THE ROLE OF METHYLATION AND CHROMOSOMAL REARRANGEMENTS INVOLVED IN THE EXPRESSION OF PATHOGENIC *BANANA STREAK VIRUS* SEQUENCES INTEGRATED INTO THE GENOME OF BANANA

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Background

Banana streak virus (BSV) endogenous pararetroviral sequences (EPRVs) are present in the genome of *Musa balbisiana*, one of the two main wild progenitors of cultivated banana, the other one being *M. acuminata*. BSV EPRVs are suspected to give rise to infectious particles through activation processes that are triggered by biotic or abiotic stresses, such as genetic crosses between *M. balbisiana* and *M. acuminata* or *in vitro* culture. Little is currently known about the mechanisms underlying the expression of pathogenic BSV EPRVs and their regulations.

Methods

The role of methylation in the expression of pathogenic BSV EPRVs was investigated. Differential cytosine methylation patterns were searched in healthy and diseased triploid (AAB) hybrids from the progeny of a cross between the virus-free diploid *M. balbisiana* female parent PKW (BB) and the virus-free autotetraploid *M. acuminata* male parent IDN-T (AAAA), using the SD-AFLP/MSAP technique. DNA from healthy and diseased hybrids was extracted and 36 primer-enzyme combinations (PEC) were used. The role of chromosomal rearrangements was also investigated, through a PCR-based analysis of both genomic DNA extracted from the progeny of the above-mentioned cross and BAC clones of the PKW parent giving positive signals when hybridised to BSV-specific probes. Strain-specific primers covering distinct parts of the genome of BSV were used.

Results

An average of 40 DNA fragments per PEC were obtained by SD-AFLP/MSAP, resulting in approximately 1,500 screened fragments, of which thirteen showing quantitative or qualitative methylation variations were cloned and sequenced. Detailed analysis of their nucleotide sequences will be presented and discussed.

PCR-based analysis of chromosomal rearrangements among triploid hybrids leads to the identification of a genetic segregation pattern between diseased and healthy individuals. Differential patterns were observed depending on the primers used, raising evidence for chromosomal rearrangements in diseased hybrids. These results were corroborated by similar analysis performed on BAC clones of the PKW parent. They pave the way to the identification of a much needed specific molecular marker of pathogenic BSV EPRVs.