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Nota di bibliografia	Includes bibliographical references and index.
Nota di contenuto	Cover; Contents; Chair's introduction; Overview of the SLC26 family and associated diseases; Discussion; Individual characteristics of members of the SLC26 family in vertebrates and their homologues in insects; Discussion; Sulfate transport by SLC26 transporters; Discussion; Sugar transport by members of the SLC26 superfamily; Discussion; SLC26A3 and congenital chloride diarrhoea; Discussion; Expression, regulation and the role of SLC26 C1/HCO exchangers in kidney and gastrointestinal tract; Discussion Anion exchangers in flux: functional differences between human and mouse SLC26A6 polypeptidesDiscussion; Physiology of electrogenic SLC26 paralogues; Discussion; Role of SLC26-mediated C1/base exchange in proximal tubule NaCl transport; Discussion; SLC26 transporters and the inhibitory control of pancreatic ductal bicarbonate secretion; Discussion; Regulatory interaction between CFTR and the SLC26 transporters; Discussion; Insights from a transgenic mouse model on the role of SLC26A2 in health and disease; Discussion

New insights into the role of pendrin (SLC26A4) in inner ear fluid homeostasis; Discussion; The renal physiology of pendrin (SLC26A4) and its role in hypertension; Discussion; Interaction of prestin (SLC26A5) with monovalent intracellular anions; Discussion; Final discussion; Index of contributors; Subject index

Sommario/riassunto

Cl⁻ absorption and HCO₃⁻ secretion are intimately associated processes vital to epithelial function, itself a key physiological activity. Until recently the transporters responsible remained obscure, but a breakthrough occurred with the discovery of the SLC26 transporters family. It is now clear that the SLC26 transporters have broad physiological functions since mutations in several members are linked to a variety of diseases. This book describes the properties of this family in detail, with contributions from the leading global researchers in the field. Complementary views from experts on ot
