THE ROLE OF IMMUNOREGULATORY CELLS IN HEALTHY AND SICK AFRICAN CHILDREN

BY

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PREFACE

The experimental work described in this thesis was carried out in the Department of Paediatrics and Child Health, University of Natal, Durban, from June, 1982 to June, 1987 under the supervision of Professor H.M. Coovadia.

These studies represent original work by the author and have not been submitted in any form to another University. Where use was made of the work of others it has been duly acknowledged in the text.

Selected results from this thesis have been published in scientific journals. Research workers who were closely associated in these studies are co-authors in these publications.

PAPERS

1. LORTAN, J.E., KIEPIELA, P., COOVADIA, H.M., SEEDAT, Y.K. (1982) Suppressor cells assayed by numerical and functional tests in chronic renal failure. Kidney International 22 : 192-197.

2. KIEPIELA, P., COOVADIA, H.M., COWARD, P. (1987) T helper cell defect related to severity in measles. Scand J Infect Dis 19 : 185-192.

3. KIEPIELA, P., COOVADIA, H.M., COWARD, P., WOODHEAD, R., BECKER, P.J. (1987) Age-related lymphocyte subpopulation changes among healthy Africans from birth to adulthood. Ann Trop Paediatr (accepted for publication).

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5. KIEPIELA, P., MICHIE, C.A., COOVADIA, H.M., COWARD, P., WOODHEAD, R., COOPPAN, R.M., BECKER, P.J. Immunoregulatory changes in single and combined, mild to moderate schistosomal infections in African children. 1. Numerical assays of T cell subsets in peripheral blood and rectal granuloma (submitted for publication).

6. KIEPIELA, P., COOVADIA, H.M., COWARD, P., WOODHEAD, R., COOPPAN, R.M., BECKER, P.J. Immunoregulatory changes in single and combined, mild to moderate schistosomal infections in African children. 2. Antigen and mitogen induced suppressor and proliferative cells in peripheral blood (submitted for publication).

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CONTENTS - VOLUME 2

LIST OF ABBREVIATIONS

ABA	-	Azobenzenearsonate
ADCC	-	Antibody dependent cellular cytotoxicity
AEF	-	Allogenic effect factor
Ag	-	Aantigen
AICC	-	Antibody independent cellular cytotoxicity
AIS	-	Antigen induced suppression
ALS	-	Anti-lymphocyte serum
BCDF	-	B cell differentiation factor
BCGF	-	B cell growth factor
BEF	-	B cell derived enhancing factor
BF	-	Blastogenic factor
C	-	Constant
CC	-	Community control
CIC	-	Circulating immune complexes
CMV	-	Cytomegalovirus
ConA	-	Concanavalin A
cpm	-	counts per minute
CRF	-	Chronic Renal Failure
CSF	-	Colony stimulating factor
CSF	-	Cerebrospinal fluid
CTL	-	Cytotoxic T lymphocytes
DMD	-	Duchenne Muscular Dystrophy
DNFB	-	Dinitro fluorobenzene
DNP	-	Dinitro phenyl
dpm	-	disintergrations per minute
DTH	-	Delayed type hypersensitivity reaction
EBV	-	Epstein Barr Virus
EDTA	-	Ethylene diamino tetraacetic acid
FACS	-	Fluorescent activator cell sorter
GAT	-	Glutamic acid-alanine-tyrosine
GA	-	Glutamic acid-alanine
GBM	-	Glomerular Basement membrane
GT	-	Glutamic acid-tyrosine
HBV	-	Hepatitis B virus
HC	-	Hospital control

HEL - Chicken egg white lysozyme
HRBC - Horse red blood cells
HSF - Histamine induced suppressor factor
IBF - Immunoglobulin binding factor
IC - Indian control
IDS - Inhibitor of DNA synthesis
IFNγ - γ -interferon
Ig - Immunoglobulin
IghV - Immunoglobulin variable heavy chain
IL-1 - Interleukin 1
IL-2 - Interleukin 2
IL-B - Interleukin B
Ir - Immune response gene
IRSF - Immune response suppressor factor
KLH - Keyhole limpet haemocyanin
LAK - Lymphocyte activated killer cell
LCM - Lymphocytic choriomeningitis virus
LIF - Leucocyte inhibitory factor
LFA-1 - Lymphocyte function associated antigen
LPS - Lipopolysaccharide
MBSA - Methylated bovine serum albumin
MCNS - Minimal change nephrotic syndrome
MHC - Major histocompatibility complex
MIF - Migration inhibitory factor
MLC - Mixed leukocyte cultures
MNC - Mononuclear cells
MRNA - Messenger ribonucleic acid
MS - Multiple sclerosis
MVA - Measles virus antigen
MW - Molecular weight
$NaIO_4$ - Sodium periodate
NK - Natural killer cell
NP - 4-hydroxy-3-nitrophenyl acetyl
NS - Nephrotic Syndrome
OVA - ovalalbumin
PBMN - Peripheral blood mononuclear cells
PFC - Plaque forming cell
pI - isolectric points

PMA - Phorbol myristate acetate

PMN - Polymorphonuclear leucocytes

PPD - Purified protein derivative

PWM - Pokeweed mitogen

RE - Reticuloendothelial system

RPI - relative proliferation index

SEA - Soluble egg antigen

SDS-PAGE - Sodium dodecyl sulphate polyacrylamide gel electrophoresis

SI - Stimulation index

SIF - Suppressor cell induction factor

SISS - Soluble immune suppressor supernatant

SIRS - Soluble immune response suppressor

SK-SD - Streptokinase streptodornase

SMA - Spinal muscular atrophy

SRBC - Sheep red blood cells

SSF - Soluble suppressor factor

SWAP - Adult worm antigen

Ti - T cell antigen receptor

TLC - T lymphocyte clones

T_{csi} - T contrasuppressor inducer

$T_{csi}F$ - T contrasuppressor inducer factor

T_{cse} - T contra suppressor effector

TD - Thymus dependent

TI - Thymus independent

$TNBSO_3$ - Trinitrobenzene sulfonic acid sodium salt

TNP - Trinitrophenyl

TRF - T cell replacing factor

TRF - Thymus replacing factor

UV - Ultraviolet light

V - Variable

VPF - Vascular permeability factor

RENAL DISEASES

RENAL DISEASES

AIM

This chapter will discuss the investigations done on Nephrotic Syndrome and Chronic Renal Failure:

A. A study of numerical and functional measures of cellular immunity in 68 children (both African and Indian) with nephrotic syndrome compared to age, sex and race matched controls. Further we relate these immunological tests to the clinical problems of relapse, remission and infection rates.

B. A study of numerical and functional measures of cellular immunity in 19 adults with chronic renal failure compared to age, sex and race matched controls.

SUMMARY: IMMUNOREGULATORY CELLS IN NEPHROTIC SYNDROME OF CHILDHOOD

Immunoregulatory cells were evaluated by studying lymphocyte subpopulations (identified by E-rosettes, surface immunoglobulin and the OKT set of monoclonal antibodies), functional assay of suppressor cells using Concanavalin A (ConA) and mononuclear cell (MNC) transformation by pokeweed mitogen (PWM) in 68 children with nephrotic syndrome of different histological types.

A decreased T4/T8 ratio was the most frequent finding but no uniform pattern of immunoregulatory abnormalities was detected in the different histological groups; the most pronounced changes were found in Minimal Change Nephrotic Syndrome (MCNS) and Membranous Nephrotic Syndrome (NS). The low T4/T8 ratio was due to a significant increase in $T8^+$ cell numbers during relapse compared to remission in MCNS. This difference remained when patients with proteinuria only were compared to those in remission in MCNS. When patients in relapse and remission were separately compared to matched controls there was a

wider range of immunological alterations mainly affecting cell proportions including T4/T8 ratio which was significantly decreased during both relapse and remission and B cell numbers which were decreased in remission. These abnormalities except for the number of B cells were transient as they were not detected in those who had been in remission for more than 5 years.

The numbers of $T8^+$ cells were increased and the T4/T8 ratio and B cells decreased in Membranous NS, the proportion of $T4^+$ of $T3^+$ cells were decreased in Proliferative NS and B cell numbers reduced in miscellaneous nephropathies. Immunological parameters (apart from B cells) were similar in HBsAg carriers without nephropathy as compared to those with nephropathy. ConA induced suppression and PWM transformation of MNC were normal in nephrotic syndrome. Significant correlations were noted between some immunological parameters and the relapse and infection rates in MCNS.

These results suggest that quantitative derangements of immuno-regulatory cells may underpin abnormalities related to immunopathogenesis and susceptibility to infection noted in the nephrotic syndrome of childhood.

A. NEPHROTIC SYNDROME

1. Immunopathogenesis of Glomerular Diseases

1.1 Immune Complexes

In the early 1960's two groups of workers led by Germuth and Dixon (Germuth 1953; Dixon et al., 1958) established through experimental work on the serum sickness model in the rabbit, that glomerular lesions were caused by immunological mechanisms. Most of these lesions were characterized by the presence of immune aggregates either along the capillary walls or the mesangium - a picture which is known as immune complex glomerulonephritis. Activation of complement components would subsequently induce damage to glomerular structures, resulting in glomerulonephritis.

However, this idea of immune complexes being deposited from the circulation was challenged since it hardly seemed possible for macromolecules like immune complexes to be able to gain access to the glomerular basement membrane (GBM) and be deposited there. Other pathogenetic mechanisms for the induction of immune complex glomerulonephritis have been described (Van Damme et al., 1978).

1.2 Size, charge and selectivity of the glomerular filter

The glomerular filter is comprised of the GBM, consisting of type IV collagen, laminin and glycoproteins; of fenestrated endothelium lined at the luminal side; and of epithelial cells with foot processes that make contact with the GBM at the side of the urinary space. This filter contains negative charges which are present along the cell membranes of epithelial and endothelial cells as negatively charged sialoproteins known as glomerular polyanions. There are additional negative charges which are present diffusely in the laminae rarae of the GBM.

These anionic sites have been identified as glycosoaminoglycans (Kanwar and Farquhar, 1979). In the filtration process two different filtration modes take place: one according to size (Rennke and Venkatachalam, 1979) and the other according to charge (Change et al., 1975). Molecules with an effective radius of less than 1,8 nm can pass the filter unhindered but with increasing molecular size the clearance of the molecules decreases until it is virtually zero at a molecular diameter of 4,2 nm. The charge - selective function of the glomerular filter resides in the overall negative charge of the glomerular filter, which will repel negatively charged molecules whereas the passage of neutral or more cationically charged molecules is facilitated. On the basis of these data, doubt was raised whether macromolecules like immune complexes were able to travel across the GBM and be deposited at the epithelial side.

1.3 "Fixed" and "Planted" Antigens

Experimental studies on autologous and heterologous immune complex glomerulonephritis provided evidence that no circulating immune complexes were deposited in the glomeruli, but instead were formed locally in the GBM (Van Damme et al., 1978; Fleuren et al., 1980b). GBM antigens are present in an interrupted pattern along the epithelial side so that circulating antibody directed against GBM antigens was able to bind to these (Couser et al., 1978; van Damme et al., 1978). Such antigens have been called "fixed antigens".

Apart from the role of "fixed antigens" other studies have demonstrated that antigens which are not related to the GBM could also be involved in the in situ formation of glomerular immune aggregates. These antigens first bind to the GBM and subsequently react with their specific antibody. Such antigens are known as "planted antigens" (Fleuren et al., 1980a). Antigens like ConA have been shown to act as planted antigens (Golbus and Wilson, 1979) as well as cationic antigens (Border

et al., 1982) which presumably bind to the anionically charged sites in the GBM. Moreover, cationic antibodies act as planted molecules and bind secondarily to their specific antigen which is filtered across the GBM (Oite et al., 1982). It is not known whether the charge of circulating immune complexes is important in localization in the GBM or in the mesangium (Gallo et al., 1983). Experimental evidence has shown that glomerular localization of circulating immune complexes occurs only in cases of low affinity immune complexes - this could mean that these immune complexes first dissociate into their constituents and are then involved in in situ formation in the glomeruli (Steward, 1979).

It is difficult to extrapolate results from animal studies to the human situation although they can be used to form a working hypothesis. Several investigators (Naruse et al., 1974; Douglas et al., 1981) have been able to detect tubular brush border antigens in glomerular immune aggregates in cases of membranous glomerulopathy. This would indicate that the pathogenesis is similar to that in autologous and heterologous immune complex glomerulonephritis. Planted antigens could be important in cases of membranous glomerulonephropathy which accompany epithelial malignancies or are associated with viral or parasitic infections. In these cases tumour antigens or antigens derived from the infectious agents have been found in the glomerular immune aggregates. In acute post-streptococcal glomerulonephritis it has been shown that in situ formation of immune aggregates in the subendothelium involves planted antigens (Lange et al., 1983)

1.4 Acute and Chronic Glomerulonephritis

In acute glomerulonephritis the antigen is usually of exogenous origin eg. post-infectious glomerulonephritis. If the reaction of the host has eliminated the antigen then immune complexes will no longer be formed either in the circulation or in situ in the

GBM, so that the glomerulonephritis will subside and heal. This happens in more than 90% of cases (Potter et al., 1982). However, when the antigen is of endogenous origin because of the constant availability of the antigen there is a continuous formation of immune complexes and consequently results in chronic glomerulonephritis. It has been shown that in 20% of cases with chronic glomerulonephritis (Row et al., 1975) there is a spontaneous healing. In this type of situation one has to assume that either the immune reaction of the host has subsided or that the antigen is no longer available. Chronicity of glomerulonephritis, can also result from immune complexes other than those originating from antigens from the infectious agent concerned. It has been shown that acid eluates of glomeruli from patients with post-streptococcal glomerulonephritis contain anti/IgG antibodies (McIntosh et al., 1978). In cases of serum sickness glomerulonephritis, immune aggregates have been removed from the glomeruli using injections of excess antigen during the first weeks of the disease (Mannik and Striker, 1980; Haakenstud et al., 1983), but later excess antigen no longer removes the aggregates (Penner et al., 1982). When an excess of rheumatoid factor was used (Rose and Lambert, 1980), the immune aggregates disappeared suggesting the presence of IgG-anti-IgG immune complexes. This might be happening in some cases of chronic glomerulonephritis which are associated with systemic lupus erythematosous (SLE) where these anti-idiotypic antibodies might interfere with the production of antibodies.

Therefore in summary: the pathogenic mechanism in the formation of in situ aggregates in immune complex glomerulonephritis appears to be in the fixed (GBM) or planted (non-GBM) antigens, although the participation of idiotype-anti-idiotype immune complexes must also be considered. The deposition of immune complexes from the circulation does not seem to be an important pathogenic mechanism.

2. Immunology in Nephrotic Syndrome

It is generally accepted that the central abnormality in the nephrotic syndrome, irrespective of the cause, is an increase in glomerular permeability to protein. The resulting proteinuria, if not compensated for by increased protein synthesis, results in reduced concentrations of a variety of plasma proteins. An obvious explanation is the readily apparent lesions due to glomerular injury seen in renal biopsy material. However, in patients with minimal change nephrotic syndrome (MCNS), such lesions are absent. The primary defect in this disease is thought to be the immune system; the release of a lymphokine may be responsible for the increased permeability of the glomeruli to albumin as well as for some of the extrarenal abnormalities in MCNS (Figure 75).

Evidence has shown that T cells are important in pathogenesis (Shalhoub, 1974; Mallick, 1977; Schulte-Wisserman, 1977). Shalhoub (1974) was the first to postulate that the disease could be produced by a systemic abnormality of T cell function. This hypothesis has been supported by the following observations:

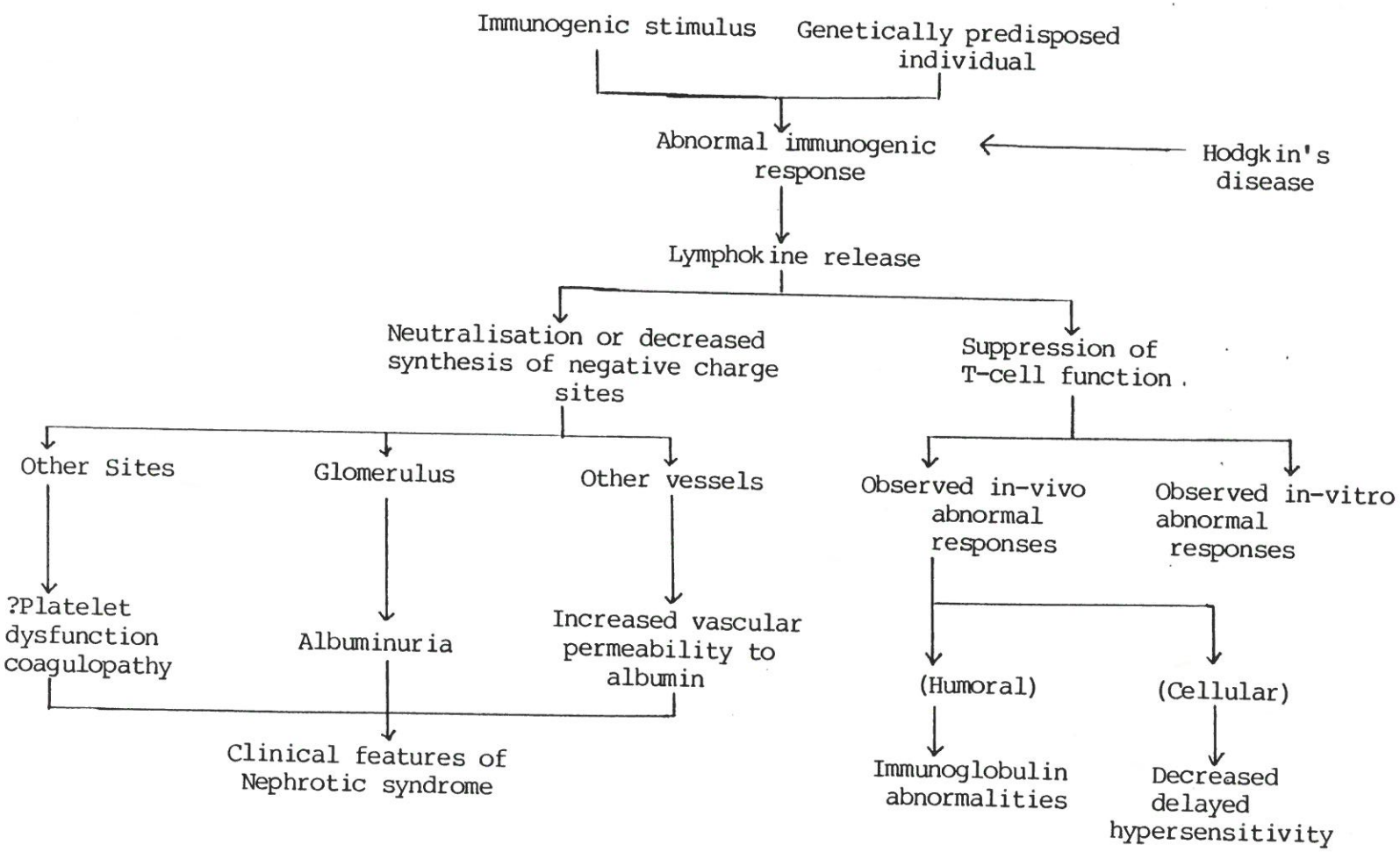
(a) There is a dramatic remission of MCNS induced by measles virus, which is known to cause immunodepression

(b) Drugs, such as steroids and cyclophosphamide, that are known to depress CMI responses, are of therapeutic benefit (Grupe, 1979)

(c) An increased susceptibility of MCNS to develope pneumococcal infections

(d) MCNS can occur during active Hodgkin's disease (Moorthy et al., 1976a)

Figure 75 A proposed scheme to explain the pathophysiology of MCNS



a) Humoral Immunity

(i) Antibody

In 1975, evidence was provided for the hypogammaglobulinaemic state seen in MCNS (Giangiacomo et al., 1975). These authors proposed that this was linked to abnormal immune circuits, suggesting a defect at the stage of production rather than due to urinary losses. Decreased levels of serum IgG and increased levels of IgM in MCNS in relapse has been found by several authors (Gupta and Yuceoglu, 1985; Fodor et al., 1982; Shakib et al., 1977; Sobel et al., 1976). The ability of lymphocytes to produce immunoglobulins when stimulated with PWM in vitro, was similarly found to be decreased in patients with membranous glomerulonephritis (Ooi et al., 1980). In another study Heslan et al., (1982) showed that in 4 categories of patients with nephrotic syndrome viz. MCNS in remission or relapse, in membranous glomerulonephritis and membranoproliferative glomerulonephritis, MCNS patients in remission had in vitro IgG production and serum IgG close to normal levels in contrast to the other 3 nephrotic groups which had decreased in vitro production of IgG, even though all groups were comparable in terms of serum albumin, serum proteins and proteinuria. This, the authors suggested to indicate that the hypogammaglobulinaemia in nephrotic patients was linked to a synthetic deficiency rather than to increased catabolism or urinary losses. Although there was this transitory dysfunction of polyclonal B cell activation correlating to the state of the nephrotic syndrome, the precise localization of the defect and the pathogenic mechanism still have to be elucidated.

(ii) Complement

Wherever, antigen-antibody complexes initiate local disease, a number of other mechanisms are involved in the mediation of a lesion. These include the coagulation and the complement system, the release of leukotoxins, anaphylatoxin and histamine from leucocytes and/or

platelets. In post streptoccoccal glomerulonephritis, it has been shown that the complement system is usually activated via the alternate pathway as reflected by decreased properdin and C3. However, early in the course of post streptococcal glomerulonephritis, serum C4 may also be reduced, indicating participation of the classical pathway. Reduced plasma C3 levels have also been found in nephritis of SLE and low plasma levels of complement may be found in membranoproliferative glomerulonephritis (Earle and Jennings, 1966).

b) Cell mediated Immunity

Recent evidence has shown that immunoregulatory cells both in number (Feehally et al., 1984; Tanphaichitr et al., 1980; Lin, 1985; Matsumoto et al., 1984a) and/or function may be abnormal in nephrosis (Gupta and Yuceoglu, 1985; Taube et al., 1984; Matsumoto et al., 1984b; Wu and Moorthy, 1982)). However, these studies have been mainly confined to those patients with MCNS both in adults and children, or to other histological categories of nephrotic syndrome mainly in adults.

(i) Numbers of cells

In children with MCNS, several groups of workers (Fodor et al., 1982; Kerpen et al., 1979; Herrod et al., 1983) found normal proportions and/or absolute numbers of T cells (E-rosette) in those who were in remission as well as in relapse. Tanphaichitr et al., (1980) found a decreased percentage of T cells (E-rosette) during relapse. However, this discrepancy in the results could be due to the fact that in the latter authors', the children in relapse had had prolonged treatment with steroids and cyclophosphamide-drugs known to decrease T cell number.

No significant changes in the proportions of $T\mu$ and $T\gamma$ have been found in the active stage of MCNS (Gupta and Yuceoglu, 1985). This is in contrast with an increase in the numbers of $OKT3^+$, $OKT4^+$ and $OKT8^+$ cells with a decrease in OKT4/OKT8 ratio in MCNS patients in relapse

(Feehally et al., 1984), although in MCNS patients in remission normal numbers were found. The increase in $OKT8^+$ cells in acute phase MCNS has also been demonstrated by Lin (1983).

(ii) Function of cells

Delayed Hypersensitivity skin reactions

No differences were found in skin reactions to purified protein derivate (PPD) and streptokinase - streptodornase (SK - SD) in children with MCNS either in remission or relapse or the control group (Fodor et al., 1982). However sensitization to dinitrochloro benzene (DNCB) was positive in all MCNS patients in remission and 93% of control children as compared to only 38% in MCNS patients in relapse (Fodor et al., 1982). Similar results were observed in adults with lipoid nephrosis (Matsumoto et al., 1981). The inability to become sensitized to a new T cell-dependent antigen was suggested to imply an alteration in the recognition of antigen and/or processing.

Lymphocyte Transformation to PHA and PPD

Lymphocyte transformation to PHA was significantly reduced in MCNS patients in relapse when compared with patients in remission and control children (Fodor et al., 1982; Gupta and Yuceoglu, 1985). Similarly in MCNS patients in relapse who also have a positive skin test to PPD, lymphocyte transformation to PPD was reduced in comparison to MCNS patients in remission with a positive skin test who had normal lymphocyte reactivity. These observations have been confirmed in both children (Fodor et al., 1982; Martini et al., 1981; Inoue et al., 1982) and adults (Iitaka and West, 1979; Sasdelli et al., 1980; Moorthy et al., 1976b).

It has also been shown that sera from MCNS patients (both children and adults) in relapse inhibit the normal lymphocytes' response to mitogens (Fodor et al., 1982; Inoue et al., 1982; Martini et al., 1981; Beale et al., 1980; Iitaka and West, 1979; Sasdelli et al., 1980

and Taube et al., 1981a). This inhibitory effect is not confined to MCNS, as it has been found in other disorders causing the nephrotic syndrome eg. focal glomerulosclerosis and membranous nephropathy (Iitaka and West, 1979; Beale et al., 1980; Taube et al., 1981a). Several factors were thought to cause this inhibitory effect of plasma from MCNS patients viz:

: hypoalbuminenia. However plasma from patients in complete remission in which the albumin deficit has been corrected still showed an inhibitory effect (Taube et al., 1981a; Fodor et al., 1982).

: Increased serum concentration of very low density lipoproteins (Chisari, 1977; Menchaca and Lefkowitz, 1980). These authors observed a decreased lymphocyte transformation response to PHA when MCNS patients' lymphocytes in remission were cultured with pooled normal serum to which they had added very low density lipoprotein fraction to equal the usual nephrotic levels.

: Diminished zinc levels in the hair and plasma has been reported in MCNS (Reimold, 1980). There is increasing evidence of the involvement of this element in the immune response (Bach, 1981) especially for the activity of serum thymic factor (Dardenne et al., 1982). Bensman et al. (1984) have reported a diminution in the biological activity of serum thymic factor secondary to zinc deficiency in children with nephrotic syndrome.

T suppressor cell activity

The finding of increased suppressor cell activity (Schulte-Wisserman et al., 1977) is not consistent, as a decrease in ConA inducible T suppressor cell activity has also been reported in MCNS patients in relapse (Gupta and Yuceoglu, 1985; Taube et al., 1984).

Vascular Permeability Factor and other Lymphokines

The mechanisms of glomerulopathy given above do not explain all the varieties of glomerulopathy and the occurrence of proteinuria. This is the case, especially in MCNS where the absence of significant pathologic lesions contrasts with the intensity of the clinical symptoms. It could be that other mechanisms of immunologic origin eg. release of humoral or cellular mediators could affect the capillary permeability independently of the presence or absence of histologic injury (Lagrue et al., 1975). It has been shown that a lymphokine termed vascular permeability factor (VPF) is released when peripheral lymphocytes of patients with nephrotic syndrome are treated by concanavalin-A (Sobel and Lagrue, 1980). These authors proposed that VPF may participate in the pathogenesis of functional and/or lesional alterations in the filtration barrier. It could be that there is an abnormal function or elaboration of interleukins in the nephrotic syndrome. The finding of an abnormal in vitro B cell activation in nephrotic syndrome may illustrate this concept (Heslan et al., 1982).

VPF has been shown to be abnormal in patients with nephrotic syndrome both by in vivo (Sobel and Lagrue, 1980) and by in vitro (Sobel and Lagrue, 1980) techniques. All types of nephrotic syndrome had increased VPF production (MCNS, focal glomerulosclerosis, membranous glomerulonephritis, membranoproliferative glomerulonephritis) as compared to MCNS in remission who had similar values to normal controls. (Sobel and Lagrue, 1980). The physicochemical characterization of VPF has shown that it is recovered in a peak emerging immediately after cytochrome C (molecular weight 12700), is sensitive to pepsin, resistant to DNAase and RNAase, heat labile at 100^{o}C and is produced by T cells only (Sobel et al., 1983).

3. The Nephrotic Syndrome among children in Africa

The following histological categories of nephrotic syndrome are found in Indian and African children in South Africa.

Table: 47 Features of nephrotic syndrome in South African children

		African	Indian
Incidence		0,17%	
Peak age		2 peaks = 4 years and 8-11 years	Pre-school (3 years)
Sex incidence	:	M>F	M>F
Aetiology	:	Unknown in majority	Unknown in majority
Histological groups	:	Dominated by obvious structural glomerular lesions (86%) Minimal change in 14%	Dominated by minimal change (80%)
Immunofluorescence	:	Deposits in most including minimal change	No deposits in majority
Response to therapy steroids, cyclophosphamide	:	Do not respond	Majority respond to steroids (97% of those with minimal change respond to steroids)
Children who relapse frequently	:	-	28%
Prognosis	:	Some evidence suggests outcome related to histological group	Excellent

Table 48 Histological categories of nephrotic syndrome in Indian and African children in South Africa

No obvious lesions:	Minimal change	
Obvious Structural lesions	Extramembranous	
	Proliferative	: Mesangial
		: Exudative and Endo-
		: capillary
		: Membranoproliferative
		: Focal
	Focal glomerulosclerosis	
	Tropical extramembranous	
	Tropical nephropathy	
	Unclassified	

In the vast majority of non-African children nephrotic syndrome is not a serious disease, there is a predictable response to drug therapy and the disease is dominated by minimal change lesions which account for ±80% of all children with nephrosis (Coovadia and Adhikari, 1982).

The nephrotic syndrome in Indian South African children resembles that of children in the West, Asia and South America. However in African children in Southern Africa, there is an unusual distribution of the histopathological types of nephrotic syndrome (Coovadia et al., 1979). Table 47 demonstrates the features of nephrotic syndrome in South African children. It should be noted that White children with nephrotic syndrome in South Africa follow a pattern similar to the Indians.

Even in the absence of the damaging effects of malaria on the kidney, African children in Southern Africa have an unusual distribution of histopathological types of nephrosis which distinguishes them from children in tropical Africa and in other continents. The disease behaves differently in African children generally compared with most non-Africans. HBs carrier status is the most frequent association with membranous nephropathy. (Table 47)

Table 47. Some associated infections in nephrotic syndrome

Nephrotic Syndrome Histology	Race	Sex	Total Number	HBsAg +ve	Associated Infections Schisto-somiasis	Malaria	Typhoid	TB
MCNS	African	Male	2	1	0	0	0	0
		Female	3	0	0	0	0	0
	Indian	Male	20	0	0	0	0	0
		Female	7	0	0	0	0	0
Membranous	African	Male	13	12	3	0	0	1
		Female	1	1	1	0	0	0
	Indian	Male	0	0	0	0	0	0
		Female	0	0	0	0	0	0
Proliferative	African	Male	4	0	1	0	0	0
		Female	0	0	0	0	0	0
	Indian	Male	4	0	0	0	0	0
		Female	3	0	0	0	0	0
Other	African	Male	2	1	1	0	1	0
		Female	4	1	0	0	0	0
	Indian	Male	5	0	0	0	0	0
		Female	0	0	0	0	0	0

1) Materials and Methods

Patients and Controls (Table 48)

Sixty-eight African and Indian children with NS were studied (Table 49). Thirty-two children had MCNS (mean age 8,20 ± 0,71 years, age range 1,75 - 18 years) and of these 16 were in remission and 2 were African. Fourteen children (all African) had Membranous Glomerulonephritis (mean age 7,29 ± 0,72 years, Range 4-10 years) of whom 1 was in remission; 13 of these patients were HBsAg carriers; 11 children had Proliferative glomerulonephritis (mean age 8,81 ± 1,18 years, Range 3-13 years) of whom 3 were in remission; 11 children had miscellaneous histological groups of NS (mean age 8,99 ± 1,42 years, Range 2,92-18 years), of these, 2 were in remission. In each group there were more males than females. All were biopsy proven except for 2 patients in the miscellaneous group. None of the patients were in chronic renal failure, had elevated serum creatinine levels or had any other chronic disease other than the nephrotic syndrome. The HBs antigen positive patients did not have liver disease but were carriers. None were on any steroid therapy at the time or 6 months prior to these investigations. Each patient was age, sex and race matched with a normal healthy control; these were obtained from a community based study of HBV prevalence. (See Chapter on Normal development of Immune Response) Eight African children of whom 6 were male (age range 1 year 5 months - 10 years) who were HBs antigen carriers without nephropathy were used as additional controls for the Membranous group. Informed parental consent was obtained in each case.

Definitions used:

Nephrotic syndrome was defined according to three essential criteria: massive proteinuria (>2gm/m^2/day), hypoalbuminaemia (<30g/l) and oedema.

Table 48 Patient details

Africans					Indians					
Histology	Sex	Condition Remission	Partial Remission	Relapse	Sex	Condition Remission	Partial Remission	Relapse	Total	
MCNS	Male	1	0	1	Male	9	6	5	22	
	Female	1	0	2	Female	5	0	2	10	
Membranous	Male	1	1	11	Male	0	0	0	13	
	Female	0	0	1	Female	0	0	0	1	
Proliferative	Male	1	0	3	Male	2	0	2	8	
	Female	0	0	0	Female	0	2	1	3	
Miscellaneous	Male	0	0	2	Male	2	2	1	7	
	Female	0	0	4	Female	0	0	0	4	
Total	Male	3	1	17	Male	13	8	8	50	68
	Female	1	0	7	Female	5	2	3	18	

Relapse was defined as the presence of all three features of nephrotic syndrome (remission was defined as the absence of all three features of nephrotic syndrome), partial remission was defined as the presence of persistent proteinuria without oedema and is included under relapse for purposes of this study. Partial remission was at times included with relapse but this will always be indicated in the text (this was done because little difference was found in immunological parameters between partial remission and relapse).

The clinical features of protein-energy-malnutrition were absent on clinical grounds and according to anthropometric measurements of the National Centre for Health Statistics (NCHS) standards of weight-for-age, height-for-age and weight-for-height ratios in both nephrotic syndrome patients and controls.

Methods:

I. Immunological

(A) Numerical Assays

(i) T and B subpopulations
(This was undertaken as described under Methods)

(ii) T cell subsets using monoclonal antibodies.
(This was undertaken as described under Methods)

(B) Functional Assays

(i) T suppressor cell function using ConA pretreatment.
(This was undertaken as described under Methods)

(ii) MNC PWM stimulation
(This was undertaken as described under Methods)

II Clinical Investigations

The following clinical features were observed simultaneously (at the time of bloodtaking): the degree of oedema, hypertension haematuria, albuminuria; the levels of serum albumin, blood cholesterol and α_2-globulin were measured; the status of HBs antigenaemia was similarly measured; therapy (previous and present) as well as the length of the condition with the number of previous mild and severe infections were noted. Other clinical investigations included were to detect schistosomiasis (both S.mansoni and S.haematobium), malaria, typhoid and tuberculosis (TB). The latter 4 investigations were done only if they were necessary.

Statistical Analysis

Results were analysed according to the non-parametric procedures using the Mann Whitney U. Results were significant at the 5% level. Correlation analysis was performed by using the Pearson correlation co-efficient at the 5% level of significance.

RESULTS

The results will be discussed as follows:

A. Effect of Relapse versus Remission

Comparison of Immune parameters in Relapse with those in Remission in MCNS patients.

B. Effect of Histological Differences

Immune parameters among children in different histological groups compared to their matched controls.

C. Effect of Treatment on the Immune Response

Effect of Previous Treatment

All patients (regardless of histology and clinical condition) previously treated with prednisone were compared to those who had previously been treated with prednisone + cyclophosphamide + chlorambucil.

D. Residual Effects after Prolonged Remission

The long term change in immunological parameters among children in remission.

(a) All patients in remission (regardless of histology) who had been in remission for more than 5 years were compared to those who had been in remission for less than 5 years.

(b) Patients with MCNS in remission for less than 5 years were compared to those MCNS patients in remission for more than 5 years.

E. The effect of Nephrotic Syndrome on the Immune Response

All nephrotic syndrome patients were compared to age, sex and race matched controls.

F. Relationship between Relapse and Immunological Indices and Infection and Imunological Indices

a) An index of infection was obtained for all nephrotic syndrome patients:

$$\text{Index of Infection} = \frac{\text{Number of previous infections (severe and/or mild)}}{\text{Length of time of nephrotic syndrome}} \text{ per patient}$$

This index of infection was then related to certain immunological parameters.

b) An index of relapse was obtained for all nephrotic syndrome patients:

$$\text{Index of relapse} = \frac{\text{Number of previous relapses}}{\text{Length of time of nephrotic syndrome}} \; / \; \text{per patient}$$

This index of relapse was then related to certain immunoogical parameters.

G. Correlations of tests

Correlations were sought between numerical and functional assays of supression and MNC PWM stimulation within each histological group of the nephrotic syndrome.

RESULTS

Note: Not all tests could be performed on all patients, the exact number studied is given in the tables. Correlations between immune parameters and disease stage (ie.. remission or relapse or partial remission) could only be done for patients with MCNS as there were too few patients in remission in the other histological groups.

: The Indian patients were compared with 25 controls who were in the same age range (3-18 years). The African patients were age and sex matched with community control children undergoing an epidemiological study on the prevalence of HBV (see Chapter on Normal Development of Immune Response).

A.1. Comparison of Immune Parameters in Relapse + Partial Remission with those in Remission in MCNS Patients (Table 49, Figure 76; see appendix table 7 for details)

MCNS patients in relapse had significantly increased absolute mononuclears, T cells (E-rosette) comprising mainly $OKT8^+$ cells and a lower T4/T8 ratio compared to those in remission. No significant differences were observed with respect to B and Null cells, ConA induced suppression and MNC PWM stimulation between patients in relapse versus those in remission

A.2. MNCS Remission versus MNS Partial Remission (Table 50)

This comparison was made to determine at which end of the spectrum "partial remission" was situated, viz. towards remission or relapse. This could only be done in MCNS as there were too few patients in the other histological groups.

Immunological Parameters

The number of absolute mononuclear cells, T cells (E-rosette and OKT3 MoAb) comprising mainly $OKT8^+$ cells were significantly higher in partial remission as compared to remission while

TABLE 49

COMPARISON OF LYMPHOCYTE SUBSETS BETWEEN RELAPSE AND REMISSION IN MCNS

Clinical Condition	N	Abs.mono-nuclears	Lymphocyte subpopulations as defined by E-rosette and SIg			T cell subsets as defined by specific monoclonal antibodies			Positive cells as % of OKT3		
			T cells (E-rosette)	B cells (SIg^+)	Null cells	OKT3	OKT4	OKT8	OKT4	OKT8	Ratio OKT4/OKT8
Remission	16	3556 ± 384°	2219 ± 386	179 ± 43	1092 ± 138	1952 ± 217	1135 ± 119	1042 ± 138	60 ± 4	54 ± 3	$1,18 \pm 0,10$
Relapse +	15	5515 ± 718	3827 ± 520	324 ± 104	1484 ± 264	3091 ± 484	1459 ± 204	1699 ± 228	50 ± 4	59 ± 5	$0,93 \pm 0,11$
p value		0,0418*	0,0116*	0,3562	0,3824	0,0892	0,3428	0,0418*	0,0576	0,5008	0,0459*

° Mean $\pm$ SEM

* Probability Value $\leq 0,05$

+ Relapse includes partial remission

Figure 76

COMPARISON OF LYMPHOCYTE SUBSETS BETWEEN RELAPSE AND REMISSION IN MCNS

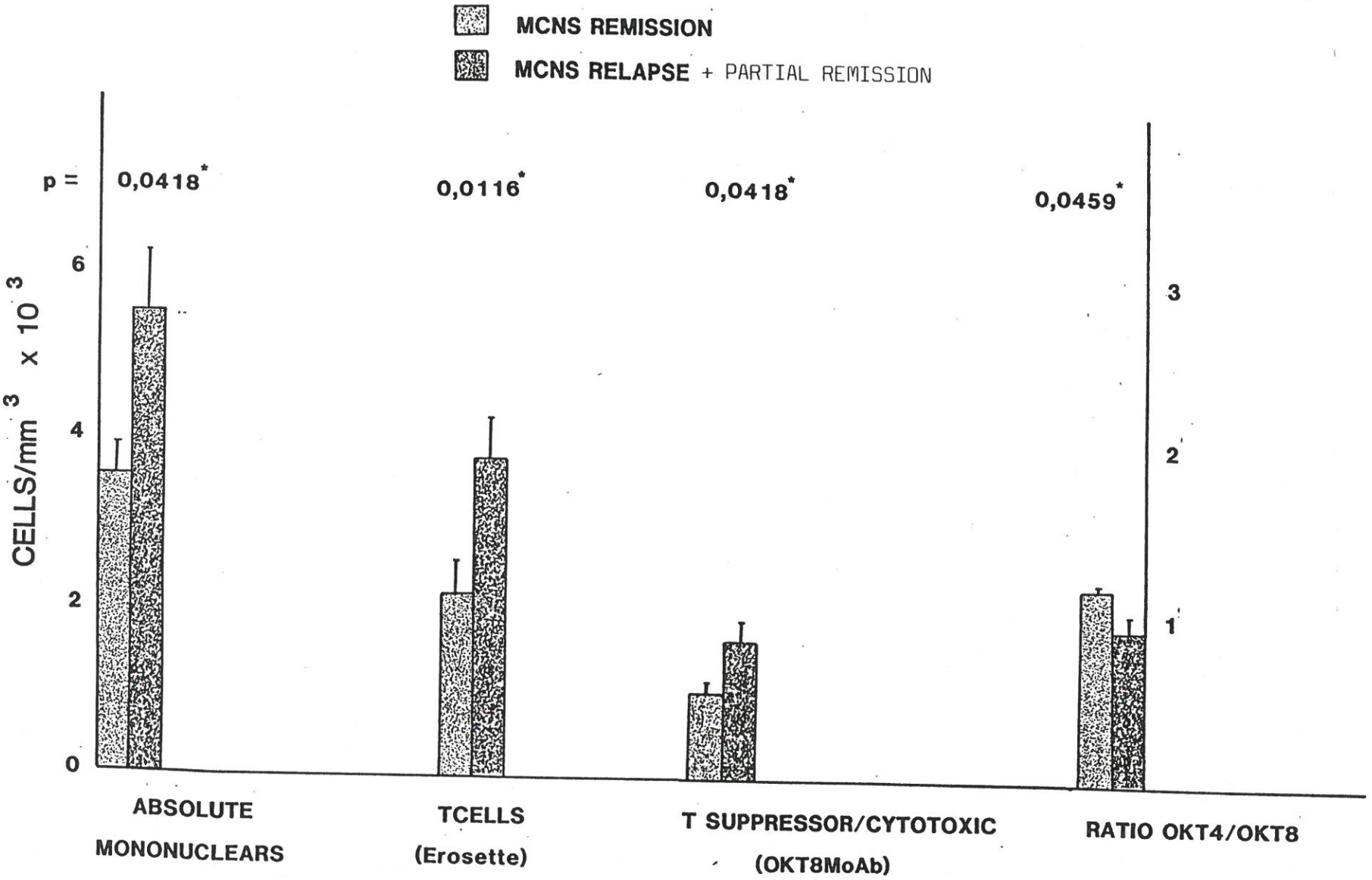


Table 50 Comparison of lymphocyte subsets beween Partial remission and remission in MCNS

Clinical Condition	N	Abs mono-nuclears	Lymphocyte subpopulations as defined by E-rosette and SIg			T cell subsets as defined by specific monoclonal antibodies			Positive cells as % of OKT3		Ratio OKT4/OKT8
			T cells (E-rosette)	B cells (SIg+)	Null cells	OKT3	OKT4	OKT8	OKT4	OKT8	
Remission	16	3556±384[o]	2219±386	179±43	1092±138	1950±217	1135±119	1042±138	60±4	54±3	1,18±0,10
Partial remission	6	5145±599	3672±433	294±103	1501±259	3204±528	1499±320	1714±305	45±4	54±3	0,86±0,10
P value		0,0270*	0,0120*	0,1833	0,1833	0,0465*	0,3020	0,0390*	0,0388*	0,9117	0,0552

[o] Mean ± SEM

*probability value ≤ 0,05

Table 51 Comparison of lymphocyte subsets between partial remission and relapse in MCNS

Clinical Condition	N	Abs mono-nuclears	Lymphocyte subpopulations as defined by E-rosette and SIg			T cell subsets as defined by specific monoclonal antibodies			Positive cells as % of OKT3		Ratio OKT4/OKT8
			T cells (E-rosette)	B cells (SIg+)	Null cells	OKT3	OKT4	OKT8	OKT4	OKT8	
Partial remission	6	5145±599[o]	3672±433	294±103	1501±259	3204±528	1499±320	1714±305	45±4	54±3	0,86±0,10
Relapse	9	5761±1156	3913±792	341±156	1474±397	3015±752	1432±279	1687±337	54±5	63±8	0,98±0,17
p value		0,9062	0,7389	0,6404	0,2740	0,4094	0,9062	0,8137	0,1949	0,4432	0,7237

[o] Mean ± SEM

* probability value ≤ 0,05

proportions of $T4^+$ cells of $T3^+$ cells were significantly lower in the former. No significant differences were observed with respect to B and Null cells, ConA induced suppression and MNC PWM stimulation between patients in partial remission versus those in remission.

A.3. MCNS Partial Remission versus MCNS Relapse

Immunological Parameters (Table 51)

No significant differences were found in any of the immunological parameters studied between children in partial remission compared to those in relapse.

B. Immune Parameters among children in different histological groups compared to matched Controls
(Table 52)

B.1 Controls versus MCNS in Remission (Table 52, Figure 77; see appendix table 8 for details)

MCNS children in remission had a significantly increased percentage $OKT8^+$ cells of $OKT3^+$ cells, significantly decreased OKT4/OKT8 raio and numbers of B cells, compared to their respective controls.

B.2 Controls versus MCNS in Relapse and Partial Remission (Table 52, Figure 78; see appendix table 8 for details)

MCNS children in relapse had a significant decrease in percentage $OKT4^+$ of $OKT3^+$ cells and in OKT4/OKT8 ratio as compared to their respective controls. Tests were similar in those with proteinuria only (partial remission) and those with oedema in addition (relapse).

B.3 Controls versus Membranous (Table 52, Figure 79; see appendix table 9 for details)

Children with membranous NS had significantly elevated numbers of absolute mononuclears, T cells (E-rosette and OKT3 MoAb) and $OKT8^+$ cells with a significantly lower number of B cells and OKT4/OKT8 ratio as compared to controls. These children, with one exception, were studied in relapse.

B.4 Controls versus Proliferative (Table 52, Figure 80; see appendix table 10 for details)

In the proliferative group the proportion of $OKT4^+$ of $OKT3^+$ cells were significantly decreased as compared to controls; this difference remained when the three patients in remission were excluded.

TABLE 52

COMPARISON OF LYMPHOCYTE SUBSETS BETWEEN CONTROLS AND DIFFERENT HISTOLOGICAL GROUPS OF NEPHROTIC SYNDROME DURING REMISSION OR RELAPSE

			Lymphocyte subpopulations as defined by E-rosette and SIg			T cell subsets as defined by specific monoclonal antibodies			Positive cells as % of OKT3		
Group	Clinical Condition	Abs.mono-nuclears	T cells (E-rosette)	B cells (SIg^+)	Null cells	OKT3	OKT4	OKT8	OKT4	OKT8	Ratio OKT4/OKT8
MCNS	Rem (16)+	3556±384°	2219±386	179± 43	1092±138	1952±217	1135±119	1042±138	60 ± 4	54 ± 3	1,18±0,10
Controls		3553±341	1989±228	378± 62	1178±187	1937±172	1291±109	896±102	67 ± 2	46 ± 2	1,54±0,09
p value		0,9426	0,9167	0,0180*	0,8835	0,6525	0,2641	0,5403	0,1652	0,0381*	0,0335*
MCNS	Rel (15)	5515±718	3827±520	324±104	1484±264	3091±484	1459±204	1699±228	50 ± 4	59 ± 5	0,93±0,11
Controls		4194±298	2515±217	463±106	1158±146	2491±239	1601±128	1143±103	66 ± 3	47 ± 3	1,48±0,11
p value		0,3451	0,0955	0,2335	0,4998	0,3955	0,4168	0,0759	0,0011*	0,0638	0,0006*
Membranous	Rem(1) Rel(13)	4387±432	3010±463	190± 38	1178±185	2691±319	1345±172	1448±214	51 ± 4	54 ± 6	1,05±0,14
Controls		3265±293	1847±159	401± 98	951±164	1861±149	1106± 97	827± 69	62 ± 4	45 ± 3	1,43±0,13
p value		0,0482*	0,0308*	0,0274*	0,3827	0,0225*	0,1594	0,0039*	0,2636	0,4512	0,0466*
Proliferative	Rem(3) Rel(8)	5132±633	3332±468	160± 44	1664±376	2956±430	1392±206	1507±262	47 ± 5	57 ± 7	1,10±0,19
Controls		3599±601	2290±433	328± 97	894±169	1844±304	1139±170	883±162	63 ± 4	47 ± 3	1,39±0,11
p value		0,0874	0,1024	0,2831	0,0527	0,0527	0,4250	0,0627	0,0250*	0,3805	>0,05
Miscellaneous	Rem(2) Rel(9)	3918±643	2752±667	124± 46	1031±272	2222±371	1160±253	1073±192	49 ± 6	47 ± 5	1,28±0,31
Controls		3248±341	1852±198	340± 76	1050±140	1835±276	1083±149	905±161	62 ± 4	48 ± 4	1,43±0,17
p value		0,3242	0,5184	0,0364*	0,6761	0,5493	0,8880	0,3786	0,0842	0,8877	0,2311

+ (Number of subjects)

° Mean ± SEM

* Probability value ≤ 0,05

Rem = Remission

Rel = Relapse + partial remission

Figure 77 Comparison of lymphocyte subsets between controls and MCNS patients in remission

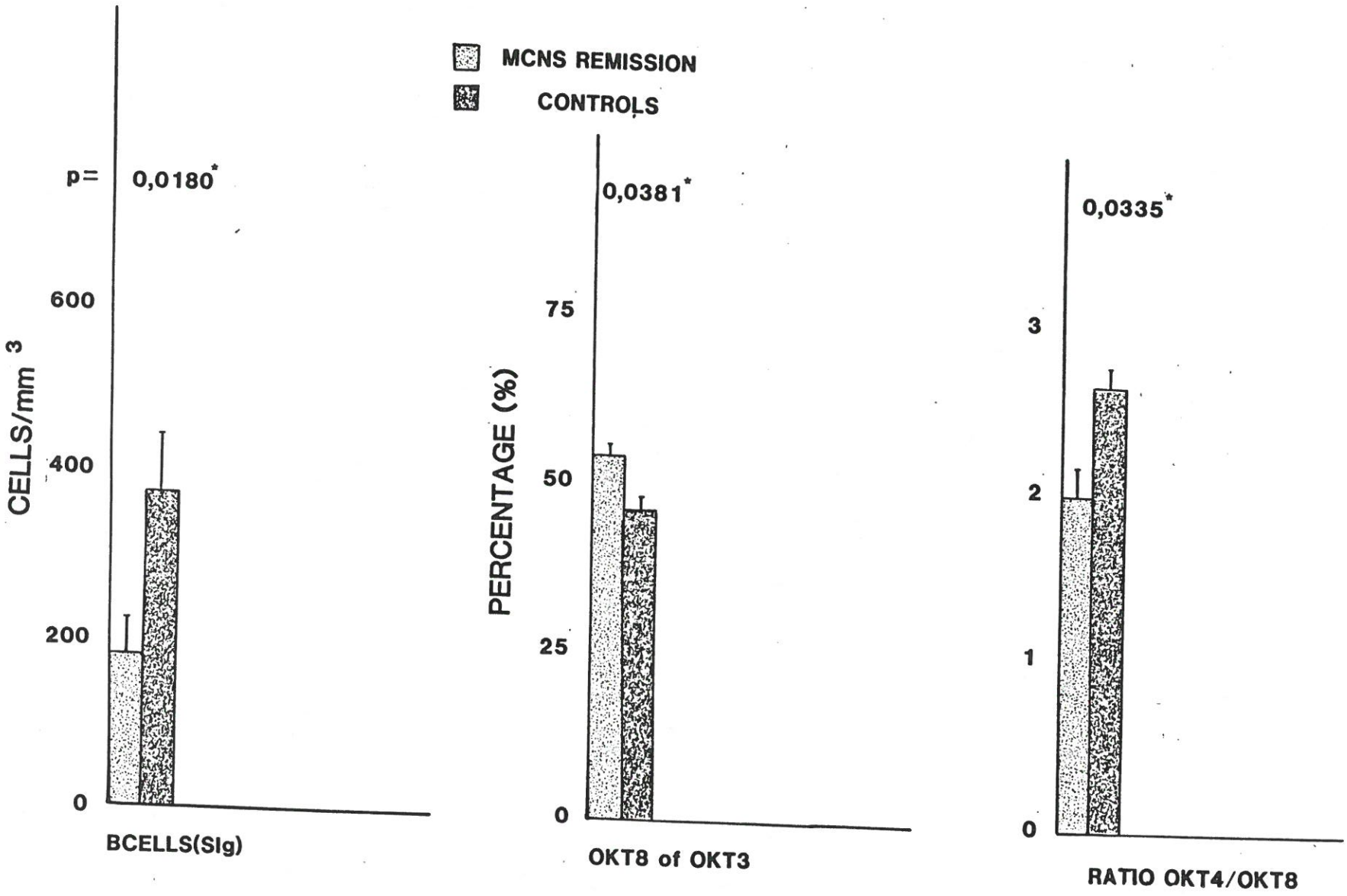


Figure 78 Comparison of lymphocyte subsets between controls and MCNS patients in relapse

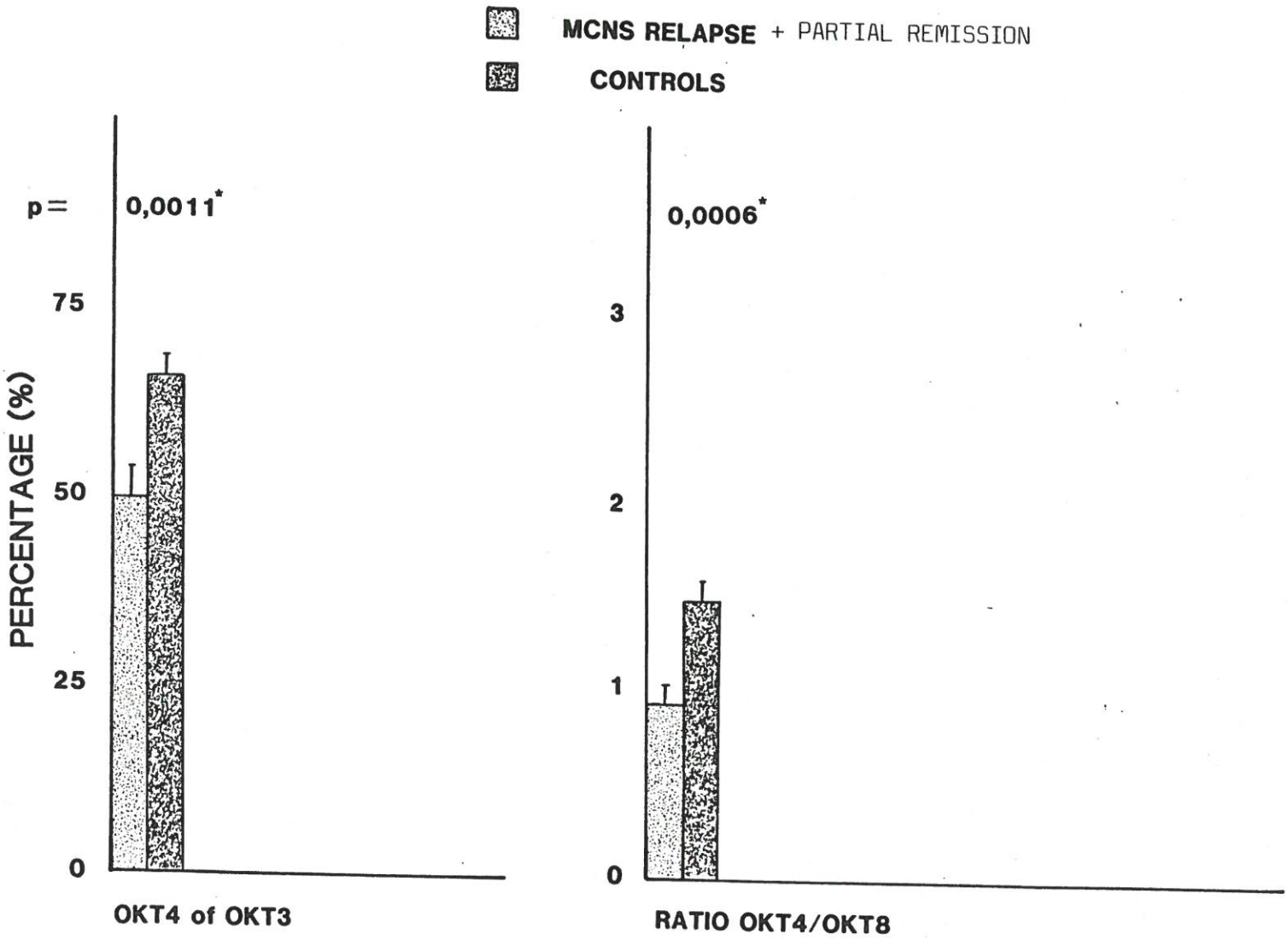


Figure 79 Comparison of lymphocyte subsets between controls and membranous group of nephrotic syndrome during remision and relapse

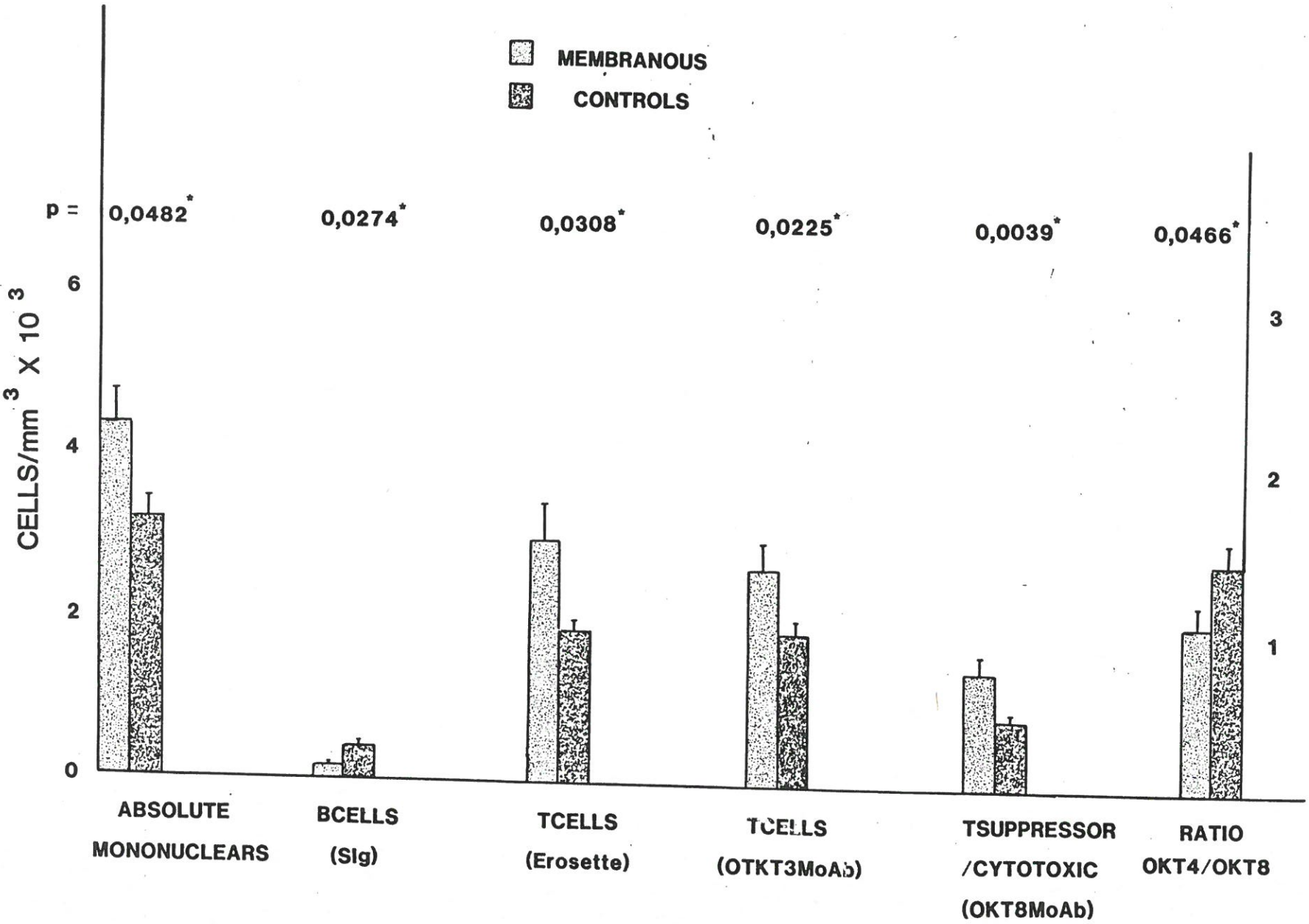
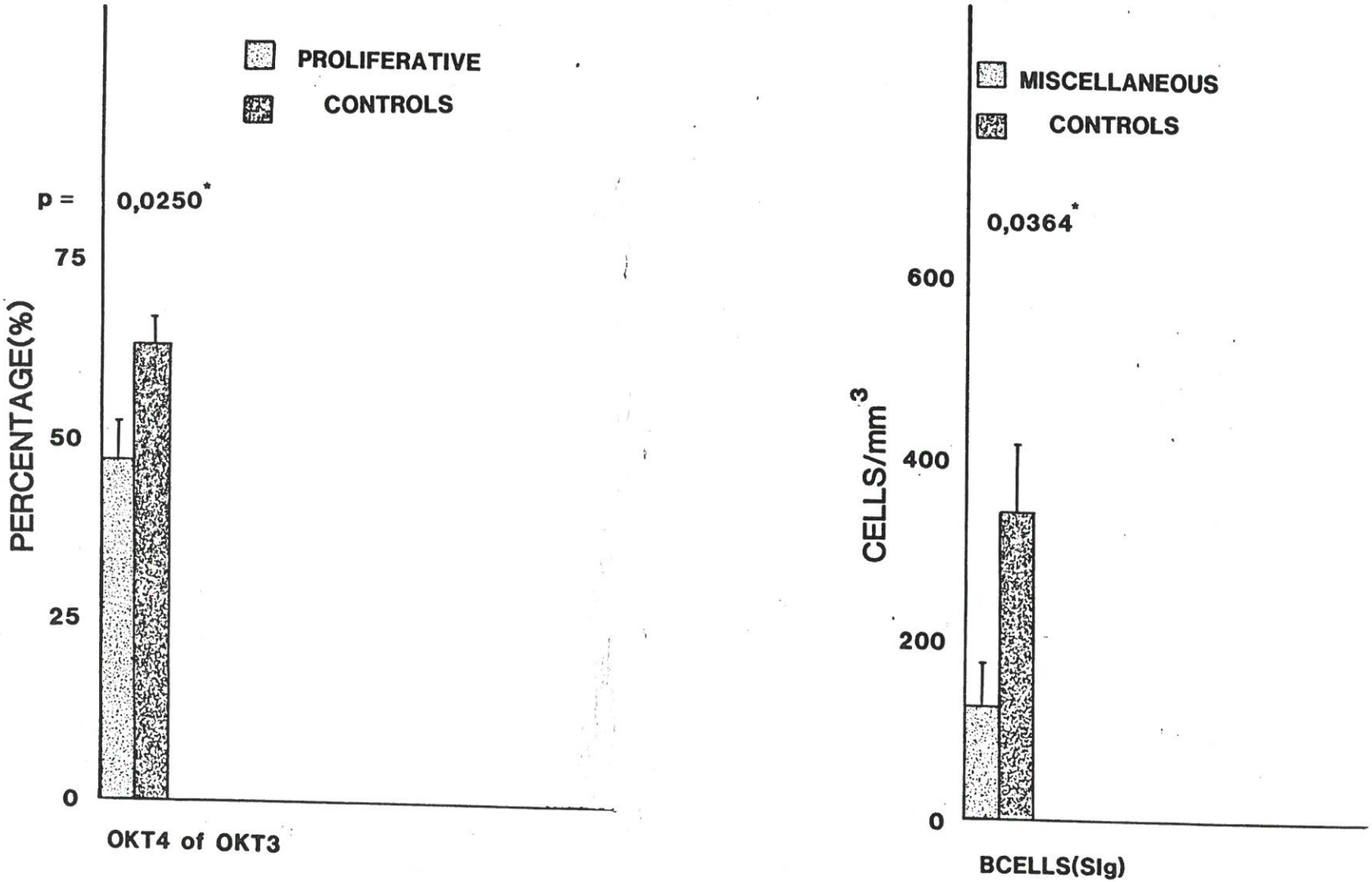


Figure 80 Comparison of lymphocyte subsets between controls and Proliferative/Miscellaneous group of nephrotic syndrome during remission and relapse + Partial remission



B.5 Controls versus Miscellaneous (Table 52, Figure 80; see appendix table 11 for details)

Children with miscellaneous nephrotic syndrome as compared to controls had significantly decreased B cell numbers; again when the two patients in remission were excluded these differences remained. No significant differences were observed in Null cells, ConA inducible T cell suppression and MNC PWM stimulation in any of the histological groups studied compared to controls and with respect to clinical condition.

B.6 Comparison between HBs Antigen carriers without nephropathy and HBs antigen membranous nephrotic syndrome (Table 53)

Note: 8 African children of whom 6 were male age range (1 year 5 months - 10 years) who were HBs antigen carriers without nephropathy were compared with the children who had HBs antigen membranous nephropathy. B cell numbers were significantly decreased in HBsAg carriers with nephropathy as compared to those without nephropathy.

C. Relationship of Treatment of the Immune Response

C.1 Effect of Previous Treatment

All Nephrotic syndrome (regardless of histology and clinical condition) previously treated with prednisone (P) were compared to those who had previously been treated with predinisone + cyclophosphamide + chlorambucil (P + C + C). This comparison could not be done as only one patient had previously been treated with P + C + C)

Table 53

ABSOLUTE VALUES FOR LYMPHOCYTE SUBSETS IN HBs ANTIGEN CARRIERS WITH AND WITHOUT NEPHROPATHY

Immunological Parameters	Without nephropathy	With nephropathy	p Value
Absolute mononuclears (cells/mm^3)	3611 ± 424°	4387 ± 432	0,2748
T cells (E-rosette) (cells/mm^3)	2080 ± 285	3010 ± 463	0,1357
B cells (SIg^+) (cells/mm^3)	457 ± 105	190 ± 38	0,0252*
Null cells (cells/mm^3)	1116 ± 214	11178 ± 185	0,8229
Total T cells (OKT3) (cells/mm^3)	2203 ± 330	2691 ± 319	0,2631
T helper/inducer (OKT4) (cells/mm^3)	1372 ± 258	1345 ± 172	0,8814
T suppressor/cytotoxic (OKT8) (cells/mm^3)	1050 ± 165	1448 ± 214	0,2961
% OKT4 of OKT3	64 ± 8	51 ± 4	0,1003
% OKT8 of OKT3	49 ± 5	54 ± 6	0,5015
Ratio OKT4/OKT8	1,42 ± 0,23	1,05 ± 0,14	0,1562

° = Mean ± SEM

* Comparison with membranous group : probability values ≤ 0,05*

D. The long term change in immunological parameters among MCNS children in Remission (Table 54, Figure 81; see appendix table 12 for details)

Children with MCNS who had been in remission for longer than 5 years had significantly lower absolute mononuclear cells, T cells (E-rosette and OKT3 MoAb) comprising mainly of $OKT8^+$ cells than children in remission for less than 5 years. No significant differences were observed in B, Null and $OKT4^+$ cell numbers, proportions of $OKT4^+$ and $OKT8^+$ cells of $OKT3^+$, OKT4/OKT8 ratio, ConA inducible suppression and PWM stimulation between the two groups. When MCNS children in remission for longer than 5 years were compared to age, sex and race matched controls, the only abnormality detected was a significant decrease in the B cell numbers in the former.

TABLE 54

LONG TERM CHANGE IN IMMUNOLOGICAL PARAMETERS AMONG CHILDREN IN REMISSION

				Lymphocyte subpopulations as defined by E-rosette and SIg			T cell subsets as defined by Monoclonal antibodies			Positive cells as % of OKT3		
Group	N	Period of Remission (years)	Abs.mono-nuclears	T cells (E-rosette)	B cells (SIg^+)	Null cells	OKT3	OKT4	OKT8	OKT4	OKT8	Ratio OKT4/OKT8
MCNS	4	≤ 5	5255±1063°	4014±1180	235±38	1533±259	2801±481	1488±299	1698±343	53 ± 7	61 ± 7	0,95±0,20
	12	> 5	2988± 218	1679±158	162±54	959±141	1666±187	1017±112	823± 80	62 ± 4	51 ± 2	1,26±0,11
p value			0,0393*	0,0180*	0,1763	0,0910	0,0393*	0,1456	0,0109*	0,2747	0,1619	0,3023
Controls△	12		3145±286	1911±276	415±80	809±114	1744±175	1206±123	821±106	69 ± 3	47 ± 3	2±0,12
p value			0,7728	0,5529	0,0176*	0,5978	0,5066	0,2253	0,9540	0,2850	0,2029	0,1489

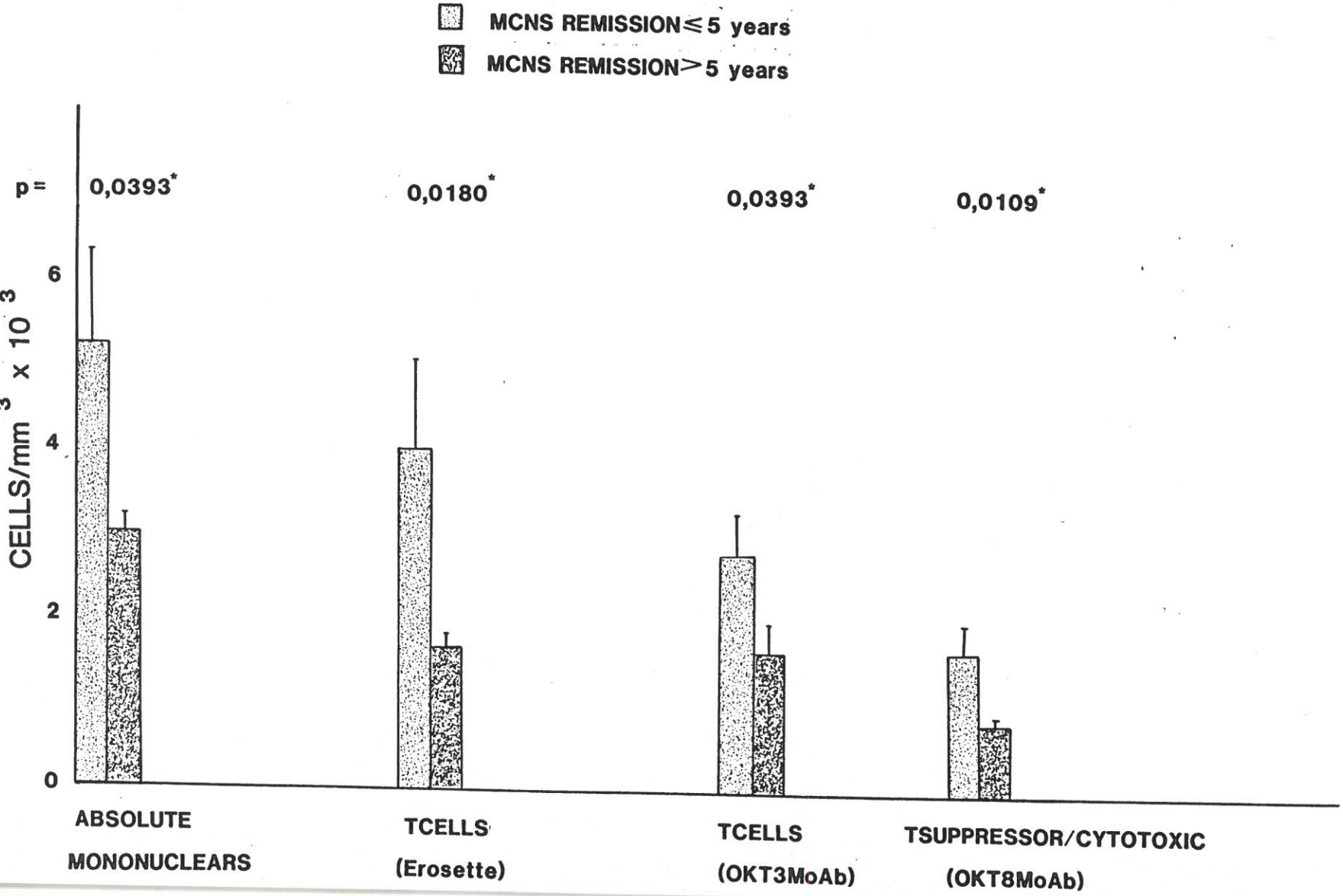
° Mean ± SEM

* Probability Value ≤ 0,05

△ Comparison: Controls versus MCNS (>5)

Figure 81

LONG TERM CHANGE IN IMMUNOLOGICAL PARAMETERS AMONG CHILDREN IN REMISSION



E. Comparison of all nephrotic syndrome patients (regardless of histology and clinical condition) with age, sex and race matched controls (Figures 82-87; see appendix table 13 for details)

WCC, absolute mononuclear cells, T cells (E-rosette), T cells (OKT3MoAb), T suppressor/cytotoxic cells and the percentage of $OKT8^+$ of $OKT3^+$ cells and percentage monocytes were significantly higher in patients with nephrotic syndrome as compared to controls. However, B cells (SIg), percentage T4 of T3, OKT4/OKT8 ratio, T suppressor cell function (using both suboptimal and optimal doses) and percentage lymphocytes were significantly lower in nephrotic syndrome patients compared to controls. No significant differences were observed between patients and controls with respect to MNC PWM stimulation and absolute lymphocytes.

Figure 82 Comparison of absolute values of cells between all nephrotic syndrome patients (regardless of histology and clinical condition) with age, sex and race matched controls

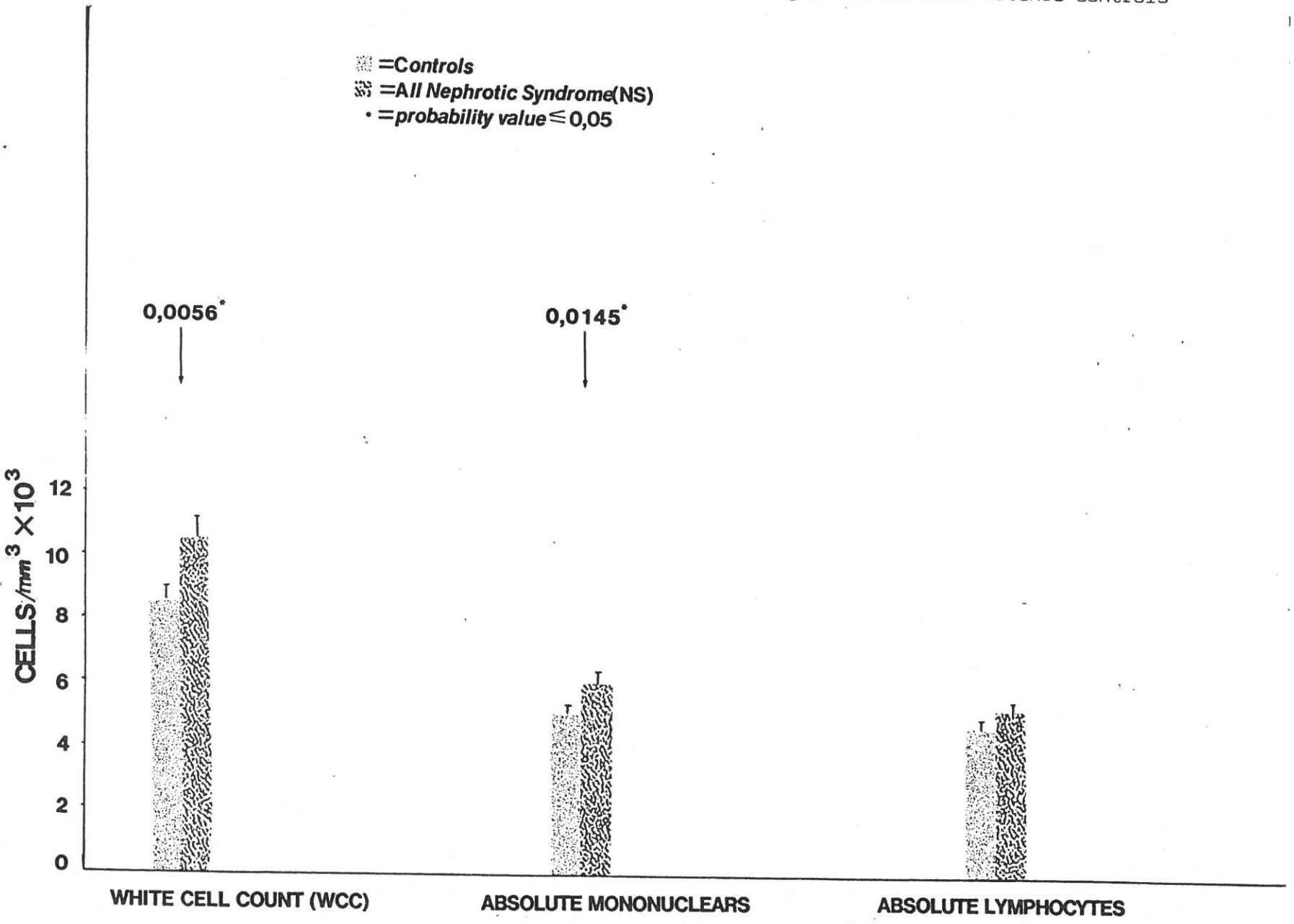


Figure 83 Comparison of lymphocyte subsets of all nephrotic syndrome patients (regardless of histology and clinical condition) with age, sex and race matched controls

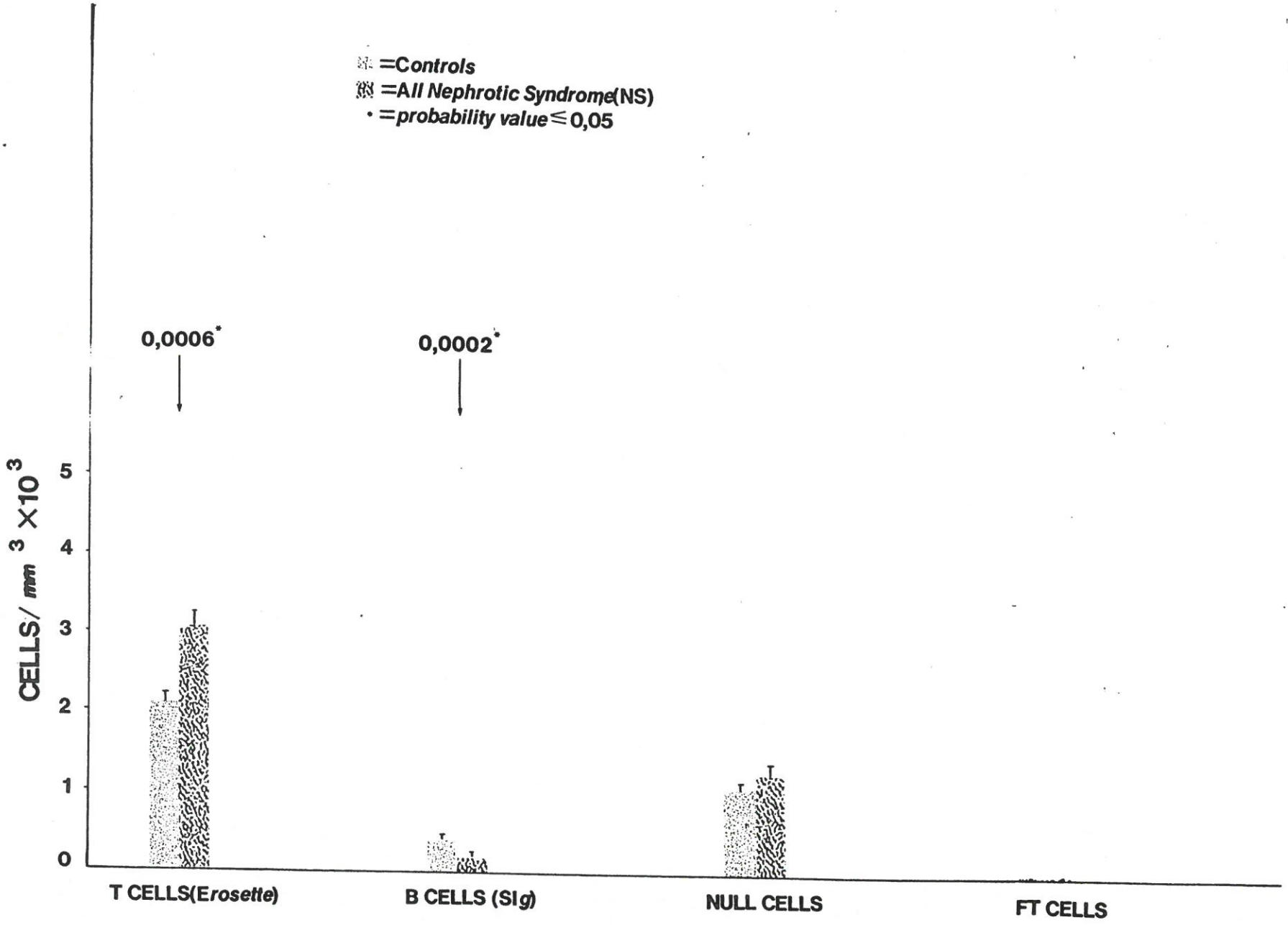


Figure 84 Comparison of lymphocyte subsets of all nephrotic syndrome patients (regardless of histology and clinical condition) with age, sex and race matched controls

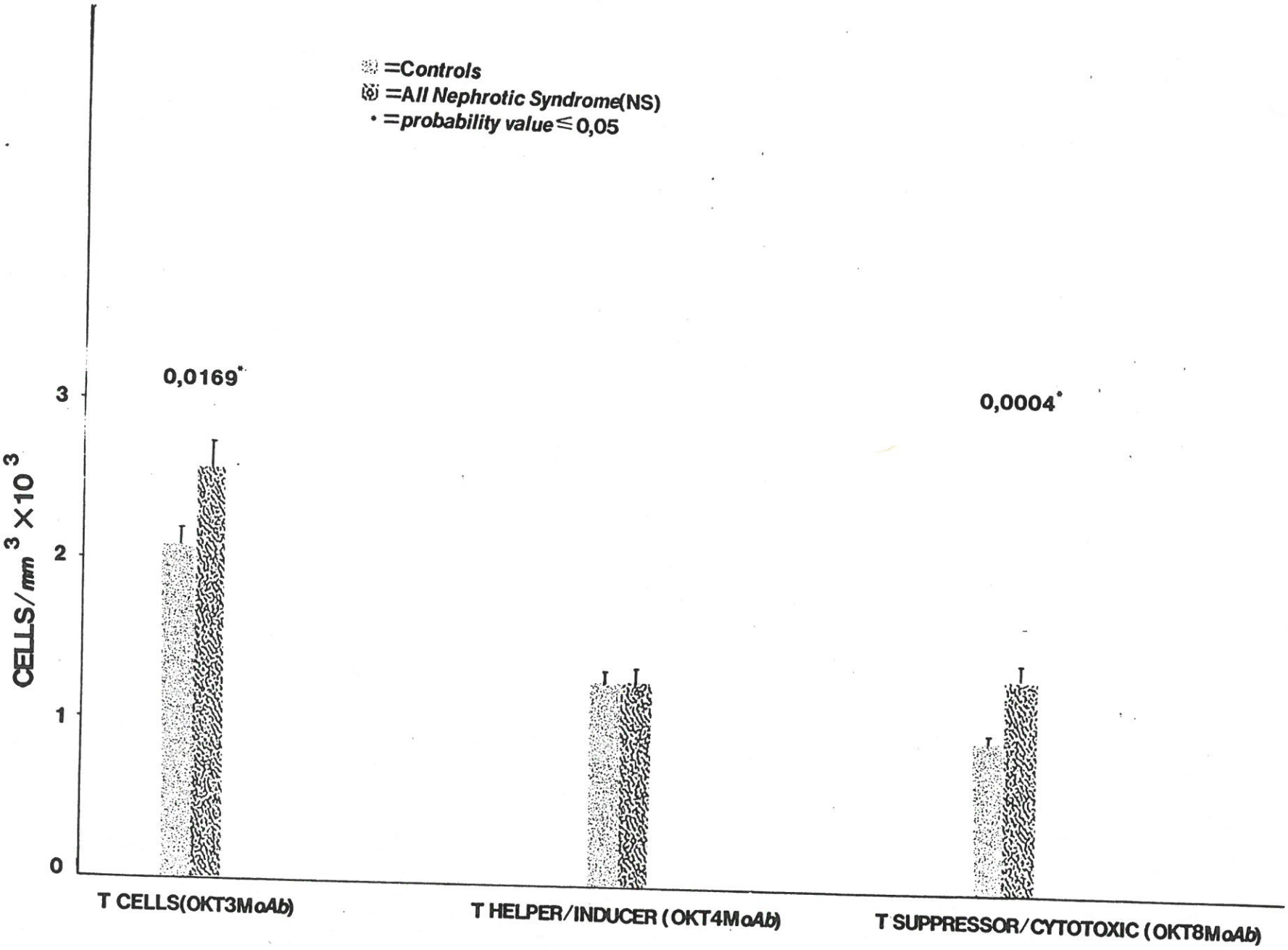


Figure 85 Comparison of proportions of lymphocyte subsets of all nephrotic syndrome patients (regardless of histology and clinical condition) with age, sex and race matched controls

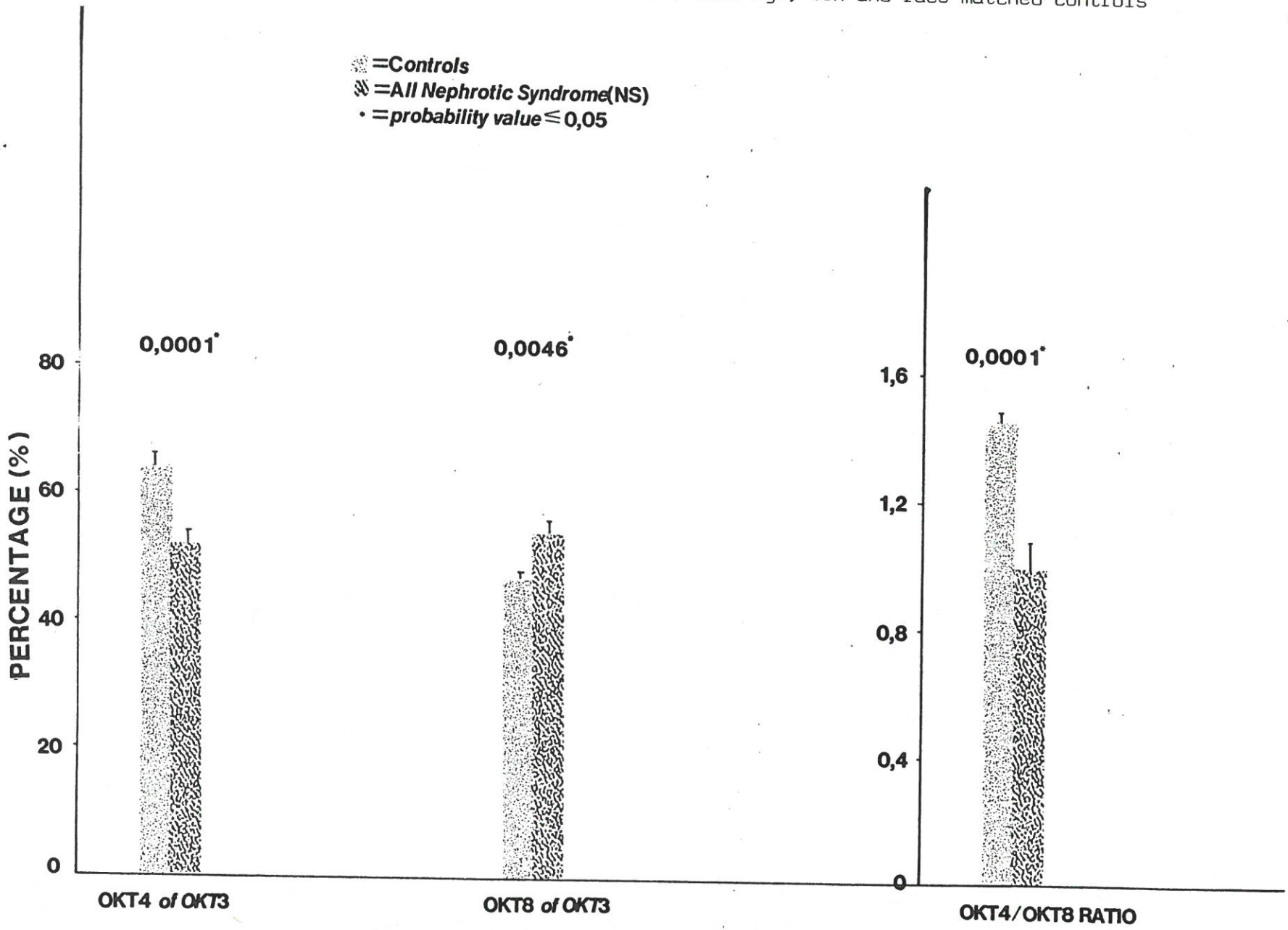


Figure 86 Comparison of suppressor cell activity and MNC PWM stimulation of all nephrotic syndrome patients (regardless of histology and clinical condition) with age, sex and race matched controls

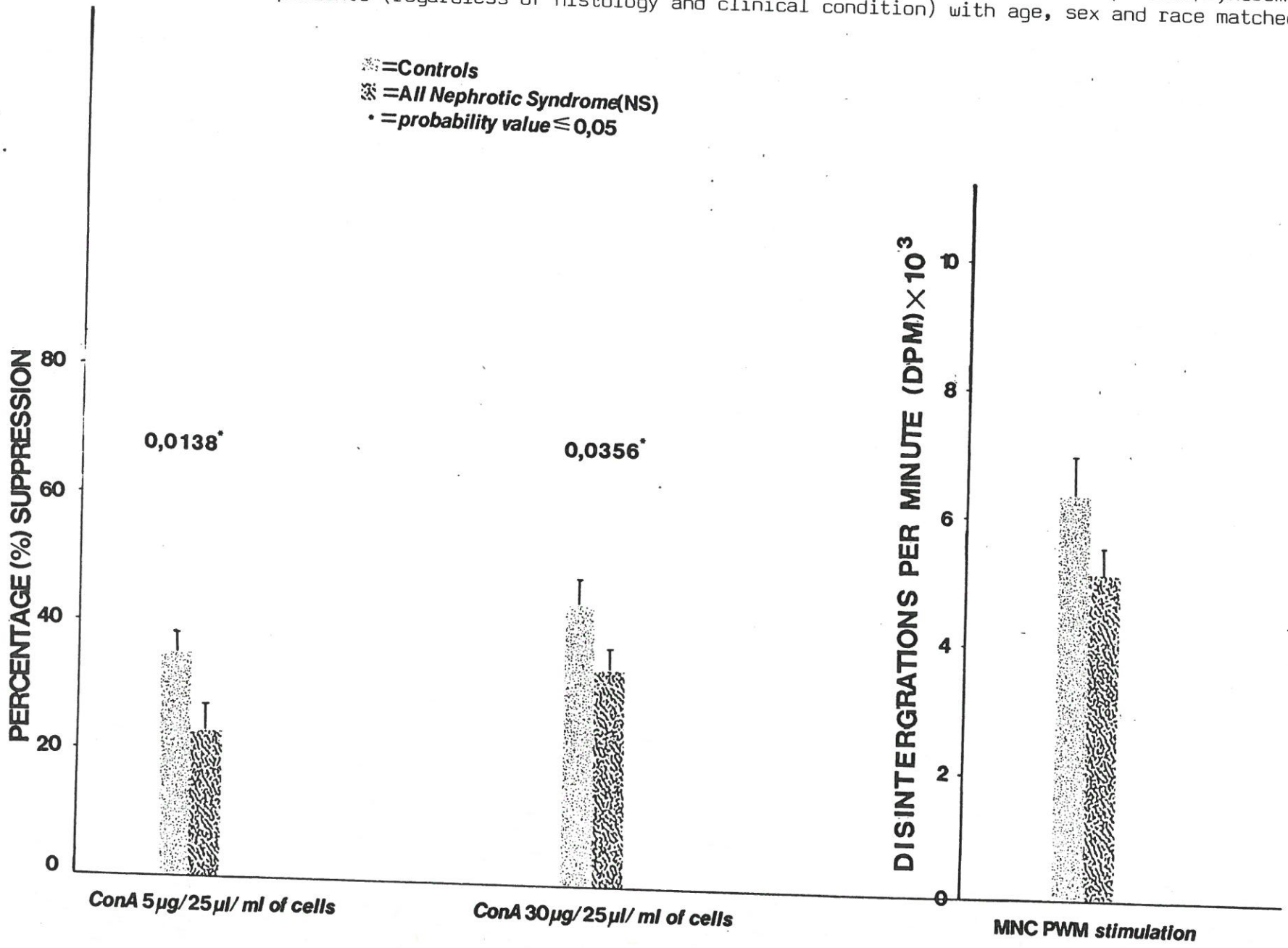
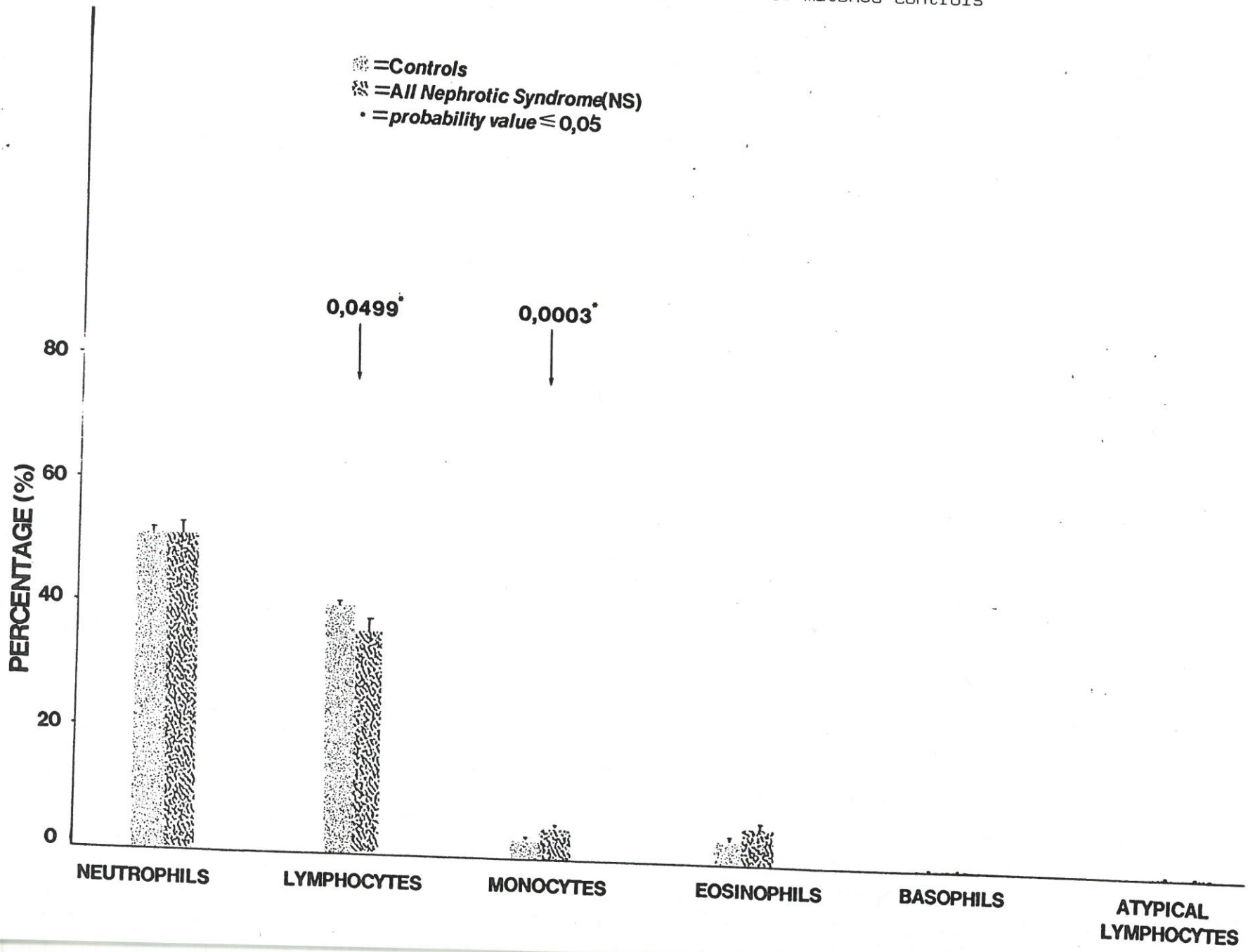


Figure 87 Comparison of the differential count of all nephrotic syndrome patients (regardless of histology and clinical condition) with age, sex and race matched controls



F. Relationship of Infection on the Immune Response

An index of infection was obtained for all nephrotic syndrome patients according to histology where:

$$\text{Index of Infection} = \frac{\text{Number of previous infections (severe and/or mild)}}{\text{Length of time of nephrotic syndrome}} \text{ /per patient}$$

This index of infection was related to certain immunological parameters.

Note: Table 55 indicates which infections were classified as mild or severe.

F.1 Immunological Parameters (Figures 88-89)

In MCNS the index of mild infection was significantly positively correlated to the number of: white cells ($r=0,572$ $p\leq 0,001$); and T cells (E-rosettes) ($r=0,389$ $p=0,049$). In the membranous, proliferative and miscellaneous groups of nephrotic syndrome none of the immunological parameters studied were related to any indices of infection ie mild or severe alone or combined. The index of infection did not correlate with certain clinical parameters such as oedema and serum albumin for any of the histological groups studied.

F.2 An index of relapse was obtained for all nephrotic syndrome patients according to histology where:

$$\text{Index of Relapse} = \frac{\text{Number of previous relapses}}{\text{Length of nephrotic syndrome}} \text{ / per patient}$$

This index of relapse was related to certain immunological parameters.

F.3 Immunological Parameters (Figures 90-92)

The index of relapse in MCNS, was significantly positively correlated to the numbers of: White cells ($r=0,634$ $p=0,001$); Null cells ($r=0,524$ $p=0,005$); and T suppressor cytotoxic cells ($r=0,443$ $p=0,013$). In the membranous, proliferative and miscellaneous types of nephrotic syndrome, none of the immunological parameters studied related to the index of relapse.

Table 55

Classification of Previous Infections

Severe	Mild
Mumps	Upper respiratory tract infection
Measles	Impetigo
Pneumoccocal infection	Scabies
Pneumonia (Klebsiella)	S.haematobium in the urine
Chicken pox	Tonsillitis
Abdominal pain	Pyoderma
Peritonitis (either pneumoccocal or pseudomonas or both)	Herpetic oral lesions
Pulmonary Tuberculosis (TB)	Angular stomatitis
Jaundice	Otitis media
Gluted boils	
Pelvic abscess	
Hepatic schistosomiasis	
Abdominal abscess	
Osteo necrosis	
Typhoid	

Figure 88 Relationship between white cell count ($x10^9/L$) and index of mild infection in MCNS

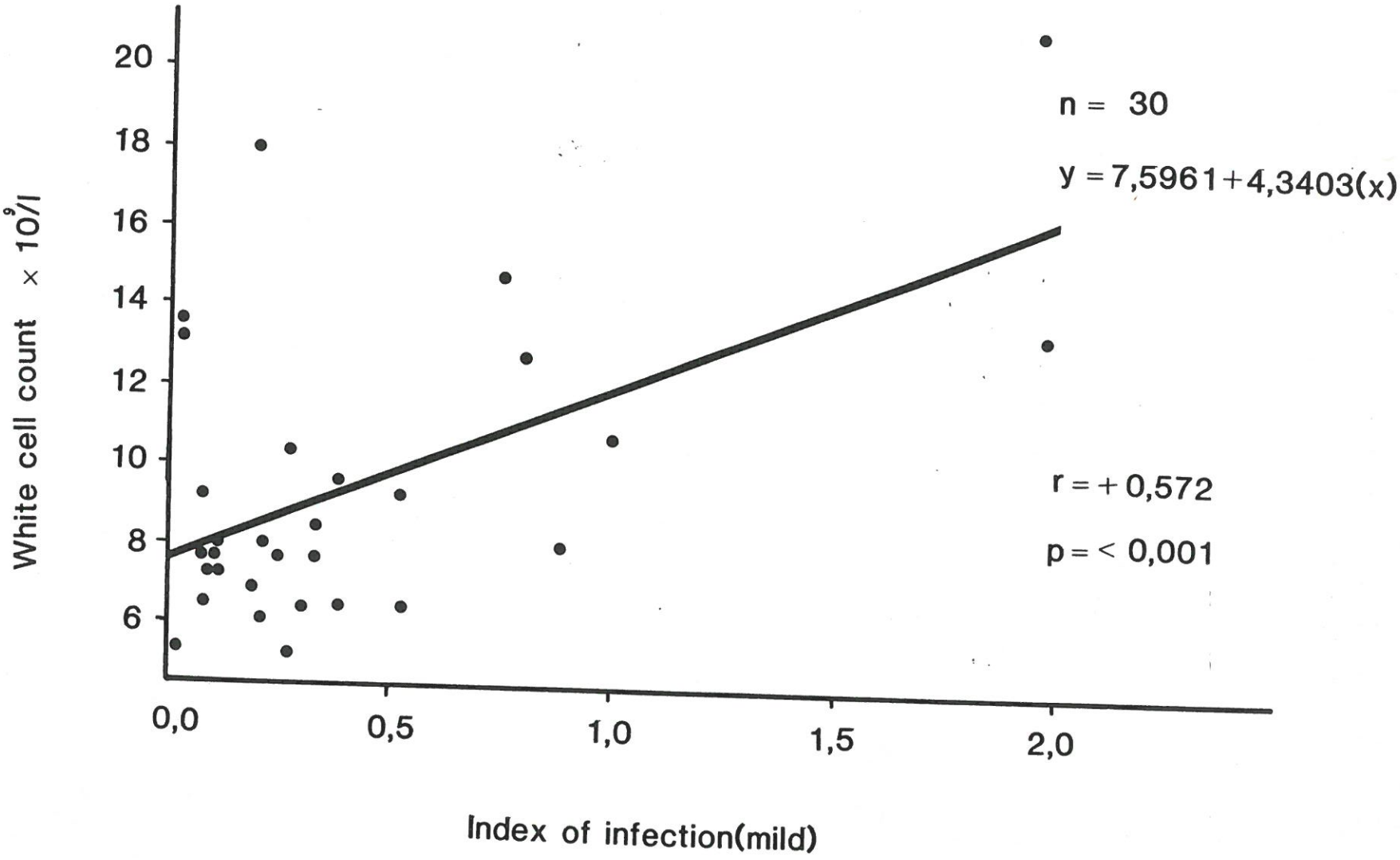


Figure 89 Relationship between T cells (E-Rosette)cells/mm^3 and index of mild infection in MCNS

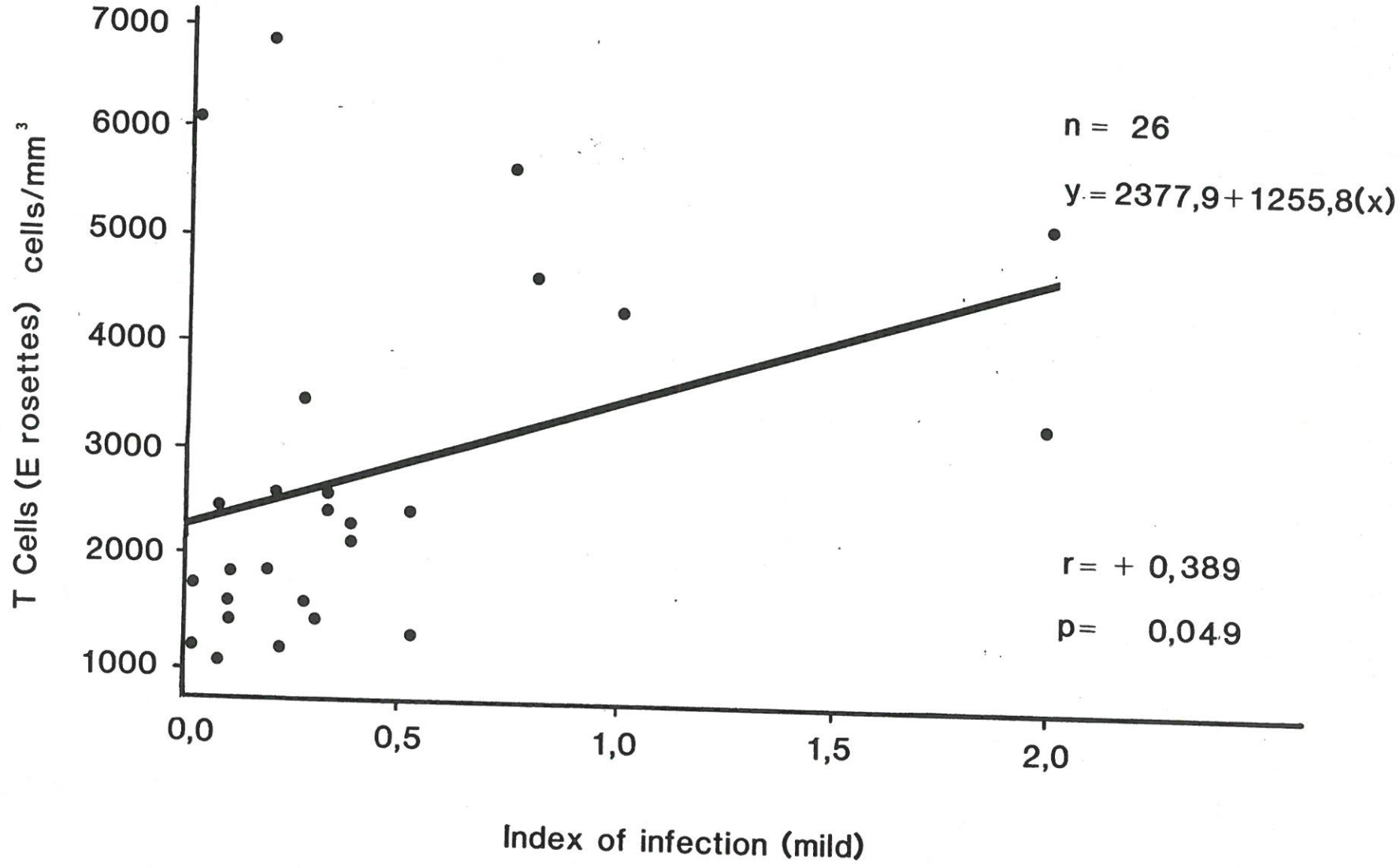


Figure 90 Relationship between white cell count ($x10^9/L$) and index of relapse in MCNS

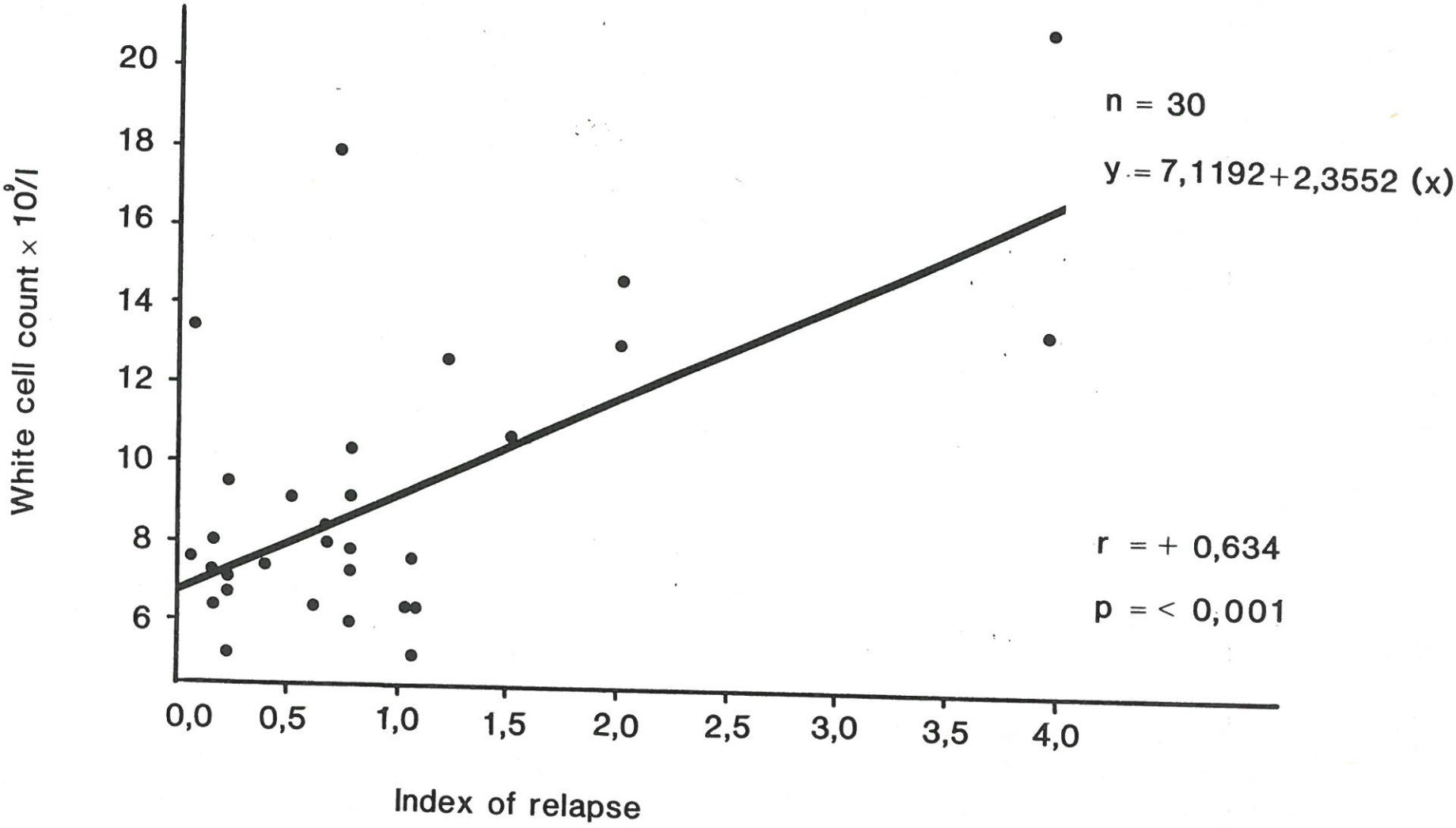


Figure 91 Relationship between Null cells/mm³ and index of relapse in MCNS

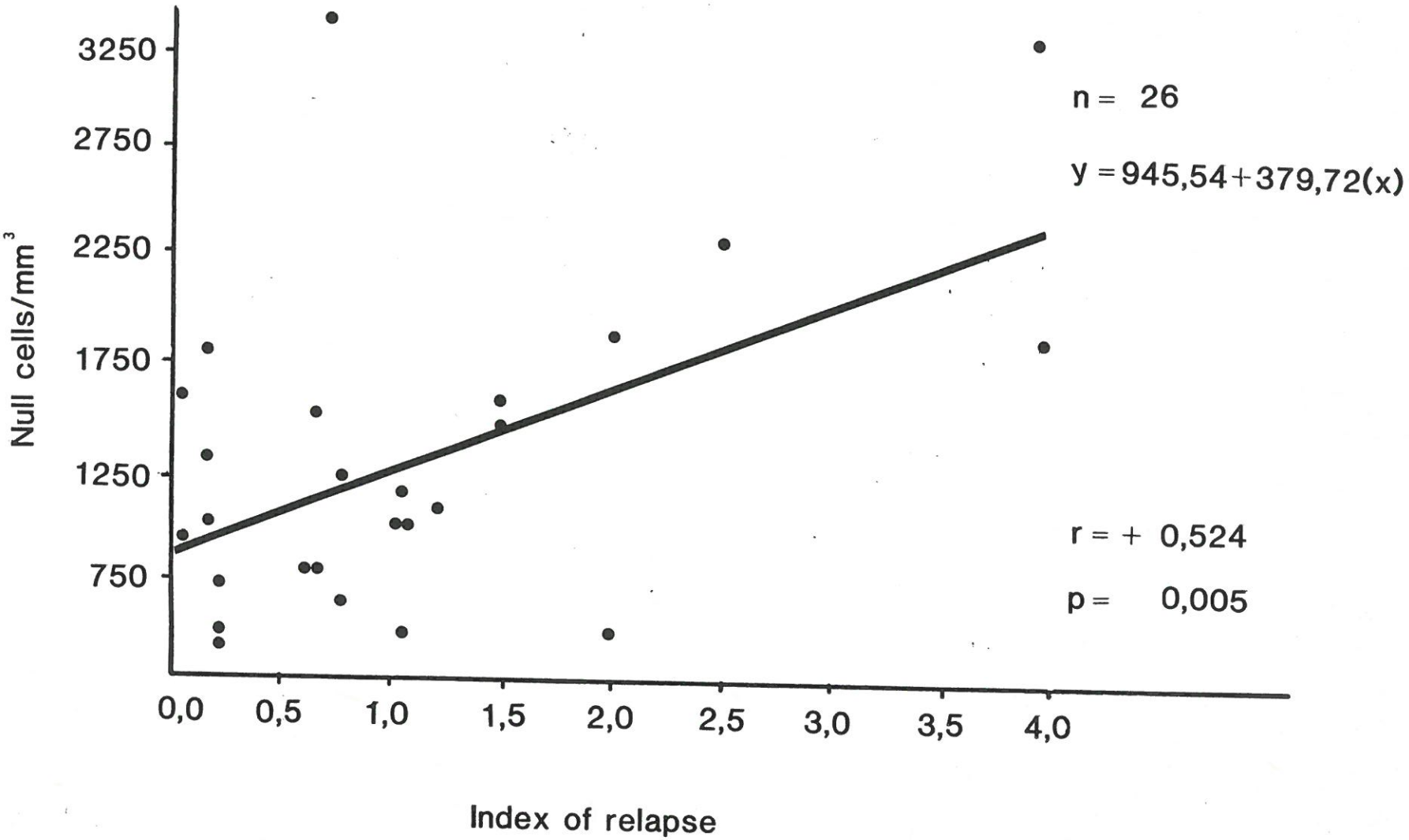
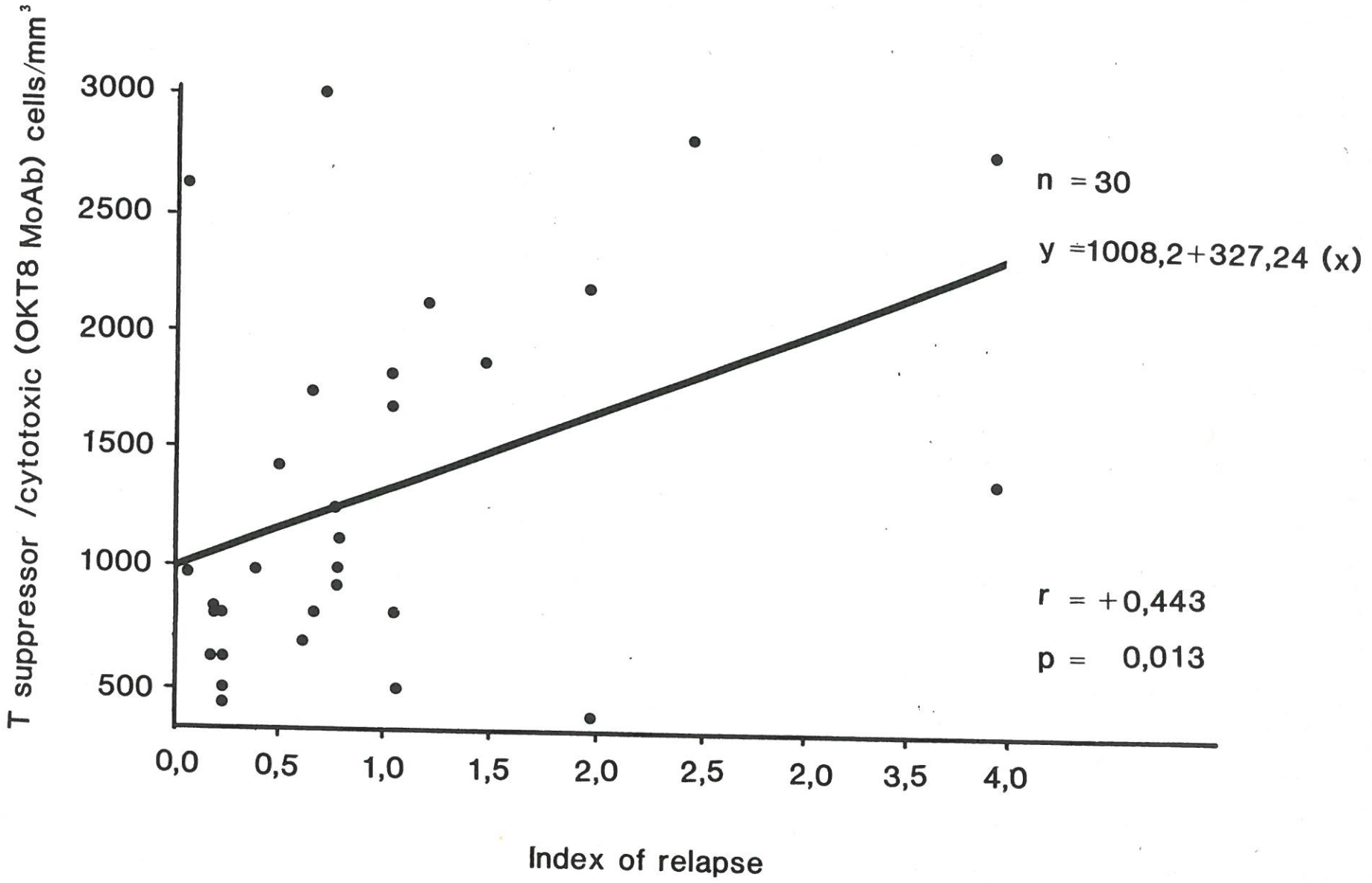


Figure 92 Relationship between T suppressor/cytotoxic cells/mm^3 and index of relapse in MCNS



G. Correlations of tests

G.1 Correlations between numerical and functional assays of suppression and MNC PWM stimulation within each histological group of nephrotic syndrome were sought.

G.1.1 Immunological Parameters

No correlations were observed between:

(a) the number of $OKT8^+$ cells and percentage T cell suppression both by ConA 5µg and ConA 30µg/25µl/ml of cells;
(b) the number of $OKT4^+$ cells and MNC PWM stimulation;
(c) the OKT4/OKT8 ratio with percentage T cell suppression both by ConA 5µg and ConA 30µg/25µl/ml of cells;
(d) the OKT4/OKT8 ratio with MNC PWM stimulation;

for any of the histological groups of nephrotic syndrome.

Summary of Findings (Table 56)

A. The Effect of Remission versus Relapse in MCNS

1) Children in relapse had reduced T4/T8 ratio due to increased T cells comprised of $T8^+$.

2) Partial remission was similar to relapse.

B. The Effect of different histolical groups on Immune response

1) A reduced T4/T8 ratio was found in two of the histological types viz. MCNS and Membranous NS.

2) Immunoregulatory cells are abnormal not only in MCNS but also in other histological types of nephrosis.

Table 56 Summary of Immunological Parameters

Histology	Condition	WCC	Abs.Monos	B cells	T cells (E-rosette + OKT3 MoAb)	$OKT4^+$ cells	$OKT8^+$ cells	% OKT4 of OKT3	% OKT8 of OKT3	Ratio OKT/OKT8	% Suppression (ConA)
All Nephrotic patients	RM+R	↑	↑	↓	↑		↑	↓	↑	↓	↓
MCNS vs Respective controls	RM			↓					↑	↓	
	R							↓		↓	
Membranous vs Respective controls	RM+R	↑	↑	↓	↑		↑			↓	
	R	↑	↑	↓	↑		↑			↓	
Proliferative vs Respective controls	RM+R							↓			
	R							↓			
Misc. vs Respective controls	RM+R			↓							
	R			↓							

Abbreviations: RM = Remission
R = Relapse + Partial remission
↑ = Increased
↓ = Decreased

3) No differences were detected when controls or HBs antigen carriers with no renal disease were independently compared to membranous nephrotic syndrome.

C. The Effect of Previous Treatment

Only one patient had been previously treated with P + C + C, hence comparisons with patients previously treated with P alone could not be done.

D. The long term effect among children in remission

There was gradual reduction in $T8^+$ cell numbers and persistence of B cell lymphopenia after 5 years of remission.

E. The Effect of Nephrotic Syndrome on the Immune Response

1) Patients with nephrotic syndrome considered as a group had abnormalities in immunoregulatory cell numbers and function.

2) This is a T cell defect due mainly to T suppressor cells.

F. Relationship of Infection on the Immune Response

Indices of previous mild infections and relapses were related to immune deficiencies , particularly in MCNS.

G) Correlation of Tests

No correlations were observed between number and function of immunoregulatory cells.

DISCUSSION

It is widely believed that immune complexes, formed either in the circulation (Wilson, 1981) or in situ (Causer and Salant, 1980), are central to the immunopathogenic mechanisms responsible for glomerular disease. "Cellular immunological mechanisms" it has been recently noted (Hoedemaker, 1983), "do not play an important role in the pathogenesis of glomerular lesions". It is acknowledged however that in MCNS, cell mediated immunity might contribute to increased permeability of the glomerular filter. A few studies have implicated T cells in the development of MCNS (Shalhoub, 1979; Mallick, 1977; Lin, 1985) although circulating immune complexes have also been detected in this syndrome (Levinsky et al., 1978). The main findings in this report suggest the strong probability that immunoregulatory cells are affected, not only in minimal change but also in other histological types of nephrosis.

1) Numerical Assays

a) Lymphocyte subpopulations

A reduced T4/T8 ratio was present in two of the histological groups studied viz. MCNS and Membranous NS. This might therefore be a feature of the nephrotic syndrome itself. This reduction was primarily due to an increase in the $T8^{+}$ subset though it was occasionally caused by a diminution in the percentage of $T4^{+}$ cells. Another frequent but unexpected finding was a decrease in the number of B cells. The majority of patients studied were in relapse during which phase these abnormalities were noted. What was equally interesting was that the lower T4/T8 ratio and B cells were evident during the clinically quiescent stage of MCNS. The clinical associations of the abnormalities in immunoregulatory cells could be best explored in MCNS as this was the only group in which there were sufficient numbers of children in remission and in relapse. A similar degree and type of immunological derangement was also documented in

membranous nephrotic syndrome; changes were more restricted in the Proliferative and Miscellaneous groups. These results probably indicate that dissimilar immunopathological processes underpin the different types of glomerular injury. It is important to note that certain intragroup difference (e.g. T8 cell numbers between remission and relapse in MCNS) were not detected when comparisons were drawn between normal controls and patients in relapse or in remission. This implies that such differences are subtle and the values occur at the extremes of the normal range during the active and inactive phases of the disease.

The detection of an increase in T8 cells leading to a decreased T4/T8 ratio in MCNS during relapse is in accordance with the findings of other workers (Feehally et al., 1984; Lin, 1985). On the other hand Gupta and Yuceoglu (1985) did not find any difference between numbers $T\gamma$ and $T\mu$ cells in MCNS patients in remission or relapse compared to controls. Their results however cannot be strictly compared to ours as the tests for the identification of T cell surface antigens differ; the use of monoclonal antibodies as in the current study gives more reliable results. The diminution in the T4/T8 ratio and in the numbers of B cells recorded during remission in MCNS differs from the findings of similar studies (Feehally et al., 1984; Lin, 1985). Feehally et al. investigated patients whilst they were on cyclophosphamide therapy, our patients were not on this treatment. This may in part account for these dissimilarities. The unmasking of immunological abnormalities during remission raises the question of whether this is a period of complete quiescence due to cessation of processes harmful to the kidney. The evidence suggests that remission is a state of reduced but not absent activity. Children with minimal change in remission continue to have plasma inhibitory factors which inhibit lymphocyte transformation (Fodor et al., 1982; Chapman et al., 1982), remain prone to severe infections resulting sometimes in death (Adhikari, 1981) and up to 46% have persistent biochemical abnormalities (Zilleruelo et al., 1984). Our findings suggest further that the immunological flaws detected in remission are not long

lasting: children who had been in remission for more than 5 years were virtually normal with significant diminution in T8 cells compared to those who had been in remission for less than 5 years. However, the B cell lymphopenia persisted when children in remission (> 5 years) were compared to controls. We do not know whether this abnormality is reversed later or is a permanent and fundamental defect in nephrosis.

The reduction in B cell numbers which was present in most patients may be caused by a number of factors. It may be an artifact of the test system in which the detection of surface immunoglobulins is compromised by interfering compounds such as the lipids present in nephrotic sera or the large variety of soluble factors released by suppressor cells. The B lymphopenia during remission may be accounted for by persistent biochemical abnormalities. The protracted duration of the B cell defect and the reduction in T8 cell numbers in long term remission make it less likely that this phenomenon is produced by suppressor T cells inhibiting B cell maturation as occurs in primary immunodeficiencies (Waldmann et al., 1976). The likelihood that the B cell defect is critical to the pathogenesis of nephrosis bears investigation especially in view of the fact that B cell numbers were low even after 5 years remission. The above observation on reduced numbers of B cells is complemented by previously reported work which showed depressed in vivo (Cathcart, 1981) and in vitro (Heslan and Loutie, 1982) function of these lymphocytes. The normal PWM transformation in this study suggests that the proliferative response of immune cells which precedes immunoglobulin release is normal; this proliferation may be due to B and/or T cells.

The increase in T8 cells might be due to more suppressor lymphocytes. This could be a compensatory mechanism to dampen excessive activity by a set of cells (T cells, B cells or macrophages) stimulated by antigen to produce glomerular damaging substances. The analogous but not identical situation is the immonosuppression of tissue damaging processes observed in chronic infections (Stobo et al., 1976). On the other hand the T8 cells could be cytotoxic cells releasing lymphokines

which increase glomerular permeability. One such lymphokine may be VPF. In the experimental situation of nephritic guinea pigs, sensitised lymphocytes can migrate to the kidney and participate directly in cytotoxic reactions (Nielson and Phillips, 1979). Lastly the T8 lymphocytosis may be a mere epiphenomenon, a distant response to some primary event such as the formation of immune complexes.

It is evident from the results that disturbances of cell mediated immunity are not restricted to MCNS but are also significant in membranous NS; the changes are more limited in the other nephropathies. Accordingly the argument that cellular immunological responses may be important in MCNS alone and not in other histological groups needs to be reconsidered. This has been previously suggested (Taube et al., 1984). Membranous nephrotic syndrome among black South African children is most often due to infection of a genetically susceptible individual by the hepatitis B virus (HBV) (Adhikari et al., 1985). Elimination of the virus cures the disease (Yamashita et al., 1983). As immunological parameters in the HBs antigen carriers without nephropathy were similar to those with nephropathy (apart from B cells) it follows that the presence of nephrotic syndrome does not affect the immunological deviation from normal of HbsAg infected individuals. To detect whether HBsAg has a particular effect in membranous nephropathy, a comparison between the HBsAg positive and HBsAg negative membranous nephropathy would have to be made. This was not possible as all patients except one were HBsAg carriers. In the parallel situation of HBV chronic active hepatitis, it has been suggested that an increase in T8 cells is at the centre of immunologically damaging mechanisms (Eddleston and Williams, 1974). It is worth noting that the most marked changes in immunoregulatory cells were found in the two disorders (MCNS and HBV membranous nephrotic syndrome) which are homogeneous among the populations studied in terms of histology, response to therapy, long term outcome and probably in triggering mechanisms (Wiggelinkhuizen and Sinclair-Smith, 1987). Proliferative and miscellaneous nephropathies represent a heterogeneous group of disorders united mostly by a common clinical presentation; this diversity probably obscures any uniform changes in immunoregulatory cells, assuming that such changes do occur.

When all nephrotic syndrome patients (regardless of remission or relapse) were compared to controls these differences remained. This overall impression however, obscures the differences between MCNS and the other three groups and between those in remission and those in partial remission or relapse. The comparison is valid in that it conveys the totality of immune abnormalities detected in nephrotic syndrome in this study. The findings would be influenced by the fact that the majority of patients (46 out of 68) were in relapse. A recent report by Lin and Hsu (1986) would confirm our findings. The two patients they studied had an increase of $OKT8^+$ cells and NK cells in relapse and a decrease of these cells in remission.

2) Functional assays

a) T suppressor cells

T suppressor cell function has been shown to be decreased in all types of nephrotic syndrome (Taube et al., 1984) but this is not a consistent finding. Normal ConA-induced suppressor cell function has been reported by Feehally et al. (1984) in children with MCNS in long term remission; Taube et al. (1981b) in children with MCNS not treated with cyclophosphamide and Gupta and Yuceoglu (1985) in three out of nine patients with MCNS. However Wu and Moorthy (1982) and Matsumoto et al. (1984b) have found increased ConA-induced suppressor cell activity in patients with MCNS. These discrepancies could be due to the different methods employed in these studies. ConA inducible T suppressor cell function in this study, was found to be lower, only when all histological groups were taken together and patients included regardless of remission or relapse as compared to controls. This difference fell away when individual histological groups were compared to their respective controls. This discrepancy between this and other studies could also be due to the different methods employed. It would also appear that depressed suppressor cell function occurs in nephrosis but requires a sizeable number of patients/control comparisons for the abnormality to be

demonstrated.

This latter observation was confirmed by looking at individual values for T suppressor cell function. At the sub-optimal level of ConA ie. 5µg/25µl/ml of cells, 9 patients (3 with MCNS, 3 with extra membranous nephropathy, 1 with proliferative and 2 with miscellaneous nephrotic syndrome) showed enhancement whereas only 4 of these same 9 patients(1 with MCNS, 1 with extra membranous and 2 with miscellaneous nephrotic syndrome) showed enhancement with the optimal level of ConA viz. 30µg/25µl/ml of cells.

b) MNC PWM stimulation

In vitro production of IgG PWM stimulation of mononuclear cells has been found to be decreased in patients with nephrotic syndrome in relapse compared to controls (Heslan et al., 1982). MNC PWM stimulation in all our patients was not adversely affected; neither histological grouping nor clinical condition had an effect. Although B cell numbers were found to be decreased (mentioned under Numerical assays), particularly in children with MCNS in remission and membranous nephrotic syndrome, this did not correlate with the functional assay we employed. It could be argued, that MNC PWM stimulation is a complex test measuring the functions of antigen presenting cells, T helper and B cells. We did not measure the final product ie. immunoglobulins and therefore these results merely suggest that the proliferative response of these cells may be normal.

3) The Effect of previous Treatment

There is conflicting evidence about the effect of steroid treatment in nephrotic syndrome. Taube et al. (1981b) found that cyclophosphamide treatment had a long term impairment of T suppression cell function in MCNS (up to 12 years mean 6,5 years) whereas Feehally et al., 1984 reported a transient defect of $T4^+$ cells up to 6 months after cyclophosphamide treatment.

Our attempts to elucidate whether prednisone on its own or in

combination with other drugs had any effect on the parameters studied failed as only 1 patient had previously been treated with prednisone + cyclophosphamide + chlorambucil.

4) The Effect of Infection and Relapse on the Immune Response

The critical event which precipitates relapse in nephrosis is usually not discernible though mild infections are often held to be responsible. Our results show that the rates of infection and of relapse correlate with white cells and T cells. This would support the commonly held view that infections tip the scales toward increased glomerular permeability and overt disease.

The findings in MCNS that certain lymphocyte subpopulations, particularly T cells comprising mainly of OKT8$^+$ cells confirms further, both in this study and as in others, that T cells have a central role in the pathogenesis of MCNS. The similarity in immunological parameters which were related to relapse as well as in infection was not surprising, as in the clinical situation, almost without fail, when the patient gets an infection he/she relapses.

In MCNS, in remission it was also observed that there was an increase in the number of infections/per year over those MCNS children in relapse. However, although T8$^+$ cells have been shown to be important in MCNS, the additional effect of cytotoxic cells cannot be ruled out.

Although in membranous nephropathy abnormalities of the immune response were detected, these did not relate to infection. In proliferative and miscellaneous types of nephrotic syndrome, it seems that infection is not related to any immunodeficiency.

5) Correlation of tests

The reasons for the lack of correlation between the numerical and functional assays of suppression as detected even in this study

has been previously discussed. (See Chapter on Measles)

6) The Effect of HBs antigenaemia

In the African child, in Southern Africa it has been suggested that the interaction of the presence of HBs antigen together with a genetic predisposition is central to the pathogenesis of membranous nephropathy (Adhikari et al., 1985). This study confirmed this observation in that 13 out of 14 children who were all African in the membranous histological group were HBs antigen positive while all the Indian children in all 4 histological groups were HBs antigen negative.

An increase in $T8^+$ has been implicated as playing a pathogenetic role in HBs antigen positive chronic active hepatitis (Eddleston and Williams, 1974). An increase in T suppressor/cytotoxic cells were found in membranous nephropathy. However, as mentioned previously, the immunological abnormalities noted in membranous nephropathy are due to renal disease.

CONCLUSION

The results obtained in this study suggest that derangements of immunoregulatory cells may underpin abnormalities related to susceptibility to infection and the immunopathogenesis noted in the nephrotic syndrome of childhood.

The changes of immunoregulatory cells reported here need to be explored further. If these findings are confirmed the therapeutic implications are that it may be possible to find alternatives to the widely used drugs, such as steroids and cyclophosphamide, which have many serious side effects. A sustained effort must be made to minimise relapses by reducing the rate of infections in nephrotic children.

B CHRONIC RENAL FAILURE (CRF)

AIM

This study was undertaken to examine the immunoregulatory changes in patients who proceed to chronic renal failure (CRF). All patients bar two adolescents, were adults. I will not elaborate too much on historical and other details (as previously) but mention them briefly and concentrate mainly on the immunological findings.

SUMMARY

CHRONIC RENAL FAILURE (CRF: SUPPRESSOR CELLS ASSAYED BY NUMERICAL AND FUNCTIONAL TESTS IN CHRONIC RENAL FAILURE).

Suppressor cells were assayed by numerical and functional tests in adults on chronic haemodialysis. Peripheral blood mononuclears (PMB) were classified as total T cells by E-rosettes and by the monoclonal antibody OKT3, as T cell subsets by OKT4 (inducer/helper T cells) and OKT8 (cytotoxic/suppressor T cells) and as B cells by the presence of surface immunoglobulin. The suppressive effect of PBM pretreated with either concanavalin A (ConA), sodium periodate, or serum rich in immune complexes, on normal homologous phytohaemagglutinin (PHA) lymphocyte transformation, was determined. Usual tests of T cell function were not done. T lymphopenia was due to significant diminution ($p \leq 0,0002$) in numbers of $OKT4^+$ cells in patients (516 $\pm$ 44 cells/mm^3 mean $\pm$ SEM) as compared to controls (906 $\pm$ 96 cells/mm^3). The number of $OKT8^+$ cells in patients was not different from normal although their percentage (45 $\pm$ 4%) was slightly higher than controls (36 $\pm$ 5%) ($p \leq 0,10$). Suppressor activity using only a suboptimal dose of ConA (5µg/ml), was significantly lower ($p < 0,002$) in uraemic patients (36 $\pm$ 12%) than in controls (67 $\pm$ 7%). An important finding was that no significant correlations were detected between the numerical and functional assays of suppression used or between any of these immunological tests and biochemical parameters studied. The implications of these results for immunoparesis in uraemia are

discussed with particular reference to the discordance between marker and functional assays of suppressor cells.

INTRODUCTION

Uremia is known to be immunosuppressive (Lawrence, 1965; Dobbelstein, 1976; Mannick et al., 1960; Hanicki et al., 1979; Sengar et al., 1975; Harris and Sengar, 1975; Kunori et al., 1980; Dammin et al., 1957) and the clinical relevance of this is seen in the high incidence of infections and neoplasia (Lindner et al., 1981) recorded in these patients. The mechanisms responsible for this relative anergy are unknown and have been ascribed to many factors including unspecified uremic toxins and malnutrition.

Impaired cell mediated immunity in uremic rats accompanied by increased activity of suppressor cells (Raskova and Morrison, 1976) and of T-cell depletion associated with retention or even augmentation of suppressor cell activity in renal failure patients on hemodialysis (Guillou et al., 1980) suggest an important role for immunoregulatory cells in this disease.

Discordant results between different functional assays of suppression in autoimmure disease (Coovadia et al., 1981) indicate that there was, at the time the study was undertaken (1981-1982), no single satisfactory test to measure this component of the immune response. These facts prompted us to compare several functional suppressor cell assays and attempt to correlate these with numerical estimates of T-cell subsets using specific murine monoclonal antibody in a further attempt to elucidate the immunodeficiency in uremia. Conventional tests of T-cell function such as delayed hypersensitivity, cytotoxicity, help and suppression of immunoglobulin synthesis were not performed. As the uremic state is characterized by wide fluctuations in biochemical indices, and as some of these have been shown to have adverse effects on the immune response (Harwick et al., 1978), we also assessed the influence of serum levels of selected metal and nutrients on the tests of immunity studied in these patients.

MATERIALS AND METHODS

PATIENTS

Patient Details (Table 57, Figure 93)

Nineteen patients undergoing regular hemodialysis (4 to 5,5 hours, three times a week), at the Addington Hospital, Durban, were studied. They ranged in age from 15 to 63 years (mean, 35 years). There were ten White and nine Indian patients: 14 were male. Chronic renal failure was ascribed to chronic glomerulonephritis in 6 patients and analgesic nephropathy in four. Chronic pyelonephritis, malignant hypertension and Alport syndrome accounted two patients, while the remaining three patients had crescentic nephritis, systemic lupus erythematosus and polycystic kidney disease. The patient with crescentic nephritis was on daily oral doses of 20mg prenisilone and 150mg cyclophosphamide. One patient with glomerulonephritis had been successfully treated surgically for a hypernephroma, diagnosed at the time of presentation with CRF.

All blood samples were taken prior to hemodialysis and immediately before systemic heparinization. As patients were lymphopenic, all the tests discussed below could not be done on every individual. The exact number studied is given in the tables.

The patients were age-, sex-, and race-matched with 19 normal healthy volunteers.

METHODS

I Immunological

(i) T and B subpopulations
(This was undertaken as described under Methods)

(ii) T cell subsets using monoclonal antibodies.
(This was undertaken as described under Methods)

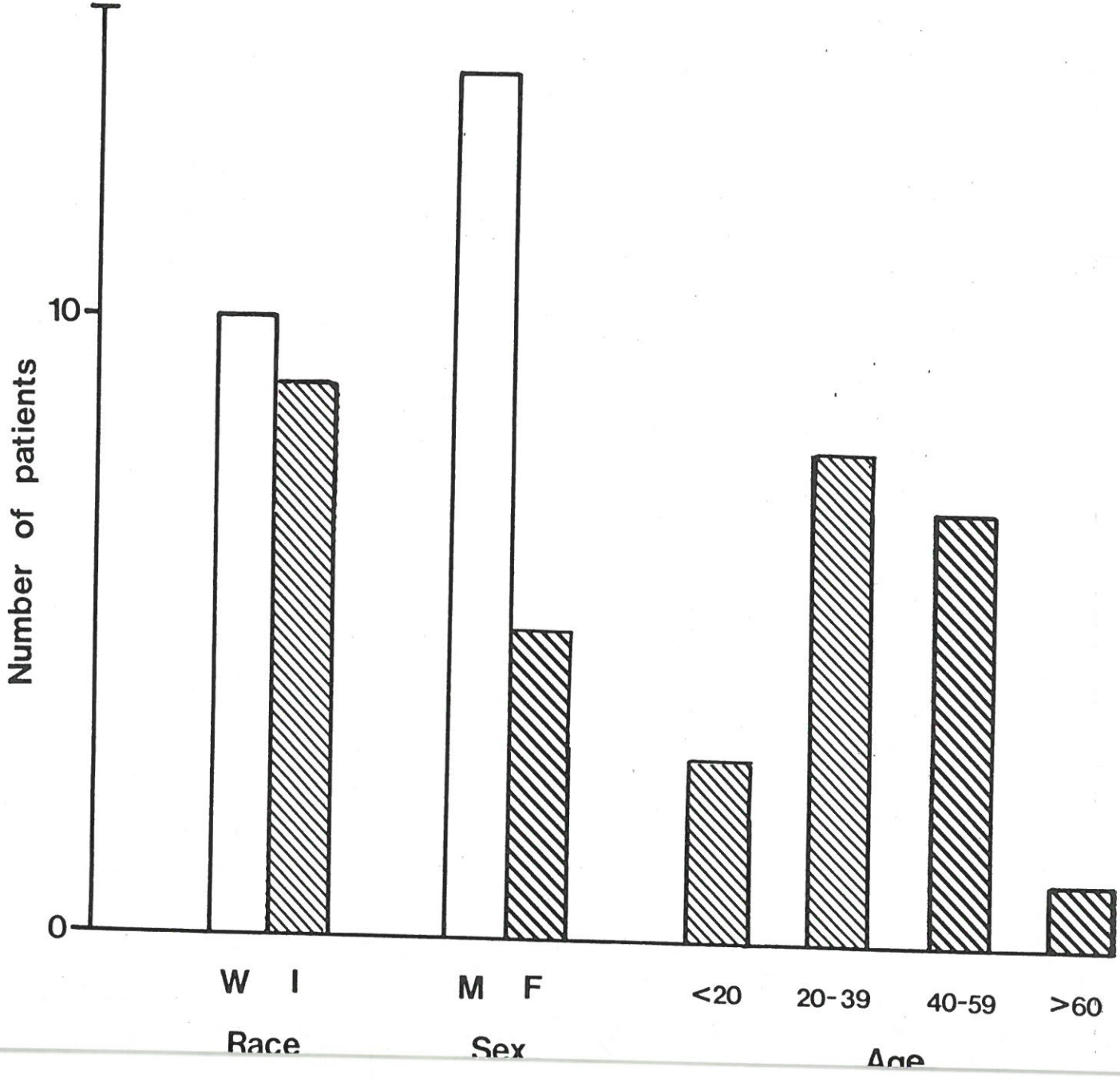
(iii) T suppressor cell function using ConA; $NaIO_4$ and circulating

Table 57 Patient details

Patient no.	Age years	Sex	Race	Duration of disease years	Plasma creatinine umole/ liter	Hyper-tension	Diagnosis Made by clinical, radiological or biopsy criteria
1	45	F	W	6	1155	No	Chronic pyelonephritis due to the vesico-ureteric reflux
2	33	M	W	8	1770	Yes	Chronic glomerulonephritis (renal biopsy)
3	29	M	1	1	1310	No	Membrano-proliferative glomerulonephritis (renal biopsy)
4	58	M	W	6	1131	No	Analgesic nephropathy
5	31	M	1	1	1020	No	Chronic glomerulonephritis (renal biopsy)
6	40	M	1	7	1310	No	Polycystic kidneys
7	52	M	1	2	1500	No	Analgesic nephropathy
8	17	M	1	1	1230	No	Alport syndrome
9	49	M	W	2	1219	No	Hypernephroma/Focal proliferative glomerulone-nephritis (renal biopsy)
10	28	F	1	2	756	No	Systemic lupus erythematosis
11	22	F	W	6	631	No	Chronic glomerulonephritis
12	54	M	W	1	1240	No	Analgesic nephropathy Renal biopsy = malignant Nephrosclerosis/marked proliferative endarteritis
13	47	M	W	5	1110	Yes	Malignant hypertension
14	15	M	1	2	775	Yes	Alport syndrome
15	22	M	W	2	1500	Yes	Rapidly progressive glomerulonephritis (renal biopsy)
16	63	M	W	2	1149	Yes	Chronic pyelonephritis and uteric valves (renal biopsy)
17	18	F	1	1	1390	Yes	Chronic glomerulonephritis (Renal biopsy)
18	21	F	1	2	1240	Yes	Malignant hypertension
19	30	M	W	2	1150	No	Analgesic nephropathy

Abbreviations: 1. Indian South African: W. White South African

Figure 93 Patient details



immune complexes.
(This was undertaken as described under Methods)

II Biochemical investigations

The following routine biochemical and haematological tests were done using standard techniques at the same time as the immunological assays on the peripheral blood of the uremic patients: urea and electrolytes; creatinine, calcium, phosphate, alkaline phosphatase, magnesium, uric acid, folic acid, serum iron and total iron binding capacity triglycerides, cholesterol, albumin, globulin, haemoglobin and white cell count. In addition, plasma zinc levels were estimated by atomic absorption (Hackley et al., 1968).

III Nutritional status

This was assessed by measurements of weight and height and by estimates of mid arm muscle circumference calculated from the mid-arm circumference and skin fold thickness, using a Harpenden skin-fold caliper (Jelliffe, 1966).

Statistical Analysis

Results were analysed using the non-parametric Mann-Whitney U test. The computed U statistic was tested at a 5% level of significance. Correlation analysis was performed by testing the linear correlation coefficient at a 5% level of significance.

RESULTS

Nutritional indices. The hemodialysis patients were found to have an adequate level of nutrition.

Biochemical Investigations (Table 58)

None had a serum albumin level below 30 g/liter; only two had serum folate levels below 5 mg/ml and percentage transferrin saturation less than 15%, and three out of 14 patients who had estimations of mid-arm muscle circumference had results below 80% of the standard (Jelliffe, 1966).

Numerical assays. The results of these assays are set out in Table 59, Figures 94-97) The patients with uremia had a significant absolute lymphopenia of total PBM as well as a T cell lymphopenia as measured both by the E-rosette technique and the monoclonal antibody $OKT3^+$. There was significant positive correlation between the total T cell population measured by these two techniques in both the normal controls (sample correlation coefficient R=0,745 p=0,001) and in the patients with chronic renal failure (R=0,917 p=0,01).

The $OKT3^+$ population was consistently smaller than the E-rosette population in the controls. $OKT3^+$ and E-rosette cells formed 49 and 74%, respectively, of the total absolute mononuclear count. The $OKT3^+$ cells amounted to 67,0% of the E-rosette subset in the controls. Similarly, in patients, $OKT3^+$ cells accounted for 44% and E-rosette cells for 74% of the total mononuclear count.

Paralleling the T cell lymphopenia, there was an absolute B cell (SIg^+) lymphopenia in patients, while there was no difference in the Null cell counts between the patients and controls. The absolute number of $OKT4^+$ cells in the patients was significantly lower than the controls, but when these results were expressed as a percentage $OKT3^+$ cells, no difference was found. The absolute number of $OKT8^+$ cells was not significantly different from normals. However, the mean percentage of $OKT8^+$ cells was slightly higher in the patients than in controls (p=0,001). The results expressed as the ratio of OKT4 cells

Table 58 Biochemical investigations in uremic patients*

Subject group	Patients	Normal range
Urea	30,03 ± 1,78	2,50 to 6,50
Creatinine, umole/liter	1193,89 ± 62,20	53,00 to 97,00
Zinc, µg/ml	1,16 ± 0,05	0,70 to 1,00
Magnesium	1,21 ± 0,04	0,74 to 0,99
Calcium	2,17 ± 0,05	2,25 to 2,75
Phosphate	2,26 ± 0,14	0,81 to 1,45
Alkaline phosphatase /Uliter	153,50 ± 27,30	30,00 to 85,00
Uric acid	0,47 ± 0,02	0,25 to 0,42
Folic acid, ng/ml	14,10 ± 1,30	5,00 to 20,00
% Saturation transferrin	38,40 ± 5,90	> 15,00
Triglyceride	2,10 ± 0,26	0,34 to 1,69
Cholesterol	4,80 ± 0,52	3,89 to 6,48
Albumin g/liter	40,00 ± 0,85	38,00 to 48,00
Globulin g/liter	27,20 ± 1,28	20,00 to 32,00
Hemoglobin, g/dl	7,30 ± 0,24	12,00 to 18,00

*All results are expressed as the mean ± SEM and in millimoles per liter unless otherwise stated.

Table 59 Subpopulations of peripheral blood lymphocytes identified by E-rosettes, SIg, and monoclonal antibodies (OKT3, OKT4, OKT8) in patients on chronic hemodialysis and in controls

	Mononuclear cells in peripheral blood smear			Mononuclear cells defined by E-rosettes and surface Ig			T-cell subsets as defined by specific monoclonal antibodies					
Patient no.	Total lymphocytes	Total monocytes	Total mononuclear cells	T-cells (E-rosette)	B cells (SIg+)	Null cells	OKT3	OKT4	OKT8	Positive cells as % of OKT3 OKT4	Positive cells as % of OKT3 OKT8	Ratio OKT4/OKT8
1	2825	0	2825	1977 (70)	565 (20)	285 (10)	1214 (43)	757 (43)	805 (28)	62	66	0,94
2	1500	450 (6)[a]	1950	1462 (75)	292 (15)	176 (9)	1073 (55)	410 (21)	546 (28)	38	50	0,75
3	1600	80 (1)	1680	1350 (81)	235 (4)	68 (4)	823 (49	638 (38)	218 (3)	78	26	3,00
4	880	264 (3)	1144	915 (80)	82 (8)	114 (10)	400 (35)	389 (34)	137 (12)	97	34	2,85
5	2560	192 (3)	2752	2202 (80)	303 (11)	248 (9)	1101 (40)	798 (29)	716 (26)	72	65	1,10
6	322	46 (2)	368	ND	ND	ND	150 (41)	63 (17)	81 (22)	42	54	0,77
7	1716	220 (5)	1936	1258 (65)	484 (25)	194 (10)	774 (40)	426 (22)	406 (22)	55	53	1,03
8	670	0	670	563 (84)	54 (8)	53 (8)	355 (53)	328 (49)	73 (11)	92	21	4,49
9	1944	378 (7)	2322	1927 (83)	186 (8)	209 (9)	975 (42)	743 (32)	743 (3)	76	76	1,00
10	2106	270 (5)	2376	2091 (88)	214 (9)	71 (3)	1212 (51)	570 (24	760 (32)	47	63	0,74
11	1020	0	1020	857 (84)	82 (8)	71 (7)	469 (46)	389 (38)	183 (18)	83	39	2,12
12	1207	213 (3)	1420	944 (70)	199 (4)	710 (50)	454 (32)	412 (32)	412 (29)	64	58	1,10
13	770	275 (5)	1045	878 (84)	115 (11)	52 (5)	449 (43)	314 (30)	188 (18)	70	42	1,66
14	1005	268 (4)	1273	891 (7)	255 (20)	127 (10)	560 (44)	547 (43)	242 (19)	98	43	2,27
15	1407	0	1407	1041 (74)	197 (4)	169 (2)	475 (34)	427 (30)	240 (17)	90	51	1,78
16	1173	0	1173	891 (76)	176 (15)	94 (8)	738 (63)	689 (59)	177 (15)	93	24	3,89
17	1242	184 (4)	1426	913 (64)	343 (24)	171 (12)	699 (49)	713 (50)	161 (11)	102	23	4,43
18	1804	44 (1)	1848	1164 (63)	517 (28)	166 (9)	813 (44)	425 (23)	129 (7)	52	16	3,25
19	1650	220 (2)	1870	1085 (58)	486 (26)	299 (16)	542 (29)	729 (39)	299 (16)	135	55	2,45
All patients	1404± 146	222±31	1606± 153	1196±131	226±37	154±18	712±70	516±44	343±57	-	-	2,08± 0,28
	-	(4±0,50)	-	(80±2)	(15±2)	(9±0,80)	(45±2)	(34±3)	(20±2)	(76±6)[c]	(45±4)[c]	-
Controls	2428± 212	173±24	2600± 217	1915±182	498±45	121±14	1275±145	906±96	426±58	-	-	2,97± 0,40
	-	(3±0,40)	-	(75±2)	(15±2)	(9±0,80)	(46±2)	(34±2)	(20±2)	(83±9)	(36±5)	-
Probability values	<0,002	>0,10	<0,002	<0,002	<0,002	>0,10	<0,02	<0,002	>0,10	>0,10	>0,05	>0,10

Abbreviation: ND not done
[a] Absolute number (%)
[b] Mean ± SEM
[c] For absolute numbers except columns 11 and 12

Figure 94 Comparison between absolute lymphocyte count in controls and CRF patients

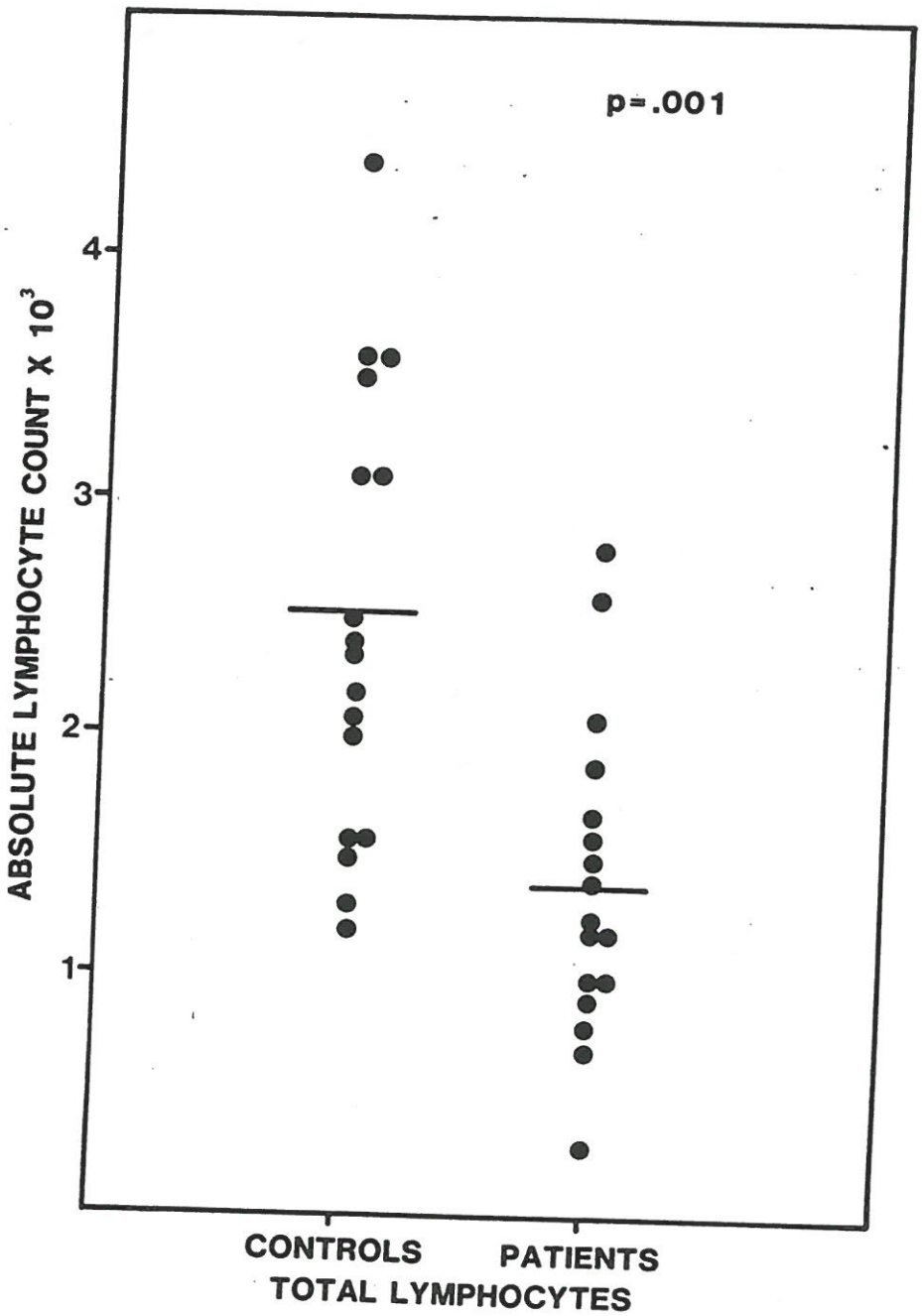


Figure 95 Comparison of T cell subsets between controls and CRF patients

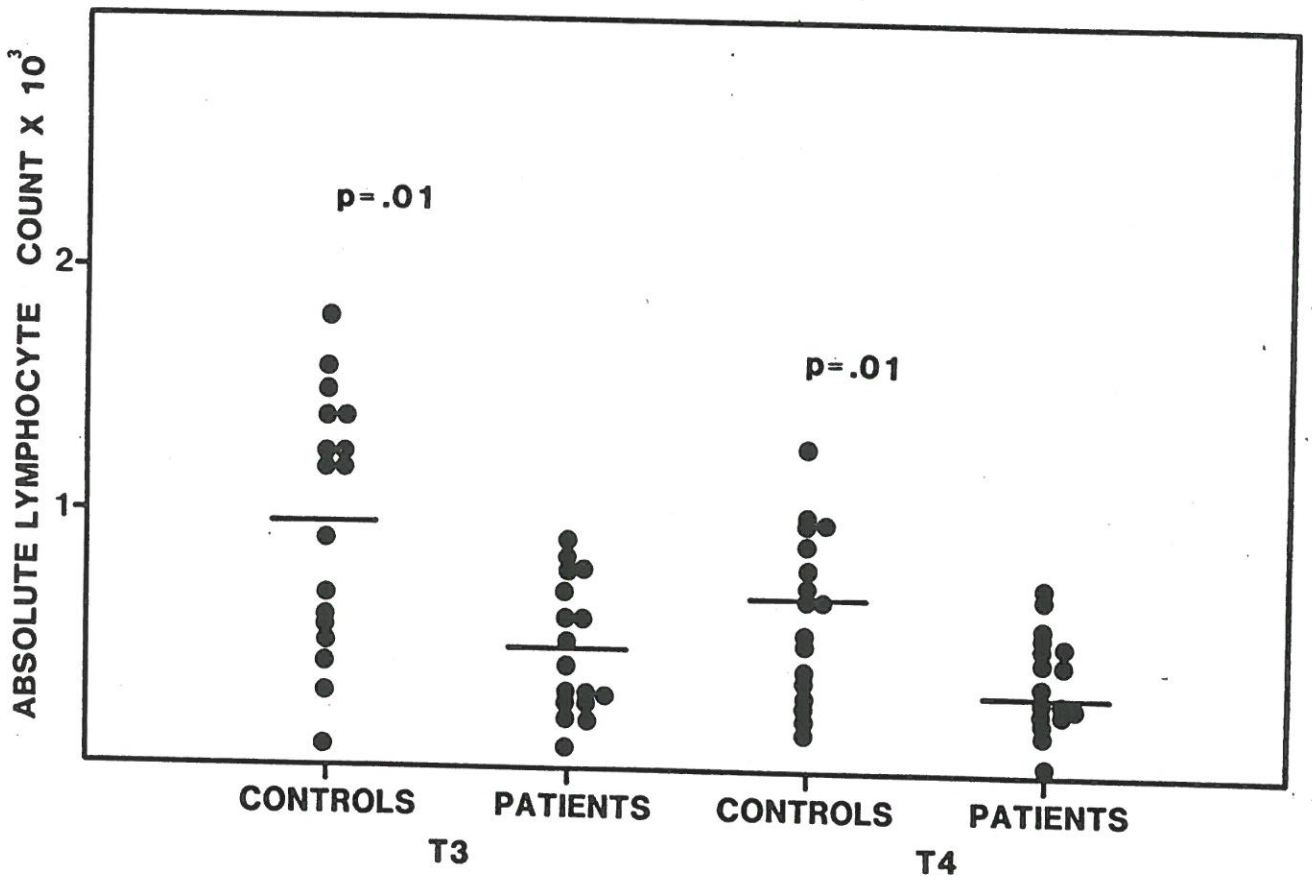


Figure 96 Comparison of proportions of $T8^+$cells between controls and CRF patients

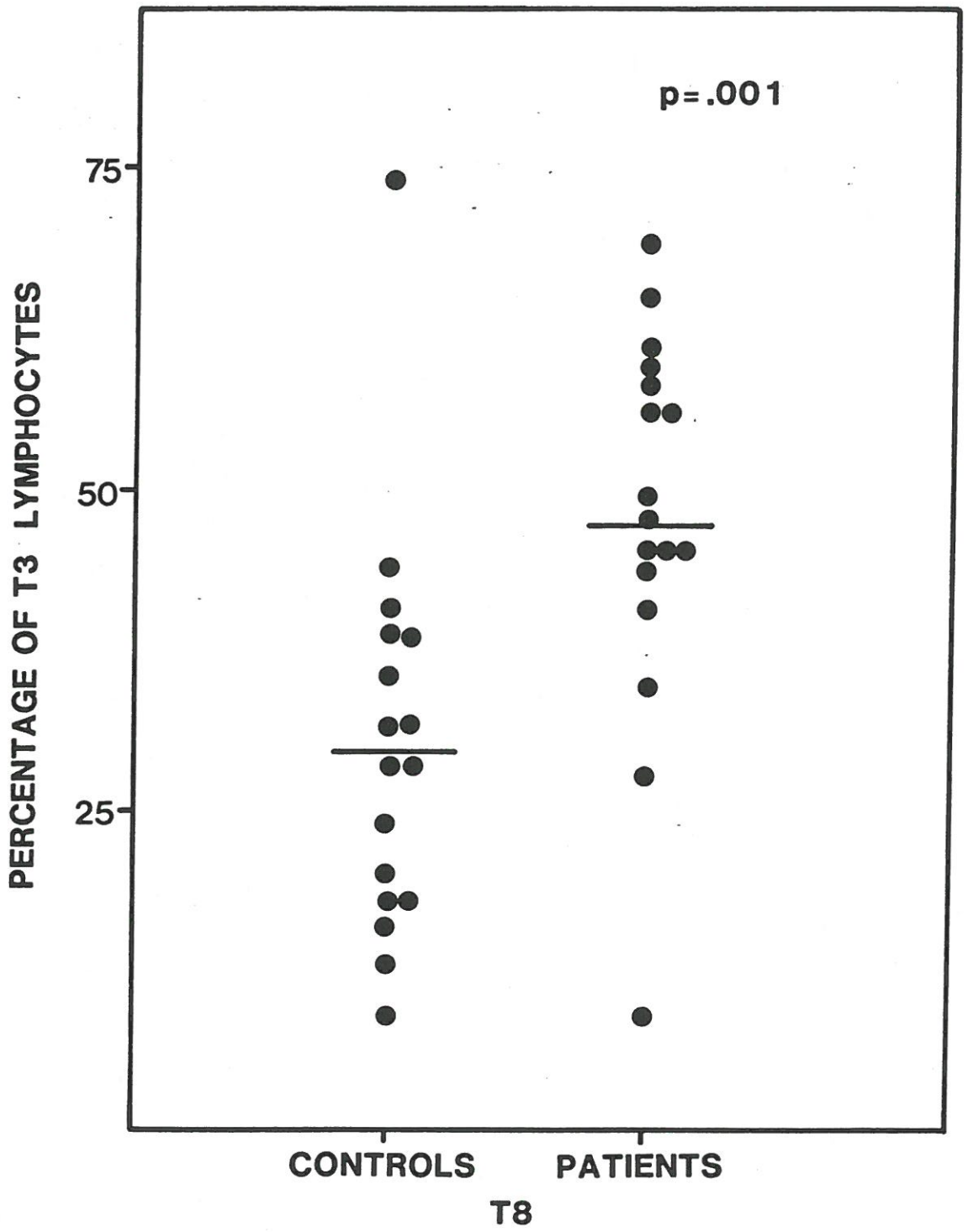
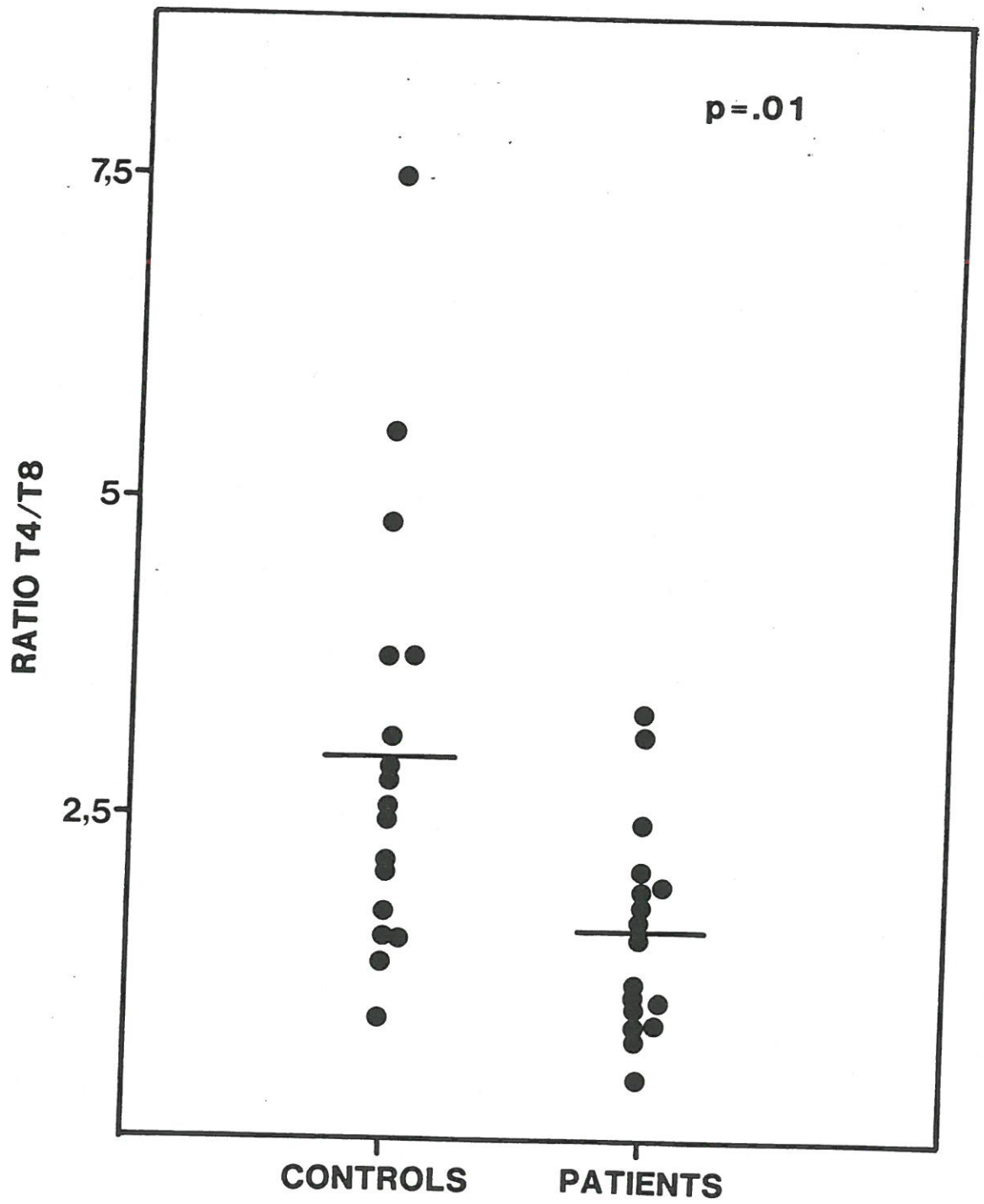


Figure 97 Comparison of T4/T8 ratio between controls and CRF patients



to OKT8 cells showed significant deviations from normal (p=0,01).

Functional assays of suppression. The results are shown in Table 60, Figure 98)

The only significant finding was a lower level of suppressor activity in uremic patients as compared to control subjects when using the suboptimal dose of ConA (5 µg/ml).

Correlations. No significant correlation was found between any of the numerical assays and the tests of suppressor cell function in both the normal control and uremic groups. Similarly, no significant correlations were detected between any of the biochemical parameters estimated and the immunological tests performed.
No correlations were observed between all numerical assays employed and the preincubation test with either PHA or ConA in the control group.

Table 60. Assays of suppressor cell function in patients on chronic hemodialysis and normal controls[a]

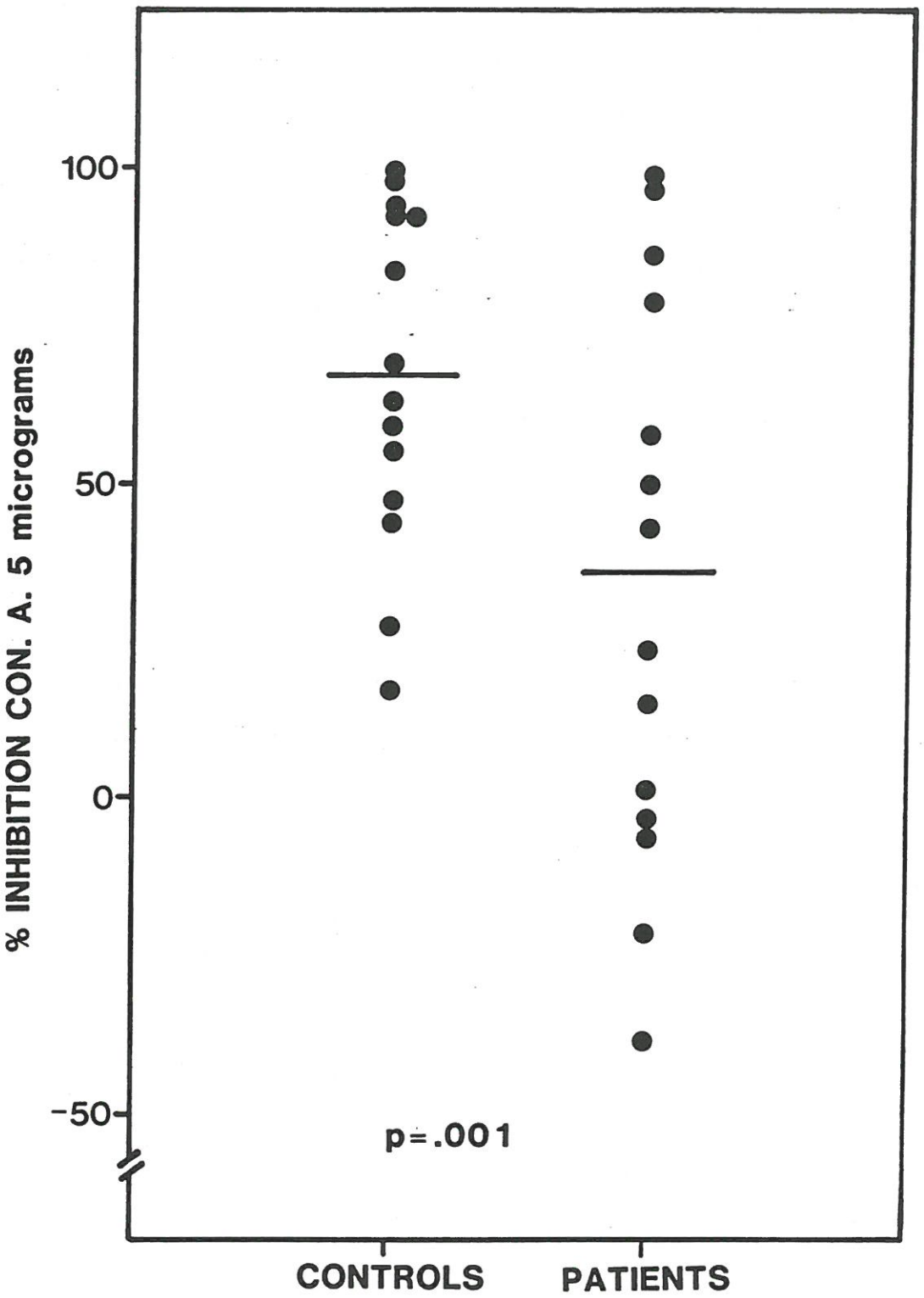
	Percentage inhibition of homologous PHA lymphocyte transformation			
	Concanavalin A			
Patient no.	5 µg/ml	30 µg/ml	Sodium periodate	Immune complexes
1	-39	ND	ND	ND
2	-6	51	-24	-12
3	97	99	99	66
4	-3	ND	-3,6	ND
5	15	84	97	80
6	ND	ND	ND	ND
7	58	86	68	95
8	ND	ND	ND	ND
9	ND	ND	ND	ND
10	23	92	24	9
11	79	ND	ND	ND
12	43	ND	87	ND
13	99	ND	ND	ND
14	1	ND	2	-14
15	50	94	24	26
16	-21	-23	-20	ND
17	86	ND	ND	ND
18	ND	ND	ND	ND
19	ND	ND	ND	ND
All patients	36±12[b]	70±16	33±16	36±17
Controls	67± 7	81± 7	-54±32	-34±44
Probability value	<0,002	>0,10	<0,10 >0,05	>0,10

a % Suppression = $\left(1 - \frac{\Delta\text{DPM mitogen-stimulated cells}}{\Delta\text{DPM non-stimulated cells}}\right) \times 100$

Δ DPM = DPM of mitogen stimulated cells - DPM of cells without mitogen.

b Mean ± SEM

Figure 98 Comparison of suppressor cell activity (ConA 5µg/ml) between controls and CRF patients



DISCUSSION

The reduction in the total circulating lymphocytes, and T- and B-cell populations confirms previous reports in uremia (Sengar et al., 1975; Harris and Sengar, 1975). In keeping with the general cell depletion is the new finding of a significant reduction of the T-helper/inducer subset. The unusual finding of a normal number of T-suppressor/cytotoxic cells in the presence of marked depletion of total lymphocytes might suggest that the relative predominance of these cells could upset the regulatory balance in the immune homeostatic mechanisms and account for the anergy in uremia. However, the certainty of this interpretation tends to be diminished by the findings of a low ConA induced suppressor activity in these patients. This significantly low level of Con A-induced suppression is similar to that described in auto-immune diseases (Coovadia et al., 1981) but contrasts with previous reports of augmented suppressor function in uremia using other experimental techniques (Raskova and Morrison, 1976); Guillou et al., 1980) which, however, are less reproducible (Coovadia et al., 1981).

The consistently smaller OKT3 population as compared to E-rosettes was not totally unexpected, as the technique of fluorescence microscopy used here, is known to give lower results than that obtained using flow cytometric analysis. In addition, it is possible that all E-rosette forming cells are not T-cells and all mature T-cells do not express the specific antigen detected by the OKT3 antibody. The sum of the $OKT4^+$ and $OKT8^+$ cells was frequently greater than the total number of $OKT3^+$ cells. This may suggest that the OKT3 antibody is underestimating the total T-cell population or that there is a population of cells that has both these antigens. A group of double-marker cells, in fact, has been demonstrated in myasthenia gravis where they were interpreted as being immature cells (Berrih et al., 1981).

The reasons for the lack of correlation between the numerical and functional assays among both controls and patients as found in this

study have been dealt with elsewhere. (See Chapter on Measles) The dissociation between marker and functional assays which we have shown here and elsewhere (Coovadia et al., 1981) has also emerged from another report (Bach et al., 1981) using the same monoclonal antibodies used in this study. In patients with leprosy, a decrease in both in vivo and in vitro tests of function attributed to T-helper cells, was not accompanied by the expected reduction of OKT4 cells.

The generation of suppressor activity by sodium periodate and circulating immune complexes produced extremely variable results with frequent and unpredictable stimulation, rather than suppression of the PHA response. The suboptimal dose of 5 µg/ml ConA in this study was most effective at demonstrating the difference between the control and patient groups.

Loss of suppressor function has been shown in patients with systemic lupus erythematosus (Bresnihan and Jasin, 1977) using the preincubation method. Our attempts, using the same method, to detect suppressor function in patients with CRF were hampered because of the insufficient number of mononuclear cells obtained.

As the majority of patients tested by us were reasonably nourished, malnutrition cannot be implicated as a cause of the anergy in uremia.

In evaluating the above findings greater reliance has to be placed on the numerical assays using monoclonal antibodies which give simple and reliable results rather than the functional assays which are subject to technical variations and whose physiological significance is unclear.

Therefore, the evidence given above on the numerical imbalance between suppressor and helper T-cells suggests that altered suppressor cell activity may be a cause of the immunoparesis in uremia.

NEUROMUSCULAR DISEASES

NEUROMUSCULAR DISORDERS: EVALUATION OF IMMUNOREGULATORY CELLS IN DMD AND SMA AMONG AFRICAN AND INDIAN PATIENTS

AIM

This study was undertaken to observe whether there is any disturbance in immunoregulatory cells in DMD and SMA.

SUMMARY

Suppressor cells were assayed by numerical and functional tests in Duchenne Muscular Dystrophy (DMD) and Spinal Muscular Atrophy (SMA) among African and Indian children in order to contribute to an understanding of the pathogenesis of these neurological disorders. Peripheral blood mononuclears (PBM) were classified as total T cells and T cell subsets by the OKT series of monoclonal antibodies and as B cells by the presence of surface immunoglobulin. The suppressive effects of PBM pretreated with concanavalin A (Con A) on normal homologous phytohaemagglutinin (PHA) transformation of mononuclear cells was determined. PBM stimulation by PHA was also assessed.

Patients with DMD had a significant increase ($p = 0,0353$) in the number of T suppressor/cytotoxic cells (1218 $\pm$ 142 cells/mm^3 mean $\pm$ SEM) as compared to controls (815 $\pm$ 95 cells/mm^3) and a significant reduction ($p = 0,0282$) in $OKT4^+$ cells expressed as a percentage of $OKT3^+$, 50% $\pm$ 3 compared to 61% $\pm$ 3.

No differences were detected in any of the numerical assays employed in SMA as compared to controls, or within SMA patients according to severity of disease.

Suppressor function and PHA transformation were normal in both groups of patients.

No significant correlations were detected between numerical and functional assays of suppression.

The implication of the results obtained for the role of immunoregulatory cells in the pathogenesis of DMD in these children is discussed.

INTRODUCTION

A vast number of clinical disorders, both acute and chronic, hereditary and acquired, affect the lower motor neurone (Dyck et al., 1975). The lesion may be in the anterior horn cell or more distally along the course of the peripheral nerve. Some disorders affect only motor nerves, others the sensory as well. In some the peripheral neuropathy occurs in isolation, in others it is associated with involvement of the spinal tracts or other parts of the central nervous system (CNS). Spinal muscular atrophy is a chronic disorder, mainly hereditary, which predominantly affect the motor nerves and therefore likely to overlap with other disorders of muscle.

The muscular dystrophies are a group of genetically determined disorders with progressive degeneration of skeletal muscle and no associated structural abnormality in the central nervous system or peripheral nerves.

It has been observed in other disorders where muscle weakness is a feature (eg. polymyositis, dermatomyositis, multiple sclerosis, that T cells and/or their products may be responsible for the inflammatory infiltrate and necrosis of muscle. Therefore it was of interest to study the above two mentioned disorders in order to show whether the direct or indirect degeneration of muscle leads to a change in the number(s) and function of cells involved in the immune regulatory network or vice versa.

A) Spinal Muscular Atrophy (SMA)

Genetics

Most forms of SMA have a hereditary basis. In the severe form as well as the milder forms, the pattern of inheritance has usually been autosomal recessive. Apart from the usual autosomal recessive mode of inheritance there have been a number of reports of SMA with dominant inheritance (Armstrong et al., 1966; Zellweger et al., 1972).

Some cases of SMA may be non-genetic and may be environmentally produced (Pearn et al., 1978) but searches for specific causative agents (Pearn, 1978; Pearn, 1979) such as trace elements, abnormalities and associated diseases have so far proved fruitless. In the African population, unlike the European and Asian communities where it is inherited as an autosomal recessive disorder, cases of SMA are mainly sporadic in nature (Moosa, A. and Dawood, 1986) and therefore presumably acquired.

There appears to be no prerequisite for inheritance as far as social class, maternal or paternal age, or area of birth is concerned.

It was not known whether the various types of progressive SMA with autosomal recessive inheritance and onset in infancy or childhood was one disease entity always due to the same gene or whether separate genes caused different types. Pearn (1980) by the use of genetic techniques on familial studies showed that there are several distinct SMA genes each with their different clinical syndromes. It is now possible to define individuals (and families) affected by these separate genes which is of extreme importance from a genetic counselling point of view in conveying the appropriate risk figures to the subject seeking genetic advice.

Pathogenesis

Apart from the genetic basis, the underlying cause and pathogenesis of the disease remain obscure. Acute onset of symptoms of SMA have been found after vaccination to: smallpox (Dubowitz, 1964); diptheria-pertussis (Gardner-Medwin et al., 1967) or miscellaneous infectious illnesses (Munsat et al., 1969). In other instances it was found that there was an increase in pre-existing weakness following or acute infection (Munsat et al., 1969) or measles (Gardner-Medwin et al., 1967). It is, however, difficult to determine whether this is of any significance particularly as SMA is in most instances a genetically determined disorder.

Beckman et al. (1970) suggested that it could be due to an in utero infection by poliomyelitis virus, perhaps associated with the widespread use of oral poliomyelitis vaccine. Hogenhuis et al. (1967) were unable to show any abnormality in RNA metabolism by autoradiographic assessment of uptake of tritiated uridine in the anterior horn cells in infantile SMA. These experiments suggested that an RNA virus, such as poliomyelitis, was unlikely. However, this picture may not be true in African children with SMA where cases are found to be sporadic and the use of oral poliomyelitis vaccine is not yet widespread, it could indeed be due to an ongoing viral process either due to poliomyelitis or coxsackie virus (Moosa, A., personal communication).

Low Vitamin E in the plasma has also been found in seven out of eight children with Werdnig-Hoffman disease (Shapira et al., 1981). High doses of vitamin E were given orally resulting in an increase of plasma levels but no clinical improvement was seen. Normal levels of Vitamin E in SMA have also been reported (Sokol and Iannaccone 1983). The role of Vitamin E deficiency in the pathogenesis of neurological deficits is therefore uncertain.

The Immune State of Patients with SMA

There is some evidence that the immune response is affected in SMA. Several authors (Hausmanowa-Petrusewisz and Fidziańska-Dolof 1975; Ryniewicz and Pawińska 1978) have observed that 50% of children with Werdnig-Hoffman disease have tonsils and adenoids which were atrophic and in 18% both these lymphatic tissues were lacking. This is in contrast to Castrovieja's findings (1984) who did not find this in his patients.

Specific Immune Response

Number of Cells

There have been no studies of immunoregulatory cell numbers in SMA to date.

Function of cells

Lymphocyte transformation to PHA has been shown to be significantly decreased in children with Werdnig-Hoffman disease as compared to normal children (Ryniewicz and Pawinska, 1978). The same authors found that the skin test to PPD was negative in 97% of these children as compared to positive in 85% of controls. They suggested that this decrease in CMI was due to a change in lymphocyte reactivity which may be related to an ectodermal defect involving both the spinal cord and the thymus, as patients' serum did not affect blast transformation to PHA of lymphocytes from healthy subjects. It has also been observed that similar low values of lymphocyte transformation were present in children with the Kughelberg-Welander type of SMA and not only in the most severely affected (Ryniewicz and Pawinska, 1978).

Non Specific Immune response

Serum IgA and IgM concentrations have been shown to be decreased in patients with SMA as compared to controls (Migaj et al., 1986).

B) Duchenne Muscular Dystrophy (DMD)

Genetics

The classical form of Duchenne dystrophy is inherited through an X-linked gene in two thirds of the cases and the remaining one third is secondary to a new mutation. Thus it is only confined to males. It is characterized by progressive muscle wasting and weakness which

become clinically evident around the age of three to five years and lead to an inability to walk by the age of 12 and death in the late teens or early twenties (Walton and Nattrass, 1954).

The locus for DMD is not within measurable distance of either the Xg locus (Blyth et al., 1965) or the colour vision locus (Emery, 1966; Greig, 1977). Cytological evidence suggests that it is in the middle of the short arm of the X chromosome (Xp 21) (Conneally, 1985).

There is also a high mutation rate. Different authors have estimated this mutation rate to be 95 per million genes per generation 65 per million and 43 per million (Dubowitz, 1978). This is one of the highest mutation rates for any human disease. The corresponding mutation rate for haemophilia is about 20 to 30 per million.

The incidence of the disease is not known for certain and estimates in male infants vary from 1/1700 in Germany to 1/6000 in France (Moosa, 1982).

It is therefore extremely important to diagnose this disorder early in order to detect carriers and offer genetic counselling. One of the investigations apart from the clinical assessment that one can do is to measure serum Creatinine Kinase (CK) levels (this enzyme is a sensitive index of myofiber damage) either of newborn male infants (Zellweger and Antonik, 1975) or of male infants who are not walking by the age of 18 months (Gardner-Medwin, 1978) in order to determine whether the child has this disorder or not. A gross elevation of CK level strongly indicates this disease. This should then be followed by the investigation of all female relatives of the patient(s) for carrier status and thereafter genetic advice should be given by experienced persons to the parents concerned. However, Gardner-Medwin (1979) has pointed out that there is no guarantee that parents will heed the genetic advice given. Perhaps for the simple reason that they would opt to take the 1-in-2 risk of having a normal son. Dubowitz has suggested that if the woman is still keen to have a child in spite of the high risk, one may be able to offer selective abortion

of a male foetus by sexing the fetus at about 14 weeks' gestation. There is at present no reliable way of telling whether a male foetus is affected by dystrophy or not. Analysis of foetal blood samples for CK levels has not proved as helpful as was originally thought. DNA probes, however, hold out real possibility of antenatal diagnosis.

Pathogenesis

DMD is a single disease entity. The clinical features are uniform, the histopathologic findings are characteristic, the serum enzymes are always elevated early in the course and the outcome, unfortunately is always predictable. However, the pathogenesis of muscular dystrophy is not clearly understood. Dystrophic muscle is composed of living, dying, dead and regenerating muscle fibres in varying proportions according to the stage of the disease. In healthy muscle fibre, necrosis is usually followed by regeneration of the affected fibres. Although regeneration also occurs in diseased muscle, it is thought that the regenerated fibres, may in turn, themselves become necrotic if the causative factor continues to act, thereby resulting in a recurring cycle of necrosis and regeneration.

Muscle fibre necrosis is seen from an early stage, even before the disease becomes clinically manifest, with hyaline fibres present in considerable numbers prior to invasion by phagocytes. The necrosis may be segmental suggesting that the factor(s) responsible for damaging the fibre act focally, or the necrotic (and regenerating) fibres may be grouped suggesting that focal ischaemia may be responsible for the muscle damage (Hathaway et al., 1970).

The actual cellular mechanism leading to muscle fibre degeneration and death in DMD is also not clear cut. Studies of muscle surface membrane (Mokri et al., 1975) have revealed gaps in the plasma membrane that may allow the permeability of abnormally large molecules and ions eg. Ca^{2+}. Whether this structural defect in the plasma membrane is a primary event in the course of muscle fibre breakdown or whether this particular lesion is secondary to a biochemical lesion

elsewhere in the muscle fibre is unknown. Furthermore, if the cause of the structural defect resided in the membrane itself, it could be caused by an abnormal lipid component or by a defective structural protein in the membrane.

Membrane abnormalities have previously been proposed in red blood cells in DMD viz. abnormality in the lipid profile and fatty acid patterns in muscle membrane phospholipids (Kunze et al., 1973); increased protein phosphorylation (Roses et al., 1975) and that red blood cells were prone to form echinocytes (Matheson and Howland, 1974). Some of these findings have not been confirmed by other workers, however, and the significance of such changes is not clear. Although many workers agree that the generalized membrane defect in DMD is genetic others have reported that these abnormalities may be induced by factors in the patients' plasma (Siddiqui and Pennington, 1977).

An abnormality in lymphocytes concerning their "capping" phenomenon has also been reported in DMD (Verrill et al., 1977; Pickard et al., 1978) but subsequent attempts to confirm this have been conflicting. Abnormalities in collagen synthesis in cultures of Duchenne fibroblasts (Ionasescu et al., 1977) and of abnormal fibroblast adhesiveness (Jones and Witkowski, 1979) have been found.

Studies of the repair processes of skeletal muscle have shown that this tissue possesses considerable powers of regeneration. Murray (1972) postulated that mononuclear muscle precursor cells are formed and that these multiply by mitosis, subsequently fusing to form the myotubes from which new muscle fibres develop. These precursor cells or myoblasts are probably derived, at least in part, from satellite cells (Maur, 1961). The same myoblast fusion mechanism appears to be operative in embryogenesis (Mintz and Baker, 1967) in normal growth, in injured and diseased muscle and in muscle grafts. The source of the mononuclear muscle cell precursors is uncertain. It could be that an undifferentiated satellite precursor cell could persist through adult life (Snow, 1978) lying between plasma and basement membranes of

the muscle fibre or it could be that myoblasts can arise by segregation of differentiated myonuclei (Walker, 1972). It also remains possible that local connective tissue cells and, in particular, circulating cells may have an accessory role in myogenesis (Toto et al., 1967).

The prospect of an eventual understanding of the basic defect in DMD lies in defining the genes that are active in neuromuscular differentiation and which are X-linked. Monaco et al. (1985) have described the successful cloning of a DNA fragment that detects a deletion which is in or very near the DMD gene. These authors hybridized excess DNA from a patient with DMD with a deletion to DNA from normal humans. The principle of the technique was that the DNA which failed to hybridize was enriched with sequences from the region of the deletion, hence identifying the chromosomal deletion of the affected gene. Monaco et al. described how seven of these probes from the region deleted in one DMD patient were used to check for deletions in DMD patients. Five of 57 males with DMD were found to have deletions, each missing at least 38 Kilobases of genomic DNA. By using these probes accurate prenatal diagnosis and carrier detection will be made available for families of affected children and most important in determining the aetiology of DMD which will hopefully provide insights into the basic mechanisms of this muscle disease.

The immune state in patients with DMD

As discussed under pathogenesis there was evidence, although controversial, to show that the "capping" phenomenon of the lymphocyte membrane structure was reduced (Pickard et al., 1978). These authors suggest that the altered membrane fluidity may be expressed as conformational changes on the surface of B lymphocytes.

In certain other myopathies eg. polymyositis and dermatomysitis and in diseases of the CNS eg. multiple sclerosis both cellular and humoral factors of the immune system have been suggested to be important factors in the aetioliogy and/or pathogenesis of these disorders.

Specific Immune Response

a) Number of cells

There have been no studies on cell numbers of the immune system in DMD to date. However, disturbances in the number of immunoregulatory T cells have been reported in other disorders of CNS involving muscle weakness eg. multiple sclerosis and myasthenia gravis. In both these conditions a reduction in the circulating T-suppressor cell subset has been demonstrated (Santoli, et al., 1978; Skolnik, et al., 1982). Bresnan et al. (1981) similarly reported a loss of circulating T-suppressor cells during the active phase of dermatomyositis in two children, while Iyer et al. (1983) failed to demonstrate any change in T cell subsets in one case of active and untreated dermatomyositis and in six cases of polymyositis.

b) Function of cells

Similarly, no studies have been done on the function of the different cells of the immune system in DMD. It has been shown, however, that lymphocytes from the cerebrospinal fluid (CSF) as well as from the peripheral blood from patients with multiple sclerosis (MS) were able to respond better to PHA and to measles antigen than to other antigens viz. rubella, mumps, and herpes simplex (Reunanen et al., 1983). These authors also found that the CSF cellular response to PHA or measles virus antigen (MVA) and the rate of intrathecal antibody synthesis to MVA showed an inverse trend, suggesting that the stimulated cells may at least partially represent suppressor cells. This excess intrathecal synthesis of certain antibody specificities in multiple sclerosis could be explained by the fact that the distribution of T cell subpopulations appear to be different in CSF and peripheral blood in MS (Merrill et al., 1980). It could also suggest that the function of controlling T cell subsets between CNS and peripheral blood are different.

Peripheral lymphocytes of patients with active polymyositis were cytotoxic to human fetal muscle cultures (Currie et al., 1971; Dawkins et al., 1973) and produced lymphokines when incubated with autologous muscle in medium free of immunoglobulins (Haas et al., 1974).

Non specific Immune Response

a) Antibody and Complement

Antibodies against muscle components have been consistently demonstrated in polymyositis and dermatomyositis (Caspary et al., 1964; Stern et al., 1967; Currie, 1981) where deposits of IgG, IgM and C3 have been found in the vessel walls. However, no studies on humoral factors have been investigated in DMD.

Materials and Methods

PATIENTS (Tables 61, 62)

20 children (of whom 19 were male) with sex-linked recessive mode of inheritance of DMD were studied at the King Edward VIII Hospital, Durban. The diagnosis was confirmed clinically and histologically. They were between 3-17 years (median age 8 years) and 14 were Indian and 6 African. One female patient in this study was a manifesting carrier. Four of the older patients were confined to a wheelchair while the rest were still ambulant at the time of study. Sixteen children and 1 adult with clinically and histologically confirmed SMA aged between 1 month-37 years (median age 1 year 7 months) of whom 2 were Indian males and 15 African (of whom 4 were males) were studied at the same hospital. Five children had severe SMA, nine intermediate and three mild.

The patients with DMD and SMA were age - sex - and race matched separately with normal healthy children who were part of an epidemiological survey of the prevalence of Hepatitis B (See Chapter on Normal development of Immune Response). Informed consent from the parents of patients and controls was obtained prior to blood samples being taken. The nutritional state of the patients was satisfactory. All were above the 5th centile of weight for height according to the NCHS growth charts and did not have any of the clinical features of protein energy-malnutrition.

Table 61

Clinical data on patients with DMD

Patient No	Age (years)	Sex	Race	
1	5	M	I	
2	8	F	I (carrier)	
3	11	M	A	
4	3	M	I	
5	$5^{1}/_{2}$	M	I	
6	8	M	A	
7	6	M	I	
8	12	M	I *	
9	17	M	I *	both not ambulant
10	8	M	A	
11	9	M	I	
12	5	M	I	
13	11	M	I	not ambulant
14	4	M	I °	
15	6	M	I °	
16	9	M	A	
17	7	M	I +	
18	7	M	I +	
19	10	M	A	
20	13	M	A	not ambulant

Abbreviations: I = Indian South African; A = African South African
F = Female; M - Male
*, °, + = Brothers of 3 separate families

Table 62

Clinical data on patients with SMA

Patient	Age (years)	Sex	Race	Degree of severity
1	9	F	A	Mild
2	37	M	A	Mild
3	3	F	A	Intermediate
4	18	F	A	Mild
5	3/12	F	A	Severe
6	2 9/12	F	A	Intermediate
7	3/12	F	A	Severe
8	1 7/12	F	A	Intermediate
9	6	M	I	Intermediate
10	9/12	F	A	Intermediate
11	6/12	M	I	Severe
12	1/12	F	A	Severe
13	3 5/12	F	A	Intermediate
14	9/12	F	A	Intermediate
15	5/12	M	A	Severe
16	1	M	A	Intermediate
17	3	M	A	Intermediate

Abbreviations: I = Indian South African; A = African South African
F = Female; M = Male

Immunological Investigations

1. Absolute lymphocyte counts were done on the Coulter counter and differential counts by routine microscopic examination of the stained slide.

2. Numerical Assays

   a) T and B subpopulations
   This was performed as previously described under Methods.

   b) Lymphocyte subpopulations identified by murine monoclonal antibodies
   This was performed as previously described under Methods.

3. Functional Assays

   a) ConA induction of suppressor cells tested on a normal homologous PHA transformation of lymphocytes
   This was undertaken as previously described under Methods

   b) MNC stimulation by Phytohaemagglutinin (PHA)

   (i) MNC were obtained as described under MNC separation

   (ii) MNC concentration was adjusted to $1,33 \times 10^6$ cells/ml with culture medium + 12,5% AB serum. The cells were dispensed in quadruplicate into each well of Titertek round bottomed microtitre plates as follows:

MNC $2x10^6$	150µl	
Culture medium	25µl	
PHA 0,4µg	25µl	(This concentration has been previously found to be optimal results not shown)

Schematic diagram:

Row number	2-5	7-10
B	MNC+O	MNC+PHA

where O = culture medium.

(iii) The outer rows of the plates were filled with sterile water to prevent evaporation, it was covered with a lid and wrapped in Jiffy wrap.

(iv) The plates were incubated at 37°C for a total of 3 days (this incubation period has been found to be optimal for PHA lymphocyte stimulation results not shown) and ^{14}C-Thymidine (0,075µCi/10µl) was added 24 hours before the plates were harvested.

(v) The plates were harvested as described under "Assay of suppressor activity on PHA transformation of normal homologous responder lymphocytes".

(vi) The results were expressed as dpm of the stimulated cultures as well as the stimulation index (SI) calculated as follows:

$$SI = \frac{\Delta \text{dpm of cultures with PHA}}{\Delta \text{dpm of cultures without PHA}}$$

Statistical Analysis

Results were analysed as described under methods, using the non-parametric Mann Whitney U test when comparing patients versus controls and patients with DMD versus patients with SMA. Correlation analysis was performed using the Spearman Rank correlation coefficient and significance was tested at th 5% level.

RESULTS

Summary of Findings

1) An increased number of $OKT8^+$ cells and a reduction in proportions of $OKT4^+$ cells were found in DMD as compared to controls. No differences in immunoregulatory cell numbers were detected in SMA as compared to controls or within SMA patients according to severity of disease.

2) Suppressor cell function and PHA transformation were normal in both groups of patients.

1) Numerical assays in DMD and SMA (Table 63, Figure 99)

The absolute number of T-suppressor/cytotoxic cells as defined by monoclonal antibody OKT8 were significantly elevated (p=0,0353) in patients with DMD (1218 $\pm$ 142 cells/mm^3 mean $\pm$ SEM) as compared to controls (815 $\pm$ 95 cells/mm^3). $OKT4^+$ subset expressed as a percentage of OKT3 was significantly reduced (p=0,0282) in patients (50% $\pm$ 3) when compared to controls (61%$\pm$ 3). There were no significant deviations from normal in absolute monocuclear cells, absolute lymphocytes, T cells (E-rosette and OKT3 MoAb) Null and $OKT4^+$ cells, although these cells were consistently higher in DMD, while the T4/T8 ratio was lower. In SMA when compared to controls, or when compared to each other with respect to the degree of severity of disease, there were no differences in any of the above numerical assays studied.

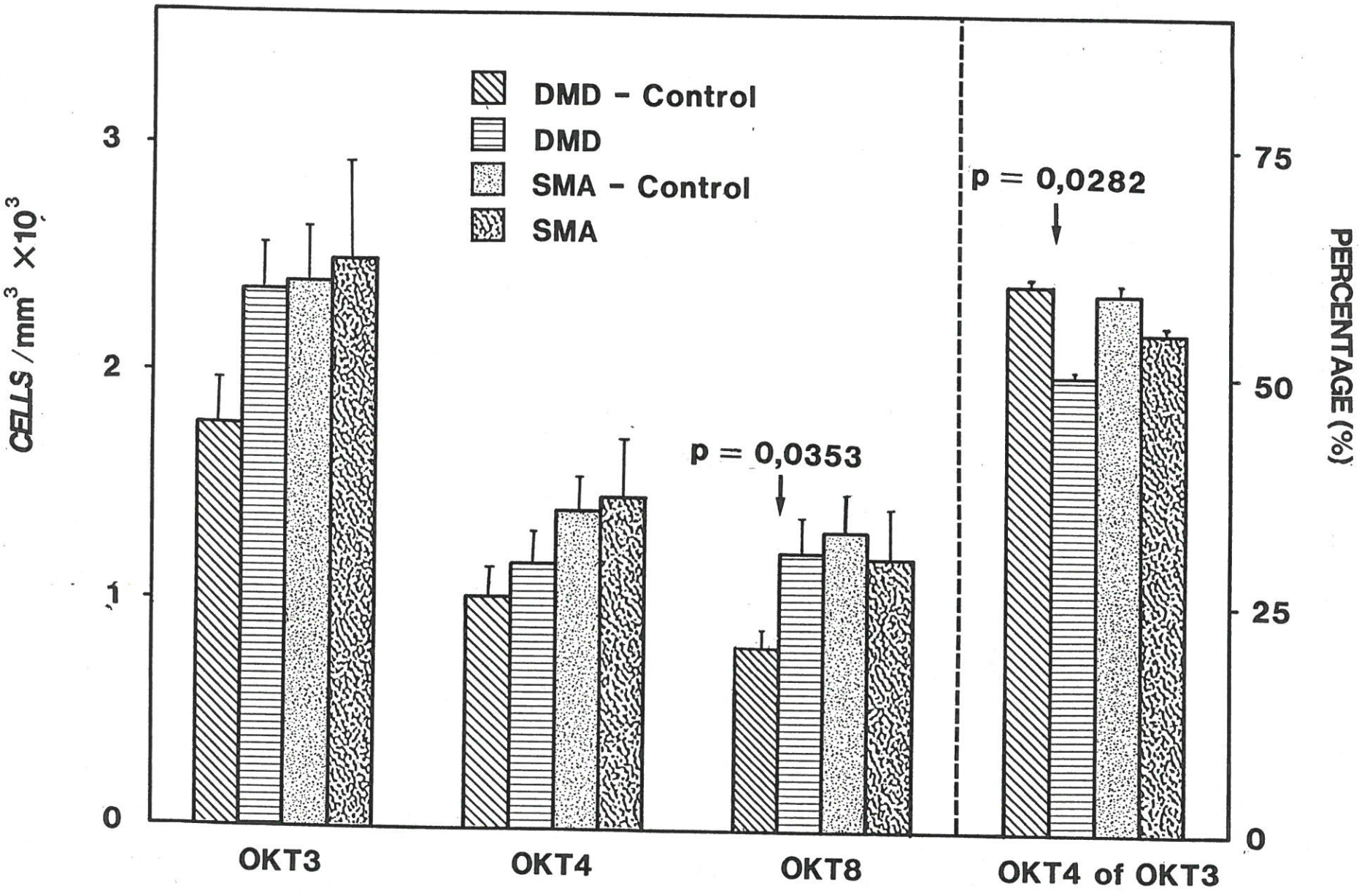
Table 63 Numbers of Mononuclear Cells, T cells and T cell subsets in DMD, SMA and their respective controls

Cell Type	DMD	DMD Control	SMA	SMA Control
n	20	20	17	17
Absolute mononuclear cells	3909 ± 386*	3190 ± 275	4659 ± 551	4785 ± 50
Absolute lymphocytes	3525 ± 329	2815 ± 254	4131 ± 545	4343 ± 51
T cells (E-rosette technique)	2293 ± 220	1860 ± 186	3145 ± 407	2345 ± 28
B cells (SIg)	283 ± 68	365 ± 61	365 ± 127	504 ± 11
Null cells	1366 ± 249	902 ± 104	1313 ± 155	1827 ± 28
T cell (MonoAb OKT3)	2370 ± 234	1771 ± 196	2510 ± 417	2395 ± 24
OKT4 cells (T helper/inducer)	1172 ± 127	1036 ± 102	1403 ± 258	1468 ± 15
OKT8 cells (T suppressor/ cytotoxic)	1218 ± 142°	815 ± 95	1202 ± 206	1334 ± 15
% OKT4 of OKT3	50 ± 3°	61 ± 3	56 ± 5	62 ± 3
OKT8 of OKT3	52 ± 3	48 ± 3	49 ± 3	56 ± 3
Ratio OKT4/OKT8	1,03 ± 0,09	1,34 ± 0,10	1,18 ± 0,14	1,18 ± 0,10

* Mean ± SEM

° Individual p values are given in the text

Figure 99 Numbers of T cell subsets in DMD, SMA and their respective controls



2) Functional assays in DMD and SMA (Table 64)

There was no difference detected in the T-suppressor cell activity as determined by ConA between patients and controls in both DMD and SMA. Similarly no difference was seen in the T-cell function as assayed by PHA between patients with each of the disorders and their respective controls.

3) Correlations

There was a significant correlation in both DMD patients ($r=+0,9000$) $p<0,0001$) and their respective controls ($r = +0,8596$)($p < 0,0001$) in the T-cell numbers detected by E-rosette technique and by monoclonal antibody OKT3. Similar findings were obtained in SMA patients and their respective controls betwen T cells detected by E-rosette technique and by OKT3 monoclonal antibody ($r = + 0,8529$ $p \leq 0,0001$; $r=+0,7696$ $p \leq 0,0002$ respectively).

There was no correlation between numerical and functional assays of T-suppressor cells in either the patient or control groups.

Table 64 T cell functional assays in DMD and SMA patients compared to their respective controls

	DMD	DMD Control	SMA	SMA Control
n	17	16	13	13
% suppression (ConA 5 µg)	18 ± 5*	25 ± 4	23 ± 8	17 ± 5
% suppression (ConA 30 µg)	32 ± 5	37 ± 4	37 ± 7	26 ± 5
PHA Transformation- (dpm)	10192 ± 1155	10557 ± 691	9283 ± 1992	12275 ± 3697
Stimulation Index (SI)	129 ± 78	138 ± 61	127 ± 73	148 ± 77

* Mean ± SEM

DISCUSSION

T-cells and/or their products have been implicated in the inflammatory infiltrate and necrosis of muscle in disorders of the CNS where there is muscle weakness eg. polymyositis, dermatomyositis and multiple sclerosis (Dawkins and Mastaglia, 1973; Bresnan et al., 1981; Santoli et al., 1971; Skolnik et al., 1982). DMD a genetically determined disorder and SMA an acquired sporadic disorder in Southern Africa (Moosa and Dawood, 1986) also are characterized by progressive muscle wasting and necrosis.

In DMD, an abnormality in the ability of B cells to "cap" has been reported (Pickard et al., 1978; Verill et al., 1977), these results have been conflicting (Gershwin et al., 1979) and the derangements have been attributed to the cytoskeleton rather than to immunity. Evidence of a cell-mediated immune effector response against muscle fibre was shown in studies of the nature of the mononuclear cell infiltrate in muscle biopsy specimens (Arahata and Engel, 1984). It has been shown (Arahata and Engel, 1984) in DMD, polymyositis and inclusion body myositis, that non-necrotic muscle fibres were invaded by $T8^+$ cells, a lesser number of $T4^+$ cells and macrophages and also those T cells expressing the Ia marker. This observation has been suggested to imply that there appears to be recognition of specific muscle fibre antigen by an antigen receptor on at least some of the invading T cells which had previously become sensitized to this putative antigen (Maritz et al., 1983). It could be that the antigen may be distributed in discrete sites. In SMA, however, several authors (Ryniewicz and Pawińska, 1978; Hausmanova-Petruswicz and Fidzianska-Dolot 1984) have shown evidence that there was a decrease in CMI as assayed by skin testing (PPD) and PHA lymphocyte transformation.

These findings prompted us to investigate the immune status of these two disorders of muscle whether it may be affected more and/or less by the direct (as in the case of DMD) or indirect degeneration of muscle

(as in the case of SMA). The availability of monoclonal antibodies to T-cells and certain functional tests of CMI were used in order to observe whether the network of immune regulation was perhaps imbalanced as a result of muscle degeneration or vice versa.

A significant increase in the number of T-suppressor/cytotoxic subset was found in DMD. Although, no difference was observed in the number of $OKT4^+$ cells, these cells when expressed as a percentage of the total OKT3 cells were significantly decreased. The increase in the $OKT8^+$ explains the decrease in the percentage of $OKT4^+$ cells and the lower T4/T8 ratio observed. This elevation in the $OKT8^+$ cells could be the way the immune response tries to dampen the continued sensitization of lymphocytes to muscle antigens. The increase in the $OKT8^+$ subset contrasts with reports in polymyositis, dermatomyositis, multiple sclerosis and myasthenia gravis where a reduction in the circulating T-suppressor subset has been demonstrated (Santoli et al., 1971; Skolnik et al., 1982; Bresnan et al., 1981). On the other hand, Iyer et al. (1983) found normal numbers of T-cell subsets in the first two conditions. The difference in our findings and those reported in polymyositis, multiple sclerosis and myasthenia may be explained by the fact that these are primarily immune mediated acquired conditions, whereas DMD is a genetic disorder in which the primary defect is thought to reside in the muscle membrane.

There was an increase, although not significantly from controls, in the absolute number of mononuclear cells and lymphocytes, in T-cells as detected by the E-rosette technique and by monoclonal antibody (OKT3), in Null cells and T helper/inducer cells. The higher number of total T cells probably reflects suppressor/cytotoxic cell increase. B cell numbers, although not significantly reduced as compared to controls, were lower. This decrease could be due, as previously suggested by Pickard et al. (1978), to changes in the conformational structure of the B lymphocyte which result in an altered membrane fluidity, thereby rendering the detection of surface immunoglobulin difficult by conventional tests.

T-suppressor cell function by pre-treatment with ConA did not show any increase in suppressor cell activity as would have been expected from the elevated $OKT8^+$ cell numbers in DMD. These discrepancies may be explained by the fact that one cannot separate the cytotoxic and suppressor aspect of T cells using OKT8 antibody. An increase in the number of $OKT8^+$ cells may in fact be due to an elevated number of cytotoxic cells enumerated in the $OKT8^+$ subset. If this is in fact so, it could explain our results of normal function of suppressor cells. Furthermore, the ConA test is a non specific measure of suppressive activity and may therefore not reflect any antigen determined reactions in DMD. The reasons for the lack of correlation between alteration in lymphocyte subset numbers with lymphocyte assays of either help or suppression have already been given. (See Chapter on Measles). As the monoclonal antibodies are much more accurate in this regard, we would suggest that there is likely to be increased suppressor activity in DMD. The normal lymphocyte response to PHA adds further evidence to the normal overall T cell function in DMD; any defect is likely to be highly antigen specific.

Patients with SMA showed no abnormalities in either immunoregulatory cell number or function compared to controls. Similar findings were obtained among SMA patients with respect to severity of disease. These findings differ from those of Ryniewicz and Pawińska (1978) who showed a significant decrease in the transformation of lymphocytes to PHA in children with SMA regardless of severity. These authors suggested that the decrease in CMI may be due to congenital defect of the thymus. These discrepancies may be explained by the fact that SMA in Africa, is a sporadic disease (Moosa and Dawood, 1986) unlike in Europe and Asia where it is inherited as an autosomal recessive disorder. The pathogenesis might also be different (Moosa and Dawood, 1986).

In summary, this study suggests that the direct degeneration of muscle in DMD is associated with regulatory changes in the number of cells of the immune system. It could be that, the genetic determinant which causes active muscle degeneration releases muscle antigen which in

turn continuously causes lymphocyte sensitization followed by release of tissue damaging lymphokines which exert a cytotoxic effect (Johnson et al., 1972). This process is dampened by an increase in the suppressor/cytotoxic population of the immunoregulatory network. A decrease in the percentage of T helper/inducer population is probably a secondary effect. This postulated mechanism would be further supported from the work of Arahata and Engels (1984) who postulated that the Class I major histocompatibility complex (MCH) gene product expressed on muscle fibres is likely to be the triggering factor for recognition by $T8^+$ cells. However, we recognize the limitations of making comparisons with results from different laboratories in different settings. Lymphokines, which may also be cytotoxic (Johnson et al., 1972) are released by sensitized cells which in turn recruit $T4^+$ cells and macrophages, with the former cells augmenting the activity of the cytotoxic $T8^+$ cells (Reinherz et al, 1979). It may be that this imbalance leads to the abnormality in the cytoskeleton of the muscle membrane reported in this disorder. Although the above are possibilities, one cannot place too much emphasis on the functional significance of lymphocyte phenotypic profile. It has been shown that both $T4^+$ and $T8^+$ subpopulations are functionally heterogeneous and $T4^+$ cells may contain subpopulations that are helper/inducer, induce $T8^+$ precursors to become suppressor cells and express cytotoxic activity against cells bearing Class II antigens (Young and Geha, 1986).

Unlike DMD, SMA is a slow process of muscle wastage where the degeneration of muscle is secondary to a defect in the anterior horn cell of the spinal cord. Hence lymphocytes may not be actively sensitized by the continuous release of muscle antigens.

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APPENDIX

Table 1 Particulars of different age groups for African Children from the community (CC)

	Age Groups	Frequency	Mean	Standard Deviation	SEM	Minimum	Maximum	Range
Immunological:								
Absolute mononuclears (cells/mm^3)	Cord	16	4925,55	1519,03	379,76	1824,00	7600,00	5776,00
	>1/12≤6/12m	14	5977,06	1630,32	435,72	3264,00	9191,00	5927,00
	>6/12≤ 1	9	6008,44	2073,27	691,09	3010,00	9660,00	6650,00
	> 1 ≤ 2	23	6100,54	3002,68	626,10	3293,00	16864,00	13571,00
	> 2 ≤ 3	24	5740,81	2798,55	571,25	2574,00	15834,00	13260,00
	> 3 ≤ 4	21	3573,90	1171,08	255,55	1827,00	7406,00	5579,00
	> 4 ≤ 5	21	3891,38	1211,76	264,43	2310,00	7504,00	5194,00
	> 5 ≤16	42	2867,14	760,05	117,28	1440,00	4420,00	2980,00
	>16 ≤60	15	2455,80	797,51	205,92	1672,00	4446,00	2774,00
Absolute lymphocytes (cells/mm^3)	Cord	16	3859,94	1120,93	280,23	1520,00	5616,00	4096,00
	>1/12≤6/12m	14	5589,56	1410,89	377,08	3200,00	8282,00	5082,00
	>6/12≤ 1	9	5273,55	1798,71	599,57	2795,00	8855,00	6060,00
	> 1 ≤ 2	23	5716,76	2943,80	613,83	2848,00	16320,00	13472,00
	> 2 ≤ 3	24	5367,40	2801,48	571,85	2244,00	15631,00	13387,00
	> 3 ≤ 4	21	3371,24	1040,89	227,14	1764,00	6762,00	4998,00
	> 4 ≤ 5	21	3705,71	1186,73	258,97	2275,00	7236,00	4961,00
	> 5 ≤16	42	2627,09	707,20	109,12	1243,00	4160,00	2917,00
	>16 ≤60	15	2179,93	769,06	198,57	1271,00	3900,00	2629,00

	Age Groups	Frequency	Mean	Standard Deviation	SEM	Minimum	Maximum	Range
T cell (E-rosette) ($cells/mm^3$)	Cord	15	2355,20	1102,70	284,72	497,00	4355,00	3858,00
	>1/12≤6/12m	14	3844,28	1136,03	303,62	2298,00	5607,00	3309,00
	>6/12≤ 1	9	3627,78	1313,96	437,99	1867,00	6182,00	4315,00
	> 1 ≤ 2	22	3903,14	1962,75	418,46	1752,00	9613,00	7861,00
	> 2 ≤ 3	24	3534,54	1869,28	381,57	1341,00	9026,00	7685,00
	> 3 ≤ 4	21	2301,09	819,69	178,87	1297,00	4814,00	3517,00
	> 4 ≤ 5	20	2559,60	878,73	196,49	1224,00	4935,00	3711,00
	> 5 ≤16	42	1607,57	397,29	61,30	749,00	2762,00	2013,00
	>16 ≤60	15	1629,20	573,63	148,11	1120,00	3468,00	2348,00
B cells (SIg) ($cells/mm^3$)	Cord	15	590,00	595,61	153,78	0,00	1987,00	1987,00
	>1/12≤6/12m	14	616,86	496,85	132,79	45,00	1628,00	1583,00
	>6/12≤ 1	9	595,33	310,06	103,35	98,00	966,00	868,00
	> 1 ≤ 2	22	499,95	315,99	67,37	56,00	1314,00	1258,00
	> 2 ≤ 3	24	620,54	709,07	144,74	0,00	3483,00	3483,00
	> 3 ≤ 4	21	278,62	232,99	50,84	39,00	1037,00	998,00
	> 4 ≤ 5	20	349,05	233,46	52,20	76,00	985,00	909,00
	> 5 ≤16	42	422,12	282,15	43,54	68,00	1591,00	1523,00
	>16 ≤60	15	197,53	271,09	69,99	18,00	1135,00	1117,00

	Age Groups	Frequency	Mean	Standard Deviation	SEM	Minimum	Maximum	Range
Null cells ($cells/mm^3$)	Cord	15	2088,93	827,06	213,55	857,00	3765,00	2908,00
	>1/12≤6/12m	14	1545,14	771,91	206,30	392,00	3279,00	2887,00
	>6/12≤ 1	9	1523,56	699,79	233,26	722,00	2512,00	1790,00
	> 1 ≤ 2	22	1802,50	1249,06	266,30	350,00	6408,00	6058,00
	> 2 ≤ 3	24	1572,37	810,58	165,46	214,00	3325,00	3111,00
	> 3 ≤ 4	21	992,33	569,65	124,31	311,00	2719,00	2408,00
	> 4 ≤ 5	20	1062,35	484,37	108,31	528,00	2776,00	2248,00
	> 5 ≤16	42	821,90	464,29	71,64	196,00	1959,00	1763,00
	>16 ≤60	15	652,73	363,67	93,90	134,00	1238,00	1104,00
FT cells ($cells/mm^3$)	Cord	15	6,93	18,44	4,76	0,00	58,00	58,00
	>1/12≤6/12m	14	0,00	0,00	0,00	0,00	0,00	0,00
	>6/12≤ 1	9	0,00	0,00	0,00	0,00	0,00	0,00
	> 1 ≤ 2	22	0,00	0,00	0,00	0,00	0,00	0,00
	> 2 ≤ 3	24	2,92	14,29	2,92	0,00	70,00	70,00
	> 3 ≤ 4	21	1,71	7,86	1,71	0,00	36,00	36,00
	> 4 ≤ 5	20	0,00	0,00	0,00	0,00	0,00	0,00
	> 5 ≤16	42	6,00	17,42	2,69	0,00	88,00	88,00
	>16 ≤60	15	2,33	6,16	1,59	0,00	18,00	18,00

	Age Groups	Frequency	Mean	Standard Deviation	SEM	Minimum	Maximum	Range
OKT3+ cells ($cells/mm^3$)	Cord	16	1636,50	648,28	162,07	662,00	2964,00	2302,00
	>1/12≤6/12m	14	3061,00	1042,48	278,61	1828,00	5328,00	3500,00
	>6/12≤ 1	9	2933,56	1215,02	405,01	1957,00	5989,00	4032,00
	> 1 ≤ 2	23	33A7,04	1811,96	377,82	1478,00	9444,00	7966,00
	> 2 ≤ 3	24	2922,79	1357,35	277,07	1132,00	5542,00	4410,00
	> 3 ≤ 4	21	1803,67	527,71	115,16	816,00	2962,00	2146,00
	> 4 ≤ 5	21	2314,67	699,33	152,61	1225,00	3977,00	2752,00
	> 5 ≤16	40	1664,35	525,02	83,01	691,00	2923,00	2232,00
	>16 ≤60	15	1475,73	429,86	110,99	974,00	2490,00	1516,00
OKT4+ cells ($cells/mm^3$)	Cord	16	1109,56	635,27	158,82	249,00	2416,00	2167,00
	>1/12≤6/12m	14	2216,43	639,21	170,84	1240,00	3868,00	2628,00
	>6/12≤ 1	9	2128,22	595,40	198,47	1460,00	3381,00	1921,00
	> 1 ≤ 2	23	1923,00	1148,44	239,47	724,00	6071,00	5347,00
	> 2 ≤ 3	24	1466,71	724,11	147,81	216,00	3452,00	3236,00
	> 3 ≤ 4	21	1117,62	370,12	80,77	605,00	1980,00	1375,00
	> 4 ≤ 5	21	1393,52	559,11	122,01	602,00	2476,00	1874,00
	> 5 ≤16	40	1022,30	335,99	53,12	581,00	1882,00	1301,00
	>16 ≤60	15	893,60	429,99	111,02	565,00	2134,00	1569,00

	Age Groups	Frequency	Mean	Standard Deviation	SEM	Minimum	Maximum	Range
OKT8+ cells (cells/mm^3)	Cord	16	732,19	324,44	81,11	270,00	1672,00	1402,00
	>1/12≤6/12m	14	1331,57	684,53	182,95	530,00	2849,00	2319,00
	>6/12≤ 1	9	1285,78	1102,02	367,34	301,00	3961,00	3660,00
	> 1 ≤ 2	23	1739,83	1185,25	247,14	494,00	5734,00	5240,00
	> 2 ≤ 3	24	1517,04	1011,76	206,52	438,00	4750,00	4312,00
	> 3 ≤ 4	21	953,24	339,28	74,04	238,00	1482,00	1244,00
	> 4 ≤ 5	21	1012,95	276,23	60,28	594,00	1637,00	1043,00
	> 5 ≤16	40	716,22	309,36	48,91	273,00	1646,00	1373,00
	>16 ≤60	15	901,00	379,86	98,08	370,00	1556,00	1186,00
% OKT4 of OKT3	Cord	16	69,50	30,03	7,51	10,00	114,00	104,00
	>1/12≤6/12m	14	74,71	14,68	3,92	59,00	114,00	55,00
	>6/12≤ 1	9	59,56	27,89	9,30	13,00	83,00	70,00
	> 1 ≤ 2	23	58,96	19,04	3,97	32,00	114,00	82,00
	> 2 ≤ 3	24	53,04	19,44	3,97	9,00	106,00	97,00
	> 3 ≤ 4	21	63,57	17,69	3,86	39,00	100,00	61,00
	> 4 ≤ 5	21	59,90	14,87	3,24	34,00	84,00	50,00
	> 5 ≤16	40	62,55	12,12	1,92	31,00	93,00	62,00
	>16 ≤60	15	61,33	17,89	4,62	32,00	86,00	54,00

	Age Groups	Frequency	Mean	Standard Deviation	SEM	Minimum	Maximum	Range
% OKT8 of OKT3	Cord	16	48,31	19,97	4,99	13,00	110,00	97,00
	>1/12≤6/12m	14	42,64	14,06	3,76	22,00	66,00	44,00
	>6/12≤ 1	9	39,78	19,45	6,48	15,00	73,00	58,00
	> 1 ≤ 2	23	50,87	15,04	3,14	22,00	83,00	61,00
	> 2 ≤ 3	24	52,33	20,95	4,28	20,00	108,00	88,00
	> 3 ≤ 4	21	53,76	18,43	4,02	22,00	103,00	81,00
	> 4 ≤ 5	21	46,00	12,42	2,71	23,00	65,00	42,00
	> 5 ≤16	40	42,98	11,91	1,88	20,00	78,00	58,00
	>16 ≤60	15	59,47	13,95	3,60	38,00	80,00	42,00
Ratio OKT4/OKT8	Cord	16	1,78	1,50	0,38	0,18	6,74	6,56
	>1/12≤6/12m	14	1,94	0,74	0,20	0,97	3,58	2,61
	>6/12≤ 1	9	2,47	1,63	0,54	0,85	6,10	5,25
	> 1 ≤ 2	23	1,31	0,73	0,15	0,38	3,57	3,19
	> 2 ≤ 3	24	1,33	0,90	0,18	0,16	3,92	3,76
	> 3 ≤ 4	21	1,39	0,87	0,19	0,49	4,38	3,89
	> 4 ≤ 5	21	1,40	0,50	0,11	0,58	2,27	1,79
	> 5 ≤16	40	1,57	0,49	0,08	0,40	2,65	2,25
	>16 ≤60	15	1,12	0,48	0,12	0,44	1,96	1,52

	Age Groups	Frequency	Mean	Standard Deviation	SEM	Minimum	Maximum	Range
% suppressor cell function (ConA 5 µg/25µl/ml cells)	Cord	10	41,10	26,91	8,51	-20,00	70,00	90,00
	>1/12≤6/12m	8	18,63	22,53	7,96	-14,00	58,00	72,00
	>6/12≤ 1	7	9,43	14,57	5,51	-5,00	39,00	44,00
	> 1 ≤ 2	20	23,35	34,32	7,68	-26,00	98,00	124,00
	> 2 ≤ 3	19	23,53	28,26	6,48	-35,00	84,00	119,00
	> 3 ≤ 4	14	25,86	22,72	6,07	-44,00	50,00	94,00
	> 4 ≤ 5	14	45,79	21,21	5,67	17,00	89,00	72,00
	> 5 ≤16	34	41,32	34,37	5,89	-13,00	95,00	108,00
	>16 ≤60	14	17,50	13,68	3,66	-10,00	36,00	46,00

	Age Groups	Frequency	Mean	Standard Deviation	SEM	Minimum	Maximum	Range
% suppressor cell function (ConA 30 µg/25µl/ml cells)	Cord	10	49,90	31,84	10,07	3,00	92,00	89,00
	>1/12≤6/12m	10	57,00	20,56	6,50	25,00	88,00	63,00
	>6/12≤ 1	7	34,57	22,80	8,62	-5,00	69,00	74,00
	> 1 ≤ 2	21	46,43	40,18	8,77	-60,00	94,00	154,00
	> 2 ≤ 3	22	42,55	33,10	7,06	-25,00	97,00	122,00
	> 3 ≤ 4	17	56,77	29,32	7,11	-30,00	94,00	124,00
	> 4 ≤ 5	16	68,81	19,41	4,85	37,00	96,00	59,00
	> 5 ≤16	39	43,03	32,36	5,18	-8,00	91,00	99,00
	>16 ≤60	14	27,21	33,06	8,83	-53,00	58,00	111,00

	Age Groups	Frequency	Mean	Standard Deviation	SEM	Minimum	Maximum	Range
MNC PWM stimulation (dpm)	Cord	9	6406,42	4650,95	1550,31	1923,73	15753,00	13829,27
	>1/12≤6/12m	5	7774,58	1578,21	705,80	6116,02	9682,63	3566,61
	>6/12≤ 1	5	4237,45	1444,39	645,95	2358,70	6058,66	3699,96
	> 1 ≤ 2	21	4929,87	2691,50	587,33	1091,14	12067,99	10976,85
	> 2 ≤ 3	19	6220,63	2671,94	612,99	581,69	11011,87	10430,18
	> 3 ≤ 4	15	5906,69	3720,95	960,74	796,18	13406,54	12610,36
	> 4 ≤ 5	17	6782,10	2127,26	515,94	2205,59	10142,61	7937,02
	> 5 ≤16	34	7324,42	3597,26	616,92	1418,06	14697,70	13279,65
	>16 ≤60	0						
WCC ($\times 10^9$/L)	Cord	16	13,56	4,20	1,05	7,60	20,50	12,90
	>1/12≤6/12m	14	9,02	1,62	0,43	6,40	12,00	5,60
	>6/12≤ 1	9	8,78	3,51	1,17	4,30	16,30	12,00
	> 1 ≤ 2	23	12,40	4,86	1,01	6,00	27,20	21,20
	> 2 ≤ 3	24	11,05	4,34	0,88	5,10	22,30	17,20
	> 3 ≤ 4	21	8,50	2,43	0,53	6,10	16,10	10,00
	> 4 ≤ 5	21	8,70	2,76	0,60	3,50	13,40	9,90
	> 5 ≤16	42	6,77	2,26	0,35	3,60	14,70	11,10
	>16 ≤60	15	5,70	1,73	0,45	4,10	10,90	6,80

	Age Groups	Frequency	Mean	Standard Deviation	SEM	Minimum	Maximum	Range
% Neutrophils	Cord	16	59,50	12,65	3,16	28,00	75,00	47,00
	>1/12≤6/12m	14	32,71	11,93	3,19	8,00	48,00	40,00
	>6/12≤ 1	9	30,11	12,14	4,05	13,00	52,00	39,00
	> 1 ≤ 2	23	46,04	13,56	2,83	23,00	72,00	49,00
	> 2 ≤ 3	24	41,79	12,54	2,56	9,00	71,00	62,00
	> 3 ≤ 4	21	54,33	11,24	2,45	28,00	71,00	43,00
	> 4 ≤ 5	21	51,48	8,95	1,95	33,00	70,00	37,00
	> 5 ≤16	41	51,32	9,20	1,44	34,00	68,00	34,00
	>16 ≤60	15	50,40	8,65	2,23	37,00	64,00	27,00
% Lymphocytes	Cord	16	30,63	12,02	3,01	12,00	54,00	42,00
	>1/12≤6/12m	14	60,86	11,07	2,96	50,00	79,00	29,00
	>6/12≤ 1	9	60,78	11,28	3,76	44,00	80,00	36,00
	> 1 ≤ 2	23	46,00	13,42	2,80	22,00	64,00	42,00
	> 2 ≤ 3	24	48,17	10,24	2,09	25,00	77,00	52,00
	> 3 ≤ 4	21	40,38	8,74	1,91	25,00	51,00	26,00
	> 4 ≤ 5	21	44,05	9,55	2,08	24,00	65,00	41,00
	> 5 ≤16	42	40,55	8,41	1,30	24,00	56,00	32,00
	>16 ≤60	15	38,73	10,17	2,62	22,00	56,00	34,00

	Age Groups	Frequency	Mean	Standard Deviation	SEM	Minimum	Maximum	Range
% Monocytes	Cord	16	7,13	4,40	1,10	1,00	16,00	15,00
	>1/12≤6/12m	14	3,79	3,14	0,84	0,00	10,00	10,00
	>6/12≤ 1	9	4,67	3,94	1,31	1,00	14,00	13,00
	> 1 ≤ 2	23	3,17	2,37	0,49	1,00	10,00	9,00
	> 2 ≤ 3	24	3,67	3,21	0,66	0,00	13,00	13,00
	> 3 ≤ 4	21	2,14	1,24	0,27	1,00	6,00	5,00
	> 4 ≤ 5	21	2,14	1,11	0,24	1,00	5,00	4,00
	> 5 ≤16	41	3,24	2,12	0,33	1,00	11,00	10,00
	>16 ≤60	15	4,73	3,92	1,01	1,00	15,00	14,00
% Eosinophils	Cord	16	1,19	0,98	0,25	0,00	3,00	3,00
	>1/12≤6/12m	14	1,21	1,37	0,37	0,00	4,00	4,00
	>6/12≤ 1	9	1,33	2,40	0,80	0,00	6,00	6,00
	> 1 ≤ 2	23	3,35	6,08	1,27	0,00	24,00	24,00
	> 2 ≤ 3	24	6,08	6,93	1,42	0,00	26,00	26,00
	> 3 ≤ 4	21	2,95	6,05	1,32	0,00	20,00	20,00
	> 4 ≤ 5	21	2,29	3,70	0,81	0,00	12,00	12,00
	> 5 ≤16	41	4,56	5,73	0,90	0,00	22,00	22,00
	>16 ≤60	15	5,20	4,43	1,14	0,00	14,00	14,00

	Age Groups	Frequency	Mean	Standard Deviation	SEM	Minimum	Maximum	Range
% Basophils	Cord	16	0,25	0,58	0,14	0,00	2,00	2,00
	>1/12≤6/12m	14	0,14	0,36	0,10	0,00	1,00	1,00
	>6/12≤ 1	9	0,33	0,71	0,24	0,00	2,00	2,00
	> 1 ≤ 2	23	0,17	0,49	0,10	0,00	2,00	2,00
	> 2 ≤ 3	24	0,04	0,20	0,04	0,00	1,00	1,00
	> 3 ≤ 4	21	0,19	0,51	0,11	0,00	2,00	2,00
	> 4 ≤ 5	21	0,05	0,22	0,05	0,00	1,00	1,00
	> 5 ≤16	41	0,17	0,50	0,08	0,00	2,00	2,00
	>16 ≤60	15	0,53	0,92	0,24	0,00	3,00	3,00
% Atypical Lymphocytes	Cord	16	0,50	0,73	0,18	0,00	2,00	2,00
	>1/12≤6/12m	14	0,93	1,69	0,45	0,00	4,00	2,00
	>6/12≤ 1	9	1,56	1,88	0,63	0,00	4,00	4,00
	> 1 ≤ 2	23	0,00	0,00	0,00	0,00	0,00	0,00
	> 2 ≤ 3	24	0,00	0,00	0,00	0,00	0,00	0,00
	> 3 ≤ 4	21	0,00	0,00	0,00	0,00	0,00	0,00
	> 4 ≤ 5	21	0,00	0,00	0,00	0,00	0,00	0,00
	> 5 ≤16	41	0,00	0,00	0,00	0,00	0,00	0,00
	>16 ≤60	15	0,07	0,26	0,0 7	0,00	1,00	1,00

	Age Groups	Frequency	Mean	Standard Deviation	SEM	Minimum	Maximum	Range
% Others	Cord	16	0,81	1,22	0,31	0,00	3,00	3,00
	>1/12≤6/12m	14	0,07	0,27	0,07	0,00	1,00	1,00
	>6/12≤ 1	9	1,22	2,54	0,85	0,00	7,00	7,00
	> 1 ≤ 2	23	0,00	0,00	0,00	0,00	0,00	0,00
	> 2 ≤ 3	24	0,00	0,00	0,00	0,00	0,00	0,00
	> 3 ≤ 4	21	0,00	0,00	0,00	0,00	0,00	0,00
	> 4 ≤ 5	21	0,00	0,00	0,00	0,00	0,00	0,00
	> 5 ≤16	41	0,00	0,00	0,00	0,00	0,00	0,00
	>16 ≤60	15	0,13	0,52	0,13	0,00	2,00	2,00
Haematological:								
RBC ($\times 10^{12}$/L	Cord	16	4,18	0,47	0,12	3,46	5,07	1,61
	>1/12≤6/12m	14	3,62	0,59	0,16	2,26	4,39	2,13
	>6/12≤ 1	9	4,43	0,27	0,09	4,06	4,81	0,75
	> 1 ≤ 2	23	4,33	0,39	0,08	3,32	5,38	2,06
	> 2 ≤ 3	24	4,31	0,47	0,10	3,39	5,17	1,78
	> 3 ≤ 4	21	4,19	0,22	0,05	3,82	4,72	0,90
	> 4 ≤ 5	21	4,29	0,39	0,09	3,82	5,29	1,47
	> 5 ≤16	42	4,49	0,33	0,05	3,42	5,14	1,72
	>16 ≤60	15	4,67	4,45	0,12	3,80	5,47	1,67

	Age Groups	Frequency	Mean	Standard Deviation	SEM	Minimum	Maximum	Range
Hb	Cord	16	15,26	1,56	0,39	12,40	18,50	6,10
(g/dL)	>1/12≤6/12m	14	9,51	1,04	0,28	6,80	10,80	4,00
	>6/12≤ 1	9	10,32	0,59	0,20	9,30	11,20	1,90
	> 1 ≤ 2	23	10,63	1,26	0,26	7,80	12,30	4,50
	> 2 ≤ 3	24	10,88	1,06	0,22	8,40	12,70	4,30
	> 3 ≤ 4	21	11,11	0,60	0,13	10,10	12,10	2,00
	> 4 ≤ 5	21	11,53	1,01	0,22	9,60	13,90	4,30
	> 5 ≤16	42	12,21	0,93	0,14	9,90	13,90	4,00
	>16 ≤60	15	14,71	0,95	0,25	13,60	16,40	2,80
HCT	Cord	16	0,47	0,05	0,01	0,38	0,60	0,22
(L/L	>1/12≤6/12m	14	0,31	0,03	0,01	0,22	0,35	0,13
	>6/12≤ 1	9	0,34	0,02	0,01	0,31	0,38	0,06
	> 1 ≤ 2	23	0,34	0,04	0,01	0,25	0,39	0,14
	> 2 ≤ 3	24	0,35	0,04	0,01	0,26	0,43	0,17
	> 3 ≤ 4	21	0,35	0,02	0,00	0,32	0,38	0,06
	> 4 ≤ 5	21	0,36	0,03	0,01	0,32	0,44	0,11
	> 5 ≤16	42	0,38	0,03	0,00	0,32	0,43	0,11
	>16 ≤60	15	0,45	0,03	0,01	0,42	0,50	0,08

	Age Groups	Frequency	Mean	Standard Deviation	SEM	Minimum	Maximum	Range
MCV	Cord	16	113,34	6,14	1,53	102,80	126,00	23,20
(fL)	>1/12≤6/12m	14	86,86	8,69	2,32	69,00	103,30	34,30
	>6/12≤ 1	9	76,92	3,26	1,09	70,40	81,90	11,50
	> 1 ≤ 2	23	77,46	7,49	1,56	58,40	87,20	28,80
	> 2 ≤ 3	24	80,53	6,50	1,33	67,50	91,50	24,00
	> 3 ≤ 4	21	84,11	4,85	1,06	78,10	95,20	17,10
	> 4 ≤ 5	21	84,52	6,80	1,48	63,20	94,80	31,60
	> 5 ≤16	42	85,10	4,61	0,71	68,80	99,50	30,70
	>16 ≤60	15	97,22	5,79	1,49	89,00	110,90	21,90
MCH	Cord	16	36,38	2,32	0,58	32,50	40,90	8,40
(f/l)	>1/12≤6/12m	14	26,63	2,94	0,78	20,30	32,40	12,10
	>6/12≤ 1	9	23,37	1,34	0,45	20,90	25,70	4,80
	> 1 ≤ 2	23	24,64	3,17	0,66	18,20	32,70	14,50
	> 2 ≤ 3	24	25,42	2,35	0,48	20,60	29,20	8,60
	> 3 ≤ 4	21	26,60	1,66	0,36	24,60	30,90	6,30
	> 4 ≤ 5	21	27,03	2,63	0,57	18,10	30,10	12,00
	> 5 ≤16	42	27,27	1,69	0,26	21,50	31,50	10,00
	>16 ≤60	15	31,67	2,01	0,52	29,60	36,00	6,40

	Age Groups	Frequency	Mean	Standard Deviation	SEM	Minimum	Maximum	Range
MCHC (g/dl)	Cord	16	32,36	0,82	0,20	30,60	33,40	2,80
	>1/12≤6/12m	14	30,60	0,66	0,17	29,40	31,80	2,40
	>6/12≤ 1	9	30,37	1,11	0,37	28,50	32,20	3,70
	> 1 ≤ 2	23	31,76	2,02	0,42	29,10	39,80	10,70
	> 2 ≤ 3	24	31,52	0,83	0,17	29,40	32,70	3,30
	> 3 ≤ 4	21	31,60	0,65	0,14	29,50	32,60	3,10
	> 4 ≤ 5	21	31,91	1,00	0,22	28,60	33,20	4,60
	> 5 ≤16	42	32,01	0,76	0,12	29,90	33,80	3,90
	>16 ≤60	15	32,58	0,74	0,19	31,10	33,60	2,50
Nutritional:								
Height (cms)	Cord	0						
	>1/12≤6/12m	14	58,64	6,05	1,62	48,00	69,00	21,00
	>6/12≤ 1	9	68,11	3,33	1,11	64,00	76,00	12,00
	> 1 ≤ 2	23	77,52	5,79	1,21	66,00	91,00	25,00
	> 2 ≤ 3	24	89,54	13,01	2,65	72,00	140,00	68,00
	> 3 ≤ 4	21	90,76	6,94	1,51	72,00	102,00	30,00
	> 4 ≤ 5	20	103,55	22,40	5,01	84,00	195,00	111,00
	> 5 ≤16	4 2	124,50	12,57	1,94	97,00	149,00	52,00
	>16 ≤60	0						

	Age Groups	Frequency	Mean	Standard Deviation	SEM	Minimum	Maximum	Range
Weight (Kg)	Cord	16	3,06	0,57	0,14	2,15	4,00	1,85
	>1/12≤6/12m	14	6,01	2,19	0,58	2,90	11,00	8,10
	>6/12≤ 1	9	9,86	0,97	0,32	8,50	11,50	3,00
	> 1 ≤ 2	23	11,72	2,25	0,47	8,50	18,00	9,50
	> 2 ≤ 3	24	12,50	2,12	0,43	8,00	16,00	8,00
	> 3 ≤ 4	21	14,60	2,66	0,58	9,00	19,00	10,00
	> 4 ≤ 5	20	16,08	2,96	0,66	12,00	22,00	10,00
	> 5 ≤16	42	24,55	6,70	1,03	12,50	44,00	31,50
	>16 ≤60	0						
Age (months)	Cord	0						
	>1/12≤6/12m	14	3,14	1,29	0,35	1,00	6,00	5,00
	>6/12≤ 1	9	8,11	1,45	0,48	6,00	10,00	4,00
	> 1 ≤ 2	23	17,35	3,74	0,78	12,00	23,00	11,00
	> 2 ≤ 3	24	29,92	3,05	0,62	24,00	35,00	11,00
	> 3 ≤ 4	21	40,71	4,00	0,87	36,00	48,00	12,00
	> 4 ≤ 5	21	51,43	3,53	0,77	44,00	58,00	14,00
	> 5 ≤16	42	101,05	26,30	4,06	61,00	159,00	98,00
	>16 ≤60	14	515,14	171,23	45,76	264,00	780,00	516,00

Table 2 Descriptive statistics of immunological and haematological parameters in sub age groups for CC

	Population	Sample size	Mean	Standard Deviation	SEM	Minimum	Maximum
Immunological:							
Absolute mononuclears ($cells/mm^3$)	Newborns	16	4925,45	1519,03	379,76	1824,00	7600,00
	>1m≤5	113	4868,03	2219,43	151,36	1755,00	9983,00
	>5 ≤16	42	2867,14	760,05	117,28	1440,00	4420,00
	>16≤60	26	2524,88	765,18	150,06	1551,00	4446,00
Absolute lymphocytes ($cells/mm^3$)	Newborns	16	3859,94	1120,93	280,23	1502,00	5616,00
	>1m≤5	113	4365,85	2165,92	147.72	1586,00	9589,00
	>5 ≤16	42	2627,09	707,20	109,12	1243,00	4160,00
	>16≤60	26	2216,30	693,55	136,02	1271,00	3900,00
T cell (E-rosette) ($cells/mm^3$)	Newborns	15	2355,20	1102,70	284,72	497,00	
	>1m≤5	113	2904,87	1510,63	1L4,49	842,00	6780,00
	>5 ≤16	42	1607,57	397,29	61,30	749,00	2762,00
	>16≤60	26	1728,54	597,47	117,17	924,00	3468,00
B cells (SIg) ($cells/mm^3$)	Newborns	15	590 00	595,61	153,78	0.00	1987,00
	>1m≤5	113	446,19	457,77	31,66	0,00	1628,00
	>5 ≤16	42	422,12	282,15	43,54	68,00	1591,00
	>16≤60	26	167,62	227,01	44,52	16,00	1136,00

	Population	Sample size	Mean	Standard Deviation	SEM	Minimum	Maximum
Null cells (cells/mm^3)	Newborns	15	2088,93	827,06	213,55	857,00	3765,00
	>1m≤5	113	1425,51	911,30	63,04	232,00	3872,00
	>5 ≤16	42	821,29	464,29	71,64	196,00	1959,00
	>16≤60	26	642,42	379,81	74,49	68,00	1292,00
FT cells (cells/mm^3)	Newborns	15	6,93	18,44	4,76	0,00	58,00
	>1m≤5	113	5,40	19,64	1,36	0,00	85,00
	>5 ≤16	42	6,00	17,42	2,69	0,00	88,00
	>16≤60	26	1,35	4,76	0,93	0,00	18,00
T cells (OKT3MoAb) (cells/mm^3)	Newborns	16	1636,50	648,28	162,07	662,00	2964,00
	>1m≤5	113	2582,52	1299,03	88,59	915,00	5831,00
	>5 ≤16	40	1664,35	525,02	83,01	691,00	2923,00
	>16≤60	26	1530,34	514,16	100,84	605,00	2490,00
OKT4+ cells (cells/mm^3)	Newborns	16	1109,56	635,27	158,82	249,00	2415,00
	>1m≤5	113	1550,82	814,96	55,58	463,00	3381,00
	>5 ≤16	40	1022,30	335,99	53,12	581,00	1882,00
	>16≤60	26	961,15	463,97	90,99	450,00	2134,00
OKT8+ cells (cells/mm^3)	Newborns	16	732,19	324,44	81,11	270,00	1672,00
	>1m≤5	113	1305,90	887,64	60,54	384,00	3961,00
	>5 ≤16	40	716,22	309,36	48,91	273,00	1646,00
	>16≤60	26	823,23	334,52	65,61	248,00	1556,00

	Population	Sample size	Mean	Standard Deviation	SEM	Minimum	Maximum
% OKT4 OF OKT3	Newborns	16	69,50	30,03	7,51	10,00	114,00
	>1m ≤5	113	60,97	16,82	1,15	32,00	100,00
	>5 ≤16	40	62,55	12,11	1,92	31,00	93,00
	>16 ≤60	26	62,69	16,40	3,22	32,00	93,00
% OKT8 of OKT3	Newborns	16	48,31	19,97	4,99	13,00	110,00
	>1m≤5	113	50,08	16,06	1,10	22,00	86,00
	>5 ≤16	40	42,97	11,91	1,88	20,00	78,00
	>16≤60	26	53,92	14,41	2,83	34,00	80,00
Ratio OKT4/OKT8	Newborns	16	1,78	1,50	0,38	0,84	6,74
	>1m≤5	113	1,40	0,73	0,05	0,51	3,57
	>5 ≤16	40	1,57	0,49	0,08	0,40	2,65
	>16≤60	26	1,26	0,49	0,10	0,44	2,29
% suppressor cell function (ConA 5 µg/25µl/ml of cells)	Newborns	10	41,10	26,91	8,51	-20,00	70,00
	>1m≤5	113	24,37	25,77	1,99	-28,00	82,00
	>5 ≤16	34	41,32	34,37	5,89	-13,00	95,00
	>16≤60	21	21,81	16,32	3,67	-10,00	59,00
% suppressor cell function (ConA 30 µg/25µg/ml of cells)	Newborns	10	49,90	31,84	10,07	3,00	92,00
	>1m≤5	113	42,21	29,17	2,18	-19,00	94,00
	>5 ≤16	39	43,03	32,36	5,18	-8,00	91,00
	>16≤60	20	31,00	31,03	6,94	-53,00	73,00

	Population	Sample size	Mean	Standard Deviation	SEM	Minimum	Maximum
MNC PWM stimulation (dpm)	Newborns	9	6406,42	4650,95	1550,31	1923,73	15753,00
	>1m≤5	113	6586,77	3081,67	265,23	381,39	12068,00
	>5 ≤16	34	7324,42	3597,26	616,92	1418,06	14697,71
	>16≤60	2	9058,77	5623,39	3976,34	5082,44	13035,00
WCC ($x\ 10^9/L$)	Newborns	16	13,56	4,20	1,05	7,60	20,50
	>1m≤5	113	9,98	3,71	0,25	4,30	19,10
	>5 ≤16	42	6,77	2,26	0,35	3,60	14,70
	>16≤60	26	5.74	1,55	0,30	10,90	4,10
% Neutrophils	Newborns	16	59,50	12,65	3,16	28,00	75,00
	>1m≤5	113	46,36	13,45	0,92	17,00	72,00
	>5 ≤16	41	51,32	9,20	1,44	34,00	68,00
	>16≤60	26	51,19	9,21	1,81	36,00	69,00
% Lymphocytes	Newborns	16	30,63	12,02	3,01	12,00	54,00
	>1m≤5	113	44,12	12,96	0,89	18,00	77,00
	>5 ≤16	42	40,55	8,41	1,30	24,00	56,00
	>16≤60	26	39,08	9,25	1,81	56,00	22,00

	Population	Sample size	Mean	Standard Deviation	SEM	Minimum	Maximum
% Monocytes	Newborns	16	7,13	4,40	1,09	1,00	16,00
	>1m≤5	113	4,06	3,32	0,23	1,00	14,00
	>5 ≤16	41	3,24	2,12	0,33	1,00	11,00
	>16≤60	26	5,23	3,73	0,73	1,00	15,00
% Eosinophils	Newborns	16	1,19	0,98	0,25	0,00	3,00
	>1m≤5	113	5,15	8,10	0,55	0,00	30,00
	>5 ≤16	41	4,56	5,73	0,90	0,00	22,00
	>16≤60	26	4,15	4,12	0,81	0,00	14,00
% Basophils	Newborns	16	0,25	0,58	0,14	0,00	2,00
	>1m≤5	113	0,20	0,68	0,05	0,00	2,00
	>5 ≤16	41	0,17	0,50	0,08	0,00	2,00
	>16≤60	26	0,42	0,76	0,15	0,00	3,00
% Atypical Lymphocytes	Newborns	16	0,50	0,73	0,18	0,00	2,00
	>1m≤5	113	0,32	1,18	0,08	0,00	4,00
	>5 ≤16	41	0,00	0,00	0,00	0,00	0,00
	>16≤60	26	0,12	0,43	0,08	0,00	2,00
% Others	Newborns	16	0,81	1,22	0,31	0,00	3,00
	>1m≤5	113	0,07	0,58	0,04	0,00	1,00
	>5 ≤16	41	0,00	0,00	0,00	0,00	0,00
	>16≤60	26	0,08	0,39	0,77	0,00	2,00

	Population	Sample size	Mean	Standard Deviation	SEM	Minimum	Maximum
Haematological:							
RBC ($\times 10^{12}$/L)	Newborns	16	4,18	0,47	0,12	3,46	5,07
	>1m≤5	113	4,31	0,50	0,04	3,37	5,44
	>5 ≤16	42	4,49	0,33	0,05	3,42	5,14
	>16≤60	25	4,70	0,42	0,08	3,80	5,47
Hb (g/dl)	Newborns	16	15,26	1,56	0,39	12,40	18,50
	>1m≤5	113	11,31	1,59	0,11	8,50	14,70
	>5 ≤16	42	12,21	0,93	0,14	9,90	13,90
	>16≤60	26	14,54	1,00	0,20	12,7	16,40
HCT (L/L)	Newborns	16	0,47	0,05	0,01	0,38	0,60
	>1m≤5	113	0,35	0,04	0,00	0,27	0,45
	>5 ≤16	42	0,38	0,03	0,00	0,32	0,43
	>16≤60	24	0,44	0,03	0,01	0,37	0,50
MCV (fl)	Newborns	16	113,34	6,14	1,53	102,80	126,00
	>1m≤5	113	81,52	7,67	0,55	62,50	96,40
	>5 ≤16	42	85,10	4,61	0,71	68,80	99,50
	>16≤60	25	95,10	5,98	1,20	82,60	110,90

	Population	Sample size	Mean	Standard Deviation	SEM	Minimum	Maximum
MCH	Newborns	16	36,38	2,32	0,58	32,50	40,90
(pg)	>1m≤5	113	26,03	2,78	0,20	19,30	32,20
	>5 ≤16	42	27,27	1,69	0,26	21,50	31,50
	>16≤60	24	31,22	1,99	0,41	27,80	36,00
MCHC	Newborns	16	32,36	0,82	0,20	30,60	33,40
(g/dL)	>1m≤5	113	31,94	1,36	0,10	29,40	34,70
	>5 ≤16	42	32,01	0,76	0,12	29,90	33,80
	>16≤60	24	32,84	0,83	0,17	31,10	34,20

Table 3a Comparisons of immunological, haematological between Males and Females of different age groups for Community Controls

Parameter	Age Group	Sex	Number	Mean	SD	SEM	Min	Max	Range
Immunological: Absolute mononuclears (cells/mm^3)	Cord blood	M	6	4915	1308	534	3192	6222	3030
		F	10	4932	1702	538	1824	7600	5776
	>1/12≤6	M	3	6839	1468	848	5248	8142	2894
		F	11	5742	1655	499	3264	9191	5927
	>6/12≤ 1	M	5	6506	2213	990	4424	9660	5236
		F	4	5386	2000	1000	3010	7824	4814
	> 1 ≤ 2	M	12	5590	1933	558	3293	10230	6937
		F	11	6658	3382	1170	3360	16864	13504
	> 2 ≤ 3	M	10	6036	3680	1164	2574	15834	13260
		F	14	5530	2090	559	2601	9589	6988
	> 3 ≤ 4	M	10	3837	1520	481	2508	7406	4898
		F	11	3335	729	220	1827	4488	2661
	> 4 ≤ 5	M	13	3831	1441	400	2310	7504	5194
		F	8	3990	786	278	3108	5456	2348
	> 5 ≤10	M	11	3279	866	261	1880	4420	2540
		F	13	2981	740	205	1786	4004	2218
	>10 ≤16	M	10	2574	617	195	1440	3520	2080
		F	8	2483	547	193	2077	3795	1718
Absolute Lymphocytes (cells/mm^3)	Cord blood	M	6	3966	1202	491	2364	5616	3252
		F	10	3796	1131	358	1520	5355	3835
	>1/12≤ 6	M	3	6333	1081	624	5084	6962	1878
		F	11	5387	1464	441	3200	8282	5082
	>6/12≤ 1	M	5	5619	1848	827	4345	8855	4510
		F	4	4841	1904	952	2795	7172	4377
	> 1 ≤ 2	M	12	5218	1782	545	2848	9300	6452
		F	11	6261	3866	1166	2926	16320	13394
	> 2 ≤ 3	M	10	5757	3662	1158	2475	15631	13156
		F	14	5089	2099	561	2244	9589	7345
	> 3 ≤ 4	M	10	3588	1322	418	2376	6762	4386
		F	11	3174	711	214	1764	4400	2636
	> 4 ≤ 5	M	13	3660	1416	393	2275	7236	4961
		F	8	3780	759	268	2832	5084	2252
	> 5 ≤10	M	11	3015	788	238	1786	4160	2374
		F	13	2713	637	177	1710	3741	2031
	>10 ≤16	M	10	2323	707	224	1243	3432	2189
		F	8	2334	472	167	1943	3450	1507

Parameter	Age Group	Sex	Number	Mean	SD	SEM	Min	Max	Range
T cell (E-rosette) (cells/mm^3)	Cord blood	M	5	2531	1237	553	1368	4355	2987
		F	10	2267	1089	344	497	4078	3581
	>1/12≤ 6	M	3	3962	1323	764	2939	5456	2517
		F	11	3812	1150	347	2298	5607	3309
	>6/12≤ 1	M	5	3585	1455	651	2760	6182	3422
		F	4	3681	1331	666	1867	4851	2984
	> 1 ≤ 2	M	11	3603	1484	448	1752	6547	4795
		F	11	4203	2385	719	2184	9613	7429
	> 2 ≤ 3	M	10	3613	2297	726	1341	9026	7685
		F	14	3479	1587	424	1691	6713	5022
	> 3 ≤ 4	M	10	2219	1000	316	1131	4814	3683
		F	11	2375	656	198	1297	3338	2041
	> 4 ≤ 5	M	12	2637	1096	316	1224	4935	3711
		F	8	2444	428	151	1935	3219	1284
	> 5 ≤10	M	11	1772	493	149	921	2416	1495
		F	13	1657	434	120	1072	2762	1690
	>10 ≤16	M	10	1419	305	97	749	1901	1152
		F	8	1536	178	63	1224	1746	522
B Cells (SIg) (cells/mm^3)	Cord blood	M	5	528	467	209	0	1104	1104
		F	10	621	672	213	18	1987	1969
	>1/12≤ 6	M	3	766	747	431	315	1628	1313
		F	11	576	448	135	45	1562	1517
	>6/12≤ 1	M	5	603	410	183	98	966	868
		F	4	585	178	89	421	814	393
	> 1 ≤ 2	M	11	494	349	105	56	1314	1258
		F	11	505	297	89	168	966	798
	> 2 ≤ 3	M	10	679	1007	318	38	3483	3445
		F	14	579	428	114	0	1438	1438
	> 3 ≤ 4	M	10	345	273	86	100	1037	937
		F	11	218	182	55	39	632	593
	> 4 ≤ 5	M	12	272	144	42	86	588	502
		F	8	464	300	106	76	985	909
	> 5 ≤10	M	11	509	444	134	132	1591	1459
		F	13	477	248	69	111	920	809
	>10 ≤16	M	10	344	111	35	184	523	339
		F	8	312	155	55	68	508	440

Parameter	Age Group	Sex	Number	Mean	SD	SEM	Min	Max	Range
Null cells (cells/mm^3)	Cord blood	M	5	2189	309	138	1867	2644	777
		F	10	2039	1007	318	857	3765	2908
	>1/12≤ 6	M	3	2110	1115	644	1058	3279	2221
		F	11	1391	636	192	392	2116	1724
	>6/12≤ 1	M	5	1718	586	262	1132	2512	1380
		F	4	1281	841	420	722	2504	1782
	> 1 ≤ 2	M	11	1656	838	253	350	3376	3026
		F	11	1949	1589	479	586	6408	5812
	> 2 ≤ 3	M	10	1745	997	315	214	3325	3111
		F	14	1449	659	176	502	2472	1970
	> 3 ≤ 4	M	10	1272	645	204	540	2719	2179
		F	11	738	354	107	311	1498	1187
	> 4 ≤ 5	M	12	1054	589	170	602	2776	2174
		F	8	1074	303	107	528	1411	883
	> 5 ≤10	M	11	915	381	115	529	1610	1081
		F	13	873	553	153	196	1959	1763
	>10 ≤16	M	10	804	373	118	298	1232	934
		F	8	632	541	191	257	1935	1678
FT cells (cells/mm^3)	Cord blood	M	5	11,60	25,94	11,60	0	58	58
		F	10	4,60	14,55	4,60	0	46	46
	>1/12≤ 6	M	3	0	0	0	0	0	0
		F	10	0	0	0	0	0	0
	>6/12≤ 1	M	5	0	0	0	0	0	0
		F	4	0	0	0	0	0	0
	> 1 ≤ 2	M	5	0	0	0	0	0	0
		F	4	0	0	0	0	0	0
	> 2 ≤ 3	M	10	0	0	0	0	0	0
		F	14	5,00	18,71	5,00	0	70,00	70
	> 3 ≤ 4	M	10	0	0	0	0	0	0
		F	11	3,27	10,85	3,27	0	36,00	36
	> 4 ≤ 5	M	12	0	0	0	0	0	0
		F	8	0	0	0	0	0	0
	> 5 ≤10	M	11	10,82	27,23	8,21	0	88	88
		F	13	4,31	15,53	4,31	0	56	56
	>10 ≤16	M	10	5,60	12,26	3,88	0	35	35
		F	8	2,62	7,42	2,62	0	21	21

Parameter	Age Group	Sex	Number	Mean	SD	SEM	Min	Max	Range
T cells (OKT3MoAb) (cells/mm^3)	Cord blood	M	6	1540	353	144	1140	2080	940
		F	10	1695	788	249	662	2964	2302
	>1/12≤ 6	M	3	3483	289	167	3149	3664	515
		F	11	2946	1152	347	1828	5328	3500
	>6/12≤ 1	M	5	3331	1513	677	2433	5989	3556
		F	4	2437	541	270	1957	3051	1094
	> 1 ≤ 2	M	12	3092	1112	321	1795	5831	4036
		F	11	3604	2390	721	1478	9444	7966
	> 2 ≤ 3	M	10	2818	1434	453	1132	5542	4410
		F	14	2998	1350	361	1239	5370	4131
	> 3 ≤ 4	M	10	1812	628	198	816	2962	2146
		F	11	1797	450	136	1060	2474	1414
	> 4 ≤ 5	M	13	2161	829	230	1225	3977	2752
		F	8	2565	321	113	2095	2982	887
	> 5 ≤10	M	11	1878	478	144	1278	2710	1432
		F	12	1811	565	163	1054	2923	1869
	>10 ≤16	M	9	1539	440	147	691	2112	1421
		F	8	1292	447	158	900	2315	1415
OKT4+ cells (cells/mm^3)	Cord blood	M	6	1157	566	231	249	1820	1571
		F	10	1081	701	222	304	2416	2112
	>1/12≤ 6	M	3	2284	76	44	2210	2362	152
		F	11	2198	727	219	1240	3368	2628
	>6/12≤ 1	M	5	2283	743	332	1460	3381	1921
		F	4	1934	345	172	1617	2425	808
	> 1 ≤ 2	M	12	1604	713	206	724	3069	2345
		F	11	2271	1445	436	918	6071	5153
	> 2 ≤ 3	M	10	1433	593	187	463	2850	2387
		F	14	1491	826	221	216	3452	3236
	> 3 ≤ 4	M	10	1042	366	116	605	1629	1024
		F	11	1186	377	114	732	1980	1248
	> 4 ≤ 5	M	13	1321	640	177	602	2476	1874
		F	8	1512	407	144	949	2237	1288
	> 5 ≤10	M	11	1087	326	98	659	1846	1187
		F	12	1104	395	114	581	1882	1301
	>10 ≤16	M	9	932	304	101	618	1514	896
		F	8	914	288	102	680	1556	876

Parameter	Age Group	Sex	Number	Mean	SD	SEM	Min	Max	Range
OKT8+ cells (cells/mm^3)	Cord blood	M	6	613	204	83	270	872	602
		F	10	804	370	117	401	1672	1271
	>1/12≤ 6	M	3	1470	707	408	787	2198	1411
		F	11	1294	708	214	530	2849	2319
	>6/12≤ 1	M	5	1701	1335	597	689	3961	3272
		F	4	767	464	232	301	1336	1035
	> 1 ≤ 2	M	12	1553	783	226	507	3581	3074
		F	11	1944	1526	460	494	5734	5240
	> 2 ≤ 3	M	10	1664	1319	417	438	4750	4312
		F	14	1412	760	203	697	3410	2713
	> 3 ≤ 4	M	10	1032	358	113	454	1482	1028
		F	11	882	321	97	238	1364	1126
	> 4 ≤ 5	M	13	959	208	58	668	1528	860
		F	8	1100	361	127	594	1637	1043
	> 5 ≤10	M	11	819	372	112	348	1646	1298
		F	12	740	352	102	273	1402	1129
	>10 ≤16	M	9	722	234	78	360	1017	657
		F	8	533	147	52	383	736	353
% OKT4 of OKT3	Cord blood	M	6	72,50	27,49	11,22	20,00	93,00	73
		F	10	67,70	32,77	10,36	10,00	114,00	104
	>1/12≤ 6	M	3	66,00	7,81	4,51	61,00	75,00	14
		F	11	77,09	15,46	4,66	59,00	114,00	55
	>6/12≤ 1	M	5	59,00	28,63	12,80	13,00	83,00	70
		F	4	60,25	31,31	15,65	14,00	80,00	66
	> 1 ≤ 2	M	12	52,42	18,10	5,23	32,00	85,00	53
		F	11	66,09	18,16	5,48	45,00	114,00	69
	> 2 ≤ 3	M	10	56,80	23,19	7,33	36,00	106,00	70
		F	14	50,36	16,65	4,45	9,00	77,00	68
	> 3 ≤ 4	M	10	59,00	13,20	4,18	39,00	74,00	35
		F	11	67,72	20,70	6,24	42,00	100,00	58
	> 4 ≤ 5	M	10	60,46	15,48	4,29	34,00	83,00	49
		F	8	59,00	14,80	5,23	35,00	84,00	49
	> 5 ≤10	M	11	58,91	13,46	4,06	31,00	78,00	47
		F	12	64,41	8,61	2,48	46,00	69,00	23
	>10 ≤16	M	9	61,78	13,55	4,52	49,00	90,00	41
		F	8	71,62	10,45	3,69	58,00	93,00	35

Parameter	Age Group	Sex	Number	Mean	SD	SEM	Min	Max	Range
% T8 of T3	Cord	M	6	41,83	14,36	5,86	13,00	50,00	37
		F	10	52,20	22,47	7,11	30,00	110,00	80
	>1/12≤6/12	M	6	41,33	17,62	10,17	25,00	60,00	35
		F	11	43,00	13,94	4,20	22,00	66,00	44
	>6/12≤ 1	M	5	47,80	20,49	9,16	27,00	73,00	46
		F	4	29,75	14,41	7,20	15,00	49,00	34
	>1 ≤ 2	M	12	50,67	16,84	4,86	22,00	83,00	61
		F	11	51,09	13,63	4,11	32,00	73,00	41
	> 2 ≤ 3	M	10	57,60	27,46	8,68	20,00	108,00	88
		F	14	48,57	14,76	3,95	21,00	84,00	63
	> 3 ≤ 4	M	10	57,90	12,66	4,00	38,00	80,00	42
		F	11	50,00	22,41	6,76	22,00	103,00	81
	> 4 ≤ 5	M	13	47,92	12,09	3,35	28,00	65,00	37
		F	8	42,88	13,11	4,63	23,00	61,00	38
	> 5 ≤10	M	11	42,91	14,65	4,42	26,00	78,00	52
		F	12	39,75	10,97	3,17	20,00	57,00	37
	>10 ≤16	M	9	47,11	8,40	2,80	33,00	62,00	29
		F	8	43,25	13,20	4,67	26,00	67,00	41
Ratio OKT4/OKT8	Cord blood	M	6	2,33	2,33	0,91	0,44	6,74	6,30
		F	10	1,46	0,82	0,26	0,18	3,00	2,82
	>1/12≤ 6	M	3	1,86	1,02	0,59	1,04	3,00	1,96
		F	11	1,97	0,71	0,21	0,97	3,58	2,61
	>6/12≤ 1	M	5	1,79	0,97	0,43	0,85	3,07	2,22
		F	4	3,33	2,02	1,01	1,34	6,10	4,76
	> 1 ≤ 2	M	12	1,18	0,66	0,19	0,38	3,00	2,62
		F	11	1,46	0,82	0,25	0,62	3,57	2,95
	> 2 ≤ 3	M	10	1,54	1,16	0,37	0,56	3,92	3,36
		F	14	1,18	0,65	0,18	0,16	2,80	2,64
	> 3 ≤ 4	M	10	1,09	0,42	0,13	0,49	1,84	1,35
		F	11	1,65	1,09	0,33	0,54	4,38	3,84
	> 4 ≤ 5	M	13	1,36	0,55	0,15	0,58	2,20	1,62
		F	8	1,46	0,43	0,15	0,85	2,37	1,52
	> 5 ≤10	M	11	1,53	0,59	0,18	0,40	2,65	2,25
		F	12	1,62	0,41	0,12	1,07	2,43	1,36
	>10 ≤16	M	9	1,35	0,36	0,12	0,79	1,79	1,00
		F	8	1,79	0,56	0,20	0,97	2,56	1,59

Parameter	Age Group	Sex	Number	Mean	SD	SEM	Min	Max	Range
% suppressor cell (ConA 5µg/25µl/ml of cells)	Cord blood	M	3	27,00	40,71	23,50	-20,00	51,00	71
		F	7	47,14	19,79	7,48	23,00	70,00	47
	>1/12≤ 6	M	2	24,00	19,80	14,00	10,00	38,00	28
		F	6	16,83	24,83	10,14	-11,00	58,00	72
	>6/12≤ 1	M	4	12,25	18,45	9,22	0	39,00	39
		F	3	5,67	9,45	5,46	-5,00	13,00	18
	> 1 ≤ 2	M	11	7,09	15,56	4,69	-26,00	27,00	53
		F	9	43,22	41,08	13,69	-3,00	98,00	101
	> 2 ≤ 3	M	7	34,29	27,32	10,33	-17,00	67,00	84
		F	12	17,25	27,98	8,08	-35,00	84,00	119
	> 3 ≤ 4	M	7	28,43	11,24	4,25	17,00	46,00	29
		F	7	23,29	31,24	11,81	-44,00	50,00	94
	> 4 ≤ 5	M	7	49,57	16,07	6,07	30,00	73,00	43
		F	7	42,00	26,13	9,88	17,00	89,00	72
	> 5 ≤10	M	10	43,40	26,77	8,46	9,00	82,00	73
		F	10	47,50	32,79	10,37	-1,00	90,00	91
	>10 ≤16	M	8	37,25	39,57	13,99	-11,00	89,00	100
		F	6	33,00	46,76	19,09	-13,00	95,00	108
% suppressor cell function (ConA 30µg/25µl/ml) of cells	Cord blood	M	3	42,67	43,88	25,33	8,00	92,00	84
		F	7	53,00	29,01	10,97	3,00	79,00	76
	>1/12≤ 6	M	2	64,50	14,85	10,50	54,00	75,00	21
		F	8	55,13	22,18	7,84	25,00	88,00	63
	>6/12≤ 1	M	4	32,50	31,76	15,88	-5,00	69,00	74
		F	3	37,33	5,03	2,91	32,00	42,00	10
	> 1 ≤ 2	M	11	49,91	29,33	8,84	-5,00	81,00	86
		F	10	42,60	51,00	16,13	-60,00	94,00	154
	> 2 ≤ 3	M	8	55,38	26,20	9,26	22,00	85,00	73
		F	14	35,21	35,23	9,41	-25,00	97,00	122
	> 3 ≤ 4	M	9	54,67	36,45	12,15	-30,00	94,00	124
		F	8	59,13	20,84	7,37	32,00	85,00	53
	> 4 ≤ 5	M	8	65,88	17,70	6,26	39,00	89,00	50
		F	8	71,75	21,78	7,70	37,00	96,00	59
	> 5 ≤10	M	11	32,00	24,19	7,29	-6,00	80,00	86
		F	11	57,18	35,27	10,63	-4,00	91,00	95
	>10 ≤16	M	10	49,00	28,16	8,91	15,00	89,00	74
		F	7	29,57	39,13	14,79	-8,00	86,00	94

Parameter	Age Group	Sex	Number	Mean	SD	SEM	Min	Max	Range
MNC PWM stimulation (dpm)	Cord blood	M	3	11084	4934	2848	5923	15753	9830
		F	6	4067	2276	929	1924	7090	5166
	>1/12≤ 6	M	1	6318	-	-	6318	6318	-
		F	4	8139	1561	781	6116	9683	3567
	>6/12≤ 1	M	3	3926	1514	814	2359	5382	3023
		F	2	4930	1596	1128	3801	6058	2257
	> 1 ≤ 2	M	12	5664	2888	834	1669	13407	10398
		F	9	3951	2181	727	1091	6814	5723
	> 2 ≤ 3	M	8	5949	3607	1275	582	11012	10430
		F	11	6418	1908	575	3592	10304	6712
	> 3 ≤ 4	M	8	5114	4590	1623	796	12307	1261
		F	7	6812	2436	921	1729	9013	7284
	> 4 ≤ 5	M	11	5833	1583	477	2206	7299	5093
		F	6	8523	1962	801	4946	10143	5197
	> 5 ≤10	M	11	6052	3102	935	1418	11279	9861
		F	9	6505	3232	1077	1854	10826	8972
	>10 ≤16	M	8	7905	4105	1451	1648	14020	12373
		F	6	10112	3350	1368	5383	14698	9315
WCC ($\times 10^9$/L)	Cord blood	M	6	11,88	4,38	1,79	7,60	19,70	12,10
		F	10	14.56	3,97	1,26	7,60	20,50	12,90
	>1/12≤ 6	M	3	9,60	1,93	1,11	8,20	11,80	3,60
		F	11	8,86	1,59	0,48	6,40	12,00	5,60
	>6/12≤ 1	M	5	8,62	2,15	0,96	6,00	11,50	5,50
		F	4	8,98	5,16	2,58	4,30	16,30	12,00
	> 1 ≤ 2	M	12	12,48	3,70	1,07	7,20	18,60	11,40
		F	22	12,32	6,08	1,83	6,00	27,20	21,20
	> 2 ≤ 3	M	10	11,16	4,20	1,33	5,60	20,30	14,70
		F	14	10,98	4,58	1,23	5,10	22,30	17,20
	> 3 ≤ 4	M	10	8,81	2,95	0,93	6,60	16,10	9,50
		F	11	8,22	1,94	0,59	6,10	12,60	6,50
	> 4 ≤ 5	M	13	8,97	3,21	0,89	3,50	13,40	9,90
		F	8	8,25	1,94	0,68	5,90	12,40	6,50
	> 5 ≤10	M	11	8,10	3,09	0,93	4,70	14,70	10,00
		F	13	7,06	1,98	0,55	3,70	9,80	6,10
	>10 ≤16	M	10	5,70	1,50	0,47	3,60	8,80	5,20
		F	8	5,82	1,04	0,37	4,10	6,90	2,80

Parameter	Age Group	Sex	Number	Mean	SD	SEM	Min	Max	Range
% Neutrophils	Cord blood	M	6	52	17,60	7,19	28	73	45
		F	10	64	6,50	2,05	55	75	20
	>1/12≤ 6	M	3	27	6,81	3,93	19	32	13
		F	11	34	12,72	3.84	8	48	40
	>6/12≤ 1	M	5	28	12,26	5,48	13	40	27
		F	4	33	13,00	6,50	22	52	30
	> 1 ≤ 2	M	12	49	14,32	4,13	27	71	44
		F	11	43	12,70	3,83	23	72	49
	> 2 ≤ 3	M	10	41	15,77	4,99	9	71	62
		F	14	42	10,25	2,74	26	63	37
	> 3 ≤ 4	M	10	53	9,00	2,85	43	71	28
		F	11	55	13,30	4,00	28	71	43
	> 4 ≤ 5	M	13	53	10,07	2,79	33	70	37
		F	8	48	5,84	2,07	40	58	18
	> 5 ≤10	M	11	51	9,00	2,59	41	66	25
		F	12	51	9,00	2,50	34	65	31
	>10 ≤16	M	10	49	9,00	2,71	35	62	27
		F	8	54	12,00	4,31	36	68	32
% Lymphocytes	Cord blood	M	6	38	16,21	6,62	12	54	42
		F	10	26	6,48	2,05	19	36	17
	>1/12≤ 6	M	3	67	10,79	6,23	59	79	20
		F	11	59	11,10	3,35	50	78	28
	>6/12≤ 1	M	5	64	12,67	5,67	50	80	30
		F	4	57	9,29	4,64	44	65	21
	> 1 ≤ 2	M	12	43	12,91	3,73	22	64	42
		F	11	49	13,97	4,21	82	64	42
	> 2 ≤ 3	M	10	50	14,30	4,53	25	77	58
		F	14	47	6,06	1,62	34	55	21
	> 3 ≤ 4	M	10	41	7,32	2,31	27	51	24
		F	11	40	10,20	3,07	25	50	25
	> 4 ≤ 5	M	13	43	11,48	3,18	24	65	41
		F	8	48	5,09	1,80	40	55	15
	> 5 ≤10	M	11	39	9,29	2,80	24	53	29
		F	13	40	7,91	2,19	28	55	27
	>10 ≤16	M	10	43	7,62	2,41	33	54	21
		F	8	41	9,86	3,49	29	36	27

Parameter	Age Group	Sex	Number	Mean	SD	SEM	Min	Max	Range
% Monocytes	Cord blood	M	6	8	5,96	2,43	1	16	15
		F	10	7	3.49	1,10	4	14	10
	>1/12≤ 6	M	3	5	4,62	2,67	2	10	8
		F	11	4	2,88	0,87	0	9	9
	>6/12≤ 1	M	5	3	2,35	1,05	1	7	6
		F	4	7	4,86	2,43	4	14	10
	> 1 ≤ 2	M	12	3	1,64	0,47	1	6	5
		F	11	4	3,01	0,91	1	10	9
	> 2 ≤ 3	M	10	2	1,96	0,62	0	6	6
		F	14	5	3,67	0,98	0	13	13
	> 3 ≤ 4	M	10	3	1,65	0,52	1	6	5
		F	11	2	0,60	0,18	1	3	2
	> 4 ≤ 5	M	13	2	0,90	0,25	1	4	3
		F	8	3	1,30	0,46	1	5	4
	> 5 ≤10	M	11	3	1,25	0,38	1	5	4
		F	12	4	3,22	0,93	1	11	10
	>10 ≤16	M	10	3	1,83	0,58	1	7	6
		F	8	2	1,07	0,38	2	5	3
% Basophils	Cord blood	M	6	0	0	0	0	0	0
		F	10	0,4	0,70	0,22	0	2	2
	>1/12≤ 6	M	3	0	0	0	0	0	0
		F	11	0,18	0,40	0,12	0	1	1
	>6/12≤ 1	M	5	0,02	0,45	0,20	0	1	1
		F	4	0,50	1,00	0,50	0	2	2
	> 1 ≤ 2	M	12	0,25	0,62	0,18	0	2	2
		F	11	0,09	0,30	0,09	0	1	1
	> 2 ≤ 3	M	10	0	0	0	0	0	0
		F	14	0,07	0,27	0,07	0	1	1
	> 3 ≤ 4	M	10	0,30	0,67	0,21	0	2	2
		F	11	0,09	0,30	0,09	0	1	1
	> 4 ≤ 5	M	13	0	0	0	0	0	0
		F	8	0,13	0,35	0,13	0	1	1
	> 5 ≤10	M	11	0,27	0,65	0,19	0	2	2
		F	12	0,25	0,62	0,18	0	2	2
	>10 ≤16	M	10	0,10	0,32	0,10	0	1	1
		F	8	0	0	0	0	0	0

Parameter	Age Group	Sex	Number	Mean	SD	SEM	Min	Max	Range
% Atypical lymphocytes	Cord blood	M	6	0,50	0,84	0,34	0	2	2
		F	10	0,50	0,71	0,22	0	2	2
	>1/12≤ 6	M	3	0	0	0	0	0	0
		F	11	1,18	1,85	0,55	0	4	4
	>6/12≤ 1	M	5	2,00	1,87	0,84	0	4	4
		F	4	1,00	2,00	1,00	0	4	4
	> 1 ≤ 2	M	12	0	0	0	0	0	0
		F	11	0	0	0	0	0	0
	> 2 ≤ 3	M	10	0	0	0	0	0	0
		F	14	0	0	0	0	0	0
	> 3 ≤ 4	M	10	0	0	0	0	0	0
		F	11	0	0	0	0	0	0
	> 4 ≤ 5	M	13	0	0	0	0	0	0
		F	8	0	0	0	0	0	0
	> 5 ≤10	M	11	0	0	0	0	0	0
		F	12	0	0	0	0	0	0
	>10 ≤16	M	10	0	0	0	0	0	0
		F	8	0	0	0	0	0	0
% Others	Cord blood	M	6	0,17	0,41	0,17	0	1	1
		F	10	1,20	1,40	0,44	0	3	3
	>1/12≤ 6	M	3	0	0	0	0	0	0
		F	11	0,09	0,30	0,09	0	1	1
	>6/12≤ 1	M	5	2,20	3,19	1,43	0	7	7
		F	5	0	0	0	0	0	0
	> 1 ≤ 2	M	12	0	0	0	0	0	0
		F	11	0	0	0	0	0	0
	> 2 ≤ 3	M	10	0	0	0	0	0	0
		F	14	0	0	0	0	0	0
	> 3 ≤ 4	M	10	0	0	0	0	0	0
		F	11	0	0	0	0	0	0
	> 4 ≤ 5	M	13	0	0	0	0	0	0
		F	8	0	0	0	0	0	0
	> 5 ≤10	M	11	0	0	0	0	0	0
		F	12	0	0	0	0	0	0
	>10 ≤16	M	10	0	0	0	0	0	0
		F	8	0	0	0	0	0	0

Parameter	Age Group	Sex	Number	Mean	SD	SEM	Min	Max	Range
Haematological:									
RBC ($\times 10^{12}$/L)	Cord blood	M	6	3,91	0,48	0,20	3,47	4,81	1,34
		F	10	4,33	0,42	0,13	3,46	5,07	1,61
	>1/12≤ 6	M	3	4,04	0,53	0,31	3,42	4,36	0,94
		F	11	3,51	0,57	0,17	2,26	4,39	2,13
	>6/12≤ 1	M	5	4,39	0,32	0,14	4,06	4,81	0,75
		F	4	4,48	0,21	0,10	4,24	4,66	0,42
	> 1 ≤ 2	M	12	4,30	0,24	0,07	4,91	4,79	0,88
		F	11	4,38	0,51	0,15	3,32	5,38	2,06
	> 2 ≤ 3	M	10	4,25	0,43	0,14	3,57	5,10	1,53
		F	14	4,35	0,52	0,14	3,39	5,17	1,78
	> 3 ≤ 4	M	10	4,18	0,18	0,06	3,98	4,55	0,57
		F	11	4,20	0,26	0,08	3,82	4,72	0,90
	> 4 ≤ 5	M	13	4,44	0,42	0,12	3,82	5,29	1,47
		F	8	4,04	0,16	0,06	3,84	4.28	0,44
	> 5 ≤10	M	11	4,46	0,33	0,10	3,63	4,92	1,29
		F	13	4,35	0,36	0,10	3,42	4,96	1,54
	>10 ≤16	M	10	4,68	0,27	0,85	4,15	5,14	0,99
		F	8	4,51	0,29	0,10	4,07	5,02	0,95
Hb (g/dL)	Cord blood	M	6	14,82	1,31	0,53	13,30	16,50	3,20
		F	10	15,51	1,70	0,54	12,40	18,50	6,10
	>1/12≤ 6	M	3	9,53	1,02	0,59	8,80	10,70	1,90
		F	11	9,51	1,10	0,33	6,80	10,80	4,00
	M>6/12≤ 1	M	5	10,26	0,69	0,31	9,30	11,20	1,90
		F	4	10,40	0,53	0,26	9,70	10,90	1,20
	> 1 ≤ 2	M	12	10,45	1,26	0,37	7,80	12,20	4,40
		F	11	10,83	1,28	0,38	8,20	12,30	4,10
	> 2 ≤ 3	M	10	10,70	0,72	0,23	9,50	11,50	2,00
		F	14	11,00	1,25	0,33	8,40	12,70	4,30
	> 3 ≤ 4	M	10	10,93	0,61	0,19	10,10	12,00	1,90
		F	11	11,28	0,56	0,17	10,60	12,10	1,50
	> 4 ≤ 5	M	13	11,65	1,16	0,32	9,60	13,90	4,30
		F	8	11,33	0,73	0,26	10,20	12,20	2,00
	> 5 ≤10	M	11	12,09	0,74	0,22	10,20	12,9	2,70
		F	13	11,60	0,94	0,26	9,90	13,40	3,50
	>10 ≤16	M	10	12,83	0,76	0,24	11,50	13,70	2,20
		F	8	12,59	0,79	0,28	11,70	13,90	2,20

Parameter	Age Group	Sex	Number	Mean	SD	SEM	Min	Max	Range
HCT (L/L)	Cord blood	M	6	0,45	0,04	0,02	0,40	0,51	0,11
		F	10	0,49	0,06	0,02	0,38	0,60	0,22
	>1/12≤ 6	M	3	0,31	0,03	0,0	10,29	0,34	0,05
		F	11	0,31	0,03	0,01	0,22	0,35	0,30
	>6/12≤ 1	M	5	0,34	0,03	0,01	0,31	0,38	0,07
		F	4	0,34	0,01	0,01	0,33	0,35	0,02
	> 1 ≤ 2	M	12	0,33	0,03	0,01	0,25	0,38	0,13
		F	11	0,34	0,04	0,01	0,27	0,39	0,12
	> 2 ≤ 3	M	10	0,34	0,02	0,01	0,31	0,37	0,06
		F	14	0,35	0,04	0,01	0,26	0,43	0,17
	> 3 ≤ 4	M	10	0,34	0,02	0,01	0,32	0,37	0,05
		F	11	0,36	0,02	0,01	0,33	0,38	0,05
	> 4 ≤ 5	M	13	0,37	0,03	0,01	0,33	0,44	0,11
		F	8	0,35	0,02	0,01	0,32	0,37	0,05
	> 5 ≤10	M	11	0,38	0,02	0,01	0,32	0,40	0,08
		F	13	0,36	0,02	0,01	0,32	0,42	0,10
	>10 ≤16	M	10	0,40	0,02	0,01	0,36	0,43	0,07
		F	8	0,39	0,02	0,01	0,36	0,43	0,07
MCV (fl)	Cord blood	M	6	116,52	6,12	2,49	108,00	126,00	18,00
		F	10	111,44	5.60	1,77	102,80	118,90	16,10
	>1/12≤ 6	M	3	77,80	8,61	4,97	69,00	86,00	17,20
		F	11	89,33	7,22	2,18	79,10	103,30	24,20
	>6/12≤ 1	M	5	77,28	1,83	0,82	74,20	78,60	4,40
		F	4	76,48	4,83	2,41	70,40	81,90	11,50
	> 1 ≤ 2	M	12	77,46	7,49	2,16	58,40	87,20	28,80
		F	11	77,46	7,84	2,36	59,90	85,70	25,80
	> 2 ≤ 3	M	10	81,06	5,34	1,69	73,00	91,50	18,50
		F	14	80,16	7,38	1,97	67,50	90,20	22,70
	> 3 ≤ 4	M	10	82,55	3,69	1,17	78,10	97,80	9,70
		F	11	85,54	5,48	1,65	78,40	95,20	16,80
	> 4 ≤ 5	M	13	83,25	7,89	2,19	63.20	94,80	31,60
		F	8	86,59	4,21	1,49	79,40	92,10	12,70
	> 5 ≤10	M	11	84,83	2,68	0,81	81,50	89,10	7,60
		F	13	84,06	7,15	1,98	68,80	99,50	30,70
	>10 ≤16	M	10	85,49	2,36	0,74	80,90	89,10	8,20
		F	8	86,65	3,80	1,34	80,20	91,50	11,30

Parameter	Age Group	Sex	Number	Mean	SD	SEM	Min	Max	Range
MCH (pg)	Cord blood	M	6	37,61	2.75	1.12	32,50	40,90	8,40
		F	10	35,64	1,77	0,56	33,00	38,30	5,30
	>1/12≤ 6	M	3	23,80	3,21	1,85	20,30	26,60	6,30
		F	11	27,40	2,47	0,74	24,30	32,40	8,10
	>6/12≤ 1	M	5	23,40	0,82	0,37	22,40	24,50	2,10
		F	4	23,33	1.97	0,99	20,90	25,70	4,80
	> 1 ≤ 2	M	12	24,33	2,67	0,77	18,20	28,20	10,00
		F	11	24,98	3,75	1,13	18,20	32,70	14,50
	> 2 ≤ 3	M	10	25,37	1,80	0,57	22,20	28,90	6,70
		F	14	25,45	2,74	0,73	20,60	29,20	8,60
	> 3 ≤ 4	M	10	26,19	1,18	0,37	24,70	27,90	3,20
		F	11	26,97	1,98	0,60	24,60	30,90	6,30
	> 4 ≤ 5	M	13	26,39	3,01	0,84	18,10	29,70	11,60
		F	8	28,06	1,48	0,52	25,60	30,10	4,50
	> 5 ≤10	M	11	27,19	0,85	0,26	25,80	28,20	2,40
		F	13	26,78	2,47	0,68	21,50	31,50	10,00
	>10 ≤16	M	10	27,43	1,26	0,40	25,90	29,50	3,60
		F	8	27,97	1,48	0,52	25,90	29,80	3,90
MCHC (g/dL)	Cord blood	M	6	32,70	0,71	0,29	31,50	33,40	1,90
		F	10	32,16	0,84	0,27	30,60	33,30	2,70
	>1/12≤ 6	M	3	30,47	0,95	0,55	29,44	31,20	1,80
		F	11	30,63	0,61	0,18	29,70	31,80	2,10
	>6/12≤ 1	M	5	30,30	1,43	0,64	28,50	32,20	3,70
		F	4	30,45	0,71	0,36	29,70	31,40	1,70
	> 1 ≤ 2	M	12	31,37	0,94	0,27	29,40	32,60	3,20
		F	11	32,18	2,76	0,83	29,10	39,80	10,70
	> 2 ≤ 3	M	10	31,28	0,73	0,23	30,10	32,30	2,20
		F	14	31,69	0,87	0,23	29,40	32,70	3,30
	> 3 ≤ 4	M	10	31,72	0,17	0,05	31,50	32,00	0,50
		F	11	31,48	0,89	0,27	29,50	32,60	3,10
	> 4 ≤ 5	M	13	31,62	1,06	0,29	28,60	32,70	4,10
		F	8	32,38	0,73	0,26	31,00	33,20	2,20
	> 5 ≤10	M	11	32,03	0,59	0,18	30,90	33,20	2,20
		F	13	31,83	0,76	0,21	30,50	33,20	2,70
	>10 ≤16	M	10	32,04	1,06	0,33	29,90	33,80	3,90
		F	8	32,24	0,58	0,21	31,10	33,20	2,10

Table 3b Comparisons of immunological, haematological and nutritional parameters between males and females 5-10 and 10-16 years in CC

	Age	Sex	Freq.	Mean	SD	SEM	Minimum	Maximum	Range	p value
Absolute mononuclears (cells mm^3)	5-10	M	11	3279,18	866,03	261,11	1880,00	4420,00	2540,00	
		F	13	2980,69	740,47	205,37	1786,00	4004,00	2218,00	0,3391
	10-16	M	10	2573,60	617,43	195,25	1440,00	3520,00	2080,00	
		F	8	2483,00	547,00	193,50	2077,00	3795,00	1718,00	0,6569
Absolute lymphocytes (cells/mm^3)	5-10	M	11	3014,73	788,21	237,66	1786,00	4160,00	2374,00	
		F	13	2713,08	636,98	176,67	1710,00	3741,00	2031,00	0,3391
	10-16	M	10	2323,10	707,36	223,69	1243,00	3432,00	2189,00	
		F	8	2334,37	472,34	167,00	1943,00	3450,00	1507,00	0,9292
T cells (E-rosette) (cells/mm^3)	5-10	M	11	1171,54	492,65	148,54	921,00	2416,00	1495,00	
		F	13	1657,46	434,21	120,43	1072,00	2762,00	1690,00	0,4689
	10-16	M	10	1419,40	305,22	96,52	749,00	1901,00	1152,00	
		F	8	1536,00	177,53	62,77	1224,00	1746,00	522,00	0,2478
B Cells (cells/mm^3)	5-10	M	11	508,82	443,59	113,75	132,00	1591,00	1459,00	
		F	13	476,54	247,86	68,74	111,00	920,00	809,00	0,7943
	10-16	M	10	344,20	111,23	35,17	184,00	523,00	339,00	
		F	8	311,87	155,11	54,84	68,00	508,00	440,00	0,7557
Null cells (cells/mm^3)	5-10	M	11	915,18	380,59	114,75	529,00	1610,00	1081,00	
		F	13	873,15	553,00	153,38	196,00	1959,00	1763,00	0,5820
	10-16	M	10	804,40	372,66	117,84	298,00	1232,00	934,00	
		F	8	632,25	541,06	191,29	257,00	1935,00	1678,00	0,2135
FT cells (cells/mm^3)	5-10	M	11	10,82	27,23	8,21	0	88,00	88,00	
		F	13	4,31	15,53	4,31	0	56,00	56,00	0,4497
	10-16	M	10	5,60	12,26	3,88	0	35,00	35,00	
		F	8	2,62	6,42	2,62	0	21,00	21,00	0,6318
OKT3+ (cells/mm^3)	5-10	M	11	1877,73	477,76	144,05	1278,00	2710,00	1432,00	
		F	12	1811,25	162,9	564,63	1054,00	2923,00	1869,00	0,7119
	10-16	M	9	1538,55	440,36	146,79	691,00	2112,00	1421,00	
		F	8	1292,12	447,01	158,04	900,00	2315,00	1415,00	0,1019

	Age	Sex	Freq.	Mean	SD	SEM	Minimum	Maximum	Range	p value
OKT4+cells (cells/mm^3)	5-10	M	11	1086,64	326,37	98,40	659,00	1846,00	1187,00	
		F	12	1103,83	395,48	114,17	581,00	1882,00	1301,00	0,8535
	10-14	M	9	931,55	304,27	101,42	618,00	1514,00	896,00	
		F	8	913,63	288,44	101,98	680,00	1556,00	876,00	0,9233
OKT8+ cells (cells/mm^3)	5-10	M	11	818,91	372,13	112,20	348,00	1646,00	1298,00	
		F	12	740,33	352,33	101,71	273,00	1402,00	1129,00	0,7119
	10-16	M	9	721,55	234,04	78,01	360,00	1017,00	657,00	
		F	8	532,87	147,84	52,06	383,00	736,00	353,00	0,1019
% OKT4 of OKT3	5-10	M	11	58,91	13,46	4,06	31,00	78,00	47,00	
		F	12	60,41	8,61	2,48	46,00	69,00	23,00	0,5994
	10-16	M	9	61,78	13,55	4,52	49,00	90,00	41,00	0,0742
		F	8	71,62	10,45	3,69	58,00	93,00	35,00	0,0742
% OKT8 of OKT3	5-10	M	11	42,91	14,65	4,42	26,00	78,00	52,00	
		F	12	39,75	10,97	3,17	20,00	57,00	37,00	0,8535
	10-16	M	9	47,11	8,40	2,80	33,00	62,00	29,00	
		F	8	43,25	13,20	4,67	26,00	67,00	41,00	0,3844
Ratio OKT4/OKT8	5-10	M	11	1,53	0,59	0,18	0,40	2,65	2,25	
		F	12	1,62	0,41	0,12	1,07	2,43	1,36	0,7119
	10-16	M	9	1,35	0,36	0,12	0,79	1,79	1,00	
		F	8	1,79	0,56	0,20	0,97	2,56	1,59	0,0833
% suppressor cell function (ConA 5 µg/25µg/ml)	5-10	M	10	43,40	26,77	8,46	9,00	82,00	73,00	
		F	10	47,50	32,79	10,37	-1,00	90,00	91,00	0,8205
	10-16	M	8	37,25	39,57	13,99	-11,00	89,00	100,00	
		F	6	33,00	46,76	19,09	-13,00	95,00	108,00	0,7960
% suppressor cell function (ConA 30 µg/25µl/ml)	5-10	M	11	32,00	24,19	7,29	-6,00	80,00	86,00	
		F	11	57,18	35,27	10,63	-4,00	91,00	95,00	0,0815
	10-16	M	10	49,00	28,16	8,91	15,00	89,00	74,00	
		F	7	29,57	39,13	14,79	-8,00	86,00	96,00	0,1430

	Age	Sex	Freq.	Mean	SD	SEM	Minimum	Maximum	Range	p value
MNC PWM (dpm)	5-10	M	11	6052,21	3102,46	935,43	1418,06	11279,16	9861,10	
		F	9	6505,14	3231,58	1077,19	1853,94	10825,94	8972,00	0,7324
	10-16	M	8	7904,53	4104,85	1451,28	1647,57	14010,42	12372,85	
		F	6	10112,41	3349,95	1267,61	5382,70	14697,71	9315,01	0,3017
WCC ($\times 10^9$/L)	5-10	M	11	8,10	3,09	0,93	4,70	14,70	10,00	
		F	13	7,06	1,98	0,55	3,70	9,80	6,10	0,8619
	10-16	M	10	5,70	1,50	0,47	3,60	8,80	5,20	
		F	8	5,82	1,04	0,37	4,10	6,90	2,80	0,7212
% Neutrophils	5-10	M	11	51,27	8,59	2,59	41,00	66,00	25,00	
		F	12	51,00	8,66	2,50	34,00	65,00	31,00	0,8529
	10,16	M	10	49,30	8,58	2,71	35,00	62,00	27,00	
		F	8	54,37	12,20	4,31	36,00	68,00	32,00	0,3740
% Lymphocytes	5-10	M	11	39,09	9,29	2,80	24,00	53,00	29,00	
		F	13	39,77	7,91	2,19	28,00	55,00	27,00	0,8617
	10-16	M	10	42,70	7,62	2,41	33,00	54,00	21,00	
		F	8	41,12	9,86	3,49	29,00	56,00	27,00	0,6564
% Monocytes	5-10	M	11	3,18	1,25	0,38	1,00	5,00	4,00	
		F	12	4,00	3,22	0,93	1,00	11,00	10,00	0,9748
	10-16	M	10	3,00	1,83	0,58	1,00	7,00	6,00	
		F	8	2,50	1,07	0,38	2,00	5,00	3,00	0,4801
% Eosinophils	5-10	M	11	6,18	8,14	2,46	0	22,00	22,00	
		F	12	4,50	5,40	1,56	0	16,00	16,00	0,7553
	10-16	M	10	4,90	4,53	1,43	0	12,00	12,00	
		F	8	2,00	2,98	1,05	0	9,00	9,00	0,2061
% Basophils	5-10	M	11	0,27	0,65	0,19	0	2,00	2,00	
		F	12	0,25	0,62	0,18	0	2,00	2,00	0,9257
	10-16	M	10	0,10	0,32	0,10	0	1,00	1,00	
		F	8	0	0	0	0	0	0	0,3711

	Age	Sex	Freq.	Mean	SD	SEM	Minimum	Maximum	Range	p value
% Atypical Lymphocytes	5-10	M	11	0	0	0	0	0	0	
		F	12	0	0	0	0	0	0	
	10-16	M	10	0	0	0	0	0	0	
		F	8	0	0	0	0	0	0	
% Others	5-10	M	11	0	0	0	0	0	0	
		F	8	0	0	0	0	0	0	
	10-16	M	10	0	0	0	0	0	0	
RBC ($\times 10^{12}$/L)	5-10	M	11	4,46	0,33	0,10	3,63	4,92	1,29	
		F	13	4,35	0,36	0,10	3,42	4,96	1,54	0,4338
	10-16	M	10	4,68	0,27	0,85	4,15	5,14	0,99	
		F	8	4,51	0,29	0,10	4,07	5,02	0,95	0,1424
Hb (g/dL)	5-10	M	11	12,09	0,74	0,22	10,20	12,90	2,70	
		F	13	11,60	0,94	0,26	9,90	13,40	3,50	0,1107
	10-16	M	10	12,83	0,76	0,24	11,50	13,70	2,20	
		F	8	12,59	0,79	0,28	11,70	13,90	2,20	0,5041
HCT (L/L)	5-10	M	11	0,38	0,02	0,01	0,32	0,40	0,08	
		F	13	0,36	0,02	0,01	0,32	0,42	0,10	0,0487
	10-16	M	10	0,40	0,02	0,01	0,36	0,43	0,07	
		F	8	0,39	0,02	0,01	0,36	0,43	0,07	0,2665
MCV (pL)	5-10	M	11	84,83	2,68	0,81	81,50	89,10	7,20	
		F	13	84,06	7,15	1,98	68,80	99,50	30,70	0,8167
	10-16	M	10	85,49	2,36	0,74	80,90	89,10	8,20	
		F	8	86,65	3,80	1,34	80,20	91,50	11,30	0,5049
MCH (pg)	5.10	M	11	27,19	0,85	0,26	25,80	28,20	2,40	
		F	13	26,78	2,47	0,68	21,50	31,50	10,00	0,8164
	10-16	M	10	27,43	1,26	0,40	25,90	29,50	3,60	
		F	8	27,97	1,48	0,52	25,90	29,80	3,90	0,5034

	Age	Sex	Freq.	Mean	SD	SEM	Minimum	Maximum	Range	p value
MCHC (g/dL)	5-10	M	11	32,03	0,59	0,18	30,90	33,10	2,20	
		F	13	31,83	0,76	0,21	30,50	33,20	2,70	0,4329
	10-16	M	10	32,04	1,06	0,33	29,90	33,80	3,90	
		F	8	32,24	0,58	0,21	31,10	33,20	2,10	0,4208
Height (cms)	5-10	M	11	116,91	8,20	2,47	104,00	130,00	26,00	
		F	13	117,15	10,88	3,02	97,00	133,00	36,00	0,7935
	10-16	M	10	135,30	7,69	2,43	124,00	148,00	24,00	
		F	8	133,37	10,18	3,60	121,00	149,00	28,00	0,6247
Weight (Kg)	5-10	M	11	20,91	3,43	1,03	17,00	29,00	12,00	
		F	13	21,35	4,88	1,35	12,50	28,50	16,00	0,6632
		M	10	29,80	6,43	2,03	21,00	41,50	20,50	
		F	8	28,19	7,60	2,69	20,00	44,00	24,00	0,4744
Age (years)	5-10	M	11	8,24	1,30	0,39	6,40	9,90	3,50	
		F	13	8,17	1,32	0,36	6,10	9,80	3,70	0,9537
	10-16	M	10	12,52	1,49	0,47	10,90	15,90	5,00	
		F	8	12,79	1,82	0,64	10,40	15,60	5,20	0,7219

Table 4 Descriptive statistics in immunological, haematological and nutritional parameters between African, Indian and White Adults

	Population	Sample size	Mean	Standard Deviation	SEM	Minimum	Maximum
Immunological:							
Absolute mononuclears (cells/mm^3)	African	11	2619,09	745,92	224,90	1551,00	4032,00
	Indian	12	2591,92	772,89	223,11	1224,00	3770,00
	White	14	2253,53	802,87	207,30	1344,00	4736,00
Absolute lymphocytes (cells/mm^3)	African	11	2265,91	608,11	183,35	1504,00	3384,00
	Indian	12	2360,83	768,34	221,80	1173,00	3654,00
	White	15	2020,67	799,06	206,32	874,00	4440,00
T cell (E-rosette) (cells/mm^3)	African	11	1864,00	629,90	189,92	924,00	2775,00
	Indian	9	1992,11	765,41	255,14	930,00	3038,00
	White	14	1729,36	750,78	200,65	383,00	3789,00
B cells (SIg) (cells/mm^3)	African	11	126,82	150,92	45,51	16,00	541,00
	Indian	9	374,33	262,89	87,63	52,00	841,00
	White	13	385,93	192,08	51,33	84,00	852,00

	Population	Sample size	Mean	Standard Deviation	SEM	Minimum	Maximum
Null cells (cells/mm^3)	African	11	628,36	418,45	126,17	68,00	1292,00
	Indian	9	374,11	416,17	138,72	24,00	1269,00
	White	14	160,00	212,76	56,86	41,00	862,00
FT cells (cells/mm^3)	African	11	0,00	0,00	0,00	0,00	0,00
	Indian	9	0,00	0,00	0,00	0,00	0,00
	White	14	2,57	9,62	2,57	0,00	36,00
T cells (OKT3MoAb) (cells/mm^3)	African	11	1604,82	625,82	188,69	605,00	2426,00
	Indian	12	1422,42	507,74	146,57	220,00	2005,00
	White	15	1063,27	545,88	140,97	308,00	2273,00
OKT4+ cells (cells/mm^3)	African	11	1053,27	512,99	154,67	450,00	1760,00
	Indian	12	913,50	361,55	104,37	306,00	1395,00
	White	15	795,66	393,57	101,62	342,00	1610,00
OKT8+ cells (cells/mm^3)	African	11	717,18	237,30	71,55	248,00	984,00
	Indian	12	551,50	269,88	77,91	98,00	956,00
	White	15	321,67	156,76	40,47	54,00	616,00

	Population	Sample size	Mean	Standard Deviation	SEM	Minimum	Maximum
% OKT4 of OKT3	African	11	64,55	14,75	4,5	43,00	93,00
	Indian	12	61,25	16,03	4,63	39,00	94,00
	White	15	61,67	19,74	5,10	7,00	85,00
% OKT8 of OKT3	African	11	146,36	11,74	3,54	34,00	69,00
	Indian	12	39,67	14,74	4,25	18,00	66,00
	White	15	33,07	17,06	4,41	9,00	75,00
Ratio OKT4/OKT8	African	11	1,46	0,46	0,14	0,88	2,29
	Indian	12	1,94	0,89	0,26	0,90	3,94
	White	15	3,03	1,90	0,49	1,00	7,56
% suppressor cell function (ConA 5 µg/25µl/ml of cells)	African	7	30,43	20,17	7,62	6,00	59,00
	Indian	8	53,75	31,42	11,11	10,00	93,00
	White	13	62,69	26,66	7,40	27,00	100,00
% suppressor cell function (ConA 30 µg/25µl/ml of cells)	African	6	39,83	26,17	10,68	13,00	73,00
	Indian	7	68,86	27,41	10,36	20,00	94,00
	White	12	61,50	29,06	8,39	-9,00	99,00

	Population	Sample size	Mean	Standard Deviation	SEM	Minimum	Maximum
MNC PWM stimulation (dpm)	African	2	9058,77	5623,39	3976,34	5082,44	13035,11
	Indian	10	4080,86	3595,15	1136,25	389,10	10479,11
	White	8	2287,19	2321,79	820,88	257,90	6194,43
WCC ($\times 10^9$/L)	African	11	5,80	1,34	0,41	4,30	7,50
	Indian	12	6,14	0,58	0,17	5,10	6,90
	White	15	5,79	1,47	0,38	3,80	8,00
% Neutrophils	African	11	52,27	10,25	3,09	36,00	69,00
	Indian	12	52,67	11,59	3,35	32,00	71,00
	White	15	56,60	8,95	2,31	36,00	67,00
% Lymphocytes	African	11	39,55	8,29	2,50	28,00	56,00
	Indian	12	39,50	11,29	3,26	23,00	63,00
	White	15	34,80	9,28	2,39	23,00	60,00
% Monocytes	African	11	5,91	3,51	1,06	1,00	13,00
	Indian	12	4,08	2,19	0,63	1,00	8,00
	White	15	5,13	3,38	0,87	1,00	13,00

	Population	Sample size	Mean	Standard Deviation	SEM	Minimum	Maximum
% Eosinophils	African	11	2,73	3,32	1,00	0,00	10,00
	Indian	12	3,17	3,51	1,01	0,00	13,00
	White	15	3,00	2,62	0,68	0,00	10,00
% Basophils	African	11	0,27	0,47	0,14	0,00	1,00
	Indian	12	0,17	0,58	0,17	0,00	2,00
	White	15	0,33	0,72	0,19	0,00	2,00
% Atypical Lymphocytes	African	11	0,18	o,60	0,18	0.00	2,00
	Indian	12	0,00	0,00	0,00	0,00	0,00
	White	15	0,00	0,00	0,00	0,00	0,00
% Others	African	11	0,00	0,00	0,00	0,00	0,00
	Indian	12	0,00	0,00	0,00	0,00	0,00
	White	15	0,00	0,00	0,00	0,00	0,00
Haematological:							
RBC ($\times 10^{12}$/L)	African	10	4,76	0,39	0,12	4,23	5,44
	Indian	12	5,20	0,57	0,17	4,35	6,14
	White	14	4,55	0,40	0,11	3,73	5,24

	Population	Sample size	Mean	Standard Deviation	SEM	Minimum	Maximum
Hb (g/dl)	African	11	14,32	1,08	0,33	12,70	16,20
	Indian	12	15,17	0,97	0,28	14,00	17,30
	White	15	14,21	1,39	0,36	11,80	16,50
HCT (L/L)	African	9	0,43	0,03	0,01	0,37	0,48
	Indian	12	0,46	0,03	0,01	0,40	0,53
	White	14	0,43	0,04	0,01	0,37	0,49
MCV (fl)	African	10	91,90	4,95	1,57	82,60	98,60
	Indian	12	85,93	4,46	1,29	80,00	95,00
	White	14	91,14	4,46	1,19	84,00	98,10
MCH (pg)	African	9	30,49	1,81	0,60	27,80	32,30
	Indian	12	29,31	2,81	0,81	24,00	33,00
	White	14	31,06	1,88	0,50	28,00	34,00
MCHC (g/dl)	African	9	33,28	0,84	0,28	31,60	34,20
	Indian	6	32,80	0,81	0,33	31,70	
	White	3	32,97	1,45	0,84	31,30	33,90

Nutritional:	Population	Sample size	Mean	Standard Deviation	SEM	Minimum	Maximum
Height (cms)	African	ND					
	Indian	ND					
	White	ND					
Weight (Kg)	African	ND					
	Indian	ND					
	White	ND					
Age (months)	African	10	355,20	126,77	40,09	108,00	516,00
	Indian	11	372,00	132,54	39,96	216,00	684,00
	White	15	509,60	170,24	43,96	288,00	756,00

ND = not done

Table 5 Comparison of immunological, haematological, biochemical, serum immunoglobulins, C3 levels, and nutritional parameters between endemic controls and infected patients with S.mansoni or S.haematobium and combined infections

		Gp.1	Gp.2	Gp.3	Gp.4	Gp.5									
		-ve S.Mansoni -ve S.Haem.	-ve S.Mansoni +ve S.Haem.	+ve S.Mansoni -ve S.Haem.	Livers ≤2 +ve S.Mansoni +ve S.Haem.	Livers > 2 +ve S.Mansoni -ve or +ve S.Haem.	Control vs Gp2	Control vs Gp3	Control vs Gp4	Control vs Gp5	Gp2 vs Gp3	Gp2 vs Gp4	Gp2 vs Gp5	Gp3 vs Gp4	Gp3 vs Gp5	Gp4 vs Gp5
Absolute mononuclears cells/mm^3	Number	24	11	15	34	19									
	Mean	3392	4224	3046	4102	3738	0,2410	0,2253	0,0265$^+$	0,9415	0,0734	0,8637	0,5047	0,0045$^+$	0,4150	0,2352
	SD	840,47	1904,3	934,78	1364,33	2062,14									
	SEM	171,56	574,17	241,36	233,98	473,09									
	Min	2067	1800	1782	897	836									
	Max	5040	8229	5250	7344	8624									
	Range	2973	6429	3468	6447	7788									
Absolute Lymphocytes cells/mm^3	Number	24	11	15	34	19									
	Mean	2977	3609	2711	3672,59	3263,32	0,2136	0,3408	0,0344$^+$	0,9512	0,0821	0,8741	0,4259	0,0098$^+$	0,5439	0,1614
	SD	866,98	1452,55	845,18	1348,69	1883,53									
	SEM	176,97	437,96	218,22	231,30	432,11									
	Min	1443	1425	1620	780	704									
	Max	4968	6327	4830	6885	7840									
	Range	3525	4902	3210	6105	7136									
T cell (E-rosette) cells/mm^3	Number	24	8	15	31	9									
	Mean	2665	3110	2267	3372	2678	0,5716	0,1659	0,0139$^+$	0,2411	0,1213	0,4441	0,2898	0,0013*	0,5312	0,0625
	SD	828,94	1426,1	761,15	1130,22	2223,03									
	SEM	169,21	504,20	196,53	202,99	741,01									
	Min	1211	1170	1172	1175	929									
	Max	4536	5959	3833	6462	7417									
	Range	3325	4789	2661	4687	6488									
B cells (SIg) cells/mm^3	Number	24	8	15	31	9									
	Mean	423	407	384	493	472	0,9673	0,9540	0,1515	0,8083	0,5613	0,4441	0,9233	0,1974	0,9287	0,4465
	SD	258,27	247,06	278,19	267,69	475,34									
	SEM	52,72	87,35	71,83	48,08	158,45									
	Min	145	72	42	76	0									
	Max	1151	701	1155	1211	1351									
	Range	1006	629	1113	1135	1351									

		Gp.1	Gp.2	Gp.3	Gp.4	Gp.5									
		-ve S.Mansoni -ve S.Haem.	-ve S.Mansoni +ve S.Haem.	+ve S.Mansoni -ve S.Haem.	Livers ≤2 +ve S.Mansoni +ve S.Haem.	Livers > 2 +ve S.Mansoni -ve or +ve Haem.	Control vs Gp2	Control vs Gp3	Control vs Gp4	Control vs Gp5	Gp2 vs Gp3	Gp2 vs Gp4	Gp2 vs Gp5	Gp3 vs Gp4	Gp3 vs Gp5	Gp4 vs Gp5
Null cells cells/mm^3	Number	24	8	15	31	9									
	Mean	304	362	388	346	1504	0,8618	0,3408	0,8122	0,0001*	0,3662	0,6764	0,0039*	0,3546	0,0005*	0,0001*
	SD	257,85	439	276,08	362,36	825										
	SEM	52,64	155	71,28	65,08	275,04										
	Min	47	26	54	31	610										
	Max	924	1332	922	1473	2848										
	Range	877	1306	868	1442	2238										
FT cells cells/mm^3	Number	24	8	15	31	9									
	Mean	5	15	1	6	8	0,4821	0,3204	0,5383	0,6307	0,1859	0,2812	0,8461	0,6837	0,2329	0,4138
	SD	12,85	31,96	5,42	20,86	15,23										
	SEM	2,63	11,30	1,4	3,75	5,08										
	Min	0	0	0	0	0										
	Max	46	89	21	96	36										
	Range	46	89	21	96	36										
T cells (OKT3) cells/mm^3	Number	24	9	15	31	10									
	Mean	1812	1782	1604	2212	2157	0,7617	0,2482	0,0790	0,8206	0,3252	0,2502	1,0000	0,0252+	0,6176	0,3466
	SD	537,47	737,12	741,03	806,01	1567,23										
	SEM	109,71	245,71	191,33	144,76	495,60										
	Min	690	450	285	911	481										
	Max	2734	2738	3360	3887	5088										
	Range	2044	2288	3075	2976	4607										
OKT4 cells/mm^3	Number	24	9	15	31	10									
	Mean	954	762	663	817	750	0,1822	0,0186+	0,1309	0,2730	0,5312	0,9355	0,9674	0,4894	0,7815	0,9758
	SD	296,46	325,13	364,33	567,52	496,45										
	SEM	60,52	108,38	94,07	101,93	156,99										
	Min	476	216	18	0	131										
	Max	1471	1243	1202	2264	1464										
	Range	995	1027	1184	2264	1333										

		Gp.1	Gp.2	Gp.3	Gp.4	Gp.5										
		-ve S.Mansoni -ve S.Haem.	-ve S.Mansoni +ve S.Haem.	+ve S.Mansoni -ve S.Haem.	Livers ≤2 +ve S.Mansoni +ve S.Haem.	Livers > 2 +ve S.Mansoni -ve or +ve S.Haem.	Control vs Gp2	Control vs Gp3	Control vs Gp4	Control vs Gp5	Gp2 vs Gp3	Gp2 vs Gp4	Gp2 vs Gp5	Gp3 vs Gp4	Gp3 vs Gp5	Gp4 vs Gp5
OKT8 cells/mm^3	Number	24	9	15	31	10										
	Mean	968	951	943	1217	1421	0,5992	0,9770	0,1640	0,7337	0,9762	0,5490	1,0000	0,1704	0,7393	0,6708
	SD	445,18	391,23	419	683,89	1423,12										
	SEM	90,87	130,41	108	122,83	450,03										
	Min	394	216	249	538	321										
	Max	2167	1531	1729	3616	5088										
	Range	1773	1315	1480	3078	4767										
% OKT4 of T3	Number	24	9	15	31	10										
	Mean	55	45	41	38	38	0,1819	0,0566	0,0081+	0,0101+	0,6981	0,3472	0,1649	0,5816	0,5056	0,8434
	SD	17,30	12,5	19,50	23,48	23,27										
	SEM	3,53	4,17	5,03	4,23	7,36										
	Min	27	21	6	0	12										
	Max	96	61	69	99	91										
	Range	69	40	63	99	79										
% T8 of T3	Number	24	9	15	31	10										
	Mean	53	54	61	56	62	0,5846	0,0643	0,5752	0,1979	0,3237	0,8968	0,4365	0,1560	0,8459	0,3385
	SD	16,75	5,81	14,75	19,81	19,40										
	SEM	3,42	1,94	3,81	3,56	6,14										
	Min	33	41	45	29	33										
	Max	107	61	90	121	100										
	Range	74	20	45	92	67										
T4/T8 Ratio	Number	24	9	15	31	10										
	Mean	1,09	0,85	0,76	0,79	0,70	0,2929	0,0734	0,0339+	0,0222+	0,6756	0,4756	0,2186	0,9160	0,6772	0,6057
	SD	0,50	0,26	0,56	0,55	0,52										
	SEM	0,10	0,09	0,14	0,10	0,17										
	Min	0,06	0,37	0,05	0	0,2										
	Max	2,50	1,22	1,92	2,07	1,88										
	Range	2,44	0,85	1,87	2,07	1,68										

		Gp.1	Gp.2	Gp.3	Gp.4	Gp.5										
		-ve S.Mansoni -ve S.Haem.	-ve S.Mansoni +ve S.Haem.	+ve S.Mansoni -ve S.Haem.	Livers ≤2 +ve S.Mansoni +ve S.Haem.	Livers > 2 +ve S.Mansoni -ve or +ve S.Haem	Control vs Gp2	Control vs Gp3	Control vs Gp4	Control vs Gp5	Gp2 vs Gp3	Gp2 vs Gp4	Gp2 vs Gp5	Gp3 vs Gp4	Gp3 vs Gp5	Gp4 vs Gp5
WCC x 10^9/L	Number	24	11	16	34	19										
	Mean	6,87	14,73	7,69	8,74	9,40	0,0077+	0,3336	0,0244+	0,0285+	0,1744	0,3283	0,6205	0,3438	0,1851	0,4580
	SD	2,06	16,49	2,24	2,99	4,50										
	SEM	0,42	4,97	0,56	0,51	1,03										
	Min	3,9	5,8	3,6	3,9	2,2										
	Max	10,6	63,3	12,7	15,5	19,6										
	Range	6,7	57,5	9,1	11,6	17,4										
% Neutrophils	Number	24	11	15	34	19	0,4030	0,1984	0,0680	0,0125+	0,1192	0,7312	0,0159+	0,0129+	0,2050	0,0008*
	Mean	41	36	45	36	50										
	SD	10,49	14,38	10,17	13,82	13,32										
	SEM	2,14	4,34	2,62	2,37	3,06										
	Min	22	6	25	15	14										
	Max	64	58	59	70	68										
	Range	42	52	34	55	54										
% Lymphocytes	Number	24	11	15	34	19	0,0218+	0,0754	0,5642	0,0025*	0,3492	0,0312+	0,9142	0,2119	0,2976	0,0096+
	Mean	45	34	38	43	34										
	SD	10,97	12,77	10,67	12,71	9,91										
	SEM	2,24	3,85	2,75	2,18	2,27										
	Min	23	9	20	20	18										
	Max	69	57	54	70	50										
	Range	46	48	34	50	32										
% Monocytes	Number	24	11	15	34	19	0,1070	0,1415	0,0257+	0,1332	0,8740	0,7275	0,7770	0,5010	0,9303	0,5550
	Mean	6	4,27	4,6	4	4,89										
	SD	3,51	2,15	2,16	2,25	3,28										
	SEM	0,72	0,65	0,56	0,39	0,75										
	Min	1	1	2	1	1										
	Max	16	8	10	10	13										
	Range	15	7	8	9	12										

		Gp.1	Gp.2	Gp.3	Gp.4	Gp.5										
		-ve S.Mansoni -ve S.Haem.	-ve S.Mansoni +ve S.Haem.	+ve S.Mansoni -ve S.Haem	Livers ≤2 +ve S.Mansoni +ve S.Haem.	Livers > 2 +ve S.Mansoni -ve or +ve Hae	Control vs Gp2	Control vs Gp3	Control vs Gp4	Control vs Gp5	Gp2 vs Gp3	Gp2 vs Gp4	Gp2 vs Gp5	Gp3 vs Gp4	Gp3 vs Gp5	Gp4 vs Gp5
% Eosinophils	Number	24	11	15	34	19										
	Mean	8	25	12	16	11	0,0001*	0,0290+	0,0003*	0,4473	0,0101+	0,0809	0,0032*	0,2586	0,2894	0,0223+
	SD	6,06	19,31	6,95	8,59	9,71										
	SEM	1,24	5,82	1,80	1,47	2,23										
	Min	0	10	1	0	1										
	Max	23	80	26	33	36										
	Range	23	70	25	33	25										
% Basophils	Number	24	10	15	34	18										
	Mean	0,17	0,1	0,2	1,59	0,39	0,8130	0,9208	0,8266	0,1218	0,7681	0,6893	0,1717	0,9289	0,2269	0,1295
	SD	0,48	0,32	0,56	8,56	0,61										
	SEM	0,09	0,1	0,14	1,47	0,14										
	Min	0	0	0	0	0										
	Max	2	1	2	50	2										
	Range	2	1	2	50	2										
Serum IgA mg/dL	Number	24	9	15	31	6										
	Mean	227,33	190,11	250,07	240,68	301,83	0,3738	0,2663	0,9932	0,8560	0,0490+	0,3392	0,5557	0,2412	0,5080	0,9836
	SD	98,41	68,24	61,75	134,96	215,28										
	SEM	20,09	22,74	15,95	24,24	87,89										
	Min	83	72	150	73	117										
	Max	515	306	378	837	655										
	Range	432	234	228	764	538										
Serum IgM mg/dL	Number	24	9	15	31	6										
	Mean	246,71	379,78	262,67	281,87	339,33	0,2411	0,6545	0,2555	0,2230	0,3870	0,5171	0,9530	0,4820	0,3502	0,6065
	SD	95,16	263,16	106,12	129,52	175,06										
	SEM	19,42	87,72	27,40	23,26	71,47										
	Min	113	105	123	55	180										
	Max	476	912	563	670	637										
	Range	363	807	440	615	457										

		Gp.1	Gp.2	Gp.3	Gp.4	Gp.5										
		-ve S.Mansoni -ve S.Haem.	-ve S.Mansoni +ve S.Haem.	+ve S.Mansoni -ve S.Haem.	Livers ≤2 +ve S.Mansoni +ve S.Haem.	Livers > 2 +ve S.Mansoni -ve or +ve S.Haem	Control vs Gp2	Control vs Gp3	Control vs Gp4	Control vs Gp5	Gp2 vs Gp3	Gp2 vs Gp4	Gp2 vs Gp5	Gp3 vs Gp4	Gp3 vs Gp5	Gp4 vs Gp5
Serum	Number	24	9	15	31	6										
IgG	Mean	1950,92	2476,67	2317	2273,71	3002,83	0,0262+	0,1703	0,1692	0,0022*	0,3252	0,2124	0,1255	0,7164	0,0516	0,0210
mg/dL	SD	442,10	588,66	726,87	826,67	732,22										
	SEM	90,24	196,22	187,68	148,47	298,93										
	Min	1005	1886	1431	1355	1828										
	Max	2610	3831	4242	5891	3899										
	Range	1605	1945	2811	4536	2071										
Complement	Number	13	8	7	26	5										
C3	Mean	101,54	101,63	105	107,5	86,8	0,1796	0,6915	0,2512	0,2999	0,2976	0,7300	0,3055	0,5088	0,1931	0,1793
mg/dL	SD	14,08	44,95	14,31	43,32	30,87										
	SEM	3,91	15,89	5,41	8,50	13,80										
	Min	76	20	90	5	45										
	Max	129	146	134	232	126										
	Range	53	126	44	227	81										
Haematological:																
RBC	Number	24	8	16	34	19	0,6633	0,7508	0,5697	0,0028*	0,8065	0,5750	0,1301	0,7789	0,0081+	0,0003*
x 10^{12}/L	Mean	4,4	3,86	4,19	4,32	3,38										
	SD	0,45	1,46	1,05	0,74	1,26										
	SEM	0,09	0,51	0,26	0,13	0,29										
	Min	3,86	0,46	0,43	0,53	0,45										
	Max	5,36	5,01	5,13	5,08	4,7										
	Range	1,50	4,55	4,7	4,55	4,25										
Hb	Number	24	11	16	34	19										
g/dL	Mean	12,09	11,51	12,33	12,15	9	0,3930	0,4298	0,6927	0,0001*	0,2771	0,2669	0,0014*	0,5322	0,0001*	0,0001*
	SD	0,89	1,92	0,98	0,93	1,80										
	SEM	0,18	0,58	0,24	0,16	0,41										
	Min	10,6	8	10,6	9,8	4,1										
	Max	14,5	15	14,1	13,9	11,7										
	Range	3,9	7	3,5	4,1	7,6										

		Gp.1	Gp.2	Gp.3	Gp.4	Gp.5										
		-ve S.Mansoni -ve S.Haem.	-ve S.Mansoni +ve S.Haem.	+ve S.Mansoni -ve S.Haem.	Livers ≤2 +ve S.Mansoni +ve S.Haem.	Livers > 2 +ve S.Mansoni -ve or +ve S.Haem	Control vs Gp2	Control vs Gp3	Control vs Gp4	Control vs Gp5	Gp2 vs Gp3	Gp2 vs Gp4	Gp2 vs Gp5	Gp3 vs Gp4	Gp3 vs Gp5	Gp4 vs Gp5
MCV fL	Number	24	10	16	34	18										
	Mean	451.25	314	468,88	254,06	459,61	0,6774	0,8144	0,4482	0,1473	0,4290	0,6337	0,6835	0,1898	0,0908	0,9310
	SD	407,13	361,39	393,82	328,53	352,83										
	SEM	83,12	114,28	98,45	56,34	83,16										
	Min	72	79	80	79	64										
	Max	971	873	905	944	829										
	Range	899		825	865	765										
Platelets x 10^9/L	Number	11	5	9	9	18										
	Mean	402	454	461	355	392	0,3356	0,4940	0,7324	0,5292	0,8415	0,2571	0,4123	0,3099	0,2368	0,9181
	SD	87,74	124,82	177,40	165,72	182,37										
	SEM	26,45	55,82	59,13	55,24	42,99										
	Min	271	256	287	0	89										
	Max	588	556	870	554	767										
	Range	317	300	583	554	678										
Biochemical (Albumin) g/L	Number	24	10	16	34	18										
	Mean	45,21	39,3	42,19	39,82	29,33	0,0532	0,2079	0,0038*	0,0001*	0,2552	0,8994	0,0023*	0,3477	0,0001*	0,0001*
	SD	5,56	6,04	6,33	6,98	8,16										
	SEM	1,14	1,91	1,58	1,20	1,92										
	Min	38	28	33	11	15										
	Max	60	45	52	50	42										
	Range	22	17	19	39	27										
AST u/L	Number	24	11	16	33	17										
	Mean	20	28,36	22,06	19,81	44,65	0,4537	0,5512	0,8713	0,0002*	0,6024	0,2114	0,0184+	0,5357	0,0023*	0,0001*
	SD	6,95	23,94	7,47	7,05	24,56										
	SEM	1,42	7,22	1,87	1,22	5,96										
	Min	6	12	12	8	12										
	Max	33	99	40	33	99										
	Range	27	87	28	25	87										

		Gp.1	Gp.2	Gp.3	Gp.4	Gp.5										
		-ve S.Mansoni -ve S.Haem.	-ve S.Mansoni +ve S.Haem.	+ve S.Mansoni -ve S.Haem.	Livers ≤2 +ve S.Mansoni +ve S.Haem.	Livers > 2 +ve S.Mansoni -ve or +ve Haem	Control vs Gp2	Control vs Gp3	Control vs Gp4	Control vs Gp5	Gp2 vs Gp3	Gp2 vs Gp4	Gp2 vs Gp5	Gp3 vs Gp4	Gp3 vs Gp5	Gp4 vs Gp5
Plasma Zinc um/L	Number	13	5	7	24	Missing value										
	Mean	0,88	0,92	1,12	0,90		0,8823	0,4507	0,6673	-	0,4160	0,4020	-	0,9059	-	-
	SD	0,25	0,19	1,09	0,38											
	SEM	0,07	0,08	0,41	0,08											
	Min	0,43	0,73	0,48	0,38											
	Max	1,29	1,12	3,57	2,11											
	Range	0,86	0,39	3,09	1,73											
Total Globulins g/L	Number	13	8	8	26	9										
	Mean	27,31	35,38	32,75	33,35	46,89	0,0164+	0,0457+	0,0082+	0,0002*	0,8745	0,4273	0,0300+	0,6986	0,0206+	0,0009*
	SD	5,94	6,35	13,13	6,08	9,78										
	SEM	1,65	2,24	4,64	1,19	3,26										
	Min	15	27	4	20	34										
	Max	35	43	46	49	61										
	Range	20	16	42	29	27										
Plasma Creatinine mmol/L	Number	13	7	7	24	2										
	Mean	27	37,86	39,29	36,38	422,5	0,0880	0,0088+	0,0085+	0,0412+	0,5627	0,8311	0,1432	0,6689	0,1416	0,1111
	SD	11,49	11,36	4,75	11,39	538,11										
	SEM	3,19	4,30	1,80	2,32	380,5										
	Min	3	25	34	3	42										
	Max	46	58	46	54	803										
	Range	43	33	12	51	761										
Parasitology																
Egg count In urine	Number	22	6	14	24	Missing value										
	Mean	0	113	0	247		0,0001*	0,0001*	0,0001*	-	0,0010*	0,9164	-	0,0001*	-	-
	SD	0	144,33	0	626,68											
	SEM	0	58,92	0	127,92											
	Min	0	0	0	0											
	Max	0	360	0	2916											
	Range	0	360	0	2916											

		Gp.1	Gp.2	Gp.3	Gp.4	Gp.5										
		-ve S.Mansoni -ve S.Haem.	-ve S.Mansoni +ve S.Haem.	+ve S.Mansoni -ve S.Haem.	Livers ≤2 +ve S.Mansoni +ve S.Haem.	Livers > 2 +ve S.Mansoni -ve or +ve Haem	Control vs Gp2	Control vs Gp3	Control vs Gp4	Control vs Gp5	Gp2 vs Gp3	Gp2 vs Gp4	Gp2 vs Gp5	Gp3 vs Gp4	Gp3 vs Gp5	Gp4 vs Gp5
Egg count in stool before	Number	24	7	15	31	7										
	Mean	0	0	12,27	13,77	44,71	-	0,0001*	0,0001*	0,0001*	0,0070	0,0073	0,0250	0,3538	0,7179	0,5081
	SD	0	0	17,38	35,34	61,31										
	SEM	0	0	4,49	6,63	23,17										
	Min	0	0	0	0	0										
	Max	0	0	60	190	152										
	Range	0	0	60	190	152										
Repeat count in stool	Number	22	5	14	25	1										
	Mean	0	0	2,86	10,36	2	-	0,0089+	0,0080	0,0001*	0,1943	0,1890	0,0253	0,9265	0,4084	0,2892
	SD	0	0	5,17	26,19	-										
	SEM	0	0	1,38	5,24	-										
	Min	0	0	0	0	2										
	Max	0	0	16	110	2										
	Range	0	0	16	110	0										
Nutritional:																
Age	Number	24	11	16	34	19										
	Mean	10,17	8,73	10.19	10,44	7,89	0,2093	0,9666	0,8543	0,0279*	0,2326	0,0737	0,3620	0,5774	0,0280+	0,0003*
	SD	3,09	2,90	2,71	1,96	2,18										
	SEM	0,63	0,87	0,68	0,33	0,50										
	Min	6	4	7	7	4										
	Max	17	13	15	13	12										
	Range	11	9	8	6	8										
Height	Number	23	7	12	28	8										
	3-10%	11	6	4	14	6	0,2000	0,6380	0,9740	0,3650	0,0820	0,2250	0,6050	0,5030	0,1530	0,4100
	10-25	0	0	0	0	0										
	25-50	10	1	6	12	2										
	> 50	2	0	2	2	0										

Weight	Number	21	10	14	33	15										
	3-10	5	1	3	5	7										
	10-25	0	2	1	2	2	0,1740	0,5770	0,3890	0,0500*	0,6160	0,4170	0,1260	0,9440	0,3200	0,0590
	25-50	13	6	7	17	3										
	> 50	3	1	3	9	3										

* $p = \leq 0{,}0042$
\+ $> 0{,}0042 \quad p \leq 0{,}05$

Table 6 Comparison between light and heavy S.haematobium infections for all parameters

Group 4 A = Light SH Light SM B = Moderate - Heavy SH Light - Moderate SM

		Group 4 A	Group 4 B
Absolute mononuclears cells/mm^3	Number	23	11
	Mean	4008	4300
	SD	1305	1527
	SEM	272	460
	Min	897	2240
	Max	5890	7344
	Range	4993	5104
Absolute Lymphocytes cells/mm^3	Number	23	11
	Mean	3628	3768
	SD	1280	1543
	SEM	267	465
	Min	780	1840
	Max	5670	6885
	Range	4890	5045
T cell (E-rosette) cells/mm^3	Number	20	11
	Mean	3260	3579
	SD	1017	1339
	SEM	227	404
	Min	1775	1859
	Max	5132	6462
	Range	3357	4603
B cells (SIg) cells/mm^3	Number	20	11
	Mean	487	505
	SD	274	268
	SEM	61	81
	Min	76	203
	Max	1211	1013
	Range	1135	810

		Group 4 A	Group 4 B
Null cells cells/mm^3	Number	20	11
	Mean	371	299
	SD	402	289
	SEM	90	87
	Min	73	31
	Max	1473	1016
	Range	1400	985
FT cells cells/mm^3	Number	20	11
	Mean	10	0
	SD	26	0
	SEM	6	0
	Min	0	0
	Max	96	0
	Range	96	0
T cells (OKT3MoAB) cells/mm^3	Number	20	11
	Mean	2461	1761
	SD	830	539
	SEM	186	163
	Min	1235	911
	Max	3887	2541
	Range	2652	1630
T Helper/inducer cells cells/mm^3	Number	20	11
	Mean	984	513
	SD	567	445
	SEM	127	134
	Min	140	0
	Max	2264	1386
	Range	2124	1386

		Group 4 A	Group 4 B
T suppressor/ cytotoxic cells cells/mm^3	Number	20	11
	Mean	1318	1034
	SD	657	724
	SEM	147	218
	Min	618	538
	Max	3616	3011
	Range	2998	2473
% OKT4 of OKT3	Number	20	11
	Mean	43	29
	SD	22	24
	SEM	5	7
	Min	5	0
	Max	99	59
	Range	94	59
% OKT8 of OKT3	Number	20	11
	Mean	55	59
	SD	14	28
	SEM	3	8
	Min	32	29
	Max	94	121
	Range	62	92
Ratio OKT4/ OKT8	Number	20	11
	Mean	0,84	0,7
	SD	0,48	0,67
	SEM	0,11	0,20
	Min	0,07	0
	Max	2,07	1,88
	Range	2	1,88

		Group 4 A	Group 4 B
WCC	Number	23	11
x 10^9/L	Mean	8,35	9,56
	SD	2,68	3,56
	SEM	0,56	1,07
	Min	3,9	6
	Max	15,5	15,4
	Range	11,6	9,4
% Neutro-	Number	23	11
phils	Mean	35	39
	SD	14	12,79
	SEM	3	3,86
	Min	15	16
	Max	70	58
	Range	55	42
% Lympho-	Number	23	11
cytes	Mean	45	40
	SD	14	10
	SEM	3	3
	Min	20	23
	Max	70	57
	Range	50	34
% Monocytes	Number	23	11
	Mean	4	6
	SD	2	3
	SEM	0,34	0,78
	Min	1	3
	Max	8	10
	Range	7	7

		Group 4 A	Group 4 B
Eosinophils	Number	23	11
%	Mean	16	15
	SD	9	7
	SEM	2	2
	Min	0	6
	Max	33	30
	Range	33	24
Basophils	Number	23	11
%	Mean	2	0
	SD	10	0
	SEM	2	0
	Min	0	0
	Max	50	0
	Range	50	0
Serum	Number	20	11
IgA	Mean	255	215
mg/dL	SD	163	55
	SEM	36	17
	Min	73	146
	Max	837	329
	Range	764	183
IgM	Number	20	11
mg/dL	Mean	252	335
	SD	127	121
	SEM	28	37
	Min	55	233
	Max	510	670
	Range	455	437

		Group 4 A	Group 4 B
IgG	Number	20	11
mg/dL	Mean	2369	2100
	SD	998	323
	SEM	223	97
	Min	1355	1659
	Max	5891	2651
	Range	4536	992
Complement	Number	15	11
C3	Mean	93	128
Mg/dL	SD	42	38
	SEM	11	11
	Min	5	89
	Max	166	232
	Range	161	143
Rbc	Number	23	11
x 10^{12}/L	Mean	4,22	4,54
	SD	0,87	0,24
	SEM	0,18	0,07
	Min	0,53	4,16
	Max	5,08	4,86
	Range	4,55	0,7
Hb	Number	23	11
g/dL	Mean	12,3	11,81
	SD	0,98	0,77
	SEM	0,20	0,23
	Min	9,8	10,6
	Max	13,9	13,3
	Range	4,1	2,7

		Group 4 A	Group 4 B
MCV	Number	23	11
f/L	Mean	333	89,81
	SD	377	7,56
	SEM	76	2,27
	Min	79	81
	Max	944	101
	Range	865	20
Platelets	Number	9	Missing value
x 10 9/L	Mean	355	
	SD	166	
	SEM	55	
	Min	0	
	Max	554	
	Range	554	
Biochemical	Number	23	11
Albumin	Mean	40	39
g/L	SD	8	4
	SEM	1,68	1,27
	Min	11	31
	Max	50	45
	Range	39	14
AST	Number	22	11
U/L	Mean	22	16
	SD	6,62	6,19
	SEM	1,41	1,86
	Min	8	8
	Max	33	28
	Range	25	20

		Group 4 A	Group 4 B
Plasma Zinc um/L	Number	13	11
	Mean	0,88	0,92
	SD	0,44	0,32
	SEM	0,12	0,10
	Min	0,38	0,60
	Max	2,11	1,70
	Range	1,73	1,1
Total Globulins g/L	Number	15	11
	Mean	33,67	32,91
	SD	6,96	4,91
	SEM	1,80	1,48
	Min	20	27
	Max	49	41
	Range	29	14
Plasma Creatinine mmol/L	Number	14	10
	Mean	37	36
	SD	10,86	12,67
	SEM	2,90	4,00
	Min	9	3
	Max	54	46
	Range	45	43
Parasitology			
Egg Count in Urine (Repeat) S.Haem)	Number	13	11
	Mean	17	520
	SD	29	867
	SEM	8	261
	Min	0	0
	Max	90	2916
	Range	90	2916

		Group 4 A	Group 4 B
Egg count in stool before	Number	20	11
	Mean	8	24
	SD	14	56
	SEM	3	17
	Min	0	0
	Max	45	190
	Range	45	190
Nutritional Age	Number	23	11
	Mean	10,70	9,91
	SD	1,79	2,26
	SEM	0,37	0,68
	Min	7	7
	Max	13	13
	Range	6	6
Height	Number		
	Mean		
	SD		
	SEM		
	Min		
	Max		
	Range		

Table 7 Comparison of immunological parameters between relapse and remission in MCNS

		MCNS remission	MCNS in relapse + Partial remission	P value
Absolute Mononuclears	Number	16	15	
(cells/mm^3)	Mean	3555	5514	
	SD	1534	2782	0,0418*
	SEM	383	718	
	Min	1988	2457	
	Max	8174	10740	
	Range	6186	8283	
Absolute Lymphocytes	Number	16	15	
(cells/mm^3)	Mean	3085	4935	
	SD	1564	2434	0,0142*
	SEM	391	628	
	Min	1716	2096	
	Max	7906	9666	
	Range	6190	7570	
T cells (E-rosette)	Number	13	14	
(cells/mm^3)	Mean	2218	3827	
	SD	1390	1944	0,0116*
	SEM	385	520	
	Min	1065	1400	
	Max	6294	7088	
	Range	5229	5688	
B cells (SIg)	Number	13	14	
(cells/mm^3)	Mean	179	324	
	SD	154	390	0,3562
	SEM	42	104	
	Min	0	0	
	Max	531	1438	
	Range	531	1438	

		MCNS remission	MCNS in relapse + Partial remission	P value
% T suppressor cell function ConA 5	Number	14	13	
	Mean	30	17	
	SD	29	23	0,4374
	SEM	7	6	
	Min	-2	-21	
	Max	102	55	
	Range	104	76	
% T suppressor cell function ConA 30	Number	15	14	
	Mean	31	38	
	SD	18	24	0,4985
	SEM	4	7	
	Min	-1	2	
	Max	57	81	
	Range	58	79	
PWM (dpm)	Number	9	12	
	Mean	4970	5010	
	SD	2362	3098	0,6440
	SEM	787	894	
	Min	500	219	
	Max	8279	9047	
	Range	7779	8828	
WCC x 10^9/l	Number	16	15	
	Mean	7,96	11,50	
	SD	2,38	5,09	0,0296*
	SEM	0,59	1,31	
	Min	5,20	5,00	
	Max	13,40	21,40	
	Range	8,20	16,40	

		MCNS remission	MCNS in relapse + Partial remission	P value
% Neutrophils	Number	16	15	
	Mean	50	46	
	SD	11	14	0,2508
	SEM	2	4	
	Min	33	22	
	Max	70	81	
	Range	37	59	
% Lymphocytes	Number	16	15	
	Mean	37	43	
	SD	9	11	0,Ø749
	SEM	2	3	
	Min	19	16	
	Max	59	60	
	Range	40	44	
% Monocytes	Number	16	15	
	Mean	6	4	
	SD	3	3	0,1406
	SEM	0,75	0,69	
	Min	2	1	
	Max	12	9	
	Range	10	8	
% Eosinophils	Number	16	15	
	Mean	4	5	
	SD	4	5	0,9045
	SEM	1	1	
	Min	1	0	
	Max	16	21	
	Range	15	21	

		MCNS remission	MCNS in relapse + Partial remission	P value
% Basophils	Number	16	15	
	Mean	0,43	0,49	
	SD	0,62	0,83	0,8509
	SEM	0,15	0,21	
	Min	0	0	
	Max	2	3	
	Range	2	3	
% Atypical Lymphocytes	Number	16	15	
	Mean	0,25	0,93	
	SD	1	1,75	0,0755
	SEM	0,25	0,45	
	Min	0	0	
	Max	4	6	
	Range	4	6	
% Others	Number	16	15	
	Mean	0	0	
	SD	0	0	0,0001*
	SEM	0	0	
	Min	0	0	
	Max	0	0	
	Range	0	0	

Table 8 Comparison of immunological parameters between controls and patients with MCNS with respect to clinical condition

RM = remission PR = Partial remission R = Relapse
MCNS = Minimal change nephrotic syndrome

Parameters		Controls	MCNS, RM	P Value	Controls	MCNS PR, R	P Value
Absolute	Number	17	16		17	15	
mononuclears	Mean	3553	3556	0,9426	4194	5514	0,3451
cells/mm^3	SD	1407	1534		1228	2782	
	SEM	341	384		298	718	
	Min	1290	1988		2100	2457	
	Max	6532	8174		6588	10740	
	Range	5242	6186		4488	8283	
Absolute	Number	17	16		17	15	
Lymphocytes	Mean	3199	3085	0,4712	3823	4935	0,3955
cells/mm^3	SD	1301	1564		1079	2434	
	SEM	315	391		262	628	
	Min	903	1716		1680	2096	
	Max	5822	7906		5856	9666	
	Range	4919	6190		4176	7570	
T cell	Number	17	13		17	14	
(E-rosette)	Mean	1989	2219	0,9167	2515	3827	0,0955
cells/mm^3	SD	940	1391		894	1944	
	SEM	228	386		217	520	
	Min	284	1065		945	1400	
	Max	4005	6294		4612	7088	
	Range	3721	5229		3667	5688	
B cells	Number	17	13		17	14	
(SIg)	Mean	378	179	0,0180*	463	324	0,2335
cells/mm^3	SD	256	154		436	390	
	SEM	62	43		106	104	
	Min	42	0		0	0	
	Max	1078	531		1643	1438	
	Range	1036	531		1643	1438	

Parameters		Controls	MCNS, RM	P Value	Controls	MCNS PR, R	P Value
Null cells	Number	17	13		17	14	
cells/mm^3	Mean	1178	1092	0,8835	1158	1484	0,4998
	SD	770	496		600	988	
	SEM	187	138		145	264	
	Min	68	437		94	497	
	Max	2825	1923		2228	3438	
	Range	2757	1486		2134	2941	
FT cells	Number	17	13		17	14	
cells/mm^3	Mean	7	3	0,4571	8	21	0,7334
	SD	19	10		25	55	
	SEM	5	3		6	15	
	Min	0	0		0	0	
	Max	76	37		88	186	
	Range	76	37		88	186	
T cells	Number	17	16		17	15	
(OKT3MoAb)	Mean	1937	1951	0,6525	2491	3091	0,3955
cells/mm^3	SD	709	867		985	1875	
	SEM	172	217		239	484	
	Min	542	934		1094	846	
	Max	3132	3842		4874	8055	
	Range	2590	2908		3780	7209	
T Helper/in-	Number	17	16		17	15	
ducer cells	Mean	1291	1135	0,2641	1601	1459	0,4168
cells/mm^3	SD	448	476		526	788	
	SEM	109	119		128	203	
	Min	297	354		781	493	
	Max	2025	2125		2701	3007	
	Range	1728	1771		1920	2514	

Parameters		Controls	MCNS, RM	P Value	Controls	MCNS PR, R	P Value
T suppressor/	Number	17	16		17	15	
cytotoxic	Mean	896	1042	0,5403	1142	1699	0,0759
cells	SD	420	552		425	884	
cells/mm^3	SEM	102	138		103	228	
	Min	271	457		483	398	
	Max	1764	2616		1850	3007	
	Range	1493	2159		1367	2609	
% OKT4 of	Number	17	16		17	15	
OKT3	Mean	67	60	0,1652	66	50	0,0011*
	SD	9	16		12	14	
	SEM	2	4		3	3	
	Min	55	34		49	31	
	Max	81	90		100	84	
	Range	26	56		51	53	
% OKT8 of	Number	17	16		17	15	
OKT3	Mean	46	54	0,0381*	47	59	0,0638
	SD	10	10		10	18	
	SEM	2	3		2	5	
	Min	29	38		29	31	
	Max	67	78		67	96	
	Range	38	40		38	65	
Ratio OKT4/	Number	17	16		17	15	
OKT8	Mean	1,54	1,18	0,0335*	1,48	0,93	0,0006*
	SD	0,38	0,40		0,44	0,41	
	SEM	0,09	0,10		0,11	0,10	
	Min	0,97	0,44		1,03	0,33	
	Max	2,29	1,79		2,75	2,04	
	Range	1,32	1,35		1,72	1,71	

Parameters		Controls	MCNS, RM	P Value	Controls	MCNS PR, R	P Value
% suppressor	Number	15	14		16	13	
cell func-	Mean	31	30	0,6943	32	17	0,0913
tion ConA 5	SD	22,89	29,83		18,97	23	
	SEM	5,91	7,97		4,74	6	
	Min	-4	-2		1	-21	
	Max	79	102		70	55	
	Range	83	104		69	76	
% suppressor	Number	16	15		16	14	
cell func-	Mean	38,44	31,33	0,2680	37,31	38	0,9172
tion ConA 30	SD	26,03	18,41		19,77	24	
	SEM	6,51	4,75		4,94	7	
	Min	-28	-1		5	2	
	Max	86	57		65	81	
	Range	114	58		60	79	
PWM (dpm)	Number	10	9		13	12	
	Mean	6408	4970	0,2207	6174	5010	0,7856
	SD	3393	2362		2709	3098	
	SEM	1073	788		751	894	
	Min	642	500		2685	219	
	Max	12520	8280		11924	9047	
	Range	11878	7780		9239	8828	
WCC	Number	17	16		17	15	
x 10^9/L	Mean	8,54	7,97	0,7456	9,20	11,50	0,1622
	SD	3,20	2,39		3,49	5,09	
	SEM	0,78	0,58		0,85	1,31	
	Min	4,30	5,20		5,20	5,00	
	Max	16,60	13,40		18,30	21,40	
	Range	14,30	8,20		18.30	16,40	

Parameters		Controls	MCNS, RM	P Value	Controls	MCNS PR, R	P Value
% Neutro-	Number	17	16		17	15	
phils	Mean	52	51	0,6915	47	46	0,4725
	SD	11	11		11	14	
	SEM	3	3		3	4	
	Min	31	33		23	22	
	Max	70	70		68	81	
	Range	39	37		45	59	
% Lympho-	Number	17	16		17	15	
cytes	Mean	38	38	0,8995	43	43	0,8649
	SD	12	10		11	11	
	SEM	3	2		3	3	
	Min	21	19		24	16	
	Max	62	59		62	60	
	Range	41	40		38	44	
% Monocytes	Number	17	16		17	15	
	Mean	4	6	0,0986	4	4	0,7442
	SD	2	3		2	3	
	SEM	0,51	0,76		0,40	0,69	
	Min	1	2		1	1	
	Max	9	12		6	9	
	Range	8	10		5	8	
% Eosino-	Number	17	16		17	15	
phils	Mean	5	5	0,8414	5	5	0,9089
	SD	5	4		5	5	
	SEM	1	1		1	0	
	Min	0	1		1	0	
	Max	15	16		21	21	
	Range	15	15		20	21	

Parameters		Controls	MCNS, RM	P Value	Controls	MCNS PR, R	P Value
% Basophils	Number	17	16		17	15	
	Mean	0,06	0,43	0,0275*	0,41	0,47	0,8712
	SD	0,24	0,63		0,71	0,83	
	SEM	0,06	0,16		0,17	0,21	
	Min	0	0		0	0	
	Max	1	2		2	3	
	Range	1	2		2	3	
% Atypical	Number	17	16		17	15	
lymphocytes	Mean	0,29	0,25	0,6388	0,35	0,93	0,4313
	SD	0,85	1		0,70	1,75	
	SEM	0,21	0,25		0,17	0,45	
	Min	0	0		0	0	
	Max	3	4		2	6	
	Range	3	4		2	6	
% Others	Number	17	16		17	15	
	Mean	0	0	0,0001*	0	0	0,0001*
	SD	0	0		0	0	
	SEM	0	0		0	0	
	Min	0	0		0	0	
	Max	0	0		0	0	
	Range	0	0		0	0	

Table 9 Comparison of immunological parameters between controls and membranous group of nephrotic syndrome with respect to clinical condition

ME = Membranous RM = Remission PR = Partial Remission R = Relapse

		Controls	ME, RM PR, R	P Value	Controls	ME, PR R	P Value
Absolute mononuclears cells/mm^3	Number	14	14		13	13	
	Mean	3265	4387	0,0482*	3276	4359	0,0858
	SD	1095	1616		1139	1679	
	SEM	293	432		316	466	
	Min	1440	1428		1440	1428	
	Max	5550	7770		5550	7770	
	Range	4110	6342		4110	6342	
Absolute Lymphocytes cells/mm^3	Number	14	14		13	13	
	Mean	3035	3657	0,2148	3034	3579	0,3173
	SD	981	1475		1021	1505	
	SEM	262	394		283	417	
	Min	1332	1020		1132	1020	
	Max	4884	6660		4884	6660	
	Range	3552	5640		3752	5640	
T cell (E-rosette) cells/mm^3	Number	14	14		13	13	
	Mean	1847	3010	0,0308*	1838	2997	0,0483*
	SD	593	1732		616	1802	
	SEM	159	463		171	500	
	Min	749	457		749	457	
	Max	2722	6760		2722	6760	
	Range	1973	6303		1973	6303	
B cells (SIg) cells/mm^3	Number	14	14		13	13	
	Mean	401	190	0,0274*	410	164	0,0111*
	SD	366	143		379	111	
	SEM	98	38		105	31	
	Min	86	0		86	0	
	Max	1591	524		1591	385	
	Range	1505	524		1505	385	

		Controls	ME, RM PR, R	P Value	Controls	ME, PR R	P Value
Null cells cells/mm^3	Number	14	14		13	13	
	Mean	951	1178	0,3827	957	1188	0,3695
	SD	613	692		638	719	
	SEM	164	185		177	199	
	Min	302	547		302	547	
	Max	2719	2706		2719	2706	
	Range	2417	2159		2417	2159	
FT cells cells/mm^3	Number	14	14		13	13	
	Mean	8	9	1,0000	9	10	1,0000
	SD	24	25		25	25	
	SEM	6	7		7	7	
	Min	0	0		0	0	
	Max	88	78		88	78	
	Range	88	78		88	78	
T cells (OKT3MoAb) cells/mm^3	Number	13	14		12	13	
	Mean	1861	2691	0,0225*	1902	2671	0,0502
	SD	536	1192		539	1238	
	SEM	149	319		156	343	
	Min	691	443		691	443	
	Max	2459	5051		2459	5051	
	Range	1768	4608		1768	4608	
T Helper/inducer cells cells/mm^3	Number	13	14		12	13	
	Mean	1106	1345	0,1594	1107	1324	0,2534
	SD	350	644		365	665	
	SEM	97	172		105	184	
	Min	619	243		619	243	
	Max	1944	2376		1944	2376	
	Range	1325	2133		1325	2133	

		Controls	ME, RM PR, R	P Value	Controls	ME, PR R	P Value
T suppressor/	Number	13	14		12	13	
cytotoxic	Mean	827	1448	0,0039*	836	1442	0,0083*
cells	SD	249	799		258	831	
cells/mm^3	SEM	69	214		74	231	
	Min	273	214		273	214	
	Max	1061	3341		1061	3341	
	Range	788	3127		788	3127	
% OKT4 of	Number	13	14		12	13	
OKT3	Mean	62	51	0,2636	60	51	0,3832
	SD	16	14		15	14	
	SEM	4	4		4	4	
	Min	44	26		44	26	
	Max	90	70		90	70	
	Range	46	44		46	44	
% OKT8 of	Number	13	14		12	13	
OKT3	Mean	45	54	0,4512	45	54	0,4622
	SD	12	23		12	24	
	SEM	3	6		3	7	
	Min	20	15		20	15	
	Max	66	110		66	110	
	Range	46	95		46	95	
Ratio OKT4/	Number	13	14		12	13	
OKT8	Mean	1,43	1,05	0,0466*	1,42	1,05	0,0508
	SD	0,46	0,51		0,48	0,54	
	SEM	0,13	0,14		0,14	0,15	
	Min	0,74	0,33		0,74	0,33	
	Max	2,43	2,22		2,43	2,22	
	Range	1,69	1,89		1,69	1,89	

		Controls	ME, RM PR, R	P Value	Controls	ME, PR R	P Value
% suppressor	Number	13	14		12	13	
cell func-	Mean	39	22	0,1092	37,25	19,08	0,0817
tion ConA 5	SD	26	30		26,41	29,03	
	SEM	7	8		7,62	8,05	
	Min	-11	-35		-11,00	-35	
	Max	78	84		78	84	
	Range	89	119		89	119	
% suppressor	Number	14	14		13	13	
cell func-	Mean	49	30	0,1128	48	26	0,0905
tion ConA 30	SD	28	28		29,17	25,40	
	SEM	8	8		8,09	7,05	
	Min	-6	-45		-6	-45	
	Max	89	81		89	63	
	Range	95	126		95,00	108	
PWM (dpm)	Number	12	6		11	5	
	Mean	5759	5506	0,7079	5637	4082	0,4615
	SD	3372	4007		3510	2206	
	SEM	973	1636		1058	986	
	Min	796	735		796	736	
	Max	14020	12624		10420	6907	
	Range	13224	11889		9624	6171	
WCC	Number	14	14		13	13	
x 10^9/L	Mean	7,70	13	0,0021*	7,71	13,54	0,0023*
	SD	3,25	4,63		3,38	4,61	
	SEM	0,87	1,24		0,94	1,28	
	Min	3,60	7,00		3,60	7,00	
	Max	13,00	22,20		13,00	22,20	
	Range	9,40	15,20		9,40	15,20	

		Controls	ME, RM PR, R	P Value	Controls	ME, PR R	P Value
% Neutro-	Number	14	14		13	13	
phils	Mean	53	58	0,4201	53	59	0,2078
	SD	9	14		9	13	
	SEM	2	4		3	4	
	Min	41	40		41	40	
	Max	70	92		70	92	
	Range	29	52		29	52	
% Lympho-	Number	14	14		13	13	
cytes	Mean	41	30	0,0106*	41	28	0,0034*
	SD	9	12		9	10	
	SEM	2	3		2	3	
	Min	28	5		28	5	
	Max	54	55		54	44	
	Range	26	50		26	39	
% Monocytes	Number	14	14		13	13	
	Mean	3	6	0,0131*	3	6	0,0065*
	SD	2	3		2	3	
	SEM	0,49	0,90		0,50	0,89	
	Min	1	1		1	2	
	Max	7	13		7	13	
	Range	6	12		6	11	
% Eosino-	Number	14	14		13	13	
phils	Mean	2	6	0,0965	2	6	0,1250
	SD	3	8		3	8	
	SEM	0,85	2		0,90	2,23	
	Min	0	0		0	0	
	Max	10	30		10	30	
	Range	10	30		10	20	

		Controls	ME, RM PR, R	P Value	Controls	ME, PR R	P Value
% Basophils	Number	14	14		13	13	
	Mean	0,14	0,71	0,9590	0,15	0,08	0,9558
	SD	0,53	0,27		0,55	0,28	
	SEM	0,14	0,07		0,15	0,08	
	Min	0	0		0	0	
	Max	2	1		2	1	
	Range	2	1		2	1	
% Atypical	Number	14	14		13	13	
lymphocytes	Mean	0	0	0,0001*	0	0	0,0001*
	SD	0	0		0	0	
	SEM	0	0		0	0	
	Min	0	0		0	0	
	Max	0	0		0	0	
	Range	0	0		0	0	
% Others	Number	14	14		13	13	
	Mean	0	0	0,0001*	0	0	0,0001*
	SD	0	0		0	0	
	SEM	0	0		0	0	
	Min	0	0		0	0	
	Max	0	0		0	0	
	Range	0	0		0	0	

Table 10 Comparison of immunological parameters between controls and Proliferative group of nephrotic syndrome with respect to clinical condition

Prol. = Proliferative Rem. = remission PR = Partial remission R = Relapse

		Controls	Prol.Rem. PR, R	P Value	Controls	Prol. PR, R.	P Value
Absolute	Number	11	9		9	8	
mononuclears	Mean	3599	5131	0,0874	3773	5558	0,0675
cells/mm^3	SD	1993	1900		2159	1504	
	SEM	601	633		720	532	
	Min	1290	1725		1290	3128	
	Max	7406	8120		7406	8120	
	Range	6116	6395		6116	4992	
Absolute	Number	11	9		9	8	
Lymphocytes	Mean	3238	4407	0,1837	3348	4786	0,1779
cells/mm^3	SD	1949	1764		2049	1443	
	SEM	587	588		683	510	
	Min	903	1380		903	2788	
	Max	6762	7308		6762	7308	
	Range	5859	5928		5859	4520	
T cell	Number	11	9		9	8	
(E-rosette)	Mean	2290	3331	0,1024	2373	3642	0,0675
cells/mm^3	SD	1436	1405		1578	1124	
	SEM	433	468		526	397	
	Min	284	845		284	1814	
	Max	4814	5071		4814	5071	
	Range	4530	4226		4530	3257	
B cells	Number	11	8		9	7	
(SIg)	Mean	327	160	0,2831	341	181	0,4914
cells/mm^3	SD	322	125		357	121	
	SEM	97	44		119	46	
	Min	16	0		16	0	
	Max	1037	325		1037	325	
	Range	1021	325		1021	325	

		Controls	Prol.Rem. PR, R	P Value	Controls	Prol. PR, R.	P Value
Null cells	Number	11	9		9	8	
cells/mm^3	Mean	894	1664	0,0527	1049	1765	0,1019
	SD	559	1129		489	1164	
	SEM	169	376		163	411	
	Min	94	716		232	716	
	Max	1699	4466		1699	4466	
	Range	1605	3750		1467	3750	
FT cells	Number	11	9		9	8	
cells/mm^3	Mean	9	0	0,0990	9	0	0,1693
	SD	18	0		20	0	
	SEM	5	0		7	0	
	Min	0	0		0	0	
	Max	56	0		56	0	
	Range	56	0		56	0	
T cells	Number	11	9		9	8	
(OKT3MoAb)	Mean	1844	2956	0,0527	1837	3176	0,0269*
cells/mm^3	SD	1009	1290		1076	1184	
	SEM	304	430		359	419	
	Min	542	1190		542	1478	
	Max	3699	5197		3699	5197	
	Range	3157	4007		3157	3719	
T Helper/in-	Number	11	9		9	8	
ducer cells	Mean	1139	1392	0,4250	1156	1476	0,3359
cells/mm^3	SD	565	619		590	605	
	SEM	170	206		197	214	
	Min	297	438		297	438	
	Max	1962	2192		1962	2192	
	Range	1665	1754		1665	1754	

		Controls	Prol.Rem. PR, R	P Value	Controls	Prol. PR, R.	P Value
T suppressor/	Number	11	9		9	8	
cytotoxic	Mean	883	1507	0,0627	936	1626	0,0675
cells	SD	538	786		585	748	
cells/mm^3	SEM	162	262		195	265	
	Min	248	552		248	672	
	Max	1850	2761		1850	2761	
	Range	1602	2209		1602	2089	
% OKT4 of	Number	11	9		9	8	
OKT3	Mean	63	47	0,0250*	65	48	0,0432*
	SD	12	15		12	16	
	SEM	3	5		4	6	
	Min	52	25		53	25	
	Max	86	73		86	73	
	Range	34	48		33	52	
% OKT8 of	Number	11	9		9	8	
OKT3	Mean	47	56	0,3805	49	58	0,4680
	SD	10	20		9	21	
	SEM	3	7		3	8	
	Min	29	31		38	31	
	Max	66	90		66	90	
	Range	37	59		28	59	
Ratio OKT4/	Number	11	9		9	8	
OKT8	Mean	1,39	1,10	>0,05	1,35	1,07	0,1390
	SD	0,36	0,56		0,32	0,60	
	SEM	0,11	0,19		0,11	0,21	
	Min	0,97	0,30		0,97	0,30	
	Max	2,05	2,05		1,81	2,05	
	Range	1,08	1,75		0,84	1,75	

		Controls	Prol.Rem. PR, R	P Value	Controls	Prol. PR, R.	P Value
% suppressor	Number	9	8		7	7	
cell func-	Mean	32	27	0,7727	33	22	0,5649
tion ConA 5	SD	20	32		23	31	
	SEM	7	11		9	12	
	Min	10	-32		10	-32	
	Max	67	60		67	57	
	Range	57	92		57	89	
% suppressor	Number	10	8		8	7	
cell func-	Mean	42	37	0,4767	46	31	0,1828
tion ConA 30	SD	23	25		25	19	
	SEM	7	9		9	7	
	Min	14	4		14	4	
	Max	95	79		95	67	
	Range	81	75		81	63	
PWM (dpm)	Number	8	6		6	6	
	Mean	7301	5660	0,2453	6810	5660	0,4233
	SD	3152	1387		2965	1388	
	SEM	1115	567		1210	567	
	Min	3175	4262		3175	4262	
	Max	11924	7501		11279	7501	
	Range	8749	3239		8104	3239	
WCC	Number	11	9		9	8	
x $10^{9/L}$	Mean	7,79	11,68	0,0366*	8,10	12,28	0,0342*
	SD	3,44	3,56		3,72	3,28	
	SEM	1,04	1,19		1,23	1,16	
	Min	4,30	6,80		4,30	6,80	
	Max	16,10	16,00		16,10	16,00	
	Range	11,80	9,20		11,8	9,20	

		Controls	Prol.Rem. PR, R	P Value	Controls	Prol. PR, R.	P Value
% Neutro-	Number	11	9		9	8	
phils	Mean	54	49	0,3599	55	46	0,1354
	SD	12	15		13	12	
	SEM	4	5		4	4	
	Min	32	26		32	26	
	Max	70	75		70	64	
	Range	38	49		38	38	
% Lympho-	Number	11	9		9	8	
cytes	Mean	41	38	0,5176	40	40	0,9615
	SD	12	12		13	11	
	SEM	4	4		4	4	
	Min	21	20		21	24	
	Max	62	63		62	63	
	Range	41	43		41	39	
% Monocytes	Number	11	9		9	8	
	Mean	4	4	0,4824	4	4	0,9218
	SD	2	3		2	3	
	SEM	0,70	0,90		0,81	1	
	Min	1	2		1	2	
	Max	9	10		9	10	
	Range	8	8		8	8	
% Eosino-	Number	11	9		9	8	
phils	Mean	2	8	0,0526	0,66	9	0,0023*
	SD	3	11		0,87	11	
	SEM	0,92	4		0,29	4	
	Min	0	0		0	1	
	Max	10	32		2	32	
	Range	10	32		2	32	

		Controls	Prol.Rem. PR, R	P Value	Controls	Prol. PR, R.	P Value
% Basophils	Number	11	9		9	8	
	Mean	0,10	0,11	0,8839	0,11	0,13	0,9314
	SD	0,30	0,33		0,33	0,35	
	SEM	0,09	0,11		0,11	0,13	
	Min	0	0		0	0	
	Max	1	1		1	1	
	Range	1	1		1	1	
% Atypical	Number	11	9		9	8	
lymphocites	Mean	0,18	0	0,3657	0,22	0	0,3458
	SD	0,60	0		0,67	0	
	SEM	0,18	0		0,22	0	
	Min	0	0		0	0	
	Max	2	0		2	0	
	Range	2	0		2	0	
% Others	Number	11	9		9	8	
	Mean	0	0,33	0,2689	0	0,38	0,2888
	SD	0	1		0	1,06	
	SEM	0	0,33		0	0,38	
	Min	0	0		0	0	
	Max	0	3		0	3	
	Range	0	3		0	3	

Table 11 Comparison of immunological parameters between controls and Miscellaneous group of nephrotic syndrome with respect to clinical condition

M = Miscellaneous RM = Remission PR = Partial Remission R = Relapse

		Controls	M, RM PR, R	P Value	Controls	M PR, R.	P Value
Absolute	Number	11	10		9	8	
mononuclears	Mean	3248	3918	0,3242	3427	4034	0,3865
cells/mm^3	SD	1132	2033		1184	2271	
	SEM	341	643		395	803	
	Min	1551	546		1551	546	
	Max	5605	6854		5605	6854	
	Range	4054	6308		4054	6308	
Absolute	Number	11	10		9	8	
Lymphocytes	Mean	2984	3363	0,4813	3168	3414	0,5637
cells/mm^3	SD	1013	1836		1036	2069	
	SEM	305	580		345	732	
	Min	1504	420		1504	420	
	Max	5130	6556		5130	6556	
	Range	3626	6136		3626	6136	
T cell	Number	11	9		9	8	
(E-rosette)	Mean	1852	2752	0,5184	2007	2851	0,7003
cells/mm^3	SD	658	2001		624	2115	
	SEM	198	667		208	748	
	Min	1046	399		1224	399	
	Max	3251	6374		3251	6374	
	Range	2205	5975		2027	5975	
B cells	Number	11	9		9	8	
(SIg)	Mean	340	124	0,0364*	381	102	0,0206*
cells/mm^3	SD	253	138		261	131	
	SEM	76	46		87	46	
	Min	16	0		16	0	
	Max	785	376		785	376	
	Range	769	376		769	376	

		Controls	M, RM PR, R	P Value	Controls	M PR, R.	P Value
Null cells cells/mm^3	Number	11	9		9	8	
	Mean	1050	1031	0,6761	1031	1075	0,8474
	SD	465	817		497	861	
	SEM	140	272		166	304	
	Min	232	104		232	104	
	Max	1601	2728		1601	2728	
	Range	1369	2624		1369	2624	
FT cells cells/mm^3	Number	11	9		9	8	
	Mean	5	3	0,5015	6	3	0,5628
	SD	17	8		18	8	
	SEM	5	3		6	3	
	Min	0	0		0	0	
	Max	56	23		56	23	
	Range	56	23		56	23	
T cells (OKT3MoAb) cells/mm^3	Number	11	10		9	8	
	Mean	1834	2222	0,5493	1948	2240	0,7361
	SD	915	1174		983	1317	
	SEM	276	371		328	465	
	Min	605	333		605	333	
	Max	3699	3699		3699	3699	
	Range	3094	3366		3094	3366	
T Helper/inducer cells cells/mm^3	Number	11	10		9	8	
	Mean	1083	1160	0,8880	1123	1233	0,9233
	SD	494	799		544	888	
	SEM	149	253		181	314	
	Min	450	140		450	140	
	Max	1962	2493		1962	2493	
	Range	1512	2353		1512	2353	

		Controls	M, RM PR, R	P Value	Controls	M PR, R.	P Value
T suppressor/	Number	11	10		9	8	
cytotoxic	Mean	904	1073	0,3786	988	1103	0,5964
cells	SD	534	607		556	684	
cells/mm^3	SEM	161	192		186	242	
	Min	248	49		248	49	
	Max	1850	2071		1850	2071	
	Range	1602	2022		1602	2022	
% OKT4 of	Number	11	10		9	8	
OKT3	Mean	62	49	0,0842	60	51	0,3120
	SD	14	20		15	22	
	SEM	4	6		5	8	
	Min	31	15		31	15	
	Max	78	85		78	85	
	Range	47	70		47	70	
% OKT8 of	Number	11	10		9	8	
OKT3	Mean	48	47	0,8877	50	47	0,6993
	SD	14	16		13	17	
	SEM	4	5		4	6	
	Min	29	15		33	15	
	Max	78	70		78	70	
	Range	49	55		45	55	
Ratio OKT4/	Number	11	10		9	8	
OKT8	Mean	1,43	1,28	0,2311	1,34	1,38	0,5637
	SD	0,58	0,98		0,56	1,09	
	SEM	0,17	0,31		0,19	0,39	
	Min	0,40	0,30		0,40	0,30	
	Max	2,35	3,89		2,35	3,89	
	Range	1,95	3,59		1,95	3,59	

		Controls	M, RM PR, R	P Value	Controls	M PR, R.	P Value
% suppressor cell function ConA 5	Number	8	10		7	8	
	Mean	38	16	0,0682	39	19	0,1176
	SD	22	27		23	27	
	SEM	8	9		9	10	
	Min	9	-22		9	-22	
	Max	76	69		76	69	
	Range	67	91		67	91	
% suppressor cell function ConA 30	Number	10	10		8	8	
	Mean	44	35	0,4055	47	41	0,5995
	SD	28	35		31	35	
	SEM	9	11		11	12	
	Min	14	-11		14	-1	
	Max	88	92		88	92	
	Range	74	103		74	93	
PWM (dpm)	Number	8	8		8	7	
	Mean	6079	4875	0,9164	6079	4951	1,000
	SD	4388	1606		4388	1719	
	SEM	1551	568		1551	650	
	Min	1418	2072		1418	2072	
	Max	14697	6601		14698	6601	
	Range	13279	4529		13280	4529	
WCC x 10^9/L	Number	11	10		9	8	
	Mean	7,65	9,29	0,4809	8,08	9,36	0,7360
	SD	2,81	4,49		2,96	4,86	
	SEM	0,85	1,42		0,99	1,72	
	Min	4,70	4,20		4,70	4,20	
	Max	14,70	17,70		14,70	17,70	
	Range	10,00	13,50		10,00	13,50	

		Controls	M, RM PR, R	P Value	Controls	M PR, R.	P Value
% Neutro-	Number	11	10		9	8	
phils	Mean	51	53	0,6215	50	53	0,5964
	SD	10	13		10	13	
	SEM	3	4		3	5	
	Min	35	28		35	28	
	Max	66	67		66	67	
	Range	31	39		31	39	
% Lympho-	Number	11	10		9	8	
cytes	Mean	40	32	0,1485	40	30	0,1119
	SD	9	12		10	12	
	SEM	3	4		3	4	
	Min	24	10		24	10	
	Max	54	46		54	44	
	Range	30	36		30	34	
% Monocytes	Number	11	10		9	8	
	Mean	3	5	0,3905	3	5	0,1701
	SD	2	3		2	4	
	SEM	0,58	1		0,62	1	
	Min	1	2		1	2	
	Max	6	10		6	10	
	Range	5	8		5	8	
% Eosino-	Number	11	10		9	8	
phils	Mean	6	11	0,4150	6	11	0,5603
	SD	7	17		7	19	
	SEM	2	5		2	6	
	Min	0	0		0	0	
	Max	21	56		21	56	
	Range	21	56		21	56	

		Controls	M, RM PR, R	P Value	Controls	M PR, R.	P Value
% Basophils	Number	11	10		9	8	
	Mean	0,27	0,30	0,6857	0,33	0,38	0,7176
	SD	0,64	0,95		0,71	1,06	
	SEM	0,19	0,30		0,24	0,38	
	Min	0	0		0	0	
	Max	2	3		2	3	
	Range	2	3		2	3	
% Atypical	Number	11	10		9	8	
lymphocites	Mean	0,45	0,30	0,6434	0,22	0,375	0,8636
	SD	1,04	0,95		0,67	1,06	
	SEM	0,31	0,30		0,22	0,37	
	Min	0	0		0	0	
	Max	3	3		2	3	
	Range	3	3		2	3	
% Others	Number	11	10		9	8	
	Mean	0	0,10	0,2943	0	0,125	0,2888
	SD	0	0,32		0	0,35	
	SEM	0	0,10		0	0,12	
	Min	0	0		0	0	
	Max	0	1		0	1	
	Range	0	1		0	1	

Table 12 Long term change in immunological parameters among all MCNS patients in remission

		MCNS Remission ≤5 years	MCNS Remission >5 years	P Value
Absolute mononuclears cells/mm^3	Number	4	12	
	Mean	5255	2988	
	SD	2126	758	0,0393*
	SEM	2063	218	
	Min	3634	1988	
	Max	8174	4158	
	Range	4540	2170	
Absolute lymphocytes cells/mm^3	Number	4	12	
	Mean	4803	2512	
	SD	2272	705	0,0522
	SEM	1136	203	
	Min	2976	1716	
	Max	7906	3542	
	Range	4930	1826	
T cells cells/mm^3	Number	3	10	
	Mean	4014	1679	
	SD	2044	502	0,0180*
	SEM	1180	158	
	Min	2344	1065	
	Max	6294	2571	
	Range	3950	1506	
B cells(SIg) cells/mm^3	Number	3	10	
	Mean	235	162	
	SD	66	171	0,1763
	SEM	38	54	
	Min	165	0	
	Max	297	531	
	Range	132	531	

		MCNS Remission ≤5 years	MCNS Remission >5 years	P Value
Null cells cells/mm^3	Number	3	10	
	Mean	1533	959	
	SD	449	447	0,0910
	SEM	259	141	
	Min	1042	437	
	Max	1923	1865	
	Range	881	1428	
FT cells cells/mm^3	Number	3	10	
	Mean	12	0	
	SD	21	0	0,0679
	SEM	12	0	
	Min	0	0	
	Max	37	0	
	Range	37	0	
T cells OKT3(MoAb)	Number	4	12	
	Mean	2801	1666	
	SD	963	648	0,0393*
	SEM	481	187	
	Min	1744	934	
	Max	3842	2841	
	Range	2098	1907	
T helper/ inducer cells/mm^3	Number	4	12	
	Mean	1488	1017	
	SD	598	388	0,1456
	SEM	299	112	
	Min	781	354	
	Max	2125	1788	
	Range	1344	1434	

		MCNS Remission ≤5 years	MCNS Remission >5 years	P Value
Suppressor/ cytotoxic cells/mm^3	Number	4	12	
	Mean	1698	823	
	SD	687	278	0,0109*
	SEM	343	80	
	Min	1018	457	
	Max	2626	1439	
	Range	1598	982	
% OKT4 of OKT3	Number	4	12	
	Mean	53	62	
	SD	15	15	0,2747
	SEM	7	4	
	Min	34	36	
	Max	71	90	
	Range	37	54	
% OKT8 of OKT3	Number	4	12	
	Mean	61	51	
	SD	15	7	0,1619
	SEM	7	2	
	Min	41	38	
	Max	78	61	
	Range	37	23	
Ratio OKT4/OKT8	Number	4	12	
	Mean	0,95	1,26	
	SD	0,40	0,40	0,3023
	SEM	0,20	0,11	
	Min	0,44	0,68	
	Max	1,32	1,79	
	Range	0,88	1,11	

		MCNS Remission ≤5 years	MCNS Remission >5 years	P Value
T suppressor cell function ConA 5	Number	3	11	
	Mean	29	30	
	SD	20	32	0,9379
	SEM	12	9	
	Min	6	-2	
	Max	47	102	
	Range	41	104	
T suppressor cell function ConA 30	Number	3	12	
	Mean	32	31	
	SD	23	18	0,8850
	SEM	13	5	
	Min	6	-1	
	Max	51	57	
	Range	45	58	
PWM (dpm)	Number	2	7	
	Mean	5442	4835	
	SD	4011	2160	0,7697
	SEM	2936	816	
	Min	2606	500	
	Max	8279	7589	
	Range	5673	7089	
WCC x 10^9/L	Number	4	12	
	Mean	10,20	7,20	
	SD	3,73	1,23	0,1813
	SEM	1,86	0,35	
	Min	6,20	5,20	
	Max	13,40	9,40	
	Range	7,20	4,20	

		MCNS Remission ≤5 years	MCNS Remission >5 years	P Value
% Neutrophils	Number	4	12	
	Mean	41	53	
	SD	8	10	0,0782
	SEM	4	3	
	Min	34	33	
	Max	53	70	
	Range	19	37	
% Lymphocytes	Number	4	12	
	Mean	46	35	
	SD	9	8	0,0449*
	SEM	4	2	
	Min	38	19	
	Max	59	46	
	Range	21	27	
% Monocytes	Number	4	12	
	Mean	5	6	
	SD	4	2	0,4625
	SEM	2	0,75	
	Min	2	3	
	Max	12	11	
	Range	10	8	
% Eosinophils	Number	4	12	
	Mean	6	4	
	SD	7	3	0,9508
	SEM	3	1	
	Min	1	1	
	Max	16	13	
	Range	15	12	

		MCNS Remission ≤5 years	MCNS Remission >5 years	P Value
% Basophils	Number	4	12	
	Mean	0,25	0,50	
	SD	0,50	0,67	0,5224
	SEM	0,25	0,19	
	Min	0	0	
	Max	1	2	
	Range	1	2	
% Atypical Lymphocytes	Number	4	12	
	Mean	0	0,33	
	SD	0	1,15	0,5637
	SEM	0	0,33	
	Min	0	0	
	Max	0	4	
	Range	0	4	
% Others	Number	4	12	
	Mean	0	0	
	SD	0	0	
	SEM	0	0	
	Min	0	0	
	Max	0	0	
	Range			

Table 13 Comparison of immunological parameters of all nephrotic syndrome patients (regardless of histology and clinical condition) with age, sex and race matched controls

Parameters		Controls	All Nephrotic Syndrome Patients regardless of Histology or condition	P value
Absolute mononuclears cells/mm^3	Number	56	64	
	Mean	3678	4475	
	SD	1370	2210	0,0145*
	SEM	183	264	
	Min	1290	546	
	Max	7406	10740	
	Range	6116	10194	
Absolute lymphocytes cells/mm^3	Number	56	64	
	Mean	3353	3873	
	SD	1248	1935	0,0794
	SEM	167	242	
	Min	903	420	
	Max	6762	9666	
	Range	5859	9246	
T cell (E-rosette) cells/mm^3	Number	56	59	
	Mean	2123	3039	
	SD	883	1753	0,0006*
	SEM	118	228	
	Min	284	399	
	Max	4814	7088	
	Range	4530	6689	
B cells (SIg) cells/mm^3	Number	56	58	
	Mean	415	206	
	SD	342	232	0,0002*
	SEM	46	31	
	Min	0	0	
	Max	1642	1438	
	Range	1643	1438	

Parameters		Controls	All Nephrotic Syndrome Patients regardless of Histology or condition	P value
Null cells cells/mm^3	Number	56	59	
	Mean	1103	1283	
	SD	651	835	0,2008
	SEM	87	109	
	Min	68	104	
	Max	2825	4466	
	Range	2757	4362	
FT cells cells/mm^3	Number	56	59	
	Mean	7	8	
	SD	21	30	0,8405
	SEM	3	4	
	Min	0	0	
	Max	88	186	
	Range	88	186	
T cells (OKT3MoAb) cells/mm^3	Number	55	64	
	Mean	2068	2564	
	SD	828	1367	0,0169*
	SEM	112	171	
	Min	542	333	
	Max	4875	8055	
	Range	4333	7722	
T helper/inducer cells cells/mm^3	Number	55	64	
	Mean	1293	1297	
	SD	500	660	0,9716
	SEM	67	82	
	Min	297	140	
	Max	2701	3007	
	Range	2404	2967	

Parameters		Controls	All Nephrotic Syndrome Patients regardless of Histology or condition	P value
T suppressor/cytotoxic cells/mm^3	Number	55	64	
	Mean	955	1355	
	SD	410	761	0,0004*
	SEM	55	95	
	Min	248	49	
	Max	1850	3341	
	Range	1602	3292	
% OKT4 of OKT3	Number	55	64	
	Mean	64	52	
	SD	13	16	0,0001*
	SEM	2	2	
	Min	31	15	
	Max	100	90	
	Range	69	75	
% OKT8 of OKT3	Number	55	64	
	Mean	47	54	
	SD	11	18	0,0046*
	SEM	1	2	
	Min	20	15	
	Max	78	110	
	Range	58	95	
Ratio OKT4/OKT8	Number	55	64	
	Mean	1,45	0,99	
	SD	0,45	0,61	0,0001*
	SEM	0,06	0,08	
	Min	0,40	0,03	
	Max	2,75	3,89	
	Range	2,35	3,86	

Parameters		Controls	All Nephrotic Syndrome Patients regardless of Histology or condition	P value
% suppressor cell	Number	49	59	
function ConA 5	Mean	35	23	
	SD	23	28	0,0138*
	SEM	3	4	
	Min	-11	-35	
	Max	79	102	
	Range	90	137	
% suppressor cell	Number	53	61	
function ConA 30	Mean	44	34	
	SD	26	26	0,0356*
	SEM	4	3	
	Min	-28	-45	
	Max	95	92	
	Range	123	137	
MNC PWM stimulation	Number	42	41	
	Mean	6333	5143	
	SD	3435	2557	0,0775
	SEM	530	399	
	Min	642	219	
	Max	14698	12624	
	Range	14056	12405	
WCC	Number	56	64	
x 10^9/L	Mean	8,59	10,67	
	SD	3,47	4,46	0,0056*
	SEM	0,46	0,56	
	Min	3,60	4,20	
	Max	18,30	22,20	
	Range	14,70	18,00	

Parameters		Controls	All Nephrotic Syndrome Patients regardless of Histology or condition	P value	ue
% Neutrophils	Number	56	64		
	Mean	51	51		,2
	SD	10	13	0,9975	
	SEM	1	2		
	Min	23	22		
	Max	70	92		
	Range	49	70		
% Lymphocytes	Number	56	64		
	Mean	40	36		32
	SD	10	12	0,0499*	
	SEM	1	2		
	Min	21	5		
	Max	62	63		
	Range	41	58		
Monocytes	Number	56	64		
	Mean	3	5		34
	SD	2	3	0,0003*	
	SEM	0,25	0,38		
	Min	1	1		
	Max	9	13		
	Range	8	12		
Eosinophils	Number	56	64		
	Mean	4	6		
	SD	5	9	0,1366	
	SEM	0,71	1		
	Min	0	0		
	Max	21	56		
	Range	21	56		