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WITH COMMERCIAL DRY RABBITS PELLETS**

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AN ASSAY OF EXPERIMENTAL MUCOID ENTEROPATHY WITH COMMERCIAL DRY RABBITS PELLETS

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ABSTRACT

This study describes an assay of experimental mucoid enteropathy with commercial dry rabbit pellets. The disease has not been reproduced in our facilities although animals and dry pellets had been introduced from farms where mucoid enteropathy had been clinically present during the previous months. It must be noted that the environmental conditions of the facilities differ significantly from those of commercial farms due to the strict hygiene practices applied through all the fattening period. Results obtained from this study could support the hypothesis that the environment and its microbism plays an important role in the appearance and /or intensity of the mucoid enteropathy (once appeared) in commercial farms.

INTRODUCTION

Mucoid enteropathy is a serious disease of rabbits. In Galicia (NW Spain), the first clinical cases were observed during the last months of 1996 and the beginning of 1997. During this period the disease showed an acute course affecting 45-50 day old rabbits. The animals loose their appetite and experienced swelling and mucosal diarrhoea before dying. Preliminary results of an epidemiological study show that cases appear to be randomly distributed among different departments and cages within the farms. Heavy animals were more frequently and severely affected. The general characteristics of the course and presentation of the outbreak coincide to the description of the French outbreaks of Epizootic Rabbit Enterocolitis. During the last months of 1997 the course of the disease appeared to be less acute and occasionally it was indistinguishable from other enteric diseases of rabbits.

Although recognised for many years in many countries, the cause the disease still remains unknown. Different causal hypothesis have been proposed, including nutritional, toxic and infectious agents, but any has been definitively proved (2, 3 and 4). The introduction of the disease in the farms has often been associated with the entry of new commercial dry pellet. However, many researches conducted in order to support the nutritional hypothesis have been unsuccessful (6). It has also been suggested that commercial dry pellets could act as a vehicle for the introduction of a hypothetical causative agent of the disease (1).

The main objective of the present study was to observe if the disease was induced or not in rabbits housed and fattened in facilities where strict environmental hygiene practices were applied, after the introduction of both, rabbits and dry pellets, from commercial farms experiencing the process.

MATERIAL AND METHODS:

A total of 142 rabbits were fattened in two assays carried out in the School of Veterinary Medicine of Lugo (Spain). Rabbits were born in farms with clinical problems of mucoid enteropathy located in Galicia (NW Spain) and arrived in the school immediately after weaning. After arrival, healthy rabbits were randomly assigned to different groups. Each group were fed *ad libitum* with different commercially available pellets. Management and environmental conditions were constant for all the animals that were kept into the same room until slaughtering.

The complete study consisted in two different assays carried out, the first one during May and the second during June 1998. 72 rabbits were fattened in the first assay and 70 in the second. A total of 142 rabbits of 31-32 days of age, coming from a farm with clinical problems compatible with mucoid enteropathy arrived in the Veterinary School. After arrival, the animals were assigned to different groups of 7-8 rabbits each and located in 10 different conventional cages in the same room. Each group was assigned a different commercial dry pellet. Characteristics of the different products used are shown in table 1.

TABLE 1: Characteristics of the different commercially pellets used for fattening.

<i>dry pellets contents</i>	<i>A</i> ¹	<i>B</i> ²	<i>C</i> ²	<i>D</i> ²
- protein	16,0%	15,3%	16,1%	16,4%
- fat	3,4%	4,2%	3,7%	3,8%
- cellulose	16,3%	16,5%	16,1%	14,5%
- draw fibre	6,8%	8,3%	6,6%	8,2%

¹ Pellets without antibiotics and coccidiostatic.

² Pellets without antibiotics.

24 hours after arrival, all the rabbits began to be fed *ad libitum*. From this day the room was visited twice a day, manure was removed by using water and the animals were visually inspected in order to identify any kind of disease event. All dead animals were necropsied.

RESULTS AND DISCUSSION

Incidences during all the fattening period were minimal. At the end of the first part of the assay, 70 of the 72 initial rabbits were shipped for slaughtering. Only two rabbits died and with no clinical or pathological signs of the disease. As in the first part of the assay, incidences during the second part were minimal through all the fattening period, after which only 3 rabbits died and 67 healthy rabbits were shipped to the slaughter-house. In none of the 5 observed deaths neither the symptoms nor the necropsy findings coincide with those reported for the clinical syndrome observed in the field. Apparently, the 5 deaths could be the consequence of other processes frequently affecting rabbits kept in intensive conditions but in a definitive lower intensity. The disease has not been reproduced in our facilities even though animals and dry rabbits pellets had been introduced from farms where the mucoid enteropathy had been clinically present in the previous months.

We were not able to reproduce the syndrome in our facilities through the introduction of animals from two farms that had previously reported the disease and different kinds of

commercial dry pellets. It must be noted that, although the animals were raised in the same manner as in a commercial farm, the environmental conditions differ significantly due to the good hygiene practices applied through all the fattening period. Results obtained from this study could support the hypothesis that the environment and its microbism play an important role in the appearance and / or intensity of the disease (once appeared) in commercial farms (6). This hypothesis could explain the important differences observed in the presentation of the syndrome in farms located in the same areas and with similarities in relation to risk factors such as feeding practices. Huge effort has been directed to the identification of new viral pathogens in order to find an explanation of the origin of the disease. The role of bacteria commonly presented in the environment of the farms as potential causes of the syndrome should not be overlooked

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