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Nota di contenuto	Front Cover; Hormones and Transport Systems; Copyright; Former Editors; Contents; Contributors; Preface; Chapter One: Dietary I- Absorption: Expression and Regulation of the Na ⁺ /I ⁻ Symporter in the Intestine; 1. The Importance of Iodide in Human Health; 2. The Na ⁺ /I ⁻ Symporter; 2.1. Molecular identification of NIS; 2.2. NIS-mediated transport: Substrates and stoichiometry; 2.3. The role of physiological Na ⁺ concentrations in NIS affinity for I ⁻ ; 3. NIS Expression Beyond the Thyroid; 4. Targeting of NIS to the Plasma Membrane; 5. Hormonal Regulation of NIS Expression; 6. Dietary I- Absorption 7. Regulation of Intestinal NIS Expression 8. Conclusions and Future Directions; Acknowledgments; References; Chapter Two: Apical Iodide Efflux in Thyroid; 1. Introduction; 2. Iodide and Thyroid Hormone Synthesis; 2.1. Thyroid organization; 2.2. Thyroid hormone synthesis; 3. Vectorial Transport Processes in Epithelia and Thyroid I- Accumulation; 3.1. Brief overview of basic epithelial transport processes; 3.2. Basolateral iodide uptake; 3.3. Apical iodide release; 4. Chloride Transport Proteins and Luminal I- Translocation; 4.1. SLC26A4 (Pendrin); 4.1.1. SLC26A4, HCO ₃ ⁻ , luminal pH 4.2. Cystic fibrosis transmembrane conductance regulator 4.2.1. CFTR and SLC26A4 interplay; 4.3. SLC5A8, a sodium-monocarboxylate

transporter (hAIT; SLC5A8; SMCT1); 4.4. TMEM16A (anoctamin 1); 5. Conclusions; Acknowledgment; References; Chapter Three: The Sodium/Multivitamin Transporter: A Multipotent System with Therapeutic Implications; 1. Introduction; 2. "Active" Transport; 3. Identification of the Multivitamin Transporter; 4. The hSMVT Gene; 4.1. Expression of hSMVT in various tissues; 4.2. An additional high-affinity hSMVT-like uptake system?; 5. From Gene to Protein; 6. Family Ties 7. The Predicted Structure of hSMVT8. The (Co)Substrates of hSMVT; 9. The Characterization of the Cloned hSMVT; 9.1. Electrogenicity of hSMVT-mediated transport; 9.2. Mechanistic implications; 10. Medical Implications; 11. Conclusion and Future Directions; Acknowledgments; References; Chapter Four: Regulation of ENaC Transcription; 1. Introduction; 1.1. Aldosterone is a ligand for the mineralocorticoid receptor and glucocorticoid receptor; 1.2. Epithelial sodium channel (ENaC) is a major target of aldosterone action and a key ion channel in regulating Na⁺ balance

2. Dot1a-Af9 Complex Mediates Repression of ENaC2.1. Histone H3 K79 methyltransferase Dot1a; 2.1.1. Dot1 proteins are a unique class of histone methyltransferases; 2.1.2. Dot1 proteins and H3 K79 methylation have diverse functions; 2.1.3. Dot1a is the first aldosterone-regulated target with a known function in epigenetics; 2.1.4. Dot1a modulates targeted H3 K79 methylation at the ENaC promoter and represses ENaC in a methyltransferase-depen...; 2.1.5. Dot1a-mediated repression apparently requires its nuclear expression as well as its methyltransferase activity and... 2.1.6. Dot1a-mediated repression of ENaC raised new questions
