



## Importation of commercial rabbits (*Oryctolagus cuniculus*) from certain Member States of the European Union. Policy review of epizootic rabbit enteropathy.

August 2012

### Summary

The Animal Biosecurity Branch of the Department of Agriculture, Fisheries and Forestry (DAFF) monitors new scientific information, and reviews its import policies, when available information indicates a change in the risk associated with importation of animals or animal products.

Animal Biosecurity Branch conducted a policy review of epizootic rabbit enteropathy (ERE) for the importation of commercial rabbits from certain Member States of the European Union as available scientific information indicates that biosecurity measures for ERE are no longer required.

### Introduction

The current import policy for commercial rabbits from certain Member States of the European Union<sup>1</sup> was published on the DAFF website on 30 November 2000 (AQPM 2000/33). The policy was developed following suspension of imports in 1997. The suspension was due to the emergence of a new, apparently infectious, disease referred to as epizootic rabbit enterocolitis in France in 1996. Following initial research, the name was changed from epizootic rabbit enterocolitis to epizootic rabbit enteropathy (ERE) to reflect the lack of inflammatory lesions in the intestines.

The current import policy details a complex protocol developed to manage the biosecurity risk of ERE.

### Discussion

Digestive disorders are the main causes of economic losses in rabbit production in Europe (Le Bouquin et al. 2009). Since 1997 ERE has become a major cause of morbidity and mortality in European rabbitries. Young rabbits at the time of weaning are commonly affected. The clinical signs include reduced appetite, weight loss, abdominal distension, limited diarrhoea and mucous excretion (Le Bouquin et al. 2009). These signs are associated with sudden high mortality.

Since the initial outbreak of ERE, similarities with another disease of rabbits, mucoid enteropathy (ME), have been described (Licois et al. 2006). Much of the research on both syndromes has focused on identification of an infectious aetiology. Despite initial indications that ERE was infectious, an infectious aetiology has not been identified. Viral and parasitic causes have been ruled out (Huybens et al. 2011; Szalo et al. 2007). Other known pathogens such as *Clostridium* spp., coccidia and rotavirus are associated with morbidity and mortality in rabbits with similar clinical signs (Dewrée et al. 2007; Marlier et al. 2006; Szalo et al. 2007). It is inconclusive whether proliferation of *Clostridium perfringens* is a consequence, or part of the aetiology, of ERE (Romero et al. 2011). Although the syndrome has been reproduced in the absence of *Clostridium* spp. (Marlier et al. 2006; Szalo et al. 2007), other studies have shown a link between the presence of *Clostridium perfringens* and ERE (Marlier et al. 2006; Romero et al. 2011; Szalo et al. 2007). These findings are similar to initial research into the aetiology of ME, which also investigated an infectious cause without result

<sup>1</sup> Austria, Belgium, Denmark, Finland, France, Germany, Greece, Ireland, Italy, Luxembourg, Netherlands, Portugal, Spain, Sweden, United Kingdom

(Harkness and Wagner 1983; Glantz 1971, Greenham 1962, Cowrie-Whitney 1970 cited in Lelkes and Chang 1987; van Kruiningen and Williams 1972). *Clostridium* spp. are also present and contribute to mortality in ME-affected animals (Harkness and Wagner 1983; LebDAH and Shahin 2011; Lelkes and Chang 1987).

Both syndromes affect animals at the time of weaning and have the same clinical signs, gross lesions, histopathological findings and methods of control. The only difference between the two diseases has been defined as the sporadic nature of ME compared with epizootic outbreaks of ERE in Europe (Licois et al. 2000). The naming of ERE was applied because of the rapid spreading or 'epizootic' nature of the disease in Europe (Licois et al. 2006). Not only has the similarity between ERE and ME been acknowledged by researchers (Licois et al. 2006), many authors refer to the two syndromes interchangeably (Haligur et al. 2009; LebDAH and Shahin 2011; Lord 2012; Vandekerchove et al. 2001). Furthermore, in the analysis of 4307 urgent veterinary visits to commercial rabbit farms in Spain and Portugal from 1997 to 2007, the majority of visits were for investigation of ME (Rosell, 2009). No visits were recorded for ERE, however both countries were reportedly affected by ERE during the 1997 outbreak (Licois et al. 2006). It appears that the same syndrome was being reported but referred to by different names.

Although ERE is endemic in Europe it has not been reported elsewhere. This is despite other countries having no import requirements for ERE, and likely to have imported rabbits from Europe. In contrast, ME has been reported in many countries outside of Europe including Egypt, Mexico, North America and Turkey (Haligur et al. 2009; LebDAH and Shahin 2011; Rodríguez-De Lara et al. 2008; van Kruiningen and Williams 1972). It appears two similar syndromes are being described with the choice of name based on geographical location.

A reduction in mortalities attributed to ERE was achieved through improved management including the adoption of a batch breeding system, improved hygiene, changes in diet (Licois et al. 2006) and the selection of breeds shown to be resistant to ERE (de Rochambeau et al. 2006). Outbreaks were also well controlled with the use of certain antibiotics (Maertens et al. 2005). The recommendations to prevent ME also include correct levels of fibre in the diet (Harkness and Wagner 1983) and improved hygiene (van Kruiningen and Williams 1972)

There is no diagnostic test available for ERE. Diagnosis is based on a combination of clinical signs, gross lesions, histopathology and an absence of other known pathogens (Licois 2004). The histopathological changes are regarded as non-specific (Licois 2004). Both clinical signs and histological findings are the same as for ME. Diagnosis can also be complicated by the presence of secondary infections (Marlier et al. 2006).

Wild rabbits in their natural environment do not seem to be affected by ERE (Licois et al. 2006) and ERE has not been reported in any other animal species or in humans. ERE is not a World Organisation for Animal Health (OIE)-listed disease and is not on the National Notifiable Animal Disease List or on the Notifiable disease list of any Australian State or Territory.

## Conclusion

Since the initial outbreak of ERE, similarities with ME have been described (Licois et al. 2006). No infectious cause has been identified for either syndrome. In addition, research has failed to identify any significant differences between ERE and ME.

In summary the following factors were considered relevant to assessing the biosecurity risk associated with ERE:

- Scientific research and investigations have not determined an infectious cause for ERE.
- ERE is similar to ME, which is associated with sub-optimal animal husbandry practices, it has been reported that improvement of those practices can ameliorate the effects of both diseases.

- ERE has not been observed in wild rabbits in their natural surrounds and is not reported in any other animal species or humans.
- ERE is not an OIE-listed disease. It is also not on the National Notifiable Animal Disease List or on the notifiable disease list of any Australian State or Territory.

Based on these considerations Animal Biosecurity concludes that the biosecurity risk associated with ERE does not justify biosecurity measures for the importation of commercial rabbits from certain Member States of the European Union.

## Reference List

de Rochambeau H, Licois D, Gidenne T, Verdelhan S, Coudert P, Elsen JM (2006) Genetic variability of the resistance for three types of enteropathy in the growing rabbit. *Livestock Science* 101: 110-115.

Dewrée R, Meulemans L, Lassence C, Desmecht D, Ducatelle R, Mast J, Licois D, Vindevogel H, Marlier D (2007) Experimentally induced epizootic rabbit enteropathy: clinical, histopathological, ultrastructural, bacteriological and haematological findings. *World Rabbit Science* 15: 91-102.

Haligur M, Ozmen O, Demir N (2009) Pathological and ultrastructural studies on mucoid enteropathy in New Zealand rabbits. *Journal of Exotic Pet Medicine* 18: 224-228.

Harkness JE, Wagner JE (1983) Mucoid enteropathy. In *The biology and medicine of rabbits and rodents*, 2nd edn, pp. 219-221. Lea & Febiger, Philadelphia.

Huybens N, Houeix J, Licois D, Mainil J, Marlier D (2011) Epizootic rabbit enteropathy inoculum (TEC4): antibiograms and antibiotic fractionation. *Veterinary Research Communications* 35: 13-20.

Le Bouquin S, Jobert JL, Larour G, Balaine L, Eono F, Boucher S, Huneau A, Michel V (2009) Risk factors for an acute expression of Epizootic Rabbit Enteropathy syndrome in rabbits after weaning in French kindling-to-finish farms. *Livestock Science* 125: 283-290.

Lebdah MA, Shahin AM (2011) Closteridia as an etiological agent of mucoid enteropathy in rabbits. *Nature* 9: 63-72.

Lelkes L, Chang CL (1987) Microbial dysbiosis in rabbit mucoid enteropathy. *Laboratory Animal Science* 37: 757-764.

Licois D (2004) Domestic rabbit enteropathies. In *Proceedings 8th world rabbit congress, 7-10 September, 2004, Puebla, Mexico*, pp. 385-403, World Rabbit Science Association, France.

Licois D, Coudert P, Ceré N, Vautherot JF (2000) Epizootic enterocolitis of the rabbit: review of current research. In *Proceedings 7th world rabbit congress, 4-7 July, 2000, Valencia, Spain*, World Rabbit Science Association, France.

Licois D, Coudert P, Marlier D (2006) Epizootic rabbit enteropathy. In *Recent advances in rabbit sciences* (eds. Maertens L, Coudert P) pp. 163-170. Institute for Agricultural and Fisheries Research (ILVO), Melle, Belgium.

Lord B (2012) Gastrointestinal disease in rabbits 2. Intestinal diseases. *In Practice* 34: 156-162.

Maertens L, Cornez B, Vereecken M, Van Oye S (2005) Efficacy study of soluble bacitracin (Bacivet S<sup>®</sup>) in a chronically infected epizootic rabbit enteropathy environment. *World Rabbit Science* 13: 165-178.

Marlier D, Dewrée R, Lassence C, Licois D, Mainil J, Coudert P, Meulemans L, Ducatelle R, Vindevogel H (2006) Infectious agents associated with epizootic rabbit enteropathy: Isolation and attempts to reproduce the syndrome. *The Veterinary Journal* 172: 493-500.

Rodríguez-De Lara R, Cedillo-Peláez C, Constantino-Casas F, Fallas-López M, Cobos-Peralta MA, Gutiérrez-Olvera C, Juárez-Acevedo M, Miranda-Romero LA (2008) Studies on the evolution, pathology, and immunity of commercial fattening rabbits affected with epizootic outbreaks of diarrhoeas in Mexico: A case report. *Research in Veterinary Science* 84: 257-268.

Romero C, Nicodemus N, Jarava ML, Menoyo D, de Blas C (2011) Characterisation of *Clostridium perfringens* presence and concentration of its a-toxin in the caecal contents of fattening rabbits suffering from digestive diseases. *World Rabbit Science* 19: 177-189.

Szalo IM, Lassence C, Licois D, Coudert P, Poulipoulis A, Vindevogel H, Marlier D (2007) Fractionation of the reference inoculum of epizootic rabbit enteropathy in discontinuous sucrose gradient identifies aetiological agents in high density fractions. *The Veterinary Journal* 173: 652-657.

van Kruiningen HJ, Williams CB (1972) Muroid enteritis of rabbits: comparison to cholera and cystic fibrosis (with color plate II). *Veterinary Pathology Online* 9: 53-77.

Vandekerchove D, Roels S, Charlier G (2001) A naturally occurring case of epizootic enteropathy in a specific-pathogen-free rabbit colony. *Vlaams Diergeneeskundig Tijdschrift* 70: 486-490.