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Trends in Consumer Litigation

## Not the Next Tobacco: Defense to Obesity Claims

*Joseph P. McMenamin, M.D., J.D.  
Andrea D. Tiglio, Ph.D., J.D.*

# Not the Next Tobacco: Defenses to Obesity Claims

JOSEPH P. McMENAMIN, M.D., J.D.\*

ANDREA D. TIGLIO, PH.D., J.D.\*\*

## I. INTRODUCTION

From a plaintiff's viewpoint, some recent tobacco litigation has been wildly successful.<sup>1</sup> In payment of both compensatory and punitive damages, hundreds of billions of dollars have changed hands under the Master Settlement Agreement.<sup>2</sup> In hopes of achieving similar successes against food companies, plaintiffs' lawyers, including many veterans of the tobacco wars, are attempting to fashion theories of recovery against such companies for health harms related to obesity.<sup>3</sup> The best known of these cases is *Pelman v. McDonald's Corp.*, a claim recently revived by the Second Circuit after it was originally dismissed in the Southern District of New York.<sup>4</sup> But the differences between tobacco and food, and between the merits of tobacco cases and obesity cases, are striking, and create serious obstacles to the success of plaintiffs' claims. Some of those are obvious; others, more subtle.

This article explores some of the differences between tobacco litigation and obesity litigation. It concludes that claims against food companies for obesity are unjustified, and that the harms to be anticipated from obesity claims may well preponderate over the possible benefits.

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\* Dr. McMenemy is a Partner in the law firm of McGuireWoods LLP, Richmond, VA.

\*\* Dr. Tiglio is an Associate in the law firm of McGuireWoods LLP, McLean, VA.

<sup>1</sup> Enthusiasts claim that the suits deserve much of the credit for decreased tobacco consumption in the United States. See, e.g., J. Alderman & R.A. Daynard, *Applying Lessons From Tobacco Litigation to Obesity Lawsuits*, 30 (1) AM. J. PREV. MED. 82 (2006). This conclusion does justice neither to the effects of decades of educational efforts, including Surgeon Generals' reports and the advice doctors give patients, nor to the development and dissemination of an enormous volume of research into the health effects of smoking.

<sup>2</sup> See, e.g., C. King & M. Siegel, *The Master Settlement Agreement with the Tobacco Industry and Cigarette Advertising in Magazines*, 345 (7) NEW ENGL. J. MED. 504-511 (Aug. 16, 2001), abstract available at <http://content.nejm.org/cgi/content/abstract/345/7/504>, (last visited July 6, 2006); see also, "Office of the Attorney General, Dept. of Justice, State of California, "Master Settlement Agreement," available at <http://ag.ca.gov/tobacco/msa.htm>, (last visited July 6, 2006).

<sup>3</sup> For an illustration of lawyers' propensity for analogizing from the tobacco cases to expand concepts of recovery in tort, see, R.F. Cochran, *From Cigarettes to Alcohol: The Next Step in Hedonic Product Liability?* 27 PEPP. L. REV. 701 (2000), and A. Lipanovich, *Smoke Before Oil: Modeling a Suit Against the Auto and Oil Industry on the Tobacco Tort Litigation is Feasible*, 35 GOLDEN GATE L. REV. 429 (2005). For an argument that "fast-food litigation will invariably follow the path of the tobacco cases," as well as an overview of the history of tobacco litigation, see, F.E. Crawford, *Fit for its Ordinary Purpose? Tobacco, Fast Food, and the Implied Warranty of Merchantability*, 63 OHIO ST. L.J. 1165, 1165 (2002). Were the U.S. to adopt the British approach to compensation claims, however, parallels between overeating and smoking might benefit defendants, not plaintiffs: Mr. Justice Stanley Burton has ruled that in the UK smokers are legally responsible for their own ill health because of their negligence in failing to quit, so the recovery of the estate of a smoker with asbestos-related lung cancer in a wrongful death claim was reduced by 20%. L. Smith & S.Lister, *Smokers Forfeit Legal Rights*, LONDON TIMES, (Dec. 17 2005), available at <http://www.timesonline.co.uk/article/0,,2-1936975,00.html>, (last visited July 6, 2006). In the same country, and on a similar rationale, a vascular surgeon was deemed to be justified in declining to treat a smoker for peripheral vascular disease until the smoker quit. J. Bale, *Smoker Told to Give up if He Wants Consultant to Treat Him*, LONDON TIMES, (Dec. 19, 2005), available at <http://www.timesonline.co.uk/article/0,,2-1939680,00.html>, (last visited July 6, 2006).

<sup>4</sup> 237 F.Supp.2d 512 (S.D.N.Y. 2003), vacated in part by *Pelman ex rel. Pelman v. McDonald's Corp.*, 396 F.3d 508 (2d Cir. 2005).

### A. Historical Context

Throughout most of human history, and indeed pre-history, humanity faced a threat not from obesity but from a clear and present danger of malnutrition or of frank starvation; nor is that battle entirely over. Much of the world still goes to bed hungry.<sup>5</sup> That hunger is comparatively uncommon in the United States must not go unrecognized.

Credit for America's extraordinary agricultural productivity belongs first to the farmer. Abundant land, fertile soil, a moderate climate, private land ownership, growing markets for farm products at home and abroad, and the application of science and technology to farm operations have all contributed to the success of American agriculture. These characteristics have enabled many consumers in the United States to enjoy access to virtually unlimited supplies of a wide variety of wholesome, nutritious food at modest cost. While obesity is not an insignificant problem in the United States, we must not lose sight of the achievements of farmers and of food companies in protecting us from famine.

### B. Food v. Tobacco

The contrast of food with tobacco is stark.

There are substantially no health benefits from smoking.<sup>6</sup> Rather, there are numerous and serious risks.<sup>7</sup> Among them are the leading causes of death in the United States and indeed the entire industrialized world: atherosclerosis (heart attacks, strokes, peripheral vascular disease), cancers of many kinds, chronic lung disease (emphysema, bronchitis), and dysrhythmias. Smoking also causes an array of less serious problems, such as rheumatologic disorders, gum disease,<sup>8</sup> and premature aging of skin. It has been estimated that tobacco causes approximately 440,000 deaths per year in the United States<sup>9</sup> Annual economic costs are estimated to range between \$5 and \$12 billion.<sup>10</sup> In contrast, the benefits of eating include survival itself. While there are no safe doses of tobacco, there is no safe way to abstain from food. There are plenty of healthy non-smokers, but no healthy non-eaters. These differences are significant not just philosophically, but also legally. The law of tort has long distinguished between necessities and luxuries,<sup>11</sup> and

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<sup>5</sup> See generally, Food and Agricultural Organization of the U.N., "The State of Food Insecurity in the World," 7th ed. (2005), available at [http://www.fao.org/documents/show\\_cdr.asp?url\\_file=/docrep/008/a0200e/a0200e00.htm](http://www.fao.org/documents/show_cdr.asp?url_file=/docrep/008/a0200e/a0200e00.htm) (last visited July 6, 2006).

<sup>6</sup> There have been reports that smokers face a lower risk of developing Parkinson's disease than do non-smokers. See, e.g., M.A. Hernan, et al., *A Meta-analysis of Coffee Drinking, Cigarette Smoking, and the Risk of Parkinson's Disease*, 52 ANN. NEUROL. 276-84 (2002). Nicotine is also thought to have anti-inflammatory properties that may have some benefit among those with ulcerative colitis. M. Longmore, I. Wilkinson, E. Torok, OXFORD HANDBOOK OF CLINICAL MEDICINE, 5th ed. (2002) at 232. Even if these effects are real, however, they are dwarfed by the harms smoking unequivocally inflicts.

<sup>7</sup> "[C]igarettes, 'are the only consumer product in existence which when used exactly as intended, and in the complete absence of any mishaps, causes injury.'" Haglund v. Philip Morris, Inc., 446 Mass. 741, 745, 847 N.E.2d 315, 321 (2006).

<sup>8</sup> PR NEWSWIRE (July 19, 2005), available at <http://www.tobacco.org/articles/category/immuno/>, (last visited July 6, 2006).

<sup>9</sup> Centers for Disease Control, "Annual Smoking-Attributable Mortality, Years of Potential Life Lost, and Economic Costs—United States, 1995-1999," 51 (14) *Morbidity and Mortality Weekly Report* 300-3 (2002). Historically, the health risks of tobacco became far more substantial, and the prevalence of smoking-related illness grew much larger, after 1915, when cigarettes could be mass-produced. G.L. Ross, K.L. Schneider, & E.M. Whelan, eds., *Cigarettes: What the warning label doesn't tell you*, 2d ed., AMERICAN COUNCIL ON SCI. AND HEALTH, New York, N.Y. (2003).

<sup>10</sup> *Id.* See also, D.W. Garner, *Cigarettes and Welfare Reform*, 26 EMORY L.J. 269, 271 n. 20, 286-90 (1977).

<sup>11</sup> See, *O'Brien v. Muskin Corp.*, 94 N.J. 169, 463 A.2d 298 (N.J. 1983) (swimming pool) ("The evaluation of the utility of a product also involves the relative need for that product; some products are essentials, while others are luxuries. A product that fills a critical need ... should be viewed differently from a luxury item." *Id.* at 184, 463 A.2d at 306).

between products that sustain life or alleviate pain and suffering and those that merely provide pleasure.<sup>12</sup>

To succeed tobacco-style, obesity plaintiffs' lawyers must characterize the food industry as a *bête noire*. In the tobacco wars, such an attack was not very difficult.<sup>13</sup> There is only a handful of companies, most quite large, that controls most of the tobacco business in America, and indeed in much of the rest of the world. Although it is theoretically possible for a smoker to grow his own tobacco and fashion cigarettes or cigars from it, only a few hobbyists actually do so. As a practical matter, the smoker must do business with one or more of several companies. An eater, in contrast, can grow his own food. Around the world, uncounted millions do. Even if he does not, though, an eater controls what foods to select from among the thousands of available choices, how much to eat at a sitting, how frequently to dine, and where, and how the food is prepared.<sup>14</sup> Even among Americans, whose prosperity is the envy of the world, most meals are prepared and eaten at home.<sup>15</sup> As we will show, reported increases in obesity in the United States have many causes, and cannot be linked to specific foods or to specific eating venues. If all fast food restaurants closed their doors tomorrow, we would not all suddenly become lean and healthy.

## II. PELMAN AND OTHER OBESITY-RELATED CLAIMS

In *Pelman I*,<sup>16</sup> plaintiffs alleged that McDonald's products caused obesity in minors who consumed the company's food.<sup>17</sup> Plaintiffs alleged that McDonald's products were unhealthful and inherently dangerous because they contained high levels of fat, cho-

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<sup>12</sup> *Brown v. Superior Court*, 44 Cal.3d 1049, 751 P.2d 470 (1988) (prescription drugs); *see also*, Restatement (Second) of Torts, § 402A, cmt. K (distinguishing between products that "may be necessary to alleviate pain and suffering or to sustain life" and those "used to make work easy or to provide pleasure.") *Id.*

<sup>13</sup> A detailed analysis of the tobacco litigation is beyond the scope of this article. The industry has numerous critics, however. *See, e.g.*, Irene Scharf, *Breathe Deeply: The Tort of Smokers' Battery*, 32 HOUSTON L. REV. 615 (1995) (arguing that tobacco manufacturers are liable in battery to smokers); Richard L. Cupp, Jr., *A Morality Play's Third Act: Revisiting Addiction, Fraud and Consumer Choice in "Third Wave" Tobacco Litigation*, 46 U. KAN. L. REV. 465 (1998); Robert L. Rabin, *A Sociolegal History of the Tobacco Tort Litigation*, 44 STAN. L. REV. 853 (1992). In his testimony at the Hearing on S. 1428 Before the Subcomm. on Admin. Oversight and the Courts of the Senate Comm. on the Judiciary, 108th Cong. (2003), in contrast, Dr. Gerard Musante, Founder, Structure House, said, "No industry is to blame and none should be charged with solving America's obesity problem."

<sup>14</sup> *See* R.H. Carmona, Acting Assistant Secretary for Health, United States Surgeon General, U.S. Department of Health and Human Services, Remarks at the American Enterprise Institute Obesity Conference, Washington, D.C., (June 10, 2003), available at <http://www.surgeongeneral.gov/news/speeches/obesity061003.htm>, (last visited July 6, 2006). The legal consequences of diversified sources of supply are significant. "Food is not a product like tobacco. There are 320,000 food products sold or served by many thousands of food manufacturers and restaurants ... in millions of households. It will be essentially impossible to prove that a person's obesity or health problems are solely caused by any particular item or place." F. M. Burtzos, *My Big Fat Greek Lawsuit*, 33 COLO. LAWYER 53 (June 2004).

<sup>15</sup> *See*, A.W. Mathews & S. Leung, *FDA Considers Nutrition Labels in Restaurants*, WALL ST. J. (Oct. 23, 2003) at B1.

<sup>16</sup> *Pelman ex rel. v. McDonald's Corp.*, 237 F.Supp.2d 512 (S.D.N.Y. 2003), *refiled as* 2003 WL 22052778 (S.D.N.Y., Sept. 3, 2003), *vacated in part*, 396 F.3d 508 (2d Cir. 2005), *remanded*, 396 F.Supp.2d 439 (S.D.N.Y. 2005). Despite its notoriety, Pelman was not in fact the first suit alleging that a food company is liable to an overweight plaintiff. In 2002, Caesar Barber, 56, alleged in a purported class action that McDonald's, Burger King, Kentucky Fried Chicken and Wendy's negligently produced food products with detrimental health effects, failed to warn consumers of the health effects of the restaurants' food, negligently marketed their food products to children and engaged in unfair and deceptive acts and practices in violation of the New York Consumer Protection Act. Complaint, *Barber v. McDonald's Corp., et al.*, N.Y. Superior Court 2002, available at <http://banzhaf.net/docs/nyccomp.html>, (last visited July 6, 2006). The case was shelved. Emily Johns, "Pending bill fights suits against the food industry," STAR TRIBUNE, (Minneapolis, Minn.), Feb. 9, 2004 at 6A. Plaintiff's counsel then filed *Pelman*. M.M. Mello, et al., *The McLawsuit: The Fast Food Industry and Legal Accountability for Obesity*, 22 (6) HEALTH AFF. 207, 208 (Nov./Dec. 2003).

<sup>17</sup> *Pelman I*, *supra* n.16, at 512-516.

lesterol, sugar and salt.<sup>18</sup> Judge Sweet dismissed the complaint because the nature and characteristics of fast food are well-known and because no one need eat at McDonald's.<sup>19</sup> The court wrote that a "[c]omplaint must allege either that the attributes of McDonalds [sic] products are so extraordinarily unhealthful that they are outside the reasonable contemplation of the consuming public or that the products are so extraordinarily unhealthful [as to be dangerous in their intended use]."<sup>20</sup> We turn to a more detailed analysis of the court's reasoning.

In *Pelman I*, Counts I and II were based on acts, practices and omissions allegedly deceptive and violative of the Consumer Protection Act in New York's General Business Law, §§ 349 and 350.<sup>21</sup> Under §§ 349 and 350, a plaintiff claiming deceptive practices must show that 1) the act was consumer-oriented, 2) it was misleading in material respects, and 3) he was injured as a result of the act.<sup>22</sup> Traditional showings of reliance and *scienter* are not required.<sup>23</sup> In alleging violations of §§ 349 and 350, plaintiffs claimed that defendant 1) deceptively advertised its food as not unhealthful and failed to provide consumers with nutritional information (Count I) and 2) induced minors to eat at McDonald's through deceptive marketing ploys (Count II).<sup>24</sup> Count II also alleged misrepresentations to children.

Judge Sweet dismissed Counts I and II because plaintiffs identified not a single instance of deceptive acts.<sup>25</sup> The advertisements that plaintiffs pointed to encouraged daily consumption of McDonald's fare, but made no suggestion that doing so could be part of a well-balanced diet. Plaintiffs offered no ad in which McDonald's claimed that its products could be eaten at every meal, every day, without ill effect.<sup>26</sup> Since there were no specific claims on which consumers could rely, the ads were mere puffery.<sup>27</sup> Plaintiffs also failed to show why any alleged omission was deceptive. More importantly, they failed to allege that the information they claimed to want was solely within McDonald's possession or that a consumer could not reasonably obtain it.<sup>28</sup> The court also dismissed Court II because it failed to "identify a specific single advertisement, promotion or statement directed at infant consumers."<sup>29</sup>

McDonald's invoked the Federal Nutrition Labeling and Education Act (NLEA),<sup>30</sup> arguing that the NLEA barred plaintiffs' claims that the restaurants' alleged "failure to provide nutritional information is deceptive" because the Act exempts restaurants.<sup>31</sup> In fact, even though it is not required to under the law, McDonald's freely discloses the nutritional values of its food.<sup>32</sup> The court held that although the NLEA allows "states

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<sup>18</sup> *Id.* at 531-32. Despite our national obsession with fat, the value of eating a low-fat diet in achieving a longer lifespan or avoiding heart attacks is not undisputed. Gary Taubes, *The Soft Science of Dietary Fat*, 291 *Sci.* 2536-2538 (2001).

<sup>19</sup> *Pelman I*, *supra* n.16, at 532-533. See generally, Andrew M. Dansicker, *The Next Big Thing for Litigators*, 37 *MARYLAND B.J.* 12-17 (2004) ("The fast food industry can fall back on the fact that Americans are, and have been, well-educated about the deadly risks with eating excessive amounts of unhealthy [sic] food but choose to do so anyway").

<sup>20</sup> *Pelman I*, *supra* n.16, at 532.

<sup>21</sup> N.Y. MCKINNEY'S GEN. BUS. LAW §§ 349 and 350.

<sup>22</sup> *Pelman I*, *supra* n.16, at 525.

<sup>23</sup> *Id.*

<sup>24</sup> *Id.* at 524.

<sup>25</sup> *Id.* at 527.

<sup>26</sup> *Id.* at 527-528. In fact, McDonald's seldom claims any nutritional value for its hamburgers. Rebecca Coombes, *The New Tobacco*, 328 *BMJ* 1572 (June 26, 2004), available at <http://bmj.bmjournals.com/cgi/content/full/3287455/1572>, (last visited June 20, 2006).

<sup>27</sup> *Pelman I*, *supra* n.16, at 528, n.14.

<sup>28</sup> *Id.* at 529.

<sup>29</sup> *Id.* at 530.

<sup>30</sup> 21 U.S.C. § 343(q)(1) and 21 U.S.C. § 343(q)(5)(A)(i).

<sup>31</sup> *Pelman I*, *supra* n. 16 at 525.

<sup>32</sup> *Id.* at 525.

to impose labeling requirements for certain food industries that are exempt under the Act, including the restaurant industry," the complaint nevertheless failed to identify "a single instance of deceptive acts," and failed to allege that only McDonald's knew the nutritional content of its food or that consumers could not obtain that information.<sup>33</sup>

Count III alleged that McDonald's negligently sold food products high in cholesterol, fat, salt, and sugar. The court dismissed Count III because the complaint failed to allege that the dangers of McDonald's products were not well-known and failed to allege with sufficient specificity that the products were a proximate cause of plaintiff's obesity.<sup>34</sup>

Count IV alleged failure to warn consumers of the ingredients, quantity, qualities, and levels of cholesterol, fat, salt, and sugar. Count IV failed because the risks of overconsumption are common knowledge.<sup>35</sup> New York recognizes obviousness as a defense to duty to warn cases.<sup>36</sup> That the plaintiffs were minors did not defeat this defense.<sup>37</sup> Moreover, the court noted, "Nobody is forced to eat at McDonald's."<sup>38</sup>

Count V alleged that McDonald's acted negligently in marketing physically and psychologically addictive products.<sup>39</sup> The court rejected Count V as excessively vague. The complaint failed to specify whether it was the combination of fats and sugars in McDonald's products that plaintiffs claimed could cause addiction or whether there was some additive, such as nicotine in cigarettes, that could do so. There was no allegation that McDonald's purposefully manufactured products with addicting qualities. The complaint failed to specify whether one could become addicted after eating at McDonald's only once or whether a steady diet was necessary. There was no allegation regarding whether, as infants, plaintiffs were more susceptible to addiction than adults.<sup>40</sup> The complaint also failed to explain sufficiently how the allegedly addictive nature of the food proximately caused plaintiffs' health problems.<sup>41</sup>

Despite these shortcomings in their pleadings, the *Pelman I* court granted plaintiffs leave to replead.<sup>42</sup>

In their second attempt, plaintiffs filed a four-count amended complaint. There were two main claims, however. First, that McDonald's products were "so processed with additives and other ingredients and preservatives" that they created a "danger and hazard," and McDonald's was negligent in failing to warn consumers of that hazard.<sup>43</sup> Second, that under New York's consumer protection laws McDonald's marketing behavior amounted to fraudulent and deceptive business practices. The plaintiffs also made, but later dropped, a failure-to-warn claim. In September 2003, the court dismissed Counts

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<sup>33</sup> *Id.* at 527-529; 21 U.S.C. § 343-1(a)(4). McDonald's, Wendy's, and Burger King disclose their products' trans fat levels on their respective websites and in their restaurants. Steven Gray, *Pressure Mounts on Fast Food Chains to Remove Trans Fats*, WALL ST. J., (Dec. 14, 2004) at D-1.

<sup>34</sup> *Pelman I*, *supra* n. 16 at 539.

<sup>35</sup> *Id.* at 533, 540. Risks "generally known and recognized" do not require warnings. See Restatement (Second) of Torts § 402A, cmt. J (1979). The Third Restatement adopts a similar formulation: "obvious or generally known." Restatement (Third) of Torts: Products Liability § 2, cmt. A (1979).

<sup>36</sup> *Pelman I*, *supra* n. 16, at 540 (citations omitted).

<sup>37</sup> *Id.*

<sup>38</sup> *Id.*

<sup>39</sup> *Pelman I*, *supra* n.16 at 520.

<sup>40</sup> *Id.* at 542.

<sup>41</sup> *Id.*

<sup>42</sup> *Pelman I*, *supra* n. 16, at 519.

<sup>43</sup> *Pelman v. McDonald's Corp.*, (No. 02-Civ. 7821), 2003 U.S. Dist. LEXIS 15202 at \*5, \*42 (S.D.N.Y. 2003) [hereinafter *Pelman II*]. The *Pelman I* court implied that the claim that McDonald's products were adulterated in ways that consumers could not reasonably anticipate may have had the greatest potential for viability. *Pelman I*, 237 F.Supp.2d at 536. The original complaint merely stated that claim "cursorily," however. *Id.* The amended complaint also failed to elaborate upon it. *Pelman II*, No. 02-Civ. 7281, 2003 U.S. Dist. LEXIS 15202.

I-III. Judge Sweet held that the plaintiffs had not sufficiently alleged the elements of a deceptive advertising claim.<sup>44</sup>

The three dismissed counts each alleged that McDonald's violated §§ 349 and 350 of the New York General Business Law, the New York Consumer Protection Business Act.<sup>45</sup> Count I alleged that McDonald's promotional representations created a "false impression that its food products were nutritionally beneficial and part of a healthy [sic] lifestyle if consumed daily."<sup>46</sup> Count II alleged that McDonald's failed to adequately "disclose that its use of certain additives and the manner of its food processing rendered certain of its food substantially less healthy [sic] than represented."<sup>47</sup> Count III alleged that McDonald's "deceptively represented that it would provide nutritional information to its New York customers when in reality such information was not readily available at a significant number of McDonald's outlets in New York."<sup>48</sup> The amended complaint also alleged that as a result of reliance upon these allegedly deceptive practices, plaintiffs had consumed McDonald's menu offerings and so developed obesity and related conditions.<sup>49</sup> Judge Sweet dismissed *Pelman II* because it lacked any express allegation that any plaintiff specifically relied to his detriment on any particular representation made in any identified McDonald's ad or promotional material.

In January 2005, the Second Circuit reversed Judge Sweet in his dismissal of Counts I-III under § 349 of the New York General Business Law.<sup>50</sup> The court vacated the District Court's dismissal of the portions of Counts I-III that related to § 349 and remanded so that discovery could proceed.

The Second Circuit held that § 349 does not require proof of actual reliance.<sup>51</sup> Bare bones notice pleading is sufficient, said the court; pleading with particularity, as required under Rule 9(b) of the Federal Rules, is not necessary.<sup>52</sup> Judge Sweet had written that, "Plaintiffs have failed . . . to draw an adequate causal connection between their consumption of McDonald's food and their alleged injuries."<sup>53</sup> The Circuit, however, decided that the complaint's failure to state what plaintiffs ate, how much they exercised, whether there were family histories of diseases, etc., were matters for discovery.<sup>54</sup>

At this writing *Pelman II*<sup>55</sup> is still pending. In decisions made so far, the court held that 1) defendant was entitled to a more definite statement of plaintiff's claims to identify the advertisements collectively amounting to the chain's allegedly deceptive scheme; 2) defendant was entitled to a brief explanation why the challenged ads were materially deceptive; 3) defendant was not entitled to confirmation that consumers saw or heard each challenged ad in New York before the action began, but plaintiffs had to provide brief explanations of how they were aware of the alleged scheme; and 4) defendant was not entitled to a description of how each of its challenged ads injured consumers, but plaintiffs did have to outline injuries that each suffered by reason of the challenged scheme.<sup>56</sup>

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<sup>44</sup> *Pelman II*, *supra* n. 43, at \*5, \*42.

<sup>45</sup> *Id.*, at \*5-6. *Cf.* n. 21, *supra*, and associated text.

<sup>46</sup> *Pelman II*, *supra* n. 43, at \*6; *see also*, *Pelman v. McDonald's Corp.*, 396 F.3d 508, 510 (2d Cir. 2005).

<sup>47</sup> *Pelman II*, *supra* n. 43, at \*6.

<sup>48</sup> *Id.*

<sup>49</sup> *Id.*, at \*6-7; 396 F.3d at 510.

<sup>50</sup> *Pelman v. McDonald's Corp.*, 396 F.3d 508, 511-512 (2d Cir. 2005).

<sup>51</sup> *Pelman*, 396 F.3d at 511.

<sup>52</sup> *Id.*

<sup>53</sup> *Pelman II*, *supra* n. 43, at \*30.

<sup>54</sup> 396 F.3d at 512.

<sup>55</sup> 396 F.Supp.2d 439 (S.D.N.Y. 2005).

<sup>56</sup> *Id.*

### A. Other Obesity-Related Suits

In addition to the *Pelman* case, there have been other obesity-related challenges to the food industry. On January 18, 2006, the Center for Science in the Public Interest (CSPI), the Boston-based Campaign for a Commercial-Free Childhood and others filed a demand letter against Nickelodeon parent Viacom and cereal maker, Kellogg. The letter, a condition precedent to a deceptive trade practices suit in Massachusetts, seeks to stop the companies from marketing on television what it dismisses as junk-food when 15% or more of the audience is 8 years old or younger. The letter targets not only commercials, but also websites, toy giveaways, contests and other marketing aimed at that age group.<sup>57</sup>

The CSPI and its co-complainants cite a report from the Institute of Medicine asserting that current food and beverage marketing practices put children's long-term health at risk. In particular, the report states that many ads aimed at children are mostly for high-calorie, low-nutrition food and drinks, thereby promoting unhealthy eating habits, and ultimately leading to the early onset of diet-related chronic diseases.<sup>58</sup> The advocacy groups further allege that although rates of childhood obesity are increasing, Kellogg and Viacom continue to market junk food to children. It appears that the letter's authors assume little or no parental control over what children view.

A purported class action has been filed in the District of Columbia against YUM! Brands' Kentucky Fried Chicken (KFC) unit by CSPI and an individual.<sup>59</sup> This suit asks that the court prohibit KFC from using partially hydrogenated oil, or in the alternative to require signs warning consumers that many of its food are high in *trans* fats. Specifically, Count I asserts a breach of implied warranty of merchantability under common law and D.C. statutory law.<sup>60</sup> Count II alleges a breach of the D.C. Consumer Protection

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<sup>57</sup> See letter, Stephen Gardner, Director of Litigation, CSPI, to Sumner W. Redstone, Chairman and CEO, Viacom, Inc., and James M. Jenness, Chairman and CEO, Kellogg Co., available at [http://cspinet.org/new/pdf/viacom\\_kellogg.pdf](http://cspinet.org/new/pdf/viacom_kellogg.pdf) (last visited July 12, 2006). As of this writing, suit has not yet been filed. The approach of the CSPI to the obesity problem may be gauged from its "School foods report cards" (June 2006), available at [www.cspinet.org/schoolreportcard.pdf](http://www.cspinet.org/schoolreportcard.pdf), (last visited July 15, 2006). Even though CSPI says in that report that its "goal is to help reduce the illnesses, diabetes, deaths, and high health-care costs caused by diet—and inactivity-related diseases," the word "exercise" does not appear in the text, although it does appear in the title of one cited article. The section entitled "Overview of state actions to promote nutrition, increase physical activity, and prevent obesity," at 12 *et seq.*, appears to say little or nothing about activity.

<sup>58</sup> Cf., Institute of Medicine, *Food Marketing to Children and Youth: Threat or Opportunity*, NATIONAL ACADEMY PRESS (Dec. 6, 2005). The report concedes, however, that "current evidence is not sufficient to arrive at any findings about a causal relationship from television advertising to adiposity among children and youth." *Id.* at 379, 380. See, <http://www.darwin.nap.edu/books/0309097134/html/272.html>, (last visited July 17, 2006). According to the UK's Institute of Practitioners in Advertising, the relationship between food advertising and obesity in other countries is doubtful: "Norway and Belgium have three or four less [sic] food ads per hour on average than Germany, Denmark, Finland, and the Netherlands, yet suffer from higher levels of obesity. Advertising to children under the age of 13 has been banned in Quebec, Canada since 1978. This appears to have had no impact on obesity levels amongst children compared to the rest of the country. In 1996, for example, 27.6% of children in Quebec were overweight with a Canadian average of 29.3%, 35.3% in the Atlantic, and 24.4% in the Prairies." H. Pringle, *Why Banning Advertising to Children Would be Naïve*, reproduced with permission from MEDIA WEEK (Jan. 16, 2004), available at [http://www.ipa.co.uk/documents/why\\_banning\\_ads\\_to\\_kids.pdf](http://www.ipa.co.uk/documents/why_banning_ads_to_kids.pdf), (last visited August 8, 2006). Advertising is intended to, and to some extent may, foster brand loyalty, but there is little evidence it has an effect on consumption of categories of food, such as foods high in salt or fat. B.M. Young, et al., *The Role of Television Advertising in Children's Food Choice*, London: Report to the Ministry of Agriculture, Fisheries, and Food (1996).

<sup>59</sup> *Hoyte v. YUM! Brands, Inc., d/b/a/ KFC*, Docket No. 06CA4526 (D.C. Super. Ct. 2006); removed to D. D.C., Docket No. 06cv1127 (2006).

<sup>60</sup> D.C. Code §§ 28:2-314.

Procedures Act.<sup>61</sup> Finally, Count III complains of allegedly negligent misrepresentation. In response, YUM! Brands has asserted that the suit is frivolous and without merit.<sup>62</sup>

A July 2002 report from the Institute of Medicine encourages consumers to limit daily *trans* fat intake to no more than one percent of total calories, which is about 2 grams per day based upon a 2,000 calorie daily diet.<sup>63</sup> CSPI's suit alleges that a KFC meal that includes three pieces of extra-crispy chicken contains 15 grams of *trans* fat.<sup>64</sup> CSPI also contends that KFC does not inform consumers that it uses hydrogenated vegetable oil in fried chicken and other products sold at its outlets nationwide.<sup>65</sup> Although a nutrition calculator on KFC's website<sup>66</sup> displays the amount of *trans* fat in its food, the plaintiffs assert that KFC fails to warn consumers that it cooks using "the worst oil available and imaginable," instead misrepresenting that its products are "the best food" and can be consumed as part of a "nutritionally healthy [sic] lifestyle."<sup>67</sup> Predicting the outcome of this suit is speculative, of course, but in the sections below we consider some of the problems plaintiffs may face in cases such as this.

### B. Legislation in Response to Food Industry Lawsuits

Since *Pelman*, both federal and state legislation has been passed to protect the food industry from lawsuits purporting to hold fast food companies responsible for weight gain and obesity. In 2004, the House of Representatives passed the "Cheeseburger Bill," officially known as the Personal Responsibility in Food Consumption Act, H.R. 339, 108th Cong. (2003), banning obesity lawsuits against the food industry in federal or state courts.<sup>68</sup> If this proposal becomes law, it would prevent "frivolous lawsuits against the manufacturers, distributors, or sellers of food or nonalcoholic beverage products" arising from obesity claims.<sup>69</sup>

In 2005, Representative Ric Keller of Florida reintroduced the Personal Responsibility in Food Consumption Act to "allow Congress and regulatory agencies to determine appropriate laws, rules, and regulations to address the problems of weight gain, obesity, and health conditions associated with weight gain or obesity."<sup>70</sup> At the same time, Senator Mitch McConnell of Kentucky introduced the Common Sense Consumption Act of 2005 in the Senate, S 908, 109th Cong. (2005). Similar bills have been introduced in more than 20 states and enacted in at least 14, including Arizona, Idaho, Louisiana, South Dakota, Utah, and Washington.<sup>71</sup> The tort reform bills in the states do not ban suits based

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<sup>61</sup> D.C. Code § 28-3904 and § 28-3905(K)(1).

<sup>62</sup> See, WebMD, *Lawsuit Targets trans Fat at KFC: Group Says It's Hard for Customers to Know Amount of Fat Used in KFC Restaurants*, available at [www.webmd.com/content/article/123/115157?src=rss\\_rxdmorg](http://www.webmd.com/content/article/123/115157?src=rss_rxdmorg), (last visited July 12, 2006).

<sup>63</sup> See, "Letter report on dietary reference taken for *trans* fatty acids," (July 10, 2002), available at <http://www.iom.edu/CMS/5410.aspx>, (last visited July 12, 2006).

<sup>64</sup> See Complaint, *Hoyte*, *supra* note 59.

<sup>65</sup> *Id.*

<sup>66</sup> See KFC Nutrition Calculator, available at [http://www.yum.com/nutrition/menu.asp?brandID\\_Abbr=2\\_KFC](http://www.yum.com/nutrition/menu.asp?brandID_Abbr=2_KFC), (last visited July 12, 2006).

<sup>67</sup> Complaint, *Hoyte*, *supra* note 59, at paragraphs 27 and 28.

<sup>68</sup> S. Morrissey, *Food Fight: 'Cheeseburger Bill' Triggers Debate Over Restaurant Liability When Diners Get Fat*, ABA J. EREPORT 11 (2004).

<sup>69</sup> H.R. at 339, § 4(5) (2004).

<sup>70</sup> Personal Responsibility in Food Consumption Act of 2005, HR 554, 109th Cong. §1 (2005).

<sup>71</sup> See, e.g., Mich. Comp. Law § 600.2974, Limitation of civil liability for weight gain or obesity; requirements for cause of action; definitions. See also, U.G. Williams & K.J. Parsigian, "Obesity Litigation—The Next Tobacco?" available at <http://articles.corporate.findlaw.com/articles/files/00338/009676>, (last visited June 20, 2006).

on deceptive advertising.<sup>72</sup> Twenty-one states have enacted “personal responsibility” laws that immunize fast food companies from obesity-related tort claims.<sup>73</sup>

### III. CAUSATION

#### A. *Legal Background*

Elementary tort law teaches that a defendant is liable to a plaintiff only if the defendant’s improper conduct harmed that plaintiff. Mere bad acts that cannot be shown to cause harm are not actionable in tort. As Prof. Pollock taught decades ago, “Proof of negligence in the air, so to speak, will not do.”<sup>74</sup> Unlike, say, a leg broken in a car accident, obesity develops gradually. More important, the cause of the leg fracture in the car case is clear and simple. In contrast, in any given individual and in society as a whole, the causes of obesity are imperfectly understood, but are known to be numerous and complex. That is often not the case with tobacco-related disorders. Consider lung cancer, for example. In oncology, we generally cannot yet identify “the cause” or even “a cause” of a patient’s malignancy, but with lung cancer in smokers we often can.<sup>75</sup>

The decision where to draw the line between “proximate” causes and all others is ultimately a policy judgment. Judges and commentators have devoted entire treatises to how and where to draw that line, and we do not attempt to summarize that scholarship here. In general, however, from among the many possible events and phenomena that could have a bearing on a given outcome, the law focuses its attention upon only those factors that play a “substantial” role in bringing about the result.

The word “substantial” [in the “substantial factor” test] is used to denote the fact that the defendant’s conduct has such an effect in producing the harm as to lead reasonable persons to regard it as a cause, using that word in the popular sense, in which there always lurks the idea of responsibility, rather than in the so-called “philosophic sense,” which includes every one of the great numbers of events without which any happening would not have occurred.<sup>76</sup>

In analyzing which factors are “substantial,” courts consider “the aggregate *number of factors involved* which contribute towards the harm and the effect which each has

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<sup>72</sup> *Id.*

<sup>73</sup> State Bill Tracking: Nutrition & Healthy Lifestyles, Washington D.C.: National Restaurant Association (2005), available at [http://www.restaurant.org/government/state/nutrition/bills\\_key.cfm#lawsuits](http://www.restaurant.org/government/state/nutrition/bills_key.cfm#lawsuits), (last visited July 12, 2006).

<sup>74</sup> F. Pollock, *The Law of Torts*, 455 (11th Ed. 1920). The prevailing test for determining causation in fact is the “but for” test. David G. Owen, et al., 1 *Madden & Owen on Products Liability*, § 12.2 (2000). The plaintiff must prove that he would not have been injured if the act complained of had not occurred. *See, e.g.*, Gen. Motors Corp. v. Davis, 233 S.E.2d 825, 827 (Ga. Ct. App. 1977). Some states use the “substantial factor” test instead of the more traditional “but for” test. F.J. Vandall, *O.K. Coral II: Policy Issues in Municipal Suits Against Gun Manufacturers*, 44 VILL. L.REV. 547, 558 (1999). Under this test, defendant’s act may be causal even though the injury would have occurred without that act if a jury determines that it was a substantial factor in causing injury. *See, e.g.*, Clark v. Leisure Vehicles, Inc., 292 N.W.2d 630, 635 (Wisc. 1980). *But see*, n. 78, *infra*, and associated text. Some states allow either test to be used. Gerst v. Marshall, 549 N.W.2d 810, 817-18 (Ia. 1996)(in groundwater contamination case, allowing “but for” test in place of substantial factor test).

<sup>75</sup> *See, e.g.*, Cipollone v. Liggett Group, Inc., 649 F.Supp. 664 (D.N.J. 1986). According to the WHO, smoking is responsible for 90% of all lung cancers. WHO, “Health Risks” in THE TOBACCO ATLAS, available at <http://www.who.int/tobacco/en/atlas9.pdf>, (last visited July 15, 2006).

<sup>76</sup> Restatement (Second) of Torts, § 431, Cmt. A, quoted in *Pelman I, supra* n.16, 237 F.Supp.2d at 538.

in producing it,” and “whether the situation was acted upon by *other forces for which the defendant is not responsible*” (emphasis supplied).<sup>77</sup> Judge Sweet was “particularly mindful of Professor Prosser’s observation that ‘no case has been found where the defendant’s act could be called a substantial factor when the event would have occurred without it.’”<sup>78</sup> In obesity, the “number of factors involved” is apt to be large, and most of them are beyond the control of farmers or food companies.<sup>79</sup> Defendants should be able to rely on the significance of such factors to defeat plaintiffs’ claims.<sup>80</sup>

*Pelman* demonstrates that the application of causation concepts to plaintiffs’ theories makes it difficult for them to prevail. One reason is that scientific literature, which plaintiffs will need to rely on, strives for balance and objectivity. The *Pelman* plaintiffs saw the consequences: “...McDonalds [sic] points out that articles on which plaintiffs rely in their Complaint suggest that a number of factors *other than diet* may come into play in obesity and the health problems of which plaintiffs complain” (emphasis supplied).<sup>81</sup> The *Pelman* plaintiffs cited, among other sources, National Institutes of Health’s (NIH’s), *Clinical Guidelines*.<sup>82</sup> As the court noted, however, this very source says: “Obesity is a complex multifactorial chronic disease developing from interactive influences of numerous factors—social, behavioral, physiological, metabolic, cellular, and molecular” and from cultural and genetic factors as well.<sup>83</sup> The *Pelman* plaintiffs also cited the Surgeon General’s *Call to Action*, which states that an individual’s weight is “determined by combination of genetic, metabolic, behavioral, environmental, cultural, socioeconomic influences.”<sup>84</sup> Any scientific discussion of the causes of obesity will necessarily list a multitude of causes, and the more complete the list the less help

<sup>77</sup> *Id.*, citing *inter alia* Restatement (Second) of Torts § 433. One reason Judge Sweet was dissatisfied with plaintiffs’ pleadings was their failure to put defendant on notice of facts sufficient to evaluate causation: The court wrote, “No reasonable person could find probable [proximate?] cause based on the facts in the complaint without resorting to ‘wild speculation.’ [citation omitted] ... First, the complaint does not specify how often the plaintiffs ate at McDonald’s ...” 237 F.Supp.2d at 538, 539.

<sup>78</sup> *Id.*, citing W. Prosser, *Handbook of the Law of Torts*, § 41 at 240 (4th ed. 1971) (applying New York law).

<sup>79</sup> Even where “common sense” suggests that a given food product might well “cause” obesity, that conclusion may well be an oversimplification. The relationship between children’s consumption of snack foods and obesity, for example, is not established. A.W. Field, et al., *Snack Food Intake Does Not Predict Weight Change Among Children and Adolescents*, 28 INT’L. J. OBESITY 1210-1216 (2004); S.M. Phillips, et al., *Consumption of High-caloric, Low-nutrient-dense Foods Over the Adolescent Period: Relationships to Body Weight and Fatness in a Longitudinal Setting*, 12 INT’L. J. OBES. RELAT. METAB. DISORD. 461-472 (2004). Even ignoring this problem, causation is complex. According to the NIH, obesity involves the “integration of social, behavioral, cultural, physiological, metabolic and genetic factors.” NIH, *Clinical Guidelines on the Identification, Evaluation and Treatment of Overweight and Obesity in Adults*, NIH publication No. 98-4083 (1998) at xi, available at [http://www.nhlbi.nih.gov/guidelines/obesity/ob\\_gdlns.htm](http://www.nhlbi.nih.gov/guidelines/obesity/ob_gdlns.htm), (last visited July 6, 2006), [hereinafter “Clinical Guidelines”]. “[O]besity is a complex disease with many contributing factors, including genetics, abnormal eating behavior, lack of exercise and cultural influences as well as cerebral mechanisms which are not yet fully understood.” *Id.* For a discussion of some of the causes of obesity, see, B.A. Swinburn, et al., *Diet, Nutrition and the Prevention of Excess Weight Gain and Obesity*, 7 PUBLIC HEALTH NUTR. 123-46 (2004); see also, J.E. Blundell & J. Cooling, *Routes to Obesity: Phenotypes, Food Choices and Activity*, 83 (suppl.) BR. J. NUTR. S33-38 (2000).

<sup>80</sup> *Cf.* *Liberty v. D.C. Police Department and Firemen’s Retirement and Relief Board*, 452 A.2d 1187, 1982 D.C.App. LEXIS 491 (D.C. Ct. App. 1982) (given evidence that disabled police officer had a history of smoking, obesity, hypercholesterolemia, and hypertension, as well as a family history of heart disease, the decision that officer’s disability secondary to ectasia of the coronary arteries was neither caused nor aggravated by work stresses was upheld as supported by substantial evidence).

<sup>81</sup> *Pelman I*, *supra* note 16, 237 F.Supp. 2d at 538.

<sup>82</sup> *Clinical Guidelines*, *supra*, n. 79. See also, “*The Surgeon General’s Call to Action to Prevent and Decrease Overweight and Obesity*, [hereinafter “Call to Action”] available at <http://www.surgeongeneral.gov/topics/obesity/>, (last visited July 6, 2006).

<sup>83</sup> *Clinical Guidelines*, *supra*, n. 79.

<sup>84</sup> *Call to Action*, *supra*, n. 82; *Pelman I*, *supra* note 16, 237 F. Supp. 2d at 538.

it is likely to offer to plaintiffs. The plaintiffs' goal will be to obscure or belittle the importance of those factors not influenced by industry, while exaggerating the impact of the remaining factors. If they prevail, science will lose.<sup>85</sup>

Plaintiffs' invocation of state consumer fraud statutes as weapons against food companies constitutes tacit recognition of their problems with causation.<sup>86</sup> They will probably argue that the causation requirements in cases predicated on these statutes, at least in some states, are more lenient than those imposed in more conventional tort claims.<sup>87</sup> This may well be one reason why, when CSPI and the Campaign for a Commercial Free Childhood sent the intent-to-sue letter to a food company and a media company in January 2006, they invoked the consumer protection law of Massachusetts. Even under the potentially applicable Massachusetts statute, however, proof of consumer fraud claims requires a showing of causation.<sup>88</sup> Often, as in Massachusetts, conduct specifically permitted or authorized under the laws of the United States or of a particular state is conduct immunized from liability under state consumer protection statutes.<sup>89</sup> The products criticized by CSPI, like other food products marketed in the U.S., bear labels regulated by the Food and Drug Administration (FDA). Hence, decisions analyzing the impact of legislation expressly authorizing conduct criticized by deceptive trade practice act plaintiffs may create a significant obstacle to other plaintiffs advancing fraud claims against food advertisers.

Deceptive trade practice claims will probably criticize companies' marketing campaigns, as plaintiffs have done in *Pelman*. While advertisers presumably act on the hope that advertising has some ability to influence behavior, the extent of that influence can easily be overestimated.<sup>90</sup> Surprisingly, there do not appear to have been many scientifically rigorous studies of the relationship between ad exposure and dietary intake. One of the few controlling for other potentially contributing factors took into account parental habits—both eating and television-watching habits—and

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<sup>85</sup> Even ignoring non-dietary factors contributing to obesity, the sources of calories in our society are innumerable and nearly ubiquitous: "The Big Mac contains only half the calories of the standard serving of fettucini Alfredo; the Quarter Pounder has seventy fewer calories than Starbuck's Venti Café Mocha with whipped cream. McDonald's is the target not because of its 'super-sized' offering but because of its deep pockets." Amity Shlaes, *Lawyers Get Fat on McDonald's; New York Lawsuit Alleges Chain is Responsible for Obesity*, CHICAGO TRIBUNE, Nov. 27, 2002 at 25, quoted in *Pelman I*, *supra*, n. 16, 237 F.Supp.2d. at 518, n.5.

<sup>86</sup> See, n. 57, *supra*, and associated text.

<sup>87</sup> Alderman & Daynard, *supra* note 1, at 85.

<sup>88</sup> Where a defendant's conduct was not the proximate cause of plaintiff's damages, a Massachusetts G.L. c. 93A claim—that is, a claim alleging unfair and deceptive trade practices under that state's statutes—will not lie. *McCann v. Davis, Malm & D'Agostine*, 423 Mass. 558, 669 N.E.2d 1077 (1996) (legal malpractice claim). See also, *Kohl v. Silver Lake Motors, Inc.*, 369 Mass. 795, 800, 343 N.E.2d 375, 90 ALR 3d 1342 (1976). In Massachusetts, even where liability can be established, it is more limited for failures to disclose than for misrepresentations. *Lawton v. Dracousis*, 14 Mass. Sup. Ct. 164, 437 N.E.2d 453, *rev. denied*, 387 Mass. 1103, 440 N.E.2d 1107 (1982).

<sup>89</sup> MASS. GEN. LAW Ch. 93A §3; see also, *Price v. Philip Morris, Inc.*, Docket No. 96236, 2005 Ill. LEXIS 2071 (Ill. 2005) (Reversing the trial court's award of \$11 billion against defendant, the Illinois Supreme Court invoked § 4 (1) of the Illinois Deceptive Trade Act, 815 ILCS 510/4 (West 1998), and noted that defendant's use of such descriptive terms as "low tar" was specifically authorized by FTC.). In at least ten states, however, consumer protection cases similar to *Price* have been filed, at least one of which has been certified. University of Maryland School of Law, Legal Resource Center for Tobacco Regulation, Litigation, and Advocacy, *Philip Morris Loses Class Action Suit on Light Cigarettes*, 2 TOBACCO REG. REV. 616 (Sept. 2003).

<sup>90</sup> In the UK, for example, the largest advertising expenditures are reportedly for beer and spirits. Yet sales of those products are in decline there. Makers of alcoholic beverages spend less to advertise white wines than they do for any other product, yet that area shows growth. *The New Tobacco*, 328 BMJ 1572 (June 26, 2004) available at <http://bmj.bmjournals.com/cgi/content/full/328/7455/1572?maxtoshow=&HITS=10&hits=10&RESULTFORMAT=1&title=the+new+tobacco&andorexacttitle=p+hrase&andorexacttitleabs=&andorexactfulltext=&and&searchid=1&FIRSTINDEX=0&sortspec=relevanc&resourcectype=HWCIT>, (last visited July 12, 2006).

found no independent relationship between television advertising exposure and children's calorie intake. Parental eating habits, in contrast, had a significant impact on the quality of the children's diets.<sup>91</sup> Indeed, several studies have demonstrated that, in children, food acceptance is affected by what they see other people eating, what foods they are offered, and how parents control food intake in the home.<sup>92</sup> To the extent that consumer choices are independent of marketing claims, deceptive trade practice suits may encounter difficulties. Moreover, truthful advertising is commercial speech protected under the First Amendment.<sup>93</sup> FDA's authority to regulate food labeling is subject to the commercial speech doctrine even though labeling is "compelled speech."<sup>94</sup> Advertisers' freedom of speech extends to speech directed to children. Congress has enacted legislation blocking the Federal Trade Commission (FTC) from preventing advertisers from targeting children.<sup>95</sup>

Claims based on marketing to children face special problems. Parents, not their children, are the ones who decide a) whether to purchase and operate a television; b) whether to allow their children to watch it; c) which programs to permit their children to see and how often; and d) whether and to what extent to permit the children's "pester power" to have any effect on the family's purse strings. After all, children cannot drive to the supermarket, and in general their parents control their funds. To a significant extent, television viewing by both children and adults has shifted over the past few decades from broadcast programming toward cable channels with fewer or no overt advertisements.<sup>96</sup> Standard children's cable channels, such as Nickelodeon, targeted specifically by CSPI, air less outside product advertising than does broadcast television.<sup>97</sup> Even in network broadcasting, there has been no substantial increase in expenditures on food advertisements or exposure to food advertisements in the last 10 years.<sup>98</sup>

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<sup>91</sup> R.N. Bolton, *Modeling the Impact of Television Food Advertising on Children's Diets*, 6 CURRENT ISSUES & RES. ADVERTISING 173, 187-91 (1983), cited in T.J. Zywicki, D. Holt, M.K. Ohlhausen, *Obesity and Advertising Policy*, 12 Geo. Mason L. Rev. 979-1001 (2004) at 1000, note 109.

<sup>92</sup> L. Cooke, et al., *Demographic, Familial and Trait Predictors of Fruit and Vegetable Consumption by Pre-School Children*, 7 PUBLIC HEALTH NUTR. 295-302 (2004); C.A. Vereecken, et al., *Influence of Mother's Educational Level on Food Parenting Practices and Food Habits of Young Children*, 43 APPETITE 91-103 (2004).

<sup>93</sup> *Virginia Board of Pharmacy v. Virginia Citizens Consumer Council, Inc.*, 425 U.S. 748, 762 (1976). Regulation of commercial speech must be narrowly tailored to directly advance a substantial governmental interest. *Hudson Gas & Electric v. Public Service Comm'n*, 447 U.S. 557, 566 (1980). A complete prohibition on dissemination of truthful, non-misleading consumer information is unconstitutional. *44 Liquormart, Inc. v. Rhode Island*, 517 U.S. 44 (1996).

<sup>94</sup> *United States v. United Foods, Inc.*, 533 U.S. 405 (2001).

<sup>95</sup> 15 U.S.C.S. § 57a(h).

<sup>96</sup> M. Greg Bloche, *Obesity and the Struggle Within Ourselves*, 93 GEORGETOWN L.J. 1335, 1341 (Apr. 2005). Children now watch substantially more cable television than "free" children's television (networks, network affiliates, syndications and Public Broadcasting System (PBS)) combined. L. Rublin, *Tune Out: Who Wins, Who Loses as Children Spend More Time on PCs and Less Watching TV*, 1999 BARRON'S 37 (1999), 1999 WL BARRONS 29061696. Premium channels such as HBO, Family, and Disney channel generally broadcast no regular advertising except for their own products (although Disney does run "sponsorship" ads for McDonald's). If television is influential, critics of advertisers have to concede that its persuasive powers are not limited to messages conveyed in ads. Overweight characters are negatively portrayed in television programming. Kaiser Foundation, *Issue Brief: The Role of Media in Childhood Obesity*, 7 (Feb. 2004).

<sup>97</sup> D. Kunkel & W.Gantz, *Children's Television Advertising and the Multi-channel Environment*, 42 J. COMM. 134, 142-143 (1992).

<sup>98</sup> See D. Jaffe, *Kids, Cartoons, and Cookies: Should We Restrict the Marketing of Food to Children?* Presentation for Cato Policy Forum (June 7, 2004) (presenting Nielsen Media Research Inc. data), available at <http://www.cato.org/events/jaffe-06-07-04.ppt>, (last visited July 6, 2006). In fact, children today may be exposed to fewer food advertisements on television than in the past. *Perspectives on Marketing, Self-regulation, & Childhood Obesity: A report on a Joint Workshop of the FTC & the Department of Health & Human Services*, Executive Summary, at viii (Apr. 2006).

Moreover, frequently engaging in video games is an important risk factor for obesity, even though the games are largely commercial-free.<sup>99</sup> Then, too, the distinction between food and tobacco that has such profound significance in other circumstances is important here as well: "Cigarette . . . consumption by minors, as a result of targeted advertisements, is illegal, and clearly affronts parental authority and autonomy. . . . Fast food does not have the same legal consequences."<sup>100</sup>

## B. *Energy Balance*

Reduced to its simplest terms, weight gain or loss depends upon the balance between the supply of and demand for energy.<sup>101</sup> The animal that ingests less energy than it expends will generally lose weight. If necessary to meet its energy requirements, the animal will catabolize its own bodily protein structure. The animal that ingests more energy than it burns, in contrast, will tend to gain weight. It stores the excess as fat against the possibility of lean times in future. In nature, times of abundance alternate with times of scarcity. One of the great achievements of agriculture and of the food industry in the United States, though, especially in the late 20th century and now the 21st, is that for the great majority of the population, there simply are no times of scarcity. Food is nearly always available, affordable and plentiful.

To point merely to our agricultural bounty, however, is to oversimplify. A superabundance of food does not compel one to overeat. Among those who do, the effect varies significantly from one person to another.<sup>102</sup> Among the risk factors for obesity are a relative decrease in basal metabolism, decreased lipid oxidation in the fasting state, reduced spontaneous physical activity and lower levels of sympathetic nervous system activity.<sup>103</sup> Besides diet, influential factors may include exercise (both recreational and occupational); genetics; metabolic/hormonal factors; cultural, socioeconomic, psychological and behavioral influences; pharmaceuticals; parental weight, fetal development and weight at birth; sleep deprivation; and even, sometimes, viral infection (adenovirus-36). We will briefly examine these factors.

### 1. *Diet*

No food is inherently good or bad.<sup>104</sup> Every food can cause harm; every food can bestow benefits. In this respect, food is similar to medication. Paracelsus (1493-1541) recognized that the only difference between a medicine and a poison is the dose. Con-

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<sup>99</sup> N. Stettler, et al., *Electronic Games and Environmental Factors Associated With Childhood Obesity in Switzerland*, 12 OBES. RES. 896 (2004).

<sup>100</sup> L.J. Munger, Comment, *Is Ronald McDonald the Next Joe Camel? Regulating Fast Food Advertisements Targeting Children in Light of the American Overweight and Obesity Epidemic*, 3 CONN. PUB. INT. L.J. 456, 478 (2004).

<sup>101</sup> D. York & C. Bouchard, *How Obesity Develops: Insights From the New Biology*, 13 ENDOCRINE 143-154 (2000).

<sup>102</sup> J.A. Levine, et al., *Role of Nonexercise Activity Thermogenesis in Resistance to Fat Gain in Humans*, 283 SCL. 212-214 (1999).

<sup>103</sup> E. Ravussin & J.F. Gautier, *Determinants and Control of Energy Expenditure*, 63 (2 Pt. 1) ANN. ENDOCRINOL. (Paris) 96-105 (Apr. 1, 2002).

<sup>104</sup> Memorandum from William R. Steiger, Special Assistant to the Secretary for International Affairs, DHHS, to J.W. Lee, M.D., Director-General, WHO (Jan. 5, 2004), enclosing the "Review of 'Diet, Nutrition, and the Prevention of Chronic Diseases,' Report of a Joint World Health Organization (WHO)/Food and Agriculture Organization (FAO) Expert Consultation (WHO Technical Report Series 916) by the U.S. Departments of Health and Human Services and Agriculture," available at <http://www.commercialalert.org/bushadmincomment.pdf>, (last visited July 6, 2006).

sider vitamins, for example. By definition, humans need but cannot synthesize vitamins. Hence, we must ingest them. Vitamin D, essential for calcium metabolism and healthy bone, can in excess be acutely toxic, causing muscle weakness, apathy, headache, anorexia, nausea, vomiting and bone pain. Chronic exposures can cause all these symptoms, as well as constipation, anorexia, polydipsia, polyuria, backache, hyperlipidemia, hypercalcemia, calcinosis, hypertension and cardiac arrhythmias.<sup>105</sup>

Vitamin A is needed for bone growth and vision. At toxic levels, however (>60-100 mcg/dL), Vitamin A is associated with headache, nausea, vomiting, and drowsiness acutely, and with skin, mucous membrane and musculoskeletal abnormalities chronically. Neurological, hepatic and skeletal problems may also arise.<sup>106</sup>

Pyridoxine, Vitamin B<sub>6</sub>, is active in the metabolism of proteins, carbohydrates and fats. It is needed to synthesize hemoglobin, the indispensable oxygen-carrying molecule in red blood cells. Pyridoxine deficiency results in growth retardation and peripheral neuropathy. Those who consume pyridoxine in excessive quantities, however, lose proprioception, which enables one to regulate posture and movement through stimuli detected by receptors in joints, tendons and muscles.<sup>107</sup>

Not surprisingly, this same principle applies to foods and their constituents. Beta-carotene is found in many leafy vegetables such as spinach, lettuce, and broccoli, and also in carrots, tomatoes and cantaloupe. No one condemns consumption of those foods. But a higher incidence of lung cancer has been reported in smokers consuming high doses of beta-carotene as compared with smokers who did not do so.<sup>108</sup> Celery, that staple of weight-loss diets, is potentially toxic to skin. It can cause primary irritant dermatitis, allergic contact dermatitis, and contact photo-sensitization.<sup>109</sup> Under the right circumstances, it may be possible, if difficult, to gain weight on a celery diet. Edelstein demonstrated that even water can be abused.<sup>110</sup> In short, any food or fluid, no matter how healthful or necessary, can be harmful in excessive quantities; apart from occasionally causing allergic reactions or food borne infections, virtually all foods, in moderation, are safe.

These ideas are neither novel nor foreign. Rather, they reflect common sense. Under the "common knowledge" doctrine, a product is not unreasonably dangerous when its inherent dangers are widely recognized.<sup>111</sup> Under the doctrine of assumption of the

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<sup>105</sup> M. Rosenbloom, "Toxicity, Vitamin," (2005), available at <http://www.emedicine.com/emerg/topic638.htm>, (last visited July 6, 2006).

<sup>106</sup> *Id.* Such effects would be expected if intake exceeded 3,000 mcg/l. Institute of Medicine, *Dietary Reference Intakes*. National Academy Press (2001) at 142.

<sup>107</sup> See, O. Sacks, *The Man Who Mistook His Wife for a Hat: And Other Clinical Tales*, New York: Simon & Schuster (1998) at 43-54.

<sup>108</sup> Alpha Tocopherol, Beta Carotene Cancer Prevention Study Group, *The Effect of Vitamin E and Beta Carotene on the Incidence of Lung Cancer and Other Cancers in Male Smokers*, 330 NEW ENGL. J. MED. 1029-1035 (April 14, 1994).

<sup>109</sup> Extonet, Extension Toxicology Network, Cornell (1993), <http://pmep.cce.cornell.edu/profiles/ex-toxnet/TIB/cutaneous-tox.html>, (last visited July 6, 2006).

<sup>110</sup> E.L. Edelstein, *A Case of Water Dependence*, 68 BR. J. ADDICT. 367 (1973).

<sup>111</sup> See, e.g., *Glassner v. R.J. Reynolds Tobacco Co.*, 223 F.3d 343, 2000 WL 1229061, \*7 (6th Cir. 2000)(affirming district court's dismissal of plaintiff's wrongful death claims against defendant tobacco companies under the common knowledge doctrine). See generally, Restatement (Second) of Torts, § 402A, cmt. I, Restatement (Third) of Products Liability § 2, cmt. j ("[A] seller is not required to warn with respect to products, or ingredients in them, which are only dangerous, or potentially so, when consumed in excessive quantity, or over a long period of time, when the danger, or potentiality of danger, is generally known and recognized. Again the dangers of alcoholic beverages are an example, as are also those of foods containing such substances as saturated fats, which may over a period of time have a deleterious effect upon the human heart." *Id.* See also, *O'Sullivan v. Shaw*, 726 N.E.2d 951 (Mass. 2000)(pool owners under no duty to warn 21 year-old-man who had swum in pool before not to dive into its shallow end).

risk, one who voluntarily exposes himself to a risk a reasonable person would recognize cannot be heard to complain about injuries occasioned by that exposure.<sup>112</sup> Every reasonable person beyond toddlerhood knows that overeating eventuates in overweight. As the *Pelman* court held, a plaintiff who, in violation of the dictates of common sense, uses a product to excess, has no claim against its manufacturer. Nor is there any duty to warn of obvious risks.<sup>113</sup>

Alcohol is a good example of a product that is lawfully marketed but capable of causing harm to those who consume it. Manufacturers and marketers of alcoholic beverages are under no duty to warn about the risks to the consumer's health generally, nor of developing alcoholism specifically, from "prolonged and excessive" consumption of their products.<sup>114</sup> The analysis does not change when the consumer was a minor who died, even where the minor's use was foreseeable.<sup>115</sup>

Similarly, the near-universal recognition that obesity has many causes, including overeating, tends to defeat both claims that industry is culpable for its prevalence in the population and claims that food companies are under a duty to warn us about what we already know.<sup>116</sup>

## 2. Fast Food

In *Pelman*, of course, the defendant is a fast food company. The fast food industry has attracted much public interest, media coverage, and considerable criticism.<sup>117</sup> Fast foods include foods from vending machines, self-service restaurants, convenience groceries and franchised food restaurants, and they are popular for meals and snacks. Such foods are rightly named: they are quickly procured. They are also reasonably priced and often a convenient alternative to home cooking. Though often high in calories, fast foods provide

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<sup>112</sup> See, e.g., *Thurmond v. Prince William Professional Baseball Club, Inc.*, 265 Va. 59, 574 S.E.2d 246 (2003) (baseball fan barred from recovery for personal injury sustained when he was hit by a foul ball); see also, Restatement (Second) of Torts § 496A (1995). Professor Epstein thinks assumption of the risk is an especially powerful defense in obesity claims: "The decision to eat with knowledge of the consequences should offer an air-tight assumption of the risk defense—except for lawyers who think that tort law always supplies remedies when legislation does not cut in the direction they want. The dangers of obesity have been raised by so many people for so long, that it is hard to find some concealed plot that even begins to resemble the alleged coverup in tobacco. The pressures from advertisements come from all sources, and people are subject to too many influences to make credible the claim that consumers are uninformed fat-craving folks with clogged arteries who are buying fast food because of irresistible urges. Assumption of risk is a nice way to summarize these results. There is enough information out there: get a grip on yourself—not a lawyer." R.A. Epstein, *What (Not) to do About Obesity: A Moderate Aristotelian Answer*, 93 GEORGETOWN L. J. 1361, 1385 (Apr. 2005). See also, Lewis Smith and Sam Lister, *Smokers Forfeit Legal Rights*, TIMES OF LONDON, (Dec. 17, 2005), available at <http://www.timesonline.co.uk/article/0,,2-1936975,00.html>, (last visited June 23, 2006).

<sup>113</sup> *Pelman I*, *supra* note 16, 237 F.Supp.2d at 540; Restatement (Third) of Torts, Prods. Liab. § 402 cmt. j.

<sup>114</sup> *Joseph E. Seagram and Sons, Inc. v. McGuire*, 814 S.W.2d 385, 386-87, 388 (Tex. 1991); *Garrison v. Heublein*, 673 F.2d 189, 192 (7th Cir. 1982).

<sup>115</sup> *Pemberton v. American Distilled Spirits Co.*, 664 S.W.2d 690, 692 (Tenn. 1984).

<sup>116</sup> "[T]he risks of eating foods that are high in fat are well known, even by lay people who lack a sophisticated understanding of the relationship between diet and health." Anthony J. Sebok, *The "Big Fat" Class-action Lawsuit Against Fast Food Companies: Is it More Than Just a Stunt?* (Aug. 14, 2002) available at <http://writ.news.findlaw.com/sebok/20020814.html>, (last visited July 12, 2006). It is fair in this context to ask what the efficacy of more elaborate warnings would be. Obesity rates have not diminished in the dozen years since nutritional labels were required on packaged foods. M. Lederhausen, paraphrased in M. Martin, *Nutrition Labels for Restaurant Meals Urged; But Firms Doubt it will Cripple Obesity*, CHICAGO TRIBUNE, (Nov. 1, 2003) at 9, zone C.

<sup>117</sup> See, e.g., E. Schlosser, *Fast Food Nation: The Dark Side of the All-American Meal*, PERENN (2002).

essential macronutrients such as fats, carbohydrates, and proteins. For some Americans, the economy of fast food may make access to needed protein much easier than would otherwise be possible. Fast food chains and restaurants have responded to the public's increasing awareness of nutrition by making ingredient and nutritional information available on their menus or in separate documentation available on request.<sup>118</sup> In fact, McDonald's recently announced that it would provide nutrition facts on the packaging for most of its menu.<sup>119</sup> This way, the consumer can look at the wrapper instead of having to go to the company's website or asking for the information at the counter. To maintain a healthful diet, of course, it is necessary to choose fast foods carefully and to eat them in moderation. Similar discretion is needed in consuming any food, however.

### 3. Carbohydrates and Proteins

Proteins, carbohydrates and lipids are the bio-macromolecules that make up the primary constituents of living things. A healthful dietary food plan includes a balance of all three.

Despite the popularity of contemporary weight-loss plans that discourage their intake, carbohydrates are the main source of blood glucose, a major fuel for all of the body's cells and the only source of energy for the brain and red blood cells. In reference to carbohydrate-rich foods, nutritionists recommend that people generally choose unrefined foods such as fruits, vegetables, peas, beans, and whole-grain products in preference to refined processed foods such as soft drinks, desserts, candy, and sugar. If eaten in excess, especially over a period of many years, the simple carbohydrates found in large amounts in refined foods may accelerate development of a number of disorders, including diabetes and hypoglycemia (low blood sugar). Yet all forms of carbohydrates (i.e., monosaccharides, disaccharides, oligosaccharides and polysaccharides) have some nutritional value, and may be consumed in moderation safely and without undue risk. At the same time, no food consumed in excess is risk-free.

Proteins are complex, high molecular weight organic compounds comprised of amino acids. They are essential to the structure and function of all living cells and are essential for growth and development. Proteins are needed to manufacture hormones, antibodies, enzymes, and tissues, and in some circumstances can be an energy source. They also help maintain proper acid-alkali balance.

Dietary proteins are categorized into two different groups, depending on the amino acids they provide. Complete proteins, the first group, contain ample amounts of all of the essential amino acids. Complete proteins are found in meat, fish, poultry, cheese, eggs, and milk. Incomplete proteins, the second group, contain only some of the essential amino acids. Incomplete proteins are also found in a variety of foods, including grains, legumes, and green leafy vegetables. We should recognize that no one food meets all our dietary requirements, and no food by itself is so dangerous as to be off-limits unless the consumer is allergic to it.

### 4. Fats

Although much attention has focused on the need to reduce dietary fat,<sup>120</sup> fat consumption is essential. Throughout life, we need fat to provide energy and support growth. Fat,

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<sup>118</sup> See, e.g., the McDonald's Corporation's "Food, fitness, and nutrition" section of its home page, available at <http://www.mcdonalds.com/usa/eat.html>, (last visited July 6, 2006).

<sup>119</sup> See <http://www.cnn.com/2005/HEALTH/10/25/mcdonalds.ap/index.html>, (last visited July 6, 2006).

<sup>120</sup> Since the early 1970s, Americans' average fat intake has actually dropped from over 40% of total calories to 34%; average serum cholesterol levels have dropped as well. G. Taubes, *supra* note 18.

in fact, is the most concentrated source of energy available to the body. Lipids, including fats and fatty acids, constitute approximately 32.8% of the energy in the American diet.<sup>121</sup> Because fat is energy-rich and provides 9 kcal of energy per gram, individuals can obtain adequate energy through reasonable daily consumption. Humans require fats to absorb Vitamins A, D, E, and K. Fats also provide essential fatty acids, important to many metabolic processes. During infancy and childhood, fat is necessary for normal nerve and brain development, including development of intelligence and visual acuity.<sup>122</sup> Total abstinence from fat would be fatal. Fortunately, it is not possible.

Lipids are small molecules found in both animal and plant tissue. They have four major functions: 1) they serve as structural components of biological membranes;<sup>123</sup> 2) they provide energy reserves, predominately in the form of triglycerols; 3) they and their derivatives may serve as vitamins and hormones; and 4) they help form lipophilic bile acids, which help the body to absorb lipids.<sup>124</sup> Lipids are characterized by their insolubility in water and can be classified into six major groups: fatty acids, triglycerides, phospholipids, lipids not containing glycerol, glycolipids, and synthetic lipids.<sup>125</sup> For purposes of this article, only fatty acids will generally be addressed.

There are three major categories of fatty acids—saturated, polyunsaturated, and monounsaturated. The majority of fatty acids are acquired through the diet. Most, however, can also be synthesized by the body, with the exception of some highly unsaturated fatty acids (linoleic acid and linolenic acid) that can be acquired by consuming a variety of vegetables.

The liver uses saturated fats to manufacture cholesterol. Cholesterol is an extremely important biological molecule that is vital to membrane structure. The synthesis and utilization of cholesterol must be tightly regulated to prevent over-accumulation and abnormal deposition within the body; over-accumulation may result in atherosclero-

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<sup>121</sup> "Trends in intake of energy and macronutrients-United States, 1971-2000," 53 (4) MMWR 80-82 (6 Feb. 2004), available at <http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5304a3.htm>, (last visited July 6, 2006). Despite recommendations that Americans reduce their fat intake, the benefits of doing can be exaggerated. Curtailing intake of fat and increasing consumption of vegetables, fruits, and grain in a large randomized cohort of post-menopausal women followed over 8.1 years did not reduce the risk of coronary heart disease, stroke, or cardiovascular disease, B.V. Howard, et al., *Low-fat Dietary Pattern and Risk of Cardiovascular Disease*, 295 (6) JAMA 655-666 (2006); invasive breast cancer, R.L. Prentice, et al., *Low-fat Dietary Pattern and Risk of Invasive Breast Cancer*, 295 (6) JAMA 629-642 (2006); or colorectal cancer, S.A.A. Beresford, et al., *Low-fat Dietary Pattern and Risk of Colorectal Cancer*, 295 (6) JAMA 643-654 (2006). In the Polyp Prevention Trial, fat, fruit, and vegetable intake were similarly modified, yet there was no effect on recurrence of colonic polyps in 2079 participants followed over four years. E. Lanza, et al., *Implementation of a 4-year, High-Fiber, High-Fruit and -Vegetable, Low-Fat Dietary Intervention: Results of Dietary Changes in the Polyp Prevention Trial*, 74 AM. J. CLIN. NUTR., 387-401 (2001). "People on low-fat diets initially lose a couple of kilograms, as they would on any diet, and then the weight tends to return. After 1 to 2 years, little has been achieved." G. Taubes, *supra* note 18.

<sup>122</sup> See, S.C. Hardy, & R.E. Kleinman, *Fat and Cholesterol in the Diets of Infants and Young Children: Implications for Growth, Development, and Long-term Health*, 125 (5 Pt. 2) J. PEDIATR. S69-77 (Nov. 1994). The human body synthesizes all of the cholesterol it needs from acetyl coenzyme A. In general, the larger the amount of dietary cholesterol absorbed, the smaller the role of its biosynthesis. Excessively restricting fat in children's diets may actually do more harm than good. R.E. Olson, *Is it Wise to Restrict Fat in the Diets of Children?* 100(1) J. AM. DIET. ASSOC. 28-32 (Jan. 2000).

<sup>123</sup> The importance of this function is clear. "Changing the proportion of saturated to unsaturated fats in the diet changes the fat composition in these membranes. This could conceivably change the membrane permeability, which controls the transport of everything from glucose, signaling proteins, and hormones to bacteria, viruses, and tumor-causing agents into and out of the cell." G. Taubes, *supra* note 18.

<sup>124</sup> See, e.g., Institute for Functional Medicine, <http://www.functionalmedicine.org/>, (visited July 6, 2006).

<sup>125</sup> There are competing ways to classify lipids. See, e.g., E. Fahy, et al., "A comprehensive classification system for lipids," 46 J. LIPID RES. 839-862 (2005), available at <http://www.jlr.org/cgi/content/full/46/5/839>, (last visited June 28, 2006).

sis, which predisposes to heart attack and stroke. On the other hand, the prevalence of obesity in the U.S. has increased during the same period that dietary fat intake (both in absolute terms and as a percentage of total dietary energy) has decreased, suggesting that increased exercise (discussed below) must be more heavily emphasized.<sup>126</sup> As with all other biologically significant molecules, balance is key; excessive cholesterol entails risk but deficiencies do, too. Low cholesterol levels may be linked to hemorrhagic strokes.<sup>127</sup> The American College of Physicians has suggested that cholesterol reduction, while valuable for those with high short-term risk of death secondary to coronary heart disease, offers “much smaller ... or ‘uncertain’ benefits for everyone else.”<sup>128</sup>

Polyunsaturated fatty acids are found in corn, soybean, safflower, and sunflower oil, as well as certain fish oils. Fish, especially oily fish, is rich in long-chain omega-3 polyunsaturated fatty acids. Consuming two servings (about 8 ounces) of fish weekly is associated with a reduced risk of both sudden death and death from coronary artery disease in adults.<sup>129</sup> In fact, fish intake is associated with decreased risks of cardiovascular disease generally.<sup>130</sup> There is recent evidence that fish oil supplementation may also have protective effects on exercise-induced bronchoconstriction in asthmatics.<sup>131</sup> More generally, polyunsaturated fats play an important role in the structure and function of cellular phospholipid membranes, which contain relatively high concentrations of polyunsaturated fatty acids, such as docosahexaenoic acid (DHA).<sup>132</sup> Without the cellular integrity that fatty acids provide, human survival would be impossible. In infancy, lipids are the most important energy source and are necessary for growth and exercise.<sup>133</sup> It is recommended that about 30-40% of a toddler’s diet, and about 25-35% of an older child’s diet, come from fat.<sup>134</sup> Maternal energy deficiency, possibly exacerbated by very low fat intake, is one key determinant in low birth weight; inadequate dietary fat in childhood can hamper sexual maturation.<sup>135</sup>

Monounsaturated fatty acids contain only one double bond and are found mostly in vegetable and nut oils such as olive, peanut, and canola. The monounsaturated fatty acids appear to lower blood cholesterol when substituted for saturated fats.<sup>136</sup>

A specific type of monounsaturated fat, which has received much press and is invoked as the basis of the allegations in the YUM! Brands suit, is known as *trans* fat (or *trans*

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<sup>126</sup> A.H. Lichtenstein, et al., *Dietary Fat Consumption and Health*, 56 (5 Pt. 2) NUTR. REV. S13-19 (May 1998).

<sup>127</sup> G. Taubes, *supra* note 18.

<sup>128</sup> *Id.*

<sup>129</sup> P.M. Kris-Etherton, et al., American Heart Association Nutrition Committee, *Fish Consumption, Fish Oil, Omega-3 Fatty Acids, and Cardiovascular Disease*, 106 CIRCULATION 2747-2757 (2002).

<sup>130</sup> B.V. Howard & D. Kritchevsky, *Phytochemicals and Cardiovascular Disease: A Statement for Healthcare Professionals From the American Heart Association*, 95 CIRCULATION 2591-2593 (1997); E.M. Balk, et al., *Effects of Omega-3 Fatty Acids on Serum Markers of Cardiovascular Disease Risk: A Systematic Review*, 184 ATHEROSCLEROSIS 237-246 (2006). Omega-3 fatty acids may also have therapeutic benefits in childhood depression. H. Nemets, et al., *Omega-3 Treatment of Childhood Depression: A Controlled, Double-Blind Pilot Study*, 163 AM. J. PSYCHIATRY 1098-1100 (June 2006), abstract available at <http://ajp.psychiatryonline.org/cgi/content/abstract/163/6/1098>, (last visited June 23, 2006).

<sup>131</sup> T.D. Mickelborough, et al., *Protective Effect of Fish Oil Supplementation on Exercise-Induced Bronchoconstriction in Asthma*, 129 (1) CHEST 39 (Jan. 2006).

<sup>132</sup> C.L. Jensen & W.C. Heird, *Lipids With an Emphasis on Long-Chain Polyunsaturated Fatty Acids*, 29 (2) CLIN. PERINATOL. 261-281 (2002).

<sup>133</sup> R. Uauy, et al., *Fat Intake During Childhood: Metabolic Responses and Effects on Growth*, 72 (5 suppl.) AM. J. CLIN. NUTR. 1354S-1360S (2000).

<sup>134</sup> Mayo Clinic, “Nutrition for kids: Guidelines for a healthy diet,” available at <http://www.mayoclinic.com/health/nutrition-for-kids/NU00606>, (last visited July 15, 2006).

<sup>135</sup> A.H. Lichtenstein, et al., *supra* note 126.

<sup>136</sup> Monounsaturated fats raise HDL, at least compared to carbohydrates, and lower LDL. G. Taubes, *supra* note 18.

fatty acids). This type of fat is formed when liquid oils are made into solid fats such as shortening and hard margarine.<sup>137</sup> A small amount of *trans* fat, however, may be found naturally in some animal-based foods. *Trans* fat behaves like saturated fat in the sense that it may increase low-density lipoprotein (“bad”) cholesterol, linked to increases in the risk of coronary heart disease.<sup>138</sup> *Trans* fat may be found in some of the same foods as saturated fat, such as vegetable shortenings, some margarines, crackers, candies, cookies, snack foods, fried foods, baked goods and other processed foods made with partially hydrogenated vegetable oils.

*Trans* fats may be created when small amounts of hydrogen are added to vegetable oil, a process called hydrogenation.<sup>139</sup> The hydrogenation process raises the melting point of the oil, thereby allowing the oil to stay solid at room temperature. Hydrogenation increases the shelf life and flavor stability of foods containing these fats.<sup>140</sup> FDA has adopted a regulation requiring food makers to report the *trans* fat content in their products. According to FDA’s labeling regulations, if a fat or oil ingredient is completely hydrogenated, the name in the ingredient list will include the term “hydrogenated” or, if partially hydrogenated, the name in the ingredient list will include the term “partially hydrogenated.”<sup>141</sup>

### C. *Tobacco v. Food*

To model tobacco litigation, proponents of obesity litigation may attempt to obscure some of the significant differences between tobacco and food. Food and nutrients provide the energy and building materials for the components essential to the growth and survival of living organisms. Proteins, fats and carbohydrates all contribute to the total energy pool. Each of these bio-macromolecules, even those that are indigestible, provides health benefits. A healthful daily food plan includes a balanced diet of protein-containing foods, dairy products, fresh vegetables, fruits, grains and water.

In contrast, tobacco confers essentially no health benefits. The harms of smoking are well-known, and will be summarized briefly here. Cigarette smoke contains more than 4000 identified constituents.<sup>142</sup> Compare dietary compounds, discussed above, with the components of cigarette, pipe and cigar smoke. Tobacco smoke contains a complex mixture of chemicals, in the form of both gases and particulates, produced by the burning materials of a cigarette. Smoke exposes us to numerous known toxins.<sup>143</sup> Among these

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<sup>137</sup> See, Center for Food Safety and Applied Nutrition (CFSAN), “Questions and answers about *trans* fat nutrition labeling,” available at <http://www.cfsan.fda.gov/~dms/qatrans2.html#s1q3> (last visited July 12, 2006).

<sup>138</sup> *Id.*

<sup>139</sup> *Id.* This step was taken in the belief that replacing saturated fats with unsaturated fats would be beneficial, because the cholesterol-raising effect of hydrogenated fats was less than that of saturated fats. A. Ascherio, M.D. Katan, & M.J. Stampfer, “*Trans Fatty Acids and Coronary Artery Disease*,” 340 (25) N. ENGL. J. MED. 1933-40 (1999). The evolution in medical thinking on this topic should make judges and policy-makers cautious about creating legally enforceable duties on farmers and food companies to achieve or promote nutritional goals.

<sup>140</sup> CFSAN, *supra* note 137.

<sup>141</sup> 21 C.F.R. § 101.

<sup>142</sup> WHO, *supra* note 75. For a partial list, see, D. Burns, *Cigarette and Cigarette Smoking*, 12 (4) CLIN. CHEST MED. 631-642 (1991). There have been many studies of the chemical constituents of tobacco smoke. See, e.g., A.W. Katzenstein, *Environmental Tobacco Smoke and Lung Cancer Risk: Epidemiology in Relation to Confounding Factors*, 18 ENVIRON. INT. 341-345 (1992), J.L. Repace & A.H. Lowrey, *Risk Assessment Methodologies for Passive Smoking-Induced Lung Cancer*, 10 RISK ANAL. 27-37 (1990), WHO, International Agency for Research on Cancer (IARC), IARC Monographs No. 38: Tobacco Smoke and Involuntary Smoking, Lyon, France, (1986) at 421. Cf., n. 6 *supra*, and associated text.

<sup>143</sup> See, e.g., L. Wallace, et al., *Exposures to Benzene and Other Volatile Compounds From Active and Passive Smoking*, 42 (5) ARCH. ENVIRON. HEALTH, 272-9 (Sept.-Oct. 1987).

are nicotine, nitrogen oxides, ammonia, formaldehyde and methyl isocyanate.<sup>144</sup> The Environmental Protection Agency (EPA) has classified 43 other components of tobacco smoke as known or suspected human carcinogens, including nitrosamines, benzene, benzo(a)pyrene, vinyl chloride, radionuclides and arsenic.<sup>145</sup> Indeed, EPA has classified environmental tobacco smoke as a Class A (known human) carcinogen; only a handful of other substances are designated as members of Class A, such as asbestos, arsenic, benzene and radon gas.<sup>146</sup> In fact, 20 chemicals in tobacco smoke have been shown to cause lung tumors in laboratory animals or humans.<sup>147</sup> Smoking is also associated with non-Hodgkin's lymphoma,<sup>148</sup> and with deep venous thrombosis and pulmonary emboli,<sup>149</sup> and it increases the risk of infection.<sup>150</sup> Smoking has adverse effects on the kidneys and excretory system,<sup>151</sup> male sexual and reproductive function,<sup>152</sup> female fertility<sup>153</sup> and fetal weight.<sup>154</sup> Chemicals in smoke that may play a major role in disease include polycyclic aromatic hydrocarbons and the tobacco-specific nitrosamine 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone.<sup>155</sup>

The mechanisms by which smoking induces harm are not entirely understood and may be complex. When smoke is inhaled through the mouth, it bypasses the filtering action of the nose. While smokers are often able to produce more mucus in response to smoking, they are less able than nonsmokers to move the mucus out of their respiratory trees. This problem arises because cigarette smoke paralyzes and eventually destroys cilia. It also changes the makeup of the mucus-secreting glands and consequently the mucus itself. In addition, a smoker's mucus glands may become plugged and less able to produce mucus. As a result, the smoker's mucus, contaminated with potentially harmful substances, is more likely to become trapped in lung tissue. Other physical changes in a smoker's lung tissue impair the ability of the lungs to take in oxygen. Smoking causes inflammation and fibrosis. It also destroys the alveoli, making the lungs less elastic, reducing surface area and obliterating capillaries. These alterations in lung architecture impair gas exchange. Consequently, both the lungs and the tissues are robbed of the nutrients and oxygen needed to be healthy and to function normally. There are no analogues between these effects and those associated with eating. In fact,

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<sup>144</sup> See, Table 3-1, EPA Report, "Respiratory Health Effects of Passive Smoking," *Lung cancer and their disorders*, EPA/600/6-90/006F (1992) available at <http://www.tabakkontrolle.de/pdf/respiratory%20health%20effects.pdf>, (last visited June 28, 2006).

<sup>145</sup> See, S.S. Hecht, *Tobacco Smoke Carcinogens and Lung Cancer*, 91 (14) J. NAT'L CANCER INST. 1194-1210 (July 21, 1999).

<sup>146</sup> See, e.g., Alaska Division of Air Quality, "Environmental Protection Agency's Class A Carcinogens," available at <http://www.dec.state.ak.us/air/anpms/as/toxics/carc/carchome.htm>, (last visited July 6, 2006). See also, National Cancer Institute, "Avoid Carcinogens at Work," available at <http://www.cancer.gov/cancertopics/understandingcancer/cancer/Slide59>, (last visited July 6, 2006).

<sup>147</sup> S.S. Hecht, *supra* note 145.

<sup>148</sup> R. Talamini, *Smoking and Non-Hodgkin Lymphoma: Case Control Study in Italy*, 115 (4) INT. J. CANCER 606-610 (2005).

<sup>149</sup> V.F. Tapson, *The Role of Smoking in Coagulation and Thromboembolism in COPD*, 2 PROC. AM. THOR. SOC. 71-77 (2005).

<sup>150</sup> L. Arcavi & N.L. Benowitz, *Cigarette Smoking and Infection*, 164 (20) ARCH. INT. MED. 2206-2216 (2004).

<sup>151</sup> See, e.g., S.J. Pinto-Sietsma, et al., *Smoking is Related to Albuminuria and Abnormal Renal Function in Nondiabetic Persons*, 133 ANN. INT. MED. 585-591 (2000).

<sup>152</sup> See, e.g., G. Dorey, *Is Smoking the Cause of Erectile Dysfunction? A Literature Review*, 10 BR. J. NSG. 455-465 (2001).

<sup>153</sup> K.M. Curtis, et al., *Effects of Cigarette Smoking, Caffeine Consumption, and Alcohol Intake on Fecundability*, 146 (1) AM. J. EPIDEMIOL. 32-41 (1997).

<sup>154</sup> I.M. Bernstein, et al., *Maternal Smoking and its Association with Birth Weight*, 106 OBSTET. GYNECOL. 986-991 (Nov. 2005).

<sup>155</sup> Curtis, *supra* note 153.

there is evidence that at least some of the harms of smoking might be diminished by sound nutritional practices.<sup>156</sup>

In addition to the effects that active cigarette smoke may have on the user, there are also passive effects on nonsmokers that can occur via secondhand smoke.<sup>157</sup> Passive or secondhand smoking is defined as breathing in the smoke produced by active smokers. In general, the nonsmoker breathes in side stream smoke from the burning tip of the cigarette and the mainstream smoke that has been inhaled and then exhaled by the smoker.<sup>158</sup> Secondhand smoke is a major source of indoor air pollution.<sup>159</sup> Secondhand smoke may affect nonsmokers in the short term in a number of ways. Large numbers of non-smokers are “exposed to, absorb, and metabolize significant amounts of second-hand smoke . . .”<sup>160</sup> Some of the immediate effects of passive smoking include eye irritation, headache, cough, sore throat, dizziness, and nausea. Asthmatics can acutely decompensate when exposed. Even brief exposure to secondhand smoke can have a measurable effect on the hearts of nonsmokers. In just 30 minutes of exposure, coronary blood flow of a nonsmoker can be reduced.<sup>161</sup> In the long term, the significance of exposure of nonsmokers to secondhand smoke is more controversial. The stronger evidence appears to support the notion that a causal relation between environmental tobacco smoke and mortality is doubtful.<sup>162</sup> There have been reports, however, of an increased risk of a range of smoking-related diseases among those exposed to secondhand smoke. For example, non-smokers exposed to passive smoking in the home are said to face a significantly increased risk of heart disease and lung cancer.<sup>163</sup> A review of the risks of cancer from exposure to secondhand smoke by the International Agency for Research on Cancer (IARC) determined that, “the evidence is sufficient to conclude that involuntary smoking is a cause of lung cancer in never smokers.”<sup>164</sup> Environmental tobacco smoke has been called the third-leading preventable cause of death, after active smoking and alcohol.<sup>165</sup> Whether secondhand smoke causes long-term problems for never smokers, however, or whether its harms are strictly short-term, the distinction is clear: A’s smoking can hurt B.

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<sup>156</sup> L.M. Butler, et al., *Dietary Fiber and Reduced Cough with Phlegm*, 170 AM. J. RESP. CRIT. CARE MED. 279-287 (2004)(high-fiber diets may reduce the incidence of chronic respiratory symptoms both in smokers and non-smokers).

<sup>157</sup> DHHS, “The Health Consequences of Involuntary Exposure to Tobacco Smoke: A Report of the Surgeon General,” Atlanta, Ga.: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, Coordinating Center for Health Promotion, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health (2006), available at <http://www.surgeongeneral.gov/library/secondhandsmoke/report/>, (last visited June 29, 2006).

<sup>158</sup> *Id.* (Message from Michael O. Leavitt, Secretary of HHS, <http://www.surgeongeneral.gov/library/secondhandsmoke/report/secretarymessage.pdf>, (last visited June 20, 2006).

<sup>159</sup> *Id.*

<sup>160</sup> See, “Respiratory Health Effects of Passive Smoking,” *supra* note 144, at 1-3.

<sup>161</sup> R. Otsuka, *Acute Effects of Passive Smoking on the Coronary Circulation in Healthy Young Adults*, 286 JAMA 436-441 (2001).

<sup>162</sup> J.E. Custons & G.C. Kabat, *Environmental Tobacco Smoke and Tobacco Related Mortality in a Prospective Study of Californians, 1960-1998*, 326 (7398) BMJ 326-336 (2003) available at <http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=155687>, (last visited June 26, 2006).

<sup>163</sup> M.R. Law, et al., *Environmental Tobacco Smoke Exposure and Ischemic Heart Disease: An Evaluation of the Evidence*, 315 BMJ 973-80 (1997). See also, A.K. Hackshaw, et al., *The Accumulated Evidence on Lung Cancer and Environmental Tobacco Smoke*, 315 BMJ 980-88 (1997); Cf., *Broin v. Phillip Morris Cos.*, 641 So. 2d 888 (Fla. D.C. App. 1994) (flight attendants alleged health harms secondary to exposure to smoke from passengers’ cigarettes).

<sup>164</sup> *Tobacco Smoke and Involuntary Smoking*, 83 IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Lyon, France (2002).

<sup>165</sup> See, S.A. Glantz & W.W. Parmley, *Passive Smoking and Heart Disease: Epidemiology, Physiology and Biochemistry*, 83 CIRCULATION 1 (1991) at 10.

The self-destructive dietary habits of even the most corpulent overeater pose no threat to those seated at the same table.

Although fast foods may have lower nutritional value than those prepared at home, if eaten in moderation they still provide some of the essential nutrients needed to sustain a healthful diet. In contrast, there is no such thing as “moderate” safe smoking. Smoking is harmful even in low doses. The lungs were designed for immediate gas exchange. Hence, absorption through the lungs is very fast and highly efficient. Carbon monoxide appears in a smoker’s blood within the first few puffs. Another distinction is notice. People who eat too much get immediate feedback, typically in the form of indigestion and, later, an expanding waistband, correctable with exercise and a properly balanced diet. In contrast, smokers can take years to develop the symptoms of lung cancer, emphysema or cardiac disease, often well after permanent anatomic change.

#### D. Exercise

Proponents of obesity litigation will likely seek to distract attention from the importance of exercise. The President’s Council on Physical Fitness and Sports, however, has declared that, “inactivity might be a far more significant factor in the development of obesity than overeating.”<sup>166</sup>

Lower rates of energy expenditure predispose to obesity.<sup>167</sup> As a nation, we are much less physically active than we once were.<sup>168</sup> Approximately 68% of U.S. adults do not

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<sup>166</sup> J.H. Wilmore, for The President’s Council on Physical Fitness and Sports (2004), available at [www.fitness.gov/activity/activity7/obesity/obesity.html](http://www.fitness.gov/activity/activity7/obesity/obesity.html), (last visited August 8, 2006), quoting J. Wilmore & D. Costill, *Physiology of Sports and Exercise*, Champaign, Ill: Human Kinetics (1994). Accord, S. Blair & M. Nichaman, *The Public Health Problem of Increasing Rates of Obesity and What Should be Done About It*, 77 MAYO CLIN. PROC. 109-113 (2002); R.P. Troiano, et al., *Energy and Fat Intake of Children and Adolescents in the U.S.: Data From the National Health and Nutrition Examination Surveys*, 72 (5) AM. J. CLIN. NUTR. 1343S-1353S (2000). Walking as little as 15 extra minutes per day might be enough to prevent increases in weight gain in most of the population. J.O. Hill, et al., *Obesity and the Environment: Where Do We Go From Here*, 301 (5633) SCI. 598 (2003). The WHO states that moderate physical activity is “of profound significance.” WHO Consultation on Obesity, *Obesity: Preventing and managing the global epidemic*, Geneva: WHO (2002) at 116. Data from the Aerobics Center Longitudinal Study suggest that low cardiovascular fitness may account for all of the excess all-cause mortality among obese men. C.D. Lee, et al., *Cardiorespiratory Fitness, Body Composition, and All-Cause and Cardiovascular Disease Mortality in Men*, 69 AM. J. CLIN. NUTR. 373-380 (1999). The same may also be true of women. S.W. Farrell, et al., *The Relation of Body Mass Index, Cardiorespiratory Fitness, and All-Cause Mortality in Women*, 10 OBES. RES. 417-423 (2002). In girls, declines in activity levels correlate with increased adiposity. S.V. Kumm, et al., *Relation Between the Changes in Physical Activity and Body-Mass Index During Adolescence: A Multicentre Longitudinal Study*, 366 (9482) LANCET 301-307 (June 2005).

<sup>167</sup> E. Ravussin & B.A. Swinburn, *Pathophysiology of Obesity*, 340 LANCET 404-408 (1992); E. Ravussin & E. Danforth, Jr., *Beyond Sloth—Physical Activity and Weight Gain*, 283, SCI. 184-185 (1999). Research from the Mayo Clinic suggests that in the course of a normal day the obese tend to be less physically active than lean individuals and generally burn around 350 fewer calories. *Mayo Clinic Discovers a Key to Low Metabolism—Major Factor in Obesity*, SCI. DAILY (Jan.27, 2005), [www.sciencedaily.com](http://www.sciencedaily.com), (last visited July 6, 2006).

<sup>168</sup> Fewer than half of American adults get the old minimum amount of recommended exercise (thirty minutes of vigorous activity daily, four days per week); and one American in four never exercises voluntarily. Centers for Disease Control and Prevention, *Prevalence of No Leisure-Time Physical Activity*, 53 (4) MMWR 82-86 (Feb. 6, 2004). Estimates from the National Center for Health Statistics are even lower: fewer than one American in three engages in a minimum recommended amount of exercise, 30% exercise “a little,” and 40% of Americans do not exercise at all. D. Brown, *Study Says 38% of Adults are Sedentary in Leisure Time*, THE WASH. POST (Apr. 8, 2002) at A-2. In an average week, one in four California adults—6.8 million adults in all—does not walk at all for transportation or leisure. Nearly half of adults there walk less than one hour during an average seven-day period, including those who do not walk at all and those who walk up to one hour. Only one-third of adult Californians walk a total of two hours or more per week. E.R. Brown, et

engage in any regular physical activity.<sup>169</sup> Sedentary lifestyle appears to be even more common among Hispanic and Latino and black adults than among white adults.<sup>170</sup> In developed countries such as ours, “people must pay for undertaking, rather than be paid to undertake, physical activity.”<sup>171</sup> The payment is the dedication of leisure time to exercise.

Nowhere is this change more apparent than among children. Children in the United States spend an estimated 75% of the day being inactive.<sup>172</sup> In 1997, 20.2% of children walked to school, but in 2001, that fraction had dropped to 12.5%.<sup>173</sup> Based on data from the Center for Disease Control and Prevention’s (CDC’s) National Health and Nutrition Examination Survey, and Youth Risk Behavior Survey and from the U.S. Department of Agriculture’s (USDA’s) Nationwide Food Consumption Survey, nutrition consultant Lisa Sutherland recently reported that children are eating only 1% more calories today than they were 20 years ago, but they are exercising 13% less.<sup>174</sup> One-third of parents acknowledge that their children exercise less than they did at the same age.<sup>175</sup> Physical education (PE) is given short shrift in many schools. According to the Institute of Medicine, only 8% of elementary schools and 6% of middle and high schools provide daily PE for the whole school year and fewer than 30% of middle and high schools require PE as part of their curricula.<sup>176</sup> The only state that requires K-12 physical education

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al., *Half of California Adults Walk Less than One Hour Each Week*, Los Angeles: UCLA Center for Health Policy Research (2005) at 1, available at <http://www.healthpolicy.ucla.edu/pubs/publication.asp?pubID=160>, (last visited July 6, 2006). More than one in four California adolescents—nearly one million in all—gets less than the recommended levels of physical activity. A reported 240,000 get no activity at all. S.H. Babey, et al., *Access to Safe Parks Helps Increase Physical Activity Among Teenagers*, Los Angeles: UCLA Center for Health Policy Research (2005) at 1, available at <http://www.healthpolicy.ucla.edu/pubs/publication.asp?pubID=159>, (last visited July 6, 2006).

<sup>169</sup> American Heart Association, “Heart Disease and Stroke Statistics—2006 Update,” Dallas Texas: American Heart Association (2006), available at <http://circ.ahajournals.org/cgi/content/short/113/6/e85#SEC12> (last visited July 12, 2006).

<sup>170</sup> A.H. Lichtenstein, *Diet and Lifestyle Recommendations Revision 2006, A Scientific Statement from the American Heart Association Nutrition Committee*, 114 CIRCULATION 82-96 (2006) at 85.

<sup>171</sup> D. Lakdawalla & T. Philipson, *The Growth of Obesity and Technological Change: A Theoretical and Empirical Examination*, Nat’l. Bureau of Econ. Research, Working Paper No. 8946, (2002) at 2.

<sup>172</sup> R.S. Strauss, *Psychosocial Correlates of Physical Activity in Healthy Children*, 155 ARCH. PEDIATR. ADOLESC. MED. 897-902 (2001). The average American child is said to spend nine hours each week playing video games, and nearly 87% of American children play video games regularly. D. Walsh, *Eighth Annual MediaWise Video Game Report Card*, Report from the National Institute on Media and the Family (2003). Many have urged promotion of more active lifestyles as critical to curbing obesity. See, e.g., C.J. Crespo, et al., *Television Watching, Energy Intake, and Obesity in U.S. Children: Results from the Third National Health and Nutritional Examination Survey, 1998-1994*, 155 (3) ARCH. PEDIATR. ADOLESC. MED. 360-365 (2001).

<sup>173</sup> R. Sturm, *Childhood obesity—What we can learn from existing data on societal trends, Part 2*, available at [http://www.cdc.gov/pcd/issues/2005/apr/04\\_0039.htm](http://www.cdc.gov/pcd/issues/2005/apr/04_0039.htm), (last visited July 6, 2006). A generation ago, nearly 80% of school children walked to school or rode their bikes. L. Frank, M. Andresen, & T. Schmid, *Obesity Relationships With Community Design, Physical Activity, and Time Spent in Cars*, 27 AM. J. PREVENT. MED. 87-95 (2004). Higher activity levels can actually decrease caloric consumption. L.H. Eptstein, et al., *Influences of Changes in Sedentary Behavior in Energy and Macro Nutrient Intake in Youth*, 81 (2) AM. J. CLIN. NUTR. 361-366 (2005), available at <http://www.jcn.org/cgi/content/fall/81/2/361>, (last visited July 16, 2006). Accord, R. Woo, J.S. Garrow, & F.X. Pi-Sunyer, *Effect of Exercise on Spontaneous Caloric Intake in Obesity*, 36 AM. J. CLIN. NUTR. 470-477 (1982).

<sup>174</sup> L. Sutherland, presentation to the Federation of American Societies for Experimental Biology, reported at CBS Healthwatch, *Why Teens Are Getting Fatter*, (May 12, 2003), available at <http://www.cbsnews.com/stories/2003/05/12/health/main553370.shtml> (last visited July 12, 2006); see also, C.M. Meerschaert, *A Call to Action*, 6 (4) TODAY’S DIETITIAN 32 (Apr. 2004).

<sup>175</sup> M. Connelly, *More Children Are Obese, and More Americans Know It*, NY Times (May 13, 2003) at F5.

<sup>176</sup> Institute of Medicine, *Preventing Childhood Obesity: Health in the Balance, 2005*, Report of the Institute of Medicine of the National Academies of Science (Sept., 2004). In a study of 814 third grade students from 10 different U.S. data-collection sites, the mean duration of physical education was 33 minutes

today is Illinois.<sup>177</sup> Whereas 42% of public schools offered PE in 1991, only 29% did so in 1999.<sup>178</sup> In some jurisdictions, driver education is classified as PE.<sup>179</sup> California randomly samples its school districts each year to determine if they are complying with state law requiring children in grades 1 to 6 to complete 200 minutes of physical education every 10 days, and older students (grades 7 to 12) to complete 400 minutes every 10 days. More than half (37 of 73) of school districts recently reviewed by California's Education Department failed to meet these requirements.<sup>180</sup>

After-school activities are often no better, and may actually divert time from exercise. For example, the American Medical Association (AMA) estimates that children watch an average of four hours of television per day.<sup>181</sup> The relationship of television viewing to obesity is well-established.<sup>182</sup> Among kids 12 to 17, the prevalence of obesity

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twice weekly, with only 25 minutes per week at a moderate to vigorous intensity level. P.R. Nader, *Frequency and Intensity of Activity of Third-Grade Children in Physical Education*, National Institute of Child Health and Human Development Study of Early Child Care and Youth Development Network, 157 ARCH. PEDIATR. ADOLESC. MED. 185-190 (2003).

<sup>177</sup> C.R. Burgeson, et al., *Physical Education and Activity: Results From the School Health Policies and Programs Study 2000*, 174 (1) J. PHYS. ED. REC. DANCE (JOPERD) 20-36 (2003). The percentage of schools that require physical education in each grade declines from around 50% in grades 1-5 to 23% in grade 8 to only 5% in grade 12. Only 8% of elementary schools, 6% of middle/junior high schools, and 6% of senior high schools provide daily physical education or its equivalent for the entire school year for all grades in the school. C.R. Burgeson, et al., *Physical Education and Activity: Results From the School Health Policies and Program Study 2000*, 71 (7) J. SCH. HEALTH 279-293 (2001) at 292.

<sup>178</sup> See Nat'l Center for Health Statistics (NCHS), NCHS Dateline, 116 PUB. HEALTH REP. 273, 274 (2001). Even where physical education is available, it is often taught by untrained instructors, it may involve little actual exertion, and it may not emphasize the enjoyability of physical activity. DHHS, "Physical Activity and Health: A Report of the Surgeon General," DHHS, Atlanta, Ga. (1996).

<sup>179</sup> See, e.g., the description of the Hanover County (VA) Public Schools' program in "Health, physical education, and drivers education," available at [http://hcps2.hanover.k12.va.us/instruction/PhyEd\\_Health/](http://hcps2.hanover.k12.va.us/instruction/PhyEd_Health/), (visited July 6, 2006).

<sup>180</sup> Nanette Asimov, *State's Schools Found Skimping on PE Classes; Less Than 20 Minutes a Day at Majority of Districts Reviewed*, SAN FRANCISCO CHRONICLE, (Thurs., June 8, 2006), at B-1.

<sup>181</sup> See, AM. MED. ASS'N, *Physician guide to media violence* (1996) at 8. Data from the 1988-1994 National Health and Nutrition Examination Survey indicated that 26% of American children (up to 33% of Mexican American and 43% of non-Hispanic black children) watched at least 4 hours of television per day, and these children were less likely to participate in vigorous physical activity. Council on Sports Medicine and Fitness and Council on School Health, American Academy of Pediatrics, "Active healthy living: Prevention of childhood obesity through increased physical activity," 17 (5) *Pediatrics* 1834-1842 at 1835 (May 2006). Children watching excessive television also had greater BMIs (see n. 271, *infra* and associated text) and skin-fold measurements than those who watched <2 hours of television per day. According to researchers David Ludwig and Steven Gortmaker, watching television is "the dominant pastime of youth throughout the industrialized world." See generally, D.S. Ludwig & S.L. Gortmaker, *Programming Obesity in Children*, 364 LANCET 226 (2004). In 99% of American homes a television can be found, and more than 50% have more than two. S.A. French, et al., *Environmental Influences on Eating and Physical Activity* 22 ANN. REV. PUB. HEALTH 319 (2001). The average American currently watches about thirty hours of television each week; the average household has a television turned on nearly seven hours per day. Nielsen Media research, *2000 Report on Television: The First Fifty Years*, New York: AC Nielsen (2000). Viewing television and playing video games begin early in life and may be increasing. V.J. Rideout, et al., *Zero to Six: Electronic Media in the Lives of Infants, Toddlers and Preschoolers*, Menlo Park, CA: Kaiser Family Foundation (2003); D.A. Christakis, et al., *Television, Video and Computer Game Usage in Children Under 11 Years of Age*, 145 J. PEDIATR. 652-656 (2004). The American Academy of Pediatrics recommends no more than 2 hours of quality television programming per day for children older than 2 years and none at all for those under 2. American Academy of Pediatrics, Committee on Public Education, *Children, Adolescents, and Television*, 107 PEDIATRICS 423-426 (2001).

<sup>182</sup> See M.H. Proctor, et al., *Television Viewing and Change in Body Fat From Preschool to Early Adolescence: The Framingham Children's Study*, 27 INT. J. OBES. RELAT. METAB. DISORD. 827-833 (2003); B.E. Saelens, et al., *Home Environmental Influences on Children's Television Watching From Early to Middle Childhood*, 23 J. DEV. BEHAV. PEDIATR. 127-132 (2002); R. Lowry, et al., *Television Viewing and its Associations With Overweight, Sedentary Lifestyle, and Insufficient Consumption of Fruits and Vegetables Among U.S. High School Students: Differences by Race, Ethnicity, and Gender*, 72 J. SCH. HEALTH 413-421 (2002);

increases 2% for every hour of weekly TV time.<sup>183</sup> Among kids watching one hour of television daily or less, obesity is found in 8%.<sup>184</sup> Among those watching four hours or more, obesity is found in 18%.<sup>185</sup> Sedentary behavior, especially watching television and videos, correlates to a higher body mass index for children and adolescents.<sup>186</sup> The relationship between sedentary behaviors and physical activity has been consistent across studies.<sup>187</sup> Reducing time spent in sedentary behaviors, including television viewing,<sup>188</sup> is an effective weight-loss strategy for youth independent of other changes in physical activity level.<sup>189</sup> Exercise is the main means by which individuals can vary energy expenditure to balance energy intake after accounting for basal energy expenditure and the body's generation of heat.<sup>190</sup> Yet, according to a recent CDC survey, 61.5% of 9- to 13-year-olds participate in no organized physical activities, and 22.6% do not partake

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T.N. Robinson, *Television Viewing and Childhood Obesity*, 48 PEDIATR. CLIN. NORTH AM. 1017-1025 (2001); B.A. Dennison, et al., *Television Viewing and Television in Bedroom Associated With Overweight Risk Among Low Income Preschool Children*, 109 PEDIATRICS 1028-1035 (2002); S.L. Gortmaker, et al., *Television Viewing as a Cause of Increasing Obesity Among Children in the United States, 1986-1990*, 150 ARCH. PEDIATR. ADOLESC. MED. 356-363 (1996). By age twenty-six, according to one report, nearly 20% of an individual's body weight could be accounted for by whether in childhood he watched more than two hours of television daily. M. Devereux, *Damaged for Life by Too Much TV*, NEW ZEALAND HERALD (July 17, 2004).

<sup>183</sup> W.H. Dietz & S.L. Gortmaker, *Do We Fatten Our Children at the TV Set? Obesity and TV Viewing in Children and Adolescents*, 75 (5) PEDIATRICS 807-812 (1985). The association with obesity persisted when controlled for prior obesity, region, season, population density, race, socioeconomic class, and a variety of other variables. "The consistency, temporal sequence, strength, and specificity of the associations suggest that TV viewing may cause obesity in at least some children and adolescents." *Id.* (abstract).

<sup>184</sup> C.J. Crespo, et al., *Television Watching, Energy Intake, and Obesity in U.S. Children: Results From the Third National Health and Nutrition Examination Survey, 1988-1994*, 155 ARCH. PEDIATR. ADOLESC. MED. 360-365 (2001).

<sup>185</sup> See, e.g., M.B. Livingstone, et al., *How Active Are We? Levels of Routine Physical Activity in Children and Adults*, 62 PROC. NUTR. SOC. 681-701 (2003). See, S.L. Gortmaker, et al., *Television Viewing as a Cause of Increasing Obesity Among Children in the U.S., 1986-1990*, *supra* n. 182. See also, See F.B. Hu, et al., *Television Watching and Other Sedentary Behaviors in Relation to Risk of Obesity and Type II Diabetes Mellitus in Women*, 289 JAMA 1785 (2003). Moreover, in discussions of obesity, there is some tendency to lump data pertinent to activity levels in with those pertinent to diet. "The first implicit assumption of the 300,000 deaths claim [ascribed to obesity] is that obesity is the sole cause of these complications, but the actual studies at best attribute the shortening of life to generalized 'diet/activity' patterns of which obesity is only a small part." R.A. Epstein, *supra* note 112, at 1369-1370, citing G. Gaesser, *Big Fat Lies: The Truth About Your Weight and Your Health* 75-80 (2002).

<sup>186</sup> J.C. Eisenmann, R.T. Baree & M.Q. Wang, *Physical Activity, T.V. Viewing and Weight in U.S. Youth: 1999 Youth Risk Behavior Survey*, 10 OBES. RES. 379-385 (2002); J. Utter, et al., *Couch Potatoes or French Fries: Are Sedentary Behaviors Associated With Body Mass Index, Physical Activity, and Dietary Behaviors Among Adolescents?*, 103 J. AM. DIET. ASSOC. 1298-1305 (2003).

<sup>187</sup> D.E. Feldmann, et al. *Is Physical Activity Differentially Associated With Different Types of Sedentary Pursuits?*, 157 ARCH. PEDIATR. ADOLESC. MED. 797-802 (2003).

<sup>188</sup> S.J. Marshall, et al., *Relationships Between Media Use, Body Fatness and Physical Activity in Children and Youth: A Meta-Analysis*, 28 INT. J. OBES. RELAT. METAB. DISORD. 1238-1246 (2004); T.N. Robinson, et al., *Reducing Children's Television Viewing to Prevent Obesity: A Randomized Controlled Trial*, 282 JAMA 1561-1567 (1999).

<sup>189</sup> B.E. Saelens, "Helping Individuals Reduce Sedentary Behavior," in: R.E. Anderson, ed., *Obesity: Etiology, Assessment, Treatment, and Prevention*, Champaign, IL.: Human Kinetics (2003) at 217-238; L.H. Epstein, L.H., *Decreasing Sedentary Behaviors in Treating Pediatric Obesity*, 155 ARCH. PEDIATR. ADOLESC. MED. 220-226 (2000). See also, W.B. Strong, et al., *Evidence Based Physical Activity for School-Age Youth*, 146 J. PEDIATR. 732-737 (2005).

<sup>190</sup> G.A. Brooks, et al., *Chronicle of the Institute of Medicine Physical Activity Recommendation: How a Physical Activity Recommendation Came to be Among Dietary Recommendations*, 79 AM. J. CLIN. NUTR. 921S-930S (2003). In a study of 9751 kindergarten students, an increase in PE instruction time was associated with a significant reduction in BMI among overweight girls. A. Datar, A. & R. Sturm, *Physical Education in Elementary School and Body Mass Index: Evidence From the Early Childhood Longitudinal Study*, 94 AM. J. PUBLIC HEALTH 1501-1506 (2004).

in nonorganized physical activity during nonschool hours.<sup>191</sup> Girls were approximately 20% less active than boys. Worse, in another study, mean activity levels decreased with age by about 7.4% per year in girls and 2.7% per year in boys.<sup>192</sup>

Increasing sedentariness among adults<sup>193</sup> may also contribute to obesity. In the aggregate, sedentary behaviors, such as driving instead of walking, exact a heavy toll: “[T]he secular trend toward increased body fat in each successive generation may be more easily explained by the availability of gasoline than by the availability of fast food.”<sup>194</sup> Adults, too, vegetate in front of their televisions, relying on the remote to channel-surf.<sup>195</sup> Only 19% of Americans exercise three or more times per week.<sup>196</sup> Twenty-five percent of adults eschew physical activity altogether.<sup>197</sup> High-level and medium-high level physical exertion seems to be even less prevalent among minorities than among whites.<sup>198</sup> With eating as with so much else, children tend to pattern their behavior after their parents.<sup>199</sup> It is hardly surprising, then, that couch potatoes are raising younger and temporarily smaller couch potatoes. We do not need as much energy as we did when

<sup>191</sup> CDC and Prevention, *Physical Activity Levels Among Children Aged 9-13 Years: United States, 2002*, 52 MMWR 785-788 (2003).

<sup>192</sup> J.J. Sallis, *Epidemiology of Physical Activity and Fitness in Children and Adolescents*, 33 CRIT. REV. FOOD SCI. NUTR., 403-408 (1993).

<sup>193</sup> “Since 1970, the percentage of Americans driving alone to work and using the car for minor tasks has increased by 15%, while biking and walking has declined by 25%.” U.S. Bureau of the Census, Website database <http://factfinder.census.gov>, (last visited July 6, 2006). For trips of less than one mile, we use our cars 75% of the time. A. Flint, *Suburban Sprawl Seen as Health Hazard*, THE BOSTON GLOBE (Oct. 8, 2001) at B-2. In part, obesity is a function of dependence on cars. L. Frank, M. Andresen, & T. Schmid, *Obesity Relationships With Community Design, Physical Activity, and Time Spent in Cars*, 27 AM. J. PREVENT. MED. 87-95 (2004). Obesity is strongly associated with a sedentary lifestyle in Europe also. M.A. Martinez-Gonzalez, et al., *Physical Inactivity, Sedentary Lifestyle and Obesity in the European Union*, 23 (11) INT’L. J. OBES. RELAT. METAB. DISORD. 1192-1201 (1999), specifically including the UK. A.M. Prentice & S.A. Jebb, *Obesity in Britain: Gluttony or Sloth*, 311 (7002) BMJ 437-9 (1995) (“...modern inactive lifestyles are at least as important as diet in the etiology of obesity and possibly represent the dominant factor.”).

<sup>194</sup> W.I. Bennett, *Beyond Overeating*, 332 N. ENGL. J. MED. 673-674 (1995).

<sup>195</sup> See F.B. Hu, et al., *supra* note 185. In this study of 50,000 women, for every two hours per day that a woman watched television, her risk for obesity increased 23%. See also, C. Geraci, “*Bush Camp Unveils Ads on Newspaper Websites*,” EDITOR & PUBLISHER (May 24, 2004) (women spend 3.3 h/d on the Internet), available at [http://www.vnuemedia.com/eandp/login/login\\_subscribe.jsp?id=xNH33mfDnH8ZL1wpPYB5kVaLXU4gP%2FfwNP%2BigZAgQHP2YevV1qEtJFtwQv8ODWsGxKfwZOuyuzV%2BlyG%2Fwdu24ss2bzIEDnQAt7YfYRqJFzTjlvvbd9JJQ%3D%3D](http://www.vnuemedia.com/eandp/login/login_subscribe.jsp?id=xNH33mfDnH8ZL1wpPYB5kVaLXU4gP%2FfwNP%2BigZAgQHP2YevV1qEtJFtwQv8ODWsGxKfwZOuyuzV%2BlyG%2Fwdu24ss2bzIEDnQAt7YfYRqJFzTjlvvbd9JJQ%3D%3D), (last visited Nov. 6, 2005).

<sup>196</sup> See, P.M. Barnes & C.A. Schoenborn, *Physical Activity Among Adults: U.S. 2000*, 333 ADVANCED DATA FROM VITAL & HEALTH STATISTICS 1, 1, 19-20 (2003), available at <http://www.cdc.gov/nchs/data/ad/ad333.pdf>, (last visited July 12, 2006).

<sup>197</sup> J. Putnam, *U.S. Food Supply Providing More Food and Calories*, 22 FOOD REV. 2, 2 (1999).

<sup>198</sup> P.M. Barnes & C.A. Schoenborn, *supra* note 196.

<sup>199</sup> See, L.V. Harper & K.M. Saunders, *The Effects of Adults' Eating on Young Children's Acceptance of Unfamiliar Foods*, 20 J. EXPERIMENTAL CHILD PSYCHOL. 206 (1975). Children's food acceptance is affected by what they see other people eating, what foods they are offered, and how parents control food intake in the home. L. Cooke, et al., *Demographic, Familial and Trait Predictors of Fruit and Vegetable Consumption by Pre-School Children*, 7 PUBLIC HEALTH NUTR. 295-302 (2004); C.A. Vereecken, et al., *Influence of Mother's Educational Level on Food Parenting Practices and Food Habits of Young Children*, 43 APPETITE 91-103 (2004). Parents more knowledgeable about nutrition tend to rear children with more healthful eating habits. J.N. Variyam, *Overweight Children: Is Parental Nutrition Knowledge a Factor?*, FOOD REVIEW (May-Aug. 2001) at 18, 20-21. Parents can increase their children's enjoyment of vegetables and other nutritious foods by regularly consuming such foods themselves. L.L. Birch, *Development of Food Preferences*, 19 ANNU. REV. NUTR. 41-62 (1999). Repeated exposure to such foods may increase liking among children. See, e.g., J. Wardle, *Modifying Children's Food Preferences: The Effects of Exposure and Reward on Acceptance of an Unfamiliar Vegetable*, 57 EUR. J. CLIN. NUTR. 341-8 (2003). To a small but significant extent, parent-child food preferences tend to correlate. J. Borah-Giddens & G. Falciglia, *A Meta-Analysis of the Relationship in Food Preferences Between Parents and Children*, 25 J. NUTR. EDUC. 102-7 (1993). When aggregated across empirically-derived groupings of foods, variations in food preferences are heritable. F.M. Breen, et al., *Heritability of Food Preferences in Young Children*, 88 (4-5) PHYSIOL. & BEHAV. 443-7 (2006). See abstract at <http://www>.

most of us worked on farms and many of the rest did other types of manual labor.<sup>200</sup> Yet many have not adjusted their food intake to account for such differences.

Not all exercise is conscious. Recently, scientists have begun to understand that energy expenditure is significantly related to “non-conscious, non-exercise activity thermogenesis” (NEAT), meaning energy expended for everything we do that is not sleeping, eating, or sports-like exercise.<sup>201</sup> Seemingly inconsequential physical activity, including fidgeting or typing, for example, increases metabolic rate. These factors in the aggregate culminate in an individual’s daily NEAT.<sup>202</sup> It turns out that manual laborers tend to have high NEAT, whereas knowledge workers typically do not.<sup>203</sup> WHO has recommended strategies augmenting non-exercise activity to increase energy expenditure by 200 calories daily, the equivalent of walking about an hour per day.<sup>204</sup>

Levine, et al.<sup>205</sup> have reported that on average obese individuals are seated two hours per day longer than lean individuals are. This postural allocation did not change when the obese persons lost weight, nor when lean ones gained it. The authors estimated that if obese individuals changed their postures to mimic those of lean ones, they would expend an additional 350 calories per day.<sup>206</sup> Hence, even if we cannot afford to join the local fitness center, nearly all of us can exercise more. Moreover, comparatively modest changes in habits may make appreciable differences in body weight and in the prevalence of obesity.

### E. Metabolic/Hormonal Factors

Endocrine effects may be important contributors to obesity as well. Although much remains to be discovered, our knowledge in this area has increased greatly in the last two decades. An exhaustive treatment of this topic is beyond the scope of this article, but some of the more important hormones will be considered briefly. A study of the endocrine influences on energy consumption and expenditure yields two valuable lessons here: 1) The complexity of the pertinent metabolic control system demonstrates that a simplistic conclusion that obesity is caused by farmers or by the food industry will lead to inaccurate conclusions. 2) The elegance of the mechanisms by which hormones regulate appetite and weight, analogous to the body’s regulation of temperature<sup>207</sup> or of plasma osmolality,<sup>208</sup> for example, mechanisms for which tobacco has no analogue, fits food’s physiologic centrality and serves once again to suggest that neither scientific nor legal analysis justifies equating overeating with tobacco abuse.

[ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list\\_uids=16750228&dopt=Citation](http://ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=16750228&dopt=Citation), (last visited August 8, 2006).

<sup>200</sup> See, D. Lakdawalla & T. Philipson, *supra*. Americans are said to burn about 20% fewer calories in household work than their parents did. J. Robinson & G. Godbey, *Time for Life: The Surprising Ways Americans Use Their Time*, UNIV. PARK: PA STATE UNIV. PRESS (1997) and D. Cutler, E. Glaeser, & J. Shapiro, *Why Have Americans Become More Obese?* National Bureau of Economic Research, Working Paper 9446 (2003). It has been estimated that 60% of America’s recent weight gains have come from labor-saving technologies. D. Lakdawalla & T. Philipson, *supra*.

<sup>201</sup> J.A. Levine, *Non-Exercise Activity Thermogenesis*, 62 PROC. NUTR. SOC. 667-679 (2003).

<sup>202</sup> S. Snitker, et al., *Spontaneous Physical Activity in a Respiratory Chamber is Correlated to Habitual Physical Activity*, 25 INT. J. OBES. RELAT. METABOL. DISORD. 1481-1486 (2001).

<sup>203</sup> J.A. Levine, *Non-Exercise Activity Thermogenesis (NEAT)*, 16 (4) BEST PRACT. RES. CLIN. ENDOCRINOL. METAB. 679-792 (2002).

<sup>204</sup> WHO, “Obesity: Preventing and Managing the Global Epidemic,” Geneva: WHO (1997).

<sup>205</sup> J.A. Levine, et al., *Inter-Individual Variation in Posture Allocation: Possible Role in Human Obesity*, 307 SCI. 584-586 (2005), abstract available at <http://www.sciencemag.org/cgi/content/abstract/307/5709/584>, (last visited August 8, 2006).

<sup>206</sup> *Id.*

<sup>207</sup> C.J. Gordon, *Temperature Regulation in Laboratory Rodents*, NY, CAMBRIDGE UNIV. PRESS (1993).

<sup>208</sup> E.M. Stricker & A.F. Sved, *Thirst*, 16 NUTRITION 821-826 (2000).

Hormonal influences are intricate and difficult to summarize succinctly. It seems clear, however, that hormones can be powerful stimulants to food consumption, and powerful deterrents. Over a period of weeks to months, food intake and energy expenditure are adjusted to compensate for day-to-day fluctuations in energy stores.<sup>209</sup> Generally speaking, satiety factors are peptide hormones secreted by the gut in response to the biochemical makeup of the food ingested. With its myriad chemoreceptors, the gut contributes to feedback inhibition. It analyzes what is being consumed, and then secretes an array of hormones that in turn trigger secretion of the proper digestive hormones and juices, all in the right concentrations and at the proper anatomic sites. The brain monitors the total nutrient load consumed. Eventually, the cumulative impact of these various factors stops the eating process. The animal or person is sated.<sup>210</sup> Admittedly, eating motivation is not controlled by a simple cycle of depletion and repletion; appetite control evolved to solve the problem of an uneven food supply, so a degree of permissiveness is built in.<sup>211</sup> Nevertheless, afferent signals continuously inform brain circuits about acute and chronic changes in energy homeostasis, which then integrate this information and respond with different signals to initiate adaptive changes and regain energy balance.<sup>212</sup>

For food consumption, as for so many other hormonally-influenced process, we have both agonists and corresponding antagonists and substantial built-in redundancy. As with all hormonal systems, feedback inhibition, designed to prevent overproduction of hormones, adds to both the elegance and the complexity of the system.

The hypothalamus regulates appetite through two major groups of neuropeptides. Here, we can mention only briefly some of the hormonal influences fostering negative energy balance and appetite control, and their antagonists, hormonal influences predisposing to positive energy balance.

### 1. *Anorexic Hormones and Negative Energy Balance*

Many hormones suppress appetite, increase energy consumption, or both. Examples include leptin, obestatin, cholecystokinin, insulin, serotonin, peptide YY 3-36, pro-opi-

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<sup>209</sup> M.W. Schwartz, et al., *Is the Energy Homeostasis System Inherently Biased Toward Weight Gain?*, 52 *DIABETES* 232-238 (Feb. 2003). See also, R. Leibel, M. Rosenbaum, & J. Hirsch, *Changes in Energy Expenditure Resulting From Altered Body Weight*, 332 *NEW ENGL. J. MED.* 621-28 (1995); R. Pool, *Fat: Fighting the Obesity Epidemic*, NY: OXFORD UNIV. PRESS (2001) and E. Shell, *The Hungry Gene: The Science of Fat and the Future of Thin*, NY: ATLANTIC MONTHLY PRESS (2002).

<sup>210</sup> S.C. Woods, *The Eating Paradox: How We Tolerate Food*, 98 (4) *PSYCHOL. REV.* 488-505 (1991) at 494. As animals become more sated, they develop decreased preference for sweet-tasting foods. M. Cabanac, *Physiological Role of Pleasure*, 173 (2) *SCI.* 1103-1107 (1971); M. Cabanac & R. Duclaux, *Obesity: Absence of Satiety Aversion to Sucrose*, 168 *SCI.* 496-497 (1970).

<sup>211</sup> P.J. Rogers & H.J. Smit, *Food Craving and Food "Addiction": A Critical Review of the Evidence From a Biopsychosocial Perspective*, 66 (1) *PHARMACOL. BIOCHEM. BEHAV.* 3-14 (2000) at 7. See, nn. 346-348, *infra*, and associated text. The acceptable range for the internal milieu respecting weight may be relatively broad. R.E. Keeseey & S.W. Corbett, "Metabolic Defense of the Body Weight Set-point," in A. J. Stunkard and E. Stellar, eds., *Eating and its Disorders*, NY: RAVEN PRESS (1984) at 87-96.

<sup>212</sup> T.R. Castañeda, et al., *Obesity and the Neuroendocrine Control of Energy Homeostasis: The Role of Spontaneous Locomotor Activity*, 135 *J. NUTR.* 1314-1319 (2005). Physiological stabilizing factors include an inhibitory effect of body fat and recent energy intake on appetite, and a "passive feedback" effect arising from the greater energy costs associated with increased body mass. The physical capacity of the digestive system to accommodate and process food also imposes limits on intake. P.J. Rogers, *Eating Habits and Appetite Control: A Psychobiological Perspective*, 58 *PROC. NUTRIT. SOC.* 59-67 (1999) at 65.

omelanocortin (POMC) and its relatives, cocaine and amphetamine-related transcript (CART),<sup>213</sup> as well as androgens, thyroid hormones, and growth hormones.

### a. *Leptin*

Leptin, from the Greek *leptos* for “thin,” is an appetite-suppressing hormone manufactured in fat cells (adipose tissue). Leptin acts on receptors on the hypothalamic neurons responsible for 1) stimulating secretion of gonadotropin-releasing hormone (GnRH) and 2) suppressing bone formation.<sup>214</sup> Leptin also acts directly on liver and muscle cells, stimulating oxidation of fatty acids in mitochondria and reducing fat storage there (but not in adipose tissue).<sup>215</sup> The level of circulating leptin is directly proportional to the total amount of fat in the body.<sup>216</sup> In studies to date, changes in leptin levels have been associated with reciprocal changes in hunger.<sup>217</sup>

Variations in leptin levels are in large measure genetically controlled, with estimates of genetic heritability ranging from 0.40-0.60.<sup>218</sup> Genetic mutations in the *Lep* gene are associated with an inability to synthesize leptin, and consequently with obesity.<sup>219</sup> A mutation in the leptin receptor has been reported in at least one human family, in whose affected members a normal birth weight was soon greatly increased by rapid weight gain in the first few months of life.<sup>220</sup> Long term, leptin inhibits food intake and increases energy utilization. Where leptin is absent, uncontrolled food intake and obesity result.<sup>221</sup> Very few leptin-deficient persons have been identified to date. Resistance to leptin’s action, however, is not uncommon among the obese.<sup>222</sup>

<sup>213</sup> Sainsbury, et al., *Hypothalamic Regulation of Energy Homeostasis*, 16 (4) BEST PRACT. RES. CLIN. ENDOCRIN. METABOL. 623-637 (Dec. 2002).

<sup>214</sup> P. Havel, *Control of Energy Homeostasis and Insulin Action by Adipocytes Hormones: Leptin, Acylation Stimulating Protein, and Adipopectin*, 13 CURR. OPIN. LIPIDOL. 51-59 (2002); R.S. Ahima, et al., *Leptin Regulation of Neuroendocrine Systems*, 21 FRONTIERS IN NEUROENDOCRINOL. 263-307 (2000).

<sup>215</sup> See, C.A. Matson, et al., *Cholecystokinin and Leptin Act Synergistically to Reduce Body Weight*, 278 AM. J. PHYSIOL. REGUL. INTEGR. COMP. PHYSIOL. R882-R890 (2000).

<sup>216</sup> M. Rosenbaum, et al., *Effects of Gender, Body Composition, and Menopause on Plasma Concentrations of Leptin*, 81 J. CLIN. ENDOCRINOL. METAB. 3424-3427 (1996).

<sup>217</sup> C. Chin-Chance, et al., *Twenty-four-hour Leptin Levels Respond to Cumulative Short-term Energy Imbalance and Predict Subsequent Intake*, 85 J. CLIN. ENDOCRINOL. METAB. 2685-91 (2000). Signaling the transition from a state of nutritional adequacy to a state of starvation may be leptin’s major function. J.S. Flier, *Clinical Review 94: What’s in a Name? In Search of Leptin’s Physiologic Role*, 83 J. CLIN. ENDOCRINOL. METAB. 1407-13 (1998).

<sup>218</sup> G. Barsh, I. Farooqi, & S. O’Rahilly, *Genetics of Body-weight Regulation*, 404 NATURE 644-51 (2000); A.G. Comuzzie, et al., *A Major Quantitative Trait Locus Determining Serum Leptin Levels and Fat Mass is Located on Chromosome 2*, 15 NAT. GENET. 273-275 (1997); C. Rotimo, et al., *Heritability of Plasma Leptin in Population Sample of African American Families*, 14 GENET. EPIDEMIOL. 255-263 (1997).

<sup>219</sup> Paracchini, et al., *Genetics of Leptin and Obesity*, 162 (2) AM. J. EPIDEMIOL. 101-114 (2005). Investigators have identified rare monogenic recessive forms of human obesity caused by mutations in the genes that encode leptin, and other hormones, which result in a phenotype of excessive energy intake relative to energy expenditure. I.S. Farooqi, et al., *Effects of Recombinant Leptin Therapy in a Child With Congenital Leptin Deficiency*, 341 N. ENGL. J. MED. 879-884 (1999) presented a striking example of the effect of leptin replacement in an extremely obese 9-year-old child with congenital leptin deficiency, in whom daily injection of leptin for a year led to a complete reversal of obesity, with sustained fat-mass loss. C.G. Bell, et al., *The Genetics of Human Obesity*, (6) (3), NATURE REVIEWS GENETICS 221-234 (Feb. 10 2005).

<sup>220</sup> K. Clement, et al., *A Mutation in the Human Leptin Receptor Gene Causes Obesity and Pituitary Dysfunction*, 392 NATURE 398-401 (1998).

<sup>221</sup> C.T. Montague, et al., *Congenital Leptin Deficiency is Associated With Severe Early-onset Obesity in Humans*, 387 NATURE 903-908 (1997); I.S. Farooqi, et al., *Beneficial Effects of Leptin on Obesity, T Cell Hyporesponsiveness, and Neuroendocrine/metabolic Dysfunction of Human Congenital Leptin Deficiency*, 110 J. CLIN. INVEST. 1093-1103 (2002); A. Strobel, et al., *A Leptin Missense Mutation Associated With Hypogonadism and Morbid Obesity*, 18 NATURE GENETICS 213-15 (1998).

<sup>222</sup> J. Korner & L.J. Aronne, *Pharmacological Approaches to Weight Reduction: Therapeutic Targets*, 89 (6) J. CLIN. ENDOCRINOL. METABOL. 2616-2621 (2004).

### b. *Obestatin*

Obestatin is a newly discovered peptide that opposes the action of ghrelin (discussed below). In humans, obestatin may delay food transit and so postpone the onset of hunger following a meal. Intriguingly, both ghrelin and obestatin, its antagonist, appear to be derived from the same gene. When the ghrelin gene is deleted, the obestatin gene is also deleted, so there is no net effect on appetite or hunger.<sup>223</sup>

### c. *Cholecystokinin (CCK)*

In response to the intraluminal presence of digestive products, cholecystokinin is released into the bloodstream from the duodenum and the jejunum.<sup>224</sup> CCK causes gall bladder contraction, intestinal motility, and release of pancreatic digestive enzymes. It rapidly inhibits eating.<sup>225</sup> Decreased plasma levels of CCK correlate with hunger, and a decreased sense of satiety.<sup>226</sup> Where the effects of CCK are diminished, obesity may be more likely.<sup>227</sup>

### d. *Insulin*

Insulin is released in anticipation of a meal and during eating. Impairment of hypothalamic insulin receptors causes hyperphagia and insulin resistance.<sup>228</sup> Insulin's concentration is proportional to the volume of adipocytes.<sup>229</sup> Insulin reduces food intake by several mechanisms. Like leptin, it decreases appetite by inhibiting CNS neurons containing neuropeptide Y (NPY) and agouti gene-related peptide (discussed below).<sup>230</sup> When levels of insulin and of leptin are low, energy intake increases, while energy

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<sup>223</sup> J.V. Zhang, et al., *Obestatin, a Peptide Encoded by the Ghrelin Gene, Opposes Ghrelin's Effects on Food Intake*, 310 (5570) *SCI.* 996-999 (11 Nov. 2005), abstract available at [http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list\\_uids=16284174](http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=16284174) (last visited June 28, 2006).

<sup>224</sup> P.M. Hellstrom, et al., *Peripheral and Central Signals in the Control of Eating in Normal, Obese and Binge-eating Human Subjects*, 92 (Suppl. 1) *BR. J. NUTR.* S47, S50 (2004).

<sup>225</sup> See G.P. Smith & J. Gibbs, "The Development and Proof of the CCK Hypothesis of Satiety," in C.T. Daurish, S.J. Cooper, S.D. Iversen, & L.L. Iversen, eds., *Multiple Cholecystokinin Receptors in the CNS*, OXFORD UNIV. PRESS: LONDON (1992) at 166-182.

<sup>226</sup> *Id.*

<sup>227</sup> S. Marchal-Victorion, et al., *Genetic, Pharmaceutical and Functional Analysis of Cholecystokinin-1 and Cholecystokinin-2 Receptor Polymorphism in Type 2 Diabetes and Obese Patients*, 12 *PHARMACOGENETICS* 23-30 (2002).

<sup>228</sup> S. Obici, et al., *Decreasing Hypothalamic Insulin Receptors Causes Hyperphagia and Insulin Resistance in Rats*, 5 *NAT. NEUROSCI.* 566-572 (2002). In the absence of insulin, fat is mobilized despite increased food consumption, and excess calories are lost in urine in the form of glucose and ketones. M.W. Schwartz, et al., *supra* note 209 at 235. Some have argued that insulin resistance, in large measure genetically controlled, may be of critical importance to weight. G. Reavan, T. Strom, & B. Fox, *Syndrome X: The Silent Killer*, NY: FIRESIDE (2000). Aerobic exercise may improve insulin sensitivity and blood lipids. B. Lamarche, et al., *Is Body Fat Loss a Determinant Factor in the Improvement of Carbohydrate and Lipid Metabolism Following Aerobic Exercise Training in Obese Women*, 41 *METAB. CLIN. EXP.* 1249-1256 (1992); P. Bjorntorp, et al., *The Effect of Physical Training on Insulin Production in Obesity*, 19 *METAB. CLIN. EXP.* 631-638 (1970).

<sup>229</sup> J. Stern, et al., *Adipose Cell Size and Immunoreactive Insulin Levels in Obese and Normal Weight Adults*, II *LANCET* 948-951 (1972).

<sup>230</sup> P.M. Hellstrom, et al., *supra* note 224. The agouti gene controls differential production of melanin pigments by a paracrine mechanism. Over 25 different alleles have been identified, some of which are associated with obesity, insulin resistance, and increased susceptibility to certain neoplasms. R.J. Miltenberger, et al., *The Role of the Agouti Gene in the Yellow Obese Syndrome*, 127 (9) *J. NUTR.* 1902S-1907S (1997). For a discussion of agouti and agouti-related peptide, *vide infra*, nn. 254-258 and associated text.

expenditure decreases.<sup>231</sup> Central administration of insulin or insulin mimetics reduces food intake and body weight.<sup>232</sup>

#### e. Serotonin

The neurotransmitter serotonin is released in states of relaxation, and generally reduces the drive to be active, sexually aroused, attentive, and hungry. Individuals with low serotonin levels have more difficulty in giving up pleasures, even pleasures that are health-threatening.<sup>233</sup>

#### f. PYY 3-36

Peptide YY 3-36 (PYY 3-36, also written PYY<sub>3-36</sub>) is a peptide produced in the distal small bowel and colon. It is released postprandially in proportion to calories ingested. It signals food intake to appetite-regulating circuits in the hypothalamus, inducing a sense of satiety and decreasing food intake through a gut-hypothalamic pathway. PYY inhibits appetite and food intake (especially after meals) by modulating appetite circuits. In obese subjects, a diminished postprandial PYY response has been reported, even though when given free access to abundant food, obese subjects consumed more calories.<sup>234</sup>

#### g. POMC and its Relatives

Another complex hormonal system influencing the body's energy balance involves pro-opiomelanocortin (POMC) and other melanocortins. Melanocortins are signaling molecules; POMC, a precursor of ACTH in the pituitary, is a prohormone regulated by leptin and chemically converted to alpha-melanocyte stimulating hormone (alpha-MSH).<sup>235</sup> Alpha-MSH can suppress appetite, decrease food intake, and increase energy.<sup>236</sup> In some patients, mutations in the brain's receptor for alpha-MSH are associated with severe obesity.<sup>237</sup> POMC deficiency causes hyperphagia and early onset obesity.<sup>238</sup>

#### h. Cocaine and Amphetamine-Related Transcript (CART)

CART is an anorectic peptide regulated by leptin. It decreases food intake and body weight,<sup>239</sup> and after central administration increases locomotor activity in rats.<sup>240</sup>

<sup>231</sup> S.C. Benoit, et al., *The Catabolic Action of Insulin in the Brain is Mediated by Melanocortins*, 22 J. NEUROSCI. 9048-9052 (2002); S.C. Woods, et al., *Signals That Regulate Food Intake and Energy Homeostasis*, 280 SCI. 1378-1383 (1998). See also, S.C. Woods, et al., *The Evaluation of Insulin as a Metabolic Signal Influencing Behavior via the Brain*, 20 NEUROSCI. BIOBEHAV. REV. 139-44 (1996); M.W. Schwartz, et al., *Central Nervous System Control of Food Intake*, 404 NATURE 404:661-671 (2000).

<sup>232</sup> E.L. Air, et al., *Small Molecule Insulin Mimetics Reduce Intake and Body Weight and Prevent Development of Obesity*, 8 NAT. MED. 179-183 (2002).

<sup>233</sup> P. Netter, "Health and Pleasure," in: *Pleasure and Quality of Life*, D.M. Warburton & N. Sherwood, eds., John Wiley & Sons, Ltd. (1996) at 84.

<sup>234</sup> R.L. Batterham, et al., *Inhibition of Food Intake in Obese Subjects by Peptide YY3-36*, 349 NEW ENGL. J. MED. 941 (2003). See also note 417, *infra*.

<sup>235</sup> C.C. Cheung, D.K. Clifton, & R.A. Steiner, *Proopiomelanocortin Neurons are Direct Targets for Leptin in the Hypothalamus*, 138 ENDOCRINOL. 4489-92 (1997).

<sup>236</sup> W. Fan, et al., *Role of Melanocortigenic Neurons in Feeding and the Agouti Obesity Syndrome*, 385 NATURE 165-168 (1997).

<sup>237</sup> H. Bays, *Current and Investigational Antiobesity Agents and Obesity Therapeutic Treatment Targets*, 12(8) OBES. RES. 1197-1211 (2004). See also, S.S. Nussey & S.A. Whitehead, *Endocrinology: An integrated approach*, <http://www.ncbi.nlm.nih.gov/books/bv.fcgi?rid=endocrin.section.1458>, (last visited July 6, 2006).

<sup>238</sup> I.S. Farooqi, *Genetic and Hereditary Aspects of Childhood Obesity*, 19 (3) BEST PRACT. & RES. CLIN. ENDOCRINOL. METAB. 359-74 (2005).

<sup>239</sup> K.E. Wortley, et al., *Cocaine- and Amphetamine-Regulated Transcript in the Arcuate Nucleus Stimulates Lipid Metabolism to Control Body Fat Accrual on a High-fat Diet*, 117 REGUL. PEPT. 89-99 (2004).

<sup>240</sup> H.L. Kimmel, et al., *Activity of Various CART Peptides in Changing Locomotor Activity in the Rat*, 36 NEUROPEPTIDES 9-12 (2002).

### i. Other Hormonal "Brakes" on Obesity

Several other hormones increase energy expenditure: androgens, thyroid hormones, and growth hormones. Whatever the organism's energy intake, these hormones help it to burn that energy, and deficiencies of these hormones have the opposite effect. We will not discuss these abnormalities here, as they are comparatively widely known.

## 2. Orexigenic Hormones and Positive Energy Balance

Still other hormones are obesity "accelerators." These include the orexigenic (appetite-stimulating) neuropeptides (hypocretins), such as ghrelin, neuropeptide Y (a feeding stimulant secreted by cells in gut and hypothalamus), agouti-related protein (AGRP), melanin-concentrating hormone, glucocorticoids, and glucagon.

### a. Ghrelin

Ghrelin, a peptide hormone derived from the GI tract, is released into the blood under fasting conditions.<sup>241</sup> Acting upon the arcuate nucleus of the hypothalamus, a region important to control of food intake,<sup>242</sup> ghrelin stimulates hunger and promotes food ingestion.<sup>243</sup> Blood concentrations are lowest shortly after eating, then rise during the fast prior to the next meal.<sup>244</sup> Fasting ghrelin concentration is negatively correlated with body fat (%) and fasting insulin and fasting leptin concentrations, all of which are elevated in obesity.<sup>245</sup> Ghrelin may be the long-suspected "meal initiation factor."<sup>246</sup> If so, it may be CCK's antagonist on this score. Tang-Christensen, et al. have suggested that ghrelin may also suppress spontaneous physical activity.<sup>247</sup> If so, ghrelin may be of even greater importance to energy balance than had been suspected initially. Gastric bypass is associated with markedly depressed ghrelin levels, possibly contributing to its efficacy.<sup>248</sup>

### b. Neuropeptide Y (NPY)

NPY is the most potent central appetite stimulant and the most abundant neurotransmitter in brain, so it promotes weight gain.<sup>249</sup> NPY release increases during fasting,<sup>250</sup> and

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<sup>241</sup> D. Weigle, *Pharmacological Therapy of Obesity*, 88 SCI. 2462 (2002).

<sup>242</sup> M. Kojima & K. Kangawa, *Ghrelin: Structure and Function*, 85 (2) PHYSIOL. REV. 495-522 (2005).

<sup>243</sup> A.J. Ven der Lely, et al., *Biological, Physiological, Pathophysiological, and Pharmacologic Aspects of Ghrelin*, 25 ENDOCR. REV. 426-457 (2004); D.E. Cummings, et al., *A Preprandial Rise in Plasma Ghrelin Levels Suggests a Role in Meal Initiation in Humans*, 50 DIABETES 1714-1719 (2001); M. Tschöp, et al., *Ghrelin Induces Adiposity in Rodents*, 407 NATURE 908-913 (2000).

<sup>244</sup> D.E. Cummings, et al., *Plasma Ghrelin Levels After Diet-Induced Weight Loss or Gastric Bypass Surgery*, 346 N. ENGL. J. MED. 1623-1630 (2002); M. Tschöp, et al., *Post-Prandial Decrease in Circulating Human Ghrelin Levels*, 24 J. ENDOCRINOL. INVEST. 19-21 (2001).

<sup>245</sup> P.M. Hellstrom, et al., *supra* note 224.

<sup>246</sup> D.C. Cummings, et al., *A Preprandial Rise in Plasma Ghrelin Levels Suggests a Role in Meal Initiation in Humans*, 50 DIABETES 1714-1719 (2001).

<sup>247</sup> M. Tang-Christensen, et al., *Ghrelin Induces Long Lasting Changes in Food Intake and Locomotor Behavior*, 497 Proceedings of the 85th Ann. Meeting of the Endocrine Society, Philadelphia, 93-94 (2003) (abs.). See also, T.R. Castañeda, et al., *supra* note 212.

<sup>248</sup> D.E. Cummings, et al., *supra* note 246.

<sup>249</sup> S.P. Kalra, et al., *Structure-Function Analysis of Stimulation of Food Intake by Neuropeptide Y: Effect of Receptor Agonists*, 50 PHYSIOL. BEHAV. 5-9 (1991); M.D. Flynn, et al., *Neuropeptide-Y Related Compounds and Feeding*, 65 PHYSIOL. BEHAV. 901-905 (1999).

<sup>250</sup> S.P. Kalra, et al., *Neuropeptide Y Secretion Increases in the Paraventricular Nucleus in Association With Increased Appetite of Food*, 38 PROC. NAT'L. ACAD. SCI. U.S.A. 10931-10935 (1991).

NPY neurons become less active with voluntary overeating.<sup>251</sup> NPY promotes fat accretion by increasing insulin and cortisol secretion.<sup>252</sup> It also acts in a variety of other ways, such as decreasing synthesis of thyrotropin-releasing hormone<sup>253</sup> (TRH, which stimulates the thyroid gland to produce thyroid hormone), thereby favoring weight gain.

### c. *Agouti-related Peptide (AgRP)*

Agouti protein and agouti-related protein (AGRP) are paracrine-signaling molecules (local mediators) that normally regulate pigmentation and body weight, respectively.<sup>254</sup> They stimulate food intake and antagonize  $\alpha$ -MSH and other melanocortins.<sup>255</sup> When AGRP is over-expressed in transgenic mice, it causes both obesity and hyperphagia. Like ghrelin, and unlike NPY, AGRP may suppress spontaneous physical activity.<sup>256</sup> Humans have a closely related homologue of the mouse agouti gene<sup>257</sup> that is expressed in adipose tissue. The human agouti gene may participate in regulation of lipid metabolism.<sup>258</sup>

### d. *Melanin-concentrating Hormone*

In animals, melanin-concentrating hormone (MCH), a neuropeptide, plays an essential role in energy expenditure, locomotion and thermoregulation. For example, rodents that were administered MCH developed hyperphagia and obesity. In contrast, ablation of the MCH gene in rodents resulted in a lean phenotype caused by an increase in metabolic rate and locomotor activity.<sup>259</sup>

### e. *Glucocorticoids*

Glucocorticoids (steroids) stimulate gluconeogenesis. Circulating levels of glucocorticoids are not elevated in most obese subjects, but owing to elevated enzyme activity (of 11 beta-hydroxy-steroid dehydrogenase [“HSD”] type 1), their local action on adipose tissue and skeletal muscle may be enhanced.<sup>260</sup> Glucocorticoid excess, growth

<sup>251</sup> J.P. Wilding, et al., *Increased Neuropeptide Y Content in Individual Hypothalamic Nuclei, But not Neuropeptide Y mRNA, in Diet-induced Obesity in Rats*, 132 J. ENDOCRINOL. 299-304 (1992); P.S. Widdowson, et al., *Reciprocal Regional Changes in Brain NPY Receptor Density During Dietary Restriction and Dietary-induced Obesity in the Rat*, 774 BRAIN RES. 1-10 (1997).

<sup>252</sup> The NPY neurons on the arcuate nucleus are stimulated by starvation, probably mediated by falls in circulating leptin and insulin, both of which inhibit NPY neurons. G. Williams, et al., *The Hypothalamus and the Regulation of Energy Homeostasis: Lifting the Lid on the Black Box* 59 J. NUTR. SOC. 385-396 (2000) at 390.

<sup>253</sup> See, M.F. McCarty, *Central Insulin May Up-regulate Thyroid Activity by Suppressing Neuropeptide Y Release in the Paraventricular Nucleus*, 45 (2) MED. HYPOTHESES 193-199 (Aug. 1995); G.A. van Haasteren, et al., *Starvation-induced Changes in the Hypothalamic Content of the Prothyrotropin-releasing Hormone (pro TRH) mRNA and the Hypothalamic Release of pro TRH-derived Peptides: Role of the Adrenal Gland*, 145 (1) J. ENDOCRINOL. 143-153 (1995).

<sup>254</sup> See n. 230 *supra*, and associated text.

<sup>255</sup> M.M. Ollmann, et al., *Interactions of Agouti Protein With the Melanocortin1 Receptor in vitro and in vivo*, 12 (3) GENES AND DEVELOPMENT 316-330 (Feb. 1998); D. Lu, et al., *Agouti Protein is an Antagonist of the Melanocyte Stimulating Hormone Receptor*, 371 NATURE 799-802 (1994). Alterations in AGRP may regulate the melanocortin system. G. Williams, et al., *supra* note 252.

<sup>256</sup> T.R. Castañeda, et al., *supra* note at 212.

<sup>257</sup> H.Y. Kwon, et al., *Molecular Structure and Chromosomal Mapping of the Human Homolog of the Agouti Gene*, 91 PROC. NAT'L. ACAD. SCI. USA 9760-9764 (1994); B.D. Wilson, et al., *Structure and Function of the ASP, the Human Homolog of the Mouse Agouti Gene*, 4 HUMAN MOL. GENET. 223-230 (1995).

<sup>258</sup> R.J. Mittenberger, et al., *The Role of the Agouti Gene in the Yellow Obese Syndrome*, 127 (9) J. NUTR. 1902S-1907S (1997).

<sup>259</sup> G. Segal-Lieberman, et al., *Melanin-Concentrating Hormone is a Critical Mediator of the Leptin-Deficient Phenotype*, 100 PROC. NATL. ACAD. SCI. U.S.A. 10085-10090 (2003).

<sup>260</sup> H. Masuzaki & J.S. Flier, *Tissue-Specific Glucocorticoid Reactivating Enzyme, 11 Beta-Hydroxy Steroid Dehydrogenase Type 1 (11 beta HSD1): A Promising Drug Target for the Treatment of Metabolic Syndrome*, 3 CURR. DRUG TARGETS IMMUNE ENDOCR. METAB. DISORD. 255-262 (2003).

hormone deficiency, and high androgen concentrations in women and low testosterone concentrations in men are associated with increased fat, reduced skeletal muscle mass, and insulin resistance.<sup>261</sup>

#### f. *Glucagon*

Briefly stated, glucagon is an insulin antagonist, and causes hyperglycemia in part by stimulating hepatic glycogenolysis.<sup>262</sup>

#### g. *Other Hormonal Influences*

Galanin stimulates appetite and regulates gut motility and the activity of the endocrine pancreas.<sup>263</sup> Other gut hormones, including gastrin-releasing peptide, neuromedin B (“NMB”), enterostatin, and amylin; and brain hormones, such as orexins-A and -B (hypocretins)<sup>264</sup> may also play a role in energy balance.<sup>265</sup> In the future, hormones not yet discovered may also prove to be important. As discussed more fully below, recent work suggests that increasing sleep deficits, perhaps related to our hectic lifestyles, may induce physiologic changes in hormonal signals promoting hunger.<sup>266</sup>

The array of hormones known to influence feeding behavior, energy metabolism, and body weight is large and growing. Humans have intricate mechanisms to control appetite, mass, and energy, and the interplay among the diverse forces set off against one another in normal physiology probably helps explain why an individual’s weight is ordinarily rather stable. Nature has provided us with a symphony orchestra. By comparison, the food industry is tooting a tin whistle.

### F. *Genetics*

For most of human existence, as noted above, people struggled to find enough food. Scientists theorize that those who could utilize available calories most efficiently enjoyed selective advantages.<sup>267</sup> For maintaining reproductive capacity, for example, energy stores are critical.<sup>268</sup>

<sup>261</sup> P. Bjorntorp, *The Regulation of Adipose Tissue Distribution in Humans*, 20 INT’L. J. OBES. RELAT. METAB. DISORD. 291-302 (1996).

<sup>262</sup> See, e.g., K. Nagai, *Possible Role of L-Carnosine in the Regulation of Blood Glucose Through Controlling Autonomic Nerves*, 228 EXPT’L. BIOL. MED. 1138-1145 (2003).

<sup>263</sup> S. Katsoulis, et al., *Human Galanin Modulates Human Colonic Motility in vitro: Characterization of Structural Requirements*, 31 (5) SCAND. J. GASTROENTEROL. 446-451 (May 1996); B. Ahren, et al, *Presence of Galanin in Human Pancreatic Nerves and Inhibition of Insulin Secretion from Human Islets*, 264 (2) CELL TISSUE RES. 263-267 (May 1991).

<sup>264</sup> In experimental animals, orexin injected into brain stimulates feeding. T. Sakurai, et al., *Orexins and Orexin Receptors: A Family of Hypothalamic Neuropeptides and G Protein-Coupled Receptors That Regulate Feeding Behavior*, 92 CELL 573-585 (1998).

<sup>265</sup> M.K. Badman & J.S. Flier, *The Gut and Energy Balance: Visceral Allies in the Obesity Wars*, 307 (5717) SCI. 1909-14 (25 Mar. 2005); K.G. Murphy & S.R. Bloom, *Gut Hormones in the Control of Appetite*, 89 (5) EXP. PHYSIOL. 507-516 (2004); S.M. Gale, et al., *Energy Homeostasis, Obesity and Eating Disorders: Recent Advances in Endocrinology*, 134 J. NUTR. 295-298 (Feb. 2004); G. Williams, et al., *supra* note 252, at 390. Many of the pertinent studies however, are of animal models. The relevance of these data to humans is not established.

<sup>266</sup> K. Spiegel, et al., Brief Communication, *Sleep Curtailment in Healthy Young Men is Associated With Decreased Leptin Levels, Elevated Ghrelin Levels, and Increased Hunger and Appetite*, 141 ANN. INT. MED. 846-850 (2004) (abstract available at <http://www.annals.org/cgi/content/abstract/141/11/846>, (last visited July 7, 2006)).

<sup>267</sup> The classic paper in this field is J.V. Neel, *Diabetes Mellitus: A ‘Thrifty’ Genotype Rendered Detrimental by ‘Progress’?*, 14 AM. J. HUM. GENET. 353-362 (1962). See also, J. Friedman, *Modern Science Versus the Stigma of Obesity*, 10 (6) NATURE MEDICINE 563, 567 (June 2004); J.C. Peters, et al., *From Instinct to Intellect: The Challenge of Maintaining Healthy Weight in the Modern World*, 3 OBESITY RES. 69 (2002); R.S. Ahima, et al., *Role of Leptin in the Neuroendocrine Response to Fasting*, 382 NATURE 250-252 (1996); D.L. Coleman, *Obesity Genes: Beneficial Effects in Heterozygous Mice*, 203 SCI. 663-665 (1979); P. Bjorntorp, *Thrifty Genes and Human Obesity: Are We Chasing Ghosts?*, 358 LANCET 1006-1008 (2001).

<sup>268</sup> R. Frisch & J. McArthur, *Menstrual Cycles: Fatness as a Determinant of Minimum Weight for Height Necessary for Their Maintenance or Onset*, 185 SCI. 949-951 (1974); S.M. Garn, et al., *Maturational Timing as a Factor in Female Fatness and Obesity*, 43 (6) AM. J. CLIN. NUTR. 879-883 (1986).

There is abundant evidence for the importance of genetics in control of weight.<sup>269</sup> Most of the variance in the incidence of obesity,<sup>270</sup> and up to 80% of variance in body mass index (BMI)<sup>271</sup> are attributable to genetic factors.<sup>272</sup> Twin studies have shown that the similarities between the weights of identical twins raised apart are greater than those between fraternal twins raised together.<sup>273</sup> Different sets of overfed twins differed

<sup>269</sup> See, e.g., J. Friedman, *A War on Obesity, Not the Obese*, 299 SCI. 856-58 (2003). Given the sad history of misuse of genetic data, this topic must be analyzed cautiously. See, e.g., Race, Ethnicity and Genetics Working Group, National Human Genome Research Institute, *The Use of Racial, Ethnic, and Ancestral Categories in Human Genetics Research*, 77 AM. J. HUM. GENET. 519-32 (2005). At the same time, the understanding of obesity is not furthered by ignoring data demonstrating association between, for example, genetic makeup and body weight. See, e.g., J.R. Fernandez, et al., *Association of African Genetic Admixture With Resting Metabolic Rate and Obesity Among Women*, 11 (7) OBES. RES. 904-911 (2003). At least one paper argues that "genetic factors are of overwhelming importance." J.C. Cogan & P. Ernsbergen, *Dieting, Weight, and Health: Reconceptualizing Research and Policy*, 55 (2) SOC. ISSUES. 187-205 (1999).

<sup>270</sup> D. Allison, et al., *The Heritability of Body Mass Index Among an International Sample of Monozygotic Twins Reared Apart*, 20 INT. OBES. RELAT. METAB. DISORD. 501-506 (1996).

<sup>271</sup> BMI = w/h<sup>2</sup>, where w = weight in kg. and h = height in meters. In the English (Imperial) system, BMI = (703.07) w/h<sup>2</sup>, where w = weight in pounds and h = height in inches. While criteria vary country to country, and experts disagree, in the U.S. a BMI over 25 is often considered overweight, and over 30, obese. Mechanical application of these figures can lead to inaccuracies, however, and a simple adjustment of criteria can increase or decrease the numbers of those deemed obese. BMI makes no distinction between the sexes, and does not account for differences in age. There is evidence that current guidelines for BMI-risk categories overestimate the risks of overweight in those age 75 or older, in whom waist-hip ratio provides a more reliable estimate. G.M. Price et al., *Weight, Shape and Mortality Risks in Older Persons: Elevated Waist-Hip Ratio, Not High Body Index, Is Also Associated with a Greater Risk of Death*, 84 Am. J. Clin. Nutr. 449-460 (2006). Although BMI is a commonly used metric, there are several others, each with advantages and disadvantages. See, e.g., M.A. Moyad, *Current Methods Used for Defining, Measuring, and Treating Obesity*, 19 SEMIN. UROL. ONCOL. 247-256 (2001); see also, R. Seid, *Never Too Thin*, NY: PRENTICE HALL (1989). There can be considerable differences in percentage of fat and main body mass between individuals with similar BMI, especially when this index is compared across different ethnic groups. P. Deurenberg, M. Yap, & W.A. Van Staveren, *Body Mass Index and Percent Body Fat: A Meta-analysis Among Different Ethnic Groups*, 22 INT'L J. OBES. RELAT. METAB. DIS. 1164-1171 (1998). Recent research suggests that BMI may be a less reliable guide to the risk of heart attack than is the waist-to-hip ratio. S. Yusuf, et al., on behalf of the INTERHEART study investigators, *Obesity and the Risk of Myocardial Infarction in 27,000 Participants From 52 Countries: A Case Control Study*, 366 LANCET 1640 (5 Nov. 2005).

<sup>272</sup> M. Rosenbaum & R. Leibel, *Pathophysiology of Childhood Obesity*, 35 PEDIATR. 73-137 (1988). See also, C. Davis, S. Strachan, & M. Berkson, *Sensitivity to Reward: Implications for Overeating and Overweight*, 42 APPETITE 131 (2004). More than 165 different gene locations that have been linked to differences in body weight. E.E. Snyder, et al., *The Human Obesity Gene Map: The 2003 Update*, 12 OBES. RES. 369-439 (2004); see also, G. Barsh, I. Farooqi, & S. O'Rahilly, *Genetics of Body-Weight Regulation*, 404 NATURE 644-651 (2000). An association between African genetic admixture and BMI has been reported. J.R. Fernandez, et al., *supra* note 269.

<sup>273</sup> See, C. Bouchard, et al., *The Response to Long-Term Overfeeding in Identical Twins*, 322 N. ENGL. J. MED. 1477-1492 (1990) (Within twin pairs intentionally overfed, weight-gain correlation was >70%, even though some twin pairs gained as little as 4.3 kg and others as much as 13.3 kg); A.J. Stunkard, et al., *An Adoption Study of Human Obesity*, 314 N. ENGL. J. MED. 193-198 (1986); T.I. Sorensen, *The Genetics of Obesity* [Review], 44 METABOLISM 4-6 (1995); E. Ravussin & C. Bogardus, *A Brief Overview of Human Energy Metabolism and its Relationship to Essential Obesity*, 55 (Suppl.) AM. J. CLIN. NUT. 242S-245S (1992). In Stunkard's study of 1,974 monozygotic and 2,097 dizygotic twin pairs, the heritability value for weight was estimated to be 0.78, which increased to 0.81 on completion of a 25-year follow-up. A.J. Stunkard, et al., *A Twin Study of Human Obesity*, 256 J. AM. MED. ASSOC. 51-54 (1986). In another paper appearing four years later, Stunkard, et al., examined identical and fraternal twins that were reared together and apart. They reported intra-pair correlation coefficients for obesity phenotypes of 0.70 for men and 0.66 for women, a measure of heritability similar to that of previous studies. Shared environment seemed to have no measurable effect and non-shared personal environment contributed about 30% of the variance. A.J. Stunkard, et al., *The Body-Mass Index of Twins Who Have Been Reared Apart*, 322 N. ENGL. J. MED. 1483-1487 (1990). A large study of 4884 twins and 2509 Finnish singletons, aged 16-17 years, estimated that 80% of the inter-individual variation in BMI was attributable to genetic effects. K.H. Pietilainen, et al., *Distribution and Heritability of BMI in Finnish Adolescents Aged 16 and 17y: A Study of 4884 Twins and 2509 Singletons*, 23 INT. OBES. RELAT. METABOL. DISORD. 107-115 (1999). A UK-based study generated similar results. G. Koepfen-Schomerus, et al., *A Genetic Analysis of Weight and Overweight in 4-year-old Twin Pairs*, 25 INT. J. OBES. RELAT. METAB. DISORD. 838-844 (2001). Restoration of normal weight after a period of overfeeding was confirmed in Czech twins that were subjected to a low calorie diet, with an intra-pair correlation of 0.88 in fat loss during treatment and a correlation of 0.77 for metabolic efficiency. C.G. Bell, et al., *supra* note

widely with respect to how they stored excess energy as fat, but within each set of twins the tendency towards increased adiposity was remarkably similar.<sup>274</sup> The adiposity of adopted children tracks the adiposity of their biologic, not their adoptive, parents.<sup>275</sup>

One of the great discoveries of modern medicine has been the recognition that hormones and drugs bring about their effects by acting on cell receptors (structures that selectively bind specific substances, such as drugs or hormones that exert specific physiologic effects). Genetics is important to the structure and function of receptors needed for energy homeostasis just as it is to weight itself. For example, some research has found differences between the prevalence of different dopamine receptor gene types in obese and non-obese individuals.<sup>276</sup>

At least 20 rare syndromes caused by discrete genetic defects or chromosomal abnormalities, both autosomal and X-linked, are characterized by obesity.<sup>277</sup> At least five monogenic defects causing human extreme obesity have been identified, namely, mutations in the genes encoding leptin, leptin receptor, prohormone convertase (PC)-1, pro-opiomelanocortin (POMC), and the melanocortin-4 receptor (MC4R).<sup>278</sup> In all of these syndromes, hyperphagia results from dysfunction of hypothalamic pathways controlling satiety.<sup>279</sup>

Examples of single-gene disorders resulting in human obesity include the Prader-Willi, Bardet-Biedl, Ahlstrom, and Cohen syndromes.<sup>280</sup> There are a variety of

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210. Twin studies reveal additive genetic influence on body size, height and weight, body mass index, daily food ingestion, and the macronutrient, alcohol, and water content of meals. In DeCastro's research, heredity accounted for 65% of the variance in energy intake, 44% of the variance in meal frequency, and 65% of the variance in meal size. On the other hand, the shared familial environments in which the twins were raised had no significant impact on the levels or pattern of intake in adulthood. J.M. De Castro, *Genetic Influences on Daily Intake and Meal Patterns of Humans*, 53 (4) *PHYSIOL. BEHAV.* 777-782 (1993). Heredity also accounted for 96% of the variance in height and 85% of the variance in weight. J.M. De Castro, *Independence of Genetic Influences on Body Size, Daily Intake, and Meal Patterns of Humans*, 54 (4) *PHYSIOL. BEHAV.* 633-639 (1993). See also, E. Shell, *supra* note 209.

<sup>274</sup> C. Bouchard & A. Tremblay, *Genetic Effects in Human Energy Expenditure Components*, 14 *INT'L J. OBES.* 49-55 (1990).

<sup>275</sup> A.J. Stunkard, et al., *supra* note 273.

<sup>276</sup> E.P. Noble, et al., *D2 Dopamine Receptor Gene and Obesity*, 15 (3) *INT'L J. EATING DISORDERS* 205-217 (1994) (The D2 receptor is one of at least five physiologically distinct dopamine receptors found on the synaptic membranes of neurons in the brain.). See also, D.P. Sibley & F.J. Monsma, Jr., *Molecular Biology of Dopamine Receptors*, 13 (2) *TRENDS PHARMACOL. SCI.* 61-69 (1992). See discussion, *infra*, at n. 398 and associated text.

<sup>277</sup> C.G. Bell, et al., *supra* note 219. See also, I.S. Farooqi & S. O'Rahilly, *Monogenic Human Obesity Syndromes*, 59 *RECENT PROG. HORM. RES.* 409-24 (2004); S. O'Rahilly, et al., *Minireview: Human Obesity—Lessons From Monogenic Disorders*, 144 (9) *ENDOCRINOL.* 3757-3764 (2003).

<sup>278</sup> Melanocortin-4 receptors are expressed in various areas of the hypothalamus, including the arcuate and the ventromedial nuclei. Activation of MC4R inhibits feeding and causes weight loss. MC4R antagonists such as agouti protein and the agouti-related peptide stimulate feeding and obesity. G. Williams, et al., *supra* note 252. Mutations in MC4R receptors cause the most common monogenic obesities, and are highly associated with binge eating. I.S. Farooqi, et al., *Clinical Spectrum of Obesity and Mutations in the MC4R Gene*, 348 *NEW ENGL. J. MED.* 1085-1095 (2003); R. Branson, et al., *Binge Eating as a Major Phenotype of MC4R Gene Mutations*, 348 *NEW ENGL. J. MED.* 1096-1103 (2003).

<sup>279</sup> B. Challis, et al., *The CART Gene and Human Obesity: Mutational Analysis and Population Genetics*, 49 *DIABETES* 872 (May 2000). See also, N. Katsanis, et al., *Exploring the Molecular Basis of Bardet-Biedl Syndrome*, 10 *HUM. MOL. GENET.* 2293-2239 (2001).

<sup>280</sup> R. Leibel, et al., *Genetic Variations and Nutrition in Obesity*, in *GENETIC VARIATION & NUTRITION*, A. Simopoulos, B. Childs, eds., Basel, Switzerland: Karger (1990) at 90-101. Prader-Willi is characterized by diminished fetal activity, hypotonia, mental retardation, short stature, hypogonadotropic hypogonadism, and obesity, all attributed to lack of a paternal segment of chromosome 15. M. Butler, *Prader-Willi Syndrome: Current Understanding of Cause and Diagnosis*, 35 *AM. J. MED. GENET.* 319-332 (1990). Bardet-Biedl syndrome features central obesity, mental retardation, dysmorphic limbs, retinal and renal problems, and hypogonadism, ascribed to abnormalities at at least eight loci. N. Katsanis, *The Oligogenic Properties of Bardet-Biedl Syndrome*, 13 (Spec. No. 1) *HUM. MOLEC. GENET.* R65-R71 (2004). Alström syndrome is characterized by childhood obesity, hyperinsulinemia, hyperglycemia, and neurosensory deficits attributed to mutations in a single gene. G.B. Collin, et al., *Mutations in the ALMS1 Cause Obesity, type 2 Diabetes*

others.<sup>281</sup> These cases constitute only a small percentage of the total number of overweight subjects, of course, but they illustrate powerfully the importance of genetic influences on body weight.<sup>282</sup> Heritability of many factors influencing body weight, including fat distribution, basal metabolic rate, changes in energy expenditure occurring in response to overeating, enzyme activity, rates of lipolysis, and even physical activity and food preferences, may be as high as 30-40%.<sup>283</sup> Heritability of BMI is estimated to range from 30-70%.<sup>284</sup> Evidence also exists for a genetic component or components related to fat cell number, a factor related to obesity.<sup>285</sup> Genome-wide scans have shown that the genetic contribution to obesity is distributed over multiple chromosomes, including 2, 5, 10, 11 and 20.<sup>286</sup> An obesity gene map, in fact, has found putative loci on all chromosomes except Y.<sup>287</sup> Genetics may also explain some of the ethnic variations in weight; for example, among certain groups of Hispanics, genetic variants on chromosomes 2 and 7 are thought to play a role in obesity.<sup>288</sup> Recently, research has suggested that a gene may play a part in up to one-fifth of the cases of Type 2 diabetes in the United States.<sup>289</sup>

Proponents of obesity litigation argue that the gene pool has not changed appreciably in one generation. This assertion does not refute the importance of genetics, nor does it damn the food industry.<sup>290</sup> If genetic makeup is unlikely to have changed in a 10- or

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*and Neurosensory Degeneration in Alström Syndrome*, 31 NAT. GENET. 74-78 (2002). Cohen syndrome is associated with mental retardation, microcephaly, characteristic facial features and eye problems arising from mutations in a gene on chromosome 8. K.E. Chandler, et al., *Diagnostic Criteria, Clinical Characteristics, and Natural History of Cohen Syndrome*, 40 J. MED. GENET. 233-41 (2003). Deletion or disruption of the single minded homologue/gene (SIM1) (associated with hyperphagic syndromic obesity) region results in either a "Prader-Willi-like" phenotype or a form of early-onset obesity that is associated with excessive food intake. See, J.L. Holder, Jr., et al., *Profound Obesity Associated With a Balanced Translocation That Disrupts the SIM1 Gene*, 9 HUM. MOL. GENET. 101-108 (2000); L. Faivre, et al., *Deletion of the SIM1 gene (6q16. 2) in a Patient With a Prader-Willi-like Phenotype*, 39 J. MED. GENET. 594-596 (2002).

<sup>281</sup> See, e.g., S.I. Farooqi, *supra* note 238 (Albright's hereditary osteodystrophy, Borjeson-Forssman-Lehman syndrome, etc.).

<sup>282</sup> The importance of genetics may have been underestimated in the past. "We used a dense whole-genome scan of DNA samples from the Framingham Heart Study participants to identify a common genetic variant near the INSIG2 gene (insulin-induced gene) associated with obesity. We have replicated the finding in four separate samples composed of individuals of Western European ancestry, African Americans, and children. The obesity-predisposing genotype is present in 10% of individuals. Our study suggests that common genetic polymorphisms are important determinants of obesity." A. Herbert, et al., *A Common Genetic Variant is Associated With Adult and Childhood Obesity*, 312 (5771) SCI. 279-283 (April 14, 2006); abstract available at <http://www.sciencemag.org/cgi/content/abstract/312/5771/279> (last visited June 23, 2006).

<sup>283</sup> C. Bouchard, ed., *Genetics of Obesity*, BOCA RATON: CRC PRESS (1994).

<sup>284</sup> C.G. Bell, et al., *supra* note 219; J. Hebebrand, et al., *Perspectives: Molecular Genetic Research in Human Obesity*, 4 OBES. REV. 139 (Aug. 2003); K. Schousboe, et al., *Sex Differences in Heritability of BMI: A Comparative Study of Results From Twin Studies in Eight Countries*, 6 TWIN RES. 409 (2003).

<sup>285</sup> R.J. Stein, et al., *Etiology and Treatment of Obesity in Adults and Children: Implications for the Addiction Model*, 15 DRUGS & SOCIETY 103-121 (1999) at 107.

<sup>286</sup> Clement, et al., *Genetics of Obesity*, 2 (3) AMER. J. PHARMACOGENETICS 177-187 (2002). See also, the Obesity Gene Map Data Base of the Human Genomics Laboratory, available at <http://obesitygene.pbrcc.edu/>, (last visited July 7, 2006).

<sup>287</sup> E.E. Snyder, et al., *The Human Obesity Gene Map: the 2003 Update*, 12 OBESITY RES. 369-439 (2004), available at <http://www.obesityresearch.org/cgi/content/full/12/3/369>, (last visited July 7, 2006).

<sup>288</sup> A. Commuzzie, et al., *A Major Quantitative Trait Locus Determining Serum Leptin Levels and Fat Mass is Located on Human Chromosome 2*, 15 NATURE GENET. 1-3 (1997) (Mexican Americans); R. Dug-girala, et al., *Quantitative Variation in Obesity-Related Traits and Insulin Precursors Linked to OB Gene Region on Human Chromosome 7*, 59 AM. J. HUM. GENET. 694-703 (1996).

<sup>289</sup> S.F.A. Grant, et al., *Variant of Transcription Factor 7-like 2 (TCF7L2) Gene Confers Risk of Type 2 Diabetes*, 38 (3) NAT. GEN. 320-3 (March 2006).

<sup>290</sup> During the same period that obesity is said to be increasing in prevalence, for example, dietary fat intake has decreased both absolutely and as a percentage of total dietary energy. A.H. Lichtenstein, et al., *supra* note 126.

20-year period, we must also recognize that in most individuals body composition generally remains stable over long periods as well.<sup>291</sup> This stability tends to persist despite wide variations in energy intake and expenditure.<sup>292</sup>

### G. Cultural, Socioeconomic, Psychological and Behavioral Influences

One's culture tends to influence his attitudes toward food.<sup>293</sup> Many psychological, social, and environmental factors affect eating, such as significant weekly and seasonal variation of food intake.<sup>294</sup> Eating is also facilitated socially. Individual intake is reported to be greater in meals eaten with companions than in meals eaten alone, for example. Intake increases as the number of others present increases.<sup>295</sup> Meals eaten in the company of others are on average 44% larger than meals eaten alone, and participants ingest larger amounts of carbohydrates, fat, protein, and alcohol.<sup>296</sup>

Quantities consumed also vary with the identities and conviviality of the diner's companions. Meals eaten with spouses, family, or friends are significantly larger than those eaten with co-workers, for example.<sup>297</sup> We generally eat more on weekends than during the rest of the week.<sup>298</sup> Among other factors that can affect the amount ingested are weather conditions, noise, atmosphere, and the distance of the food item from the eater.<sup>299</sup> A small but statistically significant increase in meal size and a decrease in alcohol intake has even been reported to occur at the time of a full moon.<sup>300</sup> An increase in total caloric intake, especially carbohydrates, in the autumn over consumption in the other seasons has also been described.<sup>301</sup> This array of influences suggests that the impact of any one factor upon an individual's food consumption or weight is likely to be modest.

<sup>291</sup> G. Glazer, *Long-term Pharmacotherapy of Obesity 2000: A Review of Efficacy and Safety*, 161 ARCH. INT. MED. 1814-1815 (Aug. 13/27, 2001).

<sup>292</sup> A. Belanger, et al., "Means at Each Examination and Inter-Examination Consistency of Specified Characteristics: Framingham Health Study, 30 Year Follow-up," in: W.Kannel, P. Wolf, & R. Garrison, eds., *The Framingham Study: An epidemiologic investigation of cardiovascular disease*, FRAMINGHAM PUBLICATION No. 88-2970, Wash., D.C.: GPO (1988), § 36.

<sup>293</sup> P. Rozin, "Sociocultural Influences on Human Food Selection," in E.D. Capaldi, ed., *Why we eat what we eat*, Wash., D.C.: American Psychological Association (1996) at 233-263. See also, L.L. Birch, *Effects of Peer Models' Food Choice and Eating Behaviors on Preschoolers' Food Preferences*, 51 CHILD DEVELOPMENT 489-496 (1980). Some have reported that black Americans see obesity in positive terms (attractiveness, sexual desirability, body image, strength or goodness, self-esteem, social acceptability) whereas Caucasian Americans generally see obesity in negative terms (unattractive sexually, undesirable, negative body image, decreased self-esteem, social unacceptability). M. Davidson & K.A. Knafel, *Dimensional Analysis of the Concept of Obesity*, 54 (3) J. ADV. NURS. 342-350 (2006).

<sup>294</sup> J.M. de Castro, *How Can Eating Behavior be Regulated in the Complex Environments of Free-Living Humans?*, 20 NEUROSCI. BIOBEHAV. RET. 119-131 (1996); abstract available at [http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list\\_uids=8622818&dopt=Abstract](http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=8622818&dopt=Abstract), (last visited July 13, 2006).

<sup>295</sup> M. Redd & J.M. de Castro, *Social Facilitation of Eating: Effects of Social Instruction on Food Intake*, 52 (4) PHYSIOL. BEHAV. 749-754 (1992); abstract available at [http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list\\_uids=1409948&dopt=Abstract](http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=1409948&dopt=Abstract), last visited July 7, 2006.

<sup>296</sup> J.M. De Castro & E.S. De Castro, *Spontaneous Meal Patterns in Humans: Influence of the Presence of Other People*, 50 AM. J. CLIN. NUTR. 237-247 (1989).

<sup>297</sup> J.M. De Castro, *Family and Friends Produce Greater Social Facilitation of Food Intake Than Other Companions*, 56 PHYSIOL. BEHAV. 445-455 (1994).

<sup>298</sup> J.M. De Castro, *Weekly Rhythms of Spontaneous Nutrient Intake and Meal Patterns of Humans*, 50 (4) PHYSIOL. BEHAV. 729-738 (1991).

<sup>299</sup> D. Engell & E. Hirsch, *Environmental and Sensory Modulation of Fluid Intake in Humans*, in D.J. Ramsay & D.A. Booth, eds., *Thirst: Physiological and psychological aspects*, Berlin: Springer (1991) at 382-390.

<sup>300</sup> J.M. De Castro & S. Percy, *Lunar Rhythms of the Meal and Alcohol Intake of Humans*, 57 PHYSIOL. BEHAV. 439-44 (1995).

<sup>301</sup> J.M. De Castro, *Seasonal Rhythms of Human Nutrient Intake and Meal Patterns*, 50 (1) PHYSIOL. BEHAV. 243-248 (1991).

Employment also has an impact. The shift towards more households with working women, whatever benefits it may have brought, may account for some fraction for the reported rise in obesity in the U.S. between 1971-1975 and 1988-1994.<sup>302</sup> Many families today have two incomes, and hence more discretionary funds than single-earner households. They also typically have fewer children than in the past, so there are more dollars available to feed fewer mouths. But part of the trade-off for two incomes is often the loss of time needed to prepare meals at home.<sup>303</sup> Cooks at restaurants, of course, lack a parent's ability to estimate the nutritional needs of the individual members of the family. Nevertheless, a parent more dependent upon others to prepare food for his children than his own parents were is still in a supervisory role. A parent bringing children to the local fast food eatery can and should control his children's choices, especially where, as is increasingly common, the establishment offers salads, fruit, and other non-traditional menu items.<sup>304</sup> All food choices, whether at home or in restaurants, are made by the consumer. We also determine how much to consume. There is evidence that our society has increased its notion of portion sizes, but that change is not confined to restaurants.<sup>305</sup> It is much in evidence at home as well.<sup>306</sup>

## H. Drugs and Alcohol

Most medications have no significant impact on weight, but some may. Steroids, and some antidepressants,<sup>307</sup> for example, may cause weight gain.<sup>308</sup> Antipsychotic medications, especially olanzapine and clozapine, may cause substantial weight gain.<sup>309</sup> Other psychotropic medications such as Risperdal (risperidone),<sup>310</sup> Prozac (fluoxetine),<sup>311</sup> Paxil (paroxetine),<sup>312</sup>

<sup>302</sup> David M. Cutler, et al., *Why Have Americans Become More Obese?*, 17 J. ECON. PERSP. 93, 97 (2003). See also, R.A. Epstein, *supra* note 112, at 1371; S-Y Chou, et al., *An Economic Analysis of Adult Obesity: Results From the Behavioral Risk Factor Surveillance System*, 23 J. HEALTH ECON. 565, 568 (2004).

<sup>303</sup> See U.S. Census Bureau, U.S. Dep't. of Commerce, *Work and Work-Related Activities of Mothers Receiving Temporary Assistance to Needy Families: 1996, 1998, and 2000*, at 3 (2002), available at <http://www.census.gov/prod/20002pubs/p70-85.pdf>, (last visited July 12, 2006). The average time mothers spend preparing meals at homes has declined by more than 50% in the last two decades. D. Cutler, E. Glaeser, & Shapiro, Jr., *Why Have Americans Become More Obese?*, National Bureau of Economic Research, Working paper 94546 (2003), *supra*, n. 213.

<sup>304</sup> See, e.g., Menu, Burger King's Website, <http://www.bk.com/@menu=2,-1,-1>, (visited July 12, 2006).

<sup>305</sup> In restaurants, consumers tend to view larger portions as better value for money. *Perspectives on marketing, self-regulation, & childhood obesity: A report on a Joint Workshop of the FTC & the DHHS*, 13 (Apr. 2006).

<sup>306</sup> S.J. Nielsen & B.M. Popkin, *Patterns & Trends in Food Portion Sizes, 1997-1998*, 289 (4) JAMA 450-453 (2003). Critics of the restaurant industry have some tendency to rely on food manufacturing data, rather than upon evidence that diners today actually consume more at each meal. J. Eric Oliver, "Food and Weight Gain: Super Sized Misperceptions," in *Fat politics: The Real Story Behind America's Obesity Epidemic*, NY: OXFORD UNIV. PRESS (2006), p. 133.

<sup>307</sup> M. Fava, *Weight Gain and Antidepressants*, 61 (Supp.) J. CLIN. PSYCHIATRY 37-41 (2001).

<sup>308</sup> See, "Obesity" in the "Merck Manual of Medical Information," 2d Home Ed. Online, at <http://www.merck.com/mmhe/sec12/ch156/ch156a.html>, (last visited July 7, 2006).

<sup>309</sup> See, A. Firestone, *Weight Gain and Diabetes With "Second Generation" Antipsychotic Drugs*, 182 (12) MED. J. AUSTRAL. 652-653 (2005) (letter).

<sup>310</sup> *Physicians' Desk Reference*, Thompson PDR: Montvale, N.J. (2005) at 1746.

<sup>311</sup> M. Fava, et al., *Fluoxetine vs. Sertraline and Paroxetine in Major Depressive Disorder: Changes in Weight With Long-Term Treatment*, 61 J. CLIN. PSYCHIATRY 863-867 (2000). Prozac has also been reported to cause anorexia and weight loss. *Physicians' Desk Reference*, *supra* note 310, at 1875.

<sup>312</sup> Paroxetine may be more likely than other drugs in its class to cause weight gain. R. Deshmukh & K. Franco, *Managing Weight Gain as a Side Effect of Antidepressant Therapy*, 70 (7) CLEVELAND CLIN. J. MED. 614-623 (July 2003), citing M. Fava, et al., *Fluoxetine v. Sertraline and Paroxetine in Major Depressive Disorder: Changes in Weight With Long-Term Treatment*, 61 J. CLIN. PSYCHIATRY 863-67 (2000). Paxil, like fluoxetine, has also been reported to cause anorexia and weight loss. *Physicians' Desk Reference*, *supra* note 312, at 1590.

may have similar effects.<sup>313</sup> Benzodiazepines increase food intake in humans.<sup>314</sup> Blood pressure agents (beta blockers, calcium channel blockers), corticosteroids, and even antihistamines sold over-the-counter can induce weight gain or make it harder for overweight patients to lose weight.<sup>315</sup> One class of drugs, the thiazolidinediones, is said to improve multiple risk factors in obese diabetics while causing significant increases in body fat.<sup>316</sup>

Nor in surveying causes of obesity should alcohol be overlooked. Alcohol consumption is energy consumption.<sup>317</sup> Alcohol's energy content is 7 k-cal per gram, second only to fat in energy density,<sup>318</sup> yet because it is metabolized fairly gradually, it is the least satiating of all macronutrients.<sup>319</sup> Alcohol reduces oxidation of fat and favors fat storage, which can result in weight gain.<sup>320</sup> Among non-abusing drinkers, food intake is generally not diminished despite intake of energy in the form of alcohol. In fact, moderate alcohol consumption before a test meal is associated with a short-term increase in food intake. Although it has long been thought that alcohol's appetite-stimulating effects did not persist beyond acute administration,<sup>321</sup> newer evidence suggests that alcohol may stimulate food intake over extended periods.<sup>322</sup> Alcohol suppresses fatty acid oxidation and increases short-term thermogenesis. It stimulates several neurochemical and peripheral systems implicated in appetite control, and also may influence energy balance in other ways, such as by inhibiting leptin and enhancing the effects of neuropeptide Y.<sup>323</sup>

### I. Weight at Birth and in Childhood

A child's weight is often related to his weight as an adult,<sup>324</sup> it is related to the weights of his parents and to his birth weight as well.<sup>325</sup> A child's weight may also be influenced

<sup>313</sup> See, D.M. Taylor & R. McAskill, *Atypical Antipsychotics and Weight Gain: A Systematic Review*, 101 ACTA PSYCHIATRICA SCAND. 416-432 (2000); I. Kawachi, *Physical and Psychological Consequences of Weight Gain*, 60 (Suppl. 21) J. CLIN. PSYCHIATRY 5-9 (1999); D.B. Allison & D.E. Casey, *Anti-psychotic Induced Weight Gain: A Review of the Literature*, 62 (Suppl.) J. CLIN. PSYCHIATRY 22-31 (2001); T. Baptista, et al., *Drug Induced Weight Gain—An Impediment to Successful Pharmacotherapy: Focus on Antipsychotics*, 5 CURR. DRUG TARGETS 279-299 (2004).

<sup>314</sup> M. Haney, et al., *Alprazolam Increases Food Intake in Humans*, 132 PSYCHOPHARMACOL. 311-314 (1997).

<sup>315</sup> M. Duenwald, *Is Your Medicine Cabinet Making You Fat?*, NY TIMES, at F-5 (Aug. 16, 2005).

<sup>316</sup> T.M. Larsen, et al., *PPARgamma Agonists in the Treatment of Type II Diabetes: Is Increased Fatness Commensurate With Long-Term Efficacy?*, 27 INT. J. OBES. RELAT. METAB. DISORD. 147-161 (2003).

<sup>317</sup> Among adult Americans, the contribution of ethanol to total daily energy intake averages 10%. E. Jequier, *Alcohol Intake and Body Weight: A Paradox*, 69 (2) AM. J. CLIN. NUTR. 173-174 (1999).

<sup>318</sup> A.M. Prentice, *Alcohol and Obesity*, 19 (Suppl. 5) INT. J. OBES. RELAT. METAB. DISORD. S44-50 (1995).

<sup>319</sup> M.R. Yeomans, et al., *Alcohol and Food Intake*, 6 (6) CURR. OPIN. CLIN. NUTR. METAB. CARE 639-644 (2003) at 640, 641.

<sup>320</sup> F. Zurlo, et al., *Low Ratio of Fat Carbohydrate Oxidation as Predictor of Weight Gain: Study of 24-h RQ*, 259 (5 Pt 1) AM. J. PHYSIOL. E 650-657 (1990).

<sup>321</sup> Yeomans, et al., *supra* note 319. There is evidence, however, that while binge drinkers and heavy drinkers are at higher risk for obesity, light-to-moderate drinkers (1-2 drinks/d) may be at lower risk than teetotalers. A.A. Arif & J.E. Rohrer, *Patterns of Alcohol Drinking and its Association With Obesity: Data From the Third National Health and Nutrition Examination Survey, 1988-1994*, 5 BMC PUBLIC HEALTH 126 (Dec. 2005), available at <http://www.biomedcentral.com/1471-2458/5/126>, (last visited July 7, 2006).

<sup>322</sup> W.H.M. Saris & M.A. Tarnopolsky, *Controlling Food Intake and Energy Balance? Which Macronutrient Should We Select?*, 6 CURR. OPIN. CLIN. NUTR. METAB. CARE 609-613 (2003) at 612; Yeomans, *supra* note 319 at 641, citing S.G. Wannamathée & A.G. Shaper, *Alcohol, Body Weight, and Weight Gain in Middle-aged Men*, 77 AM. J. CLIN. NUTR. 1312-1317 (2003); Y. Sakurai, et al., *Relation of Total and Beverage-specific Alcohol Intake to BMI and Waist-to-Hip Ratio: A Study of Self-Defense Officials in Japan*, 13 EUR. J. EPIDEMIOL. 893-898 (1997); and A. Raben, et al., *Meals With Similar Energy Densities but Rich in Protein, Fat, Carbohydrate, or Alcohol Have Different Effects on Energy Expenditure and Substrate Metabolism but not on Appetite and Energy Intake*, 77 AM. J. CLIN. NUTR. 91-100 (2003).

<sup>323</sup> Yeomans, et al., *Alcohol and Food Intake*, *supra* note 319 at 639-644.

<sup>324</sup> See, e.g., S.M. Garn & M. LaVella, *Two-decade Follow-up of Fatness in Early Childhood*, 139 (2) AM. J. DIS. CHILD. 181-185 (1985).

<sup>325</sup> D.S. Seidman, et al., *A Longitudinal Study of Birth Weight and Being Overweight in Late Adolescence*, 145 AM. J. DIS. CHILD 782-785 (1991); W.H. Dietz, *Periods of Risk in Childhood for the Development of Adult Obesity—What Do We Need to Learn?*, 127 (9) J. NUTR. 18845-18865 (1997).

by intrauterine factors. Famine during the last trimester of pregnancy and during the first months of life correlates with a low prevalence of obesity in adulthood. Famine during the first half of pregnancy, however, is associated with higher obesity rates, perhaps because nutritional deprivation at that point in development adversely affects hypothalamic centers regulating food intake and growth.<sup>326</sup> Intrauterine growth retardation is associated with reduced weight and height until at least three years of age and possibly longer.<sup>327</sup> Infants of diabetic mothers are usually macrosomic at birth.<sup>328</sup> After controlling for weight, the association between maternal and adolescent obesity may indicate that a diabetic intrauterine milieu is an independent risk factor for obesity.<sup>329</sup> Infants with the highest weight or body mass index or who grow rapidly during infancy are at increased risk of subsequent obesity.<sup>330</sup> Protracted breastfeeding, in contrast, may protect against overweight.<sup>331</sup> Patterns of growth during infancy may be associated with both childhood and adult obesity.<sup>332</sup> The likelihood that a child's body weight percentile will change appreciably decreases as the child ages.<sup>333</sup> Birth weight has been rising because we have successfully reduced the prevalence of prematurity, and we are providing better and more extensive prenatal care and maternal nutrition. While no one would argue that obesity is a public health goal, higher birth weights do correlate with reduced infant mortality.

### J. Sleep Deprivation

New evidence suggests that the duration of sleep may play a role in regulating body weight and metabolism by modulating key hormones such as leptin and ghrelin.<sup>334</sup> Chaput and colleagues found that

<sup>326</sup> G.P. Ravelli, et al., *Obesity in Young Men After Famine Exposure in utero and in Early Infancy*, 295 (17) N. ENGL. J. MED. 349-353 (1976).

<sup>327</sup> R.S. Strauss & W.H. Dietz, *Effects of Intrauterine Growth Retardation in Premature Infants on Early Childhood Growth*, 130 J. PEDIATR. 95-102 (1997).

<sup>328</sup> R.C. Whitaker, et al., *Gestational Diabetes and the Risk of Offspring Obesity*, 4 OBES. RES. 215 (1996).

<sup>329</sup> M. Rosenbaum & R.L. Leibel, *The Physiology of Body Weight Reduction: Relevance to the Etiology of Obesity in Children*, 101 PEDIATR. 525-539 (1998). Among the factors influencing childhood overweight are maternal weight before and during pregnancy, smoking during pregnancy and infant feeding, and birth weight. P.J. Salsberry & P.B. Reagan, *Dynamics of Early Childhood Overweight*, PEDIATRICS, <http://www.pediatrics.org/cgi/content/full/116/6/1329>, at 1329, (last visited July 7, 2006). Intrauterine exposure to a maternal diabetic internal milieu may cause a lifelong change in insulin production and sensitivity in the newborn, increasing the likelihood that these offspring will become overweight and develop type 2 diabetes later in life. *Id.*, citing D. Dabelea, et al., *Intrauterine Exposure to Diabetes Conveys Risks for Type 2 Diabetes and Obesity: A Study of Discordant Sibships*, 49 DIABETES 2208 (2000); M.W. Gillman, et al., *Maternal Gestational Diabetes, Birth Weight, and Adolescent Obesity*, 111 PEDIATRICS 3 (2003) available at: [www.pediatrics.org/cgi/content/full/111/3/e221](http://www.pediatrics.org/cgi/content/full/111/3/e221), (last visited July 7, 2006); R.C. Whitaker, et al., *Gestational Diabetes and the Risk of Offspring Obesity*, 101 PEDIATRICS e9 (1998) available at: [www.pediatrics.org/cgi/content/full/101/2/e9](http://www.pediatrics.org/cgi/content/full/101/2/e9), (last visited July 7, 2006); R.C. Whitaker & W. Dietz, *Role of the Prenatal Environment in the Development of Obesity*, 132 J. PEDIATR. 768-776 (1998).

<sup>330</sup> J. Baird, et al., *Being Big or Growing Fast: Systematic Review of Size and Growth in Infancy and Later Obesity*, 331 BMJ 929 (Oct. 22, 2005).

<sup>331</sup> M.S. Kramer, et al., *Infant Determinants of Childhood Weight and Adiposity*, 107 J. PEDIATR. 104-107 (1985); M.L. Hediger, et al., *Association Between Infant Breastfeeding and Overweight in Young Children*, 285 JAMA 2453-2460 (2004); M. Gillman, et al., *Risk of Overweight Among Adolescents Who had Been Breast Fed as Infants*, 285 JAMA 2461-2467 (2001); von Kries, et al., *Breast Feeding and Obesity: Cross Sectional Study*, 319 BMJ 147-150 (1999).

<sup>332</sup> M. Stettler, et al., *Infant Weight Gain and Childhood Overweight Status in a Multi-Center, Cohort Study*, 109 PEDIATRICS 194-199 (2002).

<sup>333</sup> R.C. Whitaker, et al., *Predicting Obesity in Young Adulthood From Childhood and Parental Obesity*, 337 N. ENGL. J. MED. 869-873 (1997); S.S. Guo, et al., *Body Mass Index During Childhood, Adolescence and Young Adulthood in Relation to Adult Overweight and Adiposity: The FELS Longitudinal Study*, 24 INT. J. OBES. RELAT. METAB. DISORD. 1628-1635 (2000).

<sup>334</sup> K. Spiegel, et al., *supra* note 266; S. Taheri, et al., *Short Sleep Duration is Associated With Reduced Leptin, Elevated Ghrelin, and Increased Body Mass Index*, 3 PLoS MED. e62 (2004).

[S]hort sleep duration was associated with decreased leptin levels, increased ghrelin levels and increased hunger and appetite....If the findings prove to be reproducible and generalizable, and the hormonal changes of leptin and ghrelin owing to sleep curtailment cause changes in food intake over time, we could add sleep duration to the environmental factors that are prevalent in our society and that contribute to weight gain and obesity.<sup>335</sup>

The authors noted that reduced sleeping hours is characteristic of modern society. They wrote:

It is somewhat paradoxical that sleeping, the most sedentary of all activities, may be associated with leanness. Although recommendations to get both a better night's sleep and more exercise might superficially seem to be at odds with each other from the perspective of energy expenditure and energy balance, these simple goals may well become a part of our future approach to combating obesity.<sup>336</sup>

### K. Infections

Traditionally, obesity was not classified as an infectious disease. That is not likely to change soon, but there is intriguing recent evidence that microbial pathogens may play a role.<sup>337</sup> Dhurandhar, et al. have suggested that adenovirus 36 (AD-36) may be a cause of obesity in some. They found that 15% of obese humans have antibodies to the virus, indicating prior exposure to AD-36; in contrast, 0% of normals demonstrated such antibodies. Additionally, infected monkeys followed for 18 months also demonstrated weight gain. Marmosets inoculated with AD-36 exhibited a threefold weight gain, including greater fat gain than three uninfected controls at 28 weeks post-inoculation.<sup>338</sup>

The mechanism of action of AD-36 is not yet understood. Infected animals did not eat more than uninfected ones, however. It has been suggested that the virus may decrease energy expenditure rather than increase appetite.<sup>339</sup> This theory may be plausible, but more work is needed before the effects of AD-36 are elucidated. In the meantime, researchers are investigating the possibility of creating a vaccine to prevent infections and, perhaps, some cases of obesity.<sup>340</sup> Whether other microbes may play a role remains to be seen.

Given the array of factors that contribute to energy consumption, metabolism and expenditure, and the comparatively modest impact that the practices of food companies could potentially have upon body weight, Judge Sweet's skepticism about the plaintiffs' causation case in *Pelman* was wholly justified.

## IV. ADDICTION

The numerous factors affecting weight create a significant obstacle to overweight plaintiffs attempting to blame the food industry for their problems. Invoking addiction theory is not the solution. Addiction entails repeated, compulsive use of a psychoactive

<sup>335</sup> J.P. Chaput, M. Brunet, & A. Tremblay, *Relationship Between Short Sleeping Hours and Childhood Overweight/Obesity: Results From the "Québec en Forme" Project*, 30 (7) INT. J. OBESITY 1080-5 (2006), available at <http://www.nature.com/ijo/journal/vaop/ncurrent/abs/0803291a.html> (last visited June 22, 2006).

<sup>336</sup> Chaput, et al., *supra* note 335, at 1084.

<sup>337</sup> N.V. Dhurandhar, et al., *Human Adenovirus AD-36 Promotes Weight Gain in Male Rhesus and Marmoset Monkeys*, 132 (10) J. NUTR. 3155-3160 (2002).

<sup>338</sup> *Id.*

<sup>339</sup> *Id.*

<sup>340</sup> F. Greenway, *Virus-induced Obesity*, 290 AM. J. PHYSIOL. REGUL. INTEGR. COMP. PHYSIOL. R188-R189 (2006).

substance not physiologically needed for survival despite the threat, often grave, that its use poses to health and even life.<sup>341</sup> The power of an addiction can be gauged by what the addict is prepared to sacrifice for his drug of choice. Addicts value their preferred drugs above everything else. The addict's drug of choice is more important than family, health, finances, and sometimes even food, shelter and freedom from imprisonment.<sup>342</sup> Despite shame, fear, self-loathing and the threat of criminal penalties, the addict eagerly seeks, procures and consumes his drug of choice.<sup>343</sup> Characteristically, a chemically dependent person also loses the ability to perceive and admit his addiction. This denial arises from 1) the drug's pharmacologic effect on memory and the power and influence of euphoric recall; 2) psychological ego-defense mechanisms such as suppression and repression; and 3) associates' unwitting enabling behaviors.<sup>344</sup> Addicts typically view drugs as the solution to their problems rather than the source, and they develop superficially plausible explanations for their drug use.<sup>345</sup>

### A. *The Attraction of Addiction Theory for Plaintiffs*

In an insightful article, Rogers and Smit<sup>346</sup> demonstrate that overeaters invoke addiction as a way to dodge personal responsibility for consuming highly palatable, energy-dense foods such as chocolate in quantities far beyond their needs.

By saying that "I crave chocolate," or confessing that "I am a chocoholic," the individual is able to explain why he eats chocolate frequently and why he finds it difficult to resist. Attributing what is perceived as excessive consumption of chocolate, or the use of chocolate as a mood modifier, to an addiction provides a more socially (and personally) acceptable explanation for this behavior, and thereby helps to remove individual responsibility for the difficulty. On the basis of the publicly accepted model of addictions, it implies that eating chocolate is outside the person's control, and alternatively attributes the problems of control to biological effects of the food (e.g., it contains a dependence-forming substance), or possibly to an individual pathology, thus invoking a medical model.<sup>347</sup>

Rogers and Smit explain that "attributions" are common sense explanations made in an attempt to understand and sometimes to excuse behavior.<sup>348</sup> Invoking addiction enables the overeater to evade guilt, at least to a degree, and may deter others' criticisms.

### B. *The Attraction of Addiction Theory for Plaintiffs' Counsel*

One of the reasons tobacco litigation has been so lucrative for the plaintiffs' bar is that they succeeded in convincing finders of fact that the defendants knowingly and indeed

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<sup>341</sup> H.E. Smith, G.D. Talbott, & M.A. Morrison, *Chemical Abuse and Dependence: An Occupational Hazard for Health Professionals*, 7 (3) TOPICS IN EMERG. MED. 69-78 (1985); USDHHS, 1998 Surgeon General Report, *The Health Consequences of Smoking: Nicotine addiction*, Wash., D.C.: U.S. Government Printing Office, 1988, available at [www.cdc.gov/tobacco/sgr/sgr\\_1998/index.htm](http://www.cdc.gov/tobacco/sgr/sgr_1998/index.htm), (last visited July 12, 2006). In its discussion of substance-related disorders, the American Psychiatric Association defines a "substance" to mean: "a drug of abuse, a medication, or a toxin." DIAGNOSTIC AND STATISTICAL MANUAL OF MENTAL DISORDERS, 4th Ed. (Text Revision), American Psychiatric Association: Wash., D.C. (2000) at 191.

<sup>342</sup> E. Sullivan, L. Bissell, & E. Williams, *Chemical Dependency in Nursing: The Deadly Diversion*, Reading, Mass: Addison-Wesley (1988).

<sup>343</sup> R. Coombs, *Addiction's Defining Nature*, 64 TEX. BAR J. 166, 171 (2001).

<sup>344</sup> R.M. Morse, R.M. & D.K. Flavin, *The Definition of Alcoholism*, 268 JAMA 1012-1014 (1992) at 1013.

<sup>345</sup> R. Coombs, *supra* note 343.

<sup>346</sup> P.J. Rogers & H.J. Smit, *supra* note 211.

<sup>347</sup> *Id.* at 10.

<sup>348</sup> *Id.*

intentionally manufactured an unambiguously addicting product.<sup>349</sup> Many of the same lawyers now hope to make a similar showing with foods.<sup>350</sup> There are several reasons why plaintiffs' lawyers may be motivated to attempt to portray eating as an addiction. In the tobacco context, Vanderbilt's Professor Richard Nagereda offers four: First, pleading addictiveness allows the plaintiff to attack the defense claim that individuals choose to smoke. Second, focusing on addiction "lends favorable atmospherics" to states' claim for reimbursement for tobacco-related Medicaid expenditures. Third, challenging the primacy of individual choice may facilitate regulation of tobacco. Fourth, and perhaps most significant, focusing on nicotine addiction permits litigants to allege that industry deliberately marketed cigarettes to the most vulnerable consumers—teenagers and even younger children—so manufacturers might find new recruits to replace those tobacco veterans who die from smoking-related diseases.<sup>351</sup>

A related benefit, from the plaintiff's perspective, is the damage that addiction theory may be able to do, at least in a product liability context, to the power and effectiveness of assumption of the risk as a keystone of the defense case.<sup>352</sup> Some courts see a difference between common knowledge as it relates to the general risks of smoking and common knowledge as it relates to nicotine addiction. Of the courts that have made that distinction, most conclude that the dangers of nicotine addiction, as opposed to those of smoking more generally, are not common knowledge, and are not risks that plaintiffs in fact assumed.<sup>353</sup>

As noted, overeaters invoke addiction theory to escape the burden of guilt. Analogously, their prospective lawyers may find addiction theory an attractive means to make plaintiffs appear blameless to finders of fact. The first such case in the tobacco context was a suit brought by a group of flight attendants occupationally exposed to second-hand smoke.<sup>354</sup> These plaintiffs, unlike smokers, were exposed to tobacco smoke only because it was pervasive in their work environments. Using non-smoking plaintiffs was intended to strip the tobacco industry of its "freedom of choice" defense.<sup>355</sup> In obesity litigation, plaintiffs' lawyers may allege addiction to foods for the same reason.

Of course, manufacturing an addicting substance, such as morphine or alcohol, is neither unlawful nor tortious. But labeling their obese clients as "addicts" will allow plaintiffs' lawyers to portray overeaters as victims of physiological processes that, counsel will claim, are beyond their control and not their fault.<sup>356</sup> Such theories may also be offered as a basis to impose new labeling requirements on packaged foods.

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<sup>349</sup> See, e.g., Comment, *Forcing Round Classes into Square Rules: Attempting Certification of Nicotine Addiction-as-Injury Class Actions Under Federal Rule of Civil Procedure 23(b)(3)*, 29 U. TOL. L. REV. 699, 700-701 (Summer 1998), cited in Pelman I, *supra* note 16, 237 F.Supp.2d at 531 n. 18. See also, J. Turley, *A Crisis of Faith: Tobacco and the Madisonian Democracy*, 37 HARV. J. LEGIS. 433, 443 (2000).

<sup>350</sup> See, e.g., Pelman I, *supra* note 16, 237 F.Supp.2d at 542.

<sup>351</sup> R.A. Nagereda, *Outrageous Fortune and the Criminalization of Mass Torts*, 96 MICH. LAW R. 1121, 1153-1157 (Mar. 1998).

<sup>352</sup> Assumption of the risk was a cornerstone of the decades-long success the tobacco industry enjoyed against claims. Martha A. Derthick, *Up in Smoke: From Legislation to Litigation in Tobacco Politics*, 39 (2002). Another reason plaintiffs may well prefer to proceed under state consumer protection acts is precisely to attempt to avoid this defense. U.G. Williams & K.J. Parsigian, *supra* note 71.

<sup>353</sup> See, Rogers v. R.J. Reynolds Tobacco Co., 557 N.E.2d 1045 (Ind. Ct. App. 1990).

<sup>354</sup> See Broin v. Phillip Morris Cos., *supra* note 163.

<sup>355</sup> See Donald P. Baker, *Nonsmoker's Suit Presents New Challenge for Industry; Tobacco Makers Can't Use "Freedom-of-Choice Defense"*, WASH. POST, May 31, 1997 at A3, available at 1997 WL 11585888.

<sup>356</sup> See, K. Goodrich, *Inability to Control Eating: Addiction to Food or Normal Response to Abnormal Environment?* in DRUGS & SOCIETY, Hayworth Press, Inc., Vol. 15. No. 1-2 (1999), pp. 124. Plaintiffs so arguing, however, could founder on the wrongful conduct rule, under which recovery in tort is barred if plaintiff's injuries arose while he was engaged in, or as a proximate result of, immoral or illegal conduct. Defendants might be able to prevail by showing that overeating is self-destructive and hence immoral. *Cf.*,

So far, however, lawyers arguing that food is addictive have not been very clear or detailed about the basis for their argument.<sup>357</sup> The *Pelman* complaint, for example, failed to identify what substance(s) in McDonald's products plaintiffs claimed was addictive, nor how often or in what fashion one might have to consume that substance to become addicted to it. To the extent they have shed any light on this theory, however, most plaintiffs' counsel seem to have relied upon one version or another of the Reward-Deficiency Theory, discussed below.

We shall examine this theory and its relationship to obesity claims, recognizing that at this early stage the form the argument takes may well evolve. If it does, the responsive analysis, of course, will need to be modified as well. We will first discuss the application to food of the American Psychiatric Association's (APA's) *Diagnostic and Statistical Manual* (DSM) criteria for substance dependence in order to examine the features that are often used to define the term.

### C. The Definition of Addiction

Addiction is difficult to define. Many formulations have been offered.<sup>358</sup> Terms such as drug dependence, drug abuse, and drug addiction also have many colloquial meanings.<sup>359</sup> Consensus is wanting. There has been some tendency of late to liberalize the concept, as parodied by *The Hour*, a Canadian television show airing on the CBC. *The Hour* recently ran a piece on a putative Blackberry addiction, a phenomenon it referred to as "Crackberry."<sup>360</sup> Critics of this casual approach note that to label some behavior an addiction merely because it is habitual is inconsistent with the scientist's pursuit of testable hypotheses generating useful knowledge. Excessively liberal expansion of the concept of addiction not only trivializes the tragedy of true drug dependencies, but may also provide people with a convenient excuse not to quit, and may even provide young people with an inaccurate perspective that could actually encourage experimentation with hardcore drugs.<sup>361</sup>

Today, the definition of addiction most widely used is probably that of APA's *Diagnostic and Statistical Manual of Mental Disorders*<sup>362</sup>, commonly referred to as *DSM-IV-TR* or simply as *DSM-IV* or "the *Manual*." Despite its widespread use, however, *DSM-IV-TR* needs to be interpreted in light of its origins, function, and limitations. The original *Diagnostic and Statistical Manual* was created by the APA in continuation of an effort dating to the 1840s to count and classify mental illnesses. That effort was

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Price v. The Purdue Pharma Co., No. 2003-CA-02101-SCT (Miss. 2006), available at <http://www.mssc.state.ms.us/Images/OPINIONS/CO31796.PDF> (last visited July 7, 2006) (plaintiff who violated law governing use of controlled substances could not recover against substance's manufacturer). Calling overeaters addicts may advance their litigation interests, but could, ironically, have negative therapeutic consequences. B. Magid, *Is Biology Destiny After All? Three Clinical Conundrums: Homosexuality, Alcoholism, and Obesity*, 4 J. PSYCHOTHERAP. PRAC. AND RES. 8 (1995). See discussion, nn. 575-577 *infra*, and associated text. For an argument that addictive behavior is more a function of character than of biologic or other causes, see A. Schwartz, *Views of Addiction and the Duty to Warn*, 75 VA. L. REV. 509 (1989).

<sup>357</sup> See letter from John Banzhaf to fast food chains of 6/19/03, available at <http://banzhaf.net/docs/ffltr.html>. *New Scientist*, on which Banzhaf relies, is not peer-reviewed. [http://en.wikipedia.org/wiki/New\\_Scientist](http://en.wikipedia.org/wiki/New_Scientist).

<sup>358</sup> J.R. Hughes, et al., *Should Caffeine Abuse, Dependence, or Withdrawal be Added to DSM-IV and ICD-10?* 149 AM. J. PSYCHIATRY 33 (Jan. 1992).

<sup>359</sup> G.F. Koob & F.E. Bloom, *Cellular and Molecular Mechanisms of Drug Dependence*, 4 SCI. 716 (Nov. 4, 1988).

<sup>360</sup> See, <http://www.blackberrycool.com/2005/07/15/00650/>, (last visited July 7, 2006).

<sup>361</sup> J.H. Robinson & W.S. Pritchard, *The Meaning of Addiction: Reply to West*, 108 PSYCHOPHARMACOLOG. 411-416 (1992).

<sup>362</sup> See note 341 *supra*.

stimulated in the mid-20<sup>th</sup> century by dissatisfaction with existing disease classification systems, a recognition that major differences in diagnoses existed between psychiatric practices in the U.S. and the United Kingdom (UK), and the need of third-party payers for standardized nomenclature.<sup>363</sup> Before its advent, psychiatrists often tended to use rather idiosyncratic terminology to describe their patients' problems and their own interventions. As third-party payers began to play a larger role in medical economics in the second half of the 20th century, they began to demand a degree of standardization in terminology, so that like services were paid for in like manner.<sup>364</sup> The *Manual*, then, was not intended to be a textbook of nosology.<sup>365</sup>

The *Manual's* limitations, acknowledged by its authors, include imprecision of definitions; ongoing evolution in mental health theory; the need for interpretation of symptoms, signs, and the text of the *DSM-IV* itself; the focus on medical, not legal questions; and the fact that diagnosis does not mean a lack of individual variation.<sup>366</sup> Distinctions between and among mental disorders generally remain fuzzier than is the case with, say, orthopedic problems. We should remember that psychiatry is the youngest medical specialty, and it seeks to understand some of the most complex human problems. It is no slight to psychiatrists to point out that, at this early stage in the evolution of their art, more is unknown than is known.

Among the areas where controversy among psychiatrists remains vigorous is that often most central to legal inquiry: the criteria for diagnoses. However valuable, *DSM-IV* has distinct limitations here also. Those using it must be trained not only in the subtleties of diagnosis, but in the limitations of the art, as well.<sup>367</sup>

Of particular relevance to the present discussion, the *Manual* provides an express warning specific to litigation: Diagnostic information is not congruent with the requirements of legal standards, and abilities and disabilities may vary widely within a diagnostic category.<sup>368</sup>

If we are to use the *Manual* properly, we need to recognize that it cannot be applied willy-nilly in a legal setting. As its authors have been at pains to point out, *DSM-IV* was created to serve medical, not legal needs.<sup>369</sup> *DSM-IV-TR* has distinct limits, even medically, and certainly legally. All other attempts at summarizing mental disorders, however, also have imperfections. Despite its shortcomings, *DSM-IV-TR* is used enough in clinical practice that it will likely be invoked in court. That likelihood justifies analyzing it here.

*DSM-IV-TR* does not actually define "addiction."<sup>370</sup> Instead, it sets out a seven-part test of "substance dependence":

A maladaptive pattern of substance use, leading to clinically significant impairment or distress, as manifested by three (or more) of the following, occurring at any time in the same 12-month period:

<sup>363</sup> R. Spitzer & J.W.B. Williams, "Classification of Mental Disorders and DSM-III," in H. Kaplan, A.M. Freedman, & B.J. Sadock, eds., *Comprehensive Textbook of Psychiatry III*, Williams & Wilkins, Baltimore (1980) at 1053.

<sup>364</sup> See generally, E. Shorter, *A History of Psychiatry*, NY: WILEY (1997).

<sup>365</sup> For a somewhat harsh critique of the *DSM*, see, P.H. Caplan, *They Say You're Crazy: How the World's Most Powerful Psychiatrists Decide Who's Normal*, ADDISON WESLEY PUBLISHING (1995) at 85.

<sup>366</sup> *DSM-IV, TR*, *supra* n. 341, at xxx-xxxii

<sup>367</sup> *Id.*

<sup>368</sup> *Id.* at xxxii-xxxiii.

<sup>369</sup> *Id.*

<sup>370</sup> Courts still use the term, however. An addiction is an "[h]abitual psychological and physiological dependence on a substance or practice which is beyond voluntary control." Stedman's Medical Dictionary 23 (25th ed.1990). Haglund v. Philip Morris, Inc., *supra* n.8, 446 Mass. at 750, 847 N.E.2d at 324.

- (1) tolerance, as defined by either of the following:
  - (a) a need for markedly increased amounts of the substance to achieve intoxication or desired effect
  - (b) markedly diminished effect with continued use of the same amount of the substance.
- (2) withdrawal, as manifested by either of the following:
  - (a) the characteristic withdrawal syndrome for the substance (refer to Criteria A and B of the criteria sets for Withdrawal from the specific substances)
  - (b) the same (or a closely related) substance is taken to relieve or avoid withdrawal symptoms
- (3) the substance is often taken in larger amounts or over a longer period than was intended
- (4) there is a persistent desire or unsuccessful efforts to cut down or control substance use
- (5) a great deal of time is spent in activities necessary to obtain the substance, use the substance, or recover from its effects
- (6) important social, occupational, or recreational activities are given up or reduced because of substance use
- (7) the substance use is continued despite knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been caused or exacerbated by the substance.<sup>371</sup>

With respect to obesity, let us consider two of the most important and widely-recognized criteria for addiction: the first two on the *DSM* list above. Despite the debate about addiction's proper definition, few if any formulations omit these two characteristics. Later, as an example of how the criteria might be applied to a food substance, we shall to the extent practicable use all the criteria to consider caffeine, which some have called addicting.

### 1. *Tolerance*

"Tolerance" refers to the tendency, over time, of a given dose of an addicting drug to grow less effective in achieving the desired effect. A larger and larger dose is required. Because of this characteristic, addiction is a progressive disorder.<sup>372</sup> But not all obese persons necessarily ingest foods in amounts larger than others do, and they need not necessarily ingest food over a period longer than others do. Often no differences can be found in the eating habits of obese and lean people.<sup>373</sup> An obese individual certainly need not consume progressively larger quantities of food to get his "fix." Consider the overeater who is fond of hamburgers. If overeating were truly an addiction, one would

<sup>371</sup> *DSM IV (TR)*, *supra* note 341, at 197.

<sup>372</sup> See, A. Graham & C. Glickauf-Hughes, *Object Relations and Addiction: The Role of "Transmitting Externalizations"*, 22 J. CONTEMP. PSYCHOTHERAPY 21, 28 (1992). See also, A.W. Schaeff, *When Society Becomes an Addict*, HARPER COLLINS (1987) at 18. Addicts tend to require progressively higher doses to feed their addictions. See D.P. Rice, et al., *The Economic Costs of Alcohol and Drug Abuse and Mental Illness: 1985* (1990) at 2. Many consider an increasing cost of non-consumption to be a defining characteristic of an addiction. See, e.g., C.R. Sunstein, *Legal Interference With Private Preferences*, 53 U. CHI. L. REV. 1129, 1159 (1986).

<sup>373</sup> S. Dyrenforth, O. Wooley, & S. Wooley, "A Woman's Body in a Man's World: A Review of Findings on Body Image and Weight Control," in *A Woman's Conflict: The Special Relationship Between Women and Food*, E. Kaplan, ed., Englewood Cliffs, N.J., PRENTICE HALL (1980), cited in B. Magid, *supra* note 356.

expect our hamburger-lover to consume, say, one at his first exposure, two at his second the following day, three the day after that, and so on. After several such increases, however, if not before, this pattern would clearly have to stop. Even if we assume in this hypothetical a much more gradual development of something akin to "tolerance," the satiety problem remains. Separate the increments in consumption by weeks or even months (although such slow progression would not be expected with such powerfully addicting substances as, say, crack cocaine): the result would not change. There is a limit to how much food even the most enthusiastic hamburger-lover can take in at one sitting, even if we give him years to grow his "tolerance."

Consider also treatment for tolerance. In true addiction, more and more drug must be taken to achieve the same effect. Under skilled therapy, tolerance is reduced (eventually, eliminated) by substance abstinence; the addict's capacity to metabolize his drug of choice rapidly diminishes gradually. To use the hamburger example above, it is unlikely that abstinence, even for an extended period, would significantly alter the rate at which the consumer metabolizes the burger. But the difficulty with the analysis is more subtle and broader than that. If we starve ourselves, and then eat, our bodies will replenish and even overload our fat cells in a process called "adaptive hyperlipogenesis." The more severe the food restriction, the more this overloading takes place.<sup>374</sup> Hyperlipogenesis, then, is intensified by food restriction.<sup>375</sup>

## 2. *Withdrawal*

A major feature of addiction to many drugs is a constellation of negative physiological and emotional features, loosely referred to as "withdrawal syndrome," that occur when the drug is not taken. Withdrawal is the name given to a set of symptoms and signs that the addict displays when he is unable to use his drug of choice for a length of time sufficient to allow its blood levels to drop below those where they have their accustomed effects. The syndrome of withdrawal varies with the addict and more especially with the drug, but it is usually unpleasant and may itself be dangerous.

Symptoms of withdrawal from drugs tend generally to be opposite in character to the "therapeutic" effects of these drugs. Withdrawal symptoms, therefore, may be sufficiently specific to be of diagnostic usefulness. Alcohol is a depressant; alcohol withdrawal is characterized by hyperactivity, tremors, nervousness, and often seizures. Withdrawal from stimulants is characterized by extreme depression. With opioid withdrawal, one sees a flu-like syndrome characterized by sweating, myalgia, fatigue, dysphoria and hyperalgesia.<sup>376</sup> Withdrawal from addicting substances may require treatment in and of itself. Diagnosing overeating, of course, is seldom difficult. Nor is there withdrawal from food in a medical sense. Eating does not produce the powerful neuroadaptive effects, including associated withdrawal effects, that are central to drug addiction. Labeling the perceived overconsumption of chocolate, for example, as "chocolate addiction," even if this practice is associated with high levels of comfort (emotional) eating and somewhat unstable eating patterns, risks trivializing serious addictions.<sup>377</sup>

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<sup>374</sup> See, G.T. Wilson, *The Addiction Model of Eating Disorders: A Critical Analysis*, 13 *ADV. BEHAV. RES. THER.* 27, 31 (1991).

<sup>375</sup> See, J. Field & K. O'Dea, *The Mechanism of Adaptive Hyperlipogenesis: Insulin Receptor Binding and Glucokinase Activity in Rat Liver During Fasting and Refeeding*, 29 (3) *METABOLISM* 296-301 (Mar. 1980).

<sup>376</sup> S.C. Woods, *supra*, note 210, at 498.

<sup>377</sup> P.J. Rogers & H.J. Smit, *supra* note 211, at 11.

Hence, the phenomena of tolerance and withdrawal, which so often occur with addiction, are difficult to apply to food consumption. We turn to a consideration of the reward-deficiency theory, sometimes invoked to explain the biochemical basis of addiction.

#### *D. Reward Deficiency Theory: The Reward System*

Under reward deficiency theory, snorting, shooting, or ingesting the drug of choice will eventuate in release of specific neurotransmitters, especially dopamine, at specified sites in the brain. Extending this idea to obesity, plaintiffs' argument may take this form: In modern Western societies, in which the food supply is abundant and overweight widespread, over activation of endogenous opioid peptides causes human obesity, binge eating and uncontrollable cravings for sweet and high-fat foods.<sup>378</sup> The extension of the concept of addiction as disease to eating disorders claims that some people are vulnerable to certain foods (e.g., sugar or white flour)<sup>379</sup> that can cause dependence; that the disorder is a progressive illness that can never be cured but only managed as a lifelong problem; that treatment must begin by interrupting the abuse of food (or, in the case of anorexics, the abuse of starvation); and that since the biochemical mechanisms of chemical dependence and eating disorders are similar, treatment for eating disorders should not differ fundamentally from that of alcohol or drug dependence. The recommended treatment, typically, is a 12-step program—Overeaters Anonymous (OA)—modeled after Alcoholics Anonymous (AA).<sup>380</sup>

Practical and ethical difficulties of conducting experimental work in humans with opioids and their antagonists limit the available human data.<sup>381</sup> At present, there appears to be no evidence that the overeater is an addict in the true sense of the word (i.e., that the over eater has long-term adaptive changes in dopaminergic transmission), as distinct from merely having a behavioral disturbance.

Proponents of reward-deficiency theory often therefore rely on data from experimental animals. Addiction, or what is interpreted as addiction, to many of the same compounds that "addict" humans may be induced in animals, and their behaviors can be studied as a model.<sup>382</sup> Such animals, of course, cannot tell us when they are experiencing pleasure, so data from such experiments must be interpreted cautiously. In addicted lab animals, dopamine release has been reported in specific sites in the brain when the animals are

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<sup>378</sup> See, M. Zhang, B.A. Gosnell, & A.E. Kelley, *Intake of High-Fat Food is Selectively Enhanced by Mu Opioid Receptor Stimulation Within the Nucleus Accumbens*, 285 J. PHARMACOL. EXPERIMENTAL THERAPEUTICS 908, 914 (1998). In humans, however, plasma beta-endorphin does not rise after a palatable meal. J.C. Melchior, et al., *Lack of Plasmatic Beta-Endorphin Response to a Gastronomic Meal in Healthy Humans*, 59 PHYSIOLOGY AND BEHAVIOR 1217-1221 (1991).

<sup>379</sup> As noted, the Pelman plaintiffs failed to achieve this level of specificity in their pleadings. Plaintiffs who endeavor to identify a specific ingredient as "addicting," however, face a dilemma: That ingredient is apt to be present in a great many kinds of food. Sugar, e.g., is called for in an enormous number of recipes. Moreover, glucose is of course present in every drop of blood in every living human, and it is conceptually confounding to think of any molecule that is a normal, and in fact, indispensable compound in normal tissue as an "addicting" substance.

<sup>380</sup> Wilson, *supra* note 374, citing J. Yearly, *The Use of Overeaters Anonymous in the Treatment of Eating Disorders*, 19 J. PSYCHOACTIVE DRUGS 303-309 (1987). See also, D.W. Swanson & F.A. Dinello, *Severe Obesity as a Habituation Syndrome: Evidence During a Starvation Study*, 22 ARCH. GEN. PSYCHIAT. 120, 120-121 (1970). Obesity treatment programs with the best outcomes, however, tend to be derived from behaviorism theory, a model incompatible with addiction theory. R. Stein, et al., *Etiology and Treatment of Obesity in Adults and Children: Implications for the Addiction Model*, 15 DRUGS & SOCIETY 103-121 (1999) at 116.

<sup>381</sup> M.R. Yeomans & R.W. Gray, *Opioid Peptides and the Control of Human Ingestive Behaviour*, 26 NEUROSCI. & BIOBEHAV. REV. 714 (2002).

<sup>382</sup> See, e.g., G.F. Koob & F.E. Bloom, *supra* note 359.

provided drugs. In the case of food, advocates argue, overeaters seek and obtain the same reward in a fashion that overcomes the checks and balances that might otherwise rein in consumption. Food is said to cause an increase in extracellular dopamine just as, say, cocaine does.<sup>383</sup> Rats given free access to abundant bread and chocolate, for example, reportedly gained weight, and did not curtail consumption when they had met their energy requirements.<sup>384</sup> Rats denied sugar to which they had grown accustomed are said to exhibit behaviors that have been interpreted as consistent with withdrawal.<sup>385</sup> Some investigators have reported that the same neurotransmitters that seem to be implicated in drug addiction are released from the same regions of the brain in experimental animals provided presumably palatable foods.<sup>386</sup> Some have argued that food may compete with drugs of abuse for reward sites in brain, so excessive consumption may be inversely related to drug addiction.<sup>387</sup> Taber and Fibiger and others have suggested that rewarding stimuli such as food activate the dopamine system.<sup>388</sup> Dopamine does not appear to be a reward signal itself, but it may be necessary for acquisition of reinforcing stimuli. Like serotonin, dopamine is itself an appetite suppressant.<sup>389</sup>

### 1. *Brain Structure Relevant to the Reward System*

On the basis of work in rats done originally in the mid-1950s by James Olds at McGill,<sup>390</sup> it was theorized that many of the potent motivational, and hence addictive properties of addicting substances were mediated by a common neurochemical action that played upon a relationship between neurotransmitters and their receptors in brain.

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<sup>383</sup> See, e.g., M. Gold, et al., *Overeating, Binge Eating and Eating Disorders as Addictions*, 33 (2) PSYCHIATRIC ANN. 117, 118 (2003), citing L. Hernandez & B. Hoebel, *Feeding and Hypothalamic Stimulation Increase Dopamine Turnover in the Accumbens*, 44 PHYSIOL. BEHAV. 599-606 (1988); L. Hernandez & B.G. Hoebel, *Feeding Can Enhance Dopamine Turnover in the Prefrontal Cortex*, 25 BRAIN. RES. BULL. 975-979 (1990); G.J. Mogenson, "Studies of the Nucleus Accumbens and its Mesolimbic Dopaminergic Affects in Relation to Ingestive Behaviors and Reward," In: G.B. Hoebel & D. Noven, *The Neural Basis of Feeding and Reward*, Haer Institute, Brunswick, Maine, (1982) at 275-506; F.S. Radhakishun, et al., *Scheduled Eating Increases Dopamine Release in the Nucleus Accumbens of Food-Deprived Rats as Assessed With On-line Brain Dialysis*, 85 NEUROSCI. LETT. 351-356 (1988); M. Yoshida, et al., *Eating and Drinking Cause Increased Dopamine Release in the Nucleus Accumbens and Ventral Tegmental Area in the Rat: Measurement by in vivo Microdialysis*, 139 NEUROSCI. LETT. 73-76 (1992); A.M. Young, et al., *Increased Dopamine Release in vivo in Nucleus Accumbens and Caudate Nucleus of the Rat During Drinking: A Microdialysis Study*, 48 NEUROSCI. 871-876 (1992); N.S. Miller & M.S. Gold, *A Hypothesis for a Common Neurochemical Basis for Alcohol and Drug Disorders*, 16 PSYCHIATR. CLIN. NORTH AM. 105-117 (1993).

<sup>384</sup> P.J. Rogers & J.E. Blundell, *Investigation of Food Selection and Meal Parameters During the Development of Dietary Induced Obesity*, 1 APPETITE 85 (1980).

<sup>385</sup> See, C. Colantuoni, et al., *Evidence That Intermittent, Excessive Sugar Intake Causes Endogenous Opioid Dependence*, 10 OBESITY RES. 478 (2002). See also, K. Goodrich, *supra* note 356 at 131-132 (in rats trained to binge on glucose, and then deprived of food for one or two days, teeth chattering and paw fanning reported.).

<sup>386</sup> See, e.g., N.M. Avena & B.G. Hoebel, *A Diet Promoting Sugar Dependency Causes Behavioral Cross-Sensitization to a Low Dose of Amphetamine*, 122 NeuroSci.17 (2003).

<sup>387</sup> K.D. Kleiner, et al., *Body Mass Index and Alcohol Use*, 23 J. ADDICTIVE DISORDERS 105-118 (2004).

<sup>388</sup> M.T. Taber & H.C. Fibiger, *Feeding-evoked Dopamine Release in the Nucleus Accumbens, Regulation by Glutamatergic Mechanisms*, 76 NEUROSCI.1105-1112 (1997).

<sup>389</sup> J. Blundell, *Pharmacologic Approaches to Appetite Suppression*, 12 TRENDS PHYSIOL. SCI. 147-157 (1991).

<sup>390</sup> J. Olds, *Pleasure Center in the Brain*, 195 SCI. AM. 105-116 (1956); J. Olds, *Hypothalamic Substrates of Reward*, 42 PHYSIOL. REV. 554-604 (1962). See also, B.G. Hoebel & P. Teitelbaum, *Hypothalamic Control of Feeding and Self-Stimulation*, 135 SCI. 375-377 (1962); D.L. Margules & J. Olds, *Identical "Feeding" and "Rewarding" Systems in the Lateral Hypothalamus of Rats*, 135 SCI. 374-375 (1962).

In this model, addicting drugs enhance brain reward circuits neuropharmacologically by stimulating neuronal firing or neurotransmitter release in brain reward loci.<sup>391</sup>

The brain reward cascade is a theory proposed by addictive behavior researcher Kenneth Blum.<sup>392</sup> He posits a brain reward cascade pathway composed of both central nervous system structures and endogenous neurotransmitters communicating between these structures. The theory is that the brain reward pathway evolved to promote activities essential to the survival of humans as well as of other mammals.

In general, the reward cascade is posited to involve a deficiency of the neurotransmitter, dopamine, in the limbic system of the brain.<sup>393</sup> The limbic system consists of a group of brain structures such as the hippocampus, amygdala and their interconnections and connections with the hypothalamus, septal area, and portions of the ventral tegmentum. It is thought to be involved in monitoring internal homeostasis, memory, learning, emotion and feeding behaviors. The limbic striatum is also important in the function of the limbic system and includes the nucleus accumbens (NA), the ventral caudate nucleus and the putamen.

The median forebrain bundle, NA, and ventral tegmental area are among the most highly studied structures in the brain reward cascade. The median forebrain bundle, composed of dopaminergic neurons, is sometimes described as a “power line” of neurons that connects the structures of the reward pathway with other brain structures.<sup>394</sup> Animal studies provide some support for the conclusion that the median forebrain bundle is necessary for the proper function of the brain reward cascade.<sup>395</sup> For example, animal studies have suggested that when the “power line” is severed, animals will decrease or stop self-administration of drug.<sup>396</sup>

The NA has long been considered an essential interface between “motivation and action.” It appears to play a role in behaviors related to natural reinforcers, such as ingestion, sexual behavior, incentive and instrumental learning. It is rich in dopaminergic neurons arising from the ventral tegmental area, and is postulated to be a critical substrate for the rewarding and reinforcing properties of addictive drugs.<sup>397</sup>

The ventral tegmental area (VTA) is part of the midbrain. It too is rich in dopamine and serotonin neurons and plays a role in several dopamine pathways. The VTA is thought to be one of the major sources of incentive and motivation, and an important structure in the brain reward cascade. Activities that produce pleasure tend to activate the VTA. Drugs such as cocaine are posited to target this area directly.

## 2. Neurotransmitters

In general, four neurotransmitters are thought to play a significant role in the reward cascade: dopamine in the NA and the hippocampus, serotonin in the hypothalamus and

<sup>391</sup> E.L. Gardner, “The Neurobiology and Genetics of Addiction: Implications of the ‘Reward Deficiency Syndrome’ for Therapeutic Strategies in Chemical Dependency,” in J. Elster, ed., *Addiction: Entries and Exits*, RUSSEL SAGE FOUNDATION: NY (1999) at 59.

<sup>392</sup> See, e.g., K. Blum, et al., *Reward Deficiency Syndrome: A Biogenic Model for the Diagnosis and Treatment of Impulsive, Addictive, and Compulsive Behaviors*, 32 (Suppl. iv) J. PSYCHOACTIVE DRUGS 1-112 (Nov. 2000).

<sup>393</sup> J. Lowinson, P. Ruiz, R. Millman, & J. Langrod, *Substance Abuse: A Comprehensive Textbook*, 3d Ed., WILLIAMS & WILKENS (1997).

<sup>394</sup> See, e.g., *Neuroanatomy and physiology of the ‘brain reward system’ in substance abuse*, Center for the Genetics of Anti-Social Drug Dependence, available at [http://ibgwww.colorado.edu/cadd/a\\_drug/essays/essay4.htm](http://ibgwww.colorado.edu/cadd/a_drug/essays/essay4.htm), (last visited June 26, 2006).

<sup>395</sup> M. Olds & J. Olds, *Effects of Lesions in Median Forebrain Bundle of Self-Stimulation Behavior*, 217 AM. J. PHYSIOL. 1253-1264 (1969).

<sup>396</sup> J. Lowinson, P. Ruiz, R. Millman, & J. Langrod, *supra* note 393.

<sup>397</sup> A.E. Kelley, *Ventral Striatal Control of Appetitive Motivation: Role in Ingestive Behavior and Reward-Related Learning*, 27 NEUROSCI. BIOBEHAV. REV. 765, 766 (2004).

gamma aminobutyric acid (GABA), the endogenous opioid compounds (enkephalins) in the VTA and the NA.

### a. Dopamine

Dopamine has been singled out as the primary neurotransmitter of reward. As discussed above, the median forebrain bundle “power line” of the brain reward cascade is primarily made up of dopaminergic neurons, which run from the VTA to other structures involved in the brain reward cascade and release dopamine. Thus, dopamine can be thought of as the “current” or “energy” of the brain reward system.

Dopamine is said to play an important role in the control of movement, cognition, motivation, and reward.<sup>398</sup> Higher dopamine levels can cause feelings of wellbeing or euphoria, but also psychosis. In general, drugs of abuse are thought to be associated with increased dopamine neurotransmitter levels in the brain reward cascade. In contrast, drugs that are not abused are not thought to increase dopamine concentration.

### b. Other Neurotransmitters

Three additional neurotransmitters are thought to be important in the reward cascade: serotonin in hypothalamus, and GABA and the endogenous opioid compounds (enkephalins) in the VTA and the NA. Serotonin is involved in the modulation of both drug self-administration and dopamine levels. Interactions between serotonin and dopamine release are extremely complex. GABA is an inhibitory neurotransmitter located diffusely throughout the brain. The endogenous opioid compounds (enkephalins) or endorphins are considered to be capable of motivating behavior. For example, the “runner’s high” is thought to be related to the production of such endogenous opioid compounds. They are believed to act on the same receptors as exogenous opioids, such as heroin. Through the same mechanism, they are both thought to increase dopamine concentration in the brain reward cascade. We anticipate that plaintiffs’ counsel may claim that the pleasure response to foods may be mediated by endogenous opioid compounds. Some have suggested that the opioid system may be linked to binge eating<sup>399</sup> episodes reported by obese and bulimic<sup>400</sup> women.

## 3. The Reward Cascade and Reward Deficiency Syndrome

Under the theory, all of these neurotransmitters (dopamine, serotonin, GABA and the endogenous opioids) work together in normal humans in a cascade of excitation or inhibition between complex stimuli and complex responses. These interactions (“cascade”) are said to be associated with feelings of well-being.

The so-called “reward deficiency syndrome” is said to result, generally speaking, when there is a dysfunction in the brain reward cascade. Such dysfunction has been reported to engender feelings of anxiety, anger and other negative feelings or a craving for a substance

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<sup>398</sup> K. Blum, J. Cull, & E. Braveman, *Reward Deficiency Syndrome*, 84 AMER. SCIENTIST 132-145 (Mar.-Apr. 1996). It also has numerous cardiovascular, renal, and hormonal effects. M. Velasco & A. Luchsinger, *Dopamine: Pharmacologic and Therapeutic Aspects*, 5 (1) AM. J. THER. 37-43 (Jan. 1998). See also, nn. 413-415 *infra*, and associated text.

<sup>399</sup> A.J. Stunkard, *Eating Patterns and Obesity*, 33 PSYCHIAT. QUART. 284-295 (Apr. 1959) described a “binge eating syndrome” in approximately 5% of obese persons. The authors described a sudden, compulsive, large ingestion of food in reaction to stress such as a frustrating experience. The binge is associated with self-condemnation, drastic dieting, and recurrent eating binges.

<sup>400</sup> Bulimia is an eating disorder characterized by the rapid consumption of large amounts of food in a relatively short period of time (typically less than two hours). P.S. Butterfield & S. LeClair, *Cognitive Characteristics of Bulimic and Drug-Abusing Women*, 13 ADDICTIVE BEHAVIORS 131 (1988).

that will alleviate these emotions. Since dopamine is deemed to be the most important neurotransmitter in the neurological expression of pleasure, a defect in this neurotransmitter is considered the main culprit in reward deficiency syndrome. Animal studies have suggested that dopamine agonists can decrease the consumption or self-administration of various addictive drugs or reduce drug seeking.<sup>401</sup> The results of studies in humans of the efficacy of dopamine agonists in the treatment of drug addiction and symptoms of drug withdrawal have been mixed, however, with many negative reports.

Proponents of these theories argue that carriers of the A1 allele of the dopamine D2 receptor gene may depend on dopamine-releasing substances such as marijuana, alcohol, and chocolate, for example.<sup>402</sup> Among alcoholics, cocaine addicts, compulsive gamblers, compulsive eaters, and ADHD patients, the prevalence of the A1 D2 receptor allele is said to be higher than that seen in the general population.<sup>403</sup> There are reports that obese individuals may often be A1 allele carriers—having lower levels of the D2 receptors when compared with non-obese individuals.<sup>404</sup>

It has been suggested that an individual with the A1 allele for the D2 receptor has a 74% chance of developing one of the disorders that some have categorized under reward deficiency syndrome.<sup>405</sup> In sum, the reward deficiency syndrome theory postulates that addictive, impulsive, and compulsive disorders may have a common genetic basis – dopamine hypoactivity in reward pathways. Based on this theory, one might anticipate that dopamine substitution (direct or indirect) might decrease drug taking or craving, perhaps only (or more markedly) in genetically vulnerable subjects (either animal or human).<sup>406</sup>

### E. *Limitations of Animal Models*

The obesity that the plaintiffs' bar wants to turn into "the next tobacco" is obesity not in experimental animals, but in humans. For better and for worse, humans are characterized, even defined, by their free will.<sup>407</sup> Lower animals, by definition, lack that capacity:

Unlike animals, which mostly behave like simple reinforcement machines, humans can make choices on the basis of long-term consequences of present behavior. Unlike animals, which do not seem capable of introspection, humans can have awareness of their addiction, deplore it, and fight it. Unlike animals, which lack beliefs and values of the requisite complexity, humans are embedded in a culture that shapes cognition and motivation in ways that matter for drug consumption.<sup>408</sup>

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<sup>401</sup> W. Dyr, et al., *Effects of D1 and D2 Dopamine Receptor Agents of Ethanol Consumption in the High-Alcohol Drinking (HAD) Line of Rats*, 10 ALCOHOL 10:207-212 (1993).

<sup>402</sup> G.F. Koob & F.E. Bloom, *supra* note 359.

<sup>403</sup> *But see*, J. Gelertner, D. Goldman, & N. Risch, *The A1 Allele at the D2 Dopamine Receptor Gene and Alcoholism: A Reappraisal*, 269 (13) JAMA 1673-1677 (7 Apr. 1993).

<sup>404</sup> *See, e.g.*, K. Blum, *Increased Prevalence of the Tag A1 Allele of the Dopamine Receptor Gene (DRD2) on Obesity With Comorbid Substances Use Disorder: A Preliminary Report*, 6 (4) PHARMACOGENETICS 297-305 (Aug. 1996).

<sup>405</sup> *Id.*

<sup>406</sup> E.L. Gardner, "The Neurobiology and Genetics of Addiction: Implications of the Reward Deficiency Syndrome for the Therapeutic Strategies in Chemical Dependency," in J. Elster, ed., *Addiction: Entries & Exits*, RUSSELL SAGE FOUNDATION (1999) at 80.

<sup>407</sup> World religions generally recognize the concept of free will. *See* D.J. LaFetre, *Freedom, Responsibility, and Risk: Fundamental Principles Supporting Tort Reform*, 36 IND. L. REV. 645-685 (2003) at 657, n. 75.

<sup>408</sup> *Relapse Prevention: Maintenance, Strategies and the Treatment of Addictive Behaviors*, G. Alan Marlatt & Judith R. Gordon, eds., NY: GUILDFORD (1985), Introduction at x.

Animal experimentation is clearly useful, even indispensable, in many forms of biologic investigation. In studying higher-level human behavior, however, the limitations of such experiments are at their most severe. In the study of alcohol addiction, for example, experiments with rats have clear limitations. By manipulating reinforcement schedules, rats can be induced to ingest quantities of ethanol that result in physical dependence, but on termination of the schedule they will not sustain such ingestion levels.<sup>409</sup>

Addiction is sometimes compared to a form of slavery. To the extent that metaphor is accurate, it tends to detract from the validity of animal models. An animal cannot be a slave, because an animal cannot, in the human sense, be free. Less philosophically, and more practically, there is no experimental model to duplicate or even simulate the vagaries of human will. Moreover, the biological need to eat is not so compelling as to negate the power of will. On the contrary, humans plainly malnourished may refuse to eat; humans overfed may continue to consume; bulimics may control their bingeing. As we have seen, energy intake is subject to an elaborate system of checks and balances. Young children, presumably ignorant of nutrition science, generally tend to keep energy intake fairly stable.<sup>410</sup> There is evidence, in fact, that the optimal environment for children's development of self-control of energy intake may be one in which the parents provide healthful food choices but allow children to assume control of how much they consume.<sup>411</sup> Yet older children, and adults, may choose to eat more or less than they should. Such behaviors are uniquely human, and in such particulars animal behavior cannot mimic ours.<sup>412</sup>

The experimental method works because the observer changes only one variable at a time and notes what occurs. If a single change has more than one effect, however, interpretation of ensuing observations is difficult. One may say that an animal given a dopamine blocker will not eat, because the blocker removes the neurochemical rewards of eating. But dopamine has many functions. It raises blood pressure, increases cardiac output, dilates the renal arteries, and increases urine output, characteristics that make it useful in the management of some cases of shock.<sup>413</sup> On the other hand, it suppresses ventilation,<sup>414</sup> and influences motor function.<sup>415</sup> Does the experimental animal given dopamine stop eating because it cannot move towards available food?<sup>416</sup> Similarly,

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<sup>409</sup> V.P. Dole, *On the Revelance of Animal Models to Alcoholism in Humans*, 10 ALCOHOL CLIN. EXP. RES. 361 (1986); see also HHS Publ. 87-1519, 6th Special Report to Congress on Alcohol and Health (1987), cited in G.F. Koob, & F.E. Bloom, *supra* note 359.

<sup>410</sup> M. Rosenbaum & R.L. Leibel, *supra* note 329.

<sup>411</sup> S.L. Johnson & L.L. Birch, *Parents' and Children's Adiposity and Eating Style*, 94 (5) PEDIATRICS 653-661 (1994).

<sup>412</sup> Taste alone does not control human food consumption. Eating habits are also influenced by perceived nutrition, product safety, price, convenience, and prestige. M. Kronld, et al., *Food Use and Perceived Food Meanings of the Elderly*, 80 J. AM. DIET. ASSOC. 523-529 (1982); D. Sanjur, *Social and Cultural Perspectives in Nutrition*, Englewood Cliffs, N.J.: PRENTICE-HALL (1982) at 336, cited in A. Drewnowski, *Taste Preferences and Food Intake*, 17 ANNU. REV. NUTR. 241 (1997). See also, discussion of metabolic/hormonal factors, *supra*, notes 213-266 and associated text, and discussion of cultural and related factors, *supra*, notes 293-306 and associated text.

<sup>413</sup> See Velasco and Luchsinger, *supra* note 398. See also, T.W. Robbins & B. Everitt, *Functions of Dopamine in the Dorsal and Ventral Striatum*, 4 SEM. NEUROSCI. 119-127 (1992); R.A. Felder, et al., *The Dopamine Receptor in Adult and Maturing Kidney*, 257 (3) AM. J. PHYSIOL. RENAL PHYSIOL. 315-F327 (1989).

<sup>414</sup> M.J. Welsh, et al., *Depression of Ventilation by Dopamine in Man*, 61 (3) J. CLIN. INVEST. 708-713 (1978).

<sup>415</sup> Dopamine is present in high concentrations in the corpus striatum, which participates in motor control. Deficiencies of dopamine there and elsewhere are thought to account for many of the signs of Parkinson's disease. See, National Institute of Neurological Disorders and Stroke, NIH, *Parkinson's disease: Hope through research*, available at [http://www.ninds.nih.gov/disorders/parkinsons\\_disease/detail\\_parkinsons\\_disease.htm](http://www.ninds.nih.gov/disorders/parkinsons_disease/detail_parkinsons_disease.htm), (last visited July 12, 2006).

<sup>416</sup> See, e.g., Y.S. Eyny & J.C. Horvitz, *Opposing Roles of D1 and D2 Receptors in Appetitive Conditioning*, 23 (5) J. NEUROSCI. 11584-11587 (Mar. 1, 2003).

some point to animal studies suggesting that certain animals given opioid blockers will not feed. They argue that food is rewarding in the same way opioids are, so blocking opioids blocks the rewards of food. But opioid-blocking drugs also induce nausea.<sup>417</sup> Does the animal stop eating because we have made it too sick to do so?

Then, too, there are inconsistencies in animal data that are difficult to explain. Homeostasis, the organism's tendency to maintain constant Claude Bernard's "internal milieu,"<sup>418</sup> is a hallmark of all life. If we consume too much salt, for instance, our kidneys will tend to increase their excretion of the excess so that our sodium levels rise little, if at all, and then only transiently. Against this fundamental homeostatic principle, the experimental data underpinning the reward deficiency theory are internally inconsistent. For example, consider dopamine levels in addicted animals immediately before self-administration of the drug of choice. Reward deficiency theory might plausibly anticipate that dopamine levels would be low at such times. Under the theory, a trough would be associated with decreased dopamine effect, motivating the animal to rectify the deficiency by consuming a drug and increasing dopamine back towards the "desired," "rewarding" level. Some experiments, in fact, appear to have shown just that. In other studies, however, sometimes from the very same labs, pre-dose dopamine levels are actually at their peaks, as if dopamine were itself a stimulus motivating the animal to seek out its drug of choice. For example, in heroin and cocaine self-administration experiments in lab animals, NA dopamine levels actually rose before each self-administration of drug and fell immediately after receipt of the self-administered intravenous heroin or cocaine dose.<sup>419</sup> Others have reported no elevations in NA dopamine during self-administered rewarding electrical brain stimulation in laboratory rats.<sup>420</sup> In other work, NA dopamine was said to be elevated during rewarding electrical brain stimulation that animals administered to themselves, but it disappeared after several self-stimulations, nor did it reappear for the duration of the testing session even though the animals continued self-delivering the stimuli.<sup>421</sup>

There is another problem that reward-deficiency theorists must grapple with: The two most robust stimuli for triggering reinforcement of drug self-administration in animals are stress or a priming drug dose, which tend to increase rather than decrease dopamine function within the brain's reward circuitry.<sup>422</sup> Again, one might think that

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<sup>417</sup> See, M. Bertino, et al., *Naltrexone, An Opioid Blocker, Alters Taste Perception and Nutrient Intake in Humans*, 261 AM. J. PHYSIOL. R59-R63 (1991); M.R. Yeomans & R.W. Gray, R.W., *Selective Effects of Naltrexone on Food Pleasantry and Intake*, 60 PHYSIOL. BEHAV. 439-446 (1996). Nausea may be a significant side effect of naltrexone when its use is attempted as a treatment for alcohol dependence. C. Oncken, et al., *Adverse Effects of Oral Naltrexone: Analysis of Data From Two Clinical Trials*, 54 PSYCHOPHARMACOLOGY 397-402 (2001). The appetite-suppressing effects of PYY-3-36 may be attributable in part to nausea induced by its administration. I.G. Halatchev & R.D. Cone, *Peripheral Administration of PYY-3-36 Produces Conditioned Taste Aversion in Mice*, 1 CELL METAB. 159 (Mar. 2005).

<sup>418</sup> See, e.g., Bernard's opening lecture in general physiology given to the College de France, as quoted in S.G. Schultz, *The Internal Environment*, in ESSENTIAL MEDICAL PHYSIOLOGY, 2d ed., L.R. Johnson, ed., NY: RAVEN PRESS (1998).

<sup>419</sup> A. Gratton & R.A. Wise, *Drug and Behavior-Associated Changes in Dopamine-Related Electrochemical Signals During Intravenous Cocaine Self-Administration in Rats*, 14 J. NEUROSCI. 4130-4146 (1994); A. Gratton, *In vivo Analysis of the Role of Dopamine in Stimulant and Opioid Self-Administration*, 21 J. PSYCHIAT. & NEUROSCI. 264-279 (1996).

<sup>420</sup> Z.L. Kruk, et al., *Real-time Measurement of Stimulated Dopamine Release in the Conscious Rat Using Fast Cyclic Voltammetry: Dopamine Release is not Observed During Intracranial Self-Administration*, 79 J. NEUROSCI. METH. 9-19 (1998).

<sup>421</sup> R.M. Wightman & P.A. Garriss, *Dopamine Release and Intracranial Self-Stimulation*, 22 Soc. NEUROSCI. AB. 832 (1996).

<sup>422</sup> E.L. Gardner, *Brain Reward Mechanisms*, in *Substance Abuse: A Comprehensive Text Book*, 3d Ed., J.H. Lowinson, P. Ruiz, R.B. Millman, & J.G. Langrod, eds., Baltimore: WILLIAMS & WILKINS (1997). Yet stress has also been reported to reinstate appetitive behavior to obtain cocaine but not food. S.H. Ahmed &

factors increasing dopamine in relevant brain structures would “cure” any “reward deficiency,” and discourage, rather than encourage, behaviors thought to raise dopamine levels yet further.

Reward deficiency theory proceeds from the notion that an animal will engage in behaviors that result in elevated levels of dopamine in the NA. Curiously, however, elevation of dopamine in the NA is also triggered by stress, presumably aversive to the animal as it would be to humans.<sup>423</sup>

It is possible to alter the genetic makeup of lab animals so that they lack receptors or transporters for various drugs and hormones. To our current understanding, this should mean that such drugs and hormones have no mechanism to cause any effects in these genetically modified animals. In the case of cocaine self-administration, however, scientists have observed that drug-seeking behaviors persist in addicted dopamine transporter knock-out mice—that is, mice that have been genetically altered to be unable to transport cocaine to the cells where it has its effects.<sup>424</sup> Under reward-deficiency theory, one would think that an animal rendered incapable of transporting the addicting drug, and hence incapable of feeling its effects, would no longer engage in drug-seeking activities. Such, however, is not the case. Conditioned cue (place) preference to psychostimulants can be readily established in dopamine transporter knockout mice.<sup>425</sup> Similarly, animals addicted to heroin continue to use it even after dopamine is rendered unable to have any effect.<sup>426</sup> 6-hydroxydopamine lesions in the NA, which specifically destroy dopaminergic neurons, do not affect lever press linked to opioid reward.<sup>427</sup> Mice provided with both sucrose and water demonstrated significant preference for sucrose, regardless whether they were dopamine-deficient.<sup>428</sup>

Self-administration of cocaine, but not food, results in morphological changes in dendrites and dendritic spines in the prefrontal cortex and NA. In structural volumetric studies, volume losses in the prefrontal cortex have been reported in heroin, alcohol and cocaine-dependent substance abusers. Thus, dopamine and related mechanisms in the addiction process appear to cause changes in the prefrontal cortex.<sup>429</sup> These phenomena need to be explained before the reward-deficiency theory can be accepted even in drugs in lab animals.

Even if there were no inconsistencies in observations such as these, we must remain mindful, particularly in a litigation context, of the distinction between correlation and

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G.F. Koob, *Cocaine- but not Food-seeking Behavior is Reinstated by Stress After Extinction*, 132 *PSYCHOPHARMACOL.* 289-95 (1997).

<sup>423</sup> P.W. Kalivas & P. Duffy, *Time Course of Extracellular Dopamine and Behavioral Sensitization of Cocaine: II. Dopamine Perikarya*, 13 *J. NEUROSCI.* 266-275 (1993); J.W. Tidey & K.A. Miczek, *Acquisition of Cocaine Self-Administration After Social Stress: Role of Accumbens Dopamine*, 130 *PSYCHOPHARMACOL.* 203-212 (1997). See also, C.M. Cannon & M.R. Bseikri, *Is Dopamine Required for Natural Reward?*, 81 *PHYSIOL. BEHAV.* 741 (2004) (in mice, dopamine is required neither for an hedonic response to sweets nor for their discrimination.).

<sup>424</sup> B.A. Rocha, et al., *Cocaine Self-Administration in Dopamine Transporter Knock-out Mice*, 1 *NAT. NEUROSCI.* 132-137 (1998).

<sup>425</sup> Sora, et al., *Cocaine Reward Models: Conditioned Place Preference can be Established in Dopamine- and in Serotonin-Transporter Knockout Mice*, 95 (13) *PROC. NAT'L ACAD. SCIENCES USA* 7699-7704 (1998). See, [http://www.drugabuse.gov/NIDA\\_Notes/NNVol14N2/Cocaine.html](http://www.drugabuse.gov/NIDA_Notes/NNVol14N2/Cocaine.html), (last visited July 7, 2006).

<sup>426</sup> G.F. Koob & F.E. Bloom, *supra* note 359.

<sup>427</sup> T.W. Robbins, M. Cador, J.R. Taylor & B.J. Everitt, *Limbic-Striatal Interactions in Reward-Related Processes*, 13 *NEUROSCI. BIOBEHAV. REV.* 155-162 (1989).

<sup>428</sup> C.M. Cannon & R.D. Palmiter, *Reward Without Dopamine*, 23 (34) *J. NEUROSCI.* 10827-10831 (2003).

<sup>429</sup> Ali Saffet Gönül, *Neurobiological Basis of Drug Addiction (2): Impaired Response Inhibition and Salience Attribution*, 42 (3) *Yeni Symposium* 139-140 (2004), available at [http://www.yenisymposium.net/fulltext/2004\(3\)/ys2004\\_42\\_3\\_8.pdf](http://www.yenisymposium.net/fulltext/2004(3)/ys2004_42_3_8.pdf) (last visited July 7, 2006).

causation. The evidence implicating tobacco use in certain forms of lung cancer, for example, is so well-established as to be virtually non-debatable, and causation may be taken as established. The addiction theory of obesity, in contrast, is at best a hypothesis.

#### F. *The Limitations of Dopamine as a Common Denominator*

Weaknesses in the theory include problems with its heavy emphasis on dopamine. Dopamine is released by multiple factors; once it is released there are several mechanisms that modulate its effect at the site of action.<sup>430</sup> It is rapidly removed via dopamine transporter.<sup>431</sup> Heteroreceptors (receptors regulating the synthesis and/or the release of mediators other than their own ligands) may also play a role in modulating dopamine release.<sup>432</sup> Near-total depletion of dopamine in the NA does not reduce positive affective reactions.<sup>433</sup> Natural rewards and cocaine activate different subcircuits within the nucleus accumbens.<sup>434</sup>

Under the reward deficiency theory one of the main areas of brain where dopamine is thought to exert its effects is the orbitofrontal cortex. No studies have shown abnormalities in the orbitofrontal cortex in obese humans.<sup>435</sup> In short, the linchpin of the reward-deficiency theory may not be completely secure.

#### G. *The Limitations of Reward-Deficiency Theory as Applied to Humans*

The problems with applying the reward-deficiency theory to obesity are not limited to limitations and inconsistencies peculiar to animal experimentation, nor to the difficulties in invoking deficiencies in a single neurotransmitter to explain a wide array of behaviors. Human medicine creates another set of problems for the theory.

Classically, the behavior of addicts is compulsive. In the 19<sup>th</sup> century, Benjamin Rush described an alcoholic: "When strongly urged, by one of his friends, to leave off drinking, he said, 'Were a keg of rum in one corner of the room, and were a cannon constantly discharging balls between me and it, I could not refrain from passing before that cannon, in order to get the rum.'"<sup>436</sup> Even allowing for a certain amount of hyperbole in this description, there is no comparable craving among lovers of foods.

True addicts often engage in impulsive behaviors that acutely endanger both themselves and others. The obese do not. When a true addict overdoses, as so often happens, the risks include sudden death, his own or others', as from car crashes, falls, or other trauma. Overdosing on nicotine, while admittedly much more common with nicotine patches than with smoking, can cause seizures, coma, respiratory depression, apnea,

<sup>430</sup> K.A. Keefe, et al., *Extracellular Dopamine in the Striatum: Influence of Nerve Impulse Activity in the Medial Forebrain Bundle and Local Glutamatergic Input*, 47 NEUROSCI. 325-332 (1992).

<sup>431</sup> B. Giros, et al., *Hyperlocomotion and Indifference to Cocaine and Amphetamine in Mice Lacking the Dopamine Transporter*, 379 NATURE 606-612 (1996).

<sup>432</sup> H. Moore, et al., *The Regulation of Forebrain Dopamine Transmission: Relevance to the Pathophysiology and Psychopathology of Schizophrenia*, 46 BIOL. PSYCHIATRY 40-55 (1999).

<sup>433</sup> K.C. Berridge & T.E. Robinson, *What is the Role of Dopamine in Reward: Hedonic Impact, Reward Learning, or Incentive Salience?*, 28 (3) BRAIN RES. REV. 209-269 (1998) cited in C.M. Cannon & M.R. Bseikri, *supra* note 423.

<sup>434</sup> R.M. Carelli, et al., *Evidence That Separate Neural Circuits in the Nucleus Accumbens Encode Cocaine Versus "Natural" (Water and Food) Reward*, 20 (11) J. NEUROSCI. 4255-4266 (2000).

<sup>435</sup> G.-J. Wang, et al., *Exposure to Appetitive Food Stimuli Markedly Activates the Human Brain*, 21 (4) NEUROIMAGE 1790, 1796 (2004).

<sup>436</sup> Cited in H.G. Levine, *The Discovery of Addiction: Changing Conceptions of Habitual Drunkenness in America*, 15 J. STUDIES ON ALCOHOL 493-506 (1979), available at <http://www.aabibliography.com/habitualdrunkenness.htm>, (last visited July 7, 2006).

arrhythmias<sup>437</sup> and death.<sup>438</sup> Parents are advised to store transdermal nicotine patches in locked cabinets out of the reach of toddlers.<sup>439</sup> In less severe cases of nicotine toxicity, gastrointestinal distress, pallor, weakness, dizziness and rash may be seen.<sup>440</sup> Acknowledging that smokers behave differently, if one addicted to other addiction-inducing compounds does not overdose, and uses only his accustomed, “therapeutic” dose, his judgment is impaired, his reflexes may be slowed (or exaggerated), and his capacity for self-care is often diminished. All of these phenomena create risks the obese do not face.

The behavior of a true addict and the behavior of an overeater are too different for overeating to be properly classified as an addiction. Consider, for example, the behavior of bulimics. Advocates of obesity litigation may claim that bulimics provide a good example of the validity of their position.<sup>441</sup> There have been reports, for example, that bulimics crave sweets.<sup>442</sup> Some argue that self-starvation is a form of chemical dependence—an addiction to beta-endorphins.<sup>443</sup> In the so-called “binge-and-purge” cycle, however, these unfortunate individuals overeat, then induce vomiting, diarrhea, or both. Unless they are trying to quit, true addicts never attempt to “purge” their drugs of choice. On the contrary, they devote their lives to finding and using their drugs.

Bulimics binge and purge in secret only.<sup>444</sup> Addicts abuse whether observed or not. A true addict, e.g. one addicted to crack cocaine, is not simultaneously addicted to its absence. A bulimic anorexic, however, is “addicted” both to a substance (food) and to its absence.<sup>445</sup> Treatment of addiction depends upon abstinence. The management of bulimia, however, does not entail starvation, or even fasting. Instead, it depends upon rigidly scheduled, regimented meals, without snacks in between. Specific foods are not avoided.<sup>446</sup> In contrast, an addict seeking true recovery must learn to avoid his drug of choice entirely.

In genuine addiction, spontaneous remissions, though unusual, do occur. The addict screws his courage to the sticking post and resolves to abstain. Many fail, but some

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<sup>437</sup> T. Zimmers, *Cases in Electrocardiography*, 12 (3) AM. J. EMERG. MED. 374, 375 (May 1994).

<sup>438</sup> K. Saxena & A. Scheman, *A Suicide Plan by Nicotine Poisoning: A Review of Nicotine Toxicity*, 27 VET. HUM. TOXICOL. 495-497 (1985); F.W. Lavoie & T.M. Harris, *Fatal Nicotine Ingestion*, 9 J. EMERG. MED. 133-136 (1991); N.K. Monalto, et al., *Use of Transdermal Nicotine System in a Possible Suicide Attempt*, 7 J. AM. BD. FAM. PRACT. 417-420 (1994). An 8-month old infant developed progressive obtundation and respiratory depression after ingesting only two cigarette butts. D.J. Borys, S.C. Setzer, & L.J. Ling, *CNS Depression in an Infant After the Ingestion of Tobacco: A Case Report*, 30 VET. HUM. TOXICOL. 20-22 (1988). A 17-year old adolescent suffered cardiac arrest and died shortly after ingesting a concentrated nicotine-containing solution. F.W. Lavoie & T.M. Harris, *Fatal Nicotine Ingestion*, 9 J. EMERG. MED. 133-136 (1991).

<sup>439</sup> A. Woolf, et al., *Childhood Poisoning Involving Transdermal Nicotine Patches*, 99 (5) PEDIATRICS e4 (1997), available at <http://pediatrics.aappublications.org/cgi/content/full/99/5/e4>, (last visited July 7, 2006).

<sup>440</sup> *Id.*

<sup>441</sup> See, e.g., A.S. Trotzky, *The Treatment of Eating Disorders as Addiction Among Adolescent Females*, 14 (4) INT. J. ADOLESC. MED. HEALTH 269-274 (Oct.-Dec. 2002); M. Corcos, et al., *Pertinence of the Addiction Concept in Eating Behavior Disorders*, 151 Suppl. B ANN. MED. INTERNE (PARIS) B53-60 (Oct. 2000); E. David & G. Claridge, *The Eating Disorders as Addiction: A Psychobiologic Perspective*, 23 (4) ADDICT. BEHAV. 463-475 (July-Aug. 1998).

<sup>442</sup> A. Drewnowski, “Taste Responsiveness in Eating Disorders,” in: L.H. Schneider, S.J. Cooper, & K.A. Halmi, eds., *The Psychobiology of Human Eating Disorders*, 575 ANN. N.Y. ACAD. SCI. 99-409 (1990).

<sup>443</sup> See, e.g., H.F. Heubner, *Endorphins. Eating Disorders and Other Addictive Behaviors*, NY: W.W. NORTON (1993); M.A. Marrazzi & E.D. Luby, *An Auto-Addiction Model of Chronic Anorexia Nervosa*, 5 INT’L. J. EATING DISORD. 191-208 (1986).

<sup>444</sup> See, K. Goodrich, *supra* note 356, at 125.

<sup>445</sup> G.T. Wilson, *supra* note 374.

<sup>446</sup> *Id.* at 27, 31-32 (1991).

succeed, indicating that it is in fact physiologically possible to do so. Abstaining from food, of course, is not an option for any living thing.

The reward-deficiency theory posits that addicting drugs are attractive to users because they induce rewards. Proponents of obesity litigation may argue that food induces similar rewards. If so, there should be an inverse relationship between nutrient ingestion and self-administration of psychoactive drugs. At least with respect to such compounds as cocaine, alcohol, opioids and amphetamines; however, eating disorders do not emerge upon recovery from drug dependence. There is no evidence that concurrent use of alcohol or drugs increases as eating disorder patients cease to binge.

Drugs of abuse increase dopamine in naïve as well as addicted subjects. The magnitude of drug-induced dopamine increases, and the intensity of self-reports of the drug's reinforcing properties, appear to be smaller in the addicted than in the naïve patients. Compared with opioid agonist drugs, food consumption has relatively weak effects on endogenous systems.<sup>447</sup>

Then, too, addicts generally have a drug of choice. The *Pelman* complaint did not specify what exactly plaintiffs claimed is addictive in McDonald's products, whether it is the combination of fats and sugars or some additive that may work in the same manner as nicotine to induce addiction.<sup>448</sup> Although some critics of the food industry blame carbohydrate intake for the development of obesity, the prevalence of carbohydrate cravings or rate of carbohydrate consumption is not significantly different among the nonobese, obese binge eaters, and obese non-binge eaters.<sup>449</sup> Most obese persons do not binge. Even in obese individuals who do, however, the salient feature of their binges is not cravings for specific macronutrients, but rather for the amount of food eaten.<sup>450</sup> Even according to addiction theorists, dopamine release is not limited to a specific food. Moreover, the ingredients in foods that some claim are addicting, such as cheese or white flour, are different from cocaine, amphetamines, opioids and nicotine in a physiologically important way: The addicting drugs are discrete chemical compounds. The foods are not.<sup>451</sup> Cheese may contain casein, rennin, calcium, sodium aluminosilicate, propionic acid and its salts, ascorbic acid, butylated hydroxy anisole (BHA), butylated hydroxy-toluene (BHT), benzoates, sodium nitrite, citric acid, cloves, ginger, fructose, aspartame, saccharin, FD&C Red No. 40, monosodium glutamate, caramel, annatto, limonene, and turmeric, among other things.<sup>452</sup> So, even if dopamine release could be linked to a specific food, that is by no means the same as linking it to a specific chemical moiety. And, of course, it would be a rare compound indeed that is found in only one type of food. Most molecules found in one food can be found in another. Moreover,

<sup>447</sup> P.J. Rogers & H.J. Smit, *supra* note 211.

<sup>448</sup> *Pelman I*, *supra* note 16, 237 F. Supp. 2d at 542.

<sup>449</sup> See, e.g., J. Goldfein, et al., *Eating Behavior in Binge Eating Disorder*, 14 INT. J. EATING DISORD. 427-431 (1993); M.S.J. Turner, et al., *Psychological, Hormonal and Biochemical Changes Following Carbohydrate Bingeing: A Placebo-Controlled Study in Bulimia Nervosa and Matched Controls*, 21 (1) PSYCHOL. MED. 123-133 (1991); *Binge Eating: Nature, Assessment, and Treatment*, C.G. Fairburn & G.T. Wilson, eds., NY: GUILFORD PRESS (1993).

<sup>450</sup> S.Z. Yanovski, et al., *Food Selection and Intake of Obese Women With and Without Binge-Eating Disorder*, 56 AM. J. CLIN. NUTR. 975-980 (1992).

<sup>451</sup> *Pelman I* distinguished tobacco from food by pointing out that tobacco is a discreet product producing a characteristic set of illnesses. Fat comes from all sorts of food. *Pelman I*, *supra*, n. 16.

<sup>452</sup> See, USFDA *Food additives* (1993) available at <http://www.cfsan.fda.gov/~lrd/foodaddi.html>, (last visited July 13, 2006). Short chain fatty acids, acetaldehyde, diacetyl, peptides, amino acids, acids, alcohols, esters, aldehydes, ketones, phenolics, and sulfur-containing compounds all affect the flavor of cheddar cheese; over 100 volatile compounds have been identified in it. T.K. Singh, M.A. Drake, & K.R. Cadwallader, *Flavor of Cheddar Cheese: A Chemical and Sensory Perspective*, 2 COMP. REV. FOOD SCI. FOOD SAFETY 139 (2003), available at <http://members.ift.org/NR/rdonlyres/D612A5D2-F351-4FD5-A6AB-4C8DD16B9F1F/0/crfs-fsv2n4p01390162ms20020702.pdf>, (last visited July 12, 2006).

even if there were a molecule unique to a specific food, that food might well have many sources. Even proponents of obesity litigation concede that loyalty to a specific brand is less common with food than with cigarettes.<sup>453</sup> If dopamine release in specified brain structures is the common denominator between addiction to drugs and “addiction” to food, why do we not hear claims that anyone is “addicted” to, say, carrots?

Acute opioid overdose may be specifically treated with opioid antagonists such as naloxone or naltrexone. In fact, when a patient is brought in unresponsive to an ER, naloxone is often injected, for two reasons. First, of course, if the patient responds, his life may be saved. Second, if he responds, his diagnosis is established. No similar effect is seen in overeaters, however.<sup>454</sup> There is no evidence that opioid antagonists reduce motivation to eat at the onset of a meal.<sup>455</sup> Chronic naltrexone treatment has no influence on body weight.<sup>456</sup> Naltrexone at a daily dosage of 200 mg did not appear to produce weight loss after eight weeks of treatment.<sup>457</sup> Although rated pleasantness of sweet glucose solutions declined, as did the consumption of the most preferred foods following administration of another antagonist, nalmefene, the consumption of sandwiches or salted soups remained stable after naltrexone.<sup>458</sup> In other studies, naltrexone had little effect on the palatability of sweet and salty foods.<sup>459</sup> Even those who argue that food selection among bingers may be influenced by endogenous opiates concede that there are problems with the theory. Butorphanol, for example, did not enhance hedonic preferences or food consumption in a group treated with it; a drop in consumption of sweet and high fat foods among those treated with naloxone was accompanied by an increase in popcorn consumption.<sup>460</sup> If food’s effects were mediated through some opioid-like mechanism, one would expect opioid antagonists to have unambiguous therapeutic value. They don’t.

The addiction theory oversimplifies. To make it work, one must ignore differences between groups of obese persons, and similarities between the obese and those of normal weight. Those described as obese are not necessarily the same as those who regularly overeat. There are no reproducible differences in the eating habits of thin people and some obese ones. In a study recording comparatively high average intakes (mean of 12.5 bars per week), only 15% of the 50 chocolate “addicts” surveyed reported eating three or more bars of chocolate per day, and 17% reported eating less than one bar per day.<sup>461</sup> To accept addiction theory, one must ignore these distinctions.

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<sup>453</sup> Alderman & Daynard, *supra* note 1, at 85.

<sup>454</sup> See, e.g., R.L. Atkinson, et al., *Effects of Long-term Therapy With Naltrexone [opioid antagonist] on Body Weight in Obesity*, 38 CLIN. PHARMACOL. THER. 419-422 (1985); J.D. Mitchell, et al., *High-dose Naltrexone Therapy and Dietary Counseling for Obesity*, 22 BIOL. PSYCHIATR. 35-42 (1987). Naltrexone did not prevent sulphiride-induced body weight gain in rats. T. Baptista, A. LaCruz, & L. Hernandez, *Naltrexone Does Not Prevent the Obesity Induced by Antipsychotic Drugs in Rats*, 24 NEUROSCI. ANN. MEET. 706 (1998).

<sup>455</sup> Martin R. Yeomans & R.W. Gray, *supra* n. 381 at 715.

<sup>456</sup> J.E. Mitchell, et al., *High Dose Naltrexone Therapy and Dietary Counseling for Obesity*, 22 BIOL. PSYCHIATR. 35-42 (1987).

<sup>457</sup> R. Malcolm, et al., *A Controlled Trial of Naltrexone in Obese Humans*, 9 INT. J. OBESITY 347-353 (abstract) (1985). Accord, J.E. Mitchell, et al., *High-dose Naltrexone Therapy and Dietary Counseling for Obesity*, 22 BIOL. PSYCHIATR. 35-42 (1987).

<sup>458</sup> A. Drewnowski, *Taste Preference and Food Intake*, 17 ANNU. REV. NUTR. 243 (1997).

<sup>459</sup> M.M. Hetherington, et al., *Failure of Naltrexone to Affect the Pleasantness or Intake of Food*, 40 PHARMACOL. BIOCHEM. BEHAV. 185-190 (1991); M. Mertino, et al., *Naltrexone, an Opioid Blocker, Alters Taste Perception and Nutrient Intake in Humans*, 261 AM. J. PHYSIOL. R. 59-63 (1991).

<sup>460</sup> A. Drewnowski, et al., *Naloxone, An Opiate Blocker, Reduces the Consumption of Sweet High-Fat Foods in Obese and Lean Female Binge Eaters*, 61 AM. J. CLIN. NUTR. 1206-1212 (1995). Note, however, that butorphanol is both an opioid agonist and an opioid antagonist. *Id.*

<sup>461</sup> M.M. Hetherington & J.I. Macdiarmid, “Chocolate Addiction”: A Preliminary Study of its Description and its Relationship to Problem Eating, 21 APPETITE 233-246 (1993). Cf., discussion of tolerance

Foods that eating-disordered individuals attempt to avoid because of self-imposed rules are called “feared foods” and “forbidden foods.”<sup>462</sup> Those foods identified as forbidden vary with popular beliefs. Dietary profiles of eating-disordered individuals change over time, tracking shifts in dieting fads and in nutritional information available about foods (especially carbohydrates and fats).<sup>463</sup>

Some claim that cheese is “morphine on a cracker.”<sup>464</sup> This theory does not withstand scrutiny. Opioids are CNS depressants. Appetite and weight usually decrease in depression.<sup>465</sup> Antidepressants, in contrast, tend to increase body weight.<sup>466</sup> In fact, among depressed patients, complaints of weight gain are often concomitant with clinical improvement.<sup>467</sup> Anorexia is a classic symptom of depression. Depressed patients responding to treatment, in fact, often eat more—this may give the attending physician the first clue his therapy is having some beneficial effect. In fact, a practical problem in our weight-obsessed society is that the depressed patient under treatment may actually gain weight, and for that reason become non-compliant with his antidepressant regimen. If cheese were an opioid, eating a few bites would tend to suppress appetite, and obesity would become rare. This does not seem to have happened in the U.S.

Advocates of the food-as-addicting theory point to sophisticated brain scans that suggest activity in parts of the brain thought to be affected by opioids and other drugs of abuse. There are reports that those same brain areas become active when we eat. But addiction is a clinical diagnosis, based upon history, to some extent upon physical exam, and to a limited degree, upon testing blood or urine for the presence of drugs of abuse or of their metabolites. Clinically, diagnosis of addiction does not depend upon brain scans. More important, the focus on genes associated with the brain dopamine system is preliminary in character, particularly with respect to supposed behavioral addictions.<sup>468</sup>

Most fundamental, perhaps, is this problem: However it is best defined, addiction is pathological—a disease state. Eating is physiological—a *sine qua non* for survival. From an evolutionary standpoint, it is scarcely surprising that perception of pleasure underlies behaviors that need to be repeated for preserving the species. The mere fact, if it be a fact, that there may be neurochemical similarities between the body’s metabolism of foods and its metabolism of drugs does not make drug abuse a healthful behavior nor eating a sign of disease. If food intake, necessary for the survival of the individual and of the species as a whole, were painful, the continued existence of both would be jeopardized. For the addict, heroin use is also pleasurable. But the rewards for use of heroin create not a solution but a problem. Discovery of physiological reward pathways,

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at IV.C.1, *supra*.

<sup>462</sup> See, e.g., M. Gattellari & G.F. Huon, *Restrained and Unrestrained Females' Positive and Negative Associations With Specific Foods and Body Parts*, 29 INT’L. J. EATING DIS. 377-383 (1997); E.F. Kales, *Macronutrient Analysis of Binge Eating and Bulimia*, 48 PHYSIOL. BEHAV. 837-840 (1990).

<sup>463</sup> V.M. Gonzalez & K.M. Vitousek, *Feared Food in Dieting and Non-dieting Young Women: A Preliminary Validation of the Food Phobia Survey*, 48 APPETITE 155-173 (2004).

<sup>464</sup> N. Barnard, *Breaking the Food Seduction: The Hidden Reason Behind Food Craving—and 7 Steps to End Them Naturally*, ST. MARTIN’S PRESS (2003) at 51; See also, Personal Responsibility in Food Consumption Act, Hearings before the Subcommittee on Commercial and Administrative Law of the Committee on the Judiciary, H. REP. 108TH CONG. (1st Sess.), (June 19, 2003).

<sup>465</sup> T. Baptista, *Body Weight Gain Induced by Antipsychotic Drugs: Mechanisms and Management*, 100 ACTA PSYCHIATR. SCAND. 3, 5 (1999).

<sup>466</sup> *Id.*

<sup>467</sup> G.H. Berken, et al., *Weight Gain: A Side-Effect of Tricyclic Antidepressants*, 7 J. AFFECT. DISORD. 133-138 (1984); E.J. Garland, et al., *Weight Gain With Antidepressants and Lithium*, 8 J. CLIN. PSYCHOPHARMACOL. 322-330 (1988), cited in R.W. Foltin, et al., *The Effects of Cocaine on Food Intake of Baboons Before, During, and After a Period of Repeated Desipramine*, 36 PHARMACOLOGY BIOCHEMISTRY & BEHAVIOR 873 (1990).

<sup>468</sup> A. Ibáñez et al., *Genetics of Pathological Gambling*, 19 J. GAMBLING STUDIES 11-22 (2003).

and of similarities between such pathways and the metabolism of drugs, does not prove that overeating is an “addictive” disorder, or caused by a “disease” that requires treatment. It may suggest that humans, like other mammals, are programmed for species survival to eat (and breathe and reproduce and sleep).<sup>469</sup> Perhaps the elaborate homeostatic mechanisms for balance between energy consumption and expenditure, for which there is no analog in smoking or drug abuse, reflect our constitutional preparation for and physiologic dependence on eating.

#### H. *The Limitations of Addiction Theory as Applied to Eating: Chocolate*

A number of papers have reported that women crave chocolate more than any other food.<sup>470</sup> Some have characterized such preferences as a form of addiction.<sup>471</sup> This makes chocolate a food particularly well suited to analyzing the notion that eating can be a form of addiction, and we proceed to that analysis.

One popular idea is that chocolate craving and “addiction” might be explained, at least in part, by the presence of psychoactive substances in cocoa-containing products. Relying on what is known about the concentrations of such substances in the products that are most widely eaten, however, and their probable effects on the brain when administered orally, Rogers and Smit found no convincing support for the existence of chocolate addiction. They also rejected the theory that chocolate or foods often craved are linked specifically to the effects of eating on the activity of brain serotonergic or endogenous opioid systems. Rogers and Smit point out that, apart from the compulsive eating seen in bulimia and binge-eating disorder, the vast majority of cases of (self-reported) food craving and food “addiction” should not be viewed as addictive behavior. In particular, eating does not produce the powerful neuroadaptive effects that are central to drug addiction.<sup>472</sup>

Di Tomaso, et al. and Max have argued that chocolate craving is the subjective manifestation of an addiction to psychoactive chemicals contained in cocoa.<sup>473</sup> Cocoa does contain the sympathomimetic biogenic amines tyramine and phenylethylamine and the methylxanthines, theobromine and caffeine, which could potentially be psychoactive.<sup>474</sup> For these compounds to induce a chocolate “addiction” or craving, however, Rogers and Smit note that certain conditions should be met. First, the active ingredients should be measurably stimulant, euphoric or at least reinforcing when taken orally in appropriate doses; second, such cravings should occur for all foods containing enough of these substances (assuming adequate exposure) but not for foods that do not; third, the craving should be at least partly reduced by ingestion of the active ingredients even in the absence of ingestion of the craved food. The compounds in cocoa do not meet these conditions.<sup>475</sup>

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<sup>469</sup> K. Goodrich, *supra* note 356, at 132-133.

<sup>470</sup> M.M. Hetherington & J.L. Macdiarmid, *supra* note 461; P. Rozin, et al., *Chocolate Craving and Liking*, 17 APPETITE 199-212 (1991); A. Drewnowski, *Changes in Mood Following Carbohydrate Consumption*, 46 AM. J. CLIN. NUTR. 703 (1987) (letter); S.F. Abraham & P.J.V. Beumont, *How Patients Describe Bulimia or Binge Eating*, 12 PSYCHOL. MED. 625-635 (1982).

<sup>471</sup> Hetherington & Macdiarmid, *supra* note 461; *see also*, W. Vandereycken, *The Addiction Model in Eating Disorders: Some Critical Remarks and a Selected Bibliography*, 9 INT. J. EATING DISORD. 95-101 (1990).

<sup>472</sup> P.J. Rogers & H.J. Smit, *supra* note 211, at 12.

<sup>473</sup> E. Di Tomaso, M. Beltramo, & D. Piomelli, *Brain Cannabinoids in Chocolate*, 382 NATURE 677, 678 (1996) and B. Max, *This and That: Chocolate Addiction, the Dual Pharmacogenetics of Asparagus Eaters, and the Arithmetic of Freedom*, 10 TRENDS PHARM. SCI. 390-393.

<sup>474</sup> W.J. Hurst & P.B. Toomey, *High-performance Liquid Chromatographic Determination of Four Biogenic Amines in Chocolate*, 106 ANALYST 394-402 (1981); B. Max, *supra* note 473.

<sup>475</sup> E.L. Gibson & E. Desmond, *Chocolate Craving in the Hunger State: Implication of the Acquisition and Expression of Appetite and Food Choice*, 32 APPETITE 219-240 (1999) at 220.

Data on reinforcing effects following ingestion of chocolate (or other craved foods) are scanty. Biogenic amines in food are metabolized by enzymes such as monoamine oxidase (MAO) in gut and liver, so that they would not be expected to reach brain.<sup>476</sup> Truly addicting drugs do reach brain; that's exactly why their effects are so powerful.

Rogers and Smit also point out that many of the biogenic amines found in chocolate are also found in other foods. Peas, yeast extract and pickled herrings can contain substantial amounts (1-3 mg/g) of tyramine (a sympathomimetic named from "tyrose," Greek for cheese).<sup>477</sup> Smaller amounts are present in fruits such as bananas and tomatoes as well as in a variety of meat and dairy products.<sup>478</sup> Concentrations of amines including phenylethylamines in different chocolate brands can vary by at least a factor of 10.<sup>479</sup> The presence of amines in food does not seem to provide a convincing explanation for the dominance of chocolate among craved foodstuffs.

Drewnowski, et al., have argued that chocolate craving represents a need to stimulate endogenous opioid receptors and hence resembles opioid addiction.<sup>480</sup> Rogers and Smit point out, however, that evidence for opioid mediation and responses to preferred food stimuli implicate many sweet and fatty foods for which there is little evidence of craving.<sup>481</sup>

The most widely consumed chocolate is milk chocolate and chocolate-covered confectionery. Compared with dark chocolates, these contain lower amounts of cocoa solids, and therefore a lower concentration of many of the potentially psychoactive compounds unique to chocolate.<sup>482</sup> If chocolate were truly addicting, one would expect to observe the opposite pattern.

In experimental studies, cocoa powder alone did not reduce craving any more than placebo capsules or nothing, while white chocolate, with or without cocoa powder capsules, reduced craving to some extent, but not so effectively as milk chocolate (though that may be influenced by expectation). Thus, ingestion of the cocoa ingredients had no impact on chocolate craving, while the sensory similarities of white chocolates to milk chocolate (sweetness and cocoa butter) allowed a significant or partial alleviation of craving.<sup>483</sup>

Among self-reported "chocolate addicts," eating chocolate caused increased guilt feelings. It had no effect on feelings of depression or relaxation.<sup>484</sup> And while no one

<sup>476</sup> F. Karoum, et al., *Mass Fragmentography of Phenylethylamine, m- and p-tyramine and Related Amines in Plasma, Cerebrospinal Fluid, Urine and Brain*, 33 J. NEUROCHEM. 201-212 (1979); E. Marley & B. Blackwell, *Interactions of Monoamine Oxidase Inhibitors, Amines and Foodstuffs*, 8 ADV. PHARMACOL. CHEMOTHER. 185-349 (1970).

<sup>477</sup> E. Marley & B. Blackwell, *Interactions of Monoamine Oxidase Inhibitors, Amines, and Foodstuffs*, 8 ADV. PHARMACOL. AND CHEMOTHER. 185-349 (1970).

<sup>478</sup> W.J. Hurst, et al., *Biogenic Amines in Chocolate—A Review*, 26 NUTRIT. RPT. INT'L. 1081-1086 (1982).

<sup>479</sup> W.J. Hurst & P.B. Toomey, *supra* n. 474.

<sup>480</sup> A. Drewnowski, et al., *Taste Responses and Preferences for Sweet, High-fat Foods: Evidence for Opioid Involvement*, 51 PHYSIOL. AND BEHAV. 371-379 (1992).

<sup>481</sup> E.M. Blass, et al., *Stress-reducing Effects of Ingesting Milk, Sugars and Fats: The Developmental Perspective*, 575 ANN. N.Y. ACAD. SCI. 292-305 (1989); S.J. Cooper, et al., "Endorphins, Opioids and Food Intake," in R. J. Rodgers & S. J. Cooper, eds, *Endorphins, Opioids and Behavioural Processes*, JOHN WILEY CHICHESTER, (1988) 143-186. See also M. Yeomans & P. Wright, *Lower Pleasantness of Palatable Foods in Nalmefene-treated Human Volunteers*, 16 APPETITE 249-259 (1999) and M.R. Yeomans, et al., *Effects of Nalmefene on Feeding of Humans: Dissociation of Hunger and Palatability*, 100 PSYCHOPHARMACOL. 426-432 (1990).

<sup>482</sup> P.J. Rogers & H.J. Smit, *supra* note 211.

<sup>483</sup> W. Michener & P. Rozin, *Pharmacological Versus Sensory Factors in the Cessation of Chocolate Craving*, 56 PHYSIOL. AND BEHAV. 419-422 (1994).

<sup>484</sup> J.L. Macdiarmid & M.M. Heatherington, *Mood Motivation by Food: An Exploration of the Affect and Cravings in "Chocolate Addicts"*, 35 BRIT. J. CLIN. PSYCH. 129-138 (1995).

would argue that a chocolate bar is health food,<sup>485</sup> cocoa, like tea and red wine, is rich in flavanols, which are thought to offer cardiovascular health benefits.<sup>486</sup>

Chocolate is not heroin.

### I. *The Limitations of Addiction Theory as Applied to Drinking: Caffeine*

Caffeine is present in many foods and beverages consumed frequently in America and, indeed, throughout the world. Some have argued that caffeine, often consumed in popular beverages, is addicting.<sup>487</sup> We therefore select caffeine as a second foodstuff to examine the claim that eating can be a form of addiction.

In the “Substance-Related Disorders” section of the *DSM-IV-TR*, there are diagnoses for various types of dependence (including alcohol, inhalant, and nicotine dependence), but not for caffeine dependence, and certainly none for food dependence.<sup>488</sup> Predecessor versions of *DSM-IV* did not define caffeine as addicting either.

At present, there is a dearth of evidence that caffeine meets the criteria established for substance dependence. The type of clinical data that would be necessary for adding caffeine dependence to the list might include empirical evidence that a substantial proportion of coffee drinkers or other heavy consumers of caffeine 1) try to stop and cannot, 2) have difficulty switching to decaffeinated coffee or other beverages, and 3) use caffeine despite knowledge that they have health problems that are aggravated by caffeine. Other potentially supportive data might be 4) heavy coffee drinkers’ ratings of the difficulty of stopping caffeine intake compared to stopping cigarette smoking or drinking alcohol, 5) whether tolerance to important behavioral effects (e.g., relief of fatigue) occurs, 6) whether coffee use escalates over time, 7) how often coffee is used to avoid withdrawal symptoms, and 8) what the prevalence of caffeine dependence is.<sup>489</sup> The current state of the literature does not allow one to conclude that caffeine exerts the effects characteristic of addiction.

If we consider the *DSM-IV-TR* criteria serially, the case for caffeine addiction is weak. Turning to the first part of the definition, how well does caffeine meet the test? “Maladaptive pattern of...use leading to... significant impairment or distress...” What “distress” arises from consumption of caffeine? Tachycardia? An individual with tachycardia from excessive caffeine use may have a physiological intoxication, but if this is the only symptom in the absence of maladaptive behavior, the diagnosis of caffeine intoxication would not apply. Tachycardia and diuresis *per se* are scarcely “distressing” in any meaningful sense. Nor is there any increased risk of accidents, medical complications, disrupted relationships, or vocational or financial or legal problems from use of caffeine as there is with use of, say, amphetamines.

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<sup>485</sup> Yet stearic acid, the saturated fat in chocolate, raises HDL (so-called “good cholesterol”) levels while doing little or nothing to LDL (“bad cholesterol”). G. Taubes, *supra* note 18.

<sup>486</sup> H. Schroeter, et al., (-)—*Epicatchin Mediates Beneficial Effects of Flavanol-rich Cocoa on Vascular Function in Humans*, 103 (4) *PROC. NAT’L. ACAD. SCI.* 1024-1029 (Jan. 24, 2006), available at [www.pnas.org/cgi/doi/10.1073/pnas.0510168103](http://www.pnas.org/cgi/doi/10.1073/pnas.0510168103), (last visited July 7, 2006).

<sup>487</sup> See Julia Baird, *Love-hate Bond to the Ultimate in Synthetic Foods*, *SYDNEY MORNING HERALD* (Sept. 17, 2002) at 13, cited in J. A. Cohan, Note, *Obesity, Public Policy and Tort Claims Against Fast Food Companies*, 12 *WIDENER L.J.* 103, 116 (2003). See also, *See How Caffeine Works*, at <http://howstuffworks.com/caffeine.htm> (last visited July 7, 2006); *AM. PSYCHIATRIC ASS’N, Diagnostic and Statistical Manual of Mental Disorders*, *supra* n. 341, at 177 (Table 1 lists caffeine as a substance that can create intoxication and contribute to sleep and anxiety disorders.).

<sup>488</sup> *DSM-IV, TR*, *supra* note 341, at 16-19.

<sup>489</sup> J.R. Hughes, et al., *Should Caffeine Abuse, Dependence, or Withdrawal be Added to DSM-IV and ICD-10?* 149 (1) *AM. J. PSYCHIATRY* 33, 38 (Jan. 1992), *supra* note 358.

Significant harms from caffeine ingestion are hard to show. The use of caffeine is socially accepted, so there is no reason a coffee drinker cannot be gainfully employed. In contrast, in the U.S. there is no appropriate or lawful use for heroin, for example. Overconsumption of anything can harm. The deleterious effects of caffeine ingestion, if any, are transient. Even those harms reported often turn out, on further analysis, to actually be smoking-related. The oft-cited Framingham study, the largest epidemiologic effort of its kind in the history of medicine, found no relationship between caffeine and heart disease.<sup>490</sup> This observation has been confirmed recently.<sup>491</sup> Caffeine and coffee appear to have little or no effect on blood pressure, coagulation, or serum lipids.<sup>492</sup> One small study suggested that a large single dose of caffeine actually improved exercise tolerance.<sup>493</sup>

*Tolerance:* There have been reports of tolerance to caffeine. There is reason, however, to question the accuracy of some such information. Reported drug and dietary consumption and actual consumption often differ appreciably. In one study, it was demonstrated that coffee consumption increased as caffeine content decreased, a phenomenon that could lead to an artificially low estimate of caffeine content for decaffeinated coffee drinkers.<sup>494</sup> Among subjects who reported histories of heavy coffee drinking (mean 14 cups of coffee per day), many of whom also reported problems with alcohol and/or drug abuse, Griffiths found that coffee drinking was spaced regularly among subjects given access *ad lib*. Inter-cup intervals grew longer during the day. Caffeine consumption fell as blood levels rose. The range of self-administration was narrower than with true drugs of addiction. The *ad lib* consumption of decaf was the same as that with regular. Heavier drinkers did not drink stronger coffee.<sup>495</sup> These data do not a case for addiction make. When caffeine was manipulated over a wide dose range (50, 100, 200, and 400 mg per cup), it produced a monotonic dose-related suppression of numbers of cups consumed and subject-rated coffee “liking.”<sup>496</sup>

*Withdrawal:* There have been few studies, and most of those were relatively poorly done. Unlike heroin addicts, for example, caffeine users need have no symptoms on stopping.<sup>497</sup> Those who do offer complaints mention very few. These (headache, fa-

<sup>490</sup> Dawber, et al., *Coffee and Cardiovascular Disease—Observations From the Framingham Study*, 291 N. ENGL. J. MED. 871-874 (1974).

<sup>491</sup> E. Lopez-Garcia, et al., *Coffee Consumption and Coronary Heart Disease in Men and Women. A Prospective Cohort Study*, 113 CIRCULATION 2045 (2006); abstract available at <http://circ.ahajournals.org/cgi/content/abstract/CIRCULATIONAHA.105.598664v1>, (last visited June 23, 2006).

<sup>492</sup> W.G. Thompson, *Coffee: Brew or Bane?*, 308 (1) AM. J. MED. SCI. 54 (1994).

<sup>493</sup> L.R. McNaughton, *The Influence of Caffeine Ingestion on Incremental Treadmill Running*, 20 BR. J. SPORTS MED. 109-112 (1986). Nor is this the only benefit of caffeine, which is reportedly useful in management of strokes. J.C. Grotta, et al., *Pilot Dose-Escalation Study of Caffeine Phenethanol (Caffeinol) in Acute Ischemic Stroke*, 34 STROKE 1242 (2003). Caffeine also appears to protect against development of type 2 diabetes. See, e.g., E. Salazar-Martinez, et al., *Coffee Consumption and Risk for Type 2 Diabetes Mellitus*, 140 ANN. INT. MED. 1-8 (2004). Long-term coffee consumption reportedly enhances insulin sensitivity. J. Arnlov & B. Vessley, *Coffee Consumption and Insulin Sensitivity*, 291 JAMA 1199-1201 (2004). Coffee, both caffeinated and decaffeinated, is reportedly the largest source of antioxidants in the average American's diet. J. Vinson, quoted in SENIOR JOURNAL (Aug. 28, 2005), available at <http://www.seniorjournal.com/NEWS/Nutrition-Vitamins/5-08-28CoffeeAntioxidant.htm>, (last visited July 12, 2006). It would be difficult to generate a list of benefits for a truly addicting substance such as crack cocaine.

<sup>494</sup> L.T. Kozlowski, *Effect of Caffeine on Coffee Drinking*, 264 NATURE 354-355 (1976), cited in D.M. Veleber & D.I. Templer, *Effects of Caffeine on Anxiety and Depression*, 93 J. ABNORMAL PSYCH. 122 (1984).

<sup>495</sup> R.R. Griffiths, et al., *Human Coffee Drinking: Reinforcing and Physical Dependence Producing Effects of Caffeine*, 239 J. PHARMACOL. EXP. THER. 416-425 (1986); R.R. Griffiths, et al., *Human Coffee Drinking: Manipulation of Concentration of Caffeine Dose*, 45 J. EXP. ANAL. BEHAV. 133-148 (1986).

<sup>496</sup> R.R. Griffiths, et al., *Human Coffee Drinking: Manipulation of Concentration of Caffeine Doses*, *supra* note 495.

<sup>497</sup> H.A. Reimann, *Caffeinism: A Cause of Long-continued, Low-grade Fever*, 202 JAMA 131-132 (1967); W.R. Carter, *Withdrawal of Caffeine From Regular Users: Our Study vs. the Subjects' Study*, Soc. BEHAV. MED. ABSTRACT No. C93, at 40 (1984).

tigue, drowsiness) are non-specific, and two of the three are very similar.<sup>498</sup> All three are common in the general public. And, of course, headache can be caused by caffeine consumption, too, and by dozens of other causes, so it is often difficult in those offering this complaint to determine its etiology.<sup>499</sup>

*Excessive quantities or duration:* There is little evidence here, either. In nonhumans, caffeine is not consistently self-administered at a rate greater than placebo.<sup>500</sup> In baboon studies, only erratic patterns of self-injection were observed.<sup>501</sup> In humans, self-administration of caffeine is neither readily established nor especially robust. Stern, et al. found that light caffeine users did not prefer ingesting caffeine capsules (100 mg or 300 mg) over placebo capsules.<sup>502</sup>

*Inability to Curtail Consumption:* Few reports. This phenomenon has not been demonstrated in animal studies. There is little basis to say that caffeine meets this requirement. Caffeine self-administration is not readily established and is not particularly dramatic.<sup>503</sup> Nor are there any data on the difficulty that caffeinated coffee users have with switching to decaffeinated coffee.<sup>504</sup> Relative to the widespread use of caffeine throughout the world, there are few reports in the literature of individuals who find it difficult to decrease or stop their caffeine intake.<sup>505</sup> As Hughes, et al. indicated, however, there are no prospective clinical studies on attempts to quit caffeine or on success rates of caffeine cessation programs.<sup>506</sup>

*Time Spent; Narrowed Behavioral Repertoire:* Caffeine is legal and abundant. Investing excessive time to procure or prepare it is simply not necessary. Instances of spending a great deal of time on preparation and use of beverages containing caffeine have not been reported in the recent English language literature. Most caffeine takers are flexible respecting environmentally-induced changes in intake timing. There is no need for a narrowed behavioral repertoire.<sup>507</sup> Can the DSM-IV criteria even be applied to caffeine?

*Persistent Use in Face of Harm:* What harm? As noted, caffeine users have no problems with daily functioning. They face no particular hazards when using caffeine. They experience no financial, legal, interpersonal or social problems. They suffer no loss of freedom. Those addicted to cocaine, for example, cannot make such claims. Persistent use of caffeine in the face of a known illness aggravated by caffeine has not been documented, nor have caffeine-induced accidents been documented.<sup>508</sup> Once again, one is left with the question: Does this criterion, developed by APA to evaluate

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<sup>498</sup> For an article on headaches occasioned by abstinence from caffeine, see, W.M. Davis, *Major Types of Primary and Secondary Headaches and Their Treatments*, DRUG TOPICS, (Apr. 18, 1986) at 128, 130.

<sup>499</sup> J.R. Harrie, *Caffeine and Headaches*, 213 JAMA 628 (1970); J.R. Hughes, "Caffeine Withdrawal, Dependence and Abuse," in T.A. Whidiger, A.J. Frances, H.A. Pinkus, M.B. First, R. Ross, W. Davis, eds., DSM-IV SOURCE BOOK, Amer. Psychiatric Ass'n., Wash., D.C., (1994), at 129-134.

<sup>500</sup> R.R. Griffiths & P.P. Woodson, *Reinforcing Properties of Caffeine: Studies in Humans and Laboratory Animals*, 29 PHARMACOL. BIOCHEM. BEHAV. 419-427 (1988).

<sup>501</sup> R.R. Griffiths, et al., *Comparison of Behavior Maintained by Infusions of 8 Phenylethylamines in Baboons*, 50 PSYCHOPHARMACOLOGY (Berlin) 251-258 (1976).

<sup>502</sup> Stern, et al., *Reinforcing and Subjective Effects of Caffeine in Normal Human Volunteers*, 98 PSYCHOPHARMACOLOGY (Berlin) 81-88 (1989).

<sup>503</sup> S.R. Goldberg & I.P. Stolerman, *Behavioural Analysis of Drug Dependence*, ACADEMIC PRESS: ORLANDO (1986).

<sup>504</sup> J.R. Hughes, et al., *supra* note 489.

<sup>505</sup> J.F. Greden, *Anxiety or Caffeinism: A Diagnostic Dilemma*, 131 AM. J. PSYCHIATRY 1089-1092 (1974).

<sup>506</sup> J.R. Hughes, et al., *supra* note 489.

<sup>507</sup> S.J. Heishman & J.E. Henningfield, *Is Caffeine a Drug of Dependence? Criteria and Comparisons*, 7 PHARMACOPSYCHOECOLOGIA 127-135 (1994) at 132.

<sup>508</sup> *Id.*

possible substance dependence, have any real application or any clear meaning with respect to caffeine?

Beyond the *DSM-IV-TR* criteria, there are others that have traditionally been used. Reinforcement is one. A drug is a reinforcer when its administration is a consequence of behavior and maintains behavior. The most convincing evidence of drug reinforcement occurs when an animal works hard for the opportunity to give itself a dose of the drug. Evidence of caffeine reinforcement is equivocal. Results of most animal studies have shown that caffeine does not function reliably as a reinforcer across several species and testing conditions.<sup>509</sup> *Cf.* cocaine, e.g.: Bozarth and Wise have reported that animals experimentally addicted ignore food and water unto death to get their cocaine.<sup>510</sup> Moreover, caffeine is associated with at least some aversive effects, though they are typically mild: dysphoria, anxiety, nervousness, hostility.<sup>511</sup> These may tend to defeat caffeine's capacity for reinforcement.<sup>512</sup>

### *J. Unlike Food, Nicotine's Addictiveness Is Well-Established*

There is widespread (though perhaps not quite universal) agreement that nicotine is addicting. WHO has recognized that nicotine addiction is a disease.<sup>513</sup> HHS has reached similar conclusions.<sup>514</sup> Relying on the criteria of *DSM-IV*, FDA has also declared nicotine to be addicting.<sup>515</sup> The Agency takes the position that tobacco products are intended to satisfy the cravings of persons addicted to nicotine.<sup>516</sup> FDA may have been persuaded in part by allegations that defendant tobacco companies concealed their knowledge of nicotine's addictive properties and "purposefully and deliberately emphasized efforts to addict children and adolescents as resulting in an epidemic pediatric disease. . . ."<sup>517</sup>

<sup>509</sup> C.C. Heppner, et al., *Effects of Food Deprivation on Caffeine Consumption in Male and Female Rats*, 24 PHARMACOL. BIOCHEM. BEHAV. 1555-1559 (1986); Schuster, et al., *Self-administration of Central Stimulants by the Monkey*, in F. Sjoqvist & M. Tottie, eds., *Abuse of Central Stimulants*, NY: RAVEN PRESS (1969) at 339-347.

<sup>510</sup> M.A. Bozarth & R.A. Wise, *Toxicity Associated With Long-term Intravenous Heroin and Cocaine Self-administration in the Rat*, 254 JAMA 81-83 (1985). See generally, S.J. Heishman & J.E. Henningfield, *supra* note 507.

<sup>511</sup> D.M. Veleber & D.I. Templer, abstract, *Effects of Caffeine on Anxiety and Depression*, 93 J. ABNORMAL PSYCH. 120-122 (1984); Goldstein, et al., *Psychotropic Effects of Caffeine in Man, II. Alertness, Psychomotor Coordination, and Mood*, 150 J. PHARMACOL. EXP. THER. 146-151 (1965).

<sup>512</sup> See, J.R. Hughes, *Caffeine Withdrawal, Dependence and Abuse*, *supra* n. 489.

<sup>513</sup> "Possible subjects of initial protocols: Elaboration of technical components of three possible protocols," Working Group on the WHO Framework Convention on Tobacco Control, 2d mtg., Agenda Item 6, WHO Doc. A/FCTC/WG2/4 (Feb. 15, 2000).

<sup>514</sup> HHS, *The Health Consequences of Smoking, Nicotine Addiction: A Report of the Surgeon General*, (1988) at 11 (testimony of Dr. Richard Hurt) (summarizing the major conclusions from the 1988 Surgeon General's report); D.A. Kessler, et al., *The Food and Drug Administration's Regulation of Tobacco Products*, 335 NEW ENG. J. MED. 988 (1996); J.E. Henningfield, L.M. Schuh, & M.D. Jarvik, *Pathophysiology of Tobacco Dependence*, in F.E. Bloom & D.J. Kupfer, eds., *PSYCHOPHARMACOLOGY: THE FOURTH GENERATION OF PROGRESS* (1995) at 1715.

<sup>515</sup> G.E. Kelder & R.A. Daynard, *The Role of Litigation in the Effective Control of the Sale and Use of Tobacco*, 8 STAN. L. & POL'Y REV. 63 (1997) at 76. FDA concluded that it had jurisdiction over tobacco products because "the nicotine and tobacco products is highly addictive, causes other psychoactive effects, such as relaxation and stimulation, and affects weight regulation" and because "these responses to nicotine are effects on the structure or function of the body within the meaning of [Federal Food Drug & Cosmetic] Act." *Analysis of the FDA's Jurisdiction Over Nicotine-containing Tobacco Products*, 60 *Fed. Reg.* 41, 464 (1995).

<sup>516</sup> See 61 *Fed. Reg.* at 44,628-30, 45,203-04.

<sup>517</sup> See, e.g., *Arch v. The American Tobacco Co., Inc.*, 175 F.R.D. 469, 479 (D.Pa. 1997). See also, J. Slade, et al., *Nicotine and Addiction: The Brown & Williamson Documents*, 274 (3) JAMA 225-233 (1995).

The APA agrees that nicotine is addicting. Nicotine addiction is classified as a disease under the *DSM-IV*.<sup>518</sup> APA has identified two medical disorders that pertain to nicotine addiction: 1) nicotine dependence, defined as a “pattern of repeated self-administration that usually results in tolerance, withdrawal, and compulsive drug-taking behavior;” and 2) nicotine withdrawal, which causes “clinically significant distress or impairment in social, occupational, or other important areas of functioning.”<sup>519</sup>

The addictive power of nicotine exceeds that of other, unambiguously addicting substances: “The addiction rate for smoking is higher than the addiction rates for marijuana, alcohol, or cocaine . . . symptoms of serious nicotine addiction often occur only weeks or even just days after youth ‘experimentation’ with smoking first begins.”<sup>520</sup>

### 1. *Applying the DSM-IV Criteria to Nicotine*

In concluding that manufacturers intended cigarettes and other tobacco products be used to affect the structure or function of the body, and hence to be drugs under the Federal Food, Drug and Cosmetic Act, FDA relied in part on scientific evidence concerning the addictive nature of nicotine, including evidence of physiological dependence.<sup>521</sup> FDA cited studies documenting symptoms of smoking addiction such as continued use despite knowledge of harmful consequences, withdrawal symptoms following abstinence, and continued use despite repeated attempts to quit.<sup>522</sup>

#### a. *Tolerance*

APA says that tolerance to nicotine is manifested by a more intense effect of nicotine the first time it is used during the day and the absence of nausea and dizziness with repeated intake, despite regular use of substantial amounts of the substance.<sup>523</sup> In those who initiate smoking, cigarette consumption typically escalates over a couple of years,<sup>524</sup> often leading to addiction.<sup>525</sup> More than 80% of smokers begin by age 18, and 50% of smokers have become daily smokers before age 18.<sup>526</sup> As many as 90% of children who smoke more than two or three cigarettes per day go on to become regular smokers.<sup>527</sup> On average, it takes three years from starting to smoke to becoming a daily smoker.<sup>528</sup>

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<sup>518</sup> *DSM-IV*, TR, *supra* note 341, at 264. See also, B.M. Meier, *Breathing Life into the Framework Convention on Tobacco Control: Smoking Cessation and the Right to Health*, 5 *YALE J. HEALTH POL’Y, LAW & ETHICS* 137 (2005) at 189.

<sup>519</sup> J.E. Henningfield & J. Slade, *Tobacco-dependence Medications: Public Health and Regulatory Issues*, 53 *FOOD & DRUG L.J.* 75, 79 (1998).

<sup>520</sup> CDC, “Symptoms of Substance Dependence Associated with use of Cigarettes, Alcohol and Illicit Drugs, 1991-1992,” 44 *MORBIDITY & MORTALITY WEEKLY REP.* 830 (Nov. 10, 1995), available at <http://www.cdc.gov/mmwr/preview/mmwrhtml/00039501.htm>, (last visited August 8, 2006).

<sup>521</sup> See 61 *Fed. Reg.* at 44,698-730.

<sup>522</sup> See 61 *Fed. Reg.* at 44,730-739.

<sup>523</sup> *DSM-IV-TR*, *supra*, n. 341, at 264-265.

<sup>524</sup> N.L. Benowitz, *Nicotine Addiction*, 26 *PRIM. CARE* 611-631 (1999). The development of tolerance may make it difficult for a smoker to maintain low-level tobacco consumption for extended periods of time. N. Grunberg, *Understanding the Facts About Nicotine Addiction*, 18 (6) *BROWN UNIV. DIGEST OF ADDICTION THEORY & APPLICATION* (1999).

<sup>525</sup> See *FDA v. Brown & Williamson Tobacco Corp.*, 529 U.S. 120, 169 (Breyer, J., dissenting).

<sup>526</sup> DHHS, *Children and Tobacco: The Facts*, HHS Fact Sheet (Aug. 10, 1995).

<sup>527</sup> M.J. Jarvis, *Tobacco Smoking: An Everyday Drug Addiction*, in I. Belle Glass, ed., *INT’L. HANDBOOK OF ADDICTION BEHAVIOR* (1991) at 97. There is no analogous behavior demonstratable in eating.

<sup>528</sup> Drug Abuse Advisory Committee, Center for Drug Evaluation and Research (CDER), FDA, *Issues Concerning Nicotine-containing Cigarettes and Other Tobacco Products*, (Aug. 2, 1994) (Drug Abuse Advisory Committee Meeting No. 27 transcript) at 65 (testimony of Dr. Gary Giovino, Chief of the Epidemiology Branch, CDER, Office on Smoking and Health) (citing the National Survey on Drug Abuse).

There is evidence that nicotine displays a dose-response relationship. Nesbitt gave incremental levels of electric shock to smoking volunteers, and asked them to report when the shocks became unendurably painful. Chronic smokers had significantly higher endurance thresholds when smoking than when not, with high nicotine cigarettes leading to higher endurance thresholds than low nicotine cigarettes.<sup>529</sup>

The American Health Foundation has suggested that people actually ingest far higher levels of carcinogens and nicotine from cigarette smoke than indicated by standard, governmentally-sanctioned testing.<sup>530</sup> The data suggest that whenever possible consumers generally adjust their smoking behavior to obtain a consistent dose of nicotine.<sup>531</sup>

### b. *Withdrawal*

*DSM-IV* recognizes nicotine withdrawal as a disorder.<sup>532</sup> APA says that cessation of nicotine use produces a well-defined withdrawal syndrome. Many individuals who use it take nicotine to relieve or to avoid withdrawal symptoms when they wake up in the morning or after being in a situation where use is restricted (e.g., at work or on an airplane). APA considers weight gain and a reduction in heart rate following the cessation of tobacco use to be manifestations of nicotine withdrawal and therefore, of dependence.<sup>533</sup>

Smokers suffer a source of stress not encountered by non-smokers: acute nicotine depletion.<sup>534</sup> Parrott has suggested that positive changes induced by smoking, such as relaxation and alertness, largely reflect reversal of the negative effects of abstinence (irritability and impaired concentration).<sup>535</sup> Smokers need frequent supplies of nicotine simply to maintain normal mood, and suffer from stress and irritability when they have not smoked recently.<sup>536</sup>

Symptoms of nicotine withdrawal include: craving, depressed mood, irritability, frustration, anger, anxiety, difficulty concentrating, restlessness, insomnia, headache, weight gain, and decreased heart rate.<sup>537</sup> Other symptoms of tobacco abstinence include feelings of stress, hunger and cigarette craving.<sup>538</sup> Psychomotor function can be impaired after

<sup>529</sup> P.D. Nesbitt, *Smoking, Physiological Arousal and Emotional Response*, 25 J. PERSONALITY SOC. PSYCHOL. 137-144 (1973).

<sup>530</sup> M.V. Djordjevic, et al., *Self-regulation of Smoking Intensity: Smoke Yields of the Low-nicotine, Low-“tar” Cigarettes*, 16 CARCINOGENESIS 2015 (1995).

<sup>531</sup> N.L. Benowitz, et al., *Smokers of Low-yield Cigarettes do not Consume Less Nicotine*, 309 NEW ENG. J. MED. 139 (1983).

<sup>532</sup> *DSM-IV*, *supra*, n. 341, at 244-47.

<sup>533</sup> *Id.* at 264-265.

<sup>534</sup> A.C. Parrott, *Smoking Cessation Leads to Reduced Stress, but Why?*, 30 INT'L. J. ADDICT. 1509-1516 (1995).

<sup>535</sup> A.C. Parrott, *Nesbitt's Paradox Resolved? Stress and Arousal Modulation During Cigarette Smoking*, 93 (1) ADDICTION 27-39 (1998) at 29. There is evidence, however, that even in those not dependent upon nicotine, smoking may heighten alertness, cognitive performance, and the ability to perform while fatigued, A.H. Glassman, *Psychoactive Smoke*, 37 NATURE 677-678 (1996); and that nicotine administration may enhance memory, attentiveness, and reasoning skills. S.J. Heishman, *Behavioral and Cognitive Effects of Smoking: Relationship to Nicotine Addiction*, 1 (suppl. 2) NICOTINE TOBACCO RES. S143-147 (1999).

<sup>536</sup> A.C. Parrott, *Individual Differences in Stress and Arousal During Cigarette Smoking*, 115 PSYCHOPHARMACOLOGY 389-396 (1994).

<sup>537</sup> DHHS, *The Health Consequences of Smoking: Nicotine Addiction. A Report of the Surgeon General, 1988*, Rockville, MD: Public Health Service, CDC, Office on Smoking and Health (1988) (DHHS Publication No. (CDC) 88-8406).

<sup>538</sup> J.R. Hughes, et al., “Effects of Abstinence From Tobacco,” in L.T. Kowzlowski & H.M. Annis, eds., *Recent Advances in Alcohol and Drug Problems*, NY: PLENUM (1990) at 10; J.R. Hughes, et al., *Nicotine Withdrawal vs. Other Drug Withdrawal Syndromes: Similarities and Dissimilarities*, 89 ADDICTION 1461-1470 (1994).

three or more hours of tobacco abstinence.<sup>539</sup> Tobacco withdrawal is also characterized by diminished alertness and cognitive ability, decreases in blood pressure, and disruptions in sleep pattern.<sup>540</sup> As time goes on, full blown withdrawal symptoms set in and may include tremor or shaking, decreased concentration, changes in REM sleep or inability to maintain sleep,<sup>541</sup> gastrointestinal symptoms, and slowing of brain activity on EEG.<sup>542</sup>

### c. Excessive Use

“Individuals who smoke and other individuals who use nicotine are likely to find that they use up their supply of cigarettes or other nicotine-containing products faster than originally intended.”<sup>543</sup> The reinforcing qualities of nicotine and other psychoactive drugs are not only functions of dose, but also critically involve the rate at which the dose is presented to the brain.<sup>544</sup> The use of different nicotine-delivery devices produces different patterns of blood nicotine increase. The more rapidly the nicotine level rises in the blood, the more reinforcing and dependence-producing it is.<sup>545</sup>

### d. Persistent Desire or Unsuccessful Efforts to Cut Down

Although more than 80% of individuals who smoke express a desire to stop smoking and 35% try to stop each year, fewer than 5% are successful in unaided attempts to quit.<sup>546</sup> Every year an estimated 17,000,000 smokers attempt to quit smoking for more than twenty-four hours.<sup>547</sup> According to CDC’s National Health Interview Survey, the number who make any attempt may be closer to 22,000,000 each year, because so many smokers try but relapse in the first 24 hours.<sup>548</sup> Because only about 1,300,000 smokers (about 2.5% of all smokers) succeed in becoming one-year nonsmokers each year,<sup>549</sup> probably more than 20,000,000 smokers annually try but fail to quit smoking.

Smokers’ efforts to quit have created demand for an array of products designed to help. FDA approved the first nicotine replacement, nicotine polacrilex (gum), in 1984.<sup>550</sup> As patches,<sup>551</sup> gums, sprays, lozenges, or inhalers,<sup>552</sup> nicotine replacement

<sup>539</sup> N. Sherwood, *Effects of Nicotine on Human Psychomotor Performance*, 8 HUMAN PSYCHOPHARMACOL. 155-184 (1993) at 157.

<sup>540</sup> M. Al’ Absi, T. Amunrud, & L. Wittmers, *Psychophysiological Effects of Nicotine Abstinence and Behavioral Changes in Habitual Smokers*, 72 PHARMACOL. BIOCHEM. BEHAV. 707 (2002).

<sup>541</sup> See A. Thomas & T.A. Gossel, *The Physiological and Pharmacological Effects of Nicotine*, (Suppl.) U.S. PHARMACIST 1, 3, 4 (1992) at 2.

<sup>542</sup> DSM-IV-TR, *supra* note 341, at 264.

<sup>543</sup> *Id.* at 264-265.

<sup>544</sup> J.E. Henningfield & R.M. Keenan, *Nicotine Delivery Kinetics and Abuse Liability*, 61 J. CONSULTING & CLIN. PSYCHOL. 743 (1993).

<sup>545</sup> J.E. Henningfield, et al., *Higher Levels of Nicotine in Arterial than in Venous Blood After Cigarette Smoking*, 33 DRUG & ALCOHOL DEPENDENCE 23 (1993).

<sup>546</sup> DSM-IV-TR *supra* note 341, at 264-265; M. Russell, *Help and Treatment for Addicted Smokers*, 16 WORLD HEALTH F. 19, 20 (1995). See general, respecting J.I. d., J.E. Henningfield and J. Slade *supra* note 519, at 86-90.

<sup>547</sup> G.A. Giovino, et al., *Trends in Cigarette Smoking Cessation in the United States*, 2 (Supp.) TOBACCO CONTROL S3 (1993).

<sup>548</sup> A.J. Garvey, et al., *Predictors of Smoking Relapse Among Self-Quitters: A Report From the Normative Aging Study*, 17 ADDICTIVE BEHAV. 367 (1992).

<sup>549</sup> J.P. Pierce, et al., *Trends in Cigarette Smoking in the United States: Projections to the Year 2000*, 261 JAMA 61 (1989).

<sup>550</sup> O. Ferno, *The Development of a Chewing Gum Containing Nicotine and Some Comments on the Role Played by Nicotine in the Smoking Habit*, in U.S. Dep’t of Health, Education, and Welfare (DHEW), *Smoking and Health II: Health Consequences, Education, Cessation Activities, and Governmental Action* (DHEW Pub. No. NIH-77-1413) (1977).

<sup>551</sup> T. Abelin, et al., *Controlled Trial of Transdermal Nicotine Patches in Tobacco Withdrawal*, 1 LANCET 7-9 (1989).

<sup>552</sup> J.R. Hughes, et al., *Recent Advances in the Pharmacotherapy of Smoking*, 281 JAMA 72, 75 (1999).

therapy allows nicotine maintenance or reduction while diminishing or eliminating the harms of using tobacco products, allowing smokers to modify their behaviors without additionally having to endure nicotine withdrawal symptoms.<sup>553</sup> Over time the range and availability of medications for treating nicotine dependence and withdrawal have increased substantially. Originally, transdermal nicotine patches required a prescription, but in 1996 they became available over-the-counter.<sup>554</sup> That same year, FDA approved OTC marketing of nicotine gum and two of the four prescription transdermal nicotine patch brands, as well as two new prescription nicotine medications (nicotine nasal spray and an oral vapor inhaler) and a prescription non-nicotine antidepressant (bupropion) for smoking cessation.<sup>555</sup> Bupropion's mechanism of action is unknown, but it does have dopaminergic effects that can produce stimulant-like subjective and behavioral effects that may be related to its efficacy.<sup>556</sup> The long term success rates of these replacement therapies have generally been modest. Abstinence from smoking after six weeks is estimated to be approximately 25% or more (the placebo treatment success rate is roughly half this figure), but thereafter abstinence tends to decline, so that one year later the success rate is only 15%.<sup>557</sup>

#### e. Time Spent

According to APA, "Spending a great deal of time in using the substance is best exemplified by chain-smoking. Because nicotine sources are readily and legally available, spending a great deal of time attempting to procure nicotine would be rare."<sup>558</sup> Where access to tobacco is limited, however, a smoker may spend a great deal of time trying to obtain it. Fifty years ago, Erving Goffman described the underground tobacco economy in asylums.<sup>559</sup> Malnourished prisoners of war traded food rations for cigarettes.<sup>560</sup> Now that cigarettes have been banned from psychiatric hospitals, patients once again might expend much time and energy seeking contraband tobacco products.

#### f. Giving Up Activities

APA has written: "Giving up important social, occupation, or recreational activities can occur when an individual foregoes an activity because it occurs in smoking-re-

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<sup>553</sup> J.E. Henningfield, *Nicotine Medications for Smoking Cessation*, 333 NEW ENGL. J. MED. 1196 (1995).

<sup>554</sup> A. Woolf, et al., *Childhood Poisoning Involving Transdermal Nicotine Patches*, 99 (5) PEDIATRICS E54 (1997) at E5.

<sup>555</sup> It has been reported that bupropion is associated with weight loss. See, e.g., H. Croft, et al., *A Placebo-controlled Comparison of the Antidepressant Efficacy and Effects of Sexual Functioning of Sustained Release Bupropion and Sertraline*, 21 CLIN. THER. 643-658 (1999). On approval of OTC marketing of nicotine gum, see "OTC Nicorette will be available in retail stores by late spring following Feb. 9 approval," F-D-C REP. ("THE TAN SHEET"), (Feb. 12, 1996), at 4. On approval of OTC marketing for the two nicotine transdermal packages, see "SmithKline Beecham's NicoDerm CQ Will Be the Second Transdermal Patch on the Market," F-D-C REP. ("THE TAN SHEET"), (Aug. 5, 1996), at 1.

<sup>556</sup> R.J. Baldessarini, *Drugs and the Treatment of Psychiatric Disorders: Depression and Mania*, in J.G. Hardman & L.E. Limbard, eds., GOODMAN & GILMAN'S THE PHARMACOLOGICAL BASIS OF THERAPEUTICS, 9th ed. (1996) at 431.

<sup>557</sup> J.R. Hughes, et al., *Nicotine Gum to Help Stop Smoking*, 252 JAMA 2855 (1984); J. Foulds, et al., *Effect of Transdermal Nicotine Patches on Cigarette Smoking: A Double Blind Crossover Study*, 106 PSYCHOPHARMACOLOGY 421 (1992); K.A. Perkins, et al., *Nasal Spray Nicotine Replacement Suppresses Cigarette Smoking Desire and Behavior*, 52 CLIN. PHARMACOL. & THER. 627 (1992); P. Tonnesen, et al., *A Double-blind Trial of a Nicotine Inhaler for Smoking Cessation*, 269 JAMA 1268 (1993).

<sup>558</sup> DSM-IV-TR, *supra*, n. 341, at 264-265.

<sup>559</sup> Erving Goffman, *Asylums: Essays on the Social Situation of Mental Patients and Other Inmates*, NY: DOUBLEDAY (1961).

<sup>560</sup> W.M. London, *How Addictive is Cigarette Smoking?*, 12 (3) PRIORITIES FOR HEALTH 11-15 (2000).

stricted areas.”<sup>561</sup> Given the correlation between smoking and depression,<sup>562</sup> and the general decline in activity levels among the depressed, some smokers may indeed curtail some social, occupational or recreational activities. At the entrances to office buildings, even in February, one sees small groups of smokers temporarily banished from the workplace. We have no statistics on the subject, but these workers are clearly absenting themselves from their work stations to indulge their habits for brief periods at intervals throughout the day.

### *g. Continued Use Despite Knowledge of Harm*

Efforts to prevent smoking and to help adolescents quit smoking have met with limited success.<sup>563</sup> As noted above, about one-third of smokers attempt to quit each year, but few succeed. Despite imperative medical reasons, 50% of heart attack survivors and of those hospitalized for other serious smoking-related illness relapse to cigarette consumption within weeks of leaving the hospital.<sup>564</sup> Among those who quit temporarily, “the majority persist in tobacco use for many years and typically cycle through multiple periods of relapse and remission.”<sup>565</sup> FDA has observed that “the individual continues use of the substance despite significant substance-related problems.”<sup>566</sup>

## *2. Nicotine v. Caffeine*

As noted, sources and varieties of tobacco for personal use are relatively few. The same cannot be said of caffeine. Even if we assume that caffeine is addicting, and even if producing an addicting product were *per se* actionable, it would be difficult to hold a soft drink company, for example, liable for such an addiction. There are many sources of caffeine. Among the beverages are coffee, tea, colas, root beer, orange soda, cream soda, and lemon-lime drinks. Among the foods are chocolate and cocoa. OTC drugs may contain caffeine also, including analgesics such as Excedrin, Anacin, Midol; antidrowsiness preparations such as NoDoz, e.g.; and weight-loss aids such as Metabolife and Diet Fuel.<sup>567</sup> If a plaintiff managed to persuade a jury he was “addicted” to caffeine, how is the jury to determine whose product, or products, led to that addiction?

The hundreds of compounds within tobacco smoke are highly and rapidly absorbed from lung. One reason caffeine is not addicting may be the limitations upon its absorption from gut. When a drug is absorbed quickly via the smoked or intravenous routes and thus produces a rapid onset of effect, the potential for abuse and dependence is greater than for the same drug formulated to be absorbed more slowly. A good example

<sup>561</sup> DSM-IV-TR *supra* note 341, at 264-265.

<sup>562</sup> See, e.g., N. Breslau, et al., *Nicotine Dependence and Major Depression: New Evidence From a Prospective Investigation*, 50 (1) ARCH. GEN. PSYCHIATRY 31-35 (1993); G.C. Patton, et al., *Depression, Anxiety and Smoking Initiation: A Prospective Study Over 3 Years*, 88 AM. J. PUBLIC HEALTH 1518-1522 (1998).

<sup>563</sup> S. Sussman, et al., *Effects of Thirty-four Adolescent Tobacco Use Cessation and Prevention Trials on Regular Users of Tobacco Products*, 34 SUBST. USE & MISUSE 1469, 1469 (1999).

<sup>564</sup> J.A. Dani & R.A. Harris, *Nicotine Addiction and Comorbidity With Alcohol Abuse and Mental Illness*, 8 (11) NATURE NEUROSCI. 1465-1470 (Nov. 2005).

<sup>565</sup> The Tobacco Use and Dependence Clinical Practice Guideline Panel, Staff, and Consortium Representatives, *A Clinical Practice Guideline for Treating Tobacco Use and Dependence: A U.S. Public Health Service Report*, 283 JAMA 3244, 3246 (2000).

<sup>566</sup> S.H. Carchman, *Should the FDA Regulate Nicotine-containing Cigarettes? Has the Agency Established a Legal Basis and, if not, Should Congress Grant it?*, 51 FOOD & DRUG L.J. 85 (1996) at 106 & n.154.

<sup>567</sup> R.R. Griffiths, L.M. Juliano, & A.L. Chausmer, *Caffeine Pharmacology and Clinical Effects*, in A.W. Graham, T.K. Schultz, M.F. Mayo-Smith, R.K. Ries, & B.B. Wilford, eds., PRINCIPLES OF ADDICTION MEDICINE, Third Edition, Chevy Chase, Md.: Am. Society of Addiction (2003) at 194; J.R. Hughes, et al., *supra* note 386.

of this is nicotine, which when smoked in the form of cigarettes is highly addictive, but when delivered buccally as a gum or transdermally as a patch has minimal dependence potential.<sup>568</sup> It turns out that caffeine is absorbed slowly, and caffeine in colas is absorbed more slowly than that in coffee and tea.<sup>569</sup> The reason for this difference is unknown.

## V. CONCLUSION

The health impacts of tobacco use are numerous and well-established. The health impacts of obesity are also well established, but in many cases may not be directly attributable to consumption of food, and certainly not to consumption of one particular type of food. Numerous factors entirely beyond the control of farmers and the food industry mightily influence an individual's weight.

Nicotine's power to addict is virtually beyond debate. Theories of food addiction are speculative at best, and the application of established criteria to overeating militates against, not in favor of, the conclusion that foods addict. To the rather minimal extent courts have had an opportunity to consider the question, they have rejected it. Besides *Pelman*, discussed above, the Supreme Judicial Court of Massachusetts recently dismissed in dicta an effort Philip Morris made to draw an analogy between its products and others with some potential to be hazardous: "Sugar, suntan oil, guns, and aspirin are not inherently addictive to the general public or incapable of being used reasonably."<sup>570</sup>

Ultimately, critics of industry have no choice but to agree with the concession Prof. Banzhaf makes: "Obviously there are very important differences between the problems of smoking and obesity. The argument that nicotine is addictive, and thus that smokers are not fully responsible for their actions, has no counterpart with food. Foods are not harmful when used in moderation, whereas cigarettes are."<sup>571</sup>

Even in the tobacco context, litigation has its limitations. Tort awards provide money judgments for smokers, but often fail to compel retailers to check the age of cigarette purchasers, dictate the character of tobacco advertising, or punish the position of cigarettes.<sup>572</sup> It also has its casino aspects. "Tort is a haphazard public health strategy because it is powerfully influenced ... by ever-changing normative judgments about the scruples of the contestants and extraordinary investments of lawyering activity and attempting to stage an effective appeal to moral sensibilities."<sup>573</sup>

These shortcomings are not unique to tobacco claims. They will arise in obesity litigation as well. Such lawsuits will obviously impose needless costs on food companies. Those costs will, of course, be passed on to consumers, which means: all of us. But financial harm is by no means the only reason to discourage this form of litigation. Bringing claims against food companies for obesity may actually harm those the litigation is theoretically designed to help. Emphasizing weight loss, rather than exercise, may inadvertently discourage physical activity, even among the non-obese.<sup>574</sup> Harm will be particularly likely if plaintiffs' counsel succeed in persuading the public that

<sup>568</sup> S.J. Heishman & J.E. Henningfield, *supra* note 506.

<sup>569</sup> V. Marx & J.F. Kelly, *Absorption of Caffeine From Tea, Coffee and Coca-Cola*, 301 LANCET 827 (1973).

<sup>570</sup> Haglund v. Philip Morris, Inc., *supra* n.8, 446 Mass. at 751, 847 N.E.2d at 325.

<sup>571</sup> J. Banzhaf, SAN FRANCISCO DAILY JOURNAL, (Feb. 4, 2002), <http://banzhaf.net/docs/whopay.html>, (last visited July 12, 2006), linked to in Oct. 2004 from one of Banzhaf's now-defunct websites, <http://banzhaf.net/obesity/links.html>.

<sup>572</sup> Robert L. Rabin, "The Third Wave of Tobacco Tort Litigation," in R.L. Rabin & S.D. Sugarman, eds., REGULATING TOBACCO 201 (2001).

<sup>573</sup> *Id.* at 200.

<sup>574</sup> A.K. Yancy, et. al, *Ethnic and Sex Variations in Overweight Self-Perception: Relationship to Sedentariness*, 14 (6) OBESITY 980-988 (June 2006).

overeating is an addiction, a disease. This model denies autonomy to the obese,<sup>575</sup> and teaches them that they are powerless over their own behavior.<sup>576</sup> This sends the wrong message. The disease model excuses behavior that ought not be excused and disables the overeater from helping himself.<sup>577</sup> As noted above, conflating overeating with addictions may hamper efforts to treat the addicted, by trivializing their problems, undercutting the scientific understanding of the pathophysiology of addiction, or both. Thus, the public health consequences of obesity litigation could be far more harmful than the financial costs they will impose. Then, too, products claims erode the philosophical pillars on which American freedom and democracy stand.<sup>578</sup>

Litigation against the food industry, even if it fails as it should, will hamper the efforts of all producers. It will tend to hinder innovation that could benefit us all, and decrease the variety and palatability of the American diet. Claims could ultimately be brought against farmers, who grow the food, against marketers, who promote it, and against the media, which print or broadcast the ads. Worse, obesity litigation will further erode the sense of personal responsibility on which the nation was founded. Food is not tobacco, food companies are different from tobacco companies, and the obese should seek help not from lawyers but from doctors and, more important, from themselves.

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<sup>575</sup> Speaking at the funeral of an alcoholic, a character in a recently published novel gave eloquent voice to this problem: "I just don't think it credits a man's life to say he was in the clutches of the disease and that's what ruined him. Say [the decedent] was too loyal. Say he was disappointed. ... But give him some credit for feeling, for having a hand in his own fate. Don't say it was a disease that blindsided him and wiped out everything he was.' He bit off a drink, his face flushed. 'Do the man that favor, please.'" Alice McDermott, *Charming Billy*, DELL PUBLISHING: NY(1998) at 23.

<sup>576</sup> G. Peele, *Diseases of America* (1989) at 79, 80; H. Fingarette, *HEAVY DRINKING* (1998). See also, note 356, *supra*, and associated text. "Steadiness and constancy in one's personal life reduces the risks from all quarters, of which obesity is only one." R.A. Epstein, *supra* note 112, at 1366.

<sup>577</sup> G. Peele, *supra* note 576.

<sup>578</sup> D.J. La Fetra, *Freedom, Responsibility and Risk: Fundamental Principles Supporting Tort Reform*, 36 IND. L. REV. 645 (2003).