## Endogenous negative regulators of inflammation preserve neuronal survival

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Cyanotoxins have been shown to be highly toxic for mammalian cells, including brain cells. However, little is known about their effect on inflammatory pathways. Our study investigated whether mammalian brain and immune cells can be a target of certain cyanotoxins, at doses approximating those in the guideline levels for drinking water. We examined the effects on cellular viability, apoptosis, and inflammation signaling of several toxins on murine macrophage-like RAW264.7, microglial BV-2, and neuroblastoma N2a cell lines. We have tested cylindrospermopsin (CYN), microcystin-LR (MC-LR), and anatoxin-a (ATXa), individually as well as in mixture. Searching into protective mechanism against cyanotoxins, we found that Nlrx1, a protein localized to mitochondria, ameliorates toxin effects. Decreased expression of Nlrx1 correlated with increased vulnerability of all cell types to toxin exposure. Our results demonstrate that CYN, MC-LR, and ATX-a, at low doses individually and in mixture, have potent effect inducing apoptosis and inflammation. Further research of the neuroinflammatory effects of these compounds *in-vivo* is needed to improve safety limit levels for cyanotoxins in drinking water and food.

### Speaker Biography

Denis Gris has completed his PhD in the Neuroscience program at the Western University of Ontario and moved to the University of North Carolina at Chapel Hill where he studied mechanism of immune responses. His laboratory studies a role of inflammation in the development and progression of neurodegenerative disorders and using *in-vitro* and *in-vivo* models, he aims to uncover novel endogenous pathways that limit neuroinflammation. At his lab, with his co-workers, they pair molecular work with latest state-of-the-art automated behavioral assessment technology, to study how inflammation changes CNS function.

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