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P174. THE EFFECT OF ARSENIC ON GENE POLYMORPHISM ASSOCIATED WITH OXIDATIVE STRESS

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Arsenic (As) is found commonly in nature and exposure to arsenic is unavoidable for human. Not only the consumption of drinking water and foods contaminated with arsenic but also occupational exposure to arsenic can cause toxicity. This toxicity can lead to various diseases. In addition, arsenic-induced formation of ROS and subsequent depletion of antioxidant cell defences result in disruption of the antioxidant/prooxidant equilibrium in mammalian tissues. According to its sulfydryl group binding capacity, arsenic also inhibit the activities of many enzymes, especially those involved in the uptake of glucose in cells, fatty acid oxidation and production of glutathione. The induction of oxidative stress by arsenic may influence gene expression. Genes involved in endogenous defenses against ROS thus may modify arsenic's effect. For example, MnSOD, a metalloenzyme which contains manganese cofactor, is the enzyme placed in the mitochondria to convert superoxide to hydrogen peroxide and thus has a key role in quenching free radicals generated by the electron transport chain, and overexpression of MnSOD can lead to an accumulation of reactive oxygen species (ROS), which contributors to certain diseases and tumor progression. Second important enzyme associated with oxidative stress is p22 phox, a critical enzyme for superoxide production and an essential component of nicotinamide adenine dinucleotide phosphate (NADPH) and oxidase (NOX). The effect of arsenic on polymorphism in genes associated with oxidative stress (Mn-SOD and p22phox) has been discussed in present updated overview.

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