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Research paper

The full-length genome characterization, genetic diversity and evolutionary analyses of Senecavirus A isolated in Thailand in 2016

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ABSTRACT

Senecavirus A (SVA) is a novel picornavirus that causes porcine idiopathic vesicular disease characterized by lameness, coronary band hyperemia, and vesicles on the snout and coronary bands. An increase in the detection rate of SVA in several countries suggests that the disease has become a widespread problem. Herein, we report the detection of SVA in Thailand and the characterization of full-length genomic sequences of six Thai SVA isolates. Phylogenetic, genetic, recombination, and evolutionary analyses were performed. The full-length genome, excluding the poly (A) tail of the Thai SVA isolates, was 7282 nucleotides long, with the genomic organization resembling other previously reported SVA isolates. Phylogenetic and genetic analyses based on full-length genome demonstrated that the Thai SVA isolates were grouped in a novel cluster, separated from SVA isolates from other countries. Although the Thai SVA isolates were closely related to 11-55910-3, the first SVA isolate from Canada, with 97.9–98.2%, but they are different. Evolutionary and recombinant analyses suggested that the Thai SVA isolates shared a common ancestor with the 11-55910-3 isolate. The positive selection in the VP4 and 3D genes suggests that the virus was not externally introduced, but rather continuously evolved in the population prior to the first detection. Addition, the presence of SVA could have been ignored due to the presence of other pathogens causing similar clinical diseases. This study warrants further investigations into molecular epidemiology and genetic evolution of the SVA in Thailand.

1. Introduction

Senecavirus A (SVA), formerly recognized as Seneca Valley virus (SVV), is a non-enveloped, single-stranded RNA virus in the genus *Senecavirus*, family *Picornaviridae* (Adams et al., 2015). SVA is the causative agent of a disease recognized as porcine idiopathic vesicular disease (PIVD). This disease is characterized by lameness, coronary band hyperemia, and vesicles on the snout and coronary bands, and it is indistinguishable from other vesicular diseases in swine such as foot-and-mouth disease (FMD), vesicular stomatitis (VS), swine vesicular disease (SVD) and vesicular exanthema of swine (VES).

The full-length genome of SVA is approximately 7.3 kb in length including the 5′-untranslated region (UTR), a large open reading frame (ORF), and a polyadenylated 3′-UTR. The 5′-UTR region contains two major parts. The first part is a viral protein, genome linked (VPg), that plays an important role in replication (Racaniello, 2007) and the second part is a type IV internal ribosome entry site (Willcocks et al., 2011). The ORF encodes a large polyprotein that contains leader (L) and polyprotein 1, 2, and 3 (P1, P2, and P3, respectively) regions. The P1 region is cleaved into three structural polypeptides called VP0, VP3, and VP1. VP0 further generates VP2 and VP4. VP1 contains K228, which binds to the host low-density lipoprotein receptor (LDLR) (Venkataraman et al., 2008). VP2 contains DGK and LDV motifs that have been suspected to be binding sites of α_V integrin and $\alpha_1\beta_4$ integrin host cell receptors, respectively (Venkataraman et al., 2008). The P2 region encodes 2A, 2B, and 2C, which are nonstructural polypeptides similar to the P3 region that encodes 3A, 3B, 3C, and 3D (Racaniello, 2007).

SVA was first detected in 2002 in a contaminated PER.C6 cell line culture. The first isolate was named SVV-001, and its full-length genomic sequence was subsequently reported in 2005 (Hales et al., 2008). In 2008, a suspicious vesicular disease with an unknown etiologic agent was reported in the United States (Pasma et al., 2008). The disease was identified in pigs imported from Canada, and SVA was

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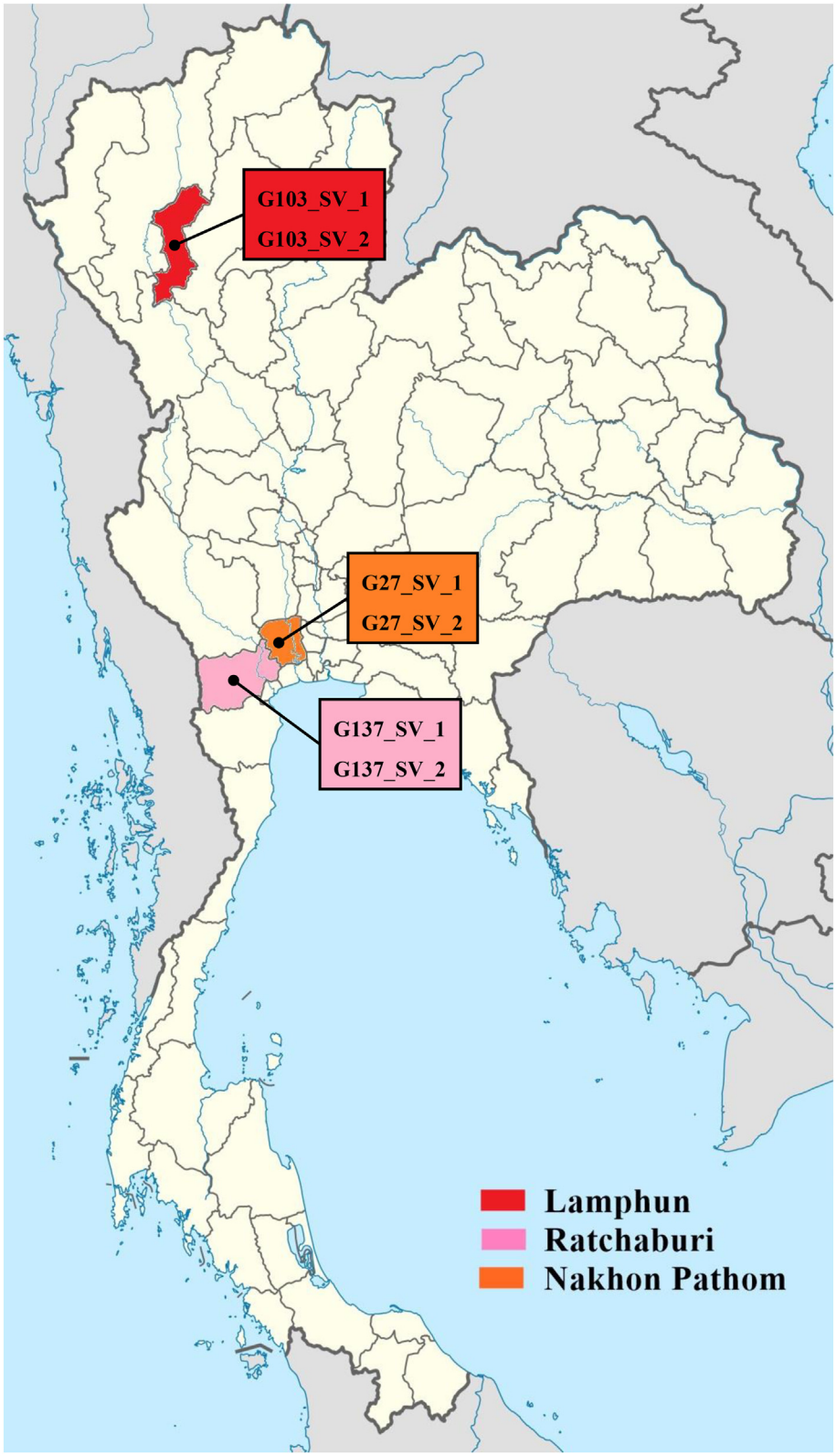

Fig. 1. Senecavirus A (SVA)-positive farms locations in three provinces in Thailand, including Lamphun (red), Ratchaburi (pink), and Nakhon Pathom (orange). (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

subsequently detected in those pigs. At present, SVA has been detected in several countries, including Canada, Brazil, and China (Bracht et al., 2016; Canning et al., 2016; Gimenez-Lirola et al., 2016; Guo et al., 2016; Joshi et al., 2016; Leme et al., 2015; Montiel et al., 2016; Vannucci et al., 2015; Wu et al., 2017).

In 2016, one swine farm in the northern region of Thailand reported a disease outbreak associated with vesicular disease characterized by lameness, coronary band hyperemia, and vesicles. The causative agent was thought to be a variant of FMD virus (FMDV), a major cause of vesicular diseases in Southeast Asia (Jamal et al., 2011). Interestingly,