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The correlation between the physiological state of the thyroid of the mother and of the fetus.

(PRELIMINARY NOTE.)

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The starting point of this investigation was some observations (incidental to other work on the thyroids) that pups born of mothers having active hyperplasia of the thyroids seemed to have much larger thyroids than the pups born of mothers with normal thyroids or with colloid goiters. The size of the thyroid in pups from mothers with marked thyroid hyperplasia is in many cases so great that they produce the distortion of the neck similar to goiter in adults. *These pups are apparently born with goiter.*

The work was begun in the spring of 1912, and so far data have been obtained on mother and offsprings in the case of 16 cats and 14 dogs; the work is being continued and extended to other species.

It is well known that goiter (active hyperplasia and colloid) is prevalent in dogs in the Great Lakes region of United States, while in cats in the same region goiter is practically unknown. The goiter of the newborn of mothers with thyroid hyperplasia may be (1) primarily hereditary, that is, due to defects in the germ plasm, or (2) it may be due to some temporary metabolic disturbance in the mother,—toxins or abnormal concentration of normal products of metabolism, acting alike both on the maternal and on the fetal thyroid. If the fetal goiter is due primarily to the constitution of the ovum rather than to the maternal environment during intrauterine life, we would expect the goitre to persist in varying degrees after birth. We would also expect to meet with fetal goiter in the case of mothers with colloid goiters, because the colloid state appears to be preceded by active hyperplasia. On the other hand, if the fetal goiter is due primarily to some intoxication or temporarily altered metabolism of the mother, acting alike on the fetal and on the maternal thyroid so as to produce

hyperplasia, there ought to be no fetal goiters in the case of mothers with normal thyroid or with colloid goiters; and the fetal goiters of mothers with active hyperplasia ought to diminish after birth.

1. *Results in Dogs.*—During intrauterine life the body increases in weight faster than the thyroid gland so that the ratio of the weight of the thyroid to the body weight becomes gradually larger. But in the case of mothers with normal (or nearly normal) thyroids, the ratio of thyroid to body weight is always greater in the mother than in the pups; in the case of mothers with colloid glands the ratio may be greater in the pups than in the mother while in the case of mothers with thyroid hyperplasia the ratio may be the same, or it may be higher or lower, depending on the degree of hyperplasia of the maternal thyroid.

The newborn of mothers with active hyperplasia have invariably much larger thyroids than the pups of normal mothers, but the thyroids of the former do not exhibit any greater degree of differentiation toward adult structure. It would thus seem that the conditions causing thyroid hyperplasia in the mother lead in the fetus to excessive growth of the thyroid rather than to specific thyroid differentiation.

In the case of mothers with colloid goiter the thyroids of the pups are on the whole of the same size as in the pups born of normal mothers. A few thyroid ratios may be cited for illustration.

	Ratio of thyroid to body weight. Mother.	Pups.
V. Normal thyroid	1–6,600	1–3,000
X. Colloid goiter	1–1,300	1–4,200
III. Thyroid hyperplasia	1–1,100	1– 480

On the whole, the relative weight of thyroids in dogs, both adult and newborn, is large in comparison with that in cats. There may be considerable variation in the thyroid:body weight ratio in pups of the same litter. We have not been able to determine whether this is due to primary difference in the ova or to the varying factor of accessory thyroids.

2. *Results in Cats.*—Active hyperplasia or colloid goiter has not yet been found in our pregnant cats. But there is some variations in relative bulk of thyroid tissue. Thus the extremes of thyroid-body weight ratio are 1–4,680, and 1–16,000 respectively.

In general the adult pregnant female weighing 3–3.5 K. has thyroids weighing (fresh) 0.20 g. to 0.30 g. While thyroid hyperplasia has not yet been found in pregnant cats, it is a striking fact that mothers with relatively large thyroids give birth to kittens with relatively large thyroids and *vice versa*. The following figures may be cited as typical:

	Ratio of thyroid to body weight. Mother.	Kittens.
No. X	1–16,250	1–7,326
No. IV	1– 4,680	1–2,000

The results so far point to the following conclusions:

1. Active thyroid hyperplasia is not associated with hypersecretion of the thyroids, because hypersecretion of the thyroids in the mother would retard rather than augment thyroid growth in the fetus.

2. Since fetal goiter is always present in the offsprings of mothers with active thyroid hyperplasia, and never present in the offsprings of mothers with normal thyroids or colloid goiter, it would seem that the fetal goiter is not due primarily to the condition of the germ cells, but to some intoxication of the mother or altered condition of the maternal metabolism. Since the maternal environment acts on the fetus only by the way of the blood, the goiter must be due to substances in the blood acting alike on the fetal and the maternal thyroid to produce cell division and growth, rather than specific thyroid differentiation and secretion. This hyperplasia is therefore not compensatory.

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Parathyroid tetany and active immunity.

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Parathyroid tetany in dogs seems to be associated with diminished resistance to bacterial invasion of the mucous membranes, as shown by the frequent infection of the eyes, the nose, and the respiratory passages. This diminished resistance may be due to (1) depression of the processes of active immunity; (2) local de-