Addiction to Food and Brain Reward Systems

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Overeating is emerging as one of the most pressing health issues affecting developed countries. While it is known that overeating leads to overweight and obesity and a number of associated health risks, the etiology of overeating remains unclear. Overeating shares many characteristics with substance use disorders. Furthermore, overeating has been characterized as an addiction and most likely arises from a combination of abnormal cognitive and neuroendocrine processes. Although emotional states have been shown to mediate reward processing, the implications for hunger mediating reward have not been fully elucidated. In this paper, we discuss the relationship between overeating and obesity with other substance addictions and the neural circuitry they share. Additionally, we discuss genetic and environmental influences on eating behaviors and the implications that these influences have on treatment.

Obesity is reaching pandemic proportions. Recent surveys indicate that 40 million Americans (approximately one-seventh of the American population) weigh 20% more than their ideal weight (McKesson Health Solutions, 2001). Among adults aged 20 to 74, obesity (body mass index [BMI] greater than 30) rates have soared from approximately 15% to 27% over the past two decades. Health problems linked to obesity are numerous and include stroke, heart disease, non-insulin dependent diabetes mellitus, osteoarthritis, and increased risk for developing cancer (Pi-Sunyer, 2002; Raman, 2002). According to a report by the American Medical Association (Allison, Fontaine, Manson, Stevens, & Vanltallie, 1999) every year, more than 280,000 deaths are associated with overeating and obesity. Obesity-related deaths rival the deaths attributed to alcohol and tobacco smoke, including secondhand smoke. Researchers generally agree that obesity is a disease, (James, Gold, & Liu, 2004) but often debate its relationship to depression, personality disorders,

or addictions. Moreover, the similarities among overeating, obesity, and classical addictions have long been demonstrated (Jonas & Gold, 1996; Gold, Johnson, & Stennie, 1997). Similarities between overeating and substance use disorders include compulsive use/behavior despite adverse consequences, craving, denial, preoccupation, increased use/consumption, guilt following excessive use/overeating, and relapse (Gold, Frost-Pineda, & Jacobs, 2003). There also is evidence of a shared neurobiological pathway. Functional brain imaging studies suggest that loss of control over eating and the resulting obesity produce changes in the brain similar to those produced by drugs of abuse (Gautier et al., 2000; James, Gold, & Liu, 2004; James, Guo, & Liu, 2001; Wang et al., 2004). Additionally, newly discovered physiological messengers such as leptin and galanin have been found to have effects in modulating eating behavior (Kalra & Kalra, 2004) and may have roles in obesity, alcoholism and other drug dependencies (Oeser, Goffaux, Snead, & Carlson, 1999; Wei, Stern, & Haffner,1997).

ETIOLOGY OF OBESITY: NATURE OR NURTURE?

Obesity results from an imbalance between energy input and energy expenditure, that is, when more calories are consumed than are needed to maintain homeostasis. Numerous theories attempt to explain the causes of obesity. A popular biologic theory is that obesity develops from abnormal neuroendocrine processes involved in the control of eating behavior and energy homeostasis. For example, the hypothalamus is a principal component of the central nervous system for maintaining energy homeostasis (Kalra & Kalra, 2004; Woods & Seeley, 2002) and changes in the hypothalamic response to anorexigenic or orexigenic signals, signals that suppress or stimulate appetite, respectively, could result in delayed sensation of central satiety. Major neuropeptides involved in regulating appetite and feeding are listed in Table 1. Conversely, a more cognitive approach towards obesity cites the social implications of food as reward (e.g., having to clean one's plate before earning dessert) and focuses upon the behavioral response to food rewards (Shizgal, Fulton, & Woodside, 2001). While these disparate approaches may initially seem irreconcilable, hunger, and satiation regulations most likely stem from interaction of these endocrine and cognitive processes (Saper, Chou, & Elmquist, 2002).

The purpose of this review is to elaborate upon psychobiological processes mediating hunger and further evaluate the link between addiction, overeating and obesity in relation to brain reward. Within this review, we discuss the neuro-anatomic reward circuitry involved with addiction, the implications of this circuitry with regard to drug and alcohol addiction, and how this can translate to an addiction model of overeating and obesity. In addition, the socioeconomic and genetic factors involved with eating disorders

TABLE 1 Neuropeptides That Regulate Food Intake (Sahu & Kalra, 1993)

Stimulate feeding	Inhibit feeding
Decrease energy expenditure	Increase energy expenditure
Anandamide	Calcitonin, Amylin, Bombesin, Somatostatin, Cytokines
β-endorphin	Cholecystokinin
Dynorphin	CRF
GABA	Dopamine
Galanin	Insulin
Ghrelin	Leptin
GHRH	Neurotensin
Neuropeptide Y	Serotonin
Norepinephrine	TRH, MSH, Glucagon, Enterostatin

and obesity and their impact on an addictive model approach of binge eating are be evaluated.

FUNCTIONAL NEUROANATOMY OF ADDICTION AND REWARD BEHAVIOR

Many reviews have examined the nature of the reward circuitry involved with addictions (Baxter & Murray, 2002; Rolls, 2000; Schultz, 2000, 2002; Tzschentke, 2001). According to these reviews, there are two main circuits for reward behavior. The first is the reciprocal connection between the prefrontal regions of the brain and the amygdala. The second is the limbic circuit that integrates the amygdala with the hypothalamus and septal nuclei. Additionally, the Papez limbic circuit integrates the hypothalamus with the hippocampus and the thalamus. The hypothalamus sits at the junction of these limbic circuits. Due to the tight connections and the structures that are integrated, the limbic system circuits are mainly focused on regulating the basic needs of life: food, sex, and water (Augustine, 1996; Denton et al., 1999). The fronto-amygdalar circuit, however, may be more concerned about rewards such as money or more abstract goals.

Nowhere is the synthesis of biologic and physiologic reward mechanisms more prominent than in addiction literature. Addictions are particularly salient to the discussion of reward since addicts continue to pursue the focus of their addiction in spite of punishing factors inherent in the drug abuse (i.e., unsanitary environments, negative health effects) and disapproval of family and peers as well as punishing external factors such as arrest and legal implications. Long-term drug abuse results in physiologic changes in the responsiveness of reward circuitry to the focus of addiction (Goldstein & Volkow, 2002).

It should come as no surprise that reward systems are activated in addicts in response to addiction-related cues. A far more interesting question is how drug abuse affects the processing of non-drug rewards. Functional neuroimaging has assessed the limbic and cortical circuitry mediating monetary reward (Elliot, Friston, & Dolan, 2000) and found that different parts of this circuitry were involved with monetary reward. The ventral striatum and the midbrain were responsive to financial rewards and the hippocampi responded to financial consequences. Elliot et al. (2000) discovered that different areas, such as the globus pallidus, thalamus, and subgenual cingulate responded to financial rewards with increasing reward systems whereas other areas were sensitive to financial consequences, such as the caudate, insula, and ventral prefrontal cortex (Elliot, Friston, & Dolan, 2000). In addition, a study on reward processing found that while smokers and nonsmokers had comparable activation of the limbic system and frontal cortex in response to monetary awards, non-monetary rewards only activated these systems in nonsmokers (Martin-Solch et al., 2001). One interpretation is that reward processing becomes fixed to the addiction and is processed only if it can assist the addict in pursuit of the addiction. However, it is possible that this decrease in activation is an inherent condition that predisposes addicts toward obtaining concrete rewards (such as drugs) over abstract ones. Further research may clarify if the observed difference in reward circuit activation results from, or drives individuals toward, substance abuse, including overeating. Another interpretation of this study proposed earlier is that the fronto-amygdalar circuit deals with abstract, goal-oriented rewards whereas the limbic system focuses upon more basic rewards. Clearly, such an attribution is far more complex than previously stated.

THE FUNCTION OF EMOTION IN REWARD STATES

An emotional state connected with an addiction also has been identified with regards to the strength of an addiction. When evaluating Obsessive Compulsive Disorder (OCD) patients, there appears to be an emotional weight that they apply to the stimuli used to evoke their obsessions and compulsions. Disgust was a prominent emotional response to contaminated food stimuli and resulted in less activity in the medial prefrontal cortex than in controls (Shapira et al., 2003; Shapira & Goodman, 2001). One possible conclusion is that OCD patients found contamination-related stimuli more disgusting and less rewarding than did control subjects. A further conclusion is that there is a connection between an emotional state and a stimulus or substance. Additionally, one could conclude that an emotional state could influence decisions made regarding rewarding or punishing stimuli or substances (James, Gold, & Liu, 2001).

Certain investigators have discovered that a hunger state can influence memory for food-related stimuli in fasting patients (Morris & Dolan, 2001). In

this particular study, brain activity differed according to what type of stimulus was used. The right anterior orbitofrontal cortex covaried with recognition of all stimuli regardless of hunger state, whereas the right posterior orbitofrontal cortex varied only with food-related stimuli in a hunger state. Overall the posterior region correlated with basic rewards, in contrast to the anterior region that correlated more with abstract goal-oriented rewards.

THE NEUROTRANSMITTER OF ADDICTION: DOPAMINE

The characterization of overeating and obesity as an addiction is still a subject of debate. However, the role of the neurochemistry of addiction is becoming pivotal in understanding overeating as an addiction. As described in Table 1, there are several neurotransmitter systems involved in feeding behaviors such as serotonin, opioids, GABA, and dopamine. Dopamine has been closely linked to feeding behavior. Rodent studies have shown that the use of dopamine agonists will increase size of meals and length of feeding time, and long-term administration of dopamine will increase body mass and feeding behavior (Clifton, Rusk, & Cooper, 1991; Schwartz et al., 2000).

Dopamine, as stated, plays a role in feeding behavior. The hypothalamus and the nucleus accumbens are two main areas of function. In the nucleus accumbens, dopamine release is associated with the reinforcement aspects of food, and in the hypothalamus, dopamine is associated with the initiation of feeding and the length of feeding (Wang, Volkow, Thanos, & Fowler, 2004). Dopamine also regulates food consumption and operates within the mesolimbic pathways and the hypothalamus. Additionally, hormones such as leptin and insulin help to regulate dopamine production. It is therefore conceivable, knowing that many drugs of abuse lead to a change in dopamine levels in the brain, specifically in the nucleus accumbens, that there is a mechanism for reinforcement that also may encompass food as a drug of abuse.

Examination of dopamine knock-out mice, mice genetically engineered to be dopamine deficient, elucidates that there are two mechanisms within the brain that regulate food intake. Dopamine deficient mice were found to die quickly because of decreased feeding behaviors likely related to the deficiency in dopamine (Wang, Volkow, Thanos, & Fowler, 2004). Mice that were given dopamine in the striatum, but not the nucleus accumbens, were able to restart feeding, whereas mice given dopamine in the nucleus accumbens were able to choose between pleasant and non pleasant foods, but did not have enough motivation to prevent the mice from dying from low caloric intake. In human studies, methylphenidate was given as a dopamine agonist in the striatum, along with food or non food stimuli. Subjects who received placebo instead of methylphenidate did not demonstrate any increase in food desire. However, subjects who received the methylphenidate and the stimuli did demonstrate increased food desire. These findings were linked to the

ventral striatum and not to the dorsal striatum, once again localizing a site for food and reinforcement.

Additionally within the genetic spectrum, the Taq allele has been identified and correlated with lower levels of dopamine D2 receptors in the brain. It is theorized that obese patients and binge eaters who have the Taq allele and low levels of dopamine D2 receptors may binge in order to increase dopamine in the brain and thereby the reinforcement that dopamine brings.

COCAINE ADDICTION AND REWARD SYSTEMS

Cocaine abuse exemplifies the changes in the neural systems that mediate reward. For example, investigators have monitored the neural activity of cocaine abusers exposed to cocaine-related cues and neutral cues (Bonson et al., 2002). Not only did cocaine abusers demonstrate increased activation of the right dorsolateral prefrontal cortex, left lateral orbitofrontal cortex, and left ventrolateral amygdala in response to cocaine-related cues over neutral cues, but the activation of these regions positively correlated to the self-reported degree of cocaine craving experienced by the subjects. An important point is that increased activity was not observed in areas not associated with reward, such as the paracentral cortex, posterior thalamus, and caudate nucleus, so the activation was specific to reward and not a global change in activity due to increased arousal. These observed patterns of reward circuitry activation generalize to other addictions (Due, Huettel, Hal, & Rubin, 2002). Due et al. (2002) discovered that nicotine-deprived smokers demonstrate increased activation of both limbic circuits (the amygdala, hippocampus, ventral tegmental area, and thalamus) in response to smoking-cues over nonsmoking-cues. These two studies stress that the introduction of addictive substances results in activation of the frontal and limbic circuitry of the brain. Even substance-related cues, such as substance-related images, may be all that is needed to activate this circuitry, thus indicating a possible permanent change in this circuitry once it is exposed to a substance.

There have been many other studies that have examined dopamine and its role in drug addiction. Cocaine blocks dopamine release in the brain, and this acts as a reinforcement of drug addiction. Dopamine D2 receptor levels in the striatum are critical for reinforcement of drug abuse, and in certain studies, higher levels of dopamine D2 receptors have been found to be protective against drug abuse in previously learned drug behaviors (Stein et al., 2001; Thanos et al., 2001).

The correlation between eating disorders and addiction is becoming evident in research that has used functional neuroimaging and neurochemistry to examine the striatums of brains in obese subjects and methamphetamine users. One study used neurofunctional imaging to examine the brains of methamphetamine users and obese subjects (Wang, Volkow, Thanos, & Fowler, 2004). The study found that both the methamphetamine users and the

obese subjects had lower levels of striatal dopamine D2 receptors compared with control subjects (Wang, Volkow, Thanos, & Fowler, 2004). These findings could possibly indicate that dopamine and the level of dopamine receptors are critical to the reinforcement of drug behavior.

THE RELATIONSHIP BETWEEN OBESITY AND SUBSTANCE ABUSE

Within the adolescent population obesity and drug abuse are some of the most concerning problems. In this population, both of these disorders are prevalent and often comorbid (Hodgkins, Cahill, Seraphine, Frost-Pineda, & Gold, 2004). Remission from one may lead to the development of the other (Hodgkins et al., 2004).

The use of tobacco, alcohol, and illicit drugs by adolescents has risen for most of the past two decades, with a plateau and then significant declines since 1999 (Johnston, O'Malley, & Bachman, 2000). However, the percentage of adolescents using alcohol and drugs in the U.S. remains high. According to the 1996 annual Monitoring the Future (MTF) Study (Johnston, O'Malley, & Bachman, 1996), about one-third of high school seniors reported being drunk in the past month, while one-fifth of 10th and 12th graders used marijuana in that same time period. In addition to high rates of use, adolescents are abusing substances at younger ages (American Academy of Pediatrics, 2001), often initiating the use of cigarettes and alcohol between the ages of 10 and 13 years and then moving to experimentation with marijuana and club drugs (Johnson, Boles, & Kleber, 2000). Club drugs are the drugs that are most widely used at all night dance parties or "raves," according the National Institute on Drug Abuse (NIDA) and represent the newest formulations of illicit drugs on the market including cocaine, crystal methamphetamines (crystal), amyl nitrites (poppers), Ecstasy, gamma-hydroxybutyrate (GHB), ketamine (Special K), and Viagra (Fernandez et al., 2005).

Additionally, the prevalence of adult and adolescent obesity has increased at alarming rates in the past three decades. In fact, adolescent obesity has been described by the Centers for Disease Control and Prevention (CDC) as an epidemic (Deitz, 2001). This striking increase has been linked to the dramatic rise in Type II Diabetes among young persons (Hodgkins et al., 2004). Today, one out of every five youths in the U.S. is overweight and one in four is at risk of becoming overweight (CDC, 2000). Among adults, half are overweight and almost one-quarter suffer from obesity. This means that there are approximately 97 million overweight adults and about 40 million obese adults (Cunningham & Marcason, 2001) The remarkable increase in obesity among adolescents in the U.S. has resulted in recommendations for more exercise, food restriction, and even bariatric surgery (Yanovski, 2001). Bariatric surgery is the surgical manipulation of the gastrointestinal tract, specifically the stomach, in order to create a smaller receptacle for food, which in turn, is meant to induce earlier satiety. At this time, bariatric surgery

appears to be an effective treatment for long-term adolescent weight loss (Yanovski, 2001), although less invasive interventions are currently being studied. Surgery is now recommended for severely obese teens (Inge et al., 2004). Complications of childhood obesity include psychosocial, psychological, neurological, cardiovascular, endocrine, musculoskeletal, renal, gastrointestinal, and pulmonary problems (Ebbeling, Pawlak, & Ludwig, 2002).

The standard definition of overweight adults is having a BMI (calculated by weight in kilograms/height in meters squared) between the 85th and 95th percentile, with severe obesity in adults being any BMI greater than the 95th percentile. Calculating BMI is simple and it correlates well with clinical measures of comorbid disease such as diabetes and hypertension, which is why it is commonly used in epidemiological studies (Bray, Bouchard, & James, 1997). Although there are some exceptions, a BMI above 25 (overweight) and above 30 (obese), is a useful guide to estimate the degree of excess fat and health risk (Bray, Bouchard, & James, 1997).

The number of adolescents with substance problems and weight issues is growing and the comorbidity of these illnesses creates difficulty with regard to treatment. Many of these adolescents will enter adulthood with substance disorders and obesity still prominent in their lives. The cost of caring for these adolescents and adults with these disorders is staggering. In addition, there appears to be a link between binge eating and substance use, as described by Ross and Ivis (1999), showing that binge eaters are more likely to use all types of substances, specifically cannabis.

In our recent study, (Hodgkins, Cahill, Seraphine, Frost-Pineda, & Gold, 2004), we described the relationship between weight gain and drug abstinence as well as the necessary changes in drug addiction treatment that need to be made to accommodate this relationship. We examined 75 male and female adolescents in a residential treatment facility for abuse of alcohol and illicit drugs from 1999 through 2002 and found that there was substantial weight gain and increase in BMI from the time of admission to the facility through a 60-day stay. The average BMI on admission adolescent smokers was 23.7 and for nonsmokers 22.23. At the 60-day evaluation, the average BMI had risen by 1.58 points. Additionally, subjects who smoked demonstrated a larger weight gain and increase in BMI than their non-smoking counterparts. Adolescents who smoke are at a greater risk of weight gain during abstinence from drugs (Hodgkins et al., 2004). Furthermore, adolescents may be replacing the reinforcement behavior of drug abuse with feeding behavior to compensate the reward systems of the brain.

EATING DISORDERS AND ALCOHOL ABUSE

A recent study by Matthews (2004) examined the relationship between eating disorders and alcohol consumption on college campuses. Her study addressed three different questions. The first examined whether there are

differences between men and women with regard to problem drinking, which incorporates binge drinking, and eating disorders with eating disorders defined as anorexia and bulimia and binge eating. The results showed that men are more likely to present with problem drinking rather than eating disorders and more women present with eating disorders rather than problem drinking. The second question addressed whether there was a relationship between problem drinking and eating disorders in college women, and the results found no such significant relationship. The third question addressed whether there was a relationship between problem drinking and subscales on the Eating Disorders Inventory-2. The results of Matthews' (2004) study demonstrated a correlation between impulsivity on the Eating Disorders Inventory and problem drinking.

When considering the possible implications on the data from the Matthews study (2004), one must also consider the limitations as well. The findings were limited by a number of factors such as the absence of investigation into the sociocultural influences on eating disorders and problem drinking in a college population. Within the study, the author also addressed the fact that screening tools, not diagnostic tools, were used in the study to identify subjects. Positive scores on a screening tool indicate that diagnostic tests need to be performed for a more thorough assessment and that subjects may be merely at risk for a disorder, not actually be diagnosed with the disorder. Additionally, the author noted that the data was skewed by the fact that there were more subjects who scored negatively rather than positively on the Eating Disorder Inventory (EDI-2) and/or the Alcohol Use Disorders Identification Test (AUDIT). Matthews (2004) analyzed the findings on the EDI-2 and the AUDIT and used a multivariate analysis to look for correlations between responses on the EDI-2 subscales and AUDIT-identified problem drinkers and problem drinking behavior. The results showed that the only subscale that correlated with problem drinking was the Impulse Regulation subscale. Matthews (2004) postulated that this EDI-2 subscale and its correlation with the AUDIT data may exemplify the relationship between substance abuse and eating disorders. One could further postulate that there may be a core element of impulsivity in eating disorders that also can be seen in other abuse disorders.

SOCIOCULTURAL VERSUS GENETIC CONTRIBUTIONS TO EATING DISORDERS

A review of the available literature (Becker, Keel, Anderson-Fye, & Thomas, 2004) indicates that further study is required in regard to eating disorders and their place in the addiction model. Can obesity and eating disorders be classified as addictions? Furthermore, should the treatment for eating disorders focus on addictive models of treatment? Becker et al. (2004) discussed

the influence of genetics versus environment on eating disorders (anorexia, bulimia, and binge eating). An amount of data supports the socioeconomic environment, such as social transitions, gender role changes, and westernization of cultures, as a trenchant influence on eating disorders. Through observational and experimental data, Becker et al. (2004) demonstrated that there are three main areas that appear to factor into the sociocultural contribution to the etiology of eating disorders. First, many developing cultures that are going through social transition or modernization, either by immigration of western peoples or cultures, are at increased risk for development of eating disorders in their community and culture. Second, with a cultural change, any change in the value of thinness or obesity can be a significant factor. Specifically, in the Fiji islands, prior to Westernization, Becker found that Fijian values encouraged vigorous appetites and discouraged weight loss (Becker, Burwell, Gilman, Herzog, & Hamburg, 2002). Following significant cultural change in Fiji in the form of Westernization and Western media influence, thinness and a slim shape were perceived as successful and desired. Third, Becker et al. (2004) discusses the change in gender roles that is seen when a culture becomes more westernized. With any transition in gender roles there is an increased risk in eating disorders. Becker et al. (2002) postulated that as women gain power in economic and social arenas, they are pressured with a higher standard of beauty. Media influence in the form of television shows, ads, and magazine material is thought to have a large effect on the development of eating disorders in a culture. Finally peer influence in the form of teasing was found to be significant when shaping a risk for eating disorders. A peer group that emphasizes thinness and a high standard of beauty contributes to a higher risk of eating disorders (Becker et al., 2002).

Mono- and dizygotic twin studies as well as molecular genetics and allele identification support a genetic contribution to the risk of eating disorders (Becker et al. (2004). Biologic relatives of subjects with eating disorders carry a higher risk of developing eating disorders. Becker et al. (2004) found this consistent with genetic clustering of disorders within a family. A diminishing risk of eating disorders is seen with second and third degree relatives, in some way as the opposite of genetic anticipation, in which genetic diseases arise earlier with each successive generation. Twin studies support a greater risk for eating disorders in monozygotic twins, though there is some speculation that there is an increased risk when development of an autonomous self is limited, as in a twin relationship (Fichter & Noegel, 1990).

Allele transmission with focus on the serotonin 5HT2a receptor also has been evaluated, but it has been found that it does not follow classic Mendelian inheritance, such as dominant or recessive allele inheritance and encompasses the restricting type of anorexia, not the binge purge type (Nacimas et al., 1999; Ricca et al., 2002; Sorbi et al., 1998). Therefore, Becker et al. (2004) concluded that it is more likely that the genetics of eating disorders is more multifactorial in nature and involves complex inheritance. This is

in contrast to strict Mendelian genetics that subscribes to the idea of strict autosomal or recessive allelic inheritance. Additionally, factors that contribute to the development of eating disorders probably encompass sociocultural and environmental factors as well as complex genetic inheritance.

In conclusion, eating disorders appear to share many characteristics with other substance use disorders. In this paper we reviewed research on reward circuits in the brain as well as neurochemistry, emotional relationships to food and hunger, and impulsivity. Further research may define overeating as an addiction. Anticipation of food, drugs, or sex activates the reward pathways of the brain. The idea that eating disorders are addictive in nature becomes salient when one considers the implications of this type of anticipation and the reward centers that are activated with eating and satiety. Whether obesity is a product of environmental cues or a genetically hard wired process cannot be easily answered. It is likely that the development of obesity results from a combination of both environmental and genetic susceptibility. However, classifying eating disorders and obesity as an addiction may prove most useful for the management and treatment of eating disorders in the future. Addiction medicine has a unique way of managing illness and this method may be most beneficial for obesity and eating disorders.

ADDICTION MODELS AS TREATMENT FOR EATING DISORDERS

As discussed above, eating disorders, and specifically binge eating disorders that lead to overweight and obesity, share striking similarities with substance disorders. These similarities are neurochemical, genetic, environmental, and behavioral in nature. In both substance disorders and eating disorders, loss of control and impulsivity are prominent. Emotional and environmental cues are common to both. Additionally, there is evidence of biologic vulnerability with both substance and eating disorders. (Gold, Frost-Pineda, & Jacobs, 2003). Due to the rising cost of obesity in the United States, a successful treatment method is needed to address binge eating, overweight, and obesity. Currently, there does not appear to be one direct area that would suffice for treatment of eating disorders, as the disorders themselves are multifactorial encompassing genetics, environment, neurocircuitry, and behavior. In our discussion we will target the therapies that may provide the most benefit in attempting to address behavior and behavior modification as it is applied to addiction medicine.

Binge eating and other eating disorders are well known to be difficult to treat as many patients with eating disorders such as anorexia or bulimia are ambivalent about treatment and are forced into treatment by concerned family and friends (Vitousek, Watson, & Wilson, 1998). When considering an addiction model approach to treatment, one must examine the work by Prochaska and DiClemente (1992). These researchers developed

the trans-theoretical model of change as a way to examine and understand how people change addiction or substance behaviors. This model involves five stages through which a person will pass on the way to eliminating a behavior. In the first stage, precontemplation, the person does not recognize the behavior as a problem. The contemplation stage is the next stage and in this stage the person can recognize the behavior but maintains ambivalence about changing. In the preparation stage, the person wants to change the behavior but is unsure of how to go about change. The action stage is the stage in which actual change takes place. The maintenance stage focuses on maintaining the new behaviors and avoiding regression into the old behaviors.

Ward, Troop, Todd, and Treasure (1996) examined the trans-theoretical model approach to eating disorders. They found that the majority of the patients were in the contemplation stage, or the stage of ambivalence, while very few patients were in the precontemplation stage. Overall, Ward, Troop, Todd, and Treasure (1996) felt that the model offered a good approach to helping patients think about changing their behavior and estimating their current need for change. Another treatment avenue that employs the addictions model is the motivational interview. Miller and Rollnick (1991) developed this method to be applied to the field of addictions. The motivational interview empowers patients to change their behavior by presenting the discrepancies between their current behaviors and their larger life goals. This technique forces patients to identify reasons for change on their own and can be a powerful motivator for those who are ambivalent.

There is one study that has examined the combination of the trans-theoretical model and the motivational interview, also known as motivational enhancement therapy (MET). Treasure, Katzman, Schmidt, Troop, Todd, and de Silva (1999) found that when MET is compared with cognitive behavioral therapy in treatment of eating disorders, specifically bulimia nervosa, MET showed significant effect in the first phase of treatment. A recent study by Feld, Woodside, Kaplan, Olmsted, and Carter (2001) evaluated the use of MET in patients with anorexia nervosa, bulimia nervosa, or eating disorder not otherwise specified. This particular study examined patients on the other end of the eating disorder spectrum, limiting study patients to those with a BMI of 27 or below. Though their population did not specifically examine those with overweight or obesity, the population they studied with regard to the bulimia nervosa patients have a degree of impulsivity that can contribute to their disorder. Feld et al. (2001) found that the use of MET in treatment increased the number of patients who viewed their disorder as a problem, moving patients away from the precontemplation stage and into the contemplation stage. Additionally, with their use of the Beck Depression Inventory, Feld et al. (2001) discovered that patients were noted to have lower depressive symptoms and an increase in self-esteem. Finally, the investigators found that the majority of subjects entered into a treatment program following the MET.

Other models aimed at eating disorder treatment include 12-Step groups such as Overeaters Anonymous. Trotzky (2002) examined the use of the 12-Step Anonymous Fellowships within the Israel Counseling and Treatment Center of the North, to examine the treatment of eating disorders through an addiction model. Bulimia nervosa and overeater/binge eaters were treated with a 12-Step approach. The results of this study showed success in the binge eating population with weight loss in 62% of the subjects. The bulimia subjects had a lower success rate, measured as abstinence from purging behaviors for sixth months, of 33%. Others have examined the success of Overeaters Anonymous, which is aimed at treating the pathologic behaviors of overeating and binge eating. Overeaters Anonymous, or OA as it is commonly known, attempts to integrate spiritual and emotional components of a person's overeating or impulse eating (Weiner, 1998). OA's approach to overeating is one that identifies overeating with a method that patients use to control their fluctuating moods and affects. According to Weiner (1998), OA then attempts to provide and encourage, through a self-help group model, more acceptable and less harmful ways of controlling affect and emotions than through food addiction or overeating.

As discussed above, there are other avenues to pursue with regard to treatment of eating disorders. These avenues include influencing the neuro-circuitry or working in the area of gene therapy. At this time, these avenues are still being investigated. The arena of eating disorders and binge eating or overeating is still comprised of multiple etiologies and modification of behavior appears at this time to be the most accessible and beneficial.

REFERENCES

Allison, D. B., Fontaine, K. R., Manson, J. E., Stevens, J., & VanItallie, T. B. (1999). Annual deaths attributable to obesity in the United States. *Journal of the American Medical Association, 282*, 1530–1538.

American Academy of Pediatrics. (2001). Committee on Substance Abuse. Alcohol use and abuse: A pediatric concern. *Pediatrics, 108*, 185–189.

Augustine, J. R. (1996). Circuitry and functional aspects of the insular lobe in primates including humans. *Brain Research. Brain Research Reviews, 22*, 229–244.

Baxter, M. G., Murray, E. A. (2002). The amygdala and reward. *Nature Rev Neurosci., 3*, 563–573.

Becker, A. E., Burwell, R. A., Gilman, S. E., Herzog, D. B., & Hamburg, P. (2002). Eating behaviors and attitudes following prolonged exposure to television among ethnic Fijian adolescent girls. *British Journal of Psychiatry, 180*, 509–514.

Becker, A. E., Keel, P., Anderson-Fye, E. P., & Thomas, J. J. (2004). Genes and/or jeans?: Genetic and socio-cultural contributions to risk for eating disorders. *Journal of Addictive Diseases, 23*(3), 81–103.

Bonson, K. R., Grant, S. J., Contoreggi, C. S., Links, J. M., Metcalfe, J., Weyl, H. L., Kurian, V., Ernst M., & London, E. D. (2002). Neural systems and cue-induced cocaine craving. *Neuropsychopharmacology, 26*, 376–386.

Bray, G. A., Bouchard, C., & James, W. P. (Eds.). (1997). *Handbook of obesity.* New York: Marcel Dekker.

Centers for Disease Control. (2000). *Youth risk behavioral surveillance.* Atlanta: Author.

Clifton, P. G., Rusk, I. N., & Cooper, S. J. (1991). Effects of dopamine D1 and dopamine D2 antagonists on the free feeding and drinking patterns of rats. *Behavioral Neuroscience, 105*, 272–281.

Cunningham, E., & Marcason, W. (2001). Obesity update. *J Am Diet Assoc, 101*, 80.

Denton, D., Shade, R., Zamarippa, F., et al. (1999). Neuroimaging of genesis and satiation of thirst and an introceptor-driven theory of origins of primary consciousness. *Proceeding of the National Academy of Sciences in the United States of America, 96*, 5304–5309.

Dietz, W. (2001). Childhood obesity. In M. Shils, J. Olsen, M. Shike, et al. (Eds.), *Modern nutrition in health and disease* (9th ed., pp. 1071–1080.) Baltimore: Williams and Wilkins.

Due, D. L., Huettel, S. A., Hal, W. G., & Rubin, D. C. (2002). Activation in mesolimbic and visuospatial neural circuits elicited by smoking cues: Evidence from functional magnetic resonance imaging. *American Journal of Psychiatry, 159*, 954–960.

Ebbeling, C. B., Pawlak, D. B., & Ludwig, D. S. (2002). Childhood obesity: Public-health crisis, common sense cure. *Lancet, 360*, 473–482.

Elliot, R., Friston, K. J., & Dolan, R. J. (2000). Dissociable neural responses in human reward systems. *J Neurosci., 20*, 6159–6165.

Feld, R., Woodside, D. B., Kaplan, A. S., Olmsted, M. P., & Carter, J. C. (2001). Pretreatment motivational enhancement therapy for eating disorders: A pilot study. *International Journal of Eating Disorders, 29*, 393–400.

Fernandez, M. I., Perrino, T., Collazo, J. B., Varga. L. M., Marsh, D., Hernandez, N., Rehbein, A., & Bowen, G. S. (2005). Surfing new territory: Club-drug use and risky sex among Hispanic men who have sex with men recruited on the Internet. *Journal of Urban Health, 82*(1 Suppl 1), 79–88.

Fichter, M. M., & Noegel, R. (1990). Concordance for bulimia nervosa in twins. *International Journal of Eating Disorders, 9*, 255–63.

Gautier, J. F., Chen, K., Salbe, A. D., et al. (2000). Differential brain responses to satiation in obese and lean men. *Diabetes, 49*, 838–846.

Gold, M. S., Frost-Pineda, K., & Jacobs, W. S. (2003). Overeating, binge eating, and eating disorders as addictions. *Psychiatric Annal., 33*(2), 117–122.

Gold, M. S., Johnson. C. R., & Stennie, K. (1997). Eating disorders. In J. H. Lowinson, P. Ruiz, R. B. Millman, & J. G. Langrod (Eds.), Substance abuse: A comprehensive textbook (3rd ed., pp. 319–330). Philadelphia: Lippincott, Williams and Wilkins.

Goldstein, R. Z., & Volkow, N. D. (2002). Drug addiction and its underlying neurobiological basis: Neuroimaging evidence for the involvement of the frontal cortex. *American Journal of Psychiatry, 129*, 1642–1652.

Hodgkins, C. C., Cahill, K. S., Seraphine, A. E., Frost-Pineda, K., & Gold, M. S. (2004). Adolescent drug addiction treatment and weight gain. *J Add Dis., 23*(3), 55–66.

Inge, T. H., Krebs, N. F., Garcia, V. F., et al. (2004). Bariatric surgery for severely overweight adolescents: Concerns and recommendations. *Pediatrics, 114*, 217–223.

James, G. A., Gold, M. S., & Liu, Y. (2004). Interaction of satiety and reward response to food stimulation. *Journal of Addictive Diseases, 23*(3), 23–37.

James, G. A., Guo, W., & Liu, Y. (2001). Imaging in vivo brain-hormone interaction in the control of eating and obesity. *Diabetes Technology and Therapeutics., 3*, 617–622.

Johnson, P. B., Boles, S. M., & Kleber, H. D. (2000). The relationship between adolescent smoking and drinking and likelihood estimates of illicit drug use. *Journal of Addictive Diseases, 19*, 75–81.

Johnston, L. D., O'Malley, P. M., & Bachman, J. G. (1996). The Monitoring the Future national results on adolescent drug use: Overview of key findings. 1995. Bethesda, MD: U.S. Department of Health and Human Services, National Institutes of Health.

Johnston, L. D., O'Malley, P. M., & Bachman, J. G. (2000). The Monitoring the Future national results on adolescent drug use, 1975–2000. 2001. Bethesda, MD: U.S. Department of Health and Human Services, National Institutes of Health.

Jonas, J. M., & Gold, M. S. (1996). Naltrexone treatment of bulimia: Clinical and theoretical findings linking eating disorders and substance abuse. *Advances in Alcohol and Substance Abuse, 7*, 29–37.

Kalra, S. P., & Kalra, P. S. (2004). Overlapping and interactive pathways regulating appetite and craving. *Journal of Addictive Diseases, 23*(3), 5–21.

Martin-Sölch, C., Magyar, S., Künig, G., et al. (2001). Changes in brain activation associated with reward processing in smokers and nonsmokers: A positron emission tomography study. *Experimental Brain Research, 139*, 278–286.

Matthews, C. R. (2004). Examining problem drinking and eating disorders from a gendered perspective. *Journal of Addictive Diseases, 3*, 67–80.

McKesson Clinical Reference Systems, San Francisco, CA, U.S. (2001). Obesity. Clinical reference systems, annual.

Miller, W. R., & Rollnick, S. (1991). *Motivational interviewing: Preparing people to change addictive behavior*. New York: Guilford.

Morris, J. S. (2001). Involvement of human amygdala and orbitofrontal cortex in hunger-enhanced memory for food stimuli. *J Neurosci., 21*, 5304–5310.

Oeser, A., Goffaux, J., Snead, W., & Carlson, M. G. (1999). Plasma leptin concentrations and lipid profiles during nicotine abstinence. *American Journal of the Medical Sciences, 318*, 152–157.

Pi-Sunyer, F. X. (2002). The medical risks of obesity. *Obesity Surg., 12*(Supp 1), 6S–11S.

Prochaska, J. O., & DiClemente, C. C. (1992). The trans-theoretical approach. In J. C. Norcross, & I. L. Goldfield (Eds.), *Handbook of psychotherapy integration* (pp. 330–334). New York: Basic.

Raman, R. P. (2002). Obesity and health risks. *Journal of the American College of Nutrition, 21*, 34S–139S.

Ricca, V., Nacimas, B., Cellini, E., et al. (2002). 5HT2A receptor gene polymorphism and eating disorders. *Neuroscience Letters, 323*, 105–108.

Rolls, E. T. (2000). The orbitofrontal cortex and reward. *Cerebral Cortex., 10*, 284–294.

Ross, H. E., & Ivis, F. (1999). Binge eating and substance use among male and female adolescents. *International Journal of Eating Disorders, 26*, 245–260.

Sahu, A., & Kalra, S. P. (1993). Neuropeptide regulation of feeding behavior: *Neuropeptide Y. TEM, 4*(7), 217–224.

Saper, C. B., Chou, T. C., & Elmquist, J. K. (2002). The need to feed: Homeostatic and hedonic control of eating. *Neuron, 36*, 199–211.

Schultz, W. (2000). Multiple reward signals in the brain. *Nature Review of Neuroscience, 1*(3), 199–207.

Schultz, W. (2002). Getting formal with dopamine and reward. *Neuron, 36*, 241–263.

Schwartz, M. W., Woods, S. C., Porte, D. Jr., et al. (2000). Central nervous system control of food intake. *Nature, 404*, 661–671.

Shapira, N., & Goodman, W. K. (2001). The psychobiology of obsessive-compulsive disorder: How important is the role of disgust? *Curr Psychiatry Rep., 3*, 281–287.

Shapira, N. A., Liu, Y., He, A. G., et al. (2003). Brain activation by disgust-inducing pictures in obsessive-compulsive disorder. *Biol Psychiatry, 1:54*(7), 751–756.

Shizgal, P., Fulton, S., & Woodside, B. (2001). Brain reward circuitry and the regulation of energy balance. *International Journal of Obesity and Related Metabolic Disorders, 25*(Supple 5), S17–S21.

Sorbi, S., Nacimas, B., Tedde, A., et al. (1998). 5-HT2A promoter polymorphism in anorexia nervosa. *Lancet., 351*:1785.

Stein, D. J., Liu, Y., Thanos, P. K., Taintor, N., Hitzemann, R., et al. (2001). The effect of ethanol drinking preference of D2 upregulation in the nucleus accumbens of the alcohol preferring (P) and non preferring (NP) rats. *Alcohol Clin Exp Res., 25*(Suppl: 56A).

Thanos, P. K., Taintor, N. B., Rivera, S. N., Umegaki, H., Ikari, H., Roth, G., Ingram, D. K., Hitzemann, R., Fowler, J. S., Gatley, S. J., Wang, G. J., & Volkow, N. D. (2004). DRD2 gene transfer into the nucleus accumbens core of the alcohol preferring and nonpreferring rats attenuates alcohol drinking. *Alcohol Clin. Exp. Res., 28*(5), 720–728.

Treasure, J. L., Katzman, M., Schmidt, U., Troop, N., Todd, G., & de Silva, P. (1999). Engagement and outcome in the treatment of bulimia nervosa: First phase of a sequential design comparing motivation enhancement therapy and cognitive behavioural therapy. *Behavioural Research and Therapy, 37*, 405–418.

Trotzky, A. S. (2002). The treatment of eating disorders as addictions among adolescent females. *International Journal of Adolescent Medicine and Health, 14*(4), 269–274.

Tzschentke, T. M. (2001). Pharmacology and behavioral pharmacology of the mesocortical dopamine system. *Progress in Neurobiology, 63*, 241–320.

Vitousek, K., Watson, S., & Wilson, G. T. (1998). Enhancing motivation for change in treatment-resistant eating disorders. *Clinical Psychology Review, 18*(4), 391–420.

Wang, G., Volkow. N. D., Thanos, P. K., et al. (2004). Similarity between obesity and drug addiction as assessed by neurofunctional imaging: A concept review. *Journal of Addictive Diseases, 23*, 39–53.

Ward, A., Troop, N., Todd, G., & Treasure, J. (1996). To change or not to change—"how" is the question? *Br J Med Psychol., 69*(Pt 2), 139–146.

Wei, M., Stern, M. P., & Haffner, S. M. (1997). Serum leptin levels in Mexican Americans and non-Hispanic whites: Association with body mass index and cigarette smoking. *Annals of Epidemiology, 7*, 81–86.

Weiner, S. (1998). The addiction of overeating: Self-help groups as treatment models. *Journal of Clinical Psychology, 54*(2), 163–167.

Woods, S. C., & Seeley, R. J. (2002). Understanding the physiology of obesity: Review of recent developments in obesity research. *International Journal of Obesity and Related Metabolic Disorders, 26* (Suppl 4), S8–S10.

Yanovski, J. A. (2001). Intensive therapies for pediatric obesity. *Pediatric Clinics of North America, 48*, 1041–1053.