Short-Term Intermittent Hypoxia Improves Stroke Outcome in Mice

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Obstructive sleep apnea (OSA) is a disorder characterized by airflow interruptions during sleep, and has been linked to increased incidence of stroke. Intermittent hypoxia (IH) in experimental animals can partially recapitulate the physiology of OSA. Previous studies have indicated that IH preconditions the brain to engage protective mechanisms against stroke. However, the temporal requirements of IH that produce preconditioning are not known. To explore the potential role of chronic IH in stroke outcome, we analyzed infarct volume and expression of proinflammatory cytokine gene expression ($TNF-\alpha$, IL-6, and $IL-1\beta$) after stroke in mice treated with IH or room air for 11 or 21 days.

Mice experiencing IH for 11 days prior to stroke and 1 day after reperfusion decreased gene expression of both IL-6 and IL- 1β . When assessed 3 days post-stroke infarct volume was decreased in mice that received IH pre-stroke and room air post-stroke compared to mice treated with air pre and post-stroke. These results were not observed in mice that experienced IH 21 days pre-stroke/3 days post-stroke. These results suggest a protective effect of subchronic IH prior to stroke in both infarct volume and inflammatory gene expression that was not present when the IH was present 21 days prior to stroke. Understanding how IH alters ischemic outcome could lead to clinical therapies for individuals at high risk for stroke.