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Opinion Article

Molecular Responses to Cellular Stress Responsive Cell

Tomoko Masui*

Department of Cell Biology, Tohoku University Hospital, Sendai, Japan

DESCRIPTION

Heat shock response was the first name given to the cellular stress response. Heat-induced chromosomal alterations were later linked to increased expression of a group of genes known as heat shock genes. These genes, which spread across numerous chromosomes as multiple families contribute to cellular viability in both normal and stressful settings. The regulation of heat shock gene expression and the function of heat shock proteins have been two major concerns. The so-called heat shock response is now understood to be a component of a larger cellular stress response. UV radiation exposure, oxidant injury, DNA damage, and glucose deprivation all cause specific reactions. Some of these responses appear to be similar to the heat shock response, while others appear to be distinct. However, one stressor can protect cells from additional stressors. Heavy metal exposure will cause a protective response against heat. Both of these cause protein damage, causing the cell to transition from normal transcriptional and translational processes to heat shock and other stress protein expression.

Specific DNA-binding proteins or transcription factors are activated to initiate the cellular stress response. According to the study, 2/3rd or more heat-inducible genes require the activation of the Heat Shock Factor (HSF), an intracellular protein. HSF1 binds to the promoter region of a stress gene and bends DNA with the help of other transcription factors to increase RNA polymerase activity. As a result, ribosomes convert stress mRNAs into stress proteins, resulting in larger quantities of messenger RNA (mRNA) encoded by stress genes. Stress proteins have been referred to as heat shock proteins in the past as they serve many functions. The size or molecular mass of these proteins is used to classify them. Heat Shock Proteins (HSPs) are made up of high, middle, and low molecular mass species, with the heat shock protein 70 (molecular mass=70 kDa). HSPs are present in most people under normal circumstances, and their levels rise when they are stressed. HSPs have a variety of roles in normal circumstances, but they become even more important during times of stress. An important role is one of the actions of cellular stress or HSP, in which other proteins are shifted from one cellular compartment to another. Damaged proteins are targeted for destruction by stress proteins. They aid in the disaggregation and possibly refolding of damaged proteins. Stress proteins assist newly generated proteins fold into a functional state by protecting them. Stress proteins may protect or promote some enzymatic activity during abnormal situations, and they may also act as molecular brakes for gene expression in specific cases.

Among the most important characteristics of the cellular stress response is that it is a model of inducible gene expression, making it a relatively well-understood system for investigating whether age influences transcriptional regulation. The heat shock transcription factor is essential for inducing the cellular stress response. HSF1 appears to be the major mediator of heat shock gene expression, despite the fact that several HSFs have been discovered in various species. HSF1 is a horse shoe-shaped molecule folded around it that can be found in the cytoplasm and nucleus of cells. Stress-induced signals cause cytoplasmic HSF1 to translocate to the nucleus, which may be aided by HSP. HSF1 unfolds and forms a trimeric structure capable of binding DNA with the help of other HSF1 molecules. The activated HSF1 trimer attaches to heat shock elements, which are DNA sequences, which found in the promoter region of heat shock and other genes.

CONCLUSION

The heat shock elements are made up of the guanine, adenosine, and adenosine nucleotide sequences (GAA). Although various co-factors and protein-protein interactions fine tune gene expression, interactions between HSF1 and GAA sequences in the promoter region of heat shock genes appear to be sufficient to boost heat shock gene production. Phosphorylation of HSF1 and HSF1-protein-DNA complexes has an additional impact on RNA polymerase processivity and mRNA synthesis rate. It is unclear that how HSF1 reverts to its inactive state; however phosphorylation and deconstruction by HSPs have been suggested as two possible mechanisms. Over activity of a potential inhibitor of HSF1-DNA binding may play a role in regulating stress responses, and it could be a clear way to change the stress response. Because HSF1 levels do not rise in response to stress, post-translational regulators appear to be in charge of this, which is a long-lived transcription factor.

Correspondence to: Tomoko Masui, Department of Cell Biology, Tohoku University Hospital, Sendai, Japan, E-mail: tomoko.masui@yahoo.co.jp

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