CHANGES IN SOME PHYSIOLOGICAL PARAMETERS OF ALBINO RATS AT DIFFERENT AMBIENT TEMPERATURES

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Summary

Five experimental groups with five adult male rats in each, were exposed to 20, 35, 40 and 45℃ air temperature for 50-70 minutes, and to 50℃ for 30-50 minutes, respectively. Food and drinking water were not permitted during the exposure. Blood samples were obtained by heart puncture immediately after the thermal treatment. All the rats were hyperthermic ($p < 0.01$) as compared to the controls (20℃). Hyperthermia was associated with hypoglycemia which was significant ($p < 0.01$) at 45 and 50℃ exposures. Plasma levels of GOT and GPT declined at 35 and 40℃ reaching the lowest ($p < 0.05$) level at 45℃, while at 50℃ GOT level was elevated by 45% but GPT was normal as compared to the controls. Differences between groups were significant ($p < 0.01$) for GOT and insignificant for GPT. Hematocrit value increased significantly ($p < 0.01$) at 45 and 50℃, indicating hemoconcentration. It could be concluded that severe heat stress (45 and 50℃) resulted in critical hyperthermia, hypoglycemia, disturbed liver function, body dehydration, and hemoconcentration leading to death.
(Key Words : Rat, Temperature, Stress, Hyperthermia, Hypoglycemia, Hemoconcentration)

Introduction

The metabolism of rats under hot climatic conditions varies possibly due to the lack of a true physiological regulation by evaporation (Herrington, 1940). Other possible factors determining the metabolic response of rats to heat are sex and duration of heat exposure (Khalil, 1981; Khalil and Kotby, 1982). Heat intensity as an important factor influencing metabolism was not extensively studied in rats, particularly the extremely high ambient temperatures.

On the other hand, heat adversely affects the blood of most mammals (Prosser and Brown, 1961). It decreases the life span of erythrocytes (Meyerstein et al., 1975) and the red cell volume (Jones et al., 1976), while increases the packed cell volume and the plasma loss due to body dehydration (Frankel, 1959; Hainsworth et al., 1968; Khalil and Kotby, 1982).

This study was to investigate the blood sugar and transaminases levels, and hematocrit value of mature male rats exposed to different high ambient temperatures for short durations.

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Materials and Methods

This study was conducted on five experimental groups, five adult male albino rat's of the Sprague Dawley strain in each. Rats were bred and housed in the rats colony at the Department of Animal Production, Faculty of Agriculture, Ain Shams university. All animals were maintained on an *ad libitum* balanced pelleted diet and drinking tap water. The five groups of rats were heat exposed in a thermally controlled incubator at 20 (control), 35, 40, 45, and 50℃ air temperatures, respectively. Relative humidity varied during exposure (60 90%), according to air temperature and water evaporation of rats. Food and drinking water were available during exposure.

Rectal temperature was measured immediately after heat exposure, using a ℃ thermometer. This was followed by blood sampling, using a heparinized syringe and heart puncture technique. Duration of exposure ranged between 50-70 minutes for the first four groups, and 30-50 minutes for the last group (at 50℃) in which animals were extremely exhausted and approached death.

Plasma glucose (PG) was determined according to the calorimetric method by Hyvarinen and Nikkila (1962), while glutamic oxaloacetic transaminases (GOT) and glutamic pyruvic transaminases (GPT) were determined in blood plasma according

to the Reitman and Frankel method (1957) using a commercially available reagent kits (Biomerieux).

Analysis of variance (Snedecor and Cochran, 1973) and Duncan's new multiple range test (Kramer, 1957) were performed on the data.

Results and Discussion

Mean values (± standard errors) of rectal temperature (RT), plasma glucose (PG), glutamic oxaloacetic transaminases (GOT) and glutamic pyruvic transaminases (GPT), packed cell volume (PCV) and plasma loss percentage (PL) for male albino rats at different ambient temperatures of 20 up to 50℃ are shown in table 1.

TABLE 1. MEANS (±SE) FOR SOME PHYSIOLOGICAL PARAMETERS OF ADULT MALE RATS EXPOSED TO DIFFERENT AMBIENT TEMPERATURES FOR 50-70 MINUTES

Parameter	Ambient temperature (℃)					Significance
	20	35	40	45	50	
RT (C)	36.8± 0.10^{a}	38.5± 0.13^{b}	39.4+ 0.20^{bc}	41.3+ 0.31^{d}	34.2± 0.10^{e}	$p < 0.01$
PG (mg/dl)	94.3±15.04^{a}	97.1± 7.45^{a}	79.4± 6.02^{ab}	58.9± 9.83^{b}	15.1± 5.88^{c}	$p < 0.01$
GOT (unit/ml)	156.9±19.97^{a}	117.3±18.82^{ab}	113.7±19.81^{ab}	64.1± 5.32^{b}	228.7±23.84^{c}	$p < 0.01$
GPT (unit/ml)	50.3± 5.42^{a}	4.8+ 3.39^{ab}	45.3± 2.74^{ab}	37.3± 2.55^{b}	53.3± 3.91^{a}	n.s
PCV (%)	42.2± 0.66^{a}	42.9± 1.10^{a}	44.2± 0.86^{ab}	46.9± 1.30^{bc}	47.7+ 1.37^{c}	$p < 0.01$
PL (%)	0	1.14	3.46	7.61	9.52	

RT, rectal temperature; PG, plasma glucose; GOT, glutamic oxaloacetic transaminases; GPT, glutamic pyruvic transaminase; PCV, packed cell volume; PL, plasma loss.

Mean superscript with different letter(s) differ significantly ($p < 0.05$-0.01).

Each experimental group contain 5 rats.

At 50℃, rats developed critical and lethal hyperthermia within 30-50 minutes, at which blood sampling was done.

Plasma loss percentage was calculated from the PCV change.

Rectal temperature (RT)

Results indicate that RT of rats was increased ($p < 0.01$) when air temperature was raised from 20℃ to 35℃. Further increase ($p < 0.01$) was obtained with increasing ambient temperature. Previously, it has been shown that the environmental air temperature of 28℃ (Herrington, 1940) and 33℃ (Bhatia et al., 1969) is the neutral temperature for rats.

In the present study, at 50℃ air temperature all rats reached the lethal body temperature (43.2℃) and died within 40-50 minutes. Frankel (1958) found at 50℃ ambient temperature that tolerance time for male rats was 44 minutes with 44.8℃ final rectal temperature. This death by heat shock is probably due to body dehydration and failure in the transport of heat, causing a high core temperature and irreversible circulatory failure (Kolk, 1969).

In other investigations, critical hyperthermia in rats was developed within 70-90 minutes at 45℃ and 70% humidity (Khalil and Kotby, 1982), within 2 hours at 45℃ and 22% humidity (Harrison, 1976), and within 5-6 hours at 40℃ (Hainsworth et al., 1968). Clearly, the time during which the animals developed critical hyperthermia is dependent, among other factors, upon the intensity of heat applied and the associated relative humidity.

Plasma glucose level (PG)

Normal level of PG (94.3 mg/dl) was unchanged at ambient temperature of 20 and 35℃, while it decreased at 40℃, reaching a level of 15.1 mg/dl at 50℃. This hypoglycemic effect of heat exposure was significant ($p < 0.01$) at 45 and 50℃. In agreement were reports of Kanter (1957 and 1959) in dogs, Harrison (1976) and Khalil and Kotby (1982) in rats. Khalil (1981) showed that blood glucose level of male mature rats began to decrease at 40 minutes of 45℃ exposure, while Megel et al. (1962) reported no change in blood glucose of male rats at 46℃ for 20 minutes. Results from this study and those from other investigations indicate that blood glucose level is greatly dependent upon heat intensity as well as heat duration.

A decreased PG level in hyperthermic rats is probably attributed to the increasing glucose utili-

zation by body tissues for meeting the increased metabolic requirements for thermoregulatory reaction against heat stress. Hyperthermia in rats was reported to be associated with an accelerated glycogenolysis (Pal and Sadhu, 1973; Harrison, 1976; Khalil and Kotby, 1982), an elevated blood lactate level (Harrison, 1976), an accelerated heart rate (Frankel, 1958), and an increased oxygen consumption by cerebral cortex (Carlsson et al., 1976).

Transaminases (GOT and GPT) levels

Normal level of GOT at 20℃ (156.9) units/ml) tended to decrease at 35 and 40℃, reaching the lowest level (64.1 units/ml) at 45℃ ambient temperature GOT level in blood exhibited a reversed trend since it increased sharply over the normal control value (228.7 units/ml). Plasma level of GPT showed a similar trend; it decreased at the temperature of 35 up to 45℃ but returned to the normal value (53.3 units/ml) at 50℃. Statistical analysis revealed that differences between groups were significant ($p < 0.01$) for GOT and insignificant for GPT plasma levels.

Elevation of GOT in blood is due to the escape of the enzyme from distributed hepatic parenchymal cell with necrosis or altered membrane permeability (Cornelius, 1974). The significant increase in GOT at lethal ambient temperature (50℃) probably indicates a disturbance in the transamination reactions in hepatic cells, with hepatic and myocardial damage (Wroblewski, 1959). Present results indicated that GOT plasma level is influenced more by thermal stress than GPT. However, hyperthermia in dogs was associated with an increase in the serum GOT and GPT levels. (Reed et al., 1964; Spurr, 1972).

In rats, exposure at 46℃ air temperature but for short duration (20 minutes) did not alter the serum level of GOT (Megel et al., 1962). Pal and Sadhu (1973) heave reported no significant change in liver tissue transaminases in rats exposed at 41℃ for 2 hours. These authors concluded that gluconeogenesis contributed little or nothing in the heat production under acute thermal stress.

Hematocrit value

Present study showed that hematocrit value increased gradually ($p < 0.01$) by the heat exposure from 42.2% in the control group (20℃) to 47.7% in the 50℃ group.

Heat intensity and duration of heat exposure seem to be equally important in heat stress research. These results agree with those reported on rats (Frankel, 1959; Hainsworth et al., 1968; Khalil and Kotby, 1982) and dogs (Kanter, 1957 and 1959).

At 45 and 50℃ ambient temperature, plasma volume was reduced by 7.61% and 9.52%, respectively as calculated from the PCV change. Khalil and Kotby (1982) have determined this percentage in adult rats exposed at 45℃ for 70-90 minutes. Plasma loss at high ambient temperature without available drinking water is due to an excessive water evaporation for body cooling purposes. This occurs in rats by active salivation (Hainsworth et al., 1968) and water cutaneous diffusion of insensible perspiration (Folk, 1969), resulting in hemoconcentration and body dehydration. When severe body dehydration occurs, animal's thermoregulatory mechanism is impaired becuase of insufficiency of tissue fluids to permit beat loss by evaporation. Adolph (1947), Brouha (1960) and Schalm (1967) reported that dehydration and hemoconcentration would lead to an added load on the cardiovascular system, resulting in uncontrolled hyperthermia and death.

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