Journal of Interdisciplinary Undergraduate Research

Volume 1 Article 2

2009

Of Mice and Men: A Comparative Study Assessing Behavioral Indicators of Sugar Addiction in Mice and College Students

Kahlilia Morris

Of Mice and Men: A Comparative Study Assessing Behavioral Indicators of Sugar Addiction in Mice and College Students

Kahlilia Morris

Abstract: Binge eating is a maladaptive behavior associated with obesity and certain eating disorders. Numerous animal studies have shown that this bingeing behavior shares qualities with those of drug addiction. Furthermore, research in rodents has shown that this addictive behavior is also characterized by the same molecular and physiological characteristics that define drug addiction. The current study purposed to assess whether bingeing on sugar could lead to behavioral indicators of sugar dependence in mice as well as humans. The results show that bingeing on sugar did not increase addictive behavior in either mice or human participants. Instead, addictive behaviors appeared to be driven by increased sugar consumption.

Obesity and certain eating disorders are often characterized by an individual's inability to control food intake. Research suggests that the behavioral component of binge eating shares many qualities with behaviors expressed during drug addiction (Riva et al., 2006). This is because food is a strong reinforcer that has the ability to highly motivate certain behavior (Epstein, Leddy, Temple, & Faith, 2007). Many scientists believe that addiction may pose a valid explanation for this maladaptive behavior because feeding development originates from the same neural pathways that are activated by addictive drugs (Kelley, et al., 2002). Avena (2007) states that the characteristics that define binge eating, such as excessive intake, aversive state, and lack of control exhibit a close resemblance to the stages of drug dependence. Therefore, it is possible that binge eating may be due to food dependence caused by the addictive nature of certain food substances.

Binge eating is usually characterized by the consumption of high calorie foods, rich in sweets that have little nutritional value (Avena, 2007). Therefore, analogous to drugs of abuse, the ingestion of sugary food substances is not motivated by a need to maintain homeostatic balance (Epstein et al., 2007). Instead, according to Avena, et al. (2008), consumption may be driven by the brain's opioid system and the release of dopamine. This suggests that sugar may be the cause of the addictive behavior displayed by binge eaters.

The neurotransmitter dopamine is believed to play an important role in the dependence of individuals to addictive drugs such as cocaine and heroin (Rothman, Baumann, Prisinzano, & Newman, 2007) and may also be a cause of sugar addiction in humans. Both sugar and drugs of abuse cause repeated release or reduced reuptake of extracellular dopamine in an area of the brain involved in reward and reinforcement (Avena, 2007; Bassero & Di Chiara, 1997). Although dopamine is released anytime an animal is exposed to novel foods, this effect diminishes with repeated exposure for satiated animals (Bassero & Di Chiara, 1997). However, this waning dopaminergic response is not observed in animals displaying sugar bingeing behavior (Avena et al., 2008).

Numerous research studies have used animal models to analyze the relationships between sugar-bingeing and drug dependence characteristics such as dopamine release, opiate-like withdrawal, and certain behavioral changes. Avena et al. (2008) found that rats with intermittent access to sugar enter a state that is similar to drug dependence on both behavioral and neurochemical levels. This is because rats with intermittent access binge on sugar when it becomes available. In addition, they also display aggression and signs of withdrawal, such as anxiety and depression (Colantuoni et al., 2002) and also exhibit altered dopamine release activity (Avena, 2007).

According to Rolls (2003) individual differences exist in the way in which the brain's dopamine response system responds to excessive food intake. In sntdies with lab rats, more dopamine is released by obese rats than lean rats during eating (Yang & Meguid, 1995). Similarly, research studies with humans have indicated differences in neuronal activity between lean and obese individuals in response to food intake and satiation (Karhunen, Lappalainen, Vanninen, Kuikka, & Uusitupa, 1997; Gautier et al., 2000). Therefore, it is possible that obese humans may also have an altered dopamine metabolism (Epstein et al., 2007) causing certain individuals to have a higher susceptibility to binge eating than others. Still, whether bingeing leads to addictive behaviors or changes in dopamine levels in humans has not been investigated.

The review of the literature indicates that certain maladaptive behaviors related to sugar intake in laboratory animals portray characteristics that are similar to those of drug abuse. On the cellular and molecular level sugar is able to affect dopamine release and other opiates. Physiologically, certain patterns of sugar intake can affect neuronal activity in a way that parallels addictive substances. Furthermore, behavioral changes comparable to those observed during drug addiction are also observed due to sugar bingeing. Researchers posit that the sugar dependencies observed in animals may provide a plausible explanation for certain maladaptive eating behaviors in humans.

The purpose of this comparative study was to analyze selected characteristics of sugar dependence in both humans and laboratory mice. It was hypothesized that bingeing on sugar would cause or exacerbate certain behavioral indicators of addiction in both mice and humans. In addition, other factors such as family history and caffeine intake were assessed in the human analysis of sugar bingeing.

Method

Participants

The participants in this study were 38 undergraduate students in General Psychology at Southern Adventist University. They received 10 extra credit points for participating in this experiment. All participants were treated in accordance with the Ethical Principles of Psychologists and Code of Conduct (American Psychological Association, 2002).

Subjects

Ten laboratory mice (Mus musculus) attained from a local pet store were used as subjects in this study. Results are reported on 8 mice due to mortality. These animal subjects were treated in accordance with federal guidelines for the ethical care of animal subjects.

Materials

The Phelps-Nourse Individual Addictiveness Profile is a self-administered test that analyzes risk factors for addiction based on an understanding of the biochemical properties of addiction. This test was created by Dr. Janice Keller Phelps, who has been an addiction specialist since 1977 (Marohn, 2004). Parts I and II of this instrument were utilized in this study. Part I assesses both behavioral and physiological indicators of addiction in terms of diet and was used as both a pre-survey and post-survey. This section is made up of 10 questions that assess how often certain behaviors or physiological symptoms occur (i.e. once/week, twice /week, once/day, etc). Each answer is given a score ranging from zero to five. Part I will be slightly modified for the pre-assessment: Four general questions regarding regular caffeine use, meals eaten per day, and sugar consumption will be added to this section. Part I will remain unmodified for the postassessment.

Part II assesses family history risk factors of addiction and will be administered as part of the pre-survey. This section of the test will be scored by giving a numerical value between one and five to each item based on the number of reported family members reported with the given condition (i.e. none, one or two, most, or a specific number). This part of test will be slightly modified in that each participant will be asked to report the number of relatives with each condition for each item.

A four-week food log was used in this experiment where participants recorded everything that they ate and drank during the duration of the study. The time taken to consume the experimental bags of candy for a 10 day period was also recorded. In addition, this log was also used to assess the number of caffeinated beverages consumed each day.

The Spielberger State Anxiety Inventory is a 20-item questionnaire that was utilized in this study. This survey assesses a person's present anxiety based on a four level scale: "not at all", "somewhat", "moderately so", or "very much so". According to Barnes, Harp, and Jung (2002), this assessment is valid for measuring anxiety and has both internal consistency and test-retest reliability (See Appendix for copies of instruments and scoring key).

Bags of candy composed of Skittles and Brach's Gummy Bears was used. Each bag contained 14 gummy bears and 14 skittles comprising at total of approximately 30g of sugar per bag.

An elevated plus maze was used to measure anxiety as a pre- and post-measure in laboratory mice.

A narrow vertical glass container, approximately 25cm high and 14 em in diameter, filled with water (21-23 C) about 17 em deep was utilized to perform a forced swim test as a pre- and post-measure with laboratory mice.

A 10% sucrose solution and lab chow will be used as food for the laboratory mice. Four cages and two mouse wheels.

Design and Procedure

Phase One: Human Participants. This study was an independent groups 1-factor experimental design. The duration of the experiment with the human participants was 27 days. Informed consent forms were given to the students at the beginning of the experiment. Then, the participants were given a packet containing the modified Part I and unmodified Part II of the Phelps-Nourse Addictiveness Profile and food logs for seven days.

After seven days the packets were retrieved from the participants. Each participant was be randomly assigned into either Group A or Group B using a table of random numbers. Participants in Group A were the 'bingers' and were asked to consume one bag of candy each day in three minutes or less. Participants in Group B were the 'nonbingers' and were asked to consume one bag of candy each day at their leisure. Each participant was given a total of 10 bags of candy as well as food logs for 12 days.

All participants were asked to abstain from sugar for approximately two days (days 18, 19, and part of day 20).

On day 20, the 12-day food logs were picked up and the SSAI will be administered to the participants. After this point, participants were told that they may discontinue sugar abstinence. A food long for seven days was given to each participant.

On day 27, the previous 7 -day food log was retrieved. The post Part I of the PhelpsNourse Addictiveness Profile was given and retrieved.

Phase Two : Rodents Subjects. This was a matched groups 1-factor experimental design. The mice were matched on coat color. Each condition contained two black mice, one white mouse, one gray mouse, and one brown mouse. The duration of the experiment with mice was three weeks. The pre-anxiety assessment using an elevated plus maze was administered to each mouse. Then a pre-forced swim test was administered to each mouse.

The mice were kept in two separate cages: one cage for control mice and one cage for experimental mice. All the mice were fed 10% sucrose solution and lab chow. The mice in the experimental condition were taken out of their cage for 12 hours, every 12 hours for 14 days. These five mice were kept in a separate cage with no access to food or sucrose solution. The control mice were also taken out of their cage for 12 hours, every 12 hours for seven days. However, the control mice had constant access to the sucrose solution and lab chow. The amount of sucrose solution consumed was recorded for both the control and experimental mice.

After the 14- day period, none of the mice had access to the sucrose solution for two days. Instead, they only had access to water and lab chow. After this, the post-anxiety measure on the elevated plus maze as well as the post-depression forced swim test was administered.

Definition of Terms

Humans. Sugar bingeing in humans is operationally defined as eating 30g of sugar in three minutes or less. Caffeine use was measured by the number of reported caffeinated drinks consumed in a 7 day period. Removing sugar from the human diet refers to abstinence from sugary products for approximately 2.5 days. 'Sugary products' in this study refer to sweet substances, high in sugar content. Family history of substance dependence was assessed by the score received by each participant on the family history section of the Phelps-Nourse assessment. Behavioral indicators of substance abuse in human participants were evaluated with the Phelps-Nourse assessment. Anxiety in human participants was measured using the Spielberger Inventory for State Anxiety.

Mice. Intermittent sugar access is operationally defined as 12-hour access to an aqueous 10% sucrose solution and lab chow, followed by 12-hour deprivation daily for two weeks. Removing sugar from the mouse diet refers to abstinence from the sucrose solution for 2 days. Anxiety in laboratory mice was measured by the amount of time spent on the exposed arm of an elevated plus-maze (Colantuoni et al., 2002). Depression in laboratory mice was assessed by analyzing passive or active escape efforts when the mice are placed in water (Avena et al., 2008).

Data Analysis

Data were coded and entered into SPSS for analysis. Independent samples t-test, paired sample t-tests, Pearson's correlation coefficient, and point-biserial correlation coefficients were used to test the hypotheses and research questions.

Results

Human Participants

Increase in sugar consumption. The first hypothesis of this study was that sugar bingers would increase the amount of sugary products in their diet. Students were given 1-week food logs prior to the experimental intervention (the phase that includes the binge/ nonbingeing) and an independent samples *t*-test showed no statistically significant difference between the two groups {t(23)=.76, p=.46}. This means that both groups were relatively similar in the amount of sugary products consumed in their diet before the experiment was administered (Fig. l). After the experimental period, sugar bingers obtained a mean of 1.90 (SD=.86) sugar products (post sugar consumption) in their diet per day. Nonbingers had an average of 1.56 (SD=l.O5) sugar products in their diet per day. Although on average nonbingers consumed less sugary products, an independent samples t-test shows that the mean difference was not statistically significant (Table 1, Fig. 1). Therefore, the null hypothesis that sugar bingeing has no effect on the amount of sugary products consumed could not be rejected {t(23)=.898, p=.379}. Interestingly, sugar consumption overall increased for both groups after the experimental treatment.

Behavioral Indicators of Substance Dependence. The second hypothesis of this study was that sugar bingers will have increased behavioral indicators of substance dependence.

Before the experimental intervention, the binge group had a mean score of 5.42 (SD-3.32) and the nonbinge group obtained a mean of 2.58 (SD= 2.31). An independent samples t-test showed that the mean difference (2.83) was statistically significant {t(22)=2.43, p=.025}. These results therefore show that these groups were not equivalent on this aspect of the experiment before experimental treatment. After the experimental period, bingers had an average score of 5.00 (SD=5.4l) on the post behavioral indicators measure and nonbingers obtained a mean score of 2. 7 5 (SD= 2 .60). The mean difference (2.25) was shown not to be statistically significant with an independent samples t-test { t(22)= 1.29, p-.21} (Table 1, Fig.2). Therefore the null hypothesis that sugar bingers would not have increased behavioral indicators of substance dependence could not be rejected.

State Anxiety. The third hypothesis was that removing sugar from the diet of sugar bingers affects levels of state anxiety. Bingers obtained a mean score of 37.38 (SD= 12. 72) and nonbingers had an average of 39.55 (SD=6.55) on the SSAI. An independent samples t-test showed that this difference was not statistically significant {t(22)- -.54, p=.60} (Table 1). The null hypothesis that removing sugar from the diet of sugar bingers does not affect anxiety levels could not be rejected.

Sugar Consumption and Caffeine Use. The fourth hypothesis was that there is a positive correlation between sugar consumption and caffeine use. During the experimental intervention (the binge/ nonbinge period), a Pearson correlation coefficient of r(31)=.253 was obtained between sugar consumption and the amount of caffeine consumed. However, this positive relationship was not statistically significant (p-.17). Post sugar consumption also showed no statistically significant relationship between post caffeine consumptions {r(30)=.19, p=.3l}. There was also no significant correlation between the sugar consumed during the experimental period {r(30)=.308, p=.lO} and post caffeine intake. Therefore the null hypothesis that there is no relationship between sugar consumption and caffeine use could not be rejected.

Gender Relationships. Does gender influence the relationship between certain behavioral indicators? Point biserial correlations analyzed correlation coefficients showed relationships between gender and a number of variables. Note that gender was coded as male= 1 and female= 2. A statistically significant positive correlation exists between gender and reported symptoms after the experimental period (r(27)= .47, p=.0l}. No statistically significant correlation was present between gender and symptoms before the experimental period {r(32)= .29, p=.l06}. Therefore, women were more likely than men to report experiencing symptoms after the treatment period. There was a negative relationship of r(26)= -.41 between gender and the amount of caffeine reported after the experimental intervention. This correlation between caffeine consumption was statistically significant (p=.04). Therefore, men were more likely to report the consumption of caffeine.

Family History. Does family history of substance dependence affect behavioral indicators of sugar dependence? Bingers had a higher mean score (M = 12.00, SD=11.38) of family substance abuse than nonbingers (M=8.77, SD=14.69). However, an independent samples t·test shows that this difference is not statistically significant {t(23)=.61, p-

.55} (Table 1). Therefore, the two groups were relatively similar in their familial backgrounds of substance abuse.

Animal Subjects

Sugar Consumption. The fifth hypothesis of this study was that intermittent access to sugar will cause an increase in the amount of sugar solution consumed by laboratory mice. Figure 3 shows a comparison of the average amounts of sugar consumed per mouse between the binge group and the nonbinge group during 12 hour periods. The binge group had a mean of 6.01 (SD=0.65) and the nonbinge group averaged 6.22 (SD=2.12). An independent samples t-test showed the difference of .212 between the group means was not statistically significant (t(22) =.330, p-.75).

Anxiety. The sixth hypothesis was that removal of access to sugar will cause an increase in mouse anxiety. Nonbingers groups had a mean pre-anxiety score of 162.33 (SD= 43.84) and bingers scored an average pre-anxiety score of 50.60 (SD- 33.76). Note that lower scores signify higher anxiety. An independent samples t-test showed that the mean difference of 117.33 is statistically significant (t(6)=4.09, p=.006). Therefore, nonbingers had higher anxiety before sugar consumption began. No statistically significant difference was found between bingers and nonbingers on anxiety after the experimental measure (t(6)=1.76, p=.129). Figure 4 shows scores obtained on the elevated plus maze after experimental treatment are generally lower than scores obtained before treatment for each mouse. Therefore, anxiety generally increased for almost all of the mice. A paired samples t-test shows that the difference is not statistically significant {t(7) = 1.927, p=.09). However, note that the p value is approaching statistical significance. An independent samples t·test showed that there were no significant differences between binge and nonbinge groups on post-anxiety scores (t(6)=1.76, p-.129}.

Depression. The final hypothesis is of this study was that removal of sugar access will cause an increase in mouse depression. Figure 5 shows post the forced swim test (FSTI scores increased for each mouse except for one. Note that higher scores signify higher depression. A paired samples t-test shows that the difference is statistically significant {t(7)=-2.312, p=.05]. An independent samples t·test showed that there were no significant differences between the post FST between binge and nonbinge groups {t(6)=5. 79, p=. 76}. Therefore, mice in both groups had increased anxiety after sugar withdrawal.

Other Interesting Findings

As part of the Phelps-Nourse Individual Addictiveness Profile (PNIAP) participants were asked to report symptoms of dependence as both a pre and post experimental assessment. On the post version of the PNIAP females had an average score of 8.33(SD=4.01) post symptoms of dependence whiles males had a mean number of 4.22 (SD= 3.38) post symptoms of dependence. An independent samples t·test showed that the mean difference of -.41 is statistically significant {t(25)= -2.63, p=.014}.

In the PNIAP, for both the pre and post measure, participants responded to how many symptoms of dependence were relieved by eating sugary foods. Table 2 shows that statistically significant correlations exist between the post symptoms relieved by eating sugary foods and the post report of sugar consumption ($r(31)=.515$, $p= .003$). There was also a positive correlation between these post symptoms relieved by eating sugary foods and their pre report of sugar consumption {$r(31)=.522$, $p=.003$}. Therefore, the more sugar that was consumed (pre sugar consumption or post sugar consumption), the more post symptoms were relieved by consuming sugar.

Pearson correlation coefficients showed that there are positive relationships between the post symptoms of dependence and sugar consumption per day. The correlations for pre sugar consumption per day ($r(30)=.432$, $p=.017$}, post sugar consumption per day {$r(29)=.390$, $p=.042$} and sugar consumption per day during the experimental period {$r(29)=.389$, $p=.037$} were all statistically significant (Table 2). This means the higher the sugar consumption, the more post symptoms of dependence were relieved by consuming sugar. Also, pre sugar consumption per day, post sugar consumption per day, and sugar consumption during the experimental period were also each positively correlated with each other on a statistically significant level. Therefore, the level of sugar consumption was not affected by the experimental procedure.

Discussion

The purpose of this study was to analyze selected characteristics of sugar dependence in both humans and laboratory mice. It was hypothesized that bingeing on sugar would cause or exacerbate certain behavioral indicators of addiction. However, the results of this study do not support this hypothesis as bingeing on sugar did not appear to cause any significant changes. Despite these findings, both the human and mice experiments showed that simply consuming large amounts of sugar may cause or predict behavioral indicators of sugar addiction.

Sugar Consumption

Bingeing on sugar had no effect on the amount of sugar participants consumed after the experimental intervention. Instead, the greatest indicator of high sugar consumption during the post interval was high sugar consumption before the experimental manipulation. In fact, the results show positive relationships between sugar consumption during all phases of the research study. Furthermore, the results suggest that people who consume large amounts of sugar in their diet may already experience the effects of sugar dependence; behavioral indicators of addiction were positively related to their sugar consumption before and after the manipulated phase. For example, the more sugar consumed, the more symptoms experienced by the participants. Moreover, the more sugar consumed the more these symptoms were relieved by eating sugary products. This may suggest that sugar dependence is present and that behavioral and physiological symptoms are alleviated by consuming high amounts of sugar.

The mice experiments also failed to show any effect of sugar bingeing on behavioral indicators of addiction. The statistically significant difference in depression after sugar withdrawal confirms the idea that simply consuming large amounts of sugar solution has the ability to induce behavioral indicators of addiction. This result is harmonious with the implications of the human experiment. Interestingly, although the mice with constant access to sugar consumed more sugar solution over time, there were no significant differences between the two groups on any of the dependent variables. A comparison of the average amounts of sugar solution consumed between each group during 12 hour periods showed no differences. However, the small sample of mice (n=8) may have contributed to these findings.

Gender Differences

Women were more likely than men to report symptoms after the experimental intervention. Higher reports by women of behavioral and physiological symptoms is a phenomena well supported by research literature. However, this result is not shown before the manipulated experimental phase. Therefore, it is possible that women may somehow be more susceptible to the effects of high sugar consumption than men.

Men were more likely to consume caffeinated beverages after the experimental intervention. Males in this study consumed more sugar after the binge/nonbinge and withdrawal period than before this experimental phase. This may suggest that men are more susceptible to cross-sensitization with psychostimulants than women. Future research should examine gender differences in the cross-sensitization between sugar and other psychoactive substances.

Conclusion

Previous research has shown that sugar bingeing has the ability to affect animals on a neurochemical level. However, no effects of this type of feeding behavior were observed in this study. Although these experiments failed to show any effect of sugar bingeing on behavior, the current research results have the capacity to open future areas of research. This comparative study showed, for both humans and mice, that simply consuming large amounts of sugar may have the ability to cause or intensify behavioral indicators of substance dependence. These results may attest to the importance of healthy eating habits. Furthermore, even though the duration of the mice experiment occurred over a shorter time interval than in previous studies, behavioral indicators of addiction were still present. Future studies should analyze the length of time necessary to observe the effects of bingeing on behavioral indicators of addiction. Also, scientists should examine if certain behavioral indicators decrease for nonbingers over time. Lastly, more studies are needed to assess behavioral indicators of sugar addiction in human populations.

References

American Psychological Association. (2002). Ethical principles of psychologists and code of conduct. American *Psychologist,* 57, 1060-1073.

Avena, N.M. (2007). Examining the addictive-like properties of binge eating using an animal model of sugar dependence. *Experimental and Clinical Psychopharmacology,* 15(5), 481-491. Retrieved January 17, 2008 from Academic Search Premier database.

Avena, N.M., Rada, P., & Hoebel, B. G. (2008). Evidence for sugar addiction: Behavioral and neurochemical effects of intermittent, excessive sugar intake. *Neuroscience* & *Biobehavioral Reviews,* 32(1), 20-39. Retrieved January 17, 2008 from Academic Search Premier database.

Barnes, L., Harp, D., & Jung, W. (2002). Reliability Generalization of Scores on the Spielberger State-Trait Anxiety Inventory. Retrieved March 21, 2008 from the ERIC database.

Bassareo, V. & DiChiara, G. (1997). Differential influence of associative and nonassociative learning mechanisms on the responsiveness of prefrontal and accumbal dopamine transmission to food stimuli in rats fed ad libitum. *Journal of Neuroscience,* 17, 851-861. Retrieved January 17, 2008 from the Online Journal of Neuroscience. Website: http:/ / www.jneurosci.org/ cgi/ content/ abstract/ 17/2/851.

Colantuoni, C., Rada, P., McCarthy, J., Patten, C., Avena, N. M., Chadeayne, A., et al. (2002). Evidence that intermittent, excessive sugar intake causes endogenous opioid dependence. *Obesity Research,* 10, 478-488. Retrieved January 17, 2008 from Academic Search Premier database.

Colantuoni, C., Schwenker, J., McCarthy, J., Rada, P., Ladenheim, B., Cadet, J., Schwartz, G., Moran, T., & Hoebel, B. (2001). Excessive sugar intake alters binding to dopamine and mu-opioid receptors in the brain. *Neuropore,* 12, 3549-3552.

Epstein, L., Leddy, J., Temple,)., & Faith, M. (2007). Food reinforcement and eating: A multilevel analysis. *Psychological Bulletin,* 133(5), 884-906. Retrieved January 17, 2006 from Academic Search Premier database. Retrieved January 17, 2008 from Academic Search Premier database.

Galic, M. A., & Persinger, M. A. (2002). Voluminous sucrose consumption in female rats: Increased "nippiness" during periods of sucrose removal and possible oestrus periodicity. *Psychological Reports,* 90, 58-60. Retrieved January 17, 2008 from Academic Search Premier database.

Gautier, J. F., Chen, K., Salbe, A. D., Bandy, D., Pratley, R. E., Heiman, M., et al. (2000). Differential brain responses to satiation in obese and lean men. *Diabetes,* 49, 838-846. Retrieved January 17, 2008 from Academic Search Premier database.

Haddock, C.K. & Dill, P.L. (1999). The effects offood on mood and behavior: Implications for the addictions model of obesity and eating disorders. *Drugs & Society*, 15(1-2), 17-47. Retrieved January 17, 2006 from Academic Search Premier database.

Hudson, J., Hiripi, E., Pope, H., & Kessler, R. (2007). The prevalence and correlates of eating disorders in the national co morbidity survey replication. *Biological Psychiatry,* 61, 348-358. Retrieved January 17, 2008 from Academic Search Premier database.

Hursh, S. R., & Bauman, R. A. (1987). The behavioral analysis of demand. In L. Green & J. H. Kagel (Eds.), *Advances* in *Behavioral* Economics (Vol. 1, pp. 117-165). Norwood, NJ: Ablex.

Karhunen, L. J., Lappalainen, R. 1., Vanninen, E.)., Kuikka, J. T., & Uusitupa, M. I. (1997). Regional cerebral blood flow during food exposure in obese and normalweight women. Brain, 120, 1675-1684. Retrieved January 17,2008 from Academic Search Premier database.

Kelley, A. E., Bakshi, V. P., Haber, S. N., Steininger, T. L., Will, M. J., & Zhang, M. (2002). Opioid modulation of taste hedonics within the ventral striatum. *Physiology* & *Behavior,* 76, 365-377. Retrieved January 17, 2008 from Academic Search Premier database.

Marohn, S. (2004). The *natural medicine* guide to *addiction.* Charlottesville: Hampton Roads Publishing Company, Inc.

O'Brien, K. & Vincent, N. (2003). Psychiatric comorbidity in anorexia and bulimia nervosa: Nature, prevalence, and causal relationships. Clinical *Psychology Review,* 23, 57-74. Retrieved January 17, 2008 from Academic Search Premier database.

Rada, P., Avena, N. M., & Hoebel, B. G. (2005). Daily bingeing on sugar repeatedly releases dopamine in the accumbens shell. *Neuroscience,* 134, 7 37-744. Retrieved January 17, 2006 from Academic Search Premier database.

Riva, G., Bacchetta, M., Cesa, G., Conti, S., Castelnuovo, G., Mantovani, F., et al. (2006). Is severe obesity a form of addiction? Rationale, clinical approach, and controlled clinical trial. *Cyberpsychological Behavior,* 9(4), 457-479. Retrieved, January 17, 2008 from Academic Search Premier database.

Rolls, B. J. (2003). The supersizing of America: Portion size and the obesity epidemic. *Nutrition Today,* 38, 42-53. Retrieved January 17, 2008 from Academic Search Premier database.

Rothman, R., Baumann, M., Prisinzano, T., & Newman, A. (2007). Dopamine transport inhibitors based on GBR12909 and benztropine as potential medications to treat cocaine addiction. *Biochem Pharmacal* 9 (3), 178-192. Retrieved January 17, 2008 from Academic Search Premier database.

Volkow, N.D., & Wise, R. A. (2005). How can drug addiction help us understand obesity? *Nature Neuroscience,* 8, 555-560. Retrieved January 17, 2008 from Academic Search Premier database.

Woods, S. (1991). The eating paradox: How we tolerate food. *Psychological Review,* 98(4), 488-505. Retrieved January 17, 2008 from Academic Search Premier database.

Yang, Z. J., & Meguid, M. M. (1995). LHA dopaminergic activity in obese and lean Zucker rats. *NeuroReport,* 6, 1191-1194. Retrieved Thursday, January 17, 2008 from Academic Search Premier database.

Appendix

Table 1.
Mean Differences (MD) for Selected Human Behavioral Indicators

Variable	MD	t	p
Human Sugar Consumption	.343	.898	.379
Behavioral Indicators	2.25	1.29	.21
State Anxiety	2.06	-.54	.60

Figure 1. Average number of sugary products consumed before and after experimental treatment.

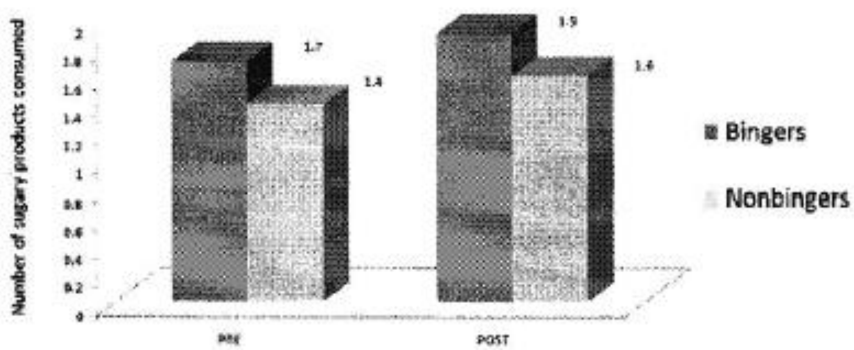

Figure 1 shows that there were no significant differences in the amount of sugar consumed between the two groups before or after treatment.

Figure 2. Average number of behavioral indicators of substance dependence pre and post treatment.

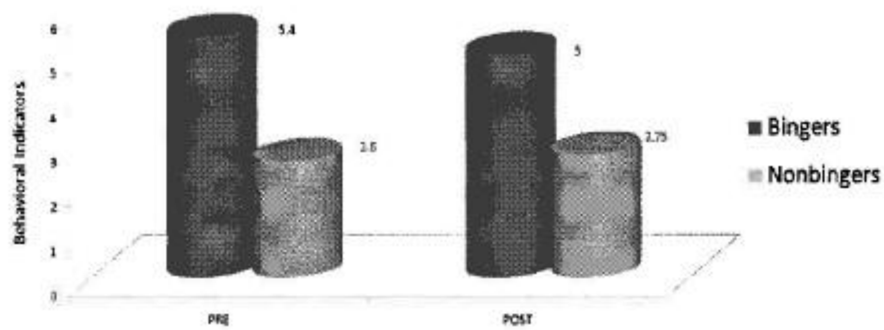

Figure 2 shows that there was no significant difference in behavioral indicators between groups before and after treatment.

Figure 3. Average amounts of sugar solution consumed during 12-hour periods

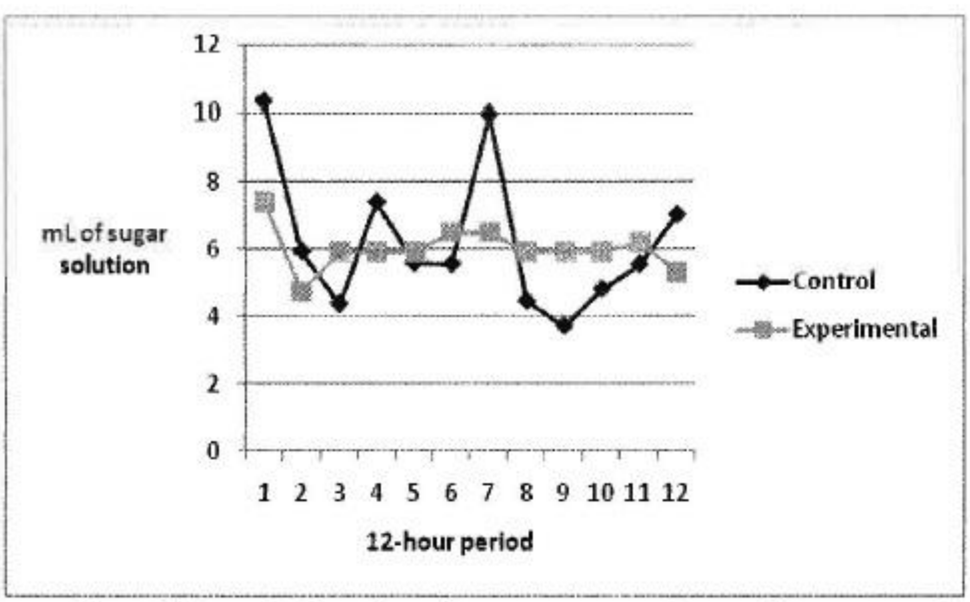

Figure 3. The graph above shows the average amount of sugar solution consumed during12-hour periods when both mice groups had access to sugar solution.

Figure 4a. Mice anxiety scores before and after experimental treatment.

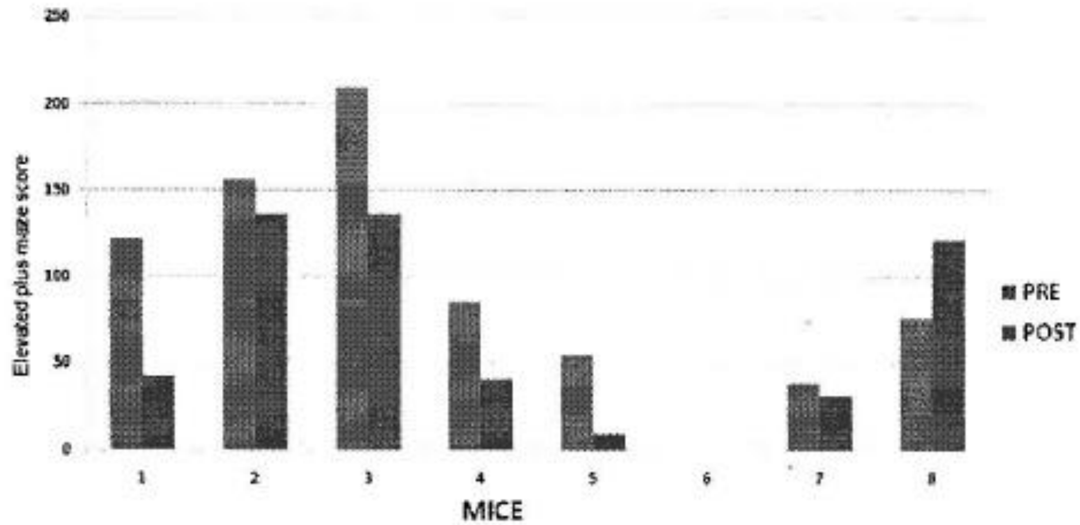

Figure 4b. Mice anxiety scores before and after experimental treatment.

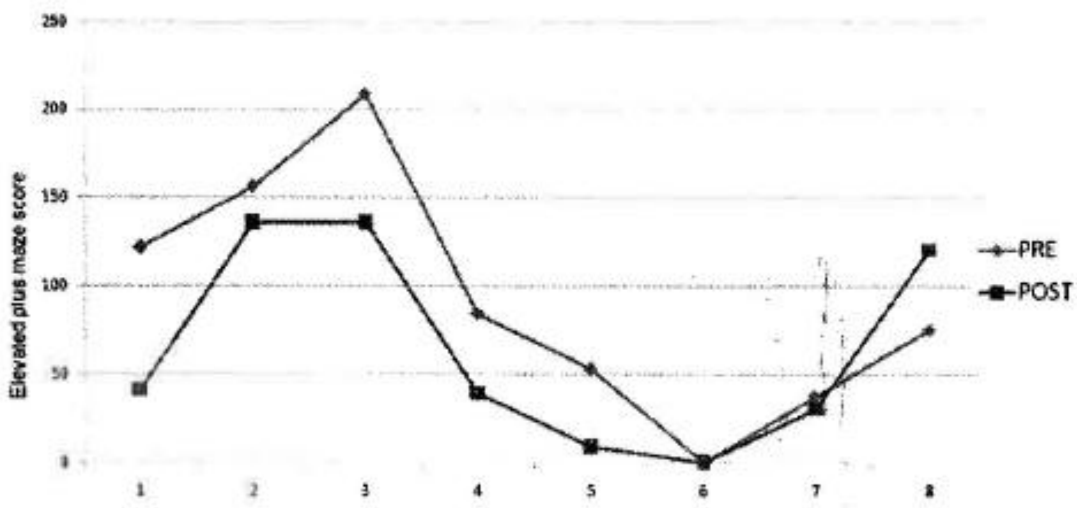

Figure 4 shows anxiety scores of mice before and after treatment. Note the lower the score, the higher the anxiety. Scores of each individual mouse (a). Line graph showing increased anxiety for almost all mice after the experimental treatment (b). This difference approaches statistical significance (p=.09).

Figure 5a. Mouse depression scores before and after experimental treatment.

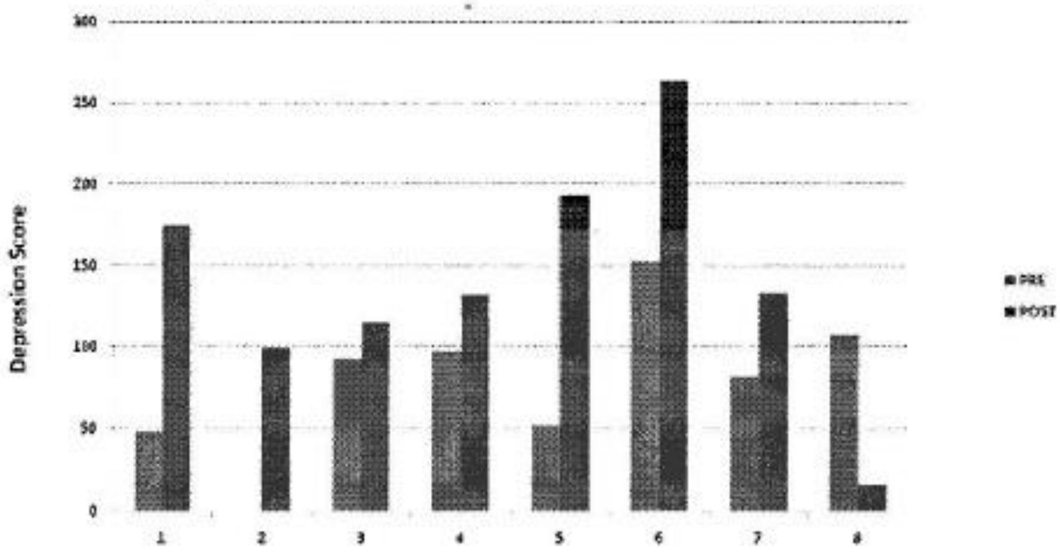

Figure 5b. Mouse depression scores before and after experimental treatment.

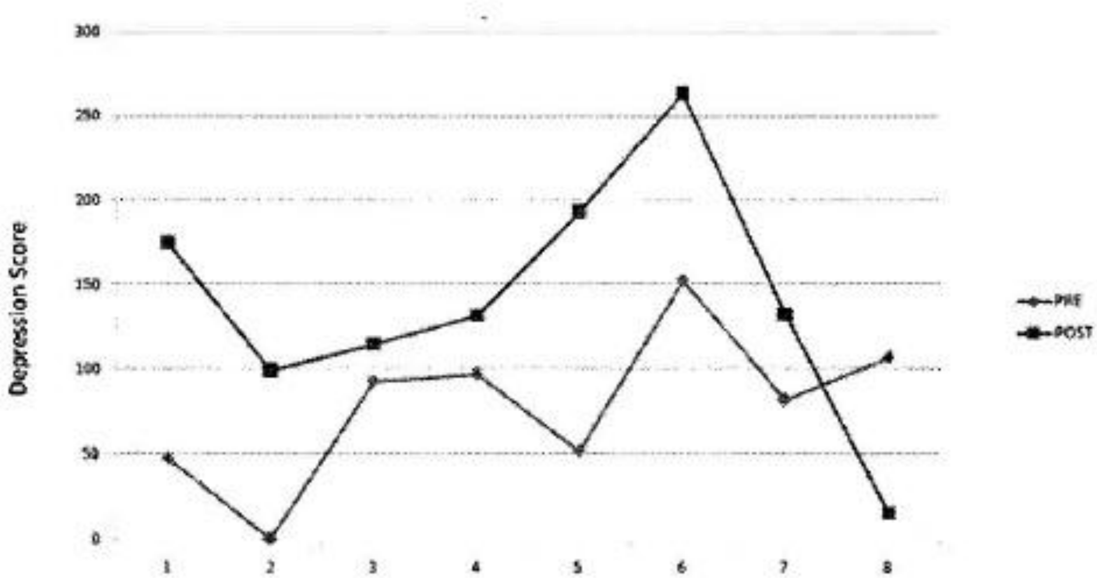

Figure 5 shows depression scores of mice before and after treatment. Note the higher the score, the higher the depression. Scores of each individual mouse (a). Line graph showing increased depression for almost all mice after the experimental treatment (b). This difference is statistically significant (p=.05).

Table 2.
Pearson Correlations Between Daily Sugar Consumption, Post-Intervention Symptoms Relieved by Eating Sugary Foods, and Post-Experiment Sugar Consumption

Variables	Post-Intervention Symptoms RESF
Pre-Intervention RSC	.522**
Post-Intervention RSC	.515**
	Post-Intervention Symptoms SUGDEP
Pre- Intervention DSC	.432*
Post-Intervention DSC	.390*
During Intervention DSC	.389*

*p < .05 **p < .01
RSC: Report of Sugar Consumption
DSC: Daily Sugar Consumption
RESF: Relieved by Eating Sugary Foods
SUGDEP: Sugar Dependence