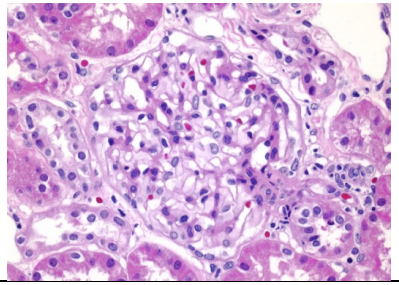
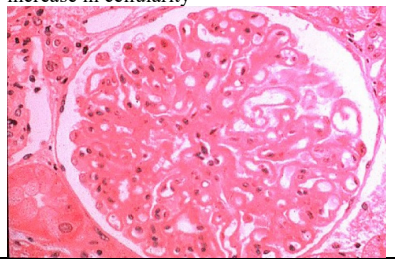
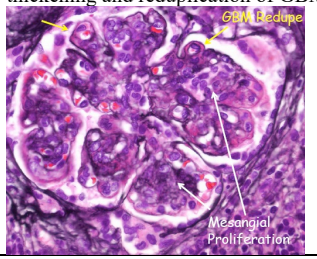
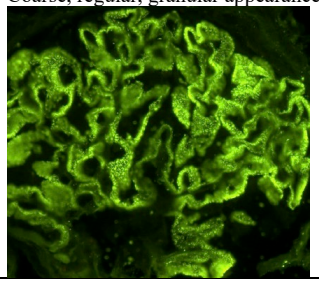
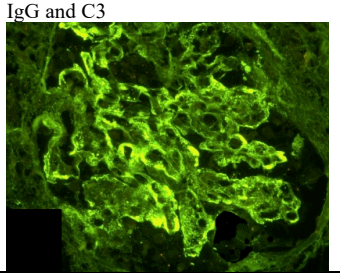
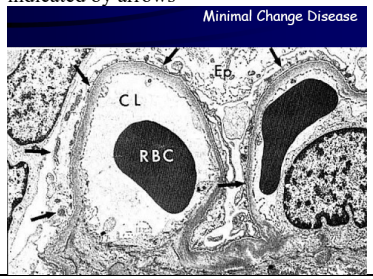
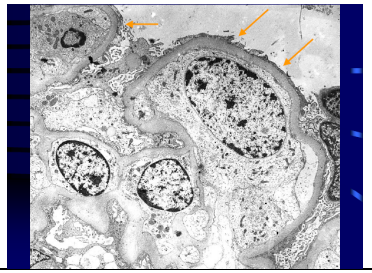
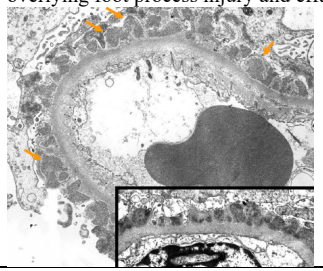
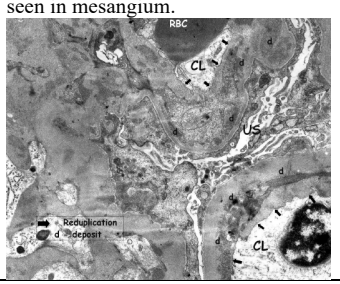
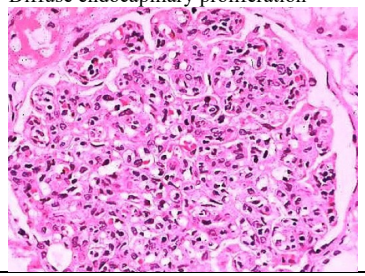
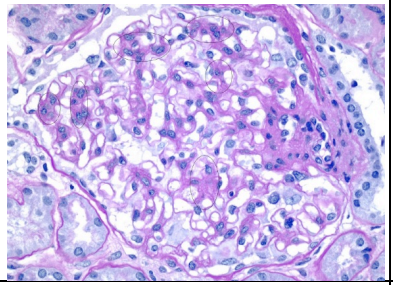
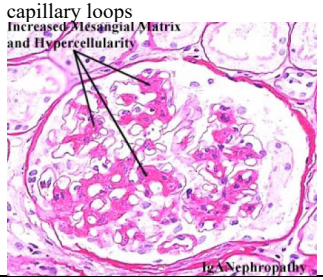
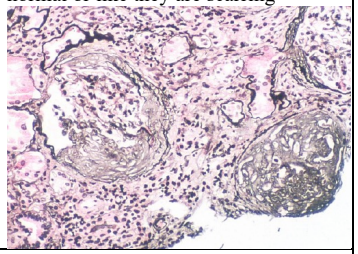
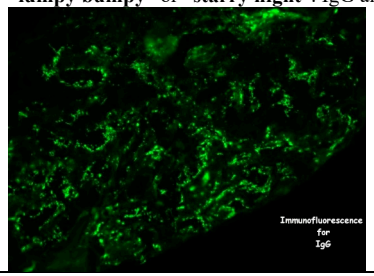
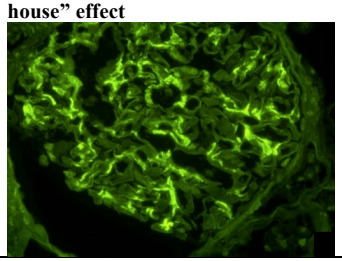
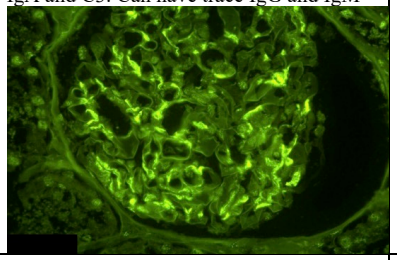
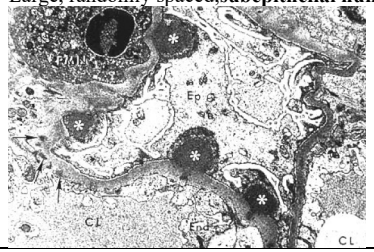
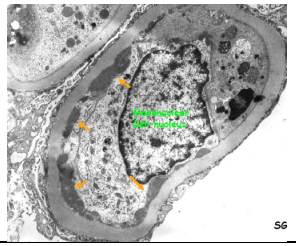
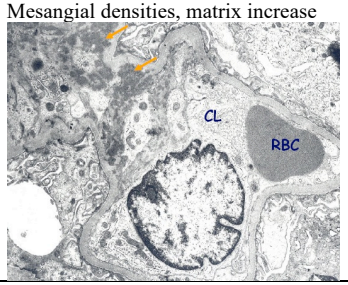
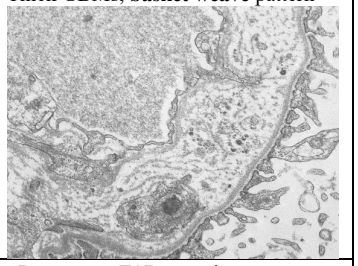
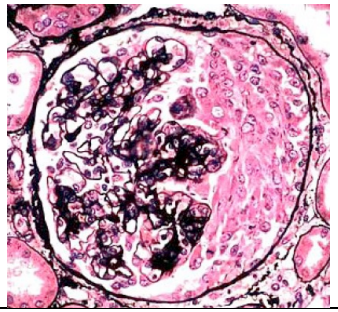
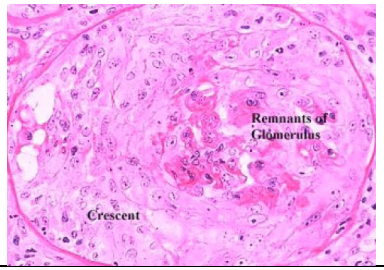
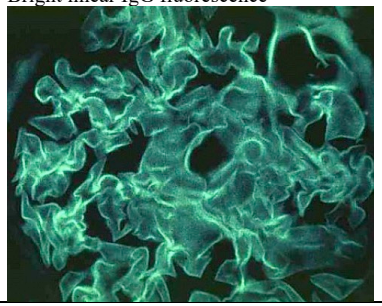
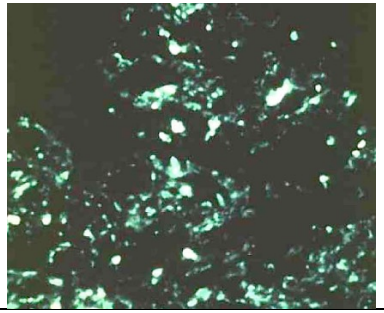
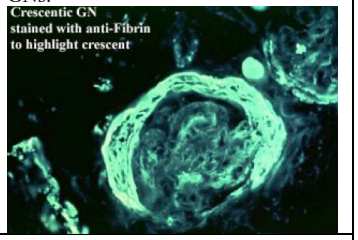
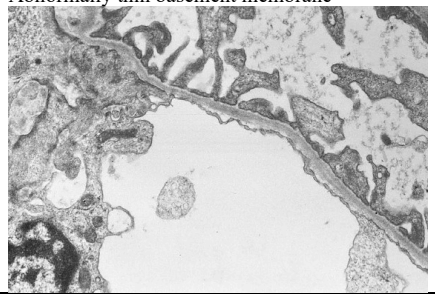
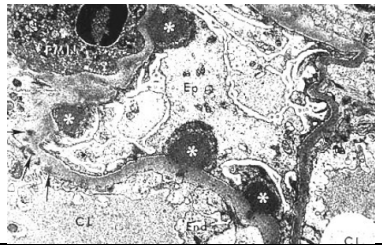
	Minimal Change Disease	FSGS	Membranous glomerulonephritis	Membranoproliferative Glomerulonephritis
Etiology	Podocyte injury of unknown cause Get reduction of polyanionic components of GBM *can be caused by **NSAIDS**	Unknown circulating factor. Serum from infected patients can infect experimental animals.	~autoimmune disorder with antibodies to podocyte cell membrane antigens ~85% idiopathic ~Secondary causes: malaria, syph, hepB, SLE, RA, solid cancers, gold, penicillamine, captopril	~Idiopathic immune complex mediated disease ~Chronic infections, immune disorders and malignancies can cause similar type of disease
Clinical Presentation	~Nephrotic syndrome ~Selective proteinuria, with albumin as predominant protein.	~Nephrotic Syndrome ~More likely to have non-selective proteinuria than MCD, more likely to progress to renal failure	~Nephrotic Syndrome ~Uncommon in children, but very common in adults.	~Nephrotic range proteinuria, hypertension, persistant hematuria, hypocompementemia, increased creatinine ~primarily children and young adults
LM	Normal 	Starts out as segmental sclerotic process – ends up global and diffuse. 	Diffuse uniform thickening of GBM without an increase in cellularity 	Hypercellular glomeruli, endocapillary proliferation, thickening and reduplication of GBMs 
IF	negative	Negative – might see a little IgM or C3, but this is NOT related to the pathogenesis of the lesion	Coarse, regular, granular appearance w. IgG or C3 	Peripheral capillary loop and mesangial staining for IgG and C3 
EM	Effacement of podocyte foot processes, as indicated by arrows 	Effacement of podocyte foot processes, much like MCD. 	Supepithelial immune complex deposits with overlying foot process injury and effacement 	Subendothelial electron dense immune deposits with mesangial interpositioning. Deposits can also be seen in mesangium. 
Prognosis	~ Usually responsive to steroids	Much less responsive to steroids than MCD, other more potent drugs are being tried.	May resolve, remain static, or slowly progress to ultimate renal failure	If idiopathic, usually progressive with poor prognosis. If secondary, and you treat cause, may be reversible.
Comments	~May follow after URTI ~Increased incidence in **Hodgkins** patients	~Can be secondary to any developmental anomaly or disease process that results in decreased glomerulus number ~**collapsing glomerulonephropathy** = variant with massive proteinuria and rapid progression. Associated with HIV and heroin use	~Get activation of complement, especially the C5b-9 complex, injuring the podocyte and adjacent GBM. ~**SUPEPITHELIAL** deposits ~certain stains can show **"spiked" or "holey"** appearance on LM due to growing up of GBM between deposits.	~Complexes are deposited in **MESANGIUM** and **SUBENDOTHELIAL SPACE**. ~Can present in various forms –asymptomatic hematuria/proteinuria, ANS, or mixed nephritic/nephritic. ~Suspect if: nephrotic range proteinuria + Large # of RBCs or increased creatinine

	Acute Nephritic Syndrome	Lupus Nephritis	IgA nephropathy	Alports Disease
Etiology	~Immune complex mediated: complexes form and activate complement subendothelially and then migrate to epithelial side ~Most commonly, post-streptococcal glomerulonephritis ~Others – SLE, severe IgA nephropathy, membranoproliferative	Immune complex mediated, complexes can deposit in any part of the kidney. Two major glomerular changes: proliferative and membranous	Unknown cause, but results from trapping of IgA subtype 1 containing immune complexes in the mesangium	~Hereditary defect in Type IV collagen production ~**X linked**: absent α5 isoform ~**autosomal**: absent α3 or α4.
Clinical Presentation	~Hematuria, proteinuria (usually not nephritic range), hypertension, mild renal insufficiency	Can present in multiple diff ways: nephrotic syndrome, ANS, asymptomatic hematuria/proteinuria, or ARF	~asymptomatic hematuria/proteinuria ~Most commonly in children and young adults ~Often history of **URTI**	Hematuria, high frequency hearing loss, various ocular defects
LM	Diffuse endocapillary proliferation 		Mesangial hypercellularity but nice open capillary loops 	Nonspecific – glomeruli may look normal or like they are scarring 
IF	"**lumpy bumpy**" or "**starry night**". IgG and C3 	Deposits in mesangium and loops. **"full house" effect** 	IgA and C3. Can have trace IgG and IgM 	~routinely negative ~can use anti-sera against various isoform chains of Type IV collagen and demonstrate lack of chains in the GBMs of affected patients.
EM	Large, randomly spaced,**subepithelial humps** 	"fingerprint-like" substructure with **tubuloreticular inclusions** 	Mesangial densities, matrix increase 	Thick GBMs, **basket weave** pattern 
Prognosis	~Kids: 95% recover spontaneously, ≤1% get RPGN, 1-2% progress to CRF ~Adults: 60% recover spontaneously, 40% develop RPGN or CRF	~better predicted by the chronic changes present at the time of biopsy than the amt. of active lesions ~good if few chronic changes	May be benign, but progresses to CRF in 10-15%. No effective treatment, trials for fish oil.	~Progress to ESD at various rates. Renal transplant is the only real treatment when failure ensues ~Problem with transplant: anti-GBM disease
Comments	~Typical post-strep presentation: abrubt onset 1-3 weeks after throat infection or 3-6 weeks after skin infection with **group A beta hemolytic strep**. Symptoms: edema, hypertension, hematuria, azotemia, mild proteinuria and oliguria. Early in disease, low C3 and C4. Positive ASO or anti-DNAseB. ~Can see C1q and C4 on IF = **classical cascade**	~Treat with steroids and cytotoxic agents like cytoxan with good results as long as there are few chronic changes present.	~Most common cause of primary glomerulonephritis in the world. ~No C4 or C1q, therefore **alternative pathway**	Very uncommon, responsible for about 2-3%of ESRD in young people.

	Thin Basement membrane disease	Type 1 RPGN	Type 2 RPGN	Type 3 RPGN
Etiology	~Autosomally inherited defect in GBM, but the etiology is unclear. ~Some evidence of abnormalities in genes coding for the α3 and α4 isoforms of Type IV collagen.	~**Anti-GBM disease** ~Caused by: idiopathic, exposure to hydrocarbon solvents, influenza A2 virus, malaria, **Hodgkins disease**	~**Immune complex mediated** ~Caused by things like: SLE, post-strep GN, membranoproliferative GN, IgA nephropathy	~**Pauci-immune disease** ~No immune complexes or anti-GBM antibodies found by IF or EM ~90% positive for ANCA ~Often found as component of **systemic vasculitis** (like MP or Wegeners)
Clinical Presentation	Asymptomatic hematuria or proteinuria, often only picked up by routine physical.		Present with renal problems as well as symptoms of underlying disease	
LM	Normal	Glomerular crescent 		
IF	Negative	Bright linear IgG fluorescence 		Crescent shape seen with anti-fibrin IM…characteristic of all crescentic GNs. 
EM	Abnormally thin basement membrane 	No immune deposits seen. Severe glomerular damage.		
Prognosis	Progression to ESRD is uncommon but can occur	Good if diagnosed early – plasmapharesis and cytotoxic drugs can restore function		
Comments		~Associated with **HLA haplotype DR2** ~Uncommon in blacks ~May be associated with pulmonary BM attack = **Goodpastures**	Crescents are caused by proliferation of cells in Bowmans space with collapse of glomerulus. Get leakage of plasma proteins.	