Gestational Diabetes Is Not Prevented By Exercise and Diet

By Chuck Dinerstein — January 9, 2019

Gestational diabetes (GD) is the development of diabetes during pregnancy. For those afflicted, it is a significant problem for these women and the children they carry. The mothers are seven times more likely to develop Type 2 diabetes, and the children “exposed” in utero have a greater lifetime risk of weight problems, obesity, and Type 2 diabetes.

Our epidemiologic data suggest that GD is like Type 2 diabetes, frequently with the same underlying bad behaviors: unhealthy foods and a sedentary lifestyle, and too many calories being taken in, with too few going out. To reduce the risk to those mothers who partake in such behavior, physicians begin with “eat better and exercise more.” How is that advice working out?

Early, preliminary studies demonstrated that reducing weight made a therapeutic difference. But when scaled up to large groups living in the real world, those injunctions about eating and exercise were ineffective despite good adherence and patients actually doing what was asked of them. Weight gain was reduced, however, gestational diabetes was not prevented. So blaming the patients is not the answer. Is the belief by physicians — that gestational diabetes can be prevented by avoiding too many calories with too little energy expenditure — wrong?

A small, well-performed, observational study reported in *Cell Metabolism* reconsiders the belief about calories, both in and out. It is a small study because it was rigorously objective in its measurements. No self-reports here. Energy intake was measured by summing daily energy expenditures, measured using doubly-labeled water and changing body energy composition. Calorie intake was based on a food photography methodology. [1]
Of the 62 women, 9 developed gestational diabetes over the 12 weeks of observation (during the second trimester). There was no difference in energy intake or expenditure, nor was there a difference in the calories or macro-nutritional composition of their meals as compared to the women not affected. Physical activity measured with calorimetry and accelerometry (a scientific way of describing a FitBit-like device) did not differ between those who did and did not develop gestational diabetes. And weight gain, in a sense, the summation of all these factors also did not vary. So what did?

The women who developed gestational diabetes tended to be heavier with more visceral fat, but that was not statistically significant. But those affected women more often had first-degree relatives with diabetes, a significantly higher fasting glucose and HbA1c – there was a greater incidence of “pre-diabetes” in these women. And it was identifiable in the first trimester. For those so inclined, it would suggest an inherited aspect to GD.

I say "inherited" because diabetes does not seem to be simply a genetic problem, but it involves “lifestyle” and perhaps some epigenetic alterations. Bottom line: gestational diabetes is not one disease; at least three distinct phenotypes have been identified. Simply being overweight or obese is not a good definition of risk, while alterations in glucose metabolism are far better.

Several consequences flow from this conclusion. First and most immediate, identifying those women at risk for GD should not be based on an eye-ball screening of weight. Instead, it requires an investigation of glucose metabolism. Weight is not a risk factor — it is the consequence.

Second, the problem of glucose dysfunction is more widespread than we think, since after all it’s clinically silent. There are many individuals who when stressed, by an acute illness or surgery, will demonstrate their glucose dysregulation. They’re difficult to identify because they are clinically silent when feeling well, their dysregulation — like that of the women with gestational diabetes — requires some additional stress. Some studies have shown that elevated blood sugars result in increased morbidity and mortality in hospitalized patients. Perhaps a more aggressive attempt to identify these patients could reduce those numbers.

Finally, while the idea of a mismatch in calories and energy expenditure contributing to diabetes is intuitively correct, it is not a panacea. Don’t stop exercising and your diet should be guided by moderation. And remember, while both actions are necessary, neither is sufficient on its own.

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[1] Let me take a run at explaining double-labeled water, which refers to water molecules with a slightly heavier form of hydrogen (deuterium) and oxygen 18. The only way for deuterium to leave our body is as water, occurring when we urinate or sweat. We can use that difference to determine how much oxygen left the body as urine and how much left the body as carbon dioxide. The only source of carbon dioxide is from our food, so knowing our carbon dioxide produced and subsequently lost tells us how much energy we are expending. As for food photography, the images can be compared in a computer application to determine the contained macronutrients – not exact but a lot more reliable than self-reports.

Source: Is Energy Balance in Pregnancy Involved in the Etiology of Gestational Diabetes in

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