Three-step anti-aMPV IgA expression profile evaluation in turkeys of different immunological status after TRT vaccination


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Abstract

Maternally derived antibodies (MDA) do not protect turkeys against rhinotracheitis (TRT) but high MDA influences upper respiratory tract (URT) immunity stimulation after avian Metapneumovirus (aMPV) vaccination. Humoral immunity cannot be considered as an indicator of protection against TRT, but specific antibodies inhibit aMPV replication and alleviate the course of TRT. Scarce reports indicate the role of IgA in protection against TRT. The aim of our study was to investigate the impact of MDA on stimulation, antigen specificity acquisition of B lymphocytes, and the production of specific IgA after TRT vaccination of turkeys.

The results of our study indicate that MDA on the day of TRT vaccination causes disturbances at different levels of specific humoral immunity expression including antigen specificity acquisition of B IgA⁺ lymphocytes as well as production and secretion of IgA. Vaccine immunity against aMPV associated with sIgA is well expressed in birds not possessing MDA on the day of TRT vaccination, whereas it is inhibited in MDA⁺ birds.

These results corroborate our previous findings and indicate that MDA could be responsible for TRT vaccination failure. These findings could explain the observed frequency of TRT field outbreaks despite aMPV vaccination of turkey flocks.

Key words: aMPV, turkeys, vaccination, IgA; ELISPOT, maternally derived antibodies

Introduction

Avian Metapneumovirus (aMPV), RNA virus being a member of the Paramyxoviridae, genus Metapneumovirus (Buys and Preez 1980, Pedersen et al. 2000), is a causative agent of turkey rhinotracheitis (TRT). aMPV has a worldwide distribution and only Australia and Canada are still considered free from TRT. Up to date aMPV have been classified into 4 subtypes (A – D) (Collins et al. 1993, Cook et al. 1993).

Turkeys respond with strong stimulation of humoral immunity to aMPV infection; however, there is a lack of correlation between specific antibodies and protection against TRT (Jones et al. 1988, Cook et al. 1989, Cha et al. 2007). On the other hand, the clinical