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Training versus Overtraining: Evaluation of Two Protocols

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ABSTRACT

Forty Standardbred trotters were divided into two groups, A and B, and subjected to two training methods with a much more intense protocol in the second group. Compared with group A, group B showed an increase in red blood cells, leukocytes, and red cell distribution width (RDW); a decrease in mean corpuscular volume (MCV) and mean corpuscular hemoglobin (MCH); and elevations of aspartate amino transferase (AST), creatine kinase (CK), lactate dehydrogenase (LDH), alkaline phosphatase (AP), total bilirubin (BUN), and α_1- and α_2-globulins. The authors believe that the concomitance of such findings may suggest a condition of overtraining.

Keywords: Training; Standardbred trotters; Overtraining; Stress; Disease

INTRODUCTION

Genetic selection by humans of trotter horses has led to the development of a breed that is a blend of harmony, speed, and potency. Trotters have a docile nature and begin training at 18 months. Their competitive life ends at 10 years for males and 7 years for females.[1] The breaking and training stages are key to optimal organ development, especially of the skeleton and muscles. They are conducted so as to prevent trauma induced by overfatigue and to ensure good future potential. During taming and training, horses perform aerobic work and undergo "basic training" consisting of approximately 30 minutes of trotting at 4 to 5 m/s.[2] After 4 months, the most precocious colts are tested on speed to assess the type of gait and hind limb forward stride. At the beginning of June, the best performers may start qualifying for official races and, if they can cover a 1-mile distance in a maximum of 2 minutes 13 seconds, they may begin their competitive careers.[3] Once the horses destined for racing careers have been identified, they undergo a very specific and intense training program, mainly consisting of 30 minutes of trotting at 4 to 5 m/s, alternating with 1,600-m or 2,000-m speed tests which simulate racing.[4] Interval training is done on racecourses with a "straight track," where the horses run for spurts of increasing speed, spaced out by 5 to 10 minutes of slow trotting to recover. This kind of training also may be used to test the extent of athletic fitness of an individual.[5]

Intense, ongoing training executed close to an individual's limit and the racing effort may trigger several pathologic conditions.[6] Statistically, lameness is certainly the most frequent condition but it is not necessarily the absolute cause of poor performance because limping horses—if warmed up properly—may cross the finish line with fairly good results.[7] This is not the case in the overtraining syndrome. Overtraining usually is associated with either a decrease in performance or consistent under-performance. Although high-intensity work with little rest between bouts is most frequently associated with overtraining, the specific causes of overtraining are not understood. True overtraining is probably uncommon in Thoroughbreds; however, it may be a problem in some Standardbreds in which training and racing tend to be much more frequent, and hard, long interval-type work loads are more likely to be used.[8] Overtrained horses lose weight without changing their food intake, produce a higher amount of blood lactate at the end of the effort, reduce their V_{200}, and constantly tend to underperform during races.[9] The overtraining syndrome is a well-known condition that causes major economic losses for the owners of racing horses. The performance of overtrained horses is inversely proportional to the intensity of exercise and frequency of racing.[10]

No agreement has been reached on how to diagnose this syndrome induced by quantitatively and qualitatively inappropriate training. Some authors suggest that increased resting hematocrit values and their decline under stress are the key discriminating factors,[11] whereas others assert that blood chemistry or biochemical parameters are not instrumental.[12] What is certain is that overtrained horses perform poorly during races, especially in terms of speed. As clearly shown by treadmill tests, under intense exercise, muscles increase their aerobic capacity but reduce their speed of contraction.[13]

In Apulia, Italy, the owners of trotters do not generally spare their horses. They tend to engage them in an excessive number of races (see ANAGT),[3] thus subjecting them to a training and racing schedule that, on average, is very intense. Given the importance of trotters in Apulian horse-breeding and the high frequency of this type of training

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management, we decided to investigate (1) whether one or more factors of the training protocols may be related to overtraining syndrome and, (2) whether changes in blood chemistry parameters were present that could reasonably lead us to confirm the physical and behavioral diagnosis of this condition.

MATERIALS AND METHODS

From June to July 2004, 40 trotters trained in Apulia were examined. The subjects were all qualified racing horses for medium-high level competitions. Information regarding the training pattern was gathered for each subject whose body condition score (BCS) was available. Based on BCS and racing frequency, two groups of 20 horses each (A and B) were identified with major differences in terms of likely onset of the overtraining syndrome. Group A comprised horses undergoing one speed training session and one 5,000-m trotting session per week and that were habituated to running once every 10 to 15 days and resting the following day. Group B consisted of horses having two speed training sessions per week and that were habituated to running once every 7 to 10 days and virtually never resting. In both groups, horses engaged in races would miss one speed training. The feed type and quantity of oats, commercial horse cubes, and hay, were the same. The energy and protein content of the diet were those recommended by the Institut National de la Recherche Agronomique (INRA),[14] equal to 6.8 UFC, calculated according to body weight and type of exercise. The daily ration was divided into three feeds, the first in the morning before exercise at 6:00 AM, the second at 12:00 noon, and the third at 7:00 PM.

One week after their last race, early in the morning while resting before their daily training session, ultrasonographic evaluations of the horses' BCS were performed, and their blood was sampled with the Vacutainer® system (Becton Dickinson, Franklin Lakes, NJ). Each blood sample was split into two test tubes: one with a serum gel separator, also used for serum protein electrophoresis and biochemistry tests, and the other with the K3 EDTA anticoagulant for a full blood count. Blood tests were performed using the Abbott cell counter analyser CELL DYN 3700® (Abbott, Chicago, IL). Hematocrit was determined by centrifugation and direct measurement. Serum protein electrophoresis was performed on agarose gel, according to the Melena BioSciences method. Serum biochemical parameters, as shown in Table 1, were assessed with ASSEL reagents and the SEAC photometer with interferential filters.

An ultrasound machine equipped with a 7.5-MHZ linear probe (ALOKA 500®; Aloka S.P.A., Italy) was used to measure fat thickness at the 14th intercostal space and tailhead to assess fattening. The BCS was computed by applying a regression equation to the ultrasound measurements.[15]

The results were examined by variance analysis, using the GLM procedure (SAS),[16] based on the following statistical model: $y_{ij} = \mu + ALL_i + \epsilon_{ij.}$ where y_{ij} = parameters under review, μ = general average, ALL_i = fixed effect of the i^{th} training technique (i: 1, 2), and ϵ_{ij} = standard error.

RESULTS

All horses had undergone training for at least 2 years (mean of age ± SD is 6.14 ± 2.19). During the study period (approximately 70 days), horses in group A had four starts, six speed training, and eight 5,000-m trotting sessions, whereas horses in group B had eight starts and 16 speed training sessions; in the last period of the study their records and performances decreased.

The results are shown in Table 1, where they are expressed as a mean of the horses present in the two groups, with the relevant standard errors.

Compared with group A, horses in group B had higher white ($P < .05$) and red blood cell counts ($P < .01$), and lower mean corpuscular volume (MCV) and mean corpuscular hemoglobin (MCH) values ($P < .05$). This is suggestive of anisocytosis with a significantly higher average red cell distribution weight (RDW) (23.66 ± 0.37 vs 21.66 ± 0.37, $P < .01$). The biochemistry tests showed significantly higher enzyme levels, such as creatine kinase (CK), aspartate amino transferase (AST) and lactate dehydrogenase (LDH) ($P < .01$), and alkaline phosphatase (AP) ($P < .05$) in group B than in group A. Markedly lower average triglyceride values ($P < .05$) were observed in group B, whereas cholesterol and glucose levels were similar in the two groups. The protein patterns only showed higher α_1 and α_2 fractions ($P < .05$) in group B than in group A, but no major difference in terms of total proteins, albumin, and other globulin fractions. Finally, the BSC of horses undergoing overtraining was markedly lower ($P < .01$).

DISCUSSION AND CONCLUSIONS

Horses in group B, which were too often engaged in racing (one start per week) and speed training (two times per week), likely were exposed to the overtraining syndrome. They presented an increased number of red blood cells with marked anisocytosis, and specifically with several small erythrocytes; as shown by the much lower values of MCV and MCH than in group A, likely due to an altered mechanism of hematopoiesis and hemocatharsis. Compared with data reported in the literature for healthy horses,[17,18] overtraining also leads to muscular fatigue with a clear elevation of muscle serum enzymes (CK, AST, LDH) and a mild increase in blood urea values, possibly attributable to an overload of amino acid metabolism in the liver and muscles.[18] The elevation of AST and CK is typical of muscle tenderness (tying up, myoglobinuria) and is a sign of muscular dysfunction, if not distress.[19] Also, the significant difference in the alkaline phosphatase values of group B horses compared with group A may be a sign of distress induced by a higher bone stress resulting from intense exercise. Alkaline phosphatase (AP) is an enzyme considered essential for mineralization, and AP serum concentrations can reflect osteoblast activity.[6] Leukocytosis with no major changes in the leukocyte formula may be ascribed to intense exercise and related to the catecholamine-induced

Table 1. Mean values (± ES) of blood parameters under review. Horses are split into two groups: training (A) and hypothesized overtraining (B). Means within rows with different superscript letters are different ($^{A,B}P < .01$ or $^{a,b}P < .05$)

Parameters under Review	Training (A)	Hypothesized Overtraining (B)
Age (yr)	6.45 ± 0.56	5.75 ± 0.56
BCS	3.2 ± 0.05^{A}	2.85 ± 0.05^{B}
WBC ($\times 10^3/\mu l$)	7.38 ± 0.31^{a}	8.34 ± 0.31^{b}
Neutrophils (%)	73.02 ± 2.23	61.77 ± 2.23
Lymphocytes (%)	30.38 ± 2.12	31.94 ± 2.12
Monocytes (%)	5.40 ± 0.39	4.86 ± 0.39
Eosinophils (%)	1.01 ± 0.2	1.32 ± 0.2
Basophils (%)	0.17 ± 0.02	0.12 ± 0.02
RBC ($\times 10^6/\mu l$)	8.53 ± 0.19^{A}	9.26 ± 0.19^{B}
HGB (g/dl)	13.61 ± 0.27	14.26 ± 0.27
HCT (automatic) (%)	37.34 ± 0.70	38.75 ± 0.70
MCV (fl)	43.74 ± 0.56^{a}	41.83 ± 0.56^{b}
MCH (pg)	16.07 ± 0.20^{a}	15.42 ± 0.20^{b}
MCHC (g/dl)	36.72 ± 0.15	36.83 ± 0.15
HCT (manual) (%)	37.85 ± 0.74	39.60 ± 0.74
PLT ($\times 10^3/\mu l$)	103.47 ± 7.87	114.30 ± 7.87
RDW	21.66 ± 0.37^{A}	23.34 ± 0.37^{B}
Albumin (%)	48.68 ± 1.36	47.01 ± 1.36
α_1 (%)	2.78 ± 0.46^{a}	4.11 ± 0.46^{b}
α_2 (%)	11.65 ± 0.49^{a}	13.06 ± 0.49^{b}
β_1 (%)	8.33 ± 0.40	7.99 ± 0.40
β_2 (%)	6.80 ± 0.51	5.99 ± 0.51
γ (%)	21.88 ± 1.01	21.79 ± 1.01
Total proteins (g/dl)	6.53 ± 0.11	6.59 ± 0.11
Albumin (g/dl)	3.66 ± 0.05	3.70 ± 0.05
Glucose (mg/dl)	82.82 ± 3.37	89.11 ± 3.28
Mg (mg/dl)	2.24 ± 0.06	2.22 ± 0.06
Cholesterol (mg/dl)	71.23 ± 3.20	72.26 ± 3.03
Triglycerides (mg/dl)	40.71 ± 4.00^{a}	31.91 ± 3.79^{b}
Calcium (mg/dl)	11.08 ± 0.29	10.59 ± 0.28
LDH (UI/L)	380.05 ± 27.54^{A}	531.20 ± 27.54^{B}
Phosphorus (mg/dl)	3.51 ± 0.16	3.42 ± 0.15
Creatinine (mg/dl)	1.35 ± 0.06	1.35 ± 0.06
BUN (mg/dl)	29.12 ± 1.7	31.98 ± 1.67
AST (UI/L)	325.50 ± 41.22^{A}	510.70 ± 41.22^{B}
ALT (UI/L)	10.65 ± 1.43	13.90 ± 1.43
Bilirubin (mg/dl)	2.21 ± 0.19	2.48 ± 0.20
γGT (UI/L)	17.82±1.51	17.21 ± 1.43
CK (UI/L)	166.35 ± 18.24^{A}	284.50 ± 18.24^{B}
AP (UI/L)	293.12 ± 29.22^{a}	345.73 ± 27.64^{b}

rise in endogenous cortisol.[20] The increase in the α_1 e α_2 globulin fractions noted in the protein pattern with no change in total protein concentrations is probably connected with overtraining syndrome because alpha-globulins are often increased after tissue injury.[18] The subjects of group B had consistently lower BCS, although their food intake never decreased, which is usually the case in overtraining.[20] Functionally, assuming that an increase in the oxidative capacity of muscle fibers of these subjects led to beta oxidation of fatty acids, thus resulting in weight loss, would be logical.

An appropriate training schedule and reasonable race frequency are key to management of a trotter. Failure to comply with such principles may lead to the onset of the overtraining syndrome, a subtle condition that initially manifests itself with the horse's poor performance and subdued liveliness.[12] Because sound scientific information on how to detect overreaching is lacking, and little is known about how to determine optimal amounts of training or ideal regenerative periods, detecting or preventing overtraining[8] is not easy. Our investigation may contribute helpful indication. Our findings did not point to a typical

picture of overtraining in absolute terms, or one that was consistently documented by laboratory results. However, in relative terms or by way of comparison, they suggest that if overtraining is suspected in a horse with a history of impaired racing performance, indices may be identified within a set of normal values that account for the subject's physical alteration and poor performance and may help confirm a diagnostic hypothesis of overtraining.

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