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# Intraventricular Glucose Administration Inhibits Feeding in Satiated but not in 24 Hours Food Deprived Cocks[1]

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ROBINZON, B. AND N. SNAPIR. *Intraventricular glucose administration inhibits feeding in satiated but not in 24 hours food deprived cocks.* PHARMACOL BIOCHEM BEHAV **19**(6) 929–932, 1983.—Injection of 5 μl isotonic glucose into the third ventricle above the basal hypothalamus but not in the posterior hypothalamus-anterior midbrain, suppressed feeding in satiated cocks. This suppression of food intake was less dramatic when glucose was injected after 5 hr of food deprivation and was eliminated if 24 hr of food deprivation preceded the glucose administration. An involvement of a glucostatic mechanism in the regulation of feeding in the chicken is suggested.

Chicken Food intake Glucose

THE involvement of a glucostatic mechanism in the short-term regulation of food intake is well documented for mammalian [18, 19, 29]. This mechanism involves insulin-dependant glucosensitive neurons located in the basal-hypothalamus [1, 2, 5, 6, 9, 15, 17, 23, 31]. Thus intracranial administration of insulin or glucose suppressed eating [14, 16, 24], while intra-cerebroventricular administration of 2-deoxy-D-glucose or intrahypothalamic injection of antiinsulin increased feeding [12, 20, 21, 31].

In contrast to the general acceptance of the glucostatic regulation of feeding in mammals there are several reports denying such mechanism in Aves: Peripheral or central administration of gold-thioglucose (GTG) to chickens and quails did not damage the basomedial-hypothalamus and was not followed by hyperphagia or obesity [4, 11, 25, 26, 27, 30]; insulin-induced hypoglycemia was not followed by hyperphagia in chickens and indeed caused hypophagia in geese [22]; Injection of 10 μl of 10% glucose into the lateral ventricle of 24 hr food-deprived chicks did not inhibit food intake [7]. However, as in Aves, GTG did not induce hypothalamic lesions or obesity in several mammalian species [3,8]. Thus, the lack of GTG effects in birds is insufficient evidence against the existence of a glucostatic mechanism in this species. Furthermore, insulin has a lipolytic effect in birds rather than the lipogenic one it has in mammals [22], thus it may be suggested that the hypophagia that follows insulin administration to geese can be the result of its effect on the lipid metabolism rather than the consequence of its effect on blood glucose levels. If this is the case, then the lack of hyperphagia after insulin injection to Aves is not necessarily the result of a lack of a glucostatic regulation of feeding in them. The glucostatic regulation of feeding in mammals suggested as a short-term mechanism that operates on a minute-to-minute level. Thus, it is possible that under starvation this mechanism would not be the dominant one in the regulation of feeding. Acceptance of this possibility denies the lack of inhibition of feeding by intracranial glucose administration from being the proof against the involvement of the glucostatic mechanism in the regulation of food intake in chickens.

In the present experiment the effect of injections of isotonic glucose into the third-ventricle or into the posterior-hypothalamus-anterior-midbrain on feeding was tested in fed, 5 hr- and 24-hr-food deprived cocks.

## METHOD

### *Animals*

Seventeen four-months old White Leghorn cocks were used for the experiment. They were kept in individual cages and were fed (Commercial Breeder Mash) and watered ad lib. The lighting regimen was 14 hr light–10 hr dark.

### *Surgery*

The cocks were anesthetized by intravenous injection of 0.6 ml of 6% sodium-pentobarbital solution (Nembutal, Abbot-Ceva, France), and inserted into a specially designed stereotaxic instrument described previously [10]. Double cannulae constructed according to Grossman's design [13] were implanted using the technique described in the x-ray atlas of the chicken diencephalon [28]. In nine cocks the tip

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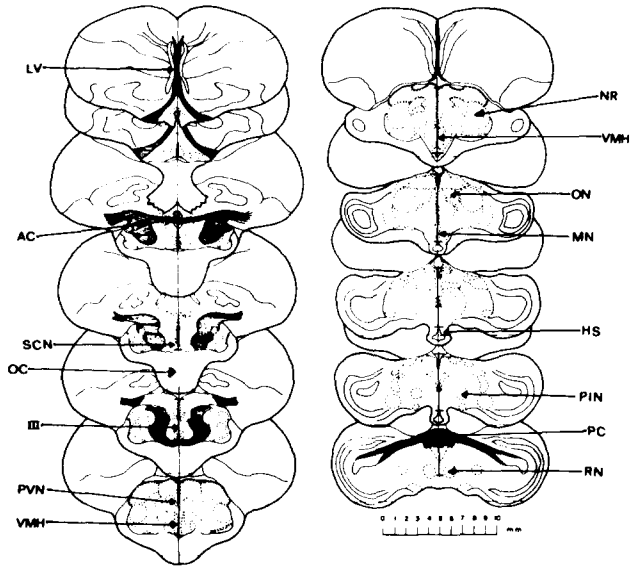


FIG. 1. Schematic drawing of frontal sections of the chicken brain [28]: AC—anterior commissure; HS—hypophyseal stalk; III—third ventricle; LV—lateral ventricle; MN—mammillary nucleus; NR—nucleus rotundus; OC—optic chiasm; ON—ovoid nucleus; PC—posterior commissure; PIN—posterointermedius nucleus; PVN—paraventricular nucleus; RN—red nucleus; SCN—suprachiasmatic nucleus; VMH—ventromedial hypothalamic nucleus.

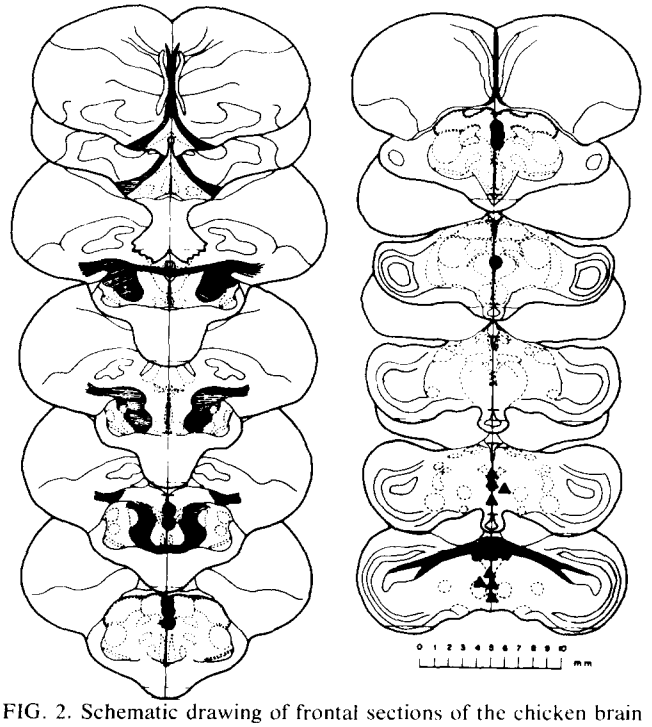

FIG. 2. Schematic drawing of frontal sections of the chicken brain showing the locations of the tips of cannulas in the third venticle (●) and in the posterior-hypothalamic-rostral-mesencephalic area (▲).

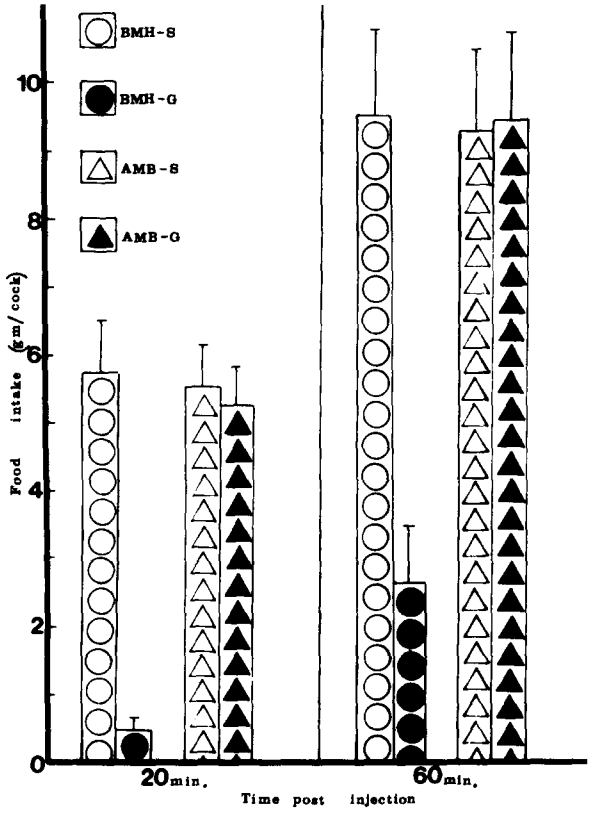


FIG. 3. Average of cumulative food intake in satiated cocks following the injection of saline (S) or glucose (G) into the third ventricle above the basal hypothalamus (BMH; n=9) and into the posterior-hypothalamus-anterior-midbrain (AMB; n=8). Vertical bars indicate S.E. of the means.

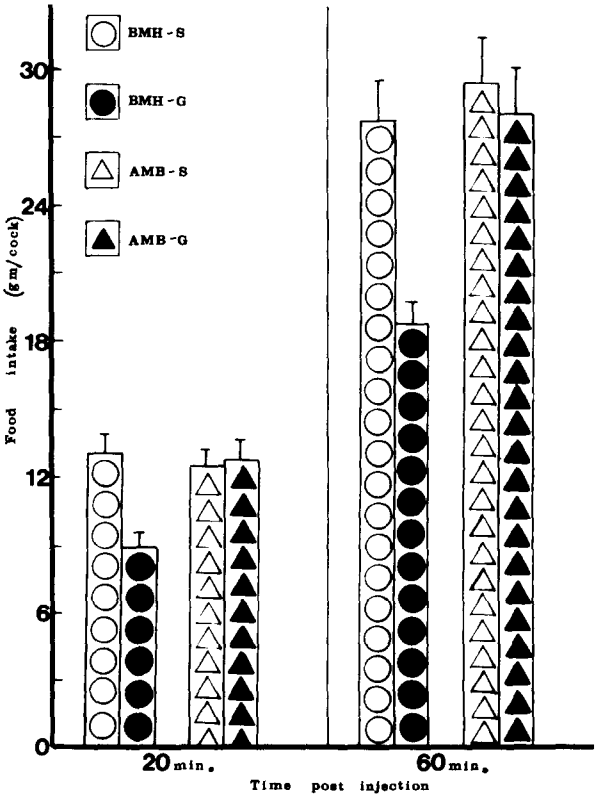


FIG. 4. Average of cumulative food intake of 5 hours food deprived cocks following the injection of saline (S) or glucose (G) into the third ventricle above the basal hypothalamus (BMH; n=9) or into the posterior-hypothalamus-anterior-midbrain (AMB; n=8). Vertical bars indicate S.E. of the means.

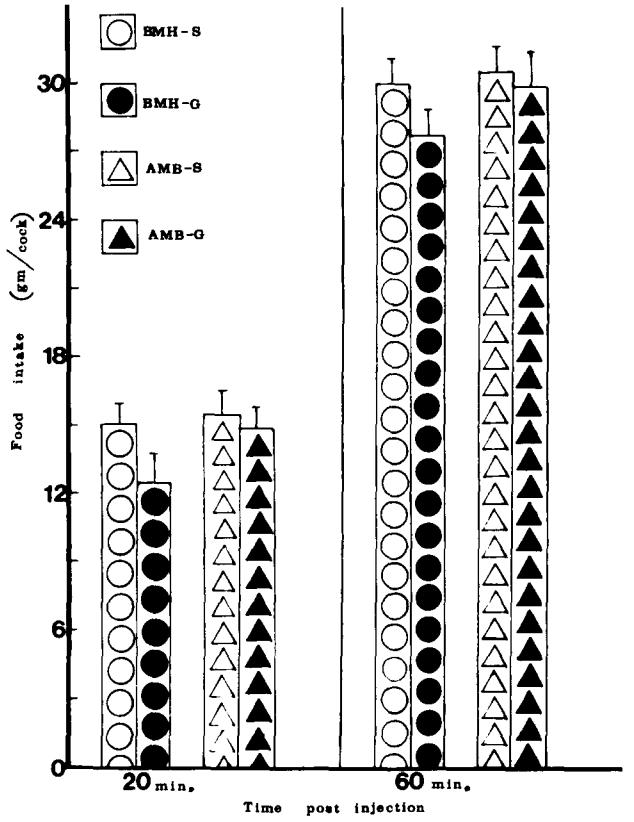


FIG. 5. Average of cumulative food intake of 24 hours food deprived cocks following the injection of saline (S) or glucose (G) into the third ventricle above the basal hypothalamus (BMH; n=9) or into the posterior-hypothalamus-anterior-midbrain (AMB; n=8). Vertical bars indicate S.E. of the means.

of the cannula was aimed at the third-ventricle, above the basal hypothalamus, while in the rest of them it was aimed at the posterior-hypothalamus-rostral-midbrain.

*Procedure*

The cocks were given a recovery period of one month post-operation, after which the injection procedure initiated.

Each cock was tested seven times for the feeding response to intra-cannula injection of 5 $\mu$l of isotonic saline and to injection of 5 $\mu$l of isotonic glucose (0.3 M) in each of the following states: 24 hr of food deprivation, 5 hr of food deprivation and no food deprivation. All tests were started at 1 p.m. and the cocks were given a recovery period of 4 days between each test. The test regimen for each cock was randomly designed. Before each test the mash in the feeders was replaced with fresh mash and food intake was measured 20 and 60 minutes post injection.

At the end of the experiment the cocks were decapitated and their brains were immediately removed and fixed in 10% neutral-buffered formalin. Serial frozen frontal sections of 25 $\mu$m thickness each were prepared and stained with thionin. The sections were examined for localization of the cannula's tips. The data were analized using ANOVA.

## RESULTS

A schematic drawing of frontal sections of the chicken brain is presented in Fig. 1. The exact location of the cannula's tips implanted in the third-ventricle and in the posterior-hypothalamus-rostral-midbrain is reconstructed on a similar drawing and presented in Fig. 2.

Injection of isotonic glucose into the third ventricle of satiated cocks significantly inhibited feeding during the following hour, while injections of the same dose of glucose to a more posterior areas of the brainstem did not suppress the eating in satiated cocks (Fig. 3). The suppression of feeding by the injection of glucose into the third ventricle was less dramatic when the injections were made after 5 hr of food deprivation (Fig. 4) and was insignificant if the glucose administration was made following 24 hr of starvation (Fig. 5).

## DISCUSSION

In the present experiment intracranial injections of isotonic glucose did suppress feeding in chickens. However, suppression of food intake was clearly observed only if the cannula's tips were in the third ventricle above the basal hypothalamus and the cocks were not food-deprived. Food deprivation for 5 hr reduced the glucose-induced inhibition of feeding and starvation for 24 hr eliminated it entirely. These results suggest that in chickens a glucostatic mechanism is involved in the regulation of feeding and that a glucosensitive system is located either in the basal hypothalamus or in the cerebroventricular system. However, since the intraventricular injection of glucose did not suppress feeding in cocks that were deprived of food for 24 hr, one may suggest that in starved chickens the glucostatic mechanism is not dominant in the regulation of food intake.

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