CONTENTS

LIST OF ABBREVIATIONS

Abs	antibodies
AD	atopic dermatitis
CAMP	cyclic adenosine monophosphate
CD	chronic dermatophytosis syndrome
CMI	cell-mediated immunity
CR	complement receptor
DNCB	dinitrocholorobenzene
DTH	delayed type hypersensitivity reaction
FcR	Fc receptor
HSV	herpes simplex virus
IFNγ	interferon-gamma
IgA	immunoglobulin-A
IgD	immunoglobulin-D
IgE	immunoglobulin-E
IgG	immunoglobulin-G
IL	interleukin
LCS	Langerhans cells
LT	leukotriene
MHC	major histocompatibility complex
NAP	neutrophil activating peptide
PUVA	psoralen with ultraviolet light-A
RAST	radio-allergo sorbent test
S. aureus	staphylococcus aureus
TH	helper T-cell
TNF	tumour necrosis factor
TP-5	thymopentin

Introduction & Aim of the Work

INTRODUCTION AND AIM OF THE WORK

INTRODUCTION:

Atopy is a genetically determined disorder in which there is an increased liability to form reagin (IgE) antibodies and an increased susceptibility to certain diseases especially asthma, hay fever and atopic dermatitis (Parish and Champion, 1973).

Atopic dermatitis is a chronic fluctuating disease which may occur at any age and is slightly more common in boys than girls. The distribution and morphology of the lesions vary with age but itching is the cardinal symptom. The age of onset is between 2 and 6 months in the majority of cases. The onset before the age of 2 months is exceptional but may occur and it is significant that coordinated scratching does not occur before this time. The disease may start at any age later in life, even over the age of 50 (Champion and Parish, 1992).

The pathogenesis of atopic dermatitis is influenced by genetic and environmental factors. The skin of affected patients is usually colonized by large numbers of staphylococcus aureus bacteria, , these bacteria may aggravate atopic dermatitis or prevent resolution of the disease (Dahl, 1983).

The mechanism by which staph aureus can trigger atopic dermatitis flares might be one of several things. Staphylococcal cell wall products (teichioc acid and peptidoglycan), as well as a staphylococcal secreted superantigen, entero-toxin B, can stimulate peripheral blood lymphocytes, and particularly in combination with IL-4, can lead to pronounced increase in IgE synthesis and FcIgE receptor expression (Neuber and Konig, 1992).

AIM OF THE WORK:

The aim of the work is isolation of staphylococcal aureus with determination of serum IL-8 of AD patients in comparison with the healthy persons toassesstheir role in the pathogenesis of AD.