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GCN5L1 Regulates Anaesthesia-Induced Nerve Injury in Newborn Through FoxO1 by Oxidation Effects

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ABSTRACT The researchers of the present study aimed to explore the effects and mechanism of general control of amino acid synthesis 5 like-1 (GCN5L1) in the anaesthesia-induced nerve injury model. In brief, the mRNA and protein of GCN5L1 expression was up-regulated in the anaesthesia-induced nerve injury model. GCN5L1 protein was increased in anaesthesia-induced nerve injury by inducing oxidative stress. Meanwhile, over-expression of GCN5L1 promoted oxidative stress in vitro. Down-regulation of GCN5L1 inhibited oxidative stress in nerve cells. GCN5L1 suppressed the protein expression of forkhead box O1 (FoxO1) in vivo or in vitro. The activation of FoxO1 attenuated the effects of GCN5L1 on oxidative stress in vitro. Finally, FoxO1 protein attenuated the effects of GCN5L protein on induced nerve injury via suppression of nerve apoptosis and oxidative stress in vivo. The researchers concluded that GCN5L1 regulated anaesthesia-induced nerve injury in newborns through FoxO1 via oxidation effects.