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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, myocytes 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 macrovascular events may be attributed to dysfunction of leukocytes and platelets respectively due to impaired glucose entry. Finally, alterations in several other metabolemics 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 metabolemics 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.

Speaker Biography

Udaya M Kabadi is a graduate of Seth G.S. Medical College, the University of Bombay in Bombay, India. He completed his internal medicine residency at KEM Hospital Parel in Bombay and a medicine residency at Jewish Memorial Hospital and Beth Israel Medical Center in New York, He also completed a fellowship in endocrinology and metabolism at VA Medical Center and Beth Israel Medical Center in New York. He is board certified in internal medicine, endocrinology and metabolism and geriatric medicine by the American Board of Internal Medicine. He is a fellow of the Royal College of Physicians of Canada, the American College of Physicians and the American College of Endocrinology. He has been a chief editor, associate editor and member of editorial boards of several medical journals. He is currently an adjunct professor of Medicine at the University of Iowa College of Medicine, Iowa City as well as Des Moines University, Des Moines, Iowa. He has over 200 publications in peer-reviewed journals. He has presentations to his credit, at regional, national, and international arenas. He has been selected as 'Teacher of the Year' many times by students, residents, and fellows in training. He has been involved in research in the area of carbohydrate metabolism and diabetes, thyroid disorders and osteoporosis as well as in clinical practice and education for several years.

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