

Does the Tear Film Lipid Layer Inhibit THE Rate of Evaporation of Tears?

Douglas Borchman*

Department of Ophthalmology, University of Louisville, USA

***Corresponding Author:** Douglas Borchman, Kentucky Lions Eye Center, University of Louisville, 301 E Muhammad Ali Blvd. Louisville KY 40202. USA.

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Editorial

A 0.1 μm thin layer of lipid, the tear film lipid layer (TFLL), on the 4 μm thick surface of tears [1-39] is produced by the meibomian glands located in the eye lids [4]. The functions of the TFLL are to dam, lubricate, and stabilize the tear film to allow for proper refraction, prevention of evaporation, degradation of mucinic clots, provide antibacterial effect, and suppress exposure to UV rays [4]. About 10 seconds after blinking, tears 'break up' and have to be reformed by another blink. Tear break up time is much lower in people with dry eye symptoms and it is believed that evaporation is associated with tear break up time and stability. Dry eye has been classified as aqueous production-deficient and evaporative [5]. Evaporative dry eye is associated with meibomian gland dysfunction and contributes to 80% of the dry eye cases [6]. Over 6 million people suffer from dry eye in the United States [7].

As a scientist experienced with lipids and membranes, intuitively I would think that the tear film lipid layer would inhibit the rate of evaporation (R_{evap}) of tears due to strong hydrophobic interactions between lipids and the fact that water and oil don't mix. Indeed there is some support for this idea. Classic studies by Archer and La Mer. [8] indicate that longer chain fatty acids offer greater resistance to evaporation compared with shorter chains. Over 50 years ago, studies using rabbits show that adding lipids to lipid depleted eyes decreased R_{evap} by over 75% [9,10]. Wax ester films about as thick as the TFLL were found to inhibit R_{evap} by 30 to 50% when they were within 2% of their melting temperature, a temperature where fluid and ordered phases co-exist [11]. Research related to R_{evap} reduction by duplex lipid films has been reviewed [12]. Two studies suggest that adding a monolayer of cetyl alcohol on the surface of large ponds inhibits R_{evap} by an average of $17 \pm 17\%$ [13].

Conversely, there are a number of studies that indicate lipid films do not inhibit R_{evap} . A study where the size of the control and experimental large water reservoirs were similar, a film of cetyl and stearyl alcohol, estimated to be about 0.14 μm thick, did not inhibit R_{evap} ($n = 4$) [13]. Experiments done 60 years ago showed that wax esters, the major lipid type of the TFLL, have a low resistance to evaporation [14], so in light of this study, it was not surprising that *in vitro* studies using human meibum spread over the surface of aqueous saline did not inhibit R_{evap} [15,16]. *In vitro*, physiological saline evaporates at a rate of $8.0 \pm 0.5 \mu\text{m}/\text{min}$ [17], similar to the R_{evap} for tears ($9.3 \pm 0.9 \mu\text{m}/\text{min}$, [17] and similar to R_{evap} measured *in vivo* for contact lens wearers ($6.97 \mu\text{m}/\text{min}$ [1]). So there is nothing unusual about the R_{evap} of tears measured *in vitro* or *in vivo*. More recently, a pool of lipids resembling the TFLL did not inhibit R_{evap} and a layer of olive oil 2,000 times the thickness that of the TFLL only decreased R_{evap} by 53% [18].

Surprised by the conflicting results, my group measured R_{evap} *in vitro* of buffer layered individually with a film of six 1-hydroxyl hydrocarbons (11 to 24 carbons) and reflex human tears layered with a film of human meibum estimated to be 34.4 μm thick [19]. R_{evap} were measured simultaneously in samples with and without lipid. R_{evap} of the samples with a film synthetic lipids or meibum was essentially similar to that of buffer or tears. The thickness of the hydroxyl lipid film did not influence R_{evap} over an estimated thickness range of 6.9×10^{-5} to $> 69 \mu\text{m}$. Experiments were repeated over 60 times by eight different investigators. Human meibum and hydroxyl lipids, regardless of their fluidity, chain length, or thickness did not inhibit R_{evap} of buffer or tears even though they form a relatively uniform surface layer. It is unlikely that hydroxyl lipids can be used to significantly (less than 10%) inhibit R_{evap} of reservoirs. Our data do not support the widely accepted (yet unconfirmed) idea that the tear film lipid layer inhibits R_{evap} of tears.

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A possible resolution to the discrepancies between *in vivo* and *in vitro* studies mentioned above is the speculation that as tears initially evaporate at the same rate as water the tear film becomes thinner and the TFL eventually contacts the higher concentrations of mucin near the surface of the cornea where the lipid layer then interacts with mucin or other proteins to inhibit the rate of evaporation. Another possibility is that conventional wisdom is incorrect and that the TFL does not inhibit evaporation and that there are other reasons for having a TFL [20].

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