At autopsy (Table), control rats ovulated the normal complement of eggs but those with only the ovarian fragment showed a 50% reduction in the expected ovulation number for the animal. Compensatory ovulation did not occur although the remaining fragment had hypertrophied to the weight of 1 ovary from control rats. Adrenal weight of the experimental rats was increased over control values; uterine weight did not differ.

12 h following the removal of 1 ovary, there is a transient rise in FSH concentration in the plasma which returns to normal level within 36 h[4]. In turn, this rise in FSH causes an increase in the rate of proliferation of smaller sized follicles into larger ones by proestrus[5] and thus accounts for the doubling of ova shed by the remaining ovary in the unilaterally ovariectomized rat.

In the present study, the fact that the ovarian fragment hypertrophied to the weight of 1 ovary in control rats is indicative of FSH stimulation. However, why the ovarian fragment did not show an increase in the number of ova shed as the remaining ovary in the hemicastrate rat does is not known. Explanations such as disruption of the intra-ovarian blood supply, removal of the larger-sized follicles, steroid and gonadotropin imbalance or surgical stress can be postulated. However, even though the amount of ovarian tissue does not affect the total number of follicles which mature during each estrus cycle, the results do demonstrate that the number of ova shed is affected by the amount of ovarian tissue present. This finding indicates that the hypothalamic-pituitary-ovarian axis is specifically regulated for the existing conditions within the rat during each estrous cycle rather than being autonomous in regards to ovulation number.

Zusammenfassung. Nachweis, dass bei der Ratte nach Entfernung von $1^1/_2$ Eierstöcken das verblieben Fragmente trotz kompensatorischer Hypertrophie eine 50%ige Reduktion der Ovulationszahl zeigt, was dafür spricht, dass die Anzahl der abgestossenen Eier durch die vorhandene Gewebemasse beeinflusst wird.

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[4] V. D. RAMIREZ and C. H. SAWYER, Endocrinology *94*, 475 (1974).
[5] R. D. PEPPLER and G. S. GREENWALD, Am. J. Anat. *127*, 9 (1970).

Magnesium Status and Iodide Uptake by Thyroid Gland

The magnesium status of the rat has been found to affect the accumulation of iodide by the thyroid gland, magnesium deficiency decreasing and loading with magnesium salts increasing the uptake of ^{125}I from the blood in vivo[1]. This could be due to a direct action of magnesium on the thyroid, or it could be secondary to other effects produced by disturbances in magnesium metabolism, and experiments were undertaken to investigate the former possibility.

Two experiments were performed in which thyroid glands attached to fragments of trachea were excised from male Wistar albino rats weighing about 75 g and incubated individually for 6 h in 3 ml of medium using a procedure similar to that described previously[1], except that magnesium salts and potassium perchlorate were omitted from the basic incubation medium. 0.04 μCi of ^{125}I, obtained as sodium iodide from the Radiochemical Centre, Amersham, England, was added to the medium containing each gland. At the end of the incubation the gland was washed free from medium and the ^{125}I activity in both the gland and the medium was measured by scintillation counting.

Initially glands from stock rats were incubated in media containing magnesium concentrations of 0, 2, 4 and 10 mg/100 ml and the results are shown in the Figure. The only statistically significant difference in ^{125}I uptake from that observed at the physiological magnesium concentration of 2 mg/100 ml was a reduced accumulation by glands in media containing the very high concentration of 10 mg/100 ml ($p < 0.05$, Students t-test). This indicated that changes in extracellular magnesium do not produce the effects observed in vivo, but it did not exclude the possibility that they may be due to changes within the thyroid cells.

Three groups of weanling rats were therefore fed magnesium-deficient (0.3 mg/100 g), control (80 mg/100 g) and magnesium-loaded (350 mg/100 g) diets for 13 days, the composition of the diets being identical apart from their magnesium content. All rats received an amount of food equal to that consumed by the deficient animals and they were fed automatically[2] to prevent any differences in feeding pattern; distilled water was provided ad libitum. The rats were exsanguinated from the heart and the thyroid glands removed immediately. Glands from deficient animals were incubated in magnesium-free medium, those from control and loaded rats in medium containing 2 mg/100 ml of magnesium. The plasma magnesium concentrations were determined by atomic absorption flame photometry and indicate the development of magnesium deficiency and loading similar in magnitude to that obtained during the previous studies in vivo, but no

Accumulation of ^{125}I by thyroid glands of magnesium-deficient and magnesium-loaded rats (means ± SEM, $n = 7$ for each group)

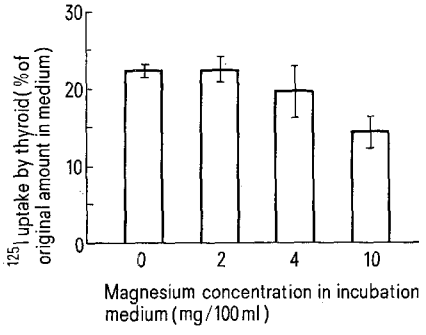
Group of animals	Plasma Mg (mg/100 ml)	^{125}I uptake by thyroid (% of original amount in medium)
Control	2.45 ± 0.10	5.05 ± 0.32
Mg-deficient	0.73 ± 0.10[a]	5.09 ± 0.23
Mg-loaded	3.66 ± 0.16[a]	4.43 ± 0.21

[a] Significantly different from control $p < 0.001$.

[1] F. W. HEATON and H. P. HUMPHRAY, J. Endocr. *61*, 53 (1974).
[2] B. W. LOVELESS, P. WILLIAMS and F. W. HEATON, Br. J. Nutr. *28*, 261 (1972).

significant differences in ^{125}I accumulation by the thyroids were observed (Table). Glands from magnesium-deficient and control rats accumulated almost identical amounts of radioiodine but there was a suggestion of reduced uptake by glands from magnesium-loaded rats ($0.2 > p > 0.1$). However, this tendency, like the reduced ^{125}I uptake observed in the first experiment, is the converse of that found in vivo during magnesium loading. The smaller ^{125}I uptake by all thyroids in the second experiment than in the first may be due to the food restriction and lower growth rate of experimental than stock rats.

The results of these experiments therefore indicate that the influence of magnesium status on iodide uptake by the thyroid in living rats is not due to a direct action of either extracellular or intracellular magnesium on iodide transport by the gland and it appears likely to be secondary to other effects of the deficiency that may be occurring elsewhere in the body.



Uptake of ^{125}I by thyroid glands of stock rats incubated in media containing different concentrations of magnesium. Vertical bars indicate ± SEM, $n = 4$ for each column.

Résumé. La quantité d'^{125}I absorbée dans le médium par les glandes thyroïdes incubées in vitro ne fut pas sensiblement affectée par la variation physiologique de la concentration du magnésium dans le médium, ni par la quantité de magnésium absorbée par les rats avant leur mort.

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Placental Impermeability to Maternal ACTH in the Rabbit

The permeability of the placental barrier to several protein hormones has been studied by means of radioactive tracers. It was shown that insulin[1], HGH[2,3], TSH[4] and glucagon[5] do not cross the placental barrier. Recently, Allen et al.[6] reported the presence of high ACTH levels in mother and foetus at delivery and, from the results observed in a Nelson's syndrome and in an anencephalic foetus, they suggested that there is no significant transfer of maternal ACTH to the foetus in humans. The purpose of the present paper was to verify this lack of placental permeability to maternal ACTH by injecting labelled ACTH into pregnant rabbits.

Materials and methods. 1–39 synthetic human ACTH, kindly supplied by Ferring A. B. (Sweden), was labelled with I^{125}, according to Greenwood et al.[7]. Purification of the labelled hormones was carried out as previously described[8] and I^{125}-ACTH was used within the first 24 h after labelling.

Two pregnant rabbit females nearing term, weighing 4.3 and 5.5 kg respectively, were used for the experiment.

In the first experiment, the animal (4.3 kg) was anesthetized with Nembutal (50 mg/kg body weight). After exposing both femoral veins, one vein was injected with I^{125}-ACTH (approx. 22.10^6 cpm) diluted in 1 ml of homologous plasma. Blood samples were collected from the other vein before and 2.5, 5, 7.5, 10 and 15 min after the injection. The uterus was then removed and blood samples were immediately taken from each foetus as well as pieces of each placenta. Fragments of kidneys, liver and adrenal gland were taken from the foetus and from the mother; 0.2 ml of blood from each sample and weighed fragments of each maternal or foetal organ were dissolved in 1 ml Soluene 100 (Packard) at 37 °C for 24 h and counted in an autogamma counter.

Table I. First experiment: Radioactivity in maternal and foetal blood (cpm/0.2 ml), and organs (cpm/mg)

Maternal blood		Fetal blood	
Prior to I^{125}-ACTH injection	0	Fetus No.	
Time after I^{125}-ACTH (min)		1	60
$2^1/_2$	18.598	2	140
5	12.465	3	190
$7^1/_2$	10.590	4	217
10	9.195	5	156
15	6.983	6	133
		7	219
		8	91
		9	177
		Mean value	153
		Standard error	18
		Maternal radioactivity at 15 min = 2.20%	

Maternal organs		Fetal organs (Mean ± standard error)	
Kidney	522.7	Kidney	0.78 ± 0.25
Liver	54.5	Liver	1.25 ± 0.30
Adrenal gland	60.5	Adrenal gland	—
Ovary	45.0	Placenta	70.35 ± 8.04