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Diabetes mellitus: Disorder of cellular dysfunction due to lack of entry into cell of glucose; the most efficient fuel for cellular function

Traditionally, diabetes mellitus has been deemed to be a chronic hyperglycemic disorder secondary to altered glucose metabolism. Alternatively, hyperglycemia may be one of several manifestations in subjects with type 1 and type 2 diabetes mellitus. Almost all tissues require insulin for entry of glucose, the possible exceptions being red blood cells, renal medulla as well as central and peripheral nervous systems. Hyperglycemia in intravascular compartment and other extra cellular milieu may be attributed to impaired glucose entry into endothelial cells of the vessel wall and almost all other cells including hepatocytes, myositis of all varieties, adipocytes and individual cells in most other organs respectively due to absence of insulin in type 1 and both the insulin resistance as well as the decline in both phases of insulin secretion in type 2 diabetes. Albeit, the decline in both phases of insulin secretion are induced by lack of glucose entry into pancreatic beta cells. Finally, hyperglycemia is perpetuated by increased hepatic glucose production caused by into sustained circulating hyperglucagonemia secondary to lack of glucose entry into the pancreatic alpha cells. Alternatively, both the decline in insulin secretion by the beta cells and the rise in glucagon release by the alpha cells are enhanced by fall in GLP1 and GIP caused by dysfunction of L cells and K cells respectively secondary to lack of glucose entry in both type 1 and type 2 diabetes. Similarly, increased prevalence of infections and thromboembolic micro and macro vascular events may be attributed to dysfunction of leukocytes and platelets respectively due to impaired glucose entry. Finally, alterations in several other metabolomics including serum concentrations of Adiponectin (Adipose cells), TNF alpha, Plasminogen inhibitor factor 1, Homocysteine, CRP, Lipids, etc., (Hepatocytes) as well as dysfunction of several organs (liver, heart, kidney, adrenal, pituitary, lungs, etc.) in both type 1 and type 2 diabetes may also be attributed to the lack of glucose entry into these specific cells. This hypothesis is validated by improvement in metabolomics and organ function on facilitation of glucose entry into cells by insulin administration and/or improvement in insulin sensitivity. Therefore, in conclusion, diabetes mellitus is a disorder manifesting dysfunction involving almost all organs and cells induced by lack of entry of glucose, the most efficient substrate for cellular function.

Biography

Udaya M Kabadi completed General Medicine residency at KEM Hospital and obtained Post-graduate degree (MD) from Mumbai University, Mumbai, India. He continued his Post-graduate training in Internal Medicine and fellowship training in New York, NY, USA. He is Board Certified in Internal Medicine, Endocrinology, Metabolism and Geriatric Medicine by American Board of Internal Medicine. He is a fellow of American College of Physicians, American College of Endocrinology and Royal College of Physicians of Canada. He is an Adjunct Professor of Medicine at Des Moines University, Des Moines, Iowa and University of Iowa, Iowa, USA. He has authored over 190 papers in peer reviewed medical journals and 2 books. He has conducted over 500 CME presentations and chaired symposia at regional, national and international meetings. He serves as a member of Editorial Board of many medical journals. He has also been elected 'Teacher of the Year' by undergraduate students and post-graduate trainees on several occasions during his career.

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