Viruses and Glomerulonephritis

K.	Kurt	Lange
----	------	-------

Dr. Kurt Lange, MD, Lenox Hill Hospital, 100 East 77th Street, Achilles, Room No. 509, New York, N.Y. 10021 (USA)

Sir,

In the Editorial Review, Nephron 31:97-102 (1982), the authors gather, in a comprehensive way, evidence that many glomerulonephritides in viral diseases are caused by circulating immune complexes. The disappearance of virus particles detected in the glomeruli of mice by immunofluorescence and electron microscopy which then disappear after 10 days must not necessarily be explained by disintegration or excretion of the virus particles. It is best explained by saturation of the viral antigen with antibody newly formed by the mice themselves which then cover the viral antigen and do not permit any longer immunologic complexing with labelled antibody used for the studies (blocking). When antigen is consistently produced as in lymphocytic choriomeningitis, absent or very low free antibody titers may result in the presence of circulating viral particles and still LCM virus may be found in the kidneys. This is especially so since the viral particles are found in this disease in association with IgG deposits probably testifying to an in situ immune complex formation rather than a primary cytotoxic effect of the virus itself. An almost identical situation can be found in the Aleutian mink disease, again in all probability, an in situ immune complex formation.

These situations resemble in the sequence of immunologic events the mechanism which we have described in human poststreptococcal glomerulonephritis [1, 2] where evidence appears incontestable that we are dealing with an in situ immune complex formation and not with the deposition of the much maligned and probably only epiphenomenal circulating immune complexes.

One may suspect that in many, if not most, instances of bacterial or viral immune reactions in the kidney, in situ IC reactions, as the above mentioned, take place as many examples show [3].

More work needs to be done to clearly demonstrate the time sequence of such pathogenetic processes, especially in viral diseases. The assumption of the localization of circulating immune complexes as pathogenetic mechanisms is not necessarily justifiable.

References

Lange, K.; Ahmed, U.; Kleinberger, K.; Treser, G.: A hitherto unknown streptococcal antigen and its probable relation to acute poststreptococcal glomerulonephritis. Clin.Nephrol. 5:207 (1976).

Lange, K.; Seligson, G.; Cronin, W.: Evidence for the in situ origin of poststreptococcal glomerulonephritis (in print).

Hamburger, J.: Proc. 8th Int. Congr. Nephrol., Athens 1981.