Conclusions and future plans
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Studies in our lab have identified two kinds of dominant RIP suppressor strains using the $Dp(erg-3)$-based assay, the seven wild-isolates and strains bearing large chromosome segment duplications. One of the dominant RIP suppressor strains, the Adiopodoumé strain, was also the only wild-isolated Neurospora strain found harboring active copies of a LINE-like transposable element named $Tad$. The dominant RIP suppressor of the Adiopodoumé strain ($Sr$) was tightly linked to $mat$ on LG IL and my main objective was to identify $Sr$.

Major conclusions

(1). I have shown that $Sr$, the dominant suppressor of RIP from the Adiopodoumé strain, shows incomplete penetrance and variable expressivity. Further, I showed that $Sr$ is identical to the Adiopodoumé allele of the $upr-1$ gene that encodes the catalytic subunit of the translesion DNA polymerase $\zeta$ (Pol $\zeta$). The suppressor Pol $\zeta$ catalytic subunit might interfere with the assembly or function of a putative RIP polymerase.

(2). I have shown that in a mosaic perithecium suppression by $Sr$ can spread into asci that do not contain $Sr$, therefore, $Sr$ acts in trans. However, suppression by $Dp$ is dominant within an ascus, it does not spread into asci that do not contain the $Dp$ and therefore, unlike RIP suppression by $Sr$, suppression via titration is autonomous to the $Dp$-bearing asci. This was demonstrated using a versatile new helper system.

(3). I have generated the first Neurospora Y family translesion DNA polymerase mutants in the gene for Pol $\eta$, Pol $\iota$, and Pol $\kappa$, and also generated an independent set of B family DNA polymerase Pol $\zeta$ mutants. These mutants were used to test whether these translesion polymerases are essential for RIP. I found that these polymerases are
dispensable for RIP. The Y family translesion DNA polymerase generated in this study can be utilized to study their role in different DNA damage repair process.

Ongoing work in the lab is testing whether dominant RIP suppression by the other non-barren suppressors also depends on their respective upr-1 alleles. It will be of interest to test whether Srp shows increased penetrance if the upstream ATGs of upr-lAd are mutated.