGovern's cyclical theory of mass torts.\(^{353}\) In their models something like market forces operate over time to bring stability to the caseload and to case outcomes. The uniform outcome imposed through the use of substantive rationing is different.\(^{354}\) In large part, this type of outcome stabilization seems to be a reaction to the jury trial as an institution.


\(^{351}\) Id. at 361.

\(^{352}\) Galanter, Case Congregations, supra note 8, at 389-90.

\(^{353}\) See McGovern, supra note 20, at 482.

\(^{354}\) In Calabresi's terms, it is more like specific deterrence—you cannot bring this type of
Substantive rationing, especially in tort law, reflects distrust of juries and the desire to have clear rules governing the legal consequences of certain facts. It shrinks, as much as possible, the grey area of tort encompassed by the phrase "a mixed question of law and fact." The effort is the judicial equivalent of negligence per se rules, but in the Bendectin Cases the rule is one of no negligence per se. The Bendectin appellate judges who have thus restricted the jury's role are heirs to Holmes' view of the jury and the judge-jury relationship. In an 1899 article Holmes commented:

I confess that in my experience I have not found juries specially inspired for the discovery of truth. I have not noticed that they could see further into things or from a saner judgment than a sensible and well trained judge. I have not found them freer from prejudice than an ordinary judge would be . . . . I do not believe that the jury have any historic or a priori right to decide any standard of conduct.

And in The Common Law, he expressed his hope regarding the ultimate allocation of duties between judge and jury:

When a case arises in which the standard of conduct, pure and simple, is submitted to the jury, the explanation is plain. It is that the court, not entertaining any clear views of public policy applicable to the matter, derives the rule to be applied from daily experience, as it has been agreed that the great body of the law of tort has been derived. But the court further feels that it is not itself possessed of sufficient practical experience to lay down the rule intelligently. It conceives that twelve men taken from the practical part of the community can aid its judgment. Therefore it aids its conscience by taking the opinion of the jury.

But supposing a state of facts often repeated in practice, is it to be imagined that the court is to go on leaving the standard to the jury forever?

The answer for Holmes was no. In the well known case of Baltimore & Ohio Railroad v. Goodman he had an opportunity to turn his thoughts into law in the context of a railroad crossing case:

When a man goes upon a railroad track he knows that he goes to a place where he will be killed if a train comes upon him before he is clear of the track . . . . In such circumstances it seems to us that if a driver cannot be sure otherwise whether a train is dangerously near he must stop and get out of his vehicle . . . . It is true . . . that the question

lawsuit—than general deterrence—given a set of general legal rules, you are unlikely to win this suit if you do bring it. GUIDO CALABRESI, THE COSTS OF ACCIDENTS 68-130 (1970).

355. The following discussion is based upon materials in GEORGE CHRISTIE, CASES AND MATERIALS ON THE LAW OF TORTS 158-64 (1983).


357. OLIVER WENDELL HOLMES, JR., LAW IN SCIENCE AND SCIENCE IN LAW, 12 HARV. L. REV. 443, 459 (1899).

358. 275 U.S. 66 (1927).
of due care very generally is left to the jury. But we are dealing with a standard of conduct, and when the standard is clear it should be laid down once for all by the Courts.\footnote{359}

As students of tort law know, Holmes' effort to ration substantive law failed. The "once for all" rule of stop, look, and listen lasted less than a decade, dismantled by Benjamin Cardozo in \textit{Pokora v. Wabash Railway Co.}\footnote{360} In a roughly similar case on the facts, Justice Cardozo declared that

Standards of prudent conduct are declared at times by courts, but they are taken from the facts of life. To get out of a vehicle and reconnoitre is an uncommon precaution, as everyday experience informs us. Besides being uncommon, it is very likely to be futile, and sometimes even dangerous. If the driver leaves his vehicle when he nears a cut or curve, he will learn nothing by getting out about the perils that lurk beyond. By the time he regains his seat and sets his car in motion, the hidden train may be upon him.

Illustrations such as these bear witness to the need for caution in framing standards of behavior that amount to rules of law. The need is the more urgent when there is no background of experience out of which the standards have emerged\footnote{361}

Needless to say, Holmes and Cardozo were speaking of standards of care, while the Bendectin judges are speaking of the causal question. The Holmes-Cardozo exchange deals with a relatively more normative question, while the Bendectin courts are dealing with a relatively more objective one. They share, however, a concern with which facts can give rise to liability. Like Holmes, the judges in \textit{Richardson, Brock,} and \textit{Ealy} concluded that on certain given facts there is no room left within which the jury may operate. \textit{Pokora,} however, stands as a warning that in a common-law system facts constantly challenge rules. If Holmes had somehow hoped that eventually each fact situation would have its own pigeonhole and that eventually judges, having considered each, would determine whether it was a "liability" hole or a "nonliability" hole, \textit{Pokora} suggests that there are too many pigeonholes to make this a practicable enterprise for all of the law of torts.

The debate about the acceptability of jury verdicts and the judiciary's obligation to intervene continues. Most frequently, it has been debated in situations where the primary evidence of liability is based on laboratory research and epidemiological studies. Charles Nesson, for example, has argued that Judge Weinstein may have erred in taking the

\footnotesize{359. \textit{Id.} at 69-70 (citations omitted).}
\footnotesize{360. 292 U.S. 98 (1934).}
\footnotesize{361. \textit{Id.} at 104.}
Agent Orange opt out case from the jury solely on the basis of adverse epidemiological evidence. Others have disagreed with this conclusion and have argued that judicial intervention is essential to control verdicts that appear to depart from "rational efforts to reconstruct reality."

From a wider perspective the dispute is not about juries per se, but about discretionary fact finding by judge or jury. When judges sit as fact finders they too can arrive at outcomes that depart from rational efforts to reconstruct reality. Note, however, that Holmes couched his thoughts in terms of a learning process. The judge should determine the standard of care, or perhaps find the facts, when nearly identical situations have been confronted repeatedly. According to Holmes, the pressure to ration law in this way is especially strong when the court finds "the jury oscillating to and fro, and will see the necessity of making up its mind for itself." The threat of inconsistent outcomes is always a cost of allowing any case to go to the jury. As Nesson notes, "[t]he force of this consideration depends on one's assessment of the system's capacity to rationalize inconsistent verdicts in terms of credibility of witnesses, ability of lawyers, variations among juries, and similar considerations." Such rationalizations may prove to be most difficult where congregations of cases present the same factual question. Inconsistent verdicts by one-shot juries lacking the benefit of the learning process that a congregation of cases provides may seem offensive to a judiciary that, as a collective entity, has tried the same question many times. Therefore, we are especially likely to observe substantive rationing as a congregation matures. This is all the more true when the central factual question is

362. Charles Nesson, Agent Orange Meets the Blue Bus: Factfinding at the Frontier of Knowledge, 66 B.U. L. Rev. 521, 526, 537-58 (1986) [hereinafter Nesson, Factfinding at the Frontier]. In part, Nesson's concern arises from his distinction between verdicts based on "a determination of what actually happened" and verdicts that are only "statements about the evidence." Charles Nesson, The Evidence or the Event? On Judicial Proof and the Acceptability of Verdicts, 98 HARV. L. Rev. 1357, 1361 (1985). Under this view, outcomes based solely on epidemiological studies are statements about the evidence and ought not overcome jury verdicts that may be based on a determination of what actually happened.


364. HOLMES, supra note 357, at 123.

365. Nesson, Factfinding at the Frontier, supra note 362, at 537. See, e.g., Dempsey v. Addison Crane Co., 247 F. Supp. 584 (D.D.C. 1965). In Dempsey, two plaintiffs injured in the same crane accident had their nonjury cases tried separately and experienced opposite outcomes. In an attempt to explain this outcome, the court was happy to be able to report that "the evidence in the two actions was not the same." See id. at 589. In Bendectin cases tried since 1985, however, the evidence on the question of general causation has been basically the same.

366. Concern with inconsistent jury verdicts is part of the reason the trial court in Lynch v. Merrell-Nat'l Lab., 646 F. Supp. 856, 862 (D. Mass. 1986), aff'd, 830 F.2d 1190 (1st Cir.
one of general causation, and therefore, by definition, the identical issue is posed time after time. Substantive rationing of law is a response to being repeatedly presented with the same fact pattern and having to determine whether the correct outcome is liability or nonliability. As the following discussion illustrates, this phenomenon is not restricted to the law of torts.

B. Substantive Rationing in the School Desegregation Cases

In the several cases leading up to Brown v. Board of Education,\textsuperscript{367} the Supreme Court ruled in favor of the plaintiffs in large part on the basis of a factual determination that the separate schools being run by the state were not equal.\textsuperscript{368} In the four cases comprising Brown, the plaintiffs presented social science testimony designed to prove that separate schools caused psychological harm to black children.\textsuperscript{369} Chief Justice Warren responded to this evidence with the following statement: "Whatever may have been the extent of psychological knowledge at the time of Plessy v. Ferguson, this finding [that state mandated segregation has a tendency to retard the educational and mental development of black children] is amply supported by modern authority."\textsuperscript{370}

Defendant school boards, however, continued to attack this factual assertion, arguing that separate may not be worse, educationally or psychologically. In this congregation of cases, as in the Bendectin Cases, the challenge soon fell on deaf ears. Appellate courts refused to hear experts on the issue, rejecting the testimony as a factual attack on Brown.\textsuperscript{371} One expert for a school district made the following comment in the face of this substantive rationing by the courts:

The legal doctrine is cast in concrete, and that's been one of my frustrations. It's as though the evidence is really immaterial . . . . I remember in one case I was talking with the judge from the witness box, and questioning some of the testimony in Brown. He asked me, "Are

\textsuperscript{367} 347 U.S. 483 (1954).


\textsuperscript{370} Brown, 347 U.S. at 494.

you questioning the facts of Brown?” And I said “Yes,” and he said, “Well, that’s not admissible for you to be doing that.”

Again, of course, the parallel should not be overdrawn. Ultimately, in the school desegregation cases courts simply did not care whether segregated schools psychologically harmed or benefitted minority students. Presumably, in the Bendectin Cases the courts still care whether the drug causes birth defects. But in both situations, the rationing response of the courts must be understood, in part, as a natural consequence of the fact that they were adjudicating cases that came relatively late in a congregation of similar cases. In the school desegregation cases and in the Bendectin Cases, substantive rationing is the ultimate rationing weapon.

VII. Conclusion: Case Congregations and the Interaction of Macro and Micro Processes

This Article began by noting Henderson and Eisenberg’s assertion that all of products liability law is changing in a prodefendant direction and that the Bendectin opinions are an example of this process. Their data indicate that the single most important component in increased defense success in products liability cases is an increased ability to win on pretrial motion, a type of judicial rationing. Henderson and Eisenberg explicitly leave for a later article their thoughts as to why this “quiet revolution” is occurring. Implicit in their discussion, however, is the argument that large scale, macro changes are occurring within the law of torts, causing the judiciary to adopt a more prodefendant position.

This Article’s micro level analysis, which attempts to explain case outcomes in terms of the internal dynamics of the Bendectin Cases as a congregation, can be viewed as a rival hypothesis to the thesis that the change in the courts’ attitude toward Bendectin suits is to be understood as part of a larger judicial retreat from proplaintiff positions. From this perspective, the future research agenda should include efforts to determine whether what we have observed in the Bendectin Cases is merely a cohort effect (a difference to be found between all products liability cases decided in 1985 and all such cases decided in 1990), as opposed to a history effect (evidencing the maturation of the Bendectin congregation). In this sense, macro and micro explanations may be seen as rival hypotheses for the findings.

372. Chesler et al., supra note 36, at 430.
373. Henderson & Eisenberg, supra note 2, at 491.
374. Id. at 531, 548.
375. Id. at 482.
There is, however, another more useful perspective, one that integrates rather than divides. Macro research on large scale patterns of change that excludes an analysis of the micro processes that generate those patterns often leads to incomplete, disembodied theories of legal change.\textsuperscript{376} Theories of micro processes enrich our theories of larger processes by placing them within the context of individual human action.\textsuperscript{377}

From this latter perspective, the present Article suggests that the large-scale change reported by Henderson and Eisenberg can be understood, at least in part, as a product of the growth of mass torts and the micro processes generated within individual congregations of cases. That mass torts have changed the law is now beyond doubt. As noted earlier, they have raised a new set of causal conundrums and forced us to engage in a new round of theorizing about the causal question.\textsuperscript{378} They have raised new issues with respect to damages.\textsuperscript{379} By causing growth in some parts of the tort caseload, they have helped precipitate another round of legislative "tort reform."\textsuperscript{380} They have reinvigorated the search for alternatives to the traditional tort system.\textsuperscript{381} This Article suggests that they may also have influenced the judiciary's approach to tort law by creating incentives for both procedural and substantive rationing.\textsuperscript{382}

The Bendictin Cases alone would not have such an effect, but mass torts in general have become the primary growth area of tort law, constituting an increasingly larger part of the total caseload.\textsuperscript{383} Their importance in terms of the judicial time they consume is even greater than is suggested by their percentage of the overall caseload. Each mass tort is a

\textsuperscript{376} Sanders, \textit{supra} note 46, at 241.
\textsuperscript{377} JAMES COLEMAN, FOUNDATIONS OF SOCIAL THEORY 18-23 (1990).
\textsuperscript{378} See Rosenberg, \textit{supra} note 215, at 855-59.
\textsuperscript{379} MARK PETERSON ET AL., PUNITIVE DAMAGES: EMPIRICAL FINDINGS 5-31 (1987).
\textsuperscript{380} Sanders & Joyce, \textit{supra} note 89, at 212.
\textsuperscript{382} The Fifth Circuit, home of Brock, has perhaps been most aggressive in applying substantive rationing to cases that are not part of a congregation. See, e.g., Christophersen v. Allied Signal Corp., 902 F.2d 362 (5th Cir. 1990), aff'd en banc, 939 F.2d 1106 (5th Cir. 1991). In Christophersen, the Fifth Circuit affirmed the district court's grant of summary judgment for the defendant against a claim that the plaintiff had not been adequately warned of the cancer causing potential of chemicals and other materials used in the manufacture of nickel-cadmium batteries. The district court had granted summary judgment after reviewing the qualifications of plaintiff's sole expert witness and the factual basis of that expert's opinion on causation. \textit{Id.} at 364.
congregation, subject to similar pressures to ration legal resources. Moreover, there are types of cases rarely thought of as mass torts that exhibit some, or even most, of the features of mass torts. Specifically, many products liability design defect cases, another growth area of tort law, present the courts with the same problems: repetitive trial of similar facts; substantial consumption of time; and the likelihood of inconsistent jury verdicts. These problems are part of the congregational effects that caused "scientific" opinions like Richardson, Brock, and Ealy to be written. In the Bendectin Cases, the problems have driven the judiciary to play an even more active role in rationing law than suggested by Galanter's congregation theory. Outcome stabilization has come not through the market forces of litigation but through the central planning of the federal judiciary.

In sum, this Article confirms the insights of Emerson and Galanter, that the analysis of litigation from a perspective other than that of the individual, atomized case offers us a new and better understanding of case processing and case outcomes. The Bendectin congregation does exhibit many of the features hypothesized by Galanter, and thereby demonstrates the value of this line of analysis. But it holds out the potential to do even more. The analysis of this and other congregations may provide a new window into the way in which, over time, individual decisions of lawyers and judges create fundamental shifts in the common law.


385. Design defect cases appear to have risen over time as a percentage of all products liability cases. Compare 3 INTERAGENCY TASK FORCE ON PRODUCT LIABILITY, U.S. DEPT OF COMMERCE, PRODUCT LIABILITY: FINAL REPORT OF THE LEGAL STUDY 84-85 (1977) (reporting that 39% of all products liability cases were design defect cases) with ALLIANCE OF AM. INSURERS, A STUDY OF LARGE PRODUCT LIABILITY CLAIMS CLOSED IN 1985 (1985) (finding that defective design was the applicable theory in 75% of the cases).

386. Products liability warning defect cases can also result in the repetitive trial of similar facts, with the attendant potential for inconsistent verdicts. In fact, some of the most important mass tort case congregations, such as asbestos and the polio vaccine, are litigated as warning defect cases. For a critique of this approach, see James A. Henderson & Aaron Twerski, Doctrinal Collapse in Products Liability: The Empty Shell of Failure to Warn, 65 N.Y.U. L. REV. 265 (1990). With respect to these and other warning cases, Henderson and Twerski "urge that judges engage more aggressively in both lawmaking and law-applying." Id. at 326.
### Table 1

**Bendectin Animal Studies**

<table>
<thead>
<tr>
<th>Author</th>
<th>Year</th>
<th>Animal</th>
<th>Exp/Con</th>
<th>Drug</th>
<th>Effect?</th>
<th>Defect Type</th>
<th>Dose</th>
<th>Dose Resp</th>
<th>Auth Conc</th>
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<tbody>
<tr>
<td>Gibson</td>
<td>1968</td>
<td>Rabbits</td>
<td>36/13</td>
<td>Do</td>
<td>No</td>
<td>—</td>
<td>(100)</td>
<td>No</td>
<td>No</td>
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<tr>
<td></td>
<td></td>
<td>Rabbits</td>
<td>38/14</td>
<td>B3</td>
<td>Yes</td>
<td>Othera</td>
<td>6</td>
<td>No</td>
<td>No</td>
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<tr>
<td></td>
<td></td>
<td>Rats</td>
<td>88/39</td>
<td>Do</td>
<td>No</td>
<td>—</td>
<td>(100)</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Rats</td>
<td>90/30</td>
<td>Di</td>
<td>Yes</td>
<td>Otherb</td>
<td>10</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Rabbits</td>
<td>36/24</td>
<td>B3</td>
<td>No</td>
<td>—</td>
<td>(30)</td>
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<td>Khera</td>
<td>1975</td>
<td>Rats</td>
<td>22/10</td>
<td>Py</td>
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<td>—</td>
<td>(80)</td>
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<tr>
<td>Gibson</td>
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<td>Rats</td>
<td>45/15</td>
<td>B2</td>
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<td>45/15</td>
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<tr>
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<td>Chicken</td>
<td>252/96</td>
<td>Di</td>
<td>Yes</td>
<td>Otherb</td>
<td>—</td>
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<td>Yes</td>
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<td></td>
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<td>1982</td>
<td>Monkeys</td>
<td>12/0</td>
<td>B2</td>
<td>Yes</td>
<td>Heart</td>
<td>7</td>
<td>?</td>
<td>Maybe</td>
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<td>Hendrickx</td>
<td>1983</td>
<td>Monkeys</td>
<td>20/0</td>
<td>B2</td>
<td>Yes</td>
<td>Heart</td>
<td>7</td>
<td>?</td>
<td>Maybe</td>
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<td>McBride</td>
<td>1984</td>
<td>Rabbits</td>
<td>48/8</td>
<td>Do</td>
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<td>Limb, Skeletal</td>
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<td>74/12</td>
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<td>69/21</td>
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<td>No</td>
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<td>(13)</td>
<td>No</td>
<td>No</td>
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<td>Tyl</td>
<td>1988</td>
<td>Rats</td>
<td>116/53</td>
<td>B2</td>
<td>Yes</td>
<td>Skeletal Death</td>
<td>200</td>
<td>Yes</td>
<td>No</td>
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</tbody>
</table>

Information Reported (by column number):

1. Year in which the research was published.
2. Animals studied: white rats, rabbits, chicken eggs, or monkeys.
3. Number of mothers in the experimental and control groups.
4. Drug tested:
   - Di = dicyclomine hydrochloride
   - Do = doxylamine succinate
   - Py = pyridoxine hydrochloride
   - B2 = two ingredient Bendectin (doxylamine succinate and pyridoxine hydrochloride)
   - B3 = three ingredient Bendectin.
5. Did the use of the drug appear to be related to some teratogenic effect?
6. Type of effect: heart, limb, skeletal, death, other (see below).
7. Minimum dose at which effect was observed. Dosages are expressed in mg/kg/day except in study 3. Where there was no observed effect this column reports (in parentheses) the maximum dose rate in the experiment. For purposes of comparison, the therapeutic dose for pregnant women was 2 to 4 tablets a day, each tablet containing 10mg of each ingredient. The dose for a woman weighing 60kg (132 lbs.) taking 4 tablets per day is .67mg/kg/day per Bendectin ingredient. Unlike the rest of the studies, Study 5—the 1981 McBride Study—involved injecting dicyclomine hydrochloride (or a saline solution for the controls) into Leghorn chicken eggs.
8. Was there a dose-response relationship?
9. Author's conclusion as to whether the drug tested has teratogenic effects?

**Other Effects (Column 6):**

- a. Contracted tendons.
- b. Focal hematomas.
- c. Kidney pelvic dilation, subcutaneous hematomas.
- d. Gastroschisis, deformed foot, micromelia, exencephaly.
Table 2
Bendectin Epidemiological Studies

<table>
<thead>
<tr>
<th>First Author</th>
<th>Year</th>
<th>Type of Study(^{a})</th>
<th>Focus on Bendectin</th>
<th>Level of Stat. Anal.(^{b})</th>
<th>Author's conclus.</th>
<th>Data Base(^{c})</th>
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<td>(2)</td>
<td>(3)</td>
<td>(4)</td>
<td>(5)</td>
<td>(6)</td>
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<td>1. Le Vann</td>
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<td>Cohort</td>
<td>No</td>
<td>1</td>
<td>No</td>
<td>24</td>
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<tr>
<td>2. Bunde</td>
<td>1963</td>
<td>Cohort</td>
<td>Yes</td>
<td>1</td>
<td>No</td>
<td>9</td>
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<tr>
<td>3. Warneke</td>
<td>1963</td>
<td>Other</td>
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<td>1</td>
<td>No</td>
<td>27</td>
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<td>4. GPRG</td>
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<td>Cohort</td>
<td>No</td>
<td>2</td>
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<tr>
<td>5. Klee</td>
<td>1964</td>
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<td>1</td>
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<td>6. Farrar</td>
<td>1964</td>
<td>Case</td>
<td>No</td>
<td>2</td>
<td>No</td>
<td>23</td>
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<td>7. Yerushalmy</td>
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<td>2</td>
<td>No</td>
<td>6</td>
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<td>8. Banister</td>
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<td>No</td>
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<td>9. Nelson</td>
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<tr>
<td>11. Newman</td>
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<td>Cohort</td>
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<td>1</td>
<td>No</td>
<td>20</td>
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<tr>
<td>12. Shapiro</td>
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<td>Cohort</td>
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<td>5</td>
<td>No</td>
<td>1</td>
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<tr>
<td>13. Greenberg</td>
<td>1977</td>
<td>Case</td>
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<td>No</td>
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<td>14. Smith</td>
<td>1977</td>
<td>Case</td>
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<td>1</td>
<td>No</td>
<td>25</td>
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<td>15. Smithells</td>
<td>1978</td>
<td>Cohort</td>
<td>Yes</td>
<td>1</td>
<td>No</td>
<td>17</td>
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<td>16. Rothman</td>
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<td>Case</td>
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<td>3</td>
<td>Maybe</td>
<td>4</td>
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<td>17. Michaels</td>
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<td>4</td>
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<td>18. Harron</td>
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<td>1</td>
<td>No</td>
<td>12</td>
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<td>19. Jick</td>
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<td>3</td>
<td>No</td>
<td>7</td>
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<td>20. Fleming</td>
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<td>Cohort</td>
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<td>3</td>
<td>No</td>
<td>13</td>
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<td>21. Clarke</td>
<td>1981</td>
<td>Case</td>
<td>Yes</td>
<td>3</td>
<td>No</td>
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<td>22. Correy</td>
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<td>Cohort</td>
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<td>1</td>
<td>No</td>
<td>20</td>
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<td>23. Cordero</td>
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<td>Case</td>
<td>Yes</td>
<td>4</td>
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<tr>
<td>24. Mitchell</td>
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<td>Case</td>
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<td>3</td>
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<td>5</td>
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<td>27. David</td>
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<td>2</td>
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<td>29. Golding</td>
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<td>4</td>
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<td>30. Aselton</td>
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<td>32. Mitchell</td>
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<td>34. McCredie</td>
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<td>39. Erickson</td>
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a. Type of Study (Column 2):
- Case = Case-Control study
- Cohort = Cohort study
- Other = A study that is neither of the above.

b. Levels of Statistical Analysis (Column 4):
- 1 = None
- 2 = Chi-square only
- 3 = Chi square and confidence interval
- 4 = Chi-square, confidence interval and something additional but no regression analysis
- 5 = 3 or 4 plus a multiple regression analysis to control for co-variants

c. For an explanation of databases, see infra app. E

January 1992] MASS TORTS 395
Table 3
Whether or Not Studies Focus on Bendectin by Whether They Are Published Before 1980 or Later

<table>
<thead>
<tr>
<th>Focus on Bendectin?</th>
<th>Published Before 1980</th>
<th>Published 1980 or Later</th>
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<tr>
<td>No</td>
<td>11 (69%)</td>
<td>4 (17%)</td>
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<tr>
<td>Yes</td>
<td>5 (31%)</td>
<td>19 (83%)</td>
</tr>
<tr>
<td>Total</td>
<td>16 (100%)</td>
<td>23 (100%)</td>
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$X^2 = 10.52, \ df = 1, \ p < .01, \ N = 39$

Table 4
Whether or Not Studies Report More Than a Chi-Square by Whether They Are Published Before 1980 or Later

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<th>Report More Than a Chi-Square?</th>
<th>Published Before 1980</th>
<th>Published 1980 or Later</th>
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<td>3 (13%)</td>
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<tr>
<td>Yes</td>
<td>3 (19%)</td>
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</tr>
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<td>Total</td>
<td>16 (100%)</td>
<td>23 (100%)</td>
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$X^2 = 18.14, \ df = 1, \ p < .001, \ N = 39.$

Table 5
Bendectin Related Cases Filed in State and Federal Courts, 1977-1988

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<th>Year</th>
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Table 6

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## Table 7

Trial Court Cases on Bendectin

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Issues (Column 4):

(0) = Other
(1) = Forum non conveniens
(2) = Federal jurisdiction
(3) = Complaint fails to state a cause of action (Note—this might be one of several causes of action.)
(4) = Summary Judgment
(5) = Directed Verdict
(6) = JNOV/New Trial
(7) = Dismissal of pharmacist or physician defendant
(8) = Statute of Limitations
(9) = Specific causation—timing of ingestion
(10) = Failure to prove general causation by a preponderance of the evidence
(11) = Failure to present any qualified expert at trial on the general causation question, summary judgment (this is case 22,30)
(12) = Motion for bifurcation, trifurcation
(13) = Collateral Estoppel
(14) = Transfer of cases in and out of consolidated litigation
(15) = Multiple

NOTE: Codes written like 4(10) indicate the type of ruling and the reason why that ruling was made: thus, in the example, a summary judgment was granted or denied based on an argument that the plaintiffs had failed to prove general causation.

Particularities of Posture (Column 4 superscript annotations):

a. Request to remove to Ohio, location of Merrell.
b. declares U.S. courts forum non conveniens for British claimants and others who survived a similar motion in Alexander (1).
c. Discovery allowed from Merrell companies abroad.
d. Class certification.
e. Lead counsel asking for money for discovery materials.
f. Plaintiffs move to return to state court because Defendant’s petition for removal was tardy.
g. Suit by military personnel against government barred.
h. Plaintiff moves for offensive collateral estoppel on causation question/ loses; Defendant pharmacy moves to dismiss/ wins; Defendant Merrell moves for summary judgment on punitive damages and fraud count/ wins.
i. Defense motion to allow statistical evidence in only if it is statistically significant (.05%) granted.
j. Quashed deposition.
k. Motion by Plaintiff to examine notes of a juror after verdict for Defendant.
l. Plaintiff’s motion to remand to state court granted. Defendant claims Plaintiff added pharmacies to destroy complete diversity, and Defendant argues this was done without hope of recovery against them.
m. Mother’s derivate action time barred.
n. Defendant makes 8 pretrial motions: (1) Exclusion of Thalidomide—denied with caution; (2) inadmissibility of early Jinks’ epidemiology studies later repudiated by Jinks in print in JAMA—denied with caution; (3) warning on UNISOM (which has doxylamine) not to be used by pregnant women—denied; (4) admissibility of published epidemiological studies—denied in advance of testimony showing their relevance and probativeness; (5) exclusion of other Bendectin related birth defects—denied; (6) exclusion of DERS and in vitro studies—sustained as to DERS not rat studies; (7) exclusion of deposition on Bunde-Bowles, sustained as it isn’t used by Defendant; and (8) exclusion of evidence on the cessation of bendectin production—sustained.
o. Objection to way jury was selected.
p. Motion to reconsider denial of new trial. Court now allows Plaintiff to photocopy jury notes and examine them further.
q. Motion by Defendant for summary judgment on punitive damages and fraud claim.
r. Defense motion to discover mother’s medical records—sustained.
s. No evidence mother ever took Bendectin—25 years before filing suit.
t. Defendant asks for summary judgment—sustained as to punitive damages, but not as to causation.
u. Motion to reconsider summary judgment rejected.
v. This opinion granted defendant a summary judgment in Daubert and Schuller v. Merrell Dow.

January 1992 | MASS TORTS 399
# Table 8
## Appellate Cases on Bendectin

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<td>Martin</td>
<td>1988</td>
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<td>No</td>
<td>8</td>
<td>P</td>
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<td>Brock</td>
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<td>1989</td>
<td>Yes</td>
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<td>Blum</td>
<td>1989</td>
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<td>Oxendine</td>
<td>1989</td>
<td>No</td>
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<td>D</td>
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<td>Vines</td>
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<td>P</td>
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<td>Wilson</td>
<td>1990</td>
<td>Yes</td>
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<td>Bernhardt</td>
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<td>Herring</td>
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<td>9</td>
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<td>Obiago</td>
<td>1990</td>
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<td>6 &amp; 8</td>
<td>P</td>
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<td>DeLuca</td>
<td>1990</td>
<td>No</td>
<td>8</td>
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<td>Coyle</td>
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<td>Ambrosini</td>
<td>1991</td>
<td>No</td>
<td>7 &amp; 8</td>
<td>P</td>
<td>D</td>
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<td>Oxendine</td>
<td>1991</td>
<td>Yes</td>
<td>9</td>
<td>D</td>
<td>D</td>
<td>No</td>
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</table>

Issue on Appeal (Column 4):

1. = Forum non conveniens
2. = Federal jurisdiction
3. = Complaint fails to state a cause of action. (Note—this might be one of several causes of action.)
4. = Dismissal of pharmacist or physician defendant
5. = Statute of Limitations
6. = Specific causation—timing of ingestion
7. = Failure to prove general causation by a preponderance of the evidence
8. = Failure to present any qualified expert at trial on the general causation question, summary judgment (this is case 22,30)
9. = Other
## Table 9

**Appellate Cases Reviewing a Jury Verdict Below**

(Numbers in parentheses are the number of the case in Table 8)

<table>
<thead>
<tr>
<th>Year</th>
<th>Type of Injury</th>
<th>Jury Verdict</th>
<th>Trial Judge</th>
<th>App. Ct. Holding</th>
<th>App. Ct. Orientation</th>
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<tbody>
<tr>
<td>(1)</td>
<td>(1) 1983</td>
<td>Limb Defect</td>
<td>Plaintiff</td>
<td>New Trial</td>
<td>Affirm</td>
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<tr>
<td>(9)</td>
<td>1986</td>
<td>Limb Defect</td>
<td>Plaintiff</td>
<td>JNOV</td>
<td>Reversed &amp; Remanded</td>
</tr>
<tr>
<td>(20)</td>
<td>1988*</td>
<td>Various</td>
<td>Defense</td>
<td>Entered Judgment</td>
<td>Affirmed</td>
</tr>
<tr>
<td>(21)</td>
<td>1988*</td>
<td>Limb Defect</td>
<td>Plaintiff</td>
<td>JNOV</td>
<td>Affirmed</td>
</tr>
<tr>
<td>(23)</td>
<td>1989*</td>
<td>Limb Defect</td>
<td>Plaintiff</td>
<td>Entered Judgment</td>
<td>Reversed</td>
</tr>
<tr>
<td>(28)</td>
<td>1990*</td>
<td>Limb Defect</td>
<td>Defense</td>
<td>Entered Judgment</td>
<td>Affirmed</td>
</tr>
<tr>
<td>(30)</td>
<td>1990*</td>
<td>Limb Defect</td>
<td>Plaintiff</td>
<td>Entered Judgment</td>
<td>Reversed Remanded</td>
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</table>

* = After MDL
Appendix B

In Vitro Studies Bibliography


Appendix C

In Vivo Studies Bibliography

(Numbers in parentheses at the end of each citation refer to the order of the studies in Table 1)

J.P. Gibson et al., Teratology and Reproduction Studies with an Antinauseant, 13 TERATOLOGY & APPLIED PHARMACOLOGY 439 (1968). (1)


John Gibson, Teratology Study With a New Antinauseant Formulation in Rats, Project Report T-75-13, Dep't of Pathology and Toxicology, Merrell-Nat'l Lab. Div. of Richardson-Merrell, Inc. Cincinnati, Ohio. (1975) (unpublished study) (on file with author). (3)


A.G. Hendrickx et al., Cardiac Embryotoxicity Studies on Bendectin in Macaques (Abstract), 27 TERATOLOGY 49 (1983). (7)

K.S. Khera, Teratogenicity Study in Rats Given High Doses of Pyridoxine (Vitamin B6) During Organogenesis, 31 EXPERIĘNTIA 469 (1975). (2)


Appendix D
Epidemiological Studies Bibliography

(Numbers in parentheses at the end of each citation refer to the order of the studies in Table 2)

Carl Bunde & D.M. Bowles, A Technique for Controlled Survey of Case Records, 5 CURRENT THERAPEUTIC RES. 245 (1963). (2)
Jose Cordero et al., Is Bendectin a Teratogen?, 245 JAMA 2307 (1981). (23)
T.J. David, Debendox Does Not Cause the Poland Anomaly, 57 ARCH. DIS. CHILD 479 (1982). (27)
Diana Elbourne et al., Debendox Revisited, 92 BRIT. J. OBSTETRICS & GYNAECOLOGY 780 (1985). (36)
David J. Erickson, Risk Factors for Birth Defects: Data from the Atlanta Birth Defects Case-Control Study, 43 TERATOLOGY 41 (1991). (39)
Brenda Eskenazi & Michael Bracken, Bendectin (Debendox) As a Risk Factor for Pyloric Stenosis, 144 AM. J. OBSTETRICS & GYNECOLOGY 919 (1982). (26)
D.M. Fleming et al., Debendox in Early Pregnancy and Fetal Malformation, 283 BRIT. MED. J. 99 (1981). (20)


E. Klees, Early Pregnancy Toxicosis Treated with Lenotan, 44 MED. WELT 204 (1964). (5)


J. H. Michaelis et al., Prospective Study of Suspected Associations Between Certain Drugs Administered During Early Pregnancy and Congenital Malformations, 27 TERATOLOGY 57 (1983). (31)

Lucille Milkovich & Bea van den Berg, An Evaluation of the Teratogenicity of Certain Antinauseant Drugs, 125 AM. J. OBSTETRICS & GYNECOLOGY 244 (1976). (10)


Suzette Morelock et al., Bendectin and Fetal Development: A Study at Boston City Hospital, 142 AM. J. OBSTETRICS & GYNECOLOGY 209 (1982). (28)


Kenneth Rothman et al., *Exogenous Hormones and Other Drug Exposures of Children with Congenital Heart Disease*, 109 Am. J. Epidemiology 433 (1979). (16)

Samuel Shapiro et al., *Antenatal Exposure to Doxylamine Succinate and Dicyclomine Hydrochloride (Bendectin) in Relation to Congenital Malformations, Perinatal Mortality Rate, Birth Weight, and Intelligence Quotient Score*, 128 Am. J. Obstetrics & Gynecology 480 (1977). (12)

Patricia Shiono & Mark Klebanoff, *Bendectin and Human Congenital Malformations*, 40 Teratology 151 (1989); Erratum 41 Teratology 250 (1990). (38)


Appendix E
Data Base List

The number preceding each data base corresponds with the number appearing in Column 6 of Table 2. The numbers in parentheses at the end of each data base reference refer to the number of the study using this database as it appears in Table 2. For example, data base number 1 was used by studies 12 (Shapiro) and 38 (Shiono).

United States

1. Collaborative Perinatal Project, 50,282 child pairs from 12 hospitals throughout the United States. (12), (38)
2. Boston City Hospital, 3222 eligible mothers. February 1977 through October 1979. (28)
3. Boston University School of Medicine and Harvard Medical School case control study. Drawn from 22 participating centers in 3 regions: (1) Boston (initiated in March 1976); (2) Pennsylvania (March 1977); and (3) Toronto (January 1979). 1565 malformed infants. (24), (32)
4. Harvard School of Health, Department of Epidemiology. Subjects chosen from all residents of Massachusetts born alive with severe congenital heart disease and with controls randomly selected from all available birth certificates filed with the Massachusetts Division of Health Statistics. (16) (between 1973 and 1985), (35) (between April 1, 1980 and March 31, 1983).
5. John B. Pierce Foundation Laboratory and the Departments of Epidemiology and Public Health and Obstetrics and Gynecology, Yale University School of Medicine. (Mothers of infants from 5 urban hospitals in central Connecticut—between November 18, 1974 and November 17, 1976). (26)
6. Northern California Kaiser Permanent Birth Defects Study (Patients from Kaiser Health Plan—also called Child Health and Developmental Studies (CHDS), 1974 to 1977. (7), (10)
7. Group Health Cooperative of Puget Sound (GHC-PS) (subjects were patients in Seattle). Data put on computer files by the Commission on Professional and Hospital Activities-Professional Activity Study (CPHA-PAS) in Michigan. (19), (30), (33), (37)
8. Metropolitan Atlanta Congenital Defects Program (MACDP) survey of birth defects "since 1967" from hospitals within the 5 central counties of the metropolitan Atlanta area. (23), (39)
9. Merrell Department of Medical Research. 18 groups (21 physicians) from California, Colorado, Illinois, Ohio, Pennsylvania, Ontario, and Quebec. (2)

United Kingdom

10. General Practitioners Research Group, patients of physician members in U.K. (4)
11. University of Edinburgh Department on Child Life and Health, Edinburgh Scotland. (9)
13. Birmingham Research Unit of the Royal College of General Practitioners, Birmingham. 22,977 women (14,684 in Scotland and 8293 in England), 620 of whom were Debendox exposed. (20)
15. The Office of Population Consensus & Surveys (OPCS) pilot study (London). (13)
16. University of Leicestershire. 40,000 perinatal births in Leicestershire. (21)
17. University of Leeds. Scan of RX records from Leeds and Liverpool. (15)
18. *Cardiff Births Survey (CBS),* (Cardiff and Aberdeen), 86,283 births between 1965 and 1979. (36)
19. Reported cases of Poland Anomaly in Britain between 1890 and 1977. (27)

Australia

20. University of Tasmania, Department of Obstetrics and Gynaecology. All deliveries in Tasmania. (11) (from 1953 to 1975), (22) (from 1975 to 1980).
21. Infants born alive in New South Wales or the Australian Capital Territory between 1970 and 1981. (34)
23. Sutherland District Hospital, Carinbaugh, Australia. Eleven mothers whose babies were abnormal and were born in September and October, 1961 and 98 similarly situated mothers with normal babies. (6)
Canada

24. University of Alberta. 33,874 births in Alberta in 1961. (1)
25. Pilot Surveillance System-Department of National Health and Welfare, Ottawa Ontario, Births in 4 Canadian Provinces (New Brunswick, Alberta, Manitoba, and British Columbia) were surveyed. (8), (14)

Germany

26. DFG Study, 21 hospitals in West Germany. (17), (31)
27. 94 women with emesis gravidarum (nausea in pregnancy). (3)

Not Certain

28. Klees Study, 30 cases of hypermises gravidum. (5)
Appendix F

District Court Opinions

(Numbers in parentheses at the end of each citation refer to the order of the cases in Table 7)


In re Richardson-Merrell, Inc. (MDL #486), 533 F. Supp. 489 (J.P.M.L. 1982). (2)
In re Richardson-Merrell, Inc. (MDL #486), 97 F.R.D. 481 (S.D. Ohio 1983). (6)
In re Richardson-Merrell, Inc. (MDL #486), 582 F. Supp. 890 (J.P.M.L. 1984). (7)
In re Richardson-Merrell, Inc. (MDL #486), 588 F. Supp. 1448 (J.P.M.L. 1984). (9)
In re Richardson-Merrell, Inc. (MDL #486), 606 F. Supp. 715 (J.P.M.L. 1985). (10)
In re Richardson-Merrell, Inc. (MDL #486), slip op. (S.D. Ohio Jan. 6, 1986) (LEXIS, Genfed library, Dist file). (14)


Appendix G
Appellate Court Cases

(Numbers in parentheses at the end of each citation refer to the order of the cases in Table 8)

Bernhardt v. Richardson-Merrell, Inc., 892 F.2d 440 (5th Cir. 1990). (29)
Brock v. Merrell Dow Pharmaceuticals, Inc., 884 F.2d 166 (5th Cir. 1989). (23b)
Brock v. Merrell Dow Pharmaceuticals, Inc., 884 F.2d 167 (5th Cir. 1989). (23c)
DeLuca v. Merrell Dow Pharmaceuticals, Inc., 911 F.2d 941 (3d Cir. 1990). (33)
Dowling v. Richardson-Merrell, Inc., 727 F.2d 608 (6th Cir. 1984). (3)

In re Bendectin Prods. Liab. Litig., 749 F.2d 300 (6th Cir. 1984). (5)


Lynch v. Merrell-Nat'l Lab., 830 F.2d 1190 (1st Cir. 1987). (15)


Martin v. Merrell Dow Pharmaceuticals, Inc., 851 F.2d 703 (3d Cir. 1988). (19)

Mekdeci v. Merrell Nat'l Lab., 711 F.2d 1510 (11th Cir. 1983). (1)


Merrell Dow Pharmaceuticals, Inc. v. Thompson, 478 U.S. 804 (1986). (10)

Urland v. Merrell Dow Pharmaceuticals, Inc., 822 F.2d 1268 (3d Cir. 1987). (14)


Wilson v. Merrell Dow Pharmaceuticals, Inc., 893 F.2d 1149 (10th Cir. 1990). (28)
Appendix H
Memorandum Opinions
(not included in data analysis)
Brock v. Merrell Dow Pharmaceuticals, Inc., 886 F.2d 1314 (5th Cir. 1989) (table disposition, reh'g en banc denied).
Appendix I

Unpublished Opinions
(not included in data analysis)

Summary Judgments Granted to Merrell Dow


Summary Judgments Denied Merrell Dow


Plaintiff Verdicts (Jury or Judge)


Defendant Verdicts (Jury or Judge)

Buell, (June 1988) (judgment for defendant on merits).
Francione, (March 1990) (Italy, judgment for defendant on merits).
Maryland Consolidated (Johnson, Kelley, Mangels), (Md. federal court, mistrial, cases dismissed by plaintiffs June 10, 1988).
Wiles, (Cal. federal court, Nov. 11, 1987) (defense verdict by judge).