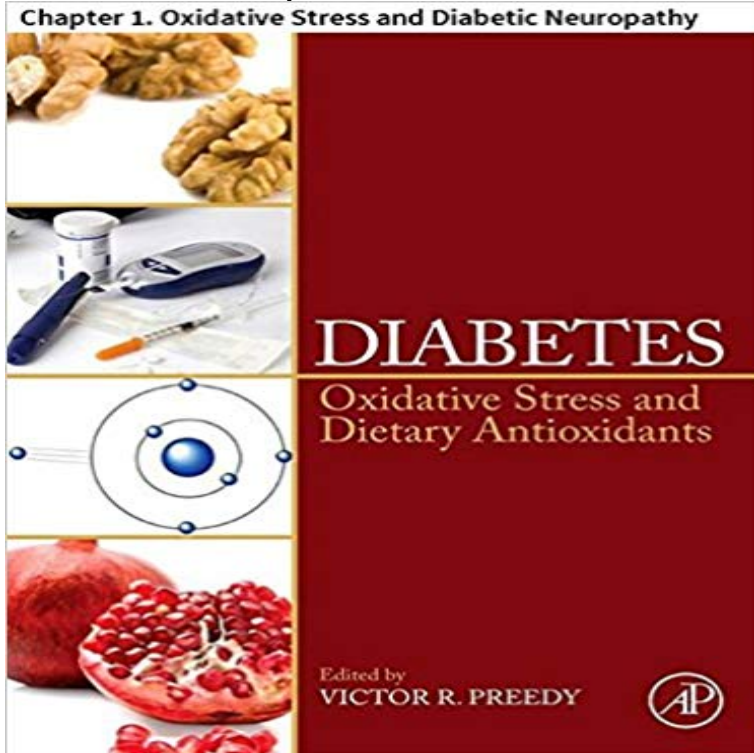


# Diabetes: Chapter 1. Oxidative Stress and Diabetic Neuropathy



Diabetic neuropathy is a serious complication in 60-70% of diabetic patients involving both type 2 and type 1. Several signaling pathways have been assumed to contribute to its development, including increased activation of the polyol pathway, oxidative stress, advanced glycation end product (AGE) formation, nerve hypoxia/ischemia, activation of protein kinase C and reduction of nerve growth factors. As a central integrating mechanism, overproduction of reactive oxygen species in peripheral nerves leads to oxidative stress, mitochondrial dysfunction, neuronal damage and finally apoptosis. Oxidative stress results in an increase in substrate for AGEs and in precursors of glycooxidation and lipoxidation products, and accelerates the free radical formation that may be accompanied by or caused by a deficiency of antioxidant and detoxification pathways. Oxidative stress is accompanied by the activation of the NF- $\kappa$ B, and then mitogen-activated protein kinases (MAPK) in Schwann cells as well as in neurons, which will lead to the activation of various inflammatory cytokines. Therefore, antioxidant treatment may improve neuronal repair in patients with diabetic neuropathy. However, the true mechanisms by which increased oxidative stress affects downstream signaling mechanisms, and how this results in the development of diabetic neuropathy have not yet been thoroughly studied. Neither is it known whether antioxidant supplementation could help the damaged neurons to regenerate, and thus enable patients with diabetic neuropathy to overcome this devastating, life-threatening situation.

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**Antioxidant-Rich Natural Products and Diabetes Mellitus** Contributors. Section I: Oxidative Stress and Diabetes. Chapter 1. Oxidative Stress and Diabetic Neuropathy. Abstract. Acknowledgments. List of Abbreviations.

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