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O-GlcNAcylation and Diabetes

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In the world 463 million adults are currently living with diabetes. One of the etiologies of diabetic complications is acceleration of the hexosamine biosynthetic pathway (HBP). The HBP is a metabolic pathway from fructose-6-phosphate to the final product uridine 5'-diphospho-N-acetylglucosamine (UDP-GlcNAc) which is the donor for O-GlcNAc modification (O-GlcNAcylation). Many nuclear and cytoplasmic proteins are O-glycosylated on their serine or threonine residue with a single monosaccharide, N-acetylglucosamine (GlcNAc), which is termed O-GlcNAc. O-GlcNAc is not elongated beyond the monosaccharide and abundant especially in the nucleus. This modification takes place at the same or adjacent sites as does phosphorylation. O-GlcNAcylation regulates transient phosphorylation. O-GlcNAc plays a role in the regulation of insulin signaling and acts as a mediator of glucose toxicity. Although glucose entering the HBP is about 2–5% of the total, this metabolic pathway is enhanced under hyperglycemia. HBP coordinates cellular metabolism in response to UTP, acetyl-CoA, glutamine, and glucose levels. The influx of glucose through the HBP and subsequent changes in O-GlcNAc levels mediate insulin signaling pathways. In the chronic hyperglycemia state O-GlcNAc levels are abnormally increased in the cells and tissues of diabetic animals and humans. Based on these and other studies, elevated levels of O-GlcNAc have been characterized in several models of diabetes and are being investigated as a marker of pre-diabetes. I will discuss on the potential roles of O-GlcNAc in diabetic complications.

Biography

Yoshihiro Akimoto is a Professor of Department of Anatomy, Kyorin University School of Medicine. He received his Ph.D. at University of Tokyo in 1986 and has worked in the Kyorin University since 1986. He did his postdoctoral work with Prof. Gerald W. Hart at University of Alabama at Birmingham. He is an Associate Editor of Acta Histochemica et Cytochemica. .