

Letter to the Editor

To

Editor-in Chief

Journal of Applied Biochemistry & Laboratory Medicine (JABLM)

Sir,

In reference to the article titled, "Glycation of Hemoglobin Among Individuals With Subclinical Hypothyroidism – An Urban CrossSectional Study" by Dr. Subhrajit Saha and Dr. Samarpita Mukherjee in the April 2022 issue, some interesting outcomes are evident. Thyroid hormones are one of the very few hormones which in fact increase blood glucose level from both ways. In cases where excess thyroid hormone production or response occurs, blood glucose levels are elevated. Ironically, deficient levels of thyroid hormones also lead to increase in blood glucose levels and its consequences e.g. an increase in HbA1c, as pointed out very aptly in the article presented by the authors as mentioned above. This letter makes an effort to highlight them in brief and extend the conclusion to their interaction with the blood glucose lowering effect of insulin.

Both of these observations have explanations, albeit different from each other. Hyperglycemia in hyperfunction of the thyroid gland or hyperthyroidism is mainly caused by increased gluconeogenesis and glycogen breakdown in the hepatic cells due to raised levels of T3 there that leads to increased release of glucose in blood raising the blood glucose level. In contrast, hyperglycemic effect in hypothyroidism results from decreased glucose uptake by the skeletal muscle cells due to reduced mobilisation of GLUT 4 transporter to their cell membranes caused by deficient T3 levels in blood. Thus, T3 stimulates the uptake of GLUT 4 proteins into the membrane and enhances cellular glucose uptake and utilisation. At this point T3 resembles the action of insulin. In contrast to this resemblance, T3 also antagonizes insulin function by inhibiting its synthesis in the beta cells of pancreas along with its increased rate of breakdown by enhancing its apoptosis. This theoretical irony of the thyroid hormone action on blood glucose is in fact, much relevant to the multivariate regulation of blood glucose by both thyroid hormones and insulin. As T3 itself enhances glucose uptake across the skeletal muscle lowering the blood glucose level, the same hypoglycemic effect by the insulin in addition would have resulted in deleterious and life threatening hypoglycemia if T3 would stimulate and stabilize the function of insulin also. This deleterious effect is prevented by the anti-insulin action of the T3 hormone thus enabling the thyroid hormones to balance the blood glucose level in conjunction with insulin. This regulatory function of thyroid hormone in both of its hyperactivity and hypoactivity on the overall blood glucose level seems as a unique property of this hormone. Thanks to the authors for presenting this article that brought out some unique functions of the thyroid hormone thereby.

With regards,

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