MOULD PRECIPITINS IN BILHARZIAL HEPATOSPLENOMEGALY

Thesis

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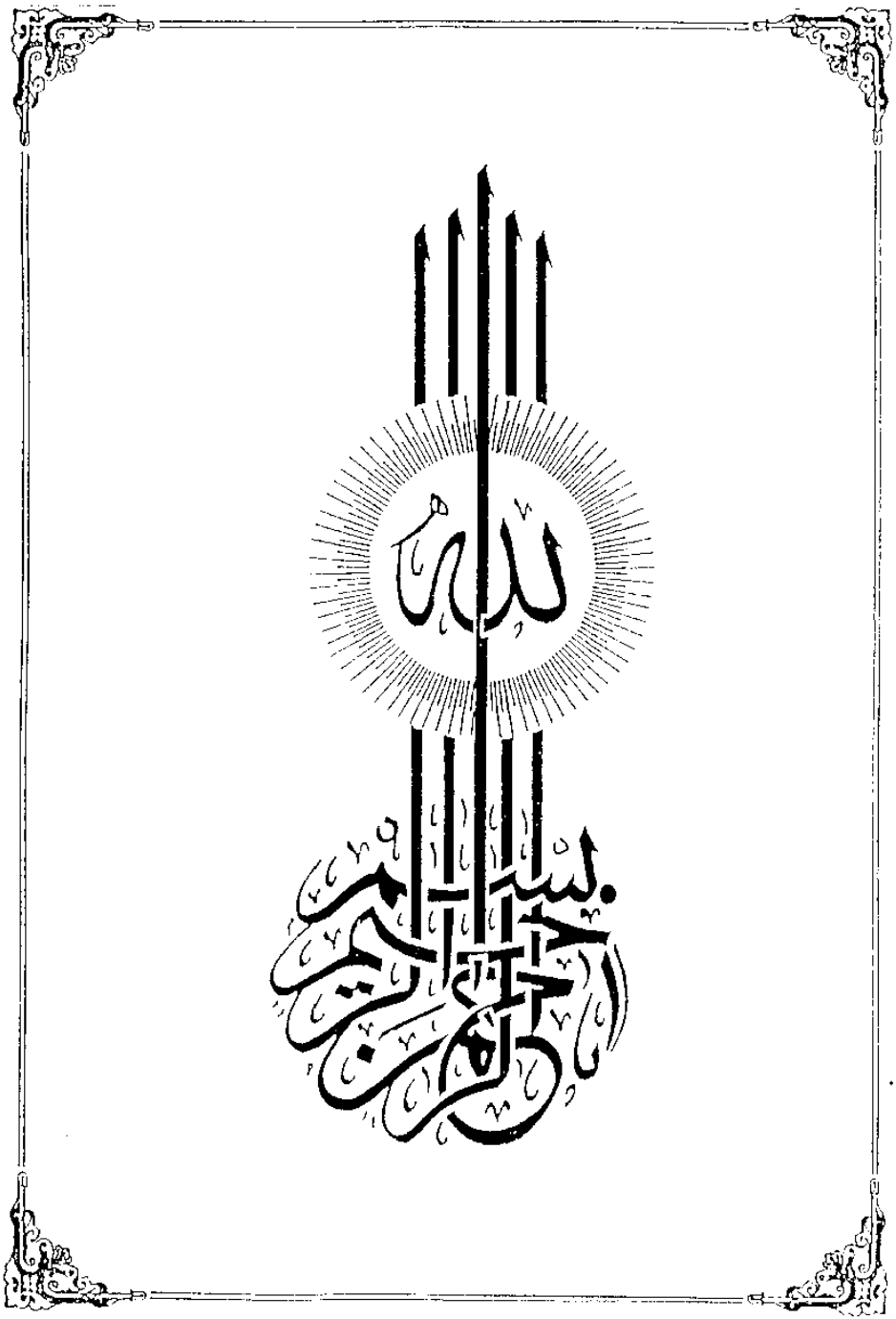
To my
Parents
Wife
Daughter



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INTRODUCTION AND AIM OF WORK

INTRODUCTION

Mycotoxins are originally produced by some strains of the mould. The metabolism of these mycotoxins are seen in some animals specially in the liver by enzymatic action of microsomal enzymes (Allcroft and Carragham, 1965).

The differences in the metabolism of the toxin are due principally to differences in enzymatic action of microsomal enzymes (Bassir and Emafo, 1981).

Aflatoxin M_1 is detected in liver extract, portal blood and some other tissues (Nesbitt et al., 1969).

In Egypt, the incidence of mixed liver disease associated wsith bilharziasis are common.

REVIEW OF LITERATURE

THE IMMUNE SYSTEM

The immune system consists of lymphocyte and monocyte and cells in the bone marrow and in lymphoid tissues, including the lymph nodes, spleen, peyer's patch and thymus. (Weir, 1985).

The prinicipal cells of immune responce is B and Tlymphocyte and cells of monocyte-macrophage lineage.

These cells and their products such as antibodies and lymphokines are responsible for the protective immunity of humans. (weir, 1985).

B LYMPHO CYTE:

Are the precursor of antibodies secreating cells, present perephral lymphoid tissue and in circulating pool of lymphocyte.

The B lymphocyte are drived from haematopiotic stem cells. These cells are found in embryonic life within the

blood islands of the yolk sac. later in gestation within the liver and postnatal life principelly within the bone marrow (Holborow, Lessof, 1981). The earliest member of B lymphocyte linage is the pre-B cell.

PRE B-CELL:

This cell lacks membrane immunoglobulin (mIg) but express in its cytoplasm one of the two constituent polypeptide chains of IgM molecules, the heavy (H) chain. As maturation progress pre B cell is associated with the apearance of mIg on the surface of the cell. (Paul, W.E., 1984).

THE IMMATURE B CELL:

This cell express mIg of IgM class and then express mIgD. This expression of mIgD in addition to mIgM are associated with new immune function such as resistance to tolerance induction and responsvness to poly saccharide Antigen (Paul, WE., 1984).

MATURE B CELL:

The majority of B lymphocyte express on the surface IgM, IgD and very few cell express surface IgG, IgA or IgE. The IgA bearing cell are present in gut.

Also mature B cells express a series of other markers that have potential function significance.

These include:-

(1) B cell have immunogloblin as their antigen receptor.

(2) Receptor for Fc portion of immunoglobulins (Fc receptor).

(3) Class II histocompetability complex (HLA-DR).these are major involved in the process by which helper Tcell recognize and interact with B cell in the course of B cell responses to antigenic stimulation.

(4) Receptor for Ebstein Barr-virus.

(5) Receptor for mouth erythracyte (ME-R).

(6) TI recptor.

(7) Receptor for complement component (R_1 for 3 b fragement of C_3 and CR_2 for 3 d fragements of C_3.

TI and ME-R are markers of immature B cells and successful in diagnosis of human lymphoprobferative disorders (Ivan Roitt, 1988b).

IMMUNOGLOBULINS:

Immunoglobulin synthesis by the B-cells is primarily a function of plasma cells, while the contribution of cells is confined to IgM. Broadly speaking Igs are involved in a number of biological process all aimed towered antigen destruction. These include complement activation antibody dependent cell mediated immunity and opsonization (Roitt, 1988).

Differentially and according to mode of action the following antibodies are recognised:antitoxins, precipitins lysins opsonins as well as aglutinizing, neuterilizing and complement fixing antibodies (Forfar, 1984).

(1) IgG:

It comprises more 80% of Igs in normal serum (1000-1500 mg%) four subclasses are recognized. It is responsible of complement activation and neutralization of toxins and viruses (Weir, 1985).It is also the main Ig responsible for cytotoxic hypersensitvity reaction, and shares with IgM in immune complex reaction. It can cross the placentar (Roitt, 1988).

Because of its relatively small molecular weight (160,000) the four classes (IgG, 65% IgG2 23%, IgG3 8% and IgG4 4%). can diffuse into the interstitial fluid (roitt, 1988).

(2) IgM:

It forms 5-10% of serum immunoglobulins. It is a macroglobulin (M.W 900.000). It biological half life is five days its serum concentration is 60-80 mg%.

IgM is the immunoglobulin produced in primary responce to antigenic challenge. It is als othe first

line of resistance against intravascular infection (Roitt, 1988).

Examples of IgM antibodies as isohaemoaglutinin to ABO blood group antigen, the rheumatoid factor, the cold agglutinins and the heterophil antibody of glandular fever.

Goodman and Wang, 1978 found that combination of IgG or IgM antibody classes with antigen stimulate the activation ofthe complement series of eleven components to produce a cascade of enzyme reaction and lead to inflammation, cell damage, coagulation and attraction of other leucocytes. The cytolysis consequent upon complement activation may useful when directed against bacterial and viruses or harmful as occurs in type II hypersensitivity reaction (Roitt, 1988).

(3) IgA:

It forms 19% of serum immunoglobulin. Its half life is six days. There are 2 forms: serum and secretory (IgA1, IgA2). The serum IgA has a concentration of 100-400 mg% and is mono, meric. Thiesecretory IgA is present

in high concentration in saliva, resperatory and gastro-intestinal secretions colustrum, tears and sweet (Weir, 1985). It neither traverse the placenta, nor fix the complement, but it can activate c3 by alternative pathways (Forfar, 1984).

IgE:

It constitutes about 0.5% antibodies. It has a low concentration in the serum 0.01 mg% = 10-130 ug with short half life 2.5 days (Weir, 1985). It does not cross the placenta and does not fix the complement, and have a special afinity to bind blood eosinophils and tissue mast cells. It is the antibody responsible for the immediate hypersensitivity. Interaction between antigens and IgE bound to the surface of mast cell products (chemical mediators) such as histamine, serotonin, kinins and slow reacting substances which are responsible for allergic and anaphylactic reaction (Malcom, 1977). The level of IgE is cheracteristically elevated in parasitic infections. Stiehm (1980), has mentioned that children in developing counteries although having high levls of IgE (due to parastism), have a low incidence of allergy. This