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Effect of Peripheral Axotomy on Gene Expression of NIDD in Rat Neural Tissues

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Abstract Peripheral nerve lesion-induced production of neuronal nitric oxide synthase (nNOS) was implicated to influence a range of postaxotomy processes necessary for neuronal survival and nerve regeneration (Zochodne et al., Neuroscience, 91:1515–1527, 1999; Keilhoff et al., J. Chem. Neuroanat., 24:181–187, 2002, Nitric Oxide, 10:101–111, 2004). Protein–protein interactions represent an important mechanism in the control of NOS spatial distribution or activity (Alderton et al., Biochem. J., 357:593–615, 2001; Dedio et al., FASEB J., 15:79–89, 2001; Zimmermann et al., Proc. Natl. Acad. Sci., 99:17167–17172, 2002). As one of the nNOS-binding proteins, *n*NOS-*i*nteracting *D*HHC domain-containing protein with *d*endritic mRNA (NIDD) has recently been identified to increase nNOS enzyme activity by targeting nNOS to the synaptic plasma membrane in a postsynaptic density protein 95/discs-large/zona occlusens-1 domain dependent manner (Saitoh et al., J. Biol. Chem., 279:29461–29468, 2004). In this paper, we established a rat model with peripheral axotomy to investigate the gene expression patterns of NIDD in neural tissues using TaqMan quantitative real-time polymerase chain reaction and in situ hybridization combined with immunofluorescence. It revealed that NIDD mRNA was upregulated after sciatic nerve transection with the similar expressing styles as that of the nNOS in the injured nerves, corresponding dorsal root ganglia, and lumbar spinal cord. These findings imply that NIDD may be involved in the different pathological conditions including nerve regeneration, neuron loss or survival, and even pain process, possibly via regulating the enzyme nNOS activity.

Keywords nNOS-interacting DHHC domain-containing protein with dendritic mRNA (NIDD) · Rat · Sciatic nerve · Axotomy · Real-time PCR · In situ hybridization

Introduction

Nitric oxide (NO), a biological signaling molecule with a diversity of functions in physiology and pathology (Prast and Philippu 2001), is synthesized from one of the essential amino acids, L-arginine by NO synthase (NOS) isozymes, which were shown in both the central and peripheral nervous system (Lefebvre 1995; Paakkari and Lindsberg 1995). Evidence indicates that NO is a mediator of nerve injury (Keilhoff et al. 2002, 2004; Zhou and Wu 2006) or the physiological response to injury and may be responsible for abnormal neural activity and pain behavior in animals (Naik et al. 2006). Once NO is synthesized normally by neuronal NO synthase (nNOS) in neuronal tissues, it simply diffuses from the nerve terminal into adjacent cells and forms covalent linkages to its targets. Therefore, the physiologic and pathologic functions of NO are primarily

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