Tackling Virus Resistance in Hot Spots

- Discriminated between resistant and tolerant cultivars in response to *Sweet potato feathery mottle virus* (SPFMV) and *Sweetpotato chlorotic stunt virus* (SPCSV). Dual infection by SPCSV and SPFMV cause sweetpotato virus disease (SPVD), that can cause devastating losses.
- Identified SPVD field resistant clones to be used as parents to breed (cross) for increased SPVD resistance in breeding populations
- Identified SPVD field resistant clones for forming a diversity panel for developing molecular markers
- Increased the frequency of SPVD resistant genotypes in breeding populations from less than 0.05% to over 20%.

SASHA Sweetpotato Action for Security and Health in Africa

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Fig 1. Sweetpotato virus disease infected plant (on left) and healthy plant (right side) (Credit R. Mwanga)

What is the problem?

Virus infection, by different types of viruses, but mainly SPCSV and SPFMV, is among the most important constraints to improving sweetpotato production globally and in sub-Saharan Africa (SSA) in particular.

Dual infection by SPCSV and SPFMV cause SPVD. SPVD can be devastating, causing up to 98% in susceptible varieties (Fig. 1). The complex genetics and large number chromosomes (90), and the presence of multiple virus infections complicate breeding for virus resistance in sweetpotato using standard breeding and molecular methods.

What objectives did we set?

The major goal is to increase the frequency of SPVD resistance in progeny of breeding populations. We would like to use more accurate and targeted breeding strategies to achieve high levels of resistance in progeny populations and in varieties by increasing the frequency of individuals with SPVD resistance. We would like our breeding methods to be more efficient in producing improved populations with high expression of SPVD resistance alongside other traits such as quality characteristics desired by farmers and consumers.

Where did we work?

Three Sweetpotato Support Platforms (SSPs) were established, with CIP sweetpotato breeders hosted by national breeding programs in Ghana, Mozambique and Uganda. The SSP in Uganda leads the SPVD resistance work in collaboration CIP, Lima and the National Crops Resources Research Institute (NaCRRI) in Uganda (Fig. 2). The SSPs provide technical backstopping in breeding at the sub-regional level for the 14 targeted countries with active sweetpotato breeding activities under the Sweetpotato for Profit and Health Initiative.

Fig 2. Screening for sweetpotato virus disease resistance at Namulonge, Uganda (Credit J. Swanckaert)

What did we achieve during SASHA Phase 2?

We used a combination of different methods focusing on increasing the frequency of resistant genotypes in breeding populations. SPVD resistance has different components, including reduced symptom expression, tolerance (giving reasonable yield despite severe infection and/or high virus titer with or without severe symptoms), recovery (the ability to recover partially or completely from virus symptoms), and reversion (the ability to recover from a particular virus infection). Starting in 2016/2017, we evaluated two populations for three seasons: population Uganda A (Pop Ug A) with 50 genotypes and Pop Ug B with 80 genotypes. These populations had previously been separated by simple sequence repeat markers for SPVD. Progeny from all possible cross combinations (crosses, 80 x 50), 5,031 genotypes in total; and a test cross of 999 genotypes from only SPVD field resistant parents (3 x 5), were also evaluated for three seasons at the Namulonge research station in Uganda, famous for being an in intense SPVD hot spot environment.

Table 1. Comparison of mean scores for SPVD (1=no symptoms; 9=severe symptoms) between the B80 x A50 trial and the test cross 3A x 5B trial.

Progeny populations/T-test	Mean offspring log (SPVD)	Mean offspring SPVD
B80 x A50 trial (5,031 genotypes, progeny of parental population)	1.42	4.40
Test cross 3A x 5B (999 genotypes, progeny of parental population)	1.17	3.39
Difference (T-test)	0.24	1.01
P-value	<0.001	
95% Confidence interval	0.23 - 0.27	

What did we achieve?

The mean SPVD score in the progeny of SPVD resistant x resistant parents increased from 10% in the parental population to 23% (Fig.3), a highly significant increase (Table 1, see P-value).

Two populations from 104 OFSPs x DLP3163 (CIP420269 cross) were phenotyped (characterized) at CIP headquarters in Lima. In the parental material OFSP genotypes, 4 of 104 Jewel descendent clones (3.8%) were resistant to SPCSV, and in the 718 offspring clones, 40 of them (5.7%) were resistant to SPCSV.

Two small sets of clones, each comprising 12 clones, were identified (a resistant set [VJ08.330 x VJ08.330, VJ08.330 x PJ05.064] and susceptible set) and phenotype and marker correlation was conducted with the following results:

- AFLP: E44M34.533, E33M48.460, E36M34.400, E33M48.343, and E39M32.440 bands were absent in the resistant bulk and present in the susceptible bulk; E39M34.156 band was present in the resistant bulk and absent in the susceptible bulk.
- Simple sequence repeat markers identified include: IBS204-172, IBS169-162, Ib-286-125, IbJ559-262, IbJ559-269, IbJ116a-229, and IBS149-225.
- Using DArT markers: 758044, 7563062, 7572542, 753123, and 7574925, 13 associations were found.

We have identified and increased the number of resistant parents for breeding.

- Parents with high SPVD field resistance have been identified and used to generate populations (botanical seed) for national programs for studies to answer and confirm methods used in other crops (heterosis, polycross vs controlled cross, genotyping by sequencing).
- The procedure to discriminate SPVD susceptible, tolerant and resistant genotypes was optimized.

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We still need to build up (increase) the numbers of SPVD resistant genotypes for validation for application of the DNA markers.

Were there any key challenges or lessons learned?

Confirmation of SPVD field resistance requires growing out the target genotypes in the hot spot areas for several seasons because genotype response to SPVD infection is influenced by other factors, such as soil fertility, temperature and rainfall. Because of the uneven distribution of the virus inoculum in the infected sweetpotato plant, confirming resistance requires testing multiple samples from the same plant. Use of molecular markers requires a large number of resistant genotypes to validate and practically apply the markers in sweetpotato breeding.

What is next?

Elimination of poor parents from the breeding populations and testing of the offspring in recurrent selection cycles will be done to increase the frequency of resistant genotypes in the progeny. We hope to use bi-parental elite crosses for generating large numbers of breeding populations for sending to national breeding programs in high SPVD pressure agro-ecologies. We also plan to exploit heterosis in virus resistant parents. We need to build up (increase) numbers of SPVD resistant genotypes for validation for application of DNA markers in sweetpotato.

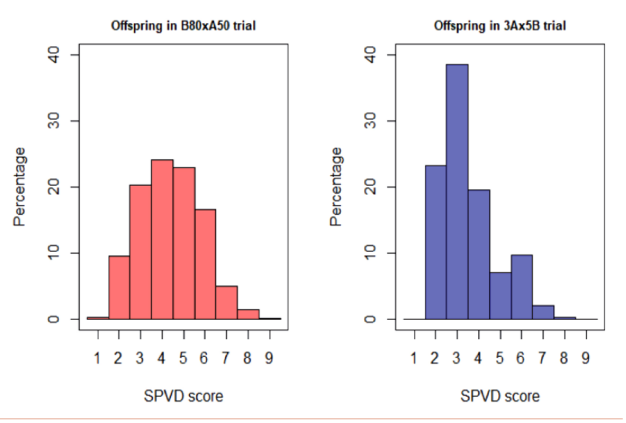

Fig 3. Offspring (left) SPVD score distribution from the B80 x A50 trial (total population) and (right) offspring from the 3A x 5B trial (resistant parents), averaged over seasons 2017B, 2018A and 2018B (A = first season and B = second season); Score ranges from 1 = no symptoms to 9 = severe symptoms (Credit J. Swanckaert).

Partners

The Support Platform (SSP) for Eastern and Central Africa is based at the National Crops Resources Research Institute (NaCRRI) in Uganda and the Kenyan Plant Health Inspection Service (KEPHIS). For Southern Africa, the SSP is based at the Agrarian Research Institute of Mozambique (IIAM) in Maputo. The West Africa platform is located at the Council for Scientific and Industrial Research-Crops Research Institute (CSIR-CRI) in Kumasi, Ghana.

CIP INTERNATIONAL POTATO CENTER
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