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New scientific breakthroughs in personalized (also known as “precision”) medicine seem to occur almost weekly. Applications of “pharmacogenomics”—the study of how individualized variations in DNA amino acid sequencing can affect a person’s response to drugs—raise the prospect of understanding many of the myriad of suboptimal reactions to prescription drugs that are today classified as “idiosyncratic.” At the same time, advances in computer technology are making genetic testing exponentially cheaper and more comprehensive. Eventually, and within many of our lifetimes, it should be possible for people to carry their entire individual genome with them on a flash drive.

Progress in pharmacogenomics will undoubtedly affect product liability litigation involving prescription drugs. These effects are likely to provide both plaintiffs and defendants with new arguments, and new evidence, useful to their cases. While a great deal has been written about pharmacogenomics from a scientific standpoint, analysis of the legal implications is less robust. This article reviews the existing law on the intersection of pharmacogenomics, personalized medicine, and prescription medical product liability litigation.

Overview of Current FDA Regulation of Pharmacogenomic Information
The U.S. Food and Drug Administration (FDA) actively has encouraged the development of personalized medicine using analysis of individual genomic variation. In 2004 it launched a “Critical Path Initiative” intended to drive pharmacogenomic innovation. The first tangible result was in 2005, when the agency authorized regulated manufacturers of prescription drugs to add pharmacogenomic information to their labeling on a voluntary basis:

The pharmacogenomic data and resulting test or tests may be intended to be included in the drug labeling to choose a dose and dose schedule, to identify patients at risk, or to identify patient responders. Inclusion of a pharmacogenomic test in the labeling would be contingent upon its performance characteristics.

FDA, Guidance for Industry, Pharmacogenomic Data Submissions, at 5 (Mar. 2005). Under the heading “Clinical Pharmacology,” the FDA states that drug labeling is allowed to include a subsection specifically devoted to pharmacogenomics. Id. An FDA presentation from 2014 indicates that New Drug Application (NDA) holders for over one hundred drugs have taken advantage of...

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Although it permits pharmacogenomic labeling, the FDA has yet to issue regulation making such information mandatory. In recent years, the FDA has released several additional guidance documents relating to the effect of individualized genetic variability on the safety and effectiveness of prescription drugs. In 2007, it facilitated the development of medical devices for individualized genetic diagnosis. See FDA, Guidance for Industry, Pharmacogenetic Tests and Genetic Tests for Heritable Markers. In 2008, the agency standardized the terminology to be used in clinical trials involving pharmacogenomic research. See FDA, Guidance for Industry, E15 Definitions for Genomic Biomarkers, Pharmacogenomics, Pharmacogenetics, Genomic Data and Sample Coding Categories. In 2013, the FDA addressed the effect of genomic information on various stages of drug development and regulatory review. See FDA, Guidance for Industry, Clinical Pharmacogenomics: Premarket Evaluation in Early-Phase Clinical Studies and Recommendations for Labeling. In 2014, the agency issued guidelines to encourage the development of “drug development tools” for assessing the impact of pharmacogenomic variation in the development and evaluation of drugs. See FDA, Guidance for Industry, Qualification Process for Drug Development Tools. A wealth of additional information related to pharmacogenomics is available on the FDA website.

Attempts by Plaintiffs to Assert Pharmacogenomic Claims

Even though the FDA does not mandate including pharmacogenomic information in drug labeling, plaintiffs in pharmaceutical litigation have asserted claims for inadequate warnings or failure to conduct adequate testing with respect to alleged instances of genetic variation that adversely affect either the safety or effectiveness of prescription drugs. As pharmacogenomic information becomes more common and less expensive, such allegations can be expected with increasing frequency in the future. One commentator has described the “ideal” scenario for assertion of pharmacogenomic-based product liability litigation:

In the ideal plaintiff’s case, a person with an allele that made him or her specifically susceptible to the action of some toxin would be exposed to that toxin, which would cause a unique and detectable biochemical change, which in turn would be shown to cause an extremely high likelihood of contracting the plaintiff’s disease. The ideal defendant’s case might occur in several ways: similar biomarker evidence would point a finger at a purely genetic cause or at some other (perhaps voluntary or nonanthropogenic) exposure; or, a person exposed to a toxin known to cause the person’s disease in susceptible people might have a gene that completely neutralized the toxic effect and also might lack a biomarker that is uniformly found in people whose disease was caused by exposure.


Already some plaintiffs have attempted to argue that genetic markers should be warned about, or in some cases, designed around. To date, most of these claims have failed due to lack of scientific support—something that will change with continued scientific and technological advances in this field. One such case is Newman v. McNeil Consumer Healthcare, 2013 WL 9936293 (N.D. Ill. March 29, 2013). Newman involved SJS/TEN, the autoimmune diseases (or different forms of the same disease), Stevens-Johnson syndrome and toxic epidermal necrosis. SJS/TEN is sufficiently serious that plaintiffs often work backwards from the diagnosis, resulting in SJS/TEN suits against a plethora of different drugs. Perhaps to bolster causation testimony that is often weak in SJS/TEN cases, in Newman the plaintiff’s expert sought to opine about pharmacogenomics. His testimony about purported genetic predisposition to SJS/TEN was rejected as speculative because no genetic link to SJS/TEN has yet been discovered:

Defendants are correct that [the expert] testimony on the subject would be speculative and irrelevant. First of all, Plaintiffs’ argument admits that the relevance and helpfulness of the information is conditioned on the discovery of a genetic link, which may not happen. Secondly, even if such a link were discovered, Plaintiffs fail to explain how it rebutts Defendants claim that SJS/TEN was unpredictable during the relevant time frame.

Id. at *8. As of the time of trial, the state of the art did not include a genetic marker for SJS/TEN. But even so, as noted by the court, “[t]hat SJS/TEN may be more predictable in the future if a particular discovery is made says nothing about Defendants’ negligence.” Id. While the genetic testimony in Newman was offered by the plaintiff, should an SJS/TEN marker be discovered, the overall effect of such a development would likely help defendants far more than plaintiffs by exonerating most of the many drugs that plaintiffs have alleged as causative agents, or perhaps all, should the as-yet unknown marker prove to be not related to a drug.

Mills v. Bristol-Myers Squibb Co., 2011 WL 4708850 (D. Ariz. Oct. 7, 2011), similar to Newman, also involved baseless allegations that the plaintiff possibly carried an adverse genetic marker. Due to a variant “CYP” gene, the plaintiff claimed not to be able to metabolize the defendant’s drug as well as most other people. The drug.
was allegedly defectively designed for those persons who, similar to the plaintiff, were slow metabolizers:

Plaintiff alleges that the chemical structure of [the drug] is defective because it carries a higher risk of adverse events for patients who carry the genetic variant CYP, who are poor metabolizers of the drug. Plaintiff contends that [the drug] is the proximate cause of her injuries because, “[u]pon information and belief,” she is a CYP carrier.

Mills, 2011 WL 4708850, at *2. As in Newman, the court in Mills did not allow the plaintiff to continue. A plaintiff’s own genetic markers, and his or her genome generally, is something uniquely possessed by that person. The court remarked, “Plaintiff’s genetic makeup is a fact solely within her control. Tests are available that can reveal whether plaintiff in fact possesses CYP” Id. Thus, when a plaintiff asserts a claim based on something in his or her own genome, he or she is responsible for producing the evidence to prove it. Id.

Similarly unsupported allegations of “genetic predisposition” making the plaintiff a “poor metabolizer” of a drug contributed to the rejection of the plaintiff’s expert in Rimbert v. Eli Lilly & Co., 2009 WL 2208570 (D.N.M. July 21, 2009), aff’d, 647 F.3d 1247 (10th Cir. 2011).

Ultimately, almost everything in [the expert’s] specific causation opinion is hypothetical and speculative, except for her conclusion. She wrote that “it is hypothesized that the plaintiff is a ‘slow metabolizer’,” aff’d, 242 F. Appx. 512 (10th Cir. 2007); Trainer v. Secretary of Health & Human Services, 2013 WL 4505803, at *7 (Fed. Cl. July 24, 2013) (“[p]etitioner, however, fails to present any evidence indicating that he has a mitochondrial DNA mutation that would make him more susceptible”); Kolakowski v. Secretary of Health & Human Services, 2010 WL 5672753, at *43 (Fed. Cl. Nov. 23, 2010) (without any testing showing “genetic predisposition,” plaintiff could not prove claim).

These cases demonstrate that plaintiffs asserting pharmacogenomic claims still have a ways to go, but with continuing advances in genetic screening, inevitably some plaintiffs will be able to support claims of pharmacogenomic injury with the necessary evidence. When that happens, defense attorneys must be ready to argue that warning claims predicated on genetic markers and pharmacogenomic susceptibilities must not become so detailed that they amount to telling physicians how to practice medicine. E.g., Swayne v. McNeil Laboratories, Inc., 807 F.2d 1464, 472 (5th Cir. 1987) (“defendant cannot control the individual practices of the medical community…, and we decline to impose such a duty”); Kennedy v. Medtronic, Inc., 851 N.E.2d 778, 786 (Ill. App. 2006) (“unreasonable, and potentially harmful, to require a [defendant] to delay or prevent a medical procedure”); In re Meridia Products Liability Litigation, 328 F. Supp.2d 791, 814 (N.D. Ohio 2004) (“[t]he law does not mandate that pharmaceutical manufacturers and marketers provide such specific instructions that they leave little room for doctors’ reasonable medical judgment”), aff’d, 447 F.3d 861 (6th Cir. 2006).

Physical & Emotional Harm §31 (2010); Restatement (Second) of Torts §461 (1965). This principle has already cropped up in some cases involving genetic conditions. Most recently, in Vanslembrouck v. Halperin, 2014 WL 5462596 (Mich. App. Oct. 28, 2014) (unpublished), the defendants asserted a “genetic abnormality” as an alternative cause. Id. at *2. The plaintiffs sought, and received, an “eggshell” plaintiff charge, which the court held appropriate under the loose “clear error” standard, because the defendant made no objection. Id. at *58–59. The same sort of unobjected-to instruction was given in Rite Aid Corp. v. Levy-Gray, 876 A.2d 115, 140 (Md. App. 2005), aff’d, 894 A.2d 563 (Md. 2006). Defendants seeking to challenge such instructions in pharmacogenomic cases first need to preserve their objections.

The “vaccine court” has applied “eggshell” plaintiff principles to reject allegations that genetic predispositions were a superseding cause of vaccine-related injuries, but only “[s]o long as the [product] was a substantial factor” in the cause. Zeller v. Secretary of Health and Human Services, 2008 WL 3845155, at *26 (Fed. Cl. July 30, 2008). Respondent proferred evidence and arguments that [plaintiff’s] genetic predisposition was a superseding cause
of her injury, rendering irrelevant the vaccine as a substantial cause…. [I]f the administration of the vaccine(s) to [plaintiff] creates or increases the foreseeable risk of harm that preexisted and coexisted in her genetic predisposition…, and the vaccine is found to be a substantial factor in causing her injury, then the genetic predisposition can-

As time advances, pharmacogenomics and identification of genetic markers will become increasingly central to product liability and other litigation, while at the same time this testing also will become more feasible.

not constitute a superseding cause…. Applying the general rule from the common law of torts, compensation is appropriate even when the vaccine operates upon a concealed physical condition, such as a latent disease, or susceptibility to disease, to produce consequences incapable of reasonable anticipation.… As every aspiring attorney learns, a defendant takes a plaintiff as he finds him. Sucher v. Secretary of Health and Human Services, 2010 WL 1370627, at *43 (Fed. Cl. Mar. 15, 2010) (citations and quotation marks omitted). Accord Byers v. Secretary of Health and Human Services, 2010 WL 5663019, at *26 (Fed. Cl. Nov. 30, 2010) (same rationale).

The possibility of a latent or otherwise unknown genetic condition, however, does not affect either the standard of care or the causation requirement. Garcia v. U.S., 2010 WL 2977611, at *20 n. 10 (D. N.M. June 15, 2010) (discussing example of the “genetic disorder” of osteogenesis imperfecta).

Thus the underlying causation question remains, “eggshell” or not. The claimed genetic predisposition must still be a “substantial factor” in causing an injury before “eggshell plaintiff” principles can expand the scope of damages allegedly aggravated in a particular case.

Pharmacogenomic Claims Concerning Efficacy Rather Than Safety

Pharmacogenomics can identify not only genetic markers that increase the risk of adverse drug reactions, but also those that reduce a drug’s effectiveness when used by particular individuals. This latter situation—that a drug was allegedly ineffective due to a genetic variation—has not been considered a legitimate product liability claim since in such cases safety is not at issue and the plaintiff would have suffered the injury in any event. Several cases establishing this principle were decided in the Plavix multidistrict litigation. In the first, Solomon v. Bristol-Meyers Squibb Co., 916 F. Supp.2d 556 (D.N.J. 2013), the plaintiff claimed that he “should have been genetically tested to determine his genetic response” to the drug because it was ineffective in some people due to genetic variation. The court first found that the effectiveness evidence did not relate to this plaintiff’s particular facts. Id. at 566–67. But then the court went on to reject the concept of product liability claims based solely on lack of effectiveness:

[It] appears that Plaintiff’s efficacy arguments are not relevant in the context of a failure-to-warn analysis…. [A] drug manufacturer is required to provide an adequate warning of its product if it knows of any potential harm that may result from the use of its product. In other words, a proper warning should adequately alert any danger or harm that may result from ingesting the drug. Permitting Plaintiff to pursue his failure-to-warn claim on an efficacy theory would, as has been found in other jurisdictions with similar laws, impermissibly expand liability under Texas law on the adequacy of pharmaceutical warning labels. Id. at 564 (citations omitted) (emphasis original).

Other cases have rejected pure effectiveness or efficacy claims. LaBarre v. Bristol Myers Squibb Co., 544 F. Appx. 120, 125 (3d Cir. 2013) (“efficacy is not relevant to a failure to warn claim”; “duty does not extend to a warning about a drug’s efficacy”); Tobin v. Astra Pharmaceutical Products, Inc., 993 F.2d 528, 536 (6th Cir. 1993) (rejecting warning “argument… relating to the studies on efficacy”; efficacy only relevant to design claim); Needham v. White Laboratories, Inc., 639 F.2d 394, 402 (7th Cir. 1981) (where plaintiff alleged only that defendant “failed to warn, comment [k] could not apply…, and evidence of the efficacy, or inefficacy, of [the drug] was irrelevant”); In re Fosamax (Alendronate Sodium): Products Liability Litigation, 2014 WL 1266994, at *15 (D.N.J. March 26, 2014) (“omission of efficacy information does not constitute a failure to warn about a drug’s risks and therefore, does not raise a genuine issue of material fact”); Carr-Davis v. Bristol Myers-Squibb Co., 2013 WL 322616, at *6 (D.N.J. Jan. 28, 2013) (“studies based on the efficacy of [the drug]… fail to raise a genuine issue of material fact on the question of whether [its] warnings were adequate”); Begley v. Bristol-Myers Squibb Co., 2013 WL 144177, at *6 (D.N.J. Jan. 11, 2013) (“although the efficacy of a drug may play a role in a physician’s decision to prescribe, the failure-to-warn doctrine is not primarily concerned with a drug’s efficacy”), aff’d, 544 F. Appx. 120 (3d Cir. 2013); In re Fosamax Products Liability Litigation, 2010 WL 1257299, at *5 (S.D.N.Y. Mar. 26, 2010) (“[t] o allow Plaintiff to pursue a claim for the ‘failure to warn’ of the efficacy of a drug would be an expansion of liability”).

Thus, pharmacogenomic product liability claims focused on efficacy face serious hurdles, although such claims might also be asserted under other theories of liability not as dependent on personal injury as strict liability.

Pharmacogenomic Conditions Supporting Alternative Cause Defenses

Pharmacogenomics also has the potential to assist the defense of prescription medical product liability cases by establishing alternative cause. Many of the most thoughtful cases doing so to date arise from federal vaccine litigation. For example, an exhaustive discussion of the genetic underpinnings of autism is found in Snyder v. Secretary of Dept. of Health and Human Services, 2009
WL 332044, at *45–50 (Fed. Cl. Feb. 12, 2009). Ultimately, the vaccine court concluded, “The evidence for autism's genetic basis and prenatal origin renders petitioner’s [vaccine-related] theory of causation improbable.” Id. at *52. Similarly, in Simanski v. Secretary of Health and Human Services, 115 Fed. Cl. 407 (Fed. Cl. 2014), aff’d, 601 Fed. Appx. 982 (Fed. Cir. Feb. 26, 2015), an inherited genetic condition was advanced as an alternative non-vaccine-related cause for the injury being alleged. The plaintiffs’ causation arguments were rejected in part because they “have done very little to refute these conclusions, including allowing genetic testing” that would have confirmed or denied the alternative cause. Id. at 427. In another vaccine case, Waters v. Secretary of Health and Human Services, 2014 WL 300936 (Fed. Cl. Jan. 7, 2014), genetic testing actually had occurred, and it revealed an alternative genetic cause that precluded a finding of vaccine-related causation:

Petitioners have failed to show by a preponderance of the evidence that [minor plaintiff’s] current condition constitutes a significant aggravation of his condition prior to vaccination. He had the SCN1A mutation before his vaccinations, his clinical course developed consistent with that condition, and his current condition is a result of his genetic mutation.


Unsurprisingly, defense-side pharmacogenomics alternative cause evidence also needs to be supported adequately by both known facts and expert testimony. Otherwise, a defendant’s assertion of a genetically based alternative cause runs the risk of failing for essentially the same reasons as the plaintiffs’ in the cases discussed above. See In re Prempro Products Liability Litigation, 586 F.3d 547, 566 (8th Cir. 2009) (pointing out that the plaintiff “submitted to every available genetic test” without any positive result); Levy-Gray, supra, 876 A.2d at 139–40 (no evidence of genetic marker for alternative cause). However, the burden of proof also needs to be taken into account. In Roberti v. Andy’s Termite & Pest Control, Inc., 2011 WL 635369 (Cal. App. Feb. 23, 2011) (unpublished), a defense expert was allowed to give testimony that the plaintiff could well have a “genetic abnormality” responsible for his condition that had not yet been discovered:

[The expert’s] opinion was not speculative or devoid of foundation. He merely pointed out that the current state of medical science does not allow him to confirm, or to rule out, a genetic or chromosomal abnormality as the cause of plaintiff’s condition. It was a factually accurate statement to say that often birth defects are linked to genetic abnormalities, but that relatively little is known about identifying the precise genetic defect responsible for various conditions…. [H]e simply said that it was possible that a genetic cause was responsible. This was not speculation; it was a statement relevant to [the expert’s] opinion regarding causation based on his scientific knowledge. In addition, we note that the opinion was offered to refute plaintiff’s counsel’s attempt to definitively confirm that plaintiff did not have a genetic disorder…. Merely suggesting that the injury could have another cause that cannot be verified is hardly prejudicial or likely to mislead the jury. Id. at *13.

Occasionally, since defendants do not bear the burden of proving lack of causation, the undefined prospect of a pharmacogenomic alternative cause can be sufficient to allow a jury to consider it.

Using Pharmacogenomic Conditions to Defeat Medical Monitoring Claims

Genetic markers can also defeat claims for medical monitoring because they destroy any assumption that exposure to a claimed toxin will affect all persons equally. Such evidence has been particularly effective in beryllium litigation, although nothing excludes equally well-established pharmacogenomic variables from leading to similar results in prescription drug cases. In Sheridan v. NGK Metals Corp., 609 F.3d 239 (3d Cir. 2010), “exposure [to the defendant’s product] itself appear[ed] to be insufficient because only persons who have a particular genetic ‘marker’” went on to develop the medical condition at issue. Id. at 244. The evidence established that “only a small percentage of the population with the known genetic marker… is at risk of becoming sensitized.” Id. at 252. Since only that small percentage was at risk of eventually developing the disease for which monitoring was demanded, the classwide monitoring claim failed as a matter of law: “[P]laintiffs did not prove they were at a significantly increased risk of developing [the disease] and thus did not present sufficient evidence to make out a prima facie cause of action for medical monitoring.” Id. Accord Pohl v. NGK Metals Corp., 936 A.2d 43, 51 (Pa. Super. 2007) (affirming summary judgment against medical monitoring claim on similar facts; “[a]ppellants cannot show they are even susceptible…, because [such] susceptibility cannot be determined by a test”).

Although Sheridan and Pohl did not involve class certification, the same logic should apply in that context, since any risk that depends on the presence of genetic markers—by definition not common to the entire population—would require individualized evidence to establish the elements of medical monitoring for every purported class member. See Norwood v. Raytheon Co., 237 F.R.D. 581, 592 (W.D. Tex. 2006) (individual “genetic makeup” one of factors
cited in denying class certification in radiation case). Medical monitoring claims are usually not viable unless aggregated into class actions. In the future, pharmacogenomic causation defenses should become a major defense weapon in medical monitoring litigation.

Using Pharmacogenomic Conditions to Defeat Failure-to-Recall Claims

Another way that pharmacogenomics can assist defendants is in those relatively rare cases in which a plaintiff claims that the drug should have been recalled. Such claims are only infrequently allowed but did survive summary judgment in *Lance v. Wyeth*, 85 A.3d 434 (Pa. 2014). Lance, however, was based in significant part on Restatement (Third) of Torts, Products Liability §6(c) (1998), which Lance indicated “at the very least overlaps or intersects” with relevant state negligence law. 85 A.3d at 459 n.37. This section, however, only would apply when the risk/benefit ratio is such that “reasonable” physicians, aware of such risks, “would not prescribe the drug or medical device for any class of patients.” Id. (emphasis added).

Advances in genomics and individualized genetic markers should narrow even further this already small exception concerning prescription drug design since such markers will assist in identifying precisely those “classes of patients” most likely to be so benefitted, even if a drug poses risks to those with other genetic characteristics. In *Mills*, for example, the court rejected a §6(c) claim because “nowhere does plaintiff allege that [the drug] would not be prescribed for any class of patients” besides those allegedly carrying the adverse genetic marker. 2011 WL 4708850, at *3.

Using Pharmacogenomic Conditions to Support the Statute of Limitations

The fact that a plaintiff sought genetic testing can be relevant to the operation of the discovery rule when the timeliness of the suit under the applicable statute of limitations is at issue. See *D.D. v. Idant Laboratories*, 374 F. Appx. 319, 322–23 (3d Cir. 2010) (plaintiff deciding to undergo genetic testing showed sufficient awareness of possible external cause to satisfy discovery rule). As in *D.D.*, a plaintiff’s decision to undergo genetic testing, as well as the results of such testing, can demonstrate sufficient awareness of the possibility of an external cause, which in many jurisdictions ceases tolling of the statute of limitations under the discovery rule.

Pharmacogenomic Discovery

Finally, since pharmacogenomic data is relevant to product liability litigation for all of the above reasons, that relevance also makes such information discoverable. As pointed out in *Mills*, plaintiffs are the sole possessor of their individual genomes. Plaintiff-side experts have all too often discounted the entire field of genomics with a flippant statement that a plaintiff has “no known history” of genetic issues, without any affirmative investigation. E.g., *Junk v. Terminix Intern. Co. Ltd.*, 577 F. Supp.2d 1086, 1096 (S.D. Iowa 2008); *Colville v. Pharmacia & Upjohn Company LLC*, 565 F. Supp.2d 1314, 1319 (N.D. Fla. 2008).

Such excuses should not be tolerated in the future. As the cost of genetic screening plummets, if plaintiffs refuse to do their own genetic testing themselves, defendants should be able to fill that gap with court-ordered discovery. Each year, more genomic research identifies more genetic markers for drug and other reactions that were previously considered idiopathic. As time advances, pharmacogenomics and identification of genetic markers will become increasingly central to product liability and other litigation, while at the same time this testing also will become more feasible.

For these reasons, discovery of genetic information should become, for plaintiffs, what e-discovery has been for defendants—only without the exorbitant cost. In 10 years, it is likely that the submission of genetic samples by plaintiffs will be as commonplace in multidistrict litigation as the completion of a preliminary questionnaire is today. Cf. In re Welding Fume Products Liability Litigation, 2006 WL 2505891, at *1 (N.D. Ohio Aug. 28, 2006) (MDL discovery order requiring plaintiff’s counsel to search medical records for “known genetic or familial susceptibility” to the conditions at issue).

Discovery of genetic information has already been permitted for some time. In *Cruz v. Superior Court*, 17 Cal. Rptr. 3d 368, 369 (Cal. App. 2004), the court affirmed compelled genetic testing of the plaintiff mother in a birth defect case to determine if the injury was, in fact, a pre-existing genetic condition unrelated to the defendant. In *Bown v. E.I. DuPont de Nemours & Co.*, 2005 WL 1952859, at *5 (Del. Super. June 23, 2005), aff’d, 906 A.2d 787 (Del. 2006), the court relied upon compelled genetic testing to exclude as unreliable the plaintiff’s expert witness’s testimony attesting to a non-genetic cause. See also *Harris v. Mercy Hosp.*, 596 N.E.2d 160, 163 (Ill. App. 1992) (although “the blood test may not conclusively determine whether [plaintiff] has a genetic disorder, we conclude that the trial court did not abuse its discretion in ordering… the blood test since the probative value of this evidence is outweighed by the potential risk”); *Bennett v. Fieser*, 1994 WL 542089, at *2 (D. Kan. Feb. 25, 1994) (plaintiff ordered to provide blood sample under Fed. R. Civ. P. 35 for genetic testing); *Dodd-Anderson v. Stevens*, 1993 WL 273373, at *1 (D. Kan. May 4, 1993) (same).

That trend should only accelerate. Recalcitrant plaintiffs were ordered to undergo genetic testing in *Vanslembrouck*, 2014 WL 5462596, at *37 (noting trial court order requiring genetic testing). In *Cutting v. United States*, 2008 WL 5064267, at *1 (D. Colo. Nov. 24, 2008), the court ordered genetic testing of the plaintiff but held that Rule of Civil Procedure 35 did not extend to attempts to compel non-party relatives of the plaintiff to provide genetic samples. See *Kirk v. Scafeffer Group USA*, Inc., 2014 WL 2807681, at *3 (W.D. Mo. June 20, 2014) (requiring plaintiff seeking recovery for auto-immune condition to identify “medical providers who provided treatment… for autoimmune illnesses or disorders”).

Not all state court rules are limited to parties. In *Johnson v. Superior Court*, 95 Cal. Rptr. 2d 864, 875 (Cal. App. 2000), the court affirmed an order compelling a non-party witness—an anonymous sperm donor—to submit to considerable genetic-related discovery, albeit not outright testing.

There may be instances under which a child conceived by artificial insemination may need his or her family’s genetic and medical history for important medical decisions. For example, such genetic and medical history can lead to an early detection of certain diseases and an increased chance of curing...
them.… While in most situations the donor's genetic and medical information may be furnished without the need of disclosing the donor's identity, there may be other situations that require disclosure of the donor's identity in order to obtain the needed information.  

*Id.* at 875 (citations omitted).

Given the pace of medical progress since 2000, full genetic testing could well be proper under *Johnson* today. Moreover, if genetic discovery can be demanded from a third party who was promised anonymity, then *a fortiori*, it is proper from a plaintiff who initiated the very litigation to which that person's genetic information is relevant, thus waiving any privilege that might otherwise have existed.

Privacy rights, of course, must be respected, even when commencing litigation waives absolute privileges. Such concerns are addressable by protective orders, and “fishing expedition” arguments can be overcome by only looking for genetic markers already identified by science as pertinent to the claim to alternative causes. If possibly significant extraneous results are uncovered, plaintiffs’ counsel can be given the option to decide, on a client-by-client basis, whether a client would want to know or should be informed of such results. Compared to e-discovery, discovery concerning pharmacogenomics will be much easier, cheaper, and most importantly, more relevant.

**Conclusion**

Despite recent advances, the science of pharmacogenomics is undoubtedly still in its infancy. The landscape 20 years from now is likely to be as unrecognizable as today's landscape undoubtedly is to litigators of the last century. Defense attorneys may, however, find the relatively limited pharmacogenomic precedent discussed here useful in pointing the way to the coming ubiquity of this type of evidence in product liability litigation.