for the thyroid-fed animals and 6,431·6 per min. for the control group. Planchet counts performed at necropsy on weighed portions of thyroid glands (corrected for total gland) averaged 36,557·8 counts per 3 min. for the experimental animals as compared with an average of 40,808·1 counts per 3 min. for the control group.

The animals receiving the exogenous thyroid had a statistically significant increase in basal metabolic rates over the control group in spite of the decreased thyroid function of the experimental animals as indicated by iodine-131 uptake studies. It is also of interest that the average heart-rate of the thyroid-fed animals was not significantly higher. Thus, it would seem that when exogenous thyroid has been administered to the aged rat over a prolonged period of time thyroid function is to some extent altered; but this depression is not sufficient to counteract the additional stimulus produced by the thyroid fed to the animal.

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Calorigenic Action of Glucagon

A recent investigation carried out in this laboratory revealed that rats treated with glucagon gained less weight and contained much less fat, protein and water than pair-fed controls. Since the calorie intake of the two groups was identical and the calorie values of the carcasses of the glucagon-treated rats was much lower than that of the controls, the effect of glucagon on the metabolic rate of rats was investigated.

Male Wistar rats were used throughout the investigation. Of the eight animals used for each experiment, four were injected subcutaneously with 1 mgm. of crystalline glucagon (Lilly, Lot No. 258–234–*B*–54–2) suspended in neutral saline. The remaining four rats served as controls and were injected with saline. The oxygen consumption of each animal was determined at an environmental temperature of 28° C., according to the method of Ferguson and Sellers[1] at the intervals shown in Table 1. Four identical experiments were performed in this manner using rats that ate *ad libitum* up to the time of injection. A second group of similar experiments was carried out using rats that had been fasted 24 hr. prior to injection.

It is evident from Table 1 that glucagon induced a rise in the metabolic rate of the fed animals of approximately 35 per cent. The increase in oxygen consumption was maximal 1 hr. after the administration of glucagon and decreased slowly throughout the 7-hr. observation period. It is also evident from Table 1 that this effect of glucagon is only slightly reduced when the animals are fasted 24 hr. before treatment.

Table 1. The Effect of Glucagon on the Metabolic Rate of Rats

No. of rats	Mean body-weight (gm.)	Metabolic rate c.c. O_2 per m.^{2} body surface per min. at N.T.P. (Hr. after injection) 1·0	2·5	4·0	5·5	7·0
		Glucagon				
16	200	205±6*	182±6	165±3	154±4	158±5
		Saline				
16	207	152±5	140±4	135±4	134±3	138±4
		Change (per cent)				
		34·9	30·0	22·2	14·9	14·5
		Fasting + Glucagon				
12	192	183±8	150±5	138±3	138±4	133±4
		Fasting + Saline				
12	190	146±4	125±3	118±2	126±3	125±3
		Change (per cent)				
		25·3	20·0	16·9	9·5	6·4

* Standard error of the mean

A stimulating effect of glucagon on the metabolic rate has not been noted previously. The mechanism(s) involved is (are) not known. It does not appear that hyperglycæmia is a significant contributing factor since in fasting rats glucagon failed to elevate the blood-sugar level but raised the metabolic rate by approximately 25 per cent. Conversely, when hyperglycæmia was induced by administering glucose to intact rats, only a slight increase (4 per cent) in the metabolic rate was observed.

Additional experiments have shown that the calorigenic action of glucagon is greatly reduced by pretreating the animals with dihydroergotamine and is abolished completely by adrenalectomy; thus the possibility that adrenal hormones are involved in this phenomenon merits careful investigation. A more detailed account of these and other experiments will be the subject of another communication.

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Transport of Potassium-42 and Rubidium-86 by Kidney Cortex Slices: the Effect of Varied Concentration Gradients

A comparison of the transport of potassium with that of rubidium has been made in earlier work[1], in which some evidence was obtained that, in contrast to sodium, the transport of both potassium and rubidium across the membrane of renal tubular cells is determined only by electrochemical gradients. A deficiency in this work was that the rubidium content of tissue slices had to be estimated from differences in potassium content as compared with control slices. For this reason, the experiments have been repeated using potassium-42 and rubidium-86, to give a direct estimation, both being present only in the incubating medium (a modified Krebs–Ringer phosphate medium containing the indicated concentrations of potassium and rubidium, with 0·005 *M* alpha-keto glutarate as substrate, at 25° C.). Activity was measured in neutralized nitric acid digests of tissue slice samples, using aluminium dishes, with values corrected for decay, dilution, extracellular space, etc. Since the specific activities of the two isotopes were different by a factor of 4·0, the rubidium