

at 09.00 h (day 7). Lymphocyte counts were significantly higher ( $p < 0.002$ ) at 16.00 h than at 08.30 h on the day of no exercise. On day 7 total leucocyte counts and absolute neutrophil counts were significantly higher ( $p < 0.005$ ) at 16.00 h than at 08.30 h. Erythrocyte parameters tended to be slightly higher at 16.00 h than at 08.30 h on day 7, but these changes were not significant.

### Discussion

The results of the first part of this study show significant changes in several haematological parameters, following 20 weeks of regular non-standardized physical training in 32 previously untrained two-year-old Thoroughbreds.

After 20 weeks of training there was a significant increase in mean Hb, RBC and PCV, which both confirms earlier work by several authors (Kitchen *et al.*, 1965; Clarkson 1968; Catling 1978) and suggests that these horses had shown a normal response to prolonged regular exercise. The mean increase in haemoglobin of 17 g/l agrees well with the 28 g/l increase found by Stewart *et al.* (1970). Since blood volume measurements were not undertaken in this study, it was not possible to assess whether the increase in haemoglobin values reflects a true increase in total body haemoglobin as shown by Persson (1967). In contrast to the increase in erythrocytes, total and differential leucocyte counts remained remarkably constant. Rose and Hodgson (1982), working with endurance horses, also found that the total and differential leucocyte count remained fairly stable during training. Although platelet counts in this study were found to be low when compared with non-Thoroughbreds (Schalm *et al.*, 1975), they remained unchanged by training.

The significant increase in total bilirubin observed after training was probably due to decreased caloric intake rather than to an abnormal rate of erythrocyte destruction. In man, severe prolonged exercise produced marked increases in serum bilirubin values caused by a combination of haemolysis and decreased caloric intake. (Lindemann *et al.*, 1978). In the horse, haptoglobins remained constant during training (Allen 1978), suggesting no increase in erythrocyte turnover. Fasting, on the other hand, is known to cause considerable increases in plasma bilirubin concentrations (Gronwall and Mia, 1975).

Plasma viscosity and fibrinogen levels were unaffected by training. It is interesting to note, however, that plasma viscosity values in the Thoroughbred are lower than in other breeds of horses and all other animals so far studied (Archer and Allen, 1970). This may be an important factor to consider when assessing the relationship between blood viscosity, exercise tolerance and fitness.

The fall in serum and erythrocyte folate levels together with a fall in serum vitamin B<sub>12</sub> levels is consistent with an increased demand for the vitamins during a prolonged training programme. Since folate levels are low and vitamin B<sub>12</sub> levels are high in the Thoroughbred compared with other species, supplementation of the diet with folic acid rather than with B<sub>12</sub> should improve the horse's haematological status. Furthermore, it has been shown in this investigation that the mean serum folate level gradually fell in a group of eight Thoroughbred racehorses during a two-year training period. This would tend to suggest that older horses, rather than previously untrained younger animals, may be more prone to folic acid deficiency states. Chanarin *et al.* (1969) have suggested that

there is a developing economy in the use of folates when body stores reach low levels. In the Thoroughbred there is the possibility that normal body functions can still be maintained on very low levels of this vitamin.

The second part of this study investigated the within-day changes in several haematological parameters in a group of healthy Thoroughbreds in training. Venepunctures were undertaken at 08.30 h and 16.00 h on two separate days, one when the horses were subjected to strenuous exercise at 09.00 h and the other when they were not. On the exercise day total leucocyte and absolute neutrophil counts were significantly higher at 16.00 h than at 08.30 h, suggesting that a time lapse of approximately seven hours is insufficient for these parameters to return to pre-exercise levels. These findings have previously been reported in horses subjected to maximal exercise and would tend to be indicative of a stress response (Carlson 1975; Catling 1978; Snow *et al.*, 1982). Conversely, on the day of no exercise leucocyte values obtained at 08.30 h agreed well with those at 16.00 h, with the exception of the absolute lymphocyte count which was significantly higher at 16.00 h. Erythrocyte parameters remained fairly constant when the horses were sedentary, but on the exercise day values were higher at 16.00 h than 08.30 h, presumably as a result of prior splenic contraction or a decreased plasma volume. There is thus strong evidence to suggest that blood samples should not be collected in the afternoon following strenuous exercise in the early morning. In addition, because of the diurnal variation of the lymphocyte count there is a need to standardize the time of venepuncture, especially for studies involving daily blood counts.

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## Exercise-Induced Alterations in Haemostasis in Thoroughbred Horses

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### Summary

*The effects of exercise on blood coagulation, platelet function and fibrinolysis were studied in five fit Thoroughbred horses which were galloped at maximal speed for 1200 m. Blood samples were collected four hours before exercise (resting sample), after saddling the horse and walking him to the track (track sample) and five minutes after exercise (post-exercise sample). Activated partial thromboplastin times, one-stage prothrombin times and thrombin times did not significantly differ in any of the samples, nor were fibrinogen/fibrin degradation products detected at any stage. The slopes of the ADP-induced platelet aggregation curves were reduced, but not significantly, when results from track samples were compared with resting sample findings. Exercise induced a significant reduction in the slopes of all ADP-induced aggregation curves. It was hypothesized that this response was an indirect effect of increased plasma catecholamine levels, possibly mediated via the release of increased amounts of prostacyclin from vascular endothelium.*

### Introduction

Normal haemostasis depends on a balanced interplay between the vessel wall and the factors responsible for blood coagulation, platelet function and fibrinolysis, which together comprise the haemostatic system. Considerable study has been made of the effects of physical exercise on blood coagulation and fibrinolysis in people, especially in relation to myocardial and vascular disease. In humans, exercise results in a shortening of clotting time (Ikkala *et al.*, 1963), an increase in platelet number and aggregation (Poller *et al.*, 1971; Warlow and Ogston, 1974), and an increase in fibrinolytic activity (Egeberg 1963). The extent of these changes varies with the length and intensity of the exercise and the physical condition of the subject. The effects of exercise on platelet adhesiveness are more variable, being either unchanged or reduced, depending on the exercise (Bennett 1972).

Little is known of the effects of exercise on the haemostatic system in horses. The effects of exercise on haemostasis are of particular interest in this species because of the

reportedly high prevalence of pulmonary haemorrhage in Thoroughbred and Standard-bred horses following maximal or near maximal exercise (Pascoe *et al.*, 1981; Raphael and Soma, 1982). The pathogenesis of this syndrome, which is referred to as exercise-induced pulmonary haemorrhage, has not been established, although it is widely believed to be associated with pre-existing pulmonary disease (Robinson 1979). It is possible that exercise-associated alterations in haemostasis may be important in the development of post-exercise pulmonary haemorrhage. Before this can be evaluated, the effects of maximal exercise on haemostasis in normal Thoroughbred horses need investigation. The results from such a study are presented in this paper.

### *Methods and Materials*

Five clinically normal mature Thoroughbred horses that were in racing condition (two mares and three geldings) were used. No medication had been administered for two weeks prior to initiating the study. The horses were housed indoors and exercised daily.

Measurements were made of selected haemostatic and haematologic parameters. The packed cell volume (PCV) was obtained with the aid of the microhaematocrit centrifuge (Damon, IEC Division), erythrocyte counts and platelet counts were performed using an automatic cell counter (Coulter Electronics), plasma protein concentration was determined using a hand-held refractometer (National), and plasma lactate concentration was determined by an enzymatic procedure (Sigma). The following coagulation recalcification tests were performed using a fibrometer (BBL Fibrosystem): activated partial thromboplastin time (APTT), one-stage prothrombin time (OSPT), and thrombin time. The thrombin time was measured with topical thrombin (Parke Davis). A commercially available test (Burroughs Wellco) was used to determine the presence of fibrinogen/fibrin degradation products by latex agglutination, after it was verified that the degradation products of equine fibrinogen were active in the assay. Platelet aggregation in response to arachidonic acid (2.0, 1.0, 0.5, 0.25, 0.125, 0.0625 mM) and adenosine diphosphate (ADP) (10.0, 5.0, 2.5, 1.25, 0.625  $\mu$ M) was determined using a chronolog aggregometer as described previously (Meyers *et al.*, 1979b). The initial slope of the aggregation response curves was determined by measuring the per cent increase in the transmission of light on a time basis. Plasma total catecholamine concentration was also measured (Upjohn Diagnostics). Values were expressed as the mean  $\pm$  standard error of the mean (SEM), unless otherwise stated.

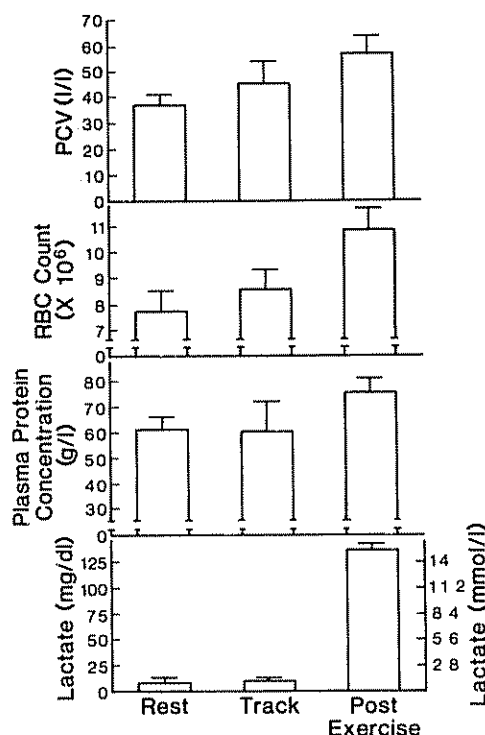
Blood was collected through 16 g needles by jugular venepuncture using the following sampling schedule: Resting values were determined from a blood sample (resting sample) taken at 08.00 h on the day of exercise. Each horse was saddled and brought to the track at 12.00 h and sampled again to determine the effects of anticipatory excitement (track sample). It was then exercised immediately on the racetrack at racing speed for 1200 m. Five minutes after exercise the horses were again sampled (post-exercise sample).

### *Results*

Placing the horses in a track environment resulted in an increase in the number of circulating erythrocytes and platelets. There was approximately a 20% increase in PCV, erythrocyte count and platelet count in the blood samples taken at the track compared to those at rest, while plasma protein concentration and plasma lactate concentration

remained essentially unchanged (Figs. 1 and 2). The increase in the number of circulating red blood cells (RBC) was accompanied by a 33% increase in the total plasma catecholamine concentration which increased from a resting value of  $946 \pm 223$  pg/ml to  $1262 \pm 257$  pg/ml when the horses were at the track.

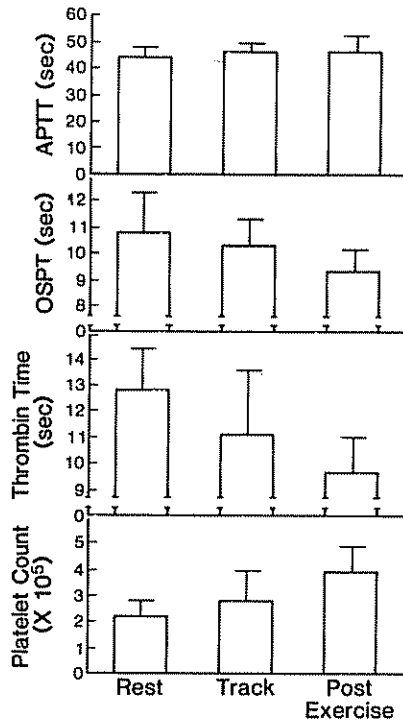
FIGURE 1. Packed cell volume (PCV), red blood cell (RBC) count, plasma protein concentration and plasma lactate concentration seen in five horses at rest, immediately before exercise and five minutes post exercise. (Data are presented as mean  $\pm$  SEM).



Bringing the horses to the track resulted in a non-significant reduction in the slope of the platelet aggregation response to ADP (Fig. 3). The height and the extent of the aggregation wave were not consistently changed (data not shown). Arachidonic acid only induced reversible aggregation, and the slope of this aggregation response was not altered. The coagulation tests that were performed did not change at this stage (Fig. 2).

The horses were subsequently strenuously exercised, and plasma lactate concentrations exceeded 14.0 mmol/l (Fig. 1). In the five-minute post-exercise sample there was a further increase in PCV, erythrocyte count, and platelet count, and these values were 50%, 40% and 80% higher, respectively, than the resting values (Figs. 1 and 2). Accompanying this increase in the formed elements of the blood was a 22% increase in plasma protein concentration (Fig. 1). In addition, there was a 189% increase in total plasma catecholamine concentration over resting values, the concentration five minutes after exercise having increased to  $2735 \pm 600$  pg/ml.

FIGURE 2. Values for parameters of haemostasis seen in five horses at rest, immediately before exercise and five minutes post exercise. (Data are presented as mean  $\pm$  SEM; APTT = activated partial thromboplastin time; OSPT = one-stage prothrombin time).



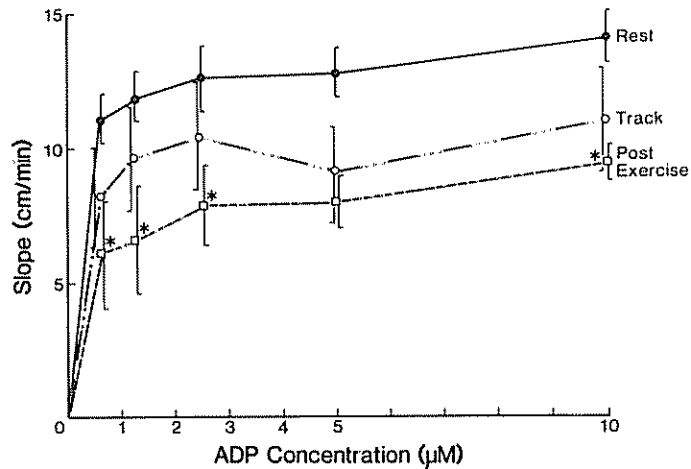
Exercise further decreased the slope of the ADP-induced equine platelet aggregation response (Fig. 3). The depression in slope was observed at all ADP concentrations. The height of the ADP-induced aggregation curve was altered in some but not all samples (data not shown). Changes in the arachidonate-induced aggregation response were not seen.

Some changes in the coagulation times were noticed after exercise. There was a decrease in the OSPT and thrombin time, although the APTT remained unchanged (Fig. 2). The Burroughs-Wellco test for fibrinogen/fibrin degradation products was negative in all samples.

### Discussion

Selected tests of haemostatic competency were conducted in Thoroughbred horses subjected to an exercise régime similar to that to which a racehorse would be exposed. This régime can be subdivided, First, there is a behavioural display of marked excitation or anticipation upon arrival at the track prior to exercise. This is associated with an elevation of heart rate, respiratory rate and haematocrit, and is thought to be catecholamine mediated. Our findings were compatible with those observations and suggestions. In addition, we found that not only was the PCV increased, but platelet counts were elevated

FIGURE 3. The slopes of ADP-induced aggregation curves seen at rest, immediately before exercise and five minutes after exercise. (Data are expressed as mean  $\pm$  SEM; \* denotes significant difference from resting value ( $p < 0.05$ )).



too. This contrasts with the results from a previous study using ponies, which indicated that there was no apparent change in platelet numbers, following moderate exercise (Lepherd 1977). In human studies, however, changes in platelet numbers similar to those seen in the present investigation have been reported, following short periods of strenuous exercise (Warlow and Ogston, 1974). The increase in erythrocyte and platelet numbers was probably due to splenic autotransfusion caused by sympathoadrenal activity (Persson 1975).

Exercise itself resulted in additional changes. In this study, the level of exertion was gauged by measuring the plasma lactate concentration five minutes after exercise. This test was selected because other assessments of the level of exercise, such as oxygen consumption and heart rate, can only be estimated with difficulty when using the type of exercise employed in this study. Previous track studies have indicated a close relationship between heart rate and plasma lactate concentrations in horses exercising anaerobically (Persson and Ullberg, 1974). The plasma lactate concentrations were determined to be in the range reported for Thoroughbred and Standardbred horses after racing or simulated racing (Asheim *et al.*, 1970), suggesting that the five horses used in this study were subjected to an appropriate exercise schedule.

A further increase in platelet and RBC numbers was observed following exercise. This well documented increase was accompanied by a marked elevation in total catecholamines and plasma protein suggesting that it was, in part, due to a reduction in plasma volume as well as continued splenic autotransfusion.

The OSPT and APTT were not significantly altered by excitement or exercise. These findings were in keeping with results from studies in humans (Ikkala *et al.*, 1963). However, the haemostatic system was changed by the pre-exercise anticipation and by the exercise itself. The changes were manifest as a reduction in the slope of the ADP-induced platelet aggregation wave and a decrease in thrombin times. The reduced thrombin time

was not followed by a change in the OSPT or the APTT, and therefore the impact of this change must be questioned.

Platelets play a prominent rôle in haemostasis, and alteration in platelet adhesion or aggregation can lead to severe bleeding. The functional capacity of the platelet can be assessed *in vitro* with an aggregometer. The slope of the aggregation response appears to be an appropriate indicator of the functional status of the platelet (Roper-Drewinko *et al.*, 1981), and it is reduced when the excitability of the platelet is decreased.

The ADP-induced aggregation was modified in this study, but arachidonic-acid-induced aggregation did not change. Differences in the aggregation response of platelets to different agonists are not unprecedented. Arachidonic acid is metabolized by the platelet into the endoperoxides prostaglandin (PG)  $G_2$  and  $PGH_2$  (Svensson *et al.*, 1976). These can then be further metabolized into thromboxane (TX)  $A_2$ , a potent platelet agonist (Svensson *et al.*, 1976). On the other hand, ADP is a weak platelet agonist (Holmsen 1978) and, as such, it may be a more sensitive indicator of changes in the responsive capacity of platelets.

The mechanism by which pre-exercise anticipation and exercise induce a decrease in platelet response to ADP is not known. The slope of the ADP-induced aggregation curve was decreased prior to and following exercise, that is, at times when plasma catecholamine levels were elevated. It is therefore possible that the responses observed were in part catecholamine mediated. Platelets from several species have alpha and beta receptors (Scrutton 1982). Alpha receptor activation potentiates the action of other agonists, while beta receptor activation results in inhibition of platelet aggregation (Scrutton 1982). Equine platelets, however, are neither potentiated nor inhibited by adrenaline (Dodds 1978; Meyers *et al.*, 1979b). A direct effect of adrenaline can therefore not account for the effects observed, and an indirect effect must be sought. Catecholamines have a major effect on the equine vasculature. The vascular endothelium in several species can produce  $PGI_2$ , a potent inhibitor of platelet aggregation (Monconda and Vane, 1978). The liberation of  $PGI_2$  during excitement or exercise could account for the reduction seen in ADP-induced aggregation.

The effects of exercise on fibrinolysis have been extensively investigated in humans. It has been demonstrated that exercise is accompanied by an increase in fibrinolytic activity which is related to the intensity of the exercise (Ogston and Fullerton, 1961; Davis *et al.*, 1976). In one study, little change in fibrinolytic activity was seen until 70 to 80% of maximum heart rate was reached (Davis *et al.*, 1976). The plasma lactate concentrations measured in the five horses in this study would suggest that their heart rates were close to maximum; however, no increase in fibrin degradation products was noted. It is possible that the duration of the exercise period was not sufficient to produce a noticeable increase in fibrinolytic activity. It has been suggested that the magnitude of the fibrinolytic response in man depends on both the intensity and duration of exercise (Rosing *et al.*, 1970), although this has been disputed in part (Davis *et al.*, 1976). It is also possible that this represents another example of the different responses produced in haemostasis by exercise, when comparing horses and people.

The observed changes in platelet function, following exercise, were detected in horses under training but not in racing conditions. Extrapolation of these data to other situations must be done with caution since exercise length and intensity and physical condition of the subject may influence the findings. Nevertheless, the reduction in ADP-induced

platelet aggregation may have clinical significance. When a vessel is traumatized, platelets adhere to the exposed surface and secrete the ADP and 5-hydroxytryptamine that is stored in intracellular granules. The release of these substances causes incoming platelets to adhere and then undergo granule secretion and results in the formation of TXA<sub>2</sub>. This occurs until a platelet plug is formed which is then solidified by the deposition of fibrin. The release of ADP and the response of the platelets to ADP appear to be critical for normal haemostasis, as there is a marked impairment of haemostasis in animals in which stored ADP granules are absent from platelets (Meyers *et al.*, 1979a, 1982; Raymond and Dodds, 1975). A reduction in the sensitivity of the platelets to ADP, as was found in this study, may therefore impair the formation of the platelet plug. Thus, if bleeding was initiated, haemostasis may be impaired, and it is conceivable that an exercise-induced reduction of platelet function could exacerbate pulmonary haemorrhaging in Thoroughbred and Standardbred horses. The total effect of exercise on haemostasis in the horse could be better evaluated by recording bleeding times. This was not done in this study because of the difficulty in making controlled measurements, which makes the interpretation of results highly equivocal.

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# TRAINING AND BLOOD CHEMISTRY

## I. Resting

### The Biochemistry and Haematology of Inherent Performance

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#### Summary

*Between 1974 and 1981 blood samples were analyzed from horses at varying intervals of time for over 28 components. Many horses exhibited marked individuality, and individual normal values for many blood components were established. When these data were computerized and animals assessed as having sub-clinical disease were removed from the survey, a study was undertaken to examine whether the remaining results which would reflect the inherent biochemistry and haematology of the horse were related to their Timeform rating at the end of a season. The results indicated that there was a relationship between plasma sodium concentration and red blood cell count in blood samples taken six hours after exercise with their Timeform rating. There was a small but significant relationship with plasma urea concentration. These observations suggest that apparently the inherent performance of the Thoroughbred is associated with its water balance and possibly its capacity to utilize protein.*

#### Introduction

This study was undertaken to ascertain whether there is a relationship between the concentration of haematological and biochemical components of blood and the inherent performance of Thoroughbreds in training and racing.

It was essential in a study of this nature, when comparing analyses of blood samples in differing horses, to ensure that results were not biased by sampling effects, analytical variation, natural physiological variation, sub-clinical or clinical disease, effects of training and diet and the more obvious effects of age and sex. The examination of these effects has been previously reported (Blackmore 1975), and the concept of individual normality for each subject under study was introduced. The analysis to be reported presents an extension of that study to confirm the individuality hypothesis and a comparison of blood chemistry and haematology with performance as assessed by Timeform rating at the end of the racing season.

#### Materials and Methods

##### *Series One: to examine individual variance*

Eight Thoroughbred yearlings were sampled at 14-day intervals for a period of ten