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Abstract

In the present study, two experiments were conducted to examine how the gerbil (Meriones Unguiculatus) responds to a chronic sodium deficiency. Adult male gerbils with a mean body weight of 79.81 grams, were maintained for a base-line period on a normal Purina Rat Chow and then they were placed on a salt-free diet. In addition they had ad lib access to a salt solution and water in a two bottle test situation. The saline concentration was cut in half and doubled to test for regulation. There was no significant overall increase in saline consumption (expressed as a percentage of total consumption), and the saline intake did not increase when concentration was cut in half nor decrease when concentration was doubled. However significant changes in absolute water intakes, food intakes and body weights did occur. Water intakes decreased as the concentration of saline decreased and water intakes increased with the increase in saline concentrations. Average food intakes decreased significantly in two of the three groups for the duration of the experimental manipulations. Average body weights decreased dramatically upon implementation of the diet but they slowly returned to baseline levels. A second group of animals was maintained on

the sodium-free diet for 28 days. They were sacrificed at this time and their adrenal weights were compared to a control group's adrenal weights. There was no significant difference in adrenal weights between the two groups. It was postulated that the gerbil has a superior capacity for the retention of sodium and thus a severe deficit was not produced. Suggestions for future study were discussed.

Regulation of Sodium Intake in the Mongolian Gerbil

Sodium is essential for the survival of mammals for a variety of reasons. It is indispensible to mammals as it is necessary for the maintenance of fluid balances in the body and is equally essential for many chemical reactions. It is chiefly responsible for the osmotic pressure of plasma (e.g. Snively, 1969), for the maintenance of appropriate blood pressure levels (e.g. Vroman, 1967) and for proper functioning of nerves and muscles (e.g. Ochs, 1965). As the organism cannot produce sodium, sodium must be ingested. It enters the body in foods and/or fluids which are eaten. The normal animal has a complex regulatory system which maintains appropriate levels of sodium and involves both intake and excretion mechanisms (Blass, 1974). This system involves central nervous system structures such as areas in and near the hypothalamus and the kidneys, adrenals and pituitary glands (Epstein, Kissileff and Stellar, 1973). Thirst and the sodium appetite are interdependent in maintaining normal levels of sodium (Stricker, 1973). Animals suffering from a lack of sodium develop a sodium appetite. There are multiple factors for the elicitation of the appetite, for example, hypovolemia, hyponatremia, and elevated levels of aldosterone (Epstein, Kissileff and Stellar, 1973).

The kidneys are the organs that either excrete or retain sodium. Whether sodium is excreted or retained is dependent upon the signals they receive from the pituitary or adrenal glands. During sodium depletion, the renin-angiotensin system plays an important role in its retention. The system is excited by multiple stimuli, such as hyponatremia, hypovolemia, renal arterial hypotension and renal sympathetic neural activity (Stricker, 1973). These stimuli promote the release of renin from the granular juxtaglomerular cells located within the arterioles of the kidneys (Blass, 1974). Renin reacts with angiotensinogen in the plasma and forms angiotensin I which is rapidly hydrolized to form angiotensin II (Stricker, 1973). Angiotensin II provides the stimulus for the secretion of aldosterone from the cortex of the adrenals (Turner and Bagnara, 1971). The increase in aldosterone secretion signals the kidneys to retain more sodium. When sodium is absorbed, it carries water back with it.

When the animal suffers from hypovolemia caused by sodium depletion, the volume receptors at the thyroid and carotid arteries send impulses to the brain and eventually to the hypothalamus. The impulses are then delivered over the hypo-pituitary tracts causing the release of ADH. The ADH passes into the arterial system supplying the anterior pituitary. ADH stimulates the production of ACTH which stimulates the adrenal cortex to produce

aldosterone, thus promoting sodium reabsorption (Woodbury, 1966).

The concentration of the extracellular fluid is regulated by excretion of ADH. If the concentration of the extracellular fluid is too high (too much NaCl), the posterior pituitary increases the release of ADH which directly affects the tubules of the kidneys, which produce a hypertonic urine. If the concentration is too low, there is a decrease in the amount of ADH secreted and the kidneys produce a hypotonic urine (Blass, 1974).

There is a paucity of data on the gerbil's regulatory mechanisms for the maintenance of sodium levels. So for the moment, we will consider the only data from which relevant conclusions can be drawn, the study of normal and sodium depleted rats. A state of sodium depletion has been created in rats through the use of sodium-deficient diets (e.g. Nachman, 1962), adrenalectomies (e.g. Richter, 1938), and through the use of an ion exchange resin (e.g. Epstein and Stellar, 1955).

Richter (cited in Denton, 1967) established that healthy rats consumed an amount of salt consistent with that of the circulating blood plasma each day when given free access to both water and a 3% salt solution, in addition to salt-poor food. He showed that normal rats have a natural preference for sodium solutions in two bottle preference tests. The preference for rats on a normal diet was maintained up to a maximum concentration

of 1%. On a low-salt diet, the rat's maximum preference level was increased to 1.2-1.4% and their preference threshold was lowered by 0.2% to 0.04%. On a high-salt diet (stock food +4% salt), their maximum preference level dropped to 0.07% and their preference threshold increased to 10 m Eq/litre.

Nachman and Pfaffmann (1963) studied the taste preferences of two groups of rats; one group maintained for 19-20 days on a sodium deficient diet, the second maintained on a standard diet. The animals were offered .4 M (2.4%) NaCl and HOH for two hours in a two bottle test situation. The experimental animals consumed significantly more NaCl than the control animals. In fact, the control animals showed a definite aversion to the salt solution. In the second part of this study, Nachman and Pfaffmann illustrated the specificity of this salt appetite. The rats were offered 0.1 M NaCl and 0.1 M KCl in a two bottle test situation. The animals maintained on a sodium-deficient diet consumed greater amounts of NaCl. Their mean intakes in a two hour test were 44.23 ml of NaCl and 0.4 of KCl. In comparison, the control animals consumed 4.1 ml of NaCl and 0.4 ml of KCl. Nachman (1962) found similar results for animals maintained on a sodium-deficient diet for 38 days. He concluded that rats made sodium deficient through a restrictive diet or adrenalectomy clearly show an immediate preference for a sodium solution.

The adrenalectomized rat cannot conserve sodium and loses it very rapidly. This was amply illustrated by Nachman (1962), who performed bilateral adrenalectomies on 52 rats. After the operations, the animals were maintained until death on a sodium-deficient diet. Fifty of the rats died, and 47 of these died within 7-15 days. As it cannot conserve salt, behavioral mechanisms are engaged to compensate for the physiological lack (Epstein, 1967). Epstein and Stellar (1955) showed that experience with salt is not a necessary condition for the development of the postoperative (after adrenalectomy) preference for NaCl. Richter and Eckert (1938) illustrated that the adrenalectomized rats also had an increased appetite for sodium salts.

Richter (1938) illustrated the adrenalectomized rat's ability to behaviorally regulate sodium concentration intake. His rats drank enough 3% NaCl to exactly compensate for the sodium being lost and gave the indication of living indefinitely on this diet. The mean latency of onset of the increased consumption was 3.3 days and consumption peaked at 20 days. Nachman (1962) also illustrated the adrenalectomized rat's ability to regulate sodium intake. His adrenalectomized rats drank the same volume of fluids as controls in a two bottle test situation, but 81% of their intake was NaCl. The control animal's consumption of NaCl was only 9% of its total fluid consumption.

Taste plays a crucial role in the specific hunger for sodium that follows adrenalectomy (Epstein, 1967). Richter (1956) showed that adrenalectomized rats that have lost the ability to taste salt will not show an increased preference for salt. This is no surprise since the animal cannot distinguish what it is consuming.

The adrenalectomized rat then behaviorally regulates the concentration of sodium in its internal environment. It appears that the amount of sodium in the internal environment of the adrenalectomized rat is of much more importance in determining the motivation to drink a 3% NaCl solution than the taste stimulation provided by the solution (Epstein, 1967). Epstein and Stellar (1955) have illustrated this experimentally in some now classic studies. The hunger for salt gradually increases in response to a gradually increasing need for it. They deprived rats of sodium for ten preoperative days. The first day after adrenalectomy, the rat's consumption of 3% NaCl reached that of the exaggerated postoperative levels normally seen much later by experienced rats. Another study by Epstein and Stellar involved the use of Resodec, an ion exchange resin. They added a 10% concentration of Resodec to the adrenalectomized rat's food. This caused approximately half the sodium ingested to be exchanged so that only about half of the sodium was available to be absorbed. Their rats had continuous access to a 3% NaCl solution

and in one day, their consumption approximately doubled, thus demonstrating again the rat's regulatory capacity.

Fregly (1958) examined the specificity of the sodium appetite. He used nine different salt solutions with at least three different concentrations. The rats only drank sodium salts. They didn't drink potassium, lithium or calcium salts and gained weight only when they consumed sodium chloride.

Experience may promote or obstruct the expression of the appetite (Epstein, 1967). Falk (cited in Epstein, 1967) illustrated that a prolonged increase in sodium drinking developed from a single episode of sodium depletion. Young (1948) notes that "New habits tend to form in agreement with body needs, but established habits tend to persist as regulators of food selection even when the selections are out of line with bodily needs." (p. 311). Oral factors are the principle cues with which these habits are formed.

As mentioned earlier, there exists a paucity of data concerning the gerbil. The area of sodium regulation, behavioral and physiological, is no exception. One study does exist. Cullen and Scarborough (1970) found that the adrenalectomized gerbil cannot behaviorally compensate for the loss of sodium, either by an increase in the consumption of 3% NaCl solution or through an increase in the consumption of a lab chow fortified with 5% NaCl. All of their animals died

within six days of surgery. They did find that the gerbil will respond to selective hormone replacement. They concluded that the gerbil is too adrenal-dependent to survive in the absence of these glands, and suggested the use of a salt-free diet for future study. Thus two major questions still exist which we will attempt to answer. Is the gerbil capable of responding behaviorally to a physiological lack of sodium, and if so, can we demonstrate regulation?

There have been a number of recent studies which give us new insights. There are species differences in preferences for salt solutions. Kozub et. al. (1979) showed that the gerbil, unlike the rat, has no preference for saline solutions at any concentration (0.01-9%). In fact, at or exceeding the 0.9% level, the intake of saline is lower than that of water in a two bottle test situation. Up to 0.4% NaCl, NaCl and water intakes are about equal. Since the gerbil can taste salt, it is assumed that it has the capacity to compensate for the sodium loss following implementation of a salt-free diet.

Another recent study lends support to the gerbil's ability to behaviorally compensate for the loss of sodium. Hauenstein (1978) found that gerbils respond to an extracellular insult in a similar manner to the rat. The gerbils were injected with a 10, 20 or 30% (vol x wt) polyethelene glycol solution. The P.G.

injected animals significantly increased their intake of a 0.9% saline solution. The gerbil was able to distinguish which solution contained sodium and increased its' consumption to compensate for the decrease in extracellular fluids. By consuming the 0.9% saline, it protected concentration and was able to increase volume. However, Hauenstein only used one concentration of sodium, it is not possible to determine if the gerbil can regulate its sodium intake in response to an acute physiological need.

The injection of formalin causes a reduction in plasma sodium and volume. Due to the related redistribution of body fluids, formalin potentiates an acute sodium appetite in the rat (Stricker, 1966). Perewiznyk (1978) found that gerbils increased their consumption of a saline solution, but this was only found when the vehicle was water. When the vehicle was isotonic saline, the formalin group increased its consumption of water. When the gerbil only had access to water, it increased its consumption of water, unlike the rat. In addition, the gerbils significantly increased water consumption even when they had a choice of varied concentrations of sodium. The results suggest that either the formalin did not elicit a salt appetite, perhaps because the sodium reservoir had not been depleted, or the gerbil does not regulate sodium and water in a similar manner to the rat. The latter rationale would be in conflict

with Hauenstein's data.

Recently we completed a pilot study in our own laboratory which was a replication of Cullen and Scarborough's (1970) study. However, the experimental procedure was improved by additionally testing the gerbil's ability to regulate sodium intake. Our results were consistent with those of Cullen and Scarborough; all of our animals were dead within six days. It is apparent that the only viable procedure for the study of sodium regulation in the gerbil is placing the animals on a salt-free diet.

If the gerbil can behaviorally compensate for a lack of sodium then it will increase its consumption of a saline solution in response to an increasing need. If the gerbil can behaviorally regulate the concentration of sodium in its' internal environment, then it will increase its consumption of a less concentrated saline solution and decrease its consumption of a more concentrated saline solution.

Experiment 1

Method

Subjects. Thirty adult male gerbils with a mean body weight of 79.81 grams were obtained from Tumblebrook Farms, West Brookfield, Massachusetts. The animals were individually housed under constant illumination in a temperature-controlled room (23-26° C). The cage floors

were lined with a fine wire mesh screen to prevent food pellet loss. Ss had ad lib access to Purina Rat Chow (1% NaCl) and water for ten days prior to the experiment.

Procedure. The animals were randomly divided into three groups of ten. All Ss had ad lib access to a diet of Purina Rat Chow for ten days of baseline data. In addition, each group had access to a solution of 0.45 or 0.90 or 1.80% NaCl and water in a two bottle test situation. The amounts of fluids consumed were recorded daily to the nearest ml. Saline intake was expressed as a function of total intake. Food intake and body weights were measured to the nearest gram. The animals were placed on a salt-free diet (Hartroft and Eisenstein, 1957) on the eleventh day in an environment devoid of sodium, with the exception of the saline provided in the drinking bottles. The animal's cages, food cups, and fluid bottles were cleansed carefully and rinsed thoroughly with de-ionized water. All other surfaces that the animals came into contact with were cleansed and rinsed in a similar fashion. During weighings, the scale was rinsed following a S's urinating on it. The cages were rinsed at least twice a week or whenever any build-up was evident upon visual inspection. E only handled the animals while wearing surgical gloves. Thus external sources of sodium were held to a minimum. Fluid, food, and body weight measurements were

plotted graphically for the duration of the experiment. The animals remained on this regimen for 21 days. The concentration of saline was then reduced to one half the baseline concentration. After 15 days on this reduced regimen, the experiment was terminated.

Results

A review of the raw data upon termination of the experiment resulted in the elimination of one S from each experimental group. This was deemed necessary because these animals persistently gnawed on their fluid bottles which resulted in invalid fluid measurements. Thus the number of Ss in each group was reduced to nine.

A single factor repeated measures ANOVA was used on each group of Ss to determine if any significant differences existed between the five mean peak intakes (defined as the five final days of a treatment level) for the various concentrations. It was hypothesized that if the gerbil could behaviorally regulate the level of sodium in its internal environment, then there would be a significant difference between the treatment levels. Sodium intake was expressed as a percentage of total intake. This analysis did not result in any significant F values. A graphic depiction of the sodium intake over the entire experiment for the three groups of subjects (see Figure 1), reveals that the last five days of a treatment were not the peak periods of sodium intake.

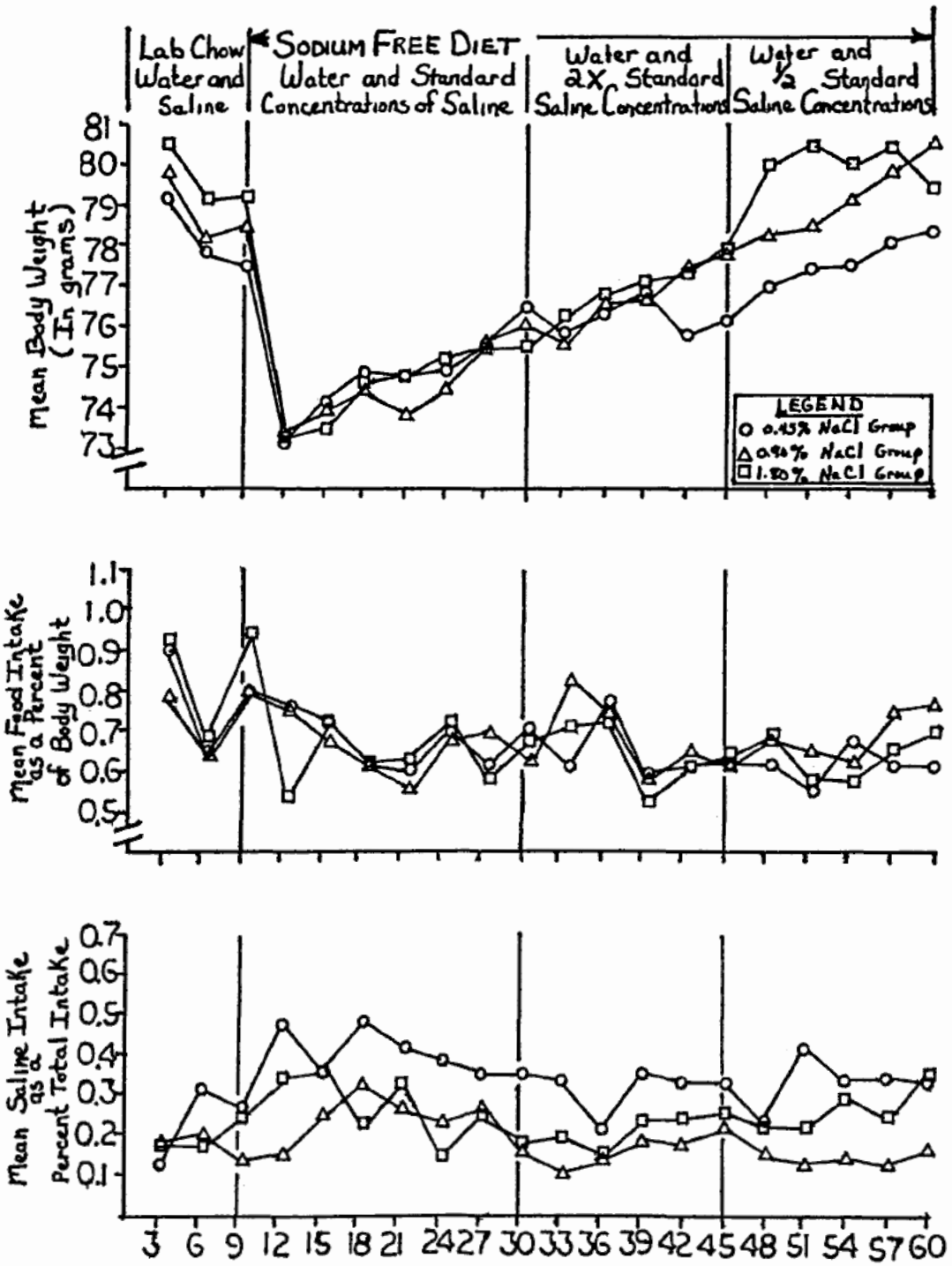

FIGURE 1. Mean body weight, mean food intake as a percent of body weight, and mean saline intake as a percent of total intake in gerbils over 60 days plotted in 3 day blocks.

A single factor repeated measures ANOVA comparing the mean intakes of sodium for the duration of each level of treatment resulted in a significant F value for the 0.90% group and the F value approached significance in the 0.45% group. A summary of the results and significance levels is depicted in table 1. The Newman-Keuls procedure for testing differences between ordered means was used on the data for the 0.90% group to determine which means differed significantly from one another. Sodium intake, expressed as a percentage of total intake, was significantly greater during the initial implementation of the sodium-free diet than during the interval when the concentration was doubled to 1.80% and when the concentration was cut to 0.45%. A summary of the results is presented in Table 2.

A two factor ANOVA repeated on the dimension of time was used to compare the initial increase in sodium consumption following the implementation of the sodium-free diet. The analysis resulted in a significant F-Max for 3 and 9 df, 6.32, p .05.

A single factor repeated measures ANOVA was used on each group of Ss to determine if any significant differences existed between the intakes of water over the four treatment levels. The average intakes are depicted graphically in Figure 2. A significant F value was found in all three groups of Ss. A summary of the results and significance levels is presented in Table 3.

TABLE 1

Significance Table for
Average NaCl Intake
(Expressed as a percentage of total intake)

	F	Crit. Value	Sig. Level
0.45% Group	2.95	3.01	N.S.
0.90% Group	4.35	3.01	.05
1.80% Group	2.15	3.01	N.S.

TABLE 2

Ordered Differences for 0.90% Group
Average NaCl Intake
(Expressed as a percentage of total intake)

Ordered Differences		½NaF	2NaF	Baseline	NaF
	½NaF		.56	1.11	10.11*
	2NaF			.55	9.00*
	Baseline				8.89
Critical Values			6.76	8.12	8.97

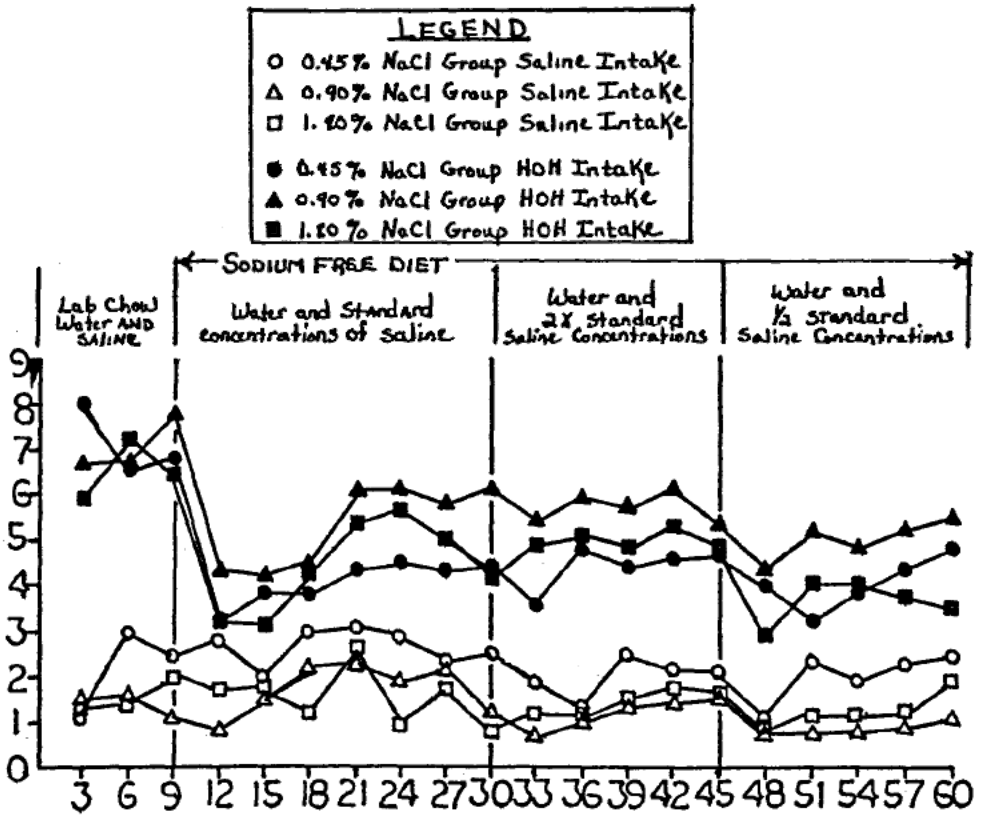

FIGURE 2. Mean absolute NaCl and water intake in gerbils over 60 days plotted in 3 day blocks.

TABLE 3

Significance Table for
Average Raw HOH Intake

	F	Crit. Value	Sig. Level
0.45% Group	121.94	7.55	.001
0.90% Group	25.53	7.55	.001
1.80% Group	14.02	7.55	.001

TABLE 4

Ordered Differences for
Average Raw HOH Intake

Ordered Differences	0.45% Group		
	NaF	2NaF	Baseline
½NaF	.15	48	3.31*
NaF		.33	3.16*
2NaF			2.83*
Critical Value		.41	.49	.55

Ordered Differences	0.90% Group		
	NaF	2NaF	Baseline
½NaF	.82	1.24 *	2.89*
NaF		.42	2.07*
2NaF			1.65*
Critical Value		.93	1.13	1.25

Ordered Differences	1.80% Group		
	NaF	2NaF	Baseline
½NaF	.40	.76	2.50*
NaF		.36	2.10*
2NaF			1.74*
Critical Value		.64	.78	.86

The Newman-Keuls procedure was used to determine which means differed significantly. Average water intake in 0.45% group decreased significantly from the average baseline intake during all three experimental conditions. The mean intake of water was lowest during the period that saline concentration was cut in half to the 0.225% level, followed by the initial implementation level 0.45% and the period of time when the concentration was doubled to 0.90%. The N-K procedure resulted in similar findings for the 0.90% and 1.80% groups. A summary of the N-K results is presented in Table 4.

A single factor repeated measures ANOVA was used on each group to determine if there were any significant differences between average raw NaCl intakes across treatment levels. The average NaCl intake is depicted graphically in Figure 2. The analysis only resulted in a significant F value for the 0.90% group. Further analysis of this data using the N-K procedure resulted in the existence of a significant difference between the initial implementation level and the level in which sodium concentration was cut in half. A summary of the analysis appears in Table 5.

The difference in average body weight across treatment levels was analyzed for each group using a single factor repeated measures ANOVA. A significant F value resulted in all three groups. A summary of this analysis appears in Table 6 and the data are depicted graphically

TABLE 5

Significance Table & Table of Ordered Differences for Average Raw NaCl Intake

	F	Crit. Value	Sig. Level
0.45% Group	1.40	3.01	N.S.
0.90% Group	3.47	3.01	.05
1.80% Group	1.93	3.01	N.S.

Ordered Differences	0.90% Group		
		2NaF	Baseline
	½NaF	2.95	5.75
	2NaF		2.80
	Baseline		
Critical Values		6.69	8.08

	NaF
	10.02*
	7.07
	4.27
	8.93

TABLE 6

Significance Table & Table of Ordered differences for Average Body Weight

	F	Crit. Value	Sig. Level
0.45% Group	6.11	4.72	.01
0.90% Group	8.58	7.55	.001
1.80% Group	46.52	7.55	.001

Ordered Differences	0.45% Group			
		2NaF	½NaF	Baseline
	NaF	.95	2.45*	3.14*
	2NaF		1.50	2.19
	½NaF			.69
Critical Values		1.69	2.04	2.26

Ordered Differences	0.90% Group			
		2NaF	Baseline	½NaF
	NaF	2.26*	4.47*	4.77*
	2NaF		2.21	2.51
	½NaF			.30
Critical Values		2.22	2.68	2.96

Ordered Differences	1.80% Group			
		2NaF	Baseline	½NaF
	NaF	2.43*	5.19*	5.56*
	2NaF		2.76*	3.13*
	Baseline			.37
Critical Values		1.11	1.34	1.45

in Figure 1. The N-K procedure was used on each group to determine which means differed significantly. In all three groups, the mean body weight was lowest during the initial implementation level, followed by the level in which sodium concentrations were doubled, and the mean body weight was greatest during the final level when sodium concentrations were cut in half. In addition, the initial implementation level was significantly lower than the other levels in all three groups. The 1.80% group was unique in that the average body weight for the double the concentration level was also significantly lower than that in the final level. Simply stated, there was a dramatic decrease in body weight following the implementation of the sodium-free diet followed by a gradual increase in average body weight to baseline levels.

The differences in average daily food intakes between the four treatment levels were analyzed for each group of Ss using a single factor repeated measures ANOVA. A significant F value resulted for the 1.80% and 0.45% groups. A summary of the analysis and significance levels is presented in Table 7. The N-K procedure for both groups resulted in a significant decrease in food consumption for the three treatment levels in comparison to the average baseline level. Thus, as can be seen in Figure 1, a significant decrease in food consumption across treatment levels resulted from the implementation

TABLE 7

Significance Table for Average Food Intake

	F	Crit. Value	Sig. Level
0.45% Group	16.46	7.55	.001
0.90% Group	1.08	3.01	N.S.
1.80% Group	13.04	7.55	.001

TABLE 8

Table of Ordered Differences for Average Food Intake

Ordered Differences	0.45% Group		
	½NaF	2NaF	Baseline
NaF	.06	.10	1.59*
½NaF		.04	1.53*
2NaF			1.49*
Critical Values	.55	.67	.74

Ordered Differences	1.80% Group		
	2NaF	½NaF	Baseline
NaF	.35	.57	2.23*
2NaF		.22	1.88*
½NaF			1.66*
Critical Values	.79	.95	1.05

of the sodium-free diet in the 1.80% and 0.45% groups. A summary of the N-K analysis is presented in Table 8.

Experiment 2

Method

Subjects. Twelve adult male gerbils, approximately 12 weeks in age, were purchased from Tumblebrook Farms, West Brookfield, Massachusetts. The animals were individually housed in the same laboratory and under the same conditions as those in Experiment 1, and the period of acclimitization was of equal duration.

Procedure. The animals were randomly divided into two groups of six. Six Ss were fed a standard diet (Purina Lab Chow) and six were placed on the sodium-free diet in an environment devoid of sodium. All Ss had ad lib access to de-ionized water for 28 days. At this time, the animals were individually sacrificed and their adrenal glands were removed by an E that was blind to the experimental conditions. The adrenal glands for each S were weighed to the nearest hundredth of a gram and an adrenal to body weight ratio was computed.

Results

A single factor independent groups ANOVA was used to determine if a significant difference existed between adrenal to body weight ratios for the control and experimental groups. The analysis resulted in a nonsignificant F value for 1 and 10 df, 1.78 p .05.

Discussion

The relevant rat studies illustrate the species' ability to behaviorally regulate its internal level of sodium. In fact, Richter (cited in Denton, 1967) established that healthy rats consumed an amount of salt consistent with that of the circulating blood plasma each day when given free access to both water and a 3% salt solution in addition to salt-poor food. Nachman and Pfaffmann illustrated the preference for NaCl in rats placed on a restrictive diet. The sodium deficient rats significantly increased their consumption of a 2.4% NaCl solution, while the controls exhibited a definite aversion to the sodium solution. The adrenalectomized rat, which cannot conserve sodium and looses it very rapidly, compensates behaviorally for the physiological lack (Epstein, 1967). Richter and Eckert (1938) demonstrated that the adrenalectomized rats have an increased appetite for sodium salts. Richter's rats drank enough 3% NaCl to exactly compensate for the sodium being lost and gave the indication of living indefinitely on this diet. The sodium deficient rat, then, can behaviorally regulate the concentration of sodium in its internal environment.

The rat's ability to behaviorally regulate its internal level of sodium has been demonstrated through a third experimental technique. Epstein and Stellar (1955) used the ion exchange resin, Resodec. They added a 10% concentration of Resodec to the adrenalectom-

ized rat's food. This caused approximately half of the sodium ingested to be exchanged, so only about half of the sodium was available to be absorbed. Their rat's consumption of a 3% NaCl solution approximately doubled in one day.

The afore-mentioned data would lead one to believe that the gerbil, if it behaves in a similar fashion to the rat, could behaviorally regulate its internal level of sodium. The gerbil, unlike the rat, cannot behaviorally compensate for the loss of sodium following adrenalectomy (Cullen and Scarborough, 1970). Their animals died within six days of the operation. However, Hauenstein (1978) found that gerbils respond to an extracellular insult in a similar manner as the rat. The gerbil significantly increased its intake of a 0.90% saline solution in response to an extracellular insult created by a P.G. injection. Thus like the rat, it protected concentration and was able to increase volume. Perewiznyk (1978) demonstrated that the gerbil did not behave in a similar fashion to the rat when injected with formalin.

The present study was an attempt to experimentally demonstrate the gerbil's ability to behaviorally regulate its internal levels of sodium in response to a chronic deficit. The deficit was created by placing the animals on a sodium-free diet in an environment devoid of sodium. It was hypothesized that if the gerbil could behaviorally

compensate for a lack of sodium, then it would increase its consumption of a saline solution in response to an increasing need. Also, it was hypothesized that if the gerbil could behaviorally regulate the concentration of sodium in its internal environment, then it would increase its consumption of a less concentrated saline solution and decrease its consumption of a more concentrated solution.

The results for the sodium intakes, expressed as a percentage of total intakes, in all three groups did not support the hypotheses. Saline intake did not significantly increase over baseline levels following implementation of the sodium-free diet. There was no significant decrease in sodium intake following the doubling of concentrations and there was no significant increase in sodium intake following the halving of the concentration. The significant F value which resulted in the 0.90% group was due to a significantly greater intake of sodium, expressed as a percentage of total, during the initial implementation level in comparison to intakes during the levels in which the concentration was cut and doubled. Again these results are inconsistent with what was expected, in that sodium intake did not increase following the halving of concentrations.

However, the gerbils did change their drinking behavior. There was a significant decrease in the consumption of de-ionized water in response to the

sodium-free diet. In all three groups, water intake was significantly lower than baseline throughout the experimental phases. The trend for intakes in all three groups was one in which the greatest decrease in water consumption accompanied the greatest decrease in the availability of sodium. Thus when the concentration of saline was cut in half, water intake was at its lowest followed by the initial 21 days of the diet and then the stage in which concentrations were doubled.

The gerbil, unlike the rat, did not significantly increase its raw saline intake of any concentration at any time in this experiment. There was a significant difference between the initial implementation level and the half concentration level. Contrary to the hypothesis, the saline intake was significantly lower during the half concentration phase.

Implementation of the diet resulted in a number of additional changes in the animals. All three groups of Ss immediately lost a significant amount of body weight. The average body weights in the 1.80% group remained at these depressed levels until sodium concentration was cut in half. The average body weights in the 0.45% and 0.90% groups increased during the phase in which sodium concentrations were doubled. In addition, average food intakes significantly decreased in the 0.45% and 1.80% groups. In both groups the intakes remained depressed throughout the experiment.

The gerbil has responded behaviorally to the experimental manipulations but not in the hypothesized manner. It did not increase its consumption of sodium or show any change in consumption as concentrations changed. This could be due to the gerbil possessing a superior capacity for the retention of sodium. Experiment 2 illustrated the gerbil's ability to live with few apparent side effects for 28 days without access to sodium. Perewiznyk (1978), also suggests that she may not have been able to deplete the gerbil's sodium reservoir. Myers (1965) found that sodium deficient rabbits have hypertrophic adrenal glands. Our gerbils did not illustrate any hypertrophism. Perhaps this was because they had not entered into a deep enough state of sodium-deficiency at the time of operation. The fact that the animals did not significantly increase their sodium intakes throughout the 50 experimental days is also consistent with this hypothesis. This capacity for the retention of sodium accompanied by the normal intake of sodium seems to have been enough to protect the animal from a chronic salt deficiency in the present situation. A change in the color of the Ss urine was noticed through casual observation. The normal gerbil has a very dense yellow urine, while the experimental animal's often exhibited a completely clear water-like urine, further suggesting that the animals were recycling their sodium rather than excreting any surplus.

Hauenstein's (1978) data suggest that the gerbil responds to an acute extracellular insult in a similar manner to the rat. They both protect concentration by increasing their intake of a sodium solution. Perhaps the diet induced insult is not great enough to produce this effect. The gerbil appears to be able to cope with the diet induced insult by decreasing its intake of water and food, thus protecting concentration.

Again, casual observations indicated a general decrease in the activity level of the animals during the 21 days following implementation of the diet. The level of activity increased when sodium concentrations were doubled. This difference in level of activity was also evident in Experiment 2. The control animals were continually gnawing on their cages or playing with their food cups, while in sharp contrast, the experimental animals slept or moved about lethargically.

To test the previous hypotheses, a number of future studies are suggested. A study replicating Experiment 1 or 2 should be conducted using daily urine analysis for sodium, potassium, chloride, calcium and possibly ph levels. This would enable one to draw conclusions about the amount of sodium and other ions being excreted or conserved following implementation of the diet. Does this level change with changes in the concentration of sodium in the fluid bottles? An experiment should be conducted to determine how long the gerbil can live

without access to sodium. Additionally an experiment should be conducted to measure activity levels in response to dietary sodium changes. An interspecies comparison between rats and gerbils on sodium-free diets would yield significant information regarding the water and sodium regulatory abilities of these two species.

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Vita

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