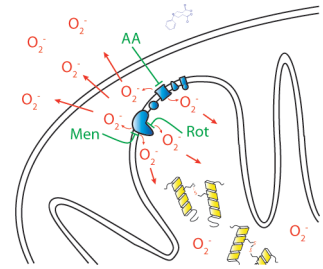
Action of a Neuroprotective Drug

Injury to the axons of neurons in the central nervous system results in cell death by a process known as apoptosis. Such injury is often associated with neurological disorders. Earlier, it was shown that this type of cell death resulting from axonal injury is mediated through superoxide anions generated inside mitochondria. It was also shown that cell-permeable borane−phosphine complex drugs that reduce oxidized sulfhydryls prevent apoptosis.

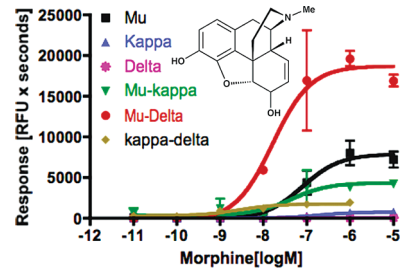
In the present study (DOI: 10.1021/cn900024r), Seidler et al. determine the site of injury within the mitochondria and find that the cell-permeable borane−phosphine complex drugs act outside of the mitochondrial matrix. These results have implications in drug design for neurological disorders involving reactive oxygen species generated in the mitochondria.



Targeting Opioid Receptors

Analgesics such as morphine mediate pain-relieving effects by targeting opioid receptors located on nerve cells in the central nervous system. There are three major types of receptors in the opioid receptor family, designated as mu (μ), delta (δ), and kappa (κ). Traditionally, analgesics were defined based on the assumption that these opioid receptors exist as monomers. However, it is now known that opioid receptors are capable of forming heterodimers in cultured cells. In addition, it is also likely that heteromers exist *in vivo*.

In a study in this issue (DOI: 10.1021/cn9000236), Yekkirala et al. examine the selectivity of standard opioid ligands with respect to both cloned homomeric and cloned heteromeric opioid receptors. They discover that these ligands usually exhibit greater functional activity at heteromeric opioid receptors when compared with their corresponding homomers. Importantly, the greater activity mediated by heteromers might have an impact on industrial screening processes using cultured cells.



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