

TAMIN-A STATUS IN EGYPTIAN CHILDREN DURING MEASLES

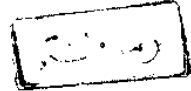
Thesis

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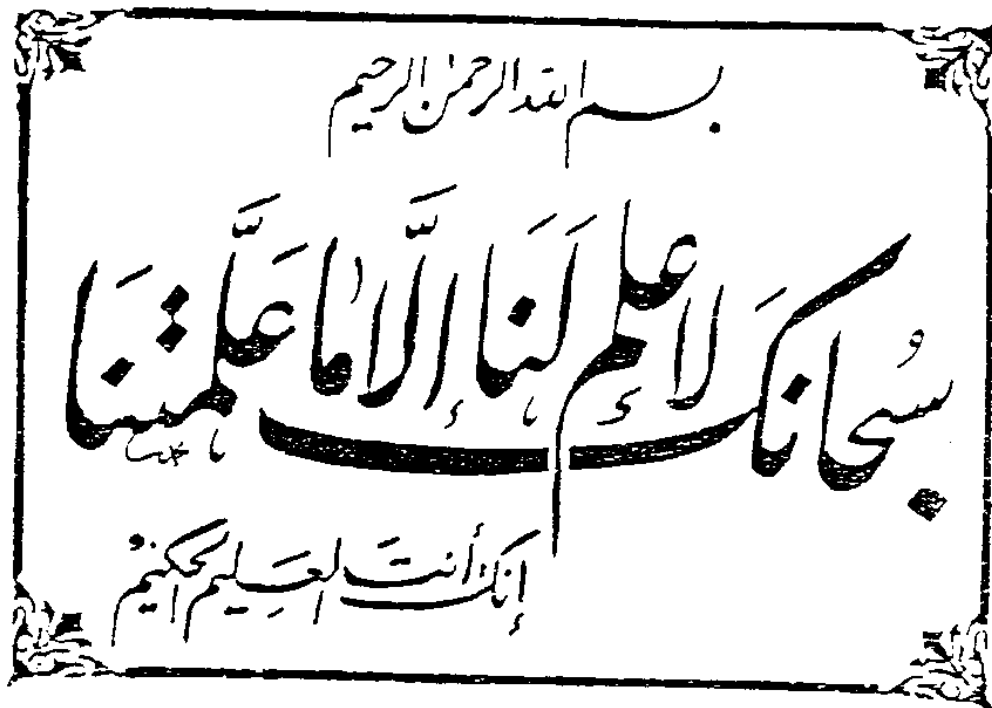
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LIST OF ABBREVIATIONS

	= Albumin
	= Complement fixation
A.B.P	= Cellular retinoic acid binding protein
.B.P	= Cellular retinol binding protein
ISA	= Enzyme linked immunosorbent assay
.A.	= Fluorescent antibody.
H.A.I	= Haemoagglutination inhibition
HLA	= Human Leucocyte Antigen
Ig	= Immunoglobulin
MMR	= Measles-Mumps-Rubella
RBP	= Retinol binding protein
RDA	= Recommended daily allowance
RNA	= Ribonucleic acid
Vit	= Vitamin
WHO	= World Health Organisation.



Introduction



INTRODUCTION

Measles is responsible for 1.5-2 million deaths per year in third world countries, accounting for half of deaths in these areas (*Arrieta et al., 1992*).

Measles remains a devastating disease for which specific therapy is lacking. Hopes for its control and eventual eradication rest in immunization but measles still kills about 2 million children each year and cripples an untold number through blindness and lung disease (*Hussey and Klein, 1990*).

It was found that measles virus infection reduces vitamin A levels, and low vitamin A in turn exacerbates measles severity possibly by decreasing antibody production (*Frieden et al., 1992*).

Hyporetinemia (serum retinol below 0.7 μ mol per liter) is associated with increased mortality from the disease particularly in children under two years of age (*Hussey and Klein, 1990*).

Children with low vitamin A are more likely to have low measles specific antibody titers. Similarly children with low measles specific antibody titers have lower vitamin A levels than children with high measles specific antibody titers (*Frieden et al., 1992*).

In almost every known infectious disease vitamin A deficiency is known to result in greater frequency, severity or mortality. Increased susceptibility to infection was one of the first features of nutritional vitamin A deficiency to be recognized and even mild deficiency appears to be associated with increased risk of pneumonia, diarrhea and death in childhood. No nutritional deficiency in the animal kingdom is more consistently synergistic with infection than that of vitamin A (*Hussey and Klein 1990*).

Abnormalities in systemic immune competence associated with vitamin A deficiency may contribute to mortality. At least in animals vitamin A deficiency interferes with humoral and especially with cell mediated immunity. Limited data suggest similar effects in man (*Sommer et al., 1986*).

High doses of vitamin A given to otherwise normal animals have been reported to produce a non specific adjuvant like increase in resistance to infection (*Sommer et al., 1986*).

The world health organization recommended vitamin A therapy for children with measles who live in communities with known vitamin A deficiency and in communities where the measles case fatality rate is 1% or greater. Recent studies suggest that this recommendation may need to be extended to all children younger than two years with severe measles (*Frieden et al, 1992*).



Aim of the work

AIM OF THE WORK

This study will be conducted to measure the levels of vitamin A during acute measles infection and to correlate these levels with the severity and complications of measles in Egyptian children.

The relationship between vitamin A levels and the levels of measles specific antibody will be analyzed. Vitamin A levels will also be measured in other acute infectious diseases, to determine whether vitamin A levels are influenced by the acute infectious process per se or is related to specific etiologic agents.



Review of Literature

VITAMINS

Vitamins are organic compounds required in trace amounts for health, growth and reproduction. vitamins are natural materials that can be isolated from animal or vegetable sources or that can be chemically synthesized (*MacCormick, 1987*).

Only small amounts of vitamins are required for the functional catalytic coenzymatic role they serve (*MacCormick, 1987*).

This is contrasted to the relatively large amounts of such macronutrients as proteins, lipids and carbohydrates (CHO) which constitute the bulk of the diet and which serve primarily as sources for energy (*MacCormick, 1987*).

Forms of vitamin A:

A1, A2, and B-carotene

R-CH₂ OH for retinol

R-CHO for retinal

R-CO₂ H for retinoic acid (*MacCormick, 1987*).(Fig.1)

Fig.1: Pro vitamin A: A1, A2 and B-Carotene (*MacCormick, 1987*):

