The Relation between Biological Consequences and Temperature on Some Non-Mammalian Species

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Abstract: The number of reports on the effects of temperature is still increasing because of the temperature is one of the most encountered stressful factors in the environment, thus it deemed important to survey the literatures for effects of temperature on the biological consequences. The objective of this review was to establish the thermoregulatory response and adaptation of some non-mammalian species during temperature. Although, there was relative scarcity of information on the relation between oxidative stress and antioxidant enzymes during temperature, this review great interest to elicit this relation in non-mammalian species. Here, this review suggests that, the increase in the oxidative stress due to temperature may be a reason for such decrease and exhaustion of antioxidant enzymes and a sequence of cellular injury or death, because of increased endogenous production of the free radicals. However, there was exception in this hypothesis because this argument is still ambiguous because of the difficulties of the direct observation of the active oxygen species in the biological systems due to their short lifetime. Taken together, because of one of the most important functions of heat shock protein is to protect the organisms from the deleterious effects of temperature, thus, it can be hypothesized that the formation of heat shock protein and antioxidant enzymes may be related to the changes in the levels of free radicals in non-mammalian species during temperature.

Key words: Thermoregulation, cellular injury, thermal protection, oxidative stress, antoxidative enzymes

Introduction

Window on the recent researches, heat stress reduces feed intake, digestibility of nutrients, egg production, egg weight and yolk index (Smith and Oliver, 1972: Wolfenson *et al.*, 1979; Donkoh, 1989) in poultry. Also, heat stress causes the release of corticosterone and catecholamine and initiates lipid peroxidation in cell membranes (Edens and Siegel, 1975; Freeman and Crapo, 1982; Sahin *et al.*, 2001). Because of temperature influences some functions of organisms strongly and others weakly (Rome *et al.*, 1992), this review will explain the various effects of temperature on non-mammalian species.

Thermoregulation
While, endothermic animals employ physiological and mechanism of behavior to keep core temperatures within a range compatible with life activities in spite of wide variations in ambient

temperature, the ectothermic animals are dependent on the heat from the environment to achieve core temperatures compatible with their activities.

In view to lizards, during heat stress, both behavioral and physiological thermoregulatory responses are employed (Schmidt-Nielsen and Dawson, 1964; Dawson, 1967). The turtles, *Terrapene ornata* (Riedesel *et al.*, 1971; Sturbaum and Riedesel, 1974), *Testudo sulcata Miller* (Cloudsley-Thompson, 1971) and *Chrysemys marginata belli* and *Chelydra serpentine* (Baldwin, 1925a, b) salivate copiously in response to high ambient temperatures. Salivation in response to high ambient temperatures is called frothing and was adopted as a good indicator of maximum physiological thermoregulation in the ornate box turtle (Sturbaum and Riedesel, 1974). *Terrapene ornata* can keep core temperatures at least 8.5°C below a 48°C ambient temperature for as long as 4 h and 10.5°C below a 51°C ambient temperature for 3 h. Evaporative water loss plays a major role in thermoregulation of this species in heat stress (Sturbaum and Riedesel, 1974). Moreover, the thermoregulatory responses to heat stress of *T. ornata* have been studied previously and it was demonstrated that this species exhibits good physiological thermoregulatory mechanisms during exposure to hot environments (Sturbaum and Riedesel 1974, 1977). Because *T. Carolina triunguis* is a woodland species, it can be hypothesized that this species does not thermoregulate physiologically in hot environments as well as *T. ornata.*

Effective thermoregulation is a function of circadian phase and arousal state in many endothermic animals (Aschoff, 1983; Heller *et al.*, 1983). In addition, thermoregulation in free-living birds is complicated by the fact that many species live and nest in exposed and often severe environments where thermoregulatory demands are intense. In the early ontogenetic phases of poultry, rapid changes in thermoregulatory abilities occur. However, the time at which adult levels of thermoregulation are reached differ among the species. The adult level is considered to be reached when the birds are able to keep a constant body temperature (±2K) under changing ambient temperatures (Tg) or when body temperature increases at low Tg (Hammel's thermoregulatory model) (Hammel, 1968). This phase takes about 22 days for laying strains of poultry (Randall, 1943; Freeman, 1965), about 31 days for Chinese quail (Bernstein, 1973), 14 days for Japanese quail (McNabb *et al.*, 1972) and roughly 40 days for the turkey (Nichelmann *et al.*, 1976). Several authors support the hypothesis that the lower body temperature of younger animals is due to their lower thermoregulatory set point and is caused not only by an unfavourable surface-volume ratio, but also by their insufficient plumage (Myhre, 1978; Pedersen and Steen, 1979; Hissa *et al.*, 1983; Dawson *et al.*, 1976).

On the other hand, there were several tested numerous species of aquatic ectotherms in the Ichthyotron, including vertebrates ranging from lamprey ammocoete larvae (Reynolds and Casterlin, 1978b) to sharks (Reynolds and Casterlin, 1978a), bony fishes (Reynolds and Casterlin, 1976; 1977; Reynolds *et al.*, 1976) and anuran amphibian larvae (Casterlin and Reynolds, 1977). All these vertebrates exhibited a range of voluntarily occupied (preferred or non-avoided) temperatures covering a span of less than 10°C, indicative of fairly precise thermoregulatory behaviour (the potential range of available temperatures spans from 0 to 50°C). The 25°C range of voluntarily occupied temperatures exhibited by *Limulus polyphemus* (the horseshoe, crap, is a primitive chelicerate arthropoda) indicates that it is much less precise in its thermoregulatory behaviour than are vertebrates similarly tested, which occupy voluntarily a range spanning less than 10°C (Robertson, 1970). *Limulus polyphemus* also exhibited the highest voluntarily occupied temperature (40°C) of any animal we have so far tested in the Ichthyotron. That *Limulus polyphemus* can tolerate this temperature for at least 8h is indicative of its considerable eurythermality (Frankel, 1960). Therefore, it can be inferred that, the ability to maintain normothermia during a heat load is the result of sufficient cooling mechanisms and/or reduced heat production in non-mammalian species.

Strategies Used by Reptiles to Regulate Their Body Temperature

In a cold environment, reptiles warm themselves by absorbing heat from the sun. This can be done by basking. Lizards orient their bodies at right angles to the sun's rays in order to maximize their exposure and seek inclined surfaces to achieve the best orientation with respect to the blunting rays of the morning sun (Florides *et al.*, 1999). In the desert where the ground becomes warmer than the air, lizards often press their bodies close to the surface, shifting slowly from side to side in the loose sand to secure better conduction of heat. On a rocky mountain side that warms up more slowly, they do their basking on pats of dead grass that insulate then from the cold ground (Bogert, 1959). Once they reach their working temperature they become active. Also when they are too hot they seek the shade or move underground. Later in the afternoon, they emerge from the shade and lie parallel to the sun's rays. In addition to the above behavioural factors, a reptile can control its body temperature by the following physiological means (Florides *et al.*, 1999):

Changes in Colour

Some lizards regulate the rate at which they absorb heat by pigmentation. When the reptile is cold, black pigmented cells (melanophores) of their skin expand, thus darkening the skin colour and increasing the rate at which the radiant energy is absorbed.

Respiration

When the air temperature exceeds body temperature, the heat exchange due to inspiration will depend both on the rate of ventilation and the rate of evaporation. Heat will be gained as the inspired air cools to body temperature and heat will be lost as water from the lungs evaporates. If it is assumed that the expired air is at body temperature and is saturated, then evaporation will be least when the body temperature is least and the heat acquired from the inspired air will be greatest when the difference between the body and air temperature is greatest (Bartholomew and Tucker, 1963).

Blood Flow

The rate of heat exchange between the reptile and the environment depends on the volume of blood flowing per unit time between the core and the surface of the body. The marked difference between the heart rates of certain lizards during heating and cooling suggest that circulation augments heat exchange during heating and retards it during cooling. While being heated, lizards maintain their heart rate at or near maximum and conversely, during cooling they reduce their heart rate to the minimum. Also circulatory factors other than heart rate must be involved in the control of thermal conductance (Bartholomew and Tucker, 1963).

Metabolism

All living cells generate small amounts of heat as a by-product of the chemical processes occurring within them. This chemical activity is called metabolism. Active cells, especially muscle cells generate much more heat than resting cells. The rate of chemical reactions depends on the temperature. This rate increases rapidly with increasing temperature and in general it can be considered that for every $10°C$ change in body temperature, the rate of physiological processes double. This phenomenon is called the Q_{10} effect (McMahon, 1991). Generally, metabolic rates in reptiles are low and thermogenesis is considered an insignificant factor in the heat balance of these reptiles. Also during activity, metabolic heat yearns tend to be offset by increased heat loss through increased evaporation.

Furthermore, eurythermic animals maintain functional and metabolic activity in a thermally fluctuating environment over a relatively large temperature range (Lehti-Koivunen and Kivivuori, 1994). They may encounter seasonal, diurnal and/or short-term temperature changes. Generally, they can also adjust physiologically to thermally variable conditions (thermal compensation).

Cellular Effects of Temperature

Indeed, it is known that natural fluctuations in environmental temperature can result in some induced cellular stress responses (Minier *et al.*, 2000; Hofmann and Somero, 1995). Exposure of cells or organisms to thermal stress can result in alterations in the integrity of the nucleolus as well as adversely affect the structure and function of the centrosome (Brown *et al.*, 1996). A delay in cell cycle progression and the inability of cells to enter mitosis have also been observed (Welch, 1990; Yang and Takahashi, 1999). Furthermore, the heat shock is a genotoxicant; causes nuclear abnormalities, chromosomal aberrations and damage to DNA (Anitha *et al.*, 2000). The data obtained in vivo showed an increasing amount of DNA damage at increasing temperatures in cells directly withdrawn from the mussels (Buschini *et al.*, 2003).

The exposure of animals to constant heating can cause death or handicap (Díaz and Bückle, 1999). Paladino *et al.* (1980) found that Catfish can resist a rises in environmental temperature for a time but muscular spasms and loss of equilibrium will occur eventually which is defined as the end point of critical thermal maximum (CTMax). These latter authors also reported that, the CTMax is a good indicator of the thermal resistance of species and is an ecologically valuable method. It is non-destructive and identifies the temperature at which the first signs of stress occur.

On the other hand, the ambient temperature effects on different blood components (Moye *et al.*, 1969; Vo *et al.*, 1978), on gases exchange (Tucker, 1968; Maeda *et al.*, 1992), on some metabolites (Marzouk, 1992) and on the changes of the air sacs composition (Bretz and Schmidt-Nielsen, 1971; Michael and John, 1983) was reported for different bird's species.

In general, the weight lost during exposure to the hot environments is indicative of water used for evaporative cooling. Because water was lost during heat exposures the hemodilution that occurred was totally unexpected. This response to heat does make sense because a lower hematocrit does result in a lower blood viscosity which reduces peripheral resistance and permits the blood to be circulated more easily thus increasing the heat delivery capacity of the blood. Thus, the drop in hematocrit could have been caused (Abdel-Salam and Mosaad, 1996) by (1) sequestering of the red blood cells by some organ or (2) an increase in plasma volume at the expense of intracellular and/or interstitial fluid. Moreover, birds exposed to the ambient temperature (45°C) showed a highly significant difference in their RBCs count, haemoglobin content and packed cell volume as compared to the normal values (Abdel-Salam and Mosaad, 1996). Also, the changes in metabolism that occur in turtles when exposed to different environmental temperatures are represented in part by changes in blood gas and substrate composition (Frankel *et al.*, 1966). In addition, an increase in blood pCO_2 with increasing temperature (Robin, 1962) and a decrease in blood glucose with cold torpor (Rapatz and Musacchia, 1957) have been reported.

Thermal Protection

In the context of thermal stresses, several studies have shown the possibility of improving survivability, growth and feed efficiency of heat-stressed chickens by prior exposure to controlled thermal stressors (Arjona *et al.*, 1988; Yahav and Hurwitz, 1996; Yahav and Plavnik, 1999; Yalcin *et al.*, 2001). When living organisms are exposed to thermal and non thermal stressors, the synthesis of most proteins is retarded but a group of highly conserved proteins known as heat shock

protein (hsp) are rapidly synthesized (Etches *et al.*, 1995). It is well documented that one of the most important functions of hsp is to protect organisms from the toxic effects of heating (Barbe *et al.*, 1988). Hsp may play roles in protein assembling and disassembling (Pelham, 1986), protein folding and unfolding (Randall and Hardy, 1986) and protein translocation (Murukami *et al.*, 1988).

Indeed, a close relationship between the threshold temperature for increased hsp70 levels and the thermal preference of fish (Poeciliopsis) was demonstrated by Hightower *et al.* (1999). Such thermal protection has been reported for cultured mammalian cells (Kampinga *et al.*, 1994; Michel *et al.*, 1994) as well as in the cells of medaka (Oryzias, pisces, Arai *et al.*, 1994), winter flounder (*Pleuronectes americanus*) (Brown *et al.*, 1992). In fish, hsp70s have been sequenced in different species including zebrafish (hsc70: Graser *et al.*, 1996; Santacruz *et al.*, 1997), tilapia (hsp70: Molina *et al.*, 2000), medaka (hsp70 and hsc70: Arai *et al.*, 1995) and rainbow trout (hsp70 partial sequence: Kothary *et al.*, 1984, Zafarullah *et al.*, 1992). The trout hsc71 and hsp70 amino acid sequences are 80 identical, whereas the human and trout hsc70 sequences are 94 identical (Zafarullah *et al.*, 1992). Furthermore, in fish, there appear to be different protein isoforms for hsp70, however, no variation in protein isoforms was evident for hsc70 (White *et al.*, 1994; Norris *et al.*, 1995; Place and Hofmann, 2001). Moreover, Zebrafish hsc70 mRNA expression was induced in embryos by heat shock (Santacruz *et al.*, 1997) and during caudal fin regeneration (Tawk *et al.*, 2000). Slightly enhanced hsc70 mRNA and protein expression was also observed after heat shock of two medaka cell lines (Arai *et al.*, 1995), whereas heat shock did not affect hsc70 expression in topminnow hepatocytes (White *et al.*, 1994; Norris *et al.*, 1995). In conclusion, these results led to hypothesize that the pattern of the cellular hsp70 response may have a close relationship with the thermal tolerance of these fish species.

There is considerable evidence that the synthesis of hsp70 is temperature dependent (Wang and Edens, 1998) and thus hsp70 response is considered as a cellular thermometer (Craig and Gross, 1991). Zulkifli *et al.*, (2002) found that exposing broiler chickens to high temperatures increased the induction of hsp70 in the brain. Higher hsp70 expression in the livers (Gabriel *et al.*, 1996), blood leucocytes (Wang and Edens, 1998), lungs and hearts (Yahav *et al.*, 1997) of hyperthermic chickens has also been reported. It is well documented that hyperthermia may increase the activity of the heat shock transcription factor, which enhances hsp70 mRNA synthesis and consequently hsp70 concentration (Craig and Gross, 1991). It has become increasingly clear that stressful experiences during the neonatal stage can elicit hsp mRNA transcription but the RNA may have been 'sequestered' and not translated until exposure to heat stress later in life (Craig, 1985). Furthermore, Wang and Edens (1998) found that heat conditioning, via a daily 1 h exposure to 41°C, enhanced in vitro hsp expression in leucocytes of poults heat conditioned for 1, 3 or 5 week. On the contrary, Zulkifli *et al.* (2003) suggest that conditioning of chicks by exposure to moderate heat stress has no profound impact on induction of hsp70 synthesis as compared to those of controls. The contradiction between these results could be attributed to differences in poultry species, samples collected for hsp70 analysis and protocol of heat challenge.

A strong correlation between the cellular heat shock response, which is defined as cellular induction of proteins including hsps under heat stress and the thermal tolerance of animals also has been reported (Parsell and Lindquist, 1993; Feder and Hofmann, 1999). Heat shock proteins have been implicated in the development of thermotolerance, in protein folding and translocation, in steroid receptor protein binding and in the onset of human autoimmune diseases, in addition to hyperthermia, a wide range of hsp inducers have been reported, such as hydrogen peroxide, ethanol, oxidizing quinines and uncoupled oxidative phosphorylation reactions, to mention a few (Omar and Lanks, 1984; Omar

and Pappolla, 1993). Other researchers have shown that the cellular defense response to either oxidative or hyperthermia-induced stress includes, in addition to induced hsp synthesis, an induction or increase in intracellular antioxidant enzyme activities, particularly superoxide dismutase (Freeman and Crapo, 1982; Omar *et al.*, 1987). In fact, the role that hsp70 plays in influencing thermal tolerance of a whole animal is not clearly understood (Parsell and Lindquist, 1993).

Effect of Temperature on Oxidative Stress and Antioxidant Enzymes

Oxidative stress has been defined as a disturbance in the balance between the production of Reactive Oxygen Species (ROS), or free radicals and antioxidant defenses, which may lead to tissue injury (Halliwell, 1994). Also, free radical can be defined as any chemical species that contains unpaired electrons in their outer orbit and thus can react virtually with all cell components (Slater, 1984; Collier *et al.*, 1992; Przekwas *et al.*, 2003). Although, reactive oxygen species are crucial to normal biological processes, they are potentially dangerous (Zimmerman, 1998; Toyokuni, 1999) and are commonly referred to as prooxidants (Mates *et al.*, 1999). The reactive oxygen intermediates (including superoxide and hydroxyl radicals as well as hydrogen peroxide) can cause direct cellular injury by including lipid and protein peroxidation and damage to nucleic acid (Takeda *et al.*, 1984; Richard *et al.*, 1990). Common examples of the free radicals include the hydroxyl radical (OH), superoxide anion (O_2), transition metals such as iron and copper, Nitric Oxide (NO) and peroxynitrite ($ONOO^-$) (Dormandy, 1983). Moreover, Betteridge (2000) reported that, the free radicals can be produced by several different biochemical processes within the body including: (1) The reduction of the molecular oxygen during aerobic respiration yielding superoxide and hydroxyl radicals; (2) By products of chemical reactions such as oxidation of catecholamine and activation of the arachidonic acid cascade product electrons, which can reduce molecular oxygen to superoxide; (3) Production of superoxide and hypochlorous acid (HOC1), a powerful oxidant, by activated phagocytes and (4) Nitric oxide production by vascular endothelium and other cells.

Moreover, the role of oxidative stress as a potential mechanism has been investigated by relatively few laboratories and in relatively few animal models (McArthur and Sohal, 1982; Sohal *et al.*, 1985; Farmer and Sohal, 1987). In general, oxidative damage to lipids and proteins and generation of reactive oxygen species have been reported to be increased at higher temperatures, although exceptions are apparent (Farmer and Sohal, 1987). The electron transport chains of mitochondria and endoplasmic reticulum are considered as the major intracellular sources of superoxide. Although the components of these chains pass the bulk of electrons onto the next component of the chain, some appear to "leak" electrons to oxygen (Halliwell and Gutteridge, 1998). Thus, the rate of superoxide radical production is increased at elevated ambient oxygen concentrations. There is enough evidence that the increase in metabolic rate and higher caloric intake enhance the oxidative stress in tissues, which supports the idea that, the more electrons are passed on to the electron transport chain, the more superoxide radicals are produced (Sohal and Weindruch, 1996). However, with few data available from vertebrate ectotherms, the expectation of broad applicability of such results (Sohal and Alien, 1990) may not be warranted.

It is well documented that high ambient temperature increased malondialdehyde (MDA) level in serum and tissue of laying hens (Naziroğlu *et al.*, 2000; Sahin and Kucuk, 2001) and Japanese quails (Sahin *et al.*, 2002) and this increase in MDA concentration consider the indicator of a lipid peroxidation, LPO (Halliwell and Gutteridge, 1989; Sahin *et al.*, 2002). The increased levels of thiobarbituric acid-reactive substances (TBARS) suggest a net increase in the levels of oxygen free radicals which could be due to their increased production and/or decreased destruction

(Giugliano *et al.*, 1996). In addition, it is reported that GSH is protective to cell membrane *in vivo* in as much as it is a substrate for glutathione peroxidase, GSH-Px, as well as being able to react with various aldehydes produced during peroxidation, thus, protect the -SH groups of membrane proteins (Halliwell and Gutteridge, 1998).

Antioxidant enzymes are an important protective mechanism against ROS and, like many other biochemical systems, their effectiveness may vary with the stage of development and other physiological aspects of the organism (Halliwell and Gutteridge, 1999; Livingstone, 2001). Also, both the development of (molecular) biomarkers (Livingstone *et al.*, 2000) and understanding of basic pro-oxidant/antioxidant processes (Livingstone, 2001) require knowledge of how the biochemical systems are influenced by key exogenous/endogenous factors such as age and sex. The balance between production and elimination of ROS is maintained by antioxidants and enzymes (Zimmerman, 1998; Toyokuni, 1999). For example;

- Superoxide Dismutase (SOD) catalyzes the conversion of superoxide anion radical to H_2O_2.
- Catalase (CAT) reduces H_2O_2 to water;

$$2H_2O_2 \rightarrow 2H_2O + O_2$$
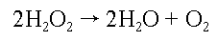
- Glutathione peroxidase (GSH-Px) acts in conjunction with other enzymes to H_2O_2 and to terminate lipid peroxidation. Changes in antioxidant activities may occur under conditions that alter the rates of formation of reactive oxygen radicals.
- The (GSSGR) enzyme catalyses the reduction of glutathione in the presence of NADPH, which was oxidized to $NADP^+$;

$$NADPH+H^++GSSG \rightarrow NADP^+ + 2GSH$$

(GSH is Reduced Glutathione form, while GSSG is the oxidized one).

Furthermore, Antioxidant systems (glutathione peroxidase, superoxide dismutase and vitamins A, C and D) are important in scavenging free radicals and their metabolic products, as well as in maintaining normal cellular physiology restoring depletion of various antioxidants in stressed poultry (Halliwell and Gutteridge, 1989). Increased excretion of H_2O_2 in C. glomerata at 26°C compared to 15°C has been shown for antifouling purposes (Chbo *et al.*, 2004). Also, vitamin A can function as an effective radical-trapping antioxidant (McDowell, 1989). There is ample evidence that vitamin A is a very effective quencher of singlet oxygen (McDowell, 1989). Also, Sahin *et al.* (2001, 2002) reported that serum vitamin A concentration decreases upon stress conditions. The plasma antioxidant vitamins and minerals such as vitamin C, E, folic acid and zinc levels declined and oxidate damage increased in stressed poultry (Feenster, 1985; Sahin *et al.*, 2002). Generally, heat stress reduced the concentrations of vitamins E, C and A (Feenster, 1985; Klasing, 1998) and it initiates lipid peroxidation in cell membrane (Sahin *et al.*, 2001). In addition, environmental stress has been demonstrated to cause an increase in oxidative stress and an imbalance in antioxidant status (Halliwell and Gutteridge, 1989; Klasing, 1998; Sahin *et al.*, 2001).

From the pre-said studies, it is also worth mentioning that the antioxidant status is modified with ambient temperature. Indeed, antioxidant enzyme concentrations (SOD, CAT and GSH-Px) are positively correlated with temperature, but different enzyme systems are affected to variable extent when the species are compared. These results demonstrate that the oxidative consequences of a given environmental condition may vary among different species. Furthermore, it is not reliable to predict

which antioxidant enzyme system is affected, if only the LPO levels are taken into consideration. In as much as GSSG was in accordance with GSH-Px activities, GSSG may be more reliable marker compared to LPO. These findings support the idea proposed by Dotan *et al.* (2004); that is, lipid peroxidation cannot be used as a universal criterion of oxidative stress." Therefore, second oxidative stress marker such as GSSG, e.g., conjugated dienes is strongly recommended for evaluation of antioxidant status. Finally, according to antioxidant theory, when the concentration of antioxidant vitamins decreases, lipid peroxidation increases in the plasma and tissues leading to damage of cell membranes (McDowell, 1989).

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