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Genetic diversity of NHE1 and resistance/susceptibility to avian leukosis virus J in birdsJiri Hejnar¹, Jiri Plachy¹, Marketa Reinisova¹, Dana Kucerova¹, Filip Senigl¹ and Michal Vinkler²¹Institute of Molecular Genetics, Academy of Sciences of the Czech Republic, Videnska 1083, Czech Republic²Charles University in Prague, Faculty of Science, Department of Zoology, Vinicna 7, Czech Republic

The J subgroup avian leukosis virus (ALV-J) infects domestic chicken, jungle fowl, and turkey and enters the host cell through a receptor encoded by *tvj* locus and identified as Na⁺/H⁺ exchanger 1 (NHE1). The resistance to ALV-J in a great majority of galliform species was explained by deletions or substitutions of the critical tryptophan 38 in the first extracellular loop of NHE1. Because several cases of ALV-J positivity have been reported in feral duck species recently in Asia, we studied the natural polymorphisms of NHE1 in wild ducks, geese, and other avian species. In parallel, we examined the NHE1 polymorphism in domestic chicken breeds where we documented differences in their susceptibility to ALV-J *in vitro*. In a panel of chicken breeds assembled with the aim to cover the maximum variability encountered in domestic chickens, we found a completely uniform sequence of NHE1 extracellular loop 1 (ECL1) without any source of genetic variation for the selection of ALV-J-resistant poultry. In wild bird species, mostly *galloanseriforms*, we demonstrate polymorphic amino-acids and the prevalent absence of the critical tryptophan residue within ECL1. In several species, the W38 was detected and the susceptibility to ALV-J was confirmed by infection of primary cells in culture. Our results will be discussed as to the chance of ALV-J to be transmitted to new hosts and to establish the natural reservoir of circulating virus with potential of further evolution.

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