Preface

Since the discovery of heat shock response by Ferruccio Ritossa in 1962, the phenomenon has been well characterized in a variety of cells and organisms as induction of a family of proteins called "heat shock proteins" (HSP). Based on their molecular weight, these proteins are classified into, at least, 6 major subfamilies named as HSP100, HSP90, HSP70, HSP60, HSP40 and small HSPs. The fact that the heat shock protein synthesis can be triggered by a variety of other stress conditions such as, infection, inflammation, exercise, starvation, oxygen-, nitrogen- or water-deprivation and exposure to chemical and physical toxins, they are also classified as "stress proteins". Then came the surprise that the HSP also exist under non-stressful conditions and perform housekeeping functions, such as folding and assisting in the establishment of correct protein conformation, mediating protein-protein interactions, intra-cellular trafficking of other proteins, preventing unwanted protein aggregation and channelizing their degradation. A new term "chaperones" evolved to express such functionality of this highly conserved class of proteins.

A new member of HSP70 family of proteins was first cloned in 1993 in a cell hybrid protein-screening assay. Since it was identified to be associated with cellular mortal phenotype, it was named 'mortalin'. Endorsing its multiple functionality, mortalin made its manifestation in many independent experimental regimes, such as those aimed to identify molecules involved in antigen processing, stress-survival and mitochondrial functions. With nearly two decades of experimentation, mortalin has been recognized as an essential protein that not only acts as a chaperone and stress-survival factor but also plays a key role in mitochondrial import motor function, energy generation, ROS management, immune response, control of centrosome duplication and activities of tumor suppressor protein p53. Stemming from these multiple functions is its role in human cancers on one-hand and neurodegenerative diseases on the other. With an aim to introduce mortalin at the graduate and advanced undergraduate levels, this book is organized as a chapter-wise description of structure, evolution and functional role of mortalin in normal and diseased physiology. We hope that this sketch of mortalin biology by the team of experts will help in asking new questions, advancing knowledge and developing mortalin-based diagnostic and therapeutic reagents and technologies.

We are very grateful to all the authors for their interest, enthusiasm and devotion to mortalin research that made this book necessary and possible. Without their hard work to contribute chapters, it was not possible to accomplish this volume suitable for general and specialized reading.

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