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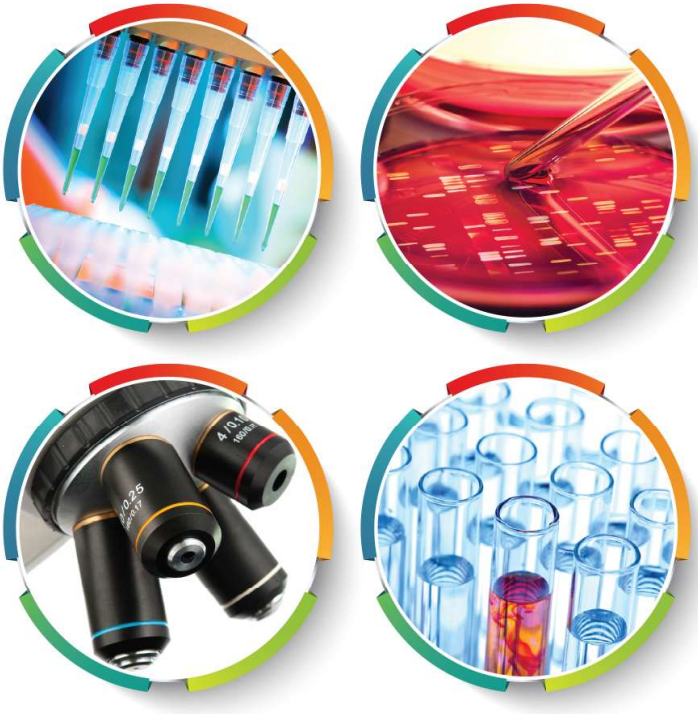

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Contents

Chapter 2

Unraveling the GRP78 Paradox: From Cancer Promotion to Suppression

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Abstract

The endoplasmic reticulum (ER) plays a vital role in numerous cellular activities like protein folding, synthesis, and post-translational modifications. ER stress can be caused by several pathophysiological conditions like the accumulation of misfolded proteins or cancer. ER stress plays a dual role in cancer by either promoting cell survival or triggering cell death depending on the imbalance between ER protein-folding capacity and load. The accumulation of misfolded proteins in the ER lumen promotes unfolded protein response (UPR) via ER stress and activates glucose-regulated protein 78 (GRP78), calnexin, and calreticulin. Moreover, moderate ER stress promotes cancer cell survival resulting in chemotherapeutic resistance, but severe ER stress leads to cancer cell apoptosis. Further, UPR signaling may activate autophagy to clear the accumulated misfolded proteins and reduce ER stress. Autophagy plays a significant role in cell metabolism, where it degrades

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