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NOTE

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Reciprocal and additive effects of hyperoncotic and hypertonic treatments on feeding and drinking in rats[1]

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Rats were given injections of a hyperoncotic colloid and/or a hypertonic saline solution. Six hours after the oncotic load and 30 min after the osmotic load, food alone was presented for 1 h. Rats had been deprived of both food and water for 24 h when food was presented. Six days later, the same Ss were given the same treatments and given water only for 1 h. Results showed that both treatments additively inhibited feeding and potentiated drinking. Feeding and drinking as influenced by the colloid and the saline treatments are reciprocally related.

Thirst and hunger are closely related. In ad lib feeding conditions there is a precise temporal relation between feeding and drinking (Kissileff, 1969) and a positive correlation between the size of food and of water intake related to a meal (Fitzsimons & Le Magnen, 1969). Limiting food intake reduces drinking proportionally to the amount eaten (Hsiao & Pertsulakes[2]), and rationing water or deterring water intake by quinine reduces food intake proportionally to the amount drunk (Collier & Levitsky, 1967; Hsiao & Lloyd, 1969). Water injection in thirsty rats reduces drinking but increases feeding proportionally to the amount injected (Hsiao & Trankina, 1969). Thus, thirst inhibits feeding, feeding induces drinking, and, conversely, reduction in thirst potentiates feeding in rats.

Increase in drinking can be induced by injections of hypertonic saline (e.g., Corbit, 1969), hyperoncotic colloid (Stricker, 1966), or by hemorrhage (Fitzsimons, 1961). Thirsts induced by hypertonic and hyperoncotic treatments or hemorrhage are additive to potentiate drinking (Corbit, 1968; Fitzsimons & Oatley, 1968).

Since (1) feeding and drinking are reciprocally related, and (2) hypertonic and hyperoncotic treatments add to potentiate drinking, it is expected that these treatments also add to inhibit feeding. This study investigated hypertonic saline and hyperoncotic colloid treatments on feeding and drinking.

SUBJECTS AND APPARATUS

The Ss were 24 female Wistar rats, about 180 days old, weighing from 210 to 291 g. They were housed individually in a constantly illuminated laboratory at a temperature of 22°-23°C; humidity varied from 38% to 42%. A 100-ml graduated tube with metal tip was mounted on each home cage and used to measure water intake. Food intake was measured by use of a 250 ml glass beaker fastened at a corner of each cage. Powdered Purina Lab Chow was introduced into the beaker for feeding. Food spillage was minimal because rats ate with their heads inside the beakers. Food intake was read to the nearest 0.1 g and water intake to the nearest 1 ml.

PROCEDURE

The Ss were adapted to a schedule of 1-day total deprivation and 1-day feeding-drinking for 8 days. On feeding-drinking days, food alone was presented for 1 h followed by water only for 1 h before both food and water were given for 22 h. By the eighth day of the adaptation period, body weight, food intake, and water intake had become stable and the experiment was begun. Ss were divided into four groups of six Ss each, equated according to their 1-h food intake measures.

Experiment 1

All Ss were deprived of food and water for 24 h and were given food only for 1 h to measure intake. Six hours before the food presentation, each S in Groups 1 and 3 was given a subcutaneous (backskin behind the neck) injection of a hyperoncotic colloid which was 5 ml of 10% (w/v) polyethylene glycol (PG) (molecular weight = 20,000) dissolved in isotonic saline. Groups 2 and 4 were similarly injected with 5 ml of isotonic saline vehicle. Ss were lightly etherized before these injections. Thirty minutes before the food presentation, each S in Groups 1 and 2 was given an intraperitoneal injection of 3 ml of 1 M NaCl solution. Groups 3 and 4 were given sham injection with needle puncture only. This was a 2 by 2 factorial design in which Group 1 received both hyperoncotic and hyperosmotic loads, Group 2 received hyperosmotic load only, Group 3 received hyperoncotic load only, and Group 4

received control conditions.

Experiment 2

The same Ss were used for this experiment 6 days after Experiment 1. The same procedures of Experiment 1 were administered to the same four groups. However, for this experiment, 1 h of water only was presented to measure the effects of those treatments on drinking.

RESULTS

Table 1 presents mean 1-h food or water intake. Combined effects of the colloid and saline treatments were to produce the least food intake but the greatest water intake; this was followed by the saline effect, the colloid effect, and the control effect in the reversed order for food intake and water intake means.

Analysis of variance indicates that, for food intake data, both main effects are statistically significant ($F = 35.81$ for the saline effect and $F = 15.62$ for the colloid effect, both $p < .01$, with df of 1/20), but the interaction effect is not significant ($F < 1$). Similar results were obtained for water intake data ($F = 33.82$, $p < .01$ for the saline effect, $F = 6.64$, $p < .05$ for the colloid effect, and $F < 1$ for the interaction effect). Apparent additivity can be noted by comparing the column differences or the row differences of food or water intake means in Table 1.

The results indicate that: (1) hypertonic saline and hyperoncotic colloid each potentiates water intake and their combined effects are additive, but (2) they each inhibits food intake, and their combined effects are also additive. Thus, hypertonic saline and hyperoncotic colloid function independently to reciprocally influence feeding and drinking.

DISCUSSION

Injection of 10% PG induces edema around the injected site which reduces fluid available for the general circulation to produce hypovolemia. However, Stricker (1968) reported that PG injection reduces plasma water concentration and induces other signs of hemoconcentration. Thus, in addition to hypovolemia, PG also seems to induce plasma hyperosmolality. However, it appears that hypovolemia is the major aspect of this treatment since isotonic saline, a volume expander, has been shown to reduce thirst produced by PG, though isotonic saline is not effective in reducing hyperosmotic thirst produced by hypertonic saline (Stricker & Wolf, 1967). Thus, functionally 10% PG can be regarded as inducing hypovolemic thirst rather than osmotic thirst. Blass (1968) showed that rats with frontal brain damage respond to PG injection as normal rats do to increase drinking, but they do not respond to cellular dehydration induced by hypertonic saline injection as normal rats do. Thus, neurologically PG-induced thirst and hypertonic saline-induced thirst appear different.

Table 1
Mean 1-H Food Intake (Experiment 1) and 1-H Water Intake (Experiment 2) as Influenced by the Hypertonic Saline and Hyperoncotic Colloid Treatments[a]

Treatments	Food Intake (g)		Water Intake (ml)	
	Control	Colloid	Control	Colloid
Control	5.25	2.96	4.2	7.2
Saline	1.86	0.23	10.8	13.7

[a] *For both food and water intake data, the saline and colloid effects are statistically significant ($p < .05$) but interaction is not ($p > .05$).*

The present study confirms the findings of Corbit (1968) and Fitzsimons & Oatley (1968), in drinking, and shows further that, reciprocal to their effects on drinking, cellular dehydration and hypovolemia add to inhibit feeding. Complex thirst (Corbit, 1969) thus appears to be a simple algebraic sum of osmotic thirst and hypovolemic thirst.

Hyperosmotic treatments that induce cellular dehydration have been known to inhibit food intake (Gutman & Kransz, 1969; Hsiao, 1967, 1970; Schwartzbaum & Ward, 1958). Gutman & Kransz (1969) reported that hypovolemia also inhibits feeding but they did not show the additivity of the effects of cellular dehydration and hypovolemia.

Reduction in thirst by water or hypotonic saline can potentiate feeding (Hsiao & Trankina, 1969); the present results show that, conversely, induction of thirst by hypertonic and hyperoncotic loadings inhibit feeding. Thus, the amount of thirst reduced or induced (as indicated by water intake) is inversely related to the amount of food intake.

It was found that (1) after ingesting dry food, rats suffer from cellular dehydration, particularly in the skin (Lepkovsky, Lyman, Fleming, Nagumo, & Dimick, 1957); (2) rat's plasma osmolality is normal after 1-day total deprivation but, after eating dry food, plasma osmolality rises (Gutman & Kransz, 1969); (3) there is hypovolemia after feeding in sheep (Blair-West & Brook, 1969). Feeding seems to induce plasma hyperosmolality, cellular dehydration, and hypovolemia, which are the conditions involved in the present study. These conditions apparently are dipsogenic and inhibit further feeding when no liquid is available to correct them.

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NOTES

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