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Autore	Kezhong Zhang
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Sommario/riassunto	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

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.