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DEPARTMENT: ANATOMY

MATRIC NUMBER: 18/MHS03/006

COURSE CODE: ANA 204

COURSE TITLE: HISTOLOGY OF SYSTEMS

1. Critically examine the renal function of desert dwellers and the anatomical basis of their unique adaptation

2. Write extensively on the clinical importance of the glomerular filtration barrier.

RENAL FUNCTION IN DESERT ANIMALS

Desert dwellers, also called xerocoles, live in dry, sandy and extremely hot places which makes their renal function different compared to animals living where there is adequate water and sunlight. Examples of desert dwellers are camels, meerkats etc. To excrete [nitrogenous waste products](https://en.wikipedia.org/wiki/Nitrogenous_waste), mammals (and most amphibians) excrete [urea](https://en.wikipedia.org/wiki/Urea#Physiology) diluted in water while such xerocoles have adapted to make their urine as concentrated as possible (i.e. use the least amount of water) to dissolve urea. Desert mammals have longer and more deeply inset [nephrons](https://en.wikipedia.org/wiki/Nephrons), as well as smaller and fewer [cortical](https://en.wikipedia.org/wiki/Cortical_nephron) and [juxtamedullary](https://en.wikipedia.org/wiki/Juxtamedullary_nephron) glomeruli, that is, glomeruli being capillary networks where both fluid and waste are extracted from blood. This in turn leads to a smaller [glomerular filtration rate](https://en.wikipedia.org/wiki/Glomerular_filtration_rate), in which, less water is transferred from the blood to the kidney. The kidneys of desert mammals are also better adapted at reabsorbing water from the [tubular fluid](https://en.wikipedia.org/wiki/Tubular_fluid), though there are fewer glomeruli, the xerocole has larger juxtamedullary glomeruli than cortical glomeruli (the former playing an important role in concentrating urine), whereas the opposite is true for non-xerocoles.

Desert mammals also have longer [loops of Henle](https://en.wikipedia.org/wiki/Loops_of_Henle), structures whose efficiency in concentrating urine is directly proportional to their length. The efficiency of their loops of Henle is augmented by the increased [antidiuretic hormone](https://en.wikipedia.org/wiki/Antidiuretic_hormone) in their blood. Desert amphibians can store more nitrogen than aquatic ones, and do so when not enough water is available to excrete the nitrogen as urea. The [African reed frog](https://en.wikipedia.org/wiki/Hyperolius) can store excess nitrogen in [iridophore](https://en.wikipedia.org/wiki/Chromatophore#Iridophores_and_leucophores), pigmented [granules](https://en.wikipedia.org/wiki/Granule_(cell_biology)) in its skin, by converting the nitrogen to [guanine](https://en.wikipedia.org/wiki/Guanine), which makes up the majority of the iridophores' composition. Reptiles, birds, insects, and some amphibious species excrete nitrogenous waste as [uric acid](https://en.wikipedia.org/wiki/Uric_acid) rather than urea. Because uric acid is less toxic than urea, it does not need to be dissolved in water to be excreted (as such, it is largely insoluble.

CLINICAL IMPORTANCE OF THE GLOMERULAR FILTRATION BARRIER

### 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: 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. 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.