

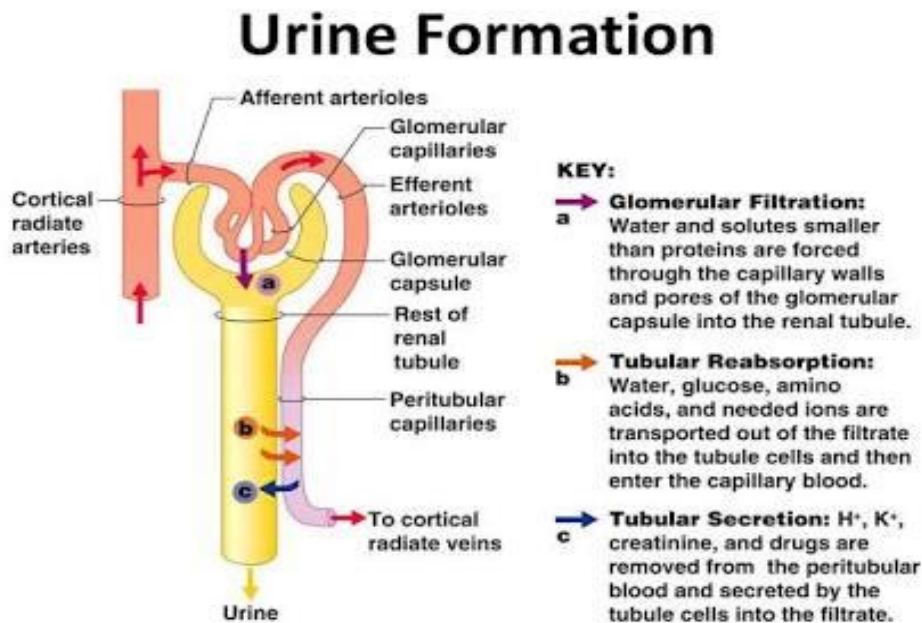
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Explain Urine Formation and concentration



Urine Formation – by filtering the blood the nephrons perform the following functions: regulate concentration of solutes in blood plasma; this also regulates pH, regulate water concentrations; this helps regulate blood pressure, removes metabolic wastes and excess substances

Urine Formation:

STEPS INVOLVED IN URINE FORMATION

- Glomerular Filtration – water and solutes are forced through the capillary walls of the glomerulus into the Bowman's capsule (glomerular capsule), Glomerular filtration occurs when glomerular hydrostatic pressure exceeds the luminal hydrostatic pressure of Bowman's capsule. There is also an opposing force, the osmotic pressure, which is typically higher in the glomerular capillary

Filtrate – the fluid that is filtered out into Bowman's capsule

Glomerular Filtration Rate is regulated by mechanisms:

- ✓ Auto regulation – the smooth muscle in the afferent arteriole responds to blood pressure changes by constricting and dilating to regulate filtration rate.
- ✓ Sympathetic control – causes afferent arterioles to constrict or dilate when activated by a nerve impulse (fight or flight response to keep blood pressure up)

Renin-angiotensin mechanism – triggered by the juxtaglomerular apparatus; when filtration rate decreases, the enzyme renin is released. Renin converts a plasma protein called angiotensinogen into angiotensin I. Angiotensin I is quickly converted into angiotensin II by another enzyme. Angiotensin II causes **changes**:

- Constriction of the arterioles – decreases urine formation and water loss
 - Stimulates the adrenal cortex to release aldosterone – promotes water reabsorption by causing the absorption of salt
 - Stimulates the posterior pituitary to release ADH – antidiuretic hormone – promotes water reabsorption
 - Stimulates the thirst and water intake (hypothalamus says we're thirsty so we get a drink)
- **Tubular Reabsorption** – occurs both passive and actively; glucose, amino acids, and other needed ions (Na, K, Cl, Ca, and HCO₃) are transported out of the filtrate into the peritubular capillaries (they are reabsorbed back into the blood); about 65% of the filtrate is reabsorbed in the proximal convoluted tubule. As these substances are reabsorbed, the blood becomes hypertonic so water easily follows by osmosis. Reabsorption in the distal convoluted tubule is under hormonal control...aldosterone causes more salt to be absorbed, ADH causes more water to be absorbed
 - **Secretion** – waste products such as urea and uric acid, drugs and hydrogen and bicarbonate ions are move out of the peritubular capillaries into the filtrate; this removes unwanted wastes and helps regulate pH
 - Urine – filtrate after it has passed through the nephron and undergone filtration, reabsorption, and secretion. The urine passes into the collecting duct, which joins with the minor calyx, major calyx, and eventually the renal pelvis. The renal pelvis joins with the ureter.
 - Color – yellow color is due to urochrome – a pigment produced from the breakdown of bile pigments in the intestine

Deep yellow to orange – more concentrated, less water

Light yellow to clear – less concentrated, more water

Urine concentration

The renal medulla produces concentrated urine through the generation of an osmotic gradient extending from the cortico-medullary boundary to the inner medullary tip. This gradient is generated in the outer medulla by the countercurrent multiplication of a comparatively small trans epithelial difference in osmotic pressure. This small difference, called a single effect, arises from active NaCl reabsorption from thick ascending limbs, which dilutes ascending limb flow relative to flow in vessels and other tubules. In the inner medulla, the gradient may also be generated by the countercurrent multiplication of a single effect, but the single effect has not been definitively identified. There have been important recent advances in our understanding of key components of the urine concentrating mechanism. In particular, the identification and localization of key transport proteins for water, urea, and sodium, the elucidation of the role and regulation of osmoprotective osmolytes, better resolution of the anatomical relationships in the medulla, and improvements in mathematic modeling of the urine concentrating mechanism. Continued experimental investigation of trans epithelial transport and its regulation, both in normal animals and in knock-out mice, and incorporation of the resulting information into mathematic simulations, may help to more fully elucidate the inner medullary urine concentrating mechanism.