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Army Biological Defense Research Lab ltr dtd 22 Oct 1971

TRANSLATION NO. 598

DATE: July 1968

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DEPARTMENT OF THE ARMY
Fort Detrick
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A.REŽEK & B.UHLIK:

Effector action of endogenous insulin upon the activity of the alkaline phosphatase of the Blood*. Lijeć.vjesn.,Zagreb, 1954, 76: 609-610(Transl.from Serbo-Croatian by Claudius F.Mayer,M.D.,July 1959).

(From the Institute of Chemistry of the Veterinary Faculty of the University of Zagreb,and the Institute of Veterinary-Medical Researches,in Zagreb).

(*FOOTNOTE: A voluminous paper on this investigation will be in print,it is announced, in a short time at another place).

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An observation of one of the authors[1] that during the development of a per-orally provoked twice repeated and successive hyperglycemia(i.e., with the Staub-Traugott type of double glucose loading) will also change the concentration of the anorganic phosphate in the blood, has suggested to the authors that under the above mentioned experimental conditions they should try to learn also the behavior of the alkaline phosphatase of the blood. In this respect, they were little or in no way interested at first in the recognized relation between the activity of the mentioned phosphatase on one hand and the action of the insulin on the other hand. Taking advantage of the known fact that during the development of hyperglycemia,provoked in the described,physiological manner, insulin will also appear in the blood, they have expected that the outlined relation would be explainable,perhaps in the best way, precisely under the above mentioned experimental conditions. The investigations were carried out on rabbits.The following were established: - during the transient hyperglycemia (provoked by double successive physiological application of the glucose) the activity of the alkaline phosphatase of the blood will also change, and the changes occur at definite symmetrical relationship to the development of hyperglycemia. The activity of alkaline phosphatase of the blood changes several times during the transient hyperglycemia. One of these changes, namely the weakening of the activity is at which the intervention of insulin begins. The analysis of the described action leads to the unquestionable conclusion that the insulin controls (brakes) the activity of the alkaline phosphatase of the blood. During the development of the above mentioned hyperglycemia, the well-known activating function of the glucose(the BACCARI-AURICCHIO effect[2]) is also well distinguished, and perceptible is also the mild increase(slight increase) of the activity in the phase of

weakening of the insulin intervention(GAMBASSI-MATARAZZO effect [3]) and the obtained results are in accordance with the observations according to which the activity of the alkaline phosphatase is also increased in alloxane diabetes [4]. On the contrary, the obtained results are entirely contradictory to the observations of HIROSHI [5] according to which the insulin does not act as an effector upon the activity of alkaline phosphatase. The results of the described investigations allow the possibility that the chronically weakened activity of phosphatase in the case of the Gierke disease could be also in a certain relation with the described inhibiting action of the insulin. And in such a case, the above mentioned disease could be characterized as an "afermentosis", and not only because of the weakening of the phosphatase but also above all as an effect of the deranged activity - of the adaptive- protease insulinase [7] which is responsible for the disappearance of the endogenous insulin.

LITERATURE.

1 Unpublished.- See in the volume v.61(p.187, 1939) of Arch.intern.pharmdyn.thér. the article by A.REZEK and M.CIGLAR.

2. BACCARI V.G. & G.AURICCHIO: Boll.Soc.ital.biol.sper.24:441(1948).Quoted by Excerpta med.,1949,3/2,p.867.- Boll.Soc.chim.biol.,31:389(1949)Quoted by C.rend.Acad., 43: 9098 a(1939).- Boll.Soc.ital.biol.sper.,23:1066(1947).Quoted ibid., 42: 5989 d (1948).

3. GAMBASSI G.& C.MATARAZZO. Minerva med.,44: 198(1953).- Ibid., 44: 204(1953).

4. LASZT L.& H.VOGEL.Nature,158: 538(1946).

5. HIROSHI F.quot.by Ber.ges.Physiol.exp.Pharm.,117: 291(1941).-See also BAUR,H. Zschr.f.t.Horm.Fermentforsch., 2: 507-595(1948/49).

6. CORI G.T.:The Harvey Lectures,ser.48:N.Y.(Acad.Press),1944,p.145.Quot.by WIELAND O.in Klin.Wschr.,32: 385-392(1954).

7. BUSCHHAUS O.& E.KRETSCHMER & G.HALL. Ferment chemical studies to the clinical and constitutional correlation research.Berl.,1941,p.141. - See also GAMBASSI G.& CICOGIO I.Minerva med., 44: 209(1953).

(Translation of German summary)

The authors utilised the formation of the endogenous insulin,during hyperglycemia, which was produced by the double glucose load according to Staub-Traugott, for the explanation of the interaction between insulin and alkaline phosphatase. Rabbits were used as experimental animals. It was shown that the activity of the al-

kaline phosphatase changed several times during the course of the experimentally produced hyperglycemia. One of these changes, namely the weakening of the activity, corresponds regularly to the formation phase of the hyperglycemia at which the effect of the insulin begins to show. The explanation of this action leads to the unquestionable conclusion that insulin inhibits the activity of the alkaline phosphatase. The authors still point out that the weakened effect of the blood phosphatase can be in the Gierke disease in a certain relation with the established inhibitory effect of the insulin.

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