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Pathogenic role of (S)IgA in IgA nephropathy

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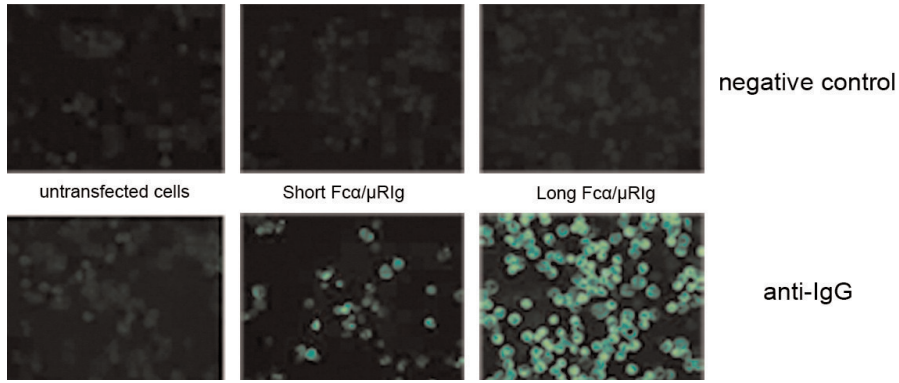
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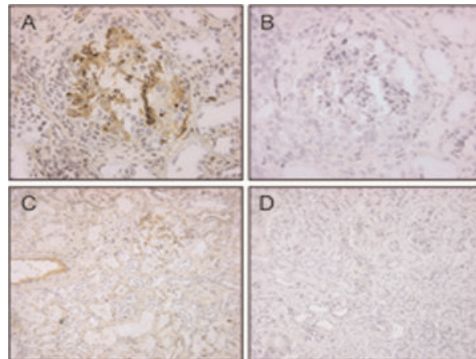
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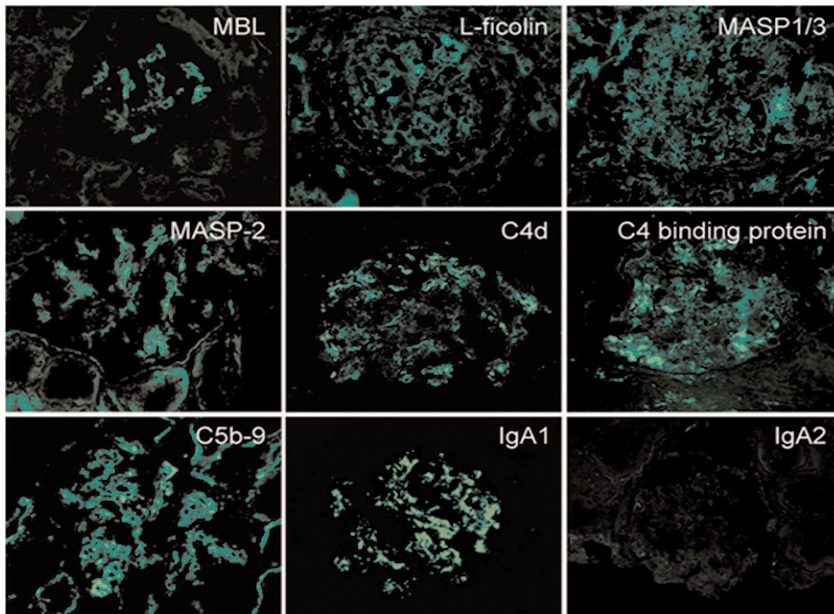
COLOR FIGURES



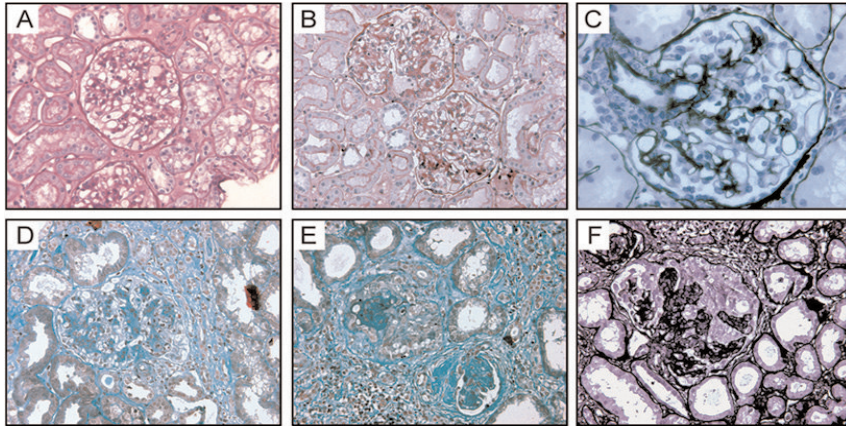
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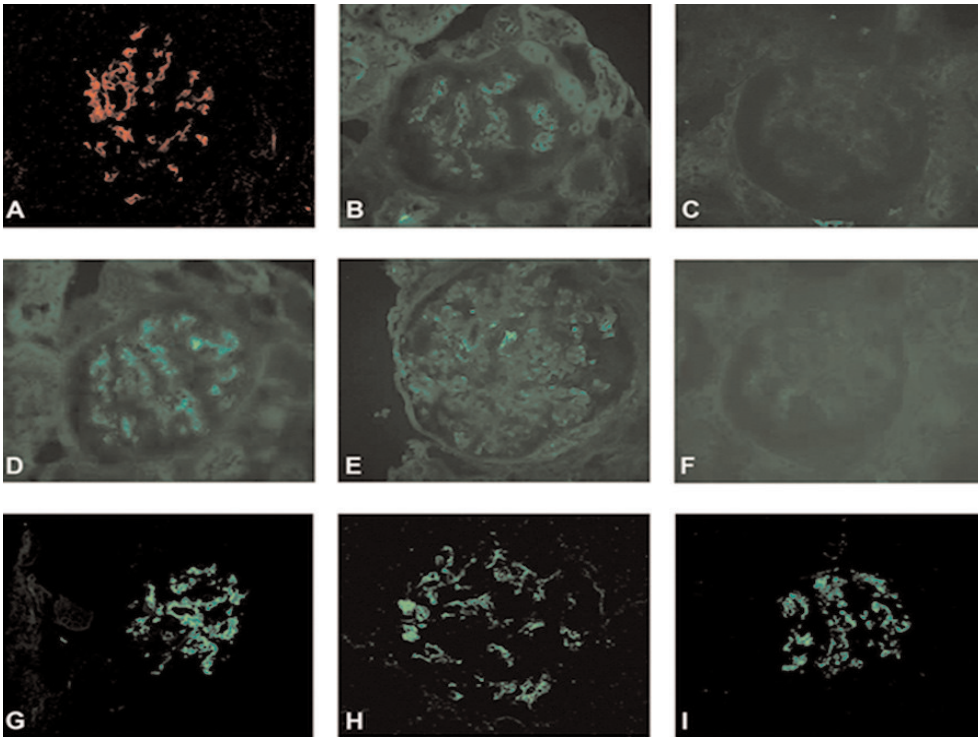
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Fc α μ Rlg fusion proteins.

B) IgG staining of transfected cells after cytopspin centrifugation.

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Glomerular MBL deposition in IgAN patients.

Biopsies from patients with IgAN were stained with mAb 1C10 (A), mAb 3E7 (C, D) or an isotype control mAb (B). Figures A, C, and D are derived from different patients who showed positive (A, C) or negative (D) staining for MBL. Figures A and B are from the same patient. Please note tubular and vascular staining for MBL in C, in addition to glomerular staining.

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Lectin pathway activation in IgAN.

Renal tissue from IgAN patients was stained for the presence of MBL (mAb 3E7), L-ficolin (mAb GN4), MASP-1/3 (mAb 1E2), MASP-2, C4d, C4 binding protein, C5b-9, IgA1 and IgA2 (mAb NI512), as indicated. Representative images are shown.

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Renal histological damage in IgAN patients.

Renal histology from three patients without glomerular MBL deposition (A-C) shows glomeruli affected by mild mesangial proliferation and mild mesangial matrix expansion. No major tubulo-interstitial lesions are visible. Images D-F represent patients with glomerular MBL deposition (N = 3). Glomerular injury is characterized by intense mesangial proliferation and mesangial matrix expansion (D), segmental and global sclerosis (E), and extracapillary proliferation (F), whereas tubular dilations, interstitial infiltration and fibrosis are evident in the interstitium (D-F). Sections were stained with PAS (A, B), PASM (C, F) or trichrome (D, E) techniques.

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Glomerular SIgA deposition in IgAN patients.

Renal tissue from patients with IgAN was stained for the presence of SIgA (A, B and C), MBL (D, E and F), IgA (G), C4d (H) and C3 (I). Representative images are shown. Renal tissues are derived from different patients who showed positive (A and B) and negative staining (C) for SIgA and positive (D and E) and negative staining (F) for MBL. All patients were positive for IgA (G). A subpopulation of patients was positive for C4d (H) and C3 (I).

