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Upregulation of Egr-1 biosynthesis in human neuroblastoma cells following activation of epidermal growth factor or muscarinic acetylcholine receptors

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The zinc finger transcription factor Egr-1 controls many biological functions in the nervous system, including neuronal plasticity, cell death and proliferation. Egr-1 connects cellular signaling cascades with changes of the gene expression pattern. Here, we show that epidermal growth factor (EGF) and carbachol stimulated the biosynthesis of Egr-1 in human neuroblastoma cells. The analysis of a lentiviral transmitted Egr-1-responsive reporter gene, embedded into the chromatin, revealed that Egr-1-dependent transcription was upregulated following stimulation. The signalling cascade initiated by carbachol involved the muscarinic acetylcholine receptors type I and III and the activation of diacylgycerol-dependent protein kinase C isoforms. Both signaling cascades, initiated by EGF or carbachol, required the activation of extracellular signal-regulated protein kinase ERK. In addition, we observed a striking enhancement of the transcriptional activation potential of the ternary complex factor Elk 1, a key transcriptional regulator of serum response element-driven gene transcription. Incubation of neuroblastoma cells with EGF or carbachol also triggered an upregulation of MAP kinase phosphatase-1 (MKP-1) that dephosphorylates and inactivates ERK in the nucleus. Lentiviral-mediated expression of MKP-1 completely blocked Egr-1 biosynthesis following EGF or carbachol stimulation, indicating that MKP-1 functions as a nuclear shut-off-device of EGF or carbachol-induced Egr-1 gene transcription in neuroblastoma cells.