

The Gap Between What We Know and What We Do About Childhood Obesity: A Multi-factor Model for Assessment, Intervention, and Prevention

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Childhood obesity in the United States has increased alarmingly and much is becoming known about multiple factors that contribute to this epidemic. These include environmental (e.g., prevalent junk foods), behavioral (e.g., inactivity), intrapsychic (e.g., depression), interpersonal (e.g., parenting style), as well as biological, socioeconomic, and cultural factors, to name just a few. A comprehensive multi-factor model of childhood obesity is presented based on extant research and its implications for assessment, intervention, and prevention are explored. Emphasis is placed on the importance of developing sound assessment approaches, on the potential value of treatment matching using interventions focused on assessment results on each child's unique combination of causal factors, and more broadly on public health implications for prevention.

Keywords: childhood obesity, obesogenic environment, treatment matching

In a well-conducted study recently published in the *Journal of the American Medical Association*, Hedley et al. (2004) demonstrated a marked increase in the prevalence of obesity in the United States over the last twenty years, finding that among adults (age 20 years or more) 65.1% are overweight, 30.4% are obese, and 4.9% are extremely obese, while among children (age 19 years or less) 31.0% are overweight or at risk for becoming overweight. Considering that increasing percentages of overweight

children will likely swell the ranks of overweight adults (Institute of Medicine, 2005; Lobstein, Baur & Uauy, 2004), concern about childhood obesity in the United States has increased dramatically in recent years (IOM, 2005; Lobstein et al., 2004; Ogden, Flegal, Carroll, & Johnson, 2002).

The economic consequences of childhood obesity alone are staggering. For adults, the medical consequences of obesity (including diabetes, cardiovascular disease, stroke, and cancer) has been estimated to cost \$70-100 billion in the United States (Olshansky et al., 2005), so the increasing number of children with obesity is likely to inflate this already astounding cost. In addition, Hampl, Carroll, Simon, and Sharma (2007) found that “increased health care utilization and charges reported in obese adults are also present in obese children” (p. 11) when compared to normal weight children, a trend they “observed even in children younger than 10 years” (p. 14). And, of course, the cost of childhood obesity is not just monetary, as quality of life and life expectancy will also be gravely impacted. Olshansky et al. (2005) called childhood obesity “a threatening storm” (p. 1139) and Goldfield and Epstein (2002) called childhood obesity “one of the most serious pediatric health problems in the United States” (p. 573).

It should be noted that there are some who dispute these dismal forecasts. For example, the actual magnitude of obesity-related increases in mortality has been challenged and government health agencies have recently given inconsistent estimates of deaths attributable to obesity, ranging from as little as 25,000 to more than 300,000 annually (Gibbs, 2005). But there is no question that that problem is increasingly trickling down to children and is associated with chronic disease, increased mortality, and personal distress, as well as social and economic discrimination. We believe the term “epidemic” is warranted in describing the prevalence and increase in childhood obesity.

In searching for ways to address this growing epidemic of childhood obesity, we should note two things. First, existing treatments for childhood obesity have demonstrated only modest success at best (Faith, Saelens, Wilfley, & Allison, 2001; Tanofsky-Kraff, Hayden-Wade, Cavazos, & Wilfley, 2003). Second, the main factors usually targeted for treatment, poor diet and physical inactivity, are merely symptoms of deeper causes of obesity, as these two factors do not deeply explain *why* so many children overeat and under-exercise. These two factors may be the end points of a chain of influences, the most obvious mechanisms by which antecedent causes are acted out. In order to more deeply understand the causes of childhood obesity, we explore a complex multi-factor model of possible causal mechanisms leading to this problem in the hope that this will lead to more effective treatment interventions and prevention efforts.

Causal Factors Associated with Childhood Obesity

The common belief that obesity is caused only by overeating and inactivity has provided a fertile ground for companies that specialize in diet and exercise products and services. However, treatments focusing solely on these two factors have not been generally effective (Tanofsky-Kraff et al., 2003), suggesting that other factors are involved (Bray, 2003; Speiser et al., 2005). Below we survey the many factors that have been described as contributing to childhood obesity.

Environmental Factors: The Obesogenic Environment

Environmental causes of obesity include availability of fast food, low cost of fattening foods, increased portion sizes, and decreases in physical activity attributable to

energy-saving machines, such as the automobile, elevators, escalators, and computers (Brownell, 2002a). The last-named problem is intensified by cell phones, labor-saving kitchen gadgets, leaf-blowers, robot vacuum cleaners, and the like, which relieve people of even the simplest physical tasks. The World Health Organization summarized studies that linked obesity with “a decline in energy expenditure that is associated with a sedentary lifestyle --- motorized transport, labour-saving devices in the home, the phasing out of physically demanding manual tasks in the workplace, and leisure time that is preponderantly devoted to physically undemanding pastimes” (WHO, 2003, p. 2). This report concludes that the evidence linking sedentary lifestyles to obesity is convincing (p. 62).

Meanwhile, the food industry produces enough to feed each American 3,800 calories a day (Nestle, 2002), far more than the 2,000 calories used as the standard figure in nutritional reports (United States Department of Health and Human Services, 2005) or the 2,247 calories of the average daily Recommended Energy Allowance (Kantor, 1999). With rates of adult overweight and obesity soaring to over two thirds of the U.S. population, obesity has become normative. One prominent obesity expert says unequivocally, “The only hope for changing the prevalence of obesity is to address these environmental causes” (Brownell, 2002a, p. 433).

Sedentary lifestyle. Television, computer games, and video games provide stimulation and amusement that many children find reinforcing (Goldfield, Raynor, & Epstein, 2002) and often choose over physical activities. Second, automobile traffic, fear of violence, and highly publicized stranger abductions make many parents reluctant to allow their children to play outside (Cohen, 2000). Third, parents are not always present. Tanofsky-Kraff et al. (2003) contend that unsupervised children are more likely to spend longer amounts of times in sedentary activities. Many single-parent households and two-parent two-career households lack time (or the resources to pay someone else) to transport children to and from activities (Cohen, 2000). Fourth, many families cannot afford fees and equipment for after-school activities (Cohen, 2000). Cohen (2000) conducted focus groups made up of parents and children in 19 ethnically and geographically diverse communities in the state of California. Participants cited “A lack of money at home,” as one of the top reasons that children do not participate in physical activities by both the adults and the children in the focus groups (p. 15).

Junk food and beverages. Children today are surrounded by “junk foods,” defined here as inexpensive, easily available food of poor nutritional quality. Junk foods are available even at school. For example, 95% of responding California schools reported the sales of fast food on school grounds, increasing meals consumed that are high in added sugar, sodium and fat (California Legislature, 2001). Many school districts contract with soft drink companies, agreeing to sell only one company’s soft drinks in exchange for a large payment (Brody, 2002). Such “pouring contracts” may forbid a school district from putting milk into vending machines (Simon, 2006, pp. 123, 221) or require the district to sell a minimum amount of soda (Brownell, 2002a). Furthermore, children are encouraged to raise funds for their schools or after-school activities by selling consumer products. One trade association (see www.afrds.org) represents 650 companies that manufacture or distribute junk food products (e.g., candy, pies, cookies, and pretzels) to non-profit organizations and schools to re-sell for fundraising

Junk food is also widely available at 24-hour fast-food restaurants, convenience

stores, and gas stations. Fast food restaurants provide busy parents, especially single parents and dual-career parents (Cohen, 2000), a quick and easy way to feed their children; some even have alluring playground areas. To protect this lucrative market from regulation, food and beverage corporations set up front groups (e.g., see www.consumerfreedom.com), which deny problems, disparage critics, and wage nationwide campaigns to minimize the perception of obesity's dangers, as well as lobby vigorously against regulation and give "grants" to supposedly impartial third parties, including the American Academy of Pediatric Dentists (Kanner & Golin, 2005).

To families on a tight budget, getting more food for a small price hike is an attractive option. *Supersizing* means to get a bigger portion of fries or soda for a small additional charge. The current supersized portion of fries at McDonald's contains 610 calories; by contrast, the only size of fries available at McDonald's in the 1950s and 1960s contained just 200 calories (Brody, 2002). Other fast food restaurant chains have followed the lead of McDonald's in offering inexpensive larger portions (Cohen, 2000).

Food advertising. Americans are barraged by food advertisements fueled by huge advertising budgets: in 2002, McDonald's spent \$1.1 billion and Coca-Cola spent \$866 million on advertising (Brownell, 2002a). In stark contrast, the National Cancer Society spent \$1 million in 2002 to promote healthy eating (Brownell, 2002 a). Many food advertisements target young audiences using popular movie and cartoon characters (Simon, 2006). Horgen, Choate, and Brownell (2001) found that the average American child watches 10,000 television food advertisements per year, 90 - 95% of them for fast food, candy, soda, and sugared cereals. Advertisers skillfully manipulate children, with the regrettable complicity of some consumer psychologists (Levin & Linn, 2004), who help corporations to use psychologically sophisticated tactics to gain "share of mind" and "life-time brand loyalty" by "viral marketing" and inculcating in children "the nag factor," "pester power," and "the fine art of whining." The marketers succeed, all too well: the WHO report concluded that heavy marketing of junk food is a probable cause contributing to obesity (2003, p. 65).

Media images and body image. Television, movies, magazines, games, and the Internet are filled with slim, attractive models and actors, with few average or plus-sized children and adults. The media image of women accurately represents fewer than 5% of actual Americans (Garner, 1997) and has become progressively slimmer in the last four decades (Sypcck, Gray, & Ahrens, 2004). Advertisements for weight-loss products and services add to the barrage of messages about the unacceptability of some body shapes, which can lead to "thin-ideal internalization" (Stice, 2002) and to body dissatisfaction (Tiggemann & Slater, 2004). The media image is a major contributor to some eating disorders, especially in girls (Garner, 1997; Stice, 2002), by inculcating body dissatisfaction that can lead to dieting and its unintended consequence, overeating.

Poverty. The USDA estimated that in 1998, 20% of American children were "food-insecure" (i.e., hungry or worried about hunger; Gardner & Halweil, 2000). Two years later, nearly one third of households headed by single women were food-insecure (Schwartz-Nobel, 2002). Food insecurity can create a "feast or famine" mindset, leading a food-insecure person to overeat when food is available (Dietz, 1995).

In sum, today's children are growing up in an environment filled with fast food, sugary beverages, heavily processed food, a barrage of advertisements, a paucity of opportunities for exercise, and media images of impossible slimness and muscularity –

an environment which Brownell and Horgen (2004) justly characterize as “toxic.” Schwartz (quoted in Okie, 2005, p. 89) summed it up well: “If you wanted to see what is the environment that would maximize the rate of weight gain – we’ve more or less created it.”

Behavioral Factors

Physical inactivity. Reductions in physical activity contribute to childhood obesity (French, Story, & Jeffery, 2001; Wadden, Brownell, & Foster, 2002). Children get less physical education at school, one result of political pressure to raise academic test scores and cut school budgets (Brody, 2002). Leisure activities are often sedentary, such as watching television, spending time on computers, or playing video games. Gortmaker et al. (1996) noted that children watch an average of 28 hours of television per week and that there is a direct correlation between hours of television viewing and obesity. A longitudinal study tracking 1,000 individuals from birth to 26 years of age found a statistically significant association between increased television viewing during childhood/adolescence and later health problems in adulthood (e.g., being overweight, poor fitness, smoking, and elevated cholesterol; Hancox, Milne, & Poulton, 2004), even after controlling for childhood socio-economic status (SES), body mass index (BMI) at age 5, parental BMI, parental smoking and physical activity level at age 15. It is unclear whether increased consumption of snacks or inactivity itself is the culprit (Kanders, 1995). Numerous researchers have discussed the link between television watching and obesity (Brownell, & Foster, 2002; Cohen, 2000; French, Story, & Jeffery, 2001; IOM, 2005; Lobstein et al., 2004). Excess weight may make it difficult for some obese children to participate in physical activities. Therefore, television viewing may be one of the few possibilities of recreation for them. No studies could be located that further explore this possibility.

Diet (food composition). Ritchie, Woodward-Lopez, Ivey, Gerstein, and Crawford (2005) reviewed childhood obesity publications from 1992 to 2004 and found six dietary intake patterns associated with child obesity: (a) high consumption of sugar-sweetened beverages, (b) high intake of food with large percentages of calories from fat, (c) low intake of fiber, (d) low intake of fruit and vegetables, (e) low intake of low-fat dairy products, and (f) skipping breakfast. In addition, Americans spend half of their food budget eating meals outside the home, twice as much as in 1970, and restaurant food typically contains 22% more fat than home-cooked meals (Winter, 2002). Especially unhealthy is fast-food consumption, whether eaten in restaurants or taken home.

Diet (quantity). Clearly, calorie intake in excess of expenditure is a major cause of obesity. A person may eat more frequently, eat more calorie-dense foods, eat larger meals, or binge. Binge eating is a behavior in which an individual consumes large amounts of food in a short time. If this activity is repeated and uncontrollable, an eating disorder (e.g., Bulimia Nervosa or Binge Eating Disorder) may be diagnosed (American Psychiatric Association, 2000). Overeating figures prominently in our model. Many factors described in this review operate by encouraging overeating.

Dietary restraint (physiological effects). Paradoxically, dietary restraint (restricting food intake to lose or maintain weight) may lead to more eating and weight gain (Faith, Matz, & Allison, 2003). Birch and Fisher (1998) stated, “Dietary restriction involving cognitive restriction of food intake involves explicit denial of hunger cues and includes stopping eating while still hungry and skipping meals” (p. 546). Furthermore,

the metabolic rate is lowered during food deprivation and remains low after normal eating is restored (Miller & Wadden, 2004).

Intrapsychic Factors

Object relations theorists note psychodynamic processes that can contribute to eating disorders, such as punitive introjects, split-off portions of the self, dissociation, and deficits in attachment, separation/individuation, and internal regulation of affect (Goodsitt, 1997). Many other intrapsychic factors may influence child obesity.

Dietary restraint (psychological effects). Dietary restraint can also promote counter-purposeful psychodynamic processes. The dieter may be ambivalent about losing weight or rebel against self-imposed restraints. The abstinence-violation effect (Carels, Douglass, Cacciapaglia, & O'Brien, 2004) can cause dieters to respond to minor slips by giving up their intention to eat less and then eating more than if they were not dieting. Dietary restraint has been found in children (Shunk & Birch, 2004) and has been found to predict the risk of obesity in teenagers (Stice & Cameron, 1999). Such well-documented effects can make dieting a self-defeating endeavor, which many nevertheless attempt repeatedly. Contemplating the fact that dieting rarely works and almost always precedes eating disorders, Polivy and Herman concluded long ago, "Perhaps dieting is the disorder we should be attempting to cure" (1985, p.200).

Affect, Low Self-esteem, Low Body-esteem and Cognitive Distortions

Depression, anxiety, low self-esteem, low body-esteem and cognitive distortions can also contribute to obesity in individuals who eat to cope with low self-esteem, body dissatisfaction, and emotional problems. It is unclear whether these negative states lead to excessive eating causing obesity or whether obesity leads to the negative states (Yanovski, 2002).

Depression. Food is a very effective regulator of mood and the line between medications and food can be blurred (Friedman, 1999). When eating is used persistently to regulate mood, some individuals lose the capacity to recognize hunger and satiety cues and ultimately eat when experiencing discomfort of any kind. Binge eating has been associated with negative affect and affective disorders (Bulik, Sullivan, & Kendler, 2002; Faith et al., 2003; Friedman & Brownell, 2002). Some research has shown increased incidence of emotional problems in obese children as compared to normal-weight children (e.g., Ringham, Levine, Kalarchain, Wisniewski, & Marcus, 2004) and significant depression and anxiety in obese adolescent bingers (Isnard et al., 2003). These studies, however, only show relationships between mental health indicators and obesity, but have not established casual pathways (i.e., have not established that obesity per se causes mental health problems, as opposed to the possible physical and social consequences of obesity).

Anxiety and stress. Some researchers found that obese individuals do not eat more when anxious (Oliver, Wardle, & Gibson, 2000). One study indicated that obese people eat more when experiencing free-floating anxiety (anxiety with no clear source) than normal-weight people who were also experiencing free-floating anxiety (van Strien & Ouwens, 2003). In one additional study, individuals who reported higher levels of stress also reported exercising less and eating less healthy diets than individuals who reported lower levels of stress (Ng & Jeffery, 2003, p. 638).

Low self-esteem and low body-esteem. For some individuals, low self-esteem and low body-esteem lead to overeating and lower levels of physical activity. According to

Tanofsky-Kraff et al. (2003), individuals who loathe themselves and their bodies are more likely than others to develop unhealthy lifestyles. Low body-esteem can also discourage heavy individuals from exercising outdoors or in public gyms (e.g., low body-esteem could possibly discourage heavy individuals from exercising outdoors or in public gyms). Once more, this only suggests relationships between esteem and obesity, but has not established casual pathways (i.e., has not established that obesity per se causes esteem problems, as opposed to the physical and social consequences of obesity).

Cognitive distortions and explanatory style. Obese adults are more likely than normal-weight adults to have weight-related cognitive distortions (O'Connor & Dowrick, 1987) such as "I will always be fat, no matter what I do" or "Everyone else can lose weight except me." Such learned helplessness can be tied to specific tasks or situations; a person may feel effective in work situations but helpless in terms of weight, whereas other people do not develop learned helplessness at all, or recover their initiative when conditions change (Seligman, 1991, 1995). As noted previously, depression, which is often a sequel of learned helplessness, is a risk factor for childhood obesity.

Interpersonal Factors

Interpersonal factors that contribute to obesity in children include neglect and family dysfunction, parental control in feeding and preoccupation with weight, stigmatization, and sexual abuse or other childhood trauma.

Neglect and family dysfunction. Lissau and Sorensen (1994) found that children who were perceived by teachers and school nurses to be neglected by their parents were more likely than other children to be obese by the age of 14. In one study, obesity in adolescent girls was found to be related to lower family cohesiveness, lower communicative expressiveness, and lower democratic family decision-making style as compared to normal-weight controls (Steinberg & Phares, 2001).

Parental feeding style. Parental prompting to eat was associated with increased food consumption and overweight in children (Klesges, Malott, Boschee, & Weber, 1986). Parents may be controlling in other ways by enforcing food rules, such as "You must eat your vegetables at dinner" and "If you put it on your plate, you have to eat it" (Puhl & Schwartz, 2003, p. 288). Faith et al. (2003) found, "Greater maternal control during feeding is associated with poorer caloric regulation by the child and increased child body fat" (p. 24). Over time, children who are prompted by parents to eat and to "clean their plates" become less prone to heed their bodies' hunger and satiety cues and, as adults, have higher rates of binge eating, weight cycling, and dietary restraint than adults who grew up in households without such rules (Puhl & Schwartz, 2003). Johnson and Birch (1994) found that the best predictor of children's ability to adjust their intake was the degree of parental control in the feeding situation. Also contributing to childhood obesity is the use of food by parents to calm, reward, or motivate a child (Agras, Hammer, McNicholas, & Kraemer, 2004).

Parental weight and parental preoccupation with weight. Parents' overweight is a risk factor for childhood eating disturbances (Stice, Agras, & Hammer, 1999) and childhood overweight (Agras et al., 2004). Parents' preoccupation with weight (their own or their children's) can influence children and adolescents to overeat (Stice et al., 1999) or conversely to value slimness and begin dieting (Field et al., 2004), which, as we have noted, is itself a risk factor for overweight. Of course, these relationships between parental factors and obesity may have some genetic predispositions (discussed later in

this paper).

Stigmatization and identity as fat. Taunting, rejection, and exclusion are often directed toward obese adults and children. Such experiences may deepen depression (Furman & Thompson, 2002), discourage a child from joining sports and other activities, and solidify an identity as a fat person. A history of being ridiculed for being fat often precedes an eating disorder (Neumark-Sztainer, Falkner, Story, Perry, & Hannan, 2002). Family members, including parents, may also engage in taunting the obese child (Schwartz, Phares, Tantleff-Dunn, & Thompson, 1999). These findings, once more, suggest but do not show possible causal relationships between obesity and mental health concerns.

Sexual abuse and other childhood trauma. There appears to be a connection in some individuals between obesity and sexual abuse (Wonderlich et al., 2001) or other childhood traumas (Felitti et al., 1998). Disordered eating helps the individual cope with negative affect and assemble a sense of self. Polivy and Herman (2002) captured this well by stating, “By refocusing one’s attention onto weight, shape, and eating, one enters a domain in which one can gain some emotional control” (p. 196). This preoccupation distracts from abuse and can provide an adaptive identity, albeit the painful identity of a person struggling with weight issues.

Sexual abuse is part of a continuum of sexual discrimination and harassment, which differentially affects females (Smolak & Murnen, 2001). Clark and Goldstein (1995) observed that successful weight loss can unmask the relationship between obesity and sexual abuse or attention:

Obesity may have the psychologically positive effect of minimizing sexual contact (either verbal or physical) and enhancing feelings of safety or security. Subsequently, weight loss may cause psychological distress as individuals experience increased sexual attention from others, increased feelings of vulnerability, or increased numbers of flashbacks. (Clark & Goldstein, 1995, p. 160)

This accords with the clinical observations of one of us (LR), who treats eating disordered patients and routinely asks obese women, “Can you think of any disadvantages to solving your weight problem?” In her 25 years of psychotherapy practice, she notes her clients almost invariably reply, “I would have to do something about attention from men.”

However, research shows only a modest association between sexual abuse and eating disorders (Gustafson & Sarwer, 2004; Smolak & Murnen, 2002). Felitti et al. (1998) note that sexual abuse is a risk factor for many psychological disorders, including eating disorders and obesity. Everill and Waller (1995) and Smolak and Murnen (2002) attribute the lack of certainty about this correlation to various mediating and moderating factors. Once more, these relational patterns do not demonstrate causality, since intervening variables could be the underlying cause (e.g., obesity may be linked with both lower SES and the higher likelihood of experiencing childhood traumas, with SES being the actual cause of both incidences of obesity and trauma).

Other childhood traumas may be relevant. Felitti et al. (1998) found a strong correlation between childhood traumas and medical or psychological disorders in

adulthood. Participants who reported four or more traumas (e.g., a parent in jail, a parent abusing alcohol or drugs, domestic violence in the home, mental illness of a parent, divorce, parental rage reactions and recurrent physical or emotional abuse by a parent) had a much higher prevalence of poor health habits (e.g., smoking), psychological disorders (e.g., depression), medical problems (e.g., hepatitis), and obesity. Again, the same point previously made about lack of evidence for causal patterns applies.

Biological Factors

The nature-or-nurture question challenges researchers in many areas, including childhood obesity. Offspring of overweight parents are more likely to be overweight than children of normal-weight parents (Whitaker, Wright, Pepe, Seidel, & Dietz, 1997), which suggests that obesity might be influenced by biological factors in addition to socialization, such as shared lifestyle and eating habits. Bottle-feeding during infancy was positively correlated to body weight in some studies (Wilfley & Saelens, 2002) but not others (Li, Parsons, & Power, 2003), and timing of weight gain in the first weeks of life has also been mentioned (Stettler et al., 2005), suggesting developmental milestones that might be biological. Certain medical conditions and medications are clearly associated with obesity: endocrine disorders, Cushing's syndrome, hyperinsulinism, growth hormone deficiency, hypothalamic syndromes, and other rare genetic syndromes (Bray, 2003, 2004). Weight gain is a demonstrated side-effect of some antidepressants, anti-psychotics, antihistamines, and steroid-based medications used to treat severe asthma and other inflammatory diseases, which are prescribed widely for children (Bray, 2003). Prenatal conditions that might be biological have also been named, including prenatal over-nutrition and undernutrition, gestational diabetes, and maternal smoking (Lobstein et al., 2004). Insufficient sleep appears to be correlated with obesity (Bass & Turek, 2005; Flier & Elmquist, 2004), including childhood obesity (Agras et al., 2004), possibly by increasing the stress hormone cortisol. Evidence suggests that other biological factors may be associated with obesity.

Genes. Genes can contribute to obesity in multiple ways, including variations in metabolism (Beamer, 2003), fat cell number (Brownell & Wadden, 1992), and how excess fat is distributed on the body (Beamer, 2003). A genetic predisposition towards anxiety or depression can also underlie a tendency to use food to regulate mood, as discussed earlier. No specific genes are associated with all cases of obesity. The gene that has received the most attention is the “ob” gene, responsible for the secretion of leptin in the body. Mice bred with a defect in their ob gene produced no leptin and overate, becoming lethargic and obese unless they received regular doses of recombinant leptin (Wadden et al., 2002). However, laboratory tests in humans found “very few, very rare mutations identified to cause significant obesity” (Beamer, 2003, p. 48). And only a few obese individuals tested were found to have low leptin levels (Wadden et al., 2002).

The Melanocortin 4 Receptor (MC4-R) gene mutation is associated with excessive hunger, overeating, binge eating, and food-seeking behavior (Branson et al., 2003). One study found an MC4-R gene mutation in 4% of children with severe, early-onset obesity (Vaisse et al., 2000) and another found it in 2.4% of severely obese children (Dubern et al., 2001). Researchers hypothesize that this mutation interferes with the appetite control centers in the hypothalamus. Thirty different mutations in the MC4-R gene seem linked to obesity (Branson et al., 2003). However, the dramatic increase in obesity within a single generation shows that genes are not the sole causes of obesity

(Wadden et al., 2002).

Metabolism. Resting metabolic rate (RMR) is another mechanism likely influenced by genes. RMR is the amount of energy expended during inactivity (just to maintain life) and accounts for approximately 70% of daily calorie expenditure in sedentary individuals (Ravussin & Bogardus, 1992). As many veteran dieters ruefully acknowledge, RMR can vary greatly between individuals by as much as 1,000 calories per day (Foster et al., 1988). Low RMR is a risk factor for overweight and obesity, even when body weight and body composition are controlled for (Clark & Goldstein, 1995). In a meta-analysis, Astrup et al. (1999) found that formerly obese individuals had a 3-5% lower RMR than a never-obese control group. It is unclear whether the previously obese individuals had a lower RMR because of their weight loss or because of pre-existing biological make-up. The set-point theory postulates a homeostatic mechanism that controls body weight within a specific range (i.e., set-point), possibly by affecting metabolism (Clark & Goldstein, 1995; Keeseey, 1995).

The number of fat cells is also a factor. Mildly obese people have about the same number of fat cells as normal weight people, but their fat cells are larger (Brownell & Wadden, 1992). Only when an individual becomes extremely obese does the number of fat cells dramatically increase (Brownell & Wadden, 1992). Fat cells can shrink, but not disappear (Bray, 2003) unless surgically removed. Other weight-related physiological processes may be genetically influenced: fat storage, thermogenesis (i.e., the process associated with the number of calories burned during digestion and exercise), and appetite/satiety (Beamer, 2003).

Social Factors

Socioeconomic status. In developed countries, there is a correlation between SES and obesity in females, especially those in ethnic minorities (Bray, 2004). Jeffery and French (1996) offer three hypotheses: (a) Obese women may have less opportunity for economic advancement due to discrimination and therefore remain in lower socioeconomic groups; (b) Women in lower socioeconomic groups may have less access to health education, healthy foods, and safe exercise; (c) Women of lower SES may have less time and energy for healthy diet and exercise and are more worried about basic necessities than about their weight.

Culture and ethnicity. The rate of obesity among ethnic minority children at all ages is higher than that among white children (Kumanyika, 2002). It is unclear whether this is due to lower SES, a sedentary lifestyle, differences in eating behavior, genetic differences, or a combination of these. According to one theory, certain racial groups contain “thrifty genes” that helped them to survive during historic periods of food deprivation by getting the most calories out of a given quantity of food. Studies comparing metabolism rates in ethnic groups have found mixed results for this theory (Jones et al., 2004; Sun et al., 1998).

Other Possible Factors

There are many other possible factors influencing childhood obesity, such as those from environmental causes. Industrialized food production (e.g., use of pesticides, hormones, and antibiotics), processing (e.g., use of salt, fats, preservatives, and corn sweeteners), and preparation (e.g., frying, canning) can exacerbate obesity in many plausible ways (e.g., Riebel, 2001). Similarly, genetic modification of food products introduces additional untested changes into our food supply. On a subtler level, the

distance from farm to table makes food a commodity and detaches consumers from respect for the sources of life.

Proposed Model of Causes of Childhood Obesity

Reflecting on the many theories of childhood obesity, Brownell and Wadden (1992) contend that each explanation is valid, “as applied to certain individuals.... Obesity is a heterogeneous disorder with multiple etiologies, and hence, multiple risk factors” (p. 505). We agree with this approach and propose there is great value at looking more complexly at the issue, rather than focusing only on the usually emphasized two main factors, diet and exercise. Also, instead of trying to find one or a few universal causes of obesity, researchers and clinicians might do better to find ways to determine how different causes may operate in each individual. Hence, the number and range of hypotheses in the literature support the need for a multi-factor model. Although some multi-factor models of causation and treatment or prevention of obesity exist (Bray, 2003; Cooper & Fairburn, 2002; Dietz & Gortmaker, 2001; Feuerstein & Papciak, 1989; Kumanyika, 2004; Lobstein et al., 2004; Mellin, 1986; Wing, 2004), none of the extant models incorporate the whole range of factors we have reviewed.

Consequently, we propose that the dozens of factors which contribute to obesity exist in a hierarchy of at least two levels (factors and mechanisms) and that each contributory factor operates through one of the following five mechanisms: encourages overeating, discourages exercise, promotes eating unhealthy foods, defeats resolutions about healthy eating and exercise, and impacts physiological processes. Some factors contribute to more than one mechanism and some mechanisms are associated with more than one factor. We assume that, absent genetic disorder or a clear medical condition (e.g., hormonal imbalance or brain lesion affecting mechanisms controlling appetite and satiety), the human organism will, when food is available, usually eat when hungry and stop eating when full. We disagree with Pinel, Assanand, and Lehman (2000), who proposed that overeating is genetically programmed to occur in common circumstances. Overeating at a level sufficient to cause obesity is considered in our model not to be a primary cause of obesity but to spring from other causes that are more fruitful to understand. Figure 1 suggests how factors may operate through mechanisms to create obesity in a hypothetical case.

The first mechanism, “encourages overeating,” includes dietary restraint, parental neglect, and depression. “Discourages exercise” includes absence of sports in schools, depression, and low body-esteem. “Promotes eating unhealthy foods” includes parental neglect, dietary restraint, and the omnipresence of junk foods and beverages. “Defeats resolutions about healthy eating and exercise” includes cognitive distortions, family pressure, and negative weight evaluations. “Impacts physiological processes” includes amount and kind of exercise, dietary restraint, and genetic factors. Table 1 demonstrates how selected factors and the mechanisms may operate to create obesity.

Table 1
Means by which Selected Factors May Operate through Five Mechanisms to Foster Obesity

Causal Factors	Encourages Overeating	Discourages Exercise	Promotes Eating Unhealthy Foods	Defeats Healthy Resolutions	Physiological Processes
<i>Environmental Factors</i>					
Fast food availability	X		X		
Slim media image				X	
<i>Behavioral Factors</i>					
Inactivity		(by definition)			X
Ignoring hunger and satiety cues	X				
<i>Psychological Factors</i>					
Depression	X	X		X	
Explanatory style	X	X	X	X	
Poor body-esteem	X	X		X	
<i>Interpersonal Factors</i>					
Family neglect	X	X	X	X	
Sexual abuse	X				
<i>Biological Factors</i>					
Low metabolic rate					X
Genetic mutations					X
<i>Social Factors</i>					
Poverty	X	X	X		
Single-parent or 2-income families	X	X	X		

Discussion

The recognition that multiple factors may cause childhood obesity offers many practical applications for successfully addressing child obesity. It is our opinion that an inclusive multi-factor model, such as presented, best describes causes of childhood obesity and would be the most effective approach for guiding treatment and prevention efforts. We believe that the most beneficial way to proceed now involves first focusing on developing adequate multi-factorial assessment techniques and second on multi-factorial intervention and prevention strategies.

Approaches to better measure these factors would be prerequisite for adequate multi-factor assessment. The only identified approach germane to childhood obesity that assesses this relatively comprehensively is *The Youth Evaluation Scale for Adolescents* (YES; Mellin, 1986), but this is applicable only to adolescents and not younger children. YES includes standardized measures of adolescent's health habits, anxiety level, depression level, family communication/cohesion/adaptability, and knowledge about weight management. Data from questionnaires are collected, including information about demographics, exercise preferences, school and peer relations, physical health and development, recent life events, weight problem history, family and peer influence on weight-control efforts, and risky behavior. A parent also completes three standardized subscales on family communication, cohesion, and adaptability, as well as other parental habits. There is a tremendous need for validation studies on the YES, as well as for the construction and validation of other measurement approaches in this area, especially of measures applicable to younger children.

Utilizing a multi-factor approach for intervention would depend on the results of a multi-factor assessment through tools such as YES. Clinicians treating obesity generally single out an intervention and recommend it to all their clients (Brownell, 1995; IOM, 2005): one clinician may advocate a prescription drug approach, another a dietary regimen (and there is vigorous disagreement about these), another a strict exercise program—and each may prescribe this particular treatment method for all his or her clients. Off the record, many childhood obesity professionals acknowledge the importance of multiple causes, but few seem to address more than one or two in their usual clinical interventions.

In contrast, we recommend efforts to study treatment matching based on the assumption that many approaches have merit and that the key to increasing successful treatment is to match individuals to the treatment(s) best suited for them. Many researchers have proposed that treatment matching would increase the effectiveness of interventions for obesity (Clark & Goldstein, 1995), but no research in this area has been conducted with children. With such an approach, one child may be offered help in moderating dietary restraint and subsequent binge eating, another child may be referred to an endocrinologist for treatment of a hormonal disorder, while a third may be referred for psychological treatment of depression or anxiety. Family therapy and nutritional counseling may be recommended for another. Last, there may be some children who, for biological reasons, fall at the extreme end of the normal weight distribution and who do not exhibit problems in eating, exercise, or other contributory factors; counseling might help them cope with stigma and prevent development of low self-esteem, low body-esteem, and depression that can be associated with being overweight in our fat-phobic society.

In order to utilize treatment matching, the clinician would need to begin with a thorough assessment. First, he or she would complete a medical exam to rule out any medical problems that could be causing or contributing to the child's obesity. Then a valid way to assess obesity, such as using skinfold calipers (the most reliable method of diagnosing obesity), would be employed to measure the child's fat ratio. Next, the clinician would assess a wide range of issues, such as explanatory style, dietary restraint, binge eating, awareness of hunger and satiety cues, self-esteem, body-esteem, dietary intake, physical activity, experiences of mastery and helplessness, family stress, style, and functioning, sexual abuse or other childhood trauma, depression, anxiety and parental feeding attitudes and behaviors. The assessment would include questions about time spent viewing television and playing computer games, about eating while viewing, and about the other factors we have described in this review. Additional data may be gathered from teachers, family, and friends. This is a daunting assessment protocol, but something we think is sorely needed for comprehensive understanding of a child with weight problems. And we acknowledge it is far easier to simply hand parents a food plan and exhort them to get the child to exercise more than it is to identify, assess, and treat many interacting factors. Yet we believe the latter is necessary, since childhood obesity is so intractable and, as we have shown, multicausal. However, we also note that it is a delicate task to help obese children without becoming part of the problem by exposing them to unwelcome attention that may further erode self-esteem or create other iatrogenic effects. Hence, we believe it is critical to address the obesogenic environment in as supportive a way as possible.

We also believe any prevention approaches should be multi-factorial. Merely exhorting children to eat less and be more active through public health campaigns is surely inadequate in the modern climate of our culture. According to Hill (2002), "The current global epidemic of obesity is fueled by an environment that overpowers the body's energy balance regulatory system.... Targeting the environment may be the only way to stop the obesity epidemic before it gets worse" (pp. 460, 463). Accordingly, important implications for public policy spring from the model, including reinstating sports and other extracurricular activities at schools, eliminating school-junk food contracts, restoring respect for physical labor, and combating poverty—and such efforts have already begun (Brownell, 2002b; Dietz & Gortmaker, 2001; Jeffery, 2002; Okie, 2005). Nestle and Jacobson (2000) support legislation that would: (a) tax soft drinks and other foods high in calories, fat, or sugar, (b) require companies to label food sold in movie theaters and convenience stores, (c) restrict advertising of high-calorie, low-nutrient foods on television programs that are watched by children, (d) improve current food assistance programs (school meal programs and food stamp programs), (e) require health professionals to learn how to counsel clients on dietary intake and physical activity, and (f) create better transportation and urban development to promote physical activity. However, vested food industry interests fight to retain their influence (Kanner & Golin, 2005; Simon, 2005, 2006) and such campaigns will require sustained effort. Despite the obstacles, Preston (2005) pointed out that mass public health initiatives have impacted smoking and drunk driving and could also be effective for combating childhood obesity.

Conclusion

An alarming increase in childhood obesity threatens the health and wellbeing of America's children. Apart from the hotly debated possibility of early mortality, obesity impedes movement and exposes the individual to ostracism, self-rejection, and chronic diseases that sap personal and national budgets, as well as hinder joy and pleasure in the life of the body. We have described a spectrum of factors contributing to obesity and placed them in an integrated framework. Some of these are deep background factors over which individuals have little control and which require broad social action to alter. Others, more readily changed, would be the immediate focus of treatment. We hope that our multi-factor model of childhood obesity will increase the understanding of this pervasive and complex problem and encourage more effective and individualized treatment approaches. We further hope that it will encourage the preventive efforts needed to systemically stem the epidemic of obesity that threatens the future of so many children.

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Figure 1. An example of how selected factors may operate through the five mechanisms to create obesity in a hypothetical child.

