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The Not So "Sweet Surprise": Lawsuits Blaming Big Sugar For Obesity-Related Health Conditions Face An Uphill Battle

Catherine Srithong Wicker

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THE NOT SO “SWEET SURPRISE”: LAWSUITS BLAMING BIG SUGAR FOR OBESITY-RELATED HEALTH CONDITIONS FACE AN UPHILL BATTLE

CATHERINE SRITHONG WICKER*

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I. INTRODUCTION

Stop into any bodega in Brooklyn, New York, and there is sure to be soda sweetened with corn syrup.¹ In Brooklyn alone, about “139 million gallons of soda are consumed each year, sweetened by 20,000 acres of corn.”² Driving a cab through his Brooklyn neighborhood of Fifth Ave. and Park Slope, Fray Mendez reminisces about “Colonial Grape,” the “sweetest grape soda [he] ever drank.”³ At

¹ KING CORN (Mosaic Films, Inc. 2007). In a documentary tracing the overproduction and subsidization of the corn industry in America, Ian Cheney and Curt Ellis attempt to trace where the nation’s surplus corn ends up. *Id.* Cheney and Ellis discover that for each kernel of corn produced, there is a 70% chance it will end up sweetening a beverage. *Id.*

² *Id.*

³ *Id.* This soda is also known as “Old Colony Uva”, and high fructose corn syrup is the second listed ingredient. SAM’S CLUB, <http://www.samsclub.com/sams/old-colony-grape-soda-11-27-fl-oz-24-pk/prod10640273.ip> (last visited Feb. 25, 2015).

his peak, he drank two liters of the soda each day, maybe more.⁴ At that time, Fray was also over 300 pounds, a size sixty in pants, and completely unaware that drinking soda was a problem.⁵ Although he lost approximately 100 pounds by eliminating soda from his diet, Fray was recently diagnosed with type 2 diabetes.⁶ Unfortunately, he is no stranger to the disease. Fray's parents and grandmother died as a direct result of their diabetes and his sister has been a diabetic for years.⁷ Fray recalls the pain his father had in his toe for six months before being diagnosed with diabetes.⁸ He describes how his father had his toe amputated; then his foot, his leg below the knee, and finally his leg above the knee.⁹ When doctors wanted to begin amputating his other leg, Fray's father had had enough and gave up.¹⁰

⁴ KING CORN, *supra* note 1. A single 11.27 fl. oz. can of Old Colony Uva contains 53 g of sugar. SAM'S CLUB, *supra* note 3. Therefore, drinking two liters of the grape soda is equivalent to consuming 0.70 lbs. of sugar.

⁵ *Id.*

⁶ KING CORN, *supra* note 1. Type 2 diabetes is a chronic condition that affects the way a person's body processes sugar (or glucose). *Type 2 Diabetes: Definition*, MAYO CLINIC, <http://www.mayoclinic.com/health/type-2-diabetes/DS00585> (last visited Feb. 25, 2015). "With type 2 diabetes, [a person's] body either resists the effects of insulin—a hormone that regulates the movement of sugar into . . . cells—or doesn't produce enough insulin to maintain a normal glucose level." *Id.* Because glucose is the body's main energy source, the inability of insulin to facilitate glucose's entry into cells means that those cells are not getting the fuel they need to properly function. *Diabetes Health Center, Causes of Type 2 Diabetes*, WEBMD, <http://diabetes.webmd.com/guide/diabetes-causes> (last visited Feb. 25, 2015). When glucose is unable to enter cells, it begins to accumulate in the blood, a condition called hyperglycemia. *Type 2 Diabetes*, MEDLINEPLUS, NAT'L INST. OF HEALTH, <http://www.nlm.nih.gov/medlineplus/ency/article/000313.htm> (last visited Feb. 25, 2015). Diabetes is clinically confirmed by meeting at least two of the following test criteria or repeating the test on different days: (1) a blood glucose level of at least 126 milligrams per deciliter after an overnight fast; (2) a non-fasting glucose level greater than or equal to 200 milligrams per deciliter with symptoms of diabetes; (3) a glucose level of at least 200 milligrams per deciliter on a 2-hour glucose tolerance test, or (4) a blood sugar (A1C) test of at least 6.5%. *Diabetes Health Center, supra*; see also *Type 2 Diabetes: Definition*, MAYO CLINIC, <http://www.mayoclinic.com/health/type-2-diabetes/DS00585/DSECTION=tests-and-diagnosis> (last visited Mar. 13, 2015).

⁷ KING CORN, *supra* note 1.

⁸ *Id.*

⁹ *Id.* Consistently high blood sugar levels can damage nerve fibers throughout the body, leading to a condition known as diabetic neuropathy. Guido R. Zanni, *Diabetic Neuropathy Symptoms and Treatment*, U.S. NEWS & WORLD REPORT (Nov. 9, 2013), <http://health.usnews.com/health-news/health-wellness/articles/2013/11/09/diabetic-neuropathy-symptoms-and-treatment>. Approximately "60 to 70[%] of people with diabetes have some form of neuropathy," the most common of which is peripheral neuropathy. *Diabetic Neuropathies: The Nerve Damage of Diabetes*, U.S. DEP'T OF HEALTH & HUMAN SERV'S, NAT'L INST. OF HEALTH 1 (2009) http://diabetes.niddk.nih.gov/dm/pubs/neuropathies/Neuropathies_508.pdf. Peripheral neuropathy causes pain or loss of feeling in the peripheral extremities, such as the toes, feet, and legs. *Id.* at 3. "Initially, it affects the foot" and "eventually spreads to the ankle and leg." Zanni, *supra* note 9. Because of nerve damage, patients do not feel pain and may be unaware of foot injuries, resulting in open sores. *Id.* These open sores may then become infected and in the most severe cases, lead to amputation of the toes or foot. *Id.* The natural progression of

Fray's story is a stark reality for many Americans, as we have become a society that walks less and eats more. As a culture, we have decreased the importance of physical activity, while simultaneously creating an unhealthy food environment.¹¹ Unfortunately for Fray, he may be without a legal remedy to sue to the makers of his favorite grape soda. Judicially created thresholds of admissibility for scientific evidence have made it increasingly difficult for food plaintiffs¹² to prove that a certain food ingredient, such as high fructose corn syrup (HFCS), caused their type 2 diabetes.

Because obesity and its associated health problems have been largely attributed to poor self-control, laziness, and various other *personal* failings, society has been unwilling to assign blame to food manufacturers for their role in contributing to this problem. But, as consumers are becoming more aware of the significantly harmful effect that poor diets can have on a person's health, the scales may be tipping in favor of bringing "Big Food"¹³ to court.

Food manufacturers, however, are not exactly vulnerable. Armed with precedent disputing the causal link between consumption of fast food and adverse health effects,¹⁴ judicially-created barriers to admitting epidemiologic evidence,¹⁵ and the defense of personal responsibility,¹⁶ food plaintiffs face an uphill battle.

This Comment explores that reality. It examines the various challenges that consumers face in holding food manufacturers liable for the dietary impact allegedly causing obesity-related health conditions. Part I briefly traces the emergence of HFCS in America's food supply and examines its possible role in the development of type 2 diabetes. It discusses previous litigation against the food industry for

diabetic neuropathy is irreversible loss of sensation in the feet, "leading to ulceration and/or amputation in 15[%] of patients." Oskar Aszmann, Patsy L. Tassler & A. Lee Dellon, *Changing the Natural History of Diabetic Neuropathy: Incidence of Ulcer/Amputation in the Contralateral Limb of Patients with a Unilateral Nerve Decompression Procedure*, 53 ANNALS OF PLASTIC SURGERY 517, 517 (2004).

¹⁰ KING CORN, *supra* note 1.

¹¹ See generally KELLY D. BROWNELL, *FOOD FIGHT: THE INSIDE STORY OF THE FOOD INDUSTRY, AMERICA'S OBESITY CRISIS AND WHAT WE CAN DO ABOUT IT* 8–9 (McGraw-Hill 2004). Scientists, James Hill and John Peters argue "the current epidemic of obesity is largely caused by an environment that promotes excessive food intake and discourages physical activity." James O. Hill & John C. Peters, *Environmental Contributions to the Obesity Epidemic*, 280 SCIENCE, 1371, 1371–74 (1998). Consequently, it is unsurprising that America's children are consuming record amounts of fat, sugar, and fast food. BROWNELL, *supra*, at 11.

¹² This Comment uses the phrase "food plaintiffs" to generally refer to litigants who bring lawsuits against food manufacturers for allegedly causing their obesity related health conditions.

¹³ Defined as the "large food companies and legacy brands on which millions of consumers have relied on for so long." Phil Wahba, *Campbell Soup CEO Says Distrust of 'Big Food' a Growing Problem*, FORTUNE (Feb. 18, 2015), <http://time.com/3714572/campbell-soup-ceo-says-distrust-of-big-food-a-growing-problem/>.

¹⁴ See *infra* Part II.B.

¹⁵ See *infra* Part IV.A.

¹⁶ See *infra* Part II.B.

allegedly causing obesity and associated health conditions; the Comment then describes a recent lawsuit targeting manufacturers of HFCS for allegedly causing a young girl's type 2 diabetes. Part II highlights epidemiology's role in demonstrating causation in product liability suits. Part III examines the current standard of admissibility for epidemiologic evidence in products liability cases after *Daubert v. Merrell Dow Pharmaceuticals, Inc.*¹⁷ Additionally, it discusses how various courts have construed *Daubert* as authorizing the creation of judge-made quantitative thresholds for admitting epidemiologic evidence.¹⁸ Part IV proposes that these judicially created standards be abandoned and suggests alternative criteria for evaluating the reliability of an epidemiologic study offered into evidence. Part V discusses various conduct-based defenses: assumption of risk, contributory negligence, and comparative fault, which limit food manufacturers' liability. Part VI explores the potential for market share liability to provide food plaintiffs a remedy by relaxing the burden of proving causation. Finally, Part VII highlights legislative attempts to curb food and beverage litigation, policy concerns, and the future of lawsuits targeting Big Food.

II. A RECENT LAWSUIT LINKING HFCS CONSUMPTION TO DIABETES IS REMINISCENT OF PELMAN V. MCDONALD'S CORP.

In 1971, Japanese food scientists discovered a way to produce a sweetener cheaper than sugar called high fructose corn syrup (HFCS).¹⁹ HFCS is six times sweeter than cane sugar and could be made from corn.²⁰ This was a boon to the corn industry, whose production rose to an all-time high in the mid-1970s.²¹ HFCS was economically produced from the huge surplus of corn grown by American farmers and thus significantly decreased the production costs of high-sugar products.²²

Initially, no warnings were raised about the significantly different metabolic path fructose takes in the human body.²³ Whereas sucrose—or regular table sugar—is

¹⁷ *Daubert v. Merrill Dow. Pharm., Inc.*, 509 U.S. 579 (1993).

¹⁸ See *infra* notes 103–05 and accompanying text.

¹⁹ GREG CRISTER, *FAT LAND: HOW AMERICANS BECAME THE FATTEST PEOPLE IN THE WORLD* 10 (Houghton Mifflin Harcourt 2003).

²⁰ *Id.* When commercial production of HFCS first began in 1967, its fructose content was approximately 15%. Lee S. Gross, Li Li, Earl S. Ford & Simin Liu, *Increased Consumption of Refined Carbohydrates and the Epidemic of Type 2 Diabetes in the United States: An Ecologic Assessment*, 79 AM. J. CLINICAL NUTRITION 774, 777 (2004). After several modifications that altered the proportion of fructose in HFCS, various food and beverage manufacturers began using HFCS sweeteners with greater fructose content. *Id.* For example, HFCS with a 55% fructose content was used as the sweetener of the soft drink and ice cream industries. *Id.* Additionally, HFCS with a 90% fructose content “became a frequent choice for use in ‘natural’ and ‘light’ foods.” *Id.*

²¹ CRISTER, *supra* note 19, at 10.

²² *Id.*

²³ *Id.* The digestive and absorptive processes are different for glucose—formed from the cleavage of sucrose—and fructose. George A. Bray, Samara Joy Nielsen & Barry M. Popkin, *Consumption of High-Fructose Corn Syrup in Beverages May Play a Role in the Epidemic of*

chemically broken down before arriving in the liver, fructose bypasses this process and arrives in the liver almost completely intact.²⁴ Although this feature of fructose, termed “metabolic shunting,” raised concern among food scientists, governmental bodies such as the USDA, did not explore the issue in depth.²⁵

Eventually, as mass production techniques made HFCS more readily available to food manufacturers, HFCS found its way into a wide assortment of foods: as a substitute for sucrose in carbonated drinks, candy, baked goods, canned fruits, and even dairy products.²⁶ Because of its unique chemical attributes, HFCS could be used in frozen foods to prevent freezer burn and in baked goods to enhance their natural appearance.²⁷ Indeed, corn syrup sweeteners now comprise greater than 20% of total daily carbohydrate intake, an increase of greater than 2100% since the beginning of the century.²⁸

A. The Possible Link Between HFCS and Global Diabetes Prevalence

Increasing scientific evidence lends support to the hypothesis that fructose consumption increases diabetes risk.²⁹ The increasing global prevalence of obesity and type 2 diabetes mirrors the worldwide increase in consumption of processed, Western style foods.³⁰ The increased availability and consumption of HFCS-

Obesity, 79 AM. J. CLINICAL NUTRITION 537, 538 (2004). Glucose and fructose are absorbed in different segments of the small intestine and once absorbed, are transported to the liver where the fructose can be converted to glucose or passed into circulation. *Id.* Addition of small amounts of fructose can help modulate liver metabolism. *Id.* However, ingestion of large amounts of fructose can potentially provide an unregulated source of precursors for fat synthesis in the liver. *Id.*

²⁴ CRISTER, *supra* note 19, at 11 (“Fructose, unlike sucrose . . . took a decidedly different route into the human metabolism. Where the latter would go through a complex break down process before arriving in the human liver, the former, for some reason, bypassed that breakdown and arrived almost completely intact in the liver . . .”).

²⁵ *Id.*

²⁶ *Id.* at 10, 138; Bray et al., *supra* note 23, at 540.

²⁷ CRISTER, *supra* note 19, at 10.

²⁸ Gross et al., *supra* note 20, at 775–76.

²⁹ Michael I. Goran et al., *High Fructose Corn Syrup and Diabetes Prevalence: A Global Perspective*, 8 GLOBAL PUBLIC HEALTH 1, 56 (2012). Using a multivariate nutrient-density model, researchers found that corn syrup consumption was positively associated with the prevalence of type 2 diabetes after controlling for total energy, fiber, fat, and protein intake. Gross et al., *supra* note 20, at 775. There has been a study, however, which found no association between HFCS and adverse health consequences. James M. Rippe & Theodore J. Angelopoulos, *Sucrose, High-Fructose Corn Syrup, and Fructose, Their Metabolism and Potential Health Effects: What Do We Really Know?*, 4 ADVANCES IN NUTRITION 236, 242 (2013) (concluding that “there is no unique relationship between HFCS and obesity.”). However, the findings of that particular study are questionable in light of the researcher’s conflict of interest. *Id.* at 236 n. 2 (“[Rippe’s] consulting fees [are] from ConAgra Foods, Pepsi Co International, Kraft Foods, the Corn Refiners Association, and Weigh Watchers International.”).

sweetened beverages is of particular concern because it provides an “an easy vehicle for excessive sugar intake.”³¹

Amid increasing public health alarm at the “concurrent global epidemics” of obesity and type 2 diabetes, scientists began investigating the possible link between consumption of HFCS and the global rise in the prevalence of diabetes.³² Researchers found that countries utilizing HFCS in their food supply as an alternative sweetener had a diabetes prevalence rate approximately 20% higher than in countries where HFCS is not used.³³

B. The Latest Battle in the Fight Against Big Food

On June 17, 2013, a lawsuit was filed in a New York District Court on behalf of a fourteen-year-old girl claiming that HFCS caused her type 2 diabetes.³⁴ This novel suit sought to hold HFCS manufacturers strictly liable for creating an unreasonably dangerous product, without warning consumers of its potential adverse health consequences, which include development of type 2 diabetes.³⁵ The complaint alleged that defendants “knew or should have known that HFCS was a *cause* of type of diabetes” and deliberately concealed this fact.³⁶ The plaintiff also claimed that the defendants were negligent “in their marketing, distribution, warning, testing and instructions to . . . consumers of the risks associated with the consumption of their product.”³⁷ As a result of the defendants’ actions, the plaintiff sought five million dollars in damages for her injuries, which included the development of “type 2

³⁰ Goran et al., *supra* note 29, at 1 (citing Barry M. Popkin & Penny Gordon-Larsen *The Nutrition Transition: Worldwide Obesity Dynamics and Their Determinants*, 28 INT’L J. OBESITY & RELATED METABOLIC DISORDERS S1, S2–S9 (3 Supp. 2004); *but see* Rippe & Angelopoulos, *supra* note 29, at 242 (noting epidemics of diabetes and obesity in countries where little or no HFCS is available, such as Mexico, Australia, and Europe).

³¹ Goran et al., *supra* note 29, at 1.

³² *Id.* Research conducted by Bray et al. showed that the increase in the consumption of HFCS preceded the rapid increase in obesity prevalence. Bray et al., *supra* note 23, at 542. Similarly, the concurrent rise of HFCS in the nation’s food supply and rates of obesity have led to the notion that consumption of HFCS sweetened soft drinks caused or contributed to childhood obesity. R.E. Morgan, *Does Consumption of High-Fructose Corn Syrup Beverages Cause Obesity in Children?*, 8 PEDIATRIC OBESITY 249, 249–50 (2013). However, a literature review of the available research investigating this specific relationship concluded that the causal mechanism behind childhood obesity is complex and therefore, increased rates of obesity should not be solely attributed to consumption of HFCS beverages. *Id.* at 252.

³³ Goran et al., *supra* note 29, at 5. Using data on food availability for forty-three countries, researchers compared diabetes estimates between countries that were HFCS users and those that were non-users, adjusting for country-based estimates of body mass index (an indicator of obesity). *Id.* at 3. The study revealed that all indicators for diabetes were higher in countries that used HFCS than those that did not. *Id.*

³⁴ Complaint at 2, *S.F. v. Archer-Daniels-Midland Co.*, No. 1:13-cv-00634-WMS (W.D.N.Y. June 17, 2013).

³⁵ *See id.* at 10–13.

³⁶ *Id.* at 14 (emphasis added).

³⁷ *Id.*

diabetes, loss of enjoyment of life, pain and suffering, emotional distress . . . and lifelong and permanent medical complications[,] including the probability of surgery and shortened life expectancy.”³⁸ The court ultimately dismissed plaintiff’s claims, noting that she offered “limited facts” to demonstrate that her consumption of foods containing HFCS—specifically HFCS manufactured by defendants—“over the course of her life[,] was a substantial factor in causing her [disease].”³⁹

This latest lawsuit is not the first time the food industry has been blamed for causing negative health outcomes.⁴⁰ In *Pelman v. McDonald’s Corp.*,⁴¹ two obese teenage girls took the fast food giant to court.⁴² The girls claimed that, as a result of

³⁸ *Id.* at 15.

³⁹ *S.F. v. Archer-Daniels-Midland Co.*, No. 13-cv-634S, 2014 WL 1600414, at *4 (W.D.N.Y. Apr. 21, 2014). The court observed that the “mere possibility of . . . causation is not enough.” *Id.* (quoting WILLIAM L. PROSSER, *LAW OF TORTS* § 41 (4th ed. 1971)). Thus, “[i]t may be possible that HFCS caused Plaintiff to develop [t]ype 2 diabetes, but [based upon the facts alleged] is it plausible?” *Id.* (emphasis added).

⁴⁰ In 2002, Caesar Barber, a fifty-six-year-old man who was 270 pounds and five-foot-ten-inches tall, brought a class action lawsuit against the fast food giants: McDonald’s Corporation, Burger King Corporation, KFC Corporation, and Wendy’s International, Inc. *Complaint of Caesar Barber v. McDonald’s Corp.*, No. 23145/2002 (N.Y. Sup. Ct. 2002); see also Geraldine Sealey, *Obese Man Sues Fast-Food Chains*, ABC NEWS, <http://abcnews.go.com/US/Story?id=91427&page=1> (last visited Feb. 25, 2015). His complaint alleged that the fast food manufacturer’s products, which were high in fat, salt, sugar, and cholesterol, contributed to his obesity. *Complaint, Barber*, No. 23145/2002, at 9–10. Moreover, Mr. Barber claimed the defendants did not disclose the ingredients of their food or adequately warn consumers that ingestion of its products are known to cause “obesity, diabetes, coronary heart disease, high blood pressure, elevated cholesterol intake, [and] related cancers . . .” *Id.* (“Barber’s lawsuit is the first broad-based action taken against the fast food industry for allegedly contributing to obesity. [Barber] claims the fast food restaurants, where [he] says he used to eat four or five times a week even after suffering a heart attack, did not properly disclose the ingredients of their food and the risks of eating too much.”). Mr. Barber’s attorney ultimately withdrew the case to pursue the potentially “ground-breaking” case, *Pelman v. McDonald’s Corp.* Saul Wilensky & Kerry C. O’Dell, *Where’s the Beef?—The Challenges of Obesity Lawsuits*, BLOOMBERG LAW, <http://about.bloomberglaw.com/practitioner-contributions/wheres-the-beef-the-challenges-of-obesity-lawsuits/> (last visited Feb. 25, 2015) (“Barber’s attorney withdrew the case to pursue *Pelman v. McDonald’s Corporation*, a class action with greater potential since, arguably, child plaintiff’s would not be accountable for their choices of food. *Pelman* was expected to be ground-breaking litigation that would purportedly expose industry documents, which, in turn, could be used to bring a flood of litigation against ‘Big Food.’”).

⁴¹ *Pelman v. McDonald’s Corp.*, 237 F. Supp. 2d 512 (S.D.N.Y. 2003).

⁴² The plaintiffs were fourteen-year-old Ashley Pelman, who was four-feet-ten-inches tall and weighed 170 pounds, and nineteen-year-old Jazlyn Bradley, who was five-feet-six-inches tall and weighed 270 pounds. FRANCINE R. KAUFMAN, *DIABESITY: THE OBESITY-DIABETES EPIDEMIC THAT THREATENS AMERICA AND WHAT WE MUST DO TO STOP IT* 218 (2005). Using a person’s height and weight, body mass index (BMI) provides an indirect measure of a person’s body fat. *About BMI for Adults*, CENTERS FOR DISEASE CONTROL AND PREVENTION (Sept. 13, 2011), http://www.cdc.gov/healthyweight/assessing/bmi/adult_BMI/. According to BMI categories, persons with BMI greater than 25 are considered overweight and those with a BMI over 30 are classified as obese. *Id.* According to her BMI of 35.5, Ashley Pelman was considered obese. *Calculate Your Body Mass Index*, NATIONAL INSTITUTES OF HEALTH, <http://www.nhlbi.nih.gov/guidelines/obesity/BMI/bmicalc.htm> (last visited Mar. 30, 2015).

their consumption of McDonald's fast food products, they became overweight, developed diabetes, coronary heart disease, high blood pressure and cholesterol levels, and other detrimental health effects.⁴³

The girls claimed that the food giant engaged in deceptive marketing and selling practices that caused them to "injure their health by becoming obese."⁴⁴ Their complaint alleged that McDonald's failed to adequately disclose the health effects of ingesting certain food items with high levels of cholesterol, fat, salt, and sugar and that it engaged in marketing designed to entice customers to purchase "value meals" without disclosing the potential negative health effects of consumption.⁴⁵

Although obesity means having a BMI greater than or equal to 30, morbid obesity begins at a BMI of 40. Daniel J. DeNoon, *Morbid Obesity Bulge Gets Bigger*, WEBMD (Apr. 9, 2007), <http://www.webmd.com/diet/news/20070409/morbid-obesity-bulge-gets-bigger>. Under this classification, Jazlyn Bradley, who had a BMI of 43.6, was considered morbidly obese. *Id.* Jazlyn's BMI places her "at greater risk for illnesses including diabetes, high blood pressure, sleep apnea, gastroesophageal reflux disease (GERD), gallstones, osteoarthritis, heart disease, and cancer." *What is Morbid Obesity?*, U.ROCHESTER MED. CTR., <http://www.urmc.rochester.edu/highland/departments-centers/bariatrics/right-for-you/morbid-obesity.aspx> (last visited Mar. 13, 2015).

⁴³ Pelman, 237 F. Supp. 2d at 519. Since she was five years old, Ashley had consumed Happy Meals and Big Macs three to four times a week. KAUFMAN, *supra* note 42, at 218. Jazlyn, a more avid consumer, ate at McDonald's for both breakfast and lunch, and sometimes after school. *Id.* Generally, she ordered whole meals each visit, consisting of a Big Mac, Chicken McNuggets, or fried fish sandwich, in addition to fries, soda or a dessert. *Id.*

⁴⁴ Pelman, 237 F. Supp. 2d at 516. The plaintiffs in *Pelman* were not the last to accuse McDonald's of deceptive practices that violated consumer protection laws. Ashley Post, *Class Action Lawsuit Against McDonald's Attacks Marketing to Children*, INSIDECOUNSEL.COM. (Mar. 1, 2011), <http://www.insidecounsel.com/2011/03/01/class-action-lawsuit-against-mcdonalds-attacks-marketing-to-children?t=litigation&page=3>. In December 2010, a California mother of two, Monet Parham, filed a class action lawsuit against McDonald's Corp., alleging that marketing Happy Meals to children violated consumer protection laws. *Id.* According to Parham's complaint, "McDonald's exploit[ed] very young . . . children and harm[ed] their health by advertising unhealthy Happy Meals with toys directly to them." Amended Class Action Complaint for Violations of the Unfair Competition Law, the Consumers Legal Remedies Act & Declaratory & Injunctive Relief at 2, *Parham v. McDonald's Corp.*, No. 10-506178 (Cal. Super. Ct., San Francisco County Apr. 4, 2014). Because young children "do not have the cognitive skills and developmental maturity" to comprehend McDonald's persuasive marketing techniques, its use of toys to "bait" children was "inherently deceptive" and violated state law. *Id.* In April 2012, however, Parham's claims were dismissed without leave to amend. Order Sustaining McDonald's Demurrers to Plaintiff's Amended Complaint at 2, *Parham v. McDonald's Corp.*, No. 10-506178 (Cal. Super. Ct., San Francisco County Apr. 4, 2014). Although viewed by some critics as frivolous, *Parham v. McDonald's Corp.* "reflects a national focus on health, and imminent industry wide marketing and product reform." Post, *supra*.

⁴⁵ Pelman, 237 F. Supp. 2d at 520. Class action lawsuits for alleged violations of consumer protection statutes may be the best approach to encourage the food industry to improve the nutritional content of its product and to change its marketing strategies. Jess Alderman & Richard A. Daynard, *Applying Lessons from Tobacco Litigation to Obesity Lawsuits*, 30 AM. J. PREVENTIVE MED. 82, 87 (2006). This is likely because "[c]onsumer protection statutes make it easier for plaintiffs to demonstrate a link between corporate behavior . . . and the public's direct losses . . . because most do not require a showing that the defendant's misbehavior caused a specific illness." *Id.* at 85. A similar approach was

Plaintiffs also accused McDonald's of negligence, arguing that the food giant sold food products high in cholesterol, fat, salt, and sugar when studies showed that consumption of such foods caused obesity and other adverse health consequences.⁴⁶ The complaint further alleged that McDonald's failed to warn customers of the nutritional content and specific ingredients of its food products.⁴⁷

In dismissing each of the plaintiffs' claims, the court provided a roadmap for future food industry lawsuits. Specifically, the court found that the plaintiffs failed to establish that McDonald's produced a product with dangerous attributes that were outside the common knowledge of the reasonable consumer.⁴⁸ The court acknowledged the well-known fact that fast food, particularly McDonald's fast food products, contained high levels of fat, cholesterol, salt, and sugar and that these ingredients were deleterious to one's health.⁴⁹ As long as consumers knowingly exercise their free choice, manufacturers will not face liability.⁵⁰ The court found that plaintiffs did not allege in their complaint that their choice to eat McDonald's

successfully used by plaintiffs against tobacco companies. See *Aspinall v. Phillip Morris, Co.*, No. Civ.A. 98-6002, 2006 WL 2971490, at *1 (Super. Ct. Mass. Aug. 9, 2006) (holding that Philip Morris engaged in deceptive business practices when it used the descriptors "light" and "lowered tar and nicotine" in the marketing of their cigarettes); *but see Price v. Philip Morris, Inc.*, 848 N.E.2d 1, 54 (Ill. 2005) (overturning \$10 billion jury award for plaintiffs by finding that Philip Morris' use of the terms "light" and "lowered tar and nicotine" were not a violation of the Consumer Fraud Act because their use was specifically authorized by the Federal Trade Commission.).

⁴⁶ *Pelman v. McDonald's Corp.*, 237 F. Supp. 2d 512, 520 (S.D.N.Y. 2003).

⁴⁷ *Id.* In a negligent failure to warn claim, a plaintiff must prove: (1) the danger was not obvious to the average consumer; (2) the product was unreasonably dangerous for its intended use; (3) the plaintiff's obesity was caused by the food product; and (4) the harm would not have occurred if a warning had been given. Michelle M. Mello, Eric B. Rimm & David M. Studdert, *The McLawsuit: The Fast-Food Industry and Legal Accountability for Obesity*, 22 HEALTH AFFAIRS 207, 208–09 (2003) (citing *Pelman*, 237 F. Supp. 2d at 540–41); *see generally* RESTATEMENT (THIRD) OF TORTS: PRODS. LIAB. § 2 cmt. i (1998). In their amended complaint, the plaintiffs in *Pelman* re-alleged negligence by McDonald's because of its failure to warn plaintiffs of the dangers of eating processed foods from McDonald's, but dropped this cause of action right before oral argument. Amended Complaint, *Pelman v. McDonald's Corp.*, No. 02 Civ. 7821(RWS) (S.D.N.Y. Feb. 19, 2003); *Pelman ex rel. Pelman v. McDonald's Corp.*, No. 02 Civ. 7821(RWS), 2003 WL 22052778, at *2 (S.D.N.Y. Sept. 3, 2003) ("Shortly before oral argument . . . plaintiffs informed the Court that they [were] dropping their fourth cause of action, which alleged negligence by McDonald's because of its failure to warn plaintiffs of the dangers and adverse health effects of eating processed foods from McDonald's.").

⁴⁸ *Pelman*, 237 F. Supp. 2d at 522. For a product to be considered unreasonably dangerous, "the article sold must be dangerous to an extent beyond that which would be contemplated by the ordinary consumer who purchases it, with the ordinary knowledge common to the community as to its characteristics." RESTATEMENT (SECOND) OF TORTS, § 402A cmt. i (1965).

⁴⁹ *Pelman*, 237 F. Supp. 2d at 532.

⁵⁰ *Id.* at 533 ("As long as a consumer exercises free choice with appropriate knowledge, liability for negligence will not attach to a manufacturer. It is only when that free choice becomes but a chimera—for instance, by the masking of information necessary to make the choice . . . that manufacturers should be held accountable.").

“several times a week [was] anything but a choice freely made” and that their decisions could not now be attributed to McDonald’s.⁵¹

Another hurdle that the *Pelman* plaintiffs faced was establishing that their particular health problems were proximately caused by consumption of McDonald’s fast food products.⁵² In order for a plaintiff to prove proximate cause, he or she must demonstrate that the defendant’s actions were a substantial factor in bringing about the harm.⁵³ The court found that the plaintiffs failed to establish proximate cause because it is “impossible as a matter of law” to blame a particular fast food establishment for causing their obesity when the plaintiffs were possibly consuming other unhealthy foods, may or may not have engaged in regular physical activity, and when their weights could have been influenced by other genetic or environmental factors.⁵⁴

In dismissing the plaintiffs’ claims for lack of proximate cause, the court provided guidance on how future plaintiff could survive a motion to dismiss. First, a complaint “must establish that the plaintiffs ate at McDonald’s on a sufficient number of occasions such that a question of fact is raised as to whether McDonald’s products played a significant role in the plaintiff’s health problems.”⁵⁵ Second, in order to allege that consumption of McDonald’s products was a substantial factor in causing a plaintiff’s health problems, the complaint must eliminate, or at least address, the factors, other than diet, that might cause or contribute to obesity and other health problems.⁵⁶

⁵¹ *Id.*

⁵² *Id.* at 537.

⁵³ *Id.* In addition to the substantial factor test, demonstrating that the consumption of fast food causes to adverse health effects requires two additional proofs. Mello et al., *supra* note 47, at 209. First, plaintiffs must establish the association between obesity and specific negative health consequences and then, convince the court that if adequate warnings have been provided, “the plaintiffs could have avoided the health problems for which they blame the manufacturer.” *Id.* at 10.

⁵⁴ *Pelman*, 237 F. Supp. 2d at 537, n.27. For additional information on factors contributing to obesity, see *Obesity Causes*, HARVARD SCH. PUB. HEALTH, <http://www.hsph.harvard.edu/obesity-prevention-source/obesity-causes/> (last visited Mar. 30, 2015).

⁵⁵ *Pelman*, 237 F. Supp. 2d at 538–39. For instance, the larger the number of McDonald’s meals consumed, the greater the likelihood that its products were a substantial factor in adversely affecting plaintiffs’ health. *Id.* at 539.

⁵⁶ *Id.* (citing *Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults*, NAT’L INST. HEALTH (Sept. 1998), http://www.nhlbi.nih.gov/guidelines/obesity/ob_gdlns.pdf (“Obesity is a complex multifactorial chronic disease that develops from an interaction of genotype and the environment. Our understanding of how and why obesity develops is incomplete, but involves the integration of social, behavioral, cultural, physiological, metabolic and genetic factors.”)). The federal Food and Drug Administration also acknowledges that “[t]he problem of obesity in America has no single cause. Rather [it] is the result of multiple factors acting together over time.” *Calories Count: Report of the Working Group on Obesity*, U.S. FOOD & DRUG ADMIN. (Nov. 10, 2011), <http://www.fda.gov/Food/FoodScienceResearch/ConsumerBehaviorResearch/ucm081770.htm>

III. USING EPIDEMIOLOGIC EVIDENCE TO ESTABLISH CAUSATION IN PRODUCT LIABILITY CASES

Generally, causation is established when a plaintiff can demonstrate a nexus between a defendant's wrongful conduct and her injury.⁵⁷ The traditional "but for" test of actual causation, commonly employed by most jurisdictions, "illustrates the 'cause and effect' relationship between an event and a consequence."⁵⁸ The test recognizes that a defendant's actions are responsible for a plaintiff's injuries if the harm "would not have occurred in the absence of the defendant's conduct."⁵⁹ Although the application of the "but for" test resolves most causation issues in tort law, the test presumes the existence of only a single causative factor.⁶⁰

Direct traceability is difficult to establish, however, when multiple factors may have played a role in bringing about a single injury. To prevent negligent defendants from escaping liability by pointing to other potential causes for the plaintiff's injury, courts developed the "substantial factor" test for multiple causation scenarios.⁶¹ Under this test, a plaintiff must prove that a defendant's actions were a *substantial factor* in bringing about her harm.⁶² Thus, in order to prevail against HFCS manufacturers under a negligence theory, the plaintiff must establish a causal link between consumption of the defendant's products and the development of type 2 diabetes. Notably, the plaintiff need not show that her consumption of HFCS was the sole cause of her diabetes, only that it played a substantial role in the

⁵⁷ PROSSER AND KEETON ON THE LAW OF TORTS 266 (5th ed. 1984).

⁵⁸ Danielle Conway-Jones, *Factual Causation in Toxic Tort Litigation: A Philosophical View of Proof and Certainty in Uncertain Disciplines*, 35 U. RICH. L. REV. 875, 876 (2002).

⁵⁹ DAN B. DOBBS, PAUL T. HAYDEN & ELLEN M. BUBLICK, THE LAW OF TORTS § 186 (2d ed. 2011) ("Under the but-for test, the defendant's conduct is a factual cause of the plaintiff's harm if, but-for the defendant's conduct, that harm would not have occurred.").

⁶⁰ Conway-Jones, *supra* note 58, at 888. Another problem with the traditional "but for" test is that it "can be applied only by comparing what happened with a hypothetical alternative series of events that would have occurred had the defendant not acted negligently. *Id.* Thus, the determination of factual cause will be based on speculation "about whether the injury really could have been avoided if the defendant had not been negligent." *Id.*

⁶¹ Conway-Jones, *supra* note 58, at 889; *Tragarz v. Keene Corp.*, 980 F.2d 411, 425 (7th Cir. 1992) ("[T]he purpose . . . of the substantial factor test . . . is aimed at alleviating the inequities that result when applying the but-for test in a multi-defendant case, not at creating such inequities."). *Id.*

⁶² RESTATEMENT (SECOND) OF TORTS § 431 (1965).

development of her disease.⁶³ To demonstrate causation, a plaintiff usually presents epidemiologic evidence “as the basis of an expert’s opinion on causation.”⁶⁴

Epidemiology is the “study of the distribution and determinants of health related . . . events in specified populations, and the application of this study to control of health problems.”⁶⁵ The goal of epidemiologic research is to quantify the causal relationship between a certain exposure and disease.⁶⁶ A two-step process is often followed when evaluating epidemiological evidence.⁶⁷ First, researchers determine whether there is an association between an exposure and a particular outcome by conducting studies of group (ecological studies) and individual characteristics (cohort and case-control studies).⁶⁸ Next, if an association is found, researchers evaluate whether the observed relationship is a causal one.⁶⁹

Epidemiological studies are increasingly used as a method for demonstrating risks to population groups from various products or practices.⁷⁰ This discipline

⁶³ *See id.* The “substantial factor test is not a comparative test in which the jury assesses all contributing causes and determines which ones are substantial.” Tragarz, 980 F.2d at 424. Instead, the test is whether each contributing cause, by itself, is a substantial factor in causing the injury. *Id.* at 424–25 (citing Lipke v. Celotex Corp., 505 N.E.2d 1213, 1221 (Ill. App. Ct. 1987)).

⁶⁴ FED. JUDICIAL CTR., REFERENCE MANUAL ON SCIENTIFIC EVIDENCE 551 (3d ed. 2011) (“Judges and juries are regularly presented with epidemiologic evidence as the basis of an expert’s opinion on causation. In the courtroom, epidemiologic research findings are offered to establish or dispute whether exposure to an agent caused a harmful effect or disease.”).

⁶⁵ JOHN M. LAST, A DICTIONARY OF EPIDEMIOLOGY 62 (4th ed. 2001).

⁶⁶ DAVID A. SAVITZ, INTERPRETING EPIDEMIOLOGIC EVIDENCE: STRATEGIES FOR STUDY DESIGN AND ANALYSIS 9 (2003); *see* FED. JUDICIAL CTR., *supra* note 64, at 552 (“Epidemiologic evidence identifies agents that are associated with an increased risk of disease in groups of individuals, quantifies the amount of excess disease that is associated with an agent, and provides a profile of the type of individual who is likely to contract a disease after exposed to an agent.”).

⁶⁷ LEON GORDIS, EPIDEMIOLOGY 204 (3d ed. 2004).

⁶⁸ *Id.*

⁶⁹ *Id.* A demonstrated association between a risk factor and disease is attributed to “chance, bias, confounding, and/or causality.” *The Role of Epidemiology in Decision-Making*, THE ANAPOLIS CENTER (1999), <http://www.accpl.org/pdf/EpidemiologyInDecision.pdf>. Using various study designs, epidemiologists strive to “reduce the influence of the first three [factors], leaving cause as the most likely explanation of the demonstrated association.” *Id.* Even if an association is found, the inability of an investigator to reduce or at least account for, the effect of chance, confounding, and bias will deem the study’s results of little value towards establishing causation. *Id.*

⁷⁰ *See In re Breast Implant Litig.*, 11 F. Supp. 2d 1217, 1224 (D. Colo. 1998) (“Epidemiology is the best evidence of causation in the mass torts context.”). In *In re Breast Implant Litigation*, plaintiffs asserted tort claims of negligence and strict liability against various breast implant manufacturers alleging that their silicone breast implants caused various autoimmune diseases. *Id.* at 1221. The court noted that, without a controlled epidemiologic study, there would be no way to determine whether autoimmune diseases “are more common in women with silicone breast implants than women without implants.” *Id.* at 1224. Thus, these studies were deemed necessary to determine causation. *Id.*; *Brock v. Merrell Dow Pharm., Inc.*, 874 F.2d 307, 315 (5th Cir. 1989) (“[S]peculation unconfirmed by

attained prominence in toxic tort cases where causation was in dispute because toxic tort injuries can remain latent for many years.⁷¹

Indeed, the difficulty of proving causation is a major difference between toxic tort and ordinary personal injury cases.⁷² Whereas a single product or incident causes an identifiable harm in personal injury suits, disease resulting from long-term exposure to a chemical is not directly observable and may not provide any physical evidence of the causative agent.⁷³ Because plaintiffs must offer epidemiologic evidence to establish the requisite causal link between an exposure and disease outcome, factfinders must understand what the discipline can, and more importantly, cannot prove.⁷⁴

IV. THE USE OF SCIENTIFIC EVIDENCE TO PROVE CAUSATION UNDER DAUBERT AND ITS PROGENY

The current standard of admissibility for expert testimony was first outlined in the leading case of *Daubert v. Merrell Dow Pharmaceuticals, Inc.*⁷⁵ In *Daubert*, two children alleged that their serious birth defects were caused by their mothers' prenatal ingestion of the anti-nausea drug, Bendectin, marketed by Merrell Dow Pharmaceuticals, Inc. (Dow).⁷⁶ Dow's expert, a physician and epidemiologist,

epidemiologic proof cannot form the basis for causation in a court of law."). The infamous "tobacco cases" also highlighted the use of epidemiologic evidence to establish causation in personal injury lawsuits. In these cases, plaintiffs attempted to use scientific evidence to demonstrate the link between cigarette smoking and lung cancer. For example, in *Pritchard v. Liggett & Myers Tobacco Co.*, the plaintiff introduced several expert witnesses each of whom testified that the plaintiff's lung cancer was caused by smoking. *Pritchard v. Liggett & Myers Tobacco Co.*, 295 F.2d 292, 294, 299–300 (3d Cir. 1961). The appellate court found that the plaintiff's evidence was sufficient to submit the issue of causation to the jury. *Id.* at 295.

⁷¹ Steve C. Gold, *Revisiting Relative Risk Rules: Garza, Blanchard, and the Ever Evolving Role of Epidemiologic Proof in Toxic Tort Cases*, 27 TOXICS LAW REPORTER 49, 49 (2012); see *Norris v. Baxter Healthcare Corp.*, 397 F.3d 878, 882 (10th Cir. 2005) ("[E]pidemiology is the best evidence of general causation in a toxic tort case.").

⁷² Steve Gold, *Causation in Toxic Torts: Burdens of Proof, Standards of Persuasion, and Statistical Evidence*, 96 YALE L.J. 376, 379 (1986).

⁷³ *Id.*

⁷⁴ Michael Dore, *A Proposed Standard for Evaluating the Use of Epidemiological Evidence in Toxic Tort and other Personal Injury Cases*, 28 HOW. L.J. 677, 683 (1985); see, e.g., *Brock*, 874 F.2d at 313 ("We find . . . the lack of conclusive epidemiological proof to be fatal to the Brock's case. While we do not hold that epidemiologic proof is a necessary element in all toxic tort cases, it is certainly a very important element.").

⁷⁵ Some consider the three U.S. Supreme Court decisions: *Daubert v. Merrell Dow Pharm., Inc.*, 509 U.S. 579 (1993), *General Electric Co. v. Joiner*, 522 U.S. 136 (1997), and *Kumho Tire Co. v. Carmichael*, 526 U.S. 137 (1999), as the "trilogy" of cases that reflect the current standard on the admissibility of expert testimony. See generally Margaret A. Berger, *The Supreme Court's Trilogy on the Admissibility of Expert Testimony*, in REFERENCE MANUAL ON SCIENTIFIC EVIDENCE 10, 10–20 (2d ed. 2000).

⁷⁶ *Daubert*, 509 U.S. at 581.

reviewed more than thirty published studies on Bendectin and birth defects, containing over 130,000 patients, none of which found that Bendectin was capable of causing human malformations.⁷⁷ Based on these reviews, Dow's expert concluded that maternal ingestion of Bendectin during the first trimester of pregnancy was not a risk factor for birth defects.⁷⁸

Although not disputing the published findings, the plaintiffs offered the testimony of their own expert witnesses, all eight of whom found that Bendectin could cause birth defects.⁷⁹ The witnesses based their conclusions on a combination of animal-cell, live-animal, and pharmacological studies, and a reanalysis of previously published epidemiological studies.⁸⁰ In granting Dow's motion for summary judgment, the district court found that the plaintiffs' reanalysis of published epidemiological studies was inadmissible because the reanalysis studies had neither been published, nor subject to the peer review process.⁸¹

Upon review of the lower court's ruling, the Supreme Court in *Daubert* held that the Federal Rules of Evidence, particularly Rule 702,⁸² replaced *Frye's* "General Acceptance" test in determining the admissibility of scientific evidence.⁸³ The Court assigned the trial judge the "gatekeeping" role of ensuring that an expert's testimony is relevant and has a solid foundation.⁸⁴

Under Rule 702, when expert testimony is offered, a trial judge must make a preliminary assessment of whether the methodology underlying the expert's testimony is scientifically valid and whether it would assist the factfinder in understanding the matter in issue.⁸⁵ The Court provided a non-exhaustive list of factors to be considered in evaluating the admissibility of expert scientific testimony: (1) whether technique or theory can be and has been tested; (2) whether the theory or

⁷⁷ *Id.* at 582.

⁷⁸ *Id.*

⁷⁹ *Id.* at 583.

⁸⁰ *Id.*

⁸¹ *Id.* For a novel statistical approach analyzing the Bendectin data, see Jon Todd Powell, *How to Tell the Truth With Statistics: A New Statistical Approach to Analyzing the Bendectin Epidemiological Data in the Aftermath of Daubert v. Merrell Dow Pharmaceuticals*, 31 HOUS. L. REV. 1241, 1291-1311 (Winter 1994).

⁸² Federal Rules of Evidence Rule 702 states:

If scientific, technical, or other specialized knowledge will assist the trier of fact to understand the evidence or to determine a fact in issue, a witness qualified as an expert by knowledge, skill, experience, training, or education, may testify thereto in the form of an opinion or otherwise, if (1) the testimony is based upon sufficient facts or data, (2) the testimony is the product of reliable principles and methods, and (3) the witness has applied the principles and methods reliably to the facts of the case.

FED. R. EVID. 702.

⁸³ *Daubert v. Merrell Dow Pharm., Inc.*, 509 U.S. 579, 597 (1993).

⁸⁴ *Id.*

⁸⁵ *Id.* at 592-93.

technique has been subjected to peer review and publication;⁸⁶ (3) the known or potential error rate of the scientific technique; and (4) general acceptance within the relevant scientific community.⁸⁷

The federal judge's gatekeeping role in screening scientific evidence was further clarified in two subsequent Supreme Court decisions: *General Electric Co. v. Joiner*⁸⁸ and *Kumho Tire Co. v. Carmichael*.⁸⁹

In *General Electric Co. v. Joiner*, the plaintiff, an electrician, alleged that workplace exposure to polychlorinated biphenyls promoted his development of small cell lung cancer.⁹⁰ The Supreme Court affirmed the district court's ruling, which excluded the four epidemiological studies the plaintiff's experts relied on because they were not a sufficient basis for their opinions.⁹¹ The Court noted that neither *Daubert* nor the Federal Rules of Evidence required a trial court to admit an expert opinion, which rests solely on the authority of the expert witness and is unsupported by the epidemiological data.⁹² Thus, "too great an analytical gap between the data and the opinion proffered," warrants exclusion of expert opinion evidence.⁹³

Later, in *Kumho Tire Co. v. Carmichael*, the Supreme Court extended *Daubert's* "gatekeeping obligation" to all expert testimony and concluded that *Daubert's* reliability inquiry is a flexible one.⁹⁴ Therefore, instead of using *Daubert's* factors

⁸⁶ Although peer review and publication is an essential component of the research process, it does not automatically attest to a study's reliability. *The Role of Epidemiology in Decision-Making*, *supra* note 69, at 18–19; *see also* Brief for Kenneth Rothman et al. as Amici Curiae Supporting Petitioners, *Daubert v. Merrell Dow Pharm., Inc.*, 509 U.S. 579 (1993) (No. 92-102), 1992 WL 12006438, at *3 (arguing that (1) statistical significance is not necessary as a requirement for drawing causal inferences from epidemiologic data; (2) by focusing on such inappropriate criteria, a court forecloses testimony about inferences that can be drawn from a combination of studies with statistically insignificant results; and (3) by suggesting an expert's "reanalysis" of published data is suspect unless published, a court "fundamentally misconstrues how epidemiologists in the real world of science function on a daily basis"). Publication deadlines often cause the review to be performed by someone less qualified in the investigator's own field and the sheer number of scientific journals increases the likelihood that a research paper can get published somewhere. *The Role of Epidemiology in Decision-Making*, *supra* note 69, at 18–19.

⁸⁷ *Daubert*, 509 U.S. at 593–94.

⁸⁸ *General Electric Co. v. Joiner*, 522 U.S. 136 (1997).

⁸⁹ *Kumho Tire Co. v. Carmichael*, 526 U.S. 137 (1999).

⁹⁰ *Joiner*, 522 U.S. at 139. The plaintiff, Robert Joiner, began working as an electrician in 1973. *Id.* at 193. His job required him to work with and around dielectric fluid, later determined to be contaminated with polychlorinated biphenyls (PCBs). *Id.* "PCB's are widely known to be hazardous to human health" and were banned by Congress in 1978. *Id.*

⁹¹ *Id.* at 147–48.

⁹² *Id.* at 146 ("But nothing in either *Daubert* or the Federal Rules of Evidence requires a district court to admit opinion evidence which is connected to existing data only by the *ipse dixit* of the expert").

⁹³ *Id.*

⁹⁴ *Kumho Tire Co.*, 526 U.S. at 141, 150.

as a definitive “checklist,” a trial judge must consider the specific circumstances and issues of each case to determine the appropriate standard of admissibility.⁹⁵

The following subparts examine how *Daubert* and its progeny have been interpreted to create a higher threshold for the admission of epidemiologic evidence, and how this poses a problem for plaintiffs attempting to prove causation in product liability cases.

A. Texas’s Strict Criteria for Determining Scientific Reliability

Various courts since *Daubert* have construed the Court’s ruling as placing stringent standards on the scientific reliability of epidemiologic evidence required to support a finding of causation.⁹⁶ The Texas Supreme Court adopted the strictest of these interpretations in *Merrell Dow Pharmaceuticals, Inc. v. Havner*.⁹⁷ In another case alleging that maternal ingestion of the anti-nausea drug, Bendectin, caused birth defects, Texas’s highest court grappled with the issue of whether the plaintiff’s causation evidence was scientifically reliable.⁹⁸ Here, the court held that if an expert’s testimony is unreliable, then the testimony itself is not evidence.⁹⁹

Further, in determining the scientific reliability of expert testimony, a court must look beyond the expert’s bare opinion.¹⁰⁰ In this regard, the *Havner* court differentiated between the admissibility of scientific evidence and its legal sufficiency in support of a finding of causation. The court declared that Rule 702 of the Federal Rules of Evidence dictates whether an expert’s opinion is admissible to support a finding of causation and offers “substantive guidelines in determining if

⁹⁵ *Id.*; Jerome P. Kassirer & Joe S. Cecil, *Inconsistency in Evidentiary Standards for Medical Testimony*, 288 JAMA 1382, 1383 (2002). For a discussion of the Daubert trilogy and why its admissibility standards necessitate the appointment of forensic experts in criminal cases, see Laurel Gilbert, *Sharpening the Tools of an Adequate Defense: Providing for the Appointment of Experts for Indigent Defendants in Child Death Cases Under Ake v. Oklahoma*, 50 SAN DIEGO L. REV. 469 (2013).

⁹⁶ See FED. JUDICIAL CTR., *supra* note 64, at 561; see also *Merck & Co., Inc. v. Garza*, 347 S.W.3d 256 (Tex. 2011); *Merrell Dow Pharm., Inc. v. Havner*, 953 S.W.2d 706, 717 (Tex. 1997); *Estate of George v. Vermont League of Cities and Towns*, 993 A.2d 367, 378 (Vt. 2010) (finding “that the trial court did not abuse its discretion in considering a relative risk greater than 2.0 as a reasonable and helpful benchmark”); *Daubert v. Merrell Dow Pharm., Inc.*, 43 F.3d 1311, 1321 (9th Cir. 1995) (“For an epidemiological study to show causation under a preponderance standard, ‘the relative risk of limb reduction defects arising from the epidemiological data . . . will, at a minimum, have to exceed ‘2.’”) (citing *DeLuca v. Merrell Dow Pharm., Inc.*, 911 F.2d 941, 958 (3d Cir. 1990)); *Sanderson v. Int’l Flavors & Fragrances, Inc.*, 950 F. Supp. 981, 1000 (C.D. Cal. 1996) (dismissing scientific expert’s probability estimate because it was not founded upon epidemiologic studies demonstrating a relative risk value greater than 2.0).

⁹⁷ *Merrell Dow Pharms., Inc. v. Havner*, 953 S.W.2d 706, 706 (Tex. 1997).

⁹⁸ *Id.* at 711.

⁹⁹ *Id.* at 714. “A flaw in the expert’s reasoning from the [epidemiologic] data may render reliance on a study unreasonable and render the inferences drawn therefrom dubious. Under that circumstance, the expert’s scientific testimony is unreliable and, legally, no evidence.” *Id.*

¹⁰⁰ *Id.* at 711–12.

the expert testimony is some evidence of probative value.”¹⁰¹ Thus, if a claimant’s evidence is deemed admissible, a court should then evaluate whether it is legally sufficient to support a finding of causation based on the totality of the claimant’s available evidence.¹⁰²

In order to be legally sufficient, a litigant must offer epidemiologic studies that not only show a “substantially elevated risk” of disease, but must also demonstrate that they are similar to the study’s participants.¹⁰³ Moreover, if there are alternative causes of the disease, the litigant must introduce evidence negating those causes.¹⁰⁴ Further, in rejecting the reliability of the Havners’ epidemiologic evidence, the Texas Supreme Court established several guidelines in considering its legal sufficiency: (1) each epidemiologic study relied upon must report a statistically significant relative risk¹⁰⁵ of at least 2.0;¹⁰⁶ (2) each study must be published or otherwise subjected to the peer review process; (3) the study must not have been prepared for litigation purposes; and (4) the claimant must provide at least two properly designed epidemiologic studies that meet the above mentioned criteria.¹⁰⁷

The surprising premise behind *Havner’s* stringent requirements was the notion that any study that did not find a statistically significant relative risk greater than 2.0

¹⁰¹ *Id.* These guidelines included: (1) the extent to which the theory has been tested; (2) the extent to which the technique depends upon subjective interpretation; (3) whether the theory has been subjected to peer review; (4) the technique’s rate of error; (5) whether the theory or technique is generally accepted as valid by the scientific community; and (6) the technique’s non-judicial uses. *Id.* at 712 (citing *E.I. du Pont de Nemours & Co. v. Robinson*, 923 S.W.2d 549, 557 (Tex. 1995)).

¹⁰² *Havner*, 953 S.W.2d at 720.

¹⁰³ *Id.*

¹⁰⁴ *Id.*

¹⁰⁵ Relative risk is an epidemiologic measure of association, which compares the rate of disease development among a group of individuals that were exposed to a particular risk factor and a group of individuals that were not exposed. See ANTONY STEWART, BASIC STATISTICS AND EPIDEMIOLOGY: A PRACTICAL GUIDE 113 (3d ed. 2010). Relative risk is calculated by dividing the disease occurrence in the exposed group by the disease occurrence in the non-exposed group. *Id.* If the relative risk value is greater than 1.0, then there is an increased risk of developing disease if one is exposed to the risk factor. *Id.*

¹⁰⁶ Recognizing the difficulty in equating scientific measures of association with legal standards, the court in *Havner* nevertheless ruled that only properly designed and executed epidemiologic studies finding that exposure more than doubled the risk of injury could be used to support a finding of causation in toxic tort cases. *Merrell Dow Pharms., Inc. v. Havner*, 953 S.W.2d 706, 17 (Tex. 1997). The court explained, however, that a relative risk greater than 2.0 is not a “litmus test,” and that a single epidemiologic study is legally insufficient to prove causation. *Id.* at 718. Indeed, the court acknowledged that a study may have a high relative risk even in the absence of a causal relationship. *Id.*

¹⁰⁷ See *id.* at 725–27.

was unreliable.¹⁰⁸ Thus, an expert's opinion that relied on that study was also unreliable and inadmissible as evidence to prove causation.¹⁰⁹

B. The Problems with a Judge-Made Threshold for Admissibility of Epidemiologic Evidence

Findings of liability in the context of toxic torts are rare because the judicial system has erected seemingly insurmountable procedural and substantive barriers to bring a claim, the most daunting of which is proving causation.¹¹⁰ Judicially created tests for the admissibility of epidemiologic evidence, such as the one developed by the Texas Supreme Court in *Havner*, place an unreasonable burden on plaintiffs and frequently result in the dismissal of otherwise meritorious claims.¹¹¹

¹⁰⁸ See *Merck & Co., Inc. v. Garza*, 347 S.W.3d 256, 265 (Tex. 2011) (explaining the court's *Havner* holding).

¹⁰⁹ See *id.*; see also *Havner*, 953 S.W.2d at 714 ("If the foundational data underlying the opinion testimony are unreliable, an expert will not be permitted to base an opinion on that data because any opinion drawn from that data is likewise unreliable."). Later, in *Merck & Co. v. Garza*, the Texas Supreme Court reaffirmed the *Havner*'s requirements for determining whether epidemiological evidence is scientifically reliable to prove causation. *Garza*, 347 S.W.3d 256, 259 (Tex. 2011). Leonel Garza, plaintiffs' decedent, had a long history of heart disease and was prescribed Vioxx, an anti-inflammatory drug, marketed by the defendant, Merck & Co. as a pain reliever. *Id.* Mr. Garza ingested 25 mg Vioxx a day for twenty-five days until his death. *Id.* In a products liability lawsuit against Merck & Co., plaintiffs introduced clinical trial studies showing an increased risk of cardiovascular disease as proof that Vioxx caused Mr. Garza's death. See *id.* at 259, 262. Although the court acknowledged the increased reliability of clinical trials, as opposed to observational epidemiological studies, it concluded that *both* types of studies must report a "statistically significant doubling of the risk" in order to meet the more likely than not standard of proof. *Id.* at 263. The Texas Supreme Court opted not to follow the holdings of other courts that had determined epidemiologic studies with a relative risk *less than* 2.0 could suffice if supplemented with other evidence of causation. *Id.* at 265 (citing *Havner*, 953 S.W.2d at 715). Other courts favor a more flexible *Daubert* inquiry over bright-line rules and view a relative risk (or odds ratio) value of 2.0 as one factor among many that the court can consider in determining the reliability of an expert's opinion. See, e.g., *Pritchard v. Dow Agro Sciences*, 705 F. Supp. 2d 471, 486 (W.D. Pa. 2010); see also *Magistrini v. One Hour Martinizing Dry Cleaning*, 180 F. Supp. 2d 584, 606 (D.N.J. 2002) ("[A] relative risk of 2.0 is not so much a password to a finding of causation as one piece of evidence, among others, for the court to consider in determining whether an expert has employed a sound methodology in reaching his or her conclusion.") (quoting *Landrigan v. Celotex Corp.*, 605 A.2d 1079, 1087 (N.J. 1992)).

¹¹⁰ Conway-Jones, *supra* note 58, at 876.

¹¹¹ *Id.*; see RESTATEMENT (THIRD) OF TORTS: LIAB. FOR PHYSICAL & EMOTIONAL HARM § 28 cmt. c(1) (2010) (advising that courts should be cautious about adopting certain scientific principles, taken out of context, in order to formulate bright-line legal rules because scientific standards for the sufficiency of evidence to establish a proposition may not be appropriate for the law and because causal inferences require judgment and interpretation); see also FAULT LINES: TORT LAW AS CULTURAL PRACTICE 273 (David M. Engel & Michael McCann eds., 2009) ("Plaintiffs can't achieve epidemiological studies by themselves, epidemiology is not the be all and end all of causal inquiry, and the courts—by and large—have turned their 'gatekeeping' function into more of a 'search and destroy' mission."); Kassirer & Cecil, *supra* note 95, at 1382 ("[C]ourts appear to be asserting standards that they attribute to the medical

By equating a relative risk of 2.0 as meeting the more likely than not burden of proof in civil cases,¹¹² courts mistakenly believe that a relative risk value greater than 2.0 is sufficient evidence for a finding of specific causation.¹¹³ Consistent with the decisions of the Ninth and Tenth Circuits, some courts have deemed as inadmissible any epidemiologic evidence that does not show at least a statistically significant doubling of the risk.¹¹⁴

Commentators have also noted that courts are increasingly ignoring other scientific evidence (such as animal studies, differential diagnoses, and chemical structure comparisons) and over-emphasizing epidemiological evidence that demonstrates a risk ratio greater than 2.0.¹¹⁵ There is an inherent problem in relying too heavily on a bright-line quantitative threshold for admissibility because statistical methods alone cannot establish proof of causation. While the concept of "doubling the risk" appears reasonable, some epidemiologists note that it does not represent a more likely than not probability that an exposure will cause disease.¹¹⁶ This is because the "causal significance of an association is a matter of judgment which goes beyond any statement of statistical probability."¹¹⁷ Indeed, the fundamental problem of relying on epidemiologic evidence alone is that causal inferences can be drawn from such studies only when coupled with additional data.¹¹⁸

By ignoring scientific evidence that does not meet an arbitrary quantitative standard, courts deprive juries of the opportunity to aggregate multiple pieces of

profession, but that are inconsistent and sometimes more demanding than actual medical practice. As a result, plaintiffs seeking compensation for an illness attributed to a toxic exposure lose the opportunity to present their evidence to a jury.").

¹¹² See Vern R. Walker, *Restoring the Individual Plaintiff to Tort Law by Rejecting "Junk Logic" About Specific Causation*, 56 ALA. L. REV. 381, 473 n. 320 (2004) ("A relative risk greater than '2' means that the disease more likely than not was caused by the event.").

¹¹³ *Id.* at 468.

¹¹⁴ *In re Breast Implant Litig.*, 11 F. Supp. 2d 1217, 1224–26 (D. Colo. 1998) (finding that if epidemiologic evidence did not show that breast implants doubled the risk of disease, then plaintiff's causation evidence was inadmissible); *Hall v. Baxter Healthcare Corp.*, 947 F. Supp. 1387, 1403 (D. Or. 1996); see also *In re Agent Orange Prod. Liab. Litig.*, 597 F. Supp. 740, 748 (E.D.N.Y. 1984) (declaring that plaintiffs must prove at least a two-fold increase in the incidence of disease allegedly caused by exposure to toxic substance). The court in *In re Breast Implant Litigation*, noted that of at least seventeen epidemiological studies of breast implants published in peer-reviewed medical journals, none consistently showed a significant elevation of risk over 2.0. *In re Breast Implant Litig.*, 11 F. Supp. 2d at 1227. Therefore, the court concluded that breast implants did *not* cause any of the diseases that plaintiffs alleged. *Id.* (emphasis added).

¹¹⁵ David Egilman, Joyce Kim & Molly Biklen, *Proving Causation: The Use and Abuse of Medical and Scientific Evidence Inside the Courtroom—An Epidemiologist's Critique of the Judicial Interpretation of the Daubert Ruling*, 58 FOOD & DRUG L.J. 223, 224 (2003).

¹¹⁶ *Id.* at 229.

¹¹⁷ *Smoking and Health: Report of the Advisory Committee to the Surgeon General of the Public Health Service*, U.S. DEPARTMENT OF HEALTH, EDUCATION & WELFARE 20 (1984), available at <http://profiles.nlm.nih.gov/ps/access/NNBBMQ.pdf>.

¹¹⁸ *Id.*

evidence in determining causation.¹¹⁹ Taking advantage of the stringent causation hurdles faced by plaintiffs, product manufacturers have urged courts to evaluate each type of study (animal, epidemiological, pathological, clinical, etc.) independently from other forms of evidence, thus attacking the very means of proving causation.¹²⁰

V. AN EPIDEMIOLOGIC STUDY SHOULD BE EVALUATED ON THE BASIS OF ITS DESIGN, CONDUCT, AND ANALYSIS.

Judicially created admissibility tests should be substituted with a comprehensive approach that mirrors how practitioners derive causal inferences in the field.¹²¹ This Comment suggests that instead of relying on arbitrary cut-offs for determining the admissibility of epidemiologic evidence, judges should evaluate the validity¹²² of an epidemiologic study on the basis of its design, conduct, and analysis.¹²³ In doing so, it further proposes, as a rubric for assessing study validity, a series of questions that

¹¹⁹ See Egilman et al., *supra* note 115, at 224.

¹²⁰ *Id.* at 231; see Kassirer & Cecil, *supra* note 95, at 1383–84 (“[A]ssessment of evidence and causal inferences depend on accumulating *all* potentially relevant evidence and making a subjective judgment about the strength of the evidence.”).

¹²¹ For example, physicians practicing the doctrine of evidence-based medicine use a “current best evidence” approach when making decisions about individual patient care. Sean C. Grondin & Colin Schieman, *Evidence-Based Medicine: Levels of Evidence and Evaluation Systems, Difficult Decisions*, in THORACIC SURGERY 13 (M.K. Ferguson ed., 2011). A tenet of evidence-based medicine is that a hierarchy of evidence exists based upon the soundness of the study’s methodology and the subsequent strength of any inferences drawn from it. *Id.* Evidence-based medicine recognizes that various forms of evidence may each provide appropriate clinical recommendations, but that some forms are more reliable than others. *Id.* at 14. Consequently, a hierarchy of the strength of evidence has been used to guide clinical decision making. *Id.*

¹²² Assessing the validity of epidemiological studies amounts to evaluating whether the causal inferences drawn from such studies are warranted. Julia H. Zaccai, *How to Assess Epidemiological Studies*, 80 POSTGRAD MED. J. 140, 140 (2004).

¹²³ *Id.*; *The Role of Epidemiology in Decision-Making*, *supra* note 69, at 21; NAT’L RESEARCH COUNCIL OF THE NAT’L ACADEMIES, HEALTH RISKS FROM EXPOSURE TO LOW LEVELS OF IONIZING RADIATION: BEIR VII PHASE 2 139 (2006) (“The first step in the interpretation of data is to assess the methods used in the study itself.”); Kassirer & Cecil, *supra* note 95, at 1383 (“[E]very [epidemiologic] study must be scrutinized not only for . . . confounders, but for the defects in study design, data quality, and the strength of the statistical correlation.”). A similar approach for summarizing epidemiologic evidence in order to derive a causal assessment is termed the “weight of the evidence approach.” Ronald H. White, et al., *Workshop Report: Evaluation of Epidemiological Data Consistency for Application in Regulatory Risk Assessment*, 6 OPEN EPIDEMIOLOGY J. 1, 6 (2013). This approach takes into account the results of all available studies and assigns “greater ‘weight’ to those considered to have the greatest reliability and validity.” *Id.* The approach advocated in this Comment, however, proposes that only validly conducted epidemiologic studies should be used as a basis from which experts can draw any causal inferences. Another approach to weighing epidemiologic evidence recommends that justifiable criteria must include “outcome ascertainment, exposure measures, and other sources of bias,” rather than set rules designed to assess study quality. *Id.*

fact finders should ask of each epidemiologic study offered into evidence.¹²⁴ If a study is deemed valid under these criteria, then experts may rely on its findings in drawing causal inferences. This Comment also examines how these guidelines can be used to assess the validity of epidemiological studies investigating dietary exposures, particularly the link between HFCS consumption and type 2 diabetes.

A. Assessing the Validity of an Epidemiologic Study.

There are many potential reasons for why a study’s reported measure of association (i.e. relative risk, odds ratio, or risk ratio) does not accurately quantify the true causal relationship between exposure and disease. Two of the most important concepts to consider in evaluating the validity of a study’s results are confounding and bias.¹²⁵ Because chronic disease epidemiology generally utilizes observational methods to detect associations between exposure and disease, uncertainty exists in understanding the causes of various chronic conditions, such as diabetes.¹²⁶ Errors in measurement or research design can produce misleading results; therefore, considerable expertise is needed to assess the quality of a study’s methodology and the validity of its purported findings.¹²⁷ A fact finder must evaluate epidemiologic evidence and the causal inferences derived from them in light of these factors.

1. Did Researchers Attempt to Minimize Confounding?¹²⁸

When an association is observed between a risk factor and disease, epidemiologists must differentiate whether this association is causal or the result of confounding by a third factor.¹²⁹

¹²⁴ The following series of questions designed to assess study validity borrow from the ideas of Julia H. Zaccai, as described in her article, “How to Assess Epidemiological Studies.” Zaccai, *supra* note 122, at 143–44.

¹²⁵ *E.g.*, CHRONIC DISEASE EPIDEMIOLOGY AND CONTROL 43 (Patrick L. Remington, Ross C. Brownson & Mark V. Wegner, eds., 3d ed. 2010).

¹²⁶ *See id.*

¹²⁷ *Id.*

¹²⁸ NAT’L RESEARCH COUNCIL OF THE NAT’L ACADEMIES, *supra* note 123, at 139 (recommending that readers consider whether there is evidence that the study addressed the potential confounding influences of other factors).

¹²⁹ GORDIS, *supra* note 67, at 228. In *Schwab v. Phillip Morris USA, Inc.*, the court examined the failure of early epidemiological studies exploring the association between lung cancer and low tar cigarettes to account for confounding variables. 449 F. Supp. 2d 992, 1199–1200 (E.D.N.Y.), *rev’d on other grounds* 552 F.3d 215 (2d Cir. 2008). Early epidemiologic studies concluded that reduced tar cigarettes were safer than their higher tar or “full flavor” counterparts. *Id.* However, these studies did not consider the possibility that people who smoked low tar cigarettes were also more likely to have healthier diets, exercise more, or better protect themselves from hazardous occupational exposures, than smokers of higher tar cigarettes. *Id.* Thus, the health consciousness of low tar cigarette smokers was a potential confounding variable because their healthier lifestyle, rather than their use of low tar cigarettes, could have accounted for their lower incidence of lung cancer. *Id.*

Confounding is a distortion of the estimated effect of an exposure on an outcome due to the presence of an extraneous factor that is associated with both the outcome and the exposure.¹³⁰ A confounding variable is both: (1) a known independent risk factor for the study disease; and (2) simultaneously associated with the exposure in question, but not a consequence of exposure.¹³¹

Confounding is a major source of error in nutritional epidemiology because it is difficult to distinguish between diet and other life style factors that contribute to obesity and its associated health consequences, such as type 2 diabetes. The distortion caused by a confounding variable can be so large as to mask the true effect of the exposure on the disease.¹³² Therefore, readers of epidemiologic studies must assess the possible role that confounding may have played in the study by considering whether any important lifestyle factors have been taken into account in the design and analysis phase of the study.¹³³ If a study fails to control for confounding variables, any reported findings should be viewed with skepticism.

2. Were There Any Biases that Affected the Study's Validity?

Inherent within each epidemiological study is the potential for bias, which is "any systematic error in the design, conduct, or analysis of a study that results in a mistaken estimate of the exposure's effect on the risk of disease."¹³⁴ Bias in an epidemiologic study can distort the true association between exposure and disease and affects the reliability of the study's results.¹³⁵ Although researchers can attempt to minimize bias by carefully planning their study's design and analysis, it is impossible to eliminate completely.

The presence of bias, however, does not automatically mean that the study's results should be rejected. Instead, each bias should be individually identified and addressed before a study's results can be used as evidence of a causal relationship. Epidemiologic studies investigating the role of nutrition in the development of chronic disease are prone to two types of biases: selection and recall bias.

¹³⁰ LAST, *supra* note, 65 at 37. A common example of confounding is illustrated in the relationship between coffee drinking and pancreatic cancer. GORDIS, *supra* note 67, at 228. In this case, smoking is the confounding variable because (1) it is a known risk factor for pancreatic cancer; and (2) is concurrently associated with coffee drinking, but is not a result of it. *Id.* Thus, the apparent association between coffee consumption and pancreatic cancer could be either that coffee drinking actually causes pancreatic cancer or that individuals who drink coffee are also more likely to smoke cigarettes, a recognized risk factor for cancer. *Id.*

¹³¹ GORDIS, *supra* note 67, at 228; JAMES J. SCHLESSELMAN, CASE-CONTROL STUDIES: DESIGN, CONDUCT, AND ANALYSIS 58 (1982) ("A confounder's association with the study disease may be either cause-and-effect or a noncausal relation resulting from the confounder's association with causal factors other than the study exposure.").

¹³² Zaccai, *supra* note 122, at 141 ("The distortion introduced by a confounding factor can be large and it can lead to overestimation or underestimation of an effect . . .").

¹³³ *Id.*

¹³⁴ SCHLESSELMAN, *supra* note 131, at 124.

¹³⁵ Zaccai, *supra* note 122, at 140.

a. Did Subject Recruitment Methods Minimize Selection Bias?¹³⁶

Ideally, investigators want study groups to be comparable on every possible variable besides the exposure of interest. Unfortunately, the procedures used to select subjects and the factors that influence study participation are potential sources of error that can distort a study’s findings.¹³⁷ Selection bias is error due to systematic differences in characteristics between subjects and non-subjects.¹³⁸

In nutritional studies, selection bias occurs when a study’s recruitment methods systematically exclude or over-represent certain types of subjects, such that the study’s results are not generalizable to the overall population.¹³⁹ Therefore, in a *case-control* study¹⁴⁰ investigating type 2 diabetes and past HFCS exposure, cases and controls should be selected independently of their likelihood to have consumed HFCS in the *past*.¹⁴¹ Similarly, in a *cohort* study¹⁴² examining HFCS consumption and *future* development of type 2 diabetes, exposed and nonexposed subjects should be chosen independently of their risk for developing diabetes.¹⁴³

¹³⁶ NAT’L RESEARCH COUNCIL OF THE NAT’L ACADEMIES, *supra* note 123, at 139 (suggesting that one of the first steps in the interpretation of data is to consider whether there is evidence that selection bias has been avoided in enrolling the study subjects).

¹³⁷ KENNETH J. ROTHMAN, SANDER GREENLAND & TIMOTHY L. LASH, MODERN EPIDEMIOLOGY 134 (3d ed. 2008). Dr. Ann McKee, a leading researcher of the long-term consequences of repetitive brain trauma and its role in the development of chronic traumatic encephalopathy (CTE) among football players, addressed the possibility of selection bias in her autopsy sample of deceased football players. *Frontline: A League of Denial* (PBS television broadcast Oct. 8, 2013), *available at* <http://www.pbs.org/wgbh/pages/frontline/league-of-denial/>. She described selection bias as the greater likelihood of families of a player who they suspect is affected by CTE to donate their brain to CTE research than a family of a player with no symptoms at all. *Id.*

¹³⁸ Zaccai, *supra* note 122, at 140; LAST, *supra* note 65, at 166. In the case of *In re “Agent Orange” Prod. Liab. Litig.*, the court discussed selection bias in the context of a cohort study consisting of young, healthy men who served in Vietnam. *In re “Agent Orange” Prod. Liab. Litig.*, 597 F. Supp. 740, 783 (E.D.N.Y. 1984), *aff’d* 818 F.2d 145 (2d Cir. 1987). The court found that comparing the mortality rate of the cohort and that of a control group comprised of civilians made it difficult to obtain useful data. *Id.* The young males in the cohort “were a highly selected, healthy group so that the expected mortality was relatively slight in their early ages and a comparison with base civilian populations difficult.” *Id.*

¹³⁹ For example, 24-hour recall interviews that are only conducted in English would necessarily exclude from participation, non-English speaking individuals.

¹⁴⁰ *See infra* note 173.

¹⁴¹ *See* FED. JUDICIAL CTR., *supra* note 64, at 584.

¹⁴² *See infra* note 170.

¹⁴³ *See* FED. JUDICIAL CTR., *supra* note 64, at 584.

b. If Performing a Case-Control Study, Did Researchers Adopt Measures to Prevent Recall Bias?

Recall bias occurs when there is a difference, among cases and controls, in being able to accurately recall past events or experiences.¹⁴⁴ The potential for recall bias to distort a measure of association is a major concern in case-control studies, particularly when the exposure involves nutrient intake.¹⁴⁵ Diagnosis can affect subject reporting by improving memory or provoking false memories of exposure.¹⁴⁶ For instance, *diseased* subjects are more likely able to remember what foods and beverages they consumed and may, in their search for the cause, overemphasize the role of diet in their disease.¹⁴⁷

Additionally, whereas cohort studies measure diet before the onset of disease, case-control studies obtain exposure information after diagnosis has occurred. Cases are thus, more likely to recollect their prior exposures differently from controls because they are more motivated to participate in the study and determine the cause of their disease.¹⁴⁸

Epidemiologists use measures of association to derive causal inferences, however, the reliability of those inferences must be considered by evaluating whether the potential biases inherent in the study invalidate its results.¹⁴⁹ Therefore, potential biases and their effect on the estimated measure of association must be

¹⁴⁴ See SCHLESSELMAN, *supra* note 131, at 135. A case-control study examines the possible relationship between a particular exposure and a disease by first identifying a group of individuals with the disease (cases) and a similar group of individuals without the disease (controls). GORDIS, *supra* note 67, at 159, 161 (“The hallmark of the case-control study is that it begins with people with the disease (cases) and compares then to people without the disease (controls).”). The relationship between a single exposure to disease is examined by comparing cases and controls in regard to how frequently the exposure is present in each group. *Id.* at 150. Using this type of study design, a positive association exists between the exposure and disease when the proportion of individuals who were exposed is higher in cases than in controls. *Id.* at 160.

¹⁴⁵ See Satu Männistö et al., *Diet and the Risk of Breast Cancer in a Case-Control Study: Does the Threat of Disease Have an Influence on Recall Bias?* 52 J. CLIN. EPIDEMIOLOGY 429, 430 (1999).

¹⁴⁶ ROTHMAN ET AL., *supra* note 137, at 111. For example, a mother whose child was born with a congenital malformation will be more likely to identify events or experiences during her pregnancy than a mother of a child born without a birth defect. See generally Martha M. Werler, Barbara R. Pober, Kathryn Nelson & Lewis B. Holms, *Reporting Accuracy Among Mothers of Malformed and Nonmalformed Infants*, 129 AM. J. EPIDEMIOLOGY 415 (1989). Contrasting recollections between mothers may artificially suggest a relationship between an exposure during pregnancy and congenital malformations. *Id.*

¹⁴⁷ Baerbel Bellach & Lenore Kohlmeier, *Energy Adjustment Does Not Control for Differential Recall Bias in Nutritional Epidemiology*, 51 J. CLIN. EPIDEMIOLOGY 393, 393 (1998) (emphasis added).

¹⁴⁸ See *id.*

¹⁴⁹ See FED. JUDICIAL CTR, *supra* note 64, at 583.

identified and weighed to determine whether the bias “exaggerate[d] the real association, dilute[d] it, or . . . completely mask[ed] it.”¹⁵⁰

B. Evaluating the Study’s Design and Conduct

1. Was the Chosen Study Design Appropriate to Answer the Research Hypothesis?¹⁵¹

In the past, epidemiological inquiries of diet and disease consisted of ecological studies, which compared “disease rates in populations with the population per capita consumption of specific dietary factors.”¹⁵² Although ecologic studies have been useful in stimulating research on the link between diet and chronic diseases, their primary drawback is the possibility that confounding factors—such as genetic predisposition, other dietary determinants of disease, and environmental or lifestyle influences—may vary between regions with high and low incidence of disease.¹⁵³ Therefore, such correlational studies cannot yield reliable conclusions regarding the relationships between dietary factors and disease and should be given the least weight when determining causation.¹⁵⁴

¹⁵⁰ *Id.* (“In reviewing the validity of an epidemiologic study, the epidemiologist must identify potential biases and analyze the amount or kind of error that might have been induced by the bias.”).

¹⁵¹ Zaccai, *supra* note 122, at 141 (“Researchers have a choice of several study designs for their investigation and a judgment must be made as to whether their choice is reasonable in relation to the question they wish to consider.”). For a more comprehensive discussion about epidemiologic study designs investigating nutritional exposures and disease outcomes, see ROTHMAN ET AL., *supra* note 137, at 581–86.

¹⁵² WALTER WILLETT, NUTRITIONAL EPIDEMIOLOGY 4 (1998). Such studies are advantageous because they reveal large contrasts in international dietary intake, the use of average dietary consumption measures offsets changing dietary patterns over time, and large population samples are subject to less random error. *Id.* at 4–5. For an example of an ecological study investigating the relationship between availability of high fructose corn syrup and prevalence of type 2 diabetes, see generally Goran et al., *supra* note 29, at 1.

¹⁵³ WILLETT, *supra* note 152, at 4–5. Another limitation of all ecological studies is the potential for what is known as the “ecological fallacy.” It is “[a]n error in inference due to failure to distinguish between different levels of organization.” LAST, *supra* note 65, at 56–57. Thus, a correlation between variables based on group characteristics may not necessarily mean there is a correlation between variables at the individual level. For example, suppose at the population level, researchers found a correlation between quality of drinking water and deaths due to heart disease in a certain region, it would be an ecological fallacy to infer that consuming low quality drinking necessarily influences an individual’s chance of dying from heart disease. *Id.* at 57. For more discussion of biases in ecologic studies, see Sander Greenland & James Robins, *Ecologic Studies—Biases, Misconceptions, and Counterexamples*, 139 AM. J. EPIDEMIOLOGY 747–60 (1994).

¹⁵⁴ See WILLETT, *supra* note 152, at 5; FED. JUDICIAL CTR., *supra* note 64, at 561 (“[Ecological] studies may be useful for identifying associations, but they rarely provide definitive causal answers.”). In *Cook v. Rockwell Int’l Corp.*, plaintiff’s expert conducted an ecological study assessing the pattern of cancer incidence among residents living near a nuclear weapons plant. *Cook v. Rockwell Int’l Corp.*, 580 F. Supp. 2d 1071, 1095–98 (D. Colo. 2006). Defendants argued that the expert’s testimony was inadmissible because he

Case-control¹⁵⁵ and cohort studies¹⁵⁶ can avoid many of the weaknesses inherent in ecological studies because confounding can be neutralized in either the design or analysis phases.¹⁵⁷ Although case-controls studies are generally easier to perform than cohort studies—because of its smaller sample size and lack of follow up—the inherent difficulties in being able to recall past food consumption makes the results of a case-control study unreliable.¹⁵⁸

Not only do case-control study subjects have to recall what they ate, when they ate it, and how much they ate, considering that HFCS concentration differs according to product, subjects must also remember the food product's brand name. Additionally, because HFCS is commonly used to sweeten beverages, subjects would also have to recollect what they drank, how much, and how often they drank it. The inability of people to accurately remember what they ate and drank on a daily basis over an extended period of time introduces systematic error, or recall bias, that will inevitably distort any measure of association derived from the study.¹⁵⁹

Cohort studies present a more promising methodology for elucidating a causal relationship between HFCS consumption and type 2 diabetes. One of the most

used an ecologic study as his method of analysis. *Id.* The court noted that such studies were useful for establishing associations but provided relatively weak evidence for establishing a conclusive causal relationship between a certain exposure and disease. *Id.* The court, however, held that this relative weakness as to causation went to the weight to be accorded the study rather than its admissibility. *Id.*

¹⁵⁵ A case-control study compares persons with the disease of interest (cases) and persons without the disease (controls). “[T]he past history of exposure to a suspected risk factor is compared between ‘cases’ and ‘controls.’” *E.g.*, LAST, *supra* note 65, at 22. A case-control study is termed “retrospective” because the study begins after the onset of disease and looks backwards in time to investigate exposure to suspected causal factors. *Id.* The measure of association derived from a case-control study is termed the odds ratio. *Id.* In this context, the odds ratio represents the odds that a person with type 2 diabetes consumed HFCS as opposed to a person without type 2 diabetes and thus asks the question: “What are the odds that a case was exposed to the study risk factor?” GORDIS, *supra* note 67, at 183 (emphasis added); see STEWART, *supra* note 105, at 115 (calculating odds ratio by dividing the odds that subjects with disease were exposed to risk factor by the odds that subjects without the disease were exposed to the risk factor).

¹⁵⁶ A cohort study follows a group of healthy individuals who are, have been, or in the future may be exposed or not exposed to a postulated risk factor. *E.g.*, LAST, *supra* note 65, at 33–34. The frequency of the outcome variable is compared between exposed and unexposed groups. *Id.* A cohort study is prospective in its design because it follows exposed and unexposed individuals forward in time to see who develops the disease outcome. *Id.* If the frequency of disease development is higher in exposed persons than in unexposed persons, it may suggest causality between the exposure and disease variables. *Id.*

¹⁵⁷ WILLETT, *supra* note 152, at 6.

¹⁵⁸ *Id.* (“[C]onsistently valid results may be difficult to obtain from case-control studies of dietary factors and disease because of the inherent potential for methodologic bias.”).

¹⁵⁹ See Karin B. Michels, *Nutritional Epidemiology—Past, Present, Future*, 32 INT’L J. EPIDEMIOLOGY 486 (2003); see also ROTHMAN ET AL., *supra* note 137, at 582; Bellach & Kohlmeier, *supra* note 147, at 398 (“The concern that cases and controls report their energy intakes with different degrees of error remains a critical consideration that must be addressed through improved measures.”).

attractive features of a cohort study design is its ability to directly measure risk of disease development.¹⁶⁰ Moreover, due to its prospective nature, dietary information is collected before the onset of disease and as the study progresses, resulting in more accurate dietary assessments. Because a cohort study is less susceptible to the potential sources of bias associated with case-control studies, it provides more reliable results from which to draw causal inferences.¹⁶¹ Therefore, among the three study designs discussed, the results of a validly conducted cohort study should be given the greatest evidentiary weight.

2. How Were Study Subjects Chosen?

The composition of a study population determines the generalizability of its results. A study is “generalizable” when its findings can be extrapolated to a target population beyond the subjects of the study itself.¹⁶² In order for readers to assess the generalizability of a study’s results, researchers must describe the characteristics of the study population, including how many and under what circumstances participants were excluded from subsequent statistical analysis.¹⁶³ What was the study’s attrition rate?¹⁶⁴ Was it so high that the main characteristics of the study and control groups changed significantly?¹⁶⁵

A study with a high attrition rate, which reduces its ability to demonstrate an association¹⁶⁶ and offsets differences in exposure between study groups, should be

¹⁶⁰ Cohort studies, which follow non-diseased exposed and unexposed subjects forward in time in order to see whether or not disease develops, is the preferred method to assess risk. GORDIS, *supra* note 67, at 149. Because the study begins with non-diseased subjects, it can establish a temporal relationship between exposure and disease outcome. *Id.* Drawbacks to this method include cost, its long follow up period, potential for subject attrition, and inapplicability for diseases with low incidence. Zaccai, *supra* note 122, at 142.

¹⁶¹ WILLETT, *supra* note 152, at 9.

¹⁶² LAST, *supra* note 65, at 184–85. Public health research attempts to extrapolate findings to the general public. SAVITZ, *supra* note 66, at 12. Extrapolation, however, does not occur from a single study, rather, findings are generalizable to the larger population when drawn from a series of validly conducted studies. *Id.*

¹⁶³ See Zaccai, *supra* note 122, at 143. Characteristics of study participants include baseline health and sociodemographic information such as sex, race, socioeconomic status, and age. *Id.*

¹⁶⁴ Attrition occurs when subjects permanently leave the study. Vicki L. Kristman, Michael Manno & Pierre Côté, *Methods to Account for Attrition in Longitudinal Data: Do They Work?*, 20 EUR. J. EPIDEMIOLOGY 657, 657 (2005). Because data is no longer collected from these subjects, attrition leads to missing exposure, confounder, or outcome data. *Id.* This loss of data threatens the validity of the study, decreases sample size, and undermines the precision of the risk estimate. *Id.*

¹⁶⁵ Zaccai, *supra* note 122, at 143. These factors are particularly relevant in evaluating nutritional cohort studies because of the study’s long follow up and potential for participant fatigue due to intensive dietary assessments.

¹⁶⁶ The ability to demonstrate an association is termed a study’s power. “The power of a study is determined by several factors, including the frequency of the condition under study, the magnitude of the effect, the study design, and sample size.” LAST, *supra* note 65, at 138.

given less weight in the causal assessment because these factors lead to selection bias and thus, a spurious estimate of association between variables.¹⁶⁷

3. How Did Researchers Define Exposure and Outcome Measures?

Measurement bias occurs when researchers do not accurately define how subjects will be classified in regards to their exposure and disease status.¹⁶⁸ A study's results can be invalidated by simply failing to choose which measurements, whether it is exposure, disease, or confounding variable, will be recorded and how.¹⁶⁹ A clear definition of what constitutes "exposed" or "diseased" must be decided beforehand.¹⁷⁰

For example, in a cohort study evaluating the association between HFCS consumption and diabetes, researchers must decide what level of HFCS consumption qualifies a subject as "exposed" for the purposes of the study. Due to the ubiquitous nature of HFCS in the nation's food supply, most people have consumed HFCS at one time or another. The relevant question thus becomes, what dosage and frequency of HFCS consumption classifies an individual as exposed? This is an important factor to consider when evaluating epidemiological studies because setting exposure to HFCS at abnormally high consumption levels would likely overestimate the effect of HFCS on health outcomes.¹⁷¹ Findings from such a study should, therefore, be cautiously viewed because its results are generalizable only to a population that similarly consumes excessive amounts of HFCS-containing foods or beverages.

4. How was Dietary Intake Measured?¹⁷²

In addition to developing an explicit definition of what constitutes "exposed," researchers must also adopt a reliable method for measuring exposure. A major hurdle in the burgeoning field of nutritional epidemiology is that investigations of dietary exposures have traditionally focused on the role of individual foods in contributing to disease.¹⁷³ The case of HFCS, however, presents a new challenge for researchers because ingredients are not consumed in isolation but, rather, as part of a

¹⁶⁷ See Kristman et al., *supra* note 164, at 657 ("Attrition leads to selection bias when the distribution of confounders and outcome among the exposed and non-exposed subjects is dependent on whether they remained in the study. This bias may lead to spurious risk estimates of unpredictable magnitude and direction.") (citation omitted).

¹⁶⁸ Zaccai, *supra* note 122, at 143.

¹⁶⁹ See *id.*

¹⁷⁰ Therefore, the criteria for including and excluding potential study subjects must be planned and clearly delineated a priori, accounting for their degree of exposure, pre-existing health conditions, relevant demographic characteristics, and disease diagnosis. *Id.*

¹⁷¹ Conversely, setting exposure to low levels of HFCS consumption would likely underestimate or fail to capture the true effect of HFCS on health outcomes. *Id.*

¹⁷² See NAT'L RESEARCH COUNCIL OF THE NAT'L ACADEMIES, *supra* note 123, at 139 (proposing that one of the first questions to consider when interpreting epidemiologic data is whether there is "evidence that information bias has been minimized in assessing exposure or disease").

¹⁷³ Michels, *supra* note 159, at 487.

complex assortment of various other ingredients, nutrients, and food products based upon individual preference.¹⁷⁴ Therefore, the single greatest limitation to investigating the role of a particular food ingredient in disease development is the difficulty of accurately measuring dietary intake.¹⁷⁵

Currently, the most widely used dietary assessment technique is the 24-hour food recall, whereby subjects are asked to report their food intake during the previous day.¹⁷⁶ Food researchers also employ food diaries, which provide “detailed meal-by-meal recordings of types and quantities of foods and beverages consumed during a specified period.”¹⁷⁷ The validity of both methods, however, is questionable due to inaccurate subject recall, highly variable day-to-day intake of specific foods, and the

¹⁷⁴ *Id.* Unlike exposures that are more easily quantifiable—such as cigarettes smoking—an individual’s diet “represents an unusually complex set of exposures that are strongly intercorrelated.” WILLETT, *supra* note 152, at 2; ROTHMAN ET AL., *supra* note 137, at 586 (“The foods we consume each day contain literally thousands of specific chemicals, some known and well quantified, some characterized only poorly, and others completely undescribed and presently unmeasurable.”). Further compounding the difficulty of studying diet is that: 1) nutritional exposures are continuous variables; 2) eating patterns change over time; and 3) individuals are generally not aware of the ingredients of the foods that they eat. WILLETT, *supra* note 152, at 2.

¹⁷⁵ WILLETT, *supra* note 152, at 3; see DAVID A. KESSLER, THE END OF OVEREATING: TAKING CONTROL OF THE INSATIABLE AMERICAN APPETITE 2 (2009) (“Most people do a poor job of reporting what they eat, and overweight people are particularly inaccurate reporters. So much of our eating takes place outside our awareness that it’s easy to underestimate how much food we actually put into our bodies.”). The next frontier of nutritional epidemiological research will thus, attempt to incorporate complex dietary patterns into less traditional analytic models. Michels, *supra* note 159, at 487. Possible statistical models include the use of factor analysis, principal component analysis, cluster analysis, and developing diet scores. *Id.*

¹⁷⁶ ROTHMAN ET AL., *supra* note 137, at 589. The 24-hour recall is the assessment method of choice for most national nutrition surveys because it requires no training or literacy and poses a minimal burden on study subjects. *Id.* The National Health and Nutrition Examination Survey (NHANES) is a national dietary survey “designed to assess the health and nutritional status of adults and children in the United States.” *National Health and Nutrition Examination Survey, 2013-2014: Let’s Improve Our Health*, CENTERS FOR DISEASE CONTROL AND PREVENTION http://www.cdc.gov/nchs/data/nhanes/nhanes_13_14/2013-14_overview_brochure.pdf (last visited Jan. 20, 2014). It collects information on food, nutrient, and supplement intake through the use of 24-hour dietary recalls (conducted in person by trained interviewers), food frequency questionnaires, survey questionnaires, and 26-item dietary screener questionnaires (DSQ). *Dietary Screener Questionnaire in the NHANES 2009-10*, NATIONAL CANCER INSTITUTE (Sept. 3, 2013), <http://appliedresearch.cancer.gov/studies/nhanes/dietscreen/>; *Measuring Guides for the Dietary Recall Interview*, CENTERS FOR DISEASE CONTROL AND PREVENTION (May 20, 2010), http://www.cdc.gov/nchs/nhanes/measuring_guides_dri/measuringguides.htm. To view a sample questionnaire assessing dietary behavior, see *Diet Behavior and Nutrition-DBQ*, CENTERS FOR DISEASE CONTROL AND PREVENTION (Jan. 15, 2013), http://www.cdc.gov/nchs/data/nhanes/nhanes_13_14/DBQ_H.pdf. The wide variety of assessment instruments illustrates the difficulty of accurately capturing dietary intake. *Id.*

¹⁷⁷ ROTHMAN ET AL., *supra* note 137, at 589. This technique should increase the accuracy of subject reporting because foods are recorded as they are consumed, however, it also places a substantial burden on subjects who must weigh and describe their meals and portion sizes. *Id.*

fact that many meals are eaten away from home.¹⁷⁸ Despite these inherent drawbacks, due to their low cost and ease of administration, the 24-hour recall and food diary will continue to be utilized until a more accurate real-time dietary assessment tool is developed. Consequently, nutritional epidemiologic studies that utilize these methods should not be discounted; instead, readers must interpret the study's findings in light of the limitations of measuring dietary exposures.

*C. Is there Evidence of a Causal Relationship using Hill's Criteria?*¹⁷⁹

Finally, demonstrating a strong association between an exposure and disease does not necessarily indicate a causal relationship. The Bradford Hill criteria¹⁸⁰ are often used as a tool for determining whether an observed association in an epidemiologic study reflects a causal association between a risk factor and disease.¹⁸¹ These criteria are: (1) strength of association;¹⁸² (2) consistency;¹⁸³ (3) temporal

¹⁷⁸ *Id.*

¹⁷⁹ *E.g.*, OXFORD TEXTBOOK OF PUBLIC HEALTH 650–652 (Roger Detels et al. eds., 4th ed. 2002) (noting that Hill's criteria is commonly used by epidemiologists to separate causal from non-causal explanations).

¹⁸⁰ The Bradford-Hill Criteria are derived from Sir Austin Bradford Hill, *The Environment and Disease: Association or Causation?* 58 PROCEEDINGS OF THE ROYAL SOC'Y OF MED. 295–99 (1965).

¹⁸¹ *See, e.g.*, FED. JUDICIAL CTR., *supra* note 64, at 597–606; *Soldo v. Sandoz Pharms. Corp.*, 244 F. Supp. 2d 434, 514–16 (W.D. Pa. 2003) (applying the Bradford-Hill criteria to evaluate whether a pharmaceutical drug causes intracerebral hemorrhage); *Amorgianos v. Nat'l R.R. Passenger Corp.*, 137 F. Supp. 2d 147, 168 (E.D.N.Y. 2001) (“Even when an appropriately designed study yields evidence of a statistical association between a given substance and a given health outcome, epidemiologists generally do not accept such an association by itself as proof of a causal relationship between the exposure and outcome. Epidemiologists generally look to several additional criteria to determine whether a statistical association is indeed causal. These criteria are sometimes referred to as the Bradford Hill criteria”) (citing ENVIRONMENTAL AND OCCUPATIONAL MEDICINE 44 (William N. Rom ed., 3d ed. 1998) and CASARETT & DOULL'S TOXICOLOGY: THE BASIC SCIENCE OF POISONS 79 (Curtis D. Klaassen ed., 5th ed. 1996)); *Missouri Pacific R.R. Co. v. Navarro*, 90 S.W.3d 747, 753 (Tex. Ct. App. 2002) (“Dr. Dayal testified that if an epidemiological study finds a relationship between an exposure and a disease, you still must apply the Bradford Hill nine-step criteria.”).

¹⁸² The strength of association is proportional to the magnitude of the estimated risk ratio—the larger the magnitude the more compelling a finding of causation. *See generally* Hill, *supra* note 180, at 296; *but see* ROTHMAN ET AL., *supra* note 137, at 26 (“[A] strong association is neither necessary nor sufficient for causality, and . . . weakness is neither necessary nor sufficient for absence of causality.”).

¹⁸³ Consistency is whether the association has “been repeatedly observed by different persons, in different places, circumstances and times.” Hill, *supra* note 180, at 296; *see also* *Smoking and Health*, *supra* note 117, at 182 (“[Consistency] implies that diverse methods of approach in the study of an association will provide similar conclusions.”).

relationship between exposure and disease;¹⁸⁴ (4) biological plausibility;¹⁸⁵ (5) coherence;¹⁸⁶ (6) specificity;¹⁸⁷ (7) dose-response relationship;¹⁸⁸ (8) analogy;¹⁸⁹ and (9) experimentation.¹⁹⁰ Although this list represents relevant characteristics in evaluating a study, the author himself warned that such "viewpoints" should not be used as a set of rigid guidelines establishing causation.¹⁹¹ Thus, the fulfillment of Hill's criteria is not dispositive.¹⁹² Instead, they should be considered additional factors in evaluating whether a study's results supports a causal association between a dietary exposure and disease outcome.

The findings of poorly designed epidemiologic studies are of little value when deriving causal inferences.¹⁹³ Therefore, interpreters of epidemiologic evidence should not accept a study's findings without considering the methodology used to

¹⁸⁴ This factor "refers to the necessity that the cause precede the effect in time." ROTHMAN ET AL., *supra* note 137, at 28. Temporality and statistical association are the only two criteria that, although not dispositive, are necessary for drawing causal inferences. CAUSATION IN LAW AND MEDICINE 453 (Ian Frecklton & Danuta Mendelson eds., 2002).

¹⁸⁵ Plausibility concerns whether there is a biological mechanism for an association. This criterion, however, is dependent upon the current state of scientific knowledge at the time of assessment. *See* Hill, *supra* note 180, at 298.

¹⁸⁶ Coherence implies that the suspected causal relationship should not contradict known facts in the natural history and biology of the disease. *Id.* (citing *Smoking and Health*, *supra* note 117, at 185).

¹⁸⁷ Considered the weakest of all Hill's viewpoints, "[a]n association is *specific* when a certain exposure is associated with only one disease." GORDIS, *supra* note 67, at 215 (emphasis added).

¹⁸⁸ Also referred to as "biological gradient," this criterion refers to a unidirectional dose-response relationship, whereby increasing exposure results in increasing disease severity. OXFORD TEXTBOOK OF PUBLIC HEALTH, *supra* note 179, at 651. For example, as the number of cigarettes smoked increases so do carcinogen exposure and tissue damage, and thus, greater opportunity for cancer formation. *Id.*

¹⁸⁹ "[A]nalogy provides a source of more elaborate hypotheses about the associations under study[] the absence of [which] only reflects lack of imagination or experience, not the falsity of the hypothesis." OXFORD TEXTBOOK OF PUBLIC HEALTH, *supra* note 179, at 652.

¹⁹⁰ Hill, *supra* note 180, at 295–99. Although Hill was unclear about what he meant by experimentation, he was likely referring to experimental evidence that was the result of removing the suspected causative agent in order to see if disease subsided. OXFORD TEXTBOOK OF PUBLIC HEALTH, *supra* note 179, at 651.

¹⁹¹ *Id.* at 299 ("None of my nine viewpoints can bring indisputable evidence for or against the cause-and-effect hypothesis and none can be required as a *sine qua non*."). Epidemiologists are also in accord with this view. *See, e.g.*, ROTHMAN ET AL., *supra* note 137, at 30 ("Other than [the necessity that the cause precede the effect in time] there is no necessary or sufficient criterion for determining whether an observed association is causal.").

¹⁹² RESTATEMENT (THIRD) OF TORTS: PHYSICAL AND EMOTIONAL HARM § 28 cmt. c(1) (2010) ("No algorithm exists for applying the Hill guidelines to determine whether an association truly reflects a causal relationship or is spurious. Because the inferential process involves assessing multiple unranked factors, some of which may be more or less appropriate with regard to a specific causal assessment, judgment is required.").

¹⁹³ *The Role of Epidemiology in Decision-Making*, *supra* note 69, at 18.

obtain those results.¹⁹⁴ Moreover, arbitrary standards for determining whether epidemiologic studies can serve as the basis for causal inferences should not be set so high that plaintiffs are barred from using this evidence to prove causation.¹⁹⁵ Indeed, it should be recognized that causal inferences “fall along a continuum of interpretation, [where] even . . . methodologically weak studies are fodder for the assessment of causality.”¹⁹⁶ Valid studies that modestly contribute to our understanding of the relationship between an exposure and disease outcome should be valued and viewed as another component in the comprehensive framework of evidence from which to make a causal evaluation.¹⁹⁷

VI. CONDUCT-BASED DEFENSES POTENTIALLY LIMIT THE LIABILITY OF HFCS MANUFACTURERS

A. Assumption of Risk

Even if HFCS plaintiffs can establish causation, they must still overcome conduct-based defenses, which limit the liability of food manufacturers. The assumption of risk doctrine is an affirmative defense that bars recovery in cases where a plaintiff voluntarily assumes a risk of harm from the conduct of the defendant.¹⁹⁸ A plaintiff, who fully understands the risk of harm and voluntarily chooses to consume the food product despite that knowledge, implicitly accepts any risks associated with the consumption and is not entitled to recovery.¹⁹⁹ Therefore, plaintiffs must establish that their decision to eat products containing HFCS was not a decision freely made because “as long as a consumer exercises free choice with appropriate knowledge, liability for negligence will not attach to the manufacturer.”²⁰⁰

Plaintiffs in *S.F. v. Archer-Daniels-Midland*, however, claim that the aggressive attempts of the Corn Refiners Association, a national trade association of corn manufacturers, to undo HFCS’s bad reputation were part of an effort to misrepresent and conceal the science and consequences of HFCS ingestion.²⁰¹ In order for plaintiffs to recover for their injuries, they must demonstrate that HFCS manufacturers did not adequately inform consumers of the risks associated with

¹⁹⁴ Zaccai, *supra* note 122, at 141.

¹⁹⁵ See SAVITZ, *supra* note 66, at 21.

¹⁹⁶ *Id.*

¹⁹⁷ *See id.*

¹⁹⁸ RESTATEMENT (SECOND) OF TORTS § 496A (1965). When a plaintiff fully understands the risk of harm caused by a food product and nevertheless voluntarily chooses to consume it, the plaintiff impliedly accepts the risk and is thus not entitled to any recovery. *Id.* at § 496C.

¹⁹⁹ *Id.* at § 496C.

²⁰⁰ Pelman, 237 F. Supp. 2d at 533.

²⁰¹ Complaint at 10, *S.F. v. Archer-Daniels-Midland Co.*, No. 1:13-cv-634S, 2014 WL 1600414, at *1 (W.D.N.Y. Apr. 21, 2014), 2013 WL 3070911 at *55.

HFCS consumption.²⁰² If consumers were unaware, uninformed, or deliberately misled about the risks of HFCS, then their decisions were not freely made and the assumption of the risk defense is inapplicable.²⁰³

B. Contributory Negligence

Plaintiffs alleging injury from HFCS consumption may also be barred from recovery under the doctrine of contributory negligence. Similar to the assumption of risk doctrine, a person is liable for contributory negligence if he or she knows, or should have known, the circumstances from which the danger arose and still intentionally exposed himself to the danger.²⁰⁴ In other words, although a food manufacturer's negligence is a proximate cause of the plaintiff's type 2 diabetes, he or she will be completely barred from recovering if his or her own conduct fell below the standard of care and proximately caused the same injury.²⁰⁵

²⁰² See John Alan Cohan, *Obesity, Public Policy, and Tort Claims Against Fast-Food Companies*, 12 WIDENER L.J. 103, 127 (2003).

²⁰³ *Id.* The Corn Refiners Association (CRA), a national trade association of corn manufacturers, argues that adverse health consequences, such as obesity, are not caused solely by HFCS consumption but by the overconsumption of unhealthy foods. See generally Veronica Louie, *Masquerading Behind Words: The Corn Refiners Association's Push to Rename High-Fructose Corn Syrup as "Corn Sugar,"* 4 NORTHEASTERN U. L.J. 293, 302 (2012). As support for their contention, they cite to numerous scientific articles that call into question claims of the negative effects of HFCS. *Id.* at 303. The CRA ran national advertisement campaigns to convince consumers that HFCS was equivalent to regular table sugar and in 2010, petitioned the Food and Drug Association to rename HFCS as "corn sugar." *Id.* at 293; see Corn Refiners Association, *Myth vs. Facts*, SWEETSURPRISE.COM, <http://sweetsurprise.com/hfcs-myths-and-facts> (last visited Jan. 21, 2014) ("[HFCS] is basically the same as sugar—both in terms of composition and in the number of calories they contain. Since [HFCS] and sugar are so similar, the human body absorbs them the same way."). In 2012, the Food & Drug Administration denied the CRA's petition to use the term "corn sugar" to describe HFCS because the term "would suggest that HFCS is a solid, dried, and crystallized sweetener obtained from corn." Michael M. Landa, *Response to Petition from Corn Refiners Association to Authorize "Corn Sugar" as an Alternate Common or Usual Name for High Fructose Corn Syrup (HFCS)*, U.S. FOOD & DRUG ADMIN. (May 30, 2012), <http://www.fda.gov/aboutFDA/CentersOffices/OfficeofFoods/CFSAN/CFSANFOIAElectronicReadingRoom/ucm305226.htm>. However, "HFCS is an aqueous solution sweetener derived from corn after enzymatic hydrolysis of cornstarch, followed by enzymatic conversion of glucose . . . to fructose." *Id.*

²⁰⁴ RESTATEMENT (SECOND) OF TORTS § 466 cmt. c and d (1965) (recognizing that assumption of risk and contributory negligence doctrines overlap when the plaintiff voluntarily consents to encounter a known danger arising from the defendant's negligent act).

²⁰⁵ 1 J. LEE & BARRY LINDAHL, MODERN TORT LAW: LIABILITY AND LITIGATION § 10:1 (2d ed. 2013); *Schmidt v. Omaha Pub. Power Dist.*, 515 N.W.2d 756, 792 (Neb. 1994) ("Contributory negligence is conduct for which the plaintiff is responsible, amounting to a breach of the duty imposed upon persons to protect themselves from injury, and which, concurring with actionable negligence on the part of the defendant, is a proximate cause of injury.").

C. Comparative Responsibility

In products liability actions, most jurisdictions apply the doctrine of comparative responsibility.²⁰⁶ Unlike assumption of risk and contributory negligence, comparative responsibility reduces the plaintiff's recovery in proportion to his or her degree of fault.²⁰⁷ Some jurisdictions have adopted a "pure" version of comparative fault that "allocates responsibility to each actor . . . in proportion to the actor's percentage of total fault."²⁰⁸

In order to prevail under this defense, a HFCS manufacturer must demonstrate that the plaintiff acted unreasonably by consuming excess amounts of food products containing HFCS, when he was aware of its potential negative health effects.²⁰⁹ Specifically, that a reasonable person would have learned of the dangers of overconsumption and modified his eating habits to reduce his risk for harm.²¹⁰

Consumers, however, could argue that because they were unaware of the health risks associated with HFCS consumption, they did not voluntarily expose themselves to the danger. Indeed, the Corn Refiners Association's "Sweet Surprise" marketing campaign that attempts to recast HFCS as "corn sugar," may demonstrate that the dangers of HFCS were not within the common knowledge of the average consumer.²¹¹

Furthermore, product misuse, a type of consumer behavior, could play a factor in apportioning liability. HFCS manufacturers may argue that overconsumption of foods containing HFCS is a misuse of the product and that such overconsumption constitutes contributory fault, thereby reducing the plaintiff's recovery.²¹² Although most consumers are generally aware of the relationship between overeating and obesity, the success of these conduct-based affirmative defenses rests on the ability of food manufacturers to prove that consumers had specific knowledge of the risk that the consumption of HFCS containing products could lead to negative health effects, such as diabetes.²¹³

²⁰⁶ RESTATEMENT (THIRD) OF TORTS: PRODS. LIAB. § 17 cmt. a (1998).

²⁰⁷ PROSSER AND KEETON ON THE LAW OF TORTS § 65, at 451 (5th ed. 1984).

²⁰⁸ RESTATEMENT (THIRD) OF TORTS: PRODS. LIAB. § 17 cmt. b (1998); RESTATEMENT (THIRD) OF TORTS: APPORTIONMENT OF LIAB. § 7 (2000) ("Plaintiff's negligence . . . that is a legal cause of an indivisible injury to the plaintiff reduces the [their] recovery in proportion to the share of responsibility the factfinder assigns to the plaintiff . . .").

²⁰⁹ See Richard C. Ausness, *Tell Me What You Eat, and I Will Tell You Whom to Sue: Big Problems Ahead for "Big Food"?*, 39 GA. L. REV. 839, 882 (2005).

²¹⁰ *Id.*

²¹¹ Louie, *supra* note 203, at 293.

²¹² See RESTATEMENT (THIRD) OF TORTS: PRODS. LIAB. § 17 cmt. a (1998). The plaintiffs in *Pelman v. McDonald's Corp.* advanced this argument.

²¹³ See generally Ausness, *supra* note 209, at 883.

VII. APPORTIONING LIABILITY AMONG SEVERAL MANUFACTURERS IS A BARRIER TO RECOVERY FOR HFCS PLAINTIFFS

Because HFCS plaintiffs must directly trace their adverse health outcome to consuming foods made with the defendant’s product, a major hurdle to recovery will be identifying which of several HFCS manufacturers is the actual maker of the HFCS that caused their type 2 diabetes.²¹⁴ Proof of causation, however, is particularly difficult when some, but not all, of the HFCS manufactured by the defendant caused the plaintiff’s injury.²¹⁵

Fortunately, some courts relax the requirement that plaintiffs prove exactly who caused their injury by adopting legal doctrines²¹⁶ that impose collective liability on defendants.²¹⁷ At first glance, the market share liability approach appears to be a potential solution to the inability of plaintiffs to identify which of several defendants manufactured the HFCS that caused their diabetes. This section argues, however, that even if plaintiffs can establish a causal relationship between HFCS and type 2 diabetes, the circumstances that typically warrant a remedy under this doctrine are absent in the case of HFCS litigation.

A. The Market Share Liability Approach

First utilized by the California Supreme Court in *Sindell v. Abbott Laboratories*,²¹⁸ market share liability allows plaintiffs to recover damages when they can identify a group of defendants engaged in the harmful conduct, but cannot

²¹⁴ Tracing diabetes to a specific manufacturer’s HFCS is inherently problematic because consumers have difficulty remembering what they eat. Plaintiffs would have to be able to recall what specific food items they ate, its brand name, and the quantity of the product they consumed. Additionally, plaintiffs’ dietary recall is influenced by their current diet and inherently biased because they are parties to a lawsuit attempting to prove causation. *See id.*

²¹⁵ *Id.* at 870.

²¹⁶ There are several other legal doctrines whereby multiple defendants are held collectively liable for causing a plaintiff’s injuries. One approach that may provide HFCS plaintiffs a remedy is the doctrine of “enterprise liability,” which was first enunciated in *Hall v. E.I. DuPont De Nemours & Co.*, 345 F. Supp. 353 (E.D.N.Y. 1972). This theory is broadly used to mean that losses caused by an enterprise should be borne by it. *Sindell v. Abbott Laboratories*, 607 P.2d 924, 928 n. 9 (Cal. 1980) (citing Howard C. Klemme, *Enterprise Liability Theory of Torts*, 47 U. COLO. L. REV. 153, 158 (1976)). Another approach for assigning collective liability emerged in the case of *Summers v. Tice*, which imposed the burden of identifying the cause of the plaintiff’s injury on multiple defendants. *Summers v. Tice*, 199 P.2d 1, 4 (Cal. 1948); *see also* DOBBS ET AL., *supra* note 59, at § 1921 (“When the plaintiff presents evidence that she suffered a single or indivisible injury at the hands of two or more tortfeasors, the burden is on the party who seeks to avoid responsibility for the entire damages to prove that magnitude of divisible damages.”).

²¹⁷ *See Sindell*, 607 P.2d at 937 (holding that if defendants could not prove that they did not make the DES liability for damages would be apportioned based on market share); Ausness, *supra* note 209, at 870; Allen Rostron, *Beyond Market Share Liability: A Theory of Proportional Share Liability for Nonfungible Products*, 52 UCLA L. REV. 151, 159 (2004).

²¹⁸ *Sindell*, 607 P.2d at 937.

prove which defendant is responsible for their injury.²¹⁹ Under this theory, a plaintiff who is unable to identify the manufacturer of the particular HFCS that caused his or her diabetes can still recover on a proportional basis from each of the defendant manufacturers that may have supplied the HFCS.²²⁰

In *Sindell*, daughters allegedly injured by their mothers' ingestion of diethylstilbestrol (DES) during pregnancy brought suit against DES manufacturers claiming that the drug caused their vaginal and cervical cancers.²²¹ Prior lawsuits seeking to hold DES manufacturers responsible for the product's carcinogenic effects were unsuccessful because plaintiffs could not identify the manufacturer of the drug prescribed to their mothers.²²² Therefore, the issue confronting the court in *Sindell* was whether a plaintiff, who cannot identify the specific manufacturer of the responsible product, may hold a manufacturer of a chemically identical drug liable for her injuries.²²³

Recognizing that the plaintiffs would be barred from recovery altogether if the court rigidly adhered to prior precedent, the court created a remedy for cases of creation of fungible goods whose source cannot be traced to a specific manufacturer.²²⁴ Unless it could demonstrate that it could not have made the DES that caused the plaintiff's injuries, the court apportioned damages among DES manufacturers in proportion to its respective market share at the relevant time.²²⁵

²¹⁹ RESTATEMENT (THIRD) OF TORTS: PHYSICAL AND EMOTIONAL HARM § 28 cmt. p (2010); Rostron, *supra* note 217, at 158; COMPENSATION FOR DAMAGE 24 (Sheila A.M. McLean ed., 1993) ("By this principle, all those who manufactured the generic product may be held responsible for the defect . . . They are not absolved from liability simply because a particular plaintiff cannot identify a particular defendant.").

²²⁰ *See* RESTATEMENT (THIRD) OF TORTS: PHYSICAL AND EMOTIONAL HARM § 28 cmt. p (2010). Market share liability is an extension of the court's holding in the famous case of *Summers v. Tice*, which held that if a party cannot identify which of two or more joint tortfeasors caused a plaintiff's injury, the burden of proof may shift to the defendant to demonstrate that they were not responsible for the harm. *Summers*, 199 P.2d at 4.

²²¹ *Sindell v. Abbott Laboratories*, 607 P.2d 924, 927–28 (Cal. 1980).

²²² *Id.* at 927–28.

²²³ *Id.* at 925. DES manufacturers had to utilize a standard chemical formula for DES outlined in the United States Pharmacopoeia. *Id.* at 933.

²²⁴ *Id.* at 936–37. When a product is fungible, market-share liability is appealing because it provides compensatory relief to plaintiffs and deters future tortious conduct by defendants. RESTATEMENT (THIRD) OF TORTS: LIAB. FOR PHYSICAL AND EMOTIONAL HARM § 28 cmt. p (2010). "If plaintiffs can demonstrate that the marketing and sale of a product was tortious and that the product caused their harm, they have a strong claim for compensation. *Id.* This approach also serves as a deterrent because it imposes liability on the manufacturers of harmful products, even when a long latency period makes identification of the responsible manufacturer impossible. *Id.*

²²⁵ *Id.* at 937. For a discussion of a new proportional liability doctrine that addresses the legal hurdles to recovery for latent diseases caused by diffused environmental hazards *see* Kenneth S. Abraham, Robert L. Rabin & Paul C. Walker, *Enterprise Responsibility for Personal Injury: Further Reflections*, 30 SAN DIEGO L. REV. 333 (1993). Under this approach, "in any case relying on epidemiological proof of causation, proportional liability rules [rather than] traditional all-or-nothing liability rules, would apply." *Id.* at 355. The authors proposed that proportional liability rules would apply when epidemiological evidence demonstrates at

B. Sindell’s “Fungibility” Requirement As a Barrier to Recovery

Courts since *Sindell*, however, have limited the reach of the market share approach by confining its applicability to cases where the product in question is “fungible,” such that “every unit of the product poses an identical degree of risk.”²²⁶ Because market share liability is premised on the notion that identical products pose identical risks of harm, the lack of fungibility of a product is often used to bar plaintiffs from recovery.²²⁷

In the case of HFCS litigation, the fungibility requirement articulated under *Sindell*, precludes a court from using market share data to allocate liability among manufacturers that do not necessarily produce chemically identical HFCS. In an effort to vary the sweetness of their product, manufacturers began altering the fructose content of its HFCS. There are two types of HFCS currently used in food products: HFCS-42 and HFCS-55, each named according to its respective proportion of fructose.²²⁸ Hence, HFCS-42 is 42% fructose.²²⁹ If a dose-response relationship exists between increasing fructose consumption and negative health effects, the risk of adverse health outcomes will vary depending upon the specific concentration of

least a twenty percent probability of causation and “damages be proportional to causation up the point when the probability is at least eighty percent . . . that the defendant’s action caused the injury in question.” *Id.* Consequently, victims who were exposed to an environmental hazard need not prove that it was at least 51% likely that their exposure caused their disease. *Id.* at 353. Rather, if the causal likelihood of exposure, as determined by epidemiological studies, was 35%, then victims would be awarded only 35% of their losses. *See id.*

²²⁶ Rostron, *supra* note 217, at 153.

²²⁷ *Id.* at 153–54; RESTATEMENT (THIRD) OF TORTS: LIAB. FOR PHYSICAL AND EMOTIONAL HARM § 28 (2010).

When market-share liability is limited to fungible products that pose equivalent risks to users who have no reasonable means to prove which manufacturer provided the product that caused the plaintiff’s harm, it has an exceedingly limited reach . . . Only products that cause harm after a lengthy latency period between exposure and development of harm are likely to create the systemic proof problems that market-share liability addresses.

Id. Additional factors in *Sindell* that augured in favor of relaxing the traditional causation requirements were the egregious actions of DES manufacturers and the circumstances of the plaintiffs’ injuries that rendered identification of a specific manufacturer impossible by either party. *See Sindell v. Abbott Laboratories*, 607 P.2d 924, 925–26 (Cal. 1980). DES Manufacturers knew or should have known of the dangers, manufacturers failed to test DES for efficacy and safety, failed to warn consumers of potential carcinogenic effects, and marketed DES on an unlimited basis without authorization from the Food and Drug Administration. *Id.*

²²⁸ Bray et al., *supra* note 23, at 537 (describing two different types of HFCS—HFCS-42 (42% fructose) and HFCS-55 (55% fructose)—made by enzymatic isomerization of glucose to fructose); *see also* Corn Refiners Association, *What is HFCS?*, SWEET SURPRISE, <http://sweetsurprise.com/what-is-hfcs> (last visited Jan. 21, 2014) (“[HFCS], a sugar made from corn, comes in two compositions—HFCS-42 and HFCS-55.”).

²²⁹ Corn Refiners Association, *supra* note 228.

fructose used in the defendant's product.²³⁰ However, because food and beverage manufacturers are not required to specify which variety of HFCS they used in their products, it is difficult to determine whether HFCS manufacturers subject consumers to equal health risks.²³¹

Furthermore, fundamental differences between HFCS and DES preclude a court from using market share liability in the context of HFCS litigation. A major difference between the two is that, unlike DES, HFCS's ability to cause harm is not as easily defined because it is one of many ingredients that comprise certain food products. This is especially true where HFCS, with its differing fructose concentrations, is present in food in varying quantities and inconsistently consumed. DES, however, was a single product, manufactured according to a single industry wide formula, ingested by women within the narrowly defined time frame of pregnancy, and responsible for a particular type of cancer.²³² Because DES posed an equivalent risk of harm, irrespective of its particular manufacturer, the court in *Sindell* was more willing to find that a reasonable approximation of a defendant's liability was its proportion of the market share.²³³

Even if HFCS plaintiffs could establish that the nature of their disease rendered identification of a particular HFCS manufacturer impossible, because HFCS is not chemically identical and because there is no available means to determine which HFCS-variety a manufacturer produced, courts cannot utilize the market share liability approach to provide them a remedy.

VIII. ISSUES OF PERSONAL RESPONSIBILITY AND PUBLIC ACCOUNTABILITY LIE AT THE HEART OF FOOD LAWSUITS

A. Food Litigation as the New Tobacco

Before obesity and its associated health consequences grabbed the nation's attention, tobacco use was a major public health concern. Recent lawsuits against the fast food industry, such as *Pelman v. McDonald's Corp.*, are reminiscent of tobacco litigation.²³⁴ Both include a personal responsibility component, allegations that the industries targeted their advertising to children, and the possibility that food

²³⁰ Indeed, current scientific evidence lends support to the notion that a dose-response relationship exists between fructose concentration in HFCS and negative health effects. See Bray et al., *supra* note 23, at 537 (finding that the increased consumption of soft drinks and concomitant increase in fructose intake from HFCS and sucrose may be an important contributor to the obesity epidemic).

²³¹ See *id.* at 540 ("HFCS is found in most processed foods; however . . . exact compositions are not available from either the manufacturer or any publicly available food composition table.").

²³² See *Sindell*, 607 P.2d at 926.

²³³ *Id.* at 937.

²³⁴ For a more in-depth discussion of the parallels between fast food and tobacco litigation, see generally Joshua Logan Pennel, *Big Food's Trip Down Tobacco Road: What Tobacco's Past Can Indicate About Food's Future*, 27 *BUFF. PUB. INT. L.J.* 101 (2008–2009).

manufacturers, like “Big Tobacco,” knew of the dangers of their products, yet continued to market them.²³⁵

These striking similarities may help assess whether future juries will assign liability to food manufacturers when consumers suffer obesity-related injuries.²³⁶ Early tobacco cases faced initial skepticism from the public, who believed that any negative health effects from tobacco use were the result of a lack of self-control.²³⁷ Similarly, food plaintiffs today face the all too familiar argument of personal responsibility.²³⁸

Lawsuits such as *S.F. v. Archer-Daniels-Midland Co.* reveal that issues of personal autonomy and public accountability affect all food consumers.²³⁹ However, the idea that HFCS manufacturers should be held strictly liable for causing diabetes may not be one that society is ready to accept because it is contrary to societal attitudes regarding individual liberty and personal responsibility.²⁴⁰ These principles reflect the belief that individuals are free to make their own food choices and must take responsibility for the consequences of their actions.²⁴¹ In other words, allowing consumers to blame the food industry for the results of their own bad judgment unfairly shifts responsibility from the individual to the manufacturer.²⁴²

²³⁵ Cohan, *supra* note 202, at 110–13; FAULT LINES, *supra* note 111, at 103–04 (arguing that food litigation followed anti tobacco strategists’ three most effective substantive claims: 1) inadequate disclosure of nutritional content; 2) marketing aimed at impressionable children; and 3) increasing public costs of healthcare due to obesity related conditions); *see generally* Jada J. Fehn, *The Assault on Bad Food: Tobacco-Style Litigation As An Element of the Comprehensive Scheme to Fight Obesity*, 67 FOOD & DRUG L.J. 65, 72–76 (2012) (noting the similarity between tobacco and food litigation).

²³⁶ Cohan, *supra* note 202, at 110. Cigarettes have been successfully recast in a negative light by the “truth campaign,” the largest anti-tobacco movement focused on young people. KESSLER, *supra* note 175, at 247. The campaign promoted the notion that a person’s desire for cigarettes did not come from the individual themselves, but rather from a “manipulative and profit-driven industry.” *Id.* The success of this campaign suggests the possibility that consumers could be willing to hold the food industry responsible for obesity related conditions. *See id.* at 247.

²³⁷ *See Fehn, supra* note 235, at 73.

²³⁸ *Id.* at 75. The theory of “conditioned hypereating,” proposed by David Kessler, calls into question the idea that individuals truly have a choice in what they consume. KESSLER, *supra* note 175, at 145. Kessler posits “chronic exposure to highly palatable foods changes our brains, conditioning us to seek continued stimulation. Over time, [the brain develops a powerful urge] for a combination of sugar, fat, and salt, [which] competes with our conscious capacity to say no.” *Id.*

²³⁹ *See Pelman v. McDonald’s Corp.*, 237 F. Supp. 2d 512, 519 (S.D.N.Y. 2003).

²⁴⁰ *See Ausness, supra* note 209, at 887.

²⁴¹ *Id.* The importance of moderation and avoidance of excessive food intake dates back to the Old Testament, in which God specifically warns against the sin of gluttony. *See Proverbs* 23:2 (New Int’l Version) (“[P]ut a knife to your throat if you are given to gluttony.”); *see also Proverbs* 23:20–21 (New Int’l Version) (“Do not join those who drink too much wine or gorge themselves on meat, for drunkards and gluttons become poor, and drowsiness clothes them in rags.”).

²⁴² This argument, however, becomes less applicable when the injured plaintiffs are children. Children and teenagers do not possess the requisite maturity to understand the

Despite increasing rates of obesity, the notion of personal responsibility still pervades the public mockery of food lawsuits and underscores legislative attempts seeking to hold Americans accountable for what they eat.²⁴³

B. Legislative Attempts to Curb Food Litigation

Recognizing that litigation is a major threat to their industry, food manufacturers lobbied members of Congress to introduce legislation that immunized them from liability.²⁴⁴ The Personal Responsibility in Food Consumption Act of 2005 prevented civil liability actions brought against food manufacturers, marketers, distributors, advertisers, sellers, and trade associations for claims of injury related to an individual's weight gain, obesity, or any health condition related to such.²⁴⁵ The Act asserted that a person's weight gain, obesity, or obesity-related health condition is based on a multitude of factors such that it cannot be attributed to the consumption of any particular food product or beverage.²⁴⁶ It also recognized that "lawsuits seeking to blame food and beverage providers for a person's weight gain, obesity, or

consequences of their eating habits. KAUFMAN, *supra* note 42, at 219–20. Consequently, they are more susceptible to the aggressive tactics that food manufacturers use to market their products. Fehn, *supra* note 235, at 69–70; KAUFMAN, *supra* note 42, at 219–20; KILLER AT LARGE: WHY OBESITY IS AMERICA'S GREATEST THREAT (ShineBox Media Prod. 2008) (suggesting that junk food marketing to children undermines parental authority to determine what their children consume and is a contributory factor to the growing rise in obesity among America's youth). Junk food marketing is a two billion dollar a year effort by the food industry to create brand loyalty among the nation's young teens and children. TEDxTalks, *Marketing Food to Children: Anna Lappe at TEDxManhattan*, YOUTUBE (Mar. 11, 2003), <https://www.youtube.com/watch?v=0bop3D7-dDM>. The food industry seeks to create brand loyalty at a young age because it knows that this loyalty ultimately generates "pester power," which works. *Id.* In fact, 75% of parents reported that they bought something for the first time simply because their children asked them to. *Id.* Juries will be more likely to assign blame to food sellers when children develop obesity-related health problems associated with the consumption of their products. Alderman & Daynard, *supra* note 45, at 85 ("Because children are more vulnerable than adults due to their lower capacity to evaluate advertising objectively, the food industry may be especially vulnerable to claims about misleading advertising directed toward children.").

²⁴³ See Shirleen Holt, *Go Ahead, Splurge on The Bulge, But Any Resulting Fact Is On You*, SEATTLE TIMES, Sept. 9, 2003, at A1, available at <http://www.freerepublic.com/focus/f-news/977597/posts>; see *infra* Part VII(B). Diners at Seattle's 5 Spot restaurant must sign a waiver promising not to file a lawsuit for any weight gain associated with consuming "The Bulge," a calorie-dense "concoction of sugar-coated fried banana, ice cream, macadamia nuts, whipped cream and two kinds of syrup." Holt, *supra* at A1. The waiver states in relevant part, "I promise to release the 5 Spot from all liability of any weight gain that may result from ordering and devouring this sinfully fattening treat. If I have to go to 'fat camp' at some time in my life, I will not mail my bill to the 5 Spot." *Id.*

²⁴⁴ KAUFMAN, *supra* note 42, at 220.

²⁴⁵ Personal Responsibility in Food Consumption Act of 2005, H.R. 554, 109th Cong. § 2(a) (2005).

²⁴⁶ *Id.* at § 2(a)(3).

a health condition . . . are not only legally frivolous and economically damaging, but also harmful to a healthy America.”²⁴⁷

Despite being reintroduced three times, the federal Personal Responsibility in Food Consumption Act failed to gain enough Congressional support.²⁴⁸ Undaunted, the National Restaurant Association, representing large food chains such as McDonald’s, has successfully lobbied state legislatures to pass various “commonsense consumption” laws.²⁴⁹ These laws, dubbed “Cheeseburger Bills,” bar civil lawsuits against food manufacturers seeking recovery for obesity-related health conditions.²⁵⁰ Between 2004 and 2012, twenty-five states enacted Cheeseburger Bills.²⁵¹

Although HFCS litigants may still bring obesity related tort claims against HFCS manufacturers in federal court, the enactment of “Commonsense Consumption” laws by over twenty-five states demonstrates how organized lobbying efforts, huge financial incentives,²⁵² and an increasingly “receptive legislative climate” can effectively shield the food industry from liability.²⁵³

²⁴⁷ *Id.* at § 2(a)(4).

²⁴⁸ Melanie Warner, *The Food Industry Empire Strikes Back*, N.Y. TIMES, July 7, 2005, at CI, [available at http://www.nytimes.com/2005/07/07/business/07food.html?pagewanted=all&_r=0](http://www.nytimes.com/2005/07/07/business/07food.html?pagewanted=all&_r=0).

²⁴⁹ *Id.* As recently as July 18, 2013, North Carolina enacted its Commonsense Consumption Act, barring civil actions against food and beverage manufacturers from claims arising from weight gain, obesity, its associated health conditions, or other conditions likely to result from long-term consumption of food. Commonsense Consumption Act, H.B. 683, 2013 Gen. Assemb. § 99E-42 (N.C. 2013). The Act also prohibits local governments from regulating the size of soft drinks offered for sale. *Id.*

²⁵⁰ Cara L. Wilking & Richard A. Daynard, *Beyond Cheeseburgers: The Impact of Commonsense Consumption Acts on Future Obesity-Related Lawsuits*, 68 FOOD & DRUG L.J. 229, 230 (2013).

²⁵¹ *Id.* Notably, fifteen of the twenty-five states with the highest obesity rates have enacted Commonsense Consumption Acts. *Id.*

²⁵² Warner, *supra* note 248 (“[T]he food and restaurant industry gave a total of \$5.5 million to politicians in the 20 states that have passed laws shielding companies from obesity liability.”).

²⁵³ *Id.*; see generally Wilking & Daynard, *supra* note 250, at 230. Boehmer and colleagues investigated state-level childhood obesity prevention legislation in order to analyze geographic patterns of bill introduction and adoption. Tegan K. Boehmer, et al., *Patterns of Childhood Obesity Prevention Legislation in the United States*, 4 PREVENTING CHRONIC DISEASE 1, 3–4 (July 2007), [available at http://www.cdc.gov/pcd/issues/2007/jul/pdf/06_0082.pdf](http://www.cdc.gov/pcd/issues/2007/jul/pdf/06_0082.pdf). During the three-year study period, 17% of the 717 introduced bills, which related to childhood obesity prevention topics, were adopted by the fifty states. *Id.* at 3. None of these adopted bills, however, sought to hold the food industry responsible. See *id.* Those bills relating to snack and soda taxes, restaurant menu, and product labeling were not adopted by any state legislature. *Id.*

IX. CONCLUSION

The New York District Court's ruling in the current HFCS lawsuit will set the stage for future food litigation and will determine if courts are willing to accept the legal theory that food manufacturers should be held liable for the adverse health outcomes associated with their food products.

Liability is contingent upon plaintiffs proving causation. In order to demonstrate that an exposure or product caused their adverse health outcome, plaintiffs are increasingly relying on epidemiologic evidence. Strict interpretations of the Supreme Court's ruling in *Daubert*, erect seemingly insurmountable barriers to admitting an expert opinion based on epidemiologic evidence. Bright-line tests, such as a risk ratio threshold of 2.0, do not reflect how practitioners derive causal inferences in the field and usually result in the dismissal of otherwise meritorious claims. Instead of utilizing these arbitrary admissibility tests, judges should determine the reliability of epidemiologic evidence by examining the methodology behind each study.

Unfortunately, even if HFCS plaintiffs succeeded in establishing causation, additional barriers, such as apportioning liability and the battle cry of personal responsibility, likely stand in their way. According to law professor and anti-tobacco litigator, John Banzhaf III, "as was the case with tobacco, it takes time for legal theories to coalesce in a way that forces major societal change."²⁵⁴ Indeed, the line between individual responsibility and public accountability for the economic and public health costs of obesity-related health conditions, has yet to be firmly drawn in the context of food litigation.²⁵⁵ *S.F. v. Archer-Daniels-Midland Co.* may

²⁵⁴ Blaine Harden, *Eatery Joins Battle with The Bulge*, GUARDIAN WKLY., Sept. 24, 2003, <http://www.theguardian.com/theguardian/2003/sep/25/guardianweekly.guardianweekly11>. Following the passage of local laws in New York and California banning trans fat in restaurants in 2006 and 2008, respectively, "[o]n [November 7, 2013], the Food and Drug Administration (FDA) announced that it is beginning the process of eliminating artificial trans fat from the U.S. food supply." Alexandra Sifferlin, *Trimming the Fat: A New FDA Effort Could Change Junk Food—For the Better*, TIME, Nov. 25, 2013, at 21. Health statistics, such as the Center for Disease Control's estimate that trans fat contributes to 20,000 heart attacks each year, "prompted the FDA to declare that trans fats are no longer 'generally recognized as safe.'" *Id.* Acknowledging that it took seven years to mandate disclosure of trans fat on food labels, this ban does not mean that trans fat will disappear from store shelves tomorrow. *Id.* Indeed, because this ban would require many businesses to completely reformulate their food production, the FDA is offering a 60-day "commenting period" for food industry advocates to suggest a more realistic timeframe. *Id.* Although it may take years before trans fat disappears for good, the FDA's newest effort to curb the obesity epidemic is a step in the right direction. *Id.*

²⁵⁵ See Pelman, 237 F. Supp. 2d at 516 ("The issue of determining the breadth of personal responsibility underlies much of the law: where should the line be drawn between an individual's own responsibility to take care of herself, and society's responsibility to ensure that others shield her?"). Entitled, "YOU ARE TOO STUPID...to make good personal decisions about foods and beverages," the Center for Consumer Freedom's ad that was launched in response to the New York City Department of Health's campaign encouraging consumers to choose beverages with less sugar, illustrates the current battle between personal responsibility and industry accountability. *You are Too Stupid*, CENTER FOR CONSUMER FREEDOM (Dec. 22, 2011), <http://www.consumerfreedom.com/2011/12/you-are-too-stupid/>;

just be one battle in the greater war to hold the food industry responsible for the nation's expanding waistline and its attendant health consequences.