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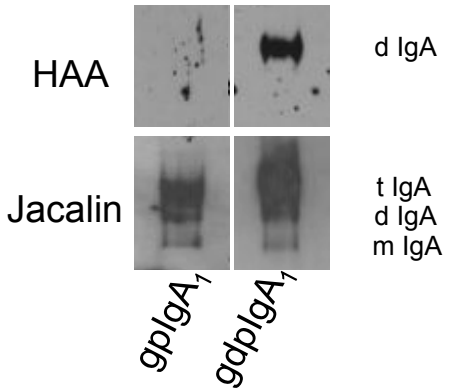
The combined role of galactose-deficient IgA and streptococcal IgA-binding M protein in inducing IL-6 and C3 secretion from human mesangial cells: implications for IgA nephropathy



Supplementary Figure 1

Supplementary figure 1: Lectin binding assay of polymeric IgA1 proteins

IgA1 proteins (gpIgA1: galactosylated polymeric IgA1 from pooled control sera, gdpIgA1: galactose-deficient polymeric IgA1-IgAN) separated by SDS-PAGE under non-reducing conditions were transferred onto nitrocellulose membranes and analyzed using biotinylated *Helix aspersa* lectin (HAA; specific for terminal *N*-acetylgalactosamine; Sigma) or jacalin (specific for disaccharide *N*-acetylgalatosamine-galactose; Vector Laboratories, Burlingame, CA) and detected with avidin-peroxidase conjugate and chemiluminescence as described (1). Galactose-deficient polymeric IgA1 characterized by binding to HAA was only detected in gdpIgA1 and was predominantly in a dimeric molecular form. In contrast, all three molecular forms of both types of IgA1 reacted with jacalin. m IgA, monomeric IgA; d IgA, dimeric IgA, t IgA, trimeric IgA.

Reference

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