

SANGER CONTACT LENS

KERATOCONUS

Who gets Keratoconus?

The actual incidence of KC is not known. It is not a common eye disease, but it is by no means rare. It has been estimated to occur in 1 out of every 2,000 persons in the general population. Keratoconus is generally first diagnosed in young people at puberty or in their late teen's. It is found in all parts of the United States and the rest of the world. It has no known significant geographic, cultural or social pattern.

What happens?

The cornea is the clear window of the eye and is responsible for refracting most of the light coming into the eye. Therefore, abnormalities of the cornea severely affect the way we see the world making simple tasks, like driving, watching TV or reading a book difficult.

In its earliest stages, keratoconus causes slight blurring and distortion of vision and increased sensitivity to light. These symptoms usually first appear in the late teens and early twenties. Keratoconus may progress for 10-20 years and then slow or stabilize. Each eye may be affected differently.

What can be done about it?

In the early stages, eyeglasses or soft contact lenses may be used to correct the mild nearsightedness and astigmatism caused in the early stages of keratoconus. As the disorder progresses and the cornea continues to thin and change shape, rigid gas permeable (RGP) contact lenses are generally prescribed to correct vision more adequately. The contact lenses must be carefully fitted, and frequent checkups and lens changes may be needed to achieve and maintain good vision. Intacs, intracorneal rings, are sometimes used to improve contact lens fit.

Corneal crosslinking is a new treatment option under investigation to halt the progression of keratoconus.

In severe cases, a corneal transplant may be needed due to scarring, extreme thinning or contact lens intolerance. This is a surgical procedure that replaces the keratoconus cornea with healthy donor tissue.

What causes Keratoconus?

The exact cause of keratoconus is unknown. There are many theories based on research and its association with other conditions. However, no one theory explains it all and it may be caused by a combination of things. It is believed that genetics, the environment and the endocrine system all play a role in keratoconus.

Genetic

One scientific view is that keratoconus is developmental (i.e., genetic) in origin because in some cases there does appear to be a familial association. From the presently available information there is less than a one in ten chance that a blood relative of a keratoconic patient will have keratoconus. The majority of patients with keratoconus do not have other family members with the disease. Some studies show that keratoconus corneas lack important anchoring fibrils that structurally stabilize the anterior cornea. This increased flexibility allows that cornea to "bulge forward" into a cone-shaped appearance.

Environmental

- **Eye Rubbing:** Keratoconus corneas are more easily damaged by minor trauma such as eye rubbing. Poorly fit contact lenses (that rub against the irregularity of the KC cornea) have been suggested as a possible cause of keratoconus; this has not been proven and remains questionable.
- **Allergies:** Many who have keratoconus report vigorous eye rubbing and also have allergies (which cause eye itching and irritation, leading to eye rubbing), however the link to allergic disease also remains unclear. A higher percent of keratoconic patients have atopic disease than the general population. Disorders such as hay fever, eczema, asthma, and food allergies are all considered atopic diseases. *Those with KC are advised to avoid eye rubbing as much as possible.*
- **Oxidative Stress:** Some studies indicate an abnormal processing of the superoxide radicals in the keratoconus cornea and an involvement of oxidative stress in the pathogenesis of this disease. Keratoconus corneas lack the ability to self-repair routine damage easily repaired by normal corneas. Like any tissues in the body, the cornea creates harmful byproducts of cell metabolism called free radicals. Normal corneas, like any other body tissue, have a defense system in place to neutralize these free radicals so they don't damage the collagen, the structural part of the cornea, weakening it and causing the cornea to thin and bulge. The keratoconus corneas do not possess the ability to eliminate the free radicals so they stay in the tissue and can cause structural damage.

Hormonal

Another hypothesis is that the endocrine system may be involved because keratoconus is generally first detected at puberty and progresses during pregnancy. This theory is still controversial and has not been proven.

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