pattern of results observed with threshold intensities.

Histological analysis indicated that the hypothalamic electrode in all three animals was located in the MFB slightly dorsolateral to the fornix. The dorsal hippocampal electrodes were located adjacent to pyramidal field CA3.

DISCUSSION

Two major findings emerge from the present study. First, unlike normal eating and drinking, which are associated with irregular or desynchronized hippocampal activity, stimulus-bound consummatory behavior is associated basically with persistence of synchronized hippocampal activity. This difference probably does not reflect variation in the intensity of the disposition to eat or drink. Rather it suggests some differences in the electrophysiological state produced by the two sets of conditions. In stimulus-evoked behavior appetitive mechanisms may continue to operate during consummatory responses unlike the normal sequencing of appetitive and consummatory phases. It is also possible that negative properties of the stimulation, as evidenced by offset responses in tests of preferred duration (Pond & Schwartzbaum, 1969), persist and contribute to the unusual agitated quality of stimulus-bound consummatory behavior.

At the same time, the results also indicate that stimulus-bound consummatory behavior can, within limits, attenuate the frequency of evoked theta patterns. This effect may relate to a reduction in feedback from locomotor movement that is otherwise elicited—paralysis with d-tubocurarine, e.g., produces similar effects (Pond & Schwartzbaum, 1970; Routtenberg & Kramis, 1968)—although consummatory activity may operate in other ways to produce “deactivating” effects on nonspecific systems that may counteract properties of LH stimulation. Evidence of such deactivating effects is available from work with cats (Marczynski & Hackett, 1969), and we have observed changes in sensory evoked response in rats as they switched from operant to consummatory behavior that are consistent with such a supposition. In any case, the attenuation of evoked theta frequency may signify some degree of change in appetitive state and/or negative properties of brain stimulation which may relate to increased preferred duration of brain stimulation observed during evoked consummatory-type behavior (Mendelson, 1969).

Finally, it is worth noting that the present data cast doubt on the generality of recent findings (Paxinos & Bindra, 1970) that conditioned immobility to

rewarding brain stimulation can block hippocampal theta otherwise evoked by the stimulation. It is likely that behavioral patterns expressed during stimulation can only alter evoked theta rhythms within limits. At higher intensities of stimulation, theta patterns may be attenuated but not blocked. Stimulus-evoked theta is not simply a function of the feedback from the evoked behavior.

REFERENCES

- CHRISTOPHER, M., & BUTTER, C. M. Consummatory behaviors and locomotor exploration evoked from self-stimulation sites in rats. *Journal of Comparative & Physiological Psychology*, 1968, 66, 335-339.
- GRASTYAN, E., KARMOS, G., VARECZKEY, L., & KELLENYI, L. The hippocampal electrical correlates of the homeostatic regulation of motivation. *Electroencephalography & Clinical Neurophysiology*, 1966, 21, 34-53.
- KRAMIS, R. C., & ROUTTENBERG, A. Rewarding brain stimulation, hippocampal activity, and foot-stomping in the gerbil. *Physiology & Behavior*, 1969, 4, 7-11.
- MARCZYNSKI, T. J., & HACKETT, J. T. Post-reinforcement electrocortical synchronization and facilitation of cortical somato-sensory evoked potentials during instrumentally conditioned appetitive behavior in the cat. *Electroencephalography & Clinical Neurophysiology*, 1969, 26, 41-49.
- MENDELSON, J. Lateral hypothalamic stimulation: Inhibition of aversive effects by feeding, drinking and gnawing. *Science*, 1969, 166, 1431-1433.
- PAXINOS, G., & BINDRA, D. Rewarding intracranial stimulation, movement and the hippocampal theta rhythm. *Physiology & Behavior*, 1970, 5, 227-231.
- POND, F. J., & SCHWARTZBAUM, J. S. Preferred duration of rewarding brain stimulation in rats and interstimulus interval. *Physiology & Behavior*, 1969, 4, 911-916.
- POND, F. J., & SCHWARTZBAUM, J. S. Hippocampal electrical activity evoked by rewarding and aversive brain stimulation in rats. *Communications in Behavioral Biology*, 1970, in press.
- ROUTTENBERG, A. Hippocampal correlates of consummatory and observed behavior. *Physiology & Behavior*, 1968, 3, 533-535.
- ROUTTENBERG, A., & KRAMIS, R. C. Hippocampal correlates of aversive midbrain stimulation. *Science*, 1968, 160, 1363-1365.
- VALENSTEIN, E. S., COX, V. C., & KAKOLEWSKI, J. W. The hypothalamus and motivated behavior. In J. T. Tapp (Ed.), *Reinforcement and behavior*. New York: Academic Press, 1969. Pp. 242-285.
- VANDERWOLF, C. H. Hippocampal electrical activity and voluntary movement in the rat. *Electroencephalography & Clinical Neurophysiology*, 1969, 26, 407-418.

Effects of ingesting nonnutritive sweet substances on subsequent sucrose consumption

ELVIS C. JONES

Frostburg State College, Frostburg, Md. 21532

An experiment with rats was conducted to verify and further study the drive-reducing effects of nonnutritive sweet substances. Ingestion of 1 ml of .25% pure saccharin or .6% sodium cyclamate solutions significantly suppressed subsequent consumption of a 16% sucrose solution. The data also indicated that the suppression effect following consumption of saccharin disappears within 5 min. It was concluded that the transitory effects of saccharin consumption may account for discrepancies between previous studies on the drive-reducing effects of saccharin.

Miller, Murray, & Roberts (1955), found that sodium saccharin suppresses subsequent food consumption if the saccharin is taken orally, but not when introduced directly into the stomach. The suppression effect was attributed to oral-pharyngeal factors.

Important details of the Miller et al experiments are not generally available, and there has been little subsequent research directly related to this effect. The bearing this phenomenon has on theories of reinforcement is such that a general verification and extension seems worthwhile, especially since these findings

are at odds with those reported by Hausmann (1933) and by Smith & Duffy (1957). These investigators found that saccharin did not significantly decrease the amount of food ingested. The discrepancy between these findings may be due to the fact that the latter two experiments measured consumption over long periods. If the effect of saccharin is highly transitory, it may have been masked in these studies.

The present experiment represented an attempt to verify the apparent drive-reducing properties of saccharin (employing pure saccharin rather than

sodium saccharin) and another sweet, nonnutritive substance (sodium cyclamate). In addition, the experiment attempted to determine if the effects of saccharin consumption would disappear if an interval was interposed between the consumption of saccharin and consumption of the test solution (sucrose).

METHOD

The Ss were 50 male Sprague-Dawley rats, 130 days old, assigned randomly to one group of 10 Ss and two groups of 20 Ss. They were maintained in individual cages and fed Purina Laboratory Chow.

The Ss were placed on a 23-h food- and water-deprivation schedule for 19 days. Beginning 6 h after being placed on deprivation each day, each S received one 2-min placement in an unpainted wooden box that contained an opaque glass food cup. The box was 12 in. long, 5 in. wide, and 6 in. deep, and was covered with a Plexiglas lid. The food cup was 1-3/8 in. in diam and 1/2 in. deep. The Ss received no liquid during these placements.

Throughout the experiment the Ss received their placements in the wooden box in a random order, a new order being determined each day. As soon as one S completed his placement, the next S was placed in the wooden box.

Once each day, 1 h after the last S completed his placement in the wooden box, the water bottle was removed from the S's home cage and replaced by a graduated drinking bottle containing a 16% sucrose solution. Each S was allowed to drink from the graduated drinking bottle for 5 min, timed from the moment he began drinking.

Tests were conducted on the 20th day of the deprivation schedule. Beginning 6 h after being placed on deprivation, each S was placed in the wooden box where he received 1 ml of liquid. The S group (consisting of 20 Ss) received a .25% pure saccharin (O-benzoic sulfimide) solution in the food cup during this placement. The C group (consisting of 10 Ss) received a .6% sodium cyclamate solution. The W group (consisting of 20 Ss) received tap water. During these placements each S remained in the box until he consumed the liquid. The cup was withdrawn and thoroughly washed and dried after each S had drank from it.

As soon as each S in the C group finished drinking the solution, he was immediately returned to his home cage and allowed free access to the sucrose solution for 5 min. Half of the Ss (selected randomly) in the S group and the W group also received the sucrose solution immediately following return to their home cages. However, the remaining Ss in

the S and the W groups received the sucrose solution following a 5-min delay spent in their home cages.

RESULTS

Dunnett's Test (Dunnett, 1955) was used to compare the S and C groups with the W group. For the purposes of this test, the S and W groups consisted of only those Ss that received the sucrose solution without delay. Mean amounts of the test liquid consumed were 4.50, 6.80, and 10.20 cc for Groups S, C, and W, respectively. Compared with the W group, which received only tap water while in the wooden box, each of the groups that received a sweet solution in the wooden box drank less of the sucrose solution (joint confidence coefficient of $P = 95\%$).

Data from the S and the W groups were analyzed using a 2 by 2 factorial analysis of variance with 10 Ss per cell. The factors and levels were: solutions (saccharin, tap water), and delay (no delay, 5-min delay). The results indicated that consumption of the saccharin solution suppressed subsequent sucrose intake, but after a 5-min delay this effect disappeared. With no delay, the mean intake was 4.50 cc for the S group and 10.20 cc for the W group. However, after a 5-min delay the means were 10.74 cc and 10.79 cc for the S and the W groups, respectively. The simple main effect of solutions with no delay was significant ($F = 18.11$, $df = 1/36$, $p < .005$), but the simple main effect of solutions with a 5-min delay was not significant ($F < 1.00$).

DISCUSSION

The results support Miller, Murray, & Roberts's (1955) conclusion that the ingestion of saccharin suppresses subsequent food intake. The possible confounding effects of sodium were eliminated in the present experiment by using pure saccharin. In addition, the results indicate that another nonnutritive sweet substance (sodium cyclamate) is also capable of suppressing subsequent consumption. Unlike saccharin, this substance does not produce aversive tastes or aftertastes.

In addition, the experiment indicated that the effects of saccharin consumption are transitory. This fact may account for the discrepancy between the findings of Miller, Murray, & Roberts (1955) and those of Hausmann (1933) and Smith & Duffy (1957). If Miller et al measured the effects of saccharin consumption over very short periods, whereas Hausmann and Smith and Duffy measured this effect over long periods, their different findings would be expected.

The mechanism by which the ingestion of sweet substances suppresses subsequent consumption is uncertain. There is

evidence that saccharin has definite effects on metabolic processes (e.g., Carlson, Eldridge, Martin, & Foran, 1923; Syllaba, 1930; Althausen & Wever, 1937; Macallum & Sivertz, 1942; Kun & Horvath, 1947; Jorgensen, 1950; Thompson & Mayer, 1959; Valenstein & Weber, 1965; Remley & Harrell, 1969; Deutsch & Siegel, 1970). However, in the present experiment the effects of saccharin were so rapid and transitory that an active inhibition of hunger via some neural mechanism seems plausible.

REFERENCES

- ALTHAUSEN, T. L., & WEVER, G. K. Effect of saccharin and of galactose on the blood-sugar. *Proceedings of the Society for Experimental Biology & Medicine*, 1937, 35, 517-519.
- CARLSON, A. J., ELDRIDGE, C. J., MARTIN, H. P., & FORAN, F. L. Studies on the physiological actions of saccharin. *Journal of Metabolic Research*, 1923, 3, 451-477.
- DEUTSCH, R., & SIEGEL, S. A mechanism for saccharin-induced sensitivity to insulin. Paper presented at the Eastern Psychological Association Convention, Atlantic City, N.J., 1970.
- DUNNETT, C. W. A multiple comparison procedure for comparing several treatments with a control. *Journal of the American Statistical Association*, 1955, 50, 1096-1121.
- HAUSMANN, M. F. The behavior of rats in choosing food: II. Differentiation between sugar and saccharine. *Journal of Comparative Psychology*, 1933, 15, 419-428.
- JORGENSEN, H. The influence of saccharin on the blood-sugar. *Acta Physiologica Scandinavica*, 1950, 20, 33-37.
- KUN, E., & HORVATH, I. The influence of oral saccharin on blood sugar. *Proceedings of the Society for Experimental Biology and Medicine*, 1947, 55, 175-177.
- MACALLUM, A. B., & SIVERTZ, C. The potentiation of insulin by sulfones. *Canadian Chemistry & Process Industries*, 1942, 26, 569.
- MILLER, N. E., MURRAY, E. J., & ROBERTS, W. W. Temporary reduction of hunger by saccharine. Unpublished, reported in N. E. Miller, Shortcomings of food consumption as a measure of hunger; results from other behavioral techniques. *Annals of the New York Academy of Sciences*, 1955, 63, 141-143.
- REMLEY, N. R., & HARRELL, E. H. Biological effects of saccharin ingestion in rats: A preliminary report. *Psychonomic Science*, 1969, 17, 295-297.
- SHEFFIELD, F. D., & ROBY, T. B. Reward Value of a non-nutritive sweet taste. *Journal of Comparative & Physiological Psychology*, 1950, 43, 471-481.
- SMITH, M., & DUFFY, M. Consumption of sucrose and saccharine by hungry and satiated rats. *Journal of Comparative & Physiological Psychology*, 1957, 50, 65-69.
- SYLLABA, G. The influence of saccharine on the respiratory exchange and on the blood-sugar level—An example of the conditioned reflex. *Guy's Hospital Reports*, 1930, 80, 230-233.
- THOMPSON, M. M., & MAYER, J. Hypoglycemic effects of saccharin in experimental animals. *American Journal of Clinical Nutrition*, 1959, 7, 80-85.
- VALENSTEIN, E. C., & WEBER, M. L. Potentiation of insulin coma by saccharin. *Journal of Comparative & Physiological Psychology*, 1965, 60, 443-446.