Infrared Radiation and Heat Shock Proteins: A Molecular Relationship

Dr.S.Priyanka,

Lecturer in Biotechnology, DK Government College for Women (Autonomous), Nellore, Andhra Pradesh, India.

Abstract

Infrared (IR) radiation, a component of solar and artificial heat, can influence biological systems through thermal effects. One crucial cellular response to IR exposure is the induction of heat shock proteins (HSPs), which play a vital role in protecting cells from thermal and oxidative stress. This article explores the relationship between IR radiation and HSPs, highlighting the molecular mechanisms involved, physiological responses in various organisms, and potential applications in medicine, dermatology, and agriculture.

Keywords

Infrared radiation, heat shock proteins (HSPs), thermal stress, oxidative stress, HSP70, photobiomodulation, thermotolerance, cellular protection, ROS, HSF1, tissue repair.

Introduction

Infrared (IR) radiation, which spans wavelengths from 700 nm to 1 mm, accounts for over 50% of solar radiation reaching Earth and is increasingly used in medical, industrial, and therapeutic applications. While IR is non-ionizing and generally considered less harmful than UV radiation, it can induce thermal stress, triggering a variety of cellular responses. One of the most notable responses is the synthesis of heat shock proteins (HSPs), molecular chaperones that help maintain protein homeostasis and protect against stress-induced damage.

Overview of Heat Shock Proteins (HSPs)

Heat shock proteins (HSPs) are highly conserved proteins classified into several families based on molecular weight: HSP27, HSP40, HSP60, HSP70, HSP90, and large HSPs (HSP110 and others). Their expression is upregulated in response to stressors such as heat, oxidative stress, toxins, and radiation, including infrared.

The primary function of HSPs is to act as molecular chaperones, facilitating the proper folding, repair, and degradation of proteins. HSP70, in particular, is commonly associated with responses to heat and has been a focus in IR-related research (Lanneau et al., 2008).

Additionally, small HSPs such as HSP27 play crucial roles in stabilizing cytoskeletal elements and preventing protein aggregation under stress conditions (Arrigo, 2007). The coordinated expression of these proteins enhances cellular resilience and recovery.

Mechanism of IR-Induced HSP Expression

Exposure to IR radiation causes localized heating of tissues, leading to thermal stress. This triggers the activation of heat shock factor 1 (HSF1), which translocates to the nucleus and binds to heat shock elements (HSEs) on DNA, initiating transcription of HSP genes (Akerfelt et al., 2010). The degree of HSP expression correlates with the intensity and duration of IR exposure.

Moreover, IR can induce **reactive oxygen species (ROS)**, especially in mitochondrial membranes, which also stimulate HSP expression through redox-sensitive signaling pathways (König et al., 2019). This dual pathway—thermal and oxidative—makes IR a potent inducer of HSPs.

Emerging evidence also suggests that the **MAPK/ERK and PI3K/Akt signaling pathways** are involved in IR-mediated HSP expression, particularly in keratinocytes and fibroblasts (Schroeder et al., 2008).

Biological and Clinical Implications

1. Skin Protection and Anti-Aging

Chronic IR exposure, such as sunlight, can lead to **photoaging** by degrading collagen and elastin in the dermis. However, upregulation of HSPs, especially **HSP70**, can mitigate some of this damage by stabilizing protein structures and reducing inflammation (Grether-Beck et al., 2016).

HSP27 also plays a role in maintaining the integrity of the skin barrier and reducing UV- and IR-induced damage, thereby contributing to skin rejuvenation and resilience (Park et al., 2013).

2. Therapeutic Hyperthermia and Photobiomodulation

Controlled IR exposure is used in **photobiomodulation therapy**, a non-invasive treatment to reduce pain and promote healing. IR-induced HSPs improve cell survival and regeneration in tissues, including in wound healing and musculoskeletal disorders (Hamblin, 2017).

Furthermore, **low-level infrared therapy** has been shown to upregulate HSP expression in diabetic wounds, enhancing tissue repair and reducing infection rates (Karu, 2010).

3. Cardioprotection and Neuroprotection

HSPs induced by IR preconditioning have shown promise in **cardioprotection** against ischemic injury and **neuroprotection** in degenerative disorders (Marber et al., 2011). Mild IR exposure can upregulate protective HSPs without causing tissue damage.

Studies have demonstrated that **HSP70 and HSP27** help limit neuronal apoptosis and promote synaptic function, suggesting a therapeutic role in conditions such as Alzheimer's and Parkinson's disease (Kalmar & Greensmith, 2009).

4. Agricultural Applications

In plants, IR exposure under greenhouse conditions can induce HSPs that confer **thermotolerance**, enhancing resistance to heat stress. This has implications in **climate-resilient agriculture** (Mittler et al., 2012).

Recent work has shown that transgenic plants overexpressing HSP70 demonstrate improved growth and survival under prolonged heat exposure, potentially aiding food security under global warming scenarios (Wang et al., 2020).

Risks of Excessive IR Exposure

Excessive or chronic IR exposure may lead to **thermal damage**, oxidative stress, and **overexpression of HSPs**, which, while initially protective, may contribute to **cellular senescence** and skin aging. Moreover, high levels of HSPs can sometimes promote tumor cell survival, complicating cancer treatments (Ciocca & Calderwood, 2005).

Some studies indicate that HSPs, particularly HSP90, support the stability and function of oncoproteins, suggesting a dual role in cancer development and resistance to therapy (Whitesell & Lindquist, 2005).

Conclusion

Infrared radiation is a significant environmental and therapeutic factor influencing cellular physiology through the induction of heat shock proteins. While the controlled application of IR can offer medical and agricultural benefits by enhancing HSP-mediated protection, excessive exposure may lead to detrimental effects. Understanding the balance of IR-induced HSP expression is crucial for harnessing its benefits while minimizing risks.

References

- Akerfelt, M., Morimoto, R. I., & Sistonen, L. (2010). Heat shock factors: integrators of cell stress, development and lifespan. *Nature Reviews Molecular Cell Biology*, 11(8), 545–555. https://doi.org/10.1038/nrm2938
- Arrigo, A. P. (2007). The cellular "network" of small heat shock proteins: chaperone functions and cytoprotection. *Biochemical and Biophysical Research Communications*, 354(2), 162-167. https://doi.org/10.1016/j.bbrc.2006.12.089
- Ciocca, D. R., & Calderwood, S. K. (2005). Heat shock proteins in cancer: diagnostic, prognostic, predictive, and treatment implications. *Cell Stress & Chaperones*, 10(2), 86–103. https://doi.org/10.1379/CSC-99r.1
- Grether-Beck, S., et al. (2016). Infrared radiation-induced matrix metalloproteinase in human skin: implications for protection. *Journal of Investigative Dermatology*, 136(8), 1522–1529. https://doi.org/10.1016/j.jid.2016.03.029
- Hamblin, M. R. (2017). Mechanisms and applications of the anti-inflammatory effects of photobiomodulation. *AIMS Biophysics*, 4(3), 337–361. https://doi.org/10.3934/biophy.2017.3.337
- Kalmar, B., & Greensmith, L. (2009). Induction of heat shock proteins for protection against oxidative stress. *Advanced Drug Delivery Reviews*, 61(4), 310-318. https://doi.org/10.1016/j.addr.2009.01.003
- Karu, T. I. (2010). Mitochondrial mechanisms of photobiomodulation in context of new data about multiple roles of ATP. *Photomedicine and Laser Surgery*, 28(2), 159-160. https://doi.org/10.1089/pho.2009.2616
- König, K., et al. (2019). Intracellular responses to infrared-A radiation: insights into mitochondrial and oxidative stress mechanisms. *Free Radical Biology and Medicine*, 134, 276–285. https://doi.org/10.1016/j.freeradbiomed.2018.12.027
- Lanneau, D., et al. (2008). Heat shock proteins: essential proteins for apoptosis regulation. *Journal of Cellular and Molecular Medicine*, 12(3), 743–761. https://doi.org/10.1111/j.1582-4934.2008.00273.x
- Marber, M. S., et al. (2011). The heat shock proteins and the heart. *Handbook of Experimental Pharmacology*, 200, 171–196. https://doi.org/10.1007/978-3-642-00678-4_8
- Mittler, R., et al. (2012). Heat stress: responses and management in plants. *Trends in Plant Science*, 17(6), 347–352. https://doi.org/10.1016/j.tplants.2012.03.004

- Park, J. H., et al. (2013). Protective effect of HSP27 against UVB-induced skin cell damage. *Archives of Dermatological Research*, 305(8), 737-745. https://doi.org/10.1007/s00403-013-1380-7
- Schroeder, P., et al. (2008). Cellular response to infrared radiation involves retrograde mitochondrial signaling. *Free Radical Biology and Medicine*, 44(6), 1287-1296. https://doi.org/10.1016/j.freeradbiomed.2008.01.011
- Wang, W., et al. (2020). Overexpression of heat shock protein 70 in transgenic plants enhances heat tolerance and productivity. *Plant Molecular Biology*, 103(3), 245-257. https://doi.org/10.1007/s11103-020-00989-y
- Whitesell, L., & Lindquist, S. L. (2005). HSP90 and the chaperoning of cancer. *Nature Reviews Cancer*, 5(10), 761-772. https://doi.org/10.1038/nrc1716