

## EXPERT REVIEW

## Neuronal plasticity and neurotrophic factors in drug responses

E Castrén and H Antila

Neurotrophic factors, particularly brain-derived neurotrophic factor (BDNF) and other members of the neurotrophin family, are central mediators of the activity-dependent plasticity through which environmental experiences, such as sensory information are translated into the structure and function of neuronal networks. Synthesis, release and action of BDNF is regulated by neuronal activity and BDNF in turn leads to trophic effects such as formation, stabilization and potentiation of synapses through its high-affinity TrkB receptors. Several clinically available drugs activate neurotrophin signaling and neuronal plasticity. In particular, antidepressant drugs rapidly activate TrkB signaling and gradually increase BDNF expression, and the behavioral effects of antidepressants are mediated by and dependent on BDNF signaling through TrkB at least in rodents. These findings indicate that antidepressants, widely used drugs, effectively act as TrkB activators. They further imply that neuronal plasticity is a central mechanism in the action of antidepressant drugs. Indeed, it was recently discovered that antidepressants reactivate a state of plasticity in the adult cerebral cortex that closely resembles the enhanced plasticity normally observed during postnatal critical periods. This state of induced plasticity, known as iPlasticity, allows environmental stimuli to beneficially reorganize networks abnormally wired during early life. iPlasticity has been observed in cortical as well as subcortical networks and is induced by several pharmacological and non-pharmacological treatments. iPlasticity is a new pharmacological principle where drug treatment and rehabilitation cooperate; the drug acts permissively to enhance plasticity and rehabilitation provides activity to guide the appropriate wiring of the plastic network. Optimization of iPlastic drug treatment with novel means of rehabilitation may help improve the efficacy of available drug treatments and expand the use of currently existing drugs into new indications.

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## INTRODUCTION

Neuronal plasticity is a process through which external and internal environment of an individual gradually becomes represented in neuronal structure and function during development and through learning. Although gross connectivity develops through genetically governed guidance, fine-tuning takes place through experience and activity-dependent plasticity, where neurons and connections that actively participate in network function are selected for stabilization and strengthened, whereas inactive contacts are weakened or eliminated.<sup>1–3</sup>

Neuronal plasticity does not only involve trophic processes such as neurogenesis and synaptogenesis but also includes atrophic processes, such as the elimination of inactive neurons and neuronal contacts. Although it is often thought that loss of neurons or synapses is harmful, elimination of connections that do not mediate useful information is, in fact, necessary for the optimal signal-to-noise ratio within the nervous system.<sup>1,2</sup> Indeed, most of the neurons and synapses formed during development are wiped out by adulthood.<sup>1,2</sup> Therefore, plasticity in itself does not have any particular direction; it is the experience-dependent activity within the neuronal network that determines which of the connections are strengthened and maintained and which ones are eliminated. Therefore, plasticity is adaptive when it is guided by beneficial environmental stimuli, but it can also be maladaptive, if the guiding experiences are adverse.

Neuronal plasticity is heightened during critical periods of postnatal development, which allows an efficient experience-driven fine-tuning of developing networks.<sup>4</sup> After the closure of

critical periods, neuronal plasticity and changes in network structure are more restricted. However, recent data indicate that several drugs used for the treatment of neuropsychiatric disorders can directly influence the plasticity and reactivate a critical period-like plasticity in the adult brain, a process known as induced plasticity (iPlasticity).<sup>5–8</sup>

To be translated into neuronal structure and function, neuronal activity needs molecular mediators<sup>9</sup> and neurotrophic factors are prime candidates for mediators between neuronal activity and plasticity.<sup>10,11</sup> In this review, we will first introduce the role of the neurotrophin family and especially of BDNF as a mediator of plasticity and drug effects. We will then discuss the role of neuronal plasticity in the mechanisms of action of drugs acting on the brain. Finally, we will review recent evidence that developmental-like plasticity, iPlasticity, can be activated in the adult brain and argue that iPlastic drugs should be combined with training, rehabilitation or psychotherapy to facilitate treatment outcome. For the role of other neurotrophic factors in neuronal plasticity, especially the family members of the glial cell line-derived neurotrophic factor, fibroblast growth factor and insulin-like growth factor, we refer to recent review articles.<sup>12–14</sup>

## NEUROTROPHINS IN PLASTICITY

The first neurotrophic factors, nerve growth factor (NGF) and brain-derived neurotrophic factor (BDNF) were discovered through their ability to support the survival of neurons and neurites during development.<sup>15–17</sup> Other members of the

# Neuronal Plasticity And Trophic Factors Symposia In Neuroscience Vol 7

**RJ Alexander**



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