Natural resistance of banana genotypes to banana streak virus is probably driven by transcriptional gene silencing.

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The genome of banana (\textit{Musa} sp.) harbours multiple integrations of \textit{Banana streak virus} (BSV), whereas this badnavirus does not require integration for the replication of its ds DNA genome. Some endogenous BSV sequences (eBSV), only existing in the \textit{Musa balbisiana} genome, are infectious by releasing a functional viral genome following stresses such as those existing in in vitro culture and interspecific crosses context. The structure of these eBSV is much longer than a single BSV genome, composed of viral fragments duplicated and more or less extensively rearranged. Wild \textit{M. balbisiana} diploid genotypes (BB) such as Pisang Klutuk Wulung (PKW) harbour such infectious eBSV belonging to three widespread species of BSV (\textit{Goldfinger} -BSGFV, \textit{Imové} – BSImV and \textit{Obino l'Ewai} - BSOLV) but are nevertheless resistant to any multiplication of BSV without any visible virus particles. We postulated these eBSV induced a natural resistance driven by gene silencing mechanisms based on their complex molecular re-arranged structure which could lead to dsRNA hairpins formation. In collaboration with the group headed by M. Pooggin (Basel, Switzerland), a deep sequencing of total siRNAs of PKW was performed using the Illumina ultra-high-throughput technology. We obtained for the first time, experimental evidence of virus-derived small RNA (vsRNA) from BSOLV, BSGFV and BSImV by blasting sequences against the 3 BSV species genomes. vsRNA are enriched in 24-nt class thus eBSV in PKW genome are likely silenced at the transcriptional level. A repartition of the vsRNA population matching eBSV will be also presented in order to determine hot and cold spots of vsRNA generation.