Pferdeheilkunde 12 (1996) 4 (Juli August) 401–404

Physiological responses to stress in the horse

J. H. Foreman[1] *and Adriana Ferlazzo*[2]

[1]University of Illinois, College of Veterinary Medicine, Department of Veterinary Clinical Medicine, Urbana, USA
[2]Universita di Messina, Instituto di Fisiologia Veterinaria, Messina, Italy.

Summary

Stress has been defined as a state in which an animal "is required to make abnormal or extreme adjustments in its physiology or behaviour in order to cope with adverse aspects of its environment and management". The most common stressors for horses are transport, exercise, lameness, and environmental temperature and humidity changes. Considerable interindividual animal variation occurs in the endocrine response observed subsequent to exposure to these stressors, particularly transport and lameness, and indicates considerable variation in individual animals' psychological responses to stress. Resting, exercise, and recovery heart rates have also been shown to be useful indicators of fatigue or lameness. Both exercise and transport have been demonstrated to have considerable effects on pulmonary defence mechanisms and other immunological functions.

Keywords: horse, stress, exercise, transport, cortisol

Physiologische Reaktionen des Pferdes auf Streß

Streß kann als ein Zustand beschrieben werden, in dem von dem Tier "eine ungewöhnliche oder extreme Anpassung seiner Physiologie oder seines Verhaltens gefordert wird, damit es mit ungünstigen Umständen in seiner Umgebung oder seinem Management fertig werden kann". Streß wird beim Pferd am häufigsten durch Transport, Belastung, Lahmheit und Veränderungen in der Umgebungstemperatur und Luftfeuchtigkeit ausgelöst. Die endokrinologischen Reaktionen auf diese Stressoren sind von Pferd zu Pferd sehr verschieden. Dies gilt insbesondere für Transport und Lahmheit und weist darauf hin, daß die individuelle psychologische Reaktion des Tieres auf Streß beachtliche Variationen aufweist. Als gute Indikatoren für Erschöpfung und Lahmheit haben sich die Herzfrequenzen unter Ruhebedingungen, bei Belastung und in der Erhohlungsphase bewährt. Für beides, Transport und Lahmheit, konnte gezeigt werden, daß sie die pulmonalen Abwehrmechanismen und andere immunologische Funktionen in beachtlichem Ausmaß beeinflussen.

Schlüsselwörter: Pferd, Streß, Belastung, Transport, Cortisol

Introduction

Stress has been defined as a state in which an animal "is required to make abnormal or extreme adjustments in its physiology or behaviour in order to cope with adverse aspects of its environment and management" (*Fraser, Ritchie* and *Fraser,* 1975). Stress is an everyday factor in the lives of many horses, particularly those horses which are actively travelling, training, and competing. The severity of such stress is routinely measured experimentally in horses and in other species by physiological variables such as heart rate, body weight, and activity level and by endocrinological variables such as plasma cortisol, cathecolamine, and beta-endorphin concentrations. The most common stressors for horses are transport, exercise, lameness, and environmental temperature and humidity changes.

Transport stress

The transportation procedure usually results in interactions of many different stressors, including the individual subjected to the interactions, the environment, other co-specifics, and always the human(s) subjecting the individual to transport. The different steps of the transportation process can have varying stressful effects on different individual animals. Recent studies on farm animals and horses have elucidated the selected influences of handling, loading and unloading procedures, and confinement on either a stationary or a moving vehicle (*Ferlazzo* 1995). In this sense, the stressors are physical and also psychological, and their severity may depend on the degree of novelty of these various procedures to the individual animal.

Several investigations have focused on the overall incidence of physical stressors in equine transport, but more recently, more specific stressors have been partitioned and examined individually. Mean heart rates are usually elevated during loading and also during 25 min of road transport (*Waran* and *Cuddeford* 1995). Abnormal body positions have also been documented (*Waran* and *Cuddeford* 1995). *Smith* et al. (1994) recently demonstrated that horses actually individually selected the positions in which they travel when trailered untethered over 32 km. Horses spent significantly more time facing backward when the trailer was moving but not when it was stationary. Heart rates were significantly higher when moving than when parked, but HR were no different when tethered either facing forward or backward or when allowed to move around untethered.

Several investigators have demonstrated increases in plasma cortisol after road transport in horses (*Petazzi* et al. 1983; *Sonnichsen* and *Jorgenssen* 1988; *Ferlazzo* et al. 1993a). Conversely, *Linden* et al. (1991) showed only slight mean changes after transport, but their low experimental number (n=5) yielded wide inter-individual variation which the investigators interpreted as indicative of "transport-induced stress being highly influenced by individual psychological factors". Cortisol changes after transport appear to be significant only at the middle distances (*Ferlazzo* et al. 1993a; *Fazio* et al. 1995; *Fazio* et al. 1996) and especially in Arabian and Anglo-Arabian crosses (*Ferlazzo* et al. 1993a). A concomitant increase in plasma triiodothyronine concentrations has also been reported after transportation of middle distances (*Fazio* et al. 1995). Initial handling of horses does not seem to cause hypercortisolemia (*Fazio* et al. 1995). Confinement on the vehicle has been shown to cause significant increases in beta-endorphin and ACTH concentrations only after short transport of <50 km (*Fazio* et al. 1996). Plasma concentration of myocardial depressant factor (MDF) peptide fraction is significantly lowered by road transport when the transport distance exceeds 100 km (*Vinci* et al. 1994).

Transport-stress induced changes in haematology (stress neutrophilia) and serum biochemistry variables have also been demonstrated to occur during air transport of 112 horses from London, England to Sydney, Australia (*Leadon* et al. 1990). During the flight, there were no differences in these variables between normal horses and those which eventually became ill upon arrival (n=7). However, after arrival, ill horses had further increases in peripheral white blood cell counts, neutrophil counts, and serum globulin and fibrinogen concentrations compared to their healthy counterparts. These additional haematological changes were thus reflective of the development of respiratory illness but the initial changes were not valid predictors for which horses would become ill, since all horses manifested some initial changes. Similar stress neutrophilia was documented after ground transportation of 337 Thoroughbred horses in Japan (*Yamauchi* et al. 1993). Other investigators have also recorded increases in serum muscle enzyme concentrations in horses and donkeys after road transport (*Ferlazzo* et al. 1982; *Ferlazzo* et al. 1983; *Ferlazzo* et al. 1984), indicating increased muscle stress in dealing with the movements of overground transport.

Transport may be a risk factor for subsequent respiratory illness partly due to the dramatic fluctuations in environmental temperature and relative humidity which have been demonstrated to occur, for instance, inside an aircraft (*Leadon* et al. 1990). Large increases in bacterial colony forming units have been detected in air sampled during flight, implying that the environment might have been the source of bacterial causes of post-transport respiratory illnesses (*Leadon* et al. 1990). Horses with their heads tied relatively tightly have a greater tendency to develop positive bacterial cultures from their tracheobronchial secretions than do horses which are left with their heads untied or tied loosely, presumably allowing them to adopt postural drainage more readily (*Racklyeft* and *Love* 1990).

A history of recent transport has been proven in a case-control study to be a significant risk factor in the development of pleuropneumonia in horses (*Austin* et al. 1995). In another study, few significant risk factors for post-transport pneumonia were identified in a cohort of 60 horses transported 515 km overground (*Hungerford* et al. 1992). There were no age, breed, or sex-related risks. Heavier, well-kept horses were surprisingly at increased risk for disease and for prolonged disease, implying that horses which had been relatively sheltered or unexposed to foreign antigens, viruses, or bacteria were at greater risk when subjected to transport stress.

Transport has been shown to have adverse effects on the immune system. *Anderson* et al. (1985) examined 9 horses with viral infections (primarily equine influenza) after recent transport. These subjects were compared to 3 horses with no history of transport stress. Affected horses' pulmonary macrophage numbers and activity were decreased compared to controls. *Bayly* et al. (1987) reported, after ground shipment of 1930 km, a significant decrease in numbers of pulmonary macrophages, neutrophils, and lymphocytes recovered from bronchoalveolar lavage (BAL) fluid. These effects peaked in severity one week after shipment and remained measurable for 4 weeks. *Crisman* et al. (1992) shipped 7 Thoroughbred horses 1050 km and demonstrated transiently decreased pulmonary macrophage numbers (1 week later), viability (week 2), and killing activity (week 2). *Traub* et al. (1988) shipped 8 horses for 12 hrs and examined the effects on the horses' BAL fluid cell numbers. While their BAL technique apparently resulted in increased cell numbers even in their control (non-transported horses), there was a delay in the development of this increased cell number for several days after transport. They concluded that transport stress decreased their subjects' ability to respond, with increased inflammatory cell numbers, to the recurrent BAL. Macrophage function was not measurably affected in transported horses compared to controls.

Recommendations for minimising transport-related problems have been made and are summarised in Table 1 (*Leadon* et al. 1990). Of utmost importance is that horses be examined for illness prior to transport, that their environment during transport be maintained in as hospitable and hygienic a manner as possible, and that they be monitored intensively before, during, and after transport for the first signs of illness.

Tab. 1: Recommendations for minimising transport stress and post-transport disease in horses travelling long distances either by ground or air (quoted from *Leadon* et al. 1990).

1. Check health status prior to and on the day of the journey.
2. Avoid unnecessary medication.
3. Take care in the administration of laxatives.
4. Ensure adequate air hygiene.
5. Provide good clean hay and an absorbent floor covering.
6. Provide water and hay every 6–8 hours.
7. Avoid delays and provide adequate ventilation.
8. Provide overnight rest periods when appropriate.
9. Express customs clearance and the relaxation of airport curfews should be provided by the relevant authorities.
10. Check health status after arrival.

Exercise stress

Exercise results in increased cortisol (*Kurcz* et al. 1988; *Ferlazzo* et al. 1993b; *McKenna* et al. 1993), catecholamine (*Snow* et al. 1992), and beta-endorphin (*McKenna* et al. 1993) concentrations in the horse. Short-term, high intensity exercise (e.g., Thoroughbred racing) and exhaustive endurance exercise both result in higher increases in plasma cortisol than exercise of moderate intensity (*Linden* et al. 1991). The introduction of exercise training to young, previously untrained horses has also been shown to be stressful as indicated by increases in plasma cortisol and lactate (*Malinowski* 1987), as has the act of weaning foals from mares (*Malinowski* et al. 1990).

Endocrinological responses to short but sporadically intensive exercise (such as competitive stadium jumping) have varied among studies. In one experiment, cortisol concentrations after jumping competition were not elevated (*Covalesky* et al. 1992), whereas after another jumping competition, cortisol was significantly elevated (*Lin-*

den et al. 1991). However, in the latter study which compared 5 different types of exercise, the post-exercise increases in cortisol were the lowest after jumping; higher after trotting racing, galloping racing (2-fold increase), and 3-day cross country; and highest (3-fold increase) after endurance racing. The investigators concluded that "the degree of increase in plasma cortisol concentration appeared to reflect both the intensity and duration of workload; endurance ride appeared to be the most exhaustive and show jumping the least" (*Linden* et al. 1991).

Motivational aspects and psychological stressors connected to competition have been studied with regard to their effects on adaptive effects on the hypothalamic/pituitary/adrenocortical axis (*Ferlazzo* et al. 1994). Lowered basal serum cortisol concentrations were recorded in resting and training sport horses before undergoing physical activity. Moreover, a pattern of adrenocortical response to exercise was observed in both sport and non-competitive horses subjected to training. There was a sudden increase (20–25%) after exercise which was more rapidly compensated (in 60 min) in sport horses. Also in sport horses, a significant increase of cortisol concentration was recorded 15–30 min after a show jumping competition.

Urinary clearance of plasma cortisol is also increased after exercise (*Toutain* et al. 1995). This exercise-induced increase in urinary clearance may result in interference in measurements of cortisol which may be present in urine as a metabolite of exogenously-administered corticosteroid or ACTH. Although not legal under the rules of racing, ACTH is commonly administered to horses as a supposed anti-inflammatory medication before racing in the USA, particularly because it is not easily detectable as excessive or exogenous.

The stress of exercise has significant effects on other homeostatic mechanisms such as immune responses. Strenuous exercise results in decreased pulmonary macrophage numbers and function (*Huston* et al. 1987); decreased lymphocyte blastogenesis responses (*Kurcz* et al. 1988); decreased neutrophil chemotactic index and peak chemiluminescence (*Wong* et al. 1992); and decreased respiratory burst activity of peripheral and BAL granulocytes (*Adamson* and *Slocombe* 1995). Conversely, moderate intensity, less exhaustive exercise has been said to be immunostimulatory rather than immunosuppressive (*Hines* and *Schott* 1995).

These immunological data (*Huston* et al. 1987; *Kurcz* et al. 1988; *Wong* et al. 1992; *Adamson* and *Slocombe* 1995) underscore the need for proper recovery periods after strenuous bouts of exercise in order to prevent any immunosuppression which may result in vivo. Of particular concern are the data which demonstrate that strenuous exercise impaired neutrophil activity (*Wong* et al. 1992) and granulocyte burst activity (*Adamson* and *Slocombe* 1995) for one or more days after exercise. Guidelines have previously been published for appropriate intervals in which to expect post-exercise heart rate to return to pre-exercise levels in normal Thoroughbred racehorses (*Foreman* et al. 1990): 20 min for trotting workouts, 40 min for galloping workouts, and 60 min for breezing or sprinting workouts. If heart rate has not returned to rest within these timeframes, the trainer should have the horse examined for suspected lameness or other occult disease which is stressing the horse and preventing a return to normal at an appropriate interval after exercise.

Lameness

Lameness is the most common cause of lost training time in athletic horses. While lameness is actually a disease, and it can be correctly argued that any disease is stressful, some initial data on lameness are presented here since so many horses are subject to lameness during their athletic careers. In two separate studies on wastage in the Thoroughbred racehorse industry, 53% of horses in training were lame at some point in one season (*Jeffcott* et al. 1982), and 67.6% of the training days which were lost to training were due to lameness over a 2-year monitoring period (*Rossdale* et al. 1985).

Using an adjustable shoe model of lameness which allows a reversible experimental lameness to be created, it has been shown that plasma cortisol increases dramatically in response to induced lameness (*Foreman,* unpublished data, 1994). Unfortunately, the longer half-life of cortisol (approximately 6 hrs in equine plasma) renders it less helpful in measuring a decrease in lameness stress in this model since it takes too long to decrease significantly in response to analgesics which are administered after lameness is induced. Heart rate, however, correlates quite well with the severity of clinical lameness induction (*Foreman* and *Lawrence* 1991) and with a clinical response to analgesic administration (*Foreman* et al. 1994), presumably in direct correlation with changes in sympathetic nervous tone and plasma catecholamine concentrations. This model has thus been used as a method of differentiating the various efficacies of commonly used nonsteroidal anti-inflammatory drugs in horses (*Foreman* et al. 1994).

Ambient temperature and humidity

Equine responses to increased ambient heat and humidity have been addressed elsewhere in these proceedings (*Foreman* 1996). Cold has also been shown to be a significant stressor in food animals but remains poorly studied in horses.

Conclusions

Stress is a significant factor in endocrinological responses to transport, exercise, lameness, and ambient temperature changes in horses. Considerable interindividual variation and the slow half-life of cortisol renders it useful as a measure of stress in a group of horses, but less reliable as a measure of decreasing stress in a short term measurement such as response to analgesic medication. Over a longer term, such as a number of days or weeks of training, cortisol measurements may be more useful as an indicator of increasing exercise or lameness stress. Resting, exercise, and recovery heart rates may also be useful indicators of fatigue or lameness.

References

Adamson, E. J. and *Slocombe, R. F.* (1995): Flow cytometric studies of equine phagocytes following strenuous exercise. Equine vet. J. Suppl. 18, 37–42.

Anderson, N. V., DeBowes, R. M., Nyrop, K. A. and *Dayton, A. D.* (1985): Mononuclear phagocytes of transport-stressed horses with viral respiratory tract infection. Am. J. vet. Res. 46, 2272–

Austin, S. M., Foreman, J. H. and *Hungerford, L. L.* (1995): Case-control study of risk factors for development of pleuropneumonia in horses. J. Am. vet. Med. Assoc. 56:1307–1316.

Bayly, W. M., Liggit, H. D., Huston, L. J. and *Laegreid, W. W.* (1987): Stress and its effects on equine pulmonary defenses. Proc. 32nd Annual AAEP Conv., 253–262.

Crisman, M. V., Hodgson, D. R., Bayly, W. M. and *Liggit, H. D.* (1992): Effects of transport on constituents of bronchoalveolar lavage fluid from horses. Cornell vet. 82, 233–246.

Covalesky, M. E., Russoniello, C. R. and *Malinowski, K.* (1992): Effects of show-jumping performance stress on plasma cortisol and lactate concentrations and heart rate and behavior in horses. J. Equine vet. Sci. 12, 244–251.

Fazio, E., Medica, P. and *Ferlazzo, A.* (1995): Effetto dello procedure pre-trasporto e del trasporto sulle funzionalita surrenalica e tiroidea nel Cavallo. Atti. Soc. it. Sci. vet. 49, in press.

Fazio, E., Medica, P., Campo, G. M., Grasso, L. and *Ferlazzo, A.* (1996): Livelli circolanti di B-endorfina, ACTH e cortisolo in Cavalli prima e dopo trasporto di differente lunghezza. Atti. Soc. it. Sci. vet. 50, in press.

Ferlazzo, A., Panzera, M., Caola, G. and *Omero, A.* (1982): Comportamento della lattacidemia e della piruvicemia nel cavallo sanfrantellano dopo esercizio di tipo fascio e posturale. Acta. med. vet. 28, 543.

Ferlazzo, A., Panzera, M., Balbo, S. M. and *Caola, G.* (1983): Su alcuni parametri ematochimici in Equus asinus L.: Influenza dello stress da trasporto. Clin. vet. 106, 238–242.

Ferlazzo, A., Caloa, G., Omoero, E. and *Panzera, M.* (1984): Modificazioni sieroenzimatiche nel cavallo sedentario dopo trasporto. Arch. vet. it. 35, 27–31.

Ferlazzo, A., Fazio, E., Murania, C. and *Piccione, G.* (1993a): Physiological responses of stallions to transport stress. Proc. Internat. Congr. on Applied Ethology (Berlin), pp. 544–546.

Ferlazzo, A., Fazio, E., Iannwelli, N., Murania, C., Panzerra, M. and *Piccione, G.* (1993b): Cortisolemia nel Cavallo atleta in corso di competetizione. Atti. Soc. it. di Ippologia 11, 25–28.

Ferlazzo, A., Fazio, E., Medica, P., Murania, C. and *Piccione, G.* (1994): Adrenal response to physical activity in training and competing sport horses. Proc. 28th Internat. Congr. on Applied Ethology, Abstract P 1.2, p. 38.

Ferlazzo, A. (1995): Animali da reddito: Indicatori di benessere e "linee guida" per la loro applicazione nel trasporto. Atti. Gli indicatori scientifici del benessere animale," in press.

Foreman, J. H., Bayly, W. M., Grant, B. D., Gollnick, P. D. (1990): Standardized exercise test and daily heart rate responses of Thoroughbred horses undergoing conventional race training and detraining. Am. J. vet. Res. 50:914–920.

Foreman, J. H. and *Lawrence, L. M.* (1991): Lameness and heart rate elevation in the exercising horse. J. Equine vet. Sci. 11:353–356.

Foreman, J. H., Grubb, T. L. and *Inoue, O. J.* (1994): Single-dose intravenous flunixin meglumine versus ketoprofen in experimentally induced lameness in horses. Proc 40th Annual AAEP Convention. R. P. Bakhaus (ed), AAEP Publications, p. 51.

Foreman, J. H. (1996): Thermoregulation in the horse exercising under hot and humid conditions. Pferdeheilkunde, submitted, under review.

Fraser, D., Ritchie, J. S. D. and *Fraser, A. F.* (1975): The term "stress" in a veterinary context. Br. vet. J. 131:653–662.

Hines, M. T. and *Schott, H. C.* (1995): Exercise and immune function. Proceedings of the 12th Annual Veterinary Medical Forum. ACVIM, Lakewood, Colorado, pp. 727–730.

Hungerford, L. L., Foreman, J. H. and *Folz, S. D.* (1992): Risk factors for respiratory disease associated with transport in horses. Equine Infectious Diseases VI, W. Plowright, P. D. Rossdale and J. F. Wade (eds.), R&W Publications, Newmarket, England, p. 317.

Huston, L. J., Bayly, W. M., Liggit, H. D. and *Magnuson, N. S.* (1987): Alveolar macrophage function in Thoroughbreds afters strenuous exercise. In: Equine Exercise Physiology 2, J. R. Gillespie and N. E. Robinson, (eds.), ICEEP Publications, Davis, California, pp. 243–252.

Jeffcott, L. B., Rossdale, P. D., Freestone, J., Frank, C. J., Towers-Clark, P. F. (1982): An assessment of wastage in Thoroughbred racing from conception to 4 years of age. Equine vet. J. 14, 185–198.

Kurcz, E. V., Lawrence, L. M., Kelly, K. W. and *Miller, P.A.* (1988): The effect of intense exercise on the cell-mediated immune response in horses. J. Equine vet. Sci. 8:237–239.

Leadon, D. P., Daykin, J., Backhouse, W., Frank, C. and *Atock, M. A.* (1990): Environmental, hematological and blood biochemical changes in equine transit stress. Proc. 36th Conv. Am. Assoc. Equine Practnrs., pp. 485–490.

Leadon, D. P., Frank, C. and *Atock, A.* (1990): Recommendation to horse owners and their representatives on the transport of horses. Federation Equestre Internationale, Berne, Switzerland, pp. 1–18.

Linden, A., Art, T., Amory, H., Desmecht, D. and *Lekeux, P.* (1991): Effect of 5 different types of exercise, transportation and ACTH administration on plasma cortisol concentration in sport horses. In: Equine Exercise Physiology 3, S. G. B. Persson, A. Lindholm and L. B. Jeffcott (eds.), ICEEP Publications, Davis, California, pp. 391–396.

Malinowski, K. (1987): Effects of training and exercise stress on plasma cortisol and lactate in Standardbred yearlings. J. Anim. Sci. 65, 222.

Malinowski, K., Hallquist, N. A., Helyar, L., Sherman, A. R. and *Scanes, C. G.* (1990): Effect of different separation protocols between mares and foals on plasma cortisol and cell-mediated immune response. J. Equine vet. Sci. 10, 363–368.

McKenna, B., Lambert, M and *Evans, J. A.* (1993): A study of beta-endorphin and cortisol levels in the exercising horse. Proceedings from the Twelfth Meeting of the Association of Equine Sports Medicine, . Foreman (ed.), Veterinary Practice Publishing Company, Santa Barbara, California, pp. 39–43.

Petazzi, F., Zarilli, A. and *Ceci, L.* (1983): Comportamento del T3, T4 e del cortisolo in cavalli adulti sottoposti a stress da transporto. Obiettivi Doc. vet. 4, 55.

Racklyeft, D. J. and *Love, D. N.* (1990): Influence of head posture on the respiratory tract of healthy horses. Aust. vet. J. 67, 402–405.

Rossdale, P. D., Hopes, R., Wingfield-Digby, N. J. and *Offord, K.* (1985): Epidemiological study of wastage among racehorses 1982 and 1983. Vet. Record 116, 66–69.

Smith, B. R., Jones, J. H., Carlson, G. P. and *Pascoe, J. R.* (1994): Body position and direction preferences in horses during road transport. Equine vet. J. 26, 374–377.

Snow, D. H., Harris, R. C., MacDonald, I. A., Forster, C.D. and *Marlin, D. J.* (1992): Effect of high-intensity exercise on plasma catecholamines in the Thoroughbred horse. Equine vet. J. 24, 462–467.

Sonnichsen, H. V. and *Jorgenssen, K.* (1988): Untersuchungen zum Transportstress beim Pferd. Pferdeheilkunde 4:35–36.

Toutain, P. L., Lassourd, V., Popot, M. A., LaRoute, V., Alvinerie, M. and *Bonnaire, Y.* (1995): Urinary cortisol excretion in the resting and exercising horse. Equine vet. J. Suppl. 18:457–462.

Traub, J. L., McKinnon, A. O., Bruyninckx, W. J., Thrall, M. A., Jones, R. L. and *Blancquaert, A. M. B.* (1988): Effect of transportation stress on bronchoalveolar lavage fluid analysis in female horses. Am. J. vet. res. 49, 1026–1029.

Vinci, R., Calastroni, A. and *Ferlazzo, A.* (1994): Valutazione del peptide MDF, Myocardial Depressant Factor, nel plasma di cavalli trasportati. Atti Soc. it. Sci. vet. 48, 146–149.

Waran, N. K. and *Cuddeford, D.* (1995): Effects of loading and transport on the heart rate and behaviour of horses. Appl. Anim. Behav. Sci. 43, 71–81.

Wong, C. W., Smith, S. E., Thong, Y. H., Opdebeeck, J. P. and *Thornton, J. R.* (1992): Effects of exercise stress on various immune functions in horses. Am. J. vet. Res. 53, 1414–1417.

Yamauchi, T., Oikawa, M. and *Hiraga, A.* (1993): Effects of transit stress on white blood cell counts in the peripheral blood in Thoroughbred race horses. Bull. Equine Res. Inst. 30, 30–32.

Dr. Jonathan H. Foreman

1008 West Hazelwood Drive
Urbana
Illinois 61801 USA

Phone: (217) 333-2000
Fax: (217) 244-1475
e-mail: jforeman@cvm.uiuc.edu