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Recent Studies on Human Glomerulopathies Using Various Lectins

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Summary. **The results of our current study demonstrate that each disease entity expresses its unique lectin binding profile and that increases, decreases, appearances, and disappearances of lectin binding site(s) are predictable. We feel that these types of data may be used to generate valuable computer profiles of renal diseases. These profiles then can be used for diagnostic and prognostic indices and to subclassify renal diseases to predict responses to therapy.**

INTRODUCTION

Study of glycoconjugates provides valuable information on biochemical structure and functions of normal and diseased organs. Since the early eighties, lectins, highly specific and effective non-immune molecular probes, have been used to study the kidney in animals and humans. Works reported in the literature so far are mainly focused on distribution of lectin receptors in normal kidneys and role of glycosylation in the development of nephron.[1-3] Only few studies in humans, using limited numbers of lectins, have examined the lectin binding in glomerular diseases.[4,5]

MATERIALS AND METHODS

Thirty cases from our pathology archive were selected for the study. The cases included 3 to 5 patients in each of the following disease groups: Minimal change disease (MINCH DIS), Focal and segmental glomerulonephritis (FSGN), Membranous glomerulonephritis (MEMBGN), Membranoproliferative glomerulonephritis (MPGN), Crescentic glomerulonephritis (CRESGN) and Lupus glomerulopathy (LUPUSGN). We have selected seven chemically pure lectins for their carbohydrate specificity to study common clinical renal diseases mentioned above. Lectins were obtained from commercial source (Vector Lab. Burlingam, CA, USA). The lectins used were WGA {NeuNAc & (D-glcNAc)2}, CON-A (α-D-Man & α-D-Glc), PNA {β-D-gal(1-3)-D-galNAc}, UEA-1 (α-L-fucose), RCA-1 (β-D-Gal), BSL-1 (α-D-galNAc & α-D-Gal), and SBA (D-galNAc). Strepavidin-biotin (SABC) immunoperoxidase technique was applied on 6 micron thick tissue sections. To control the staining reaction, lectins pretreated with corresponding blocking sugars were also used. The results were scored as follows: the percentage of positive cells and the intensity of staining reaction were recorded an assigned an arbitrary value of 0% (no stain and no intensity) to 100% (all cells show positive stain and 2+intensity).

RESULTS

From the results presented in Figs. 1-5, following generalization on the lectin binding patterns can be made.

1. Normal glomerulus is remarkable by absence of PNA, presence of large amounts of UEA-1 on endothelial cells, CON-A and RCA-1 on parietal epithelial cells and WGA in the glomerular basement membrane. 2. Diseased glomeruli show expression of PNA on intrinsic cells in MPGN and on GBM in FSGN. In addition, marked increases of WGA, CON-A and RCA-1 on glomerular endothelium and loss of SBA from glomerular cells and GBM in nephrotic diseases are quite striking.

DISCUSSION

In comparison to the lectin binding pattern of normal glomeruli, the diseased glomeruli show detectable changes which appear to be quite unique to the corresponding disease entity (Figs. 1-5). For example,

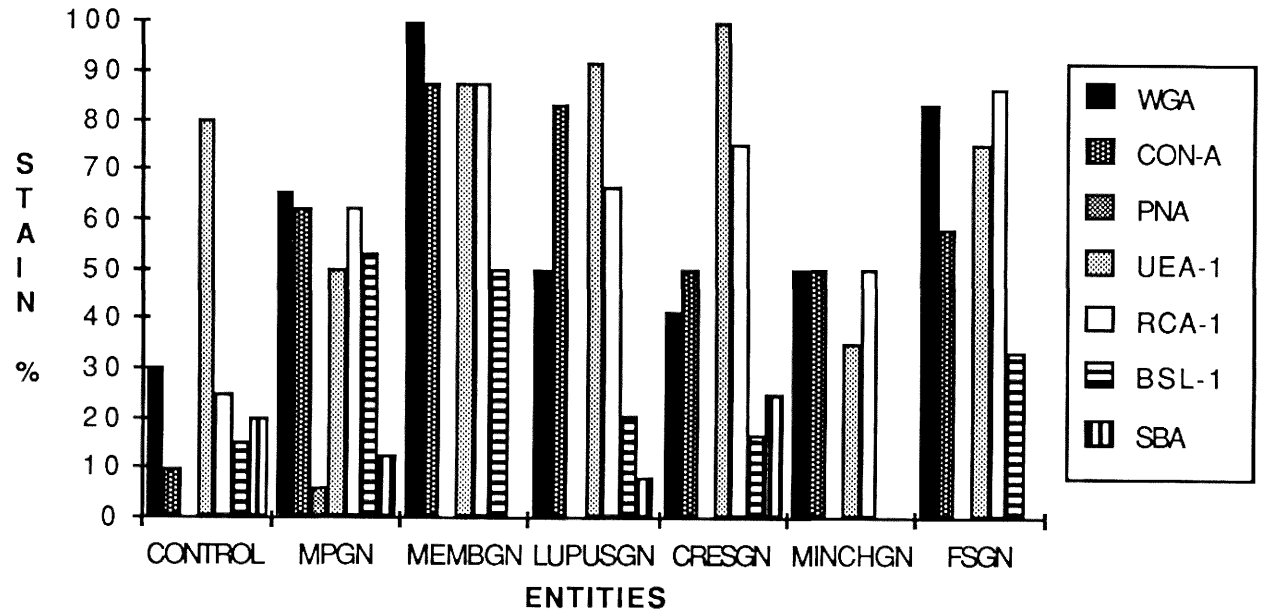

Fig. 1. Glomerular endothelium lectin binding pattern.

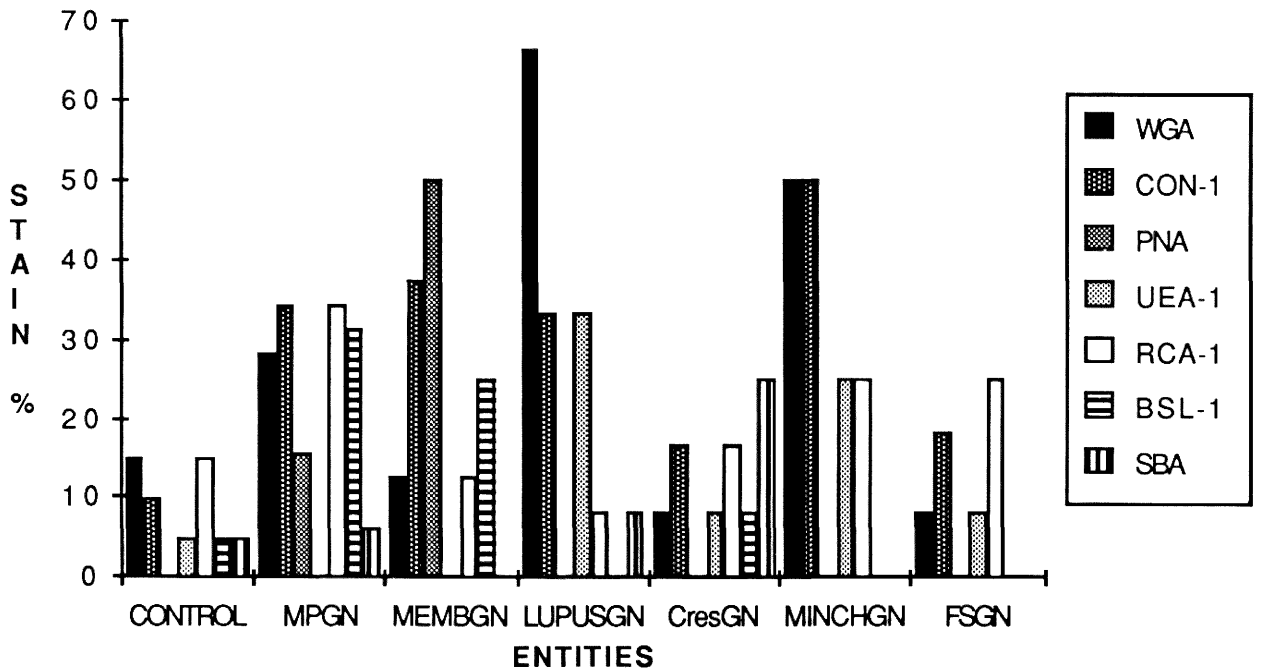

Fig. 2. Mesangial cell lectin binding pattern.

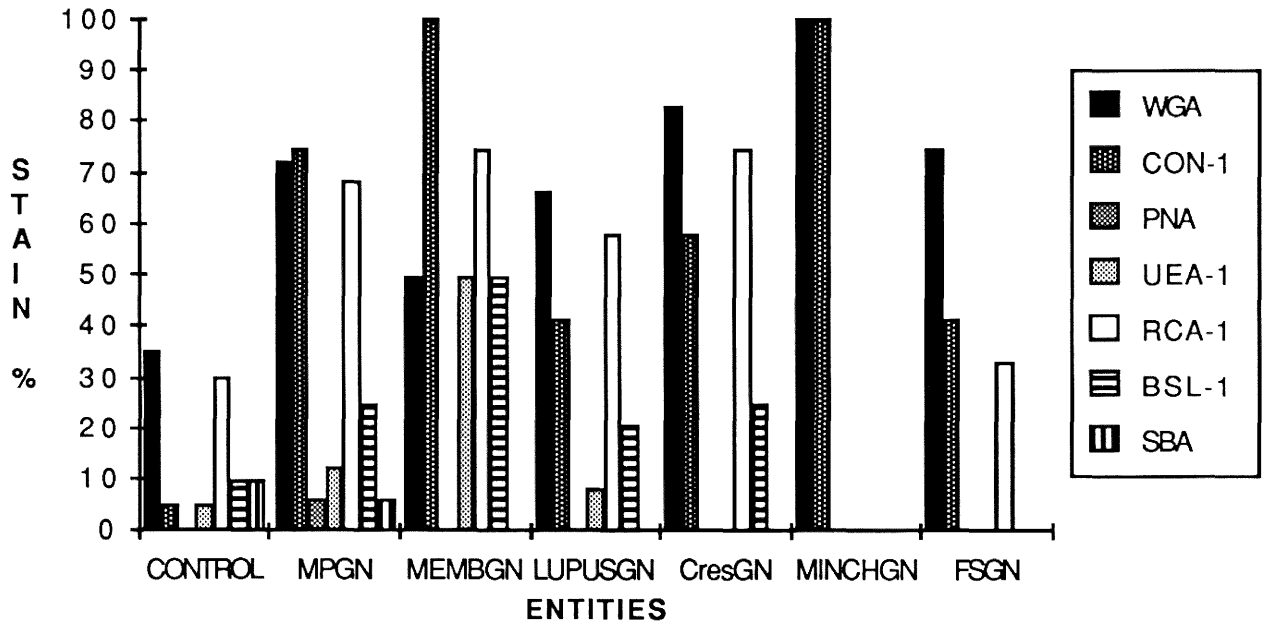

Fig. 3. Visceral epithelial cell lectin binding pattern.

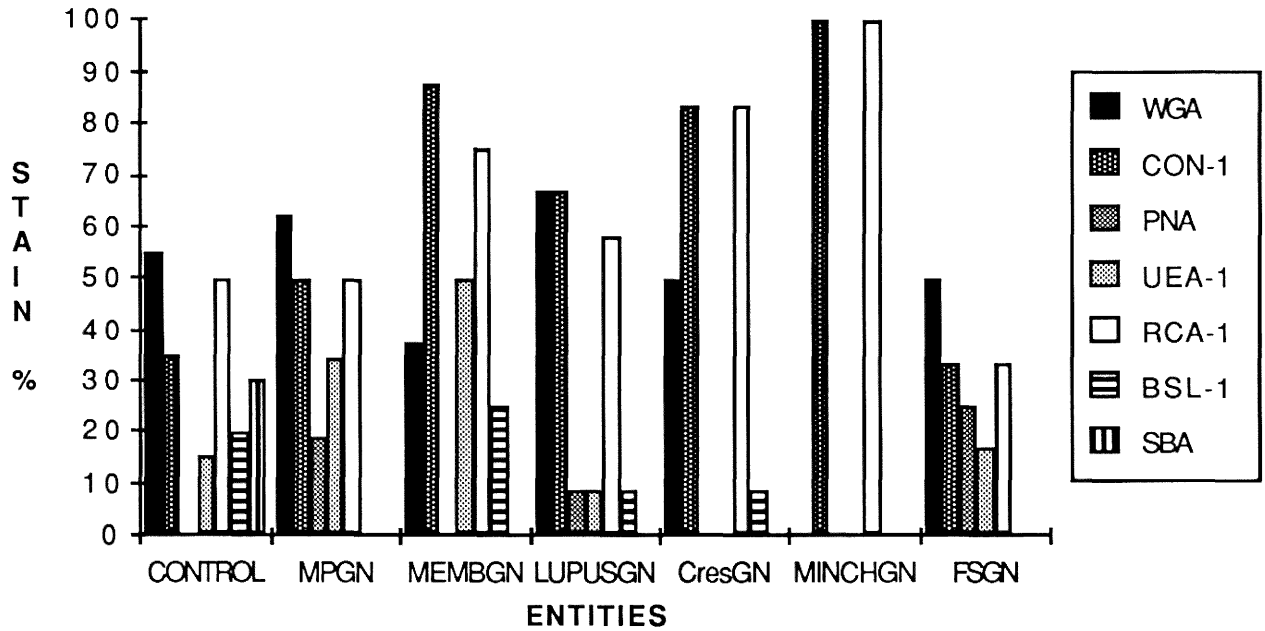

Fig. 4. Glomerular parietal epithelium lectin binding pattern.

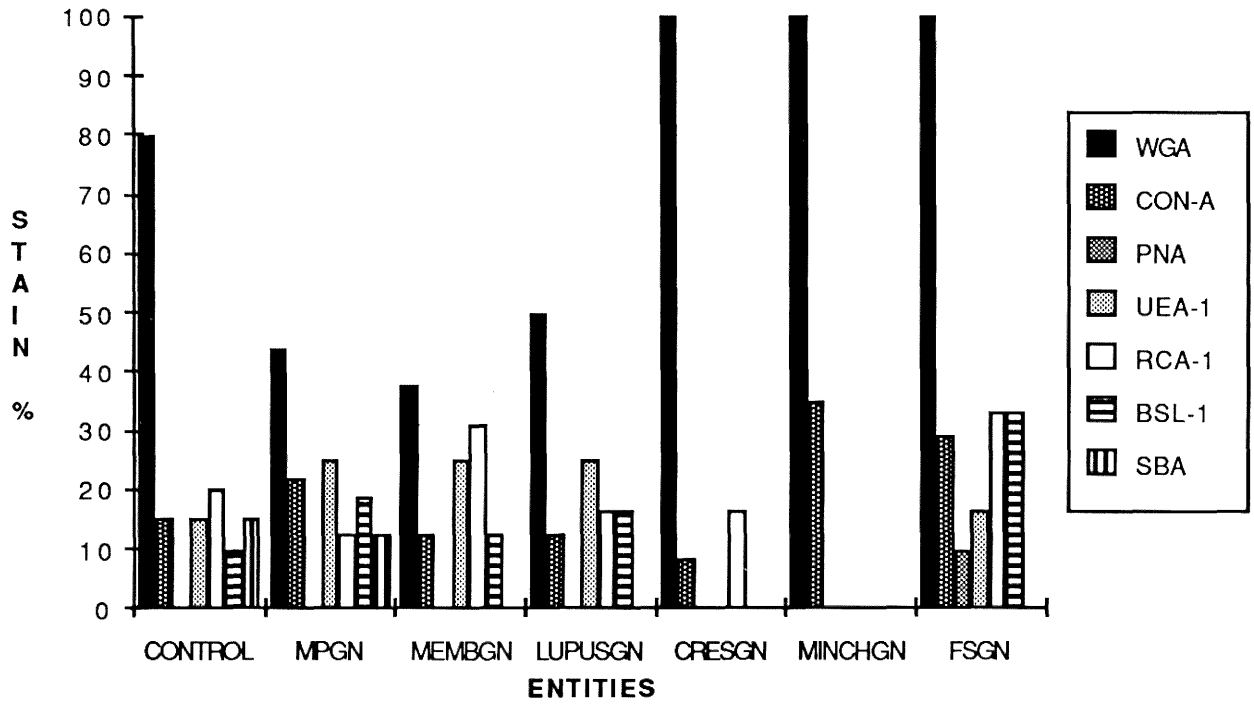

Fig. 5. GBM lectin binding pattern.

SBA binding sites were lost from glomeruli of nephrotic diseases like MEMBGM, MINCH DIS and FSGN; PNA sites were expressed on glomerular cells in MPGN, and on GBM in FSGN. Our WGA and PNA data confirm the reported results in the literature.[4,5] The other observations are new and we consider them to be preliminary at this time. We believe that diseases are manifestations of disorders occurring at or near molecular and/or macromolecular levels brought about by changes in their microenvironment by various etiologies. Studies conducted at the molecular levels are the best avenues available to us to understand better the molecular events which occur in health and diseases.

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