Inflammation and Cosmetic Intolerance

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Introduction

Consumers using topical products often find that many products cause a variety of reactions ranging from irritation to apparent allergic dermatitis. As the numbers of topical products on the market as well as the number of end users have increased dramatically, so has the number of adverse incidents with these products. These incidences have become so prevalent in society that it has approached epidemic proportions. Maibach et al have recently identified these phenomena and have coined the term Cosmetic Intolerance Syndrome (CIS) to describe the issue.

CIS can be described as the inability of an end-user to tolerate topical application of cosmetic products. The individual who suffers from this syndrome is often reactive to a single component or a combination of components. The intolerance of the skin to these components manifests itself in apparent pseudo-allergic reactions and a reduction in their quality of life. The cause of this syndrome is unknown but likely lies in disruption of the sequence of events leading to the inflammatory response.

The Inflammatory Response

UV exposure and the inflammatory responses generate a multitude of adverse changes such as lipid oxidation, loss of enzyme function, protein glycation, thus leading to depletion of natural cellular protectants, a compromised immune system and DNA damage. Protection of the skin from inflammatory reaction is of paramount importance, as the response results in damage to the skin at the visible, cellular, molecular and sub-molecular level.

Inflammatory mediators cause an increase in the production of prostaglandins (PG), the molecules that cause the inflammatory response. The increase in PG production is brought about by an induction (an increase in the production) of the gene that codes for the enzyme prostaglandin endoperoxide synthase, also known as COX2. This enzyme is the rate limiting step in the production of PG and is therefore the most significant reaction in the inflammatory response. Numerous non-steroidal anti-inflammatory drugs (NSAIDs) have been developed that inhibit this reaction, reduce PG synthesis and inhibit the inflammatory response.

The NSAID Dilemma

Although numerous NSAIDs have been successfully developed, each of these has prevalent side effects. NSAIDs are rarely specific to the inhibition of COX2. Often they inhibit other enzymes, in particular COX1. COX1 produces an alternate form of PGs with protective effects and therefore non-selective inhibition of this enzyme leads to deleterious side effects. The side effects of NSAIDs are well known to the public and this has lead to apprehension in their widespread use.

ActiSoothe™

ActiSoothe™ is a patent pending bio-processed extract of Coriolus versicolor with multi-functional skin attributes. Designed to reduce both short and long term forms of skin irritation and redness, it dramatically reduces Cosmetic Intolerance Syndrome (CIS) by normalizing hyperactive skin by down regulation of key enzymes involved in the inflammatory response. In particular, ActiSoothe™ downregulates the synthesis of COX2, the rate limiting step in the inflammatory response without affecting COX1 synthesis. By decreasing the active concentration of COX2, we have developed a selective method of inhibiting COX2 activity and thus reduced inflammatory PG production without the serious side effects of other anti-inflammatory actives. See Figure 1 overleaf.