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Nrf2 signaling and cytoskeleton as targets of α -synuclein overexpression: new insights into pesticide-induced neurotoxicity.

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Overexpression of α -synuclein (α -syn) and pesticide exposure are considered triggering factors of Parkinson's disease. Previously we demonstrated that α -syn overexpression downregulates neurofilament light chain expression and alters actin organization in neuroblastoma cells. In this work, we studied the effect of α -syn overexpression in neuronal cytoskeleton organization and how this could affect the antioxidant response during pesticide-induced neurotoxicity. For this purpose, neurons stably expressing wild type α -syn gene (WT α -syn) were exposed to the pesticide Maneb (Mb). Overexpression of α -syn triggered actin polymerization and Tau phosphorylation. Cytoskeleton changes were associated with differential activation and subcellular localization of focal adhesion (FA) and LIM kinases, respectively. FAK and Tau phosphorylation and the expression of Nrf2-dependent antioxidant enzymes were increased by Mb exposure in WT α -syn cells. The upregulation of antioxidant defenses was associated with a neuroprotective effect against pesticide neurotoxicity. However, Nrf2 nuclear localization induced by Mb exposure was not altered by α -syn overexpression. Our results demonstrate that Nrf2 trafficking seems to be affected by cytoskeleton disturbances induced by WT α -syn overexpression. Further studies are necessary to decipher the crosstalk between cytoskeleton reorganization induced by α -syn overexpression and Nrf2-dependent signaling during Mb neurotoxicity.