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Sensory Responses, Dietary-Induced Obesity and Biochemical Values in Sprague-Dawley Rats[1]

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GRINKER, J. A. AND W. D. BLOCK. *Sensory responses, dietary-induced obesity and biochemical values in Sprague-Dawley rats.* BRAIN RES BULL **27**(3/4) 535–540, 1991.—Food intake and body weight gain variability in Sprague-Dawley (SD) rats exposed to a palatable high-fat diet were examined in relation to sensory responses and biochemical parameters in two experiments. In the first experiment, varying sucrose concentrations (4–32% wt./vol.) were randomly presented for 20 minutes to ad lib chow-fed rats. Each rat's sensory response was expressed as Beta (B), or the slope of the regression between solute intake and concentration, and used to assign rats to diet groups. In the second experiment, responsiveness to fat emulsions (1–37%) were similarly measured and categorized. In both experiments sensory responses to sucrose were significantly related to weight gain/fatness on the high-fat diet (lab chow-corn oil). Sensory responsiveness to the fat emulsions was unrelated to sucrose responsiveness or to high-fat feeding. Biochemical parameters (insulin, cholesterol, triglycerides, lipoproteins) reflected increased caloric (fat) intake, as well as sucrose responsiveness. Predictors (sensory responses, biochemical values) of response to chronic (4 months) or short-term (<2 months) high-fat diets are discussed.

Dietary-induced obesity Taste Sucrose Cholesterol Triglycerides Insulin

SUSCEPTIBILITY to diets leading to the development of obesity has been studied among different rat strains; the Osborne Mendel rat is highly responsive to high-fat feeding and sucrose solutions, while the S 5B/Pl strain is resistant (20,21). Significant variability in weight gained, percent fatness, or adipose cellularity in response to such diets has also been reported in studies of rats of the same genetic strain (5–9, 15). Sprague-Dawley (SD) rats show great variability in food intakes and body weight gains when fed obesity-producing diets, e.g., high fat or high carbohydrate (CHO). Even prolonged exposure (3–5 months) to these diets may not produce marked obesity in all rats, compared with rats on control diets (5, 7, 15).

Dietary responsiveness among several rat strains has been related to a variety of behavioral measures; "tail pinch"-induced feeding latency, magnitude of acoustic startle reflex, meal sizes or sensory responses to sweet solutions have all been positively related to subsequent responsiveness to obesity-producing diets (5–9). Glucose-induced activitation of the sympathetic nervous system was related prospectively to dietary-induced obesity (DIO) among rats given chronic exposure to a condensed milk diet (16).

Initial studies reported variations of 30% to 130% gain in body weight among SD rats on a supermarket diet, compared with weight gains of 15–30% on laboratory (lab) chow. Early over/under-nutrition (litter size) did not alter susceptibility, although behavioral/nutritional factors were predictive of the development of DIO. The meal patterns (frequency and size) of rats on lab chow allowed division of the rats into "gorgers" and "nibblers" (rats with smallest-sized meals). Gorgers gained more weight (44%) than did nibblers (27%) when subsequently fed the supermarket diet and had enhanced fat deposition and increased adipose cell number in the retroperitoneal (RP) depot (5).

In the present study, we measured the sucrose intake of SD rats given brief access to a series of sucrose solutions (range 4–32% wt./vol.). The sensory responsiveness of each rat was defined as the slope of the regression between solute intake, and concentration and was related to percent weight gain on high-fat feeding. In a second study, we compared sensory responsiveness to fat-CHO emulsions (liquid dairy products of 1–37% wt./vol. fat) with sensory responsiveness to sucrose and to subsequent adiposity following high-fat feeding. Biochemical data (e.g., liproproteins, triglycerides, insulin) in blood plasma from rats at sacrifice and, in Experiment 2 during both baseline lab chow and high-fat feeding, are also reported.

EXPERIMENT 1

Method

Animals. Forty (one-month-old) male SD rats (Charles River) constituted the initial pool from which two experimental and one control group were selected. Weights ranged from 90–100 grams at arrival. Rats were individually housed in suspended steel-wire cages in a laboratory maintained at 20±2°C with a 12-hour light-dark cycle (0700–1900). Rats were fed ad lib water and Purina lab chow (containing 23.4% protein, 4.5% fat and 52.1% CHO) (3.3 kcal/g). Daily food and water intake and body weights were monitored on all rats. After two months on the dietary regime, rats were tested for sensory responses to sucrose and assigned to different diet conditions at 6 months.

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TABLE 1

SENSORY RESPONSIVENESS TO SUCROSE AND FAT SOLUTIONS

Group	N	Mean and Range of Sensory Responsiveness Sucrose Solutions*	N	Fat Emulsions†
High Responders				
Exp. 1	13	0.1605 (.1308–.2080)		
Exp. 2	16	0.1051 (.09002–.14553)	15	13.528 (9.2366–28.038)
Medium				
Exp. 1	9	0.1165 (.1078–.1258)		
Exp. 2	15	0.0790 (.06855–.08835)	21	8.6044 (6.4271–11.692)
Low				
Exp. 1	13	0.0855 (.0345–.1033)		
Exp. 2	16	0.0411 (.01324–.06808)	11	6.1098 (4.3101–8.2181)

*Expressed as the slope of the regression (Beta) of the amount of solute (g of sucrose) consumed as a function of concentration (4, 8, 16, 32% wt./vol.). For this analysis the 'y' axis intercept was ignored. Goodness of fit (r^2) was .70 or better.

†Expressed as the slope of the regression (Beta) of the amount of solute (milk fat) consumed as a function of concentration (dairy products: skim, regular milk, half and half and whipping cream). For the purpose of this analysis the 'y' axis intercept was ignored. Inconsistent rats ($r^2 < .70$) were placed in the medium group.

Procedures

Sensory responsiveness. Sucrose solutions in varying concentrations to 32% wt./vol. were used (4, 8, 16, 32%). Tests were conducted for 20 minutes in the afternoon, immediately after regular lab chow and water had been removed. At the end of the testing period, regular food and water were restored. Solutions were always presented in a paired comparison procedure, with water and one sucrose solution available. To control for side preference, each sucrose solution was presented twice, on both the right and left sides of the cage, and the order of the concentrations was randomized. Final data consisted of the average intake/concentration converted into grams of solute consumed. An individual rat's sensory responsiveness was defined as the regression of solute intakes (g of sucrose) against sucrose concentrations, i.e., the slope (beta). A steeper slope indicated a higher sensory response. Since the magnitude of change or increase in intake with increasing sucrose concentration was the measured variable, the 'y' axis intercept was ignored for purposes of analysis and assignment to experimental condition. After analysis of the distribution of the individual rat's regression scores, they were divided into 'high,' 'medium' and 'low' sucrose taste responders (HST, MST and LST) and assigned to one of three different diet groups: one group (n = 15) was changed from laboratory chow to a high-fat diet (HFI) for the remaining four months. This diet consisted of powdered Purina lab chow, supplemented with 30% wt./wt. Crisco (5.01 kcal/g). A second group (n = 12) remained on lab chow for 2 additional months and was then transferred to the high-fat diet (HFII) for approximately 2 months. The third group (control-C) (n = 9) remained on ground lab chow for the entire period. Assignment to experimental conditions was made on the basis of sensory responsiveness, as well as prior average weight gain.

Each of the three diet groups included high and low sucrose taste responders, with equivalent food intakes and body weight gains during lab chow feeding. The HFI group (4 months on high fat) consisted of 5 HST responders, 5 MST responders and 5 LST responders. The HFII group (2 months on high fat) consisted of 5 each from the HST and LST responding groups and 2 rats from the MST group, one of which subsequently began losing weight and was removed from the study. The C group consisted of 3 rats from each of the 3 taste response groups (i.e., 3 HST, 3 LST and 3 MST). Due to the theoretical interest of the experiment and the limited number of rats in the MST-HFII group, all sensory comparisons were limited to HST vs. LST groups.

Biochemical and physiological procedures. At approximately 10 months of age, all rats were sacrificed by guillotine after an overnight fast (12 hours). Truncal blood was placed in a tube containing NaEDTA (1 mg/ml), centrifuged at 800 × g for 10–15 minutes. Aliquot portions of each plasma specimen were stored at 4°C until analyzed. Fat depot wet weights from four depots (RP, mesenteric-MES, epididymal-EPID, and inguinal-ING) and the weights of the gastrointestional (GI) tract and liver were determined.

The various plasma lipoprotein subfractions were separated by the combination of density gradient and dextran precipitation (10, 17, 25). Plasma cholesterol (CHOL), triglycerides (TRIG) and lipoprotein fractions HDL CHOL and HDL_3 CHOL were determined by enzymatic techniques (1,3). HDL_2 CHOL was determined by subtraction.

Statistical analysis. Data were analyzed by analysis of variance (ANOVA) for sensory groups, diet conditions and MANOVA for selected parameters (2, 4, 26). The MST responding group in the HFII condition was eliminated for analysis of all biochemical and physiological variables, and these comparisons included only HST and LST responders among the three diet conditions. Post hoc *t*-tests were done on selected parameters with appropriate controls (Bonferroni correction).

Results

Rats were categorized into one of three sensory response groups on the basis of the individual beta score (see Table 1). Table 1 (Experiment 1) shows mean values and ranges for rats in the sensory groups. The medium group included all rats whose scores were intermediate. Rats (n = 4) that failed to meet the criteria of a goodness of fit of an r^2 of at least .70 were eliminated.

TABLE 2

MEAN (SD) BODY AND FAT DEPOT WET WEIGHTS AND PLASMA VALUES (CHOL, TRIG, HDL CHOL) FOR DIET CONDITIONS BY SUCROSE TASTE GROUP

		Experimental Condition		
	Taste Group	High-Fat I*	High-Fat II†	Control‡
Body wt. (g)	High	817.52 (82.88)	692.14 (104.50)	601.43 (24.35)
	Low	731.72 (70.50)	655.88 (29.00)	550.63 (48.00)
		Fat Depots (g)		
ING	High	14.74 (3.15)	9.60 (3.85)	5.44 (1.56)
	Low	12.05 (2.61)	8.67 (1.79)	5.75 (1.89)
MES	High	28.36 (6.42)	17.98 (5.26)	8.02 (0.64)
	Low	23.74 (6.02)	16.50 (5.48)	9.88 (2.53)
		Plasma Values (mg/dl)		
CHOL	High	89.20 (12.79)	93.67 (22.92)	97.00 (7.81)
	Medium	83.67 (7.39)		80.66 (5.69)
	Low	74.80 (9.09)	74.67 (11.00)	82.00 (15.13)
TRIG	High	115.80 (18.00)	161.17 (33.04)	67.00 (10.39)
	Medium	105.67 (31.05)		94.33 (14.22)
	Low	99.80 (17.51)	119.17 (43.48)	81.00 (14.18)
HDLC (total)	High	55.00 (9.03)	55.33 (8.26)	57.33 (11.24)
	Low	46.00 (6.04)	49.33 (6.89)	53.33 (5.69)
$HDLC_2$	High	26.50 (5.36)	27.00 (9.76)	37.00 (16.46)
	Low	19.00 (4.69)	24.83 (5.00)	21.67 (4.51)
$HDLC_3$	High	26.50 (3.64)	30.50 (3.78)	20.33 (10.69)
	Low	26.40 (4.93)	24.33 (5.28)	31.67 (6.51)

*Six months on lab chow followed by 4 months on lab chow + 30% corn oil.
†Eight months on lab chow followed by 2 months on lab chow + 30% corn oil.
‡Ten months on lab chow.

Table 2 shows mean body weights for each group of rats at sacrifice. ANOVA for rats in all conditions showed main effects of both diet condition and sensory groups. Rats in the HFI group were heavier than rats in the HFII group, which were heavier than C rats; Diet condition, $F(2,20)=15.18$, $p<0.001$. However, HST rats were heavier than LST rats; Taste groups, $F(1,20)=4.10$, $p=0.05$; $t=3.96$ $p<0.03$. There were significant differences between the HST and LST responders in both high-fat diet conditions in weight gained/week and % weight gain; $F=5.121$, $p<0.03$; $F=2.90$, $p=0.05$. Weight differences were primarily due to differences in food intake: During the first 11 weeks of diet, HFI rats consumed an average of 8343.2 (±595) calories, compared with 7487 (±784) calories by C rats, and 7752.7 calories (±595) by HFII rats (fewer weeks on high fat) during this same time. The average caloric intake/week was significantly different between diet groups, $F(2,406)=69.07$, $p<0.001$. Both HFI and HFII groups continued to eat more than the C group until the termination of the experiment. In addition, HST responders consumed more calories than MST or LST responders, $F(2,406)=6.51$, $p<0.01$.

There were significant differences in the weights of the various fat depots, $F(3,63)=134.8$, $p<0.001$, as well as among diet conditions, $F(2,21)=24.52$, $p<0.001$. ING and MES fat depots showed a main effect of experimental condition, $F(2,21)=22.85$, $p<0.001$, as well as an interaction among experimental condition and fat depots, $F(2,21)=9.50$, $p<0.01$ (see Table 2). Both ING and MES depots showed significant increments as a consequence of high vs. low sucrose responsiveness, $t(26)=5.43$, $p<0.01$; $t(26)=5.21$, $p=0.01$. Rats showing high sucrose taste responsiveness were slightly fatter. In the EPID and RP depots, only diet condition differentiated among these tissue weights. EPID weights ranged from 27.2 and 25.3 g in the HFI-HST and LST groups, respectively, and 11.3 and 10.3 in the C HST and LST groups, respectively. Similarly, the RP depot ranged from 54.4 g in the HFI condition, to 17.7 in the C condition.

Fasting liver and GI tract weights reflected the increased fatness produced by the high-fat diet and the differing amount of time on this diet, $F(2,21)=5.38$, $p<0.02$. Liver wet weights ranged from 22.2 g in the HFI-HST condition, to 15.9 g in the C-LST condition. There were no significant taste differences, $F(1,21)=2.56$, $p=0.125$. GI tract weights ranged from 23.2 g to 21.8, with no differential effects of diet condition or taste group.

Biochemical parameters. Table 2 shows mean plasma values for total CHOL and TRIG, and total HDLC (heavy density lipoprotein CHOL) and the subfractions, for all diet conditions for HST and LST groups. There were separate effects for taste groups on CHOL levels (MANOVA), $F(1,24)=5.93$, $p<0.023$, and diet conditions on TRIG, $F(1,24)=6.12$, $p<0.02$. Within both HFI and II conditions there was a significant effect for HST vs. LST rats on CHOL, $t(20)=2.71$, $p<0.03$, and total HDLC, $t(20)=2.35$, $p<0.03$. HST responding rats had higher CHOL and total HDLC levels than LST rats on the high-fat diet. TRIG differed significantly between both diet conditions and the control group, $t(26)=4.01$, $p<0.01$. There were also significant differences in $HDLC_2$, $F(1,27)=4.43$, $p<0.05$, between HST and LST responders and a significant taste × diet condition interaction for $HDLC_3$, $F(2,27)=3.27$, $p<0.05$. Differences in

TABLE 3

MEAN (SD) ORGAN AND TISSUE WET WEIGHTS AND PLASMA VALUES OF HIGH-FAT DIETARY RESPONSE GROUPS AND CONTROL GROUP

	High-Fat Diet Responders*			
Organ/Tissue (g)	High (n = 12)	Medium (n = 14)	Low (n = 12)	Control† (n = 9)
Liver	19.35 (2.1)	18.63 (2.2)	16.80 (2.4)	18.31 (3.0)
GI Tract	34.42 (4.6)	33.61 (4.4)	29.86 (4.2)	39.21 (4.8)
Heart	1.53 (0.13)	1.66 (0.14)	1.46 (0.10)	1.47 (0.21)
ING	7.81 (2.8)	6.91 (1.6)	4.57 (2.0)	3.28 (1.3)
MES	19.48 (5.8)	14.55 (4.2)	10.19 (3.5)	8.14 (3.5)
EPID	19.69 (6.1)	16.12 (3.4)	13.69 (4.8)	9.04 (3.9)
RP	31.26 (10.4)	25.06 (4.8)	18.78 (6.0)	11.76 (4.4)
	Baseline Plasma†			
CHOL (mg%)	71.00 (18.18)	66.50 (12.17)	66.67 (10.66)	69.78 (13.90)
TRIG (mg%)	67.00 (16.01)	70.86 (13.44)	64.17 (14.24)	63.22 (10.43)
Insulin (u-unit/ml)	3.42 (2.74)	2.84 (1.61)	4.07 (4.62)	3.06 (2.45)
	Experimental Plasma§			
CHOL (mg%)	86.50 (14.43)	80.93 (22.08)	76.50 (11.00)	80.78 (17.62)
TRIG (mg%)	96.00 (26.61)	91.43 (25.04)	81.42 (18.38)	84.22 (19.71)
Insulin (u-unit/ml)	25.06 (14.89)	24.56 (16.57)	23.13 (14.01)	8.14 (11.13)

*Defined on the basis of dietary responsiveness (amount of weight gain) to high-fat diet (6 weeks) of lab chow plus 30% wt./wt. Crisco (corn oil).

†Remained on ground Purina lab chow until the end of the experiment. Rats were sacrificed nonfasting which is reflected in the weights of GI tract.

‡Fasting baseline measures taken on lab chow prior to high-fat feeding.

§Fasting measures taken after experimental diet for 6 weeks (high-fat) or lab chow (control group).

total HDLC were probably a consequence of differences in $HDLC_2$. Differences in TRIG were produced as a consequence of diet condition or duration of exposure to high-fat feeding. Differences in total CHOL, however, reflected differences in taste responsiveness.

Discussion

Longer exposure (4 months) to a high-fat diet induced greater increments in body weight and fatness and greater variability than a shorter exposure (2 months). Since HST responders gained more weight and fat than LST responders, taste responsiveness to sucrose solutions while on lab chow may predict the degree of dietary and biochemical responsiveness to high-fat feeding. Whereas the high-fat diet had a major effect on TRIG levels, CHOL levels may have also differed as a function of taste responsiveness. There were also significant interactions between duration of exposure to high-fat feeding and taste responsiveness, suggesting that longer exposure to high-fat feeding may potentiate the predictive value of the taste responsiveness to sucrose.

EXPERIMENT 2

This experiment added several dependent measures that were not included in the first experiment. Sensory responses to fat were included to ascertain whether the response to one type of fat, i.e., corn oil in the diet, was related to responses to other fats. Rats' sensory responses to fat emulsions or liquid dairy products were measured, and rats classified in taste groups using methodology similar to that for sensory responses to sucrose. Sensory responses to sucrose were also measured. In addition, blood samples were taken for biochemical determinations during control lab chow feeding and during exposure to the high-fat diet. Procedures were similar to those outlined above for the first experiment.

Method

Animals. Forty-seven 33-day-old male SD rats (Charles River) were individually housed in stainless steel cages in a temperature-controlled room (20 ± 2°C) on a 12-hour light-dark cycle (0800–2000). Mean weights and ranges were 105.2 g and 93–119 g, respectively, at arrival. After a two-week adaptation period on Purina lab chow pellets, body weight and food and water intake were measured daily. During the last three days of the adaptation period, rats were switched to ground Purinia lab chow, which was used for the remainder of the experiment. Sensory tests were then conducted.

Sensory response to sucrose. Sucrose solutions in water ranging from 4 to 32% wt./vol. were randomly presented to all rats for 20 minutes, immediately after food and water had been removed. At the end of the 20 minutes, regular food and water were restored. Only one solution and water were simultaneously tested at each trial (see the Method section—Experiment 1). The slope of the regression line (beta) of grams of solute consumed as a function of sucrose concentration was individually determined for all rats. Rats were then separated into low (LST), medium (MST), and high (HST) sucrose taste responders.

Sensory responses to fat. Liquid dairy products ranging from less than 1 to 37% fat [skim milk (0.5%), regular milk (3.25%), half & half (10.5%), and heavy cream (37%)] were used to determine sensory responses to fat. Fat emulsions were tested using the procedures for sucrose responsiveness (see above). An individual rat's response to the fat emulsions was similarly analyzed, and rats separated into low (LFT), medium (MFT), and high (HFT) fat taste responders.

Procedures

The high-fat diet (HF) was identical to that in Experiment 1 (30% Crisco added to Purina lab chow). The experimental condition (HF) consisted of 38 rats selected to represent all variations of sucrose and fat sensory responsiveness. The control condition (C) (n = 9) was selected to include major variations of

sucrose and fat responsiveness (high-low and low-high) and remained on ground Purina lab chow. Starting mean weights (SD) for the HF and C conditions were 477.3 (50) g and 474.6 (24) g, respectively. After six weeks on high-fat feeding, rats in the HF condition were separated into low (LHF) (n = 12), medium (MHF) (n = 14), and high (HHF) (n = 12) dietary responders, based on the amount of food consumed and the change in percent body weight since the beginning of high-fat feeding. Body weight changes averaged 150 g, 111 g, and 85 g in the HHF, MHF, and LHF groups, respectively, compared with an average of 61 grams for C condition rats.

Biochemical measures. Fasting blood samples were taken after an overnight fast, one day prior to the initiation of high-fat feeding, and again after six weeks of high-fat or control diets. Blood samples (2 ml) were obtained through tail bleedings, and analyzed for plasma insulin, TRIG, total CHOL, total HDLC and HDL_2C and HDL_3C subfractions (see Experiment 1 for procedural details). Plasma insulin was determined by radioimmunoassay (11).

Tissue and organ weights. All rats were sacrificed (nonfasting) by decapitation seven weeks after the initiation of the high-fat diet. [The brain was immediately removed and frozen for later determination of catecholamine binding (8).] Fat depot wet weights (ING, MES, RP and EPID) were determined. The liver, GI tract, and heart were dissected and wet weights determined.

Statistics. Data were analyzed by *t*-tests, ANOVA and least squares analyses. Data were initially analyzed as a function of dietary response and subsequently by taste responsiveness to both sucrose and to fat. Post hoc *t*-tests and Bonferroni corrections were performed where appropriate.

Results

Table 1 shows sensory responses to sweet solutions and fat emulsions by rats tested during chow feeding (see Table 1). Inconsistent responders, i.e., $r^2 < .70$, were placed in the medium group. Only 4 rats failed to meet the r^2 criterion for sucrose responses. Similarily, rats that presented inconsistent responses to the fat emulsions were also placed in the medium group. Only 26 rats met the criterion of $r^2 > .70$ and, therefore, all others as well as true mediums were placed in the medium group (n = 21). The rat's individual sensory response to fat was not correlated with that rat's individual response to sucrose.

Six weeks were sufficient to create a significant difference between the HF and C conditions in terminal body weights. Rats in the HF condition averaged 593.32 (SD = 76.42) g, while rats in the C condition averaged 533.2 (35.09) g. Based on percent weight changes, it was possible to divide rats easily into three groups of dietary responders. The cumulative weight change ranged from 31.3%, 24.9%, 18.0% among rats in the HHF, MHF and LHF response groups, to 12.8% among C rats. Again, these increments in body weight or fatness were primarily a consequence of increased food intake. The average weekly food intake of rats in the high-fat diet condition ranged from 24.1–28.4 g in the HHF, 22.2–26.9 g in the MHF, and 19.5–23.3 g in the LHF groups. Weight gain and food intake were correlated ($r = .36$, $p < 0.05$). When data were analyzed by taste response groups, only sucrose responsiveness showed a relationship to weight/fatness increments, $F = 4.52$, $p < 0.03$, for high vs. low sucrose responders on the high-fat diet. Sensory responsiveness to fat emulsions was unrelated to dietary response on high-fat ($p > 0.10$). However, both terminal body weight and body weight gain were significantly correlated with sucrose responsiveness (regression $p < 0.045$ for terminal body weights). As predicted, sensory responsiveness to sucrose and to fat were unrelated ($r = .2587$, $F = 3.229$, $p > 0.10$).

Fat depot wet weights also showed marked differences between HF and C conditions (see Table 3). In addition, there were significant differences within HF rats as a function of taste response groups. Average MES fat depot weights for experimental rats differed significantly as a function of sucrose responsiveness ($p < 0.022$). Mean weights were 11.86 (SD = 3.05) g, 18.36 (6.43) g, and 15.00 (6.33) g for LST (n = 14), MST (n = 10), and HST (n = 14) responders, respectively, on high-fat feeding. ING wet weights showed a similar pattern. A six weeks' duration of high-fat feeding was sufficient to create significant differences in organ weights of GI tract and liver as a function of the rat's response to the high-fat diet (HHF > LHF group).

Since the duration of high-fat feeding was only 6 weeks, most biochemical parameters (CHOL and TRIG) showed changes primarily as a function of increased weight or fatness (see Table 3). Analysis of pooled plasma samples for CHOL subfractions suggested a slight increase with increased responses to the high-fat diet. These results were obtained in both HF and C groups. Only insulin was significantly related to sucrose responsiveness. Initially, sucrose responsiveness showed a borderline, but nonsignificant, positive relationship with fasting insulin levels ($r = .293$, $p = 0.0745$). By the termination of high-fat feeding, however, there was a significant negative correlation between sucrose responsiveness and insulin levels ($r = -.388$, $p < 0.02$).

GENERAL DISCUSSION

Results confirm the first experiment regarding the effectiveness of utilizing sucrose responsiveness, or the slope of the regression line of solute intake, to sucrose concentration as a predictor of response to high-fat feeding. Higher sucrose responders gained more weight and had significantly increased fatness. The sensory responses to fat emulsions were unrelated to subsequent weight/gain in fatness on this unique high-fat diet, suggesting that the type of fat sampled (liquid dairy products) may not generalize to responsiveness to a composite high-fat diet consisting of liquid Crisco added to lab chow. Alternatively, fat responsiveness may be unrelated either to weight gain on a high-fat diet or to changes in biochemical parameters. This latter interpretation is supported by suggestions (unpublished data, Spiegel, Stellar) that human subjects' preferences for fat are more stimulus specific, whereas preferences for a given level of sweetness generalize from one model food system to another. Preferences for fat are learned as a result of texture and flavor dimensions, whereas sweet is one of the four basic tastes for both humans and rats.

The finding of a significant negative relationship between sucrose responsiveness and fasting insulin levels in rats at the termination of high-fat feeding, suggests that 6 weeks on a high-fat diet is sufficient to induce not only increased fatness, but also insulin resistance. Certainly, obesity in man and animals is often accompanied by insulin abnormalities, which are often reversed with weight loss.

The relationship between sucrose responsiveness and body weight/fatness gains proved robust, even with 6 weeks of high-fat feeding to young rats. Sucrose responsiveness might be considered as a predictor of response to obesity-producing diets. Thus, other parameters could be analyzed in high- and low-sensory responders to sucrose prior to, or even without placement, on a high-fat diet. The data from the combined experiments suggest that age (older), as well as duration or length of time on the high-fat diets, interact with sensory responsiveness to sucrose. Older rats maintained for longer periods of time on a

high-fat diet appear to show the most consistent and sustained relationship. These data are supported by data from other studies (14,22) in which older male rats (or mice) were more susceptible to dietary-induced obesity, and were also more feed-efficient than younger rats. Other investigators have examined age-related changes in lipid and carbohydrate metabolism in genetically obese mice (18), as well as age, sex and dietary effects among rats exposed to obesity-producting diets (12, 13, 22, 24).

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