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Nota di contenuto	Handbook of ATPases; Contents; Preface; List of Contributors; Part I P-type ATPases; 1 Yeast Plasma-membrane H(+)-ATPase: Model System for Studies of Structure, Function, Biogenesis, and Regulation; 1.1 Introduction; 1.2 Structure; 1.2.1 Ca(2+)-ATPase as a Model; 1.2.2 Applicability of the Ca(2+)-ATPase Structure to Other P(2)-ATPases, Including the Pma1 H(+)-ATPase; 1.2.3 H(+)-ATPase Oligomers; 1.2.4 Associated Proteolipids; 1.3 Reaction Mechanism; 1.3.1 Overview of the Reaction Cycle; 1.3.2 ATP Binding and Phosphorylation; 1.3.3 E1-E2 Conformational Change; 1.3.4 H(+) Pumping 1.4 Biogenesis1.4.1 Pma1 Mutants with Defects in Folding and Biogenesis; 1.4.2 Use of Pma1 Mutants to Screen for Other Genes that Play a Role in Biogenesis and Quality Control; 1.4.3 Role of Lipid Rafts; 1.5 Regulation; 1.6 Emerging Knowledge of Other Yeast P-type ATPases; Acknowledgments; References; 2 Regulation of the Sarco (endo)plasmic Reticulum Ca(2+)-ATPase by Phospholamban and

Sarcolipin; 2.1 Introduction; 2.1.1 Background to Ca(2+) Signaling; 2.1.2 -Adrenergic Signaling in the Heart; 2.2 Phospholamban-SERCA Interactions; 2.2.1 SERCA Structure and Function 2.2.2 PLN Structure and Function 2.2.3 Approaches to the Study of PLN-SERCA Interactions; 2.2.4 SERCA Residues Essential for Cytoplasmic Interaction with PLN; 2.2.5 PLN Residues Essential for Cytoplasmic Interaction with SERCA; 2.2.6 PLN Residues Essential for Transmembrane Interactions with SERCA; 2.2.7 SERCA Residues Essential for Transmembrane Interactions with PLN; 2.2.8 Structural Modeling of the PLN-SERCA Inhibitory Interaction; 2.3 Physiological Role of PLN in Basal Cardiac Function; 2.3.1 Alterations in PLN Levels and Function by Transcription and Phosphorylation 2.3.2 Targeting of PLN 2.3.3 Role of PLN in Smooth and Skeletal Muscles; 2.3.4 Overexpression of PLN; 2.3.5 Physiological Role of PLN in -Adrenergic Stimulation; 2.3.6 Superinhibitory PLN Mutants; 2.4 Phospholamban in Heart Failure; 2.4.1 Introduction; 2.4.2 Potential Therapies; 2.5 Human PLN Mutations as a Cause of Cardiomyopathy; 2.5.1 PLN R9C Mutant; 2.5.2 PLN L39stop Mutant; 2.6 Sarcolipin; 2.6.1 Introduction; 2.7 Physiological Role of SLN; 2.7.1 SLN Expression; 2.7.2 Overexpression of SLN; 2.7.2.1 Response of the SLN Gene to Chronic Stimulation 2.7.3 Inhibition of SERCA Function by SLN Plus PLN 2.7.4 Modeling of the SLN-SERCA and SLN-PLN-SERCA Interactions; Acknowledgments; References; 3 Catalytic and Transport Mechanism of the Sarco-(Endo) Plasmic Reticulum Ca(2+)-ATPase (SERCA); Summary; 3.1 Introduction; 3.2 Experimental Systems; 3.3 Functional Characterization; 3.4 Structural Characterization; 3.4.1 Extramembranous Region and the Catalytic Domains of E1·2Ca(2+); 3.4.2 Transmembrane region of E1·2Ca(2+); 3.4.3 Enzyme Structure in the Absence of Ca(2+) (E2·TG); 3.4.4 Thapsigargin-binding Domain; 3.4.5 Interaction with Phospholamban 3.5 Binding of Ligands, Catalytic Events and Conformational Changes

Sommario/riassunto

As the first comprehensive overview of this important class of enzymes, this two-volume handbook summarizes recent knowledge about the molecular mechanism of ATPases, relating this information to the physiology and pathophysiology of ion transport, mitochondrial function, vesicle transport and lysosomal acidification. All important P-type, F-type and V-type ATPases are treated systematically, complemented by a special section on the cell biology and physiology of acidic compartments, and backed by an extensive bibliography and index. This premier reference source for physiologists, molecular