Cytotoxic and genotoxic effects of epoxiconazole on F98 glioma cells

Titre	Cytotoxic and genotoxic effects of epoxiconazole on F98 glioma cells
Type de publication	Article de revue
Auteur	Hamdi, Hiba [1], Abid-Essefi, Salwa [2], Eyer, Joël [3]
Editeur	Elsevier
Type	Article scientifique dans une revue à comité de lecture
Année	2019
Langue	Anglais
Date	Aout 2019
Pagination	314-323
Volume	229
Titre de la revue	Chemosphere
ISSN	1879-1298
Mots-clés	Apoptosis [4], Cell cycle arrest [5], cytoskeleton [6], Epoxiconazole [7], F98 cells [8], mitochondria [9], Ros [10]
Résumé en anglais	Epoxiconazole (EPX) is a very effective fungicide of the triazole family. Given its wide spectrum of use, the increased application of this pesticide may represent a serious risk on human health. Previous studies have found that EPX is cytotoxic to cells, although the exact mechanism remains elusive. In particular, the effect on the nervous system is poorly elucidated. Here we evaluated the implication of oxidative stress in the neurotoxicity and studied its apoptotic mechanism of action. We demonstrated that the treatment by EPX reduces the viability of cells in a dose dependent manner with an IC50 of 50 µM. It also provokes the reduction of cell proliferation. EPX could trigger arrest in G1/S phase of cell cycle with low doses, however with IC50, it induced an accumulation of F98 cells in G2/M phase. Moreover, EPX induced cytoskeleton disruption as evidenced by immunocytochemical analysis. It provoked also DNA fragmentation in a concentration dependent manner. The EPX induced apoptosis, which was observed by morphological changes and by positive Annexin V FITC-PI staining concurrent with a depolarization of mitochondria. Furthermore, the cell mortality provoked by EPX was significantly reduced by pretreatment with Z-VAD-FMK, a caspase inhibitor. Moreover, N-acetylcysteine (NAC) strongly restores cell viability that has been inhibited by EPX. The results of these findings highlight the implication of ROS generation in the neurotoxicity induced by EPX, indicating that the production of ROS is the main cause of the induction of apoptosis probably via the mitochondrial pathway.
URL de la notice	http://okina.univ-angers.fr/publications/ua19644 [11]
DOI	10.1016/j.chemosphere.2019.05.018 [12]
Lien vers le document	https://www.sciencedirect.com/science/article/pii/S0045653519309142?via%... [13]
Autre titre	Chemosphere

Identifiant (ID) PubMed	31078888 [14]

Liens

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