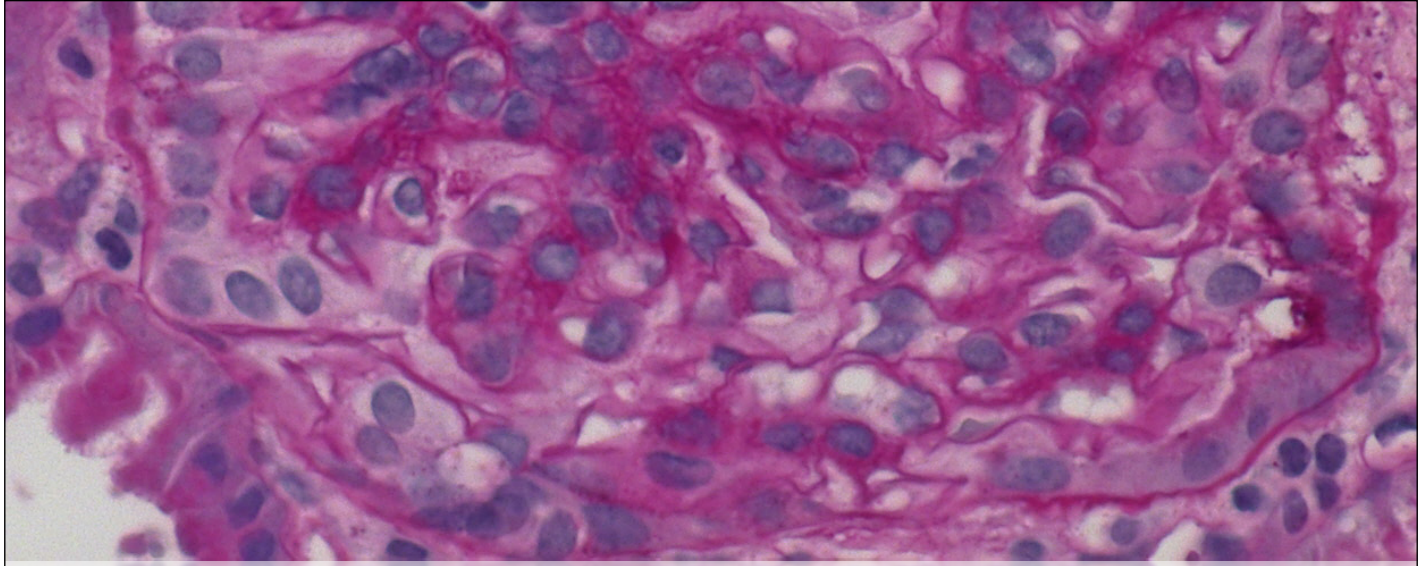
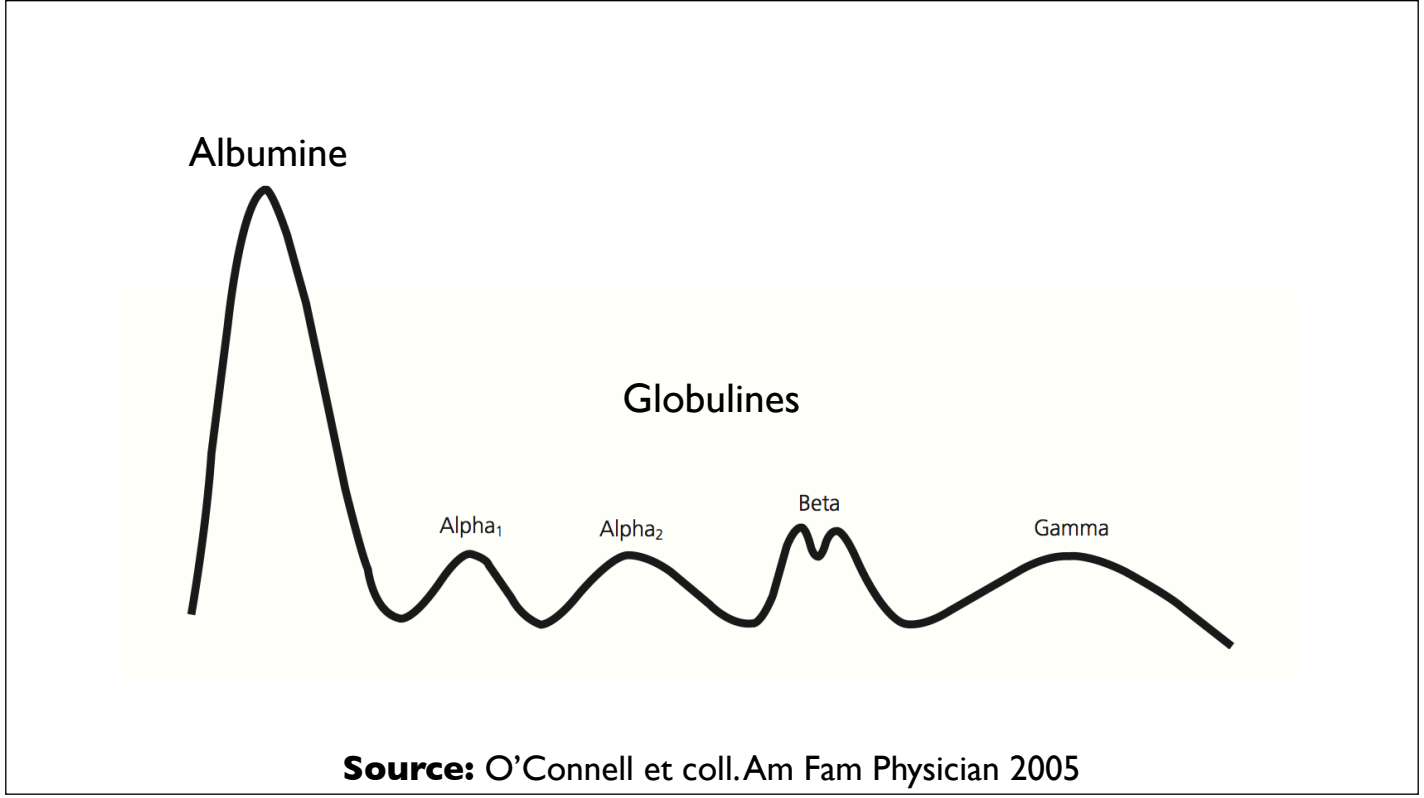


Protéinurie en bref

Dr Vincent Bourquin - service de néphrologie - http://nephroblog.org



Source: O'Connell et coll. Am Fam Physician 2005

ALBUMIN

The albumin band represents the largest protein component of human serum. The albumin level is decreased under circumstances in which there is less production of the protein by the liver or in which there is increased loss or degradation of this protein. Malnutrition, significant liver disease, renal loss (e.g., in nephrotic syndrome), hormone therapy, and pregnancy may account for a low albumin level. Burns also may result in a low albumin level. Levels of albumin are increased in patients with a relative reduction in serum water (e.g., dehydration).

ALPHA FRACTION

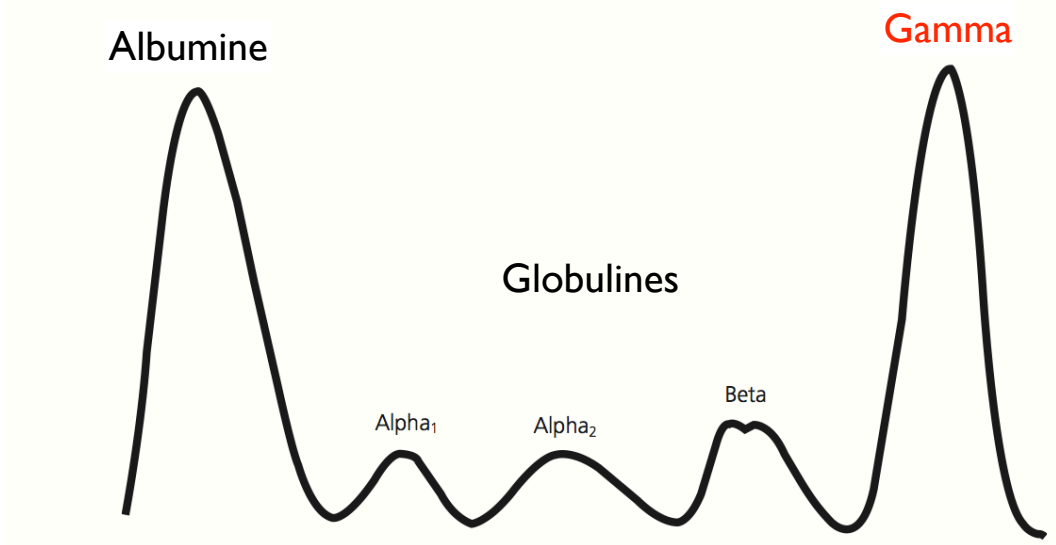
Moving toward the negative portion of the gel (i.e., the negative electrode), the next peaks involve the alpha1 and alpha2 components. The alpha1-protein fraction is com- prised of alpha1-antitrypsin, thyroid-binding globulin, and transcortin. Malignancy and acute inflammation (resulting from acute- phase reactants) can increase the alpha1- protein band. A decreased alpha1-protein band may occur because of alpha1-anti- trypsin deficiency or decreased production of the globulin as a result of liver disease. Ceruloplasmin, alpha2-macroglobulin, and haptoglobin contribute to the alpha2-protein band. The alpha2 component is increased as an acute-phase reactant.

BETA FRACTION

The beta fraction has two peaks labeled beta1 and beta2. Beta1 is composed mostly of transferrin, and beta2 contains beta-lipoprotein. IgA, IgM, and sometimes IgG, along with complement proteins, also can be iden- tified in the beta fraction.

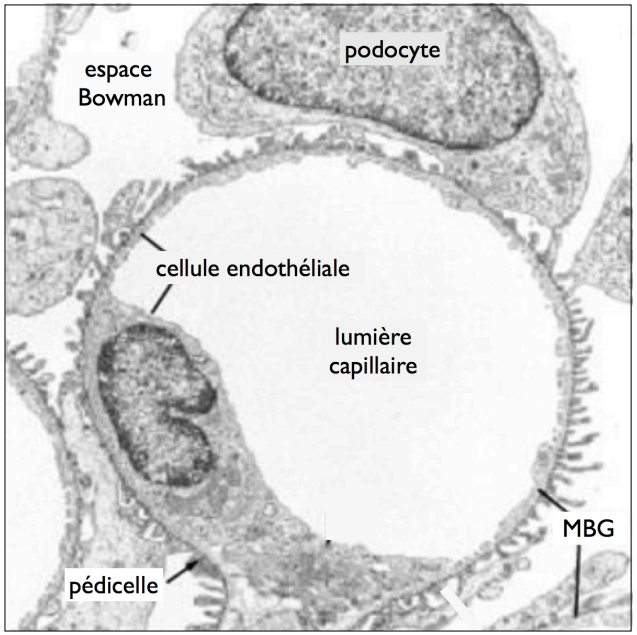
GAMMA FRACTION

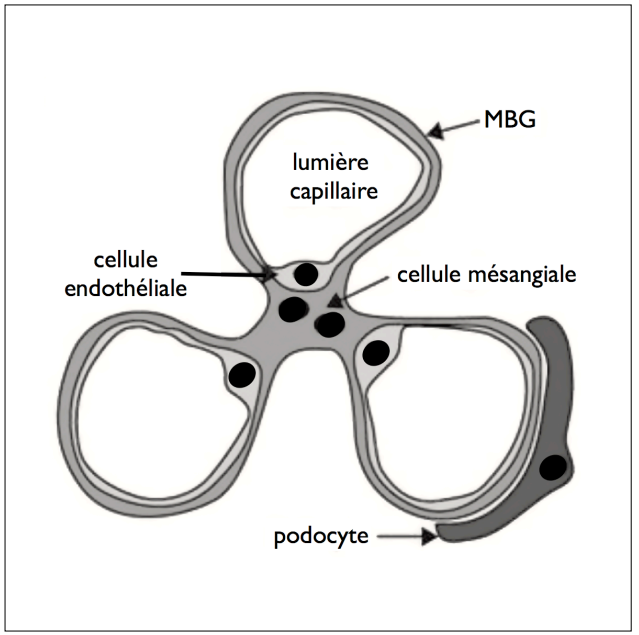
Much of the clinical interest is focused on the gamma region of the serum protein spectrum because immunoglobulins migrate to this region. It should be noted that immunoglobulins often can be found throughout the electrophoretic spectrum. C-reactive protein (CRP) is located in the area between the beta and gamma components.[1]

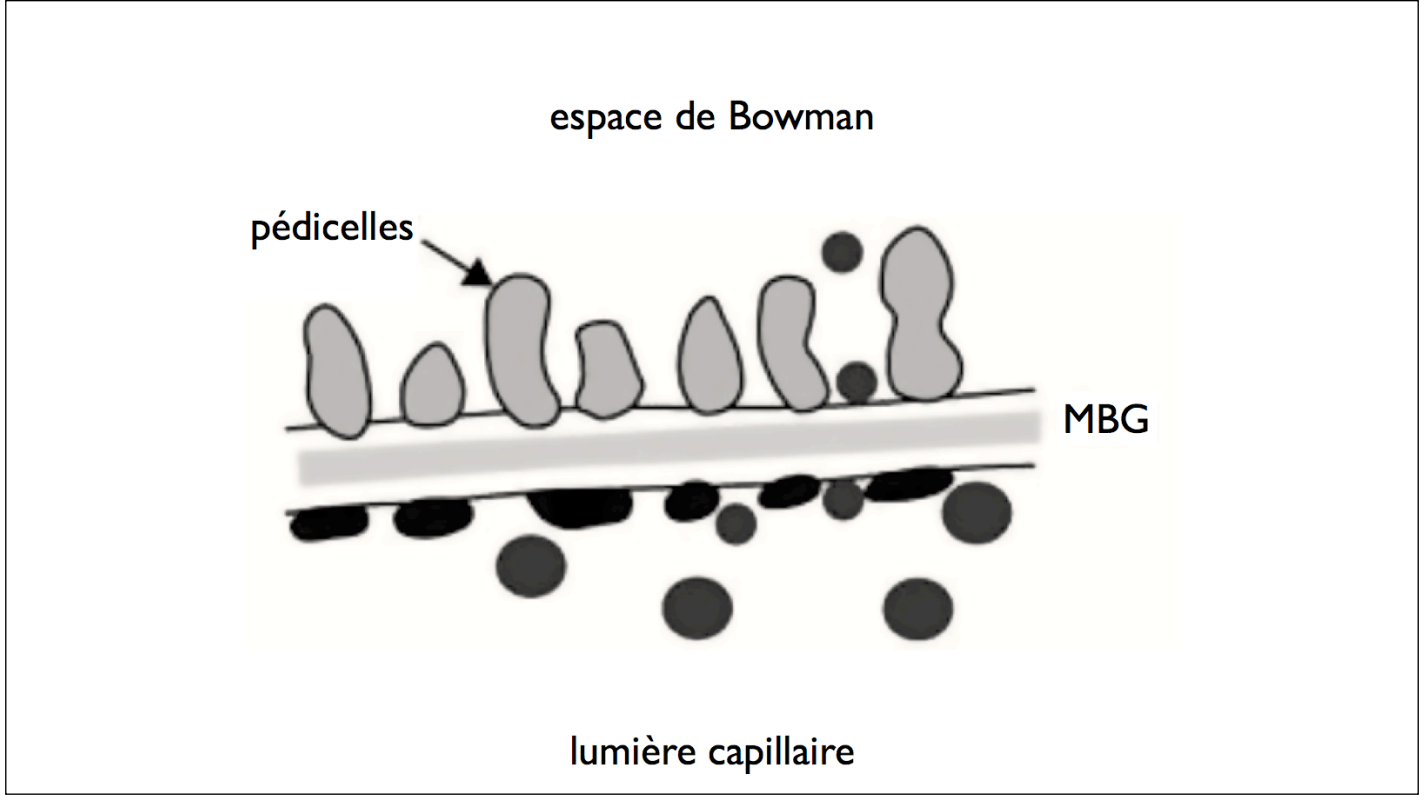


Source: O'Connell et coll. Am Fam Physician 2005

Protéine	Masse moléculaire relative kD	Sérum: Intervalle de référence mg/L	Urine: Intervalle de référence mg/L
alpha2-macroglobuline	250	M 1'200-2'700 F 1'400-3'200	<10 <10
Immunoglobuline G	150	8'000-17'000	<10
Transferrine	79.5	2'300-4'300	< 2.5
Albumine	**67**	**37'000-53'000**	**< 20**
alpha1-antitrypsine	54	1'400-2'300	< 3.5
alpha1-glycoprotéine acide	41	400-1'300	< 10
alpha1-microglobuline	30-33	25-100	< 12
Retinol-binding protein	21	30-60	< 0.5
Lysozyme	14	< 6	< 0.3
Cystatine C	13.3	0.63-1.63	
Beta2-microglobuline	13	1-3	< 0.3

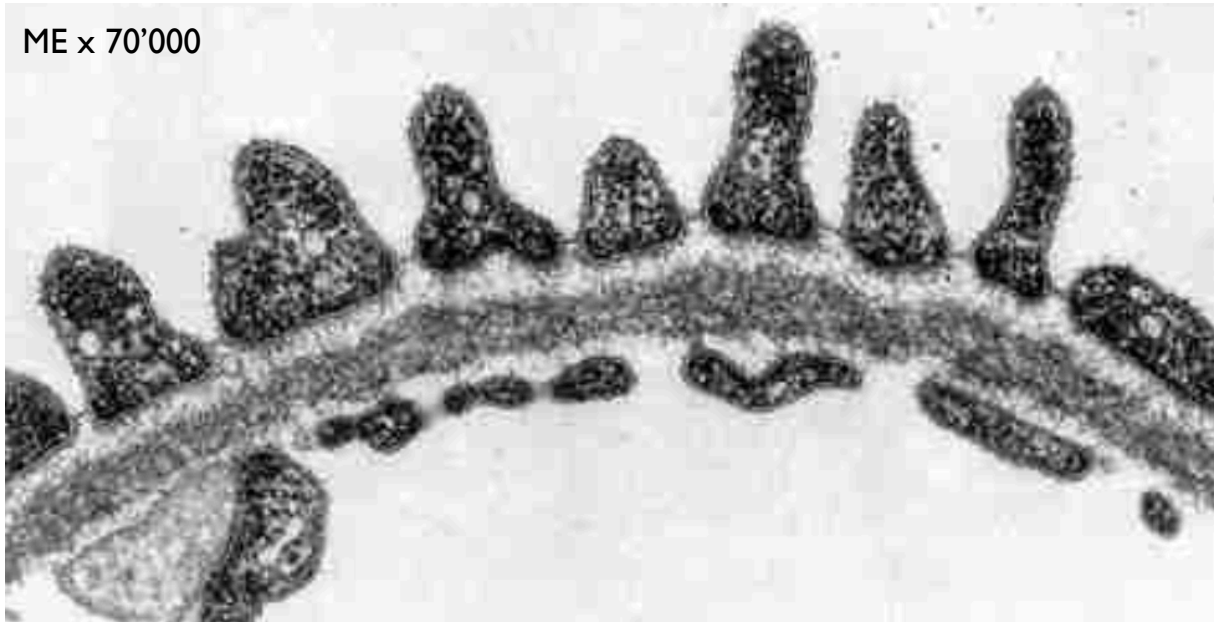

espace
Bowman
podocyte
cellule endothéliale
lumière
capillaire
MBG
pédicelle


MBG
lumière
capillaire
cellule
endothéliale
cellule mésangiale
podocyte



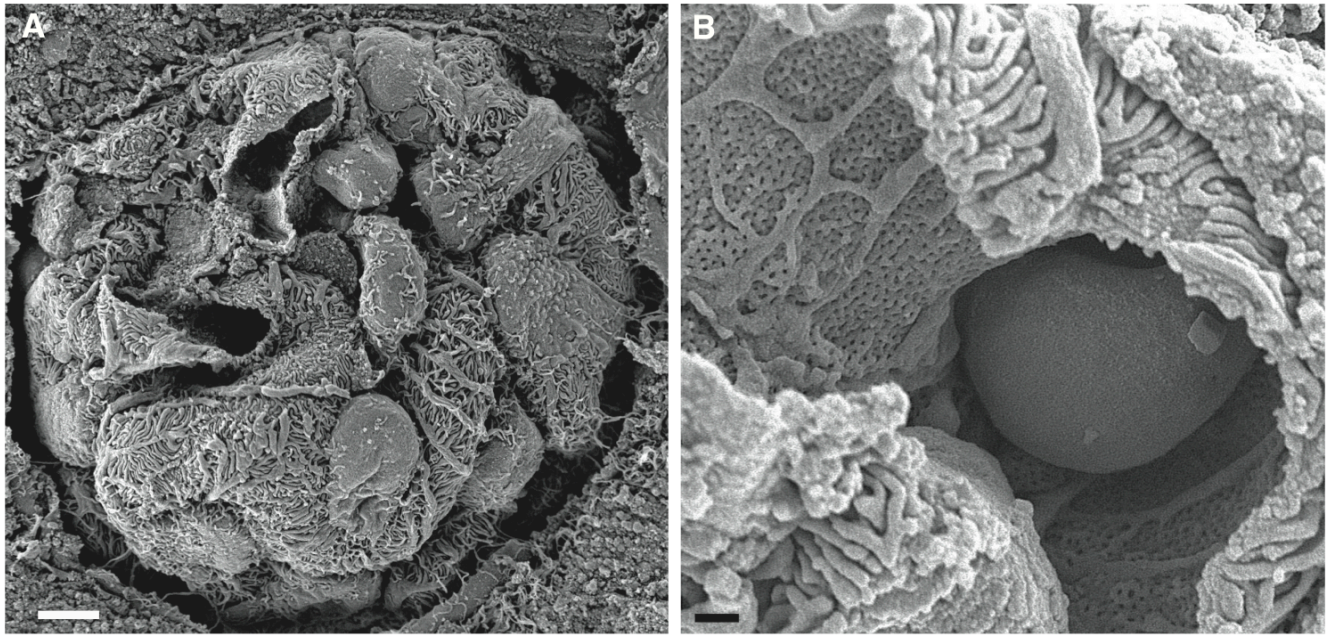
Barrière de filtration glomérulaire composée de ses trois couches:

- **L'endothélium capillaire glomérulaire largement fenestré** qui n'assure aucune restriction de taille.
- **La membrane basale glomérulaire**, assemblage complexe (maillage) de glycoprotéines. La subdivision classique en 3 couches : lamina rara, interna, externa, résulte d'artefacts de microscopie électronique. Cette matrice est perforée de pores d'environ 40 nanomètres de rayon. La membrane basale assure environ 30 % de la restriction de passage de taille. Sa composition chimique riche en glycoprotéines anioniques assure une partie importante de la restriction au passage de charge.
- **Les cellules épithéliales ou podocytes** recouvrent le versant externe de la membrane basale glomérulaire. Ces cellules fortement différenciées étendent des pieds ou pédicelles, eux-mêmes à l'origine d'interdigitations extrêmement fines, entrecroisées entre-elles et qui recouvrent complètement l'ensemble de la membrane basale glomérulaire. Chaque interdigitation est recouverte d'un diaphragme de fente, lui-même perforé par des pores rectangulaires d'environ 60 x 40 nanomètres. Cette couche épithéliale assure l'ensemble de la restriction au passage de taille empêchant notamment le passage de protéines de poids supérieur à 60 kilodaltons (KD). Les pédicelles sont également recouverts de glycoprotéines polyanioniques qui assurent une restriction au passage de charge. Outre leur rôle dans l'hindrance aux macromolécules, les podocytes assurent une fonction très importante de maintien de l'architecture normale des floculus.

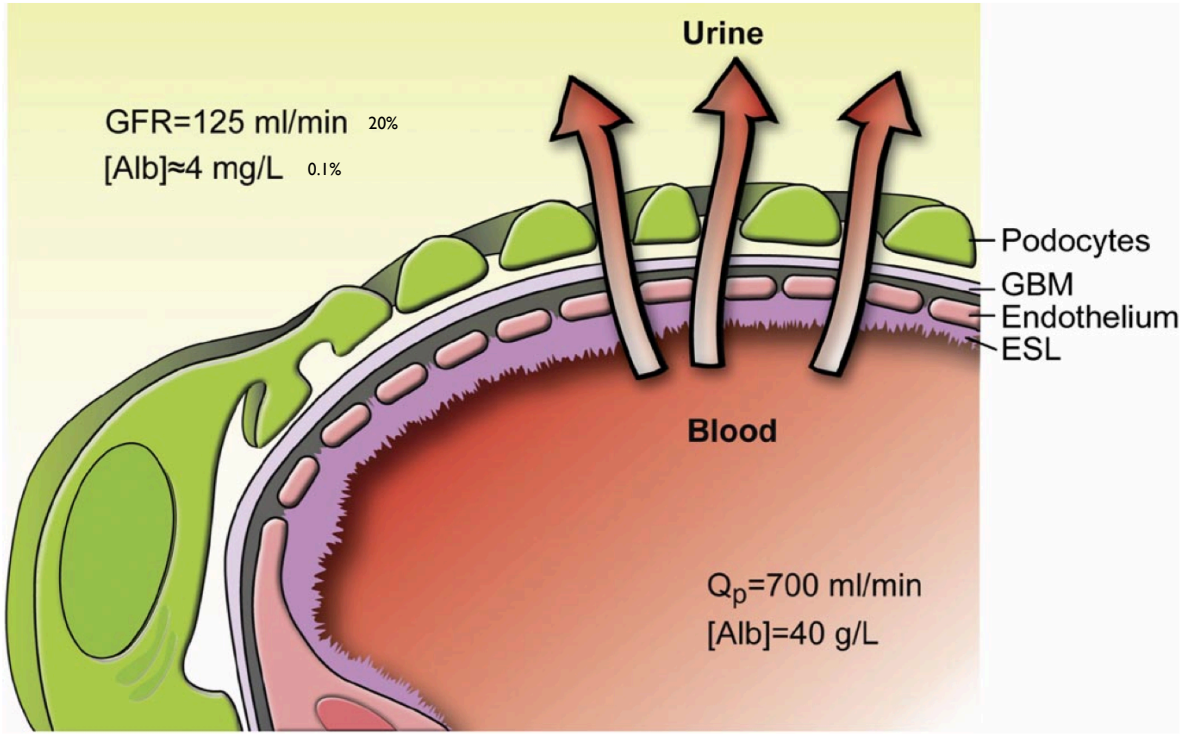


Source: http:nephrohus.org

Barrière de filtration capillaire glomérulaire composée de ses trois couches : l'endothélium fenestré, la membrane basale glomérulaire, et les digitations des cellules podocytaires séparées par des fentes de filtrations, elles-mêmes recouvertes par les diaphragmes de fentes.

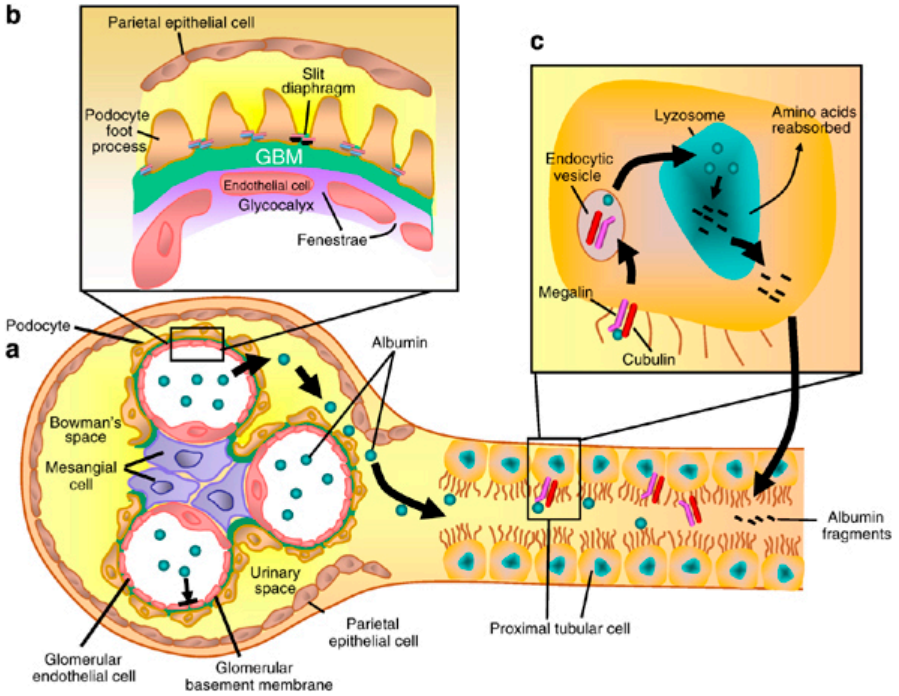


Source: Haraldsson et coll. Physiol Rev 2008



Source: Haraldsson et coll. Physiol Rev 2008

Schematic drawing of the glomerular barrier. Podo, podo- cytes; GBM, glomerular basement membrane; Endo, fenestrated endo- thelial cells; ESL, endothelial cell surface layer (often referred to as the glycocalyx). Arrows indicate the filtration of plasma fluid across the glomerular barrier, forming primary urine at a glomerular filtration rate (GFR) of 125 ml/min in humans. The plasma flow rate (Q_p) is close to 700 ml/min, giving a filtration fraction of 20%. Also shown are the concentrations of albumin in serum (40 g/l) and estimated concentration in primary urine 4 mg/l (i.e., 0.1% of that in plasma). The sieving coefficient of albumin across the glomerular barrier in humans is estimated to be 10% of that in rodents.



Source: Jefferson et coll. Kidney International 2008

Normal renal handling of albumin. (a) Normal glomerulus and proximal tubule. The individual cells and constituents of the glomerulus and proximal tubules are shown. Albumin (represented by green spheres) normally remains within the capillaries of the glomerular tuft, and does not escape into the urinary (Bowman's) space. (b) Glomerular filtration barrier. This barrier comprises the innermost glomerular endothelial cells, GBM, and outermost podocytes; and serves to serially limit albumin escaping from the capillary loops. Fenestrae within specialized endothelial cells are covered by a negatively charged glycocalyx. Podocytes attach to the outermost aspect of the GBM by foot processes, between which are proteins comprising the size barrier slit diaphragm. (c) Proximal tubule. The albumin that is physiologically filtered at the level of glomerulus into the urinary space is taken up by the megalin/cubulin receptor lining the brush border of proximal tubular cells. Albumin is internalized by vesicles, and upon lysozyme action, the resultant fragments are either reabsorbed or secreted back into the tubular lumen as albumin fragments.

La capacité d'une substance à traverser une membrane est exprimée par le **coefficient de partage**

Source: **Bourquin et coll. Néphrol ther 2009**

original article http://www.kidney-international.org

see commentary on page 479

The normal kidney filters nephrotic levels of albumin retrieved by proximal tubule cells: Retrieval is disrupted in nephrotic states

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The origin of albuminuria remains controversial owing to difficulties in quantifying the actual amount of albumin filtered by the kidney. Here we use fluorescently labeled albumin, together with the powerful technique of intravital 2-photon microscopy to show that renal albumin filtration in non-proteinuric rats is ~50 times greater than previously measured and is followed by rapid endocytosis into proximal tubule cells (PTCs). The endocytosed albumin appears to undergo transcytosis in large vesicles (500 nm in diameter), identified by immunogold staining of endogenous albumin by electron microscopy, to the basolateral membrane where the albumin is disgorged back to the peritubular blood supply. In nephrotic rats, the rate of uptake of albumin by the proximal tubule (PT) is decreased. This is consistent with reduced expression of clathrin, megalin, and vacuolar H^+-ATPase A subunit, proteins that are critical components of the PT endocytotic machinery. These findings strongly support the paradigm-shifting concept that the glomerular filter normally leaks albumin at nephrotic levels. Albuminuria does not occur as this filtered albumin load is avidly bound and retrieved by PTCs. Dysfunction of this retrieval pathway leads to albuminuria. Thus, restoration of the defective endocytotic and processing function of PT epithelial cells might represent an effective strategy to limit urinary albumin loss, at least in some types of nephrotic syndrome.

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KEYWORDS: albuminuria; proximal tubule; glomerular filtration barrier; nephropathy; pathophysiology of renal disease and progression

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The major barrier to the passage of albumin into the urine has long been thought to reside in the glomerular capillary wall, partly on the basis of massive proteinuria associated with genetic diseases of the glomerular epithelial cell (podocyte)[1] and the very low concentrations of albumin measured in the filtered fluid by micropuncture methods[2] in the early tubular lumen. The restrictive permselectivity of the glomerular filter to albumin has been attributed to exclusion of albumin based on its size and in particular its electrical charge. However, recent studies have shown that electrical repulsion of anionic polysaccharides by the glomerular capillary wall could not account for the apparent low permselectivity of albumin.[3–6]

In order to directly measure albumin filtration, we have used the minimally invasive, non-toxic technique of intravital 2-photon microscopy which allows a four-dimensional (volume and time) examination of physiological processes in the kidney.[7] This technique has previously been used to demonstrate subcellular trafficking and transcytosis in proximal tubular cells (PTCs)[10] as well as determining the glomerular sieving of different molecular weight dextrans.[8] Here we evaluate the second to second dynamics of albumin filtration and cellular uptake *in vivo* in normal and nephrotic rats using fluorescent-labeled albumin. Electron microscopy was used to identify intracellular transport of endogenous albumin. Further, we use immunocytochemistry to determine changes in critical components of the proximal tubule (PT) endocytic machinery including expression of the albumin receptor megalin, vacuolar H^+-ATPase A subunit (v-ATPase) and clathrin in nephrotic rats.

These data provide fundamental new insights into the extent of albumin filtration by the kidney and the critical role of post-glomerular albumin reabsorption in the prevention of nephrotic levels of albuminuria.

BRIEF COMMUNICATION www.jasn.org

Impaired Tubular Uptake Explains Albuminuria in Early Diabetic Nephropathy

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*Program in Membrane Biology, Center for Systems Biology, Department of Medicine, Massachusetts General Hospital and Harvard Medical School, Boston, Massachusetts; †Department of Medicine, Division of Nephrology, Indiana Center for Biological Microscopy, Indiana University Medical Center, and ‡Roudebush Veterans Affairs Medical Center, Indianapolis, Indiana; §Exosome Diagnostics Inc., New York, New York; and ‖Department of Biochemistry and Molecular Biology, Monash University, Victoria, Australia

BRIEF COMMUNICATION

ABSTRACT

Understanding the pathogenesis of albuminuria in diabetic nephropathy is important to improve methods for early diagnosis and treatment. In this study, we addressed whether albuminuria in diabetes results from altered glomerular filtration and/or altered processing of filtered albumin by the proximal tubule. Type 1 diabetic Munich Wistar rats developed albuminuria after 12 wk of diabetes. Intravital two-photon microscopy revealed similar glomerular permeability in the diabetic and control animals, assessed using both albumin-Alexa568 and 69-kD FITC-dextran; however, diabetic animals demonstrated significantly less filtered fluorescent albumin in renal proximal tubule (PT) cells compared with control animals. We also observed increased albumin-derived urinary peptide excretion in diabetic animals, and hyperglycemia modulated this peptideuria. In conclusion, in the early stages of diabetic nephropathy, the PT plays a major role in the development of albuminuria, which may be preceded by peptideuria.

J Am Soc Nephrol **20:** 489–494, 2009. doi: 10.1681/ASN.2008050503

There has been much focus on the kidney filter as the primary factor for the manifestation of albuminuria in kidney disease, including diabetic nephropathy.[1,2] Many studies have emphasized the importance of various structural elements of the glomerular capillary wall (GCW), including podocytes, in governing albuminuria in diabetes[3–5]; however, previous studies were unable to make direct measurements of GCW albumin permeability. There is now a debate as to whether the albuminuria that develops in diabetes and other renal diseases is of glomerular or PT origin.[5] Here, we use intravital two-photon microscopy of infused albumin-Alexa568 and 69-kD FITC-dextran to examine directly and for the first time the *in vivo* processes of albumin filtration and proximal reabsorption in an animal model of diabetes.

We induced type 1 diabetes in rats using streptozotocin (STZ), a drug that targets and destroys β cells of the pancreas, producing a widely used and recognized model of insulin-dependent diabetes. Analysis of physiologic parameters after 12 wk of diabetes demonstrated significantly decreased body weight and increases in blood glucose, kidney weight, and osmotic diuresis accompanied by increased water intake, *versus* age-matched control rats (Supplemental Table 1). Analysis of urinary albumin excretion demonstrated significantly increased intact albumin excretion in the diabetic *versus* control groups ($P < 0.05$; Figure 1), consistent with the induction of diabetic nephropathy.

Intravital two-photon microscopy was used to determine the origin of the albuminuria. Anesthetized rats were administered an intravenous injection of rat serum albumin conjugated to Alexa568 (albumin-Alexa568; red).[6] After the injection, fluorescent albumin was immediately filtered across glomerular capillaries, entered the urinary space, and avidly bound to the base of the PT cell brush border in both control and diabetic animals (Supplemental Movies 1 and 2, respectively). Superficial glomeruli and associated PTs were imaged for the first 3 min after albumin-Alexa568 injection, and short movies were also recorded at 12 and 24 min after injection for use in determining the albumin glomerular-sieving coefficient (GSC). We calculated GSC as the ratio of albumin-Alexa568 (or FITC-dextran in some experiments) in the glo-

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The Proteinuria Controversy

One of the largest controversies in the field of proteinuria/nephrotic syndrome research derives from a 2007 Kidney International paper published by Russo et al. Briefly stated, the authors suggest the paradigm-shifting idea that the glomerulus filters massive amounts of albumin, and that nephrotic syndrome is a defect in tubular reabsorption of albumin. This flies in the face of decades worth of research on proteinuria, which based on a combination of micropuncture and other physiologic experiments in mice and man has led to the conventional model in which albumin is prevented from entering Bowman's space by the charge-selectivity of the glomerular filtration barrier, and nephrotic syndrome results from a breakdown of this barrier. What are we to make of such a debate? Who is right and who is wrong? The following is a (hopefully unbiased) list of the pros and cons of each side of the argument.

Pro: Normal glomeruli filter nephrotic levels of albumin.

The major piece of data in support of this hypothesis comes from the Russo et al paper in which the authors use a relatively new imaging technique, intravital 2-photon microscopy, which enables in vivo imaging of the kidney using injected fluorescent compounds at a resolution previously unachievable. Essentially, the crucial experiment involved injection of a fluorescently-labeled albumin into the vasculature of rats; by quantifying the degree of fluorescence in the plasma compared to Bowman's space, the authors were able to calculate a "sieving coefficient" for albumin. The sieving coefficient they arrived at was about .02--which is orders of magnitude higher than the previous value obtained via the micropuncture method, about .0006. This implies that nephrotic levels of proteinuria are being filtered from normal glomeruli, and the authors postulate a proximal tubular-based mechanism of rapid albumin reclamation. The nature of such a mechanism is unclear, but the authors suggest that vesicles of intact albumin are transcytosed through proximal tubular cells, providing evidence of this by showing an electron micrograph of endogenous albumin within proximal tubular cells. Finally, in a 2008 JASN article by Dr. Wayne Comper, the author sites various methodologic problems with some of the initial experiments used to demonstrate the "charge selectivity" model of the glomerulus. A recent follow-up paper in JASN by Russo et al furthermore suggests that it is an impaired tubular uptake of filtered albumin which accounts for the changes seen in early diabetic nephropathy.

Con: The case against the "tubular proteinuria" model.

Not surprisingly, this newer model has been met with much resistance. In an article by Christensen et al forcefully entitled, "Controversies in nephrology: renal albumin handling, facts, and artifacts!", the authors describe their opposition to the idea that the glomerulus exhibits such large permeability to albumin. First, they point out several methodologic concerns with the technique of 2-photon microscopy, suggesting that the low fluorescent signal they observed is subject to misinterpretation; perhaps some of the "filtered albumin" seen in Bowman's space is fluorescent bleed-through from nearby blood vessels. The authors also point out a very logical question: if massive amounts of protein are retrieved by proximal tubular cells, why haven't we seen evidence of this throughout decades of research in this field? Many veterans of the field have simply not observed proximal tubular cells chock-full of vesicles containing endogenous albumin, and suggest that the published electron micrographs by Russo et al could be fixation artifact. Furthermore, the authors point out that mice lacking megalin function--thought to be a major player in protein uptake in proximal tubular cells--show only a mild degree of proteinuria, not nearly enough to be consistent with the massive amounts of albumin purported to be filtered by the glomerulus. Finally, virtually all of the mutations identified in patients with congenital nephrotic syndrome target genes known to be important in podocyte function: nephrin, podocin, alpha-actinin 4, etc, which would seemingly point to the podocyte, rather than the proximal tubular cell, as the primary player in regulating proteinuria.

So: Who's right and who's wrong? I don't think we know yet, but for now my money is on the conventional explanation--in general, it seems that the "tubular etiology of nephrotic syndrome" is supported predominantly by a new technique, the limitations of which are not yet fully known, and in order to lend it further credence, alternative techniques which support this theory would be necessary. If it does turn out to be true, however, this would really represent a major paradigm shift in nephrology.

Physiologiquement, il existe une **protéinurie de faible abondance** (40-80 mg/j, max 150 mg/j)

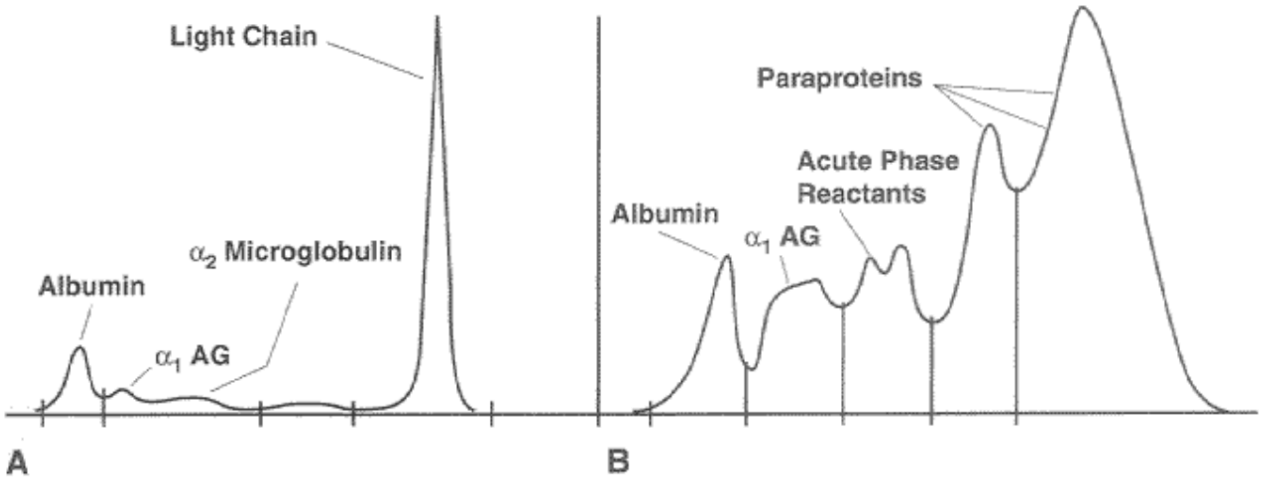
Source: http:nephrohus.org

Physiologiquement, il existe une protéinurie de très faible abondance. Chez les sujets normaux, cette proténurie physiologique est d'environ 40-80 mg/j avec une valeur supérieure haute de 150 mg/24 h (au-dessus de cette valeur la protéinurie est considérée comme pathologique). Cette protéinurie est composée pour moins de 10 mg/j d'albumine vraie, pour 30 à 50 mg/j de mucoprotéine de Tamm-Horsfall (protéine synthétisée et sécrétée spécifiquement dans la branche ascendante large de l'anse de Henle et ajoutée à l'urine après la filtration glomérulaire ; cette protéine constitue aussi la matrice de la plupart de cylindres urinaires) et enfin moins de 20 mg/j d'immunoglobulines et de fragments d'immunoglobulines et d'autres protéines de petit poids moléculaire.

Les mécanismes physiopathologiques de la protéinurie peuvent être classifiés comme étant **glomérulaires**, **tubulaires** ou de **surcharge**.

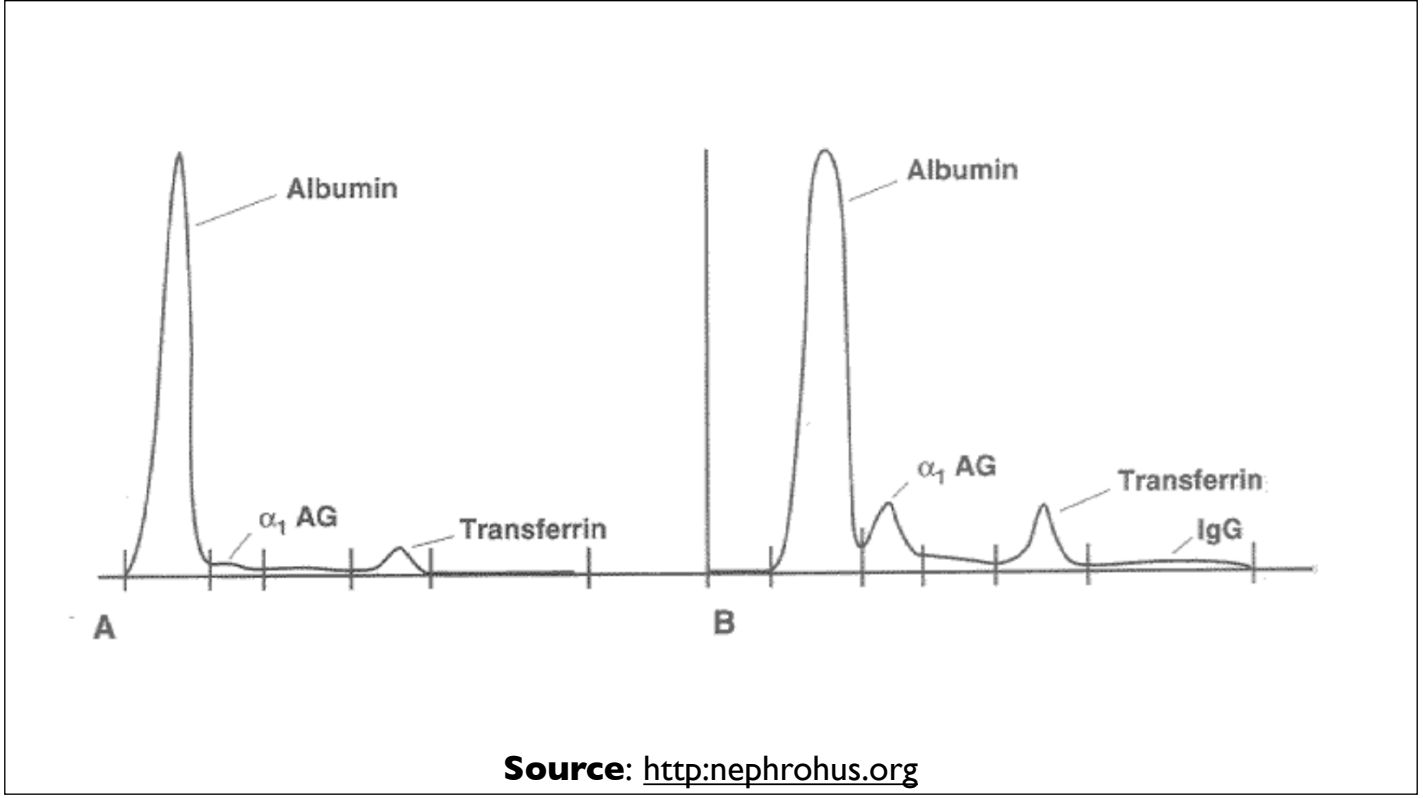
Source: Bourquin et coll Forum Med Suisse 2007

Dans un certain nombre de situations pathologiques, la BFG est altérée et va laisser passer dans l'urine des quantités importantes de macro-molécules dont notamment des protéines. Les mécanismes physiopathologiques de la protéinurie peuvent être classifiés comme étant glomérulaires, tubulaires ou de surcharge. L'atteinte glomérulaire est la cause la plus fré- quente de protéinurie pathologique. Un certain nombre d'anomalies glomérulaires modifie la perméabilité de la MBG, causant une perte urinaire d'albumine et d'immunoglobulines. Celle-ci provoque d'importantes pertes protéiques; une excrétion urinaire de plus de 2 g par jour est généralement le résultat d'une maladie du glomérule. L'atteinte tubulaire se produit quand une maladie tubulo-interstitielle empêche le tubule proximal de réabsorber normalement les protéines de bas PM. Il s'agit habituellement d'une protéinurie de faible importance, inférieure à 1 g par jour. Dans la protéinurie de surcharge, il s'agit de protéines de bas PM entre 20 et 30 kDa, libérées en quantité massive dans la circulation sanguine, librement filtrées par le glomérule mais insuffisamment réabsorbées, car le processus est saturé au niveau du tubule proximal. Les principaux exemples sont les chaînes légères d'immunoglobulines kappa ou lambda au cours des myélomes à chaînes légères et la myoglobine au cours des rhabdomyolyses.



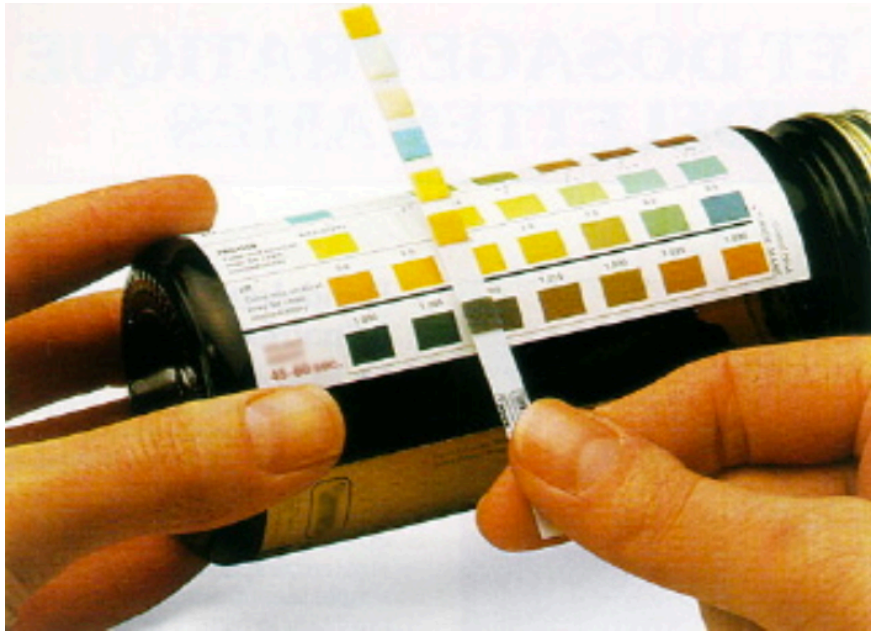
Source: http:nephrohus.org

Electrophorèse des protides urinaires : Protéinurie de surcharge (A) myélome à chaine légère, pic de chaine légère monoclonale constituant la majorité de la protéinurie. (B) SIDA avec syndrome inflammatoire responsable d'une protéinurie modérée constituée de protéines de la réponse inflammatoire, aspect polyclonal.



Source: http:nephrohus.org

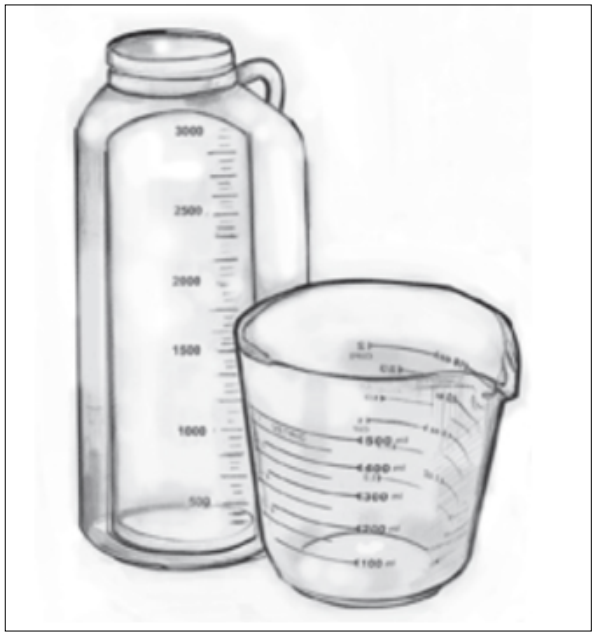
Electrophorèse des protides urinaires au cours de protéinuries glomérulaires : (A) protéinurie sélective constituée presque exclusivement d'albumine au cours d'un syndrome néphrotique à LGM ; (B) protéinurie non sélective avec présence de globulines notamment d'IgG au cours d'une glomérulonéphrite extramembraneuse.



Source: http:nephrohus.org

Le dépistage d'une protéinurie repose sur la bandelette réactive. Le principe de la bandelette est celui du virage d'un indicateur coloré (bleu de bromophénol) à pH constant. L'échelle des couleurs a un intérêt limité car elle ne permet qu'une estimation semi-quantitative de la concentration d'albumine dans un échantillon. Ces bandelettes réactives sont très sensibles à la présence d'albumine (elles détectent une concentration d'albumine de l'ordre de 50 mg/l). Par contre elles ne détectent pas les autres protéines, notamment les immunoglobulines et les chaînes légères d'immunoglobuline. L'évaluation est semi-quantitative cotée en croix selon l'intensité de la réponse.

Certains faux positifs doivent être connus en cas de bandelettes trop anciennes, d'urines alcalines (présence de germes uréase+) ou de la présence sur le récipient de recueil de détergents ou d'ammonium quaternaire. Les bandelettes doivent donc être conservées dans un flacon hermétique, à l'abri de la lumière et de la chaleur, à pH très acide (pH 3). Il faut aussi faire la recherche sur des urines fraîches en l'absence de détergents (ammonium quaternaire).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Demande n°
Prélèvement du

Copie(s)

Renseignements cliniques (RC)

Diurèse	2900	ml		24/09/12	2900		
Durée de récolte	24	h			24		

Chimie clinique

Marc Bagnoud 022 719 75 83 et Olivier Seffert 022 807 12 43

	Dosage		Normales		Antériorité 1	Antériorité 2
Urines de 24 h						
Créatinine	4.4	mmol/L		24/09/12 09:30	5.4	
Créatinine	12.9	mmol/d	7.0-18.0	24/09/12	15.6	
Protéines totales urinaires 24 h	34	mg/L		24/09/12 09:30	43	
Protéines totales urinaires 24 h	99	mg/24h	<150	24/09/12	125	
Micro-albumine	<3.0	mg/L		24/09/12 09:30	<3.0	
Micro-albumine	Calcul non réalisable			24/09/12	(*)	

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Demande n°
Prélèvement du

Chimie clinique

Marc Bagnoud 022 719 75 83 et Olivier Seffert 022 807 12 43

	Dosage		Normales		Antériorité 1	Antériorité 2
Bilan urinaire						
Créatinine urinaire	9.4	mmol/L	3.4-22.9	31/07/12 08:30	8.2	
* **Protéines totales urinaires**	**946**	mg/L	<150		**2028**	
* **Rapport protéines / créatinine**	**100.28**	mg/mmol cre	<11.20		**246.84**	
* **Micro-albumine urinaire**	**39.9**	mg/L	<20.0		**293.7**	
* **Rapport albumine / créatinine**	**4.23**	mg/mmol cre	<2.27		**35.75**	
Rapport albumine / protéines (urines)	0.04	g/g	<0.6		0.14	

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Demande n°
Prélèvement du

Renseignements cliniques (RC)

Diurèse	1800	ml
Durée de récolte	24	h

Chimie clinique

Marc Bagnoud 022 719 75 83 et Olivier Seffert 022 807 12 43

	Dosage		Normales	Antériorité 1	Antériorité 2
Urines de 24 h					
Créatinine	5.8	mmol/L			
Créatinine	10.4	mmol/d	7.0-18.0		
Protéines totales urinaires 24 h	2813	mg/L			
• **Protéines totales urinaires 24 h**	**5063**	mg/24h	<150		
Micro-albumine	2407.9	mg/L			
• **Micro-albumine**	**4334**	mg/24h	<30		


merci de votre attention
Dr Vincent Bourquin - service de néphrologie - http://nephroblog.org