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Is *Banana streak virus/Arabidopsis thaliana* a viable pathosystem?

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Banana streak viruses (BSV) infect bananas and plantains (*Musa* spp.) worldwide. BSV became a major constraint for banana breeding programmes and germplasm exchanges since the discovery a couple of decade ago of the presence of infectious BSV integrations (eBSV) in the genome of *Musa balbisiana* (B genome) which can under certain stress conditions released spontaneously fonctional and infectious BSV. To date, BSV is known to only infect banana but for several reasons working with banana is very time consuming and not an easy task (space needed, long life cycle, no mutant database available, transient and stable transformations are difficult and laborious…). To overcome most of those problems and to both understand the mechanisms underlying the regulation of eBSV activation as well as eBSV non-activation and gain knowledge in the BSV biology, we tried to establish infection of Arabidopsis thaliana (At). We stably transformed two different ecotypes of At via agrobacterium using a more than a full length BSV viral genome clone containing twice the promoter region because of the poly A signal is in front of the promoter. T2 transformants did not show any abnormal phenotype neither BSV symptoms. RT-PCR experiments showed that viral transcription was very weak if not undetectable amongst more than 30 independent transformants. One hypothesis to explain this absence of transcription could be due to methylation of the viral genome. Using the cleavage activity of the McrBC methylation-dependent enzyme which recognizes 5-methylcytosines (a hallmark of plant DNA methylation) we demonstrated for all independent transformants tested (4 in total) than the viral construct was always methylated hampering potentially the BSV transcription. To validate and explore this hypothesis, we are crossing selected transformants with mutants affected at different stage of the epigenetic pathway.