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Original Article

Assessment of the Ameliorative Effect of Vitamin A on Heat Stress Using White Albino Mice

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Abstract

BACKGROUND AND AIM: Biofortification of food and food addictives with vitamin A is basically aimed at improving vision and or preventing the occurrences of vision related diseases. This usually entails the inclusion of a defined amont of the said micronutrient in a target food. Thus, fortification of animal feed with vitamin A to ameliorate heat stress and increase animal production is possible if the ideal concentration of the said micromutrient is determined for inclusion. Therefore, Aim: this study aims at assessing the ameliorative role of vitamin A (retinol) in the management of Heat Stress (HS).
MATERIALS AND METHOD: Thirty adult male albino mice were divided into six (6) groups of five (5) rats per groups. Methodology: Group I was the normal control and was not exposed to heat stress.While groups III and IV were exposed to 57 mw/cm^2 of solar radiation at 37^{o}C, groups V and VI were exposed to 98 mw/cm^2 of solar radiation at 47^{o}C. Exposure to solar radiation lasted for 21 days during which groups II, III-VI were administered 30 mg/kg body weight of vitamin A for 21 days as well.
RESULTS: Feed intake for groups III-VI was significantly ($p<0.05$) lower than that of groups I and II. Finial body weights for groups III-VI were significantly ($p<0.05$) lower than the initial body weight. Thiobarbituric Acid Reactive Substance (TBARS) for groups III-VI was significantly ($p<0.05$) higher than that of groups I-II.
CONCLUSION: It can be deduced from this study that 30 mg/kg of vitamin A did not ameliorate varying magnitudes of heat stress studied.

Keywords:
Heat, Stress, Vitamin A, Thiobarbituric Acid, Body Weight

INTRODUCTION

Redox reaction commonly known as oxidation-reduction reaction qualifies any reaction in which there's a transfer of electron between two species. It is a common sight in a plethora of chemical reactions occurring in biological systems. Despite the importance of the oxidation-reduction reaction to life, its damaging effect is well established and is linked to its characteristic ability to generate highly reactive oxygen species which once formed initiates a chain of reactions that consumes the integrity of cellular components such as lipids, carbohydrates, deoxyribonucleic acids and more thereby undermining their biological roles (Engwa, 2018). Fortunately, the biological system is endowed with complex antioxidant defense systems which include the enzymatic system characterized by the catalase, superoxide dismutase and perioxidase and the non-enzymatic system notably the vitamin A Among others which react with free radicals to halt the chain reaction by eliminating free radical intermediates and inhibiting subsequent reactions by oxidizing themselves (Hamid *et al*., 2010). However, disruption of the antioxidant defense mechanism by an offensive factor automatically, translates to accumulation of free radicals to deleterious proportions and its attendant consequences (Amit and Priyadarsini, 2011).

In animals, heat stress occurs as a result of an imbalance between heat production within the body and its release. It can be triggered by a prolonged exposure to excessive heat from the environment in addition the metabolically generated heat in the body (Sunil Kumar *et al*., 2011). Heat stress has been implicated in enhanced generation of reactive oxygen species known for their harmful effects in the biological systems (Feng *et al*., 2008; Tan *et al*., 2010).

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Vitamin A is a fat-soluble vitamin which possesses multiple functions in supporting vision as well as the non-enzymatic antioxidant defense system (Combs, 1998). It scavenges the free radicals and reactive species by donating an electron and maintaining a chemical balance (Chaudhary *et al*., 2023). Vitamin A has been extensively involved in food fortification, a programmes which entails the inclusion of a definite amount or concentration of the said micronutrient in food. This simply suggests that such programmes with Vitamin A can be extended to animal feed if the right amount sufficiently effective to ameliorate heat stress is determined. Therefore, substantiating the need to perform this study in an effort to develop a dependable solution to heat stress.

MATERIALS AND METHOD

Chemicals

Vitamin A was purchased from Sigma Aldrich

Experimental animals

White albino mice weighing 20-25 g were purchased from the Animal House of the Department of Veterinary Pharmacology, University of Maiduguri were housed in well ventilated transparent plastic cages. The mice were exposed to 12 hours' light and dark. They were allowed uninterrupted access to feed and water. The animals were allowed 14 days acclimatize prior to commencement of the experiment. The mice were handled in accordance with International guiding principles for biomedical research involving animal use (Council for International Organization of Medical Sciences, 2012).

Animal Grouping

Thirty adult male mice were divided into six groups of five rats per group. Group I (Normal control) was fed with rat chow and water only. Group II was administered 30 mg/kg body weight Vitamin A only orally. Group III was exposed to low simulated solar radiation (57/mw/cm^2) without Vitamin A. Group IV was exposed to low simulated solar radiation (57/mw/cm^2) administered 30 mg/kg b.w of Vitamin A orally. Group V was exposed to intense simulated solar radiation (98 mw/cm^2) without Vitamin A. Group VI was exposed to intense simulated solar radiation(98 mw/cm^2) administered 30 mg/kg b.w of Vitamin A orally.

The respective groups of mice were exposed to radiation for one minute. This was repeated intermittently four times at a regular interval of one minutes for both high and low radiation. The height of the solar heat simulator (500 w lamp) on the clamp were adjusted to 60 and 45 cm respectively for low and high intensity. Exposure of animals to heat stress lasted daily for 21 days at 37^{o}C and 47^{o}C for 57/mw/cm^2 and 98mw/cm^2 respectively after which the animals were sacrificed and blood sample collected.

Biochemical Analysis

Determination of thiobabituric acid reactive substance (TBARS)

Exactly 100 µL of the supernatant was deproteinized by adding 2 mL of 14% trichloroacetic acid (TCA) and 2 mL of 0.67% thiobarbituric acid solution. The mixture was heated in a water bath at 80^{o}C for 30 minutes to complete the reaction and then cooled rapidly on ice for 5 minutes. After centrifugation at 2000 g for 10 minutes, the absorbance of the colored product (TBARS) was measured at 532 nm with a UV spectrophotometer.The concentrations of TBARS were determined in triplicate and calculated using the molar extinction coefficient of malondialdehyde -1.56×105 mol/L/cm (Yavuz *et al*, 2004; Sivonova *et al*, 2007). All TBARS concentrations were expressed in µmol/g tissue protein.

Data analysis

Data analysis was carried out using Statistical Package for the Social Sciences (SPSS) Version 23. Data were analyzed using one way Analysis of Variance (ANOVA) and differences in mean were compared with the aid of Turkey's post hoc test. P-values less than 0.05 were considered statistically significant.

RESULTS

The effect of heat stress on feed intake is shown on Figure 1, indicating that feed intake reported for groups III-VI were not significantly ($p>0.05$) but significantly ($p>0.05$) lower than that reported for group I which was the normal control and group II which was administered vitamin A only without prior exposure to heat stress.

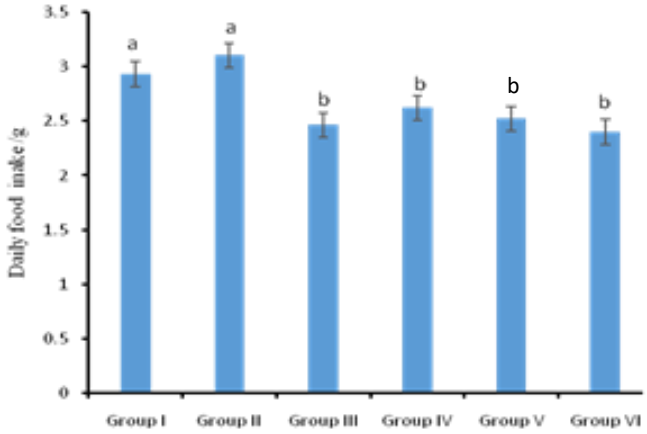

Figure 1: Effect of heat stress on feed intake
Hint: Bars with the same alphabets represents values that are not significantly ($p>0.05$) different

The body weight of mice exposed heat stress is expressed on Figure 2 showing that the final weight recorded on groups III-IV was significantly ($p<0.05$) lower than that recorded on the initial weight. However, a contrary observation was made on groups I and II.

The levels of thiobabituric acid reactive substance in mice exposed to heat stress is depicted on Figure 3 indicating that the levels of TBARS reported for groups III-VI were not significantly ($p>0.05$) different but were significantly ($p>0.05$) lower than that reported for groups I and II which also presented levels of TBARS which were not significantly ($p<0.05$) from each other.

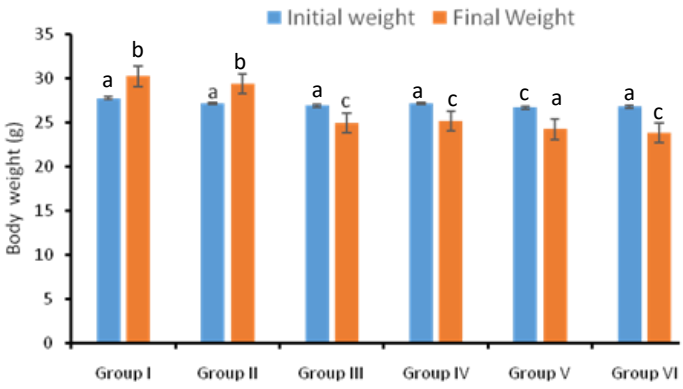

Figure 2: Body weight of mice exposed to heat stress
Hint: Bars with the same alphabets represents values that are not significantly ($p<0.05$) different

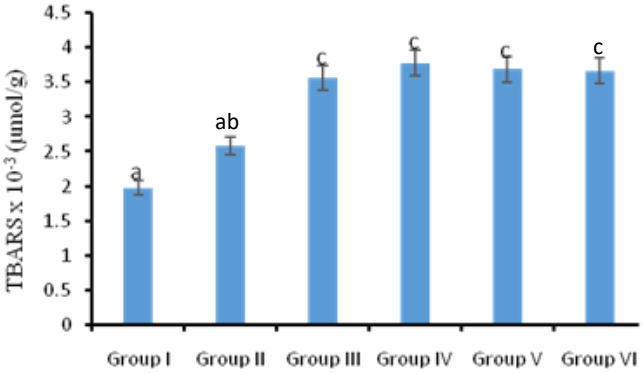

Figure 3: Thiobabituric acid reactive substance in mice exposed to heat stress
Hint: Bars with the same alphabets represents values that are not significantly ($p<0.05$) different

DISCUSSION

An animal's physiological response to stress involves a variety of systems notably the digestive systems. Heat stress can impair feeding process, nutrient absorption and utilization (Richards *et al.*, 2010 and Morera *et al.*, 2012). The reduction in body weight observed on groups III-VI that could be attributed to the stimulation of the hypothalamic axis by heat stress due to increased adiponectin and leptin levels which translates to declined feed feed consumption and consequently weight loss (Hoyda *et al.*, 2012). This observation is consistent with the finding of West *et al.* (2003) which showed that heat stress decreased intake of dry matter (DM) dairy cattle. The concentration of thiobarbituric acid reactive substances (TBARS) in plasma is a pointer to lipid peroxidation and oxidative stress. The increased TBARS levels observed on groups III-VI could be attributed to the effect of oxidative stress which could have been triggered by heat stress (Sunil Kumar *et al.*, 2011).

Conclusion: From the outcome of this study, it can be deduced that vitamin A supplementation did not ameliorate heat stress, evident by the fact that the studied heat stress (HS) indicators are obvious in exposed animals even with vitamin A supplementation. However, higher doses of vitamin A may be tried in future studies.

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