PHAGOCYTIC FUNCTIONS CORRELATED WITH VITAMIN D LEVEL IN TUBERCULOSIS IN EGYPTIAN CHILDREN

THESIS

Submitted for the partial fulfilment of the
M.D. Degree in Pediatrics

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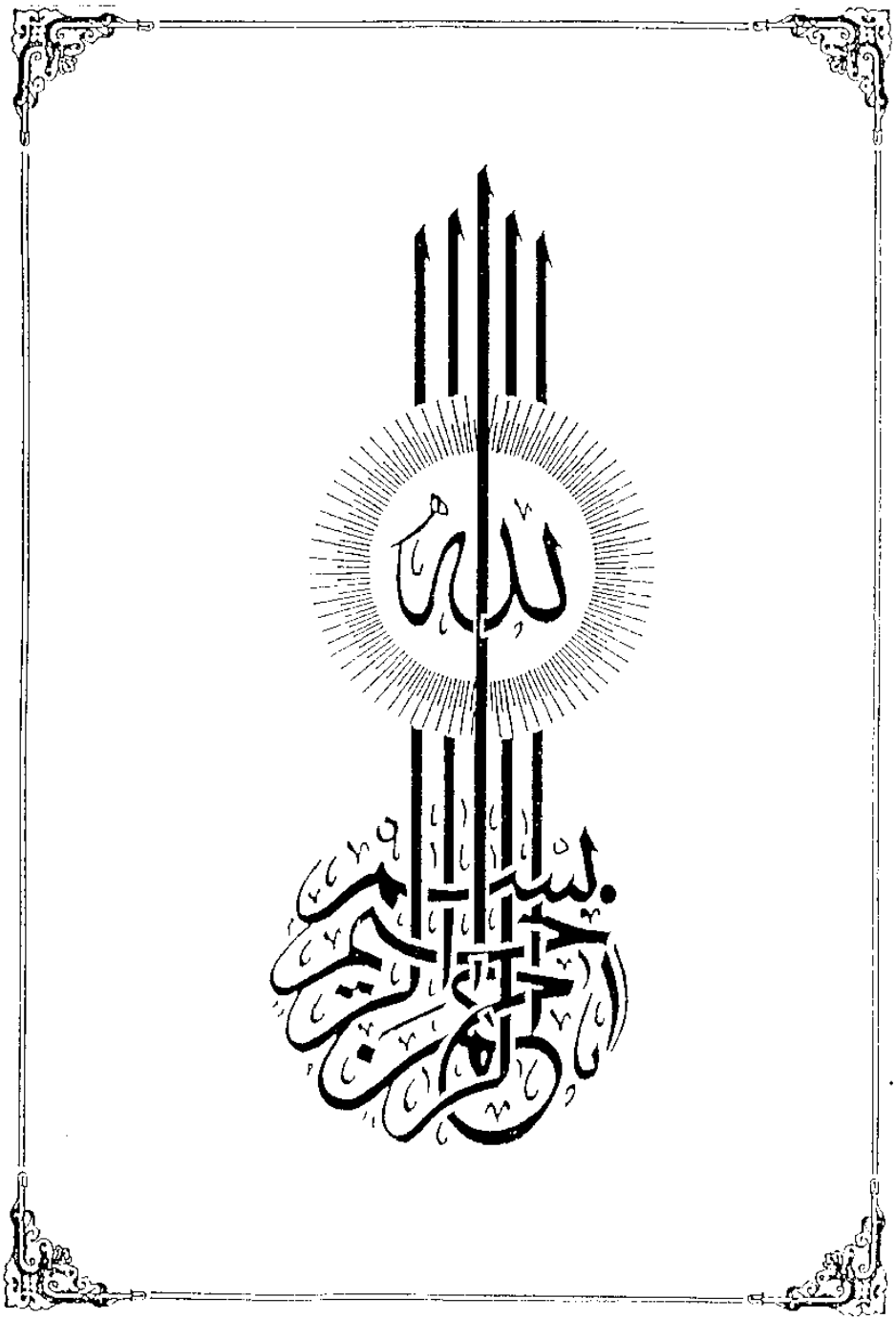
بسم الله الرحمن الرحيم

« وعلمك ما لم تكن تعلم

وكان فضل الله عليك عظيما »

صدق الله العظيم

(سورة النساء آية ١١٣)



ACKNOWLEDGEMENT

I am greatly indebted to ***Prof. Dr. Saadia Mohamed Abd El-Fattah****, Professor of Pediatrics, Ain Shams University, for this precious opportunity of working under her supervision. Working with her is all at once both pleasurable and educational. Her kind help, sound advice and guidance were indispensable for accomplishing this work.*

I am also indebted to ***Prof. Dr. Karima Ahmed Abd El-Khalik****, Professor of Pediatrics, Ain Shams University, for her valuable guidance, kind supervision and motivation to excel. I will always remember her prolific supervision and constructive criticism throughout this work.*

Very special thanks go to ***Prof. Dr. Laila Abd El-Aala El Shawarby****, Professor of Clinical Pathology, Ain Shams University. Her exemplary perseverance and meticulousness were crucial for the practical part of this work.*

I wish to express my special thanks to ***Dr. Zeinab Anwer El-Kabbany****, Assistant Professor of Pediatrics, Ain Shams University, for her kindness and generous help. I'll always cherish her care and advice in every step of this work.*

I am also grateful to ***Dr. Mona Mohamed El-Tobgui****, Assistant Professor at Child Health Laboratory, National Research Center, who carried the burden of the practical part of this work. Her valuable guidance and advice were essential for accomplishing this work.*

I would also like to thank ***Dr. Nahla Zakaria****, Lecturer of Clinical Pathology, Ain Shams University, who helped me much in the practical part of this work. Her enthusiasm and ardor are truly matchless.*

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LIST OF ABBREVIATIONS

AD	Adenosine deaminase
AIDs	Acquired immune deficiency syndrome
ALP	Alkaline phosphatase
ALT	Alanine aminotransferase
AST	Aspartate aminotransferase
B.C.	Before Christmas
BCG	Bacille Calamette Guerin
C	Complement
Ca^{2+}	Calcium
CD	Cluster differentiation
CR	Complement receptor
CSF-1	Colony stimulating factor-1
DTH	Delayed type hypersensitivity
ELISA	Enzyme linked immunosorbent assay
ESR	Erythrocyte sedimentation rate
Hb	Hemoglobin
HBBS	Hank's balanced salt solution
HIV	Human immune deficiency virus
HLA	Human leukocytic antigen
HPLC	High performance liquid chromatography
IFN-γ	Interferon gamma
IgG	Immunoglobulin-G
IL-1	Interleukin-1
INH	Isoniazid
IUATLD	International Union Against Tuberculosis and Lung Diseases
LPS	Endotoxins

LIS

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patients and the

M. tuberculosis	Mycobacterium tuberculosis
MNC	Mononuclear layer
NK	Natural killer
NO	Nitric oxide
OT	Old tuberculin
PABA	Para-amino benzoic acid
PASA	Para-amino salicylic acid
PCR	Polymerase chain reaction
PDD	Purified protein derivative
Phos	Phosphorous
PTH	Parathyroid thyroid hormone
RNI	Reactive nitrogen intermediate
T. bil	Total bilirubin
TB	Tuberculosis
TCR	T-cell receptor
TNF-α	Tumor necrosis factor-alpha
TU	Tuberculin unit
VDR	Vitamin D receptor
WHO	World Health Organization
$1,25(OH)_2D_3$	1,25-dihydroxy cholecalciferol
<	Less than
>	More than

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Introduction

INTRODUCTION

Despite the existence of a vaccine and highly effective chemotherapy, tuberculosis today remains a major global health problem. The World Health Organization (WHO) calculates probably 8-10 million new cases a year in the world, and 3 million deaths (*Crofton et al.*, 1992). One reason for the slow progress towards tuberculosis control has been the inadequacy of our understanding of the immune mechanisms that function in tuberculosis (*Rook*, 1987).

There is growing evidence that the immune responses to tuberculosis are regulated by various extrinsic and intrinsic immune modulators. Among the important extrinsic immune modulators is host resistance. Several conditions associated with reduced cellular immunity predispose to tuberculosis. Low vitamin D levels may be associated with unusual susceptibility to tuberculosis (*Onwubalili*, 1990).

Vitamin D therapy cured skin tuberculosis in the pre-chemotherapy era (*Dowling and Prosser Thomas*, 1946).

There is evidence to suggest that vitamin D influences monocyte maturation and increases macrophage capacity for oxygen reduction by interferon activated macrophages, thus increases their inhibition of mycobacterium tuberculosis (*Rook et al.,* 1986). It has been found that exposure of macrophages to 1,25-dihydroxy cholecalciferol renders them more liable to release tissue damaging macrophage products including tumor necrosis factor (TNF) upon exposure to live mycobacterium tuberculosis. TNF together with interleukin-1 may account for fever and weight loss that characterize tuberculosis (*Rook et al.,* 1987).