Hormonal Profiles of African Pygmy Goats Exposed to Heat Environment

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Abstract Hormonal profiles were measured on 5 non-lactating African Pygmy goats exposed to thermoneutral and 3 levels of high temperature. Animals were progressively exposed to each environmental temperature (18°C for 5 days, 30°C for 3 days, 34°C for 9 days and 37°C for 4 days). Plasma prolactin concentration increased to a maximal during 37°C exposure, and epinephrine, norepinephrine and cortisol concentrations during 34°C exposure. Plasma epinephrine, norepinephrine and cortisol then tended to decline toward thermoneutral levels, which may be an expression of a state of acclimation. In this stage of acclimation phase, plasma prolactin remained elevated and T3 remained decreased.


Key words: heat stress, African Pigmy goat, hormonal profiles

The African Pygmy goat has evolved in relatively harsh environments of various tropical, humid and desert climates. It is known to have a high resistance to many diseases and is adaptable to a wide range of nutritional environments. Of special interest in this investigation is the determination of neuroendocrine and physiological mechanisms of adaptability of the African Pygmy goats to heat environment. BELL et al.3) conducted initial investigations on metabolic, evaporative, thermal balance mechanisms of the Pygmy goat in response to high temperatures based on the similar experimental design to that of this study. In that study, the increased rectal temperatures, respiration rates and water intake, as well as the decrease in feed intake were observed at the high temperature treatments. However, body weights did not decrease as expected. Therefore, it should be noted that urine production did not keep pace with water consumption, and it is possible that retained water accounts for part of the higher than expected body weights. As described above, the African Pygmy goats is interest to physiologists due to its adaptability to hot-humid and hot-dry conditions. Little data concerning the physiology of Pygmy goats as it relates to the environment, however, is available.

The objective of this investigation was to establish the hormonal profiles of non-lactating Pygmy goat during exposure to moderate and extreme high temperatures.

Materials and Methods

Experimental procedure:

Five non-lactating African Pygmy goats of a line originating in Cameroon, Africa were obtained from Carol Foster of Peculiar,
Missouri, USA. Ages were between 1.5 to 2.5 years of age and were non-lactating at the time of study. All animals were exposed to a series of environmental temperatures, ranging from thermoneutral (18°C) to extreme high temperature (37°C) in the Animal Climatology Laboratory at the University of Missouri. The treatments consisted of 3-9 days of exposure at constant temperatures 18°C (50% rh), 30°C (30% rh), 34°C (30% rh) and 37°C (30% rh), with three days rest intervals at thermoneutral for all conditions except 37°C. The animals were given feed and water ad libitum. Rectal temperatures and respiration rates were recorded daily. Two 10 ml blood samples were taken with heparinized vacutainer tubes from the jugular vein on each daily sampling day. The whole blood was centrifuged at 1,500 × g for 15 min. A portion of the plasma was stored and frozen at −20°C for growth hormone, prolactin, cortisol and T₃ analyses; the remainder of the plasma was deproteinized for catecholamine analyses and kept frozen at −70°C.

Analysis:
Norepinephrine and epinephrine in plasma were analyzed by High Performance Liquid Chromatography/Electrochemical Detection (HPLC/EC). Two ml of plasma was deproteinized with 0.1 N perchloric acid. The extract was adsorbed on alumina with microfilter tubes (Bioanalytical Systems Inc. Indiana). The filtrate was injected into the HPLC/EC system, to obtain quantifiable recording of the catecholamines. External standards were used to calculate the unknown concentrations based on the peak height.

Prolactin and growth hormone were analyzed by double antibody radio-immunoassay described by Prapa et al.13. Ovine prolactin and ovine growth hormone were used as standards. The recoveries of pools spiked with prolactin at 20, 40, 80, 160 and 200 ng were 97.0, 97.1, 96.4, 99.4 and 97.3%, respectively. The inter and intra assay variations were 6.1 and 4.6%, respectively. The recoveries of pools spiked with growth hormone at 5, 10, 20 and 30 ng were 98.0, 99.0, 97.8 and 97.9%, respectively. The inter and intra assay variations were 3.9% and 3.4%, respectively.

Plasma T₃ and cortisol were analyzed using solid phase radio-immunoassay kits produced by Diagnostic Products, Los Angeles, CA. The standard curve ranged from 0-100 ng/ml for plasma cortisol. Samples ranging from 50 to 200 µl were linear to the standard curve. Sample volumes of 50 µl were used for analysis of plasma cortisol. The recoveries spiked with pools at 10, 20 and 50 ng were 99.4, 96.8 and 97.0%, respectively. The inter and intra assay variations were 5.5 and 6.5%, respectively. The standard curve for plasma T₃ ranged 0-6.0 ng/ml. The pools were tested for linearity from 50 to 250 µl. The sample size for analysis was 100 µl. The spiked recoveries at 1.0, 2.0 and 6.0 ng were 99.1, 96.4 and 97.3%, respectively, and inter and intra assay variations were 3.6 and 5.9%, respectively.

The differences between the treatments were analyzed by analysis of variance for split-plot in time with repeated measurements (ANOVA) using a least significant difference (LSD) procedure of Statistical Analysis System (SAS).

Results
Fig. 1 presents the data on rectal temperature, respiration rate and water intake as indicators of the ability of the goats to maintain thermal balance. The time trends may be observed for the rectal temperature, respiration rate as well as water intake under each temperature condition. The rectal temperatures and respiration rates increased at each high temperature. Similar responses were observed for water intake. These data, especially rectal temperatures, indicate the degree of thermal stress or thermal imbalance, which persisted from 30°C to 37°C exposure.

Fig. 2 illustrates the effects of temperature on plasma prolactin and cortisol. Prolactin
increased at 30°C, 34°C and 37°C. As observed earlier, water intake and rectal temperature also declined after 4 days at 34°C, reflecting the influence of stress reduction on prolactin. However, the prolactin remained elevated during the entire heat acclimation period and paralleled the deep body temperature response with the exception of the rectal temperature overshoot upon first exposure to 34°C. Further increase in the prolactin was found with a concomitant increase in the rectal temperature during exposure to 37°C. The cortisol showed an increasing tendency with some fluctuations after first exposure to 34°C but after 4–5 days the values tended to decline, and the declining tendency continued during following exposure to 37°C.

Maximal values of norepinephrine and epinephrine were observed on day 2 of exposure to 34°C and returned to near thermoneutral levels by day 3 of exposure to 34°C for norepinephrine and day 6 for epinephrine. Even the continued exposure to more severe heat stress (37°C) did not re-stimulate the secretion of norepinephrine and epinephrine. This decline occurred even though the body temperature remained elevated and higher at 37°C (Fig. 3).

Plasma growth hormone responded to high

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**Fig. 1.** Daily mean values of rectal temperature, respiration rates and water intake during thermoneutral (18°C) and 30°C, 34°C, 37°C environmental high temperatures. Each value represents the mean ± standard error.

**Fig. 2.** Daily mean values of plasma prolactin and cortisol during thermoneutral (18°C) and 30°C, 34°C, 37°C environmental high temperatures. Each value represents the mean ± standard error.
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Fig. 3. Daily mean values of plasma norepinephrine and epinephrine during thermoneutral (18°C) and 30°C, 34°C, 37°C environmental high temperatures. Each value represents the mean ± standard error.

Fig. 4. Daily mean values of plasma growth hormone and triiodothyronine (T3) during thermoneutral (18°C) and 30°C, 34°C, 37°C environmental high temperatures. Each value represents the mean ± standard error.

temperature (34°C) with an increase upon initial exposure followed by gradual declines to thermoneutral levels or lower (Fig. 4).

The progressively declined plasma T3 during exposure to high temperatures was observed (Fig. 4), and this could be a reflection of the adjustments in endogenous heat production and a declining calorigenic intake as observed in an earlier study by Bell et al.3).

Table 1 summarizes the means, standard error, and significance for rectal temperature, respiration rate, water intake and plasma hormones at thermoneutral and each high temperature.

Discussion

In the previous studies on cattle exposed to high temperatures (28°C-32°C for 2 weeks), initial exposure to heat resulted in stimulation of chemical thermogenesis by increased levels of many hormones such as growth hormone, cortisol, prolactin, epinephrine and norepinephrine, and only epinephrine and norepinephrine remained elevated with continued heat exposure1,7,8,9). Those hormonal profiles found in cattle showed similar tendencies to those observed in this study.

These hormonal and calorigenic changes conform to the alarm and resistance phases of general adaptation syndrome described many years ago12), and the progressive decline during prolonged exposure to heat at 34°C and 37°C may serve to avoid suppression of immune processes10). However, the only hormone meas-
Table 1. Means, standard error and significance of temperature effects on rectal temperature, respiration rate, water intake and plasma hormones in African Pygmy goats

<table>
<thead>
<tr>
<th></th>
<th>18°C</th>
<th>30°C</th>
<th>34°C</th>
<th>37°C</th>
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</thead>
<tbody>
<tr>
<td>Rectal temp.</td>
<td>38.9±0.06</td>
<td>39.2±0.05</td>
<td>39.4±0.07</td>
<td>39.5±0.06</td>
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<td>(°C)</td>
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<tr>
<td>Respiration</td>
<td>39.4±1.9</td>
<td>91.5±8.1</td>
<td>145.5±6.1</td>
<td>187.8±2.8</td>
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<td>rate (per</td>
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<td>minute)</td>
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<tr>
<td>Water intake</td>
<td>0.91±0.08</td>
<td>1.5±0.11</td>
<td>1.6±0.14</td>
<td>1.2±0.20</td>
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<td>(/day)</td>
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<tr>
<td>Prolactin</td>
<td>10.4±1.4</td>
<td>31.7±3.6</td>
<td>36.0±3.0</td>
<td>92.3±7.2</td>
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<tr>
<td>(ng/ml)</td>
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<tr>
<td>Cortisol</td>
<td>16.3±0.3</td>
<td>15.7±1.3</td>
<td>26.3±1.5</td>
<td>19.8±1.3</td>
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<tr>
<td>(ng/ml)</td>
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<tr>
<td>Norepinephrine</td>
<td>92.8±5.5</td>
<td>144.8±22.8</td>
<td>165.9±33.9</td>
<td>55.0±1.3</td>
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<td>(pg/ml)</td>
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<tr>
<td>Epinephrine</td>
<td>73.9±8.4</td>
<td>65.6±22.8</td>
<td>213.5±39.1</td>
<td>69.8±1.4</td>
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<tr>
<td>(pg/ml)</td>
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<tr>
<td>Growth hormone</td>
<td>1.8±0.2</td>
<td>1.7±0.3</td>
<td>2.7±0.4</td>
<td>2.6±0.3</td>
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<tr>
<td>(ng/ml)</td>
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<tr>
<td>Triiodothyronine</td>
<td>1.7±0.07</td>
<td>1.6±0.11</td>
<td>1.4±0.04</td>
<td>1.0±0.03</td>
</tr>
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* a, b, c, d Means in the same row without a common superscript differ (P<0.05).

ured that does not decline is prolactin and reflects not only an initial exposure to a heat stressor and possibly an "alarm" action, but under thermal stress remains elevated throughout the resistance phase as long as the thermal stress persists. The intensity of the thermal stress is reflected closely by the levels of plasma prolactin.

The elevated plasma prolactin has been reported to stimulate the release of 5-hydroxytryptamine (5-HT) which may be an advantage to heat balance of the goat. 5-HT is known to be beneficial in heat loss during heat exposure and also believed to play a role in fluid balance.

Elevated plasma cortisol began to decline during the latter phase of the 34°C exposure and the following 37°C exposure. The decline may have been partially due to decline in adrenocorticotropic stimulation of adrenal cortisol secretion and the epinephrine stimulated release of cortisol. Indeed, sharp increase in the plasma epinephrine and norepinephrine were found on day 2 of 34°C exposure, and they rapidly declined as did the cortisol with time lag.

These adjustments may have other significant effects on the animal's ability to tolerate longer period of heat stress. The decline in cortisol can possibly be due to the decline in the heat induced emotional reaction upon initial heat exposure or merely passage of the animal system into the resistance phase of the stress syndrome. Plasma T₃, as did feed intake and energy metabolism, declined throughout the first few days or the early alarm stage and especially the latter exposure or resistance phase of the stress syndrome. All of these factors significantly reduced the endogenous heat production and thus could alleviate some of the heat imbalance problems.

References
3) Bell, B.A., W. Hainen and H.D. Johnson,


12) Selye, H., The stress syndrome was subsequently called the general adaptation syndrome by Selye. This syndrome proceeds in three stages: (i) the alarm reaction, (ii) the stage of resistance, and (iii) the stage of exhaustion. Nature 138: 32. 1936.