THE IMPORTANCE OF ADENOHYPOPHYSIS AND ITS "CONTROLLED" ENDOCRINE GLANDS IN THE OCCURRENCE OF BROODINESS IN CHICKENS

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In 1935, Riddle, Bates, and Lahr demonstrated that prolactin, the hormone which produces milk secretion in mammals, was capable in inducing incubation behaviour in laying hens. Since then, this hormone has often been referred to as the principal factor in building the mental states characteristic of parents during incubation and care of the young in birds. According to Riddle and Dykshorn (1932) and Schooley, Riddle, and Bates (1937), prolactin is effective in stimulating the crop sac of pigeons in the absence of the pituitary, adrenals, thyroids, or testes. Recently, a study made by Lehrman and Brody (1961), showed that prolactin is normally not principally responsible for the initiation of incubation behaviour in the ring dove.

It, therefore, seemed very desirable to determine whether injected prolactin in inducing broodiness in chickens is independent of the presence of the adenohypophysis and its "controlled" endocrine glands, i.e. gonads, thyroids, and possible adrenals, and if there is any other hormone, i.e. progesterone, which is responsible for the initiation of broodiness or incubation behaviour in chickens.

MATERIALS AND METHODS

Nine one-year old laying New Hampshire hens, weighing 1.60–2.85 kgs., were used in this experiment. They were divided into three groups of three hens each: pseudo-hypophysectomized, hypophysectomized, and hypophysectomized with adenohypophyseal autotransplants in the kidney capsules. The techniques of hypophysectomy and autotransplantation of adenohypophysis into the kidney were as described by Nalbandov and Card (1943) and Ma (1960). As to the technique of pseudohypophysectomy, it went as the same as that of hypopysectomy but remained the adenohypophsis *in situ* instead of sucking the adenohypophysis out.

After operation, all hens were placed in a growing cage with two head-on hanging infrared lamps for three weeks. Hens then were individually placed in the individual laying cages and treated with semi-darkness. Besides, each hen was intramuscularly injected with 140 I. U. of prolactin daily until she expressed the characteristic of broodiness. Two months later, all hens have completely recovered from the broodiness, they were treated with semi-darkness again and were intramuscularly injected with 0.5 mg. of progesterone every three days for three weeks. The purpose of this injection was tried to find that if this injection could induce broodiness in hens or not.

During the prolactin and the progesterone treated phases, a brood of chicks in the brood batteries were placed within the room for promoting psychologic stimulant to the hens in hastening the breakout of broodiness. Feed and water were *ad libitum* throughout the whole experiment. Body weight and comb size were recorded before operation, at the beginning of the prolactin injection and thereafter once every week. Environmental temperatures were also recorded.

RECOGNITION OF BROODINESS: Riddle, Bates, and Lahr (1935) stated that the full expression of broodiness (clucking and nesting) is induced only in those breeds showing a natural tendency to go broody by the injection of prolactin. Non-broody hens clucked, but did not set, when injected. Hens used in this experiment were New Hampshire of non-broody nature. Consequently, an indication of clucking from the hormone treated birds will be considered as the expression of broodiness.

RSULTS AND DISCUSSION

The principal results are shown in Table I.

Birds after hypophysectomy and autotransplantation not only stopped laying but also had a marked decrease in comb size. The comb size almost reached to the smallest extent in three weeks after operation and thereafter it maintained size without any practical change until the end of the experiment. All operated birds including pseudo-hypophysectomized, had a weight loss after operation, but they gained their weight again later on. Most of the autotransplanted and hypophysectomized birds, however, did not attain their body weight as that before operation. Birds excluding those pseudo-hypophysectomized, had molted and their newly growing feathers were of the characteristics as described by Ma (1960). Pseudo-hypophysectomized birds began to lay eggs again after they gained their weight, but they occasionally laid soft shelled eggs during the progesterone treated period. Progesterone may exert an inhibit effect of the formation of egg shells in chickens since such inhibit effect has already been demonstrated in pigeons and doves (Dunham and Riddle, 1942).

For four days after the daily injection of prolactin, all hens started to cluck. Further injections for another four days did not show any progress towards the full expression of broodiness. It definitely agreed with the finding of Riddle, Bates, and Lahr (1935) that non-broody hens clucked only when they were injected with prolactin. Evidently, the incubation behaviour response to the prolactin injection was independent of the presence of adenohypophysis and its "controlled" endocrine glands, i.e., gonads, thyroids, and possible adrenals. In four days after the stopping of prolactin injection, clucking was completely disappeared in all hens. It might be considered that the injection of prolactin is the principal factor for the initiation of incubation behaviour or broodiness in hens. However, the hypophysectomized hens showed the broody response to the injection of progesterone. All hypophysectomized hens started to cluck after the second injection of progesterone and lasted as long

Table I. The interaction of prolactin or progesterone injection and the adenohypophysis and its "controlled" endocrine glands on the occurrence of broodiness

(A: before operation; B: at the beginning of prolactin injection; C: at the end of experiment)

Hen number	Operation	Body wt. (Kg.)			Comb size (cm^2)			Expression of broodiness	
		A	B	C	A	B	C	Prolactin treatment	Progesterone treatment
1497	Pseudo-hypophysectomy	2.85	2.60	2.80	13.50	14.40	15.00	+	−
1459	Pseudo-hypophysectomy	2.05	2.20	2.50	10.85	12.25	12.25	+	−
1422	Pseudo-hypophysectomy	1.60	1.80	2.00	8.40	9 38	10.00	+	−
66	Autotransplantation	1.80	1.60	1.75	15.40	5.00	5.00	+	−
1436	Autotransplantation	2.40	1.60	2.00	15 17	6.00	5.80	+	−
1470*	Autotransplantation	2.40	2.00	——	15.58	7.00	——	——	——
519	Hypophysectomy	2.85	1 80	2.00	21.00	7.75	7.25	+	+
1445	Hypophysectomy	2.40	2.50	2.50	10.05	6.20	6.20	+	+
1495	Hypophysectomy	2.80	2.40	2 45	15.60	7.80	7.50	+	+

* Hen number 1470 died after the first injection of prolactin.

** Environmental temperatures were 60°F and 76°F during prolactin and progesterone treatment period, respectively.

as the injection continued. It has already been found in the dove that changes in the weight and histology of the dove's crop, which highly specific indicators of the presence of prolactin (Riddle and Bates, 1939), occur not before, but after the beginning of incubation (Beams and Meyer, 1931; Lehrman, 1958). Riddle and Lahr (1944) induced broodiness in pigeons and doves by administration of progesterone. These authors considered that the injection of progesterone caused the pituitary to release prolactin. Working on ring doves, Lehrman and Brody (1961) found that at dosage levels of prolactin inducing full crop growth, only 40% of the individuals tested sat on eggs while progesterone induces incubation behaviour in 70% of the individuals. This fact in addition to the findings of Beams and Meyer (1931) and Lehrman (1958), leads Lehrman and Brody to conclude that prolactin is normally not principally responsible for the initiation of incubation behaviour in the ring dove. It is not understandable why the injection of progesterone caused broody behaviour in hypophysectomized hens but not in the normal hens and the hypophysectomized hens with hypophyseal autotransplants while the injection of progesterone with a dosage of 1 mg. per bird normally is capable of stimulating the pituitary to release LH and thereby of inducing ovulation in laying hens (Rothchild and Fraps, 1949). Evidence from Ma (1960) and Ma and Chung (1961) showed that hypophysectomy leads to a rapid and complete degeneration of gonads, combs, and thyroids. In the group with autotransplanted pituitary glands, the gonads and combs degenerated completely. The thyroids, however, apparently retained their ability to function at a level somewhat below that of intact controls. So far as the adrenal glands concern, the adrenals in both hypophysectomized and autotransplanted birds retain their quantitative ability to function but they function at qualitatively different levels. It would be very significant that there is a common difference between hypophysectomized birds and those birds bearing an adenohypophysis either in sella turcica or in the kidney capsule. This difference is the complete degeneration of thyroid glands in the hypophysectomized birds, not in the adenohypophyseal autotransplanted and the normal birds. Would this difference make the expression of broody behaviour in hypophysectomized hens only after the injection of a small amount of progesterone?

SUMMARY

The injection of prolactin induced broody behaviour, clucking, in all pseudohypophysectomized, hypophysectomized, and hypophyseal autotransplanted New Hampshire laying hens. The effect of prolactin in inducing broodiness is independent of the presence of pituitary gland and its "controlled" endocrine glands. The injection of a small amount of progesterone could induce broody behaviour, clucking, in hypophysectomized hens, too. This situation suggests that the initiation of broodiness might be a very complicated physiological phenomenon. The action of progesterone in chickens was fully discussed within the body of this paper.

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