



Why inhaling salt water changes what we exhale

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Abstract

We find that inhaling salt water diminishes subsequently exhaled biomaterial in man and animals due to reversible stabilization of the airway lining fluid (ALF)/air interface as a novel potential means for control of the spread of airborne infectious disease. The mechanism of this phenomenon relates to charge shielding of mucin or mucin-like macromolecules that consequently undergo gelation; this gelation alters the physical properties of the ALF surface and reduces its breakup. Cations in the nebulized solution and apparent surface viscoelasticity of the ALF (more than any other ALF intrinsic physical property) appear to be responsible for the reduced tendency of the ALF to disintegrate into very small droplets. We confirm these effects in vivo and show their reversibility through nebulization of saline solutions to anesthetized bull calves.

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1. Introduction

Studies have shown that humans and animals tend to exhale small bioaerosol droplets formed of airway lining fluid, or ALF, owing to the passage of air over the ALF during normal breathing [1–3]. Recent work suggests that humans breath out highly varying numbers of such expired bioaerosol particles, with certain individuals breathing out far more particles than others [4].

Furthermore, delivery of sufficient quantities of aerosolized isotonic saline (aqueous 0.14 M NaCl) to human lungs can markedly diminish the number of expired bioaerosol particles over time, notably among those individuals who breath out the largest numbers of particles [4]. In vitro experiments using simulated respiratory maneuvers with a mucus mimetic indicate that delivered saline diminishes the mobility of droplets formed on passage of air over the ALF, primarily by promoting a larger

aerosol droplet size [4]. Many mammals including humans commonly inhale salt water through such natural phenomena as sea mist. In addition, therapeutic delivery to patients of nebulized drugs dissolved in saline is accompanied by no known adverse side effects. Indeed, the idea of inhaling saline for medical benefit has existed since the time of Hippocrates [5]. The mechanism of this phenomenon remains unclear, as does the reason for the dramatic exhaled aerosol differences among human individuals.

In the present study, we performed in vitro and in vivo experiments to test a hypothesis that the immobilization of bioaerosol via inhaled saline (and, possibly, the diminished exhaled aerosol numbers in certain individuals relative to others) stems from an increased airway lining fluid/air interface rigidity caused by a relative abundance of free ions near the ALF/air surface. We report results from in vitro experiments using airway fluid models that mimic ALF in terms of rheology and charge heterogeneity, as well as in vivo experiments using Holstein bull calves. The in vitro studies reveal that delivery of saline to ALF promotes a reversible physical change in

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