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Involvement of gap junctions in astrocyte impairment induced by manganese exposure

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Highlight

- The impairment of primary astrocytes by excessive manganese exposure was related to glutamate exitotoxicity.
- The permeability of gap junction communication among astrocytes was disrupted after high level manganese exposure.
- The forming protein of gap junction, connexin43 abnormal expression contributed to gap junction function disorder.

Abstract

Glutamate excitotoxicity, characterized as excessive glutamate stress, is considered to be involved in cerebral ischaemia, brain trauma, and neurodegenerative diseases such as Parkinson’s disease and