Fighting virus disease in sweetpotato

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Resistant varieties of sweetpotato

Sweetpotato (*Ipomoea batatas*) is one of the most important food crops in the world, with an annual production of 122 million tonnes. In developing countries, it is a famine reserve crop and consumed by the poor rural populations for subsistence. However, it is attacked by a number of diseases, especially viruses.

Sweetpotato chlorotic stunt virus (SPCSV) is one of the most important pathogens of sweetpotato. It can reduce yields by 40 percent, but what makes SPCSV most harmful is its ability to break down the natural resistance of sweetpotato to other viruses and open up the plant to other viral diseases. The most common and severe of these diseases is known as sweetpotato virus disease (SPVD) and is caused by co-infection with SPCSV and sweetpotato feathery mottle virus (SPFMV). Yield losses of 70–100 percent are regularly observed in infected plants. Most sweetpotato cultivars are extremely resistant to SPFMV infection alone, but few have any real resistance to SPCSV or SPVD. Although SPVD-resistant landraces grow in East Africa, most of them have various inadequacies such as poor and late yield. Furthermore, their resistance is controlled by a complex genetic structure that makes conventional breeding methods very difficult.

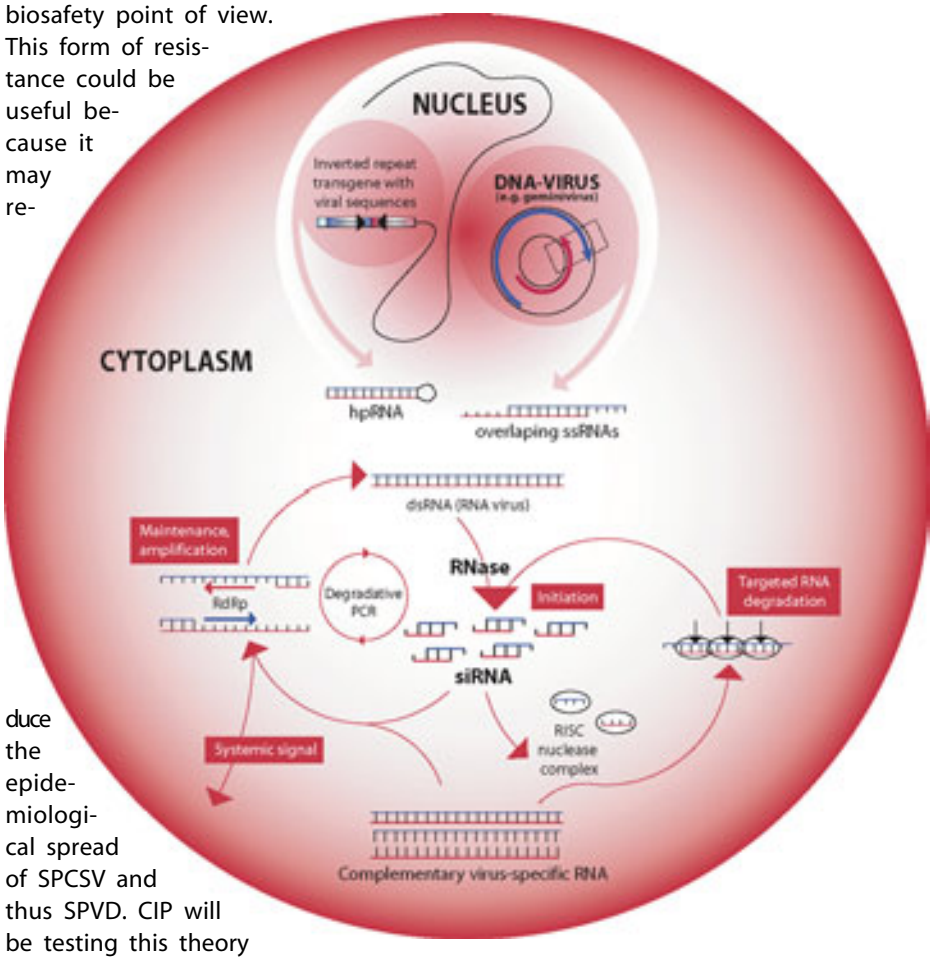
A fundamental natural defence in plants against viruses is called RNA silencing, where the plant produces small strands of RNA, called siRNA, that cut the virus genome into pieces and so silences them. Sweetpotato uses this mechanism very efficiently to resist most viruses, including SPFMV. However this is apparently not the case for SPCSV. No sources of true resistance to SPCSV are available in sweetpotato germplasm.

One of the few

alternative ways of introducing resistance to SPCSV into sweetpotato is gene technology. Introducing pieces of the virus genome into the plants through genetic transformation will induce plants to form siRNAs against the virus prior to infection. This renders the plants resistant as its defense system is already prepared for the virus. However, using biotechnology to transform sweetpotato is not easy. The protocols tend to function only with a few genotypes and at a low efficiency, and results often show low reproducibility. "One of our objectives was to improve the procedure to introduce genes that confer resistance to pathogens into sweetpotato varieties using the bacterium *Agrobacterium tumefaciens*," said CIP molecular virologist Jan Kreuze.

The transgene construct used in this study was relatively efficient in conferring resistance to SPCSV. "While the transgenic varieties we produced were not fully immune to SPCSV," said Kreuze, "Half of them showed mild or no symptoms following infection with the virus. This in itself is a significant step forward."

There are few previous reports of inducing such transgenic resistance to viruses of the family Closteroviridae in cultivated plants. Furthermore, this technology does not result in an accumulation of the transgene transcripts, nor expression of a foreign protein in the plant, which makes it attractive from the biosafety point of view. This form of resistance could be useful because it may reduce the epidemiological spread of SPCSV and thus SPVD. CIP will be testing this theory through field trials in Uganda in the future. However, the resistance was not enough to prevent SPVD after co-infection with SPFMV. "The key question was how SPCSV, even in the very low amounts found in the transgenic plants, causes the dramatic general loss of resistance to other viruses," said Jari Valkonen, professor at the Department of Applied Biology, University of Helsinki, Finland, who has been working with Jan Kreuze. Collaborative research between the University of Helsinki and CIP revealed a novel virus–mediated mechanism that causes an overall failure of the antiviral defence and makes plants vulnerable to many unrelated viruses. "The results pave new ways towards preventing globally important virus diseases", says Valkonen and co-researcher Wilmer Cuellar of the University of Helsinki. To that he adds "This is a complex viral disease that is much more challenging to solve than any other for which transgenic resistance has been attempted to date. But now we have a much better understanding of how to get ahead".



Introducing pieces of virus DNA into the plants 'immunizes' them against virus infection