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**Bioluminescence-based detection of brain immune cells apoptosis and ATP depletion induced by aflatoxin B<sub>1</sub>**

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Caspases-mediated apoptosis/cell death activation is key regulatory response in many physiopathological conditions. Application of bioluminescence and the reaction of luciferase would provide a powerfully novel *in vitro/vivo* assay for apoptosis detection. As key brain immune cells, astrocytes and microglia, are vital part of the central nervous system (CNS); they are the main responder to inflammation in CNS; any disruption on their function would lead to CNS damage. Aflatoxin B<sub>1</sub> (AFB<sub>1</sub>) is commonly found in foodstuffs, and can be the cause of many diseases including cancer. AFB<sub>1</sub> and its metabolites cause oxidative stress in especially the CNS-derived cells, adversely affecting their normal activities, thus leading to the neurodegenerative diseases including multiple sclerosis (MS), Alzheimer's and Huntington's diseases. Considering the importance of astrocytes and the inevitable existence of AFB<sub>1</sub> in the feed/foods, worldwide, the study of astrocytes-AFB<sub>1</sub> interactions is valuable. We therefore investigated the impact of AFB<sub>1</sub> on the apoptosis of one of the key accessory supportive CNS, astrocytes, using several biochemical experimentations including intracellular ATP and caspases 3/7 measured by bioluminescence and luciferase reactions. The release of cytochrome c and apoptosis/necrosis of AFB<sub>1</sub>-treated astrocytes with various concentration of AFB<sub>1</sub> and exposure time was also tested using Western blotting and flow cytometry techniques, respectively. Bioluminescence results revealed decreased intracellular ATP, increased caspases 3/7 activities, cytochrome-c release and apoptotic/necrotic of astrocytes particularly at higher timepoints and doses of AFB<sub>1</sub>. Considering the broad roles of astrocytes in CNS, this finding deepens our understanding of the molecular mechanisms and functional consequences of the neural cells damage neurotoxicity triggered by AFB<sub>1</sub> exposure in mammals.

### Biography

Jalil Mehrzad has completed his PhD at the age of 32 years from Ghent University, Faculty of Veterinary Medicine, and postdoctoral studies from McGill University. He is an associate professor of Immunology in Ferdowsi University of Mashhad (next year will move to Tehran University as full-time scientific member of department of Microbiology and Immunology). With H-index and citations of 16 and 1315, respectively, he has published more than 45 papers in reputed journals and has been serving as regular reviewer for many journals in the area of immunobiology, molecular biotechnology and medicine.

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