Effect of Hormones on Renal Gluconeogenesis

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It was demonstrated previously that glucose production in the kidney cortex was increased in diabetic, growth-hormone-treated and cortisone-treated rats (Joseph & Subrahmanyam, 1968). Pyruvate carboxylase (EC 6.4.1.1) activity, which was 3.20 ± 0.31 units in normal rats, was increased by 96% in the diabetic condition and by 67% by cortisone treatment. Growth-hormone treatment had no effect on the activity of this enzyme. Phosphoenolpyruvate carboxylase (EC 4.1.1.32) activity was increased by 49%, 58% and 69% in diabetic, cortisone-treated and growth-hormone-treated rats respectively. These findings, together with the previous finding of an elevated fructose 1,6-diphosphatase activity in all these conditions, explain the elevated glucose formation in the kidney cortex from gluconeogenic precursors.

The glycogen content of kidney cortex ($1.31 \pm 0.15 \mu\text{mol}$ of glucose equivalents/g wet wt.) was increased about fourfold in the diabetic condition and twofold by growth-hormone treatment. It was, however, unaltered by cortisone treatment. The activity of glycogen synthetase (EC 2.4.1.11) was not changed in any of these conditions. Phosphorylase (EC 2.4.1.1) activity was depressed by 53% and 41% respectively in the diabetic condition and by growth-hormone treatment. Cortisone treatment had no effect on this activity.

Thus the gluconeogenesis (glucose and glycogen production) in the kidney cortex of hormone-treated rats can be explained on the basis of enzyme activities.

Joseph, P. K. & Subrahmanyam, K. (1968) *Arch. Biochem Biophys.* 127, 288

Effect of L-Glutamate on Glucose Metabolism in Rat Cerebral Cortex

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Cerebral cortical slices of normal rats that had been injected with glutamate, glutamine and γ -aminobutyrate before they were killed were incubated with $[\text{U-}^{14}\text{C}]$ glucose. It was found that, whereas glutamate depressed the incorporation of the label from 9500 ± 608 to 5461 ± 350 c.p.m./g wet wt. of cerebral-cortex slice into glycogen and from

180760 ± 923 to 139116 ± 467 c.p.m./g wet wt. into CO_2 , there was no significant change as a result of injecting glutamine or γ -aminobutyrate.

One slice of cerebral cortex of normal rats was incubated with $[\text{U-}^{14}\text{C}]$ glucose and the slice from the other hemisphere with $[\text{U-}^{14}\text{C}]$ glucose + glutamate. The incorporation into glycogen was decreased from 9500 ± 608 to 4422 ± 503 c.p.m./g wet wt. and that into CO_2 from 180760 ± 923 to 84612 ± 1766 . Thus glutamate has a marked effect on the utilization of glucose in the cerebral cortex both *in vivo* and *in vitro*. Addition of insulin to the slices during incubation was not found to annul the depressing activity of glutamate on glucose metabolism.

In diabetic rats the incorporation into glycogen and CO_2 , which is already depressed (compared with normal slices), is markedly decreased further to about 20% for glycogen and 60% for CO_2 .

Studies on the permeability of radioactive glucose into the slices indicated that glutamate does not have any effect on this process. It is concluded that glutamate has a pronounced effect on the metabolism of glucose in the cerebral cortex of rats; this is being studied further.

Ligand-Induced Conformational Changes in Amylose, Studied by Spectrophotometric and Enzymic Methods

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Amylose is known to form helical complexes with butan-1-ol, cyclohexanol, dimethyl sulphoxide, iodine etc. The ligand-induced conformational changes in the polymer chain in an aqueous environment are detected by studying the influence of these complexing agents on the formation of the blue amylose-iodine complex (λ_{max} , 640nm) spectrophotometrically, and by the action of α -amylase (EC 3.2.1.1) and β -amylase (EC 3.2.1.2) on these as modified substrates. These complexing agents do not react with iodine in aqueous solutions at pH 4.8. The action of these enzymes on amylopectin is unaffected in the presence of complexing agents, as judged from the action pattern, optimum pH and K_m of these enzymes.

The influence of concentration of these complexing agents, time and temperature on the formation of the amylose-iodine complex is critically investigated. It is observed that there is a gradual increase in the absorption (λ_{max} , 640nm) of the blue amylose-iodine complex with increase in concentration of the complexing agent, reaching a maximum, followed by a decrease in the absorption, which in some cases is accompanied by a shift in λ_{max} , towards the blue region of the spectrum (640 to 600nm).