INTRODUCTION AND AIM OF WORK

Acne vulgaris is the most common cutaneous problem of adolescence affecting more than 80% of individuals in their second and third decades (Msliborski & Lumpkin 1981).

The Pathogenesis of acne vulgaris is multifactorial and involves the combined roles of the pilo sebaceous unit, microorganisms and host’s response to the events that occur within the pilosebaceous unit. The abnormal keratinization that develops at puberty probably represents the earliest change in the development of acne (comedo). The increased amounts of sebum, and proliferations of the normal bacterial flora, form a plug which prevents normal follicular emptying (Cullen 1985). Propionbacterium acne appears to be the microbiologic factor, that correlates best with acne (Cove et al., 1980; Shalita et al 1979) though there is no correlation between their number and severity of the lesion (Shalita et al., 1979). The organism secretes various extracellular products including lipases, hyaluronidases, proteases and chemotactic factors (August 1985).

Current management of acne comprises various topical and systemic drugs; including benzoyl peroxide, antibiotics and Retinoids. However, despite the established therapeutic efficacy of these drugs, recurrences with acne are nevertheless frequently encountered due to the chronic nature of the disease. Thus repeated long term courses of treatment may be required
which emphasizes the need for therapy safety (Katsambas et al., 1989).

Azelaic acid is a c-q dicarboxylic acid which is an antibacterial agent (King et al., 1985) and has been reported to be neither toxic nor teratogenic (Mingrone et al., 1983). In an open study, it was shown to be of therapeutic benefit in acne (Nazzaro-Porro et al., 1983)

**AIM OF WORK**

The aim of the present study is to evaluate the efficacy, tolerability and safety of azelaic acid 20% cream in the treatment of acne vulgaris.