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Investigating the Effects of Hypoxemia on Traumatic Brain Injury

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Traumatic brain injury, brain injury caused by an external force, remains a high cause of death and disability. In preclinical settings, hypoxemia, or episodes of low oxygen levels in the blood, immediately after traumatic brain injury (TBI) has been observed to exacerbate clinical symptoms. It is yet unknown whether hypoxemia several hours after the initial injury has an effect on injury in the white matter of the brain. We developed a clinically relevant mouse model of TBI and delayed hypoxemia. We placed mice in hypoxic conditions for 30 minutes, 24 hours after controlled cortical impact (CCI), a standard way of inducing reproducible TBI. Mice which were injured and exposed to hypoxemia had significantly elevated levels of axonal injury compared to injured mice alone. These results were consistent across two markers of axonal injury. Our model of delayed hypoxemia following TBI allows for quickly processing potential candidate therapeutics for preventing and protecting against axonal injury and cell death. We then investigated whether there were sex-dependent differences in responses to injury and hypoxemia. We explored differences between female and male mice, as well as between female mice in proestrus, one of the four stages of the reproductive cycle of mice, compared to female mice in the other three stages. There were no significant differences in measures of axonal injury, microglia activation, or astrocyte activation.