## THE ROLE OF TRANSLESION POLYMERASES IN THE GENERATION OF GENETIC VARIABILITY OF AN EMERGENT ssDNA PLANT VIRUS

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Single-stranded DNA (ssDNA) viruses such as animal circoviruses or plant geminiviruses are important emergent viruses. SsDNA viruses are as variable as their RNA equivalents and evolve quickly, with high mutation rates and mutation frequencies around  $10^{-3}$ - $10^{-5}$  mutations/nt. Several factors are responsible for the elevated substitution rates of ssDNA viruses including polymerase replication fidelity, mismatch repair, exogenous and endogenous DNA damage, nucleotide imbalances and the action of other cellular DNA modifying enzymes. Indels can also be introduced during replication or as a consequence of recombination. Unlike RNA viruses, which owe their genetic variability in part to their error prone RNA-dependent RNA polymerases, ssDNA viruses do not encode DNA polymerases. They are replicated by unknown cellular DNA polymerases in the host nucleus via a rolling circle mechanism. Mutation bias compatible with the deamination and oxidation of single-stranded DNA has been observed in geminiviruses. This kind of DNA damage is a substrate for Translesion Polymerases (TLS), involved in lesion bypass. TLS pols of the Y family lack proofreading activity and have low nucleotide selectivity, and thus exhibit high error rates for base substitutions and indels, even in the absence of damage. Here, we have addressed the involvement of TLS polymerases in the replication of the Tomato Yellow Leaf Curl Virus (TYLCV) geminivirus in Arabidopsis thaliana. To this end, wt A. thaliana, homozygous AtRev1 and AtPolK, and heterozygous AtPolH TLS mutants were infected with TYLCV. TLS expression and viral loads were analysed at 7, 14, 21 and 28 days post infection (dpi). We observed an absence of AtRev1 transcripts, a significant reduction in AtPolK expression, while AtPolH was expressed at wt levels in the corresponding mutants. In each mutant, expression of the other TLS polymerases was unaffected. In addition, viral loads in AtRev1, AtPolH and AtPolK were comparable to those of wt A. thaliana TYLCV infections. Preliminary results of the effect of altered TLS levels on TYLCV variability measured by next generation sequencing will be shown.