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# NGF, TrkA-P and neuroprotection after a hypoxic event in the developing central nervous system

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Highlights

- Programmed cell death involves the neurotrophin–neurotrophin receptor system
- An hypoxic event changes the expression level of ligand NGF and the p75 receptor
- An hypoxic event produces a bimodal profile in the activation of the TrkA receptor
- NGF possesses a neuroprotective effect in the developing central nervous system
- NGF neuroprotection requires the activation of receptor TrkA

**ABSTRACT**

A decrease in the concentration of oxygen in the blood and tissues (hypoxia) produces important, sometimes irreversible, damages in the central nervous system (CNS) both during development and also postnatally. The present work aims at analyzing the expression of nerve growth factor (NGF) and p75 and the activation of TrkA in response to an acute normobaric hypoxic event and to evaluate the possible protective role of exogenous NGF. The developing chick optic tectum (OT), a recognized model of corticogenesis, was used as experimental system by means of in vivo and in vitro studies.

Based on identification of the period of highest sensitivity of developmental programmed cell death (ED15) we show that hypoxia has a mild but reproducible effect that consist of a temporal increase of cell death 6 h after the end of a hypoxic treatment. Cell death was preceded by a significant early increase in the expression of Nerve Growth Factor (NGF) and its membrane receptor p75. In addition, we found a biphasic response of TrkA activation: a decrease during hypoxia followed by an increase -4 hours later- that temporally coincide with the interval of NGF