Respiratory Research

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Development of a lung slice preparation for recording ion channel activity in alveolar epithelial type I cells

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Published: 27 April 2005

Respiratory Research 2005, **6**:40 doi:10.1186/1465-9921-6-40

Received: 21 December 2004
Accepted: 27 April 2005

This article is available from: http://respiratory-research.com/content/6/1/40

Abstract

Background: Lung fluid balance in the healthy lung is dependent upon finely regulated vectorial transport of ions across the alveolar epithelium. Classically, the cellular locus of the major ion transport processes has been widely accepted to be the alveolar type II cell. Although evidence is now emerging to suggest that the alveolar type I cell might significantly contribute to the overall ion and fluid homeostasis of the lung, direct assessment of functional ion channels in type I cells has remained elusive.

Methods: Here we describe a development of a lung slice preparation that has allowed positive identification of alveolar type I cells within an intact and viable alveolar epithelium using living cell immunohistochemistry.

Results: This technique has allowed, for the first time, single ion channels of identified alveolar type I cells to be recorded using the cell-attached configuration of the patch-clamp technique.

Conclusion: This exciting new development should facilitate the ascription of function to alveolar type I cells and allow us to integrate this cell type into the general model of alveolar ion and fluid balance in health and disease.

Background

Of fundamental importance to the optimisation of gas exchange in health and disease is the role of the alveolar epithelium in regulation of the liquid sub-phase in the postnatal lung. There is now unequivocal evidence to support both constitutive and stimulated Na^+-driven, active vectorial transport of water from lung lumen to interstitium in the neonatal and adult lung (see [1-5] for reviews). During the final days of fetal development, steroid hormone-induced transcriptional upregulation of the amiloride-sensitive epithelial Na^+ channel (ENaC) ensures that, during labour, the huge surge in fetal adrenaline leads to channel opening and massive Na^+ flux out of the fetal lumen [6-9]; this drives osmotically-linked fluid reabsorption in preparation for the neonatal lung to take on the gas exchange role previously undertaken by the placenta. Maturation of the response appears to be under control of a number of environmental factors, most importantly oxygen [10-12]. Key to understanding this mechanism was the observation that transgenic mice