AJP-Cell begins a Theme series on Cellular Mechanisms of Endoplasmic Reticulum Stress Signaling in Health and Disease

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OVER THE PAST DECADE, endoplasmic reticulum (ER) stress signaling has emerged as an essential mechanism at the crossroads of cellular functions. Indeed, beyond the instrumental roles of the ER in the biogenesis of secretory and transmembrane proteins, and in the control of lipid balance or in calcium homeostasis, this subcellular compartment can now be seen as a signal integration platform that manages information on ER homeostasis on cellular metabolic status, the interactions of a cell with its (micro)environment, and, in multicellular organisms, the status of the organism as a whole (signaling control through cell nonautonomous mechanisms). The afferent signals (noted above) produce dramatic changes at the level of the ER membrane, thereby eliciting the activation of signaling pathways collectively known as the unfolded protein response, which in turn reprogram the cell (at the transcriptional, posttranscriptional, translational, and posttranslational levels) to either promote adaptation or death.

These novel functions of the ER have significant impact on the understanding and treatment of disease states in which protein homeostasis is perturbed (for instance, in secretory protein misfolding diseases or neurodegenerative diseases) but also in pathologies that have more complex traits such as cancer, diabetes, or inflammatory diseases.

We have thus invited several leading experts to write review articles for a theme series on the cell physiology of ER stress signaling so as to provide readers with current insights and to identify major unsolved questions. The series begins in this issue with an introductory article by C. Hetz that explores the role of ER stress in cell life and death decisions (“ER stress signaling mechanisms: an overview”). Subsequent reviews will highlight the different functions of the ER in the control of secretory and transmembrane protein homeostasis (J. E. Chambers and S. J. Marciniak, “Protein misfolding and ER stress”) and explore the role of ER stress signaling in cancer (S. Manié and E. Chevet) and in pancreatic β-cells (F. Urano).

We believe that readers will find the review articles in this Theme of interest. In addition, we hope that the ideas and results provided in the articles will stimulate experiments that address unanswered, important questions regarding cellular mechanisms in which ER stress signaling plays essential functions. We look forward to receiving original manuscripts in response to the accompanying Call for Papers on “Endoplasmic Reticulum Stress” that provide such results for publication in AJP-Cell Physiology.

DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the author(s).

AUTHOR CONTRIBUTIONS

F.F. and E.C. drafted manuscript; F.F., S.L., P.A.I., and E.C. edited and revised manuscript; F.F., S.L., P.A.I., and E.C. approved final version of manuscript.

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