there is no longer competition for nutrients and yet growth of the T-1 oocytes is prevented. This suggests that the event of ovulation is physiologically important to the animal in releasing the inhibition of the T-1 oocytes. The nature of this inhibition is unknown, although in other insects it has been suggested to be humoral[3,8]. It would be of interest to determine if the location of the T oocytes in M. domestica reproductive tract influences the production or mode of action of the oostatic factor. However, it is possible that the nature of the inhibition differs in houseflies and locusts, particularly in view of the fact that in other flies (Glossina), the presence of mature oocytes within the ovarioles does not prevent the maturation of other oocytes in the same and other ovarioles[17].

The results presented above demonstrate that in S. gregaria, the growth of the T-1 oocytes is inhibited by the presence of mature chorionated T oocytes *within* the ovarioles but *not* by T oocytes in the oviduct. Using this information, I have examined the activity of the CA in animals in which the growth of T-1 oocytes was inhibited and in animals in which ovulation had occurred, to determine if inhibition of oocyte growth was the result of 'inactivity' of the CA and conversely, if active vitellogenesis and growth of oocytes could be associated with high CA 'activity'. Figure 2 shows that the rate of synthesis of C_{16}JH in animals with inhibited T-1 oocytes is high in many instances (solid circles) while in animals in which ovulation had occurred, the rate of JH synthesis is lower (open circles). The mean rate of JH synthesis in animals with inhibited T-1 oocytes = 13.5 pmole h^{-1} per pair (n = 31) whereas in animals which had ovulated, mean JH synthetic rate = 8.0 pmole h^{-1} per pair (n = 18). Figure 2 demonstrates conclusively that in S. gregaria, the inhibition of growth in T-1 oocytes is not the result of CA inactivity, contrary to previous suggestions[11]. It might be argued that our assay procedure in vitro does not accurately reflect the synthetic capabilities of the CA in vivo. The validity of our assay has been discussed elsewhere[10] and we are confident that it accurately reflects the activity of the glands in vivo. It should also be noted that under all experimental conditions used to date, synthesis of JH in the CA of S. gregaria is followed immediately by release[18] – thus, there is no storage of JH within the CA. In the present experiments, by determining the C_{16}JH content of glands and incubation media separately, a similar relation has been observed.

Because inhibition of T-1 oocyte growth is not the result of low rates of JH biosynthesis, it is necessary to look elsewhere for the nature of the inhibition. At present, this is unknown but is has been suggested that an antigonadotropin emanating from the ovary is responsible for inhibition of T-1 oocytes in R. prolixus[8]. It is possible that a similar factor is operative in S. gregaria and in fact, the observation that T-1 oocytes become vitellogenic in partially ovariectomized animals[9] might be interpreted to indicate that an inhibitory factor is present in the ovary – when part of the ovary is removed, the inhibition is no longer effective. Work is in progress to define the nature of this inhibitory factor.

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Effects of cerebral lateral, ventricular infusions of phloridzin on feeding and body weight in Gallus domesticus (L.)

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Summary. Contrary to earlier findings in rats, cerebral lateral ventricular infusions of 1×10^{-3} M or 2×10^{-3} M solutions of phloridzin at a rate of 2.5 µl/min for 90 min had no significant stimulating effects on food intake and weight gain in hens and cocks. These different responses to intraventricular phloridzin might reflect a difference of sensitivity to the inhibitory action of phloridzin on glucose transport in cerebral cells or certain peculiarities of mechanisms controlling food intake in chickens.

Glick and Mayer[2] have found that cerebral lateral, ventricular infusions of phloridzin caused marked hyperphagia and excessive weight gain in rats. They attributed the overeating to inhibition of glucose uptake in neural tissue by phloridzin and interpreted the phenomenon as definite proof for the existence of cerebral glucoreceptors which are involved in the regulation of food intake.

The purpose of the present experiments was to investigate the effects of intraventricular phloridzin on feeding and body weight in Gallus domesticus. This is a species of bird which, taking into account its anorectic response to insulin, seems to have certain peculiarities in the mechanisms controlling food intake[3–5].

Material and methods. The experiments were carried out on 6 laying hens (White Leghorn) and 6 4-month-old cocks (White Rock × Cornish) housed individually and fed commercial chicken mash and water ad libitum.

To perform the cerebral ventricular infusions on unanesthetized birds, a technique developed by Goodrich et al.[6], and modified by us, was used. According to this technique, the lateral ventricle is punctured at the time of each infusion through an extradural guide-tube mounted chronically in the parietal bone. Details of construction for our modified guide-tube and probe assembly are shown in figure 1. Implantation of guide-tube was conducted under pentobarbital anaesthesia, using stereotaxic coordinates from the stereotaxic atlas of van Tienhoven and Juhasz[7].

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At least 1 week following surgery, when postoperative food intake returned to normal, the effect of cerebral lateral, ventricular infusions of phloridzin on eating, drinking and body weight of hens and cocks was tested. Infusions of 1×10^{-3} M or 2×10^{-3} M solutions of phloridzin ($C_{21}H_{24}O_{10} \cdot 2H_2O$, BDH) were carried out from a 5 ml motor drivensyringe at the rate of 2.5 µl/min for 90 min. In the control tests, similar infusions of physiological saline were made. The infusions for each bird were repeated 2–3 times at 7–14-day-intervals. Some of the hens were given initially phloridzin and others physiological saline. All clocks were infused at first with physiological saline and afterwards with phloridzin.

Individual daily food intake, water intake and body weight were measured throughout the experiments. For the evaluation of phloridzin or physiological saline action, the values recorded during the first 7–14 days after infusion were compared with those recorded during the last 3 days before infusion.

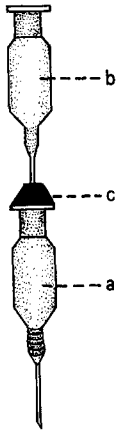

Fig. 1. Guide-tube and probe assembly: *a* guide-tube, constructed from the basal end of a No. 17 hypodermic needle from which the shank was extracted, and having a screw on its top in order to be screwed in the parietal bone; *b* a No. 17 hypodermic needle probe which penetrates tightly through the guide-tube; *c* collar of acrylic dental resin that set the depth of penetration of the needle probe into the brain so as the tip of the probe enters the lateral ventricle.

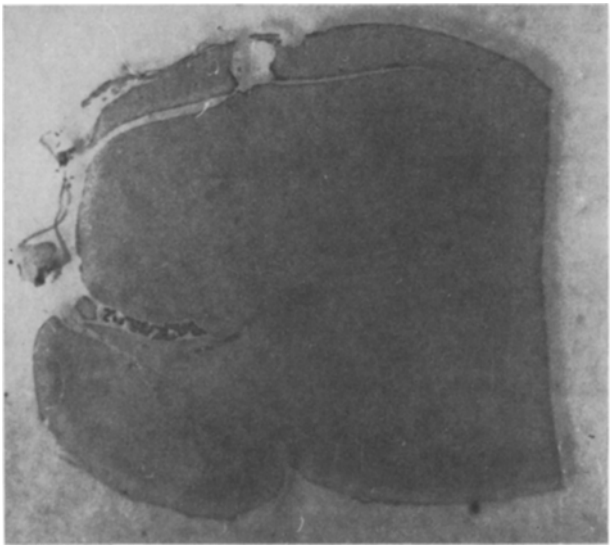

Fig. 2. Histological representation of a correct cerebral, lateral ventricular infusion.

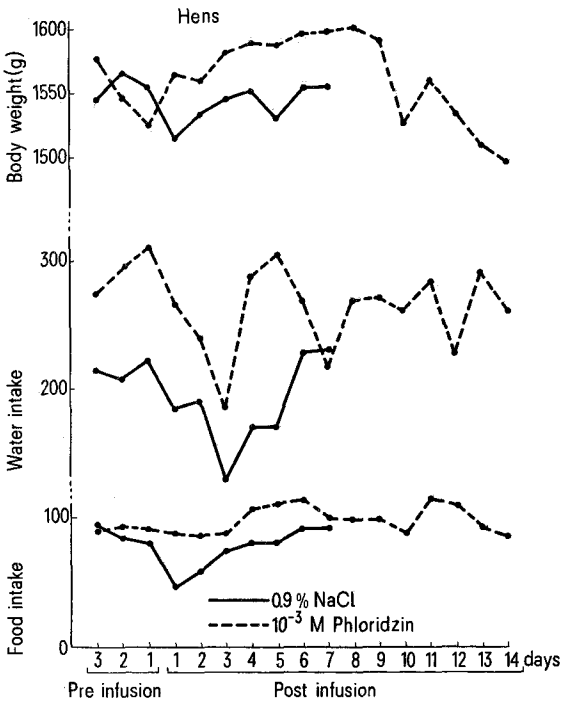

Fig. 3. Eating, drinking and body weight of hens following intraventricular infusions of physiological saline or 1×10^{-3} M solution of phloridzin (mean daily values).

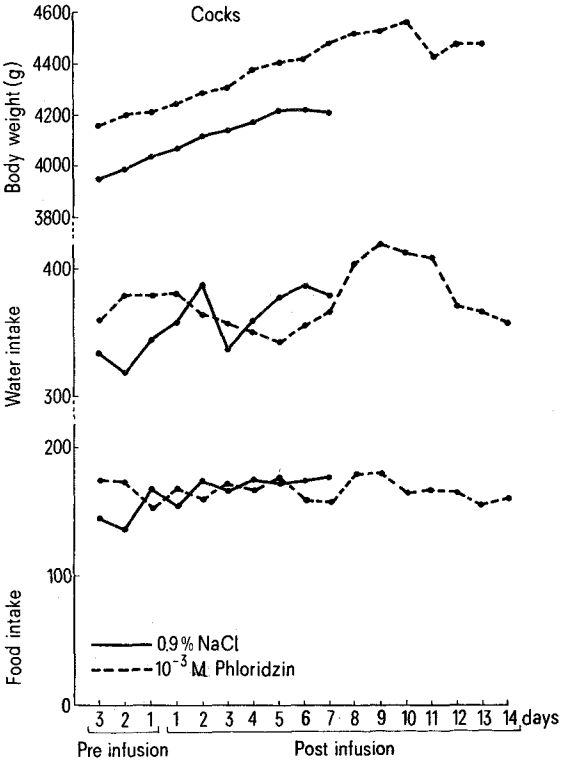

Fig. 4. Eating, drinking and body weight of cocks following intraventricular infusions of physiological saline or 1×10^{-3} M solution of phloridzin (mean daily values).

To detect the immediate effects of infusion, hourly measurements were also made during the first 3 h following it. Results were statistically evaluated by use of the t-test. At the end of the behavioral tests, all birds were sacrificed, and the fixed brains examined using paraffin block sections, stained with hematoxylin-eosine (figure 2).

Results. Pre- and postinfusions eating, drinking and body weight of hens and cocks are shown in figures 3 and 4. As we see in these figures, food intake diminished slightly on the first day following intraventricular infusion of physiological saline, returning thereafter to initial value in hens or exceeding this value in cocks.

Cerebral lateral, ventricular infusions of a 1×10^{-3} M solution of phloridzin did not result in significant changes of food intake in chickens. Slight increases were recorded in the first 3 days following infusion in cocks, or thereafter in hens. Only 2 of the hens presented mild hyperphagia starting as early as the first days following phloridzin.

Water intake varied significantly from one day to another. However, a clear decrease of drinking can be detected in the first days following intraventricular infusions of phloridzin and physiological saline in hens or only following phloridzin in cocks.

Body weight curves are more or less parallel to food intake curves. Most of the birds gained weight during the experiments. Mean daily weight gain did not differ significantly ($p > 0.1$) between the 2 treatments. Thus, weight gain in cocks was 35 ± 5.59 g following phloridzin and 27 ± 7.98 g following physiological saline. In both cases it did not exceed an average of 10 g in hens.

As figure 5 shows, there were not significant alterations of food and water intake in cocks during the first 3 h following cerebral intraventricular infusions of a 1×10^{-3} M solution of phloridzin. Lateral ventricular infusions of a double concentrated solution of phloridzin (2×10^{-3} M) rested also without a reliable effect on feeding and drinking of cocks.

Discussion. Glick and Mayer[2] have shown that rats doubled their daily food intake and gained up to 15 g/day following cerebral lateral, ventricular infusion of a 1×10^{-3} M solution of phloridzin at a rate of 1–2 µl/min for 50–90 min. These effects lasted from 48 h to 2 weeks, depending on the rate of infusion.

Although the concentrations of phloridzin solution were the same or 2fold higher and the rate of infusion was larger than the maximal rate used by Glick and Mayer, our results in chickens were not comparable with theirs. Hens and cocks did not significantly increase their food intake and gain in weight following intraventricular infusions of phloridzin.

It is not easy to interprete this lack of response to intraventricular phloridzin in chickens. It is possible that the dose of phloridzin and the rate of infusion used here were not sufficient to produce such an inhibition of glucose uptake that gives rise to glucoprivation in cerebral cells and elicit feeding. On the other hand, this could demonstrate a difference of sensitivity to the inhibitory action of phloridzin on the glucose transport in neural tissue. The failure of gold thioglucose to induce hyperphagia and obesity in the Japanese quail and chicks was also explained by a difference of sensitivity to the specific lesioning action of this substance on hypothalamic glucoreceptors[8,9].

If, besides the lack of hyperphagia following intraventricular phloridzin, we take into account anorexia produced by insulin in chickens[3–5], the hypothesis is then much more plausible that something other than, or in addition to, glucose metabolism is regulating feeding in this species of birds.

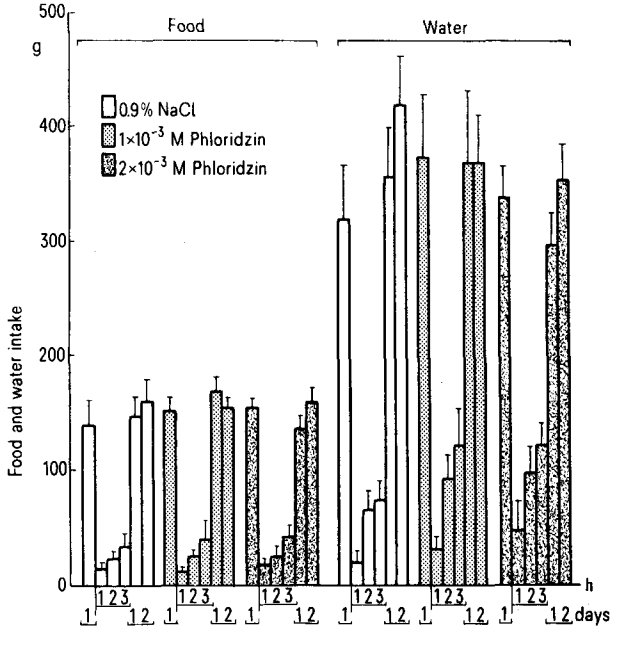

Fig. 5. Food and water intake in the first 3 h and respectively in the first and the second day following intraventricular infusion of physiological saline, phloridzin (1×10^{-3} M) or phloridzin (2×10^{-3} M) in cocks.

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