## Viral Replication 2

306

### Trans-complementation studies with atypical pestiviruses provide new insights in the compatibility of pestivirus non-structural proteins

I. Reimann[1], Mar. Richter[1], P. D. Kirkland[2], M. Beer[1]

[1]Friedrich-Loeffler-Institut, Institute of Diagnostic Virology, Greifswald-Insel Riems, Germany
[2]Elizabeth Macarthur Agricultural Institute, Virology Laboratory, Camden, Australia

Among the atypical pestiviruses, Bungowannah virus is the most divergent member of the genus *Pestivirus*. In former studies, we used heterologous complementation to clarify the phylogenetic relationship and to demonstrate the exchangeability of the structural proteins. Here, we analysed the functional replaceability of non-structural (NS) proteins.
Using a bovine viral diarrhea virus (BVDV) backbone, several chimeric constructs, generated by the substitution of the NS proteins p7, NS2, NS3 and NS4A were investigated. While constructs with substitutions in the NS3 and/or NS4A encoding regions were not able to replicate in transfected bovine cells, substitutions of p7 and/or NS2 resulted in autonomous replication. Interestingly, infectious chimeric virus could only be observed after replacing the p7-encoding region (vCP7_p7-Bungo), which had the lowest amino acid homology (28.1%) to the respective BVDV proteins. In contrast, BVDV chimeras expressing NS proteins of the atypical HoBi virus (vCP7_NS3-HoBi, vCP7_NS3NS4A-HoBi) grew to high virus titres. However, both the complementation of Bungowannah virus- and HoBi virus-NS2 resulted in replicons only, not able to generate infectious virus progeny.
Our data demonstrate, that in contrast to the putative viroporin p7 the compatibility of Bungowannah virus-NS2, -NS3 and -NS4A with a BVDV backbone is severely reduced compared to other atypical pestiviruses. Furthermore, especially NS2 needs additional interactions with homologous proteins to allow the generation of virus progeny.

Corresponding author:
**Maria Richter**
Maria.richter@fli.bund.de