Aging and Nerve Injury – Neuropathic Pain – Evoked Behavior

**M e c h a n i c a l H y p e r s e n s i v i t y**

- **A g e d  -  S N I**
- **A d u l t -  S N I**
- **A g e d - S h a m**
- **A d u l t - S h a m**

**Cold (Acetone) al l o d y n i a**

- **A c e t o n e  r e s p o n s e  ( s / 6 0 s )**

**Aging and Neuroinflammation – Gene Expression**

- **Spinal Cord Cytokine Expression - 5dp**
- **Spinal Cord Cytokine Expression - 56dp**

**Aging and Dynamic Allodynia**

**D y n a m i c  m e c h a n i c a l  a l l o d y n i a**

**RESULTS**

Surprisingly, aged animals took significantly longer to develop signs of neuropathic pain than young adult rats as measured by both mechanical hypersensitivity and cold alldynia (21 days aged vs. 5 days young). When we assessed DRG and spinal cord tissue ipsilateral to the injury for inflammatory and ER stress genes we observed an age-and surgery-related upregulation of ATF4, IL-6, and SK2 5-days post surgery. However, there was no difference in behavior or gene expression 60-days post surgery.

**CONCLUSIONS & FUTURE**

- Surprisingly, aged animals take longer to develop neuropathic pain post nerve injury.
- This delay could be related to the upregulation of SK2 channel expression.
- Collectively our work demonstrates an age-related mechanism to the development of a chronic pain state.

**REFERENCES**

- Weyer AD, Zappia KJ, Garrison SR, O'Hara CL, Dodge AK, Stucky CL. Nociceptor Sensitization Depends on Age and Pain Chronicity(1,2,3). eNeuro. 2016 Feb
- Godbout JP, Chen J, Abraham J, Richwina AF, Berg BM, Kelley KW, Johnson RW. Exaggerated neuroinflammation and sickness behavior in aged mice following activation of the peripheral innate immune system. FASEB J. 2005

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