学術講演会開催のご案内

このたび、下記により学術講演会を開催いたします。
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演題　**Antidepressants and neurogenesis**

講演者　**Prof. Nuno Sousa**
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場所　京都大学医学研究科 A 棟 4 階セミナー室（402 号室）
日時　平成 22 年 9 月 1 日(水)　午後 4 時半～午後 5 時半

New neurons are continually being generated in the adult brain, particularly in the olfactory bulb and in the dentate gyrus of the hippocampus. It is generally believed that the incorporation of newborn cells in the pre-existing neuronal network, in parallel with molecular, synaptic and morphological alterations of individual cells, represents an additional plastic process for the adult brain to adapt to novel conditions. Indeed, during the last two decades it has been shown that adult neurogenesis is regulated by several different factors; moreover, variations in the rate of adult hippocampal neurogenesis have been linked to behavioral changes. Three independent lines of evidence provided substantial support to the interaction between hippocampal neurogenesis and behavior. First, it was demonstrated that stress, a condition associated to major depression, downregulates hippocampal neurogenesis. Secondly, all major classes of antidepressants and electroconvulsive shock stimulate hippocampal neurogenesis. Finally, it was suggested that the behavioral effects of antidepressants require hippocampal neurogenesis.

The implications of the observation that antidepressant action is dependent on hippocampal neurogenesis are colossal, especially if applicable to human depression. Importantly, recent studies have shown that the suppression of neurogenesis is apparently not essential for the induction of depression in humans, as hippocampal precursor proliferation was not reduced in post-mortem tissue of depressed subjects. In addition, impaired hippocampal neurogenesis was also not observed in rats that develop learned helplessness. Furthermore, a careful scrutiny of the only study claiming that the behavioral actions of antidepressants require hippocampal neurogenesis reveals several points that demand a reappraisal of the evidence supporting this link. In fact, the most relevant of these technical issues relates with the fact that the novelty-suppression feeding protocol used to determine the behavioral effects of antidepressants is a test that assesses anxiety-, and not depressive-like, behavior in rodents.

To investigate a causal link between these observations, we analyzed the impact of blocking adult hippocampal neurogenesis in the emotional (depressive- and anxiety-like) behavior of animals submitted to chronic-mild stress that were treated with different antidepressants. We report that antidepressants revert depressive-like behavior in an animal model of depression (assessed by learned helplessness and anhedonic behaviors) regardless of their hippocampal neurogenic capability, providing evidence that the newborn cells do not mediate these effects of antidepressants. However, we showed that neuroplastic events are triggered by antidepressants and these are correlated with behavioral improvement.

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