SUMMARY AND CONCLUSION

The mechanism rsponsible for MCNS still remains unknown. T-cell dysfunction was postulated as an etiological factor.

The present work was an attempt to study the T-cell function in nephrotic syndrome of different stages and to try to interpret the cause of T-cell dysfunction.

This study was carried out in Mounira Children Hospital of Cairo University during the years 1984-1985. The material of the study included 42 children of both sexes; their ages ranged from 2 to 12 years. They were classified into four groups; the corticosteroid-responsive group in active stage (11 patients), the resistant group (9 patients), the remission group (12 patients) and the control group (10 normal children).

In addition to thorough clinical examination, all children were subjected to the following investigations:

I. Routine investigations(complete urine analysis and complete blood picture).

II. Chemical study (blood urea, serum creatinine, protein loss in 24 hours urine, plasma proteins,

serum cholesterol, serum LDL cholsterol, serum VLDL cholesterol, plasma zinc and hair zinc).

III. Lymphocyte study (separation and enumeration of T-lymphocytes. PHA induced blastoid transformation was done in three different situations; the washed lymphocytes alone, the lymphocyte culture in presence of autologous plasma and finally the lymphocyte culture in presence of autologous plasma devoid of its LDL-& VLDL-cholesterol).

IV. Renal biopsy (was done for resistant cases).

The data of the work were statistically analysed, with following results:

Rosette formation was significantly increased in active nephrosis in both the responsive and the resistant groups($P < 0.001$), however the latter group showed an insignificant higher resutls than the former one . In remission, rosette formation was insignificantly different from the control group but significantly lower than the responsive group ($P < 0.001$).

The blastoid transformation of the washed lymphocytes was significantly lower in the active nephrosis

of either the responsive or the resistant group ($P < 0.001$), while the resistant group was significantly lower than the responsive one ($P < 0.001$). During remission the blastoid transformation was significantly higher than the active group ($P < 0.001$), but significantly lower than the control group ($P < 0.001$).

The addition of the autologous plasma to the cultured lymphocytes, showed a significant inhibition of tansformation in the responsive, the resistant and the remission group ($P < 0.001$).

Separation of lipoprotein (LDL and VLDL) from the field of the cutlure, resulted in partial relief of the inhibitory effect of plasma which was signficant in ($P < 0.001$). In the responsive group that relief was correlated with serum levels of LDL, however in the resistant group it was correlated with serum levels of both LDL and VLDL. In remission there was insignificant relief.

The rest of the inhibitory effect of plasma was significant in all groups ($P < 0.001$). In the active groups the difference was inversely correlated with plasma zinc but not correlated during remission.

This study proved that T-cell dysfunction was

evident in nephrotic syndrome whether the responsive or resistant types, while during remission the dysfunction improved.

The cause of T-cell dysfunction was due to an intrinsic defect in addition to extrinsic factors namely raised serum levels of the immunosuppressive lipoproteins (LDL & VLDL) and the decreased zinc concentrations. Thus T-cell dysfunction could be a manifestation of nephrotic syndrome as a consequence of its metabolic changes, while intrinsic cell defect could denote an aetiological factor.