

Increased electrical activity in eye may relieve short-term dry eye pain

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A boost of electrical activity in the eye's mucous membranes may lead to new treatments for the painful condition known as dry eye. The study, published ahead of print in the *American Journal of Physiology—Cell Physiology*, was chosen as an APSselect article for June.

Dry eye is a common condition often caused by a disruption in the eye's fluid balance. A long-term imbalance of too much salt and not enough water (hyperosmolarity) in the thick layer of fluid in the eye (tear film) can lead to inflammation and cell damage that may become irreversible.

Extremely fast electrical signals carry messages throughout the body, telling it how to function. This process plays a role in how the body responds to various stimuli. In this case, little is known about how the eye's surface adapts to the fluid imbalance seen in dry eye. Donald G. Puro, MD, Ph.D., from the University of Michigan, studied the bioelectrical responses of [cells](#) in the [mucous membranes](#) that line the eyelids ([goblet cells](#)). Goblet cells release a protein called mucin—the basis of mucous—which slows down the evaporation of tears and helps maintain the tear film's balance. In a rat model of dry eye, Puro found that [electrical activity](#) in the goblet cells increases as hyperosmolarity rises in the tear film, which in turn allows the cells to produce more mucin. However, this voltage boost is short-lived. If the salt-to-water ratio of the tear film remains unbalanced in the long-term, the goblet cells' electrical activity returns to normal levels without producing additional mucin.

"Continued progress in elucidating the bioelectric mechanisms by which the ocular surface responds to dryness [and] hyperosmolarity should provide novel strategies for [improving] the uncomfortable sight-impairing condition of dry eye," Puro wrote.

More information: Donald G Puro, Role of ion channels in the functional response of conjunctival goblet cells to dry eye, *American Journal of Physiology-Cell Physiology* (2018). [DOI: 10.1152/ajpcell.00077.2018](#)

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