# INTRODUCTION

# CHAPTER 1

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Psoriasis (a term that comes from the Greek word 'Psori' meaning 'Itch') is a chronic, common inflammatory and proliferative disease of the skin characterized by sharply demarcated dull red, scaly papules and plaques distributed mainly on extensor aspects of the extremities, trunk and scalp. Its prevalence in the general population is 0.5 to 2.5% worldwide and 0.44 to 2.8% in India (Dogra and Yadav, 2010). Abnormal keratinocyte proliferation, differentiation and infiltration of inflammatory components into the skin characterize the pathology. Psoriasis is a multifactorial disease, generally believed to be a T-cell mediated disorder in which environmental factors

precipitate the disease in a genetically predisposed individual (Griffiths and Barker, 2007). However, other mechanisms might be involved. Recently, psoriasis has been postulated to result from imbalance in oxidant/antioxidant system of the skin (Yildirum *et al.*, 2003). The involvement of antioxidant enzyme systems in the regulation of keratinocyte proliferation has also been reported (Grashin *et al.*, 2010).

Zinc is an essential trace metal involved in virtually all aspects of metabolism by acting as a metal moiety of more than 300 metalloenzymes (King and Keen, 2003). It also plays an important role in the maintenance of normal immune functions (Prasad, 2008). Besides, zinc is also considered an important antioxidant for skin playing a vital role in the protection against free radical damage (Rostan *et al.*, 2002). The role of trace minerals in the aetiopathogenesis of psoriasis has been investigated by a few workers only (Hinks *et al.*, 1987; Basavaraj *et al.*, 2009). Earlier studies regarding the relationship between the serum zinc status and psoriasis have revealed conflicting results (McMillan and Rowe, 1983; Bruske and Salfeld, 1987; Kreft *et al.*, 2000; Ozturk *et al.*, 2001). One study (Bruske and Salfeld, 1987) has reported increased serum zinc level in psoriasis patients while another study (McMillan and Rowe, 1983) has shown low plasma zinc level that was inversely proportional to the area of psoriatic involvement. Yet, serum zinc level has been found not to have statistically significant change in

some other studies (Kreft *et al.*, 2000, Ozturk *et al.*, 2001). Levels of inherently zinc-dependent alkaline phosphatase enzyme have also been found to increase 4-fold in psoriatic skin (Hajini *et al.*, 1977). Moreover, patients with psoriatic arthritis have been reported to benefit from oral zinc sulphate therapy (Clemmensen *et al.*, 1980) and addition of zinc to topical steroids in the treatment of psoriatic skin has produced better results (Thomas and Kandhari, 2001). Only a few studies have been reported from India on the aspect regarding the relationship between the serum zinc status and psoriasis (Basavaraj *et al.*, 2009; Saxena *et al.*, 1990; Bhatnagar *et al.*, 1994; Nigam, 2005). Moreover, different methods have been used for serum zinc determination by the earlier investigators. Though the main method adopted for serum zinc determination has been atomic absorption spectrophotometry, a new enzymatic method (Demir *et al.*, 1993; Erel and Avci, 2002) of measurement of serum zinc level has also been described. In view of the conflicting data of serum zinc level in psoriasis patients as well as paucity of such study particularly from the north-eastern part of India, the present investigation has been carried out aiming at (i) adopting a simplified enzymatic method of serum zinc determination based on re-activation of apocarbonic anhydrase by $Zn^{2+}$, (ii) determining the nature of variation in serum zinc level in psoriasis patient population in Manipur, and (iii) defining the variation of the serum zinc level as a function of severity of psoriasis. Hundred confirmed cases of psoriasis attending the

Outpatient Department of Dermatology, Regional Institute of Medical Sciences Hospital, Imphal were included in the study. For every patient, a detailed history was taken and a record was kept about the age, sex, age at onset, duration of the disease, etc. Control comprising fifty individuals was selected from normal healthy population. After obtaining informed consent, venous blood was withdrawn and serum was prepared. Estimation of serum zinc level was carried out by a specific enzymatic method based on $Zn^{2+}$-concentration dependent apocarbonic anhydrase re-activation.