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Causes of obesity

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HONORS THESIS

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In America, overnutrition has become a greater problem than undernutrition. One out of every five children in the country is overweight as a result. This is important since four out of five overweight children become overweight adults. Cappon (1973) calculated that the stored energy in the overweight women of North America would be sufficient to provide heat and light to New York for one year!

The prevalence of obesity in the United States is estimated at between 40 to 80 million people (Stuart and Davis, 1972). A marketing research study done in 1964 (Wyden) found that 9.4 million Americans were dieting, 16.4 million were watching their weight, and an additional 26.1 million were concerned about their waistlines. There were an additional estimated 27 million who were unwilling to acknowledge their obesity, resulting in a total of 79 million Americans who had reason to be concerned about their weight. This represented fifty-eight percent of the population at that time.

The preoccupation with obesity finds expression in the popularity of books and articles dealing with dieting and weight control, the rapid growth of weight reduction groups such as Tops and Weight Watchers, as well as the full marketing of numerous low calorie and dietetic foods. Much of the interest in weight control is a consequence of the current standards of physical beauty in our society. But another more pressing reason for interest in weight control is the documented evidence relating obesity to various somatic disorders.

Weinhaus (1969) found that for each five pounds above ideal weight, life expectency is reduced about one year. These excesses in mortality rates are attributed to the greater number of deaths from degenerative diseases among obese people.

Mayer (1968) has noted that obese individuals in the United States comprise a minority group which suffers from prejudice and discrimination. As a nation, we are programmed to believe that the slender figure is a necessary criterion for acceptance into society. Clothing fashions, hair styles and commercial advertisements are geared towards the petite figured woman. Life insurance rates, job promotions, and social popularity are based on physique. The nature and extent to which this prejudice effects the obese individual is hard to determine. Evident are the psychological hazards for the obese-including feelings of inferiority, self rejection, and overdependence on others for acceptance.

Obesity is an excessive amount of subcutaneous nonessential fat (Craig, 1969). Fat is metabolically formed when more food energy or calories are consumed than the body needs. The excess energy is stored in the tissues, usually in the form of fat. The cause of obesity, by its simplest explanation, is an excessive caloric intake beyond that required for energy expenditure. Three thousand five hundred calories equals one pound of fat. A man twenty-five to thirty percent, and a woman thirty to thirty-five percent, above their recommended weights are considered obese. The average non-obese man usually consumes about one million calories per year and stores fifteen percent body weight-enough to provide for all caloric needs for nearly one month. His body stores remain quantitatively unchanged because he expends an equal number of calories. An error of ten percent in either intake or output leads to a

thirty pound change in body weight within one year (Leitenberg, 1976).

A vital factor in the regulation of body weight is physical activity. In our affluent society there has been a marked decrease in physical activity which contributes to the recent rise of obesity as a major health problem. Obesity is rarely found in underdeveloped countries. Not just the lack of food but also their increased amount of physical activity, especially without the modern conveniences of our luxurious living, enable them to avoid obesity.

Though lack of physical activity may explain why obesity has been increasing in this country, it is not an adequate explantion for the extremely obese individual whose excessive caloric intake far exceeds the needed quantity. The mechanisms which regulate food intake are complex. Mayer (1968) has provided evidence to support a multicausal etiology and has noted that it is an oversimplification to say overeating or inactivity is the sole cause. There is a multiplicity of interactions leading to the caloric imbalance which causes obesity. Mayer (1968) implicated that various factors may influence the development of obesity. For example: biological factors including genetic predisposition or heredity and damage to the hypothalamus; social factors including eating habits and environmental cues; and psychological factors including personality and emotions. Mayer notes that obesity may be a characteristic of more than one disorder and may be the result of interactions between biological and psychosocial factors.

A popular belief is that obesity is caused by metabolic factors but this theory has little empirical support. Kaplan and Kaplan (1957) found no relationship between various metabolic factors and obesity. They reported that glandular abnormalities could be related to fewer than five percent of the total number of cases

surveyed.

Recent studies indicate that the brain contains a special control center called the hypothalamus which motivates feeding and registers satiety. The lateral hypothalamus (an area of the brain to the immediate right and left of the ventromedial hypothalamus) can be described as the "feeding center" of the brain. Electrical stimulation of the lateral hypothalamus evokes stimulus bound eating and drinking, whereas destruction of this area by lesions stops ingestion and results in eventual death. (Anand and Brobeck, 1951).

The ventromedial hypothalamus appears to be involved in the cessation of eating and drinking, making it the "satiety center". Stimulation of this area of the brain causes the animal to stop eating, while destruction causes the animal to overeat and become obese (Hetherington and Ranson, 1942). When food intake is sufficient, the satiety center is thought to signal other parts of the brain to reduce the feeding drive. But when the satiety center is destroyed, the source of stimuli to reduce feeding is lost and eating becomes continuous.

Miller, Bailey, and Stevenson (1950) found the hunger drive of ventromedially rats to be much lower than that of normal rats. This suggests that ventromedial lesions interfere with the mechanisms which stop eating, not hunger. The above findings imply that obese individuals may have some type of ventromedial disorder which interferes with their stop eating mechanism causing them to overeat.

Mayer (1968) has proposed a glucostatic theory relative to control of food intake by the satiety center. In general, the theory proposes that the hypothalamus contains very sensitive

receptors for measuring glucose utilization and when utilization of glucose is high, satiety occurs. The hypothalamus reacts to the rate of passage of glucose or related ions. A low level of glucose utilization inhibits satiety and initiates hunger. If the hypothalamus is malfunctioning, food intake may be out of balance. Metabolic obesity is a state reflected by a biochemical error in the animals mechanism rather than the regulatory center.

Nisbett (1972) proposes that most obesity is a function of an individuals biologically determined "set point". Heredity and early nutritional conditions determine the total number of fat cells. This number is fixed and cannot be altered. Weight reduction must come from a decrement in the size of the cells (Hirsch and Knittle, 1971). An obese individual with enlarged cells suffers from an energy deficit and literally starves when trying to lose weight (Nisbett, 1972). It is not surprising that the obese individual finds it very difficult to adhere to low-calorie diets in order to maintain weight losses.

Keys (1950) found another interesting phenomenon based on this set point theory. Normal weight individuals displayed a remarkable capacity to return to their normal "set point" weight even after large fluctuations such as weight losses during an illness. Successive body compensations are mechanisms whereby a deficit or excess in daily food intake is corrected over a period of time. Sims (1968) found that an obese individual's weight also stabilized this way; however his "set point" was higher than that of a normal weight individual.

Mayer (1968) presents a convincing argument for the role of heredity as a causal factor in obesity. He points out that heredity is readily accepted as an explanation for adiposity in

animals (greyhounds tend to be leaner than bulldogs), but this same argument is rejected when applied to humans. Mayer (1957) found that eighty percent of the offsprings of two obese parents are likewise obese. The percentage drops to forty percent when only one parent is overweight and ten percent when neither parent is obese. Davenport (1923) compared parents to their children. He found that out of fifty-one children of slender parents, none were overweight and out of thirty-seven children of obese-nonslender parents-all were at least average weight and a third were obese. Important to note is that these findings may be due to environmental factors such as eating habits adopted from parents.

Mayer (1968) compared body weight similarities between fraternal and identical twins and found that fraternal twins showed more variability in body weight than do identical twins. However, expected similarities among common gene pool members may not occur when their environmental differences are extreme. Although hereditary make-up may increase the likelihood of overeating or under exercising, environmental factors can overcome genetically transmitted weight predispositions.

Overfeeding an infant or young child can lead to lifelong obesity since the number of fat cells in a person's body are not hereditarily fixed but are determined by the amount of food consumed during the early years of life. The number of fat cells becomes permanent once physical growth ends, around late adolescence. It is the number, and not the size, which preposes the individual to obesity. Dieting reduces the size of the cell but never the number. Parents should not overfeed their children and food should not be used to pacify an infant, especially if he or she has just recently

been fed. Fat babies are believed to be healthy babies but this is not so. One way to prevent obesity in adulthood is not to overfeed infants.

Hicks (1970), in an extensive study of 3,444 preschoolers at various socioeconomic levels discovered that mothers implant bad eating habits on their children, causing them to overeat. Twenty-three percent of the parents in this study used food as a reward for good behavior; ten percent held back food as punishment and twenty-nine percent used food as a pacifier. Mothers who overestimated food needs tried to force children to eat too much. Another frequent practice when a small child dawdled at meal times was forcing him to clean up his plate before he could do anything else.

The above practices equate food, particularly those of high caloric content, with "reward". The individual will then continue to reward himself as an adult by overeating or eating too many desserts and other sweets.

To an unusual degree, obesity is under social environmental control, regardless of its genetic or biochemical components. The age of obesity onset may be the critical factor. Mayer (1968) has noted that this onset in childhood is more disruptive to personality than if the onset occurs during adulthood. Childhood obesity is associated with emotional disturbances and increases the tendencies toward overweight problems later in life. Eighty-six percent of overweight boys remain overweight as men compared to only fourteen percent which reduce to normal weight. (Abraham and Nordsieck, 1960). Similarly, eighty percent of obese girls remain obese as women compared to only twenty percent which became normal weights. Odds against an overweight child becoming a normal weight adult are

four to one at age twelve and increasing to twenty-eight to one for those not reducing during adolescence.

Childhood-adolescent obesity may initially be caused by glandular dysfunction or genetic structural make-up, but as soon as the child-adolescent puts on enough weight to feel "different" from others, the psychological as well as biochemical factors come into play. The obese adolescent is a victim of serious threats to his physical, social, and emotional well being.

In the area of physical activity, Reindeau(1958) reported that excess body fat exerted an adverse effect upon motor ability-imposing a limitation to motor fitness in general. If the obese child is unsuccessful in play activities, he will refrain from participation, thus eliminating an important avenue for the elimination of excess calories. A vicious cycle may be created, especially with the absence of the socializing aspect of play. The child's alienation from play contributes to improper psychological development at a critical stage in life.

Social development is also hampered through rejection by peers and psychological disturbances develop from abnormal self-loathing caused by cruel teasing from peers (Cole, 1964). Since appearances are important at this age, overweight girls have little appeal to boys. They need help from their family, teachers, and doctors to find happiness and a normal adjustment. The fat adolescent boy is not much better off but canfind acceptance in becoming a clown. His extra poundage makes it awkward for him to participate in games-the surest road to admiration among peers. The fat under his nipples and buttocks give rise to comments in the locker room. He is often interested in pornography, partly due to rejection by girls and the necessity of receiving attention from peers. If he has money, he

will often try to buy acceptance (Cole, 1964).

Although there is considerable variation among biologically based theories, they are uniform in their implications for treatment. Obesity is built in at birth or shortly thereafter. If the physiological malfunction causing the obesity is not or can not be corrected, then attempts to diet are doomed to failure.

There are various psychological factors presumed to cause obesity but much of this theorizing is based on psychoanalytic personality theory. Schopbach and Matthews (1945) characterized mothers of obese individual's as dominant and powerful, whereas the father's were seen as weak, submissive and unable to provide guidance for the child.

Many theories have sprung up suggesting hidden meanings behind overeating. Kaplan and Kaplan (1957) have summarized these theories by stating "The present authors believe that the great number and variety indicate that any emotional conflict may eventually result in the symptom of overeating; it is their conclusion that the psychodynamic factors causing obesity are non-specific (pp. 196-197)." They go on to explain that the central notion of these theories is that eating serves as an anxiety-reducing mechanism for the obese.

This view has popularly been accepted, but empirical investigations have yielded conflicting results. Schachter, Goldman, and Gordon (1968) tried to arouse fear by the threat of painful electric shock. Although obese subjects were significantly more frightened than their normal counterparts, their food consumption was not significantly greater. Abramson and Wunderlich (1972) also tried to assess the effects of fear of electric shock and interpersonal anxiety on the eating behaviors of obese and normal-weight subjects and came up with similar results. The obese subjects were more

responsive to manipulation; however, the anxiety did not affect their eating behavior. McKenna (1970) found conflicting results which indicated that obese subjects consumed more food under high-anxiety conditions.

Evidence supporting eating as an anxiety-reducing mechanism was provided by Holland, Masling, and Coplay (1970). Interview data showed that obese individuals eat more often when anxious and depressed than when hungry. Leon and Chamberlain (1973) compared eating habits of obese individuals with those of normal-weight persons participating in a weight reduction group. For normals, hunger was the most frequently cited stimulus for eating. The obese; however, were more likely to choose one of several arousal states as factors triggering eating. Boredom was found to cause increased eating in both obese and normal subjects. (Abramson and Stinson, in press).

Any conclusions concerning the part emotions play in the eating behavior of obese individuals would be premature in light of the conflicting results. Much of the difference may be attributed to research methods. Obese individuals may not accurately report their food consumption during interviews. Other experimental studies may also be inadequate since the subjects had only one type of food, such as crackers. Leon and Chamberlain (1973) have pointed out that results from similar experiments would not be adequate since a large variety of more appetizing foods is available in the natural environment. Although the effects of emotions on eating are uncertain, there is supportive evidence that obese individuals are more reactive to emotional stimuli than normals (Abramson and Wunderlich, 1972, Rodin, 1973).

The psychological approach to dealing with obesity caused by anxiety arousing stimuli requires that the emotional conflict causing overeating be resolved. Bruch (1973) states that the aim of psychotherapy is to effect a meaningful change in the personality so that the individual can live a constructive life without misusing eating in bizarre and irrational ways. The problem is how to achieve this. (p. 334). Focusing in on eating as an anxiety-reducing mechanism has prompted the use of behavioral techniques for controlling eating.

Another perspective from which to look at obesity is from the social-environmental point of view. Eating habits frequently cause obesity. This is particularly true of those individuals who gain weight with age. As the person gets older, his metabolic rate decreases as does his energy output. He needs to eat less, but since the amount he consumes is determined more by habit than by body requirements, he may continue to eat the same as when he was in his twenties.

External cognitive and social cues such as taste, smell, and sight of food are believed to control eating in obese individuals. In contrast, the food consumption of normal weight people is believed to be a function of internal, visceral cues such as gastric motility and hypoglycemia. The lack of correspondence between internal states and subjective feelings of hunger was first demonstrated by Stunkard and Koch (1964). Using a balloon to measure stomach contractions, they found that obese subjects were more likely to report hunger when their stomachs were not contracting and conversely, denied hunger when their stomachs were contracting. In contrast there appears to be a relatively close correspondence between gastric contractions and reports of hunger for normal-weight

subjects.

Schachter, Goldman, and Gordon (1968) had subjects report to their laboratory after missing a meal. Half of both the obese and normal weight group were fed prior to the start of the task, which was rating the taste of various crackers. Deprived normals ate more than their peers who had been fed, whereas no difference was found in the cracker consumption between the two obese groups. Again, eating by the obese appeared to be independent of visceral cues that trigger eating for normal-weight individuals. Nisbett (1968) found similar results when he gave either one or three sandwiches to obese and normal subjects several hours after not eating. Subjects were told additional sandwiches could be found in the refrigerator. Obese ate less when given one sandwich and more when given three. Normals ate the same regardless of the number of sandwiches given.

A series of ingenious studies by Schachter and his students illustrated the various external cues that can determine food consumption. For example, Schachter and Gross (1968) used doctored clocks to manipulate time. Obese subjects ate almost twice as much when they thought it was 6:05 as they did when the clock read 5:20. The role of time as an external cue regulating food consumption for the obese was supported by the results of a field study using trans-atlantic flight crews. The likelihood of being troubled by the discrepancy resulting from crossing time zones was less for heavier crew members. The overweight flier adjusted his eating according to the local time, independent of actual food deprivation (Abramson, 1977).

Ross (1969) demonstrated that cue salience affected an obese person's eating habits. Significantly more cashew nuts were eaten

when illumination was provided by a 40 watt bulb than by a 7 1/2 watt red bulb.

In a study exploring the role of taste as a determinant of eating, Demke (1971) provided obese and normal-weight subjects with either a good-tasting vanilla milkshake or with a milkshake that had quanine added to it. Obese subjects drank more than normals when the milkshake was good, but drank less when it had been adulterated. Other studies have shown that the more effort is involved in food (such as shelling nuts) the less obese individuals will eat. Nisbett and Kanouse (1969) found that obese individuals purchased more food in a grocery store, shortly after eating than normal weight individuals who purchased less food immediately after eating. These findings suggest that obese are more stimulus bound, basing their self-attribution on external cues.

Recently several investigators have demonstrated that the externality of obese individuals goes beyond the realm of food consumption. Rodin (1973) found that obese subjects, while working on tasks requiring concentration, were more disrupted by outside disturbances. Pliner, Meyer, and Blankstein (1974) demonstrated that obese subjects responded more strongly to positive affective stimuli than did normal weight subjects. The clear implication of these and similar studies is that obese persons are more responsive than normals to a variety of external cues. The external-cue hypothesis suggests a variety of specific techniques that have been incorporated in behaviorial self-control weight reduction programs. The underlying idea is to reduce the frequency of occurence of the various external cues that cause eating, thereby decreasing food consumption.

As evident throughout this report, obesity has multiple causes. These multicausal factors must be taken into account to understand what predisposes the individual to excessive body weight. Many reducing regiments are available for the individual including such things as diet pills, fad diets, and reducing machines. However, these regiments may only provide minimal short-term assistance while the long term problem procures.

The various behavioral treatments are based on the assumption that obesity results from excessive eating and inadequate energy expenditure. Behavioral modification techniques are used to reduce or eliminate the maladaptive behaviors leading to overeating and teaching more appropriate behaviors to the individuals.

In this way, the individual learns to maintain his normal weight by not reverting back to his old eating habits which originally lead to the obesity.

Permanent weight loss by dieting can only be accomplished through calorie reduction. This can be accomplished by either cutting down on the portion sizes of the food the individual is accustomed to eating or by restricting food intake by actually counting the calories consumed. In any case, the individual must meet the nutritional requirements of his body.

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