Increased food consumption in thirsty rats after water satiation: Inhibition by salts*

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Thirsty rats gradually learned to eat excessively after water satiation. The excessive eating subsequent to water consumption was antagonized by one M NaCl injection (IP) but not by an injection of distilled water.

The multiple mechanisms whereby rats regulate their food and water intake are obscure (Adolph, 1947). In the one aspect of water regulation, there is evidence that cellular dehydration (Corbit, 1965, 1968; Fitzsimons, 1961a; Gilman, 1937; Holmes & Gregersen, 1950) and hypovolemia (Fitzsimons, 1961b; Oatley, 1964; Stricker, 1966) are stimuli for thirst. The experiment reported here suggests that dilution of plasma salts or water loading are stimuli for eating. In addition, these experiments were designed to test the hypothesis that the sudden dilution of the plasma produces a "motivational state" for excessive eating.

Previously, acute administration of water through intragastric tube (Smith et al, 1949) or single intravenous infusion of water (Smith, 1966) was shown to produce small increases in food consumption. However, these alternate methods of water loading are stressful and require extensive handling of the experimental Ss. Experiments herein described made use of spontaneous drinking in thirsty rats to introduce large amounts of water into the body.

METHOD

Thirty adult male Sprague-Dawley derived rats (Charles River Farms), weighing 350-400 g at the beginning of the experiment, were divided into three groups. Two groups (water-deprived experimental and water-deprived control) were deprived of water but allowed food ad lib. The third group was deprived of food and water. All animals were given premeasured quantities of food and water during the experimental session. The experimental sessions were run during the morning hours only in order to minimize the effect of circadian rhythm on hunger and thirst.

During the experimental session, the water-deprived experimental rats and the rats deprived of food and water were allowed free access to a measured quantity of distilled water for 30 min. It was previously determined that approximately

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25 min of drinking produced water satiation and that no further water consumption occurred. The water was then removed and a weighed quantity of food was placed in the otherwise clean cages for 60 min. In water-deprived control rats, 30 min of water followed the 60 min of food after an interval of 2 to 4 h. The water and food consumed by each rat was measured for each experimental session.

After the quantity of daily food consumption was stabilized, rats of each group were divided randomly into two subgroups. One of the subgroups was given injections (IP) of 3 ml of distilled water, while the other subgroup was injected with 3 ml of salt solution,¹ prior to the 60 min of food availability. On the subsequent day, these treatments were repeated but reversed (i.e., the distilled-water subgroups were given salt solution, while the other subgroup received distilled water). The data from both subgroups were pooled after the two sequences of injection were found not to differ from each other ($p > .05$).

RESULTS

In order to determine the effect of water satiation on food consumption, the latter was measured during the 60 min after water satiation daily throughout the experiment. There was no water satiation before 60 min of food in the control group except during the first 2 days. This group was run so that the food consumption due to hunger could be estimated under our experimental conditions in the rats that initially showed increased food intake after water satiation. For comparison, food and water consumption were measured for 5 days in six rats not deprived of water and food. On the average these rats consumed $0.04 (\pm 0.01)$ g of food and $2.3 (\pm 0.66)$ ml of water in 60 min of experimental period during the last 3 of the 5 days.

Data summarized in Fig. 1 show that, after water satiation, food consumption increased markedly in both of the experimental groups. The food consumption increased gradually until it tripled by the 5th day (Day 1 vs Day 5, $p < .005$ in both groups) in both experimental groups.

Data from two experimental groups summarized in Fig. 2 show that the rats deprived of water only, consumed more water than those deprived of both food and water ($p < .005$ in all three comparisons). The food consumption of those rats that were deprived of water only, was slightly less than the food consumption of those that were deprived of both food and water. The difference was significant only in the distilled-water group ($p < .05$). Injection of distilled water did not alter food consumption, but the

Fig. 1. Food consumed during 60 min after water satiation. The rats were deprived of water or water and food for 22 h. Control rats were treated similarly to water-deprived rats except that, beginning on Day 3, they were water satiated 2-4 h after 60 min of food.

Fig. 2. Water and food consumed by two groups of rats. Food consumption was measured during 60 min after 30 min of free water.

injection of salt solution markedly decreased food consumption of the water-satiated rats in both groups ($p < .01$). Injection of distilled water or salt solution did not affect water consumption in either group of rats. In a separate experiment it was established that the injection of salt solution depressed operant lever pressing for not more than 3 min. Thus injection of salt solution was not a disabling treatment in our experimental conditions.

DISCUSSION

Food and water deprivation increases significantly the serum sodium concentration (Stevenson et al, 1950), which is known to stimulate drinking (Kutscher, 1966), possibly through cellular dehydration and hypovolemia (Corbit, 1968). Water deprivation alone may further increase serum salts due to either the salt content of food ingested during that period or the decreased urine output to conserve water (Dicker & Nunn, 1957), or both. Either would explain the greater stimulation of water drinking in water-only deprived rats.

Uninterrupted consumption of large quantities of water would be expected to result in the salt dilution of plasma that apparently triggered eating, since the injection of salt solution immediately after water consumption blocked this response. In our experiment the reduction in food consumption was not due to the discomfort of the IP injection, since drinking behavior was not depressed. Absence of the reduction of drinking behavior due to concentrated salt solution was also reported by Corbit (1968) without any sign of physical disability.

The progressive increase in food consumption with time indicates that the animals learned to eat in order to correct the physiological imbalance produced by excessive drinking. This learned behavior seems to depend upon the "motivational state" induced by plasma dilution. Eating on the first day could not elevate plasma tonicity instantaneously because of considerable delay in digestion and absorption processes. Thus, the lack of instantaneous feedback from eating, with regard to restoration of plasma tonicity, would require several trials, separated in

time, in order for the animals to associate eating with eventual correction of the salt deficiency that was created by excessive drinking. In other words, ingestion of food served as secondary reinforcement for delayed availability of salt to the body.

It should be admitted that our data do not rule out the possibility of osmotically induced cellular hydration acting as a stimulus for eating, instead of salt deficiency acting by itself as an eating stimulant.

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NOTE

1. Each milliliter of the salt solution contained: NaCl, 60 mg; KCl, 0.075 mg; CaCl_2 , 0.1 mg; NaHCO_3 , 1.0 mg.