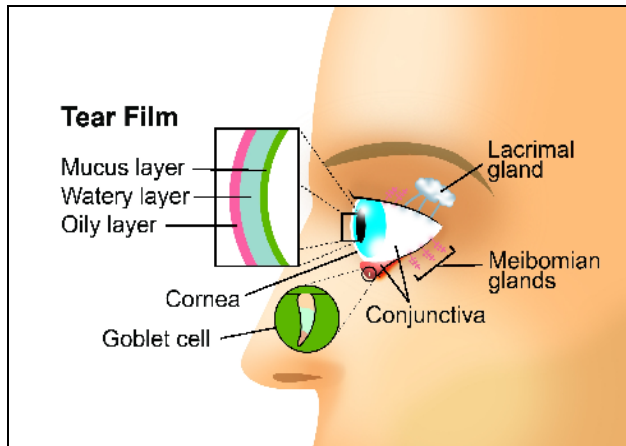


## Dry Eye Syndrome

Dry eye syndrome (DES), also known as keratoconjunctivitis sicca (KCS), is one of the most common disorders of the eye. It is caused by a lack of tear quantity, or lack tear quality, adequate tear production without the necessary elements to maintain them. Those suffering from DES present with a sense of dryness, grittiness, foreign body sensation, or



visual disturbances depending on the severity of the condition. DES is much more complex than originally thought, involving secretions from three separate glands, comprehensively known as the lacrimal system.

What are commonly known as tears are actually a film comprised of three layers. The outermost lipid (oily) layer is secreted by the meibomian glands. This oily layer coats the middle aqueous (water) layer secreted by the

lacrimal glands and retards evaporation. Once thought only to wash away debris, it is now understood that the aqueous layer supplies oxygen and a special mixture of electrolytes to surface eye cells since these cells have no blood supply. The innermost or base layer of the tear film is a mucous layer produced by the conjunctiva goblet cells which attaches the tear film to the corneal surface. The hydrophilic quality of the mucous layer allows the aqueous to spread over the corneal surface. The mucus is secreted by goblet cells on the ocular surface. Each of these layers is critical to producing and maintaining tear film that hydrates, nourishes, and protects the ocular surface from infection.<sup>i</sup> Dysfunction of one or more of these elements may cause DES.

Central to virtually all dry eye disorders is a loss of water from the tear film that increases solutes (osmolarity) on the ocular surface such as sodium and potassium. This loss of water and increase in osmolarity may result from any condition that either decreases tear production or increases tear evaporation; either of which causes the eye surface to dry, become irritated, itchy, or have the sensation of foreign matter. Tear production can decline due to lacrimal gland disease, as a result of anything that decreases the delivery of lacrimal gland fluid to the ocular surface, or even in reaction to decreased corneal sensation. Increased evaporation, meanwhile, commonly comes from meibomian gland dysfunction, which causes a drop in the amount of protective lipid in the tear film. Whatever the initial causes of dry eye, chronic dryness of the ocular surface results in inflammatory reactions and gradual destruction of the lacrimal glands and conjunctival epithelium. Once dry eye disease has developed, inflammation is the key mechanism of ocular surface injury, as both the cause and consequence of cell damage.<sup>ii</sup>

In the last decade, it has become well-recognized that sex hormones such as androgens, estrogens, and progesterones help regulate the function of the lacrimal and meibomian glands.<sup>iii</sup> An imbalance in these hormones may lead to DES and irritation to the ocular

surface. Perceiving the eye to be under attack, the body's immune system produces T lymphocytes, immune cells that destroy infection. These T cells in turn release proteins known as cytokines, which cause inflammation on the ocular surface.<sup>iv</sup> The proper effects of these hormones are critical to glandular secretions which comprise tear film and also determine who is susceptible to DES.

In several population-based studies DES have been found to be much more prevalent in women.<sup>v</sup> The decrease in serum androgens in women as they reach menopause has been shown to increase the incidence of DES. This has been confirmed in Sjogren's syndrome, an autoimmune disease causing the most severe dry eye condition, which occurs almost exclusively in women.<sup>vi</sup> Both the aqueous-producing lacrimal glands and the lipid-producing meibomian glands are sensitive to androgens. As women age, androgen production decreases, the functionality of these glands can wane, causing either insufficient aqueous production from the lacrimal glands or a lack of lipid secretion from the meibomian glands, decreasing the evaporation time of tears produced, or both.

- <sup>i</sup> Lemp MA, Foulks GN et al. The Therapeutic Role of Lipids, Managing Ocular Surface Disease. *Refractive Eye Care for Ophthalmologists*, Vol. 9, Number 6, June 2005.
- <sup>ii</sup> Gilbard JP. Dry Eye - Natural History, Diagnosis and Treatment. *Optometric Management*.
- <sup>iii</sup> Sullivan DA. Gender and sex steroid influences in dry eye syndrome. *Dry Eye and the Ocular Surface*. New, NY: Macel Dekker Inc.
- <sup>iv</sup> Connor CG. Symptomatic Relief of Dry Eye with Progesterone Cream. Presentation. American Academy of Optometry 2006 Annual Meeting. December 2006.
- <sup>v</sup> McCarty CA et al. The epidemiology of dry eye in Melbourne Australia. *Ophthalmology*. 1998; 105:1114-1119. As cited by Pflugfelder SC in Hormonal Deficiencies and Dry Eye. *Arch Ophthalmol*, Vol 122, Feb 2004.
- <sup>vi</sup> Valtyskottir ST, Wide L, Hallgren R. Low serum dehydroepiandrosterone sulfate in women with primary Sjogren's syndrome as an isolated sign of impaired HPA axis function. *J Rheumatology*. 2001;28: 1259-1265. As cited by Pflugfelder SC in Hormonal Deficiencies and Dry Eye. *Arch Ophthalmol*, Vol 122, Feb 2004