Special Issue

Molecular Structure and Mechanisms of Neurotoxicity of Amyloid Prefibrillar Oligomers

Message from the Guest Editors

It has been proposed that a common core of pathologic pathways exists for amyloid-associated diseases based on the cellular membrane permeabilization and subsequent abnormal Ca2+-influx induced by aggregates of the involved proteins, independently from their primary sequence. An intriguing hypothesis has been formulated, that amyloid diseases were caused by aggregates that mimic bacterial pore-forming toxins, which, in general, form well-ordered oligomeric membrane-spanning pores. Up until now, emerging evidence has been focused on low molecular weight prefibrillar oligomers (PFOs) as the toxic species. On the other hand, many studies have indicated that the neuronal membrane composition and its chemical microenvironment play a pivotal role. It is now generally accepted that “lipid-rafts”, which are ordered nanodomains formed by sphingolipids, play a special role. However, the existence of a specific common toxic structure, and a common mechanism by which it induces neuronal damage and death, is still an open hypothesis. This Issue aims to gather as much information to test this hypothesis.

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