

Major extra and intracellular electrolytes

Definition – *Electrolytes* are ions (charged atoms or molecules) that dissociate in water and conduct electricity. “Major” physiological ions are those present in millimolar quantities in body fluids and essential for life. They are grouped by their **chief location**:

Extracellular (outside cells)	Intracellular (inside cells)
Na^+ , Cl^- , HCO_3^- , Ca^{2+}	K^+ , Mg^{2+} , PO_4^{3-} , SO_4^{2-}

Body-fluid Compartments

Total body water \approx 60 % of body mass

- **Intracellular Fluid (ICF)** \approx 40 %
- **Extracellular Fluid (ECF)** \approx 20 %
 - Plasma 5 % – Interstitial 15 %

Osmotic equilibrium is preserved by electrolytes; water follows ions across semipermeable membranes (Van 't Hoff principle – $\pi = nRT/V$).

Roles of the Major Physiological Ions

Ion	Key Physiological Roles	Indian Formulations
Sodium (Na^+, extracellular)	<ul style="list-style-type: none">• Primary ECF cation \rightarrow maintains osmotic pressure• Acid–base balance via NaHCO_3 buffer• Nerve impulse initiation	Normal-Saline Inj. (0.9 % w/v, <i>Nirlife</i>); ORS sachet per IP monograph
Chloride (Cl^-, extracellular)	<ul style="list-style-type: none">• Balances cations electrically• Gastric HCl formation (parietal cells)	Ringer's Lactate (Cl^- 109 mEq L ⁻¹ , <i>Baxter</i>)
Bicarbonate (HCO_3^-, extracellular)	<ul style="list-style-type: none">• Principal blood buffer – Henderson-Hasselbalch: $\text{pH} = \text{pKa} + \log [\text{HCO}_3^-] / (0.03 \times \text{pCO}_2)$	Sodium Bicarbonate Inj. 7.5 % (<i>Neon Healthcare</i>)
Calcium (Ca^{2+}, extracellular)	<ul style="list-style-type: none">• Bone/teeth mineral (hydroxyapatite)• Muscle contraction (binds troponin-C)• Second messenger (IP_3-Ca^{2+} pathway)	Calcium Sandoz® + D3 tablets

Potassium (K^+, intracellular)	<ul style="list-style-type: none"> Action-potential repolarisation Enzyme cofactor for glycolysis (pyruvate kinase) 	Potrate-MB6 (K^+ citrate) – renal stone prophylaxis
Magnesium (Mg^{2+}, intracellular)	<ul style="list-style-type: none"> Cofactor ≥ 300 enzymes (ATP-Mg complex) Stabilises ribosome structure 	Magnesium Sulphate Inj. (eclampsia kit, Govt. supply)
Phosphate ($HPO_4^{2-}/H_2PO_4^-$)	<ul style="list-style-type: none"> Intracellular buffer ($pK_{a2} = 7.2$) High-energy bonds (ATP, creatine-P) 	K-Phos® tablets (Sandoz)
Sulphate (SO_4^{2-})	<ul style="list-style-type: none"> Detoxification: phase II conjugation in liver (sulfation) 	Sodium Sulphate IP used in colon-cleansing regimens

Functions of the Major Physiological Ions

Ion (normal serum range)	Key Roles (recap)	High Level (Hyper-...) – Common Indian Causes	Low Level (Hypo-...) – Common Indian Causes
Sodium Na^+ 135-145 mmol L ⁻¹	<ul style="list-style-type: none"> Controls ECF osmolality & BP Drives secondary transport Starts nerve/muscle depolarisation Part of $NaHCO_3$ buffer 	<ul style="list-style-type: none"> Dehydration with water loss > salt loss – profuse sweating in heat waves Osmotic diuresis in uncontrolled diabetes Hyperaldosteronism (Conn's) 	<ul style="list-style-type: none"> Diarrhoea / vomiting (ORS topic, IP monograph) SIADH due to CNS infection or carbamazepine Congestive heart failure (dilutional)
Potassium K^+ 3.5-5.0 mmol L ⁻¹	<ul style="list-style-type: none"> Sets RMP & repolarises APs Enzyme cofactor Maintains ICF osmotic balance H^+/K^+ exchange in kidney 	<ul style="list-style-type: none"> Acute renal failure / CKD (reduced excretion) K^+-sparing drugs – spironolactone, ACE-inhibitors Massive cell lysis (tumour lysis, rhabdomyolysis) 	<ul style="list-style-type: none"> Loop/thiazide diuretics without K^+ supplement Severe diarrhoea or fistulas Insulin overdose or β-agonist therapy
Calcium Ca^{2+} (ionised)	<ul style="list-style-type: none"> Bone/teeth (hydroxyapatite) Muscle 	<ul style="list-style-type: none"> Primary hyperparathyroidism Malignancy- 	<ul style="list-style-type: none"> Hypo-parathyroidism / post-thyroidectomy

1.0-1.3 mmol L ⁻¹	contraction & NT release • IP ₃ -Ca ²⁺ signalling • Activates clotting factors II, VII, IX, X	associated (PTH-rP secretion, bone mets) • Prolonged thiazide use	• Vit-D deficiency (nutritional rickets, osteomalacia) • Acute pancreatitis (saponification)
Magnesium Mg ²⁺ 0.7-1.1 mmol L ⁻¹	• Stabilises ATP (300+ enzymes) • Dampens NMJ excitability • DNA/RNA synthesis • Needed for PTH release	• Renal failure (diminished clearance) • Excess MgSO ₄ infusions (eclampsia)	• Chronic PPI therapy (↓ gut absorption) • Alcoholism / malnutrition • Osmotic diarrhoea (lactulose misuse)
Chloride Cl ⁻ 98-106 mmol L ⁻¹	• Maintains electroneutrality • Forms gastric HCl • CO ₂ transport (Cl ⁻ shift)	• Renal tubular acidosis type I or II • Large 0.9 % NaCl infusion (“saline load”)	• Prolonged vomiting / NG suction (HCl loss) • Diuretic overuse with Na ⁺ /K ⁺ wasting
Bicarbonate HCO ₃ ⁻ 22-26 mmol L ⁻¹	• Main extracellular buffer • CO ₂ shuttle in blood	• Metabolic alkalosis – vomiting, diuretic therapy • Compensation for chronic CO ₂ retention (COPD)	• Metabolic acidosis – diabetic ketoacidosis, renal failure, profuse diarrhoea
Phosphate PO ₄ ³⁻ 0.8-1.5 mmol L ⁻¹	• Buffers ICF pH • Forms ATP/creatine-P • DNA/RNA, phospholipids • Regulates 2,3- BPG in RBCs	• Renal failure (↓ excretion) • Tumour lysis syndrome • Hypocalcaemic states with ↑ PTH-rP	• Refeeding syndrome after prolonged starvation • DKA therapy (insulin drives PO ₄ ³⁻ into cells) • Antacid abuse with Al(OH) ₃
Sulphate SO ₄ ²⁻ 0.3-0.4 mmol L ⁻¹	• Phase-II drug sulphation • Structural GAGs (cartilage)	• Rare—may rise in advanced CKD (reduced filtration)	• Protein-energy malnutrition (low methionine/cysteine) • Neonates on long- term parenteral

			nutrition without sulphate
--	--	--	---------------------------------------