The Impact of Systemic Inflammation on Post-Hypoxic Recovery



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Background: Hypoxia

- What do Sudden Infant Death Syndrome (SIDS), sleep apnea, and traumatic brain injury (TBI) have in common?
 - Hypoxic insult to the central nervous system (CNS) followed by reoxygenation
- <u>Reoxygenation</u> = Post-hypoxic recovery
 - Period after the hypoxic episode (10% O2) has ended and *normoxic* levels of gases are restored (21% O2)







- CNS inflammation has been linked to sleep apneas and SIDS (Lorea-Hernández et al., 2016).
- Little is known on how inflammation affects the *post-hypoxic reoxygenation period*



Methodology

- CD1 mice at p6-p8
- Intraperitoneal injection of LPS (3 hr)
 - 15 µl lipopolysaccharide (LPS) (2 µg/µl) + 15 µl saline
 - 30 μ l saline
- Baseline whole-body plethysmography
 - Measured <u>tidal volume</u> and <u>respiratory rate</u>
- Intermittent Hypoxia (10% O₂, 5 min x 3)
 - Reoxygenation 10 min
- Whole-body pleth





Intermittent Hypoxia Protocol





NX = Normoxia (Reoxygenation, 21% O₂)

HX = Hypoxia (10% O₂)



Hypothesis

- Systemic inflammation induced by LPS will *reduce* an animal's ability to recover after intermittent hypoxia
 - The average <u>respiratory rate</u> of animals during post-hypoxic recovery will *decrease* after LPS injection in comparison to no LPS
 - The average <u>tidal volume</u> during recovery from hypoxia will decrease after LPS injection in comparison to no LPS



Reoxygenation Before and After LPS (Animal 1)

Reoxygenation





Reoxygenation Before and After LPS (Animals 2 & 3)



Conclusions

- Our preliminary data supports the hypothesis that LPS-induced systemic inflammation reduces respiratory rate and tidal volume during post-hypoxic reoxygenation in comparison to animals that lack LPS.
 - This suggests that mild inflammation has a negative impact on the respiratory system of neonatal mice.
- Inflammation in neurological diseases and disorders which are tied to hypoxic episodes could impact respiratory recovery.

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