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# INTERLEUKIN-2 IN SERUM OF CHILDREN WITH NEPHROTIC SYNDROME

## Thesis

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بسم الله الرحمن الرحيم

# ﴿وقل رب زدني علماً﴾

”صدّق الله العظيم“

(سورة طه آيه رقم (١٤))



**TO...**  
*Our Beloved, Great Prophet*  
**MOHAMED**  
**Who learned us and learned all the  
world .. how to be human beings**

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## ABBREVIATIONS

<b>AIDS</b>	Acquired immunodeficiency syndrome
<b>ARS</b>	AIDS-related complex
<b><math>\alpha</math>2P</b>	$\alpha$ -2-plasma inhibitor
<b>ATIII</b>	Antithrombin III
<b>CGN</b>	Chronic glomerulonephritis
<b>C3</b>	Complement 3
<b>C3NEF</b>	Complement 3 nephritic factor
<b>Con A</b>	Concanavalin A
<b>cAMP</b>	Cyclic adenine monophosphate
<b>cGMP</b>	Cyclic guanosine monophosphate
<b>CBC</b>	Complete blood count
<b>FSGS</b>	Focal sclerosing glomerulopathy
<b>GBM</b>	Glomerular basement membrane
<b>Anti-GBM</b>	Anti-glomerular basement membrane
<b>GFR</b>	Glomerular filtration rate
<b>G-CSF</b>	Granulocyte colony stimulating factor
<b>GM-CSF</b>	Granulocyte-macrophage colony stimulating factor
<b>GTPase</b>	Guanosine triphosphatase
<b>HIV</b>	Human immunodeficiency virus
<b>IL-1</b>	Interleukin-1
<b>IL-2</b>	Interleukin-2
<b>IgG</b>	Immunoglobulin G
<b>IFN<math>\alpha</math> &amp; B1</b>	Interferon $\alpha$ and interferon B1
<b>IFN-<math>\delta</math></b>	Interferon- $\delta$
<b>ISKDC</b>	International study of kidney disease in children
<b>LGL</b>	Large granular lymphocyte
<b>LPS</b>	Lipopolysaccharide
<b>LDL</b>	Low density lipoproteins
<b>LAK</b>	Lymphokine activated killer cells
<b>LT &amp; TNF</b>	Lymphotoxine and tumour necrosis factor

<b>M-CSF</b>	Macrophage colony stimulating factor
<b>Mag-GAR</b>	Magnetic goat anti-rabbit
<b>MHC</b>	Major histocompatibility complex
<b>MGN</b>	Membranoglomerulonephritis
<b>MPG</b>	Membranoproliferative glomerulonephritis
<b>MCNS</b>	Minimal change nephrotic syndrome
<b>MIF</b>	Migration inhibitory factor
<b>NK</b>	Natural killer cells
<b>NS</b>	Nephrotic syndrome
<b>PBL</b>	Peripheral blood lymphocytes
<b>PGE2</b>	Prostaglandin E2
<b>PHA</b>	Phytohemagglutinin A
<b>RIL-2</b>	Recombinant IL-2
<b>RIA</b>	Radioimmune assay
<b>EDTA</b>	Sodium ethylene diamine tetra-acetate
<b>SDS</b>	Sodium docyl sulphate
<b>SD</b>	Standard deviation
<b>T4</b>	Thyroxine
<b>TSH</b>	Thyroxine stimulating hormone
<b>TGFB</b>	Transforming growth factor B
<b>25, OH D2</b>	25, hydroxy chole claciferol
<b>VDBG</b>	Vitamin D binding globulin
<b>VPF</b>	Vascular permeability factor

# INTRODUCTION

## INTRODUCTION

Although the etiology of minimal change nephrotic syndrome (MCNS) is not yet established, an immune pathogenesis is suspected. *Shalhoub (1974)* postulated that patients with MCNS had two underlying effects: primarily an abnormality in T-cell regulation resulting in uncontrolled proliferation of a T-cell clone or T-cell subclass; and secondly an increase in circulating levels of a thymic hormone or lymphokine capable of alternating glomerular permeability to proteins.

Interleukin-2 is a critical component of the immune response of T-lymphocytes and is an essential lymphokine for the clonal expansion and maturation of antigen triggered T-cell activity (*Welter and Mertelsmann, 1985*). Decreased Interleukin-2 (IL-2) production has been observed in primary immunodeficiencies (*Flomenberg et al., 1983*) and in autoimmune diseases (*Linker-Israeli et al., 1983*).

*Hinoshita et al., (1990)* studied in vitro the IL-2 production of T-cells when stimulated with autologous non T-cells and found that IL-2 was significantly decreased in patients with MCNS, regardless the stage of the disease. They postulated possible primary interleukin-2 defect in MCNS rather than a secondary phenomenon.

## **AIM OF THE WORK**

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The present study aims at estimation of serum level of IL-2 in children with nephrotic syndrome, and determination of its possible correlation with the disease stage as well as the mode of therapy, possible familial changes of IL-2 will be also studied.