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Long-term effects of sucrose and carbohydrate on blood glucose and aggression in mice.

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Long-Term Effects of Sucrose and Carbohydrate on Blood Glucose and Aggression in Mice

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CHAPTER 1

INTRODUCTION

1.1 Background.

The study of nutritional effects on behavior is in its infancy. The literature is rapidly growing but still small and scattered, and most issues are considered far from resolved. The macronutrient that has received the most attention in the popular literature but that has only recently returned to the scientific literature since its brief popularity there in the 1960's, is sucrose. Sucrose has been linked to practically every physical and mental disorder. Some of the most common symptoms alleged to be related to sucrose consumption include fatigue (Wurtman, J. 1986), cognitive impairment particularly in terms of memory problems (Behar et al., 1984), low activity level (Behar et al., 1984), high activity level (Buckalew & Hickey, 1983; Conners & Blouin, 1982/83), hyperkinesis (Gross, 1984; Prinz et al., 1980; Walraich et al., 1985), mood shifts (Brody & Wolitzky, 1983), abnormal blood sugar metabolism (Hallfrish et al., 1983; Liu et al., 1984; Cohen & Teitelbaum, 1964), and aggression (Benton et al., 1982; Neideffer et al., 1977; Schoenthaler & Doraz, 1983). With a few exceptions, studies have been poorly controlled and results inconclusive or conflicting.

The primary problems with most of the studies done to date are the brevity of the studies, the lack of time before the manipulation for controlling the previous diet, and the small amount of substance used compared with typical levels of sucrose ingestion in human beings. There are three reasons that a lengthier study than has commonly been done is crucial for this type of research. First, and most important, is that as long as two weeks are needed before behavioral effects from some diet changes are manifest (Kantak &

Eichelman, 1982). While it is common practice in drug studies to take factors such as this into account, most sucrose studies last no more than a few hours, particularly those with human beings. The second reason is that even after physiological changes have occured, all corresponding behavioral changes will not immediately follow. It takes time for learned behaviors to diminish even when a cause of the behavior is removed. Most sugar-behavior studies have been conducted with hyperkinetic, or at the least, problematic children. Even if removing sugar briefly from or adding it to their diets makes a difference physiologically, the change may not be immediately manifest: Behaviors in a classroom situation are well learned and should therefore be expected to be maintained for a while. This effect can be reduced with a long-term study, particularly if animals are used as subjects. Animals exhibit learned behaviors as human beings do, but the environmental interactions to which their behaviors are linked are fewer and less complex. The third reason for a longer study is that it permits for differentiation between short and long term effects, which in many aspects of physiological functioning differ dramatically. In summary, a long-term study would allow the time needed for both physiological and behavioral changes to occur, and would also reduce the risk of confounding short-term and long-term effects.

Just as it is problematic to neglect to follow a study for a sufficient period of time after experimental manipulation, it is important to consider both short-term and long-term pre-experimental diets. What is consumed the day of a short-term study can affect the subject's reactions to the experimental food challenge. For example, Conners (1986) found that hyperkinetic children had a larger rise of blood glucose than normals did after a glucose challenge if they had a carbohydrate meal before the challenge. Long-term differences in pre-experiment diet may present an even greater confound

than short-term differences. The physiological adjustments made to handle 50% versus 10% of calories as sucrose for many years would seem to differ. Different long-term pre-experimental diets among subjects may therefore result in different degrees and speed of accomodation to the sucrose challenge regardless of the experimental manipulation. A subject regularly exposed to large quantities of dietary sucrose may be less reactant to a small sucrose challenge than a subject less accustomed. Correspondingly, the dose needed to affect a subject used to large versus small quantities of dietary sucrose would seem to differ. The most common sucrose dose in human challenge studies has been the equivalent of the sugar content of one soda, and sometimes this has been spread over one to three hours. Such a manipulation may not serve as a sugar "loading" for most subjects: Since most U.S. citizens consume much more sucrose daily than the equivalent of a soda, the body may accommodate fairly well to that dosage in order to avoid continual extreme reactions. Two conclusions follow: First, long-term analyses controlling the pre-experimental diet are needed. Using animals it is possible to control for dietary sucrose content from birth. Second, the experimental dose of sucrose needs to be at least large enough to simulate the relative equivalent of a common human intake level.

A question central to many of these issues is whether or not the body does in fact make accomodations over time to a high sucrose diet. Kanarek and Marks-Kaufman (1979) looked at fasting blood glucose levels of rats 46, 57, and 70 days old. At 46 and 57 days, the rats that had been on a high sucrose diet since weaning had significantly lower blood glucose than rats fed only Purina Chow. This trend continued at 70 days but not significantly. The rats were then sacrificed. Whether blood glucose levels of the high sucrose group would decrease, level off, or continue upward in comparison to the low glucose group

after 70 days of age is yet to be determined. Cohen and Teitelbaum (1964) conducted an intriguing study with rats in which they substituted various percentages of sucrose for starch in nutritionally balanced long-term diets. The glucose tolerance of the rats in the sucrose groups became impaired. Correspondingly, the fat content of the liver increased, and the insulin-like activity of the sera decreased. There were no differences in total body weight, nor in fat body content, nor in weight of livers as determined by grams per 100 g body weight. The less sucrose in the diet the longer it took for the impairment to take place, with 21-40 days needed in the 67% sucrose group, 40 days in the 40% sucrose group, and 50-100 days in the 33% group. The results were reversible: Glucose tolerance tests of animals on the 67% sucrose group for 78 days returned to normal after being put for 15 days on the diet in which other carbohydrates replaced the sucrose. After at least 21 days on this non-sucrose diet the animals were returned to the 67% sucrose diet. Instead of requiring 21-40 days again for abnormal glucose tolerance conditions to be reached, this time it only took 6 days. Cohen and Teitelbaum suggest that the frequently repeated rapid absorption of the glucose may, through overstimulation, impair the insulin system, resulting in a reduced insulin reserve. More long-term studies are indicated to replicate these studies and to further explore the possibility of cumulative effects or even of relatively permanent physiological changes.

In doing this research it will be important to consider whether or not there is a difference between the effects of sucrose and those of carbohydrates in general. In other words, if there is a sucrose effect, is that because of sucrose uniquely or is the effect because sucrose is a carbohydrate? It is widely taught and accepted that sucrose is just like any other carbohydrate. After all, it is argued, sucrose breaks down into the same components as any

other starch. However, the Cohen and Teitelbaum study (1964) would indicate that sucrose consumption leads to results that are profoundly different from those from the consumption of other carbohydrates. Effects from a high sucrose diet were reversed when the sucrose was replaced by another carbohydrate. Also, there were no significant differences between the carbohydrate group and the control group, whose diet was composed of 60% rather than 67% carbohydrate. It would have been interesting to see, however, if there would have been a difference in a high versus low carbohydrate diet if the difference in percentage had been greater. Also in support of a unique sucrose effect are long-term human studies by Reiser et al.: Replacing complex carbohydrates with sucrose affected various aspects of carbohydrate and lipid metabolism and indices of glucose tolerance even in a low-saturated fat, high-fiber diet (Reiser et al., 1981; Reiser et al., 1986).

Perhaps there are ways in which sucrose uniquely affects physiology as described above in addition to having other effects that are in common with any carbohydrate. These "other effects" could involve effects of sucrose and any other carbohydrate on the amino acid tryptophan, the precurser of the neurotransmitter serotonin. Tryptophan has been receiving a lot of attention recently as a substance whose flow into the brain is affected by diet, and specifically by carbohydrate consumption. It has been found that consumption of carbohydrates increases levels of serotonin (Wurtman et al., 1981; Wurtman, 1982) in the brain. For this effect, it makes no difference whether the carbohydrate is sucrose or some other starch (Wurtman, J. 1986). Except for fructose, any starch will lead to insulin secretion. The insulin depresses the serum levels of most large neutral amino acids which normally compete successfully with tryptophan for transport into the brain. The levels of tryptophan do not fall because the tryptophan binds to a serum globulin. If

little protein is consumed with the carbohydrates, the insulin will be able to clear away enough of the large amino acids for an increased amount of tryptophan to enter the brain (Wurtman, J., 1986).

Several studies indicate that carbohydrate consumption can affect behavior in many of the ways often attributed to sucrose, and in ways that could actually have to do with this tryptophan effect. Prinz et al. (1980) found that restlessness and destructive aggressiveness in hyperactive children correlated with the amount of sugar consumed, the ratio of sugar products to other foods, and the carbohydrate-protein ratio of the food they consumed over the one week charted period of the study. However, since the study was correlational, it is possible that the activity or aggression led to the high sugar/carbohydrate consumption. Chiel and Wurtman (1981) found that a diet with a higher carbohydrate-protein ratio resulted in a higher activity level in rats within 3 days. This change in spontaneous motor activity was not due to amount of food eaten, change in weight, or increase in fat content from 15% to 45%. Rather, Chiel and Wurtman conclude that it is likely that the effect is from the relatively high percentage of carbohydrate to protein facilitating an increased flow of tryptophan into the brain. Here then is a possible way in which sucrose could in the same way as any carbohydrate lead to physiological, and then behavioral, change.

The study of factors that may influence blood glucose levels and tryptophan/serotonin levels is important for many reasons, one of which is common to both. That is, both have implications for aggression. Low blood glucose levels have been linked in both human and animal studies to aggression (Benton et al., 1982; Neideffer et al., 1977; Shively & Kaplan, 1984; Virkkunen, 1984; Virkkunen, 1982). Dominant rodents were found (Shively & Kaplan, 1984) to have lighter adrenal glands and lower plasma glucose

concentrations than subordinates. Does the low blood sugar cause the aggression, does the aggression result in low blood sugar, is there some interaction, or is none of these hypotheses correct? If chronic high sucrose consumption leads to low blood glucose (Kanarek & Marks-Kaufman, 1979) then sucrose consumption may be related to at least one type of aggression. With longitudinal studies these issues can begin to be resolved. The purpose of Part I of this study is to collect systematic short-term and long-term data on the effect of dietary sucrose on blood glucose in mice.

Decreased serotonergic inhibitory control in the brain is the most commonly accepted explanation for the pathology of aggressive behavior (Valzelli, 1981). One way in which high levels of sucrose and of carbohydrates in general may both have an impact on aggressivity is by affecting the level of serotonin in the brain. There is currently evidence that manipulations with tryptophan can affect brain levels of serotonin (Spring et al., 1987). One result of this can be a change in aggression level. A study (Gibbins et al., 1979) in which rats were put on a tryptophan-free diet for 4-6 days resulted in mouse killing by non-killer rats and a decreased latency of mouse killing by killer rats, along with a 26% reduction in brain serotonin (5-HT) and a 29% reduction in brain 5-hydroxyindoleacetic acid (5-HIAA), a metabolite of serotonin. When then supplimented with L-tryptophan, 5-HT and 5-HIAA both went above levels of those of the controls and killing responses returned to normal. In another study (Thurmond et al., 1977), however, no effects were found when looking at the relationship between tryptophan and aggression in mice. The likely explanation is that there was protein in every diet, as protein would have blocked any extra tryptophan from entering the brain. Part II of this study will look at possible effects on aggression of chronic diets high in sucrose, carbohydrate, or carbohydrate eaten separately from protein.

Diets high in carbohydrates, particularly when the carbohydrates are not consumed at the same time as proteins, enable a large amount of tryptophan to enter the brain. The resulting short-term effect is that the level of serotonin correspondingly increases (Wurtman, J., 1986). This, clearly, would not lead to aggression. But what if the short-term effect is different from the long-term effect? Often the acute effect of a manipulated substance on a neurotransmitter level is the opposite of the chronic effect. Could it be then that the long-term effect of a diet high in carbohydrate relative to protein could be a decrease, rather than an increase, in serotonin? That low levels of serotonin, associated with aggressive behavior, may result from a long-standing high carbohydrate diet, particularly if the carbohydrate is frequently consumed in the absence of protein, is a possibility that needs to be explored. In this study, diets will be manipulated in such a way as to enable an initial look at this question.

1.2 Summary.

The purpose of this study is to explore possible effects of chronic high sucrose or carbohydrate consumption on the two biochemical factors most commonly accepted as being causes of aggression: low blood glucose levels and low serotonin levels.

CHAPTER 2

METHOD

2.1 Subjects.

The subjects were male C57BL/6 mice purchased from Charles River Breeding Laboratories where they were maintained on a standard pelleted diet. There were 64 experimental animals, ages 21 and 22 days old on the day of delivery. They were randomly distributed among four treatment groups at age 31 days.

2.2 Housing Conditions.

The subjects were housed in 6" X 8" bins with 900 ml. shavings. Cage covers were stainless steel with suspended water bottles. During the first 24 hours, the mice were group housed by age with 3 or 4 animals per compartment. During this period, they were provided with both the Charles River Laboratory food and with a generic powder similar to the diets they would soon be put on. In this way, the mice who recognized the powder as food could model eating it before all of the animals were isolated. The second day, the mice were housed individually in random order and were provided with only the generic diet. The compartments were cleaned weekly. Room temperature was 69 degrees Fahrenheit. Dark onset was at 19:00 and light at 7:00. From 19:00 until 21:00 the room was lit with a 25 watt red light bulb.

2.3 Food.

Food was available ad libitum for all groups except between 16:00 and 22:00 daily. Water was available ad libitum at all times. The food was held in a 2 oz. glass Qorpak jar inside each compartment. The jar was covered except for a

drilled hole of one inch diameter. This allowed the animal's head to reach the food while discouraging the animal from climbing into the jar, and in general limited spillage. To further deter spillage, the food was directly covered by a nickel-coated brass sink drain of one and a half inch diameter that functioned inside the jar as a cup with holes. To assure accessibility of the food through the holes, the food was fluffed daily. Also, droppings and shavings were removed before daily food weighings.

New food was made weekly. Weeks were defined according to blood testings: Week #1 ended with the first blood testing. Week #2 ended with the second blood testing, and so on. (Note that the week defined as #2 was the first week during which the animals were on the experimental diets.) Each new week started with new food and a clean compartment, as both were changed right after blood testing.

The food (see Table 1) was given as powder mixed with corn oil. The constituents of the diets were from Nutritional Biochemicals. The proportions of nutrients in the base sixty-five per cent by calorie of each diet were identical for each group, providing a simple standard protein diet: The total protein intake of each diet was 16%. This was obtained from Casein Vitamin Free supplimented with .24% DL-methionine. AIN Vitamin Mixture made up 1% of the total diet, and AIN 76 Mineral Mixture 3.5%. As is standard, the largest portion of the diet - 34.26%- was carbohydrates. The carbohydrate was all rice starch except in the sucrose groups in which 24.26% was rice starch and 10% was sucrose. Corn oil made up 9.8% of the diet. To equate the palatability of the diets, it was necessary to add an artificial sweetener to the non-sucrose diets. There are indications that aspartame may have a neurologic and behavioral impact (Pardridge, 1986) and that, if mice are like gerbils, it may not stimulate the animal's taste receptors (Jakinovich, 1982).

Saccharin sodium salt was therefore used, as it presents neither of these problems. The .2% saccharine in the non-sucrose groups was balanced by .2% hydrolyzed alphacel in the other groups. The remaining 35% calories varied depending on treatment group. In Diet #1 (Suc) it was sucrose, in Diet #2 (Carb) it was complex carbohydrate in the form of rice starch, and in Diet #3 (Fat) it was fat in the form of corn oil. Diet #4 (Suc2) was of the same total composition as Suc. However, the constituents of the Suc2 diet were available to the animals in a different manner than in Suc: From 18:00 to 20:00, they were fed a snack made up only of sucrose, complex carbohydrate, and fat. (Fat was actually not added until week 4. It was added in hopes of correcting for an inequality of amount of snack consumed between Suc2 and the other groups.) Between 16:00 and 18:00 and between 20:00 and 22:00 no food was available in order to prevent protein consumption with or shortly before or after the sucrose/carbohydrate loading. Then the remaining ingredients of the diet were available for the other 18 hours. The amount of sucrose mixture was determined by allotting 11-14% (varied by week) of the average daily intake of all the animals from the previous week. (Note: The 1st two weeks this amount was based on the daily mean consumption by week of pilot animals who were 7 days older. Week 7 was based on week 5's consumption due to abnormal consumption week 6.) The snack was given in the same type of jar as was used for the regular diet except that there was no sink drain. The other groups were on the same feeding schedule, but their snack was the same as their normal food. Within each week, the snacks of all mice of all groups were identical (+/- .005 grams) by Kcals. The amount of snack consumed was rated on a 4 point scale:

0 = no food remained

(0) = 20% or less of the food remained

2 = approximately 1/2 of the food remained, with an allowable range of 21 to 75% of the food remaining

3 = very little or no food eaten: at least 76% of the food remained

Interobserver reliability on snack ratings ranged from percent agreement scores of 94% to 98% based on 128 ratings. Intraobserver reliability ranged from 91% to 97% based on 33 ratings. Raters were never off by more than 1 point.

2.4 Overall Description of the Research.

This research consisted of two interrelated studies. Part I looked at blood glucose levels over time while also setting the stage for possible differences in levels of serotonin. Part Ia consisted of 12 weeks of weekly fasting blood testing. Part Ib consisted of a blood test 3 hours after a glucose challenge (given by gavage) week 13 and 30 minutes after a glucose challenge (given by gavage) week 14. Part II of this research looked at behavioral effects of the manipulations of Part I: The subjects from Part I were tested for aggression levels.

2.5 Part I: Blood Testing.

2.5.1 Part Ia: Blood Testing After Fast.

In order to minimize the extent of an adrenalin response during blood testing, two steps were taken. First, during the week each compartment was carried to a different section of the room for the 4:00 and 6:00 feedings each day in order that moving would not be associated only with blood testing. Second, testers were trained to work quickly and to not touch each compartment until ready to begin.

Blood testing was conducted in a corridor outside of the room in which the mice were housed. It took place over 2 consecutive days each week such that all mice were of the same age when tested. Initial blood tests were taken at 31 days of age, followed by the beginning of diet manipulation. Food of the animals to be tested was removed each week at 8:00 the morning of testing. Testing was begun at 1600 and completed at approximately 1800. Animals were tested sequentially but starting with a randomly chosen number each week. Two 75 microliter capacity heparinized microhematocrit capillary tubes were filled from the tail. Within at most 3 hours of collection, the blood was centrifuged for 15 minutes at 4000 rpm's in an IEC clinical centrifuge. The plasma was collected and then frozen below -70 degrees Celsius. Except for a 20 minute interlude for the centrifuging process, the blood was kept in ice from immediately after collection until being frozen. For analysis, the frozen plasma was thawed rapidly and analyzed promptly for glucose levels with a Yellow Springs glucose analyzer.

2.5.2 Part Ib: Blood Testing After Sucrose Challenge.

Initial intentions were to begin gavage trials week 6. Most of the animals were therefore gavaged 1-4 times with water that week so that the procedure would not itself be stressful when used on blood testing day. Many of the animals became injured by twisting during the procedure. To avoid the risk of further injury and to allow the experimenter with the best technique a long period of time during which continue to practice the procedure, gavage was delayed until week 13. At that time, all gavaging was conducted by the highly trained experimenter, and there were no casualties.

Week 13 the food of the animals to be tested was removed at 5:00 the day of testing. Eight hours later the subjects were given by gavage a solution of 2 grams of sucrose per Kg of body weight, diluted by 50% with water. Blood

testing took place at the normal time, which was 3 hours after the sucrose challenge. Week 14 the food of animals to be tested was removed at 7:30 the day of testing. Eight hours later the subjects were given by gavage 2 grams of sucrose per Kg of body weight. Blood testing followed by 30 minutes.

2.5.3 Values Used in Analyses.

Although the 4 groups each began with 16 subjects, statistical analysis of most of the individual weeks was conducted with slightly fewer than 16 values per group, and occasionally with many fewer (see Table 2 for N values by diet and week). As one animal was sick during most of the study, its results were not included at all in the analysis. As a result, the N of Carb became 15. The other missing values were particular to specific technical incidents individual weeks. In particular, weeks 1, 2, and 6 have many missing values. A large number of the missing values weeks 1 and 2 were due to a difficulty with analysis: The experimenter analyzing the plasma erroneously allowed a small amount of air to be mixed with each sample, thereby diluting it, with consequent low values. The samples mixed with the most air were not used. Week 6, values of subjects who had strong reactions to being gavaged were not used.

In addition to the missing values, it should be noted that blood collection time decreased weekly, thereby possibly adding noise to early weeks. However, analysis relating blood collection time to glucose value was not able to tease out a significant relationship between the two.

2.6 Part II: Aggression Testing.

2.6.1 Target Animals

For the aggression experiment, only within strain inter-male aggression was considered. The stimuli for the aggression tests were younger,

smaller, male C57BL/6 mice, ages 43 to 49 days, maintained on Purina Chow. Each target was randomly matched with one experimental animal. Between 20 and 25 minutes before testing, the target was marked with a spot of white paint behind each ear for purposes of identification.

2.6.2 Testing Conditions and Procedures.

Testing was conducted between 9:00 and 14:00 over a 7 day period. The testing did not follow snack time in order to avoid measuring acute effects of the protein-free snack. Aggression testing took place in a room near but out of sound range from the room in which the animals were regularly housed. Room temperature was 65 degrees Fahrenheit. The testing chamber was a 48 X 28 X 27 cm glass aquarium divided in half by a removable folded aluminum partition which was 24.5 cm high and which kept the mice 4.5 cm apart. The partition stood up firmly due to a .2 cm wide portion of it which extended along all 4 edges of each side of the testing chamber. Beneath the partition, the floor was lined with Benchkote lab cover absorbent paper. Between each testing, the paper was changed and the aquarium and partition were cleaned with a light amonia solution.

The order of testing was randomly assigned. Each experimental animal was randomly assigned to the left or right side of the chamber with the limitation that half would be on each side. Both during testing and later during coding, the experimenters were blind as to the identification of the subject.

The S and its partner were placed at the same time into separate halves of the chamber. The partition was removed after 5 minutes, allowing the animals to interact for 15 minutes. Tests were videotaped for future coding.

2.6.3 Aggression Coding.

Behaviors were checked if present in each of 90 10 second intervals. Behaviors coded were: Lunge; Rattle, Offensives, Circle; Aggressive Groom; Bite; Biting Attack; Fight. The codes "bite" and "biting attack" were further coded with a letter to indicate target behavior immediately preceeding each attack (see Table 3). For this study, several behaviors that were similar were combined into new variables. The following variables were used for analyzing aggressive behavior in the present study:

MinorA = Lunge + Rattle + Offensives + Circle

AGroom = Aggressive Groom

Ag = Bite + Biting Attack

Code = Code Value of First Bite + Code Value of First Biting Attack

AllAg = Agroom + Ag + Fight

All coding was done by 1 rater, for consistancy. Intraobserver reliability on the above 5 variables ranged from .90 to 1.0 (see Table 4). After all subjects were coded, random subjects were chosen for reliability checks by another trained observer. Interobserver reliability ranged from .77 to .82.

In addition to the coded behaviors, several latencies and time periods were taken from the data of each subject. These were:

1) TLAO1: latency to 1st lunge, rattle, circle, or offensive

2) TAGGR: latency to the 1st aggressive groom, bite, biting attack, or fight

3) TFABAF: latency to the last aggressive groom, bite, biting attack, or fight

4) AGTIME: TFABAF - TAGGR1 (ie., total time between 1st and last aggressive groom, bite, biting attack, or fight)

5) ETIME: TFABAF - time of first target escape code (ie., total time between 1st efforts by target to escape a bite or biting attack, and last aggressive groom, bite, biting attack, or fight)

Table 1

Diet Composition in Grams and KCal

	Suc		Carb		Fat		Suc2		Snack Suc2		Generic Diet for Pre-week1 Blood Testing	
BASE:	grams	Kcal's	grams	Kcal's	grams	Kcal's	grams	Kcal's	grams	Kcal's	grams	Kcal's
Mineral Mix	3.50	1.40	3.50	1.40	3.50	1.40	3.50	1.40			3.50	1.40
Vitamin Mix	1.00	3.88	1.00	3.88	1.00	3.88	1.00	3.88			1.00	3.88
Casein	16.00	64.00	16.00	64.00	16.00	64.00	16.00	64.00			16.00	64.00
DL Methionine	0.24	0.00	0.24	0.00	0.24	0.00	0.24	0.00			0.24	0.00
Corn Oil	9.80	88.20	9.80	88.20	9.80	88.20	8.33	74.97	1.47	13.23	9.80	88.20
Carbohydrate:												
as Rice Starch	24.26	97.04	34.26	137.04	34.26	137.04	21.01	84.04	3.25	13.00	34.26	137.04
as Sucrose	10.00	40.00					10.00	40.00				
Variable:											4.44	40
Corn Oil					15.56	140.00					25.00	100
Rice Starch			35.00	140.00								
Sucrose	35.00	140.00					25.26	101.04	9.74	38.96	0.19	
Saccharin			0.20	0.00	0.16	0.00						
Alphacel	0.20	0.00					0.20	0.00		0.00		
Totals:												94.43
grams	100				80.52		85.54		14.46			434.52
Kcal	434.52				434.52		369.33		65.19			4.601
Kcal/gram	4.345				5.397		4.318		4.508			

Table 2

Numbers of Subjects With Non-Missing Glucose Values by Diet and Week

	Suc (N=16)	Carb (N=15)	Fat (N=16)	Suc2 (N=16)
w1	2	7	9	8
w2	13	11	13	12
w3	16	15	15	15
w4	16	12	15	16
w5	16	15	16	16
w6	13	13	13	12
w7	16	15	15	16
w8	16	14	16	15
w9	16	15	16	15
w10	14	15	16	16
w11	16	15	16	16
w12	16	15	16	16
w13	08	08	08	07
w14	15	14	15	15

Table 3

Description of Behavioral Criteria for Aggression Testing

Lunge: "Rapid thrust of head or fore-part of body towards opponent. Head does not contact opponent's body."

Rattle, Circle, Offensives: Tail Rattle - "Rapid lashing of tail from side to side. Produces "rattle" when solid object (e.g. testing chamber wall) is struck"
Circle - "Circuit is made away from ("leave") then toward ("Approach") opponent with no pause in ambulation"
Offensive hunch - walking with back hunched. Often includes wobbling shuffling movement
Upright offensive - "Bipedal stance with back hunched. Head and body oriented (and leaning) towards opponent. Eyes slitted and ears flattened"
Sideways offensive - "Body presented laterally to the opponent and rotated away from it. Head oriented towards opponent with eyes slitted and ears flattened. May shuffle around or sideways (towards) opponent"

Aggressive groom: "Vigorous tugging of opponent's fur, generally in the back or shoulder region," and mounting is often present. Grooming is differentiated from bite and biting attack by the absence of vocalization by opponent

Bite: A single "rapid leap, or darting of head and forebody, towards opponent, ending in mouth contact with opponent's body" with one corresponding vocalization by opponent

Biting Attack: Two or more bites in rapid succession accompanied by vocalization by opponent. May include chase of fleeing opponent and/or circling of testing chamber with no pause in ambulation before continuing the attack

Fight: "Animals roll around floor biting, kicking and wrestling, their bodies clasped tightly together"

Context codes for biting and biting attack :
A = Aggression/threat by target
I = Mild Investigation (instigation) by target. May include sniffing, nosing, social grooming
N = Target is Neutral - standing in corner away from Experimental, or exploring environment
E = Active Escape effort by target. May include defensive postures, fleeing, jumping, charging, withdrawing, vocalizations
note: C, I, A, and N supercede E as codes for first initiation of bites and biting attacks, as code is for indication of behavior that preceeded the unit of attack. If the attack and escape behavior continues into the next time segment, however, an E is coded.

Note: Quotations are from Jones' and Brain's (1985) coding descriptions.

Table 4

Reliability Ratings For Aggression Coding

Reliability Based on Product Moment Correlation.
"Code" reliability based on Agreement Score as "Code" has several possible values: Reliablity = Agreement/(Agreements + Disagreements)

	Intraobserver N=4 mice	Interobserver N=6 mice
MinorA	Psi = .90	Psi = .82
AGroom	Psi = .92	Psi = .81
Ag	Psi = .94	Psi = .82
Codes	27/27 = 1.00	9/13 = .77
Allag	Psi = .95	Psi = .81

Note: The "Codes" rated here is not the variable"Code" that is analyzed and graphed. "Codes" refers to the ratings of all the bites and biting attacks of the N subjects rated. "Code" refers only to the code of the first bite and the first biting attack for each subject. "Codes" was used for the reliability rating in order to rate a larger number of scores than "Code" would have provided.

CHAPTER 3

RESULTS

3.1 Glucose Analysis.

3.1.1 Glucose Data as Affected by External Sources Weeks 1, 6, and 9

Mean glucose values by diet and week are presented in Figure 1. One aspect of the data that stands out is the very low values week 1 and the dips weeks 6 and 9. What is striking about each of these weeks is that the dips are consistant among all treatment groups. This indicates that the variation was caused by an external source rather than being random fluctuation due to noise or lack of a steady progression of a process. Rather, except for those three weeks, the curves are fairly regular. Week 1 there were many methodological problems, and accordingly a lot of missing data. The problem of air dilution in glucose analysis was major in week 1. Although the samples mixed with the most air were not used, samples with less air were, deflating the "true" glucose values in all the groups.

Weeks 6 and 9 are different from week 1. As the dips weeks 6 and 9 both corresponded with a similar decrease in food consumption, the dips are unlikely to be a reflection of the blood testing experience or the glucose analysis. The dip is also unlikely to be fully explainable by the extent of missing data (week 6 N=13 for Suc, Carb, and Fat and 12 for Suc2; week 9 there is only one missing value). Week 6 the answer is clear: That is the week that the subjects were gavaged with water, and many responded poorly and consequently ate abnormally little. Although glucose analysis that week did not include samples from the subjects that responded the most poorly to the gavage, it did include subjects who had had minor reactions. Many of these subjects had eaten less than they did ordinarily. Corresponding to the abnormally low food intake were abnormally low blood glucose levels. Week 9

the explanation is not known. There was probably a third variable affecting both food intake and blood glucose levels, or more likely affecting food intake which in turn affected blood glucose levels. One likely possibility would be a considerable drop in room temperature that week.

3.1.2 Fasting Glucose Levels.

Repeated measures analysis of variance was conducted using all weeks except weeks 1, 6, and 9, as those three weeks artificially caused almost every possible curve to appear significant. The analysis showed significant diet differences ($p<.01$). The differences were in part due to the linear contrast ($p = .07$), but were primarily due to a strong week by diet interaction ($p<.01$). While Figure 1 shows the steady relationships between groups within each week, the time effect is difficult to see due to weeks 1, 6, and 9. By removing these weeks, a clearer picture of the trends emerges (see Figure 2). Although the slope of each group is essentially flat, there is a differentiation among the groups that increases over time. Figure 2 shows this gradual slight spreading, with Suc having the highest values and rising slightly, Suc2 with the next highest values, then Fat, and finally Carb with the lowest values, sloping slightly downwards over time.

Weekly analysis of variance comparisons among the 4 diet groups were made followed by a Scheffe test for each of the 12 weeks of fasting blood testing, and for weeks 13 and 14 as well. Bonferroni T tests were then also used in order to correct for the number of tests. The results are shown in Table 5. The first column of significance values for the differences between the weekly glucose levels are those obtained with 14 Scheffe tests. The second column of significance levels are in accordance with Bonferroni criteria:

For p< .01 the value had to be less than .01/14 = .0007

For p< .05 the value had to be less than .05/14 = .00357

For p< .10 the value had to be less than .10/14 =.007

The groups began the same even according to the less conservative Scheffe p values (see Figure 2). Week 1, the blood test measuring fasting glucose values before the diet manipulation began, showed no differences between any two groups. There continued to be no significant differences week 2, indicating that if there were effects of the dietary manipulation, the effects were not immediate. Differences began to emerge week 3 according to Scheffe p values and week 7 according to the more conservative Bonferonni p values, each showing the mean of Suc2 to be significantly greater than the mean of Carb. Beginning week 4 there were also significant differences between Suc and Carb. The mean differences were even greater than the differences between Suc2 and Carb, and the p values were correspondingly stronger and more consistant. Both sucrose groups, then, had after the first few weeks on the high sucrose diets significantly and consistantly higher mean glucose values than Carb. The differences between the sucrose groups and Fat were less pronounced. While the means of both groups were always greater than the mean of Fat after week 4 (except for week 10 when the mean of Fat barely exceeded the mean of Suc2 by .6 Mg/Dl), the differences were only at times significant. Suc began to be significantly different from Fat week 7 with Scheffe values of p<.05 weeks 7, 9, and 11 and p<.01 week 8. It was not until week 12 that there was a significant difference by Bonferroni criteria, and with a p value of only .1. Suc2 and Fat were never significantly different. Although the mean of Fat was always greater than the mean of Carb after week 2, Fat and Carb were never significantly different by Bonferroni

criteria, and were only different weeks 4 ($p<.05$), 7 ($p<.05$), and 11 ($p<.05$) by Scheffe. Suc and Suc2 were never significantly different by either criteria.

3.1.3 Glucose Levels Following Three Hour and Thirty Minute Sucrose Challenges.

Although weeks 13 and 14 tested somewhat different parameters than the other 12 weeks, all weeks (except 1, 6, and 9) are presented together in Figure 3. In this way group relationships can be most easily compared between fasting and challenge manipulations.

Only half of the data from week 13 were analyzed, as the remaining plasma was saved for possible future insulin analysis. Significant differences were found nevertheless, with the mean glucose values of the sucrose groups again significantly exceeding those of the other groups. In fact, in most cases in both weeks 13 and 14, the differences were even greater than those of the previous weeks. Again, Suc and Suc2 did not differ from each other, nor did Fat and Carb.

3.1.3.1 Week 13.

The mean glucose value for Suc week 13 was greater than that of Carb by 37.25 Mg/Dl, a difference meeting the Bonferroni criteria for significance at $p<.01$. The mean difference between Suc and Fat was significant ($p<.05$) by Bonferroni criteria, with a mean Suc value 29.62 Mg/Dl greater than that of Fat. Suc2's mean value were also greater than Carb's and Fat's, by 32.68 and 25.05 Mg/Dl respectively. The Bonferroni significance level of the first was $p<.01$. The difference between Suc2 and Fat was not significant by Bonferroni criteria. It was, however, significant ($p<.05$) using Scheffe criteria. By comparison, the mean difference value between Suc and Suc2 is a nonsignificant 4.57 Mg/Dl, and between Fat and Carb a nonsignificant 7.63 Mg/Dl. Besides that the group relationships held for the 3 hour challenge, the

mean differences between Suc2 and Fat and Suc 2 and Carb grew, thereby increasing the spread between the sucrose groups and the other groups.

3.1.3.2 Week 14.

Week 14 showed similar results as week 13, although the values were all elevated due to the more recent sucrose challenge. Also, the mean of Suc2 rose slightly more than the mean of Suc. Consequently, this time it was Suc2 that had a Bonferonni value of $p<.05$ in the comparison with Fat, and Suc that had a Scheffe value of $p<.05$ in the same comparison. Also, the Bonferonni p of the Suc-Carb difference was .05 rather than .01.

3.1.3.3 Comparison of Fasting and Peak Glucose Levels.

Blood glucose tests week 13 were taken at a time when most of the glucose ought to have been used up. The tests would therefore be likely to show only subtle differences from fasting levels. Blood glucose levels at a 30 minute challege, however, are quite different from fasting levels. In order to assess whether or not the peak values in this study paralleled the fasting values, repeated measures analysis of variance was conducted. The values of week 14 were compared to those of week 12, as week 12 was the most recent and therefore most appropriately comparable week from which fasting glucose values were obtained. Although the values of all 4 groups rose as expected from the sucrose challenge, Suc2 rose more than the other groups did. Comparing the mean differences of each group from week 12 to the levels produced by the 30 minute challenge, the following differences were found: Suc2 rose more sharply than the other groups did, with the result that Suc2 and Suc ended up with very similar values as each other (see Figure 4). Suc2's mean was 37.8 Mg/Dl higher than its mean week 12, compared with 16.5 Mg/Dl for Suc, 23.0 for Carb, and 15.4 for Fat. Repeated measures analysis of weeks 12 and 14 showed that this diet by week interaction was significant ($p<.05$).

3.2 Other Variables.

3.2.1 Mouse Weight.

Until week 5, there were no significant differences among the diet groups in terms of subject weight. From weeks 5-15, Suc was significantly heavier than Carb every week, according to weekly Scheffe tests, sometimes at $p<.05$ and sometimes at $p<.01$. No other groups were significantly different at the $p<.05$ level at any week. Over time, however, one can see a gradual and slight spreading of the groups with Suc the heaviest, then Suc2, then Fat, then Carb (see Figure 5).

3.2.2 Total Food Consumed.

Each week of data was analyzed by analysis of variance followed by a Tukey test. There were no significant differences in Kcal consumed among the 4 diet groups the first week that the mice were on the experimental diets (which was "week 2" of the study, as it was the food which preceeded the 2nd blood testing). It can therefore not be rejected that the groups started out the same. Throughout the study, there were no significant differences between the carbohydrate and fat groups, nor between the two sucrose groups. Except for weeks 3 and 7, neither sucrose group was significantly different from the carbohydrate group. Both sucrose groups, however, ate significantly more than the fat group during many of the weeks through week 10. Suc2 weighed significantly more than Fat week 13 as well. However, whereas Suc's significant differences from Fat usually corresponded with a p value of less than .01, Suc2 did only weeks 3 and 13.

3.2.3 Snacks Consumed.

Again, each week of data was analyzed with a Tukey test. The amount of snack consumed varied quite a bit among the groups, particularly between Suc2 and the other groups. Until week 10, the mean snack consumption of

Suc2 was consistantly quite a bit lower than that of the other three groups, and was lower than Suc and Fat throughout the study: Except for weeks 13 and 14 Suc2 ate significantly less snack than Suc every week, with $p<.01$ all but weeks 6, 7, and 12. Suc2 ate significantly less snack than Carb weeks 2-5, 8, and 9 with $p<.01$, and week7 with $p<.05$. Suc2 ate significantly less snack than Fat every week but 14, with a p value less than .01 all but weeks 7, 11, and 13. In addition, Fat ate less than Suc week 6 ($p<.01$) and less than Carb weeks 6 and 11 ($p<.o1$) and weeks 10 and 12 with $p<.05$. Except for week 11, Suc and Carb were never significantly different.

Although the mean snack consumption of Suc2 did gradually increase, two of the mice in that group continued through week 9 for one and 10 for the other to consistantly eat less than half of the snack. This contrasts with most of the animals in all the groups generally eating about 90% of their snacks. The diet manipulation of those 2 subjects, then, was more like that of Suc than that of Suc2. Neither of the two mice displayed any aggressive acts.

3.2.4 Sucrose Consumption in Suc and Suc2.

Until week 7 Suc2 on the average ate a lot less snack than Suc, and somewhat less most of the weeks of the study. They therefore ate slightly less sucrose than Suc, as sucrose in Suc2 was highly concentrated in the snack. There were no statistically significant differences in food consumption between Suc and Suc2. Suc2 ate the same or at times slightly more than Suc, with each gram of the Suc2 diet containing fewer Kcal of sucrose than in the Suc diet. The result of the similar food consumption and decreased snack consumption was that Suc2 generally ate slightly more than 1 Kcal less per day in sucrose than Suc did. Also, while the percentage of total diet consumed in the form of sucrose had to remain at a constant 45% in Suc, the percentage in Suc2 was actually approximately 37% throughout the study.

3.3 Aggression

3.3.1 Targets.

Weights of the target animals did not significantly differ among the groups. The mean of Suc was 20.66 (SD=.927), of Carb was 20.37 (SD=.81), of Fat was 20.29 (SD=.99), and of Suc2 was 20.37 (SD=.992).

3.3.2 Differences in Degree of Aggressive Behavior Across Groups.

Each aggression variable was analyzed with the Mann-Whitney rank sum test and the Kruskal-Wallis one way analysis of variance. These tested the differences in location of the two populations. By using rankings, an extreme case would not artificially inflate or deflate a difference value. It also eliminated the problem of unequal variances assumed in a parametric analysis of variance. However, the degree of aggressivity was an important consideration in this study, both for each subject and across groups. Analysis by rankings left out much of the richness of this information. In diet behavior research it is common for some subjects to have stronger reactions to the experimental manipulations than others, and these strong reactions are important to consider. In order to consider degree of aggressivity beyond that taken into account by ranking, parametric analysis of variance was also conducted: The results of these parametric analyses are presented in Appendices A, B, C, and D. Both measures yielded similar results, but with one primarily providing information on position and the other primarily on degree within that position. Both measures showed a consistant trend across measures in favor of higher aggression levels in Suc2 and Fat than in Suc and Carb, but the differences were not statistically significant.

3.3.3 Frequency Variables and "Code" (see Table 6 and Figure 6).

3.3.3.1 MinorA.

The minor aggresssive behaviors were lunges, tail rattles, offensive postures, and circles. Therefore, the higher the frequency value the higher the aggression as measured by this variable. The mean rank of Fat was the highest, followed by Suc2, Carb, and Suc (Suc rank x=422.0, Carb rank x=481.5; ; Suc2 rank x=552.5, Fat rank x=560.0; p=.45).

3.3.3.2 AGroom.

The rank for number of aggressive grooms of Fat exceeded that of the other groups but not significantly (Carb rank x=467.5; Suc rank x=491.5; Suc2 rank x=495.5; Fat rank x=561.5; p=.82).

3.3.3.3 Ag.

With bites and aggressive attacks; Suc2 was most aggressive; followed by Fat; then Suc; and then Carb. Again; however; the differences are not significant (Carb rank x=486.5; Suc rank x=496.5; Fat rank x=511.0; Suc2 rank x=522.0; p=.99).

3.3.3.4 Code.

Code took into account the immediate precipitants of the first bite and biting attack for each subject that exhibited biting behavior. It was determined before the testing began that a subject would be considered most aggressive if attacking a target who was trying to escape (Escape: Code E = 4), next most aggressive if attacking a target who was not interacting with him (Neutral: Code N = 3), less aggressive (more defensive) if the target was exhibiting investigatory, social behavior (Instigating: I = 2), and least aggressive if reacting to aggressive behavior by the target (Aggressing: A = 1). According to these criteria, a high value indicates higher aggression. The

mean rank of Suc2 was the highest (x=522.0) followed by Fat (x=511.0), Suc (x=496.5), and Carb (x=486.5). This difference was not statistically significant (p=.96).

3.3.3.5 Fight.

Fight is a variable that more than the others has a great deal to do with the target. It is in fact defined according to the target's behavior. It may or may not be defensive behavior depending on the situation. For example, athough 6 10-second intervals were recorded for "fight" for the mouse in Suc, it should be noted that previous to and during the first two fighting segments, 25 segments of the experimental animal's fleeing was recorded (against a mean of approximately 1.5 for "fleeing" segments). In addition, there were no biting attacks and only 1 bite by the experimental animal, and the bite was coded "A" indicating that it was in response to aggressive behavior by the target. In that N was only 1 in Suc, 0 in Suc2, and 2 in Carb and Fat, and in that the behavior had a defensive element that could not be separated out, a separate analysis on "fight" was not conducted. However, since the fights did occur, rather than escape behavior which in this study was a much more usual behavior, fight must be considered. It was therefore taken into account as part of several combination variables (Allag and all the time variables except TLAO1).

3.3.3.6 AllAg.

Allag (All Aggressions) was a combined variable to take all the aggressive acts with physical contact into account at the same time. In addition to the aggressive grooms, bites, and biting attacks already analyzed individually, it includes "Fight." As the aggression levels of most of the components of this variable favored Suc2 and Fat, that trend continued in this

new variable (Suc rank x=486.5; Carb rank x=451.0; Suc2 rank x=536.0; Fat rank x=542.5; p=.85).

3.3.4 Time Variables (see Table 7 and Figure 7).

3.3.4.1 TLAO1.

The longer the animals were together before the subject displayed any aggressive postures, the lower the aggression level. Suc2's latency was the lowest (x=466.0) followed by Suc (x=471.5), Carb (x=494.5), and Fat (x=584.0). Although the differences were not significant (p=.56), the mean rank of Fat was for the first time lower than those of Carb and Suc.

3.3.4.2 TAggr.

TAggr was the variable measuring the latency to the first aggressive act involving physical contact. Except for TAggr, the mean rank of Suc2 indicated a higher level of aggression than Suc and Carb for every variable. For TAggr, however, the ranks of both Suc and Carb indicated higher aggression levels than that of Suc2. Also, as with the time to the first offensive act, Fat again appeared to have the lowest aggression as measured by this variable. None of the differences were significant (Fat rank x=551.0; Suc2 rank x=515.0; Suc rank x=500.0, Carb rank x=450.0; p=.88).

3.3.4.3 TFABAF.

Unlike the first two time variables, the latency to the final aggressive act involving physical contact would be high for an aggressive animal. Suc2 and Fat showed a trend for higher aggression, followed by Suc and then Carb (Carb rank x=435.0; Suc rank x=489.0; Suc2 rank x=535.0; Fat rank x=557.0; p=.72).

3.3.4.4 AgTime.

The total time between the first and final physically aggressive behaviors would be high for an aggressive animal. The trend was again in

favor of Suc2 and Fat as the more aggressive groups followed by Suc and Carb (Carb rank x=451.5; Suc rank x=508.5; Fat rank x=522.5; Suc2 rank x=533.5; p=.94).

3.3.4.5 ETime.

Escape Time measured the time between the first escape attempt by the target and the last aggressive act that involved physical contact between the target and the subject. A high value for ETime could therefore be interpretted as indicating a higher aggression level. Again, Fat and Suc2 had the highest mean ranks, and Suc and Carb had the lowest (Carb rank x=404.0; Suc rank x=504.5; Suc2 rank x=531.5; Fat rank x=576.0; p=.42).

3.3.5 Summary.

No statistically significant differences were found among the diet groups for any of the aggression variables, with p values ranging from .42 to .99. While the differences were not significant, the relationships among the groups were quite consistent across variables. Except for the two variables measuring latency to the onset of aggressive behaviors (TLAO1 for Fat and TAggr for Suc2 and Fat), the mean ranks of both Suc2 and Fat were always in the direction indicative of a higher level of aggression than both Suc and Carb.

3.3.6 Subjects Displaying Aggressive Behavior.

Overall, there were no significant differences in aggression levels among the groups as a whole. However, it is quite common in diet research for the dietary manipulation to affect only some of the subjects, and for there to be a range of degree of response among those subjects. If such differences were present in this study, they would have been extremely difficult to detect from the analyses presented above. The difficulty would have arisen from the

fact that fewer than half of the subjects were aggressive.[1] Consequently, the high number of zero values for each variable considerably diluted the group means and caused enormous variances, thereby making significant findings extremely unlikely. For example, with the time variables, a normal value when existent was at times as high as 700 or 800. Zero values mixed with values this high created an enormous contrast. The variances were so large that, when using parametric analysis, even if there were differences among the groups, the possibility that the differences were due to chance would not be able to be rejected. Also, the means were reduced to values that were only minimally reflective of the meaning they stood for: For example, the largest difference between groups on a time variable was for TLAO1, the latency to the first lunge, rattle, circle, or offensive. Considering only the animals whose value for this variable was not zero, the mean of Suc was 494 seconds with a standard deviation of 258. For Suc2, the mean was 136 with a standard deviation of 98. These values of these means provide the actual amount of time that it took for aggressive behavior to occur, when it occurred: Animals in Suc, on the average, were in the compartment with the target for eight and a quarter minutes before displaying offensive behavior, if they displayed that behavior. In Suc2, it took on the average only two and a half minutes. The difference appeared substantial. Indeed, the p value was .0068. However,

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1 N was 16 in each group except Carb, in which it was 15. Of these, 10 of the subjects in Suc, 7 in Carb, 7 in Fat, and 8 in Suc2 (including the 2 who ate little snack - otherwise 6) displayed no aggressive behavior as defined by the measured criteria. In Suc, the only aggressive behavior of one of the remaining subjects was one segment in which "lunge" was coded, and one subject in Carb had only 1 "offensive" act coded as well. In isolation, the behaviors coded may not have been aggressive ones. In another of the "aggressive" Suc subjects, the aggression was defensive. Even calling those subjects aggressive left only 6/16 aggressive animals in Suc, 8/15 in Carb, 9/16 in Fat, and 8/14 in Suc2 if the 2 subjects who ate very little snack are exluded from the group.

analyzing data with all the subjects whether aggressive or not yielded less meaningful results. The mean of Suc was 154 with a standard deviation of 272! The mean of Suc2 was 59 with a standard deviation of 93. Yet the first offensive act certainly did not occur that quickly, and with variances larger than means statistical significance was improbable. The large standard deviation led to a p value of .20. In addition, this value had to be considered invalid because Levene's test for equality of variances failed. Using nonparametric measures Levine's test was not a problem, but the large mean differences were totally lost: Compared to rank differences of 46 for Suc and 32 for Suc2 and a p value of .028 when including only aggressive animals in the analysis, using all animals yielded a mean rank of 261.5 for Suc, 266.5 for Suc2, and a p of .91. Although the tests including all the subjects indicated that the populations were not different, it is also possible that such tests obscured real differences that the diet manipulation may have led to in some but not all of the subjects. For this reason, with the recognition that despite suggestive differences the groups as a whole were not statistically different on any variable, analysis was conducted on the aggression data using only non-zero values. The goal was to respond to the following question: Of the subjects who exhibited "X" behavior, did the subjects in one group do so more than those in another?

The results of nonparametric analysis using non-zero values is shown in Tables 8 and 9 and Figures 8 and 9. Parametric analysis results can be found in Appendices E, F, G, and H. The trends that appeared from the analysis of all the subjects did show up more strongly in this re-analysis, but the differences could still not be considered significant: The p value in the nonparametric analysis of MinorA was less than .05. However, due to the large number of comparisons, this could easily be explained by chance. In fact, the parametric

analysis of the same variable did not show significance (p=.08). The trend, however, was still apparent. The trend was notably strong for Code (parametric p=.08, nonparametric p=.11) and TLAO1 (parametric p=.11, nonparametric p=.07) as well. These analyses, then, found the same trend of higher aggression in Suc2 and Fat than in Suc and Carb as did the previous analyses, but even more powerfully.

Although the analysis using the non-zero values did not show significant differences, the trends did appear even more strongly than previously, thereby lending further support to the possibility that something subtle could be occurring and that further analyses could therefore be of interest. Rather than the sucrose groups clustering together as differentiated from Carb, as in the blood glucose portion of the study, Suc and Carb clustered together as differentiated from Suc2 in the aggression data. That Fat often fell in between accentuated the differentiation. In the blood glucose portion of the study the primary comparisons were between the glucose groups and Carb, as there was no reason to believe that the snack manipulation would affect fasting blood glucose levels. Fat was present as an imperfect control to help to clarify whether or not the 3 groups were very similar to each other despite any differences that might unfold. The sucrose groups were in fact different from Carb, although not in the hypothesized direction: The sucrose groups had higher rather than lower blood glucose than the other groups. While differences in aggression caused by the dietary components (as opposed to the snack manipulation) could still have occurred, they would not have been the result of the hypothesized low blood glucose in the sucrose groups. In fact, the sucrose groups did not differ from Carb in aggression. But for the aggression portion of the study, the snack manipulation caused an additional comparison to be of particular interest. Namely, were the subjects with the "protein-free"

snack different from those with the "balanced" snack? Might this snack manipulation have had an effect on aggression levels? It clearly did not cause a significant differentiation among groups as a whole, but could it have had a subtle effect manifesting itself only in the differences in trends? The trends from the previous analyses are in the direction that would be expected from such an effect. The most precise way to take a closer look at the possibility would be to compare the group with the "protein-free snack" (Suc2) with the group with the same diet as a whole but with the "balanced" snack (Suc). Once again, then, analysis on the non-zero values was conducted, but this time with only Suc2 and Suc.

Aggression levels of aggressive subjects in Suc2 were significantly higher than those of Suc as measured by 3 of the variables (see Tables 10 and 11 and Figures 10 and 11 for the result of the nonparametric analysis, and Appendices I, J, K, and L for the results of the parametric analysis). The variables for which the differences were not significant showed stronger trends for higher aggressiveness in Suc2 than did the previous analyses. The variables for which analysis yielded significant results were MinorA (Suc rank x=19.0, N=5; Suc2 rank x=59.0, N=7; $p<.05$), Code (Suc rank x=11.0, N with scores for bite and/or biting attack=4; Suc2 rank x=25.0, N with scores for bite and/or biting attack=4; $p<.05$), and TLAO1 (Suc rank x=46.0, N=5; Suc2 rank x=32.0, N=7; $p<.05$). Except for the variable ETime, Suc2 showed a trend for higher aggression for every other variable as well: AGroom (Suc rank x=16.5, N=4; Suc2 rank x=19.5, N=4; p=.66); Ag (Suc rank x=11.5, N=4; Suc2 rank x=24.5, N=4; p=.058); Allag (Suc rank x=25.0, N=5; Suc2 rank x=41.0, N=6; p=.36); TAggr (Suc rank x=35.0, N=5; Suc2 rank x=31.0, N=6; p=.36); TFABAF (Suc rank x=26.0, N=5; Suc2 rank x=40.0, N=6; p=.45); and AgTime (Suc rank x=25.0, N=5; Suc2 rank x=41.0, N=6; p=.36). ETime was the only variable that changed direction by

varying the statistical test: The parametric test yielded a barely higher aggression level for Suc (Suc2 x=470, Suc2 SD=340; Suc x=544, Suc SD=374), and the nonparametric test showed the reverse (Suc rank x=31, Suc2 rank x=35). P values of .7 and .85 further support the probable lack of difference with this variable.

3.3.7 Summary of Sub-Group Analysis.

The latency to and number of non-contact aggressive behaviors both showed that of the subjects who displayed these behaviors, the subjects in Suc2 were significantly more aggressive than those in Suc. They also initiated the first bite/biting attack without instigation significantly more frequently. For ETime the groups were the same. For every other variable, there was a strong but non-significant trend in which the value of the mean rank of Suc2 indicated a higher aggression level than did that of Suc. In sum, analysis comparing the aggressive subjects who had eaten the "protein-free" snack (Suc2) with the aggressive subjects who differed by having instead eaten a "balanced" snack (Suc) showed a consistent trend toward higher aggression in the "protein-free" snack group.

Table 5

Significance Levels of Mean Differences in Glucose Values by Week

Significance Level Determined by an ANOVA followed by a Scheffe Test for Each of the 14 Weeks
*** = .01 ** = .05 * = .1 blank = none

Overall Bonferroni Significance Level
+++ = .01 ++ = .05 + = .1 blank = none

	Suc-Carb	Suc-Fat	Suc-Suc2	Suc2-Carb	Suc2-Fat	Fat-Carb
w1	-24.129	-3.312	-32.000	7.871	28.688	-20.817
w2	9.688	16.375	11.938	-2.250	4.437	-6.687
w3	16.450	3.875	-5.125	21.575 **	9.000	12.575
w4	22.367 **	3.500	3.125	19.242 **	0.375	18.867 **
w5	18.833 *	13.062	0.687	18.146 *	12.375	5.771
w6	20.071	8.563	-6.875	26.946 **	15.438	11.508
w7	35.430 ***+++	18.000 **	5.875	29.555 ***+++	12.125	17.430 **
w8	25.663 ***+++	15.188 *	1.625	24.038 ***++	13.563	10.475
w9	24.642 ***++	19.125 **	5.062	19.580 **	14.063	5.517
w10	26.196 ***+++	12.563	13.125	13.071	-0.562	13.633
w11	36.846 ***+++	17.813 **	12.375	24.471 ** +++	5.438	19.033 ***
w12	39.258 ***+++	23.812 ***+	14.812	24.446 ***+	9.000	15.446
w13	37.250 ***+++	29.620 ***++	4.570	32.680 ***+++	25.050 **	7.630
w14	32.810 ***++	24.940 **	-6.460	39.270 ***+++	31.400 ***++	7.870

Table 6

Kruskal-Wallis One Way Analysis of Variance Test Results Using All Subjects in the Four Diet Groups: Frequency and Code Variables

			MinorA	AGroom	Ag	Code	AllAg
Mean	Rank	Suc	422.0	491.5	496.5	492.5	486.5
Mean	Rank	Carb	481.5	467.5	486.5	496.0	451.0
Mean	Rank	Fat	560.0	561.5	511.0	505.5	542.5
Mean	Rank	Suc2	552.5	495.5	522.0	522.0	536.0
p			0.45	0.82	0.99	0.96	0.85
X⇒A			Fat	Fat	Suc2	Suc2	Fat
next			Suc2	Suc2	Fat	Fat	Suc2
next			Carb	Suc	Suc	Carb	Carb
least	aggressive		Suc	Carb	Carb	Suc	Suc

Table 7

Kruskal-Wallis One Way Analysis of Variance Test Results Using All Subjects in the Four Diet Groups: Time Variables

	TLAO1	TAggr	TFABAF	AgTime	ETime
Mean Rank Suc	471.5	500.0	489.0	508.5	504.5
Mean Rank Carb	494.5	450.0	435.0	451.5	404.0
Mean Rank Fat	584.0	551.0	557.0	522.5	576.0
Mean Rank Suc2	466.0	515.0	535.0	533.5	531.5
p	0.56	0.88	0.72	0.94	0.42
X=>Aggr.	Suc2	Carb	Fat	Suc2	Fat
next	Suc	Suc	Suc2	Fat	Suc2
next	Carb	Suc2	Suc	Suc	Suc
least aggressive	Fat	Fat	Carb	Carb	Carb

Table 8

Kruskal-Wallis One Way Analysis of Variance Test Results Using All Subjects that Manifested the Aggressive Behaviors: Frequency and Code Variables

	MinorA	AGroom	Ag	Code	AllAg
Mean Rank Suc	59.5	45.5	20.5	16.5	50.5
Mean Rank Carb	87.0	44.0	34.5	44.0	56.0
Mean Rank Fat	131.5	51.0	35.0	29.5	66.5
Mean Rank Suc2	157.0	49.5	46.0	46.0	80.0
p	0.048	0.42	0.3	0.082	0.58
Kruskal-W.	7.9	2.79	3.67	6.71	1.98
X⇒A	Suc2	Fat	Suc2	Suc2	Suc2
next	Fat	Suc2	Fat	Carb	Fat
next	Carb	Suc	Carb	Fat	Carb
least aggressive	Suc	Carb	Suc	Suc	Suc

Table 9

Kruskal-Wallis One Way Analysis of Variance Test Results Using All Subjects that Manifested the Aggressive Behaviors: Time Variables

	TLAO1	TAggr	TFABAF	AgTime	ETime
Mean Rank Suc	98.5	64.0	53.0	41.0	58.0
Mean Rank Carb	105.5	55.0	40.0	28.0	20.0
Mean Rank Fat	143.0	75.0	81.0	55.0	88.5
Mean Rank Suc2	59.0	59.0	79.0	66.0	64.5
p	0.11	0.76	0.86	0.33	0.57
Kruskal-W.	6.11	1.18	0.76	3.43	2.01
X=>Aggr.	Suc2	Carb	Fat	Suc2	Fat
next	Suc	Suc2	Suc2	Fat	Suc2
next	Carb	Suc	Suc	Suc	Suc
least aggressive	Fat	Fat	Carb	Carb	Carb

Table 10

Kruskal-Wallis One Way Analysis of Variance Test Results
Using All Subjects from Suc and Suc2
that Manifested the Aggressive Behaviors:
Frequency and Code Variables

	MinorA	AGroom	Ag	Code	AllAg
Mean Rank Suc	19.0	16.5	11.5	11.0	25.0
Mean Rank Suc2	59.0	19.5	24.5	25.0	41.0
N Suc	5	4	4	4	5
N Suc2	7	4	4	4	6
p	0.028	0.66	0.058	0.037	0.36
X⇨A	Suc2	Suc2	Suc2	Suc2	Suc2

Table 11

Kruskal-Wallis One Way Analysis of Variance Test Results
Using all Subjects from Suc and Suc2
That Manifested the Aggressive Behaviors:
Time Variables

	TLAO1	TAggr	TFABAF	AgTime	ETime
Mean Rank Suc	46.0	35.0	26.0	25.0	31.0
Mean Rank Suc2	32.0	31.0	40.0	41.0	35.0
ND1	5	5	5	5	5
ND4	7	6	6	6	6
p	0.028	0.36	0.45	0.36	0.85
X=>Aggr.	Suc2	Suc2	Suc2	Suc2	Suc2

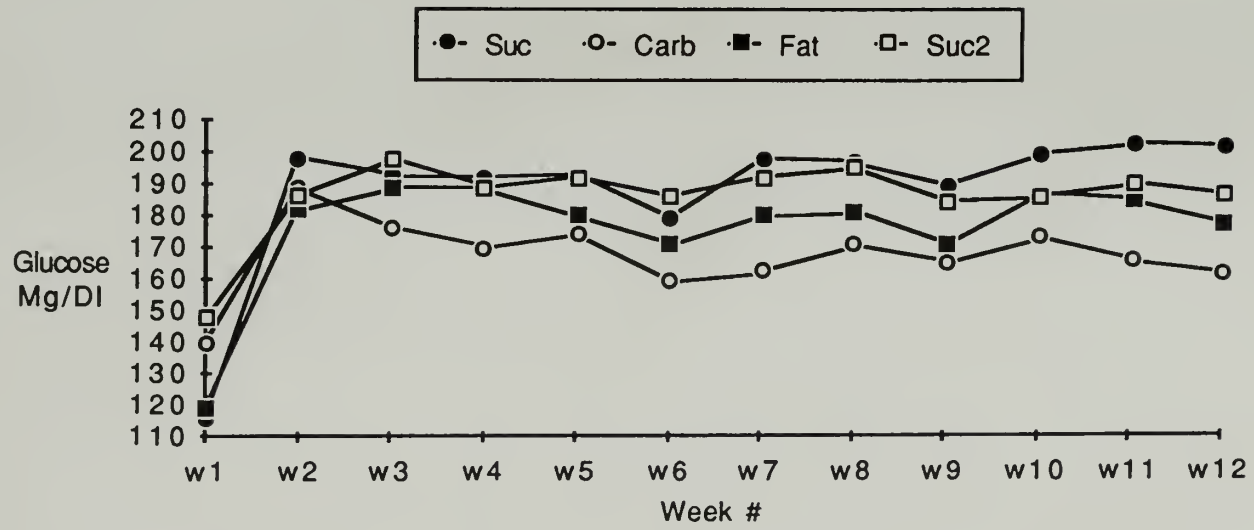

Figure 1

Mean Glucose Values by Diet and Week

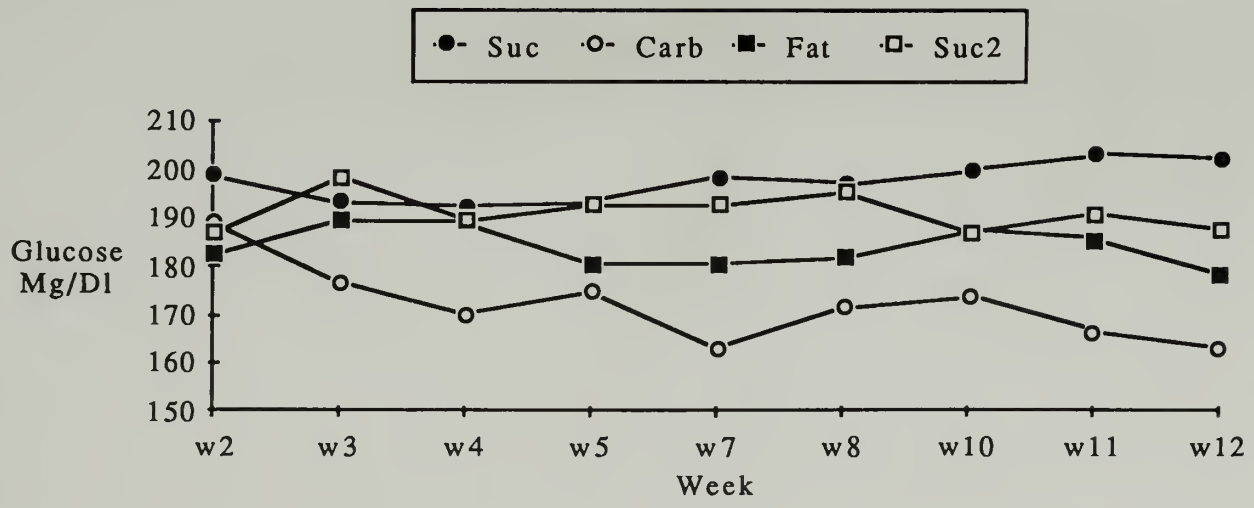

Figure 2

Mean Glucose Values by Diet:
Weeks 1-12 Without Weeks 1, 6, or 9

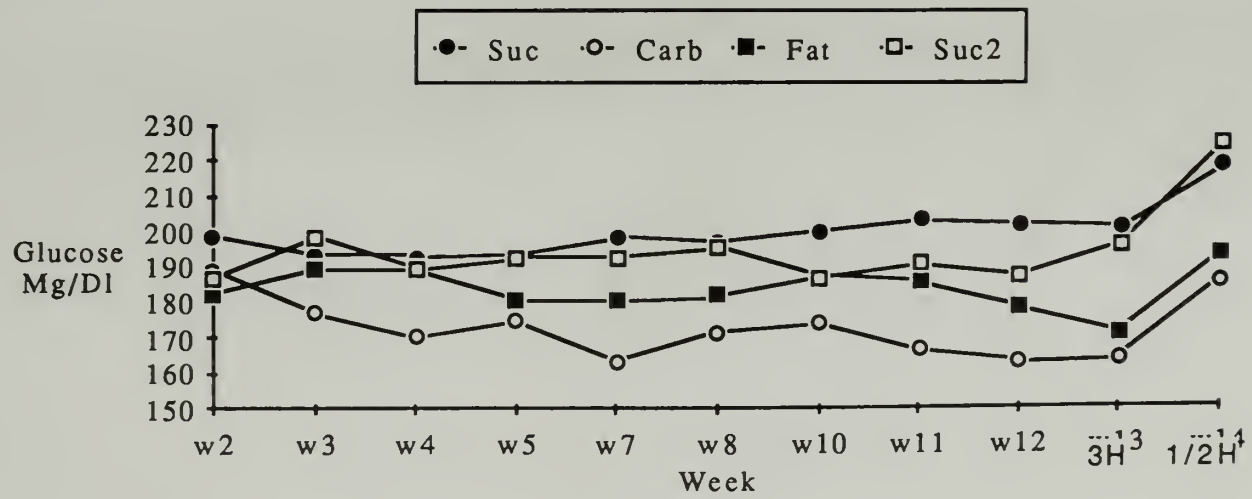

Figure 3

Mean Glucose Values by Diet:
Weeks 1-14 Without Weeks 1, 6, or 9

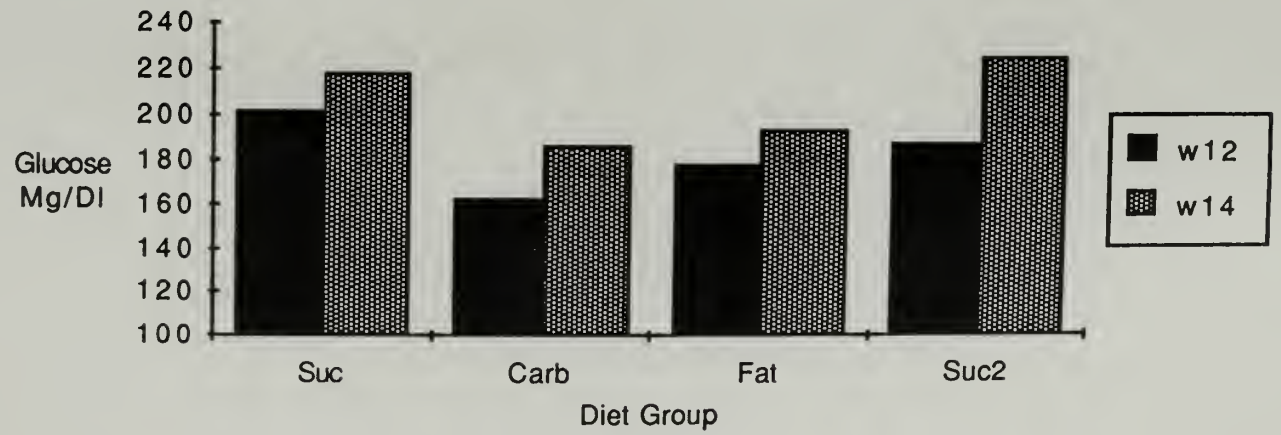

Figure 4

Mean Glucose Values by Diet:
Final Fasting Glucose (Week 12) Compared
with 30 Minute Sucrose Challenge (Week 14)

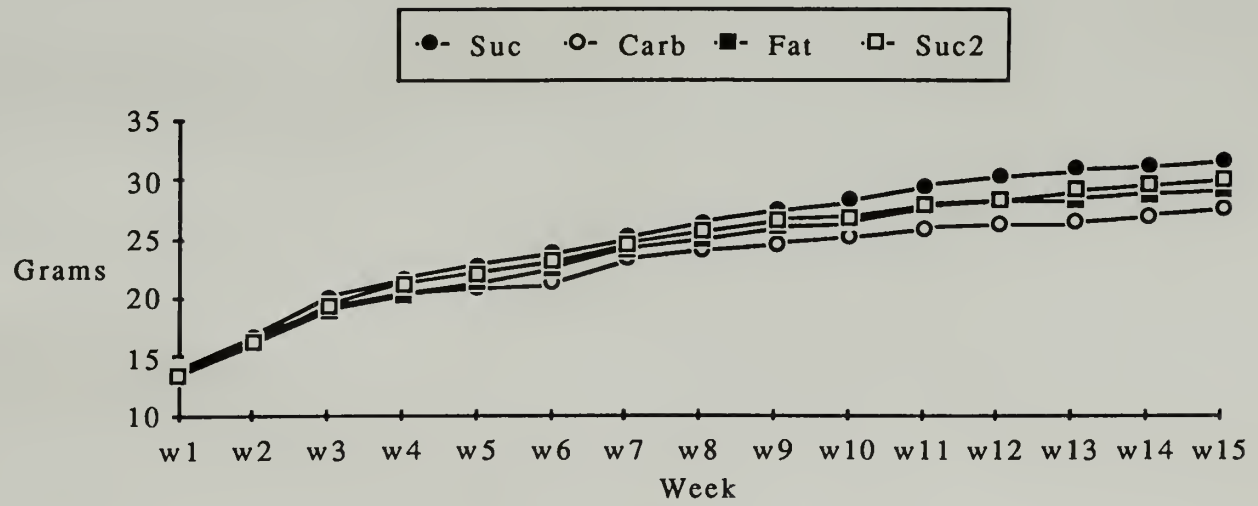

Figure 5

Mean Mouse Weight in Grams
by Group and Week

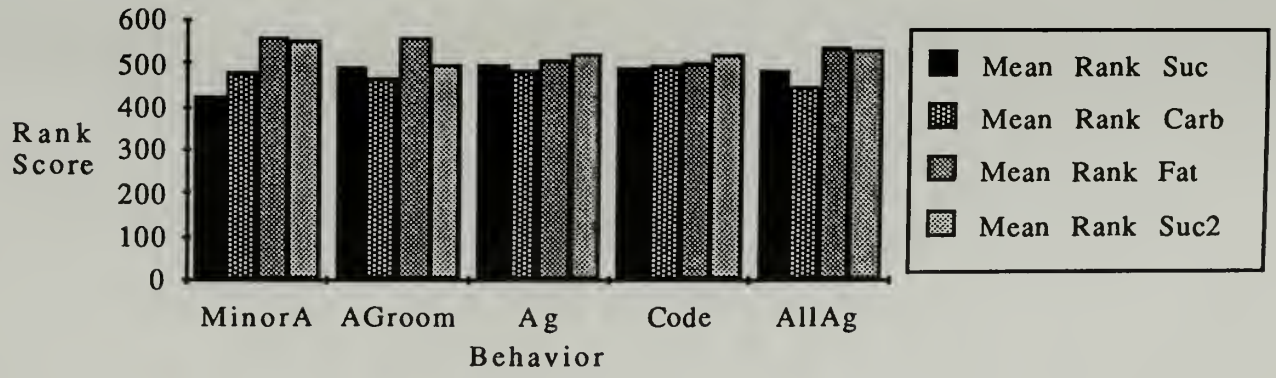

Figure 6

Kruskal-Wallis One Way Analysis of Variance Test Results Using All Subjects in the Four Diet Groups: Frequency and Code Variables

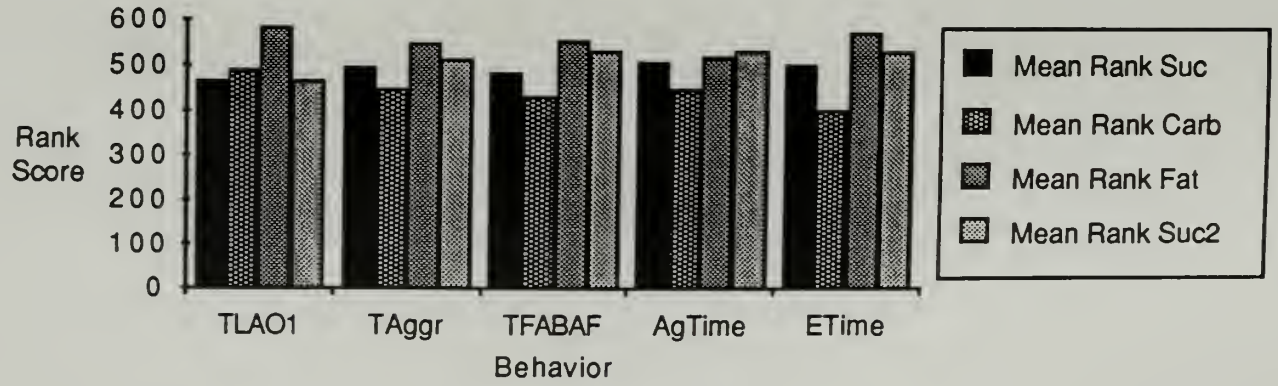

Figure 7

Kruskal-Wallis One Way Analysis of Variance Test Results Using All Subjects in the Four Diet Groups: Time Variables

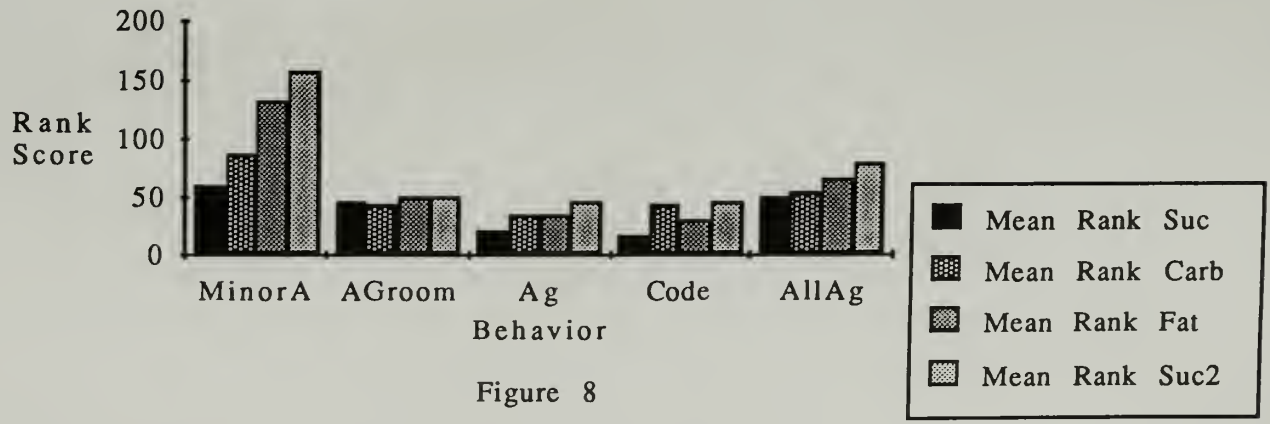

Figure 8

Kruskal-Wallis One Way Analysis of Variance Test Results Using All Subjects That Manifested the Aggressive Behaviors: Frequency and Code Variables

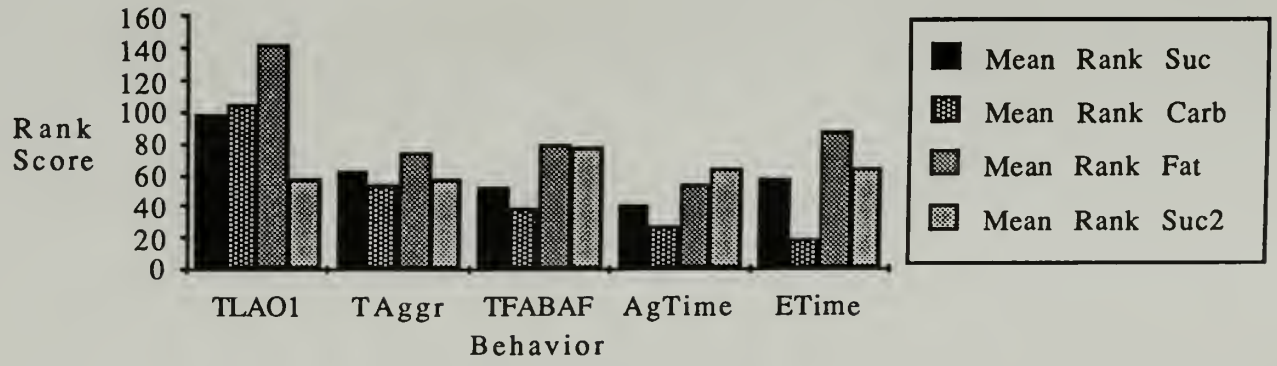

Figure 9

Kruskal-Wallis One Way Analysis of Variance Test Results Using All Subjects that Manifested the Aggressive Behaviors: Time Variables

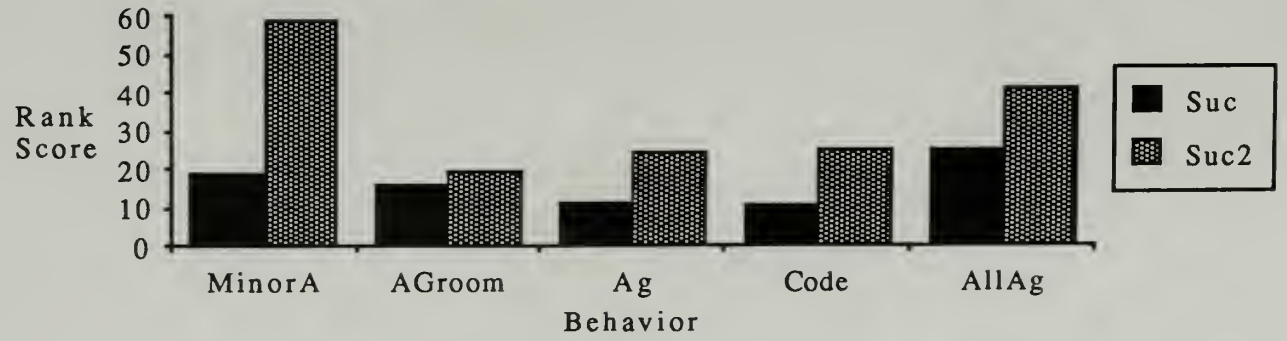

Figure 10

Kruskal-Wallis One Way Analysis of Variance Test Results Using All Subjects from Suc and Suc2 that Manifested the Aggressive Behaviors: Frequency and Code Variables

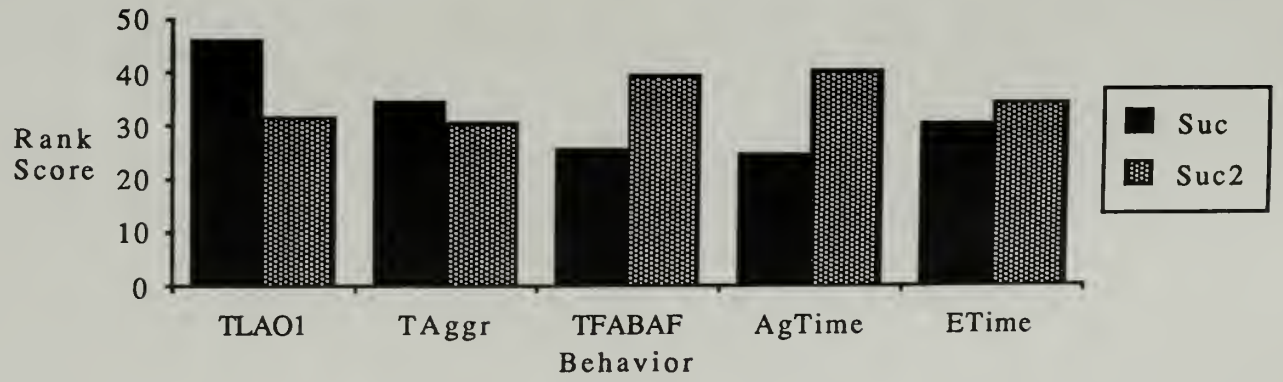

Figure 11

Kruskal-Wallis One Way Analysis of Variance Test Results Using All Subjects from Suc and Suc2 that Manifested the Aggressive Behaviors: Time Variables

CHAPTER 4

DISCUSSION

4.1 Sucrose Versus Other Carbohydrates, and Blood Glucose Levels.

4.1.1 Fasting Glucose Levels.

It is logical that Suc and Suc2 were never significantly different from each other in terms of blood glucose levels, as the subjects in the two groups ate essentially the same food. Except for the slight difference in sucrose consumed and the timing of which parts of their food was provided when, the groups were treated the same. That these two groups did yield the same results as each other increases the confidence with which one can say that the groups were under essentially the same conditions as each other in terms of factors that could affect blood glucose levels. No unknown conditions that may have differed between groups were great enough in any case to result in differing mean glucose values. Why, then, did the sucrose groups have higher mean glucose values than the carbohydrate group, which was also treated the same? All three groups had the same amount of carbohydrate in their diets. The only difference was the form of the carbohydrate. If what was important in producing high glucose values in this portion of the study was the high level of carbohydrate, the three carbohydrate groups should not have significantly differed from one another. That they did differ is strong support for the explanation that the high glucose levels in the sucrose levels was due to sucrose as differentiated from carbohydrates. The consisent trend of Suc's mean value slightly exceeding Suc2's lends support to the hypothesis that it was the sucrose in the diet that caused both to have high values, as Suc consistently ate slightly more sucrose than Suc2 did. Further support comes from the data from the high fat group, although this support is less conclusive

due to the complications that fat itself brings to such a study. That the mean of Fat fell consistently lower than that of the sucrose groups yet consistently higher than that of the carbohydrate group, rather than lower than it too, suggests again that the groups were different. Finally, the gradual spreading of the means of the groups indicates a gradually increasing differentiation arising among them. In conclusion, there is evidence to support the hypothesis that the dietary manipulations separated the original population into three different groups in terms of blood sugar levels, one made up of Suc and Suc2, one of Fat, and a third of Carb. This differentiation among groups grew greater over time, indicating the presence of long-term effects. One possible partial explanation for the differences between sucrose and carbohydrate could be that perhaps sucrose gets into the system much more quickly than a complex carbohydrate, if the complex carbohydrate takes longer to break down and therefore enters the system more gradually. Having to frequently deal rapidly with high sucrose loads could result over time in changes in the system. One example of such a possible change could be reduced insulin binding to erythrocytes (Reiser et al., 1986).

4.1.2 Glucose Levels Following Three Hour and Thirty Minute Sucrose Challenges.

The general relationships among the groups remained the same for the sucrose challenges as for the fasting glucose levels. However, there was also some evidence that the sucrose challenge affected the sucrose groups slightly differently than the other two groups: the sucrose groups clustered together and the other two groups clustered together, with a large spread in between.

4.1.2.1 Three Hour Challenge.

Week 13, the differences are not different enough compared to the other weeks to permit the drawing of any conclusions. Two explanations are

possible. The first is that the groups may not have been different. The second possibility is that only one week of data and only half of the subjects did not provide a sufficient test for finding significant differences for a 3 hour challenge. This could have been particularly true in that by 3 hours after the challenge most if not all of the glucose was used up, as indicated by the lack of elevation in all of groups except for Suc2. Blood glucose levels should therefore not have been very different from fasting levels so that significant differences between the weeks would be unlikely. In light of this unlikelihood, the hint of differences raises questions. One might speculate, for example, that perhaps the further differentiation of the sucrose groups from the other groups due to Suc2's rise, Suc's already high value, and Fat's fall in the 3 hour challenge was in fact a remnant of a greater difference that might have been caught had the testing been done earlier. Such a case would indicate metabolic differences, with a particularly long time needed for blood glucose levels to return to normal after the sucrose challenge, at least in Suc2. A conclusion such as this can certainly not be drawn with only the current evidence. However, the data do suggest that further exploration could be worthwhile.

4.1.2.2 Thirty Minute Challenge.

The 30 minute challenge was conducted primarily to check for diabetic or near diabetic reactions. The glucose peak of a diabetic animal would be higher than that of other animals due to the diabetic animal's inability to deal with the sucrose as quickly as others can. A significant diet by week interaction was found, indicating that at least one group's glucose values were more different from the other groups at peak than at fasting blood testing. One explanation could be that a few of the subjects in Suc2 and not in the other groups had diabetic reactions, causing the mean of its group to rise more than

the mean of the other groups. However, most of the subjects in both Suc and Suc2 were elevated at a similar level, strengthening the likelihood of a different possibility: Maybe a number of subjects in those two groups had some degree of glucose intolerance but not necessarily to the level of having a full blown diabetic response. The individual glucose values do indicate a wide but elevated range. A repeat of the 30 minute test with both sucrose groups again having the same mean values would have lent even further support to this possibility.

In any case, the results of the 30 minute challenge showed that the groups did respond to the sucrose challenge more differently from each other than they did for fasting values, indicating that in some way the groups' glucose metabolisms differed. To get a better sense of the nature of the differences it would be interesting to do additional tests at times between 30 minutes and 3 hours, to measure values for more than 1 week at various phases of the cycle, and to analyze insulin values along with glucose values.

4.1.3 Food Consumption Versus Glucose Curves.

The food consumption curves are similar to the glucose level curves: Both begin with a lack of differentiation, become more differentiated over time, and have low values weeks 1, 6, and 9. Except that Carb ate more than Fat while having a lower glucose value than Fat,[2] the relationships among the groups are comparable as well. There is therefore a strong direct or indirect relationship between glucose levels and food consumption. However, other factors show that neither variable fully explains the other. For example, the

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2 It is possible that Carb did not actually eat more than Fat: There appeared to be a small amount of food spillage by many of the subjects in Carb, probably due to the powdery consistency of Carb's food. The spilled food may or may not have been eaten in its entirety.

relationship between Fat and Carb is different in the two graphs. This factor is made more complex, though, by the interesting fact that Carb weighs less than Fat despite eating more. In fact, the body weight relationships among the groups is the same as those of the glucose levels. Animal weight, food consumption, and blood glucose levels are clearly related. However, the relationships are far from perfect. The glucose curves are different in several ways from the others, the most notable way being that the spread of the glucose curves is larger than the spread of either of the other curves. This indicates that something besides food consumption and body weight is impacting blood glucose levels. That something, which most likely is the diet manipulation, may be impacting food intake and body weight as well.

4.1.4 Long-term effects.

The spread of the blood glucose curves of the groups over time shows that the diets had long-term differential effects on the blood glucose metabolism of the subjects. Since all the curves were almost flat, though, one cannot determine conclusively from this study that the spread was due only to the sucrose diets rather than to the carbohydrate and fat diets, or to the level of food consumption in general. However, the escalated differences weeks 13 and 14 in combination with the response by Suc2 to the 30 minute challenge strongly suggest that the effects were in fact due at least largely to the sucrose manipulation. Sucrose challenges early in addition to late in the study would have further clarified this issue, as well as the question of how much time on the diets was needed before the abnormal responses to the challenge would manifest themselves.

4.2 Sucrose Versus Other Carbohydrates, and Aggression.

This study did not find long-term effects of a diet high in sucrose to be low blood glucose levels. On the contrary, values were higher than those of subjects on other diets. Consequently, it makes sense that the study did not find the hypothesized low blood glucose levels leading to higher aggression in the sucrose groups than in the other groups. In fact, the sucrose groups did not differ at all from the other groups in aggression level. The conclusion one can draw from the current study, then, is that chronically high levels of sucrose consumption do not result in a change in level of aggressivity.

Although this study showed no behavioral effects of the sucrose as compared to the carbohydrate or high fat diets, there is reason to speculate about whether such a difference might have been found had the aggression testing been conducted after a fast:

4.2.1 Chronic High Sucrose Diet and Hypoglycemia.

The glucose values of the mice on the high sucrose diets were consistently higher than those of the mice in the other groups even after an eight hour fast. An implication is that a diet consistently high in sucrose may lead to consistently high blood glucose levels. Since conducting the study I have learned that it has been shown that even when blood glucose levels are normal, or even high, brain glucose levels may be very low (Thurston, 1976). Studies (Gjedde and Crone, 1981; McCall et al., 1982) have shown that chronic hyperglycemia leads to a substantial reduction of glucose transport into the brain, possibly due to a decrease in the availability of hexose carrier molecules at the blood-brain barrier. This may function as a protective mechanism, keeping brain glucose levels stable despite high peripheral levels. However, an individual who is chronically in a state of hyperglycemia, perhaps due to a chronic high-sucrose diet, may be particularly susceptible to hypoglycemia:

If sucrose consumption is decreased to a below normal or even normal level due to a change in diet or to a fast, too little glucose may be carried into the brain. Therefore, if a long-term high sucrose diet leads to hyperglycemia, it is possible that an individual on such a diet could at times have abnormally low brain glucose levels concurrently with normal or high blood glucose levels. In such a case, perhaps higher aggressivity would result. It would be interesting to replicate the present study but with the aggression testing following a period of fasting, as that is when brain glucose levels would have most likely been low if the above mechanism was operational.

4.3 Carbohydrate Snack and Aggression.

Aggression is a term with so many definitions that no one study can explore all of them. This study did not seek to. It looked at a natural interaction (rather than aggression in response to shock, for example) between two animals of the same species in the territory of neither animal. Aggression by the subject would be likely to occur at least at times due to its having lived in isolation, and it would be more likely to occur than aggression by the target due both to the isolation of the subject and also to the differences in age and corresponding body weight. The variables chosen as measures of aggression, while certainly only measuring certain aspects of aggression, and being particularly limited in information about target behavior, did take target behavior into account to some extent and did cover several different aspects of aggressive behavior. Various behaviors were considered along a range from aggressive postures to aggressive grooms to biting and fighting. These behaviors were defined as aggressive by Jones and Brain (1985) as determined by sequence analysis that did consider target behavior. The discontinuous time measure took into account the fact that aggressive

behaviors are not discrete but continuous, with the beginning and ending difficult if not impossible to define. By using small intervals, long periods of behaviors would be differentiated from shorter ones. The use of intervals over time also allowed for the collection of latency and time variables, further filling out the picture of aggressivity of the subjects in the treatment groups. At least in terms of the variables measured in this study, this picture did not show statistically significant differences among the four groups. However, when pinpointing only the animals that displayed aggressive behavior in the two groups that most precisely tested for snack effects, there was a strong trend of higher aggression by the group that had a daily protein-free snack than by the comparable group whose snack was balanced. The implication is that a long-term diet including daily periods of carbohydrate consumption in the absence of protein may lead to higher levels of aggression by some individual responders, although not in the population at large. However, with such a small N and with significant differences found only with analysis conducted on non-zero values, it is too early to conclude that the differences between these sub-groups were in fact due to a snack effect. This is particularly true because the overlap of many of the variables could account for some of the consistancy of the trend. If the groups did not begin equal in terms of aggressiveness, the current results could have occurred. With such a small number of aggressive animals on which to base conclusions, a slight difference in initial aggressiveness would be powerful. However, there is no reason to believe that the randomly distributed groups were different from each other before the manipulation. Replication with a larger N could help to clarify whether the trends were a result of the manipulation, or whether they were an artifact of some other factor such as initial group differences. A within-subjects design would be an even better test of the "responders"

hypothesis, but the additional complication of carry over effects from one testing to the next would need to be taken into account.

4.3.1 Other Factors to Consider.

4.3.1.1 Target Differences.

Perhaps the aggression differences between the two sub-groups can be explained by differences between the corresponding target groups. The random distribution of the targets and their almost identical mean group body weights make that an unlikely, but still possible, explanation. It is certainly an explanation that warrents further discussion, as each test was an interaction between both animals. If target behavior varied, subject response variation would logically follow. For example if targets in Suc2 initiated earlier and more extensive investigatory behavior, it would have likely instigated earlier and more escalated reactions from the heretofore isolated mice (Cairns & Nakelski, 1971). However, the scores of the variable "Code" indicated that if there were differences between the target groups, they may have favored aggression in Suc rather than in Suc2. The higher Suc2 "Code" score says that "neutral" or "escape" behavior by targets immediately preceeded each subject's first bites and biting attacks more than did "aggressive" or "investigatory" behavior, with the reverse true for Suc. Even in the event that the "escape" code actually functioned as an instigation to aggressive behavior, Suc2 would have still come out as more aggressive - In all but 1 of its first bites or biting attacks, the code was "Neutral." While this score does not account for the full range of behaviors preceeding the first attack, it at least suggests that if there were differences between target behavior preceeding the subjects's initial attacks, that the differences were in favor of less rather than more response in Suc2. Whether this shows a difference between targets, or whether it shows more self-instigated aggressive behavior

by Suc2, Suc2 comes out as more aggressive than Suc. The possibility does still remain, however, that escape behavior after the initial attacks, may, like investigatory behavior, have caused an escalation in the subjects' behavior. Full sequence analysis would help to clarify what role the targets played in promoting the subjects' behaviors in the two groups.

4.3.1.2 Diet Differences.

Although the diets of the two groups were essentially the same, there were slight differences due to the lower snack consumption of Suc2 and the correspondingly slightly higher protein/carbohydrate ratio of its diet. While these differences cannot be absolutely ruled out as an alternative explanation for the differences in aggression, the possibility of these slight differences having had such a strong effect is highly unlikely. Studies looking at effects of varying carbohydrate protein ratios have found that greater differences than these were needed.

4.3.1.3 Brain Serotonin.

The possibility exists that the snack manipulation did affect aggression levels but not due to serotonin levels changing. To determine whether or not a change in serotonin levels occurred, serotonin would need to be measured directly. It would have been of interest to have had actual direct measures of brain serotonin levels upon sacrifice. However, as experiences with aggression affect serotonin levels (Eleftheriou & Church, 1968), the aggression testing would have confounded any analysis of serotonin levels which would have followed. Such analysis was therefore not conducted in the present study. However, considering the trend of increased aggression in Suc2, and particlarly if those results hold up under replication with a larger N, it would be worthwhile to conduct a long-term study with the diet groups of Suc and Suc2 followed directly by serotonin analysis.

4.4 Summary of Aggression Results.

Due to the lack of significant differences found in the aggression portion of this study, it cannot be concluded that the aggressive animals were aggressive for any reason but chance. Comparing sucrose to other carbohydrate and a high fat diet showed no indication of behavioral differences. Comparing carbohydrate (in the form of sucrose) eaten in isolation from protein, to carbohydrate eaten concurrently with protein, again showed no differences in level of aggression. However, the data are not inconsistent with the hypothesis that the effect of chronic protein-free snack consumption is increased aggression for some individuals. Trends in the data give cause for speculation that perhaps some mice in Suc2 responded to the snack manipulation whereas others did not. If only a small percentage of the mice were responders, than each group as a whole would not have been affected enough to result in significant group differences. Standard analysis does not take such possibilities into account. The hypothesis would best be tested with a within-subject design using those subjects whom one has reason to believe would be most likely to respond. While such a pre-selection would not be feasible with mice, it could be done with humans.

4.5 Clinical Implications.

Whereas in this study, due to its design, one can only speculate as to whether the higher aggressiveness found when comparing the non-zero values of Suc2 to those of Suc was due to outliers or to responders, it is important in general to take into consideration the possibility of individual responders in diet behavior research. Accordingly, it makes sense to consider the use of "responders" or even of "possible responders" (as in the current study) rather than all subjects for one level of analysis in such research. It is

not uncommon for different subjects to have widely varying reactions to dietary manipulations. A few may have very strong reactions while others will have weak ones and some will have no reactions at all. Treating the potentially strong responders as outliers and the non-responders as evidence for lack of effect is often not appropriate in this type of research (King, 1981). Much information is lost. Likewise, knowledge from diet behavior research can generally not be applied to the population at large. Rather, it may be helpful to some particular individuals.

Clinical ecologists are discovering that certain foods can evoke reactions in particular individuals, some reactions so strong as to even mimic mental illness. Again, when tested with the short-term acute food challenge, only a tiny percentage of people respond. Some of these responders have emotional while others have physical complaints, and many of them have both. Knowledge about the possibility of such responses to the food challenges results in the possibility that these people, though few, can be helped when they otherwise could not have been. Also, other people may be affected in milder ways that cannot be detected in the kinds of studies that have been done so far. The fact that *some* people *have* been found to respond gives credence to the connection between food and emotional as well as physical well being. Some people who seek help from medical and mental health professionals may in fact be suffering in part from an unidentified food or environmental contaminant. If so, these causes need to be acknowledged, identified, and removed. Then, if some symptoms remain, they can be treated appropriately.

The current study was conducted with mice, and therefore the results cannot be assumed to be directly applicable to humans. First, one must replicate the results through direct human testing. The findings of this study indicate that a parallel human study could be quite interesting. If similar

results are found, the current tightly controlled study would support that the results of the human study were due to the dietary manipulation rather than to the confounding factors inherent in human work.

If the findings of the current research can be replicated with humans, there will be important implications. First, in the current study, it appears that diets chronically high in sucrose rather than complex carbohydrates lead to elevated blood glucose levels. In addition, such diets may impact glucose metabolism. If there is a similar effect on humans, and if the degree of the effect is substantial enough to be potentially harmful, then considering the extremely high intake of sucrose in the United States, particularly by children, it would seem warrented to work towards greater substitution of sucrose with more complex carbohydrates in the American diet.

Second, this study provides no evidence for the hypothesis that sucrose per se has any effect on aggression level. In fact, if differences had occurred, one might speculate that they would have been in the opposite direction from that hypothesized: Since the sucrose groups had higher blood glucose levels than the other groups, perhaps diets high in sucrose slightly protect from the low blood glucose levels that correlate so highly with aggression. If such was occurring, however, the effects would have been extremely subtle and not true of the population as a whole, as no differences were found. Much has been written about the possibility of reducing aggressive behavior through dietary sucrose reduction. This study indicates that such measures are not warrented.

Third, Suc2 provided an opportunity to consider a diet in which carbohydrates, including sucrose, are consumed in the absence of protein. In doing so it presents a similarity to a diet typical of human beings in the United States - for example a donut for breakfast on rushed mornings with no protein

until later in the day. Although the results of the study are not sufficient to conclude that there is an effect, the results do point to the possibility that the long-term result of such a behavior in certain individuals could be higher aggression levels. This tentative finding compliments the correlational study by Prinz et al. (1980): Diets chronically high in sucrose and diets with a chronically high carbohydrate-protein ratio correlated with greater destructive-aggressive and restless behaviors in hyperactive boys. At first look the current study seems to contradict the finding of high sucrose levels being correlated with aggressivity. But what if it was not the sucrose or the high carbohydrate-protein ratio per se that made the difference in the Prinz et al. study? What if both of those factors correlated highly with a third factor? Both variables could quite easily be correlated with the amount and frequency of sucrose or other carbohydrate eaten in the absence of protein. Prinz et al. do not report on how much of the sucrose and other carbohydrates were consumed in isolation. As it is typical in children's high sucrose diets that some is consumed as soda or candy, it is likely that protein-free snacks were not uncommon.

In that Prinz's study was correlational, one could not be certain about the direction of causality. Perhaps the diet directly caused the behavioral problems. More likely, perhaps the diet led to behavioral problems in these particular boys due to an already present susceptibility to the triggering of such behaviors in these particular boys. Such an explanation might explain the results in Suc2 as well, particularly in that the mice were not free to design there own diets so that the direction of causality is more conclusive: If there was an effect, it was most likely due to the diet, yet the diet did not affect all of the subjects. But in the Prinz et al. study there is another possible explanation for the results. The boys, all of whom had behavioral problems,

could have self-selected their food for reasons related to their aggressiveness. Perhaps this selection was of sucrose or other carbohydrate products in isolation. If so, could it be that the boys in Prinz et al.'s study made such a selection to fulfill a physiological need?

If a diet like that of Suc2 can lead to higher aggression levels in some responders, one possible explanation could be a decrease in serotonin levels. Research (Rosenthal & Heffernan, 1986; Spring et al., 1987) has shown several populations of humans who crave carbohydrates, and the researchers attribute this craving to low serotonin levels. So far the research shows that these people include such populations as drug addicts attempting to quit, obese, and people with seasonal affective disorder. These people, it seems, eat large quantities of carbohydrate in the absence of protein as a way of self-medicating, as perhaps did the boys in the Prinz et al. study. If in fact short-term effects lead to the desired increase in serotonin, but long-term effects are the opposite, this craving can be understood: Serotonin levels decline and then carbohydrate is consumed in isolation to increase the levels. The increase is successful, but if part of a long-term pattern of such consumption, the initial increase will be followed by an even greater decline. This in turn will signal, through craving, the need for more carbohydrate in isolation, and the cycle continues. If this cycle proves through further research to be the case, clinical implications will be far reaching.

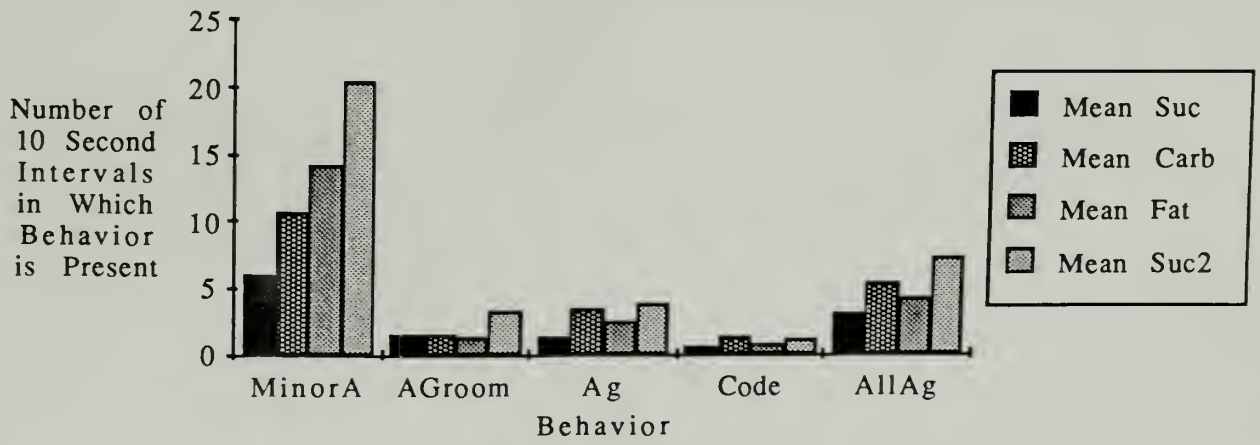
Appendix A

Mean Frequencies of Aggressive Behaviors Using All Subjects in the Four Diet Groups

	MinorA	AGroom	Ag	Code	AllAg
Mean Suc	6.1	1.5	1.3	0.6	3.1
Mean Carb	10.7	1.5	3.5	1.3	5.5
Mean Fat	14.3	1.3	2.6	0.75	4.4
Mean Suc2	20.5	3.4	3.9	1.2	7.4
SD Suc	11.7	3.3	3.4	1.1	5.7
SD Carb	19.5	3.1	9.4	2.4	13
SD Fat	19.2	1.9	6.2	1.5	8.3
SD Suc2	28.8	9.9	7.6	2.2	14.6
p	0.26	0.66	0.72	0.67	0.73
F	1.37	0.54	0.45	0.52	0.43
Levine	fail	pass	pass	fail	pass
X⇔A	Suc2	Suc2	Suc2	Carb	Suc2
next	Fat	Suc	Carb	Suc2	Carb
next	Carb	Carb	Fat	Fat	Fat
least Aggr.	Suc	Fat	Suc	Suc	Suc

Appendix B

Mean Frequencies of Aggressive Behaviors
Using All Subjects in the Four Diet Groups



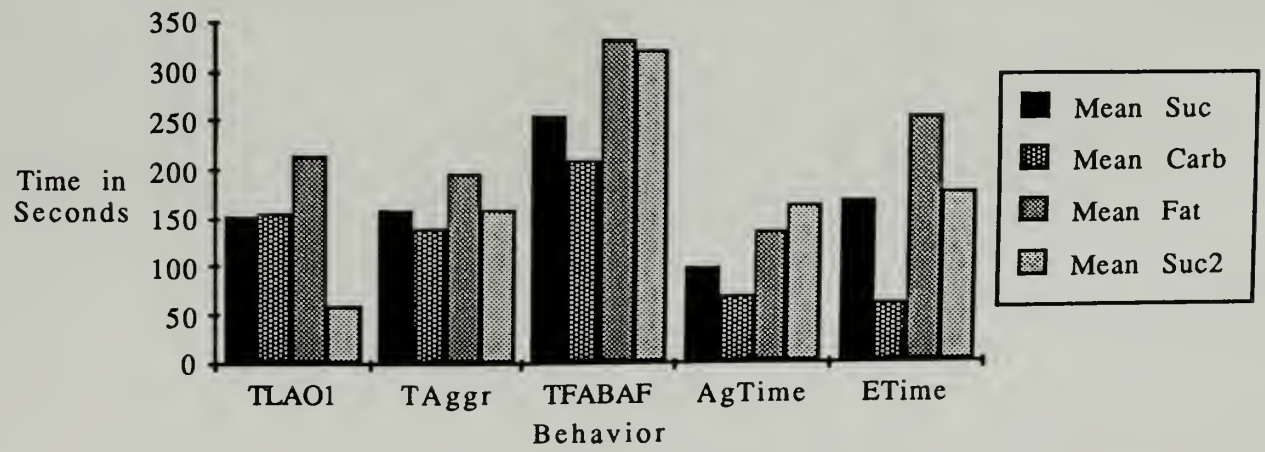
Appendix C

Mean Time of Aggressive Behaviors Using All Subjects in the Four Diet Groups

	TLAO1	TAggr	TFABAF	AgTime	ETime
Mean Suc	154.4	157.5	256.3	98.8	170.0
Mean Carb	156.7	139.3	210.0	70.7	62.0
Mean Fat	215.6	195.6	331.9	136.3	255.0
Mean Suc2	59.4	158.1	322.5	164.4	176.3
SD Suc	271.5	248.7	396	161.4	324.2
SD Carb	234.9	273.0	369.2	189.3	170.8
SD Fat	278.5	266.9	407.6	235.3	325.5
SD Suc2	93	263.3	430.7	283	306.0
p	0.3	0.94	0.81	0.66	0.33
F	1.24	0.13	0.32	0.53	1.15
Levine	fail	pass	pass	pass	fail
X⇨A	Suc2	Carb	Fat	Suc2	Fat
next	Suc	Suc	Suc2	Fat	Suc2
next	Carb	Suc2	Suc	Suc	Suc
least Aggr.	Fat	Fat	Carb	Carb	Carb

Appendix D

Mean Time of Aggressive Behaviors
Using All Subjects in the Four Diet Groups



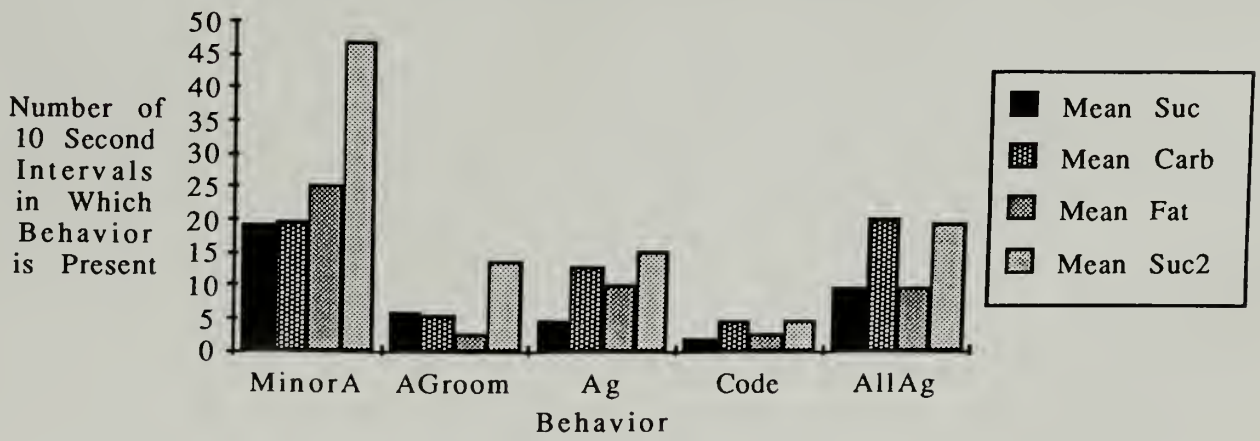
Appendix E

Mean Frequencies of Aggressive Behaviors of Subjects that Manifested Those Behaviors in All Diet Groups

	MinorA	AGroom	Ag	Code	AllAg
Mean Suc	19.6	6.0	5.0	2.3	10.0
Mean Carb	20.0	5.5	13.0	4.8	20.8
Mean Fat	25.4	2.9	10.5	3.0	10.1
Mean Suc2	46.9	13.8	15.5	4.75	19.7
SD Suc	13.6	4.4	5.7	0.96	6.0
SD Carb	23.4	4.0	15.6	2.2	19.2
SD Fat	19.3	2	9	1.4	10.2
SD Suc2	25.2	17.3	6.8	1.5	18.7
p	0.08	0.26	0.52	0.11	0.45
F	2.5	1..5	0.8	2.54	0.91
Levine	pass	fail	pass	pass	pass
X⇒A	Suc2	Suc2	Suc2	Carb	Carb
next	Fat	Suc	Carb	Suc2	Suc2
next	Carb	Carb	Fat	Fat	Fat
least Aggr.	Suc	Fat	Suc	Suc	Suc

Appendix F

Mean Frequencies of Aggressive Behaviors of Subjects that Manifested Those Behaviors in All Diet Groups



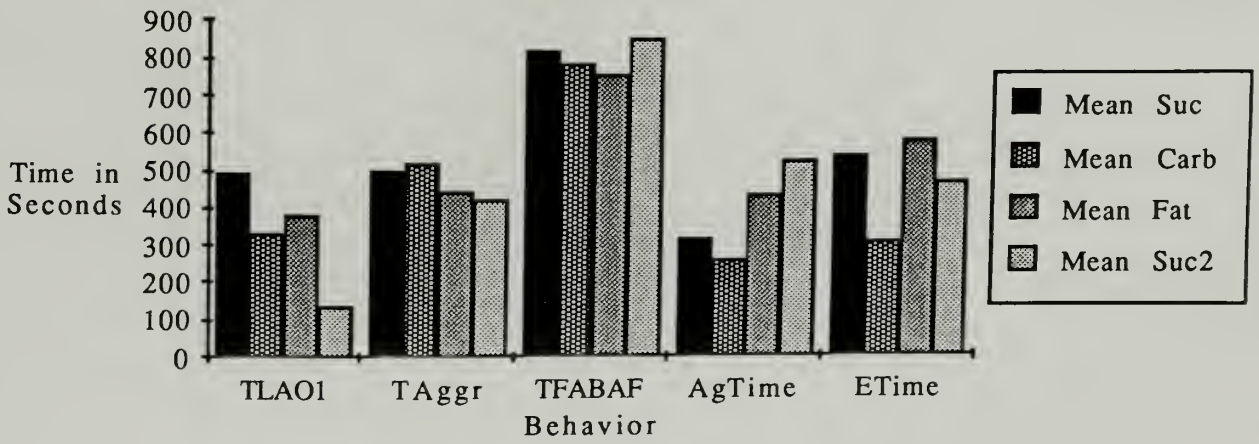
Appendix G

Mean Time of Aggressive Behaviors
of Subjects that Manifested Those Behaviors
in All Diet Groups

	TLAO1	TAggr	TFABAF	AgTime	ETime
Mean Suc	494.0	504.0	820.0	316.0	544.0
Mean Carb	335.7	522.5	787.5	265.0	310.0
Mean Fat	383.3	447.1	758.6	436.0	582.9
Mean Suc2	135.7	421.7	860.0	526.0	470.0
SD Suc	258.3	117.2	100.0	109.2	373.9
SD Carb	242.1	284.3	172.1	314.0	298.2
SD Fat	270.4	216.5	194.2	210.5	204.7
SD Suc2	97.6	273.3	43.8	250.3	340.1
p	0.07	0.88	0.6	0.33	0.61
F	2.69	0.22	0.59	1.25	0.62
Levine	pass	pass	fail	pass	pass
X⇒A	Suc2	Suc2	Suc	Suc2	Fat
next	Carb	Fat	Suc2	Fat	Suc
next	Fat	Suc	Carb	Suc	Suc2
least Aggr.	Suc	Carb	Fat	Carb	Carb

Appendix H

Mean Time of Aggressive Behaviors of Subjects that Manifested Those Behaviors in All Diet Groups



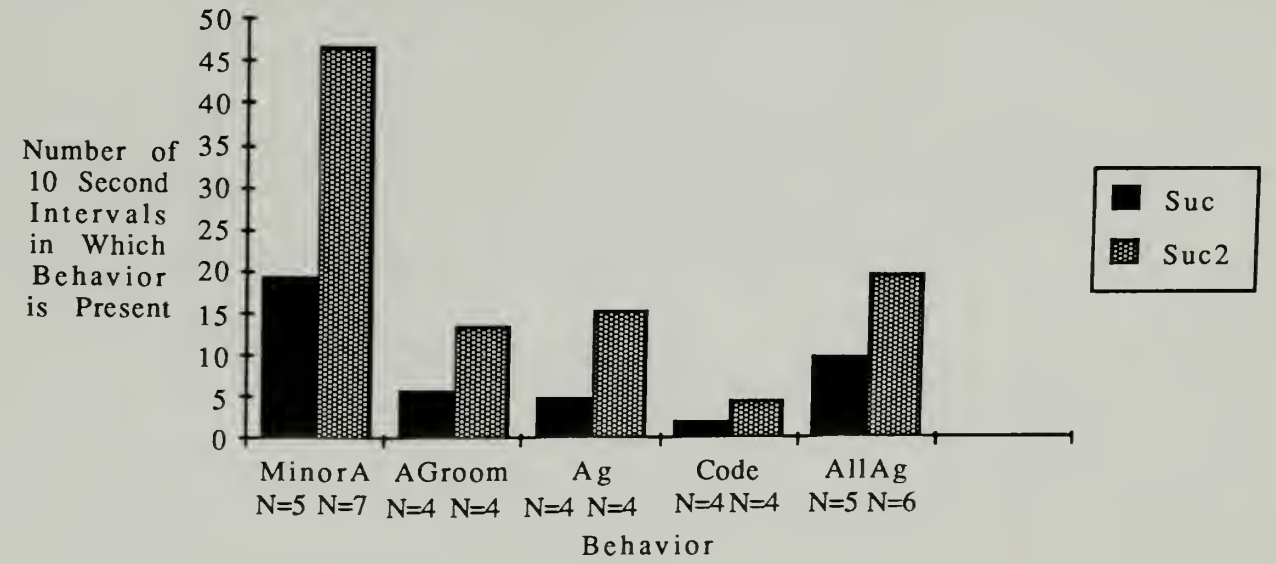
Appendix I

Mean Frequencies of Aggressive Behaviors of Subjects that Manifested Those Behaviors in Suc and Suc2

	MinorA	AGroom	Ag	Code	AllAg
Mean Suc	19.6	6.0	5.0	2.3	10.0
Mean Suc2	46.9	13.8	15.5	4.8	19.7
N Suc	5	4	4	4	5
N Suc2	7	4	4	4	6
SD Suc	13.6	4.4	5.7	0.96	6.0
SD Suc2	25.22	17.4	6.8	1.5	18.7
Max Suc	34	11	13	3	18
Max Suc2	78	39	25	6	54
Min Suc	1	1	1	1	4
Min Suc2	5	1	9	3	1
p	0.054	0.42	0.055	0.031	0.3
Levine	pass	pass	pass	pass	pass
X⇔A	Suc2	Suc2	Suc2	Suc2	Suc2

Appendix J

Mean Frequencies of Aggressive Behaviors of Subjects that Manifested Those Behaviors in Suc and Suc2



Appendix K

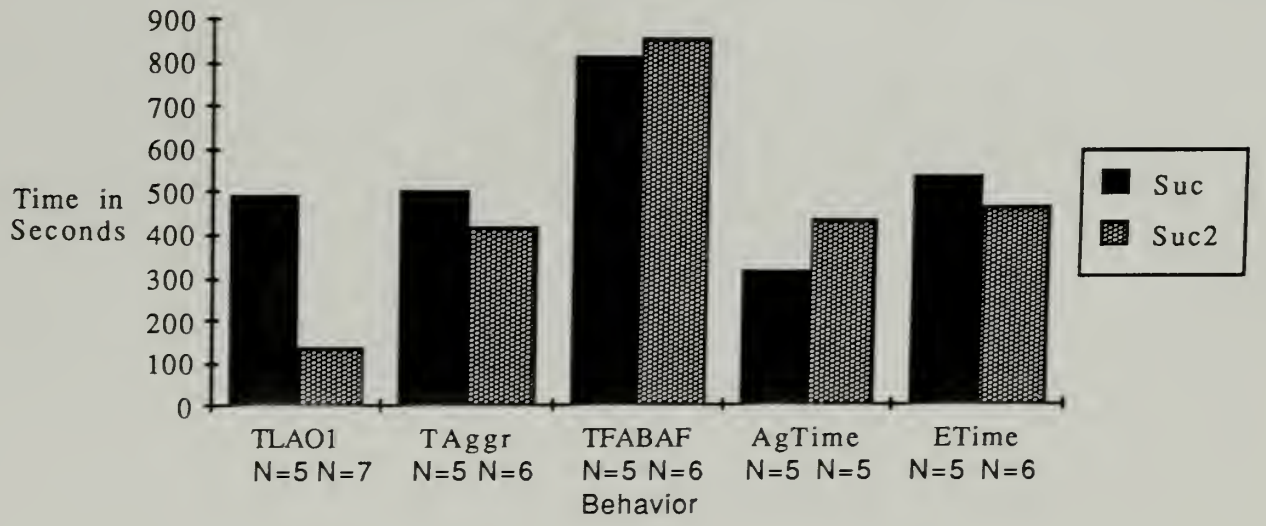
Mean Time of Aggressive Behaviors of Subjects that Manifested Those Behaviors in Suc and Suc2

	TLAO1	TAggr	TFABAF	AgTime	ETime
Mean Suc	494.0	504.0	820.0	316.0	544.0
MeanSuc2	135.7	421.7	860.0	438.3	470.0
N Suc	5	5	5	5	5
N Suc2	7	6	6	6	6
SD Suc	258.3	117.2	100	109.2	373.9
SD Suc2	97.6	273.3	43.8	310.2	340.1
Max Suc	730	710	890	420	880
Max Suc2	290	780	890	740	890
Min Suc	90	420	650	180	30
Min Suc2	20	130	780	0	90
p	0.0068	0.55	0.40	0.43	0.74
Levinc	pass	pass	pass	fail	pass
X=>Aggr.	Suc2	Suc2	Suc2	Suc2	Suc

Note: AgTime. One of the values in Suc2 was a 0, as the 1st and last aggressive act were the same. This value greatly affected the mean and the SD. Without the 0, the mean of Suc2 was 526 (rather than 438) compared to Suc's 316. The Levine test did not fail and the p value was .12 (rather than .43).

Appendix L

Mean Time of Aggressive Behaviors of Subjects that Manifested Those Behaviors in Suc and Suc2



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Appendum to Masters Thesis

When re-conducting analyses in improved ways, I discovered several mistakes in the original which warrented correction. The following reflects both a correction of the errors, as well as some changes in analyses conducted.

1. I always saw Diets Suc, Carb, and Fat as one study, asking one question, and diets Suc and Suc2 as another study, asking another question, yet had not treated them that way until now.

2. I had conducted an analysis of variance on the blood glucose levels each week, adjusting the significance value by using a Scheffe test, and then further accounting for the 12 weeks by using a Bonferroni test. Adjusting my significance level with both tests was extreme/much too conservative. The primary problem, however, was my use of a separate analysis for each week in the first place, particularly with my concluding that a significant result one week meant that that week was when the change occured, rather than that that week was when the sample size and the effect were large enough for there to be enough power for the gradual change to manifest itself. Trend analysis would be more appropriate, to show change over time. I therefore reanalyzed my glucose data by taking the slope and intercept of each mouse and then doing an analysis of variance of the slopes and intercepts by diet group. I also analyzed the first and last weeks to further show that the subjects started out the same and did end up different. Not only was trend analysis more appropriate for the reasons mentioned, but it is also

a more powerful test than what I had done. Consequently, Suc and Suc2 came out as significantly different in this re-analysis, whereas they had not previously. Similarly, I conducted slope intercept analyses on food intake and body weights, finding no significant differences across groups.

3. The variable "Code" was a catagorical variable that I put on a problematic scale in order to analyze it. I have now more appropriately analyzed it with a χ^2. In a post hoc analysis, there was some indication that Suc2 subjects may be more likely than subjects of other diets to be more prone to react aggressively to non-interacting than to interacting target animals: (χ^2 (1)=3.81, p=.051).

4. Also having to do with target behavior, I removed the variable ETime. First, n was small. More important, ETime's meaning would be clear if the subjects had been human but was not with mice.

5. I had analyzed, as a secondary analysis, only some of the aggression data, using a zero value as a cut-off for whether a score would be included or not. That meant that for each behavior, a score of a subject was analyzed only if the subject exhibited that behavior. I was therefore comparing the amount of each behavior across diet groups, for subjects exhibiting that behavior. I was conducting that analysis because of the possibility that only certain animals may be responders. What I really wanted to know was whether subjects, not individual behaviors, differed across groups. What I realized, and therefore adjusted for the re-analysis, is that the more appropriate analysis would

be to select the subjects that are aggressive at all (ie., possible responders), and use those animals for the secondary analysis for all behaviors whether they exhibited each particular behaviors or not.

6. I changed the definition of an aggressive mouse to exclude mice for whom only one interval of one aggressive behavior was coded, such that there was no intensity or continuation. In such a case where there was only one stance or groom that was labeled as aggressive, it is likely that the behavior was not actually an aggressive one. For example, it is difficult to discriminate between social and aggressive grooming. With an otherwise social context, what looks like an aggressive groom is probably not. This change of definition meant changes in which animals would be analyzed as aggressive.

7. I added the variable Flee, to provide slightly more of a picture of the interaction than the subject instigated aggressive behaviors offer.

8. I added the variable ATime, the total time between the first and last contact or non-contact aggressive behavior.

9. I had defined TLAO1 and TAggr in terms of latency to aggressive acts, such that a low value meant high aggression. With all the other variables, a high value meant high aggression. In order that the figures be more readily understandable, I redefined the variables as time following aggressive act. Then, the new TLAO1 equals 900 seconds total minus the old value of TLAO1. I re-analyzed accordingly.

10. I enlarged the definition of TLAO1 to include aggressive groom for the rare instances that aggressive groom occured before any non-contact aggressive behaviors were coded, such that TLAO1 accounted for the first aggressive behavior of any sort.

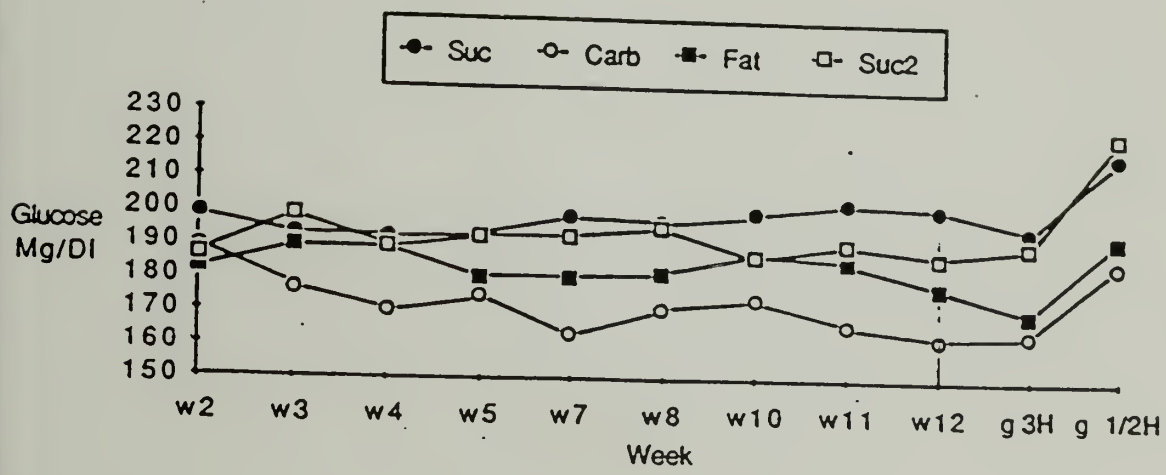
11. The aggression data was coded by modified frequency scoring, which was a good decision. However, when I combined more than one variable for my analysis, I forgot that that could result in intervals being counted more than once. In fact, this error did occur on several occasions. I therefore went back through the raw data and counted non-overlapping intervals for each of my combined variables, and re-analyzed the variables accordingly.

12. I did blood glucose analysis on the rest of the plasma from week 13, and then re-analyzed and re-graphed the gavage data and graphs accordingly.

13. My data was abnormal enough for it to make sense in a thesis to analyze it with both parametric and non-parametric analysis, as while both analyses were problematic in some ways, they complemented each other. However, for the re-analysis I chose non-parametric as the more appropriate.

14. Finally, I have let go of the conviction that the mean differences in my full group aggression data was a trend that I was just unable to capture in my significance tests.

Note: Many of these changes are in part due to discussions with Arnie Well, David King, George Wade, and Melinda Novak, all from the University of Massachusetts.



Mean Glucose Values By Diet:
Weeks 1-14 Without Weeks 1, 6, or 9

RESULTS

Fasting Blood Glucose Levels:

Slope over 12 weeks, trend analysis :

Suc > Carb $p<.01$

Suc > Suc2 $p<.05$ (Note, Suc2 ate slightly less sucrose than Suc)

Week 12, Tukey tests:

Suc > Carb $p<.01$ (Suc M = 202; Carb X =169)

Suc > Fat $p<.01$ (Fat M = 178)

Fat > Carb $p<.05$ (Fat M = 182)

Suc > Suc2 $p<.05$ (Suc2 M = 187)

Challenge Glucose Levels:

Week 13 (3 hour), Tukey tests

Suc > Carb $p<.01$ (Suc M = 195; Carb X =164)

Suc > Fat $p<.01$ (Fat M = 170)

Week 14 (30 minute), Tukey tests

Suc > Carb $p<.01$ (Suc M = 219; Carb X =186)

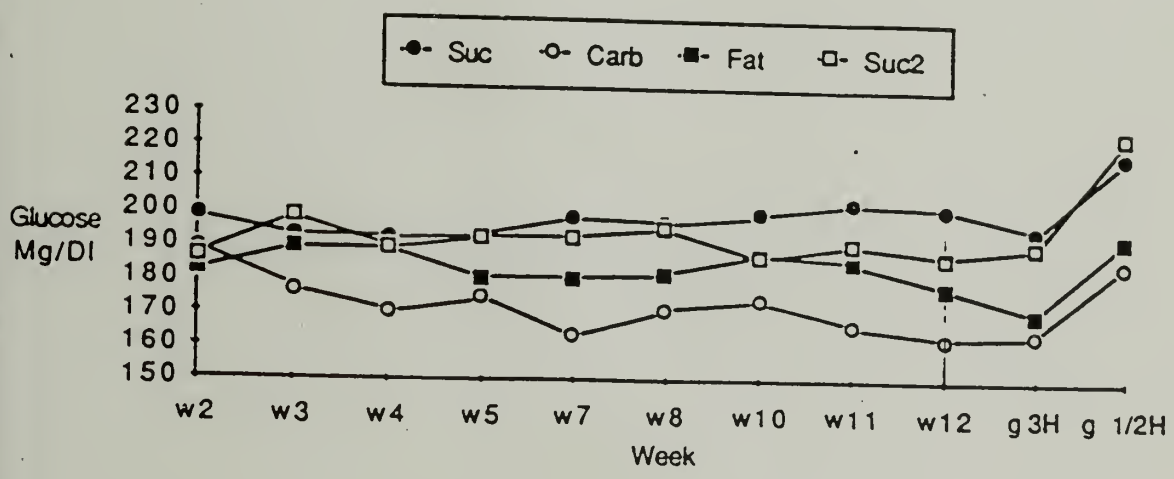
Suc > Fat $p<.01$ (Fat M = 194)

Challenge versus Fasting Glucose Levels:

Week 14 (30 minute, peak) minus Week 12 (fasting),

Tukey tests on difference scores to check for differential reaction to challenge

Suc2 > Suc $p<.05$ (Suc D = 17; Suc2 D = 38)



Mean Glucose Values By Diet:
Weeks 1-14 Without Weeks 1, 6, or 9

Kruskal-Wallis One Way Analysis of Variance Test Results: Suc, Carb, and Fat

All Subjects

			Flee	MinorA	AGroom	Ag	AllAg
Frequency Variables	Mean Rank Suc	(n=16)	365.5	302.5	345.0	355.0	354.0
	Mean Rank Carb	(n=14)	345.5	335.0	312.5	332.0	306.0
	Mean Rank Fat	(n=15)	324.0	397.5	377.5	348.0	375.0
	Kruskal-Wallis H		0.45	3.11	0.91	0.18	0.72
	p		0.80	0.21	0.64	0.92	0.70
	most aggressive			Fat	Fat	Fat	Fat
	next			Carb	Suc	Suc	Suc
	least aggressive			Suc	Carb	Carb	Carb

			TLAO1	Taggr	TFABAF	AgTime	ATime
Time Variables	Mean Rank Suc	(n=16)	299.5	351.0	352.0	367.0	301.5
	Mean Rank Carb	(n=14)	350.0	294.0	297.0	307.0	329.5
	Mean Rank Fat	(n=15)	385.5	390.0	386.0	361.0	404.0
	Kruskal-Wallis H		3.01	1.15	1.37	0.29	3.50
	p		0.22	0.44	0.5	0.87	0.17
	most aggressive		Fat	Fat	Fat	Suc	Fat
	next		Carb	Suc	Suc	Fat	Carb
	least aggressive		Suc	Carb	Carb	Carb	Suc

Aggressive Subjects

			Flee	MinorA	AGroom	Ag	AllAg
Frequency Variables	Mean Rank Suc	(n=5)	67.0	51.0	66.5	60.0	69.0
	Mean Rank Carb	(n=7)	72.0	70.5	72.0	81.0	69.0
	Mean Rank Fat	(n=9)	92.0	109.5	92.5	90.0	93.0
	Kruskal-Wallis H		1.07	0.56	0.93	0.46	1.39
	p		0.59	0.76	0.63	0.79	0.50
	most aggressive			Fat	Fat	Fat	Fat
	next			Carb	Carb	Carb	S/C
	least aggressive			Suc	Suc	Suc	S/C

			TLAO1	Taggr	TFABAF	AgTime	Atime
Time Variables	Mean Rank Suc	(n=5)	45.5	66.0	67.0	71.0	44.0
	Mean Rank Carb	(n=7)	78.0	57.0	60.0	63.0	74.0
	Mean Rank Fat	(n=9)	107.5	108.0	104.0	97.0	113.0
	Kruskal-Wallis H		0.68	2.38	1.96	2.15	1.23
	p		0.71	0.3	0.37	0.34	0.54
	most aggressive		Fat	Fat	Fat	Fat	Fat
	next		Carb	Suc	Suc	Suc	Carb
	least aggressive		Suc	Carb	Carb	Carb	Suc

Kruskal-Wallis One Way Analysis of Variance Test Results: Suc and Suc2

All Subjects

Frequency Variables		Flee	MinorA	AGroom	Ag	AllAg
Mean Rank Suc	(n=16)	276.0	234.5	256.0	257.5	250.0
Mean Rank Suc2	(n=16)	252.0	293.5	272.0	270.5	278.0
Kruskal-Wallis H		0.23	1.63	0.14	0.10	0.39
p		0.63	0.21	0.70	0.75	0.53
more aggressive			Suc2	Suc2	Suc2	Suc2
less aggressive			Suc	Suc	Suc	Suc

Time Variables		TLAO1	Taggr	TFABAF	AgTime	ATime
Mean Rank Suc	(n=16)	234.0	251.0	252.0	256.5	240.0
Mean Rank Suc2	(n=16)	294.0	277.0	276.0	271.5	288.0
Kruskal-Wallis H		1.55	0.33	0.29	0.12	1.08
p		0.21	0.56	0.59	0.73	0.30
more aggressive		Suc2	Suc2	Suc2	Suc2	Suc2
less aggressive		Suc	Suc	Suc	Suc	Suc

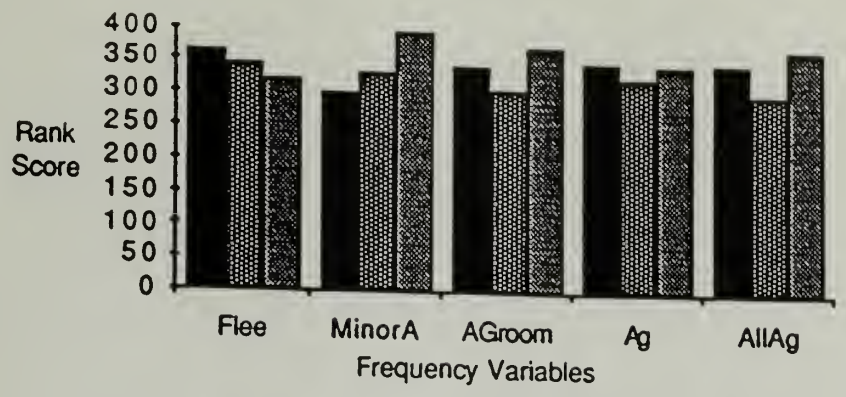
Aggressive Subjects

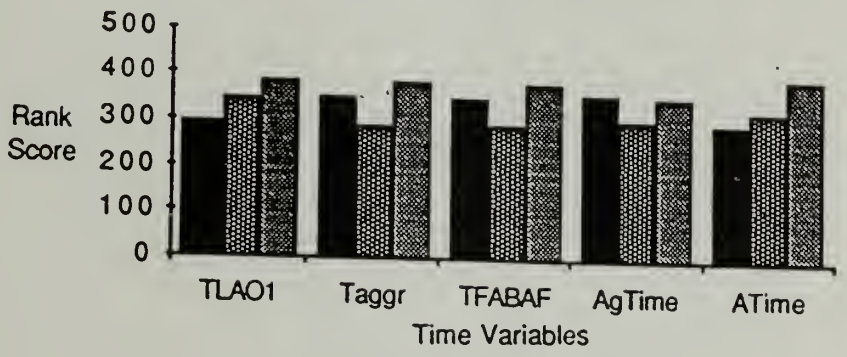
Frequency Variables		Flee	MinorA	AGroom	Ag	AllAg
Mean Rank Suc	(n=5)	38.0	19.0	35.0	30.0	29.0
Mean Rank Suc2	(n=7)	40.0	59.0	43.0	48.0	49.0
Kruskal-Wallis H		0.91	4.81	0.17	0.17	0.32
p		0.34	0.03	0.68	0.68	0.57
more aggressive			Suc2	Suc2	Suc2	Suc2
less aggressive			Suc	Suc	Suc	Suc

Time Variables		TLAO1	Taggr	TFABAF	AgTime	ATime
Mean Rank Suc	(n=5)	19.0	30.0	32.0	30.0	24.5
Mean Rank Suc2	(n=7)	59.0	48.0	46.0	48.0	53.5
Kruskal-Wallis H		4.81	0.17	0.01	0.17	1.69
p		0.03	0.68	0.93	0.68	0.19
more aggressive		Suc2	Suc2	Suc2	Suc2	Suc2
less aggressive		Suc	Suc	Suc	Suc	Suc

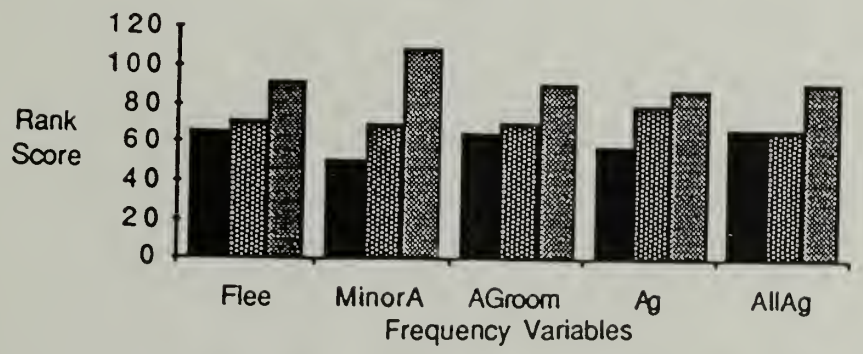
Kruskal-Wallis One Way Analysis of Variance Test Results: Suc, Carb, and Fat

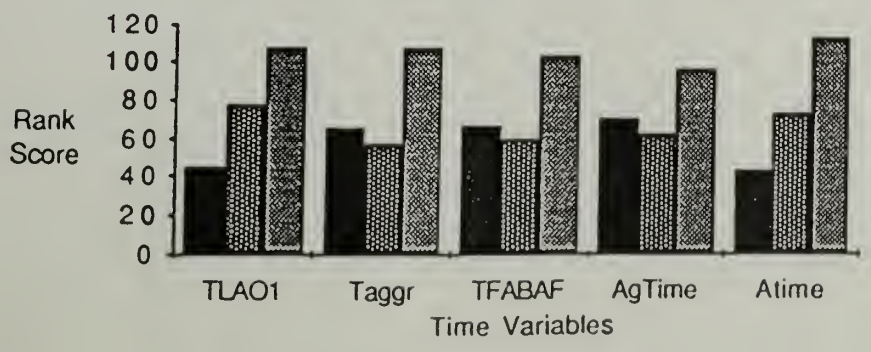
All Subjects

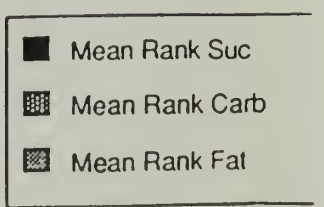




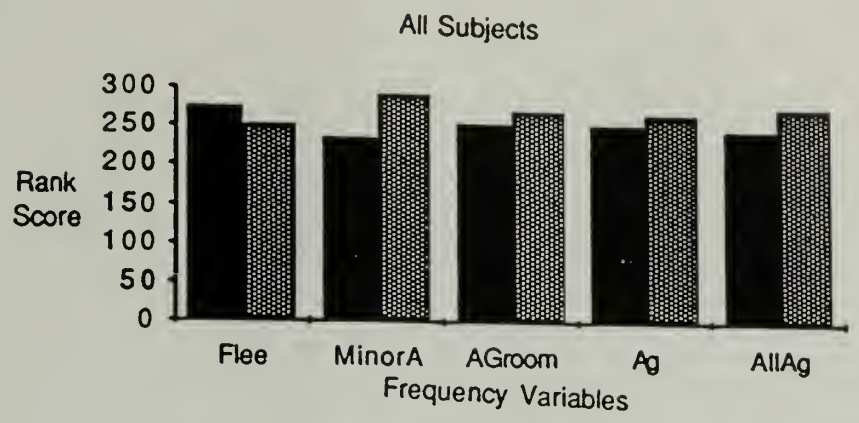
Aggressive Subjects

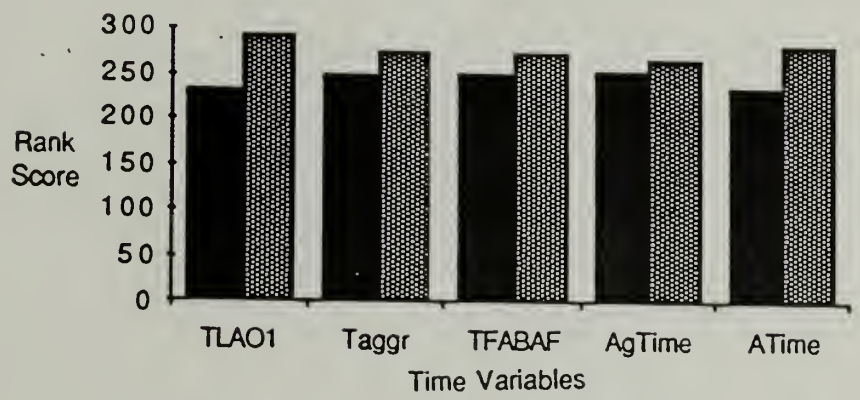


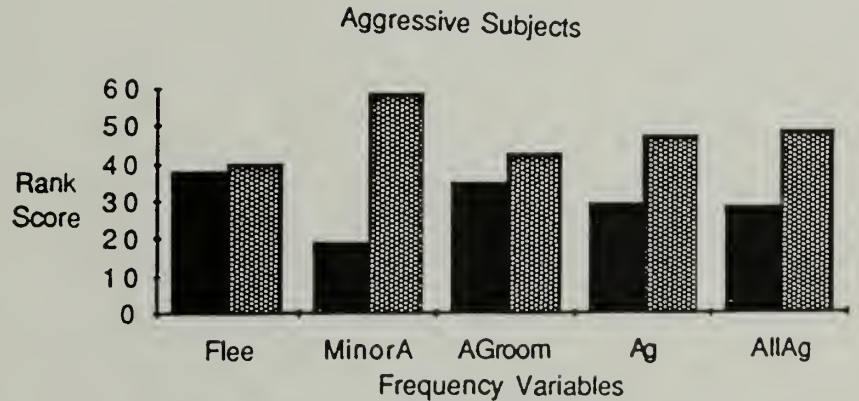


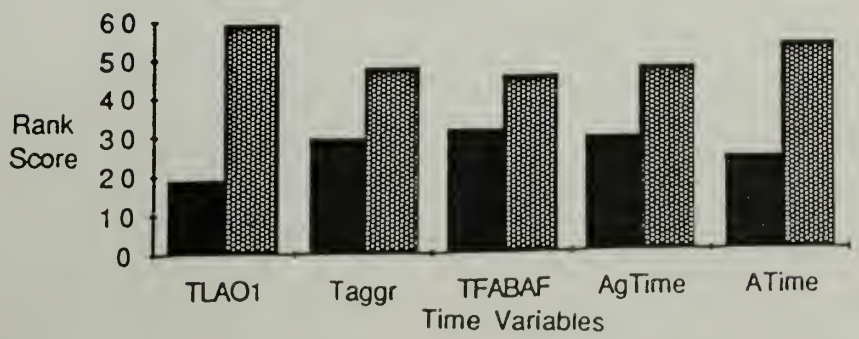


Kruskal-Wallis One Way Analysis of Variance Test Results: Suc and Suc2









Suc2 > Suc $p < .05$ (Suc D = 17; Suc2 D = 38)

Aggression Results:

Analysis of variance on each variable

No significant results

Exploratory sub-group aggression results:

Kruskal-Wallis analysis of variance on each variable, using only aggressive subjects, keeping in mind possibility of individual responders to manipulation

MinorA (number of non-contact aggressive behaviors):

Suc2 more aggressive than Suc $p<.05$ (rank: Suc=19; Suc2 = 59)

TLAO1 (time remaining after first aggressive behavior):

Suc2 more aggressive than Suc $p<.05$ (rank: Suc=19; Suc2 = 59)