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

Research Report

By

Kahlilia Morris

Southern Scholars Faculty Committee

April 4, 2008
Of Mice and Men: A Comparative Study Assessing Behavioral Indicators of Sugar Addiction in Mice and College Students

Southern Adventist University

Kahlilia Morris
Evidence of Sugar Addiction: A Comparative Study

Obesity and certain eating disorders are often characterized by an individual’s inability to control food intake. Some researchers (Haddock & Dill, 1999) believe that food dependence may be caused by the psychoactive effects of simple carbohydrates. Apparently the pathways involved in responding to food and other natural rewards are also activated by addictive drugs (Avena, Rada, & Hoebel, 2008). Furthermore, sugar’s ability to release dopamine and its interaction with the opioid system has led scientists to speculate that sugar may have definite addictive qualities (Avena, 2007; Avena, et al., 2008).

The following literature review examines empirical research related to animal and human susceptibilities to sugar addiction. First, the relationship between food dependence and binge eating is described. Then, studies that assess the neurochemical commonalities between sugar consumption and the utilization of substances of abuse are presented. Finally, an animal model of sugar addiction that addresses both behavioral and neurochemical implications are summarized.

Binge Eating and Food Dependence

Eating is a homeostatic mechanism that helps maintain nutrients, minerals, and vital fluids at important physiological levels (Woods, 1991). However, some individuals display maladaptive eating behaviors such as binge eating that can lead to obesity, or eating disorders (Avena, 2007). According to the American Psychiatric Association (2000), binge eating occurs when individuals consume large amounts of food in a short period of time. This behavioral phenomenon is found in a significant portion of the population (Hudson, Hiripi, Pope, & Kessler, 2007) and may pose risks to health (O’Brien & Vincent, 2003).
Research suggests that the behavioral component of binge eating shares many qualities of those expressed during drug addiction (Riva et al., 2006). Like addictive drugs, food is a strong reinforcer that has the ability to highly motivate certain behavior (Epstein, Leddy, Temple, & Faith, 2007). Some researchers posit that in certain situations food may be an even stronger reinforcer than drugs of abuse (Hursh & Bauman, 1987). 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. Many scientists believe that addiction may pose a valid explanation for food dependence because feeding behavior developed from the same neural pathways that are activated by addictive drugs (Kelley, et al., 2002). Therefore, it is possible that binge eating may due to a food dependence caused by the addictive nature of certain food substances.

Sugar as an Addictive Substance

Volkow and Wise (2005) found that neurobiological similarities exist between binge eating and addictive drugs. 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.

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. According to Avena (2007) the nucleus accubens is a part of the brain involved in reward and reinforcement. Both sugar and drugs of
Sugar addiction causes the repeated release or the reduced reuptake extracellular dopamine in this area of the brain (Bassero & Di Chiara, 1997). Indeed, dopamine is released anytime an animal is exposed to novel foods, but 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 due to intermittent access to sugar (Avena et al., 2008).

**Animal Models of Sugar Addiction**

Animal models have provided evidence for sugar's congruity to drug addiction. For example, the expression of addictive behavior (Colantuoni et al., 2002; Galic & Persinger, 2002), certain neurochemical changes (Bassareo & Di Chiara, 1997), and cross-sensitization (Avena, et al., 2008) observed in animals after certain modes of sugar consumption suggest that humans may also be susceptible to sugar addiction.

Animal models have provided a possible explanation for bingeing behavior observed in some humans. For example, according to Rolls (2003) individual differences exist in the way in which the brain's dopamine response system responds to excessive food intake. In studies 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.

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. Apparently giving rats intermittent access to a sugar solution and lab chow causes them to binge on the sugar solution when it becomes available. Rats placed on this feeding schedule have altered accumbens dopamine release activity (Avena, 2007). As rats binge on sugar, they also display aggression and signs of withdrawal, such as anxiety and depression (Colantuoni et al., 2002).

Summary and Critique of Literature

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 sugar dependencies observed in animals may provide a plausible explanation for certain maladaptive eating behaviors in humans.

Hypotheses

Null Hypotheses. Seven null hypotheses were tested in this study:

1. Sugar bingeing has no effect on the amount of ‘sugary’ products consumed in diet.
2. Sugar bingers do not have increased behavioral indicators of substance dependence.
3. Removing sugar from the diet in humans does not increase state anxiety.
4. There is no relationship between sugar consumption and caffeine use.
5. Intermittent access to sugar has no effect on the amount of sucrose solution consumed in laboratory mice.

6. Removal of access to sugar does not affect mouse anxiety.

7. Removal of access to sugar does not affect mouse depression.

**Research Hypotheses.** Seven research hypotheses will be addressed in this study:

1. Sugar bingers will increase the amount of 'sugary' products in their diet.
2. Sugar bingers will have increased behavioral indicators of substance dependence.
3. Removing sugar from the diet of sugar bingers will increase their state anxiety.
4. There is a positive correlation between sugar consumption and caffeine use.
5. Intermittent access to sugar will cause an increase in the amount of sucrose solution consumed laboratory mice.
6. Removal of access to sugar will cause an increase in mouse anxiety.
7. Removal of access to sugar will cause an increase in mouse depression.

**Research Questions**

Two research questions guided this study:

1. Is there a relationship between gender and certain behavioral indicators of sugar dependence?

2. How does a family history of substance dependence affect behavioral indicators of sugar dependence?

**Definition of Terms**

Eleven terms are operationally defined in this study:

1. Sugar bingeing in humans is operationally defined as eating 30g of sugar in three minutes or less.
2. 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 three weeks.

3. Caffeine use was measured by the number of reported caffeinated drinks consumed in a 7 day period.

4. Removing sugar from the human diet refers to abstinence from sugary products for approximately 2.5 days.

5. Removing sugar from the mouse diet refers to abstinence from the sucrose solution for 3 days.

6. 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).

7. Depression in laboratory mice was assessed by analyzing passive or active escape efforts when the mice are placed in water (Avena et al., 2008).

8. ‘Sugary products’ in this study refer to sweet substances, high in sugar content.

9. Family history of substance dependence was assessed by the score received by each participant on the family history section of the Phelps-Nourse assessment.

10. Behavioral indicators of substance abuse in human participants were evaluated with the Phelps-Nourse assessment.

11. Anxiety in human participants was measured using the Spielberger Inventory for State Anxiety.

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. 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 post-assessment.

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 cm in diameter, filled with water (21-23°C) about 17 cm 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 Phelps-Nourse 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.

Data Analysis

Data were coded and entered into SPSS for analysis. Independent samples t-test, paired sample t-tests, and Pearson’s correlation coefficient 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. 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=1.05) sugar products in their diet per day. An independent samples t-test shows that the mean difference was not statistically significant (Table 1). Therefore, the null hypothesis that sugar bingeing has no effect on the amount of sugary products consumed could not be rejected \( t(23)=.898, p=.379 \).

**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=5.41) on the post behavioral indicators measure and nonbingers obtained a mean score of 2.75 (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). 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 ($r(30)=.19$, $p=.31$). There was also no significant correlation between the sugar consumed during the experimental period ($r(30)=.308$, $p=.10$) 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 using Pearson correlation coefficients showed the relationship 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 ($r(27)=.47$, $p=.01$). No statistically significant correlation was present between gender and symptoms before the experimental period ($r(32)=.29$, $p=.106$). Therefore, women were more likely than men to report experiencing symptoms. 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 1 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 2 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 of this study was that removal of sugar access will cause an increase in mouse depression. Figure 3 shows post the forced swim test (FST) 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 while 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\}.

Pearson correlation coefficients showed that there are positive correlations 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. 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.

In the PNIAP, for both the pre and post measure, participants responded to how many symptoms of dependence were relieved by eating sugary foods. Statistically significant correlations existed 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.

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.
Still, both the human and mice experiments showed that 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.

Gender Differences

Women were more likely than men to report symptoms after the experimental intervention than men. 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 sugar 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 proper 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


Rothman, R., Baumann, M., Prisinzano, T., & Newman, A. (2007). Dopamine transport inhibitors based on GBR12909 and benztropine as potential medications to treat cocaine addiction.


Appendix

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

<table>
<thead>
<tr>
<th>Variable</th>
<th>MD</th>
<th>t</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Human Sugar Consumption</td>
<td>.343</td>
<td>.898</td>
<td>.379</td>
</tr>
<tr>
<td>Behavioral Indicators</td>
<td>2.25</td>
<td>1.29</td>
<td>.21</td>
</tr>
<tr>
<td>State Anxiety</td>
<td>2.06</td>
<td>-.54</td>
<td>.60</td>
</tr>
</tbody>
</table>

Figure 1. The graph above shows the average amount of sugar solution consumed per mouse during 12-hour periods when both groups had access to sugar solution.
Figure 2. Pre and Post Elevated Plus Maze Scores

This graph shows scores obtained on the elevated plus maze before and after sugar withdrawal. All the mice except for one have increased anxiety. Note that lower scores signify higher anxiety.
Figure 3. This graph shows depression scores before and after the withdrawal period. All the mice except for one have increased depression scores.
Name: Kanilua Morris  Date: 11/6/08

Major: Psychology

A significant scholarly project, involving research, writing, or special performance, appropriate to the major in question, is ordinarily completed the senior year. The project is expected to be of sufficiently high quality to warrant a grade of "A" and to justify public presentation.

Under the guidance of a faculty advisor, the Senior Project should be an original work, should use primary sources when applicable, should have a table of contents and works cited page, should give convincing evidence to support a strong thesis, and should use the methods and writing style appropriate to the discipline.

The completed project, to be turned in in duplicate, must be approved by the Honors Committee in consultation with the student's supervising professor four weeks prior to the last day of class for the semester the project is turned in. Please include the advisor's name on the title page. The 2-3 hours of credit for this project is usually done as directed study or in a research class.

NOTE - Senior Project Proposal Due Date: The senior project proposal is due in the Honors Program Director's office two weeks after the beginning of the semester the project will be completed. The proposal should be a detailed description of the Honors Project's purpose and proposed methodology.

Keeping in mind the above senior project description, please describe in as much detail as you can the project you will undertake. Attach a separate sheet of paper.

Signature of faculty advisor: 

Expected date of completion: April 1, 2009

NOTE: An advisor's final project approval does not guarantee that the Honors Faculty Committee will automatically approve the project. The Honors Faculty Committee has the final vote.

Approval to be signed by faculty advisor when the project is completed:

This project has been completed as planned (date)__________

This is an "A" project_______________________________________

This project is worth 2-3 hours of credit_______________________

Advisor's Final Signature: ______________________________ Date: ____________

Chair, Honors Committee: Mark Peach Date Approved: 1/24/08

Dear Advisor,

(1) Please write your final evaluation on the project on the reverse side of this page. Comment on the characteristics that make this "A" quality work.

(2) Please include a paragraph explaining your specific academic credentials for advising this Senior Project.
Evaluation of Kahlilia Morris’ Senior Project April 2008

Kahlilia’s project utilizing both an experimental and a quasi-experimental design with human participants and animal subjects was ambitious in its formulation and well executed in its implementation. The following Research Report format was used to evaluate her senior project which is the research report of her sugar addition study. It is rare to find an undergraduate paper in which a student goes to such great lengths to answer scientific questions of interest. The paper is of A caliber and raises interesting questions that could well be addressed in future research.

Signed,

Ruth Williams-Morris
Research Project Faculty Advisor

RESEARCH REPORT (FINAL)

Cover Page
Title Page
Abstract
Title

Introduction (do not type sub-title)
  Introductory Paragraph (no sub-title)
  Literature Review (no sub-title, but sub headings in body of review)
  Purpose of study (no sub-title)
  Definition of terms, if applicable
  Hypotheses/Research questions

Comprehensive coverage of addiction research

Method
  Participants
  Materials/Apparatus
  Design and Procedure
  Data Analysis

Complicated research procedure, but well managed
Results
Describe sample using descriptive statistics as appropriate
Repeat Hypotheses and Research Questions; state which statistical test was used
to test each hypothesis or answer each research question; provide results first in
statistical terms relevant to the statistical test used and then in lay terms.
Be sure to refer to tables and graphs within the text of this section
Describe any unexpected or interesting findings under the subheading of
Other Interesting Findings

Discussion
Apply the “Seven-year-old Nephew Test” to this section: Use lay terms as
much as possible.
Restate the purpose of your study.
Summarize the Research Questions and Hypotheses
State your findings. Why do you think you found what you did or did
not find what you expected? Discuss.
State the limitations and weaknesses in your study.
Relate your current findings to the theoretical/empirical base of your literature
review.
State the importance of your study
Describe an agenda for future research

Well done

References
Author’s Note
Tables
Figure Captions page
Figures

Not applicable for Senior Project

Appendix
Informed Consent Form (blank)
Questionnaire/Survey/Recording Form
Scoring Key for Questionnaire
Coding Sheet
Additional Graphs/Tables
SPSS output
Proposal