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Necrostatin-1 counteracts neuronal cell loss induced by aluminum

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Investigators have linked aluminum accumulation in the brain as a possible contributing factor to neurodegenerative disorders such as Alzheimer's disease. The present study aimed at the mechanism underlying aluminum-induced neuronal cell death and identifies necrostatin-1, a specific inhibitor of necroptosis (programmed necrosis), as a substance which counteracts several of aluminum's neurotoxic effects. When aluminum was injected into the cerebral ventricles of living mice, brain tissue analysis revealed shrunken and abnormal-looking neurons. When Nec-1 was injected simultaneously with aluminum into the ventricles, more surviving neurons could be seen, especially when higher doses of Nec-1 were used. Cell death-related proteins in the brain, a marker protein of necroptosis known as RIP1 showed the most changes, compared to marker proteins of apoptosis or autophagy. Similar findings were found for Alzheimer-related proteins: aluminum exposure increased the expression of mGluR2, mGluR5, Aβ, and Tau levels while Nec-1 treatment resulted in dose-dependent reductions of these protein levels. Progressive cell loss in specific neuronal populations associated with typical learning and memory dysfunction is a pathological hallmark of neurodegenerative disorders, especially in AD. The present study demonstrates that Nec-1, in addition to its use as a therapeutic agent for cell death, might therefore be of use in slowing the progression of the cognitive deficits associated with neuronal degeneration.

Biography

Qinli Zhang has completed her Ph.D. and postdoctoral studies from Shanxi Medical University, China. She is now enrolled in Purdue University as a visitor scholar in Prof. Wei Zheng's lab. She has published more than 40 papers in reputed journals and has been serving as an editorial board member of repute.

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