Type II diabetes may affect stem cell niche resulting in down regulation of glucose transporters and insulin receptors in cells

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Abstract

Characterized by high blood glucose concentration, resistance of cells to glucose uptake and reduced insulin sensitivity, Type II diabetes is a major health problem afflicting both developing and developed countries in increasing extent as populations around the world increasingly adopt high energy diets. Given that, in Type II diabetes, successive generations of various cell types in the body ranging from muscles, tissues, blood, and organs are resistant to glucose uptake and exhibited reduced sensitivity to insulin, the underlying aetiology of Type II diabetes might involve the altered gene expression of stem cells in stem cell niches that adapted to a high glucose diet through an evolutionary conserved mechanism that aimed at homeostasis. Specifically, faced with a high energy and high sugar diet, stem cells in stem cell niches around the body possibly activated an evolutionary conserved mechanism aimed at reducing glucose uptake by cells for reducing weight gain by the body. Thus, successive generations of cells generated from the stem cell niche would exhibit an epigenetically controlled programme of gene expression that exhibited down regulation of genes for glucose transporters and insulin receptors. Such cells would display a phenotype of reduced glucose uptake together with reduced sensitivity to insulin; thereby, resulting in a high blood glucose concentration characteristic of Type II diabetes. The above hypothesis helped explain why high sugar intake by the body could result in impaired sensitivity to insulin and reduced glucose uptake by cells, and more importantly, the widespread nature in which many cell types (principally muscle cells) are affected by a possible epigenetically controlled gene expression programme which hitherto appeared clinically irreversible. Specifically, the most important clinical question for diabetes treatment and care remains the reasons underlying the clinically observed irreversible nature of the disease that progressively, with age and poor glucose control, worsens with complications to many organs of the body such as the eyes, kidneys, cardiovascular system and brain (stroke). Interested readers are invited to expand on the ideas presented in this abstract preprint.

Keywords: epigenetics, stem cell niche, high blood glucose, impaired insulin sensitivity, reduced glucose uptake, gene expression, Type II diabetes, muscle cells, homeostasis, weight gain,

Subject areas: diabetes and endocrinology, biochemistry, cell biology, metabolic sciences, internal medicine,
Conflicts of interest
The author declares no conflicts of interest.

Author’s contribution
The author thought about the systemic nature in which body cells became resistant to glucose uptake and insulin, and inferred that the stem cell niche of the body might be affected by high sugar consumption. Specifically, through an evolutionary conserved mechanism, a cellular programme might be activated where expression of glucose transporters and insulin receptors were down regulated through epigenetic memory. Thus, successive generations of body cells ranging from muscle to blood and cells in organs became resistant to glucose uptake, leaving a high blood glucose concentration that characterized the clinical symptoms of diabetes. He wrote the abstract preprint.

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