

**TITLE:** Neonatal Pain Accelerates Meal Onset and Increase Body Mass in Adult Female Rats with Poor Spatial Memory

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The hippocampus is a brain area that is important for memory of personal events. Emerging evidence indicates that the hippocampus is also involved in energy regulation. For example, rats with hippocampal lesions eat larger meals and more frequently (i.e., accelerated meal onset) relative to rats with an intact hippocampus. We have recently shown that pain on the day of birth (AKA neonatal pain) impairs hippocampal (HC)-dependent memory in middle-aged rats. Given that impaired hippocampal function results in meal onset acceleration, we hypothesized that neonatal pain accelerates meal onset resulting in an increase in body mass. To test this hypothesis, female Sprague-Dawley rats were injured on the day of birth (Postnatal day [P] 0) with an injection of the inflammatory agent carrageenan (1%) to the right hind paw or handled in a similar manner. In adulthood (P 250), their food intake, body mass, and HC-dependent spatial learning and memory were tested. Rats were then separated into those with better or poorer spatial memory using a median split based on the average proximity to the platform over 30 seconds. We found that neonatally injured rats with poorer memory had significantly accelerated meal onset and increased body mass relative to controls with poorer spatial memory. These differences were not observed in rats with better spatial memory. The results suggest that neonatal injury may accelerate meal onset and increase body mass in the presence of hippocampal-dependent memory deficits. Future studies should determine whether these effects are sex-specific.

**KEYWORDS:** Neonatal Stress, Pain, Inflammation, Hippocampus, Meal Onset, Body Mass, and Female