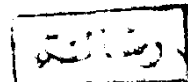


**INCIDENCE OF TOXOPLASMA AND  
TOXOCARA ANTIBODIES AMONG  
OUTPATIENTS IN THE OPHTHALMIC  
RESEARCH INSTITUTE**

**Thesis**

Submitted for Partial Fulfilment of  
The Master Degree in Parasitology



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بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ



## Acknowledgment

I would like to express my thanks and gratitude to Professor Dr. **Tosson. A. Morsy**, Head of Medical Parasitology Department, Faculty of Medicine, Ain-Shams University for his great help and support.

I'm really indebted to Professor Dr. **Fathy M. Abd El-Ghaffar**, Professor of Medical Parasitology, Faculty of Medicine Ain-Shams University who planned and supervised all the steps of this work for his help, generous supervision, continuous support and honest guidance.

I wish to express my deepest thanks and gratitude to Professor Dr. **Elmeya H. Safar**, Professor of Medical Parasitology and Head of Parasitology and Microbiology Department, Research Institute of Ophthalmology, who planned and supervised all the steps of this work for her generous help, kind support and sincere supervision.

My deepest appreciation and gratitude to Professor Dr. **Salwa A. Soffar**, Professor of Medical Parasitology, Faculty of Medicine, Ain-Shams University, who supervised all the steps of this work for her sincere devotion, continuous help and support.

I would like to express my deepest gratitude to Dr. **Karam M. Mokled**, Lecturer of Medical Parasitology, Faculty of Medicine, Ain-Shams University. I'm greatly indebted to her for all her help, continuous support and valuable advice.

I'm greatly thankful for Dr. **Khalid S. Habeeb**, Lecturer of Medical Parasitology, Faculty of Medicine, Ain-Shams University for his kind help and advice

I would like to express my thanks to **Dr. Rawia El Abiad** Research worker, Ophthalmic Research Institute who carried out the ophthalmoscopical examination throughout this study for her kind help and support.

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## List of Abbreviations

C <sub>3</sub>	Complement 3
CIEP	Counter current immuno electro phoresis
ELISA	Enzyme linked Immuno Sorbent Assay
FC	Fragment Crystallizable
IDT	Intra-Dermal test
IFAT	Indirect Immuno fluorescent antibody test
IFN - $\gamma$	Interferon-gamma
IgE	Immunoglobulin E
IgG	Immunoglobulin G
IgM	Immunoglobulin M
IHAT	Indirect haemagglutination test
IL-2	Interleukin 2
IU	International unit
L-2	2 <sup>nd</sup> stage larva
LA	Latex agglutination test
LAK	Lymphokine activated killer cells
ml	millilitre
$\mu$ l	microlitre
MRC5	Human embryonic fibroblast cell line
NK	Natural Killer cells
OLM	Ocular larva migrans
PAT	Percipitin absorption test
PBS	phosphate buffer saline
rpm	revolution per minute
<i>T.canis</i>	<i>Toxocara canis</i>
TES	<i>Toxocara</i> excretory-secretory antigens
<i>T.gondii</i>	<i>Toxoplasma gondii</i>
VLM	Visceral larva migrans
WB	Western-Blott
WHO	World Health Organization

# *Introduction*

## Introduction

Previous serological surveys in Egypt had indicated that the frequency of *Toxoplasma* antibodies in different groups of Egyptian population was 16.8% among inhabitants of some governorates (*Rifaat et al.*, 1975) , 25% in human females in the reproductive period (*Fikry et al.*, 1980) and 64% among school children in Manshiat El-Bakary School in Giza (*El-Gammal et al.*, 1992) .

*Toxoplasma gondii* infection is either congenitally transmitted or acquired (*Beaver et al.*, 1984). The former may not manifest in the newly born, but later on (*Remington & Desmonts*, 1989), while the latter is frequently subclinical and possibly persisting for the life of the host (*Warren and Mahmoud*, 1984).

Congenital infection presents mainly either with central nervous system manifestations in the form of convulsions, hydrocephalus (*Martinovic et al.*, 1982), mental retardation (*Wilson et al.*, 1980). Or, more commonly, with ocular affection mainly as retinochoroiditis (*Harvas et al.*, 1987 and *Rao et al.*, 1992).

Early diagnosis and treatment of ocular affection may prevent hazardous complications, which can take place, such as cataract, nystagmus, microphthalmos, and even blindness (*Remington & Desmonts*, 1989).

Regarding detection of *Toxocara* antibodies in apparently healthy Egyptian control groups, *Bebars* (1979); *Nasr* (1987) and

*Safar et al. (1990)* recorded an incidence of 2.5%, 10% and 0% respectively. However, clinical reports about toxocariasis are still few (*Schantz, 1989*), because *Toxocara* infection is frequently asymptomatic unless it affects a vital organ (*Beaver et al., 1984* and *El Ganayni, 1990*). It manifests either as visceral larva migrans (V.L.M.) or as ocular larva migrans (O.L.M.) (*Barriga, 1988*). Retinal granuloma, or chronic endophthalmitis are the presenting forms of ocular larva migrans (*Molk, 1983* and *Beaver et al., 1984*).

It is evident that both *Toxoplasma* and *Toxocara* infections share common pathogenicity in the eye, causing confusing lesions in the retina (*Azab et al., 1990*). So, the aim of the present work is to detect the incidence of both *Toxoplasma gondii* and *Toxocara canis* antibodies among outpatients attending the Ophthalmic Research Institute's clinics.

## *Review of literature*

## **Toxoplasma gondii**

### **Historical Review**

The history of *Toxoplasma* began when *Nicolle and Manceaux (1908)*, observed a parasite in mononuclear cells of the spleen and liver of a North African rodent, the gundi (*Ctenodactylus gundi*); which so closely resembled *Leishmania*, this is why it was originally named *Leishmania gondii*. *Janku (1923)* in Prague described the recognized case of toxoplasmosis in human. However, the recognition of toxoplasmosis as a disease entity in human with rare impact on medicine, was not valid until *Wolf et al., (1939)* reported a fatal case of encephalomyelitis which was due to toxoplasmosis.

In recent years, most physicians recognized the important position of *Toxoplasma* among pathogens of humans and animals. The organism is responsible for some diseases that were previously classified to be due to unknown causes. (*Remington & Desmonts, 1989*).

## **Taxonomy:**

According to (*Beaver et al., 1984*).

phylum	Apicomplexa
Class	Sporozoea
Subclass	Coccidia
Order	Eucoccidiida
Suborder	Eimeriariina
Family	Sarcocystidae
Genus	Toxoplasma
Species	gondii      which is the only species of this genus

**Morphology:** *Anderson and Remington (1975)* identified three forms of the organism.

### ***a) Tachyzoites*** (Trophozoites):

The trophozoite is the proliferative form of *Toxoplasma gondii*.

It is crescentic or banana-shaped with a pointed anterior pole. It is 2-4  $\mu\text{m}$  in width and 4-8  $\mu\text{m}$  in length. The nucleus is composed of loose chromatin granules surrounded with a definite membrane. It may be rounded or oval, with big nucleolus and a karyosome. It lies just below the middle of the organism. There are no flagella, cilia or pseudopodia.