Sodium Ascorbyl Phosphate Shows In Vitro and In Vivo Efficacy in the Prevention and Treatment of Acne Vulgaris

Authors: Jochen Klock, DSM Nutritional Products, Basel, Switzerland

Keywords: Sodium Ascorbyl Phosphate, acne vulgaris, Proprionibacterium acnes, lipid oxidation, clinical efficacy

Abstract

Acne vulgaris is the most common inflammatory skin disorder and jeopardises seriously the facial impression of a person. Development of acne involves a complex relation among several causes. Treatment and prevention success can be archived by affecting the main contributors positively like Proprionibacterium acnes or lipid oxidation leading to inflammatory reactions and follicular keratinization.

Vitamin C tends to break down in cosmetic formulations resulting in a brownish discoloration. Sodium Ascorbyl Phosphate represents a stable precursor of vitamin C that ensures a constant delivery of vitamin C into the skin.

We were able to show that 1% Sodium Ascorbyl Phosphate has a strong anti-microbial effect with a log reduction of 5 after 8 hours on P. acnes in a time-kill study. Further on in a human *in vivo* study with 20 subjects a Sodium Ascorbyl Phosphate O/W formulation significantly prevents the UVA-induced sebum oxidation up to 40%. Finally we performed an open *in vivo* study with 60 subjects with a 5% Sodium Ascorbyl Phosphate lotion over 12 weeks. The efficacy of Sodium Ascorbyl Phosphate was 76.9%, ranked as excellent and good, and was superior to a widely prescribed acne treatment.

In conclusion this data shows that Sodium Ascorbyl Phosphate is efficient in the prevention and treatment of acne vulgaris. Sodium Ascorbyl Phosphate can be used in a non-antibiotic and effective treatment or co-treatment of acne with no side effects, which makes it particularly attractive for cosmetic purposes.

Introduction

The etiology of acne vulgaris is mainly applied to skin areas with a high density of pilosebaceous units such as face, chest and upper neck. Acne lesions arise from pilosebaceous units, which consist of sebaceous glands and small hair follicles. Acne vulgaris is a multifactor disorder, with seborrhea, follicular hyperkeratinization, and bacterial colonization by Proprionibacterium acnes (P. acnes). Inflammation plays a significant role in the pathogenesis as well[1, 2]. Obstruction of the pilosebaceous canal is the primary cause of acne and occurs because of a variety of factors[3]. The first factor is sebum overproduction stimulated by intrinsic signals. Excess sebum production combined with an increase in epithelial cell turnover leads to formation of comedones[4]. The combination of sebum and desquamated cells provides a rich environment for the growth of P. acnes, the principal organism in inflammatory acne lesions. Proliferation of P. acnes leads to sebum hydrolysis into free fatty acids by lipases causing irritation and stimulation of an immune response. Inflammatory lesions are induced by secreted inflammatory mediators from bacteria and involved skin cells.

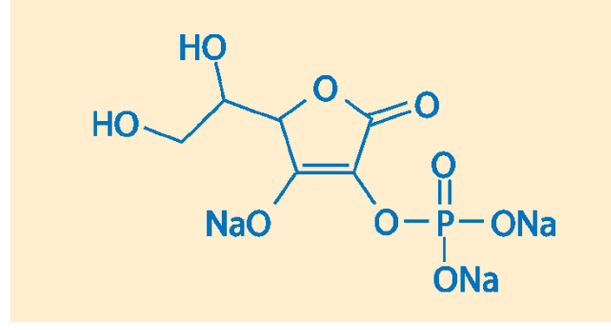

Figure 1: Chemical structure of Sodium Ascorbyl Phosphate (SAP)

Further on reactive oxygen species resulting in oxidation of sebum are thought to play an important role in inflammatory processes as well[1,3,5]. It's very important to promote a long-term healthy skin by preventing side-effects such as re-colonization of adverse skin bacteria after antibiotic treatment. Therefore healthy skin bacteria are important for the balance of healthy skin and should not be affected by an acne treatment. An example for these healthy skin bacteria are Staphylococcus epidermidis.

Vitamin C or ascorbic acid is one of the most widely used antioxidants for protecting the skin. Unfortunately, endogenous vitamin C is easily depleted when the skin is exposed to the sun, and by other external stressors such as smoking[6-8]. Maintaining