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.

One of the central problems in current classification and treatment of diabetes is that it is based on century old concepts and simplifications. As stated by the authors, the original use of insulin did not even consider dividing the disease into type 1 and type 2 diabetes. Current division of diabetes into type 1 and type 2 is still based on observational laboratory values of glucose and insulin with the molecular pathology being an explanatory after-thought. Even worse, most research is trying to define/defend single unifying molecular defects and pathologies as explanation for all cases off either type. This paper is making a convincing case for an overlooked molecular pathology, as playing a role in what we traditionally define as type 2 diabetes.

The paper challenges the current models to explain type 2 diabetes and suggests adding additional complexity to better explain the observations. It also makes a convincing case that some of the unexplained observations in diabetes may be explained by this expanded model.

However, in my opinion it may be better to take the next step and suggest that we completely discard the traditional lab-based classifications into two numbered subtypes – or even try to build a single model for type 2 diabetes.

Maybe it's time to redefine diabetes into (potentially dozens of) new endotypes based on specific molecular pathologies – each with its own diagnostic criteria and optimal pathology targeted treatments. Patients with hyperglycemia could even be diagnosed with multiple types of diabetes at the same time. Just as there is no reason to think that all diabetes patients have all observed pathologies there is no reason to think that most would have just a single cause/contributor to their hyperglycemia.

It is tempting for us humans to try and simplify a complex biological and pathological state, but it may be more productive to accept a complex reality where each patient have their own personal disease constructed from one or more interacting pathological processes. Treatment could then try to focus on each applicable pathology in any given patient.