The noble gas argon modifies extracellular signal-regulated kinase 1/2 signaling in neurons and glial cells.

Recently, the noble gas argon has been identified as a potent neuroprotective agent, but little is known about its cellular effects. In this in vitro study, we investigated argon’s influence on the extracellular signal-regulated kinase (ERK) 1/2, a ubiquitous enzyme with numerous functions in cell proliferation and survival. Primary neuronal and astroglial cell cultures and the microglial cell line BV-2 were exposed to 50 vol.% argon. Further possible effects were studied following stimulation of microglia with 50 ng/ml LPS. ERK 1/2 activation was assessed by phosphorylation state-specific western blotting, cytokine levels by real-time PCR and western blotting. Total phosphotyrosine phosphatase activity was examined with p-nitrophenylphosphate. After 30 min exposure, argon significantly activated ERK 1/2 signaling in microglia. Enhanced phosphorylation of ERK 1/2 was also found in astrocytes and neurons following argon exposure, but it lacked statistical significance. In microglia, argon did not substantially interfere with LPS-induced ERK1/2 activation and inflammatory cytokine induction. Addition of the MEK-Inhibitor U0126 abolished the induced ERK 1/2 phosphorylation. Cellular phosphatase activity and the inactivation of phosphorylated ERK 1/2 were not altered by argon. In conclusion, argon enhanced ERK 1/2 activity in microglia via the upstream
kinase MEK, probably through a direct mode of activation. ERK 1/2 signaling in astrocytes and neurons in vitro was also influenced, although not with statistical significance. Whether ERK 1/2 activation by argon affects cellular functions like differentiation and survival in the brain in vivo will have to be determined in future experiments.

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