

ant point bearing upon what next transpires. The condition is unassociated with hyperglycæmia, but hyperglycæmia should be present if the sugar molecules passed as such into the blood. It seems to me that we are driven to the conclusion that the sugar molecules must enter into combination with a constituent of the blood and then become set free again by the influence brought to bear when the kidney is reached. If a transport of sugar in one way or another occurs, as the evidence before us denotes it does, and it is not effected by passage in a free state, the only inference to be drawn is that the sugar is thrown out of sight by entering into a combined state.

Physiologists accept this explanation of the phenomenon of transport connected with phloridzin glycosuria. If admitted here, why should it not be equally admitted as applicable to what happens in connexion with the ordinary occurrences of life? Forced exercise leads to a rapid emptying of the liver of its glycogen, without producing any show of the passage of sugar through the circulation in a free state. If the sugar goes into combination in the one case, why should it not do so in the other? The circumstances stand in reality upon an analogous footing. The abstraction of carbohydrate from the blood to compensate for its consumption in the muscles during forced exercise, will create a demand for its replenishment, and thence lead to the store of glycogen in the liver being drawn upon. In the case of phloridzin glycosuria, the outflow of sugar that occurs in the kidney will, similarly to forced exercise, draw off carbohydrate and give rise to a demand for reinstatement from the glycogen store. The line of procedure is the same in the two cases, but the initial condition that leads to the demand for the replenishment of carbohydrate in the blood is different.

Support is given to the view that has been set forth by the modified conditions belonging to pancreatic glycosuria. Here hyperglycæmia precedes the glycosuria. The disappearance of glycogen occurs just as in phloridzin glycosuria, but the source of disappearance is conversion into sugar at the seat of storage. The kidney simply eliminates the sugar that is conveyed to it in the blood, and if the elimination is prevented by extirpation of the kidneys, the sugar goes on increasing, with the result of exceedingly high percentages having been noticed. The abnormality proceeds from a local transformation of glycogen into sugar, which recognisably passes into the blood in a free state, and subsequently flows off with the urine.

The instance that has been cited in reality shows what occurs when sugar from vanishing glycogen reaches the blood in a free state. In phloridzin glycosuria, there is equally a disappearance of glycogen, but