1. Kidney plays an important role in glucose homeostasis, both in the post-absorptive and postprandial period. Kidney produces glucose by gluconeogenesis in the renal cortex and uses glucose for covering energy needs of the medulla. Kidney participates also to the reabsorption of filtered glucose in order the terminal urine was devoided of glucose, as long as blood glucose did not exceed 180mg/dL. Reabsorption of glucose is mediated by sodium-glucose cotransporters (SGLT1 et SGLT2) expressed in S1 and S3 segments of proximal tubule. SGLT2 is the main sodium-glucose cotransporter responsible for 90% of glucose reabsorption. In type 2 diabetics, renal gluconeogenesis and glucose utilisation are increased by 30%. Surprisingly, renal glucose reabsorption is increased, participating to worsening of hyperglycemia. This results from the increase in the renal threshhold of glucose reabsorption (220mg/dL) and from an overexpression of SGLT2 in response to hyperglycemia and of cytokine secretion. The administration of SGLT2 inhibitors to type 2 diabetic patients induced a decreased in the renal threshhold of glucose reabsorption (80mg/dL) and strongly reduced kidney glucose reabsorption. The inhibitors of SGLT2 are the only antidiabetic molecules able to correct the excessive renal glucose reabsorption in type 2 diabetics and thus to contribute, by an original mechanism, to the lowering of blood glucose level.

2.)Micturition process consists of two phases:

* Storage phase
* Voiding phase

Storage Phase

The urinary bladder is a balloon-shaped, hollow, muscular, organ that acts as the storage organ for urine. The urinary bladder in a healthy urinary system can store up to 16 ounces of urine for 2 to 5 hours easily. The circular sphincter muscles prevent leakage of urine. They close tightly around the opening of the bladder into the tube (urethra) that allows the passage of urine outside the body.

Voiding Phase

When the bladder is filled with urine, the nerves in it are triggered, which in turn stimulates the need to urinate. The brain signals urinary bladder to contract. The receptors of the urinary bladder send a signal to the [central nervous system](https://googleweblight.com/i?u=https://byjus.com/biology/central-nervous-system/&hl=), in response to which the nervous system sends a signal that incites the contraction of the urinary bladder.  Through the urinary opening at the urethra, the urine is eliminated, and the process is called micturition. The neural mechanism involved is called the micturition reflex.

**3.)Juxtaglomerular Apparatus**

The juxtaglomerular apparatus is a specialized structure formed by the distal convoluted tubule and the glomerular afferent arteriole. It is located near the vascular pole of the glomerulus and its main function is to regulate blood pressure and the filtration rate of the glomerulus. The macula densa is a collection of specialized epithelial cells in the distal convoluted tubule that detect sodium concentration of the fluid in the tubule. In response to elevated sodium, the macula densa cells trigger contraction of the afferent arteriole, reducing flow of blood to the glomerulus and the glomerular filtration rate. The juxtaglomerular cells, derived from smooth muscle cells, of the afferent arteriole secrete renin when blood pressure in the arteriole falls. Renin increases blood pressure via the renin-angiotensin-aldosterone system. Lacis cells, also called extraglomerular mesangial cells, are flat and elongated cells located near the macula densa. Their function remains unclear.

4.) Evidence that the kidneys play a key role in blood pressure regulation comes from the fact that chronic abnormalities of blood pressure control, such as hypertension, almost always begin with some abnormality of renal function. For example, Goldblatt hypertension begins with stenosis of one or both of the renal arteries; mineralocorticoid hypertension begins with increased renal tubular sodium reabsorption; and hypertension caused by infusion of vasoconstrictors such as angiotensin III (AII) or norepinephrine may be associated with increased tubular reabsorption as well as renal vasoconstriction. As hypertension develops, many of these initial changes in renal function are obscured by various compensatory mechanisms that act to restore renal excretory function toward normal. Secondary to increased arterial pressure, a cascade of circulatory alterations occurs that in many instances is much more striking than the disturbance of renal function, even though the original abnormality was in the kidney. For this reason, the importance of changes in renal function in causing hypertension has often been underestimated.

5.) Plasma calcium concentration is maintained within a narrow range (8.5-10.5 mg/dL) by the coordinated action of parathyroid hormone (PTH), 1,25(OH)2D3, calcitonin, and ionized calcium (iCa2+) itself. The kidney plays a key role in this process by the fine regulation of calcium excretion. More than 95% of filtered calcium is reabsorbed along the renal tubules. In the proximal tubules, 60% of filtered calcium is reabsorbed by passive mechanisms. In the thick ascending limb, 15% of calcium is reabsorbed by paracellular diffusion through paracellin-1 (claudin-16). The calcium sensing receptor (CaSR) in the basolateral membrane of the thick ascending limb senses the change in iCa2+ and inhibits calcium reabsorption independent to PTH and 1,25(OH)2D3. The fine regulation of calcium excretion occurs in the distal convoluted tubules and connecting tubules despite the fact that only 10-15% of filtered calcium is reabsorbed there. Transient receptor potential vanilloid 5 (TRPV5) and 6 (TRPV6) in the apical membrane act as the main portal of entry, calbindin-D28K delivers Ca2+ in the cytoplasm, and then Na2+/Ca2+ exchanger (NCX1) and plasma membrane Ca2+-ATPase in the basolateral membrane serve as an exit. In the cortical collecting duct, TRPV6 is expressed, but the role might be negligible. In addition to PTH and 1,25(OH)2D3, acid-base disturbance, diuretics, and estrogen affect on these calcium channels. Recently, klotho and fibroblast growth factor 23 (FGF23) are suggested as new players in the calcium metabolism. Klotho is exclusively expressed in the kidney and co-localized with TRPV5, NCX1, and calbindin-D28K. Klotho increases calcium reabsorption through trafficking of TRPV5 to the plasma membrane, and also converts FGF receptor to the specific FGF23 receptor. FGF23:klotho complex bound to FGF receptor inhibits 1α-hydroxylase of vitamin D, and contributes to calcium reabsorption and phosphate excretion in the kidney.