International Journal of
Diabetes and Clinical Research

SHORT COMMENTARY

Changing the Face of Diabetes

*Stanley S Schwartz, MD, FACP, FACE*[*]

Emeritus Associate Professor, Perelman School of Medicine, University of Pennsylvania, Philadelphia, PA, USA

*****Corresponding author:** *Stanley S Schwartz, MD, FACP, FACE, Emeritus Associate Professor, Perelman School of Medicine, University of Pennsylvania, 771 Country Line Rd, Villanova, PA 19085, USA, Tel: (610)-547-2000*

It is time to embrace a more nuanced view of what it means to be "diabetic" and how to manage our patients with diabetes. To be clear, this is not a criticism of current doctrine or those that came before us. In fact, this is a call to recognize that time, knowledge, and understanding marches forward and we should not be too slow to appreciate this fact.

To wit, rather than diabetes classifications being fixed in stone, let us recognize that, great variation and overlap of "typical features" exists between the current DM classifications [1]. For example, approximately 30% of patients diagnosed with Type 1 diabetes mellitus (T1DM) have insulin resistance where as T-cell mediated autoimmunity is prevalent in patients diagnosed with Type 2 DM (T2DM) with reports of cellular and humoral islet autoimmunity in 41% and 14%, respectively, of patients with T2DM on metformin in the National Institute of Diabetes and Digestive and Kidney diseases GRADE (Glycemia Reduction Approaches in Diabetes) clinical trial [2,3]. Additionally, it is imperative to realize our ability to detect frank complications is limited and the damage and processes begin early in the disease continuum [4]. Further, it is important to widen our view of the causes of diabetes and its complications to incorporate common pathophysiologic processes beyond "just metabolic" [5,6]. Indeed the pathologic mechanisms that damage the b-cell (i.e. genetic/epigenetics, inflammation, abnormal environment, and insulin resistance) also contribute to cell and tissue damage elevating diabetes-related complications but also link to other common conditions including cancer, dementia, psoriasis, atherosclerotic cardiovascular disease, nonalcoholic fatty liver disease, and nonalcoholic steatohepatitis (Figure 1) [6].

With this said, this opens up the potential to begin to think differently with regard to how we manage our patients. Rather than feel stuck in the mud with single medication therapies for DM, we should fell emboldened to approach its management with multimodal approaches. Think of the money wasted trying to determine which drug was "better" when we could be considering drugs with complimentary benefits and mechanisms of action that address the multifaceted etiology of the DM spectrum [6]. In addition, some newer therapeutic classes not only have proven benefits for managing hyperglycemia, but also reduce cardiovascular and kidney risk as well as promote weight loss [6]. Furthermore, moving our understanding of a patient "unlucky enough" to get more than 1 disease besides DM toward recognizing the interconnection between common conditions allows us to further add to our armamentarium of potential drugs to address common etiologies across disease [6]. This broader view brings us closer to precision medicine for our patients. Importantly, as espoused by Dr. Sniderman of the McGill University Health Centre, clinicians must continue to value clinical reasoning and logic as well as evidenced-based medicine in order to move individualized patient care safetyforward [7]. We should endeavor to treat each patient as if they are our own family, recognizing that clinical reason AND science-based approaches are best, as we await evidenced-based corroboration of current therapies and as well as potential new advanced technologies (e.g, CRISPER, artificial intelligence [AI]) and novel drugs [8-10]. Indeed, imagine a future with AI-powered precision medicine building algorithms to classify key pathophysiologic processes and markers enabling physicians to identify those at risk for disease
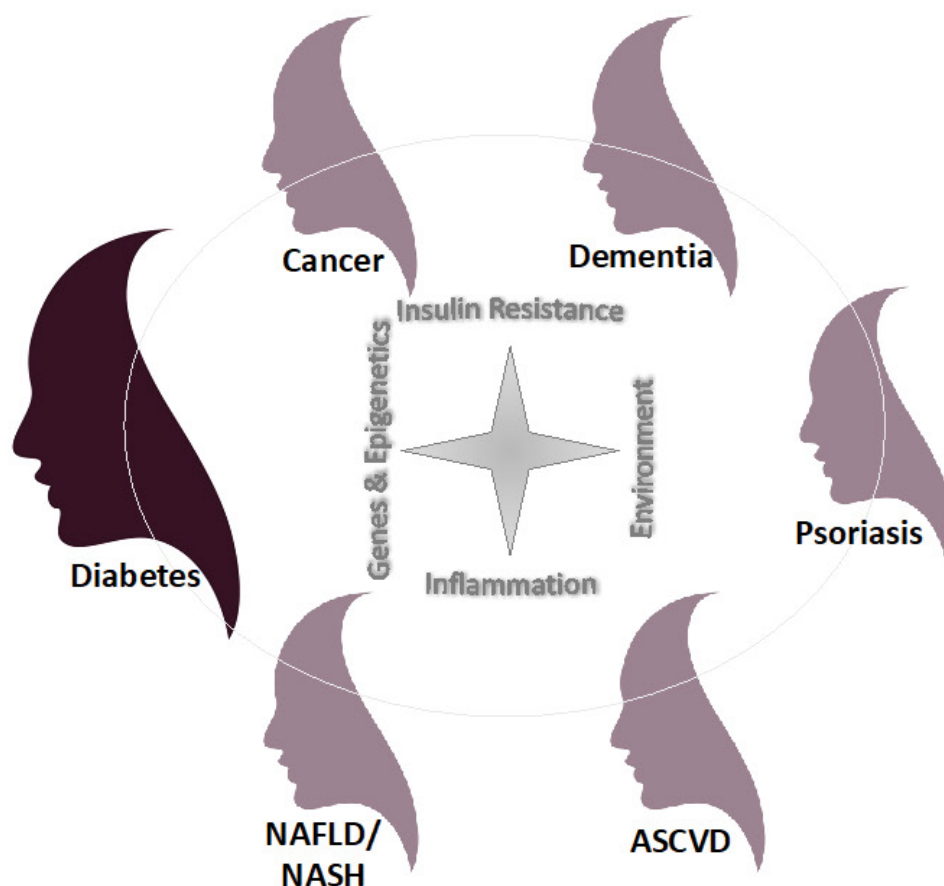
Figure 1: Common pathophysiologic mechanisms and triggers that an individual patient has or is exposed to can determine the clinical presentation or "face" of diabetes and other commonly linked conditions.

ASCVD: Atherosclerotic Cardiovascular Disease; NAFLD: Nonalcoholic Fatty Liver Disease; NASH: Nonalcoholic Steatohepatitis

and complications and determine the best therapeutic options for their patients on an individual basis [9,10]. Till then, we should continue to open to the recognition and treatment of diabetes in all its different faces with individualized approaches to deliver the best care for our patients.

Acknowledgements

Sources of Support

References

1. Schwartz SS, Epstein S, Corkey BE, Grant SF, Gavin JR 3rd, et al. (2016) The time is right for a new classification system for diabetes: Rationale and implications of the β-cell-centric classification schema. Diabetes Care 39: 179-186.

2. Leslie RD, Palmer J, Schloot NC, Lernmark A (2016) Diabetes at the crossroads: Relevance of disease classification to pathophysiology and treatment. Diabetologia 59: 13-20.

3. Brooks-Worrell B, Hampe CS, Hattery EG, Palomino B, Zangeneh SZ, et al. (2022) Islet autoimmunity is highly prevalent and associated with diminished β-cell function in patients with type 2 diabetes in the grade study. Diabetes, db210590.

4. Schwartz SS, Rachfal AW, Corkey BE (2022) The time is now for new, lower diabetes diagnostic thresholds. Trends Endocrinol Metab 33: 4-7.

5. Schwartz SS, Epstein S, Corkey BE, Grant SFA, Gavin Iii JR, et al. (2017) A unified pathophysiological construct of diabetes and its complications. Trends Endocrinol Metab 28: 645-655.

6. Rachfal AW, Grant SFA, Schwartz SS (2021) The diabetes syndrome - a collection of conditions with common, interrelated pathophysiologic mechanisms. Int J Gen Med 14: 923-936.

7. Sniderman AD, LaChapelle KJ, Rachon NA, Furberg CD (2013) The necessity for clinical reasoning in the era of evidence-based medicine. Mayo Clin Proc 88: 1108-1114.

8. Matboli M, Kamel MM, Essawy N, Bekhit MM, Abdulrahman B, et al. (2021) Identification of Novel Insulin Resistance Related ceRNA Network in T2DM and Its Potential Editing by CRISPR/Cas9. Int J Mol Sci 22: 8129.

9. Ellahham S (2020) Artificial intelligence: The future for diabetes care. Am J Med 133: 895-900.

10. Rashid J, Batool S, Kim J, Wasif Nisar M, Hussain A, et al. (2022) An augmented artificial intelligence approach for chronic diseases prediction. Front Public Health 10: 860396.

Schwartz. Int J Diabetes Clin Res 2022, 9:157

• Page 2 of 2 •