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A Short Commentary on Oxidative Stress in Diabetes Mellitu

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DESCRIPTION

Diabetes complications has both micro vascular and cardiovascular, are exacerbated by oxidative stress. Diabetes-related metabolic changes result in mitochondrial reactive oxygen species overproduction in endothelial cells of both small and large vessels, as well as in the myocardium. This increased reactive oxygen species production activates five major pathways involved in the pathogenesis of complications: polyol pathway flux, increased formation of advanced glycation end-products (AGEs), increased expression of the AGE receptor and its initiating ligands, activation of protein kinase C (PKC) isoforms, and hexosamine pathway over activity. Also it inhibits the activity of two significant antiatherosclerotic enzymes, e NOS and prostacyclin synthase, Increased intracellular ROS start causing defective angiogenesis in reaction to ischemia, activate several pro-inflammatory pathways, and end up causing long-lasting epigenetic changes that drive persistent expression of proinflammatory genes after glycaemia is normalized ('hyperglycemic memory'). Atherosclerosis and cardiomyopathy are partially caused by path that leads insulin resistance that also tends to increase mitochondrial ROS production from free fatty acids and causes ROS to inactivate anti-atherosclerosis enzymes, Superoxide dismutase overexpression in transgenic diabetic animals precludes diabetic retinopathy, nephropathy, and cardiomyopathy. The purpose of this literature review is to highlight recent advances in our understand the role of metabolite-generated ROS in the development of type 2 diabetes complications. Overall, diabetic micro vascular complications are affected by exposure to high levels of glucose. The extent of diabetic tissue injury is also determined by genetic determinants of individual susceptibility, as well as the presence of independent accelerating factors such as hyperlipidemia, as with atherosclerosis. Large-scale prospective studies for both type 1 and type 2 diabetes have defined the importance of hyperglycemia. The vast majority of articles published on the underlying mechanisms reactive hypoglycemia diabetic vascular damage focus on five key methods: increased flux of glucose and other sugars through the polyol pathway, increased intracellular formation of advanced glycation end-products (AGEs), increased expression of the receptor for AGEs and its activating ligands, activation of protein kinase C (PKC) isoforms, and over activity of the hexosamine pathway. However, medical trials where only one of these paths is blocked have yielded disappointing results, in 2000; it was proposed that all five mechanisms are activated by a single upstream event: mitochondrial overproduction of reactive oxygen species (ROS). The polyol pathway is predicated on a family of aldo-keto reductase enzymes that can use a wide range of carbonyl compounds as substrates and decrease them to their respective sugar alcohols via nicotinic acid adenine dinucleotide phosphate (NADPH) (polyols). This was originally believed that the enzyme aldose reductase converts glucose to sorbitol, which will then be oxidized to fructose by the enzyme sorbitol dehydrogenase (SDH), with NAD+ as a cofactor, Aldose reductase is found in a variety of tissues, including nerve, retina, lens, glomerulus, and vascular cells. Carbohydrate uptake in many of these tissues is mediated by insulin-independent GLUTs; thus, intracellular glucose concentrations rise in tandem with hyperglycemia. A few hypotheses have been put forward to explain how hyperglycemia-induced increases in polyol pathway flux could harm the tissues involved.

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CONFLICT OF INTEREST

We have no conflict of interests to disclose and the manuscript has been read and approved by all named authors.

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