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Lineage diversification of pigeon paramyxovirus effect re-emergence potential in chickens

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ABSTRACT

Genotype VI-paramyxovirus (GVI-PMV1) is a major cause of epidemic Newcastle-like disease in Columbiformes. This genotype of avian paramyxovirus type 1 has diversified rapidly since its introduction into the US in 1982 resulting in two extant lineages, which have different population growth properties. Although some GVI-PMV1s replicate poorly in chickens, it is possible that variants with different replicative or pathogenic potential in chickens exist among the genetically-diverse GVI-PMV1s strains. To determine if variants of Columbiform GVI-PMV1 with different phylogenetic affiliations have distinct phenotypic properties in chickens, we investigated the replicative properties of 10 naturally circulating pigeon-derived isolates representing four subgroups of GVI-PMV1 in primary chicken lung epithelial cells and in chicken embryos. Our data demonstrate that GVI-PMV1 variants have different infection phenotypes in their chicken source host and that properties reflect subgroup affiliation. These subgroup replicative properties are consistent with observed dynamics of viral population growth.

Introduction

Genotype-VI paramyxovirus (GVI-PMV1) arose from cross-species transmissions from chickens to pigeons and now has strong host fidelity to pigeons (Chong et al., 2013). GVI-PMV1 strains cause systemic infection in pigeons resulting in respiratory disease (Toro et al., 2005) and with notable symptoms in the central nervous system and gastrointestinal tract (Barber et al., 2010). GVI-PMV1 is most frequently detected in Columbiformes (*i.e.* pigeons and doves) populations (Kim et al., 2008) but continues to cause sporadic outbreaks in chickens (Abolnik et al., 2008; Hassan et al., 2010; Pedersen et al., 2004). Despite possessing the genetic signature for virulence at the F gene cleavage site, GVI-PMV1 isolates typically replicate poorly and are not virulent in chickens in experimental studies (Meulemans et al., 2002). The virulence potential of GVI-PMV1 in chickens is determined by virus strain as well as by the age of chickens, route and intensity of viral inoculations used in the experimental settings (Gelb et al., 1987; King, 1996; Pearson et al., 1987; Toro et al., 2005). For example, the consequences of infection by different GVI-PMV1 strains range from mild respiratory disease to neurotropic disease and mortality in experimentally infected chickens (Gelb et al., 1987; Pearson et al., 1987; Toro et al., 2005). Serial passage in chickens or embryonated eggs increased virulence (Dortmans et al., 2011; Fuller et al., 2007; Kommers et al., 2001) and enhanced the replication of GVI-PMV1 (Dortmans et al., 2011), and in one study isolates recovered from experimentally passaged virus in chickens had only three mutations in two viral genes, P and L (Dortmans et al., 2011). This data suggest that small changes in the viral genome are sufficient to allow GVI-PMV1 infections in chickens.

After the emergence of GVI-PMV1 in European Columbiformes in the 1980s, there were at least three virus introductions to North America. GVI-PMV1 in North American Columbiformes has evolved at a high substitution rate and there is measurable genetic diversity in contemporary strains (Chong et al., 2013). Two extant lineages, GVIbii-*d* and -*g*, have diversified locally; the former has maintained a fairly constant viral population size over time, whereas the later has experienced exponential population growth (Chong et al., 2013). Despite the apparent fidelity of GVI-PMV1 for pigeons, outbreaks in poultry continue to be reported in many countries (Abolnik et al., 2008; Hassan et al., 2010; Mase et al., 2002; Pedersen et al., 2004; Ujvari et al., 2003) and GVI-PMV1 can be isolated from chickens in the absence of an epizootic. It is therefore of substantial interest for both risk assessment and diagnostics to determine if the genetic diversification of the GVIbii-*d* and -*g* lineages derived from the pigeons has

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