Blanchard v. Goodyear Tire and Rubber and Connecticut River Development Corp. (2010-250)

2011 VT 85

[Filed 05-Aug-2011]

ENTRY ORDER

2011 VT 85

SUPREME COURT DOCKET NO. 2010-250

MARCH TERM, 2011

Paul H. Blanchard	}	APPEALED FROM:
	}	
v.	}	Superior Court, Windsor Unit,
	}	Civil Division
	}	
Goodyear Tire & Rubber Company and Connecticut River Development Corporation	}	DOCKET NO. 837-12-07 Wrcv
	}	
	}	Trial Judge: Harold E. Eaton, Jr.

In the above-entitled cause, the Clerk will enter:

¶ 1. Plaintiff Paul Blanchard appeals the superior court's order granting summary judgment to defendants with respect to his toxic tort personal injury action. We affirm.

¶ 2. In 2005 at age forty-nine, plaintiff was diagnosed with a rare type of non-Hodgkin's lymphoma—Primary CNS (Central Nervous System) Large B-Cell Lymphoma. He attributes the onset of the disease to benzene exposure that allegedly occurred between 1968 and 1973 while he was a teenager playing on a ballfield on the grounds of the former Goodyear rubber manufacturing plant that operated in Windsor, Vermont from 1936 to 1986. In December 2007, plaintiff filed a personal injury action against the Goodyear Tire and Rubber Company and the Connecticut River Development Corporation (CRDC), the current owner of the property on which the plant was located, alleging that the field itself was polluted and that there was a gully in the outfield that transported foul-smelling and oily stormwater discharge away from the manufacturing plant. He claims that the discharge carried benzene from the plant through the field and that his exposure to the benzene caused his cancer. There is no evidence that Goodyear used benzene in the plant's manufacturing process, but the chemical is contained in petroleum products that were used at the plant.

¶ 3. In late 2009, Goodyear and CRDC filed their respective motions for summary judgment. Plaintiff opposed the motions, and a hearing was held in May 2010. Following the hearing, the superior court granted both motions and entered a final judgment in favor of defendants. The court concluded that plaintiff was not entitled to present his case to a jury because he had provided neither circumstantial evidence sufficient to support an inference that he had been exposed to benzene in any amount, let alone an amount that could have caused his illness, nor expert testimony sufficient to eliminate other potential causes of his disease. On appeal, plaintiff argues that his circumstantial evidence of causation was sufficient to present his case to the jury. Thus, what this appeal boils down to is whether the superior court erred by not allowing the case to go to trial for the jury's consideration.

¶ 4. This Court exercises de novo review on appeal from a grant of summary judgment, applying the same standard as that applied by the trial court. Field v. Costa, 2008 VT 75, ¶ 14, 184 Vt. 230, 958 A.2d 1164. We will uphold a grant of summary judgment when the pleadings and other pre-trial documentation are sufficient to show that no genuine issue of material fact

exists and the prevailing party is entitled to judgment as a matter of law. Id. In determining whether a genuine issue of material fact exists, the nonmoving party receives the benefit of all reasonable doubts and inferences. Messier v. Metropolitan Life Ins. Co., 154 Vt. 406, 409, 578 A.2d 98, 100 (1990).

¶ 5. We stress, however, that while plaintiff, as the nonmoving party, is entitled to all reasonable inferences regarding the state of the evidence, he cannot survive Goodyear's motion for summary judgment on his toxic tort claim unless he is able to point to evidence suggesting a probability, rather than a mere possibility, that (1) he was exposed to the specified chemical at a level that could have caused his physical condition (general causation); and (2) the exposure to that chemical did in fact result in the condition (specific causation). Golden v. CH2M Hill Hanford Group, Inc., 528 F.3d 681, 683 (9th Cir. 2008); see also White v. Dow Chem. Co., 321 F. App'x 266, 273 (4th Cir. 2009) (holding that proof of causation must suggest probability, not mere possibility, of exposure through evidence demonstrating amount, duration, intensity, and frequency of exposure); Bland v. Verizon Wireless, L.L.C., 538 F.3d 893, 898 (8th Cir. 2008) ("Critical to a determination of causation is characterizing exposure." (quotation omitted)). Indeed, "proof of causation must be such as to suggest 'probability' rather than mere 'possibility,' precisely to guard against raw speculation by the fact-finder." Sakaria v. Trans World Airlines, 8 F.3d 164, 172-73 (4th Cir. 1993).

¶ 6. "In a toxic tort case, general causation addresses whether a substance is capable of causing a particular injury or condition in a population, while specific causation addresses whether a substance caused a particular individual's injury." King v. Burlington Northern Santa Fe Ry. Co., 762 N.W.2d 24, 34 (Neb. 2009). As we recently explained in Estate of George v. Vermont League of Cities and Towns, epidemiological studies assess the existence and strength of associations between a suspected agent and a disease or condition and thus focus on general causation—whether the agent is capable of causing the disease—rather than specific causation—whether the agent actually caused the disease or condition in a particular individual. 2010 VT 1, ¶ 18, 187 Vt. 229, 993 A.2d 367. In addition to showing general causation through epidemiological studies, plaintiffs in toxic exposure cases must demonstrate specific causation by submitting evidence concerning "the amount, duration, intensity, and frequency of exposure." Dow Chemical, 321 F. App'x at 273; see Henricksen v. ConocoPhillips Co., 605 F.

Supp. 2d 1142, 1157 (E.D. Wash. 2009) (citing several appellate court cases holding that experts testifying as to specific causation must pay careful attention to amount, intensity, and duration of exposure).

¶ 7. Of course, in many, if not most, toxic tort cases it is impossible "to quantify with hard proof—such as the presence of the alleged toxic substance in the plaintiff's blood or tissue—the precise amount of the toxic substance to which an individual plaintiff was exposed." Plourde v. Gladstone, 190 F. Supp. 2d 708, 721 (D. Vt. 2002). "Thus, expert testimony on toxic injuries may be admissible where dosage or exposure levels have been roughly established through reliable circumstantial evidence." Id. But, while "it is not always necessary for a plaintiff to quantify exposure levels precisely," courts generally preclude experts from testifying "as to specific causation without having any measurements of a plaintiff's exposure to the allegedly harmful substance." Henricksen, 605 F. Supp. 2d at 1157 ("Defendant's concession that its product contains a known carcinogen—benzene—does not excuse Plaintiffs from having to show the benzene contained in Defendant's gasoline is capable of causing the illness at issue.").

¶ 8. When direct evidence of the precise amount of exposure to a toxic substance is limited, courts have allowed expert witnesses to use "differential diagnosis" as an accepted method of proving specific causation. Id. at 1157-58; Plourde, 190 F. Supp. 2d at 722. Differential diagnosis is a scientific analysis entailing "the weighing of relevant evidence, listing all likely causes of the patient's observed symptoms or injury, then eliminating all but one cause." Plourde, 190 F. Supp. 2d at 722; see Westberry v. Gislaved Gummi AB, 178 F.3d 257, 262 (4th Cir. 1999) (defining differential diagnosis as "standard scientific technique of identifying the cause of a medical problem by eliminating the likely causes until the most probable one is isolated" and noting that differential diagnosis is typically done based on physical examinations, medical histories, and clinical test results). "However, courts are reluctant to admit causation testimony based on a differential diagnosis where the proffered expert possesses only weak circumstantial evidence that some exposure occurred and makes no effort to scientifically evaluate or roughly estimate the degree of exposure or dosage." Plourde, 190 F. Supp. 2d at 722. Courts have also "excluded experts' differential diagnoses where they failed to adequately account for the likelihood that the disease was caused by an unknown factor." Henricksen, 605 F. Supp. 2d at 1162. "Standing alone, the presence of a known risk

factor is not a sufficient basis for ruling out idiopathic origin in a particular case, particularly where most cases of the disease have no known cause." Id. In such cases, "analysis beyond a differential diagnosis is required." Id.

¶ 9. Here, plaintiff relies upon three sources of evidence to avoid summary judgment. First, he offers statements made by himself and boyhood friends concerning their alleged exposure to toxic chemicals from the Goodyear plant when they were teenagers playing ball on a field adjoining the plant in the late 1960's and early 1970's. Plaintiff estimated that between 1968 and 1973, he spent thirty hours a week in the summer, and somewhat less during the school year, playing ball on the Goodyear field. He and friends stated that the grass was discolored and that a gully frequently filled with an oily, pungent, and discolored liquid ran through the outfield. Second, plaintiff relies on the report and deposition testimony of Robert Nicoloro, the project manager for an environmental firm hired by Goodyear in 2007 to conduct a site investigation in response to a clean-up agreement reached by Goodyear and the State of Vermont. The 2009 report stemming from the investigation listed contaminants of concern, including petroleum products containing benzene, that could have been released into the environment by way of normal plant operations. Given the standard of review on summary judgment, the court concluded that neither the 2009 report nor Mr. Nicoloro's testimony precluded the possibility that petroleum products containing benzene could have migrated via discharge stormwater from the plant and through the gully into the ballfield. Third, plaintiff relies upon the testimony of his two experts, Dr. David Goldsmith and Dr. Camilo Fadul. Dr. Goldsmith, an epidemiologist, testified that occupational exposure to benzene is generally associated with a risk of non-Hodgkin's lymphoma, of which Primary CNS Large B-Cell Lymphoma is a subtype. Dr. Fadul, a neuro-oncologist, testified that plaintiff's cancer was not caused by an immunodeficiency disorder, one of the known causes of that form of cancer.

¶ 10. In sum, plaintiff proffered evidence indicating that, as a teenager some thirty-five years earlier, he frequently played on a field adjoining the Goodyear plant. A gully that ran across the field may have contained water contaminated by petroleum products containing benzene. Benzene has been associated with non-Hodgkin's lymphoma, a general category of cancer under which plaintiff's subtype falls. Plaintiff's lymphoma was not caused by an immunodeficiency disorder, a known cause of that type of lymphoma.

¶ 11. Assuming that we accept all of this evidence as true, it falls well short of what plaintiff would be required to show in order to prevail in a jury trial. Indeed, if a jury were to find in favor of plaintiff on the evidence relied upon by plaintiff, we would have to overturn the verdict. In the end, plaintiff's suspicion that his cancer was caused by exposure to benzene on the Goodyear ballfield when he was a teenager is purely speculative. As plaintiff's own expert acknowledged, there is no way to know whether any benzene-containing product actually contaminated the ballfield. It is possible, of course. Although benzene itself was not used at the plant, plant operations entailed the use of petroleum products, including gasoline, that contain benzene. But even if we were to assume that benzene-containing products made their way into the gully and through the field, there is no evidence indicating the amount or concentration of benzene that was present. Nor is there any evidence indicating plaintiff's level of exposure to any benzene that may have been present on the field. Nor is plaintiff able to point to studies indicating a risk of cancer posed by exposure to limited amounts of benzene from petroleum products in an outside environment. Putting aside plaintiff's failure to demonstrate the presence of benzene in the field, a jury could only wildly speculate on the level of plaintiff's exposure to any such benzene and on the relationship between any such exposure and plaintiff's disease.

¶ 12. Further, plaintiff cannot rely upon differential diagnosis to overcome the complete lack of evidence as to the level of any exposure to benzene. His own expert acknowledged that a large percentage of cases of plaintiff's type of lymphoma are of unknown origin, so any attempt to establish causation by ruling out other causes must fail. See Bland, 538 F.3d at 897 (holding that because most cases of plaintiff's condition are of unknown origin, expert could not properly conclude, based on differential diagnosis, that plaintiff's exposure to freon was probable cause of her condition); Whiting v. Boston Edison Co., 891 F. Supp. 12, 21 n.41 (D. Mass. 1995) (concluding that differential diagnosis cannot be used to explain plaintiff's disease where 90% of cases of disease are of unknown origin).

¶ 13. Nonetheless, relying upon our discussion of epidemiological studies in George, plaintiff asserts that his experts' testimony regarding general and specific causation was sufficient to have a jury weigh the evidence on causation. As we noted in George, "[e]pidemiological studies quantify the degree of association between a given substance and a disease by assigning a 'relative risk' factor to the association." 2010 VT 1, ¶ 20. "When the relative risk reaches 2.0,

the risk has doubled, indicating that the risk is twice as high among the exposed group as compared to the non-exposed group." Id. (quotation omitted). "Thus, the threshold for concluding that an agent was more likely than not the cause of an individual's disease is a relative risk greater than 2.0." Id. (quotation omitted). Based on this reasoning, we concluded in George that "the trial court reasonably found the 2.0 standard to be a helpful benchmark in evaluating the epidemiological evidence underlying [the expert's] opinion." Id. ¶ 26. In George, we upheld the trial court's refusal to admit an expert opinion, which was based on epidemiological studies, that the plaintiff's firefighting responsibilities caused his lymphoma, noting that the expert did not specify the precise weight given to each study or how he reached his conclusion that the studies, taken together, demonstrated a statistically significant result. Id. ¶¶ 33, 36.

¶ 14. Here, in contrast, the trial court accepted the testimony of plaintiff's experts, but determined that the testimony along with other evidence proffered by plaintiff was insufficient on the element of causation to present his case to the jury. Plaintiff's first expert, Dr. Goldsmith, did not directly interview plaintiff or review his medical records. He specifically testified that his statement of opinion as to the derivation of plaintiff's illness was limited to general causation, and not specific causation. Indeed, when asked whether he was "in this case, giving an opinion as to the specific causation of [plaintiff's] disease," he responded as follows: "No. I'm speaking about the general causation of his disease." At one point, Dr. Goldsmith testified "[t]hat the epidemiology literature shows that it is more likely than not that exposure to benzene from rubber and tire manufacturing and other solvents are an explanation for [plaintiff's] non-Hodgkin's lymphoma." (Emphasis added.) Upon further questioning, Dr. Goldsmith emphasized, however, that his opinion was meant to indicate only that there is a general association between non-Hodgkin's lymphoma and exposure to benzene.

¶ 15. As for the strength of that association, Dr. Goldsmith testified: "My reading of the literature is that there is a substantial body of new epidemiology research . . . that has now linked so that one can say that the relative risks are greater than 2.0 and in that sense is a strong relative risk to the link between benzene and non-Hodgkin's lymphoma." Plaintiff has seized upon this statement and our statements in George noted above regarding relative risks to assert that Dr. Goldsmith's testimony is sufficient to have a jury make a determination on causation. We

disagree. First, neither Dr. Goldsmith's testimony nor any other evidence cited by plaintiff would allow a jury to find it more likely than not that benzene was located on the Goodyear field or, in particular, that plaintiff was exposed to benzene at a level that could have caused his illness. Indeed, Dr. Goldsmith himself acknowledged that no-one will ever know if there was benzene on the field and that plaintiff's statements as to the amount of time he played on the field say nothing about his level of exposure. Dr. Goldsmith's statement concerning his understanding of the recent epidemiological literature cannot overcome these shortcomings.

¶ 16. Further, in addition to being unable to estimate or evaluate the likely degree of exposure, cf. Plourde, 190 F. Supp. 2d at 723 n.10 (rejecting expert opinion in part because expert made no attempt to estimate or evaluate likely degree of exposure), Dr. Goldsmith conceded that different types of lymphomas had different etiologies and that he was unaware of any studies particular to plaintiff's rare lymphoma. Cf. George, 2010 VT 1, ¶ 21 (noting that trial court found studies relied upon by expert may have been overinclusive in reflecting associations between other types of lymphomas and generic cancers in firefighters). In the end, Dr. Goldsmith offered his opinion that studies showed an association between occupational exposure to benzene and non-Hodgkin's lymphomas. Nothing in his testimony was sufficient to support a jury finding of specific causation.

¶ 17. Nor was Dr. Fadul's testimony sufficient to get plaintiff's case to the jury. As explained above, Dr. Fadul was able to exclude only one known cause of plaintiff's lymphoma. Unfortunately for plaintiff, the vast majority of cases concerning his type of lymphoma are of unknown etiology. Therefore, the jury could not find more-probable-than-not specific causation based on Dr. Fadul's testimony.

¶ 18. Finally, we address the issue of spoliation. Without citing any case law or pointing to anything in the record to support his vague accusations, plaintiff suggests that Goodyear was obligated to keep records of its release of contaminants from the plant but either failed to do so or destroyed any records that were kept, making it virtually impossible for him to prove his case. Plaintiff fails to cite a specific legal basis for the obligation he claims Goodyear had to keep records. Nor does he cite any evidence of spoliation or note any extensive attempt on his part to discover Goodyear's past records. Under these circumstances, we find unavailing

plaintiff's unsupported argument that Goodyear's lack of records should result in an inference "favorable to the plaintiff"—presumably that benzene made its way from the plant to the ballfield at a level of concentration sufficient to cause plaintiff's illness.

Affirmed.

BY THE COURT:

Paul L. Reiber, Chief Justice

John A. Dooley, Associate Justice

Denise R. Johnson, Associate Justice

Marilyn S. Skoglund, Associate Justice

Brian L. Burgess, Associate Justice