Genetic and Physiological Contributions to Obesity
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Abstract

Obesity can be observed through the use of both animal and human models. The most important contribution of the two models is the causal link of genetics and physiology to obesity. Through chromosomal mapping, animal models provide evidence that a gene mutation is the source of obese (ob/ob) rats. This presents the hypothesis that the only difference that exists between lean and obese rats is in phenotypes and not in behavioral disposition. Human models emphasize the genetic cause of obesity through use of twin and adoption studies, supporting the concept of nature over nurture. Through the use of body mass index (BMI), classifications can be made for individuals as being “overweight” or “obese”. Many physiological factors contribute to obesity, such as the roles of specific neuropeptides, which have influence on food intake, digestion, and energy levels. Such neuropeptides include Neuropeptide Y (NPY), Cholecystokinin (CCK), and Ghrelin. A second link of obesity to physiology is the presence of leptin, which acts to suppress the amount of food consumed. Specific peptide systems also play a part in the physiological contribution to obesity, and include dopamine, melanin-concentrating hormone (MCH), and interleukin. Finally, the role of adipose tissue, which functions as a storage place for fats, is examined. It plays an important role during times of fasting when energy is needed. However, in excess, adipose tissue can cause complications that result from obesity. Treatment options, which include gene therapy and the use of administered leptin, have also been considered.
Genetic and Physiological Contributions to Obesity

Introduction

Obesity is becoming a world wide epidemic affecting both children and adults. This disease is caused by an imbalance in food intake and energy expenditure. Genetics can also play a role in the development of obesity in both animal and human models. The animal model for obesity is observed in obese (ob/ob) rats that become obese due to a genetic mutation. On the other hand, the role of genetics in human models is apparent in a variety of sources, such as adoption and twin studies, which show that one’s phenotype more closely resembles that of their biological parents rather than their adoptive parents, demonstrating that environment plays little to no role in obesity.

The body mass index (BMI) is a universal system by which obese and overweight individuals can be classified and has become the measure by which to determine and identify obesity in humans. BMI is calculated by taking weight in kilograms divided by the square of the height in meters (Allison, 1999). A BMI of 25 or greater designates the individual as being “overweight”, whereas a BMI greater than 30 indicates that the individual is “obese” (World Health Organization, 2000).

Several physiological factors can also contribute to obesity. Such influences include neuropeptides, leptin, peptide systems of dopamine, melanin-concentrating hormone, and interleukin, and the storage of fat by adipose tissue, which is the body’s main energy source. Fat storage increases in adipose tissue when caloric intake is greater than the amount of energy expenditure. All of these physiological contributions can interact to produce obesity and related side effects.
Due to the growing epidemic of obesity around the world, actions need to be taken to ensure that the problem doesn’t become uncontrollable. One such strategy for controlling obesity is through treatments that are being discovered through animal models. For example, gene therapy, using leptin and neuropeptide Y, have produced weight loss results in rodents over short periods of time. Focusing in on the unbalanced equation of more food intake to less energy expenditure is where treatment need to start. Other treatments involve manipulating the energy intake balance through diet and exercise, group therapy, and even surgery, such as gastric bypass.

The factors that cultivate obesity are far-reaching; from the foods we eat to the sedative lifestyles promoted by advancements in technology. These factors, along with a genetic disposition dramatically increase one’s probability at becoming obese. As a nation, we need to come together in the fight against obesity as a root of many other diseases to improve our health.

Obesity in Animal Models

Using rat and mice models as a means to study the genetics of obesity is due to the rapid development of their genetic map in a short time frame. Such models consist of lean and obese rats. An obese rat can be defined as overweight, hyperphagic, hyperinsulinemic, and insulino-resistant (Chaouloff, 1994) and is usually linked to a mutation of the \( \text{ob} \) gene. A mutation of this gene causes a shortened and dysfunctional protein, resulting in the rat’s \( \text{ob/ob} \) phenotype (Walder, 1997). It has recently been found that cloning can also be a precursor for obesity in mice. Obesity in cloned rats and mice is indicative of epigenetic abnormalities, which results from defective nuclear programming (Inui, 2003).
Identification of the animal as being lean or obese can be determined as early as 7
days old. Skin biopsies and observing the morphology can be used as a technique to
determine the phenotype of the rat. This technique requires minor surgery, allowing for
repeated examinations of a single animal. Increased age produces more accurate
predictions of the rat’s classification (Hausman, 1983).

Though a physiological difference can be observed between lean and obese rats,
there has not been shown to be a behavioral difference. In a study, ten lean and ten obese
rats were exposed to novel experiences, which included an elevated plus-maze, the open
field, and the black/white box. These experiences are designed to produce anxiety in the
rats. However, the performance of the ten lean and ten obese rats did not differ based on
their specific phenotype, for such factors as locomotion, grooming, time, and rearing
(Chaouloff, 1994). The only behavioral difference between the animals is the amount of
food intake, which results from an inability of the obese rats to become satiated and stop
eating. This shows that the difference between lean and obese rats lies only in their obese
phenotype.

It has also been found that certain chromosomes are linked to dietary obesity.
Quantitative trait locus (QTL) is the procedure used to determine the mapping of the
chromosomes and the phenotype for which they influence. The mapping is done on
mice, referred to as F1, that have been intercrossed with one another. This interbreeding
of two different mice species creates a generation known as F2. Through continued
intercrossings and backcrossings, the mice become sensitive to the diet they are given.
For example, the F2 mice that were backcrossed and given a low-fat diet, became obese
spontaneously, though none of the parental lines showed evidence of this spontaneous
obesity. This is due to the polygenetic mutations that can result from backcrossing, promoting obesity. However, those rats that were intercrossed a single time showed no signs of obesity, though they were given a high-fat diet (West, 1994). The genetic cause of obesity is suggested to result from polygenetic mutations, due to the repeated backcrossing of the mice (Horvat, 1999).

Mapping has also determined that two chromosomes, 9 and 15, are responsible for the obese phenotype and are known as dietary obese 2 (chromosome 9) and dietary obese 3 (chromosome 15) (West, 1994). The animals in this study, which demonstrated positive traits of the obesity chromosomes, were placed on a high fat diet in order to induce obesity. Upon the rat’s weight gain and generated obesity, it was supported that both genetics and environment influence obesity. However, chromosomes 2, 12, and X have also been found to demonstrate positive traits for obesity (Horvat, 1999).

Establishing that these three chromosomes have been found to be a precursor for obesity. It is possible to correlate the chromosomes within the mice to those of humans, providing us with greater insight to human obesity (Lembertas, 1997).

Animal models can be used to study the genetic influences of obesity. The observation of behavior and chromosomal mapping are two methods used to study obesity in animal models. By using both rat and mice models, more can be learned about obesity in humans.

**Obesity in Humans**

It is estimated that 300,000 adults will die of obesity related causes each year in the United States alone (Mokdad, 2003) and more than 80% of those death will be among
those with a BMI of 30 kg/m or greater (Allison, 1999). One such risk factor for death in those considered to be obese is diabetes. A telephone questionnaire, known as the Behavioral Risk Factor Surveillance System (BRFSS), was conducted by the Center of Disease Control and Prevention, as well as through many local heath Departments. It was found that the incidences of diagnosed diabetes had increased by 0.6% from 2000 to 2001, with the highest rate found among blacks, those aged 60 years or older, with less than a high school education. However, some confounding variables include the way in which the survey is administered. For example, since it is conducted by telephone, subjects can over or under estimate for such questions pertaining to height, weight, and income and can fail to provide accurate information about past health records and current physical health. These confounding issues were addressed in the article and were taken into consideration when stating certain rates of diabetes among obese in the United States. It was also found that those individuals deemed as obese ranked the highest not only in diabetes, but also high blood pressure, high cholesterol, asthma, arthritis, and overall poor general health (Mokdad, 2003).

Obesity has become a growing epidemic in the United States. In 2001, approximations were made to determine that roughly 22.9 million women and 21.4 million men were considered obese in the United States (Mokdad, 2003). There are many risk factors for obesity, most of which appear around adolescence. Diet, sedentary behaviors, and physical activity have all been examined as causal links to childhood obesity, however, none provide strong support (Patrick, 2003). Metabolism, physical activity, and diet are all factors that are influenced by genes. For example, resting energy expenditure, or metabolism, is thought to be influenced by genetics, though the
correlation to weight gain is small. However, a stronger correlation has been found between weight gain and total daily energy expenditure. The lower the total daily energy expenditure, the more likely weight gain will occur. This is believed to be a predisposition to weight gain, though not as influential as metabolism and even living conditions. However, metabolism and living conditions prove to not contribute significantly to weight gain (Weinsier, 1998). Poor eating habits have been found among normal and over weight adolescence, suggesting that most children are not meeting daily nutritional requirements. Normal weight children participate in a greater amount of physical activity and only slightly less time watching television than overweight children (Patrick, 2003). It appears that weight gain and an increased BMI can be attributed to a multitude of external factors, as well as genetics, which proves to be the greatest influence on obesity. Genetic mutations of interleukin-6 receptors found in Pima Indians have been attributed to an increased vulnerability to obesity (Wolford, 2003). Genetics, and any subsequent mutations in humans, can be correlated to a greater susceptibility of becoming obese.

The role of genetics in human obesity can be best demonstrated through adoption studies. These studies have shown that the BMI of the adopted child more closely resembles that of their biological parents and not of their adoptive parents. This shows a genetic contribution to obesity, rather than the environment in which one is raised. A study was conducted by the Swedish Adoption/Twin Study of Aging (SATSA), which looked at the importance of genetic and environmental factors on weight gain and BMI. The study’s sample consisted of 93 pairs of identical twins that were reared apart, 154 pairs of identical twins reared together, 218 pairs of fraternal twins who were reared
apart, and 208 pairs of fraternal twins reared together. Those twins who compose the 2 sample reared apart were separated early in life and those reared together were identified through the Swedish Twin Registry. There were three principle findings in this study. The first proved strong evidence for the influence of genetics on the BMI. The correlation between genetics and BMI for the 93 pairs of twins who were reared apart was found to be 0.70 for men and 0.66 for women. The study also found that there was great significance in the influence of nonadditive genetic variance later in life. A previous study was conducted on American twins at the age of 20 and then again at the age of 45. No evidence was found at the age of 20 for nonadditive variance, whereas when tested at age 45, there was proof of this variance. Thirdly, this study found that there was no correlation between the environment in which the twins were reared together or apart and BMI. The influence of the environment on BMI is unique to the individual and is not caused by the impact of family and other surroundings (Stunkard, 1990). This idea was further supported by another study conducted on the influences of genes and shared family environment on adult BMI. No strong statistical evidence was found to support the idea that the shared influences of a familial environment correlated with weight gain and an increased BMI (Volger, 1995). Any familial resemblance of BMI can be attributed to genetics and not environment. One possible confound that could have underestimated the data was the self reported questionnaire of height and weight, both current and maximum, by the adoptees. However, the validity for this method was determined acceptable since a second questionnaire was also sent to and completed by the adoptees, biological parents and siblings, and adoptive parents and siblings (Vogler, 1995).
This idea of genetic influence on BMI and weight gain is further supported by a third study conducted on male twins (Fabsitz, 1994). Three examinations were performed on the male twins, who were war veterans and served in the Military during World War II or the Korean War. The first examination took place during 1969-1973, a second in 1981, and a third in 1986. At the third examination, the subjects were asked to self-report their maximum weight and the corresponding age at which this weigh was reached. This could pose as a confounding variable, due to inaccurate self-reporting of the weight and age. From this data, it was found that the bulk of the twins reached their maximum weight around 60 years of age. This trend of weight gain around a central point is attributed to genetics. However, the variation around this central point was found to be environmental, due to the fact that no genetic link was found (Fabsitz, 1994).

The use of twin and adoption studies is imperative to finding genetic links to obesity in humans. By finding genetic causes of obesity, it can be determined that human obesity is more greatly attributed to nature over nurture. Such findings can also play an important role in discovering a cure or treatment options for human obesity.

**The Role of Neuropeptides**

Neuropeptides play an important role in weight gain and obesity. They have influence on such factors as food intake, digestion, and energy levels. Three such neuropeptides include Neuropeptide Y, Cholecystokinin, and Ghrelin.

The first specific neuropeptide is Neuropeptide Y (NPY), which is an orexigenic molecule that acts to increases the desire to eat (Inui, 2003). NPY is one of the signals involved in the eating patterns that trigger appetite and satiety. When one is satiated, low
and slow levels of NPY are secreted as pulses in the arcuate nucleus-paraventricular nucleus (ARC-PVN) axis, which plays an important role with the hypothalamus in daily meal planning. However, just before mealtime or after fasting, the secreted pulses of NPY are accelerated in an attempt to sustain the energy drive until it is replenished. NPY is also important in maintaining the homeostasis of weight by increasing and decreasing levels and concentration as needed (Kalra, 2004).

There are four NPY receptors, which have been demonstrated to have effects on one’s body weight and eating habits. These receptors are Y1, Y2, Y4, and Y5 (Hohmann, 2003). A study was conducted Herbert Herzog (2003), which looked at mice known as knockout mice, meaning that the specific coding sequence for NPY has been replaced with another gene known as lacZ gene. It was speculated that with the deletion of the given gene, the mice would have a reduction of weight and food intake. However, this didn’t prove to be the case. More specifically, the Y1 knockout mice did not show any great changes in food intake. The mice with the pre-synaptically expressed Y2 receptor deletion resulted in two different outcomes. It was observed by Naveilhan et al. (2001) that there was an increase in food intake as well weight gain. On the other hand, Sainsbury et al. (2002) found that the mice exhibited a reduced body weight. There is little evidence concerning the Y4 knockout mice, therefore not much is known about its function. What has been shown is a reduction in body weight and food consumption in these mice. However, it has been found that the deletion of the Y4 receptor has no effects on ob/ob mice in regards to food intake and weight loss. Finally, the mice with the deletion of the Y5 receptor, known as “the feeding receptor”, have shown normal growth and development in young mice, but later develop obesity with an increase in the
amount of food consumed (Herzog, 2003). The Y5 receptor may be activated later in
life, leading to a later onset of obesity in the rats.

A second neuropeptide involved in obesity is Cholecystokinin (CCK). The
neuropeptide CCK works to control the amount of food consumed, meal size, and
facilitates digestion of food within the small intestine. CCK is present in the brain as
well as the gastrointestinal tract. Since CCK reduces meal size, it has been shown that
rats injected with the neuropeptide will stop eating to engage in other activities such as
exploration, grooming, and sleeping. There are two different CCK receptors, both of
which are members of the G-protein family. The first receptor is CCK-A. It has been
shown that when a CCK-A agonist is administered, it causes a decrease in food intake.
The second receptor, known as CCK-B, fails to produce satiation in the presence of an
agonist (Bi, 2002).

Studies have been conducted on a specific species of rats known as the Otsuka
Long-Evans Tokushima Fatty (OLETF) rat. It was developed as a model for obesity
when it was discovered among Long-Evans rats in 1984 and established for breeding in
1992 (Bi, 2002). This species lacks the CCK-A receptor. Therefore, if the OLETF rat is
given a high fat diet, it will increase in size and weight, whereas, lean rats on the same
diet will compensate for the caloric increase by limiting the amount of food intake
(Chandler, 2003).

One such study that examined the relationship of CCK and obesity in OLETF rats
was conducted by P.C. Chandler et al. (2003). Using CCK-8, which has full biological
activity and contains 8 amino acids, the study focused on the response of the peptide due
to diet-induced obesity (DIO). DIO is when animals are given a high fat diet in order to
increase their weight. There were two groups of DIO rats used in the study. The first group was DIO-prone, which means these rats are more susceptible to weight gain due to the high fat diet. The second group consisted of DIO-resistant rats, which means they are less likely to gain weight on a high fat diet. It was found that CCK-8 exerted much greater satiety effects in the DIO-prone rats and not in the DIO-resistant. This is due to the inability of the DIO-prone rats to limit their food intake as the DIO-resistant rats were able to do, thereby, exceeding the amount of food needed to meet metabolic needs resulting in weight gain (Chandler, 2003).

Ghrelin is a third neuropeptide that plays a role in obesity and is involved in the management of body weight and food intake (Wang, 2003). Ghrelin is a peptide that is found in the periphery and central nervous system (Beck, 2004). In healthy men, the level of ghrelin is inversely correlated with BMI, thereby increasing appetite and food intake. For example, in patients that are exhibiting signs of anorexia nervosa, high level of ghrelin have been found. On the contrary, in those considered obese, the levels of ghrelin are much lower (Wang, 2003). This is due to the fact that the concentration of ghrelin is lowest after a meal, whereas it increases just prior to the consumption of another meal. Obese people are always eating, which maintains the low levels and those who are anorexic rarely eat which keeps the levels of ghrelin raised.

Beck et al. (2004) conducted a study, which examined the amount of ghrelin in both lean and obese rats during different weight gain periods. At two months of age, measurements of ghrelin, insulin, and leptin were recorded. Also noted was the weight and food consumption of the rats. When leptin levels were measured at 2 months, it was found that the concentration in the lean rats was more than 10-fold higher than in the
same lean rats at 6 months. However, at both 2 and 6 months, the levels of leptin in obese rats was higher by nearly 20-fold than in the lean rats. It was found that the obese rats ate about 60% more than the lean rats and weighed about 50% more. The obese rats also had much lower concentrations of ghrelin at 2 months than the lean rats. The second measurements were taken at 6 months of age. The results revealed a 47% increase in food consumption in the obese rats and were overweight by more than 70%, as compared to the lean rats. However, the difference in ghrelin concentration that was seen at 2 months was no longer present at 6 months. The ghrelin levels do not correlate with body weight, due to the fact that when the lean rats were 6 months old, they had the same body weight as the obese rats at 2 months. The data observed in this study shows that it is not body weight that determines the down regulation of ghrelin, but rather body composition is what is important. This establishes a defense mechanism for the animal against developing obesity in the early years of its life.

**Leptin**

The leptin gene was first described in 1994 and acts to suppress the amount of food consumed as well as increase the expenditure of energy (Weinsier, 1998). Leptin, which is secreted by adipocytes, or fat cell, is a part of a feedback loop, which helps to maintain fat storage. It acts as an inhibitor to orexigenic molecules, such as NPY (Inui, 2003). Therefore, when there is an increase in the amount of available leptin, NPY signaling in the ARC-PVN axis is inhibited, acting as an appetite suppressant (Kalra, 2004). However, the opposite is true of activated anorexigenic molecules, such as melanocortin, when activated lead to weight loss due to the stimulation to expend energy
(Inui, 2003). Such molecules demonstrate that leptin administration can be used as a weight loss method, since it works to suppress appetite and increase energy expenditure (Fan, 2003).

Souza et al. (2004) found that leptin concentrations correlate with total body weight in both children and adults. Their study looked at the concentration of leptin levels in the body upon completion of an aerobic exercise test. In fit adults, the levels of leptin should be low following the recovery phase of exercise. However, the results showed that the initial leptin levels in obese children were very high and in the range of those levels found among obese adults. This could possibly lead to the assumption that obesity is linked to one’s resistance to leptin, since the presence of leptin results in decreased appetite and increased energy expenditure (Souza, 2004).

Leptin gene therapy has the potential to treat obesity. A study attempted to prevent weight gain in mice through gene therapy. The procedure involved transferring naked plasmid of humans, containing leptin cDNA, into the muscles of mice. Upon completion of the procedure, the amount of food consumption was observed. It was noted that the amount was much lower than that observed in the control mice. Also, there was no weight gain among the treated mice. In fact, seven weeks after the procedure, the weight of the mice treated with gene therapy was 20% less than that of the control. This demonstrates the successfulness of leptin treatment for obesity in mice (Wang, 2003).

**Peptide Systems**

The increase or decrease of feeding and weight can be caused by different peptide systems, including melanin-concentrating hormone (MCH), dopamine, and interleukin.
The first peptide, MCH, releases its neuropeptide and is thought to stimulate rodent feeding. However, by incorporating a MCH-1 receptor antagonist, reductions in food intake are observed in rodents (Shearman, 2003). One such antagonist is T-226296. A study was conducted by T.J. Kowalski et al. (2004), which looked at the cause of the receptor antagonist and determine if it was due to a decrease in the frequency of the meals, the size of the meals, or a combination of both. This experiment was performed on DIO-prone rats. The rat received 1 mg, 3 mg, or 10 mg of the receptor antagonist T-226296. The food intake was observed for a 24-hour period and showed that the 10 mg dose dramatically decreased the body weight of the rat. It was found that rat’s decrease in body weight was in fact due to the reduced size of the meal it received over the 24-hour period. The rat’s behavior was not affected by the MCH-1 receptor antagonist, thereby concluding that the reduction in food intake was not caused by a downturn in behavior, but rather to the meal size reduction (Kowalski, 2004).

The second peptide involved in obesity is dopamine, which is believed to control the stability of energy and ingestion. A study examined the characteristics of body weight and fat, as well as food intake in a mouse that has had their dopamine-3 receptor disrupted (Drd3). Two sets of mice were used for the experiment. The first was a wild-type mouse with no alteration of the dopamine receptor and the second set was mutant mice with a disruption of Drd3. The mice were then divided into groups according to the diet they would receive. The first diet consisted of standard rodent chow and the second was a high fat diet. The animals were exposed to the specific diet for 3 months, at which point they were sacrificed. The mice were all weighed and had fat extracted in order to be tested for fat, water, and lean tissue content. It was found that the male Drd3 mice had
an increase in body weight to the high fat diet, but not to the diet, which consisted of standard rodent chow. However, the female Drd3 mice did not show an increase in body weight in response to either diet, but rather showed an increase in body fat to both diets. Body fat in Drd3 mice will increase despite being given a low or high fat diet. From this, it can be determined that the dopamine-3 receptor is involved in the regulation of both body fat and body weight in mice (McQuade, 2003).

Certain peptide systems found in humans can also have an impact on obesity. For example, the Pima Indians are a populace that is prone to weight gain and an increase in adipose tissue. Wolford et al. (2003) examined the peptide interleukin 6 (IL-6). High circulating levels of IL-6 can suggest the development of T2DM, which has found to be linked to obesity. The subjects for the research experiment were a part of an ongoing study of T2DM in members of the Gila River Indian Community, comprised of 332 families. Genotyping was done on the Pima Indians who were of full heritage in an attempt to find a relationship between IL-6 and BMI. This revealed that the individuals who carried the allele had a high BMI as compared to those individuals with the wild-type allele. It was concluded that any genetic variation in the interleukin 6-receptor gene might play a role in the increased vulnerability to obesity (Wolford, 2003).

**Adipose Tissue**

Through evolution, the body has needed storage of fat to be used during times of fasting. Fat is stored in adipose tissue and is the body’s energy source when little to no food has been consumed (Frayn, 1995). However, the excess storage of fat in adipose tissue is a primary characteristic of obesity, posing a factor for such problems as
hypertension and cardiovascular disease, as well as obesity. These two problems, in conjunction with an increased amount of adipose tissue, form a cause and effect relationship. For example, obesity, in combination with hypertension, tends to promote cardiovascular disease. However, cardiovascular disease weakens the heart, demanding more energy to be spent on daily activities, resulting in lower energy expenditure, promoting weight gain. Cardiovascular disease also exacerbates hypertension. Because the diseased heart is weaker, it must work harder to pump blood, increasing one’s blood pressure. Increased amounts of adipose tissue are directly linked to all three diseases, showing the impact it has on many aspects of health (Frühbeck, 2003).

The area of the body that has the greatest amount of adipose tissue collection can also affect one’s health. For example, those with a high BMI, large waist circumference, and high waist to hip ratio are at a larger risk for developing health problems. One’s body type can play a role in the formation of large amounts of adipose tissue, resulting in disease (Frühbeck, 2003). Such as women who are apple shaped, meaning they are larger on the top than the bottom, are more susceptible to having a heart attack, due to the build up of adipose tissue around the heart.

A relationship between leptin mRNA levels and adipose volume has been established and has been found to be positively correlated with one another. It is suggested that leptin may function as a signal to fat mass as a response to the regulation of energy. Since leptin increases energy expenditure, it may function to decrease the amount of adipose tissue in an attempt to lower the amount of fat stored in the body. However, obesity may be a response to the decrease in leptin expression per fat unit in adipose tissue in mice. If there are lower levels of leptin found in the body, it will
respond in a manner to increase food intake as well as a decrease the amount energy cost, resulting in weight gain (Guo, 2004).

Another relationship has been found between adipose tissue and ghrelin, which is involved in the amount of food consumed established by means of an orexigenic outcome. An active administration of ghrelin resulted in the decrease of arterial pressure, thereby attempting to reverse the side effects and symptoms of heart disease. By reversing the effects of hypertension, ghrelin reduces the possibility of developing cardiovascular disease, thereby lowering one’s chance of fat storage in adipose tissue causing weight gain (Frübeck, 2003).

Discussion

The prospect of being able to treat and control obesity through gene therapy and the administration of a peptide is very exciting. If we are able to treat rodent models with these procedures, the use of humans needs to be more closely examined. Gastric bypass is much too risky and permanent to be the only “cure all” for obesity, though the concept of the procedure is appropriate in the treatment of obesity. However, instead of surgically reducing the size of one’s stomach, why not work to find a way to reduce individual’s craving for foods and the amount that they consume, which is shown to be the result of leptin that is administered from an outside source.

Due to the growing problem of obesity in the United States, greater action needs to be taken in an effort to alleviate this disease. One such undertaking is a greater involvement by the government. For example, standards need to be set for food serving sizes at restaurants. Portion sizes continue to increase every year, promoting over eating
of unhealthy foods. By having the government set a standard for the serving size of food and regulate the portions, people would eat less, resulting unintentional weight loss. It is much easier to lose weight by still eating all of the foods one loves, but by having a controlled portion, the caloric intake and the amount consumed are reduced, resulting in weight loss.

Another step that could be taken to control the obesity epidemic is to have organizations offer free exercise and health sessions that include tips to better eating, exercise classes, weight loss support groups, and education about obesity. Most people who are considered obese are of a low socioeconomic status, therefore can’t afford to pay for a gym membership, nutritionist, or personal chef. By offering free health sessions at places like the YMCA or YWCA, or at local churches, more people could become educated on how to improve their obese lifestyle.

The rise in obesity can be attributed to the lifestyle we, as Americans, are encouraged and thereby enforced to live as a result of many outside factors. For example, the serving size of a meal at restaurants has increased, directly causing the patron to increase the intake of that food. Also, the use of cell phones, Internet, and automobiles have encouraged a more sedentary lifestyle. We as a society seem to participate in activities that require the smallest amount of energy and effort. For example, at colleges nationwide, the use of Instant Messenger is overwhelming. Instead of walking down the hall to ask a friend what they are doing for dinner, messages are sent from the comforts of a computer chair, only exerting energy through one’s fingers. The use of cars has also increased, since driving everywhere has become standard. Our nation has become creatures of habit by driving everywhere and using the phone and Internet for
all forms of communication and information. Though the benefits of ease and immediate
gratification derived from these technological advancements tend to outweigh the
negative, we must realize that the small effort we make today will be advantageous for
our lives in the future.
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