Contact Lens Update
CLINICAL INSIGHTS BASED IN CURRENT RESEARCH

Effect of Contact Lens Materials and Designs on the Anatomy and Physiology of the Eye

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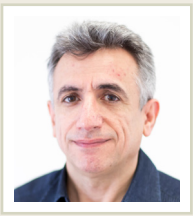

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Morgan PB, Murphy PJ, Gifford KL, et al. CLEAR – Effect of materials and designs on the anatomy and physiology of the eye. Cont Lens Anterior Eye 2021;44(2):192-219.

This CLEAR paper reviewed the effect of contact lens materials and designs on the anatomy and physiology of the eye, covered ocular changes caused by contact lens wear and the clinical significance of these changes.[1] This review was conducted in a systematic manner that followed the possible order of examining the ocular structures during a routine contact lens examination. The report also covers the differences among different ocular structures and their sensitivity and adaptation to the physical presence of the lens as well as their sub-clinical inflammatory response to lens wear. These elements were examined to provide some insights into features of the ocular surface sensory and immunological responses to lens wear along with their possible relationship to contact lens discomfort and/or adverse events.

Eyelids and adnexa

There is an increased risk of ptosis especially with long-term wear of rigid corneal lenses, possibly due to excessive eyelid manipulation during their insertion and removal. Blinking is an important ocular surface mechanism for maintaining ocular comfort through continuous ocular surface and lens lubrication, as well as providing a smooth optical surface for good vision. The blink rate increases in both dry eye disease (DED) and contact lens wear. Incomplete blinking can increase the risk of DED, meibomian gland atrophy and poor tear film stability and lead to exposure of the inferior ocular surface, which may affect lens comfort in digital device users. Meibum in contact lens wearers appears to have a higher melting point than in non-wearers. Possible changes to meibomian gland function and/or structure from lens wear can affect the meibum secretion or its lipid composition resulting in compromised tear film integrity and hence increased tear evaporation, which can affect wearing comfort and overall contact lens success.

Bulbar and limbal conjunctiva

Bulbar and limbal conjunctival hyperemia occurs as a result of increased dilation of the limbal arcade or bulbar conjunctival arterioles from mechanical irritation, hypoxia, hypercapnia, acidic shift of the conjunctival environment, increased osmolarity, toxic reactions to preservatives, or as part of the inflammatory response to allergens or infections. Many of these factors can be present in contact lens wearers and can be acute or chronic in nature. Conjunctival staining induced by the edge of a contact lens is rarely symptomatic or accompanied by hyperemia. However, comfort can be reduced in the presence of conjunctival indention and can

be improved by changing to a lens with a thinner edge design and/or a lower modulus. Lid-parallel conjunctival folds (LIPCOF) may be caused by increased friction between the ocular surface and the lid, which results from reduced lubrication due to a deficient tear film. It has been shown that LIPCOF is highly correlated with DED and its associated symptoms. a similar etiology to DED (i.e. increased friction between the moving eyelid and the ocular surface) has been suggested for LIPCOF in contact lens wear, however, a clear relationship between the coefficient of friction of contact lenses and LIPCOF has not yet been proven.

Palpebral conjunctiva

The palpebral conjunctiva forms the back surface of both eyelids. Changes to papillary conjunctival redness and roughness are a complication of all types of contact lens wear and are caused by the interaction between the front surface of the lens and the back surface of the eyelid. The interactions can be mechanical or allergic/inflammatory in nature. The increased redness and roughness of the palpebral conjunctiva can be alleviated by switching to daily disposables or lower modulus lenses in symptomatic patients. Increased friction between the lid wiper and ocular surface is thought to be the mechanism that results in Lid Wiper Epitheliopathy (LWE). However, a link between contact lens coefficient of friction and LWE has not been established yet. Furthermore, the relationship between LWE and contact lens discomfort remains unclear.

Cornea and contact lens-induced hypoxia

Corneal hypoxia can affect all corneal layers. Clinical signs of corneal epithelial hypoxia include epithelial microcysts, compromise in junctional integrity, neovascularization and decreased corneal sensation. Corneal epithelial swelling rarely occurs with lens wear, but it can occur from exposure to a hypotonic environment or from significant trauma. Epithelial microcysts are a delayed response to high levels of chronic hypoxia and they are mainly associated with overnight wear or contact lenses of low oxygen transmissibility. Contact lens-induced corneal neovascularization may be triggered by chronic corneal hypoxia. It is now rarely seen in daily wear of modern high Dk/t lenses. However, one should remain cautious with the risk of corneal neovascularization in high-swellers (individuals with a higher swelling response to hypoxic conditions), particularly when using higher minus power, thicker lens edge profiles and especially in overnight wear. With the advent of high Dk/t lens materials, the effects of hypoxia on corneal physiology in an average cornea have been minimized. However, the same word of caution relevant to neovascular changes is also true for preventing chronic hypoxia-induced stromal swelling, or for preventing unwanted corneal endothelial cell loss from chronic hypoxic stress in high-swellers. Progressive thinning of the cornea is a long-term side effect of contact lens wear. Stromal thinning can affect the suitability for future refractive surgeries; current knowledge on its etiology is inconclusive and points to mechanical and/or hypoxic mechanisms.

Short-term hypoxia-related responses include stromal swelling, striae, folds and haze. Corneal swelling mainly occurs in the posterior stroma. Stromal haze from increased amounts of back scattered light from the anterior cornea is more likely to occur with higher levels (i.e. >15%) of swelling after closed-eye wear of a low Dk/t lens and/or in high-swellers. However, haze is more commonly seen in corneal pathological conditions (e.g. Fuchs' dystrophy). Stromal striae and folds are two important short-term clinical indices of corneal swelling that are more commonly seen with low Dk/t hydrogel lenses and especially after closed-eye lens wear. Striae and folds are reported at swelling levels as low as 4 and 7%, respectively. Transientendothelial blebs are associated with localized areas of endothelial swelling from acidosis caused by reduced oxygen supply and increased carbon dioxide in the cornea. Blebs have no known clinical significance and they historically were seen with low Dk/t lenses. The number of blebs peaks after 30 minutes of lens wear and then drops over the following hours.

Ocular surface sub-clinical inflammatory responses

Dendritic cells are one of the subclinical biomarkers of ocular inflammatory response to physical presence of a

lens. Elevated levels of corneal epithelial dendritic cells (CEDC) have been found in the corneal centre of soft lens wearers. Daily replacement of lenses reduces the CEDC response. A similar increase in dendritic cell density of the lid margin, palpebral conjunctiva and central cornea was reported after one week of wearing a reusable silicone hydrogel (SiHy) lens but not in daily disposable wear. The effect of lens material and oxygen transmissibility on CEDC is unclear. Also, the effect of lens wear, lens material and edge design on the density of limbal and bulbar conjunctival dendritic cells is not clear. Further research is required to investigate the relationship between sub-clinical inflammatory response of the ocular surface to contact lens wear with adverse events and lens discomfort.

Ocular surface sensitivity and neural adaptation

Corneal sensitivity adaptation to soft lens wear does not occur to the same extent as to rigid lenses. However, it is unclear if the lack of adaptation could be a contributor to discomfort in soft lens wearers. The bulbar conjunctiva is less densely innervated than the cornea and there is no evidence for a change in conjunctival sensitivity with current contact lenses. Both hydrogel and SiHy lenses could increase limbal conjunctival sensitivity because of mechanical interactions between the lens and the limbal conjunctival sensory nerves. Increased limbal sensitivity from this mechanical interaction can contribute to symptoms of lens discomfort. The mechanism of the interaction between soft contact lenses and limbal conjunctiva in symptomatic lens wearers is unclear. However, the strength of the mechanical interaction might be influenced by lens edge design, lens modulus and lens thickness. Soft lens wear produces a reduction in upper lid palpebral conjunctival sensitivity. However, an increased sensitivity in symptomatic lens wearers could contribute to lens discomfort. Lid margin sensitivity is the highest of all the conjunctival areas, although it is generally reduced in both rigid corneal and soft lens wear as a result of the mechanical interaction between the eyelid margin and the edge of the contact lens during blinking.

Ocular growth modification with contact lenses

Previous studies showed that the control of axial length growth or myopia progression in children could not be achieved by single vision rigid or soft contact lenses. In contrast, in numerous myopia control clinical trials in children with either rigid ortho-K or soft multifocal lenses, both axial length growth and vitreous chamber depth were effectively controlled, resulting in slower progression of myopia compared to controls. The mechanism for controlling axial elongation with ortho-K lenses is not fully understood. However, like in soft multifocal lenses for myopia control, the signal for slowing down myopia in rigid ortho-K has been mainly attributed to an increased myopic relative peripheral defocus caused by differential central and peripheral retinal stimulus from the lens design.

In summary, this CLEAR paper emphasizes that the desired effect from the interaction of a contact lens with the complex anatomy and physiology of the eye is that the ocular surface structure and physiology should remain unchanged. However, it is also recognised that a contact lens can be intentionally designed to alter a targeted structure e.g. limiting axial length growth for myopia control. The report pointed to significant improvements in the past two decades in the safety of contact lenses through advances in oxygen permeability, lens material and design along with lens replacement schedules. The review also pointed to shortcomings in other important areas of research relating to the interaction of contact lenses with the eyelids, blinking, meibomian gland function and structure, ocular surface sensitivity, LIPCOF and LWE, and their relationship with contact lens discomfort.

The report paid particular attention to contact lens-induced subclinical inflammatory responses of different ocular surface structures and their possible relation to adverse events and lens discomfort, highlighting the importance of further research in this field which may help to prevent dropout from wear. The report predicts that this topic along with the two other important research fields of rigid ortho-K and soft multifocal CLs for myopia control will be at the forefront of future research.

REFERENCES:

1. Morgan PB, Murphy PJ, Gifford KL, *et al*. CLEAR – Effect of contact lens materials and designs on the anatomy and physiology of the eye. *Cont Lens Anterior Eye* 2021;44:192-219.