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Review

Transport and interaction of cosmetic product material within the ocular surface: Beauty and the beastly symptoms of toxic tears

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ABSTRACT

Eye cosmetics such as mascara, eye shadow and eyeliner are used extensively to highlight the eyes, and are normally applied external to the ocular surface. Adverse reactions of cosmetics within the ocular surface include mild discomfort, eyelid dermatitis, pre-corneal tear film instability, and keratitis. These are attributed mainly to the preservative (benzalkonium chloride (BAC)) constituent of cosmetic product material (CPM).

Transport of CPM from an external environment to any location on the ocular surface, essentially precedes the adverse interactions occurring at the location, and the control of these transport modes is therefore of clinical relevance.

The inter-transport of CPM across the TF occurs due to both diffusion and drift processes. Diffusion of neutral species is driven by concentration gradients, and the drift of cationic BAC is influenced by the inherent electric field; determined by the distribution of the various ions secreted into the aqueous layer, and the negative glycocalyx charge at the mucin layer.

In the presence of mucin deficiency, the corneal epithelium is exposed to invasion by both incident BAC and lipophilic species. The transport of cationic BAC across the TF may be controlled by regulating the secretion of various electrolytes at the lacrimal gland. This is of clinical significance in reducing corneal epithelial adverse effects. However, the risks of adverse effects at the corneal surface due to invasion by the lipophilic species remain. Patients with mucin deficiency, and especially those on eye ointment/drops medication, should be discouraged from using cosmetics in a way likely to contaminate the TF.

1. Introduction

Cosmetics [1–9] such as mascara, eye shadow and eyeliner are used extensively world-wide to highlight and emphasise the eyes. Most of these are applied away from the ocular surface (Fig. 1) but some may exist precariously close to the eyelash margin. However adverse reactions [10–15] to these eye cosmetics, ranging from simple irritation, keratitis, corneal epithelium inflammation, eyelid dermatitis and dry eye [16–18] symptoms have been reported. This suggests migration of cosmetic product material (CPM) from an external environment onto the ocular surface including the pre-corneal tear film (TF). It is also obvious that adverse reactions at any distal location on the ocular surface essentially occur due to the transport of CPM from an external environment to this location and the resulting chemical/biological interactions at the location determine the nature of the adverse activity. Thus the control/suppression of CPM transport modes within the ocular surface is of relevance in the prevention of adverse effects of eye cosmetics.

The chemical composition of eye cosmetics is complex, but is well documented [1–9]. Additives such as fragrance and preservatives in cosmetics are well known to give rise to toxic and allergic reactions [11,19]. The current manufacturing trend is to avoid the use of fragrance, but the inclusion of preservatives is necessary in order to prevent growth of bacteria during storage and control of infection. Benzalkonium chloride (BAC) is the most common preservative used in eye cosmetics [11,19–23].

Significant observed deterioration [24,25] in contact lenses during wearing has also been attributed to the use of ocular cosmetics. Mascara pigmentation of the bulbar conjunctiva is associated with rigid gas permeable lens wear [26].

In this review, the expected CPM transport modes within the various regions of the ocular surface will be discussed. Transport driving factors and the resulting adverse effects are identified. For completeness, the properties of popular eye cosmetics i.e. eyeliner, mascara and eye shadow are also summarised.

The initial migration of CPM from the external environment onto the immediate ocular surface i.e. adjacent to the eyelash margin, occurs [27,28] due to mechanical push, suction due to tear surface tension or wrapping/blinking of the eyelids. Conjunctival absorption, epithelial pigmentation and posterior blepharitis are

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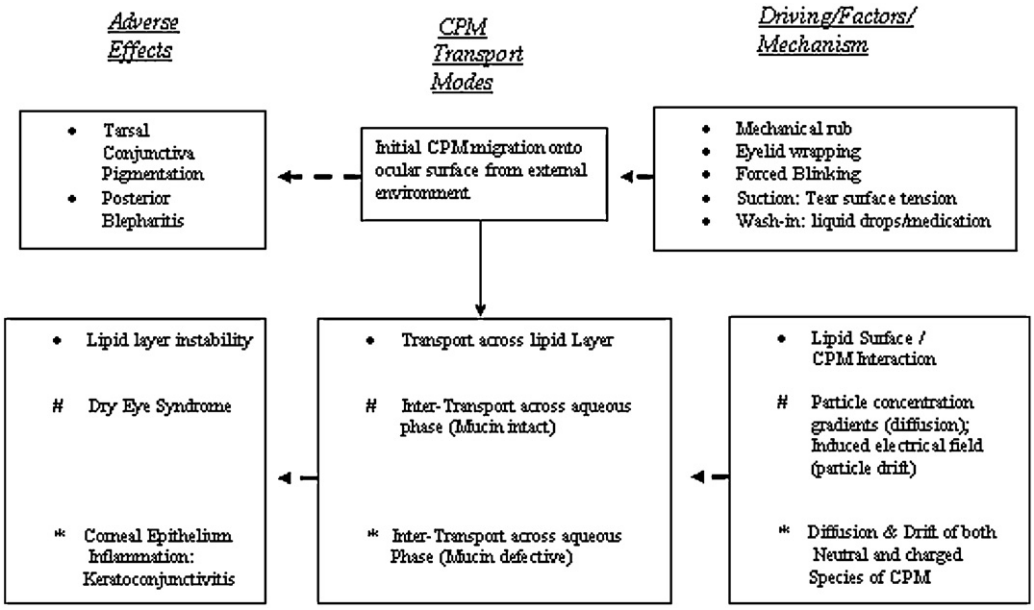

Fig. 1. Interrelationship between various cosmetic product material (CPM), transport modes, driving factors/mechanisms, and the resulting adverse effects of eye cosmetics within the ocular surface.

the typical expected adverse effects during this phase of transportation.

The subsequent interaction and transport of CPM across various layers of the TF are largely determined by the characteristic properties of its constituents, the chemistry of the individual TF layer and the driving forces such as an electric field or concentration gradient. The distribution of +ve and −ve ions (electrolytes secreted by the lacrimal gland) within the aqueous layer, in conjunction with the −ve glycocalyx at the mucin, give rise to the electric field, leading to drift of the charged species within the TF. Due to the constant scavenging effect of the mucin (removal of incident debris and pathogens), the net charge in the region at any time and hence the electric field is difficult to predict. Concentration gradients, leading to diffusion processes, arise mainly due to the aggregation of the neutral species within the medium. For the purpose of the present work, CPM is considered as a mixture of BAC (cationic polar molecules) and neutral lipophilic species.

The initial penetration of BAC across the lipid layer of the TF, in the absence of an electric field, is concentration dependent. In vitro experimental study [29] concerning interaction of BAC with lipids, suggests that at low concentration <0.01%, adsorbed BAC within the lipid layer exists as individual monomers and as micelles at higher concentration >0.01%. Monomers are generally unable to penetrate the lipid layer, but the few that do penetrate, alter its structure and are trapped within the layer.

We suggest that cationic polar BAC, because of its affinity for water/aqueous; preferentially align along the lower polar lipid layer arrangement adjacent to the aqueous layer. Thereafter, the majority of charged BAC species drift into the aqueous phase due to the force of the electric field. Others, form neutral complexes with polar lipids and are displaced as neutral species into the aqueous phase. This process is probable, as charged cationic BAC is deficient in electron and the polar lipid bilayer is ready to be ionised. It is similar to ionisation of polar lipids in the presence of an ion that facilitates the transfer of electrons leading to formation of free radicals [30,31]. However due to the weak forces involved, complete transfer of an electron does not occur, rather an electron is shared between electron-deficient BAC and polar lipid to form neutral BAC complex.

Both neutral and cationic BAC species thus exist within the aqueous layer beneath the lipid layer, and directly affect the osmolarity and viscoelasticity of the TF, leading to dry eye symptoms.

Within the aqueous layer, the drift of the cationic BAC species is dictated by the prevailing electric field, and the neutral species diffuse according to the laws of passive diffusion governed by concentration gradient. Net excess −ve charge within the TF gives rise to a negative field that promotes drift of cationic BAC towards the mucin layer, whereas net +ve charge leads to positive field that retards the drift of charged BAC towards the mucin layer. Neutral BAC complexes after aggregation within the aqueous phase beneath the lipid layer move according to the laws of diffusion.

Lipophilic species of CPM initially diffuse through the lipid bilayer according to the laws of simple diffusion [32]. Thereafter the diffusing neutral lipophilic species are insoluble within the host aqueous layer and tend to aggregate beneath the lipid layer, leading to passive diffusion within the aqueous phase, driven by concentration gradient.

Further along the aqueous layer, the deep interposing mucin layer acts as a barrier, and under normal circumstances the corneal surface is protected from invasion by incident BAC and lipophilic species.

However, in the presence of mucin deficiency, both BAC and lipophilic species are able to interact with the corneal epithelium.

The interaction of the cationic BAC with the epithelium may be reduced by creating net +ve charge within the aqueous layer by enhancing the secretion of positive electrolytes at the lacrimal gland, and this is of clinical significance particularly during mucin deficiency.

However, in patients with mucin deficiency, adverse effects at the corneal epithelium can occur due to lipophilic species alone i.e. even with preservative-free cosmetics. These patients and especially those on eye ointment/drops medication should be informed of the beastly aspects of cosmetics and discouraged from using cosmetics in a way likely to contaminate the TF.

Fig. 1 illustrates the interrelationship between the various CPM transport modes, the driving factors/mechanisms and the resulting adverse effects of eye cosmetics within the ocular surface.