Study of polymorphisms in *tir* and *eae* genes of enterohemorragic and enteropathogenic *Escherichia coli* of serogroup O26.

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Enterohemorragic *Escherichia coli* strains (EHEC) represent an important problem for public health in developed countries. Indeed, EHEC strains can infect humans via vegetal and animal food consumption and cause food poisoning with diarrhoeas, frequently accompanied by hemorrhagic colitis with, in 10 % of the cases, apparition of renal sequelae (Haemolytic Uraemic Syndrome) that can lead to death. Domestic ruminants (especially cattle) are considered to be the main reservoir of EHEC strains for human infections. In the veterinary field, several serogroups of EHEC strains (O26, O111, O118 for example) are also associated with digestive disorders in two weeks- to two months-old calves.

Pathogenicity of EHEC is divided into four stages: (1) colonisation of intestine by specific adhesins, (2) translocation of a signal into the enterocyte by the type III secretion system of the bacteria and integration of the Translocated Intimin Receptor (Tir) into the host cell membrane, (3) intimate adhesion of bacteria to eukaryote cells by specific adhesins (Intimins) that bind to Tir, and (4) production of verocytotoxins (or Shiga toxins). Major polymorphisms of the Tir receptor and Intimin adhesin have been described between EHEC strains and are related, to some extent to the EHEC serogroups. F.i. EHEC srains belonging to the O26 serogroup produce Intimin and Tir of the β types. On the other hand the minor polymorphisms of these two proteins within a serogroup have not often been defined.

The aim of this study was to (1) investigate the polymorphism of the *tir* and *eae* genes (coding respectively for Tir and Intimin) existing amongst O26 EPEC and EHEC strains isolated from bovines and from humans; and (2) determine if these polymorphisms are specific to bovine or human strains.

A 2941 pb fragment of the *eae* gene and a 1559 pb fragment of the *tir* gene of 77 EPEC and EHEC strains of serogroup O26, isolated from humans and bovines, were amplified by PCR and sequenced. Sequences were aligned and compared. Several minor polymorphisms were detected amongst O26 human and bovine EHEC strains. Nevertheless, none was specific to the host. Additional work is currently ongoing to analyze the different polymorphisms found among the collection of strains in relation to the intracellular mechanism of action.