Toward an electro-physiological identification of a primary drive factor¹

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Kangaroo rats, which do not drink water, were implanted with monopolar electrodes in order to measure changes in electrical brain resistance. The manipulation of water balance and caloric intake resulted in slow resistance changes over a period of days. The direction and magnitude of the changes were consistent with extracellular osmotic theories. The results are more suggestive of changes in drive parameters than start-stop mechanisms of water and food regulation.

Novin (1960) demonstrated that measurements of brain resistance fluctuate with the hypotonicity and hypertonicity of the laboratory rat. Brain tissue resistance has been directly related to the amount of fluid intake, water deprivation, dry food consumption, and hypertonic saline injections (Novin, 1962). If one measures the electrical tissue resistance of animals who have drunk their fill of water, this state of hypotonicity (increased resistance) could be operationally defined as water satiation. In any case, the tissue need for water should be at a low ebb. This type of definition would be independent of the usual *ex post facto* performance definitions.

The data reported here were collected to determine if it would be feasible to use measurements of brain resistance to identify a tissue need and thereby isolate a primary drive factor. Drive stimulus variables of peripheral cues were minimized in order to increase the correspondence between water need and its primary drive factor. This was accomplished by using animals that do not drink water. Considerable work has been done on the water metabolism of kangaroo rats (Schmidt-Neilson & Schmidt-Neilson, 1950, 1951; Chew, 1961). These small mammals use metabolic and residual water in food to meet their tissue needs for water.

Method

Twenty male kangaroo rats of two different species (*dipodomys spectabilis* and *dipodomys ordi*) weighing from 40-70 g, were live trapped. These Ss were implanted with two monopolar electrodes placed bilaterally 1.5 mm anterior to bregma and two electrodes placed bilaterally 1.5 mm posterior to bregma. All of the electrodes were located 3 mm left or right of the midline. The electrode depth sought was 5.5 mm, approximately the thalamic level. The electrodes were constructed of 6 mm lengths of .01 in. nichrome milar-coated wire and fastened to miniature Amphenol strip connectors. The distal ends of these electrodes were electroplated with platinum-black deposits (Novin,

1960). The resistance measuring device was a Wheatstone bridge with a 1 KHz sine wave generator as the signal source. The 1 KHz resistance of the Ss brain tissue was balanced against a precision resistor in parallel with a step variable capacitor (Novin, 1960).

Results

The data in Figs. 1 and 2 are presented in terms of fluctuations in brain resistance. Each discrete data point represents an average of six electrode readings obtained during each 5 min. experimental session.

Figure 1 represents the changes in brain resistance for typical Ss following two 12-h periods during which lettuce was available *ad lib*. The vertical lines in Fig. 1 represent the midpoints of the 12-h periods. All Ss except one showed an increase in brain resistance after lettuce was made available. The peak brain resistance occurred approximately 44 to 49 h after the midpoint of the 12-h lettuce period. This measured peak is followed by a decrease in brain resistance. On Day 9 (Fig. 1) lettuce was again introduced. This time the measured peak resistance across Ss occurred approximately 24 to 34 h after the midpoint of the *ad lib* period. The Ss were also deprived of lettuce for a period of 15 days. The broken line histograms in Fig. 1 indicate the day-to-day fluctuations for approximately the last seven days of this "water deprivation" period. It can be seen that the base line resistance during water deprivation is lower than during high water balance. This difference may be as small as 60 ohms or as large as 640 ohms. It can also be seen that the variance of the fluctuations is small during water deprivation.

Figure 1 is representative of three consistent trends seen in all of the Ss: (a) A large increase in brain resistance following the introduction of lettuce, (b) An overall decrease in brain resistance after a period

Fig. 1. Resistance (solid lines) plotted against days for typical animals during availability of high water content food (lettuce). Heavy vertical lines indicate the midpoint of 12-h periods of *ad libitum* lettuce. The broken lines indicate resistance measurements during lettuce deprivation (during this period the only source of food was dry seeds).

Fig. 2. The change in resistance (solid lines) as a function of two oral injections (broken vertical lines) of .05 cc of D-Glucose ($\text{CH}_2\text{OHCH}(\text{CHOH})_3\text{CHO}$), mixed in the ratio of 1.5 mg to 1 cc tap water.

of lettuce deprivation, (c) A decrease in the daily fluctuations of brain resistance following a relatively prolonged period of lettuce deprivation.

Figure 2 represents typical changes in brain resistance which follow oral injections of .05 cc of glucose. The vertical lines represent the times of the injections. All Ss except one demonstrated dramatic increases in resistance within 18 to 20 h after the second injection of glucose. Of interest in Fig. 2 is the large fluctuations that are functions of caloric intake and much greater than any seen by directly manipulating water balance.

Discussion

This study sought to isolate a primary drive factor in terms of a reproducible electrophysiological technique. It was found that in using the present technique, water satiation could only be defined relative to measures of water deprivation. That is, during water deprivation electrical brain resistance will stabilize at a low and constant baseline. If nondrinking rodents are given a high water content food, the resistance measures increase to a much higher mean value, and daily variance becomes quite large. It is this latter problem which makes an accurate identification of water satiation difficult.

The data presented here are best explained by an extracellular hypothesis. That is, the control of water balance could be explained in terms of changes in extracellular volume and subsequent changes in electrolytic concentration. The theory would predict that during lettuce deprivation the extracellular volume has decreased, thus resulting in an increase in electrolytes which, in turn, causes the observed decrease in resistance. The relative invariance of the measures during water deprivation could be caused by a large extracellular pool of ions necessitating a large influx of ions to show measurable resistance change. In short, at extreme ionic concentrations any additional increase would be held in check by physiological transport mechanisms. On the other hand, when Ss

received lettuce and increased their extracellular volume, any small change in electrolytic concentration would be measured as a concomitant change in resistance.

Novin (1962) reported but did not explain why eating dry food is first followed by an increase in electrolytes and then a subsequent decrease in electrolytes, without any access to water. In the present study we found similar results using oral injections of glucose. Our long term increase in brain resistance would be similar to Novin's stage of subsequent decrease in electrolytes.

Jacobs (1964) has suggested that since glucose loads decrease water intake over a 2 h period, and since this decrease is similar to that obtained with water loads, the glucose loads must allow the release of enough free water to hydrate the Ss. This is exactly the type of metabolic hydration effect which is needed to maintain an extracellular water balance in rodents that do not drink water.

In summary, the present technique is an approach toward physiologically identifying a primary drive. Furthermore, the reported electrophysiological measure not only reflects rapid changes in tissue states but also demonstrates the long term effects of reinforcers.

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Notes

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