

1. Record Nr.	UNISA996397343103316
Autore	Altham Michael <1633-1705.>
Titolo	A vindication of the Church of England from the foul aspersions of schism and heresie unjustly cast upon her by the Church of Rome [[electronic resource] ] : in two parts
Pubbl/distr/stampa	London, : Printed by J.H. for Luke Meredith ..., MDCLXXXVII [1687]
Descrizione fisica	[3], 30, [2], 5-40 p
Soggetti	Schism
Lingua di pubblicazione	Inglese
Formato	Materiale a stampa
Livello bibliografico	Monografia
Note generali	Attributed by Wing (2nd ed.) to Michael Altham. Advertisements: p. [3] and p. 40. Incorrectly identified as Wing A2963A. Reproduction of original in: Newberry Library.
Sommario/riassunto	eebo-0101

2. Record Nr.	UNINA9910137530103321
Autore	Kezhong Zhang
Titolo	Endoplasmic reticulum stress response and transcriptional reprogramming // topic editor Kezhong Zhang
Pubbl/distr/stampa	Frontiers Media SA, 2015 Switzerland : , : Frontiers Media SA, , 2015
ISBN	9782889194360 (ebook)
Descrizione fisica	1 online resource (97 pages) : illustrations
Collana	Frontiers Research Topics
Soggetti	Cytology Biology Health & Biological Sciences
Lingua di pubblicazione	Inglese
Formato	Materiale a stampa
Livello bibliografico	Monografia
Note generali	Bibliographic Level Mode of Issuance: Monograph
Nota di bibliografia	Includes bibliographical references.
Sommario/riassunto	<p>Endoplasmic reticulum (ER) is an intracellular organelle responsible for protein folding and assembly, lipid and sterol biosynthesis, and calcium storage. A number of biochemical, physiological, or pathological stimuli can interrupt protein folding process, causing accumulation of unfolded or misfolded proteins in the ER lumen, a condition called "ER stress". To cope with accumulation of unfolded or misfolded proteins, the ER has evolved a group of signaling pathways termed "Unfolded Protein Response (UPR)" or "ER stress response" to align cellular physiology. To maintain ER homeostasis, transcriptional regulation mediated through multiple UPR branches is orchestrated to increase ER folding capacity, reduce ER workload, and promote degradation of misfolded proteins. In recent years, accumulating evidence suggests that ER stress-triggered transcriptional reprogramming exists in many pathophysiological processes and plays fundamental roles in the initiation and progression of a variety of diseases, such as metabolic disease, cardiovascular disease, neurodegenerative disease, and cancer. Understanding effects and mechanisms of ER stress-associated transcriptional reprogramming has high impact on many areas of molecular genetics and will be particularly informative to the</p>

development of pharmacologic avenues towards the prevention and treatment of modern common human diseases by targeting the UPR signaling. For these reasons, ER stress response and transcriptional reprogramming are a timely and necessary topic of discussion for *Frontiers in Genetics*.

---