cytochrome *c* for oxidative phosphorylation. This may point to a basic difference in the surface configuration of these structural elements of liver and brain cells. It may also be argued that the observed uncoupling of oxidative phosphorylation in liver mitochondria by chlorpromazine is secondary to the effect of the drug on mitochondrial structure.

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Effects of Pituitary Growth Hormone on the Insulin and Hyperglycæmic-glycogenolytic Factor extractable from the Pancreas of Obese-Hyperglycæmic Mice

IN two separate experiments it has been found that, 24 hr. after the administration of bovine pituitary growth hormone to mice with the hereditary syndrome of obesity and hyperglycæmia (1 mgm. of growth hormone per mouse), changes had occurred in the amounts of insulin and hyperglycæmic-glycogenolytic factor extractable from the pancreas using a standardized acid alcohol technique[1]. In each experiment values for insulin and hyperglycæmic-glycogenolytic factor values were compared in the following four groups of animals (ten or twelve animals per group): obese-hyperglycæmic mice and their non-obese sibs treated with growth hormone, and two groups of the same description but not treated with growth hormone.

The growth hormone treatment resulted in significant rises of 37 and 169 per cent in the average (non-fasting) blood-sugar level in the groups of obese-hyperglycæmic mice above pre-injection non-fasting levels. For the hyperglycæmic-glycogenolytic factor assays, concentrations of test materials were measured on a logarithmic scale, and the above rises were associated with logarithmic increases in the factor of 1·52 and 2·42 respectively, relative to untreated obese controls. The insulin extractable from the pancreas decreased by 48 and 86 per cent in the two experiments. No consistent changes were observed in the hyperglycæmic-glycogenolytic factor or insulin extractable from the pancreas of the non-obese mouse following injection of growth hormone.

Rise of the concentration of glucose in pancreatic blood plasma results in increased release of insulin by the pancreas in the rat[2] and in the dog[3,4]. The fall in the extractable insulin of the pancreas which followed the rise in blood-sugar level in the obese-hyperglycæmic mouse, treated with growth hormone in our experiments, may signify that the pancreas of these animals is not able, following a sudden increase in demand, further to increase insulin production[5] to compensate for the increased rate of release. The alternative explanation for the fall in the extractable insulin, namely, that it resulted principally from decreased insulin production without increased insulin secretion, is not, in our opinion, so well supported.

Mice with the obese-hyperglycæmic syndrome have a turn-over of liver glycogen several times faster than their non-obese sibs[6] and have six times as much liver phosphorylase activity as their non-obese litter-mates (Shull, K. H., and Mayer, J., unpublished work). In view of Sutherland's findings concerning the action of hyperglycæmic-glycogenolytic factor on liver phosphorylase[7], these observations are consistent with the previously postulated hypothesis[8] of a hypersecretion of the factor in these animals. These obese mice are unusually sensitive to the hyperglycæmic action of growth hormone[9], which has been observed to cause the release of a hyperglycæmic factor by the pancreas in rats and cats[10] and in dogs[3]. In spite of apparent differences between the conditions of action of this agent and the extracted hyperglycæmic factor of pancreas[11], the hypothesis that growth hormone in the obese hyperglycæmic mouse leads to a rise in blood-sugar level due to a hyperglycæmic factor originating in the pancreas is still supported. The observed co-existent increase in the hyperglycæmic-glycogenolytic factor extractable from the pancreas apparently indicates that growth hormone administered to the obese hyperglycæmic mouse caused the rate of production of extractable hyperglycæmic-glycogenolytic factor to exceed its rate of disappearance from the pancreas. Experiments which are in progress will, perhaps, provide more information than is available at present on these processes.

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Mechanism of Anorexia in Vitamin-deficient Hyperphagic Animals

RECENT findings[1] have demonstrated that the ventromedial hypothalamic nuclei act as 'satiety' brakes which, in response to certain stimuli (particularly metabolic), inhibit a constantly activated 'feeding' mechanism. This mechanism, in turn, appears to be initiated at the level of the anterolateral nuclei[2]. Destruction of the ventromedial nuclei does