

# Magnesium disorder

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REVIEW ARTICLE

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## Magnesium Disorders

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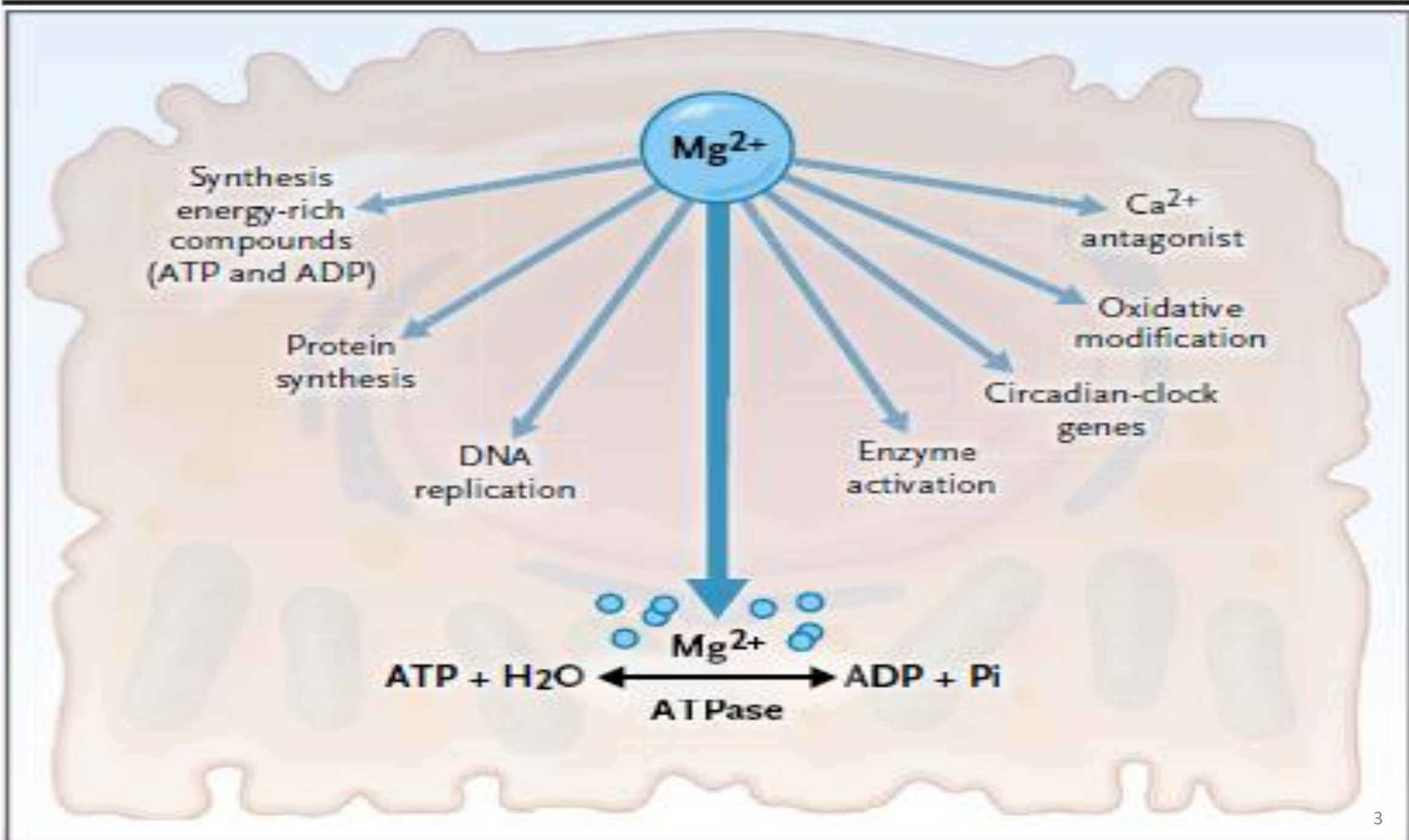
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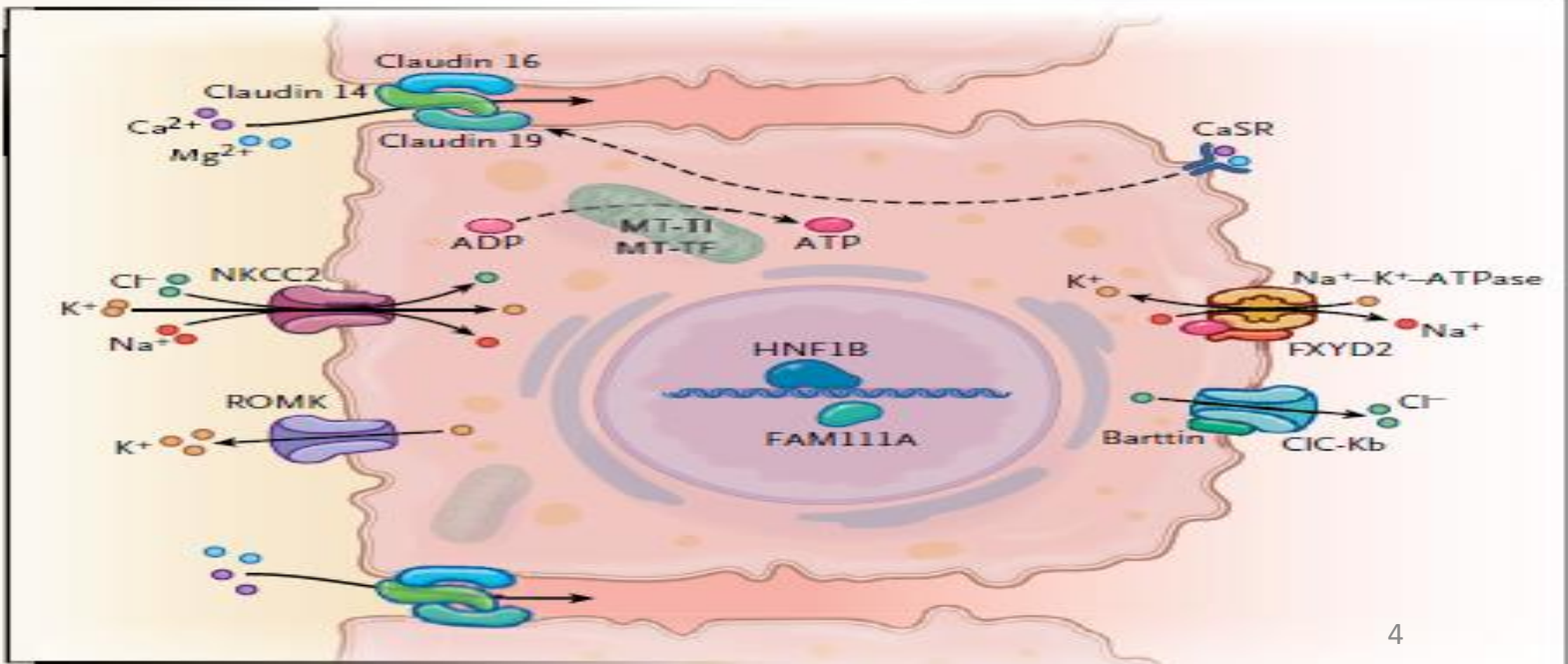
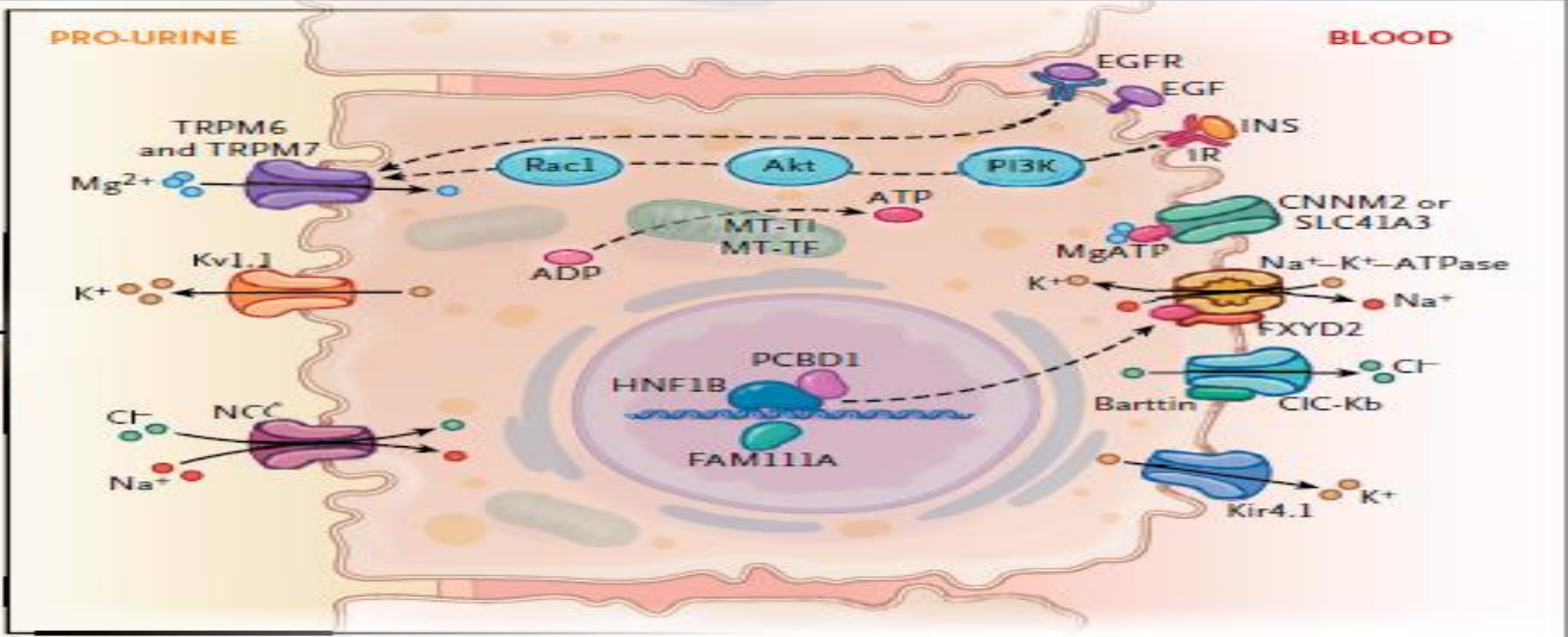
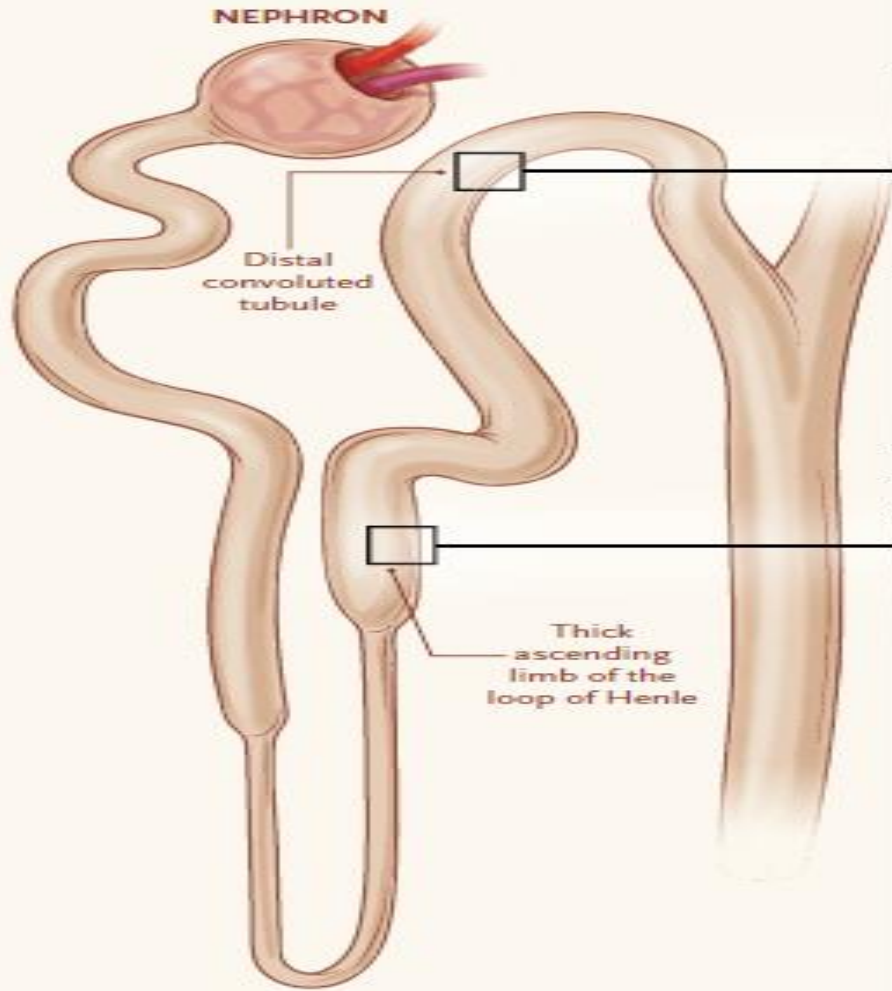
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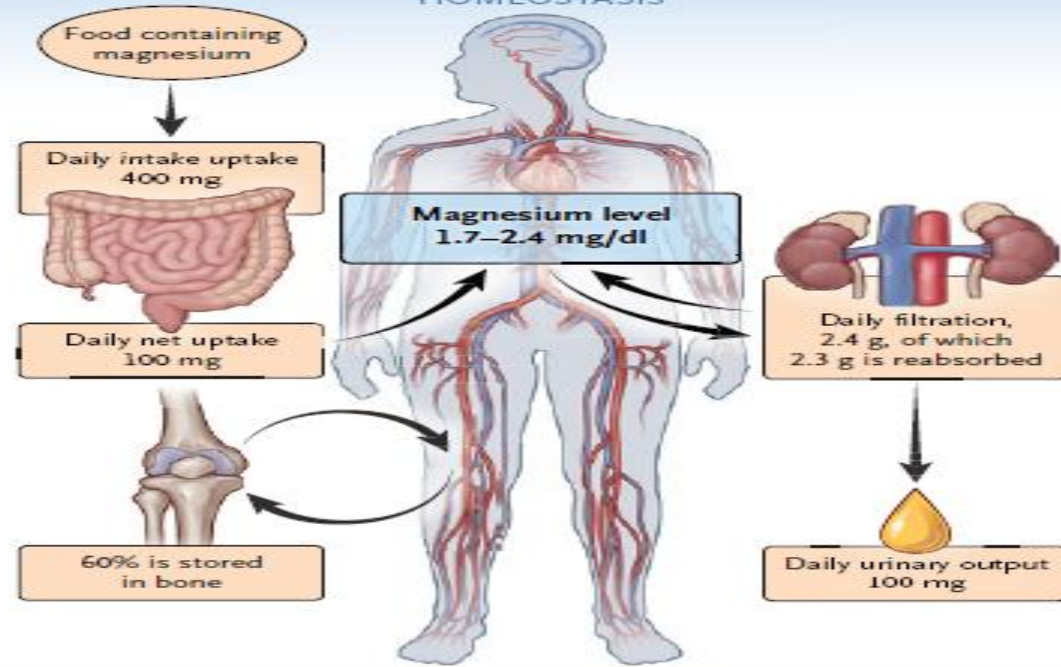
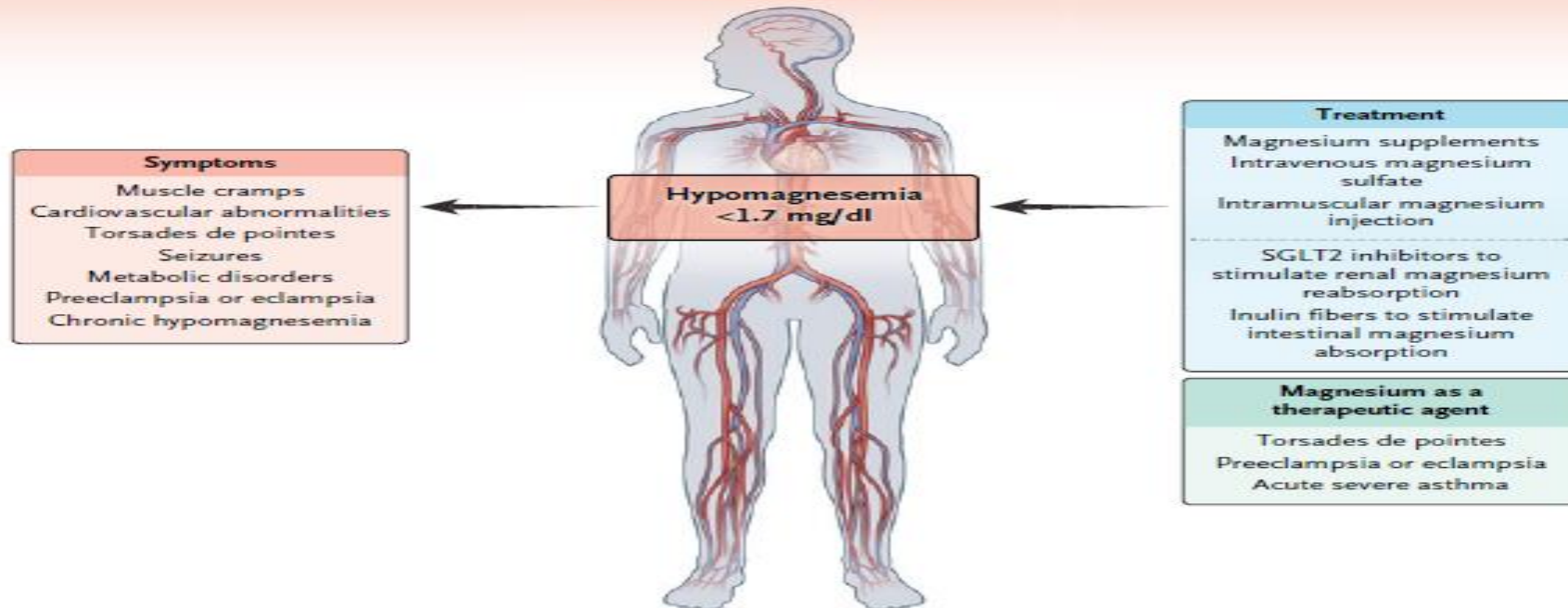


**A**LTHOUGH THE PAST SEVERAL YEARS HAVE SEEN SUBSTANTIAL ADVANCES in the understanding of molecular and cellular mechanisms regulating sodium, potassium, calcium, bicarbonate, and volume homeostasis in health and disease,<sup>1-3</sup> there has been a paucity of clinically relevant information about disorders of magnesium. Around 1980, magnesium was described as the “forgotten electrolyte,”<sup>4</sup> even though it was and remains recognized as “nature’s ... calcium blocker.”<sup>5</sup> Reasons for the apparent lack of appreciation for the clinical significance of magnesium may be due, at least in part, to the lack of information regarding the regulatory processes of this cation at the cellular, tissue, and systems levels.

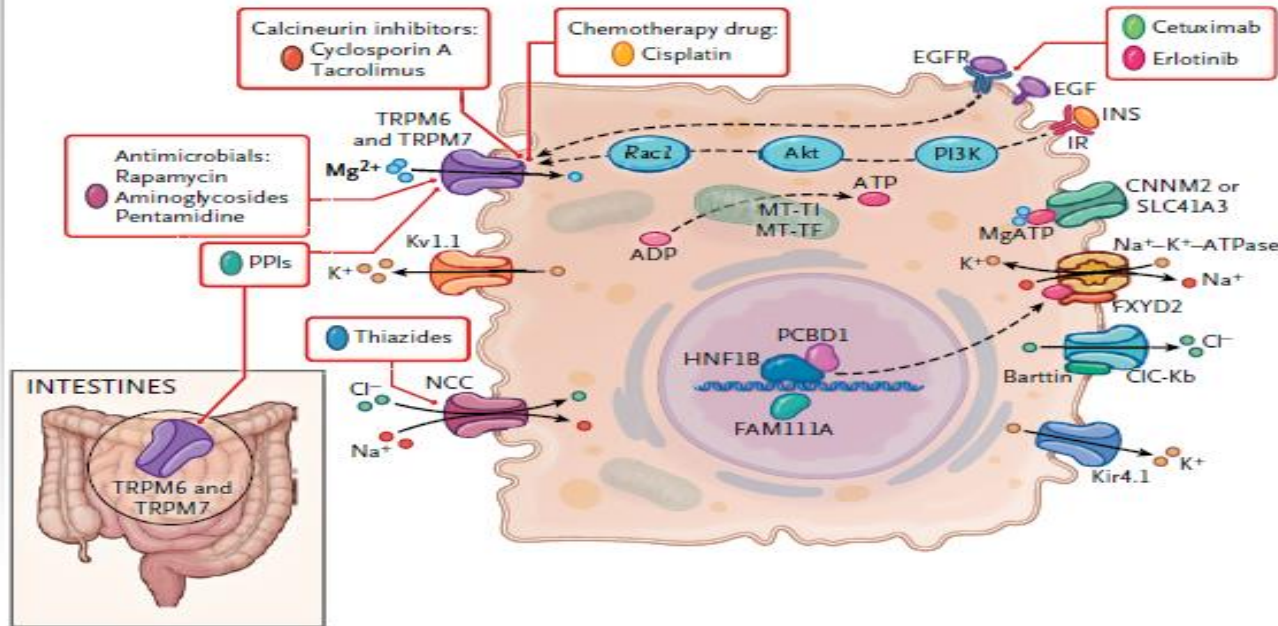
Although Murphy suggested at the turn of the millennium that it was high time to “unravel ... the mysteries of magnesium,”<sup>6</sup> her call was heeded only recently, with a growing appreciation of the role of magnesium in clinical medicine. This change has been facilitated by the discovery of magnesium-specific channels and transporters, as well as the characterization of physiological and hormonal processes that regulate magnesium homeostasis.<sup>7</sup> This review focuses on recent discoveries in how magnesium functions in the body, concentrating on hypomagnesemia, the most common clinical magnesium disorder. Hypermagnesemia is rare and occurs primarily in patients with kidney disease who are receiving magnesium-retaining drugs.<sup>8</sup>



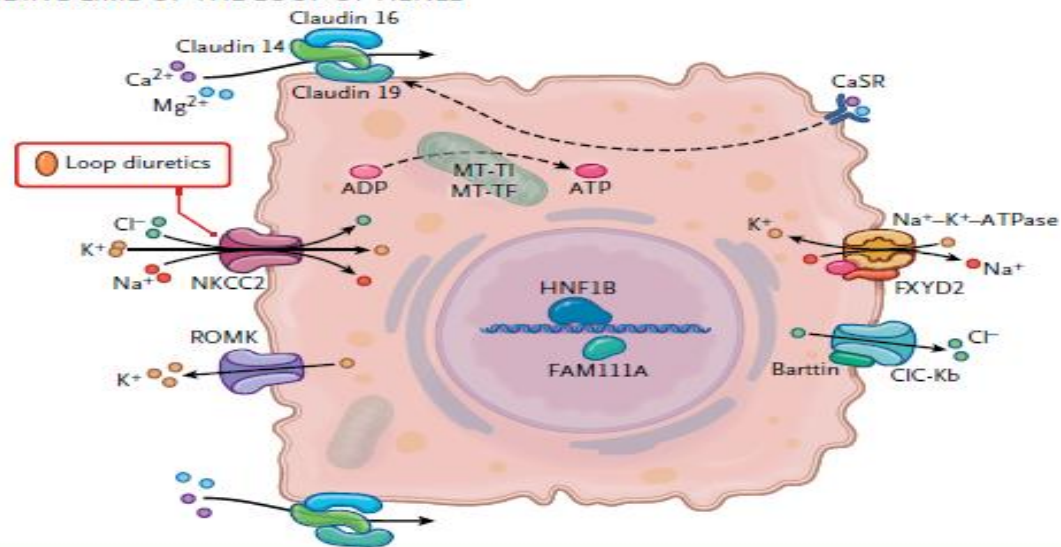


**A****HOMEOSTASIS****B****MAGNESIUM DEFICIENCY**

### DISTAL CONVOLUTED TUBULE



### THICK ASCENDING LIMB OF THE LOOP OF HENLE



**Figure 4. Drug-Induced Hypomagnesemia.**

Most drugs that cause magnesium wasting involve mechanisms that alter renal magnesium reabsorption. PPIs denotes proton-pump inhibitors.

# magnesium disorder

- Forgotten electrolyte
- Calcium blocker
- discovery of magnesium-specific channels and transporters
- cofactor for ATP(mg<sup>2+</sup>-ATP ase)
- glucose, lipid, and protein metabolism
- neuromuscular function, cardiac rhythm, vascular tone, hormone secretion
- circadian-clock

- 1.7 to 2.4 mg per deciliter (0.7 to 1.0 mmol per liter)
- intestinal absorption, renal excretion, and storage in bone
- Hypomagnesemia ( 3 to 10% general population), type 2 diabetes(10-30%) and hospitalized(10-60%) , I.C.U(65%).
- Hypocalcemia , hypokalemia, and metabolic alkalosis
- antibiotics, diuretics, biologic agents, immunosuppressants, proton-pump inhibitors, and chemotherapies
- TRPM6 and TRPM7



# The Intestine–Bone–Kidney Axis and Magnesium Homeostasis

- Cereals , beans , nuts, and green vegetables
- Dietary magnesium, intestinal lumen pH, hormones (estrogen, insulin, EGF, FGF23, [PTH]), gut microbiota
- hypomagnesemia ( <1.2 mg per deciliter [0.5 mmol per liter])
- neuromuscular irritability (carpopedal spasm, seizures, and tremors)
- cardiovascular abnormalities (arrhythmias and vasoconstriction)
- metabolic disorders (insulin resistance and chondrocalcinosis).

## Hypocalcemia, Hypokalemia , and Hypomagnesemia

- promoting potassium secretion in the collecting duct
- inhibit  $\text{Na}^+-\text{K}^+-\text{ATPase}$  pump activity-increase (ROMK) channels
- suppresses the release of PTH
- **Drug-Induced Hypomagnesemia**
- antibiotics, diuretics , biologic agents, immunosuppressants, proton pump inhibitors (PPIs), and chemotherapies
- **Nondrug Causes of Hypomagnesemia**
- chronic alcohol – type 2 diabetes -

Hypomagnesemia cause:

- cardiovascular disease(RF , torsade de point , long QT)
- vascular system(vascular contraction)
- Preeclampsia and eclampsia(calcium-channel blockade and vasodilation)

## Hereditary Hypomagnesemia

- affect magnesium reabsorption in the distal convoluted tubule-trpm6, trpm7
- **Gitelman syndrome**
- Sodium wasting disorder-hypomagnesemia, hypokalemia , and metabolic alkalosis
  - mutations in NCC.

- **Evaluating Hypomagnesemia in the Clinic**
- 24-hour magnesium excretion, fractional excretion of magnesium
- and the magnesium-loading test
- **Magnesium Replacement**
- no clear treatment guidelines -presence and severity of clinical manifestations –
- Organic salts (magnesium citrate, aspartate, glycinate, gluconate, and lactate) –  
diarrhea
- Injection =short-bowel syndrome, tetany or seizures,
- **Magnesium as a Therapeutic Agent**
- Torsades de pointes, acute asthma exacerbations, preeclampsia or eclampsia