

STUDY OF CELL MEDIATED IMMUNITY  
IN PATIENTS WITH SENSITIZATION  
CONTACT DERMATITIS (ECZEMA)  
BY IN VIVO AND IN VITRO TESTS

THESIS SUBMITTED IN PARTIAL FULFILMENT  
FOR MASTER DEGREE IN CLINICAL PATHOLOGY

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**INTRODUCTION  
AND  
AIM OF WORK**

## INTRODUCTION

E-rosette test is now accepted as a very valid indicator of T cell population in the peripheral blood (Sepaha et al., 1972). The delayed skin reaction is mediated by sensitized T lymphocyte and is taken as evidence of the reactivity of T cells.

The combination of both Tests provides therefore aprobably more accurate representation of the T lymphocyte function (Pepys, 1975).

Contact Dermatitis is assumed to be the classic example of delayed Hypersensitivity, (type IV Hypersensitivity), (Gell and Combs, 1968).

Owing to the above facts, both E-rosette test and delayed skin test were studied in patients suffering from Allergic C.D.

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AIM OF THE WORK

The aim of this work is to clarify some aspects of cellular immunity status of patients suffering from allergic contact dermatitis (Eczema), by E-rosettetest and delayed skin testing .

E-rosette test used was a modification of previously described techniques (Brain et al., 1970).& Kelly and Shell 1977). The method of skin testing was that described by Sokal (1975) for delayed skin testing.

Contact Dermatitis is a classical example of delayed Hypersensitivity reaction in which The cell mediated immunity plays the major role (Gell and Coombs, 1968).

In this work, we aim to detect an increase in the number of rosette forming cells, and an increase response to delayed skin test in Patients exhibiting the clinical signs of Allergic C.D. as eczematous skin manifestations, as a result of their immunization by the Allergens.

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# REVIEW OF LITERATURE

## CELL MEDIATED IMMUNITY

### 1. Historical:

Chase & Landsteiner in 1942 managed to transfer immunity successfully in some diseases by taking living lymphoid cells from an immune individual and transferring it to a non immune one.

People suffering from this group of diseases, that need cells rather than serum to acquire immunity, often show a delayed skin reaction to intradermal challenge with the appropriate antigen.

The two terms, cell mediated immunity and delayed hypersensitivity, are sometimes confusingly used as synonyms. The former may be defined as specifically provoked slowly evolving (24 - 48 hours) mixed cellular reactions involving particularly lymphocytes and macrophages, which can be transferred in experimental animals by means of such cells but not by serum (Turk, 1967).

The latter is restricted to the actual tissue damage resulting from the physiologic normal defensive reaction to the actual pathological tissue destruction (Turk, 1967). Hence, in situations such as graft rejection, antibodies are only secondarily

involved and the immune response is cell mediated (Hudson, 1972).

2. Biological significance of cellular immunity:

The cell mediated immunity has been shown to be involved in the following biological reactions;

(1) Resistance to infection:

The cell mediated immunity is responsible for the protection against slowly growing intracellular pathogens (Bacteria, virus, protozoa) by means of:

- a) Formation of effector killer cells.
- b) Activation of macrophages.

The latter mechanism is far more important than the former in resisting infection (Bowry, 1975).

(2) Tumour rejection:

It is now generally accepted that the immune system eliminates the neoplastic cells which arise by mutation of actively regenerating tissues (Hellstrom et al., 1971); this function is known as immunological surveillance.

(3) Delayed hypersensitivity reaction:

Delayed hypersensitivity is a reaction

characterized by tissue damage 24 - 48 hours after contact with an antigen (Gell and Coombs, 1968). This reaction occurs in many conditions e.g. contact dermatitis and some autoimmune diseases and transplant rejection. In such conditions it can not be considered as a defence mechanism.

(4) Regulation of the immune system:

This mechanism is mainly influenced by release of the so called lymphokines (Graddock et al., 1971).

CELLS CONCERNED IN IMMUNE RESPONSE

(1) Mononuclear cells.

(a) Lymphocytes.

(b) Macrophages.

(2) Polymorphnuclear cells

(a) Neutrophils.

(b) Basophils and mast cells.

(c) Eosinophils.

(1) Mononuclear cells:

( ) Lymphocytes:

Lymphocytes are derived from lymphoblasts, and dispensed into the blood where they represent

20 - 45% of the circulating leucocytes (Barrett, 1978). They are carried by the blood through many organs, but critical events take place in the thymus and bursa of Fabricius or its mammalian equivalent, where it appears to imprint the lymphocytes with special functions and regulate the response of the lymphocytes as either T or B lymphocytes to antigens.

Lymphocyte is the only cell in the body shown to be intrinsically capable of recognising an antigen as foreign and initiating mechanisms to get rid of the invader. It is also capable of distinguishing "self" from "non self" protein (Eisen, 1974).

1. T lymphocytes:

T lymphocytes are formed from lymphoblasts that leave the bone marrow and mature in the thymus. Maturation is accompanied by the acquisition of a specific antigen for T lymphocytes, = antigen (Adinolfi and Humphrey, 1969). T lymphocytes can be recognised by their ability to react with antitheta sera (Sela, 1975).

A simple method for enumeration of T lymphocytes is by measuring their ability to

form rosettes with sheep red cells (Cantor, 1976).

Subpopulation of T cells:

T cells can be differentiated into either helper T cells or suppressor T cells, in response to thymus dependent antigens.

a. Helper cells:

Cooperation between T and B cells has been demonstrated in the induction of antibody production (Davies, 1969, Arther, 1973).

Davies and his co-workers (1967) suggested the release of mediators from specifically stimulated T cells to act on B cells. The recognition molecule on T cells is a special type of IgM-like immunoglobulin which can mediate T/B cell cooperation in vivo and in vitro (Rajewsky, Eichman, 1977).

The T helper cells can cooperate as well by activating the alternate pathway of the complement releasing activated  $C_3$  which can act as trigger for B cells to release antibodies (Miller et al., 1971).

b. The suppressor cells:

Baker and his co-workers (1970) showed

that passively transferred T lymphocytes could suppress an anti type III pneumococcal polysacchride response.

Suppressor T cells may act via cell to cell contact (Cohen et al., 1973) or by release of soluble mediators. Kapp et al., (1976) and Tada (1976) have demonstrated the biological activity of antigen specific suppressor mediators from T cells on antibody responses in vivo.

#### T cell T cell interaction:

The sensitized T lymphocytes produce the mitogenic factor, this factor is able to attract non sensitized T lymphocytes and stimulate them to produce lymphokines thus expanding the cellular reaction (Cantor and Asfosky, 1972).

#### 2. B lymphocytes:

B lymphocytes arise from the bone marrow as lymphoblasts, arrive to the bursa of Fabricius or its mammalian equivalent, where they are imprinted with the characteristics of B cells.

The B cells acquire the B antigen on