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**DEPARTMENT:** ANATOMY

**COURSE:**  ANA 204

**QUESTION**: **Critically examine the renal function of desert dwellers and the anatomical basis of their unique adaptation.**

The anatomical structures for urine concentration found in animals living in desert or arid environments, although not all occurring in one particular animal, are wide medullae, long loops of Henle, long proximal tubules, long collecting tubules, small renal corpuscles, extension of the renal pelvis, well developed elongated papillae, occurrence of giant vascular bundles, specialized ultrastructure of Henle's loops, epithelial changes in the collecting tubule, zonation of the vasa recta and peculiarity of the arterial supply to the kidney. The renal renin content is moderately high in these species. The renin-angiotensin-aldosterone system is very active, retaining Na+ with water. The urine is concentrated at the expense of other electrolytes. Both the renal blood and urinary flow rates are lower than in species with access to unlimited water supply. The juxtaglomerular apparatus components are topographically intimate for effective tubuloglomerular autoregulation of renal blood flow.

**QUESTION: Write extensively on the clinical importance of the glomerular filtration barrier.**

**The glomerular filtration barrier is a main component for the filtration of the plasma and formation of primary urine. It is composed of specialized cells and noncellular structures that, together, can avoid the loss of important plasma components but permit the passage of water and undesirable molecules**

The [glomerular filtration](https://www.sciencedirect.com/topics/biochemistry-genetics-and-molecular-biology/glomerulus-filtration) barrier determines the composition of the plasma ultrafiltrate. It restricts the filtration of molecules primarily on the basis of size. In general, molecules with a radius smaller than 20 Å are Filtered freely, molecules larger than 42 Å are not filtered, and molecules between 20 and 42 Å are filtered to various degrees.The glomerular filtration barrier functions as a highly organized, semipermeable membrane preventing the passage of the majority of proteins into the urine. This barrier is composed of the glomerular basement membrane, the podocyte, and the slit diaphragm between the podocytes.

Damage to the glomerulus by disease can allow passage through the glomerular filtration barrier of red blood cells, white blood cells, platelets, and blood proteins such as albumin and globulin. Underlying causes for glomerular injury can be inflammatory, toxic or metabolic.[[8]](https://en.wikipedia.org/wiki/Glomerulus_%28kidney%29#cite_note-8) These can be seen in the urine ([urinalysis](https://en.wikipedia.org/wiki/Urinalysis)) on microscopic and chemical (dipstick) examination. Examples are [diabetic kidney disease](https://en.wikipedia.org/wiki/Diabetic_kidney_disease), [glomerulonephritis](https://en.wikipedia.org/wiki/Glomerulonephritis), and [IgA nephropathy](https://en.wikipedia.org/wiki/IgA_nephropathy).

Due to the connection between the glomerulus and the GFR, the GFR is of clinical significance when suspecting a kidney disease, or when following up a case with known kidney disease, or when risking a development of renal damage such as beginning medications with known [nephrotoxicity](https://en.wikipedia.org/wiki/Nephrotoxicity).

Clinical Applications: Nephrotic Syndrome

The nephrotic syndrome is a set of symptoms that include the following:

* + protein in the urine;
	+ low blood protein levels;
	+ swelling or edema.

It may also include elevated levels of serum lipids, anemia, and vitamin D deficiency, all because of loss of plasma proteins into the urine. This can have multiple causes, but all involve defects in the glomerular barrier to proteins so that excess proteins are filtered and thereby excreted in the final urine. The three barriers were discussed in the text: the fenestrated endothelial cell layer, the GBM, and the podocyte and slit [diaphragm](https://www.sciencedirect.com/topics/engineering/diaphragms).

Nephrotic syndrome can be primary or secondary. Primary causes are described by their histological changes: minimal change disease, focal segmented glomerulosclerosis, and membranous nephropathy. Secondary causes are described by their underlying cause, which include diabetes mellitus, sarcoidosis, hepatitis B, hepatitis C, bacterial infections, parasitic infections, and more.

All of the diseases are characterized by protein in the urine, at least 3.5 g per 24 h. The loss of protein can cause hypoalbuminemia, with resulting edema that may show as puffiness around the eyes, pitting edema in the legs, and pleural effusion. Loss of proteins stimulates liver synthesis, including lipoproteins. Because lipoprotein lipase levels fall, lipoprotein levels increase. Loss of vitamin D binding protein can lead to vitamin D deficiency diseases, with calcium malabsorption and bone disease.

Mutations of nephrin, a protein of the filtration slit, cause nephrotic syndrome. Mutations of podocin also cause nephrotic syndrome that is insensitive to steroid treatment. Podocin is an integral protein of the podocyte cell membrane that segregates into [lipid rafts](https://www.sciencedirect.com/topics/engineering/lipid-raft) and is required to recruit nephrin into those rafts. Current thought is that podocin and nephrin form a signaling complex that activates protein kinases involved in glomerular structural integrity. These mutations cause minimal change diseases in which structural changes are evident only at the [electron microscope](https://www.sciencedirect.com/topics/engineering/electron-microscope) level and not at the histological level. Until recently, these were part of the set of nephrotic syndrome called idiopathic nephrotic syndrome.

Membranous glomerulonephritis is one of the more common causes of nephrotic syndrome in adults. It is an inflammatory disease, believed to be caused by binding of antibodies to antigens in the GBM that triggers the formation of a [membrane attack complex](https://www.sciencedirect.com/topics/engineering/membrane-attack-complex) from complement (see Chapter 5.3). This triggers release of proteases and [oxidants](https://www.sciencedirect.com/topics/engineering/oxidant) that damage the [capillary walls](https://www.sciencedirect.com/topics/engineering/capillary-wall), causing them to become leaky. Histology reveals thickened basement membranes.

Treatment depends on etiology. For all nephrotic syndromes, monitoring and maintaining normal fluid levels and distribution among the body compartments are the goal. This could include restriction of fluid intake, restriction of salt intake, regular monitoring of blood pressure and urine output, and the use of diuretics. Inflammatory causes of nephrotic syndrome are treated with immunosuppressants such as prednisolone and dietary modificaton.

REFRENCES:

<https://www.sciencedirect.com/topics/biochemistry-genetics-and-molecular-biology/glomerular-filtration-barrier>

<https://pubmed.ncbi.nlm.nih.gov/3051651/>