INNATE MECHANISMS FOR
REGULATION OF SODIUM INTAKE

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I am going to review three recent experiments which indicate that the relationship between body sodium deficiency and sodium appetite in the rat is mediated by innate mechanisms. The experiments attempt to show that the emergence of sodium appetite is not dependent upon prior learning that the ingestion of sodium will relieve the symptoms of the deficiency, but that the appetite emerges spontaneously upon the rat’s very first experience with sodium deficiency. Before describing the experiments I want to discuss a general assumption underlying their design and interpretation.

It is assumed that the experimental animals never experienced sodium deficiency prior to the experimental depletion of body sodium. The animals used for the experiments were adult male laboratory rats which had been fed standard rat chows ad libitum from the time of weaning. These chows contain a surplus of sodium in comparison to minimal amounts necessary for normal growth. Thus, while we cannot be certain that the rats never experienced a transient deficiency of body sodium, the assumption that they never did seems quite plausible. It should be noted that rigorous verification of this assumption would require continuous records of body sodium levels from infancy to the time of experimentation. Simply increasing the sodium content of the diet or otherwise administering supplementary sodium would not provide an unequivocal control, for it would still remain possible that a reflexive decrease in the activity of the hormonal system for sodium retention1 could occasionally result in sodium loss in excess of intake.

There have been a number of experiments, most notably that of Nachman,2 showing that “inexperienced” sodium-deficient rats will ingest sodium salts but reject nonsodium salts (except lithium) soon after the initial taste and before any significant alteration of body sodium could occur. An experiment by Handal2 attempted to determine the minimum amount of time necessary for such differential behavior to occur. The rats were depleted of body sodium by subcutaneous formalin injection. (The sodium “depletion” induced by subcutaneous formalin is due to the sequestering of body sodium at the injection site.)3 They were then washed to remove all salts from the surface of their bodies and put in clean cages with distilled water and salt-free food ad libitum. The rats had no opportunity to taste salt from the time of injection to the time of testing, 24 hours later. The test was a “single stimulus” test, in which each rat was given only one solution, which contained either a sodium or a nonsodium salt. Intake of the solutions was monitored by a drinkometer.
Figure 1 shows the results of the experiment. Sodium-depleted rats given sodium salts drank rapidly and continuously while those given nonsodium salts stopped drinking after the first few licks. The difference between these groups was statistically significant within 5 seconds after the commencement of drinking. Note also that a group of nondepleted rats rejected a strong sodium chloride solution which was accepted by depleted rats.

It seems to me that the most plausible explanation of these results is that sodium-deficient rats immediately recognize sodium by taste and ingest it without previous experience that such behavior will relieve the deficiency. Or, in more operational terms, ingestive behavior is elicited immediately upon conjunction of adequate in-

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Figure 1  Drinkometer records of individual rats. Every fifth lick is represented by a vertical mark. Records within the upper brackets are from 9 sodium-depleted rats drinking solutions of sodium salts. Records within the middle brackets are from 6 sodium-depleted rats drinking solutions of nonsodium salts. Records within the lower brackets are from 3 nondepleted rats drinking a sodium salt solution. (From Handal19)
ternal (body sodium deficiency) and external (sodium taste) stimuli. While we cannot be sure that the rats never experienced sodium deficiency prior to the experiment, we know that they never previously tasted sodium salts in isolation from other taste stimuli, because their only source of sodium was their laboratory chow and their excretions, both of which contain many other effective taste stimuli. Under these maintenance conditions, the only way the rat could specifically associate the sodium with the reduction of the need is by some innate mechanism which makes it especially sensitive to the taste of sodium during deficiency.

The next experiment, done by Quartermain, Miller, and Wolf, indicates that the ingestion of sodium by inexperienced sodium-deficient rats is not merely a reflexive act but represents "voluntary motivated behavior" (see Teitelbaum for definitions). Rats which had been pre-trained in bar pressing (by being given water as reinforcement when thirsty) were injected with various amounts of formalin to induce varying degrees of sodium depletion. The rats were prevented from tasting sodium prior to testing, but were given water and salt-free food ad libitum as in Handel's experiment. The next day they were put in the Skinner box where bar presses now yielded a saline solution.

The results of the experiment are shown in Figure 2. When bar pressing yielded saline, the rate of pressing increased with the degree of sodium depletion. This effect was statistically significant within 10 minutes after commencement of pressing, at which time only a small amount of sodium (about 0.3 mm) had been delivered, so that the differences between the groups could not have been due to repletion of body

Figure 2  Mean cumulative bar presses for 4 groups of rats injected with 0 ml, 0.5 ml, or 1.0 ml of 1.5% formalin and given 0.33 M NaCl solution as reinforcement, and 1 group injected with 1.0 ml of 1.5% formalin and given water as reinforcement. There were 4 rats in each group. (From Quartermain, Miller, and Wolf)
sodium. A group of sodium-depleted rats given water instead of saline reinforcement did not press the bar. This experiment suggests that the tendency for the sodium-deficient rat to ingest sodium salts is not merely a reflexive act triggered by adequate need and taste stimuli, but that there is an innately determined relationship between the degree of deficiency and the degree of motivation to obtain sodium.

While the above experiment demonstrates a relationship between sodium need and motivated behavior, it does not elucidate the degree to which the increased propensity to press the bar (motivated behavior) is due to increased internal drive stimuli or to increased gratification from the taste of sodium. The final experiment suggests that the initial sodium deficiency elicits a specific desire for sodium that precedes and hence is not dependent upon reinforcement from the taste of sodium. The experiment was designed to show that inexperienced sodium-deficient rats are motivated to obtain sodium prior to experiencing the effects of ingesting it while in the deficient state.

Six groups of rats which had presumably never experienced sodium deficiency were deprived of water and trained to press a bar to receive as reinforcement one of six aqueous solutions. Three of the solutions contained sodium salts, while the other three did not (sodium chloride, sodium phosphate, and sodium acetate vs. potassium chloride, calcium chloride, and pure water). After a few days of bar-pressing training, when rats in all groups were pressing at similar rates, the rats were injected with formalin to induce sodium depletion. Two additional groups of rats trained with sodium chloride solution or with water reinforcement were sham injected to serve as nondepleted controls. As in the previous experiments, all the rats were washed and given water ad libitum until the next day, when they were put back in the Skinner boxes under extinction conditions (i.e., bar presses did not yield any reinforcement).

Figure 3 shows that the groups which had previously learned that bar presses yield sodium salts and which were sodium-depleted during testing pressed the bar at a rate

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*Figure 3  Horizontal bars indicate mean number of bar presses during 1-hour extinction session for various groups of 12 rats as a function of solution used as reinforcement during training (denoted inside individual bars) and of sodium balance during testing. The salt solutions are all 0.15 M and sodium and nonsodium solutions are balanced for palatability. The figure combines the results of experiments #2 and #3 of Kriechhaus and Wolf.*
two to three times faster than did groups in the other three conditions, even though they now received no reinforcement. Kriechkhaus has recently succeeded in also demonstrating that rats can learn the location of sodium in a T-maze when thirsty and utilize the information when subsequently depleted of sodium (personal commun.). (The initial study of Kriechkhaus and Wolf reports a failure to demonstrate this phenomenon. This was apparently attributable to insufficient training.) The above results are interpreted to show that rats have a specific desire for sodium that emerges spontaneously the very first time they are deficient in sodium and that precedes and directs behavior toward sodium acquisition. In addition, the results indicate that rats can remember how and where they obtained sodium even though they did not need it at the time of ingestion.

In summary, the three experiments investigated the innate mechanisms for the behavioral regulation of body sodium. According to the foregoing assumptions and interpretations of the results, the first experiment showed that rats experiencing sodium deficiency for the first time ingest sodium salts and reject nonsodium salts within seconds after the initial taste. This phenomenon precluded the possibility that such discriminative behavior is based upon learning that only sodium salts will relieve the symptoms of the deficiency. The phenomenon could, however, be explained on the basis of ingestive reflexes, which are released when stimuli of sodium need and sodium taste are simultaneously present. The second experiment showed that the alimentary behavior of the sodium-deficient rat is not simply reflexive but is motivated, because rats will perform a learned response to obtain sodium. The third experiment showed that the motivated behavior could not be attributed simply to an increase in the rewarding properties of sodium (e.g., increased palatability), but that sodium deficiency elicits an internal drive state which guides behavior towards acquisition of sodium.

The fact that the motivational response to sodium deficiency appears to be mediated in large part by innate mechanisms does not necessitate the conclusion that there exists a special neural system whose sole function is the regulation of sodium intake. While it seems likely that certain correlates of body sodium deficiency are monitored by a specialized receptor system, all that is necessary to account for the major phenomena described in the present paper is some innately determined relationship between the coding of neural signals of body sodium deficiency and the coding of neural signals of the taste of sodium.

I would like to utilize the above concept of similarly coded drive and taste signals in a rather preliminary and incomplete hypothesis attempting to account for the phenomenon demonstrated in the third experiment—clearly the most inclusive and complex of the various phenomena described in this paper. Stimulation of the taste receptors by sodium elicits a certain consistent pattern of neural activity in the central nervous system. Each time the taste receptors are stimulated by sodium this pattern is recorded in memory in association with temporally related stimuli from other sensory modalities. Sodium deficiency, in addition to eliciting general tension and arousal,
gives rise to a pattern of neural activity which has a configuration similar to the sensory pattern for sodium taste. Thus, via scanning and matching mechanisms, the motivational state is immediately related to the taste of sodium and to the complex of memories associated with previous experiences of sodium ingestion. Mechanisms for scanning and matching similar neural impulse configurations have been discussed by Pitts and McCulloch and by John. I sometimes think of the neural pattern of the sodium drive as a “negative” of the neural pattern of sodium taste or, more specifically, that the two patterns have a similar over-all configuration but have opposing facilitatory-inhibitory effects on common target neurons.

The above model provides a potential neural mechanism for an innately organized drive which has an experiential component associated with a specific taste sensation—for example, a craving to ingest a salty-tasting substance. The system can be summarized in behavioral terms as follows. When the sodium drive is activated, the taste of sodium is charged with reinforcement value and stimuli associated with previous sodium ingestion gain secondary reinforcement value in accordance with a spatial-temporal gradient of contiguity to the ingestive act. Sequential secondary reinforcements, increasing in intensity as the goal is approached, guide the sodium-deficient rat toward places or responses which previously yielded the primary reinforcer—sodium.

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REFERENCES