Effect of sugar on behavior and mental performance[1,2]

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ABSTRACT Sugar is one of the major dietary components and any relation between sugar and behavior is of major interest. We review the scientific evidence supporting or refuting the relation between sugar and behavior or cognitive function. Little evidence supports the claim that refined sugar intake significantly influences behavior or cognitive performance in children. A few children may have idiosyncratic reactions or rare allergic syndromes and may therefore respond adversely, but this has yet to be substantiated by carefully controlled research. A role for glucose in the potentiation of memory processing appears clear. Further research is required to define the clinical relevance of this role and to elucidate the mechanisms involved. Sucrose may have a sedative effect, at least in infants, but there are no substantial data suggesting that any sugar has analgesic properties. *Am J Clin Nutr* 1995;62(suppl):242S–9S.

KEY WORDS Sugar, sucrose, behavior, performance, cognitive function

INTRODUCTION

Ingestion of refined sugar first became a concern in the United States after the Civil War. At that time the main issue was refined sugar's lower nutritive value (1). The idea that foods containing sugars, mostly sucrose, might have an adverse effect on behavior was first hypothesized in 1922 by Shannon (2). This hypothesis was revisited in 1947 by Randolph (3) in his description of tension fatigue syndrome. Sucrose later appeared as a major offending agent in the 1970s as the result of much coverage in the lay literature of the condition called functional reactive hypoglycemia (1). The belief in the relation between sugar and behavior has since become so strong that it has been used in court cases in the United States as a self-defense, referred to there as the "twinkie defense," and reference to it appears in cartoons and on television shows (4).

Because sugar is one of the major dietary components, any relation between sugar and behavior is of great interest. It is estimated that each child in the United States consumes ≈84.2 g total sugars minus lactose/d (5). Even if sugar were to affect a small portion of the population, this would have significant implications (6). We therefore review the scientific evidence supporting or refuting the relation between sugar and behavior or cognitive function. The first and most prominently believed relation is that between sucrose and hyperactivity and aggressive behaviors. A second, less-well-known relation has been suggested between glucose and enhanced memory, particularly in elderly individuals. A third reported relation is sugar's effect in the opposite direction of hyperactivity, namely sedation.

SUGAR AND HYPERACTIVITY

The belief in the relation between sugar and hyperactivity is based on two theories. The first, a possible allergic response to refined sugar, was conceived of in the first half of this century as tension-fatigue syndrome, a behavioral correlate to the vomiting reaction to milk proteins (7). Two more recent investigations used this theory to study the effects of placing children on restricted diets. In one of these investigations, children were challenged with the presumed offending foods or placebos while being kept on restricted diets (8). The researchers demonstrated significant differences in the children challenged with the food compared with placebo as determined by the behavior ratings by the parents and physician. However, these differences were not found in the psychological testing or the behavior ratings of the independent psychologist. In addition, because the diets were tailored to foods the children were believed to be allergic to, not all of the children had their dietary sugar restricted nor were they challenged with dietary sugar, and only some of the children initially treated were then enrolled in the controlled study. Unfortunately, the report does not indicate how many of the diets included sugar in their restrictions. In the second study, hyperactive preschool children were provided restricted diets that were compared with baseline and placebo diets; significant improvement was found with the restricted diet (9). Again, simple sugars were only one of the restrictions and the only reported differences were in the parents' behavior ratings. Despite clear objective evidence to the contrary, an allergic response, not to the sugar but to protein impurities, that affects behavior may still exist in a few children but this has yet to be documented.

The second suggested etiology for the relation between sugar and hyperactivity is that some children may experience functional reactive hypoglycemia, as reported in adults (10). These individuals experience glucose concentrations in the hypoglycemic range while consuming diets high in carbohydrates, and it was hypothesized that children would display increased motor activity at low blood glucose concentrations. This hypothesis became prominent after it was found in a study of 271 hyperactive children that following a glucose tolerance test

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many children had abnormal blood glucose curves that were similar to the pattern seen in adults with functional reactive hypoglycemia (11), and after hypoglycemia was diagnosed in aggressive criminal offenders (12). Subsequent studies in children showed that the patterns found by Langseth and Dowd (11) were actually normal variations in blood glucose (13).

Separate from any of the underlying etiologies, interest in sugar as a single offending agent peaked in the mid-1970s because of several correlational studies. One early correlational study suggested an association between sugar intake and hyperactivity: hyperactive children who consumed more sugar seemed to display more hyperactive behavior (14). A similar but smaller effect was found in a second study (15). Because the main limitation of correlational studies is that no causal relation can be determined, it is equally possible that the behavior caused the increased sucrose intake as it is that the increased sucrose intake caused the behavior. More definitive examination of the issue has required research with an intervention methodology.

Most intervention research has entailed controlled, double-blind challenge studies. Children receive challenges with foods or drinks containing sucrose or an artificial sweetener; the children, their parents, and the researchers are not aware of the composition of the foods or drink, and behavior and cognitive performance is closely assessed within the few hours after the ingestion. In reviewing these studies, there are some important considerations that include the age and prior existing conditions of the subjects being studied, the doses of agents used in the challenge design, the type of placebo used, the specific dependent measures, and the diet of the subjects with respect to overall intake and timing of meals. The possible mechanisms of any adverse response to sugar help to dictate the timing, duration, and control of other variables in the study such as diet. For example, to study idiosyncratic reactions, a large sample size of adverse responders is needed. Investigating an allergic etiology of sugar requires strict dietary control and a longer duration of study.

The first important consideration is characteristics of the subjects. The immediate effects of sugar have been examined in a wide variety of children. Studies have involved normal children, subjects historically identified as behaving poorly after sugar ingestion, children with diagnosed hyperactivity or attention deficit disorders, and aggressive or delinquent children. The studies have used subjects ranging in age from preschool children to adolescents.

There are few if any guidelines regarding the type or quantity of sugar likely to affect behavior. Sucrose has been the most prominent sweetening agent, although many foods are now sweetened with corn sweeteners, that is, combinations of glucose and fructose in various proportions. Fructose and glucose have been included in a few of the studies. Most challenge studies have used the quantity used in glucose tolerance tests (1.75 g/kg), although doses as high as 2 g/kg have been studied.

The diet condition before challenge may also be a factor in the interpretation of intervention studies. This issue has been raised particularly regarding the specific manipulation of the ratio of carbohydrate to protein or fat. However, study designs have varied greatly, from no dietary control to restricted diets and including overnight fasts and postprandial states. With this degree of variation one would expect variations in findings if preexisting diet was a factor, and yet the results from almost all of the studies consistently found no effect when the children were challenged with sugar.

The final important issue is the measurement of the proposed effects of refined sugar. Most measures have focused on the behavior of children with attention deficit hyperactivity disorder, who are characterized as having a short attention span, impulsive behavior, and increased motor activity compared with other children. Reports from parents and teachers have been used to assess behavior. Numerous behavior-rating scales with reasonable psychometric properties were used, depending on age and range of behaviors. Other neuropsychological measures were also used to assess vigilance, impulsivity, memory, and motor skills. Some studies used electronic motion-detector devices to record activity.

Despite the high degree of variation in subjects, challenge agents, and dependent measures, the results are surprisingly consistent for most of the studies. Results from 14 published papers describing 16 controlled challenge studies and representing >400 subjects do not support the hypothesis that refined sugar affects hyperactivity, attention span, or cognitive performance in children. This is highly significant because many of the subjects were originally considered to be adversely affected by intake of sucrose. Such a population was studied by Gross (16), who found no difference in the behavior of 50 hyperkinetic children identified as adverse responders to sugar. This is especially remarkable because diet was not manipulated other than by sucrose challenge and observations were by parents who identified their children as behaving poorly after ingestion of sucrose. Six additional challenge studies investigated this population without demonstrating a clear effect of sugar consumption (17–22). Behar et al (17) investigated 21 boys aged 6.5–14 y, whose parents believed that they experienced deterioration in behavior after refined sugar intake. Nine of these children met DSM III (a diagnostic manual devised by the American Psychiatric Association) (23) criteria for attention deficit disorder with hyperactivity. Standardized measures for attention and memory as well as behavioral observations and an actometer were used. No effects on behavior or cognition were found. However, a small but statistically significant decrease in motor activity was found after sucrose ingestion. This was also found by Saravis et al (24) in normal children.

Several other studies used children who were diagnosed as having attention deficit disorder for which response to sugar was not a criterion. Wolraich et al (25) challenged two groups of 16 hyperactive males with 1.75 g sucrose/kg each and found no differences in activity, behavior, or cognitive performance despite using 37 measures. The first group was challenged 30 min after a standard lunch and the second group was challenged after an overnight fast. In a third study, also of 16 males with attention deficit disorder, behavioral effects were examined in a classroom setting (26). Again, no effects were found.

On the basis of one study, it was postulated that a more marked reaction might be established in younger children. Goldman et al (27) observed some increased motor activity and decreased attention in eight normal preschool children completing a continuous performance task 1 h after a sucrose challenge. However, similar results were not found in other studies dealing with this age group. Kruesi et al (18) challenged 18 boys aged 2–6 y who were reported to have adverse behav-

ioral responses to sucrose and found no effects of sucrose on behavior or activity level, even when the person who reported the child to be an adverse responder was used as a rater. Rosen et al (19), who studied 30 preschool children, reported no statistically significant differences between the sucrose challenge and placebo although small but not statistically significant increases in activity of all children after sugar ingestion were reported on the teacher rating scales. Additionally, Roshon and Hagen (28) challenged 12 normal 4–5-y-old boys and girls and found no difference in behavior, attention span, or activity level between the study and placebo groups.

To answer the question of a possible dose effect of sucrose on behavior, Ferguson et al (20) used a range of doses of sucrose in their cohort of eight children who were reported by their parents to have adverse behavioral responses to sugar consumption. No consistent effects were observed. In the study by Goldman et al (27) reported above, when a high dose of 2 g sucrose/kg was used, some increased motor activity and decreased attention was found. The authors cautioned, however, that these children still exhibited activity well within normal limits. Mahan et al (21) used a challenge of 2 g sucrose/kg in a group of 16 subjects considered to be adverse responders to sucrose and observed no effects. Roshon and Hagen (28) also used a challenge of 2 g/kg and found no effects.

A possible effect of sugar on delinquent or aggressive behavior was also postulated (29). Virkkunen (30) reported a significantly higher rate of glucose tolerance test patterns consistent with functional reactive hypoglycemia in a prisoner population. These findings led to the concept of the "twinkie defense," an argument that violent criminals were not responsible for their actions because diets high in carbohydrates made them prone to violent behaviors (4). Bachorowski et al (31) did not observe any impairment in behavior or cognitive performance in their study of 115 juvenile delinquents. Wender and Solanto (32) studied 17 children with attention deficit disorder with hyperactivity and oppositional defiant disorder and 9 age-matched control subjects. No differences in aggressive behaviors were identified after a sucrose challenge was compared with placebo for either group. However, a decrease in performance on a continuous-performance task was found in the group with attention deficit disorder. This was the same finding as reported above in the Goldman et al (27) study, and a similar finding was reported by Connors (CK Connors, unpublished observations, 1983).

The studies mentioned above all used a challenge design similar to that used in assessing the effects of medications: subjects in some studies were challenged with sucrose after a fast, others after an isoenergetic meal matched to controls, and still others with no dietary manipulation. In addition to the importance of controlling energy intake in a design, some investigators have argued that the carbohydrate-to-protein ratio is important. As this ratio increases, subjects may experience drowsiness and a drop in cognitive performance (31, 32). Challenge studies are also unable to address any effects that might occur from chronic exposure. One argument raised by those postulating an allergic etiology is that unless the children are denied the offending agent for a period of time, a challenge with the offending agent is not likely to show a change in behavior.

Few studies have examined the effect of sucrose consumed in high doses over time. Wolraich et al (22) used a controlled diet design with sucrose, aspartame, and saccharin and complete control of the 48 subjects' dietary intake for 9 wk. Preservatives, dyes, and food additives were removed from the diet. In addition, the sucrose diet had up to a 30% higher carbohydrate-to-protein ratio and slightly higher energy intake than did the aspartame and saccharin diets. The subjects included 23 children aged 6–10 y who were identified by their parents as experiencing deterioration of behavior with refined sugar ingestion and 25 normal children aged 4–6 y. Thirty-nine and 31 measures, respectively, were used in the two groups of children, including parent and teacher behavior ratings and a battery of neuropsychological measures. The analyses included examining the effects when the subjects received sucrose as the last of the three diets, giving them 6 wk on a low-sucrose diet before the challenge. Even so, no evidence was discovered implicating sucrose as affecting the behavior, cognitive performance, or motor activity of these children. Further analysis to identify any individual subjects who might respond adversely did not identify any responders. Bachorowski et al (31) reported similar results despite a carbohydrate-to-protein ratio in the sucrose diet almost twice that in the control diet.

It is curious that despite the of clinical proof, many parents remained convinced of the association between sugar and adverse behavior. This may be explained by coincidence and expectancy. For example, excited states in children are common with birthday parties and holidays, for which large sugar intake is likely. Because sugar is commonly ingested and enjoyed by children, variation in their behavior may be mistakenly correlated with consumption. Additionally, Hoover and Milich (33) found that when parents who believed that sugar caused adverse effects expected their children to be challenged with sugar even though they were challenged with an artificial sweetener, they rated their children's behavior significantly worse and were more demanding than parents who rightly expected their children to be challenged with an artificial sweetener. The differences in this study were greater than were found in the studies examining the effects of sugar. If behavior changes are noted when children receive candy even if there are other possible causes, and parents expect that behavior, they are likely to be convinced that a relation does exist between sugar and adverse behavior.

In summary, clinical investigations have not demonstrated a significant effect of sucrose on aggressive or disruptive behavior, motor activity, or cognitive performance in children. Studies mostly used challenge methods similar to those used in investigating the effects of food additives and preservatives. The diets of the subjects at the time of the challenges varied greatly with respect to their protein-to-carbohydrate ratios. A wide variety of subjects have been studied and no particular group of children was identified as reacting differently from the general population. This evidence is even beginning to reach the lay press. Since 1988 some articles in the popular press have reported the clinical trials described previously (34, 35) and attitudes toward the effects of dietary sugar on behavior are beginning to change. More extensive coverage in the lay press occurred recently with the publication of the controlled diet study (22).

GLUCOSE AND MEMORY

There is increasing evidence that sugar, specifically glucose, can influence central nervous system activity. Although memory enhancement was not demonstrated in any of the challenge studies that measured memory in children, there is evidence that glucose concentrations influence memory functioning in rats and humans, locomotor activity and sleep patterns in rats, and the distress associated with painful procedures in human infants. The focus of research in this area has been to establish how glucose acts to mediate these effects.

Because the retention of memory is an important central nervous system function in the process of cognition, central nervous system mechanisms salient to this function, such as noradrenergic and cholinergic systems, have been investigated. Sternberg et al (36) demonstrated memory enhancement in aged rats and mice with peripheral injections of epinephrine. To investigate the positive effects of epinephrine on memory processing further, Gold and his colleagues (37, 38) have systematically studied the effects of glucose on both animal and human subjects. Using a foot shock avoidance task on rats, Gold (37) observed, similar to the epinephrine effects, significantly improved memory retention in animals who received an injection of 10–100 mg glucose/kg immediately after training. No effect was observed if the injection was delayed by 1 h or if higher or lower doses were used. In a subsequent study, glucose had effects similar to other memory modulators in that its administration with low-foot-shock training enhanced the rats' memory storage, whereas its administration with high-foot-shock training impaired memory retention, possibly because of endogenous epinephrine produced by the foot shock (38). This study also confirmed glucose facilitation of memory in the presence of alpha or beta adrenergic blockers, suggesting that glucose release in response to epinephrine release is the mechanism of memory modulation.

Using intraventricular injections of glucose, Lee et al (39) further elucidated that glucose acts directly on central nervous system processes. Rats injected in this manner showed significantly improved memory retention compared with controls 24 h after training. Ragozzino and Gold (40) presented additional evidence that glucose interacts with central cholinergic function. They found that glucose attenuated the activity of muscarinic cholinergic antagonists by improving memory test performance and potentiating decreased locomotor activity in rats. Epinephrine and glucose have similar inverted U-shaped dose-response curves (41). This implies an optimum dose for memory enhancement. Lower and higher doses of epinephrine or glucose gradually impair the subjects' ability to retain the learned information. The peak effect was observed at doses of 100 mg/kg. The similarity of these dose-response measurements adds further evidence that glucose released by epinephrine is a mediator of memory enhancement. Other monosaccharides such as fructose also improve performance of memory tasks but at extremely high doses (2 g/kg) and are therefore unlikely to have a similar role in physiologic pathways (42).

Extending the postulate that glucose improves memory functioning in humans, Gonder-Frederick et al (43) demonstrated significantly improved memory processing by using a standardized measure in 9 of 11 elderly human subjects after administration of oral glucose compared with placebo. Enhancement of memory in elderly humans 24 h after learning was significantly improved by glucose administration before or after the learning task (44). Just like the finding in rats that memory potentiation in elderly rats was more marked than that in young adult rats, a similar phenomenon may be seen in humans (45). Similar results were found in a recent study by Benton et al (46) in young women. None of the studies of sugar in children showed any effect on memory, whereas those completed in elderly subjects did. However, most of the studies with children used sucrose, and only a few specifically tested memory.

McGlynn et al (McGlynn, Lennartz, and Gold, unpublished observations, 1992) demonstrated that the medial septum of the brain stem in rats, analogous to the thalamic region in humans, is at least one site for glucose to act in improving memory function. They found that rats with septal lesions failed to show improved retention with peripheral glucose administration. Additionally, interseptal and peripheral injections of glucose attenuated the memory impairment produced by intraseptal injections of morphine (47).

The potentiation of memory processing in humans was achieved by administering single oral doses of glucose which caused a rise in blood glucose in the physiologic range. The precise anatomical areas of influence and the particular mechanisms of action have not been elucidated. For example, which is more important, the peak glucose concentration or the rate of the rise in central nervous system tissues? Possible pharmacologic enhancement of memory awaits research aimed at discovering these and other related mechanisms (41).

In summary, there is evidence that glucose is discretely involved in neuroendocrine modulation of memory storage in both rats and humans. This influence is demonstrated best in elderly subjects. One site of action for glucose is the medial septum, which is rich with communications to the hippocampus, an area of the brain important in memory processing. Although the precise mechanism of the effects of glucose on memory is not known, these findings may have far-reaching implications for pharmacologic treatment of memory impairment resulting from old age or head trauma. Other mechanisms of memory enhancement may also be important and the influence of blood glucose is but one aspect in a complicated web of interactions within the brain.

SEDATIVE EFFECTS OF SUGAR

Further evidence of the effect of glucose on central nervous system function in rats was noted by Stone et al (48) in their study of memory enhancement in rats. They observed that the duration of paradoxical (rapid eye movement) sleep in aged rats was attenuated by glucose administration. In another study, sleep patterns in rats were shown to become normal with glucose administration (49). The only finding of a similar phenomenon in humans is by Blass and Hoffmeyer (50), who reported that sucrose was an effective analgesic in infants exposed to painful procedures. They noted that infants given 2 mL of a 12%-sucrose solution before a heel stick cried one-half as much as did control infants. Similarly, infants who underwent circumcision and were given a pacifier moistened with a sucrose solution cried 18% less than infants who were given a water-moistened pacifier. The degree of calm also appeared to be related to the quantity of sucrose. These observations may

be due to other factors such as reduction of anxiety or personality factors of the individual infants. There is no substantial evidence that sucrose acts as an analgesic.

A possible mechanism explaining the effects on sleep in rats and the calming of human infants may be related to the influence of carbohydrate on serotonin concentrations in the brain. Serotonin can affect arousal and has a particular influence on sleep induction. Carbohydrate intake alone, whether as monosaccharides or starch, increases brain serotonin by increasing the proportions of tryptophan, tyrosine, and phenylalanine with respect to other neutral amino acids (leucine, isoleucine, and valine). This effect is not observed if protein is consumed concomitant with the carbohydrate (51). This phenomenon may have been noted by Oberlander et al (52) in their investigation of feeding on sleeping and crying in human infants. An association between carbohydrate (lactose) ingestion alone and sleep duration was not observed. Crying was diminished in infants fed carbohydrate compared with water, but no significant difference between lactose and formula was observed. The significance of the carbohydrate-serotonin interaction on behavior is unclear, and further research is needed.

CONCLUSION

There is little evidence to support the claim that refined sugar intake significantly influences behavior or cognitive performance in children. A few children may have idiosyncratic reactions or rare allergic syndromes and may respond adversely, but this has yet to be substantiated by carefully controlled research. The preliminary findings suggest a role for glucose in the potentiation of memory processing. Further research is required to define the clinical relevance of this role and to elucidate the mechanisms involved. Sucrose may have a sedative effect, at least in infants, but there are no substantial data suggesting that any sugar has analgesic properties.

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COMMENTARY

Authors: We are providing, as requested, the following table (**Table 1**), which includes more comprehensive details about the studies. We encourage readers to review the table in conjunction with the descriptions of the studies in the text or with the articles themselves. The studies vary greatly in design and quality even though they are all double-blind, controlled studies.

Sherman: It has been proposed that fatigue during prolonged exercise may be related to exercise-induced changes in the availability of tryptophan (Trp) to the brain and to the synthesis of serotonin (1). It is proposed that the increase in brain 5-hydroxytryptamine (5-HT) that occurs with prolonged exercise influences the central nervous system in an unknown way that contributes to the process of fatigue (2, 3). Nutritional feeding of branched-chain amino acids (BCAAs) during prolonged exercise that decreased the ratio of BCAAs to Trp compared with the control ratio improved the running performance of so-called slow runners and improved their mental performance after exercise (4). Davis et al (5) reported that compared with placebo, feedings of a liquid carbohydrate solution during prolonged cycling improved endurance performance and attenuated the increase in free Trp and the free Trp:BCAA ratio. Thus, increasing blood BCAA concentration by BCAA ingestion and reducing blood fatty acid capabilities via carbohydrate ingestion may have some direct effects on the central nervous system. This is plausible because acute short-term swimming exercise apparently increases permeability of the blood brain barrier, presumably via the influences of serotonin on 5-HT receptors (6).

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McDonald: White and Wolraich have reviewed evidence suggesting that glucose may improve memory and have described findings in rats of improved memory storage after foot-shock stress and infusion of glucose. On the basis of these and other data, they have concluded that glucose is discretely involved in neuroendocrine modulation of memory storage. Although not referenced, the findings of Elliot and Sapolsky (1) and Stein-Behrens and Sapolsky (2) at Stanford University are consistent with the concept of an interaction among memory function, stress hormones, and aging. These investigators have found that the age-related loss of memory resulting from high concentrations of glucocorticoids can be restored by elevating serum glucose concentrations. Some questions arise, however, as to the relevance of these data to normal aging. First, normal aging is not characterized by increased concentrations of stress hormones. It is more likely that memory function is related to disease rather than to aging per se. Second, serum glucose is well maintained or even slightly elevated in aging (3–7). If glucose is artificially raised to improve memory, does one

TABLE 1
Summary of sugar challenge studies

Reference	Type, number, and age of subjects	Sugar and dose	Placebo	Diet control	Dependent measure[1]	Results
16	Hyperactive; *n* = 36 M, 14 F; 5–17 y	Sucrose (75 g)	Saccharin	None	Mother rates behavior change	No significant differences
25	Hyperactive; *n* = 16 M; 7–12 y	Sucrose (1.75 g/kg)	Aspartame	1 h after standard lunch with 3-d low-sucrose diet	CPT, PAL, PRS, PO, ACT, and MFF	No significant differences
25	Hyperactive; *n* = 16 M; 8–12 y	Sucrose (1.75 g/kg)	Aspartame	After overnight fast with 3-d low-sucrose diet	CPT, PAL, PRS, PO, ACT, and MFF	No significant differences
26	Hyperactive; *n* = 16 M; 6–9 y	Sucrose (1.75 g/kg)	Aspartame	After overnight fast	CO and TRS	No significant differences
17	Sugar sensitive; *n* = 21 M; 6–14 y	Sucrose and glucose (1.75 g/kg)	Saccharin	After overnight fast with 3-d high-carbohydrate diet	CPT, ACT, and PO	Significant decrease in motor activity with both sugars
18	Sugar sensitive, *n* = 18 M, and normal, *n* = 12 M; 2–7 y	Sucrose and glucose (1.75 g/kg)	Aspartame and saccharine	None	TRS, PRS, ACT, and aggression	No significant differences
24	Normal; *n* = 10 M, 10 F; 9–10 y	Sucrose (1.75 g/kg)	Aspartame	Given with a constant breakfast after an overnight fast	ACT, PO, and associate-learning task	Significant decrease in motor activity with sucrose
19	Normal; *n* = 26 M, 19 F; 5–7 y	Sucrose (50 g)	Aspartame	In conjunction with constant breakfast	PAL, CO, and TRS	No significant differences
27	Normal, *n* = 8; 3–7 y	Sucrose (2 g/kg)	Aspartame	None	CPT and PO	CPT errors greater with sucrose; increase motor activity with sucrose
32	Hyperactive to aggressive, *n* = 17, and normal, *n* = 9; 5–7 y	Sucrose (1.75 g/kg)	Saccharin and aspartame	Given with a consistent breakfast	CPT and PO	More CPT errors with sugar diet for hyperactive children only
28	Normal; *n* = 6 M, 6 F; 3–5 y	Sucrose (2 g/kg)	Aspartame	None	PAL and PO	No significant differences
21	Sugar sensitive, *n* = 5; 3–10 y	Sucrose, honey, and starch (3 g/kg)	Aspartame	None	ACT and CO	2 subjects, no effect; 1 subject, worse with placebo and honey; and 2 subjects, increased activity with sucrose
31	Delinquents and normal adolescents, *n* = 154; 14–19 y	Sucrose (78 g)	Aspartame	Given with consistent breakfast	Finger tapping, trail making, digit span, CPT, TRS, and PRS	More disturbed delinquents improved with sucrose; better behaved delinquents did worse
20	Sugar sensitive, *n* = 6 M, 2 F; 5–13 y	Sucrose (1.75 g/kg)	Aspartame	None	CPT, PAL, memory task, CO, PBS, and TRS	No evidence for a sucrose effect
20	Normal, *n* = 18; 3–5 y	Sucrose (1.75 g/kg)	Aspartame	None	PRS, TRS, CO, PAL, ACT, and developmental drawing	Significantly worse in developmental drawing with sucrose
CK Connors, unpublished, 1983	Psychiatric inpatients, *n* = 28 M, 9 F; 6–12 y	Sucrose and fructose (1.25 mg/kg)	Aspartame	None	CPT, CO, reaction time, and TRS	Decreased minor motor activity with sucrose and decreased gross motor activity with fructose

[1] ACT, actometer; CO, classroom observation; CPT, continuous performance test; MFF, matching familiar figures; PAL, pained associate learning; PO, play room observation; PRS, parent rating scale; TRS, teacher rating scale.

not run the risk of inducing diabetes-like problems? Finally, these findings are related to glucose, not dietary sucrose.

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