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Letter to Editor **Open Access**

A New Member of Cardiovirus

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Animal experiments have been carried out in order to study the pathogenicity of SAFV, although SAFV does not infect rodents naturally. The experiments of different two groups suggest that SAFV is neurotropic in mice. Of special note is the presence of inflammation in the spinal cord white matter, because the closely related mouse cardiovirus, TMEV, causes a demyelinating central nervous system disease that resembles multiple sclerosis [3]. Therefore, the recombination between SAFV and TMEV, which could alter the host range, will be a serious problem to humans. However, Himeda et al. reported in the other issue of J Plant Pathol Microbiol that the recombination of capsid proteins between SAFV and TMEV did not occur [4]. Another important issue is that the major viral load following SAFV intraperitoneal inoculation is the pancreas. Several viruses in the family Picornaviridae, particularly Coxsackie B viruses have long been implicated in the etiology of type I diabetes. In addition, EMCV is known to induce pancreatitis and type I diabetes in rodents [3]. Recently, several European groups started the study to investigate the relationship between SAFV and type I diabetes. The answer to this issue awaits the data from them.

From these observations, the pathogenicity of SAFV still remains unclear, although the potential pathogenicity of SAFV to humans is thought to be varied (respiratory, gastrointestinal and neurological diseases and type I diabetes etc.). In order to clarify the pathogenicity of SAFV, the further epidemiological studies including the data of healthy persons as a control group is required. In addition, the researches of viral factors involved in the pathogenicity of SAFV using a reverse genetic technique [5] are needed. Furthermore, the identification of the receptor(s) for SAFV infection is also important in order to establish the transgenic mice as a novel animal model to study the pathogenicity of SAFV.

Cardiovirus A is a member of the Picornaviridae family. Infection with the virus causes encephalomyocarditis and reproductive disease in pigs. Although a variety of mammalsmay host the virus, pigs are classed as the domestic host as they are most easily infected. It is thought to be spread by rodents. The disease can be found worldwide but is of greatest economic importance in tropical areas. It is not thought to be zoonotic.

Picornaviruses are classed under Baltimore's viral classification system as group IV viruses as they contain a single stranded, positive sense RNA genome. Their genome ranges between 7.1 and 8.9 kb (kilobases) in length.[1] Like most positive sense RNA genomes, the genetic material alone is infectious; although substantially less virulent than if contained within the viral particle, the RNA can have increased infectivity when transfected into cells. The genome RNA is unusual because it has a protein on the 5' end that is used as a primer for transcription by RNA polymerase.

The genome is non-segmented and positive-sense (the same sense as mammalian mRNA, being read 5' to 3'). Unlike mammalian mRNA picornaviruses do not have a 5' cap but a virally encoded protein known as VPg. However, like mammalian mRNA, the genome does have a poly(A) tail at the 3' end. There is an un-translated region (UTR) at both ends of the picornavirus genome. The 5' UTR is usually longer, being around 500–1200 nucleotides (nt) in length, compared to that of the 3' UTR, which is around 30–650 nt. It is thought that the 5' UTR is important in translation and the 3' in negative strand synthesis; however the 5' end may also have a role to play in virulence of the virus. The rest of the genome encodes structural proteins at the 5' end and non-structural proteins at the 3' end in a single polyprotein.