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In This Issue

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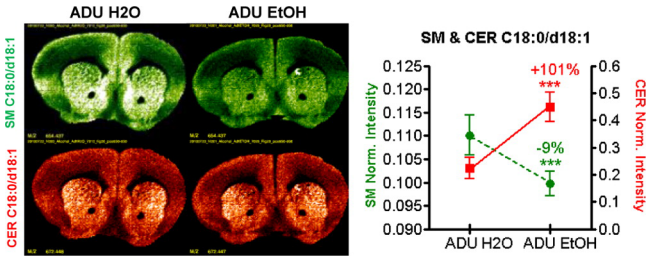
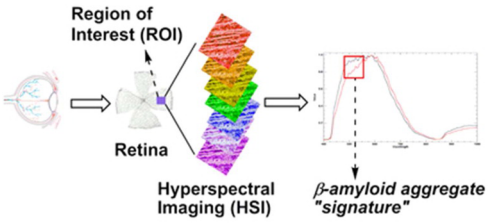
ALCOHOL CONSUMPTION AFFECTS BRAIN SPHINGOLIPIDS



Consumption of high doses of alcohol has been shown to cause several different neurological dysfunctions. However, the mechanisms underlying neurotoxicity is unclear. Roux et al. (DOI: 10.1021/cn500174c) investigate one potential mechanism, i.e., the involvement of sphingolipids in ethanol-induced neuroinflammation.

The authors describe a series of findings suggesting that long-term exposure to ethanol results in substantial changes in the cerebral content of two sphingolipid classes, ceramides and sphingomyelins. The authors use two complementary techniques, ESI and MILDI, which provide quantitative data on whole-brain concentrations of analytes of interest (ESI) or qualitative information on local analytes in coronal sections cut at various levels of the brain (MILDI). The two approaches reveal the existence of alcohol-dependent alterations in brain sphingolipid concentrations, which are strongly influenced by age.

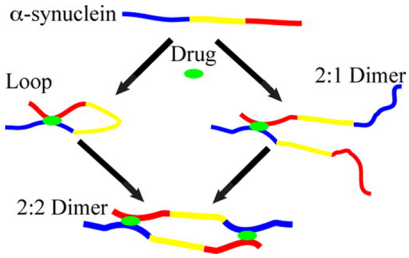
EYE TEST FOR EARLY DETECTION OF ALZHEIMER'S DISEASE



Therapy for Alzheimer's disease remains unsatisfactory, in part due to the lack of early diagnostic tools. Recent developments utilizing retinal imaging require an external dye or a fluorophore, thus introducing a regulatory factor while also rendering difficult the screening of therapeutic agents because of chemical or biomolecular interactions with the labeling entity. Now, More and Vince (DOI: 10.1021/cn500242z) provide the first-of-its-kind approach for monitoring Aβ aggregation in the asymptomatic stages of Alzheimer's disease.

The authors employed near-IR and visible electromagnetic transmission measurements acquired over a wide wavelength range (for high spectral resolution) and at pixel-level isolation (for high spatial resolution), known otherwise as hyperspectral imagery (HSI). This method enabled the acquisition of a "HSI signature" of soluble Aβ aggregates. In this way, Aβ pathogenesis could be detected in the APP/PS1 mouse as early as 4 months of age by visualization of excised mouse retina. To put things into perspective, symptomology begins at 6–7 months of age in this , and brain plaque deposition is observable at 6 months of age.

RASAGILINE BINDS α-SYNUCLEIN



Parkinson's disease is caused by the aggregation of the protein α-synuclein, which eventually leads to neuronal cell death. Rasagiline is a drug used to treat symptoms of Parkinson's disease. The mechanism of action for this drug is thought to be through the effect of an increase in dopamine levels. In the current issue, Kakish et al. (DOI: 10.1021/cn5002914) show that the mechanism of action of rasagiline may actually be through the binding to α-synuclein.

The study shows that rasagiline binds to α-synuclein by interacting with both the N- and C-termini of the protein. This direct binding to α-synuclein forms a structure that is much less likely to aggregate, thus serving as a neuroprotectant. Similar drugs might be useful in the long term treatment of Parkinson's disease.

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