Preface

This is my delight, thus to wait and watch at the wayside where shadow chases light and rain comes in the wake of summer. Messengers with tidings from unknown skies greet me and speed along the road. My heart is glad within and breath of the passing breeze is sweet.

Rabindranath Tagore
(Gitanjali: Song of offerings)

The biological membranes of cellular organization enfold an important group of membrane proteins called the ATPases, which not only are versatile in maintaining chemical gradient and electrical potential across the membrane but also bring metabolites necessary for cell metabolism and drive out toxins, waste products, and solutes that otherwise can curb cellular functions. ATPases are distributed virtually in all forms starting from unicellular to multicellular and also in viruses. There are different types of ATPases, which differ in function and structure and in the type of ions they transport. The three main types of the ion pump ATPase family are (1) P-type ATPases that transport different ions across membranes. Plasma membrane Ca$^{2+}$-ATPase (PMCA) utilizes ATP as the energy to extrude Ca$^{2+}$ from the cells. The main calcium controlling organelle in the cell is within the sarco(endo)plasmic reticulum (SERCA). This pump transports Ca$^{2+}$ from the cytosol to the lumen of the SR (or ER). The involvement of phospholamban in the regulation of SERCA by phosphorylation has been described. Interactions of SR Ca$^{2+}$-ATPase with phospholamban was shown to have functional roles in different types of diseases, for example, pulmonary hypertension; (2) F-type ATPase in mitochondria, chloroplasts, and bacterial plasma membranes produce ATP using the proton gradient; and (3) V-type ATPase catalyzes ATP hydrolysis to transport solutes and maintains acidic pH in organelles like lysosomes. Genetic defects in either of the ATPases cause several diseases. For example, mutations expressed in osteoclasts and intercalated cells lead to diseases such as osteoporosis, tumor cell invasion, and renal tubule acidosis. Furthermore, H$^+$-ATPase gene mutations cause distal renal tubular acidosis, a condition characterized by impaired renal acid secretion resulting in metabolic acidosis. A number of
researches have demonstrated the involvement of several members of the ATPase family in the cell pathology and diseases, thereby penetrating exciting new areas of our understanding.

In this book, the authors summarize recent knowledge about the molecular mechanisms associated with Ca\(^{2+}\)-ATPase, V-ATPase, and F-ATPase in intracellular and extracellular Ca\(^{2+}\) transport, mitochondrial ATP synthase, vesicular H\(^+\) transport, and lysosomal pH regulation. This book thereby bridges the gap between fundamental research and biomedical and pharmaceutical applications. It also provides an informative resource to improve ATPase research and modern therapeutic approaches toward different life-threatening diseases that are associated with dysregulation of the ATPases.

This book contains 29 chapters, which have been arranged under four parts, namely: (1) Plasma Membrane Ca\(^{2+}\)-ATPases; (2) Sarco(endo)plasmic Reticulum ATPases; (3) Vacuolar ATPases; and (4) F\(_1\),F\(_0\)- and other ATPases, for the convenience of our readers. It is hoped that the readers will find each chapter stimulating and thought inciting, which will add new dimensions of future ATPase research. It is well said that: all endings are also beginnings, we just don’t know it at the time!

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