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A single head injury progressively alters corpus callosum function and limb coordination in mice

Axonal injury and demyelination following traumatic brain injury (TBI) may contribute to chronic and progressive neurodegeneration and behavioral deficits. The corpus callosum (CC) is unusually vulnerable to TBI. After a single closed-head injury (CHI), the chronic effect of CHI on CC function is assessed by ex vivo evoked compound action potential (CAP) responses measured at 3, 14, 90, and 180 days post injury (DPI). At 3DPI, ipsilesional N1 (myelinated axons) and N2 (unmyelinated) components of the CAP are significantly reduced compared to shams. Ipsilesional CAP responses recovered to sham levels by 14DPI. At 180DPI in the contralesional CC, CAP responses are significantly increased compared to shams. The behavioral effects of CC damage are assayed in CHI and sham mice by analyzing the running patterns on simple and complex wheels at 14 or 180 DPI. Simple wheel contains evenly spaced rungs. Complex wheel contains irregularly spaced rungs increasing limb coordination which involves CC. At 14 DPI, injured mice show speed deficits in simple but not complex wheel without altering limb coordination. At 180 DPI on simple wheel, injured mice improve running speed on simple wheel using a compensatory running strategy of increasing step frequency variability that produces sham-level limb coordination. On complex wheel, these mice are unable to use this compensatory running strategy resulting in impaired limb coordination. These data suggest that injured mice have declining limb coordination between 14 and 180DPI. These data also suggest that CAP responses recover at 14DPI followed by increased CAP amplitude in the contralesional CC at 180DPI. Thus, the altered CC function as seen in ex vivo recordings may underlie the impaired limb coordination at 180DPI. These data suggest a single impact produces both acute and chronic effects of CC function that coincide with delayed deficits in CC-dependent motor tasks.