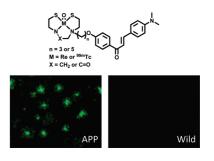
In This Issue

SPECTacular β-Amyloid Probes

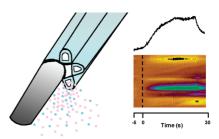
Detection of β -amyloid plaques in the brain is useful in the detection of Alzheimer's disease and as such, many positron emission tomography (PET) probes which target β -amyloid plaques in the brain have potential clinical utility. Unfortunately, due to the short half-lives of many of these labeled probes, usage is often limited to academic PET facilities with onsite cyclotrons and sophisticated radiochemistry laboratories. Single photon emission tomography (SPECT) probes offer an inexpensive alternative in clinical settings. Ono et al (DOI: 10.1021/cn100042d) set out to identify imaging probes labeled with SPECT isotopes useful in the detection of β -amyloid plaques. The authors synthesize novel chalcone derivatives conjugated with ^{99m}Tc or Re complexes, which provide simple SPECT imaging methods for detecting and eventually quantifying β -amyloid plaques.



Dopamine Release and Iontophoresis

Iontophoresis is an electrical technique that allows the introduction of small amounts of chemicals into a highly localized region of the brain. However, traditionally, iontophoresis was not widely used to deliver neuropharmaceuticals because of the difficulty in quantifying drug delivery using this approach. Previously, the mechanism of ejection of chemicals using a carbon-fiber

electrode attached to iontophoresis barrels was quantified. Here, Herr et al (DOI: 10.1021/cn100056r) utilize this technique to characterize pharmacological factors that presynaptically modulate dopamine extracellular levels in anaesthetized rats. Taken together, these experiments show that iontophoresis has potential in neuroscience research.



β-Amyloid Aggregation Inhibition

The design of peptides or peptidomimetics that bind and interfere with β -amyloid aggregation is a major focus of neuroscience research. Now, Bett et al. (DOI: 10.1021/ cn100045q) perform a systematic analysis of the aggregation-mitigating effect of various α , α -disubstituted amino acids in the hydrophobic core $(A\beta 17-20)$. The authors describe amyloid aggregation-mitigating peptides that are active against preformed fibrils and associated with gross changes in aggregate morphology. These results should be useful in the design of β -strand mimics that help mediate protein-protein interactions.

