The exploration of the pathosystem BSV/*Musa* sp.: How does it work?

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As several other plants, the genome of banana and plantain contains integrations of *Banana streak virus* (BSV) sequences even though integration is not an essential step in the replication cycle of this virus. In banana two types of BSV integrants exist. Ones are non functional sequences present in both common *Musa* species, *Musa acuminata* (denoted A) and *Musa balbisiana* (denoted B) and it is now assumed that the integrants of the other type, containing the complete viral genome and restricted to *M. balbisiana* genome, become infectious by reconstituting a complete replication-competent viral genome. Thereby, an increasing record of BSV outbreaks was observed fifteen years ago among banana breeding lines and micro propagated inter-specific *Musa* hybrids, worldwide.

Today, three widespread BSV species, *Banana streak Obino l'Ewai virus* (BSOlV), *Banana streak Imové virus* (BSImV) and *Banana streak Golfinger virus* (BSGfV) are known to occur as infectious integrants in the *M. balbisiana* genome. However, even though such integrations are known to be infectious, their presence is not sufficient to induce infection

We demonstrated that the process of genetic hybridization and abiotic stresses such as micropropagation by *in vitro* culture contributed in triggering episomal expression from EPRVs. Two mechanisms at least are involved in the BSV expression: the ploidy of the *M. balbisiana* in *Musa* genotypes and an additional genetic factor called BEL for BSV expressed locus concerning the triploids (*Musa* AAB) resulting from inter-species genetic crosses between virus-free diploid *M. balbisiana* (BB) and tetraploid *M acuminata* (AAAA) parents. Then, diploids *M. balbisiana* such as PKW and Pisang Batu harboring pathogenic BSV EPRVs are resistant to any multiplication of BSV while haploid genotypes such as triploids (AAB, French clair) or tetraploids (AAAB, FHIA 21) expressed BSV. Thereby, we characterized the segregation of three BSV species appearance among the AAB F1 progeny as a monogenic allelic system conferring the role of carrier to the *M. balbisiana* diploid parent. BSOlV and BSImV appeared in almost all infected hybrids (50% of the progeny) depending of BEL regulation while BSGfV are restricted in only half of these hybrids and subordinated by BEL.

Three BAC libraries from accession of *M.acuminata* Cavendish subgroup cv petite naine (AAA), a wild *M. acuminata* subsp *burmannicoides* Calcutta 4 (AA) and a wild *M. balbisiana* PKW (BB) are explored for the pattern of integration of infectious BSV EPRVs by testing a set of different viral probes representing each time the BSOlV, Im and Gf complete genome.

BSV positive BAC clones are characterised by RFLP fingerprints approaches. This analysis revealed that the three BSV species represent low-copy loci and their integration is specific to the PKW *Musa balbisiana* genome. BSGfV EPRVs in PKW is twice and are fully annotated after sequencing. Each BSGfV is composed of back-to-back viral sequences representing more than a whole BSV genome very similar each other. We developed molecular markers (PCR, PCR-RFLP) to distinguish each others and analysed the BSGfV EPRV segregation in the AAB F1 progeny. BSGfV EPRVs are found to be allelic, located at the same locus. In theory, both allelic EPRVs could be involved in the restitution of virions through a set of recombination events.

BSV References

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