tion may be clarified by plotting the longitudes at which it is found at different universal times (Fig. 2). As may be seen, the observed points, which are based on three years results in most cases, lie on a pair of straight lines separated by 180° (or 24 hr.), each of which repeats every 48 hr. Thus we can demonstrate the existence, at any instant, of two centres of activity on the D-region spaced 180° apart which rotate once around the earth every 48 hr. In contrast to the storm E, the probability of occurrence of storm D near the centre of the storm belt is almost constant throughout the western hemisphere between Sweden and Alaska.

The less intense forms of storm D perturbation, which do not completely attenuate all signals, have more complicated variations which combine the characteristics of the storm E and storm D perturbations discussed above.

An examination of individual disturbances shows that there is a close connexion between the areas affected by active D centres, $F2$-region perturbations and abnormally intense local magnetic activity.

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Insulin and the Permeability of Cell Membranes to Glucose

The effect of insulin in accelerating the peripheral utilization of glucose is generally ascribed to an action on the enzymic carrier system responsible for the intracellular oxidation of glucose, in which the first step is the hexokinase reaction.

More recently, evidence has accumulated suggesting that the locus of action of insulin may be, not on intracellular enzymes, but upon the transport mechanism whereby glucose is transferred across the cell membrane from the extracellular to the intracellular fluids. Such evidence is to be found in the work of Levine and his colleagues[1] on the effect of insulin on the distribution of galactose between the blood and the tissues, and in the effect of insulin in accelerating the rate of transfer of glucose across the blood-aqueous barrier[2].

This concept is further strengthened by studies on the uptake of glucose and other sugars by the isolated rabbit lens. Pairs of lenses immediately on removal from the eyes of anæsthetized rabbits were decapsulated and quickly weighed. They were then placed in tubes containing 3 ml. of Krebs–Henseleit–Ringer phosphate solution, pH 7·4, at 37° C. A sample of aqueous humour from one of these eyes had previously been removed by paracentesis and its glucose concentration estimated. Glucose was added to the medium so that its glucose concentration was identical with that of this aqueous humour. One unit of soluble insulin was added to one of the tubes, and both were incubated at 37° C. for 3 hr.; samples were removed from both tubes after 10, 60 and 180 min.

The mean uptake of glucose by the normal lens was 0·202 mgm./gm. lens substance/hr.; in the presence of insulin the uptake was 0·71 mgm./gm./hr. Insulin had thus accelerated the rate of uptake by 350 per cent. When homogenates of lens tissue were used, the rate of uptake of glucose was accelerated by only 33 per cent. When galactose was added to the medium and one hour allowed for equilibration, there was no uptake of galactose by the normal lens; but in the presence of insulin the uptake was 0·11 mgm./gm./hr. When sucrose was similarly employed, there was no uptake either in the presence or absence of insulin.

Insulin greatly accelerates the rate of glucose assimilation by the intact lens, but does so to a much less extent in homogenates in which the cell membranes have been broken down. Although the normal lens did not utilize galactose, nevertheless galactose is transferred into the cell in the presence of insulin; it is of interest to note that such lenses became opaque much more quickly than did lenses in the absence of galactose, suggesting that galactose blocks the glucose utilization mechanism. There is no specialized system for the transport of sucrose across the cell membrane, and hence insulin cannot promote the uptake of sucrose.

This work affords added support for the concept that an important action of insulin is to increase the permeability of cell membranes to biologically important sugars by accelerating an enzymic transport mechanism, and that in its absence such substances must enter the cell by solution in the cell membrane or by passage through 'pores'. The low lipoid solubility and high molecular weight of the sugars mean that their rate of entry by these routes would be slow, so that the organism, in the course of evolution, has developed these specific accelerator mechanisms to facilitate the entry of an adequate amount of glucose into the cell. The hyperglycæmia of diabetes mellitus is the result of the inability of glucose to enter the cells, rather than of a failure of intracellular enzymic oxidations. The intracellular oxidations essential for the life of the cell are maintained in diabetes mellitus largely by the utilization of 2-carbon-atom fragments obtained from the breakdown of fats, which are able to enter the cell because of their high lipoid solubility.

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Histochemical Detection with Ditetrazolium Chloride of some Enzymatic Activities in Isolated Mitochondria

In an earlier paper[1], I have reported that ditetrazolium chloride, a substance introduced by Seligman and Ruthenburg[2] for the histochemical detection of succinoxidase in tissue sections, can also be used for the detection of the same enzyme in mitochondria isolated from kidney and liver. This has been recently confirmed by Schiebler[3] on the granula isolated from the neurohypophysis by differential sedimentation.

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