Introduction

Atopic dermatitis (AD) is a chronic relapsing condition of pruritus and eczematous lesions that affects 15-20% of the childhood population and 1-3% of adults worldwide with increasing prevalence in highly industrialized countries (Roll et al., 2004).

Staphylococcus aureus (S. aureus) is the most important microorganism of the normal skin flora, the bacterial skin flora of patients with atopic dermatitis is different from that in healthy people. In addition, such patients more often suffer from microbial infections such as impetigo, folliculitis, and furunculosis S.aureus infection is perceived not only as a secondary complication of AD, but also worsening AD (**Pezesk et al.,2007**).

Atopic skin provides a favourable environment for the colonization and proliferation of S. aureus. Secondarily infected patients show greater clinical improvement to combined treatment with anti- staphylococcal antibiotics and topical corticosteroids, compared with topical corticosteroids alone, supporting the concept that S. aureus contributes to skin inflammation in AD (**Donald, 2008**).

Once the skin is colonized by S. aureus, it can aggravate AD by various mechanisms, for example through bacterial super-antigens and other exotoxins. Superantigens of S.aureus activate various cells including T cells, eosinophils, Langerhans cells, macrophages, mast cells and

keratinocytes, which are important in the pathogenesis of AD [Bieber, (2008) and Leung et al., (2008)].

Up to 65% of S. aureus strains isolated from patients with AD colonized with this microorganism express superantigens (**Llewelyn and Cohen , 2002**), which polyclonally activate T cells, resulting in increased cytokine production which can enhance presentation of allergen to T helper2 (Th2) cells by keratinocytes Furthermore superantigens induce the production of superantigen specific IgE, which contributes to inflammation in AD through mast cell Degranulation (**Baker,2006**), and is also thought to activate mechanisms of pruritus, in addition, it has also been found that S. aureus superantigens can inhibit the suppressive activity of regulatory T cells (**Gomes et al.,2010**).