RESEARCH ABSTRACT
SWINE HEALTH

Title: Neuropathogenesis of a brain-derived *Porcine teschovirus* type 11 strain or a brain-derived *Porcine sapelovirus* strain in 3-week-old CDCD pigs-NPB #13-211

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Introduction

A severe highly fatal polioencephalomyelitis affecting pigs of all ages was first reported in 1929 in Teschen, Czechoslovakia (Trefny 1930). Known as Teschen disease, the etiologic agent was a virulent neurotropic strain of type 1 porcine enterovirus that spread and caused devastating losses across Europe until it disappeared in the 1950s. Later, a milder form of the disease characterized by lower morbidity and mortality, less common mentation defects and predominantly posterior paresis and/or paralysis was reported in Talfan, Wales (Harding et al. 1957). The cause was determined to be a less virulent neurotropic strain of type 1 porcine enterovirus. Since then, this less virulent polioencephalomyelitic disease, known as Talfan disease, is reported in countries around the world in association with predominantly type 1, but less commonly types 2-6, 9 and 10 of porcine enterovirus (Alexandersen et al. 2012). Porcine enteroviruses were historically serotyped by virus-neutralization and 13 serotypes were described. They were further subdivided by physiochemical properties i.e. replication properties in various cell lines into three cytopathic effect (CPE) groups: group I (serotypes 1-7 and 11-13), group II (serotype 8) and group III (serotypes 9 and 10) (Zoletto 1965, Knowles et al. 1979). Subsequent analysis of genomic sequences confirmed the distinctness of the three groups and led to complete reclassification (Kaku et al. 2001, Zell et al. 2001) as follows: Porcine enterovirus group I serotypes 1-7, 11-13 are assigned to the new genus *Teschovirus* as porcine teschovirus (PTV) serotypes 1-10 and additional serotypes 11 – 13 are also described; porcine enterovirus group II, serotype 8, is assigned to genus *Sapelovirus* as porcine sapelovirus (PSV); and porcine enterovirus group III, serotypes 9 and 10, remain as porcine enterovirus B (PEV-B).

In general, one or more strains of various types of PTV are endemic in commercial swine operations and most of growing and adult pigs have a serologic evidence of exposure to the virus. In one long-term longitudinal study spanning 26 months in a single swine herd, waves of

infection by six different serotypes of PTV were demonstrated (Singh and Bohl 1972). The virus is known to be a resident in the pig gut and is commonly found in feces (Alexandersen et al. 2012). In contrast, Talfan-like nervous disease with polioencephalomyelitis is relatively uncommon and inconsistent in North America. Typically the problem in affected herds is sporadic and self-resolved. Recent evidence suggests that this may be changing. Beginning in 2009, a regional outbreak of teschoviral polioencephalomyelitis occurred in Haiti affecting swine of all ages with 60% morbidity and 40% mortality (Deng et al. 2012), marking the first report in the Western hemisphere of a highly virulent strain of PTV. The outbreak was caused by a type 1 PTV with 84% and 86% nucleotide identity by whole genomic sequencing with the index strains of Teschen and Talfan viruses, respectively. And, starting in December of 2012, Iowa State University's Veterinary Diagnostic Laboratory (ISUVDL) has received neurologic cases with posterior paralysis and sometimes encephalitic signs (abnormal mentation) 5 to 10 times more than previously from swine operations in wide geographic locations (Iowa, Illinois, Minnesota, North Carolina). These outbreaks have been in predominantly nursery-age pigs, but have also less commonly been in grower pigs or in adults. Outbreaks have also been apparently lasting longer and affecting more pigs than previously. Polioencephalitis and/or myelitis is/are consistently observed and PTV and/or PSV are detected by PCR in brain and/or spinal cord. Sequencing of the PTV has frequently identified it as type 11. There have been no previous reports of type 11 PTV in cases of polioencephalomyelitis in North America. Likewise, porcine sapelovirus historically has not been associated with neurologic disease and is also frequently found in feces or intestine from clinically normal pigs. Hence, it is unexpected to detect PSV alone in brains and/or spinal cords from neurologic pigs. The significance of these findings is not clear, especially since fecal contamination during harvest of brains and spinal cords (often performed under field conditions by submitting veterinarians) may be the source of PCR-detected PTV and/or PSV. Therefore, the causative role of PTV11 and PSV in polioencephalomyelitis needs careful evaluation under experimental conditions. In addition, diagnosticians need a laboratory method such as in-situ hybridization or immunohistochemistry which can reliably visualize PTV or PSV within affected neurons in typical brain and spinal cord lesions, so that causality and not contamination can be interpreted from positive PCR test results.