gether with the various test substances. The glucagon content of the incubation media was then determined by radioimmunoassay. The release of glucagon from the islets in these conditions has been shown to respond to glucose and to amino acids in a similar manner to glucagon release from isolated rat islets and from perfused dog pancreas (Edwards, Howell & Taylor, 1969). Glucagon secretion from guinea-pig islets *in vitro* was found to be inhibited in the presence of low concentrations of fatty acids and of β -hydroxybutyrate. A very small inhibition was also found with high concentrations of glucose (Edwards *et al.* 1969).

More recently the effects of metabolic inhibitors have been studied. Cyanide (1mM), 2,4-dinitrophenol (0.25mM) and iodoacetate (0.4mM) all stimulated the release of glucagon from guinea-pig islets incubated in the presence of glucose alone. Inhibition of release by fatty acids was abolished by 2,4-dinitrophenol but not by iodoacetate.

Release of glucagon therefore is stimulated by a number of cell poisons and agents that disrupt the supply of intracellular energy. It is suggested that, in contradistinction to the release of insulin from the β -cell, expenditure of energy is necessary to retain glucagon within the α -cell.

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Glucose and Glycogen Metabolism in Hypertrophied Denervated Rat Hemidiaphragm

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Section of the phrenic nerve to one half of the rat diaphragm initiates a pronounced, although transient, hypertrophy of the muscle on that side. Feng & Lu (1965), from histological studies, found that the hypertrophy was solely a function of the 'red' fibres of the tissue, the 'white' fibres apparently only being capable of atrophy. Since red fibres and white fibres differ characteristically in their capacity to phosphorylate glucose and to participate in glycogen metabolism (Bär & Blanchaer, 1965), the activities of enzymes associated with these processes were investigated as a function of time after denervation to see if they reflect changing proportions of red cells and white cells in the fibre population.

Expressed per unit weight of tissue, the extractable activity of hexokinase remained largely unchanged for up to 15 days after denervation. During the hypertrophy phase, however, the tissue mass increases by some 30–40%, so that it appears that net synthesis of hexokinase is taking place. The concentration of glycogen in the tissue taken from fed rats was found to fall markedly as a result of denervation: after 3 days the value was 50% of that in the control tissues. Since Buse & Buse (1959) have established that glucose uptake is unaltered during denervation hypertrophy, it seems that glucose entering the cell tends to be metabolized rather than be stored as glycogen. Glycogen phosphorylase activity decreases significantly in the hypertrophied tissue; this decrease is reflected in the higher glycogen contents present in denervated muscle taken from rats starved for 16h before assay. Similarly activities of phosphoglucoisomerase and phosphoglucomutase were both found to have decreased significantly in concentration as soon as 3 days after denervation. These changes in enzyme activities, and especially in the decrease of the phosphorylase/hexokinase activity ratio, are considered to be indicative of a decreasing proportion of glycogenolytic, i.e. white-fibre, metabolism in the hypertrophying tissue.

Activities of glucose 6-phosphate dehydrogenase and 6-phosphogluconate dehydrogenase are found to increase immediately after phrenicectomy, reaching a maximum after 3 days and thereafter declining; this behaviour is compatible with the observed increase in nucleic acid content of the denervated muscle (Manchester & Harris, 1968), and is suggestive of a reorientation of metabolism to direct glucose 6-phosphate towards pentose synthesis.

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Glycerol Metabolism in Perfused Rat Liver

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Livers from starved female albino rats weighing about 200g were perfused by the Miller technique with haemoglobin-free Ringer solution. The preparations were stable for at least 2h. It was found that glycerol was metabolized at a constant rate by each liver, but that the rates varied from liver to liver for reasons as yet unknown. To achieve a steady perfusate concentration glycerol was added to the medium at the beginning of the perfusion, and from its rate of disappearance a rate of constant infusion of 33% glycerol was calculated that would