Review of: "Reduced Blood to Brain Glucose Transport as The Cause For Hyperglycemia: a Model That Resolves Multiple Anomalies in Type 2 Diabetes"

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Potential competing interests: No potential competing interests to declare.

The authors have put a great effort in explaining their model using a highly professional language.

The model assumed has shown gaps and controversy statements, which could be presented as follows:

1- They have mentioned as a definitive concept that the lateral ventricles controlling the glucose levels and energy homeostasis which could be a little divergent from what is known to be the role the hypothalamus and the brain-stem in taking this function.

2- The liver role in liberating glucose in the fasting period leading to high glucose levels is not clearly explained and rather make a gap in explaining this phenomenon

3- It is one known that the adult averagly weighted person is the reference model for staplishing the basis of health anatomy and physiology standers, and hence T2DM starts usually in 3^{rd} o 4^{th} decate of life in who where normal average adult does not support the theory of cerebral vascular anatomical or physiological disorders, and assuming vascular role in patients who are prediacetic or recentally diabetic in T2DM is not a true assumption , at such changes appear latter in T2Dm and the role of the vascular circulation could be appreachiated.

4- Amyloidosis is assumed to be in this model as taking role in the impaired function of Beta cells in the acute stage which is quite contradicting with the development of amyliodosis in acute stages, it is a rather complication of unknown origin appearing in chronic debilitating diseases

5- Autoimmune disorders have a contribution impairing the function of the Beta cells as well as viral infection as in mumbs and other pancreatic infections

6- The insulin resistance model could be supported by the over secretion of insulin by the pancreas and the sustained hypoglycemia in patients who are taking oral hypoglycemic tabs like Glibenclamide in which the driving force of high insulin level produced is counteracting the insulin resistance

7- Unlike anatomy pathological changes, unfortunately physiological phenomena can not be proved by postpartum examination.

Use of the terms like post-meal and after- feeding are not exact terms in case of diabetes, it is more specific to use 1-2

hours postprandial (PP) and the word feeding fits more with pediatrics patients and parenteral support.

I hope this is beneficial in this context to the authors and constructive in looking into the building of this model.