UPREGULATION OF LSD1 IN THE TRIGEMINAL SYSTEM CONTRIBUTES TO NEUROPATHIC OCULAR PAIN IN A RAT MODEL OF LACRIMAL GLAND EXCISION

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Abstract - Dry eye disease is a condition associated with ocular surface inflammation and sensory nerve abnormalities, often accompanied by significant pain and reduced quality of life. Lysine-specific demethylase 1 (LSD1) regulates several neural processes, including neuronal development, synaptic plasticity, and cell survival. Previous studies have shown that LSD1 expression is upregulated in the dorsal root ganglion (DRG) in neuropathic pain models such as Chronic Compression of the DRG (CCD) and Spinal Nerve Ligation (SNL), contributing to nociceptive sensitization. In this study, a rat model of dry eye was established by lacrimal gland excision (LGE) to mimic tear film instability and neuropathic ocular pain, and behavioral assessments and protein analyses were performed to examine changes within the trigeminal system. Our results showed that LSD1 expression was markedly increased in the trigeminal system, including the trigeminal ganglion (TG) and trigeminal brainstem nuclear complex (TBNC), in the LGE dry eye model, and this upregulation was associated with enhanced pain behaviors. Furthermore, administration of an LSD1 inhibitor reduced LSD1 expression and significantly attenuated pain responses in rats. Overall, these findings suggest that LSD1 plays a critical role in dry eye–related neuropathic pain and may serve as a potential therapeutic target for Neuropathicocular pain.

Keywords - LSD1, Dry Eye Disease, Ocular Pain