Mechanical stress increases vocal fold transepithelial potential difference

Christopher Atkins, MA
Kimberly V. Fisher, Ph.D.

Northwestern University Department of Communication Sciences and Disorders
Evanston, IL. USA

Vocal fold epithelium functions as a physical barrier that defends against numerous biological threats including mechanical stress inherent in voice production, airway protection, and swallowing. It is well established that eukaryotic cells and tissues are mechanosensitive and this sensitivity can be biologically communicated through numerous chemical and molecular responses to external mechanical stressors. We hypothesized that a mechanosensitivity of vocal fold epithelium could be detected by changes in the tissue's bioelectric properties in response to stretch. We further questioned whether vocal fold epithelial sodium channels (ENaC) contribute to any observed mechanosensitivity of this tissue. Fresh ovine vocal fold mucosa (N=20) were mounted in an Ussing Chamber System. The transepithelial potential difference (PD) and short-circuit current (I_{sc}) were acquired from both a control group (n=10), and an experimental group (n=10), before, during, and after stretch. For both groups, imposition of a hydraulic pressure gradient (5 cm H_2O applied to the basal surface) stretched the tissues. In the experimental group,10μM of amiloride, an inhibitor of epithelial sodium channels, was added prior to stretch to determine the role of Na^+ transport in supporting mechanosensitivity. Tissues were maintained in a viable state for the duration of the experiment. Stretch increased the PD ($p < 0.001$) for both groups with no concomitant change in I_{sc} ($p = 0.833$) which supported an increase in the electrical resistance for all tissues. This response to stretch was not abolished by ENaC inhibition ($p = 0.541$). Our results are consistent with the hypothesis that vocal fold epithelial mechanosensitivity can be physiologically expressed through changes in the tissue's electrical properties while maintaining its viability in response to stretch induced by a low hydraulic pressure. We speculate that sensitivity to low hydraulic pressure is not directly ENaC dependent but rather may be biologically transduced through cytoarchitectural changes.

Key words: Vocal fold biology, epithelial defense

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