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Short Communication

The effect of the global warming and environmental temperature on the animal's molecular response and enzymatic activity

Abstract

The impact of climate change has been significant to threaten human health via different parameters. Temperature is a dangerous abiotic factor which affects organisms on its ecological level through infiltrating its molecular and cellular levels. Temperature is a measure of the kinetic energy of the molecules in a system. Environmental temperature has direct effect on molecular response and enzymatic activity of animals. This article is a short communication of the effect of the global warming and environmental temperature on the animal's molecular response and enzymatic activity.

Keywords: environmental temperature, climate change, molecular response, enzymatic activity

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Introduction

The intergovernmental panel on climate change officially released an important report that addressed the global warming impact on living phenomena on the earth.[1] The environmental temperature might have many fluctuations for many creatures throughout their lives. This effect may occur yearly, seasonally, or daily and usually does not remain constant. There is a question to ponder on, and that is that how an organism struggles with long-term or severe temperature changes.[2] Initial heat shock research investigates the mechanisms behind the response to critical heat stress by observing the heat shock in Drosophila. In this study, puffs characteristics had been studied in the salivary gland chromosomes of Drosophila.[3] Later, it was understood that these studied chromosomal puffs were associated with RNA synthesis and heat shock protein expression. Since 1974 up till now, it is well known that heat shock protein family including (e.g, Hsp100, Hsp90, Hsp70, Hsp60, Hsp40 and small heat shock protein families) are regulated under "heat shock response" mechanisms in almost all organisms.[4] The heat shock proteins are grouped by size or categorized by function. They are also famed as molecular chaperones because they are involved in protein folding. It has also been reported that they are preventing the formation of protein accumulation inside the cell.[5] Additionally, it has been reported that they have functions in ATP-independent` (small heat shock proteins) ATP-dependent (Hsp60, Hsp70) mechanisms.[6] Initial studies revealed that the binding of the heat shock factor (HSF) to cis-regulatory heat shock element (HSE) regions induced heat shock regulatory networks. Furthermore, interaction between Hsp70 with HSF and can cause heat shock auto regulation or block it.[7]

Researchers have approved that the heat stress response is a complicated mechanism. Research has also confirmed that the heat shock response has a variation in the initiation or termination timing or in the stress intensity. It furthermore depends on different types of Hsps in different organisms. It also may comprise from the induction of many other non Hsps genes.[4,8-11]

The comparison between Drosophila and mussel *Mytilus trossulus* showed that in the Drosophila heat shock protein is synthesized during heat stress, but it will only be expressed after stress in mussel *Mytilus trossulus*. A range of HSPs proteins involved in response to heat were detected by faint SDS-PAGE bands in marine snails, yeast, and mussels. Hsps protein also maintains a fundamental influence for thermos-tolerance in yeast.[12]

Moreover, recent work on Drosophila and yeast has exposed no limitation in the binding target of the HSF to heat shock proteins. This confirmed that almost 3% of genomic loci are available as binding target.[13] The recent interest in gene and mechanisms characterizations has risen by the development of genomic tools. The accessibility to the heat shock response studies by gene expression monitoring on a wide-ranging scale by high throughput technology and quantitative PCR has increased.[14] Post research has shown that the level of change in gene and protein expression are not essentially correlated. Recent researches have confirmed that by some protein regulations. For instance (e.g., elongation factors) showed that these proteins are expressed at the translational level, but not in protein level or vice versa. This means that an increased in this gene expression generally does correlate with an increase in their protein expression.[15] Moreover,

additional research on Drosophila confirmed a parallel linkage between genomic and metabolomic profiles resulting in heat stress.

Previously, most studies had focused on simple modeled organisms such as prokaryotes, Drosophila, and yeast. Recent studies; however, focused on the heat shock response in higher organisms.[12] Heat stress can cause many types of changes, fluctuations or variations in higher organisms or animals? Higher organisms including animals are living in different habitat and are widely exposed to a range of biophysical challenges. They are widely exposed to temperature fluctuations. These creatures may counteract these challenges by increasing or decreasing their body temperatures through adaptation process.[16]

A noteworthy example is the T. funerals an aquatic animal which was monitored in a study for 26 days from March-April. Nearly after half of the 26 days, the water temperature was midday low, but the body temperature was 27°C, its body temperature was high enough to induce Hsp70 and Hsp90 gene expressions.[17]

Fascinatingly, in the similar heat stress condition, before midday (2.5h at 30°F), Hsp70, Hsp38, Hsp90, and Hsp77 expression was increased and after 6h, 30min., 6h, and 14h it dropped to normal levels, this showed that *T. funebrails* can tolerate the stress response during the two low-temperature waves.[17] In addition, the up-regulated HSPs levels that contribute to survival with reversible protein denaturation also exist following heat stress in the studied area. Reversible protein denaturation is the mechanism that can handle the reversible denaturation through protein degradation.[16]

There are some studies on the recovery from temperature fluctuation of intertidal specious such as mussel *M. trossulus* and crab, Petrolisthes cinctipes. Studies on the heat shock response in these species have confirmed the immersion of Hsp70 and Hsp90 in reversible protein denaturation.[18] In this stage, a question arises that, what other mechanisms might be affected in response to the thermal stress? And a serious question will rise, which does of the temperature have effect on the enzymatic Activity?

All enzymes have a range of temperatures for their activity. In eukaryotes the enzymes have an optimum temperature that is the best reaction for their optimum enzymatic activity which in human is around 37°, 98.6°F, the normal body temperature. Enzymes activity has interdependent interaction with temperature.[19] All the animals have the adaptation capacity to the limited range of the environmental temperature. Animals from hot climates such as desert and tropical climates adopt their enzymatic activity to the highest optimum range while animals from cold climate adapt their enzymatic activity to the lowest optimum range.[20] Even though that the animals have this adaptation capacity to the temperature limits, still there is a limited tolerance range for their enzymatic activity and survival.[21] This limited ranges are the two ends of an enzyme activity in which the enzyme activations start up to the protein break down. Enzymes are proteins and they will break down at temperatures above 40 degrees Celsius or 104°F.[22]

Enzymes are protein molecules that would be activated in their tertiary structure. An enzyme may become inactive by an inhibitor or under advert thermal circumstances. The enzyme activity would reach to its highest range in the optimum thermal condition and will decrease in high temperature due to denaturation. Most of the animal enzyme will lose their activity above 40°C. In high temperature the active site of the enzyme will be denatured and loose its 3D structure. So, temperature has a strong effect on enzyme activity.[23] And the deficiency in enzymatic activity will subsequently case an cause respiratory diseases due to the Cardiovascular disease, mental health,[24–27] and different types of the cancer.[28]

Conclusion

Even though the IPCC reports which addressed the global warming impact on earth had been officially released in 2014, the effect of temperature on animal health had been neglected. It's clear that the environmental temperature has a direct effect on body temperature and subsequently on the enzymatic activity as an environmental stressor. All animals are threatened by the fluctuations in environmental temperature, caused by global warming. It has become critical to determine the scope of molecular research on climate change and the associated animals and human health impact. So, to the authors point of view, such studies that report gene-environment interaction under different circumstance, contribute to make more insights into the role of the genes, proteins and cell structures in living organisms' adaption to the advert climate circumstances.

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Conflicts of interest

The author declares that there are no conflicts of interest.

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