Effect of drinking soda sweetened with aspartame or high-fructose corn syrup on food intake and body weight

Michael G Tordoff and Annette M Alleva

ABSTRACT  To examine whether artificial sweeteners aid in the control of long-term food intake and body weight, we gave free-living, normal-weight subjects 1150 g soda sweetened with aspartame (APM) or high-fructose corn syrup (HFCS) per day. Relative to when no soda was given, drinking APM-sweetened soda for 3 wk significantly reduced calorie intake of both females (n = 9) and males (n = 21) and decreased the body weight of males but not of females. However, drinking HFCS-sweetened soda for 3 wk significantly increased the calorie intake and body weight of both sexes. Ingesting either type of soda reduced intake of sugar from the diet without affecting intake of other nutrients. Drinking large volumes of APM-sweetened soda, in contrast to drinking HFCS-sweetened soda, reduces sugar intake and thus may facilitate the control of calorie intake and body weight.  Am J Clin Nutr 1990; 51:963-9.

KEY WORDS Human food intake, aspartame, high-fructose corn syrup, sugar, sweetness, body weight, weight control

Introduction

It is generally believed that artificial sweeteners provide the benefit of a desirable taste without calories (1). Indeed, foods and drinks containing these substances are frequently labeled “diet.” However, the possibility that sweet, low-calorie foods and drinks actually lead to a reduction in body weight has not been examined in detail.

There is mounting evidence that in the short term (< 12 h), consumption of artificial sweeteners increases the motivation to eat. Rats increase food intake after drinking a saccharin solution (2). Humans report increased hunger after drinking solutions of aspartame (APM), saccharin, or acesulfame-K (3, 4). Food intake is greater after eating a saccharin-sweetened yogurt than after a glucose-sweetened or unsweetened yogurt (5). These results are not caused by a postingestive or pharmacological effect of the artificial sweeteners; rats eat more food after sham-drinking (ingesting but not absorbing) sucrose solution (6), and humans increase hunger ratings after chewing a gum base sweetened with as little as 0.6 mg APM (7). Moreover, subjects who have normal sweetness perception while drinking a sweet milk shake subsequently eat more food than do subjects who cannot perceive the milk shake as sweet [because of treatment with gymnemic acid (8)]. These and other findings (9) suggest that sweet oral stimulation initiates a cephalic-phase metabolic reflex that increases appetite (10).

The long-term effects of artificial sweeteners on food intake and body weight are less clear. Although some investigators report weight gain in animals given artificial sweeteners to eat or drink (11-13), the majority reports no effects (11, 14-17). What little work has been done in humans does little to answer the question. Two correlative comparisons of users and nonusers of artificial sweeteners showed that the sweeteners had no effect on body weight (18, 19). In contrast, an epidemiological study of 78 694 women found that reported weight gain was greater in those who used artificial sweeteners than in those who did not (20). There are only three published studies that have used a causative approach. In one, dieters who were either encouraged or discouraged to use APM-sweetened products lost the same amount of weight (21). In the other two, hospitalized lean and obese subjects ate fewer calories during a 6- or 12-d period when APM replaced all sucrose in the diet than when they were fed a high-sucrose diet (22, 23).

None of the work to date has examined the effect on food intake or body weight of adding artificial sweeteners to the normal diet. In the present study, we attempted to do this by determining the effect on long-term (3-wk) food intake and body weight of consuming APM given in soda, the most prevalent vehicle for artificial sweeteners. By comparing periods when subjects drank APM, HFCS, and no soda, we planned to examine the effect of APM both as an addition to the diet and as a sugar substitute.

Methods

Recruitment of subjects

The experiment was run in two replications, held in the fall of 1987 and the spring of 1988. It was approved by the Committee on Studies Involving Human Beings at the University of Pennsylvania. Potential subjects were first attracted by advertisements posted on local university campuses. Upon ar-

1 From the Monell Chemical Senses Center, Philadelphia.
2 Supported by the US Department of Agriculture’s Competitive Research Grants Program grant 87-CRCR-1-2316.
3 Address reprint requests to MG Tordoff, Monell Chemical Senses Center, 3500 Market Street, Philadelphia, PA 19104.
4 Received May 30, 1989.
5 Accepted for publication August 9, 1989.

rival at the laboratory for an initial appointment, each prospective subject received a written description of the study and signed a participation consent form. The study's purpose was stated as "an ongoing project to examine basic mechanisms of food preference, food intake, and appetite." The only procedural details given were the requirement to keep a dietary record and "you will receive beverages to drink on various days," but "we cannot tell you at this time how many drinks you will receive or what they contain." The description also included notice of the requirement to attend a weekly interview at the laboratory and a schedule of remuneration, totalling $100 for satisfactory completion of the experiment.

Subjects were administered the 40-question eating attitudes test (EAT-40) (24), the 51-question Restrained Eating Questionnaire (25), and other questionnaires to assess medical history, food preferences, eating attitudes, and dietary restraint. On the basis of questionnaire responses, applicants were excluded if they were recently or currently dieting, were avoiding caffeine, had a family history of diabetes, or were pregnant.

Initial training period

An experienced registered dietician instructed each subject on how to complete dietary records. The 45-min lesson emphasized the necessity of timely and accurate record keeping and included demonstrations with food models and household measures. To augment compliance, subjects were told, "We could determine what you have eaten from analysis of urine samples" (although this was untrue). To ensure understanding of the instructions, subjects kept a practice dietary record for 2 or 3 d. The completed record was scrutinized by the dietician (with the subject present) to clarify any ambiguities and to familiarize subjects with the rigor required for keeping a dietary record. At this stage six females and eight males elected to quit the experiment. Two males who kept insufficiently detailed records were also eliminated.

Experiment design and procedure

Each subject maintained a dietary record continuously for 9 wk. During this period they received, in counterbalanced order, for 3 wk each, soda sweetened with APM, soda sweetened with high-fructose corn syrup (HFCS), or no experimental drinks. The cola-flavored soda was provided in ~300-mL glass bottles. There was an alphanumeric code on the cap or sleeve of each bottle but nothing to inform the subject of the identity of the drink. During the appropriate periods, subjects were required to drink four bottles (1135 g) of soda daily (Table 1).

At the start of the test period and then at weekly intervals, each subject was weighed (wearing casual clothes, to the nearest 100 g; the weight was not revealed to the subject), the dietary record from the previous week was examined for ambiguities, and printed instructions for the following week were given. In the two soda conditions, subjects were directed to drink four sodas a day, keep unopened bottles in a refrigerator, and record the time each bottle was consumed. In the no-soda condition, they were notified, "There are no special instructions for this week." At the end of the weekly visit, subjects were given 28 bottles of soda for the following week (if necessary). Because carrying the sodas was somewhat cumbersome, a few subjects collected them in smaller batches more frequently.

Debriefing and taste tests

At the end of the 9-wk test period, taste tests were conducted to see if subjects could recognize differences between soda containing APM and HFCS. First, each subject received a series of 16 counterbalanced triangle tests: the subject attempted to pick the disparate soda from three 10-mL samples of soda, two of one variety and one of the other. Second, the subject was allowed to drink as much as he or she wanted from four cups of soda. He or she was asked to identify whether the soda was a diet or regular type. Unbeknownst to the subject, two glasses contained APM-sweetened soda and two, HFCS-sweetened soda. Finally, we asked what the subject thought the study was about.

Analysis of dietary records

Dietary records were analyzed by use of NUTRITIONIST-3 diet-analysis software (release 3.0, N-Squared Computing, Silverton, OR) by trained personnel who were unaware of the treatment conditions. Components of foods not listed in the database were obtained directly from the manufacturers or by chemical analysis. For simplicity, we combined fructose, glucose, sucrose, and other mono- and disaccharides as "sugar." After inspection of initial results, separate values were derived for sugar in beverages (ie, soft drinks, coffee, and tea) and food (all other sources of sugar).

Results

Preliminary analyses found there were no differences between the results of the two replications of this study, so they were combined. Of the 13 female and 28 male subjects who started the study, 1 female and 5 males stopped keeping dietary records or failed to keep appointments at the laboratory. Three females were eliminated because of chicken pox, pneumonia, and relocation away from the area. Two males complained about having to drink so much soda, so they were also dropped from the study. Analyses and data presentation are based on the remaining 9 females and 21 males.

Subject characteristics

Anthropometric measures are shown in Table 2. Body mass indexes of the females and males were 25.4 ± 1.4 and 25.1 ± 0.5 kg/m², respectively, which fall just below the 75th percentile of body weight distribution (26). With the exception of four males who ate fixed meals four times per week, all subjects controlled their own food choice and meal size. There were minimal re-

<table>
<thead>
<tr>
<th>Constituent</th>
<th>APM</th>
<th>HFCS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight (g)</td>
<td>1135</td>
<td>1135</td>
</tr>
<tr>
<td>Water (mL)</td>
<td>1130</td>
<td>1000</td>
</tr>
<tr>
<td>APM (mg)</td>
<td>590</td>
<td>0</td>
</tr>
<tr>
<td>HFCS (g)</td>
<td>1</td>
<td>133</td>
</tr>
<tr>
<td>Calories (kcal)</td>
<td>3</td>
<td>530</td>
</tr>
</tbody>
</table>
Table 2

Physical characteristics of subjects*

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Female (n = 9)</th>
<th>Male (n = 21)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y)</td>
<td>28.2 ± 2.7</td>
<td>22.9 ± 0.8</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>165.5 ± 2.2</td>
<td>174.5 ± 1.2</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>69.6 ± 4.3</td>
<td>76.6 ± 2.1</td>
</tr>
</tbody>
</table>

* x ± SEM.

ports of food allergies or aversions. No subjects had extreme scores on the EAT-40, a measure of eating disorders (females 11.8 ± 2.0, males 9.9 ± 1.1). The 51-question Restrained Eating Questionnaire revealed normal eating behavior except that two females and one male had high (> 2 SD above the mean) restraint (factor 1) scores and five males had high disinhibition (factor 2) scores. None of the questionnaire responses correlated significantly with food intake or weight change during the experiment, except for a correlation between hunger (factor 3 of the Restrained Eating Questionnaire) and calorie intake during the no-soda (baseline) period (r = 0.37, p < 0.05).

Body weight

Subjects gained slightly but significantly more weight after 2 wk of drinking HFCS-sweetened soda than after the same period drinking APM-sweetened soda or no experimental soda (Appendix A). This difference was more marked after 3 wk (Fig 1). Females lost significantly more weight than did males during the control (no-soda) period. While drinking HFCS-sweetened soda, females gained weight significantly (0.97 ± 0.25 kg, p < 0.01) and males gained slightly (0.52 ± 0.23 kg, NS). While drinking APM-sweetened soda, females gained weight slightly (0.25 ± 0.29 kg, NS) but males lost weight significantly (0.47 ± 0.22 kg, p < 0.05). Thus, the effect on both sexes combined of drinking HFCS-sweetened soda was to significantly increase body weight, whereas the effect of drinking APM-sweetened soda was to nonsignificantly decrease it.

Because of the counterbalanced design of the study, the decrease in weight seen when males drank soda sweetened with APM could reflect either a direct influence of the soda or recovery from the weight gain caused by a previous period of HFCS-sweetened-soda consumption. To discriminate between these possibilities, we compared body weight changes of the three male and three female subgroups of subjects during the first 3 wk of the experiment (Appendix B) and during each of the three 3-wk periods of the study (Appendix C). The pattern of results for each of the periods was more-or-less similar to that seen overall, although because of the smaller group sizes and loss in sensitivity produced by the use of between-subject comparisons, the only significant difference for females was present during the first 3-wk period (Appendix C). Judging by the decrease in the weight of males who drank APM-sweetened soda before any possible carry-over effects of body weight gain could occur, it appears that the weight loss seen when subjects drank soda containing APM was due to the soda per se.

Food intake

Intakes of the various nutrients and of total calories were analyzed by three-way ANOVAs with factors of sex, treatment, and days (1–21 d). Separate analyses were performed either including or excluding the ingredients from the experimental sodas. All the analyses found that females consumed significantly less than did males, and there was no interaction between sex and treatment (Appendix A). None of the analyses produced a main effect or interaction involving the days factor, indicating that intakes were stable across the 21-d treatment periods. The possibility of carry-over effects from one period to another was examined using the same procedure as for body weight data. Results from the first 3-wk period were analyzed separately by using between-subject comparisons (Appendix B). The results of these analyses from a period before carry-over effects could have occurred were similar to those from the complete set of data, indicating that carry-over effects were either absent or, if present, undetectable and thus of minor significance.

Calories. Relative to calorie intake during the no-soda condition, drinking 530 kcal HFCS-sweetened soda/d produced a large and highly significant increase in total calorie intake (including calories in the experimental soda). Drinking the same volume of APM-sweetened soda decreased calorie intake. Both APM and HFCS consumption significantly reduced intake of calories from the diet (ie, calories excluding the sodas) to the same extent (by 179 and 195 kcal/d, respectively; Table 3).

The decrease in dietary calorie intake produced by drinking either form of soda was due entirely to a decrease in sugar intake (Fig 2). Drinking soda did not affect the intake of protein, fat, alcohol, or complex (nonsugar) carbohydrate (Table 3).

Sugar and soda. During the period without experimental sodas, average intake of sugar-sweetened soda was 292 ± 133 g for females and 414 ± 85 g for males. Three females and two males drank essentially no (< 25 g/d) HFCS-sweetened soda; one female and two males drank > 1135 g/d. Intake of APM-sweetened soda during the same period was 159 ± 82 g for females and 88 ± 40 g for males, which included 6 females and 16 males who did not drink any. The total intake of both types

![Figure 1](https://example.com/fig1.png)

**FIG 1.** Changes in body weight during 3-wk periods when subjects drank 1150 g/d of soda sweetened with aspartame (APM), an equal weight of soda sweetened with high-fructose corn syrup (HFCS), or had no experimental manipulation (no soda). *p < 0.05 relative to weight gain in no-soda period.
of capita soda intake (425–550 g/d) (27). There were no discern-
tal" sodas on calorie intake and body
weight. Not surprisingly, providing subjects with "experimental"
sodas of soda (488 ± 65 g/d) corresponds well to estimates of US per
capita soda intake (425–550 g/d) (27). There were no discern-
cible relationships between spontaneous soda consumption,
seen in the baseline period, and the effects of the "experimen-
tal" sodas on calorie intake and body weight.
Not surprisingly, providing subjects with "experimental"
soda displaced most (but not all) consumption of discretionary
soda. Although this was responsible for some of the decrease in
sugar intake, it could not account for it all, even if all sugar
ingested in liquid form (soft drinks, juice, tea, and coffee) was
combined (Fig 2). The remaining decrease in sugar intake pro-
duced by drinking experimental soda could not be attributed
to any particular item but appeared to be a general reduction
in all sugar-containing foods in the diet. Subjects did not eat
enough APM-sweetened products during the no-soda period
for us to determine whether drinking soda also reduced the in-
take of artificial sweeteners.

Taste tests
It was difficult to tell from the results of our tests whether
subjects could reliably distinguish between HFCS- and APM-
sweetened soda. In the triangle tests, the "odd" soda was cor-
rectly identified by females on 53 ± 4% and males on 52 ± 4%
of occasions; neither group performed significantly better than
chance (ie, 33%; for females, Z = 1.22, NS; for males, Z = 1.80,
NS). In the identification test, HFCS-sweetened soda was cor-
rectly identified by females and by males on 83 ± 8% and on
83 ± 6% of the tests, respectively. On the other hand, APM-
sweetened soda was correctly identified by females on only 39
± 11% of the tests and by males on 50 ± 7%. Because by chance
subjects would be correct 50% of the time, this implies that
subjects could identify regular but not diet soda. There was no
evidence that the effects of the sodas on calorie intake and body
weight were influenced by the subjects' ability to identify the
soda.
None of the subjects guessed the experiment's purpose; most
thought we were performing market research of some kind on
a new brand of soda. None noticed that drinking the sodas
changed their body weight or altered their patterns of food
intake or selection.

Discussion
Imposing the requirement to drink 1135 g/d of APM-sweet-
ened soda on normal-weight, freely feeding subjects decreased
calorie intake significantly (by 7%) and reduced body weight
slightly (significantly in males). This was in marked contrast to
the highly significant, 13% increase in calorie intake and sig-
nificant increase in body weight produced by consumption of
the same amount of HFCS-sweetened soda. The two types of
soda produced an identical, 33% decrease in dietary sugar in-
take (excluding the sugar in the soda), without affecting intake
of other macronutrients. This was caused in part by the "exper-
imental" sodas displacing discretionary beverages; subjects
given four bottles of soda per day have little motivation to pur-
chase and drink their own. However, drinking either form of
soda also reduced intake of all sugar-containing foods in the
diet.
Because dietary sugar intake was reduced equally by drink-
ing APM- and HFCS-sweetened soda, it seems likely that their
effects are mediated by a common property, rather than, for
example, a pharmacological action of APM (28). Both APM-
and HFCS-sweetened soda had approximately the same vol-
ume, water content, carbon dioxide content, and caffeine con-
tent and so in principle these could be responsible for the re-
duced calorie intake. However, why they should reduce sugar
intake specifically is difficult to explain. For example, high

![FIG 2](image-url)
doses of caffeine (eg, 300 mg, equivalent to 2.7 kg soda) affect short-term calorie intake but, in contrast to our results, they decrease intake of the three macronutrients equally (29). Similarly, there is no reason to suspect that ingesting large volumes of fluid or carbon dioxide should specifically suppress sugar intake. A more satisfactory explanation is that the orosensory effects of the soda inhibit the intake of other sweet foods. In short-term tests, ingestion of APM, cyclamate, or sugar solutions reduces the perceived pleasantness of sucrose (3, 30). This is usually considered to be a form of sensory-specific satiety, ie, consumption of a particular food decreases the hedonic preference for that food without affecting hedonic ratings or intake of other foods (31). In this case, however, the inhibition of intake generalizes from one sweet food item (the soda) to others, indicating a modality-specific rather than a flavor-specific satiety.

These conclusions involve several assumptions that are a consequence of the methodological limitations imparted by measuring food intake in free-living humans. Subjects were asked to maintain a diet record continuously for 9 wk. Although we repeatedly implanted them to maintain accurate diet records, it is likely that occasional lapses in record keeping occurred. Controls for the effect of such errors were imparted by collecting many days' records (which reduces the statistical impact of each aberration) and using a counterbalanced design (which spreads aberrations evenly across treatment conditions). The counterbalanced design also controlled for possible confounding factors associated with temporal effects (eg, weather, weekends, and the possibility that record keeping deteriorated as the study progressed) and for individual subject bias (a subject who overestimated the portion size of a food during one treatment would be likely to provide the same overestimate when eating the same food during a different treatment). The possible disadvantage of a counterbalanced design, which is that carry-over effects from one treatment to another might occur, did not seem to be an issue here because we could not identify a significant influence of any of the treatments on the ones that followed.

In common with several studies that have covertly increased energy intake (32-34), there was little compensation for the 530 kcal/d provided in HFCS-sweetened soda. The magnitude of the effect seen here is striking. Compared with intake during the no-soda period, calorie intake from the diet decreased by 195 kcal/d when subjects drank HFCS-sweetened soda (indicating a 37% compensation). However, the same decrease was seen when subjects drank APM-sweetened soda. Thus, with the controls for sweetness, bulk, and caffeine content afforded by the APM condition, there was 0% compensation for calories derived from the HFCS. The reason for this total failure to compensate is unclear. Judging by the increase in body weight when subjects drank HFCS-sweetened soda, most (if not all) of the energy provided by the HFCS was probably stored as fat. It has been suggested that fuel oxidation controls food intake and that fuel stored as fat bypasses this control (35). Perhaps the HFCS in a liquid vehicle is more prone to be stored as fat (and thus not to inhibit food intake) than are the solids typically used in other studies. This is consistent with work showing that sugar given in a liquid vehicle produces greater obesity more consistently than does sugar given as a solid (36).

The 7% decrease in calorie intake seen when subjects drank APM-sweetened soda does much to allay fears (37, 38) that consumption of artificial sweeteners may increase food intake and body weight gain. This possibility is based on two lines of evidence. First, in one study, women who used artificial sweeteners reported gaining more weight than did nonusers (20). However, the investigators did not attempt to discriminate between artificial sweetener use as the cause or as the effect of the reported weight gain. Second, several laboratory studies have found that subjects who taste or ingest artificial sweeteners increase appetite and short-term food intake (3, 4, 5, 7). Indeed, we found that hunger increases in subjects after they drink 500 mL APM-sweetened soda (unpublished observations, 1987). The present results suggest that these short-term changes in the motivation to eat do not accumulate into an increase in long-term calorie intake. Perhaps with repeated exposure, adaptation occurs to the appetite-stimulant effect of artificial sweeteners. Alternatively, the relatively subtle influence of sweetness may be counteracted by other controls of feeding behavior (10).

The effectiveness of artificially sweetened drinks for the control of body weight rests heavily on their intended use. When covertly substituted for sugar, APM produces a dramatic reduction in daily calorie intake and a tendency to weight loss rather than weight gain in both free-living and hospitalized subjects (present results; 22, 23). It seems reasonable to infer from this that substitution of APM for sugar can facilitate body weight control (21). More speculative is the possibility that simply adding to the diet large volumes of APM-sweetened soda reduces calorie intake and body weight. In the present experiment, drinking APM-sweetened soda decreased the calorie intake of both sexes significantly and reduced the body weight of males (but not of females) significantly. It remains to be seen whether this finding using normal-weight subjects given large volumes of APM-sweetened soda for relatively short periods (3 wk) generalizes to other populations, other food products, and more prolonged periods of artificial-sweetener consumption.

References
TORDOFF AND ALLEVA


APPENDIX A

F values from analyses of variance (results from a 9-wk period)*

<table>
<thead>
<tr>
<th></th>
<th>Sex [1, 28]</th>
<th>Treatment [2, 56]</th>
<th>Sex × treatment [2, 56]</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body weight gain</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1st week</td>
<td>0.00</td>
<td>2.85</td>
<td>2.06</td>
</tr>
<tr>
<td>2nd week (cumulative)</td>
<td>0.24</td>
<td>5.79*</td>
<td>1.62</td>
</tr>
<tr>
<td>3rd week (cumulative)</td>
<td>1.65</td>
<td>10.29*</td>
<td>3.62*</td>
</tr>
<tr>
<td>Alcohol</td>
<td>5.29*</td>
<td>0.09</td>
<td>0.71</td>
</tr>
<tr>
<td>Fat</td>
<td>4.99*</td>
<td>0.12</td>
<td>0.10</td>
</tr>
<tr>
<td>Protein</td>
<td>9.77†</td>
<td>0.28</td>
<td>0.27</td>
</tr>
<tr>
<td>All carbohydrate</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sodas included</td>
<td>8.40‡</td>
<td>42.34§</td>
<td>0.17</td>
</tr>
<tr>
<td>Sodas excluded</td>
<td>8.40‡</td>
<td>13.68§</td>
<td>0.26</td>
</tr>
<tr>
<td>Sugar</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sodas included</td>
<td>4.81†</td>
<td>72.23§</td>
<td>0.22</td>
</tr>
<tr>
<td>Sodas excluded</td>
<td>4.81†</td>
<td>23.43§</td>
<td>0.38</td>
</tr>
<tr>
<td>Nonsugar carbohydrate</td>
<td>8.96†</td>
<td>0.18</td>
<td>1.70</td>
</tr>
<tr>
<td>Calories</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sodas included</td>
<td>14.16‡</td>
<td>4.09§</td>
<td>0.29</td>
</tr>
<tr>
<td>Sodas excluded</td>
<td>14.16‡</td>
<td>15.51§</td>
<td>0.25</td>
</tr>
</tbody>
</table>

* Numbers represent F values from three-way ANOVAs with factors of sex (between subjects, male or female), treatment (within subjects, no soda, APM, and HFCS), and days of treatment (within subjects, days 1-21). F values involving days of treatment are not presented because in no case was the main effect or interaction involving days of treatment significant.
† Numbers in brackets are degrees of freedom.
‡ p < 0.01.
§ p < 0.001.
|| p < 0.05.
APPENDIX B

Means and $F$ values from analyses of variance of body weight change (kg) and food intake (kcal) of males during the first 3-wk period*

<table>
<thead>
<tr>
<th></th>
<th>No soda</th>
<th>APM</th>
<th>HFCS</th>
<th>$F_{[2,18]}$</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$(n = 5)$</td>
<td>$(n = 7)$</td>
<td>$(n = 9)$</td>
<td></td>
</tr>
<tr>
<td>Body weight gain</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1st week</td>
<td>$+0.36 \pm 0.27$</td>
<td>$-0.11 \pm 0.25$</td>
<td>$+0.11 \pm 0.25$</td>
<td>$0.77$</td>
</tr>
<tr>
<td>2nd week (cumulative)</td>
<td>$+0.24 \pm 0.29$</td>
<td>$-0.26 \pm 0.25$</td>
<td>$+0.77 \pm 0.47$</td>
<td>$1.08$</td>
</tr>
<tr>
<td>3rd week (cumulative)</td>
<td>$+0.14 \pm 0.29$</td>
<td>$-0.71 \pm 0.26$</td>
<td>$+0.56 \pm 0.40$</td>
<td>$3.74^*$</td>
</tr>
<tr>
<td>Alcohol</td>
<td>$240 \pm 85$</td>
<td>$235 \pm 75$</td>
<td>$225 \pm 75$</td>
<td>$0.55$</td>
</tr>
<tr>
<td>Fat</td>
<td>$1024 \pm 133$</td>
<td>$861 \pm 117$</td>
<td>$993 \pm 69$</td>
<td>$0.88$</td>
</tr>
<tr>
<td>Protein</td>
<td>$408 \pm 71$</td>
<td>$380 \pm 34$</td>
<td>$379 \pm 30$</td>
<td>$0.16$</td>
</tr>
<tr>
<td>All carbohydrate</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sodas included</td>
<td>$1384 \pm 140$</td>
<td>$1096 \pm 188$</td>
<td>$1614 \pm 68$</td>
<td>$3.59^*$</td>
</tr>
<tr>
<td>Sodas excluded</td>
<td>$-$</td>
<td>$-$</td>
<td>$1084 \pm 72$</td>
<td>$1.54$</td>
</tr>
<tr>
<td>Sugar</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sodas included</td>
<td>$732 \pm 92$</td>
<td>$448 \pm 102$</td>
<td>$982 \pm 36$</td>
<td>$5.20^\dagger$</td>
</tr>
<tr>
<td>Sodas excluded</td>
<td>$-$</td>
<td>$-$</td>
<td>$452 \pm 36$</td>
<td>$4.45^\dagger$</td>
</tr>
<tr>
<td>Nonsugar carbohydrate</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Calories</td>
<td>$3114 \pm 213$</td>
<td>$2478 \pm 262$</td>
<td>$3252 \pm 207$</td>
<td>$3.65^*$</td>
</tr>
</tbody>
</table>

* $\overline{x} \pm$ SEM. $F$ values are the results from one-way between-subject ANOVAs comparing the effect of the three treatments (no soda, APM, and HFCS) in male subjects during the first 3 wk of the experiment. These data are free of possible carry-over effects caused by the body weight changes produced by drinking the sodas. There were insufficient female subjects to make a similar analysis for females practical.

$^\dagger$ $p < 0.05$.

$^\dagger\dagger$ $p < 0.01$.

$^\dagger\dagger\dagger$ $p < 0.10$.

APPENDIX C

Effect on body weight of drinking soda (1135 g/d) sweetened with APM or HFCS over each 3-wk period*

<table>
<thead>
<tr>
<th></th>
<th>No soda</th>
<th>APM</th>
<th>HFCS</th>
<th>$F$</th>
<th>$p$</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$(n = 5)$</td>
<td>$(n = 7)$</td>
<td>$(n = 9)$</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Females</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1-3 wk</td>
<td>$-0.80 \pm 0.30 [3]$</td>
<td>$-0.68 \pm 0.23 [3]$</td>
<td>$+0.57 \pm 0.39 [3]^\dagger$</td>
<td>$F_{[2,14]} = 5.27$</td>
<td>$&lt;0.05$</td>
</tr>
<tr>
<td>4-6 wk</td>
<td>$+0.39 \pm 0.31 [3]$</td>
<td>$+0.36 \pm 0.35 [4]$</td>
<td>$+0.65 \pm 0.25 [2]$</td>
<td>$F_{[2,14]} = 0.45$</td>
<td>NS</td>
</tr>
<tr>
<td>7-9 wk</td>
<td>$-0.67 \pm 0.07 [3]$</td>
<td>$-0.20 \pm 0.50 [2]$</td>
<td>$+0.63 \pm 0.53 [4]$</td>
<td>$F_{[2,14]} = 2.81$</td>
<td>NS</td>
</tr>
<tr>
<td>Overall</td>
<td>$-0.36 \pm 0.14 [9]$</td>
<td>$-0.11 \pm 0.23 [9]$</td>
<td>$+0.61 \pm 0.19 [9]^\dagger$</td>
<td>$F_{[2,16]} = 14.3$</td>
<td>$&lt;0.001$</td>
</tr>
<tr>
<td>Males</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1-3 wk</td>
<td>$+0.14 \pm 0.29 [5]$</td>
<td>$-0.71 \pm 0.26 [7]^\dagger$</td>
<td>$+0.56 \pm 0.40 [9]$</td>
<td>$F_{[2,18]} = 3.74$</td>
<td>$&lt;0.05$</td>
</tr>
<tr>
<td>4-6 wk</td>
<td>$+0.42 \pm 0.25 [6]$</td>
<td>$-0.38 \pm 0.39 [8]^\dagger$</td>
<td>$+0.72 \pm 0.21 [7]$</td>
<td>$F_{[2,18]} = 4.03$</td>
<td>$&lt;0.05$</td>
</tr>
<tr>
<td>7-9 wk</td>
<td>$-0.07 \pm 0.26 [10]$</td>
<td>$+0.11 \pm 0.26 [6]^\dagger$</td>
<td>$+0.64 \pm 0.39 [5]^\dagger$</td>
<td>$F_{[2,18]} = 3.61$</td>
<td>$&lt;0.05$</td>
</tr>
<tr>
<td>Overall</td>
<td>$+0.12 \pm 0.19 [21]$</td>
<td>$-0.35 \pm 0.22 [21]^\dagger$</td>
<td>$+0.64 \pm 0.20 [21]^\dagger$</td>
<td>$F_{[2,40]} = 6.06$</td>
<td>$&lt;0.01$</td>
</tr>
</tbody>
</table>

* $\overline{x} \pm$ SEM. $n$ in brackets. Group sizes differ slightly from period to period because of subject dropout from the counterbalanced design.

$^\dagger$ Different from no-soda control group, $p < 0.05$ (between-group comparison).

$^\dagger\dagger$ Different from weight gain during no-soda control period, $p < 0.05$ (within-subject comparison).