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Journal of Proteomics and Bioinformatics

Commentary

A Brief Note on Proteostasis and its Mechanisms

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DESCRIPTION

The dynamic regulation of a balanced, functional proteome is known as proteostasis. The proteostasis network is made up of competing and interconnected biological mechanisms within cells that regulate protein synthesis, folding and degradation both inside and outside the cell. Proteostasis loss is crucial for understanding diseases caused by excessive protein misfolding and degradation, which results in loss-of-function phenotypes and aggregation-related degenerative syndromes. These disorders may be treated or resolved with therapeutic restoration of proteostasis. Successful development, healthy ageing, resistance to external stimuli, and minimising homeostatic disturbances from pathogens such as viruses all require cellular proteostasis. Regulated protein translation, chaperone-assisted protein folding, and protein degradation pathways are all cellular strategies for preserving proteostasis. To sustain all cellular processes that rely on a properly folded proteome, each of these pathways must be adjusted based on the demand for specific proteins.

Mechanisms of proteostasis

The roles of the ribosome in proteostasis: Translation is one of the initial points of regulation for proteostasis. This is performed by the ribosome's structure, which is crucial to translation. These two properties determine how the protein folds and how it interacts in the future. The ribosome's ability to synthesis a new peptide chain is slow, and it can even halt when it encounters a rare codon, a codon found in low amounts in the cell. These pauses give an individual protein domain the time it needs to fold before the creation of subsequent domains. This makes it easier for multi-domain proteins to fold correctly. Through the narrow ribosome escape channel, the freshly synthesized peptide chain exits the ribosome and enters the cellular environment. The nascent chain has already formed secondary and restricted tertiary structures because to space constraints in the exit route. Simultaneously, the exit channel avoids premature folding by preventing large-scale interactions within the peptide chain that would demand more space.

Molecular chaperones and post-translational maintenance in proteostasis: The cell uses molecular chaperones, which aid in the assembly or disassembly of proteins, to maintain protein homeostasis post-translationally. They work to encourage the appropriate development of non-covalent interactions that lead to the desired folded state by recognizing exposed regions of hydrophobic amino acids in the nascent peptide chain. When a nascent chain of more than 60 amino acids exits from the ribosome exit channel, chaperones begin to assist in protein folding. Trigger factor is one of the most researched ribosome binding chaperones. Trigger factor helps to stabilize peptides by promoting their folding, preventing aggregation, and promoting the refolding of denatured model substrates. In vivo, ribosome profiling experiments revealed that TF primarily targets ribosomes translating outer membrane proteins, and that ribosomes translating inner membrane proteins are underrepresented. Trigger factor not only helps the protein fold properly, but it also attracts other chaperones to the ribosome, including Hsp70.

Chaperonins are a type of chaperone that cyclically encapsulates the peptide chain to facilitate native state folding. There are two types of chaperonins. Bacteria, chloroplasts, and mitochondria all include Group 1 chaperonins. Group 2 chaperonins are found in the cytosol of both eukaryotic and archaeal cells. Unlike Group 1, which relies on an extra cochaperone to act as a lid, Group 2 chaperonins have an additional helical component that works as a lid for the cylindrical protein chamber.

Regulating proteostasis by protein degradation: The protein degradation machinery is the third component of the proteostasis network. When cellular signals indicate that total cellular protein levels must be reduced, protein breakdown happens in proteostasis. Protein degradation can have local impacts, with the cell just feeling the effects of the degraded protein itself, or it can have global implications, with the entire protein landscape changing as a result of the loss of other proteins' interactions with the damaged protein. Proteostatic degradation targets a variety of substrates. Nonfunctional protein fragments formed by ribosomal stalling during translation, misfolded or unfolded proteins, aggregated proteins, and proteins no longer required for cellular function are all examples of degradable substrates. There are several alternative strategies to carry out these degrading processes.

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Received: 02- Feb-2022, Manuscript No. JBP-22-16040; **Editor assigned**: 04-Feb-2022, PreQC No. JBP-22-16040 (PQ); **Reviewed:** 18- Feb-2022, QC No. JBP-22-16040; **Revised**: 25- Feb-2022, Manuscript No. JBP-22-16040 (R); **Published**: 04-Mar-2022, DOI:10.35248/0974-276X.22.15.571.

Citation: Petrovsky N (2022) A Brief Note on Proteostasis and its Mechanisms. J Proteomics Bioinform.15:571.