Sodium transport in the kidney

- sodium in the kidneys is not secreted, it is only reabsorbed
- about 99% of the filtered amount of sodium is reabsorbed

Content

Mechanisms of resorption

- Na + - K + ATPase , located in the basolateral membrane of tubules. Pumps primarily *actively* (ATP consumption) out into the blood, whereby K + enters the cytoplasm of the tubular cell
- this creates driving forces supporting further sodium transfer:
  - chemical gradient for Na +
    - sodium actively pumped from the tubular cell into the blood causes other sodium to pass from the lumen into the cell according to its gradient
  - electric potential – electric driving force for Na +
    - it is created by actively pumping K + into the tubules, its excess creates a positive charge here, while it is negative in the tubular cell
    - the negative charge pulls more sodium into the cell
- *passive* flow of Na + into cells – its driving force is the electrochemical gradient. It takes place differently in individual sections of the nephron:
  - proximal tubule
    - resorption of about 65% of the filtered sodium (the concentration of the luminal fluid does not change, because water also leaves with the sodium)
    - about a third of the resorption is active
    - Na + flows passively from the tubule lumen into the tubular cells via:
      - Na + - H + antiport – electroneutral exchange of Na + into the cell and H + into the lumen
      - various cotransport carriers for secondary active transport
        - positively charged particles are removed from the lumen, and therefore a negative charge is created there, the transported cations continue into the blood, where a positive charge is generated - depolarization of the first section of the proximal tubule - the formation of a negative transepithelial potential (LNTP) in the lumen
        - LNTP can be used for paracellular resorption of Cl - into the blood, but this resorption is delayed, so that the luminal concentration of Cl - increases and then diffuses down its gradient, creating a lumen positive transepithelial potential (LPTP)
  - thick segment of the ascending limb of the loop of Henle
    - Na + resorbed by the action of the Na + - 2Cl - - K + transporter
    - transport is primarily electroneutral, but K + are immediately driven back into the lumen, and thus LPTP is formed
  - distal tubule
    - Na + - Cl - cotransport
  - collection channels
    - On channels (activation: ADH , aldosterone , inhibition: ANP , prostaglandins)

On + leaves the cell with

1. Na + - K + ATPase – on the basolateral side
2. Na + - 3HCO 3 - by contransport – tertiary active

Regulation of resorption

- important for maintaining the constancy of the extracellular fluid (individual points connect to each other)

A. lack of salt – hyponatremia (with normal concentration of H 2 O)

- results in a decrease in blood osmolarity
- inhibition of ADH secretion
- increased excretion of water
- reduced volume of extracellular fluid (thus also blood plasma and blood pressure reduction)
- by activating the ARAS (activating reticular ascending system)
- angiotensin induces thirst and, via aldosterone, Na + retention
- secondarily induces water retention (through ADH secreted to increase Na + concentration)

B. an excess of salt

- increased plasma osmolarity
- thirst and stimulation of ADH release
- an increase in the volume of extracellular fluid
- ARAS attenuation

- increased secretion of ANP (atrial natriuretic peptide)
- higher excretion of NaCl and with it H 2 O
- equalization of extracellular fluid volume

Links

References

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