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Redox regulation in health and disease: Novel hypothesis

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In view of the critical role of redox system in numerous physiological and pathophysiological processes, it is important to clearly understand the constitution and regulatory mechanism of redox system. In this work, we will systematically review the current data detailing the reactive oxygen species (ROS), enzymatic and non-enzymatic antioxidants and redox sensitive transcription factors and we give a brief description of redox-exerted epigenetic and post-translational regulation. We propose that the redox system functions as a "Redox Chain", consisting of "ROS-generating Enzyme Chain", "Combined Antioxidant Chain" and "Transcription Factor Chain". We suggest that redoxomic techniques should be extensively applied to understand the biological effects of redox alterations in a more integrated way. A stable and standardized "redox index" is urgently needed for the evaluation of the general redox status. We suggest that for the redox intervention of an individual, an individualized assessment of the redox status in the body should be conducted. The strategy of intervention is to maintain general redox balance rather than to conduct simple pro-oxidant or anti-oxidative interventions. These findings provide valuable new insights into redox system and open up new paths for the control of redox-related disorders.

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Detoxification of aflatoxin B1 using dietary molecular species

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Aflatoxin B_1 (AFB_1) is a class 1 carcinogen and a common food contaminant worldwide. It is also a major cause of the development of hepatocellular carcinoma (HCC), making dietary exposure to this toxin very concerning. Existing strategies to reduce AFB_1 exposure are limited and as a result, many people are exposed to this toxin worldwide. Issues with current detoxification strategies include harmful byproduct formation, incomplete removal, or the requirement of sophisticated infrastructures. Our study aims to develop a new chemical treatment process to modify AFB_1 into a non-carcinogenic form using benign reagents found in human diets. Our strategy targets themutagenic site of the AFB_1 molecule, the 8,9-double bond, by adducting it to selected amino acids in dietary proteins.Identification and quantification of aflatoxins was performed using high performance liquid chromatography-electrospray ionization-time of flight mass spectrometry (HPLC-ESI-TOFMS). Optimization of AFB_1 hydration was carried out by incubating in various organic acids as well as increasing temperature. Newly formed AFB_{2a} was introduced to alkaline solutions containing amino acids, peptides, and other biological molecules. Products were identified based on changes in retention times and accurate mass values. Mutagenicity of the resulting adduct was determined using an Ames' test with and without the presence of hepatic microsomes. This study provides a basis for developing a safe and effectivedetoxification method for contaminated foods, reducing exposure to AFB_1 worldwide.

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