An Attempt to Establish an Unbiased Classification of Diabetes Mellitus.

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Q-technique of factor analysis was applied to obtain optimum classification of diabetes mellitus. This is based on the assumption that the characteristic symptoms inherent in diabetes are such as to make each individual more alike to his contemporaries than to patients of other groups. Correlations between different subjects are established to yield the inter-individual degree of resemblance. From the resulting correlation patterns, factors can be extrapolated which may be interpreted as classification schemes. — We analyzed the data on 149 diabetics. The onset of their diabetes was almost equally distributed throughout the first seven decades of age. In males there resulted a demarcation between the childhood diabetics, the young diabetics, and the adult diabetics. The first two formed clear groups, contrary to the adult diabetics who could not be combined to one uniform group. In females we found a simple bipartition with a division of the age of onset of diabetes at about 35 years of age.

Serial Post Prandial Blood Glucose Levels in 332 Subjects with and without Diabetes.


Venous blood glucose (BG) was assayed (autoanalyzer) in 332 subjects, 45, 60, 90 and 120 min after beginning of midday-lunch (usual meal or calibrated for diabetics) between noon and 1.30 pm, without any change in antidiabetic or other treatment. BG mean maximal values ranged between 113.3 ± 6.0 and 123.7 ± 6.8 mg/100 ml in healthy subjects (n = 45), and between 128.3 ± 5.8 and 132.5 ± 5 in non-diabetic patients (n = 60) according to

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sex, age and obesity. BG declined after 45 min. Among maturity-onset diabetics the closest to normal patterns were observed in non-obese; biguanide-treated \( (n=10, m=137\pm 6) \), diet-treated \( (n=10, m=158.5 \pm 12.6) \), and biguanide + sulfonylurea-treated \( (n=12, m=161.8 \pm 25.4) \) with a peak \( (T) \) for BG at 45 or 60 min. For obese diabetics \( (n=37) \), the maximal value was \( 165.4 \pm 14.1 \) in diet-treated \( (n=12) \) and \( 166.7 \pm 12.5 \) in biguanide-treated with an earlier decline of BG in the biguanide-treated \( (T=45 \text{ versus } 60 \text{ min}) \). With sulfonylureas alone the BG peak was higher and delayed. Among insulin-treated the more frac- tionated was the daily insu- lin therapy, the better were the post prandial BG patterns (in terms of BG increment and speed of decline): lente insulin once a day \( (n=20) \); \( m=244.2 \pm 30.1 \) \( T=120 \text{ min} \); intermediate twice a day \( (n=58) \); \( m=218.4 \pm 12.3 \) \( T=90 \text{ min} \); insulin 3 times a day \( (n=41) \); \( m=181.5 \pm 13.1 \) \( T=60 \text{ min} \). In maturity-onset diabetics the better results observed in some groups may be related simply to a milder form of disease. In insulin-treated pa- tients the better pattern obtained with multiple daily injections may be due to the use of regular insulin just before meals and/or the use of larger doses (+30% daily) in some cases.

**Analysis of Hyperglycaemic Effect of Adrenergic Stimul- ation and of Hypoglycaemic Effect of Adrenergic Blockage in Normal and Diabetic Rats.**

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The effect and mechanisms of combined or isolated alpha and beta adrenergic stimulation by adrenaline, noradre- naline and isoproterenol or blockage by phentolamine, propranolol and dihydroergotamine were estimated from changes of glycemia on the periphery (tail blood) and in the blood of v. hepatica sin. v. portae, aorta and v. cava caudalis, which were cunlubulated, as well from changes of hepatic glycogen determined simultaneously in normal and alloxan diabetic non-anesthetized or with pentobar- bital anesthetized and laparotomized rats. — The hyper- glycemic effect of adrenaline or beta stimulation in normal and diabetic rats is accompanied by a decrease in aorto-caval glucose difference, by an insignificant rise in hepatoportal glucose difference and by a decrease in hepatic glycogen, which together cannot, however, explain the sharp and large increase in glycemia. — Similarly, the decrease of glycemia in normal, but not in diabetic rats, after combined alpha and beta adrenergic blockage cannot be explained by changes of these parameters only. The enhanced blood flow after adrenergic stimulation is a contributing factor to hyperglycaemia and the slowed blood flow after adrenergic blockage to hypoglycaemia.

**Induction of Glycerokinase by Insulin in Adipose Tissue and Liver of BHob-Mice and Wistar Rats.**

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Using a modification of the radiochemical enzyme test described by Newsholme and co-workers, it was possible to demonstrate Glycerokinase (GK) in isolated epidydimal fat cells and liver tissue of obese hyperglycaemic mice \( (ob/ob) \) which was age-dependent and significantly higher than in lean controls \( (ob+ob) \). Changes of GK activity were significantly correlated to changes in serum IRI levels. — The dependence of GK activity from serum IRI was also observed in ob/ob mice made insulin-deficient by streptozotocin and thereafter substituted with insulin. The insulin effect on GK in fat cells and liver tissue was suppressed by actinomycin D. — *In vitro* incubation of epidydimal fat pads and liver slices obtained from Wistar rats has also shown a significant rise of GK activity after the addition of insulin. This effect was suppressed by actinomycin D. — It is concluded that GK in adipose tissue and liver of rats and ob/ob mice is induced by insu- lin. The pathophysiological significance of these results for the regulation of fat and glyceral metabolism will be discussed.

**Molecular Size of Circulating Glucagons in the Duck.**


Previous work using extracts from the duck pancreas and gut showed the existence of two pancreatic glucagon species ("small" and "large") and one gut glucagon ("largo"). In order to know what are the secreted forms, serum extracts were obtained from a) normal fasted, b) totally pancreatectomized and c) partially eviscerated (duodenal loop and pancreas retained) ducks. Ten animals were used for each extraction, the product was passed through previously calibrated Sephadex G 50 columns. — In all cases only glucagon species were detected: the "largo" one, with a molecular weight of about 6000. — The elution profiles obtained with glucagon from normal serum and eviscerated subjects (i. e. glucagon derived from the pancreas) coincide: that from the gut is marginally smaller. This may mean that there is more than one form of "largo" glucagon: in pancreas and gut extracts, however, these appear identical. The minor difference observed in the molecular weight is not felt sufficient to ascribe circulating glucagon to the pancreas rather than the gut. What is clear is that in ducks, unlike in mammals, the "small" pancreatic form (molecular weight 3800) is not released in significant quantities.

**Monolayer Culture of Pancreatic Cells: Evidence for Insulin Synthesis and Release.**


A monolayer cell culture technique was applied to neo- natal rat pancreas and the preparation characterized during an 8.5-day culture period by a) histology, b) assay of immunoreactive insulin (IRI) content and release. By light microscopy, the cell population was dominated by clusters of epithelioid cells, part of which were aldehyde- diazoxide each inhibited glucose-induced insulin release.

**The Importance of Insulin in Normal and Partially Pan- createctomised Chickens.**