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SCRENNING THOROUGHBREDS FOR ANTIBODIES TO THE EQUINE INFECTIOUS ANEMIA VIRUS

Z Asgarali¹, M Campbell¹, D Coombs¹, E Caesar¹, F Mohammed¹

ABSTRACT

One hundred and seventy-two Thoroughbreds were screened for the presence of antibodies to the capsid protein, p26 of the equine infectious anemia (EIA) virus using the agarose gel immunodiffusion (AGID) Coggins test. Horses ranged in age from 1 month to 21 years old and were either imported or locally bred. The majority were involved in racing and breeding and were housed either at the Santa Rosa Racing Complex at Arima or at privately owned farms. Complete blood counts (CBCs) were performed on all horses. Low haemoglobin concentrations were found in 18 (10.5 %), high white blood cell counts in 17 (9.9 %) with neutrophilia in 13 (7.6 %). Low red blood cell counts were present in 28 (16.3 %) and high platelet counts were seen in 11 of 154 horses (7.1 %). At least 12 horses had evidence of clinical babesiosis, but only 7 were confirmed infected by examination of Giemsa stained blood smears. Racehorses from Trinidad and Tobago occasionally move inter-island for racing and increasingly come in contact with foreign horses with the increasing importation of horses from countries known to harbour the virus. All 172 horses tested negative for antibodies to EIA virus. This implies that the strict adherence to import and quarantine regulations may have contributed to keeping the country free from the EIA virus. This ongoing study is the first to provide sero-prevalence data and document the prevalence of EIA in the equine population in Trinidad and Tobago.

Key Words: Equine infectious anemia; Coggins’s test; Trinidad and Tobago

INTRODUCTION

Equine infectious anaemia (EIA) is one of the most feared diseases in the equine industry since many cases show little or no clinical signs. There is no known cure or treatment for the affected horses and a vaccine is not yet available (1). Classified as a notifiable disease and placed on List B by the Office International Des Epizootics (OIE) (2), control of its spread remains a high priority. The EIA virus which belongs to the family Retroviridae and the subfamily, Lentivirinae, can infect all Equidae. While older strains were known to be highly virulent and can kill, many newer field strains induce few or no overt clinical signs of the disease (3).

EIA has a worldwide distribution. In the Americas it has been reported in the United States for more than 75 years where presently “hot spots” are well recognized. The disease is also enzootic in Canada, Argentina, and Guyana (4, 5). The prevalence varies with location, horse population density, vector abundance, and climate. (3).

There are three clinical forms of the disease, acute, chronic and asymptomatic. The incubation period is usually one to three weeks but can take as long as three months. The acute form is characterized by pyrexia followed by a syndrome of recrudescing fever, weight loss and ventral oedema (1). The disease can also cause abortion, infertility, colic and ataxia. Despite rapid replication of the virus, most animals progress to the chronic stage ending with the asymptomatic form of infection. At this stage they are considered inapparent carriers and remain infected for life (3). Inapparent infections may become symptomatic during concurrent illnesses, severe stress or hard work (3).

A consistent immunopathological feature includes thrombocytopenia, which is often present in the acutely infected horses. Leukopenia, anaemia, leukocytosis and monocytosis may be also seen in horses undergoing multiple febrile episodes (1). The virus possesses two main surface glycoproteins, gp90, gp45 and four major non-glycosylated internal core proteins, p9, p11, p15 and p26. A major characteristic of the EIA virus is rapid mutation of the variants gp90 and gp45 (6). The capsid protein p26 is the major core group specific antigen and is conserved among all viral strains. Therefore, antibodies produced to p26 allow for the universal serologic detection of the virus (7).
ARTICLES

BRAIN STEM LOCALIZATION OF NEURONS OF THE SUBDIAPHRAGMATIC VAGUS NERVE FIBRES IN THE FERRET (MUSTELA PUTORIUS FURO): A WGA-HRP NEUROHISTOCHEMICAL STUDY.

A Odekunle1; A J Bower 2

ABSTRACT

The brain stem localization of neurons of nerve fibres in the ventral and dorsal abdominal vagal trunks were studied in the ferret. A total of 14 adults ferrets (Six experimental and eight controls) were used for the study. Following anesthesia with pentobarbitone sodium, an upper midline laparotomy was done to expose the abdominal trunks of the vagus nerve. After dissecting the trunks clear from the abdominal oesophagus and the cardia of the stomach the nerve trunks were cut and WGA-HRP was applied to the proximal stump of the cut trunks. Control ferrets were divided into four groups of two ferrets. In the first group normal saline instead of the tracer was applied to the proximal stump of the vagal trunks. The second group was treated in a similar manner as the experimental animal except that the application of tracer was preceded by bilateral cervical vagotomy. In the third group of controls 0.1ml of WGA-HRP was injected into the abdominal cavity and the fourth group had tracer injection into the hepatic portal vein. All animals were allowed a survival period of 48-72 hours after tracer injection following which each animal was perfused with normal saline, fixative and buffered sucrose. The brain stem was extracted and cut in transverse section (40µm thick) with the freezing microtome. Sections were then processed for WGA-HRP neurohistochemistry and subsequently viewed and analyzed under light/dark-field illuminations. In the experimental ferrets labeled cells were seen bilaterally in the dorsal motor nucleus of the vagus nerve (DMNV), the nucleus dorsomedialis (nDm), the nucleus ambiguous (nA) and the nucleus retroambiguus (nRA). The DMNV was the most intensely labeled nucleus. Sporadic distribution of labeled cells was also observed in the reticular formation (rf) between the nA and the DMNV. Labeled neurons were not seen in any of the control experiments.

Key words: Abdominal vagus; vagal nuclei; WGA-HRP; ferret.

INTRODUCTION

Over the years, the sources of vagal preganglionic parasympathetic fibres to various abdominal viscera/structures have been investigated in various species using different nerve tracing techniques (1-11). To date, the brainstem origins of vagal fibres to the stomach, pylorus, small intestine, colon, liver, spleen and the pancreas have been investigated and documented in the ferret (12-19). The central origin of preganglionic fibres innervating several abdominal organs still remained uninvestigated in the ferret.

While several vagal brain stem nuclei have been shown to project to the abdominal viscera already studied, it is not known if there are other brain stem nuclei associated with the vagus nerve which have not been identified or labeled by earlier investigations in this species. Since all the abdominal branches of the vagus nerve are derived from the ventral and dorsal abdominal vagal trunks, it is logical to assume that tracing the origins of the vagal fibres in these trunks would reveal all the brain stem nuclei associated with the abdominal portion of the vagus nerve in this species.

The present report is the result of our investigations of the sources of fibres in the ventral and dorsal abdominal trunks of the vagus nerve in the ferret.

MATERIALS AND METHODS

Fourteen male and female adult ferrets weighing between 0.8 and 1.5 kg were used for the study. All the animals were kept in a well-ventilated and illuminated facility in the animal house and fed with ferret cubes.