Addendum

Addendum: Gomes et al., Excitotoxicity Downregulates TrkB.FL Signaling and Upregulates the Neuroprotective Truncated TrkB Receptors in Cultured Hippocampal and Striatal Neurons

While the article "Excitotoxicity Downregulates TrkB.FL Signaling and Upregulates the Neuroprotective Truncated TrkB Receptors in Cultured Hippocampal and Striatal Neurons" by João R. Gomes, João T. Costa, Carlos V. Melo, Federico Felizzi, Patricia Monteiro, Maria J. Pinto, Ana R. Inácio, Tadeusz Wieloch, Ramiro D. Almeida, Mário Grãos, and Carlos B. Duarte, which appeared on pages 4610–4622 of the March 28, 2012 issue, was under review, a related paper was published by Vidaurre et al. (2012). Both papers report the role of calpains in TrkB.FL downregulation in cultured neurons subjected to excitotoxic conditions, although different cultures and distinct stimulation protocols were used. Furthermore, both studies show that excitotoxic stimulation induces *de novo* synthesis of truncated TrkB receptors (TrkB.T). Similar results were obtained in both studies using the MCAO model of transient focal brain ischemia, and the work of Vidaurre et al. (2012) also showed a neuron-specific increase in TrkB.T1 protein levels after stroke in the human brain. The most important disagreement between the two papers concerns the neuroprotective role of TrkB.T receptors which was unraveled for the first time in our work through experiments where cultured hippocampal neurons were incubated with BDNF.

Reference

Vidaurre OG, Gascón S, Deogracias R, Sobrado M, Cuadrado E, Montaner J, Rodríguez-Peña Á, Díaz-Guerra M (2012) Imbalance of neurotrophin receptor isoforms TrkB-FL/TrkB-T1 induces neuronal death in excitotoxicity. Cell Death Dis 3:e256.

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