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# Elevated Cortisol and/or Simple Carbohydrates in Stress and Stress-Related Diseases

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## Opinion

Cortisol, a steroid hormone, in the glucocorticoid class is produced in humans by the zona fasciculata of the adrenal cortex within the adrenal gland [1] and released in response to stress and low blood-glucose concentration [2]. It represents a potentially important mechanism linking chronic stress with accelerated aging. In a study, a 24-hour urinary excretion of cortisol was found to link with an elevated marker of DNA and RNA damage in older adults [3]. Previously, Du et al. [4] reported that, long-term high-dose cortisol administration dramatically decreased mitochondrial function and promoted cell death in culture cells.

In the early fasting state, cortisol stimulates gluconeogenesis (formation of glucose from non-carbohydrate source), and activates anti-stress and anti-inflammatory pathways. Cortisol also plays an important role in liver and muscle glycogenolysis (glycogen to glucose-1-phosphate and glucose) [5]. However, in the late fasting state, cortisol increases glycogenesis, where the livers takes up glucose not being used by the peripheral tissue and turn it into liver glycogen stores to be used by the body during fasting state [6]. In the later case, cortisol counteracts insulin and contributes insulin resistance [7] by decreasing the translocation of glucose transporters, especially insulin-sensitive glucose transporter (GLUT)-4 to the cell membrane [8].

Glucose is a major circulating sugar and primary fuel for the animal brain for energy production. Not only GLUT-1, but also GLUT-4 also helps to enter glucose into neural cells [9]. Thus, an increase in cortisol level contributes stress in the animal's brain. The fight or flight response to emergency or stress involves glycogenolysis, gluconeogenesis, and so on decrease motility of the digestive system, cause secretion of the cortisol [10]. Hyperglycemia is evident to contribute to cellular and tissue injury by increasing oxidative stress (by overproduction of ROS, such as superoxide radical) and cause DNA damage [11,12]. In a study, cisplatin was found to induce nephrotoxicity in rat renal cortical slices, where a reduction in gluconeogenesis was seen along with the down-regulation of reduced glutathione (GSH), while up-regulation of lipid peroxides (LPx) [13]. In this study, it was seen that the amino acids such as glycine and L-arginine showed the promising nephroprotective capability as they reduced LPx, while increased in GSH levels in the experimental animals. An excess of fructose and glucose in the bloodstream causes extensive glycation and damage the proteins [14]. Advanced glycated products (AGEs) play a critical role in aging, diabetes, atherosclerosis and cardiovascular diseases, and in neurodegenerative diseases. Some of these AGEs are also reported to link with an increased fatty acid oxidation in arterial endothelial cells [15,16].

The polyol pathway transforms excess of glucose into sorbitol, which in turn produces a variety of intracellular changes in vascular tissues, including conversion of glucose to fructose by sorbitol dehydrogenase [17]. Fructose at high concentration can increase reactive oxygen species (ROS) production [18], that can cause excessive cytokine (pro- and/or inflammatory) production, thus the inflammatory response. Excess ROS is also evident to cause damage of proteins, lipids, and genetic materials (e.g. DNA and RNA). ROS can alter signal transduction and cause insulin resistance *via* mitogen-activated protein kinase (MAPK), protein kinase C (PKC) and c-Jun N-terminal kinase (JAK) mediated pathways.

Diabetes is characterized by an increased production of sorbitol, that can induce nitric oxide synthase (iNOS), reactive oxygen/nitrogen species (ROS/RNS); cause oxidative-nitrosative stress, endogenous antioxidant depletion, enhanced lipid peroxidation, and so on [19].

Substances such as methylglyoxal (MG) cause insulin resistance [20] may associate with an induction of hypertension [21], which can increase the levels of uric acid [22], thus the increase in nicotinamide adenine dinucleotide phosphate (NADPH) oxidase activity and oxidative stress [23].

In summary, simple carbohydrates after ingestion readily available in the blood, increase cortisol to let them convert into

glycogen. Both, glycogenolysis and gluconeogenesis are also invloved in stress induction by multiple pathways. Moreover, hyperglycemia is also evident to induce oxidative stress in human and other animals, by increasing glycation and increase in fatty acid oxidation.

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