Gestational Diabetes: The Field to Test the Validity of Insulin Resistance in Type 2 Diabetes

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Short Note

Globally, 382 million people have Type 2 diabetes and this is projected to increase to 592 million by 2035 [1]. The health care expenditure for adults with diabetes was estimated to be US $612 billion in 2014 [2]. It is clear that significant savings in healthcare expenditure cannot be realized other than through prevention. However, in order to prevent people from developing diabetes, we need is a much clearer understanding of what Type 2 diabetes really is.

Current medical science believes that Type 2 diabetic patients retain some insulin secretion, but insulin levels are low relative to the magnitude of “insulin resistance” and ambient glucose levels. Hyperinsulinemia in the basal state of any origin produces widespread insulin resistance [3]. The mechanism of insulin resistance is believed to be the improper functioning of the insulin receptor, resulting in a diminution of signal transmission to the interior of the affected cells, such as fat, muscle, and liver cells. Without the cell recognizing the presence of insulin, glucose cannot enter and remains in the bloodstream, causing high blood sugar. This explanation has come to be interpreted for using high blood sugar as a surrogate marker for insulin resistance.

Gestational diabetes mellitus (GDM) is defined as glucose intolerance of variable severity with onset or first recognition during pregnancy [4]. Although pregnancy-related factors such as placental lactogen can contribute to this, the cause of gestational diabetes is thought to be precisely the same as that in people with Type 2 diabetes. In both situations, insulin resistance of muscle fibers, the biggest user of glucose as fuel, is thought to contribute the most to the elevation of blood sugar.

Since insulin resistance appears during pregnancy and resolves completely within 6 to 13 weeks after pregnancy, in most cases, gestational diabetes offers an ideal field to test the validity of insulin resistance as the primary causative factor of type 2 diabetes. I suggest testing the degree of insulin resistance at the most important site, muscles, as soon as blood glucose level is elevated in a pregnant person. (If a site-specific insulin resistance test can’t be designed, even after 80 years of promoting insulin resistance as a causative factor of type 2 diabetes, that itself is a basis for doubting the validity of such a claim.) The change in the value of insulin resistance of muscles can be tracked, along with changes in blood sugar levels. More importantly, it can also be correlated with reduction in blood sugar levels as a consequence of treatment with insulin in pregnant women with gestational diabetes.

Such a test will clarify the role of insulin resistance not only in gestational diabetes but also in type 2 diabetes, much faster compared to the often-sited United Kingdom Prospective Diabetes Study [5] that took over a decade of treatment with medications to show significant improvement.

If such a study cannot be done, or shows that insulin resistance cannot explain elevated blood sugar, I propose an alternate possibility. What causes high blood sugar is that muscles use fatty acids for energy even when glucose is available, much like what happens inside the engine of a hybrid automobile that can burn gasoline or use electricity. The release of fatty acids from triglycerides by the action of external lipase outside fat cells that are too full to accommodate them has already been postulated [6]. High levels of fatty acids and triglycerides long before blood sugar levels start climbing have been demonstrated.

The global pandemic of diabetes, a non-communicable condition, along with the disproportionately high incidence of diabetes in less affluent populations in the world can become clear if one considers the link between diabetes, carbohydrate consumption, and obesity. The manufacture of cheap grain-based products and the promotion and marketing of “whole grains” as healthy foods are the causes of filling the fat cells of the consumers, whether they are poor, affluent, young, old, lean, obese or pregnant—setting off the metabolic switch to burn fatty acids and leave glucose in the blood.

This theory can explain why pregnant women can become diabetic within a matter of months, given how likely it is that they have filled their fat cells, creating an excess of fatty acids in the blood that muscles burn for fuel. My proposal is that this can be easily tested using pregnant women as the study group, as follows.

As soon as elevation of blood sugar is detected in a group of pregnant women, randomly assign them into two groups. The study group should then be asked to eliminate the consumption
of grain-based complex carbohydrate. Since consumption of carbohydrate is not essential for the wellbeing of a human being, this restriction should not adversely affect the developing fetus. The control group can continue the same diet they were on prior to the finding of elevated blood sugar level. Both groups can be followed with regards to the need for medications to keep blood sugar levels within accepted limits for a pregnant woman.

If this test proves that overconsumption of grain products rather than insulin resistance is the cause of type 2 diabetes, we will have two important targets for modification—one by governments and the other by individuals. Governments must begin promoting regional agriculture of fruits and vegetables rather than grain farming. Secondly, consumers must be reeducated to reduce and even eliminate consumption of grains whether they are presented as whole or mixed, and grain products even when they are fortified with vitamins or gluten free.

This can be the start of a new approach to diabetes care and prevention, one that can save the US federal government billions of dollars each year in healthcare costs from an aging population, 1 in 4 of whom are diabetic.

REFERENCES
3. Diabetes Care February 2008 vol. 31 no. Supplement 2 S262-S268