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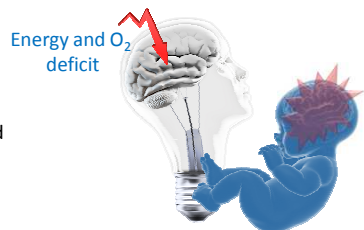
Lactate as a neuroprotective treatment after a neonatal hypoxic-ischemic event

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Context:

Neonatal hypoxia-ischemia:

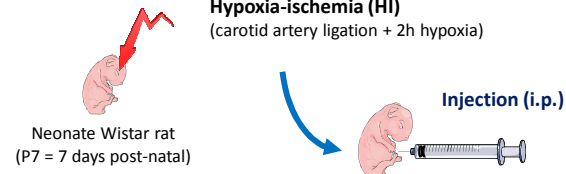
- 1-6 % cases per live births
- 15-20% of death during perinatal period
- 25% of motor and cognitive deficits



- ↳ brain oxygen
- ↳ energetic substrate supply
- ⇒ Energy deficit
- ⇒ Neuronal death
- **Therapeutic care: hypothermia**
- BUT : 50% of infants don't respond to it**

Methods:

Hypoxia-ischemia (HI)
(carotid artery ligation + 2h hypoxia)

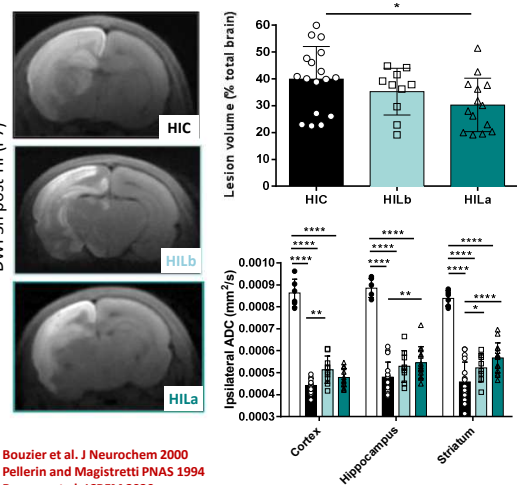


HIC: NaCl
HIG: glucose
HIP: pyruvate
HILb: lactate before HI
HILa: lactate after HI
HI3L: 3x lactate after HI
HILO: lactate + oxamate

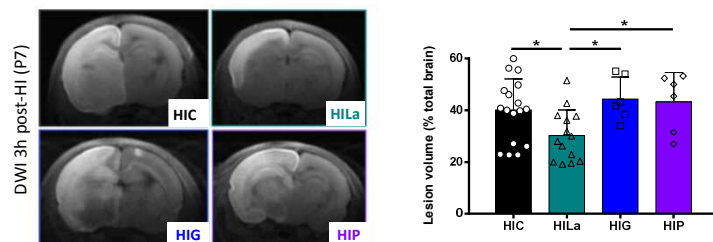
Experiments:

- DWI (4.7T, Bruker) for lesion volume + Apparent Diffusion Coefficient
- Behavioral tests (motor and memory)

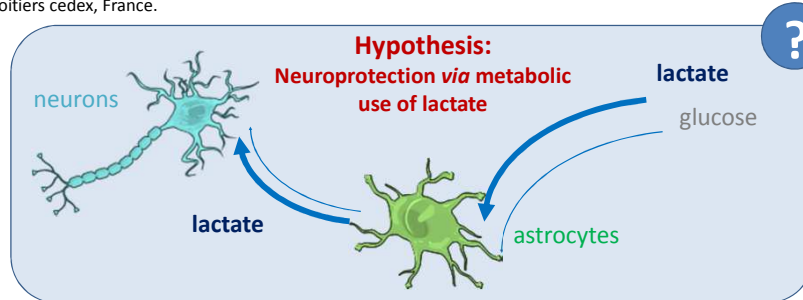
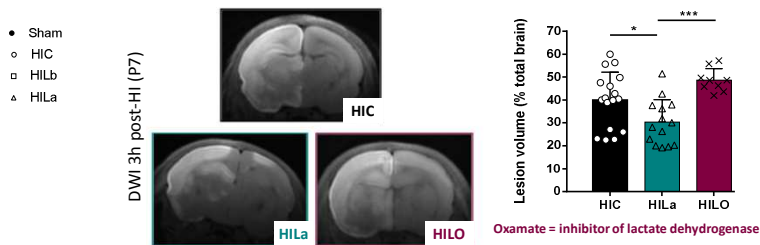
Result 1: Post-HI lactate administration is neuroprotective and ↓ edema



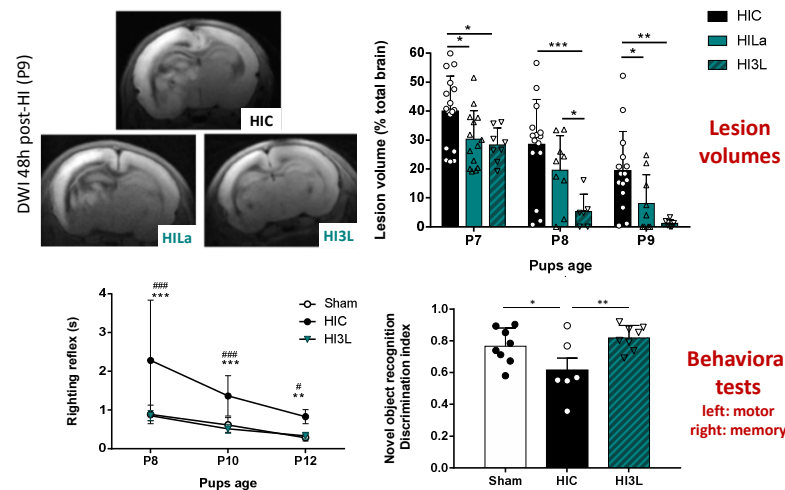
Result 2: Only lactate decreased brain damages



Result 3: Lactate neuroprotection implies its metabolic use



Result 4: Best neuroprotection obtained with 3 lactate injections (1/day)



Conclusion:

Lactate, a neuronal substrate¹, was neuroprotective against HI brain damages, via the astrocyte-neuron lactate shuttle (ANLS)², sparing glucose for its use in the pentose phosphate pathway (which helps fighting against ROS) and restored brain functions³.

