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EFFECTS OF TRANSPORT AND RACING ON IONIC CHANGES IN THOROUGHBRED RACE HORSES

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Abstract—1. Packed cell volume (PCV), blood glucose, total plasma proteins (TPP) and plasma electrolytes, osmolality, cortisol and aldosterone alterations produced by transport and racing, were investigated in race horses.

2. Plasma cortisol, sodium and blood glucose, found after transport, were higher, while aldosterone was lower than control levels.

3. After racing, PCV, blood glucose, TPP and plasma cortisol, sodium and osmolality were higher than control, while chloride diminished and aldosterone returned to control values.

4. These results demonstrate that transport and racing are different kinds of stressors, suggesting that the sympathetic system and hypophysis–suprarenal cortex axis have a dissimilar contribution to the physiological response.

INTRODUCTION

The performance of thoroughbred race horses participating in short races (between 1 and 2 min), with supramaximal exercise, is dependent on energetic metabolic supplies, and neuromuscular and psychological conditions (Physick–Sheard, 1982; McMiken, 1983; Revington, 1983). These conditions could be modified by stress situations occurring before a race (changes of horseshoes, transport in trucks, clinical examination, public exposition, starting location), and by psychological as well as the physics and traumatic consequences of racing (Carlson *et al.*, 1975; Dybdal *et al.*, 1980; Revington, 1983; Bayly *et al.*, 1986). These stressors could produce neuroendocrine and ionic alterations decreasing the locomotive capacity of the animal (Knochel, 1982; Martínez *et al.*, 1988; White *et al.*, in press).

Physical and psychological stress produce hypophysis–adrenal axis activity determining plasma levels of cortisol (Snow and Mackenzie, 1977) and sympathetic-adrenal axis activation with catecholamines release (Dybdal *et al.*, 1980; Dimsdale and Moss, 1980; Revington, 1983; Freg *et al.*, 1985; Martínez *et al.*, 1988). The release of catecholamines produce several physiological events in response to this stress, for instance: glycogenolysis stimulation (McMiken, 1983), hyperglycemic effect by alpha adrenergic blockade of insulin release in pancreatic beta cells (Vargas *et al.*, 1974; Dybdal *et al.*, 1980), plasma potassium regulation (Wolfe *et al.*, 1977; Sterns *et al.*, 1981; De Fronzo *et al.*, 1981; Thornton, 1985; Williams *et al.*, 1985), enkephaline and endorphine release (Snyder, 1984; Pasternak, 1988), ionic channel regulation (Tsien, 1987) and a greater release of acetylcholine in the neuromuscular junctions (Bowman and Nott, 1969).

The neuromuscular excitability and muscular blood perfusion capacity are strongly dependent of ionic equilibrium through cell membranes (Tsien, 1987; Hoffmann and Simonsen, 1989). In order to characterize the effect of neuroendocrine changes produced by transport and racing on ionic metabolism, PVC, blood glucose, TPP and plasma osmolality, sodium, potassium, chloride, cortisol and aldosterone were measured under control conditions, just after transport and immediatly after the race, in blood samples of thoroughbred race horses. These parameters are related to the animals athletic performance (Persson, 1967; Soliman and Nadim, 1967; Williamson, 1974; Physick-Sheard, 1982; Snyder, 1984; Muylle *et al.*, 1984; Thornton, 1985).

MATERIALS AND METHODS

Experiments were performed on 18 male and female thoroughbred race horses, between 3 and 5 years old, trained on Chilean tracks and transported to other tracks located 10 to 120 km from the training place. The time of transport varied between 1–4 hr. The animals were maintained on a diet of standard mixed grain and hay, with water *ad lib.*

Three blood samples were collected from each animal, in less than 25 sec, by jugular venepuncture, while the horses were restrained by their usual attendants. The blood samples were obtained after descending from the truck, 5 min after racing and in basal conditions. False transport blood samples were taken simultaneously with the experimental group, from animals not stressed by transport. Basal samples were obtained in the stall, exactly 24 hr before transport.

Glucose and PCV were immediately assayed in 1 ml of blood sample. Glucose was determined by glucoxidase Dextrostix Ames strips in a Glucometer® and PCV by microcentrifugation in a portable Compur M 1100. Furthermore, 20 ml of the same sample, obtained with heparin as

Table 1. Packed cell volume and total plasma protein concentration in race horses in basal condition, after transport and racing

	Basal	After transport	After race
PCV (%)	43.3 ± 1.0 (16)	44.4 ± 1.0 (16)	61.6 ± 0.9 (15)*
TPP (g/dl)	6.1 ± 0.1 (18)	6.2 ± 0.1 (18)	8.0 ± 0.2 (17)*

Values are expressed as mean ± SE of mean.
*$P < 0.001$ in relation with basal values.
Number of animals studied is given in parentheses.

anticoagulant, were maintained on ice and the plasma was quickly separated by centrifugation at 800 *g* for 15 min. The plasma samples were harvested and frozen at −20°C for subsequent laboratory analysis.

Plasma parameters determined were: TPP in a Goldberg refractometer, sodium and potassium in a IL 343 flame-photometer, chloride in a Büchler chloridometer, cortisol and aldosterone by radioimmunoassay, using Coat-a-count kits and osmolality in a Advanced Instrument 3W2 osmometer. In cortisol analysis was previously denatured plasmatic transcortin by dilution and heating at 60°C for 30 min.

The results were statistically analysed by the Student's *t*-test.

RESULTS

After transport PCV and TPP are not statistically different from control basal values. Nevertheless, a significant increment in both was observed in after race samples (Table 1).

When PCV and TPP are expressed in percentage with respect to the basal values, the significant increment observed in PCV, in after race samples, is higher (41.9%) than in TPP values (31.1%) (Fig. 1).

Post-transport and after race blood glucose values are significantly different, both of them are significantly higher than basal values. In animals not stressed by transport the observed value was not significantly different from basal (Fig. 2a).

Plasma cortisol concentration values found in post-transport condition and in samples taken after racing are significantly different. Both are significantly higher than basal value. The plasma cortisol level found in animals not stressed by transport was not significantly different from basal (Fig. 2b).

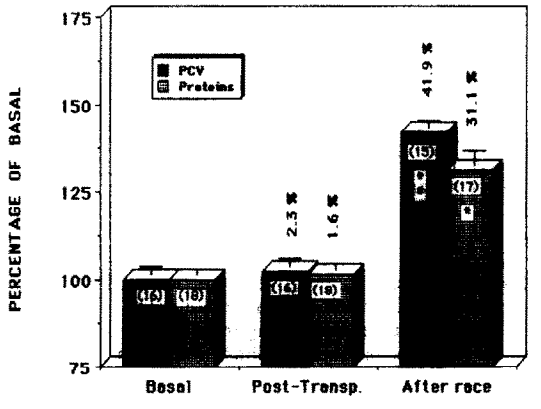

Fig. 1. Comparison of the variation in PCV and TPP experimented in response to stress conditions. Values are expressed as percentage of basal values (mean ± SE of the mean). After race values were significantly higher than basal levels (*$P < 0.001$). The percentage of after race PCV increment is significantly different from TPP (# $P < 0.01$).
Number of animals studied is given in parentheses.

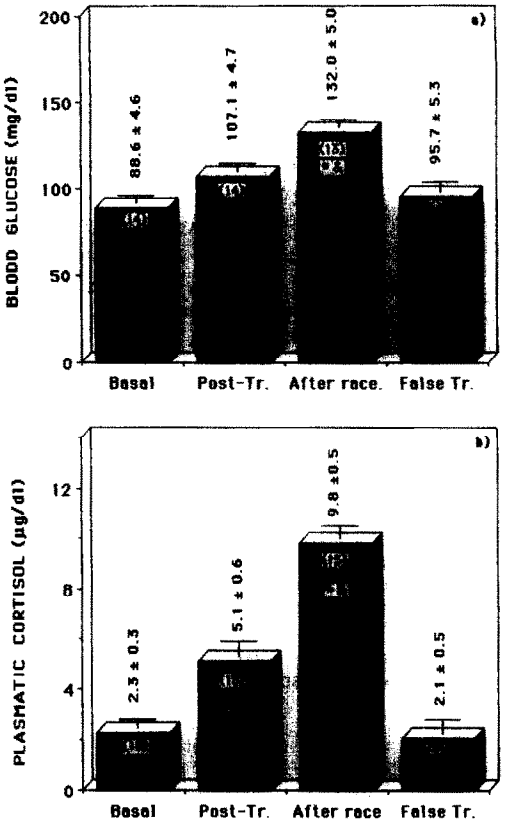

Fig. 2. Variation in blood glucose and plasmatic cortisol levels in response to stress conditions. Values are expressed as the mean ± SE of the mean. After transport and after race values were significantly different (&$P < 0.001$) and both were significantly higher than basal and false transport levels (*$P < 0.001$). Number of animals studied is given in parentheses.

Concerning ion metabolism, plasma chloride concentration is slightly but significantly lower than basal values only in samples taken after the race. The level found in post-transport samples is not significantly different from other experimental groups (Table 2).

Plasma potassium concentration of the basal group is slightly higher than post-transport and after race samples. Nevertheless, obtained values are not significantly different in all the experimental groups studied (Table 2).

Plasma sodium concentration of post-transport and after race samples are significantly different. Both are significantly higher than basal values (Table 2).

In addition, plasma osmolality of basal and post-transport samples are not statistically different. Both are significantly lower than after race values (Table 2).

Finally, the concentration of aldosterone found in post-transport plasma samples is significantly lower than basal and after race levels. No difference is observed between basal and after race values (Fig. 3).

Table 2. Plasma electrolytes and osmolality of race horses in basal condition, after transport and racing

	Basal	After transport	After race
Chloride (mEq/l)	97.9 ± 0.7 (18)	96.4 ± 0.7 (18)	95.4 ± 0.9 (17)*
Potassium (mEq/l)	4.0 ± 0.1 (18)	3.8 ± 0.1 (18)	3.8 ± 0.1 (14)
Sodium (mEq/l)	131.5 ± 0.8 (17)	134.9 ± 1.2 (18)*	143.6 ± 1.8 (17)‡
Osmolality (mOsm/KgH20)	274.5 ± 1.5 (18)	274.6 ± 1.7 (18)	312.1 ± 3.4 (17)†

Values are expressed as mean ± SE of mean.
* and †$P < 0.05$ and $P < 0.001$ respectively in relation with basal values.
‡$P < 0.001$ in relation with after transport and basal values.
Number of animals studied is given in parentheses.

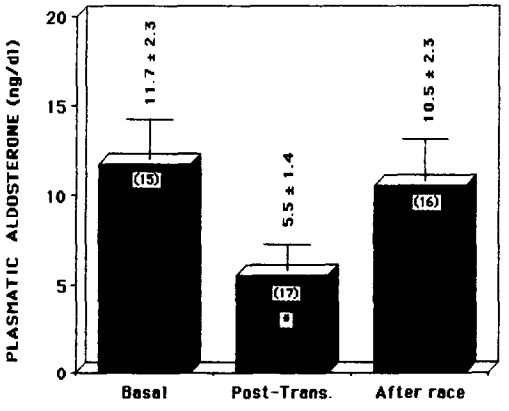

Fig. 3. Post-transport aldosterone concentration in comparison with basal and after race condition. Values are expressed as the mean ± SE of the mean. Basal and after race values were similar and both were significantly higher than post-transport level (*$P < 0.001$). Number of animals studied is given in parentheses.

DISCUSSION

This work demonstrated that thoroughbred race horses transported in trucks before racing, arrive stressed at the race tract, as is suggested by the significant increment found in blood glucose and plasmatic cortisol values (Chin and Evonuk, 1971; Vargas *et al.*, 1974; Snow and Mackenzie, 1977; Martínez *et al.*, 1988).

During transport, animals are 1–4 hr inside the trucks subjected principally to psychological stress. On the other hand, since the race lasted 1 or 2 min at a maximal physical activity, an important adrenergic and hypophysis–suprarenal cortex axis response is developed.

This study was performed on blood samples taken in less than 25 sec to avoid hematological changes produced when jugular venepuncture is more prolonged (Persson, 1967; Revington, 1983).

The small, but significant, increment found in plasma sodium after transport could be related to metabolic alterations, with water redistribution. This result agrees with the conditions observed in the horses just after transport: sweating, excited and thirsty (Kerr and Snow, 1983; Köhler, 1987). Other parameters as PCV, TPP and plasma osmolality do not change under this stress condition.

The lower values observed for plasma aldosterone concentration in post-transport samples may be in response to the increased plasma sodium concentration. Other possibilities are the adrenal exhaustion of "zona glomerulosa" or low levels of plasma potassium concentration. The increase in plasma aldosterone levels observed in samples taken after racing returning to around basal values, could be due to simultaneous and antagonistic response to the inhibitory effect of high levels of plasma sodium concentration produced by water redistribution, with water loss by breathing and perspiration, and stimulatory effect of ACTH and catecholamines in response to psychological and physical stress (Guthrie *et al.*, 1980, 1982; Missale *et al.*, 1986). In our experiments aldosterone levels should not be modified by potassium levels, because after racing plasma potassium concentration does not change with respect to basal values (Rose and Allen, 1985; Williams *et al.*, 1985).

The unchanged plasma potassium concentration after transport and racing, could be explained by the equilibrium between alpha and beta-adrenergic effect of the catecholamines released. Alpha-adrenergic potassium releasing action should occur first and mainly on liver, muscle and erythrocytes. While muscle and liver potassium uptake should be mediated later by beta-adrenergic action (Bowman and Nott, 1969; Mukherjee and Lefkowithz, 1976; Wolfe *et al.*, 1977; Silva and Spoker, 1981; Sterns *et al.*, 1981; Martínez *et al.*, 1988, 1990). The stability of plasma potassium is important for normal membrane excitability (Wang and Clausen, 1976; Clausen and Flatman, 1977; Sterns *et al.*, 1981; Duane, 1986) and regulate the red cell volume, allowing membrane deformability when blood perfuses the muscular microcirculation (Kjellmer, 1965; Durán, 1977; Hoffmann and Simonsen, 1989; Chasis and Schrier, 1989). These results agree with the decrease of red cell potassium concentration in after race condition, without hyperkalemia, previously described by these authors (White *et al.*, in press).

The increment observed in after race PCV and PPT blood samples, as well as plasma sodium concentration and osmolality, could be due to water loss by breathing and perspiration, and water redistribution. The percentage of increment found in PCV (41.9%) and PPT (31.1%) are higher than the rise of plasma sodium (9.2%) and osmolality (13.7%) probably because red cells and proteins stay in the intravascular, while sodium and electrolytes are transported to the extravascular compartment (Kerr and Snow, 1983; Köhler, 1987). The difference observed between the increments found in PCV and PPT is probably due to the important amount of red cells produced by splenic contraction (Persson, 1967; Martinez *et al.*, 1988).

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