



BACKGROUND

- Chronic pain is estimated to affect 1.5 billion people worldwide.
- CD44 functions as a receptor, and has been implicated in inflammation associated with neuronal injuries.
- VCAM-1 helps regulate inflammation-associated vascular adhesion.
- An increase of proinflammatory factors and a reduction of neurotrophic factors have been reported to modulate the hippocampal neurogenesis and neuroplasticity in chronic pain.
- The spared nerve injury (SNI) model induces symptoms of neuropathic pain.

METHODS

- On day 0, the SNI surgery is performed; half of the mice get the surgery, the other half get the sham surgery.
 On day 1, the mice get injected intraperitoneally with 0.1
- mL of buprenorphine.
- On day 1, 7, 28, the mice get behavior testing. They go through the Y maze, open field, zero maze and von Frey analysis.
- Five of the sham mice and five of the injured mice are perfused with PBS solution, while the other ten are perfused with the microphil solution.
- The mice are then sacrificed and the sectioning of their brains takes place later on.
- Using IHC, the brain sections are stained and then using the Keyence are imaged.
- The images will be further processed and then quantified by cell count and intensity using ImageJ.

CONCLUSION

This study will allow for a better understanding of how chronic pain affects the microvasculature of the brain.

Alterations of Brain Microvasculature due to Chronic Pain

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RESULTS



The above images are brain sections stained blue with DAPI, red with VCAM, green with CD44 and the overlap.

REFERENCES

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- Institute of Health (NIH).





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