Integrating Fundamental Concepts of Obesity and Eating Disorders: Implications for the Obesity Epidemic

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Physiological mechanisms promote weight gain after famine. Because eating disorders, obesity, and dieting limit food intake, they are famine-like experiences. The development of the concept of meeting an ideal weight was the beginning of increasing obesity. Weight stigma, the perception of being fat, lack of understanding of normal growth and development, and increased concern about obesity on the part of health providers, parents, and caregivers have reinforced each other to promote dieting. Because weight suppression and disinhibition provoke long-term weight increase, dieting is a major factor producing the obesity epidemic. The integrated eating disorder–obesity theory included in this article emphasizes that, contrary to dieters, lifetime weight maintainers depend on physiological processes to control weight and experience minimal weight change. (*Am J Public Health.* 2015;105:e71–e85. doi:10.2105/AJPH.2014.302507)

Since 1960, the Centers for Disease Control and Prevention has done periodic surveys of representative samples of the US population, which include measured heights and weights.[1] From the 1960 to 1962 to the 1976 to 1980 measurement periods, there was little change in population weight. However, the next survey (1988–1994) showed increases in body mass index (BMI; defined as weight in kilograms divided by the square of height in meters [kg/m^2]) that were unanticipated and inexplicable.[2] Most of the increase occurred in those with BMI of 30 or greater.[3] In 2006, a prominent Centers for Disease Control and Prevention researcher expressed frustration with her incapacity to explain why this happened.[2]

Losing weight and recuperating from that weight loss is part of the biological heritage of every human being.[4–6] However, in the past 70 years, self-induced famine (dieting to achieve and maintain a lower weight)[7] became the societal norm.[8–11] This suggests that much of the behavior observed in people who perceive they are overweight and who diet, manage, control, maintain, or suppress their weight could be manifestations of starvation and recovery from starvation.[4–6]

We breathe in and out, our hearts beat steadily, our temperature is automatically maintained without our thinking about it. The human body has similar mechanisms that produce weight gain after weight loss[5,6] or weight loss after weight gain.[5] However, this control is more flexible than the control of breathing: short-term differences in food intake are extremely variable over a period of hours or days whereas the mechanisms to moderate weight to previous levels take effect over a period of weeks or months.[5] These mechanisms work similarly and are equally efficacious in people of all weights.[6]

The researchers and practitioners who study energy balance, weight control, obesity, and eating disorders reflect different disciplinary viewpoints that function independently and publish their research in different genres of professional journals.[5] In this article, I sought to integrate this literature and show how the physiology of weight control is of paramount importance in explaining why the long-term trend in obesity changed.

BIOLOGICAL MECHANISMS

The basic premise of this article is that dieting is a self-imposed, voluntary famine. The most definitive study of famine is the Minnesota Experiment.[4] The validity of this study has been questioned because no formal control group was used.[12,13] However, an autocontrol of baseline measurements of all the parameters included in the study was established during a 12-week period before the men were placed on the semistarvation diet.[4(ch4)] In addition, 12 men who lived under the same conditions as the Minnesota Experiment and participated in a similarly rigorous but non–weight-loss nutritional experiment presented no evidence of the weight loss or behavior changes found in the Minnesota Experiment.[12]

The average age for the men selected for the Minnesota Experiment was 25.5 (±3.5) years. They were sociable and in good physical and mental health. Their initial BMI was 22.1 (±2.0). During the 24-week semistarvation phase, their intake averaged 1570 kilocalories per day (45% of autocontrol kcal) producing an average weight loss of 37 pounds in 24 weeks. (1.5 lb/wk; 24% of autocontrol weight). During 12 weeks of limited rehabilitation they were divided into 4 different groups on the basis of distinct percentages of autocontrol caloric levels.[4(ch4)] The men in the group with the highest caloric intake (88% to 116% of autocontrol levels) regained an average of 19 pounds during this time (1.5 lb/wk) recuperating 60% of their lost weight. Those in the lowest group (55% autocontrol period kcal) remained essentially the same weight for 6 weeks, gained an average of 3 pounds during 4 weeks on 78% autocontrol caloric levels, and an additional 3 pounds during 2 weeks on 88% autocontrol caloric levels. No participant returned to his autocontrol weight during limited rehabilitation.[4(pp1133–1140),12]

To celebrate the end of the experiment, the men attended an all-you-can-eat banquet. The next day they began eating prodigious amounts of food and frequently felt powerless to stop. They ate large meals, and then snacks between meals even though they were not subjectively hungry.[4(p846),12] Five months after terminating semistarvation, 4 men still reported cravings and excessive eating.[4(p848)]

Recent reviews report that binge eating and binge eating disorder (BED) exist as a continuum of symptoms that differ in magnitude but not in type of problem.[14–18] The size of a binge varies from quite small to extremely large[14–16] and the frequency of bingeing ranges from infrequent (<1/wk)[14,15,17] to bingeing more than 2 hours in a day.[15,17] Loss of control over eating is an important criterion for BED[14–16] but the presence of overweight or obesity are not criteria.[15,19] Those with low BMI and high weight suppression show the highest frequency of binge eating,[20,21] rapidity of weight regain,[22] and onset or maintenance of bulimic symptoms over a 5- or 10-year period.[22,23]

What links famines, eating disorders, and obesity is limitation of food intake. The behavior following semistarvation in the Minnesota Experiment complies with the more extreme descriptions of BED.[14–17] This close relationship has been confirmed by 3 studies that used questionnaires based on poststarvation eating to assess behavior of people diagnosed with BED.[24–26] Hagan et al.[24,25] used diagnosed BED participants and people from a general population. Both studies decisively showed that BED behavior is a poststarvation response to food. Sibilia[26] used descriptions of poststarvation eating interspersed with more neutral eating behaviors. High or low agreement with the question: "At times I feel an unbearable hunger"[26(p347)] was used to divide 311 college students into 2 groups. Only questions similar to those used in the Hagan studies showed statistically significant differences between the groups.

Table 1 shows the physiological, behavioral, and psychological ramifications of the different phases of the Minnesota Experiment. During the R1-to-R12 period (weight maintenance), the level of dietary restraint was imposed by the experimenters and adhered to by the participants. If one considers the severity of binge eating after R12, it is highly probable that if they had not conformed to the experimental protocol they would have binged during this period. The citations in Table 1 are not exhaustive but are indicative of similarities between the Minnesota Experiment and recent information found in the literature.[24–49]

IDEAL WEIGHT

The first widely available ideal height–weight tables were published by insurance companies in the 1940s based on actuarial analysis of longevity. They consisted of heights with corresponding weights for small, medium, and large frames. With the publication of these tables the idea of a healthy weight and losing weight to improve lifespan became plausible and desirable because higher premiums were charged for those who weighed more than their "ideal" weight.[50]

Currently, the National Institutes of Health defines a healthy weight as a BMI of 18.5 to 24.9, overweight as 25.0 to 29.9, and obesity as 30.0 or greater.[51] It is recommended that a person with a BMI of 25 or greater should lose 5% to 10% of current weight, at a rate of 1 to 2 pounds per week, with a focus on balancing calories in–energy out, and following a healthy eating plan. The recommended caloric intake is 1000 to 1200 kilocalories per day for women and 1200 to 1600 kilocalories for men.[52]

However, for most people, ideal weight is related to societal appearance norms rather than health and longevity.[53] Evidence that an ever-thinner ideal woman has evolved is found in an analysis of *Playboy* centerfolds that shows a decreasing trend in average BMI from approximately 19.8 in 1953 to approximately 17.9 in 2001.[54]

WEIGHT STIGMA

Weight stigma has 3 components: cognitive (stereotype), affective (prejudice), and behavioral (discrimination).[55] Together these 3 components produce self-stigma.[56,57] Weight stigma is part of the everyday experience of people with obesity.[57] Link and Phelan[58] designated it as structural discrimination: behavior that determines social relationships and whether a person receives recommendations of colleagues and friends.[59] The frequency of experiencing weight stigma is similar to gender,[60] race,[60,61] and gay or lesbian stigma.[61] Current health policy regarding obesity seems to have inadvertently supported all 3 aspects of weight stigma.[62]

Weight stigma is found at all ages[63–68] and professions[62] including teachers,[69–71] dietitians,[72,73] nurses,[73,74] and physicians.[73–76] Physicians who specialize in the treatment of obesity[75,76] consider fat people lazy, stupid, and worthless on both implicit and explicit attitude measures.[76] Because weight is considered controllable, not achieving weight-loss goals is attributed to lack of intelligence, motivation, will-power, self-discipline,[74,75] and knowledge.[74] People with obesity accept responsibility for their condition, attributing it to failure on their part even though they have tried multiple weight-loss remedies.[74]

Antifat bias of children increases with age.[77] Recipients are subject to bullying, teasing, and verbal harassment.[78] The problems caused by teasing and bullying related to weight stigma do not seem to be the events as such, but the frequency of the events,[79] whether the person who receives it is bothered by it,[80,81] and whether the person perceives herself or himself to be "too fat."[82] Of the people who perceive weight discrimination, those who are not obese are more likely to become obese and those already obese more likely to maintain obesity over a 4-year period.[83]

PERCEPTION THAT "I AM TOO FAT" IS A PROBLEM

Internalization of the cultural norm of thinness occurs when a person adopts the norm of extreme thinness as his or her private ideal. The BMI of men and women portrayed in commercial television is highly skewed compared with the actual weight distribution in the US population: 31% of the women on television are underweight versus 5% of the actual population, and only 3% obese, versus 25% of the actual population. Weight prejudice is reflected in these programs.[84,85] Media exposure to thin-ideal images engenders a significant increase in body dissatisfaction compared with controls that did not view such images.[86] A 5-year prospective study shows that those who read magazines that present articles and advertisements about dieting and weight loss put higher importance on weight,[87] and engaged in a greater number of eating disorder–related weight control measures than their peers who do not.[88] Older children and adolescents may be especially vulnerable because they look to the media, including advertisements, for information to shape their own identity.[89]

People of all ages and BMI levels perceive they are "too fat."[90–94] Being "too fat" has been expressed as early as age 5 to 6 years.[90] Kurth and Ellert[91] measured 11- to 17-year-olds

TABLE 1—Observations and Measurements of Minnesota Experiment Participants During Weight Loss, Limited Rehabilitation, and Weight Regain[4,12] With Citations to Similar Observations Reported in the Current Eating Disorders and Obesity Literature

Category	Observations and Measurements
	Observed during semistarvation phase (weeks S1-S24); period of weight loss
Physiological	Increased appetite, hunger, desire for salt, edema, alopecia, vertigo, faintness on arising, momentary blackouts, craving for food, fatigue, weakness, hearing acuity, desire for hot food, unsteadiness, and sensitivity to cold.[27]
	Decreased production of sex hormones, blood pressure, blood glucose, tolerance of cold temperatures, endurance, strength, reaction time, coordination, sex drive, interest in opposite sex, pulse rate, metabolic rate, and capacity to lift.
	Cuts and wounds slower to heal, muscle cramps, soreness, and polyuria.
Behavioral	Food and eating became dominant concern; collected cook books, etc.; possessive attitude toward food; total attention to food while eating; eats food formerly disliked[24,25]; initially gulped food, but at end ate very slowly[25]; all food consumed to last crumb; intolerant of food waste; daydream about foods[25,26]; created unusual food concoctions[30]; poor table manners[24]; added ingredients to thicken food[24]; smoked,[31, 32] chewed gum to alleviate hunger; some started smoking[31]; and consumed large amounts of water, coffee, tea.
	Increased moodiness,[38,39] irritability,[25,38,39] self-centeredness, narrowing of interests, and gastrointestinal distress[24,25]; complaints about lack of alertness, poor comprehension, lack of judgment, and deterioration of memory.[33–36]
	Decreased ambition, self-discipline, self-control, social initiative,[39] sociability,[39] ability to concentrate,[39] interest in opposite sex,[39–42] sense of humor, and interest in learning.
Psychological	Increased depression,[39,43,44] apathy, dissatisfaction with their appearance, inability to sustain mental or physical effort,[39] "nervousness" and general emotional instability, social withdrawal,[39] and narrowing of interests.
	Decreased decisiveness, ability to make personal plans,[25] willingness to participate in group activities,[39] and sexual desire.[39,40,42]
	Increased MMPI scales[4]: hypochondriasis, depression, and hysteria at end of semistarvation ($P<.01$); depression increased >2 SD.
	Observed during limited rehabilitation phase (weeks R1-R12): period of weight maintenance)
Physiological	Increased appetite, hunger; decreased strength (had not returned to control levels at R12).
Behavioral	Increased irritability,[25,38,39] impatience, tenseness, and general unrest.[43]
	At R12 had not resumed academic studies and intellectual pursuits[25] to level during autocontrol; inefficiency and poor study habits continued.[25,33–36,39]
	Recovery from dizziness, apathy, and lethargy most rapid; tiredness,[44] loss of sex drive,[39–42] and weakness were slow to improve. Many were argumentative and negativistic[45,46] and failed to reestablish efficient nonexperimental lab and work assignments.[44,46–48] Food concoctions continued.[30]
	Lack of strength, endurance; as weight increased regained humor, enthusiasm. Sociability progressively reappeared and irritability and nervousness decreased.[39]
Psychological	Psychobiological stress continued to greater or lesser degree depending on level of refeeding. For those who maintained weight there was a return to the depressed levels experienced during semistarvation (famine).[44,46,49]
	Feeling of well-being, range of interests, emotional stability, and sociability returned more rapidly than strength, endurance, normal eating habits, and sexual drive.
	Observed during no restrictions on eating phase (weeks R13-R58): period of complete disinhibition of dietary restraint)
Physiological	Banquet with unlimited food served on last day of R12 produced LOC (binge days),[14,15,24–26] GI distress.[24,25]
	Tiredness, unusual sleepiness; improvement in sex drive and spontaneous activity parallel increase in hunger, appetite.
	At R58 were similar to pre-experimental norm.
Behavioral	3 men restricted food intake to return to autodetermined weight levels.[4,49]
	Increased irritability[38]; food concoctions continued[30]; attempts to avoid wasting food even though supplies unlimited; and eats fast or slowly.
	At R20 (5 wks) 5 of 15 who reported for follow-up still binge eating. At R33 (8 wks) 4 of 14 still bingeing.
Psychological	Distress associated with LOC. 3 men: distress with regaining "too much" weight.
	Psychological stamina of the participants was unquestionably superior to that likely to be found in any random or more generally representative population sample.[4]

Notes. GI = gastrointestinal; LOC = loss of control over eating; MMPI = Minnesota Multiphasic Personality Inventory.

regarding physical well-being, emotional (psychological) well-being, feelings of self-worth, well-being in the family, well-being in relation to friends or same-age peers, and well-being in the school domain. For every parameter measured, the behavior of those who perceived themselves to be "far too fat" was the same no matter what was their objective BMI classification. All those who self-perceived they were "just the right weight" also had the same behavior regardless of their BMI.

Of the 11- to 14-year-olds studied by Viner et al.,[92] 2% of the girls defined by BMI as underweight considered themselves "too heavy" but 5% were currently dieting and 10% had previously dieted. Thirteen percent of those who were normal weight and 36% of those who were overweight perceived themselves to be "too fat" and the percentage currently dieting or who had ever dieted was about double these percentages. For the obese girls, 63% considered themselves to be "too fat," 81% were currently dieting, and 77% had previously dieted. For boys, lower

misperceptions of being "too heavy" were recorded but the proportional increase in percentage of those who were currently trying to lose weight or had ever dieted was similar.[92] People with BMIs of 35 to 39.9 divided themselves 50–50 between those who perceived that they were overweight and those who considered themselves obese. Those who considered themselves overweight reported fewer objective binge-eating episodes (eating an objectively large amount of food accompanied by loss of control) and less distress about eating compared with those who considered themselves obese.[95]

Dieting leads to long-term weight gain. Longitudinal observational studies or prospective cohort studies that last 4 to 5 years[96–102] or more[97,103–110] in preadolescent girls[96,97] and boys,[97] in girls and boys at puberty,[97,98] in adolescent to early adult girls[99–101,103–105] and boys,[101,103,105] in women,[106–109] and men[102,107–110] (including 2 studies of male and female twins[105,107]), show that those who are dieting at the beginning of the study end up weighing more than their nondieting counterparts, even though they are not overweight at the beginning of the study.[103,110,111] Two longitudinal studies[112–114] show no relationship between dieting and an increase in BMI. These studies included yearly measurements of height and weight coupled with yearly administration of diet and weight-control questions.[112,113] They also show selective loss to follow-up of individuals with a higher BMI.[112,114] Studies of Olympic wrestlers show weight gain over time compared with nonathletes[110] or athletes who have less severe cycling,[110,115] but a study of collegiate athletes did not show this pattern.[116]

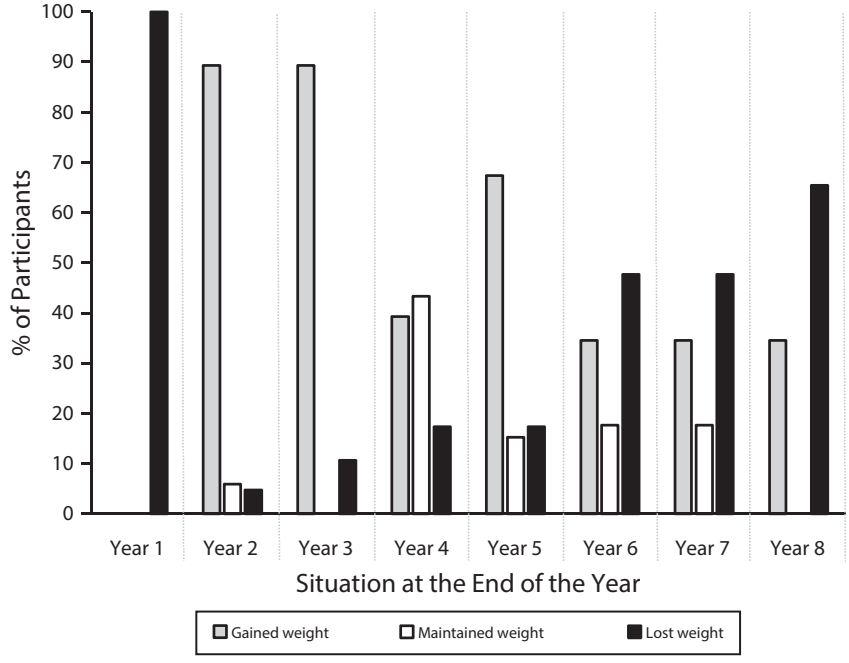
WEIGHT MAINTENANCE VERSUS SUPPRESSION

Maintaining a lower weight is considered a virtue in the obesity literature (weight maintenance) but the eating disorder literature considers it problematic (weight suppression). "Successful weight loss," defined by the National Weight Control Registry as losing at least 10% of initial body weight and maintaining that weight loss for at least 1 year,[117] is used to evaluate most weight-loss studies. However, a review of weight-loss studies that lasted 2.5 years or more shows that the ineffectiveness of dieting is frequently underestimated because of 4 methodological problems: very low follow-up with inadequate reporting of the characteristics of those who continued versus those who dropped out, not asking about additional dieting that might have occurred after the diet that was being studied, self-reported weight, and confounding the effect of dieting with that of increased physical activity. They concluded that each of these factors biases studies toward showing successful weight-loss maintenance.[118]

The effect of these factors can be seen in the Minnesota Experiment[4] and other reports.[49,117] For the Minnesota Experiment, only 21 of the original 32 men reported for follow-up at week R58. Of these, 12 (57%) had gained more than 10 pounds (R58 weight minus maximum autocontrol weight), 4 (19%) had gained between 5 and 10 pounds, 2 (10%) were within 5 pounds (gain or loss), and 3 (14%) were known to have actively dieted after their poststarvation weight increased to levels they considered unacceptable. Two men who had previously reported considerable weight gain did not show up for this evaluation.[4(p1140)]

Look AHEAD (Action for Health in Diabetes) was a multicenter randomized controlled trial designed to assess the efficacy of weight loss for adults with diabetes. It compared an intensive lifestyle intervention (ILI) with diabetes support and education. The goal was to lose 10% or more of initial weight during the first 6 months eventually stabilizing at a mean weight loss of 7% or more. The ILI groups initially met frequently, used portion control, and was encouraged to use meal or snack replacement products. If the participant lost 5% or less body weight at 6 months, pharmacotherapy was encouraged.[119]

At the end of the eighth year the participants were grouped according to their year-1 weight loss (≥ 10%, 5%–9.9%, 0%–4.9% of initial weight) and then subgrouped by each category on the basis of those who in year 8 had lost 10% or more, 5% to 9.9%, 0% to 4.9%, or gained weight. Figure 1 shows that all groups lost weight during the first year. In the



Note. AHEAD = Action for Health in Diabetes. Calculated from grouped data included in Figures 4_a to 4_c of Look AHEAD Research Group.[120]

FIGURE 1—Year-by-year changes in percentage of intensive lifestyle intervention participants in groups that were gaining weight, maintaining weight, or losing weight during 8 years of the Look AHEAD study.

subsequent 2 years, 89% were in groups that gained weight. In year 4, a measurement year, 43% were in groups that maintained weight but in all other years less than 20% were in groups maintaining weight. By year 8 all groups were either gaining or losing weight.[120]

At year 4, the ILI group had, on average, improved weight, fitness, and cardiovascular indicators compared with the diabetes support and education group.[121] At year 10, the ILI group, on average, showed improved weight loss and waist circumference, with inconsistent improvement in glycerated hemoglobin between year 6 and 10. However, no differences were found between ILI and diabetes support and education groups in terms of cardiovascular disease morbidity or mortality.[122]

Qualitative studies indicate that successful weight-loss maintainers constantly think about maintenance,[123,124] use clothing fit as feedback,[123–126] weigh themselves frequently[123,125,126] (some several times a day),[125,126] and keep track of their actions through journaling or worksheets.[124–126] When challenges or setbacks occur they immediately institute changes to reduce their weight.[125,126] But many get bored with monitoring, or decide that it is no longer worth paying constant attention to what they are eating.[123,125] In one study, partners, family, and friends were reported as being saboteurs of maintenance efforts.[124] In contrast, lifelong weight maintainers take a relaxed attitude toward food, do not weigh themselves frequently, nor do they stress about a few pounds.[126]

This analysis indicates that "successful weight loss" usually refers to weight cycling rather than lifelong maintenance. Both the National Weight Control Registry[117] and the qualitative studies of weight maintainers[123–126] show that people who attempt to maintain weight use activities associated with eating disorders: frequent weighing,[127] continuous monitoring of weight and appearance,[128] and high levels of physical activity.[129] Ironically, weight loss or maintenance efforts did not produce the health benefits that the obesity literature anticipated.[122]

EARLY-ONSET OBESITY

Retrospective studies of the age of onset of bingeing and dieting show 2 groups: one that dieted before bingeing (D_1) and another that binged before dieting (B_1)[28,130–140] with the B_1 sequence always starting at a younger age. In studies that reported the age at which the person recalls becoming overweight, the D_1 sequence is diet, binge, overweight,[130–133] which is the sequence alluded to in the previous sections of this article. However, the B_1 sequence is binge, overweight, diet[130–133] except for young men (average age: 18.0 ±10.5 years), who dieted frequently and show a sequence of overweight, binge, diet.[133] This different perception is probably attributable to their adolescent growth spurt.[141] Spurrell et al.[130] found that 90% of their B_1 and 59% of D_1 began bingeing before their 20th birthday. Women with early onset obesity (aged <18 years) have a greater lifetime maximum weight, more dieting attempts, greater weight loss on a single diet, and increased binge eating.[142]

The cited studies did not differentiate between bingeing with loss of control and overeating. Loss of control over eating is a physiological response to a previous lack of food[4(p846–849)] but not necessarily dieting as such.[143] It can result in the development of partial- or full-syndrome BED.[144] Overeating is a precursor to rapid growth that occurs in childhood[145] or at puberty.[141] It also may occur at any age as a response to nonverbalized emotions.[146–148] A person that has a sense of control over eating may eat a lot (overeating) but does not show typical eating-disorder symptoms.[149] Loss of control over eating can occur with lower or higher body dissatisfaction. However, the higher the body dissatisfaction the more severe the eating-disorder symptoms.[149] Children who binge with loss of control have significantly greater weight concerns,[150] disordered eating attitudes,[145] eating in the absence of hunger,[145] and depressive and anxiety symptoms[145,150,151] as well as greater use of other maladaptive strategies[151] compared with overeaters.

To better understand the factors that underlie early onset obesity, I will first discuss the nature and timing of the adolescent growth spurt and the physiological changes that take place in both sexes during puberty. I then discuss how concern about obesity affects the feeding practices and interrelationships between child and parent from infancy to adolescence. This is followed by the effect of childhood trauma on the development of obesity.

Normal Growth and Development

Body mass index is currently used to indicate relative adiposity in people. However, people come in all sizes and shapes. Some are naturally more rounded, others more angular. Some have small, light bones; others have large, dense bones. Some have long trunks and short legs; others have long legs and short trunks. Those with large, dense bones; long trunks; and short legs automatically have a higher BMI than those with small, light bones; long legs; and short trunks even though they do not have higher fat mass.

The BMI cut-offs for underweight, normal weight, overweight, and obesity for children are based on cross-sectional analysis of children from birth to 20 years.[152] However, longitudinally, weight increase precedes height increase.[153] The age at which peak weight or height velocity occurs, the amplitude of that velocity, and the duration of the growth spurt all vary in distinct ways among individuals.[154,155] Because of this, Tanner, a renowned authority on human growth,[156] concluded that there can be no satisfactory weight for height standards between the ages of 7 to 18 years (girls)[154] or 9 to 20 years (boys).[154,155]

Every organ, muscular, and skeletal dimension of the body takes part in the adolescent growth spurt. Early in the process the growth of the heart, stomach, and other visceral organs speeds up.[157] In both boys and girls, the head, hands, and feet reach their adult status first. Leg length reaches peak growth velocity before trunk length. The body broadens later: hips in girls, shoulders in boys.[155]

During adolescence a person gains about 50% of his or her adult body weight.[158] From infancy to about age 5 to 7 years, BMI decreases after which it begins to increase. This increase has been designated "adiposity rebound."[159] An earlier adiposity rebound is accompanied by being taller in childhood, having earlier pubertal development, and greater growth increase during puberty.[155]

Fat mass and BMI correlate well,[160] but BMI is a weight-to-height ratio (kg/m^2), not a measurement of body composition. Fat-free mass increases steadily in childhood and at the beginning of adiposity rebound increases more rapidly than fat mass, which does not increase appreciably until about 2 years later.[161] Childhood overweight has been

consistently associated with larger long-bone cross-sections in both sexes.[162] In girls, nearly one third of total skeletal mineral is accumulated in the 3- to 4-year period immediately after puberty onset.[158] Both boys and girls who enter puberty early have the same average adult height as those who enter later,[163,164] and their comparative increased fat-free mass and bone density continues as adults.[162] Thus, they naturally have higher BMIs as adults compared with their later-maturing companions. As there is an increase in fat-free mass and bone density as well as fat mass during adiposity rebound, a more appropriate designation would be preparation for pubertal growth rather than a critical period in childhood for the development of obesity.[159]

In adolescence, both boys and girls worry about looks, popularity, and relationships with the opposite sex.[165] In girls, the worry about looks concentrates on thinness and weight[165] whereas boys want to be more muscular.[166] The normal physical development of girls includes increased size of visceral and sexual organs in the lower part of the trunk, increased hip breadth, and increased fat as a natural part of the process of becoming a woman. These changes are at odds with the very thin ideal for women promoted by US norms.[9,10] Limiting food intake because of body image concerns, increasing exercise to burn up energy, and seriously wanting to lose weight all increase substantially in girls between 6 and 24 months postmenarche.[167] The increased muscularity desired by boys is not compatible with a situation in which their strength has not caught up to their height and they are effectively managing an adult-size body with child-strength and child-size muscles.[155]

Parental Concerns

Parents are concerned about their children eating the right amount of food and the right kind of food. If they perceive that a child is overweight or "eats too much" the response is to restrict intake of the total amount of food or of foods high in fat and sugar.[168] If the child is perceived to be underweight or not eating enough of the "right foods" the response is to pressure the child to eat more total food or more vegetables or other healthy foods.[169] Restricting food leads to an increased desire to eat the restricted foods,[168] and pressuring a child to eat leads to increased negative comments about the food and less desire to eat it,[169,170] which sometimes lasts a lifetime.[170]

High concern about a child's overweight or the possible development of overweight in the future is associated with increased weight between ages 3 and 5 years.[171] Parents of children studied between birth and 5 years of age reported that 34% of the children experienced the emergence of overeating, 18% hid favorite foods, 9.7% seemed shy or inhibited when eating with parents compared with eating alone, and 9.7% ate so much that they became sick or vomited.[172] Longitudinally, restriction at age 5 years predicts whether a girl eats although not hungry at age 7 years[173] and 9 years[174] as well as an increase in eating in the absence of hunger during these 4 years.[174] A review of the literature confirmed the association between restricting foods and later increased BMI or food intake.[175] These studies indicate that mothers who worry about their own weight and eating have higher concerns about daughters' weight, higher levels of restricting daughters' intake, and encourage daughters more frequently to lose weight.[176,177] Fathers are involved but no longitudinal studies were found.

A mother's concern that a child might become overweight seems to be dependent on trunk and leg fat related to total fat,[178] eating more sweets and other palatable foods,[178] eating rapidly,[179] and eating more than presumed to be normal.[145] Although most studies of children depend on parent observations, there is little concordance between a 6-year-old girl[180] or 6- to 12-year-old child[181] and his or her parent's answers regarding the child's food-related behavior. This discrepancy in parent–child observations could be critical in terms of future excessive weight gain. Shomaker et al.[145] documented a group of children who were classified by their mothers as eating excessively but who actually were overweight with a lower fat mass than the other groups in the study (high BMI, not high fat). This indicates that, for the child, the increase in intake is best characterized as a response to the physiological need for enough food for his or her body. Because the mother sees it as overeating, this could lead to attempts to restrict intake with concomitant increased hiding or eating food when mother is not around.[145,172]

Childhood Trauma

Longitudinal studies show that childhood trauma (emotional neglect,[182] physical neglect,[183,184] physical abuse,[182,183] sexual abuse,[184,185] and lack of parental support[182,186]) result in increased BMI in adulthood. Child emotional neglect and abuse are common[187] but difficult to define and measure.[188] Estimates for child sexual abuse range from less than 3%[182,183] to 30% in girls and 15% in boys.[189] Noll et al.,[185] using cross-sequential technique, found no differences between sexually abused girls and a comparison group in terms of the percentage who are obese at ages 6 to 14 years ($P = .52$), nor at 15 to 19 years ($P = .09$). However, at age 20 to 27 years, more than 40% of the sexually abused girls were obese compared with 30% of the comparison group ($P < .009$). A study of adults who reported childhood physical or sexual abuse found increased odds ratio (≥ 3) for recurrent fluctuations in weight, strict dieting, obesity, any eating disorder, and self-induced vomiting compared with those who had not been abused.[184] Although childhood trauma occurs more frequently than we would like to admit, the incidence of child maltreatment reported in 2012, 2004, and 1999 indicates that the per capita rate has decreased.[190] Thus, these factors cannot be cited as causing an increase in the prevalence of obesity.

Alexithymia, Interoceptive Awareness

Restriction of foods or physical or psychological abuse can begin at an age in which a child has little or no capacity to identify and verbalize emotions (alexithymia). Experiences of overeating to quell inexpressible emotions or hiding restricted food may result in lack of ability to distinguish hunger and satiety signals (interoceptive awareness). Several studies have cited these and related concepts as problems that lead to the development of bulimia nervosa and BED.[146–148,191–193] Alexithymia may be more important in the development of obesity in men than in women.[192]

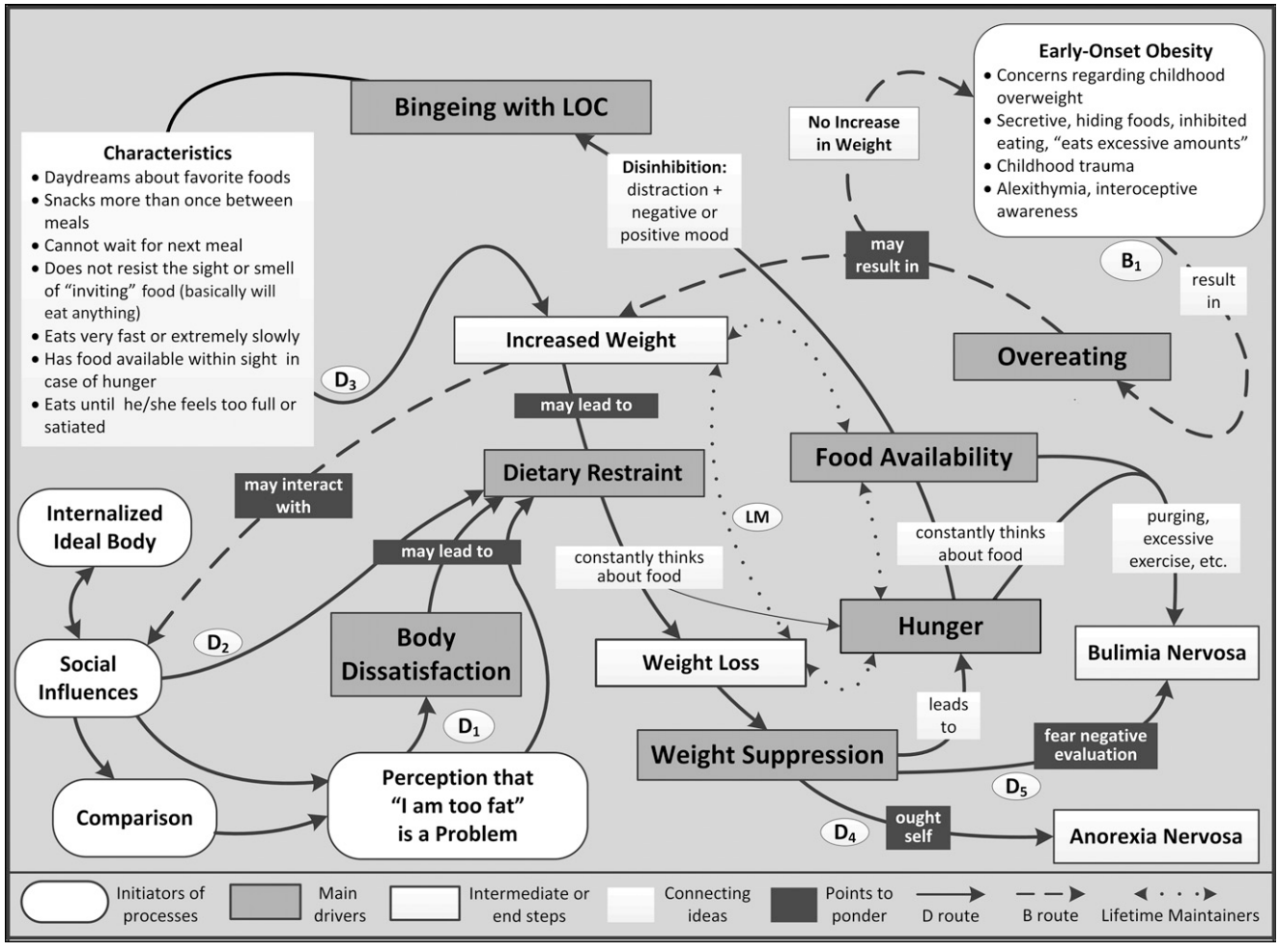
AN INTEGRATED THEORY OF DIETING, EATING DISORDERS, AND OBESITY

A good theory can be tested, receives empirical support, has predictive power, is

internally consistent, and is as simple as possible to explain known facts.[194] The basic components necessary for an integrated theory of eating disorders and obesity include sociological or psychological parameters that lead to the perception that "I am too fat" is a problem, limitation of food intake (dietary restraint) to correct that problem, hunger (from eating less food), the physiological reaction to hunger and limited food intake (disinhibition), and the psychopathology caused by lack of food (Table 1).

The transdiagnostic theory for eating disorders uses a core cycle in which overvaluation of eating, shape, and weight produces strict dieting, which in turn produces binge eating. This then loops back to renew the strict dieting. Alternative pathways show binge eating may be in balance with compensatory vomiting and laxative misuse (bulimia nervosa [BN]), whereas for anorexia nervosa (AN) the binge–compensatory cycle may be present but the behavior is basically diverted to a "starvation syndrome."[195(p521)]

Figure 2 is a concept map of the integrated eating disorders–obesity theory, which amplifies and updates the transdiagnostic theory core cycle[195] to align it with recent literature. The gray background represents the pervasive weight stigma that underlies obesity and eating disorders. The dieting routes to eating disorders and obesity (D_{1-5}) begin in the lower left-hand corner based on the tripartite influence model.[166,196-199] Social influences (media, peer influences, parental–family concerns) and comparison (judging one's self-worth by comparison with media stereotypes, and to actual people in the immediate social environment of the individual)[196,198] lead to the "perception that 'I am too fat' is a problem." This phrase incorporates 3 concepts: perception of fatness, I am fat, and this is a problem I have to solve. If fatness is considered a problem, it leads to



Note. LM = lifetime maintainer; LOC = loss of control over eating. The gray background indicates the weight stigma that permeate any consideration of obesity. The D routes (solid-line arrows) begin in the lower left hand corner with "social influences," etc. Starting with dietary restraint they begin a cycle that goes to weight suppression. From weight suppression they can follow a cycle that ends up with weight gain, or go to anorexia nervosa or bulimia nervosa. The B route (dashed-line arrows) begins in the upper right-hand corner and ends with "Social Influences." It can also loop back on itself with no increase in weight. The life time maintainer route (dotted-line arrows) begins with hunger and food availability and can cycle in either direction.

FIGURE 2—Integrated eating disorder–obesity theory.

dietary restraint ("dieting" [D_1]).[82,91,92] High internalization of the thin ideal occurs equally frequently in high and low BMI females even though the person is not aware of it.[200] Thus, comments within the immediate peer group[201,202] augmented by schoolwide norms[200,202] can result in girls with low to average BMI becoming increasingly concerned about weight and shape without expressing overt body dissatisfaction[200] (D_2).

Effective dietary restraint leads to weight loss. The period of weight loss is characterized by lower levels of disinhibition, practically no objective binge eating episodes, decreased depression, lack of subjective feelings of hunger, increased body satisfaction, and increased self-esteem.[203] Lack of hunger results from a neurologic response to lack of food.[204] The feelings of well-being and reduction of depression stem from feeling control over their weight.[205]

Once a lower weight is realized the person is then confronted with long-term weight suppression.[120,123–126] Although negative affect is clearly seen after a famine (Table 1) this reality has been ignored in the obesity literature. The eating-disorder literature frequently assumes that it is psychological in origin.[194,206,207] Body dissatisfaction is induced by peer pressure but is not accompanied by negative affect.[208] Therefore the "negative affect" cited in the eating-disorder literature has to have its origins in attempting to maintain lower weight. This may explain why those who have lost weight report that friends and family act as saboteurs of their efforts to maintain the lower weight.[124]

Weight suppression has 3 potential outcomes: binge eating and weight regain, AN, or BN. Bingeing with loss of control is the most common and can range from levels that are indiscernible (<150 calories/d) to daily large binges.[14–18] The variability of response in the Minnesota Experiment indicates that the reaction of a specific individual is probably genetic.[209]

Contrary to reports in the eating-disorder literature that binge eating is produced by negative affect,[207] in the Minnesota Experiment, it began after a banquet.[4(p843)] Ecological momentary assessment studies show that binges occur most frequently when a person has moderate hunger,[210,211] is alone,[211] and distracted[212] or in a negative[211,213] or positive[211] mood. Thus, the path from weight suppression to bingeing with loss of control includes hunger, constantly thinking about food, food availability, and disinhibition: distraction plus negative or positive mood. The connecting idea characteristics summarizes important aspects of bingeing with loss of control and indicates that under these conditions people will eat anything, not just highly desirable foods.[4(p6),23–25] This process leads to increased weight (D_3). If this is accepted, the route stops. If it is not acceptable, dieting eventually occurs again.[214(p113),215] Repeated cycling through this route can lead to significant weight gain.[96–110,215]

It is generally thought that AN and BN (D_4, D_5) occur only in normal weight or very thin individuals. However the antecedents may be overweight[216] or actual obesity.[216,217] Dieting behavior of future AN is characterized by reduction in portion sizes, fixed meal times, and stereotyped food choices, which produce a slow, unrelenting weight loss.[218] In BN, the concept of the thin ideal self is very strong and includes need for approval,[219] as well as fear of negative evaluation.[219,220] Future BN starts out with weight control behaviors that the obesity literature designates as unhealthy (fasted, ate little food, use of a food substitute, skipped meals) or extreme (taking diet pills, laxatives, diuretics, or vomiting).[216] The weight loss results are generally poor, which leads to more restrictive diets, binge eating, and large fluctuations in weight.[218] People with AN eat very slowly whereas those with BN eat rapidly. Training AN to eat progressively more rapidly, after they have agreed to gain at least 5 pounds,[204,221] and BN, who have been assured that they will not gain weight, to eat less rapidly[204] leads to normalization of their eating behavior and complete remission of their eating disorder and psychiatric symptoms.[204,221]

In Figure 2, the B_1 route of early onset obesity begins in the upper right-hand corner and is shown by dashed-line arrows. Its antecedents are the indicated social factors rather than lack of food. The overeating that occurs usually includes sweets, snacks, and high-fat foods because they are readily available to a child in the community and are frequently restricted at home. If overeating is accepted, the child may become plump, but his or her body will balance the excessive calories with times in which less food is eaten. The result may be a slightly overweight person, but high levels of obesity will seldom occur. If an issue is made of the "excessive" intake and the limited plumpness that develops is considered worrisome by health professionals or concerned parents and caregivers, then these social influences may well initiate dieting in the child. This means they then follow the D_{3-5} routes to increased weight, AN, or BN.

The experience of lifetime maintainers[126] is shown with dotted-line arrows in the middle of Figure 2. Flexible hunger and satiety determine the amounts eaten[214] and physiological processes balance it out over time.[5,6] At what level of lack of food, dietary restraint, and weight suppression the autonomic lifetime maintainers route turns into the perception of loss of control is unknown.

CURRENT THEORETICAL DEVELOPMENTS IN EATING DISORDERS

The terms "dietary restraint" and "disinhibition" originated with studies of dieting: the person inhibits caloric intake (dietary restraint), which is then disinhibited in response to food being available. The Restraint Scale was the first questionnaire available for measuring eating behavior in eating disorders. It was severely criticized because it measured concern for dieting and weight fluctuations rather than dietary restraint.[222] The Three Factor Eating Questionnaire (TFEQ)[223] and the Dutch Eating Behavior Questionnaire (DEBQ)[224] were developed to correct these problems. The TFEQ purports to measure 3 physiologically sound constructs: dietary restraint, disinhibition, and hunger.[223] The DEBQ purports to measure restrained eating, but the concept of disinhibition was substituted with emotional eating (eating in response to negative affect) and external eating (eating in response to food-related stimuli regardless of the internal state of hunger).[224]

When the transdiagnostic theory was developed,[195] Fairburn and Harrison showed awareness of the close similarity between AN and postfamine psychology and behavior.[39] However, the Fairburn et al. transdiagnostic theory did not indicate that other eating disorders might show less extreme aspects of this postfamine psychopathology.[195]

Stice et al. subtyped women with BN[225] and BED[226] based on measurements of dietary restraint, depression, and self-esteem. Two

groups were formed: dietary (D) and dietary negative affect (DNeg). A prospective study of this dual pathway model indicated that body dissatisfaction predicted both dieting ($P<.001$) and negative affect ($P<.01$), but, although dieting predicted bulimic symptoms ($P<.001$), it did not predict negative affect ($P<.1$).[227] This directly contradicts the findings of the Minnesota Experiment, which showed that mean depression scores increased to pathological levels at the end of the semistarvation period and then returned to normal in parallel with improvement in caloric intake.[4(p868)] In the original study with BN, although no significant difference was found between the subtypes with the TFEQ hunger scale, a significant difference was found on the BED component of the Axis I diagnostic scale ($P<.01$).[225] In the BED study the TFEQ hunger scale shows a significant difference between BEDs (D<DNeg; $P<.001$).[226] This suggests that if the subtyping had been based on the TFEQ hunger scale, the studies might have shown that dieting produced negative affect.

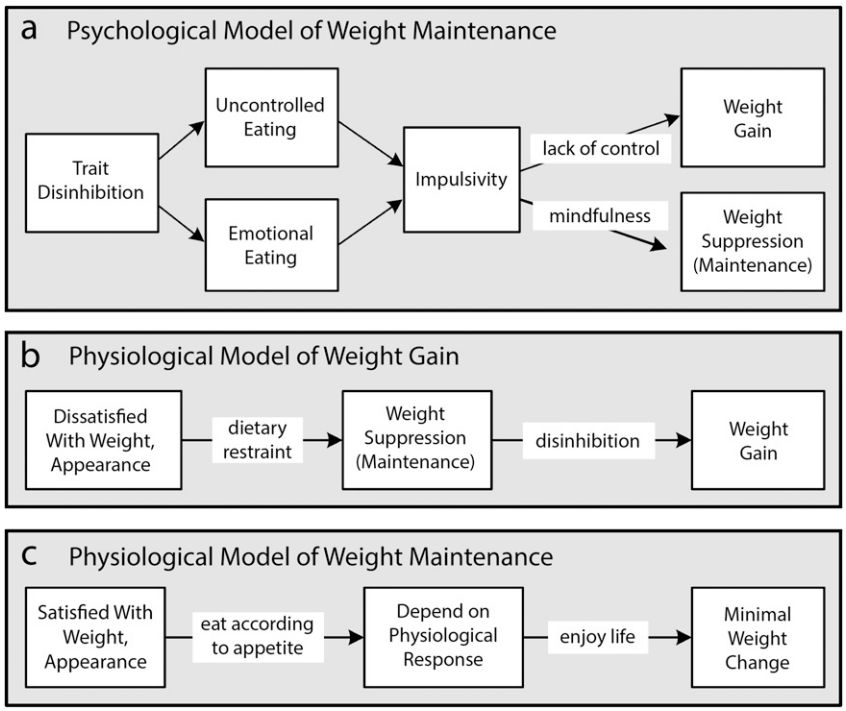
The lack of understanding of the primacy of limitation of food on subsequent psychopathology[4,12] and the development of the emotional and external eating scales of the DEBQ[224] opened the possibility to interpret binge eating (disinhibition) as a response to negative moods and the availability of desirable food[228] rather than a response to whatever appears to be edible.[24–26] A 2007 review defined disinhibition as measured by the TFEQ as "a tendency towards over-eating and eating opportunistically in an obesigenic environment."[228(p409)] The review was "not concerned with the inhibition of Restraint but with the durable trait of Disinhibition" (disposition for opportunistic eating).[228(p410)] It concludes that "trait disinhibition" is associated with obesity and mediating variables that constribute to poorer health as well as increased severity of eating disorders.[228(p409)]

IMPLICATIONS FOR PUBLIC HEALTH

United States food guides based on vitamin and mineral needs have been issued periodically since 1943. The 1980 Dietary Guidelines included weight control for the first time.[229] Since then the insistence on weight management to control and prevent obesity has become stronger[230–232] and has increasingly been aimed at younger age groups.[233] In the 2005 and 2010 Dietary Guidelines, weight control was no longer a separate goal but permeated the entire document with the exception of the section on micronutrients of concern. This increased emphasis is concomitant with a greater percentage of the population dieting[234–236] and greater prevalence of obesity in the ensuing years.[1] As people who are overweight or obese diet more frequently[92,108] this produces a more rapid increase in weight for those at the higher end of the BMI curve.[103,108]

Thus, the insistence of the medical community and the dietary guidelines on weight loss, compounded by the commercial diet and weight-loss industry that makes more than $60 billion per year,[237] result in dieting as a major factor producing the obesity epidemic.

A good theory is as simple as possible to explain the known facts.[194] Figure 3 contrasts a recent psychological explanation of weight control with autonomous physiological weight control. A 2011 article used the TFEQ-21 in which the 3 components refer to cognitive restraint, uncontrolled eating, and emotional eating to assess the relationships between "trait disinhibition,"[228] impulsivity, mindfulness, and adverse psychological symptoms that are caused by weight suppression. Figure 3a diagrams their conclusions: "trait disinhibition" has 2 components: uncontrolled eating and emotional eating. Impulsivity, another concept that the psychological literature substitutes for disinhibition is positioned as a mediator for weight gain for both components. Since they found that mindfulness is negatively correlated to impulsivity, they conclude that it could be used to counteract the lack of control that leads to weight gain.[238] However, the problem after dieting is *loss* of control as the result of physiological mechanisms that over several months propel the body to restore lost weight,[5,6] not *lack* of control. Since mindful eating emphasizes controlling eating,[239] it



Note. The grey background shows the weight stigma that permeates all considerations of obesity and eating disorders.

FIGURE 3—Comparison of (a) a psychological explanation of weight gain and maintenance with (b) the physiological impetus to weight gain after weight loss and (c) the simplicity of weight maintenance when physiological mechanisms control weight.

could be helpful in dealing with alexithymia or lack of interoceptive awareness, but will have minimal long-term effect on loss-of-control eating.

Figure 3b gives a succinct summary of the D_{1-3} routes of Figure 2. Dietary restraint is used to produce a lower weight. Disinhibition (loss-of-control eating) reflects the breakdown of that restraint and causes long-term weight gain. This diagram reinforces the conclusion of the review that disinhibition produces weight gain[228] but attributes it to its correct source: physiological processes.

Figure 3c is a succinct statement of how lifetime maintainers[126] keep their weight within plus or minus 5 pounds.[108] Health at Every Size,[240] Eating Competency,[240–245] the Trust Model,[246] and Intuitive Eating[246] all reflect this diagram. A recent review shows that most programs based on these principles are successful in terms of weight maintenance and physiological measurements presumably related to future health problems.[247] However, many of these programs do not acknowledge that eating for emotional reasons is part of life: all over the world people eat to celebrate weddings, holidays, and other special occasions, as well as offer food to console a person in difficulty. Physiological processes are perfectly capable of accommodating this variability of intake.[5,6,126] ■

About the Author

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Human Participant Protection

Institutional review board approval was not necessary for this article deals with the development of theory based on the discussion of the investigators. The study, Community Based Participatory Research to Improve Health in Children, fomented the faculty, student, and community conversations that resulted in this article. It was approved by the committee to protect humans in research studies of the University of Puerto Rico, Mayagüez Campus.

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