been speculated that athletes with a particular inherited pattern of arrangement of myocardial fibres develop hypertrophy that is asymmetrical,[5] and that T-wave abnormalities in asymptomatic, highly trained athletes could have a neurogenic basis.[10] Normalisation of T-waves occurred during exercise and after isoproterenol infusion.[10] These authors suggested that a decreased resting sympathetic tone in trained subjects could unmask, in genetically predisposed athletes, a latent functional asymmetry of the cardiac sympathetic nerves.

The principal conclusion of the current study is that T-wave vectors in the resting ECG of the Thoroughbred racehorse are altered by training. Approximately 80% of trained Thoroughbreds develop a pattern of ventricular repolarisation which has been described in the literature as abnormal and pathological. However, presence of these T-wave patterns in the resting ECG does not indicate that the trained horse is likely to perform badly. The training-induced changes in ventricular repolarisation should be regarded as a normal physiological response to training, rather than as evidence of myocardial disease.

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The Effect of Training on Adrenocortical Function in Thoroughbred Racehorses

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ABSTRACT. The effects of training, and of age, on basal and ACTH-stimulated plasma cortisol concentrations were evaluated in 11 two year old and 16 three year old Thoroughbred colts which achieved racing fitness while in flat race training. Cortisol concentrations were measured in samples collected on the morning of rest days, before, and 2 hours after i.v. administration of synthetic ACTH (1 mg). There was no significant difference between the two age groups with respect to baseline and ACTH-stimulated cortisol concentrations; thus they were considered as a single group to evaluate the effect of training. Baseline and ACTH-stimulated cortisol concentrations declined significantly ($p<0.005$) with training but there was no significant change in the ACTH-induced increase in cortisol concentration. It is unlikely that altered sensitivity of the adrenal cortex to endogenous ACTH was responsible for the observed decline in basal plasma cortisol concentrations. This study failed to provide evidence that training induces an increase in "adrenocortical reserve" in Thoroughbred horses.

Key words Cortisol; adrenocorticotrophic hormone; ACTH; horses.

INTRODUCTION

The endocrine and metabolic responses of horses to maximal and submaximal exercise have been extensively documented.[4,5,8,12,14,18,19,20] The results of these studies indicate that both the intensity and the duration of exercise influence the magnitude and timing of increases in plasma cortisol concentration and that the cortisol response appears to be closely associated with changes in other hormones such as insulin, glucagon, catecholamines and thyroid hormones which are involved in the mobilization of metabolic substrates.[4,5,12,20,24] The effect of training on exercise-associated alterations in endocrine and metabolic functions has been studied in an attempt to evaluate adaptation of these integrated responses to increased work demands.[4,18,19] Limited information is available regarding the effects of training on indices of adrenocortical function measured in the resting horse,[4] despite the popularly held belief that "adrenal exhaustion" may be an important cause of poor performance, especially in the over-trained horse.[9,14,21]

Based on studies in Standardbred trotting horses, which showed higher resting baseline and ACTH-stimulated cortisol concentrations in a group of trained racehorses than in a group of untrained horses, it was suggested that these measures of adrenal function may be useful indicators of state of training.[14] These results also were interpreted as supporting Selye's hypothesis of adrenal adaptation in response to increasing stress demands[17] and the finding that adrenal mass increases in response to increased demand.[3,15,23] A subsequent longitudinal training study involving a small group of Thoroughbred horses failed to demonstrate any significant effect of training (2 months) on resting plasma concentrations of ACTH or cortisol, or in the cortisol response to administration of exogenous ACTH.[4]

The purpose of this study was to evaluate the effect of training to racing fitness on basal and ACTH-stimulated cortisol concentrations in a large group of Thoroughbred horses and to determine whether age-related differences exist in these parameters of adrenal function.

MATERIALS AND METHODS

Animals

Forty-one Thoroughbred colts (22 two year olds and 19 three year olds) in an English flat race training stable were used in the study. The selection of horses was based on their lack of resistance to venipuncture, absence of known pre-existing orthopedic or medical abnormalities which might preclude training to racing fitness, normal complete blood count, and the willingness of the trainer to regularly report training and racing schedules, health status, and other pertinent aspects of management and training. The 3 year old horses had been in race training the previous year as two year olds and had been maintained on a routine of light exercise (walk, trot, slow canter but no fast galloping) 6 days each week, weather permitting, up to the first sampling day in early February. The 2 year old horses had entered training in the late autumn and winter, at which time they were broken to ride and were being ridden 4 to 6 days each week at the walk, trot or slow canter up to the initial sampling period. Thirty-eight of the 41 horses entered the study in early February and 3 horses entered the study in early April after initiation of training had been delayed by minor ailments. The intensity of work was increased progressively during the training period, as deemed appropriate by the trainer for each horse, until the horses were judged to be fit enough to race (racing fitness). Factors other than fitness (e.g. the availability of suitable races, the class of the horse and ground conditions) which impacted the trainers decision to race an individual horse, were not considered in determining the time at which the horses achieved racing fitness.

Sampling protocol

Each of the horses which remained in training were sampled 5 times between early February and late September. All samples were collected on the morning of rest days and baseline samples from each horse were collected at the same time (± 30 min) on each test day. Sampling was not performed from 5 days before a race to 3 days after. Baseline blood samples were collected, between 8:30 a.m. and 9:50 a.m., by jugular venipuncture and immediately transferred to tubes containing lithium heparin. Synthetic ACTH (Synacthen™, Ciba Laboratories, Horsham), 1 mg in aqueous solution, was administered i.v. immediately after collecting the baseline blood sample, and a second blood sample was collected into heparinized tubes 2 hours later. Blood samples were centrifuged at $600 \times G$ within 2 hours of collection and the plasma was transferred to screw-capped, plastic storage tubes which were then sealed and maintained at $-20°C$ until assayed for cortisol.

Cortisol assay

Plasma cortisol concentrations were measured using a direct radioimmunoassay procedure with I^{125}-labelled cortisol tracer and polypropylene tubes covalently coated with anti-cortisol antibody (Corti-cote™, Becton Dickinson Immunodiagnostics, Orangeburg, N.Y.). Samples and standards were assayed in duplicate with appropriate high, medium, and low concentration equine control samples included in each run. All samples from each individual animal were assayed in a single run to minimize the effects of interassay variation.

Statistical methods

The cortisol concentration in samples collected from each horse early in the training period, before the introduction of maximal exercise (early February for all except 3 horses), were designated "early training". The cortisol concentration in samples collected when each horse achieved racing fitness, closest to the time of its first race, were

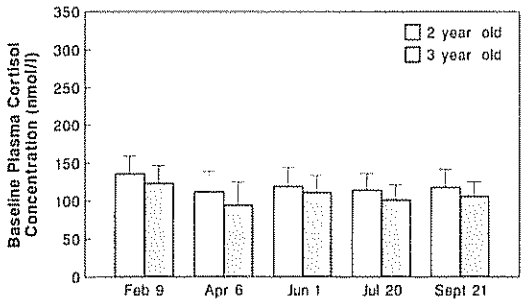

Fig 1. Baseline concentrations of cortisol in plasma samples collected on the morning of rest days from 2 year old and 3 year old Thoroughbred colts in flat race training.

designated "racing fitness". The effect of age on values for baseline cortisol concentration, ACTH-stimulated cortisol concentration, and the difference between ACTH-stimulated and baseline cortisol concentration was evaluated by comparing the results for 2 year old and 3 year old horses at both the early training and racing fitness time points using the Mann-Whitney Rank Sum Test (an unpaired *t*-test). The effect of training on the above parameters was determined by comparing the values for early training and racing fitness time points using the Wilcoxon Signed Rank Test (a paired *t*-test).

RESULTS

Inter-assay precision for 22 runs of the cortisol radioimmunoassay for the high, medium and low cortisol concentration control samples were 5.7%, 5.7%, and 8.3%, respectively. Intra-assay precision determined for quadruplicate samples of the high and low control samples included in each run were less than 7% for all runs and averaged 4.7% and 4.6%, respectively.

Twenty-seven of the 41 horses achieved racing fitness during the study. Of the remainder, 6 were removed from training before racing due to injury, illness or other reason and 8 remained in training but did not race. The number of horses in active training and the number which had achieved racing fitness at a particular sampling time are shown in Table 1. As expected, a larger number of 3 year old than 2 year old horses raced during the training period. The mean values for baseline and ACTH-stimulated plasma cortisol concentration for all 2 year old and 3 year old horses in active training at each sampling period are shown in Figs 1 and 2. The mean values for both baseline and ACTH-stimulated plasma cortisol concentration were highest for both age groups in February, early in the training program. A decline in mean plasma cortisol concentration was noted at the next sampling time in early April. Thereafter, the mean baseline and ACTH-stimulated plasma cortisol concentration for those horses remaining in active training showed only minor fluctuations at each sampling period.

Eleven of the 2 year old and 16 of the 3

Table 1. *Number of horses in active training and number having achieved racing fitness at each sampling time*

	Number in active training		Cumulative number achieving racing fitness	
Sampling time	2 year old	3 year old	2 year old	3 year old
February	20	18	0	0
April	20	18	0	1
June	19	17	0	9
July	18	16	2	16
September	17	13	11	16

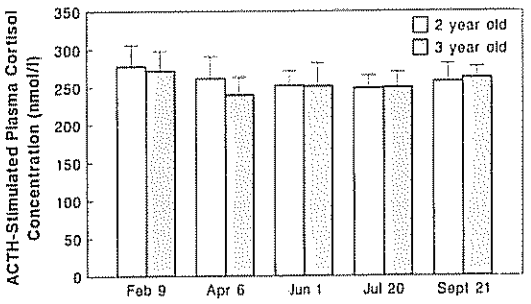

Fig 2 Concentrations of cortisol in plasma 2 hours after i.v. administration of synthetic ACTH (1 mg) to 2 year old and 3 year old Thoroughbred colts in flat race training.

year old horses became fit enough to race during the study period, permitting a comparison of baseline and ACTH-stimulated plasma cortisol concentrations and the ACTH-induced increase in cortisol concentration to be made between samples collected early in the training period with those collected when the horses achieved racing fitness. There were no significant differences between 2 year old and 3 year old horses with respect to values (mean ± SD) for early training baseline (135.9±22.8 nmol l^{-1} vs 127.6±20.3 nmol l^{-1}) and ACTH-stimulated (271.5±23.0 nmol l^{-1} vs 272.8±25.4 nmol l^{-1}) cortisol concentrations or for racing fit baseline (116.5±22.2 nmol l^{-1} vs 107.8±17.74 nmol l^{-1}) and ACTH-stimulated (263.7±21.6 nmol l^{-1} vs 248.9±23.2 nmol l^{-1}) cortisol concentrations. The 2 year old and 3 year old horses were therefore considered as a single group to evaluate the effect of training. The mean (± SD; n=27) baseline plasma cortisol concentration declined significantly ($p<0.002$) in response to training, from 131.0±22.1 nmol l^{-1} early in the training period to 111.3±20.5 nmol l^{-1} when the horses achieved racing fitness (Fig. 3). The mean ACTH-stimulated cortisol concentration also declined significantly with training ($p<0.001$), from 272.3± 25.0 nmol l^{-1} during early training to 254.9±24.2 nmol l^{-1} when the horses became racing fit (Fig. 2). There was significant difference in the mean values for the exogenous ACTH-induced increase in plasma cortisol concentration between samples collected during early training (141.3±28.9 nmol l^{-1}) and those collected when the horses were racing fit (143.6±23.6 nmol l^{-1}).

DISCUSSION

The baseline and ACTH-stimulated cortisol concentrations for the horses in this study were similar to reported values.[4,6,8,11,14] Two hours after i.v. administration of synthetic ACTH (1 mg), mean plasma cortisol concentrations were increased to more than double the baseline concentration on each occasion that the horses were sampled. These results agree with those of previous studies in which the same protocol was used for the ACTH-stimulation test.[6,8] The absolute, rather than percentage, ACTH-induced increase in plasma cortisol concentration was used to assess cortisol output in this study because it is less affected by basal plasma cortisol concentration and is, therefore, a better indicator of the functional reserve of the adrenal cortex.

No significant differences were noted between 2 year old and 3 year old horses with regard to basal and ACTH-stimulated cortisol concentrations during either the early training period or when the horses achieved

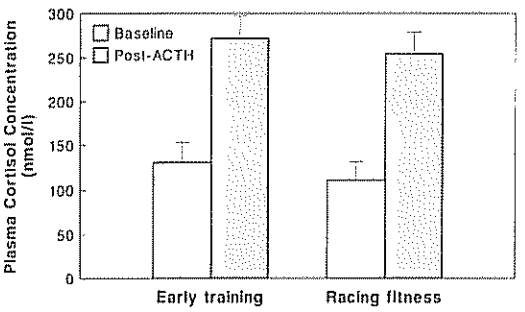

Fig 3 Concentrations of cortisol in plasma samples collected before (baseline) and after the i.v. administration of synthetic ACTH (1 mg) to 27 Thoroughbred colts during early training and again when the horses achieved racing fitness

racing fitness. These results are consistent with those of previous studies which failed to demonstrate any significant effect of age on plasma cortisol concentration in horses.[6] Statistical comparisons between the two age groups at individual sampling times were not performed because the 3 year old horses were generally at a more advanced stage of training and a greater proportion were racing fit at each sampling time after initiation of the study.

The horses used were in active training in a commercial racing stable; thus it was not possible to completely standardize experimental conditions or to objectively quantify the fitness of each horse at each sampling time. However, experimental conditions were controlled as much as possible and a large number of horses were included in order to minimize the effects of day-to-day variation and other factors which limit the value of single measures of plasma cortisol concentration for assessing adrenocortical function. The effects of time of day, feeding, activity in the stable, exercise, and the sex of the horse were minimized by collecting all samples in a standardized order on the morning of rest days and by using only colts. Baseline samples were collected at a time of day when plasma cortisol concentrations should have been at, or near, the peak of their diurnal cycle.[6 8 11 25] The possible effect of changing day length and environmental conditions could not be evaluated or standardized because a similarly managed matched group of young male Thoroughbred horses, not undergoing active training, was unavailable for study. All of the horses had been stabled on the premises for at least 2 months prior to the start of the study and were well acclimatized to the management and training routine. It is therefore unlikely that increasing familiarity with the stable routine contributed to the observed changes in cortisol concentrations, but this effect cannot be ruled out since only one set of samples was collected before the intensity of exercise was increased in the training program. It is also possible, but unlikely, that some of the observed decline in resting plasma cortisol concentrations during the first 2 months of training could have been due to changes in day length, with consequent alterations in the diurnal and episodic patterns of cortisol secretion, although there are no published reports which document such an effect in horses. Rather, the decline in basal cortisol concentrations appeared to parallel increases in the intensity of exercise during the training program. The lowest mean baseline concentration of cortisol was found at the second (April) sampling time, a time when more strenuous galloping exercise had been introduced into the training program of a large number of horses and all sound horses were experiencing an increase in the intensity of exercise performed. Despite a continued increase in day length between April and June, baseline concentrations of cortisol did not decline during this time.

The finding of a highly significant decline in baseline and ACTH-stimulated cortisol concentrations in horses in response to training for maximal exercise has not been reported previously. This may reflect the fact that smaller numbers of horses have been used in previous studies,[4 18] since, in one study, resting plasma cortisol concentrations before an exercise test appeared to be lower in trained horses than in untrained horses.[4] The observed decline in basal plasma cortisol concentrations with training may represent changes in plasma volume, increased utilization or elimination of cortisol, reduced production of cortisol from the adrenal cortex due to reduced sensitivity to endogenous ACTH, or reduced secretion of ACTH by the hypothalamus due to changes in feedback sensitivity in the hypothalamic-pituitary-adrenal axis. Since endogenous ACTH concentrations were not measured, it is not possible to accurately define which of these mechanisms were active. A diminished response of ACTH and cortisol to the administration of corticotrophin-releasing hormone has been noted in highly trained human endurance runners[13] and a blunting of the ACTH response to submaximal exercise has

been reported following endurance training in humans.[2] These studies suggest that training results in down-regulation of hypothalamic-pituitary feedback sensitivity to circulating cortisol concentrations. However, this blunting mechanism is not operational during maximal exercise following endurance training, even when interval sprint training is incorporated,[10] and studies in Thoroughbred horses have failed to demonstrate an effect of training on either resting plasma ACTH concentrations or the ACTH response to treadmill exercise.[4]

Diminished adrenocortical sensitivity to ACTH in response to training was suggested by the results of a study in which isolated adrenals from treadmill-trained rats released less corticosterone in response to ACTH than did adrenals from untrained rats and rats in the early phases of training, despite a significant increase in adrenal weight.[23] The lack of effect of training on the adrenocortical response to exogenous ACTH in the horses in the present study suggests that altered sensitivity of the adrenal cortex to endogenous ACTH is unlikely.

Training studies in humans have yielded conflicting data with regard to resting plasma cortisol concentrations. Prolonged strength training in weightlifters was associated with a decline in resting plasma cortisol concentrations.[1] In runners, sprint training induced significant increases in resting plasma cortisol concentrations, whereas endurance training did not induce changes.[10,16] Intense (90% VO_2 max) bicycle interval training in young men resulted in progressive increases in resting morning plasma cortisol concentrations during the first 4 weeks of training, followed by a decline to baseline concentrations as training continued for a further 2 weeks.[22] Similar results have been noted in rats undergoing training on a treadmill.[3] The varied results of these studies may reflect differences in experimental protocol and the degree of fitness of individuals entering the studies. However, it does appear that exercise intensity, particularly anaerobic work, during the training period may be more important than the total amount of exercise in determining the adaptive response of the hypothalamic-pituitary-adrenal axis.[10,22] This conclusion is supported by the results of the present study, since almost all of the horses showed a drop in basal plasma cortisol concentration shortly after the intensity (speed) of exercise was increased in the training program, but did not decrease further as training intensity was maintained.

The decline in cortisol concentrations with training likely reflects, at least in part, increased utilization of cortisol with increasing work demands. Strenuous training has been shown to lower plasma concentrations of cortisol-binding globulin in humans and in horses; the effect being most pronounced in males.[1,7] This results in a higher percentage of the plasma hormone being present in the free or metabolically active form and may increase the fraction taken up and utilized by the target tissues,[7] as well as making a greater proportion of the cortisol in plasma available for excretion. The net effect would be a reduction in the plasma concentration of total cortisol, but maintenance of the concentration of metabolically active free cortisol for a given adrenocortical output of cortisol.

The present study failed to provide evidence to support the concept that adrenal reserve or adrenal output increases in response to training for maximal exercise in horses. Despite a significant decline in both baseline and ACTH-stimulated cortisol concentrations in response to training, the absolute increase in cortisol concentration induced by ACTH administration showed no significant change. The latter result agrees with the findings of published studies involving smaller numbers of Thoroughbred racehorses, and with the findings of Persson et al. in studies with Standardbred racehorses, although interpretation of the results of these studies differed.[4,14] To our knowledge, no similar studies have been performed in humans to evaluate the effect of training for maximal exercise on adrenal responsiveness to ACTH at rest. However,

studies in female endurance runners and joggers failed to demonstrate any effect of training on the cortisol response to exogenous ACTH or any difference between sedentary women and women who exercised regularly.[16]

None of the horses used in the present study developed signs of over-training or poor performance, so it is not possible to evaluate whether adrenal exhaustion is a cause of poor performance in the horse. However, the parallel decline in basal and ACTH-stimulated cortisol concentrations in response to training resulted in a number of horses having basal cortisol concentrations of less than 80 nmol l^{-1} and ACTH-stimulated cortisol concentrations of less than 230 nmol l^{-1} at a time when they were in hard training and racing at a high level. Measurement of cortisol concentrations in similar individuals presented for clinical evaluation of poor racing performance could lead to an erroneous diagnosis of hypoadrenalcorticism if the presumed normal response to training was an increase in plasma cortisol concentration. Despite experimental conditions being standardized as much as possible in this study, the variation between horses and, perhaps, between different days for the same horse, suggests that in an individual horse the measurement of a single plasma cortisol concentration would be of limited value for the assessment of fitness and adrenocortical function.

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Effects of Ozone on Exercising Horses: A Preliminary Report

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ABSTRACT. Air pollution is a problem in metropolitan areas where some racetracks are located. The objectives of this study were to evaluate the effects of ozone, a major component of oxidant air pollution, on treadmill performance by exercising horses and to determine the characteristics and distribution of lesions induced by ozone. Five Thoroughbred geldings ran on a treadmill at speeds up to 16 m s^{-1}. Three were exposed to 0.8 ppm ozone and 2 to 0.25 ppm ozone on 2 consecutive days during a 9 min exercise protocol and a 20 min cooling off period. The exposure regimen simulated a "breeze" and a race on consecutive days during an air pollution episode. All horses performed well during their first ozone exposure, but one horse exposed to 0.8 ppm ozone refused to complete the exercise protocol the second day. Ozone did not alter oxygen consumption ($\dot{V}O_2$) or respiratory frequency (f_R), but did increase heart rate (f_H). Two of the 3 horses exposed to 0.8 ppm had pulmonary hemorrhages and edema. Necrosis and sloughing of type I alveolar epithelium in the centriacinar regions were seen in all horses exposed to 0.8 ppm, but their distribution was not uniform. Lesions in horses exposed to 0.25 ppm ozone were less severe and limited to ciliated cells in terminal bronchioles.

Key words Exercise; horses; ozone; air pollution; respiratory system; lungs

INTRODUCTION

Air pollution occurs in many areas of the United States and other countries at concentrations that exceed the National Ambient Air Quality Standards (NAAQS) established by the US Environmental Protection Agency. Oxidant air pollution, with ozone as its archetypical component, is characteristic of smog in the California South Coast Air Basin, which includes the Los Angeles area and 5 major racetracks. In 1988, the most recent year for which data are available, the NAAQS for ozone, 0.12 ppm, was exceeded 178 days. During 78 of those days the concentration was 0.20 ppm or greater.[5] As in previous years, the highest and second highest concentration reported in that air basin in 1988 were 0.35 and 0.34 ppm.[5]

Inhalation of low concentrations of ozone results in physiological, biochemical and morphometric changes in the respiratory system.[20,21] While few physiological changes are reported in resting humans exposed to ozone, exercising humans have significant decrements in pulmonary functions.[1,15] Exercise increases the total dose of ozone inhaled by increasing ventilation, and is a major factor influencing both the total dose and the dose rate.[1,13,15,16] In a review of a large series of studies it was reported[11] that inhalation of ozone during exercise in humans results in reductions in forced vital capacity (FVC) and forced expiratory volume at 1 s (FEV_1), and an increase in airway resistance (R_{aw}). Some studies suggest that these physiological changes may be accompanied by a reduction in performance by trained athletes.[1,9,10,15,16] Although many biochemical