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Utilizing a 3D Human Corneal Epithelial Tissue Model to Study Oxidative Stress in Corneal Injuries of Various Origin

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ABSTRACT

The current study's goal was to promote ocular treatments by using a three-dimensional (3D) corneal epithelial tissue model to examine dry eye disease and oxidative stress-related corneal epithelial damage. Normal human corneal epithelial cells were grown in air-liquid interface cultures to create 3D corneal epithelial tissues suitable for physiologically relevant exposure to environmental variables. By subjecting the tissues to non-toxic amounts of ultraviolet (UV), hydrogen peroxide, the vesicant nitrogen mustard, or desiccating circumstances, oxidative stress was created. These stimuli induced morphological, cellular, and molecular alterations related to dry eye illness. Barrier function, tissue viability, reactive oxygen species (ROS) accumulation, lipid peroxidation, cytokine release, histology, and gene expression were all assessed as part of the evaluation of the specific responses in the cornea. Key elements of molecular responses to diverse types of oxidative stress-induced ocular injury were physically and functionally replicated in a 3D corneal epithelial tissue model. The effects of UV irradiation, hydrogen peroxide exposure, nitrogen mustard exposure, and desiccating circumstances on intracellular ROS buildup, barrier disruption, lipid peroxidation, and IL-8 release were the most notable ones for each treatment.

Keywords: Corneal epithelial tissue

INTRODUCTION

The cornea, an exposed mucosal surface of the eye, protects against numerous types of infection, trauma, and injury to the cornea. The stratified, non-keratinized squamous epithelial corneal epithelium, which has 5 to 7 cell layers, has a direct role in shielding the cornea from UV radiation, dangerous chemicals, and damage. Antioxidant enzymes that remove free radicals and reactive oxygen species are abundant in the healthy corneal epithelial tissue (ROS). The mammalian cornea's main soluble protein, corneal aldehyde dehydrogenase (ALDH), plays a significant role in the detoxification of harmful aldehydes produced in cells as a result of membrane lipid peroxidation. The eye is susceptible to the harmful effects of oxidation because it is constantly exposed to a variety of oxidative circumstances, such as ambient oxygen, photo-oxidation, ionising radiation, cigarette smoke, and many types of contaminants. Oxidative stress (OS) can alter the activity of enzymes, prevent the synthesis of nucleic acids and proteins, upregulate the expression of proinflammatory genes, and activate the inflammatory response, which frequently exacerbates OS and sets off a self-reinforcing, vicious cycle of oxidation and inflammation. OS has been linked to a wide range of ocular conditions, including glaucoma, bullous keratopathy, keratoconus, age-related macular degeneration, cataracts, diabetic retinopathy, dry eye disease (DED), and fuchs' endothelial dystrophy. Additionally, UV irradiation-related ocular damage, chemical burns, and exposure to vesicating agents all include OS.

In this study, we investigated the corneal epithelial response to a variety of oxidising circumstances, including DED caused by desiccating conditions, exposure to UV irradiation, and chemical agents. We did this by using an in vitro rebuilt 3D human corneal epithelial (3D-HCE) tissue model.

CONCLUSION

In conclusion, we have effectively created an in vitro reconstructed 3D-HCE tissue model that, when subjected to various oxidative stress conditions, is comparable to the human corneal epithelium *in vivo*. This is a significant breakthrough because it is challenging to analyse changes to the ocular surface in vivo. The 3D corneal epithelial tissue model can be used to provide physiologically relevant amounts of a substance, simulate in vivo exposures, and conduct analysis right away after administration. Access to both the apical and basolateral sides, as well as the potential for high throughput screening of drug candidates, are additional benefits of the in vitro 3D-HCE tissue model.