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Temporary Suppression of Cell-Mediated Immunity in Standardbred Horses with Decreased Athletic Capacity

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Jensen-Waern M, Persson SGB, Nordengrahn A, Mérza M, Fossum C: Temporary suppression of cell-mediated immunity in Standardbred horses with decreased athletic capacity. Acta vet. scand. 1998, 39, 25-33. – Eighty Standardbred horses, originating from 5 training campuses, with decreased athletic performance in association with symptoms such as intermittent fever and mild pharyngitis were examined. As control animals, 10 horses from a stable with normally performing horses were used. Virus isolation and clinico-chemical and serological tests were performed. Lymphocyte proliferation tests were carried out to evaluate the capacity of the cell-mediated immunity. In addition, a bioassay for equine type I interferon, as a marker for early viral infections, was established. No specific microbe could be linked to these symptoms, but there was a temporary suppression of the cell-mediated immunity, which might be explained by the serological evidence of an EHV-2 and/or rhinovirus infection.

poor performance; lymphocyte proliferation; equine interferon; virus; serology.

Introduction

Loss of performance is a common problem among racing Standardbred horses. It may either be due to non-infectious factors, such as cardiovascular and energy metabolic insufficiency (*Persson* 1967, *Persson* 1968, *Eaton* 1994), musculoskeletal impairments (*Freestone & Carlson* 1991) and respiratory tract abnormalities (*Morris & Seeherman* 1990), or have an infectious aetiology. Different microbiological agents such as equid influenza, herpes and rhinoviruses, *Mycoplasma, Pasteurella,* and *Streptococcus* spp. have been associated with decreased athletic performance (*Burrows* 1970, *Allen & Bryans* 1986, *Wood et al.* 1993, 1994).

During the winter and early spring of 1994, several trainers in central Sweden reported a decrease in the performance capacity of a majority of their racing Standardbred trotters. In addition, one or several of the following symptoms were observed: intermittent fever, mild pharyngitis without nasal discharge, swollen and sore pasterns, lymphangitis, and dull skin which sometimes was covered with crusts. On the basis of these symptoms and clinical examination, including ECG, the increased incidence of poorly performing horses appeared to have an infectious origin.

In the present study, horses originating from 5 training campuses with these health problems and from one stable with normally performing healthy racehorses were examined. Virus isola-

tion and clinico-chemical and serological tests were performed. Since viral infections may impair the immune response in animals (*Larsson* 1988, *Carter et al.* 1989, *Hannant et al.* 1991) as well as in humans (*Bloomfield & Mateer* 1919, *Mills* 1984, *Wainberg et al.* 1985), lymphocyte proliferation tests were carried out to evaluate the capacity of the cell-mediated immunity. In addition, a bioassay for equine type I interferons (IFN), as a marker for early viral infections, was established.

Materials and methods

Horses

Eighty Standardbred trotters, aged 2-7 years, actively involved in training or racing, from 5 different training campuses (A, B, C, D, E) were examined between February and April 1994. A majority of the horses at campuses A and B showed "loss of performance" for about 2 months, and in the other 3 training stables (C, D, E) for at least 3 months, prior to the first sampling occasion. As control animals, 10 normally performing Standardbred horses from another campus (F) were used.

In all horses, a general physical examination including ECG was undertaken. Blood samples were collected from the jugular vein for total and differential white blood cell counts, virus isolation, lymphocyte stimulation tests and IFN analyses. In 40 horses, originating from stables A (n = 17) and B (n = 23), total and differential white blood cell counts and lymphocyte stimulation tests were performed a second time, 5 weeks later.

On each sampling occasion, haematological and lymphocyte stimulation tests were carried out on control horses concomitantly with the patients.

Paired serological tests were performed in horses from stables A and B and controls on 2 occasions 5 weeks apart.

Haematology, serology and virus isolation

The total white blood cell count was performed with a microcell counter (Sysmex F-800, Toa Medical Electronics Co, Kobe, Japan) and conventional differential counts were carried out by standard procedures at the Department of Clinical Chemistry of the Swedish University of Agricultural Sciences.

Sera were tested for the presence of antibodies to Equine Herpes virus (EHV) types 1 and 2, equine arteritis virus (EAV) and rhinovirus types I and II by routine procedures at the Department of Virology, National Veterinary Institute, Uppsala, Sweden. Antibody titres were determined as the highest serum dilution that was reactive in the test used, i.e. virus neutralization tests (rhinovirus types I and II), and complement fixation test (EHV-1) or ELISA (EHV-2 and EAV). The assay of antibodies to EHV-2, was run essentially as described by *Voller* (1978), while antibodies to EAV were assayed according to the instructions of the manufacturer (SVANOVA Biotech, Uppsala, Sweden).

Attempts to isolate virus from nasopharyngeal swabs and blood were made by co-cultivation of the samples with equine endothelial lung cells. The cells were monitored for any cytopathic effect during 5 passages. Virus isolates were identified by using reference sera in virus neutralization tests.

Lymphocyte proliferation test

The proliferative response of lymphocytes to mitogen stimulation was determined in affected horses on 2 occasions, 5 weeks apart (15 horses, first test in the middle of Feb., stable A; 23 horses, first test in the beginning of Mar., stable B) and on one occasion (39 horses, stables C, D, E). On all test occasions lymphocytes from the control horses (stable F) were examined simultaneously.

The lymphocyte proliferation tests were carried out in whole blood cultures, i.e. heparinized

blood diluted 1:10 in growth medium, consisting of RPMI 1640 medium (Flow Laboratories, Irvine, Scotland, UK) with 20 mM HEPES buffer, L-glutamine (2 mM), penicillin (200 IU/ml), streptomycin (100 μg/ml) and 5% FCS (Myoclone; Gibco, Scotland, UK). The magnitude of proliferation (mean cpm value for triplicate cultures) was determined after stimulation with the following mitogens; concanavalin A (Con A; Pharmacia, Uppsala, Sweden); phytohaemagglutinin (PHA; Wellcome, Dartford, England, UK); or pokeweed mitogen (PWM; Boehringer-Mannheim, Mannheim, Germany), at concentrations of 20, 10 and 5 μg per ml, respectively. In addition, the spontaneous proliferation was determined in triplicate cultures without mitogen. After 4 days of culture at 37 °C in a humid atmosphere with 7% CO_2 in air, all cultures were pulsed for 24 h with 25 μl growth medium containing 0.5 μCi ^{3}H-thymidine (specific activity 185 GBq mol^{-1}; Amersham International, Amersham) per well and the radioactivity in each culture was determined in a liquid scintillation counter (betaplate counter; LKB Wallac, Turku, Finland).

Interferon type I bioassay

A cytopathic effect inhibition assay, mainly carried out as described for detection of porcine IFN-α (*Artursson et al.* 1989) was adapted for detection of antiviral activity in equine serum and nasal secretion. For that purpose, a laboratory standard of equine IFN was prepared by infecting equine peripheral blood mononuclear cells with Aujeszky's Disease Virus (ADV, strain Phylaxia) for 24 h. The cells were removed by low speed centrifugation, the supernatant was treated with HCl (pH 2 for 24 hours at +4 °C), and pH was restored to 7 by addition of 1M NaOH. Based on the IFN inducer used, the type of IFN-producing cell, and the acid stability, the antiviral activity of this laboratory standard is, in accordance with *Yilma et al.* (1982), referred to as equine IFN-α (EqIFN-α). No difference in the ability of EqIFN-α to protect various cell lines from challenge with vesicular stomatitis virus (VSV) was found when tested on porcine (PK-15), bovine (MDBK) and equine (primary testis, HP2915) cells. The bioassay for EqIFN-α was therefore carried out by incubating confluent monolayers of MDBK cells with serial dilutions of the samples in 96 well microtitre plates overnight at 37 °C. The samples were replaced by medium, containing VSV in a sufficient amount to cause complete destruction of the cells in unprotected wells in 24 h. Antiviral activity in samples was compared with that of the laboratory EqIFN-α standard after staining of residual MDBK cells with crystal violet. The EqIFN-α standard was obtained from parallel runs with a human laboratory standard of IFN-α (calibrated against the National Institute of Health reference leucocyte IFN-α GA-23-902-530), which contained 250 U per ml.

Statistical analysis

The results from the lymphocyte proliferation tests were compared by the Mann Whitney U test and are expressed as mean counts per min ± SEM.

Results

Physical examinations

Stable A. A majority of the horses in stable A showed poor athletic performance in the middle of February. In addition, intermittent fever, mild pharyngitis, dull skin with crusts and sore pasterns were common symptoms. ECG revealed no signs of cardiopathy.

Stable B. The horses in stable B, which were examined in the beginning of March, displayed similar symptoms to those in stable A. No abnormalities were found on ECG.

Stables C, D and E. Besides the decreased performance capacity, the horses in stables C

Table 1. Incidence of antibodies against EHV-1, EHV-2, rhinovirus types 1 and 2 and EAV in sera from horses in stables A and B and control animals. Sera were considered positive when the titres were >4, ≥8,000, ≥559, ≥90 and >10, respectively. Paired samples (I, II) were taken at a 5-week interval.

	EHV-1			EHV-2			Rhino 1			Rhino 2			EAV	
	No. of sera examined	No. of positive sera		No. of positive sera		% with increased antibodies	No. of positive sera		% with increased antibodies	No. of positive sera		% with increased antibodies	No. of positive sera	
		I	II	I	II		I	II		I	II		I	II
Stable A	17	0	0	11	15	65%	11	14	59%	3	9	82%	0	ND
Stable B	23	0	0	23	23	17%	19	20	22%	2	3	57%	ND	ND
Controls	10	0	0	8	8	10%	4	6	30%	5	5	10%	ND	ND

"% with increased antibodies" = percentage number of sample with increase in antibody level between sampling occasions I and II respective of whether the sera at test I had positive or negative titres.
ND: not determined.

(n = 21), D (n = 6) and E (n = 13) showed intermittent fever and mild pharyngitis, but the clinical signs in the skin and pasterns were minimal. These animals were examined physically in the middle of and at the end of March.

Haematology, serology, and virus isolation
In all but 3 horses, the total and differential white blood cell counts were within the normal range ($5.1\text{-}10.3 \times 10^9$/L). In the 3 exceptions there was an increase in the polymorphonuclear cells, associated with a bacterial infection in the lower respiratory tract.
The paired serological data (Table 1) show that the number of horses with antibodies to EHV-2 increased in stable A between the first and second test occasions. Sera obtained from 17 horses were examined; of these, 11 were positive on the first test occasion and 15 on the second test occasion. All 23 tested horses in stable B and 8 out of 10 tested controls showed positive titres against EHV-2 on both occasions.
In the horses of stable A, the number of sera positive for antibodies to rhinovirus types I and II increased from 11 to 14 and from 3 to 9, respectively. In the stable B and control horses only minor increases in the number of samples positive against rhinovirus types I and II were noted.
In general, the proportions of samples showing an increase in the level of antibodies to EHV-2 and rhinovirus types I and II between the 2 sampling occasions, irrespective of whether the titres were above or below the limit of a positive reaction, were higher in stables A and B than in the control horses. No positive sera against EHV-1 and EAV were found.
In 2 cases EHV-2 was isolated from the white blood cells, but no other virus was recovered in either the nasopharyngeal swabs or in the blood samples.

Lymphocyte proliferation test
On the first test occasion, the response to stimulation with PWM had decreased ($p<0.01$) in cultures of whole blood obtained from the horses in stables A and B, compared with the control horses (Fig. 1). Similarly, both the Con A- (stable A; $p<0.05$) and PHA-induced (stable B; $p<0.001$), and the spontaneous proliferation (stables A and B; $p<0.05$) were poorer in the patients. On the second test occasion, 5 weeks

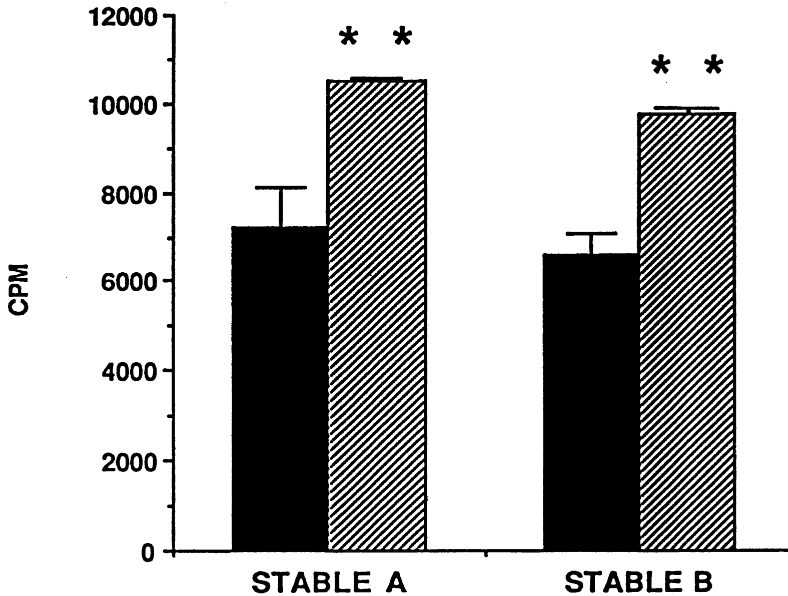

Figure 1. Proliferative responses of lymphocytes, obtained on the first sampling occasion, from horses in stable A ■ (n = 15), stable B ■ (n = 23) and controls ▨ (n = 10) after stimulation with pokeweed mitogen. Values are given as cpm ± SEM.
** $p<0.01$ indicates a significant difference between the horses in stables A and B and the respective controls.

later, no such difference in proliferative capacity was observed either between the horses in stable A or between those in stable B and the control animals (data not shown).
The proliferative capacity of lymphocytes from horses in stables C, D and E was only tested once. The overall response to the stimulation by different mitogens can be summarized as follows:
Stable C: The response of lymphocytes from 16 horses was of the same order of magnitude and from 4 horses below that of cells from control animals.
Stable D: The cells from 4 horses responded similarly to those of the controls, but a poorer response was noted in lymphocytes from 2 horses.
Stable E: The proliferative response was of similar magnitude in 8 horses, and below that of the controls in 5 horses.

Interferon type I (IFN) bioassay
The applicability of the bioassay for EqIFN-α was tested in a pilot study, including 17 racing Standardbred horses with an ongoing influenza virus infection, and a Shetland pony from a separate stable. Body temperature was measured for 3 consecutive days and on the third day the levels of EqIFN-α was determined in samples of nasal secretion and serum (Table 2). At this time, 11 samples from nasal swabs were analyzed for A2 influenza virus by immunofluorescence according to standard procedures at the Department of Virology, National Veterinary Institute, Uppsala, Sweden. In general, the IFN response was higher in nasal secretion than in the serum. In 13 out of 17 horses, an IFN response which coincided with elevated body temperature was detected in the nasal secretion and/or serum. The infection was confirmed by

demonstration of viral antigens, using immunofluorescence (IF), in samples from nasal swabs collected from 9 of these horses. In 2 horses, which were also negative in IF, no antiviral activity was detected in the serum or nasal secretion.

In total, 166 serum samples from horses in stables A-F were analysed for IFN-α. During the course of the study only 6 samples were positive (6-24 U IFN-α/ml) and 2 of these originated from horses in stable F, that is, the campus with normally performing horses.

Discussion

The clinical examinations of the horses with a decreased athletic capacity, and also the high incidence of poor performers in a stable, pointed towards an infectious origin of the disorder. The horses recovered from their episode of decreased athletic performance and were all racing with their normal performance capacity within 6 to 9 months after the first signs of illness.

Both bacterial (*Wood et al.* 1993, 1994) and viral (*Burrows* 1970) infections have been found to be associated with loss of performance in racehorses. In the present study, a purulent infection in the lower respiratory tract was observed in only 3 out of 80 horses. This infection was reflected in the total and differential white blood cell counts as an increased number of neutrophils. The proliferative capacity of lymphocytes obtained from these horses was however of the same magnitude as that of control horses, and the paired serological samples were all considered as positive against EHV-2 and rhinovirus types I and II. In all the other horses sampled, no haematological aberrations from the normal range were noted. No other clinical signs of bacterial infection, such as nasal discharge and swollen lymph nodes of the respiratory tract, were observed. An attempt to isolate virus from the blood and nasal swabs was therefore made and serum samples were analysed for IFN-α by a bioassay.

The presence of IFN-α in the serum has been used in several species, including man, as a marker for ongoing viral infections (*Gresser* 1984). In pigs, serum IFN-α has been very useful for monitoring the incidence of viral infections on a herd basis (*Artursson et al.* 1989, *Wallgren et al.* 1993). However, the IFN-α response is of short duration, lasting approximately one week, and local replication of virus might not induce detectable levels of IFN-α in serum. For instance, IFN was detected in nasal secretion 2 to 10 days after intranasal inoculation of SPF foals with EHV-1 (*Chong & Duffus* 1992). The bioassay used in the present study was sufficiently sensitive to detect IFN-α in nasal secretion and serum obtained from horses with an ongoing influenza virus infection (Table 2). However, IFN was not detected in serum from the poorly performing horses, neither was virus isolated from the blood or nasal swabs. Thus, it was concluded that no acute viral infection was present at the time of the clinical examinations.

However, since the horses had shown a decreased athletic capacity for 2 to 3 months before the attempts to isolate virus or detect IFN-α were made, antibody titres to EHV-1, EHV-2, EAV, and rhinovirus types 1 and 2 were determined in paired serum samples as indicators of viral infections in retrospect. As shown in Table 1, several of the affected horses acquired serological evidence of infection with EHV-2 and rhinovirus types I and II during the course of the study. Equid Herpesvirus type 2 is widespread in the horse population, and persistent infections in which virus can be recovered intermittently from the nasopharynx and white blood cells are well documented. However, whether this virus is associated with clinical disease or not is still unclear (*Palfi et al.* 1978).

Table 2. Body temperature, virus detection* and levels of IFN-α* in serum and nasal secretion collected from horses during an acute infection with equine influenza virus.

Horse No.	Temperature °C			Virus (A2) Nasal swab (IF)	IFN-α Serum	IFN-α (U/ml) Nasal secretion
	28 Aug.	29 Aug.	30 Aug.			
1	38.8	37.9	38.2	+	(+)	125
2	37.4	38.8	38.7	+	(+)	27
3	37.6	38.0	37.8	not tested	(+)	negative
4	39.7	38.2	38.2	not tested	+	18
5	40.5	40.6	39.1	not tested	+	25
6	39.1	38.2	37.9	not tested	+	36
7	37.4	38.8	38.1	not tested	(+)	10
8	37.4	38.2	38.5	not tested	+	1,400
9	37.4	39.1	38.7	+	(+)	188
10	41.2	39.2	39.2	+	(+)	18
11	37.7	37.8	37.8	+	–	not tested
12	37.6	37.9	37.9	+	–	3
13	37.4	38.0	37.8	–	–	negative
14	37.7	37.9	38.1	+	+	4
15	37.7	37.4	37.6	+	–	50
16	37.1	38.4	37.2	not tested	(+)	5
17	37.3	37.4	37.6	+	–	not tested
18			"no fever"	–	–	negative

* Determined in samples collected August 30, 1994.

Infection with rhinovirus can either lead to an overt clinical disease (*Plummer & Kerry* 1962) or, quite often, be subclinical (*Wilson et al.* 1965, *Burrows* 1970). According to *Burrows* (1970), rhinovirus type 1 is the most common subtype, a finding in conformity with the present results. Also, the present incidence of horses with neutralizing antibodies to rhinovirus type 1 is in accordance with that found in adult British Thoroughbreds (*Plummer & Kerry* 1962, *Burrows* 1970).

Herpesvirus infections have been associated with immunosuppression in many species, including man (for review see *Wittmann et al.* 1984). In horses, secondary bacterial infections are common after EHV-1 infections of the respiratory tract (*Bryans* 1980). Equid Herpesvirus type 1 has been shown to impair the ability of lymphocytes to respond to PHA stimulation (*Wilks* 1976, *Thomson & Mumford* 1977), and in ponies experimentally infected with EHV-1, a significantly reduced response to PHA stimulation for at least 40 days after infection was observed. The lymphocyte function was approaching normal levels on about day 90 post-infection (*Hannant et al.* 1991). In the present study, a similar temporarily reduced response of the lymphocytes obtained from poorly performing horses in stables A and B was observed. On the second test occasion, 5 weeks later, the lymphocyte function was of the same magnitude as in normally performing control horses. At that time the horses had shown failing performance for about 3 months. However, no serological indication of infection with EHV-1 was noted in any of the horses tested (Table 1). On the other hand, the levels of antibodies to EHV-2 increased in 65% of the

sera collected from horses in stable A and in 17% of those from horses in stable B compared with 10% among the controls, during this period of time. Thus, it may be possible that the observed suppression of the cell-mediated immunity was associated with an EHV-2 infection. An immuno-compromising effect of EHV-2 has previously been demonstrated in a study on foals from a farm with endemically occurring *Rhodococcus equi* infections (*Morein & Merza* 1991). In that study it was clearly shown that the incidence of pneumonia decreased dramatically after vaccination with an iscom vaccine against EHV-2. However, no subsequent infection has so far been observed in our study.

In summary, in these racing Standardbreds poor performance was associated with symptoms such as intermittent fever and mild pharyngitis. No specific microbe could be linked to these symptoms, but there was a temporary suppression of the cell-mediated immunity, which might be explained by the serological evidence of an EHV-2 and/or rhinovirus infection.

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Sammanfattning

Temporär suppression av det cellmedierade immunförsvaret hos varmblodiga travare.

Åttio varmblodiga travare med nedsatt prestationsförmåga från 5 olika träningsstallar undersöktes. Förutom försämrad prestation uppvisade flertalet av hästarna intermittent feber och lindrig faryngit. Många var även matta och krustösa i hårremmen.

Kontrollgruppen bestod av 10 normalpresterande tävlingshästar från ett annat träningsstall.

Virusisolering, serologi, hematologi samt lymfocytproliferationstester, som speglar det cellmedierade immunsystemet, utfördes. Som en markör för en tidig viral infektion etablerades en bioassay för ekvint interferon typ 1.

Ingen specifik mikrob kunde knytas till den nedsatta prestationsförmågan, däremot kunde en temporär suppression av det cellmedierade immunsystemet beläggas. Möjligen kan denna immunosuppression associeras till de serologiska indikationerna på EHV-2 och/eller rhinovirus infektion. Samtliga hästar med nedsatt kapacitet tävlade och tränade på sin förväntade förmåga efter en konvalescens på 6 till 10 månader.

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