Comparative Cytotoxicity and Mitochondrial Disruption in H9c2 Cardiomyocytes Induced by Common Pesticides

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Abstract

Chronic exposure to pesticides is believed to be associated with various human diseases, including cardiovascular diseases. However, the mechanisms by which pesticides lead to cardiovascular diseases remain unclear. In our study, we selected the following commonly used pesticides as typical examples: the herbicides glyphosate (GLY) and glufosinate ammonium (GLA); the insecticides imidacloprid (IMI) and thiamethoxam (THM); and the fungicides pyraclostrobin (PYR) and azoxystrobin (AZO). We employed H9c2 cells as a model to investigate the cytotoxic effects of these pesticides on myocardial cells at concentrations of 1, 10, 100, and 1000 mg/L. The results indicate that these pesticides can affect cell viability, alter the cell cycle, and significantly impact ATP content and mitochondrial complex levels, ultimately triggering oxidative stress responses in the cells. However, compared to herbicides GLY and GLA, insecticides IMI and THM, and fungicides PYR and AZO pesticides are more toxic to H9c2 cells. Additionally, GLY, GLA, IMI, THM, PYR, and AZO were found to cause structural changes in the mitochondria of H9c2 cells. Molecular docking results suggest that these pesticides can bind to proteins related to mitochondrial dynamics. Furthermore, IMI, THM, PYR, and AZO exhibit stronger binding abilities to mitochondrial dynamics-related proteins. These findings indicate that these pesticides significantly adverse effects on myocardial cells, mainly by causing mitochondrial dysfunction and inducing oxidative stress. Our findings highlight the importance of considering the differential toxicity of various classes of pesticides when assessing their risks to human health, particularly concerning cardiovascular diseases.

Keywords

Pesticides, H9c2, Cardiomyocytes, Cytotoxicity, Mitochondrial dynamics.