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Biography

Stanley Schwartz is an affiliate of the main line health system and an emeritus associate professor of medicine at the University of Pennsylvania, currently in a private practice in Ardmore, Pennsylvania. Stanley Schwartz received his MD in 1973 from the University of Chicago in Chicago, Illinois. He then completed his residency at the University of Pennsylvania, followed by a fellowship in endocrinology and metabolism at the University of Chicago.

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Note:

A UNIFIED PATHOPHYSIOLOGIC CONSTRUCT OF DIABETES AND ITS COMPLICATIONS, INCLUDING MALIGNANCIES, IN THE CONTEXT OF THE β-CELL–CLASSIFICATION OF DIABETES

We have previously presented a proposal for a new, beta-cell centric classification of diabetes based on a consilience of genetic, metabolic, and clinical research that have accrued since the current classification was instituted. It recognizes that the beta-cell is the core defect in all patients with diabetes. Differences in the genetics, insulin resistance, environment and inflammation/immune characteristics of the damage to the beta-cell in each individual will determine the phenotypic presentation of hyperglycemia and allow for a patient-centric, precision-medicine therapeutic approach, part of which we labeled 'the Egregious Eleven'.

We now recognize the same pathophysiologic mechanisms that account for damage to the beta-cells govern the susceptibility of the cells involved in the complications of diabetes to damage by the now well-defined abnormal metabolic environment that typifies beta-cell dysfunction. This abnormal metabolic environment is typified by oxidative stress which alters metabolic pathways a la Brownlee's Hypothesis model, alterations in gene expression, epigenetics, and inflammation. This unified pathophysiologic construct of diabetes and its complications, including malignancies, in the context of the β-cell–Classification of Diabetes allows us to understand the varied risk of developing complications of diabetes with similar levels of glycemic control, how non-glycemic effects of some medications for diabetes result in marked complication risk modification and the value treating co-morbidities of diabetes in effecting complication risk.

Principles we outlined in using 'the Egregious Eleven' model- use agents that preserve beta-cell function, treat with least number of agents that treat most number of mechanisms of hyperglycemia- can be extended to use those agents, in combination, that also engender weight loss, and decrease CV outcomes. This approach allows for a more accurate assessment and treatment of each patient's disease and effecting true precision medicine.

We also believe that the same pathophysiologic mechanisms that account for damage to the beta-cells and govern the susceptibility of the cells involved in the complications of diabetes are likely to explain the association of cancer and cognitive deficiencies to diabetes and obesity, explaining why a diabetic medication may affect cancer risk and therapy.