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MicroRNA-210 mediates the hippocampal neurogenesis following traumatic brain injury

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Motivation and Aim: Adult neurogenesis is a crucial process for brain tissue repair and remodeling after traumatic brain injury (TBI). MiR-210, a unique and pleiotropic microRNA, has been known to be upregulated in various tissues under hypoxic condition. This study is aimed to investigate the role of miR-210 in TBI-induced neurogenesis and the implicated mechanism.
Methods and Algorithms: TBI was induced by weight-drop device. Immunofluorescence staining of BrdU- and DCX-labeled neurons were used to qualify neurogenesis among different groups after TBI. The expression of miR-210 was evaluated by real-time PCR and the expression of ERK/MEK/Raf cascade was detected by Western blot.
Results: In this study, we found the level of MiR-210 significantly increased within 1 hour after TBI and reached a peak 4 hours after TBI. Administration of a shRNA against miR-210 not only reversed the TBI-associated upregulation of miR-210, but also attenuated the TBI-induced neurogenesis. The upregulation of miR-210 after TBI was found to be mediated by HIF-1α and regulated the phosphorylation of ERK/MEK/Raf cascade, a signal pathway also involved in adult neurogenesis. TBI triggered an approximate 3.0–3.5 fold stimulation in ERK/MEK/Raf phosphorylation, and the administration of miR-210 shRNA effectively dampened the TBI-induced activation of ERK/MEK/Raf cascade.
Conclusions: These results strongly suggest that the increase in miR-210, conferred by TBI, is mediated by HIF-1a expression and might have led to the stimulation of ERK/MEK/Raf cascade, which in turn promotes the TBI-induced neurogenesis.