# ANTIBODY LEVEL AFTER MEASLES VACCINATION

THESIS

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By

**Maha M. Fathy M. Amin**

M.B , B.Ch

Under supervision of

**Prof . Dr . Medhat A . Darwish**

Prof of Microbiology and Immunology

Faculty Of Medicine

Ain Shams University

**Dr. Taghreed Hamed T. EL Khashaab**

Lecturer of Microbiology and Immunology

Faculty of Medicine

Ain Shams University.

Faculty of Medicine

Ain Shams University

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( وَقُلْ رَبِّ زِدْنِي عِلْمًا )

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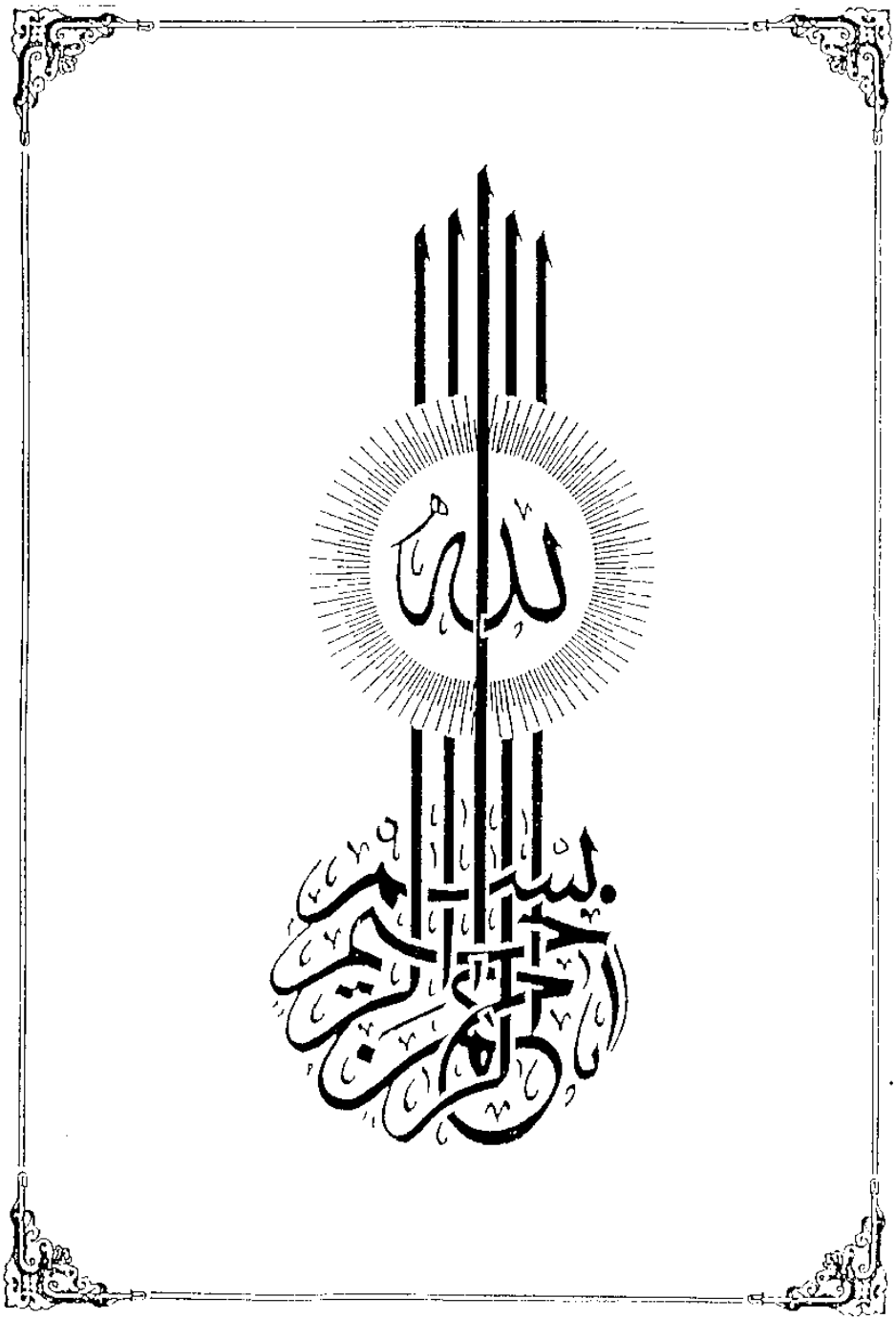

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# I Introduction

# INTRODUCTION

Measles is a highly contagious world wide acute viral disease. The clinical spectrum of measles ranges from a mild self limiting illness to a fatal disease. More than one million children a year die from acute measles. In addition delayed mortality as a result of measles infection is now being realized (Aaby and Clements, 1989).

Infants acquire immunity transplacentally (virus neutralizing IgG antibodies) from mothers who had measles. This immunity usually lasts for the first four to six months of life and disappears at a varying rate. Several attempts to discover safe and effective means of inducing active immunization against measles have been done including both inactivated and live vaccines. The use of live attenuated vaccines began in 1963 in United States and has extended to many parts of the world.

Despite the marked decline in the incidence of measles since the introduction of the live virus vaccine, outbreaks continue to occur among both its recepients and non recepients leading to questions about the efficacy of measles vaccine (McCormick et al., 1977).

# II Aim of The Work

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Measles vaccine was licensed in 1963. Despite the availability and wide spread use of measles vaccine, epidemic measles is still occurring. The failure to eradicate measles is a matter of major concern and has been the subject of considerable studies and analysis. In Egypt infants are immunized against measles at the age of nine months according to the Expanded Programme of Immunization of WHO. Despite the use of measles vaccine 20 years ago, measles disease continues to occur.

The present study was designed to estimate the level of measles IgG antibody in infants early after vaccination and in pre-school children to determine their immune status before joining school. For this purpose measles IgG antibody was measured in three groups as follows: a group of non vaccinated infants, a group of recently vaccinated ones and a group of vaccinated pre-school children.

# III Review of Literature

## A. *HISTORY OF MEASLES*

There is some doubt about the origin of the name measles. Most probably it comes from the latin term "misellus" or "misella" itself a diminutive of the latin miser meaning miserable. It was used in this way for the suffer from various skin eruptions and sores by Langland in the 14th century and also later on by Shakespear. The anglicized form of misellus namely measles became applied not to the suffer of illdefined skin lesions but to the specific disease morbilli (measles) (Wilson, 1962).

Morbilli is a diminutive of morbus (a disease) and the term morbilli was used to distinguish measles from another disease which closely resembles measles named "il morbo" or the plague (Christie, 1987).

Measles appears to be a relatively new disease of humans. The first written description of measles is attributed to Abu Becr, a 10th century persian physician also known as Rhazes. He reffered to measles as "Hasbah", which means eruption in Arabic and distinguished it from smallpox but he did not consider measles infectious. Thomas Sydenham's remarkable description of an outbreak of measles in London in (1670) provided an accurate clinical picture of the disease. Peter Panum who was sent to assist with an epidemic of measles in the Faroe Islands in (1846) confirmed that measles was contagious and transmitted directly from person to person. He defined the 14-day incubation period between exposure and the appearance of the rash, and he demonstrated that patients were most infectious at the end of the prodrome when the rash was just breaking out. He also observed an attack rate of almost 100%, documented increased mortality in children under one year of age and in adults over 50 years demonstrated the efficacy of quarantine and

showed that infection conferred life long immunity. In (1883). Hirsch described the devastating impact of measles on virgin populations in the Fiji Islands and Amazon Basin, recording mortalities in excess of 20%. Transmission of measles to monkeys was first reported by Josias in (1898). Anderson and Goldberger repeated these experiments in (1911) and confirmed the viral etiology of measles. Several workers succeeded in propagating measles virus on the chorioallantoic membrane of the developing chicken embryo and in minced chicken embryo tissue culture. In 1954. Enders and Peebles reported the isolation of measles virus in rollertube cultures of human and rhesus monkey kidney cells and described the characteristic cytopathic effects (CPE) that accompanied measles virus replication. thus provided a basis for virus isolation procedures, for infectivity assays and for the measurement of neutralizing antibody. This discovery which was quickly followed by the adaptation of measles virus to growth in a variety of cell cultures permitted the detailed analysis of virus structure, virus replication and virus cell interactions and led directly to the development of effective measles vaccines (Norrby and Oxman, 1990).

## B. *MEASLES VIRUS*

### 1. *Classification:*

Measles virus is a member of the genus Morbilli virus in the family Paramyxoviridae (Kingsbury et al., 1978).

The Morbilli virus genus includes three other well defined members which do not infect man: canine distemper virus affecting dogs and other canines, rinderpest virus affecting all artiodactyls and peste des petits ruminants virus affecting small ruminants. Recently a serious distemper like disease outbreaks among European seals were demonstrated to be caused by a newly recognized member of this genus that proved to be closely related to canine distemper virus: phocid distemper virus (Osterhaus and Vedder, 1988). Another morbilli virus was isolated from Siberian seals with similar symptoms and this virus also proved to be closely related to canine distemper virus (Osterhaus et al., 1989).

Measles, canine distemper and rinderpest viruses show close immunological relationships which involve to a varying extent all of the structural proteins (Norrby et al., 1986). Each one of these three viruses is an antigenically stable monotypic virus as determined by neutralization tests with polyclonal sera. However minor epitopic variations between strains have been demonstrated with measles and distemper (Sato et al., 1985).

Morbilli viruses are distinct from the paramyxoviruses in that they do not have any detectable neuraminidase activity and interact with cellular receptors which are insensitive to neuraminidase treatment. In addition they cause the formation of

intranuclear inclusion bodies as an important part of their cytopathology (Norrby and Oxman, 1990).

2. ***Morphology and structural components***

Like other paramyxoviruses, measles virions are spherical, enveloped particles with a centrally located helical nucleocapsid (Norrby, 1964). (Fig. (1))

The diameter of the pleomorphic particles varies between 100 and 250nm with a mean value of about 150nm. The viral genome is a linear single strand RNA of negative polarity with an estimated MW of 4.5 x $10^6$ daltons (Lund et al., 1984).

Thera are six virus specific proteins participating in the formation of virions, three of these proteins occur in the envelope and the other three are associated with the internal structures (Rima, 1983).

The envelope contains a bimolecular lipid layer of cellular origin with one of the virus envelope proteins, the matrix (M) protein located on its inside. The other two components are transmembranous proteins forming nine to 15 nm long radial projections (peplomers) (Varsanyi et al., 1984).

There are two different types of peplomers which when isolated have distinct morphologies. The hemagglutinin (H) peplomers appear conical. They are oligomer of a protein with a capacity to anchore the virus particles to specific receptors on cells. Since similar receptors occur also on erythrocytes of Old World monkeys, these cells are agglutinated by measles virions or envelope fragments, hence the term hemagglutinin (H) for this protein (Norrby, 1988).