TABLE 1

Values of total and lipoprotein cholesterolin normal subjects with different age (Mean ± S.D)

Group	Total cholesterol (T-c)	HDL-c	LDL-c	VLDL-c	T-c / HDL-c	Blood pressure Systolic Diastolic
I(87)	201·7 ± 21·2	65·3 ± 10·2	92·3 ± 36·7	42·4 ± 14·2	3·2 ± 0·9	118 ± 8/82 ± 6
II(93)	231·5 ± 30·2[a]	59·1 ± 14·2[b]	123·2 ± 32·2[c]	60·7 ± 15·4[c]	3·94 ± 0·7[b]	125 ± 11/85 ± 8
III(95)	235·3 ± 36·7[b]	55·2 ± 13·2[c]	124·3 ± 48·7[c]	62·7 ± 24·8[c]	4·1 ± 0·7[c]	127 ± 10/89 ± 9
IV(107)	239·4 ± 40·2[b]	56·2 ± 14·2[c]	122·7 ± 39·7[c]	59·7 ± 19·7[c]	3·9 ± 0·7[b]	122 ± 10/89 ± 7

HDL-c High density lipoprotein cholesterol
LDL-c Low density lipoprotein cholesterol
VLDL-c Very low density lipoprotin cholesterol

a, $P<0·05$; *b*, 0·01; *c*, 0·001

aged 20–29 years, 30–39 years, 40–49 years and 50 years and above respectively. Fasting blood samples from healthy individuals were collected into EDTA vials (1 mg/ml of blood). Plasma was separated and processed for lipoprotein fractionation using heparin, manganese chloride and sodium dodecyl sulphate double precipitation techniques[6,7]. The cholesterol content in different fractions of lipoproteins was estimated[8].

In the present study, significant increase in the mean total cholesterol, low density lipoprotein cholesterol (LDL-c) and very low density lipoprotein cholesterol (VLDL-c) observed in the age group above 30 years (table 1). This may be because of decreased endogenous metabolism with the advancement of age. Significant increase in high density lipoprotein cholesterol (HDL-c) and decrease in LDL-c observed in Indian healthy individual as compared to age and sex matched healthy individuals of western countries[4,10] may be because of high intake of fat by western people. VLDL-c in Indian subjects was significantly higher as compared to those of the western countries in same age and sex matched groups.[11] This may be explained on the basis of the increase in carbohydrate intake with less of fat by Indians as compared with their counterpart in the west, resulting in an increased synthesis of VLDL.

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DISTRIBUTION OF COMPLEMENT FIXING ANTIBODY TO EQUINE RHINOPNEUMONITIS VIRUS IN FOALS AND STALLIONS

A. M. JANA, G. PANDYA AND K. M. RAO
Department of Microbiology,
Defence Research and Development Establishment,
Gwalior 474 002, India

RESPIRATORY disease in young horses and subclinical infection in adult horses owing to equine rhinopneumonitis virus (ERV) have been recognized in many parts of the world[1–3]. Reports on ERV infection caused by equine herpes virus type 1 (EHV1) in India are generally restricted to abortion syndrome in mares[4,5]. Preliminary serological survey of ERV infection among army horses and mules in India

reveals its presence in the country[6]. In the present study we report the relative distribution of complement fixing (CF) antibody to ERV among different categories of colt, filly and stallion.

Serum samples from 78 foals (38 colts and 40 fillies aged 2 to 6 months and 32 stallions (10 horse stallions and 22 donkey stallions) were collected from an equine breeding stud in August 1980. Serum samples preserved with merthiolate were stored at − 20° until used. Sera were screened by microtiter system of complement fixation test (CFT)[7,8], using hamster adapted Kentucky D strain of EHV1 antigen. Positive and negative sera were also simultaneously used in the test. Two units each of EHV1 antigen, guinea pig complement and haemolysin were employed in the test proper using veronal buffer as diluent. More units of complement were utilized for determining anti-complementary activity of the serum. A CF titre of 1 in 16 and above was considered as significant, 1 in 8 as doubtful and below 1 in 8 as insignificant. Low levels of CF titres (1/4-1/8) as evidence of recent EHV1 infection have been reported to be doubtful because of antigenic cross reactions with other herpes viruses[8,9] and the composition of EHV1 CF-antigen[7].

Thirty eight colts were further grouped into 26 horse young stock (H-YS) and 12 mule young stock (M-YS). Similarly 40 fillies were grouped into 21 H-YS and 19 M-YS. The pattern of CF antibody distribution in these categories of foals is represented in table 1. The titres were significant in only 2 M-YS fillies (10·53%) and one H-YS filly (4·76%) gave doubtful reaction. Out of 12 H–YS colts only one (8·33%) gave doubtful reaction. The other colts and fillies in both categories did not reveal significant CF antibody titres. Among M–YS, out of 19 fillies 14 (73·68%) and out of 12 colts 7 (58·33%), had 1:4 CF titre. Comparatively in H-YS only 7 fillies out of 21 (33·33%) and 8 colts out of 26 (30·77%) showed the same titre. Thus the CF reaction was more pronounced in M-YS colts and fillies than in those of the H-YS group.

Results on age-wise CF-antibody distribution among horse and donkey stallions have been depicted in table 2. Sera of all the 10 horse stallions aged 4 to 11, 12 years and above showed insignificant CF-reactions. On the contrary out of 16 donkey stallions of 4 to 11 age group, 13 (81·25%) were detected with significant titres. However, among 6 donkey stallions of 12 years of age and above, only one (16·67%) was found to be significant and 3 (50%) had doubtful titre. Anti complementary (AC) activity was also noted in donkey sera.

A common feature noted among 2 to 6 month old colts and fillies was the lack of significant EHV1 antibodies. The only exceptions were two, 3 month old M-YS fillies which showed significant titres. The results are comparable to the findings of De Boer[1] and Thomson *et al.*[3], who however, used serum neutralization test to detect antibody titres in foal sera. Our findings show detectable antibody levels even though they were insignificant among H-YS and M-YS foals. A fairly large number of M-YS sera exhibited AC activity, a feature demonstrated by their adult counterparts[6].

The significance of absence of CF antibodies in foals of less than six months of age has been explained by several workers[10,11]. Foals inherit passive immunity from colostrum of their dams and in sequel remain free from respiratory syndrome induced by EHV1. The passive immunity derived from their dams may persist in foals for nearly 4 months which eventually declines afterwards. However, despite their

TABLE 1

Distribution of CF antibody to ERV in different categories of foals (2 to 6 months old)

Category of foals	No. of sera tested	Reciprocal of serum dilutions (CF titre)			
		2 and <2	4	8	16 and above
		Insignificant		Doubtful	Significant
Colt H-YS	26	18 (69·23)	8 (30·77)	—	—
Filly H-YS	21	13 (61·90)	7 (33·33)	1 (04·76)	—
Colt M-YS	12	4 (33·33)	7 (58·33)	1 (08·33)	—
Filly M-YS	19	3 (15·79)	14 (73·68)	—	2 (10·53)
TOTAL	78	38	36	2	2

H-YS Horse young stock; M-YS Mule young stock; Figures in parentheses indicate percentages

TABLE 2
Distribution of CF antibody to ERV in different categories of stallions

Category of animals	Age in years	No. of sera tested	Reciprocal of serum dilutions (CF titre) 16 and above Significant	8 Doubtful	8 Insignificant
Horse stallion	4–11	7	—	—	7 (100)
Horse stallion	12 and above	3	—	—	3 (100)
Donkey stallion	4–11	16	13 (81·25)	—	3 (18·75)
Donkey stallion	12 and above	6	1 (16·67)	3 (50·0)	2 (33·33)
TOTAL		32	14	3	15

Figures in parentheses indicate percentages

susceptibility to EHV1 infection foals do not manifest clinically symptoms of the disease owing to active immunity which generates thereafter[12].

Our study on the incidence of CF antibody in horse and donkey stallions yielded contrasting results. While none of the horse stallions showed significant or doubtful titres, 17 (77·27%) of 22 donkey stallions (both age groups included) had significant and doubtful titres. Preliminary study[6] on army horses and mules indicated that only 0·60% horses had 1 in 16 CF titre. The present study shows that donkey stallions between 4 and 11 years of age reveal high incidence of CF antibody to EHV1 which substantially recedes after this age. Matumoto[13] found 100% positive sera in 1–12 year old Egyptian donkeys. Shimizu *et al*[14] reported that CF antibody titres fluctuate up to the age of 11 years in male horses and retard after this age.

The authors thank the Commandant and staff of Equine Breeding stud for invaluable help and cooperation, and to Dr. T. Shimizu (National Institute of Animal Health, Tokyo, Japan) for the supply of CF antigen (Ky D strain EHV1) and positive and negative sera. Keen interest evinced in the work by Dr. P. K. Ramachandran, Director, is thankfully acknowledged.

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THE NERVOUS SYSTEM AND ESTERASE DISTRIBUTION IN *SCHISTOSOMA SPINDALIS* (TREMATODA)

S. LAKSHMAN RAO, G. VENKATA RAMA KRISHNA AND S. S. SIMHA
Department of Zoology, Osmania University, Hyderabad 500 007, India

THE role of esterases in synaptic or neuromuscular transmission of nerve impulse is well-known in vertebrates and invertebrates[1]. The role, distribution and functions of these enzymes in trematodes has also been studied[2–6]. Though much work has been done on esterases in different groups of trematodes, the blood flukes have received little attention. The only available literature in this field on blood flukes is on human Schistosomes[7,8]. Unfortunately no data are available on the esterase distribution in cattle blood flukes and Indian blood flukes in particular. The present study