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Displacement of US-1 clonal lineage by a new lineage of *Phytophthora infestans* on potato in Kenya and Uganda

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Phytophthora infestans, the pathogen responsible for causing late blight in potato and tomato, is a rapidly evolving and highly adaptable pathogen. Following the appearance of a new clonal genotype in Kenya (KE-1 lineage) in 2007, there was a need to document possible displacement of the US-1 genotype by the newer lineage. Samples from Kenya (260) collected on potato and from Uganda collected on potato (134) and tomato (32) were genotyped with simple sequence repeat (SSR) and mitochondrial DNA haplotype (mtDNA) markers. Results show that, four years after its discovery, the KE-1 lineage has completely displaced the US-1 lineage on potato in Kenya. The KE-1 lineage has also migrated into Uganda through the eastern part of the country that borders western Kenya. All potato samples from eastern Uganda were of the KE-1 lineage. Moreover, samples from eight fields in western Uganda were also of the KE-1 lineage, which is in agreement with an ongoing westward displacement. However, the new lineage has not displaced the old lineage on tomato, as all samples from tomato were US-1. Principal coordinate analysis (PCA) scatter plots revealed clustering that indicated separation between the lineages and also between the subpopulations of the US-1 lineage on potato and tomato.

Keywords: displacement, late blight, population structure, potato, tomato

Introduction

Late blight, caused by the oomycete plant pathogen *Phytophthora infestans*, is a devastating disease of potato and was responsible for the epidemics that led to the Irish potato famine in 1845. The pathogen genome codes for a large number of effector proteins (Haas *et al.*, 2009) and exhibits a very high risk of rapid evolution owing to high mutation rates, large effective populations, a high gene flow, a mixed reproduction system and efficient directional selection (McDonald & Linde, 2002). Populations of *P. infestans* undergo major population shifts in agricultural systems via the successive emergence and migration of asexual lineages (Cooke *et al.*, 2012). Although the phenotypic and genotypic bases of these selective changes are largely unknown, effective management strategies, including the use of host resistance, need to adapt to reflect the changing pathogen population.

Before the 1980s, the global population of *P. infestans* was dominated by a single clonal lineage known as the US-1 'old' population (Goodwin *et al.*, 1994). However, this changed as a result of pathogen migrations, believed to have arisen in 1976 following the importation from Mexico into Europe of potatoes that contained a population of *P. infestans* of diverse mating types and with novel alleles (Fry *et al.*, 1993, 2009; Goodwin & Drenth, 1997). Because Europe exports seed tubers throughout the world, the new genotypes of *P. infestans* were widely distributed in Europe and subsequently to South America, North Africa and Asia. Sexual reproduction has been shown to occur in several parts of Europe, e.g. in Scandinavia (Andersson *et al.*, 1998; Widmark, 2010), and both mating types have been reported to occur elsewhere, for example in India (Chimote *et al.*, 2010) and the UK (Cooke *et al.*, 2003).

In North Africa, the A1 and A2 mating types have been reported in Algeria, Egypt and Morocco (Sedegui *et al.*, 2000; Hammi *et al.*, 2001; Corbière *et al.*, 2010). Less is known about the *P. infestans* population in East and Southeast Africa. The earliest reports involving marker data indicate that some isolates collected in Rwanda were not US-1 lineage (Forbes *et al.*, 1998; Gavino & Fry, 2002). Studies conducted in late 1990s in South Africa found the population from varied hosts (tomato, potato, petunia) to be exclusively US-1 (McLeod *et al.*, 2001). In the eastern and western highlands of Uganda, potato isolates collected in 2001 were also found to be US-1 lineage (Ochwo *et al.*, 2002), as were isolates collected in Kenya and Uganda from tomato and potato in 1995–97 (Vega-Sánchez *et al.*, 2000). Thus, excluding the study from Rwanda, the US-1 lineage and its variants have been the dominant genotypes in Kenya, Uganda and South Africa until very recently.

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There is less information about the *P. infestans* population in the Horn of Africa. If the US-1 lineage were dominant at one time, it must have been displaced more than a decade ago: Schiessendoppler & Molnar (2002) found no US-1 isolates in Ethiopia, but instead found a population characterized by the mitochondrial (mt) DNA haplotype Ia.

The appearance of a new lineage in Kenya, KE-1, was first detected in 2007; isolates have a unique glucose-6-phosphate isomerase genotype (90/100), some private simple sequence repeat (SSR) alleles, mtDNA haplotype Ia and a unique restriction fragment length polymorphism (RFLP) fingerprint with probe RG-57 (Pule *et al.*, 2013). In the 2007 survey of eight African countries (Burundi, Kenya, Malawi, Mozambique, Rwanda, South Africa, Tanzania and Uganda), only two fields in Kenya had the new KE-1 lineage; all the other isolates were of the US-1 lineage that, as noted above, was dominant in eastern sub-Saharan Africa. Another survey in 2009 also identified isolates belonging to a new European lineage, 2_A1, with the new lineage found on more farms than the US-1 lineage (Were *et al.*, 2013). Both Were *et al.* (2013) and Pule *et al.* (2013) found at least one field with a mixture of the US-1 and new lineages. Markers used in these two studies did not permit an assessment of whether KE-1 and 2_A1 were the same lineage, but they were found in nearby locations.

This current study was carried out to test the hypothesis that displacement of US-1 by one or more new lineages is ongoing in Kenya and Uganda. An additional objective was to provide an initial map of the major lineages of *P. infestans* on potato and tomato in Kenya and Uganda.

Materials and methods

Field sampling

Leaves with young sporulating single lesions of late blight were sampled between November 2011 and July 2012 from farmers' fields in the potato-growing areas of Uganda and Kenya. In Kenya, collections were made from 65 fields in seven districts with four samples taken per field. Fields were chosen based on reported locations where the new lineage was previously found as well as areas with extensive potato farming. Moreover, multiple samples per field were collected to detect lineage mixtures or within-lineage variants occurring within a single field. No tomato samples were obtained from Kenya because tomato was not found growing in the areas visited during the survey.

In Uganda, a more extensive sampling approach was taken to be able to cover all production areas in the east and west parts of Uganda, bordering Kenya and Rwanda, respectively. One sample per field was collected from 166 fields in 15 districts, of which 32 samples were tomato.

DNA for fingerprinting was collected by squashing individual leaflets with freshly sporulating lesions on FTA cards (Whatman; Li *et al.*, 2013). To do this, each lesion-containing leaflet was placed between a piece of Parafilm and the appropriate section of the card with the back of the leaflet facing towards the card. The card was placed on a small plastic board in the field and the sample rubbed with a pestle until sap soaked through to the back of the card. The cards were allowed to air dry for at least 5 min before storage. The materials (scissors, crushing board and pestle) were wiped with 70% ethanol between each sample to avoid cross-contamination and a new piece of Parafilm was used for each lesion.

DNA extraction

The FTA cards were stored at room temperature for between 2 and 10 months after samples were collected before DNA extraction was carried out. A single 6 mm disc was punched out from the dry sample spot and transferred to a 1·5 mL microfuge tube. To ensure that no residue was carried over to the next sample, the punch tip was rinsed in 70% ethanol, wiped with a paper towel and punched through unused chromatography paper. After adding 400 μL FTA purification reagent (Whatman) to each tube, the tubes were vortexed and the samples incubated for 4 min at room temperature. The FTA purification reagent was then discarded and the FTA purification reagent wash was repeated once. The disc was then rinsed twice with 400 μL $T_{10}E_{0.1}$ buffer (10 mM Tris, 0·1 mM EDTA, pH 8) in a similar manner. Using a pipette tip, the disk was transferred to a 0·5 mL microfuge tube and 80 μL $T_{10}E_1$ buffer (10 mM Tris, 1 mM EDTA, pH 8) was added. After centrifugation, the disc was incubated in the buffer for 5 min at 95°C, cooled on ice and the tube (containing the buffer and disc) was stored at −20°C.

Microsatellite (SSR) analysis

Eight polymorphic microsatellite loci were chosen from previously published markers and PCR was conducted in separate reactions for each locus. The markers used were Pi4B, PiG11 (Knapova & Gisi, 2002), Pi04, Pi16, Pi56, Pi70, Pi89 and D13 (Lees *et al.*, 2006). The forward primers for loci PiG11 and Pi70 were labelled at the 5′ end with the fluorescent dye NED, while Pi04, Pi16, Pi56, Pi89, Pi4B and D13 were 5′-labelled with FAM.

PCR was performed in a 15 μL volume containing 3 μL of the solution eluted from the FTA cards, 1 × PCR green buffer, 0·2 mM each dNTP, 2 mM $MgCl_2$, 0·3 μM each forward and reverse primer, and 0·36 U DreamTaq (Fermentas). Amplifications were performed in a PTC200 thermocycler (MJ Research) under the following conditions: initial denaturation at 94°C for 2 min; followed by 33 cycles of 30 s at 94°C, 30 s at 62°C (Pi04, PiG11), 60°C (Pi16), 58°C (Pi56, Pi70, Pi89, Pi4B) or 50°C (D13), 45 s at 72°C; and finished with an elongation cycle of 25 min at 72°C.

The PCR amplification products were diluted with distilled deionized water to approximately 10 ng μL^{-1}. Sample dilution depended on band intensities that were visually estimated on 1·5% agarose gels. The lengths of SSR amplicons were determined using an ABI 3730xl DNA Analyzer (Uppsala Genome Center at Rudbeck Laboratory, Uppsala University, Sweden). The peak positions of the SSR alleles were scored using the software GENEMARKER (Soft Genetics) to identify polymorphisms. Allele sizes were calibrated to reference DNA alleles kindly provided by David Cooke of The James Hutton Institute, UK.

Mitochondrial DNA haplotype analysis

In total, 250 lesion samples were genotyped for mtDNA haplotype using PCR-RFLP analysis, as described by Griffith & Shaw (1998) with minor modifications. Two primer pairs (F2/R2 and

F4/R4) were used. Amplification was as follows: final concentrations of 1 × Green buffer, 0·1 mM dNTPs each, 1·5 mM $MgCl_2$, 0·32 μM of each forward and reverse primer and 1·5 U DreamTaq, in 25 μL final volume. The PCR conditions were: 1 cycle of 94°C for 3 min; 30 cycles of 94°C for 30 s, 62°C for 30 s and 72°C for 60 s; followed by a final extension at 72°C for 5 min. Amplified PCR products (10 μL) of the primer pair F2/R2 and F4/R4 were cleaved with *Msp*I and *Eco*RI restriction endonucleases, respectively. The resultant DNA fragments were separated by agarose gel electrophoresis to discriminate between the Ia and Ib haplotypes. Electrophoresis was carried out for 3 h in TAE (Tris–acetate–EDTA) buffer in a horizontal 2% agarose gel containing GelGreen stain (Biotium; 1 μL/50 mL).

Data analysis

Seven of the eight SSR markers produced two alleles at each locus of the *P. infestans* samples. The other marker (Pi04) generated three alleles, of which two were common to all samples and were consequently included with the other SSR markers used for genetic analyses described below to identify multilocus genotypes (MLGs).

Analysis of molecular variance (AMOVA) was conducted in GenAlEx v. 6.5 (Peakall & Smouse, 2006, 2012) based on clone-corrected SSR data to avoid over-representation of frequently occurring clones. This analysis was used to test for population differentiation based on the pairwise comparisons of KE-1 subpopulations in Uganda and Kenya, and US-1 subpopulations on potato and tomato in Uganda. A principal coordinate analysis (PCA) was performed from a genetic distance matrix generated in GenAlEx v. 6.5 based on the SSR data to reveal clustering of genotypes.

Results

The 426 *P. infestans* samples genotyped from both Kenya and Uganda were classified into 80 unique MLGs (Table 1), 50 of which were represented by a single sample (Table S1). Nonetheless, based on mtDNA haplotype (either Ia or Ib) and SSR genotype, all samples were classified into one of two lineages: US-1 or KE-1 (Table 1).

All potato samples from Kenya and eastern Uganda were KE-1; potato samples that were US-1 lineage were only found in western Uganda (Fig. 1). All samples taken from tomato were US-1, even in eastern Uganda.

All the putative KE-1 samples collected on potato from the two countries had a Ia mtDNA haplotype while all the US-1 samples from both potato and tomato in Uganda had a 1b mtDNA haplotype.

Table 1 Clonal lineage and number of SSR multilocus genotypes (MLGs) of *Phytophthora infestans*

		Lineage					
		KE-1		US-1		Total	
Country	Host	*n*	MLG	*n*	MLG	*n*	MLG
Kenya	Potato	260	16	0	0	260	16
Uganda	Potato	58	9	76	38	134	47
	Tomato	0	0	32	27	32	27
	Potato and tomato	58	9	108	60	166	69
Total		318	20	108	60	426	80

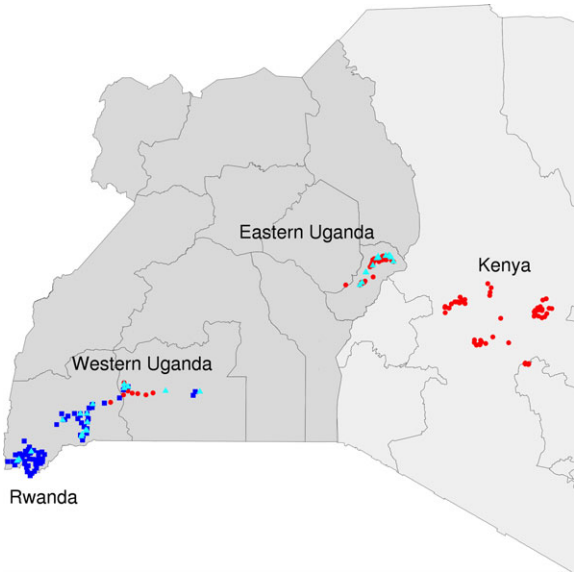

Figure 1 The *Phytophthora infestans* population in Kenya and Uganda showing occurrence and distribution of US-1 and KE-1 lineages. Red circle = KE-1 from potato; blue square = US-1 from potato; cyan triangle = US-1 from tomato.

PCA of the SSR data confirmed separation of the KE-1 and US-1 lineages, and also separated US-1/potato from US-1/tomato genotypes (Fig. 2). Several putative KE-1 genotypes that did not cluster with any of the groups were also identified. An AMOVA based on SSR data indicated a significant difference between US-1/potato and US-1/tomato subpopulations ($P = 0{\cdot}002$; Table 2) and was thus consistent with the PCA.

Based on the frequency of MLGs (Table 1), it was clear that there is more diversity within the US-1 lineage than within the KE-1 lineage. A total of 108 US-1 samples from potato and tomato in Uganda produced 60 MLGs, while 318 KE-1 samples from potato in both Kenya and Uganda produced only 20 MLGs (Table 1). An AMOVA based on SSR data from KE-1 samples from Kenya and Uganda found no evidence for geographic substructuring between the two countries ($P = 0{\cdot}232$; Table 3).

Discussion

This study used two genetic markers, microsatellite fingerprinting and mtDNA haplotypes, to demonstrate that the *P. infestans* population in East Africa has changed since the pathogen population study in 2007 (Pule *et al.*, 2013), in which the US-1 lineage was dominant throughout sub-Saharan Africa except in two fields, one in western and another in central Kenya, where the new KE-1 lineage was found. A comparison of the SSR data from the current study with that of Pule *et al.* (2013) indicates that the new lineage in the current study is the same as

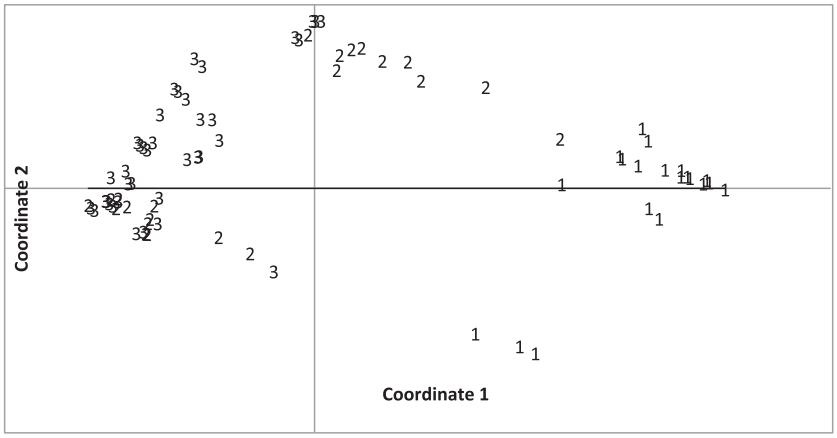

Figure 2 Principal coordinate analysis (PCA) based on the calculated genetic differences of SSR multilocus genotypes of *Phytophthora infestans* samples. 1 = lineage KE-1 from potato (Kenya and Uganda); 2 = lineage US-1 from tomato (Uganda); 3 = US-1 from potato (Uganda).

Table 2 Analysis of molecular variance (AMOVA) based on eight SSR loci for US-1 lineage *Phytophthora infestans* samples collected from tomato or potato plants in Uganda

Source	df	SS	MS	Est. Var.	% Var.	*P*
Between hosts	1	9·894	9·894	0·263	9	0·002
Within hosts	58	148·756	2·565	2·565	91	
Total	59	158·65		2·828	100	

df, degrees of freedom, SS, sums of squares; MS, mean squares; Est. Var., estimated variance within and among populations; % Var., percentage of estimated variance within or among populations; *P*, probability of observing the data assuming there is no differentiation.

Table 3 Analysis of molecular variance (AMOVA) based on eight SSR loci for KE-1 lineage *Phytophthora infestans* samples collected from potato plants in Kenya and Uganda

Source	df	SS	MS	Est. Var.	% Var.	*P*
Between countries	1	2·998	2·998	0·072	3	0·232
Within countries	18	41·202	2·289	2·289	97	
Total	19	44·2			100	

df, degrees of freedom; SS, sums of squares; MS, mean squares; Est.Var., estimated variance within and among populations; % Var., percentage of estimated variance within or among populations; *P*, probability of observing the data assuming there is no differentiation.

KE-1 (data not presented). Within the 4 years between the sampling of Pule *et al.* (2013) and the current study, displacement of US-1 on potato by KE-1 appears to have been more or less complete in Kenya and eastern Uganda; no US-1 was found on potato in these regions.

The fact that a mixture of lineages was found on potato in midwestern Uganda, but only US-1 was found on potato in southwestern Uganda, suggests that the current study represents a snapshot of KE-1 in a process of migration toward the west. Future sampling in Uganda would enable a test of this hypothesis. As KE-1 was not found in Tanzania or Rwanda by Pule *et al.* (2013), it would also be useful to sample there to monitor the spread of the lineage.

While it is impossible from the current study to know which fitness advantages KE-1 has over US-1 to explain the displacement, it is noteworthy that displacement of US-1 in the past by other lineages has frequently been associated with increased difficulty in disease management (Cooke *et al.*, 2011). It is possible that fungicide resistance might have played a role in the KE-1 spread, but there is no evidence to support this hypothesis. Furthermore, at least one previous study demonstrated high frequencies of metalaxyl resistance in the US-1 lineage in the region (Mukalazi *et al.*, 2001).

The results of the present study are consistent with an earlier study in the region demonstrating host specialization on potato and tomato of the *P. infestans* US-1 population (Vega-Sánchez *et al.*, 2000): the SSR data separated the potato and tomato US-1 subpopulations found in Uganda. In the current study there was no indication that KE-1 is displacing US-1 on tomato. Displacement of US-1 on potato but not tomato has been noted in other regions (Oyarzun *et al.*, 1998; Ghimire *et al.*, 2003).

Although this study was not designed to determine the origin of KE-1, some speculation is possible. At the onset of the study, it was not clear whether KE-1 found by Pule *et al.* (2013) was the same lineage as found in 2009 in the same region by Were *et al.* (2013), which was subsequently identified as identical to 2_A1, an old European lineage. However, recent analyses in the authors' laboratory and by A. K. Lees (The James Hutton Institute, UK, personal communication) indicate that 2_A1 and KE-1 are the same lineage, indicating a possible European origin for KE-1.

Genotypes similar to some variants of KE-1 have been found in Ethiopia (D. Shimelash, Hiramaya University, Ethiopia, personal communication) and KE-1 shows limited similarity to some isolates found in the 1990s in Rwanda (Pule *et al.*, 2013). Although KE-1 was originally reported from only two fields, Pule *et al.* (2013) found unexpected variation for a recent clonal introduction, with at least seven genotypes identified from 15 isolates collected. These observations indicate that the question of when, where and how the KE-1 was introduced into the region remains unanswered.

Despite the uncertainty concerning the origin of KE-1 noted above, the data in the current study indicated that the present KE-1 population is less diverse than the US-1 population. This implies that US-1 has been evolving in the region significantly longer than KE-1.

The two genetic markers used in this study clearly differentiated KE-1 from the US-1 lineage. From the eight SSR markers used, all putative KE-1 samples had two private alleles: 193 at locus Pi70 and 172 at locus Pi56 (Table S1). These alleles may prove useful as simple markers in studies to simply distinguish between the two lineages. All KE-1 samples were mtDNA haplotype Ia, which differentiated them from the US-1 lineage, which was mtDNA haplotype Ib. Both lineages are of the A1 mating type as earlier reported by Pule *et al.* (2013), indicating that, although displacement is occurring, there is no evidence for sexual recombination in the region.

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References

Andersson B, Sandström M, Strömberg A, 1998. Sexual reproduction of *Phytophthora infestans* on potato in Sweden. In: Schepers H, Bouma E, eds. *Proceedings of the Workshop on the European Network for Development of an Integrated Control Strategy of Potato Late Blight, 24–27 September 1997, Carlow, Ireland, PAV-Special Report No. 3*. Lelystad, Netherlands: Applied Research for Arable Farming and Field Production of Vegetables, 92–6.

Chimote VP, Kumar M, Sharma PK, Singh PH, Singh BP, 2010. Characterization of changes in phenotype and genotype of *Phytophthora infestans* isolates from India. *Journal of Plant Pathology* **92**, 669–77.

Cooke DEL, Young V, Birch PRJ *et al.*, 2003. Phenotypic and genotypic diversity of *Phytophthora infestans* populations in Scotland (1995–97). *Plant Pathology* **52**, 181–92.

Cooke LR, Schepers HTAM, Hermansen A *et al.*, 2011. Epidemiology and integrated control of potato late blight in Europe. *Potato Research* **54**, 183–222.

Cooke DEL, Cano LM, Raffaele S *et al.*, 2012. Genome analyses of an aggressive and invasive lineage of the Irish potato famine pathogen. *PLoS Pathogens* **8**, e1002940.

Corbière R, Rekad FZ, Galfout A, Andrivon D, Bouznad Z, 2010. Phenotypic and genotypic characteristics of Algerian isolates of *Phytophthora infestans*. *PPO-Special Report* **14**, 133–46.

Forbes GA, Goodwin SB, Drenth A, Oyarzun P, Ordoñez ME, Fry WE, 1998. A global marker database for *Phytophthora infestans*. *Plant Disease* **82**, 811–8.

Fry WE, Goodwin SB, Dyer AT *et al.*, 1993. Historical and recent migrations of *Phytophthora infestans*: chronology, pathways, and implications. *Plant Disease* **77**, 653–61.

Fry WE, Grünwald NJ, Cooke DEL, McLeod A, Forbes GA, Cao K, 2009. Population genetics and population diversity of *Phytophthora infestans*. In: Lamour K, Kamoun S, eds. *Oomycete Genetics and Genomics: Diversity, Interactions and Research Tools*. Hoboken, NJ, USA: John Wiley & Sons Inc, 139–64.

Gavino PD, Fry WE, 2002. Diversity in and evidence for selection on the mitochondrial genome of *Phytophthora infestans*. *Mycologia* **94**, 781–93.

Ghimire SR, Hyde KD, Hodgkiss IJ, Shaw DS, Liew ECY, 2003. Variations in the *Phytophthora infestans* population in Nepal as revealed by nuclear and mitochondrial DNA polymorphisms. *Phytopathology* **93**, 236–43.

Goodwin SB, Drenth A, 1997. Origin of the A2 mating type of *Phytophthora infestans* outside Mexico. *Phytopathology* **87**, 992–9.

Goodwin SB, Cohen BA, Fry WE, 1994. Panglobal distribution of a single clonal lineage of the Irish potato famine fungus. *Proceedings of the National Academy of Sciences, USA* **91**, 11591–5.

Griffith GW, Shaw DS, 1998. Polymorphisms in *Phytophthora infestans*: four mitochondrial haplotypes are detected after PCR amplification of DNA from pure cultures or from host lesions. *Applied and Environmental Microbiology* **64**, 4007–14.

Haas BJ, Kamoun S, Zody MC *et al.*, 2009. Genome sequence and analysis of the Irish potato famine pathogen *Phytophthora infestans*. *Nature* **461**, 393–8.

Hammi A, Msatef Y, Bennani A, El Ismaili A, Serrhini MN, 2001. Characterization of populations of *Phytophthora infestans* (Mont.) de Bary in Morocco using aggressiveness, mating type and metalaxyl resistance. *Journal of Plant Pathology* **83**, 226.

Knapova G, Gisi U, 2002. Phenotypic and genotypic structure of *Phytophthora infestans* populations on potato and tomato in France and Switzerland. *Plant Pathology* **51**, 641–53.

Lees AK, Wattier R, Shaw DS, Sullivan L, Williams NA, Cooke DEL, 2006. Novel microsatellite markers for the analysis of *Phytophthora infestans* populations. *Plant Pathology* **55**, 311–9.

Li Y, Cooke DEL, Jacobsen E, van der Lee T, 2013. Efficient multiplex simple sequence repeat genotyping of the oomycete plant pathogen *Phytophthora infestans*. *Journal of Microbiological Methods* **92**, 316–22.

McDonald BA, Linde C, 2002. Pathogen population genetics, evolutionary potential, and durable resistance. *Annual Review of Phytopathology* **40**, 349–79.

McLeod A, Denman S, Sadie A, Denner FDN, 2001. Characterization of South African isolates of *Phytophthora infestans*. *Plant Disease* **85**, 287–91.

Mukalazi J, Adipala E, Sengooba T, Hakiza JJ, Olanya M, Kidanemariam HM, 2001. Metalaxyl resistance, mating type and pathogenicity of *Phytophthora infestans* in Uganda. *Crop Protection* **20**, 379–88.

Ochwo MKN, Kamoun S, Adipala E, Rubaihayo PR, Lamour K, Olanya M, 2002. Genetic diversity of *Phytophthora infestans* (Mont.) de Bary in the Eastern and Western Highlands of Uganda. *Journal of Phytopathology* **150**, 541–2.

Oyarzun PJ, Pozo A, Ordoñez ME, Doucett K, Forbes GA, 1998. Host specificity of *Phytophthora infestans* on tomato and potato in Ecuador. *Phytopathology* **88**, 265–71.

Peakall R, Smouse PE, 2006. GenAlEx 6: genetic analysis in Excel. Population genetic software for teaching and research. *Molecular Ecology Notes* **6**, 288–95.

Peakall R, Smouse PE, 2012. GenAlEx 6.5: genetic analysis in Excel. Population genetic software for teaching and research – an update. *Bioinformatics* **28**, 2537–9.

Pule BB, Meitz JC, Thompson AH *et al.*, 2013. *Phytophthora infestans* populations in central, eastern and southern African countries consist of two major clonal lineages. *Plant Pathology* **62**, 154–65.

Schiessendoppler E, Molnar O, 2002. Characterization of *Phytophthora infestans* populations in sub-Saharan Africa as a basis for simulation modeling and integrated disease management. In: Lizárraga C, ed. *Late Blight: Managing the Global Threat, Proceedings of the Global Initiative on Late Blight Conference, 11–13 July, Hamburg, Germany*. Lima, Peru: International Potato Center, 140.

Sedegui M, Carroll RB, Morehart AL *et al.*, 2000. Genetic structure of the *Phytophthora infestans* population in Morocco. *Plant Disease* **84**, 173–6.

Vega-Sánchez ME, Erselius LJ, Rodriguez AM *et al.*, 2000. Host adaptation to potato and tomato within the US-1 clonal lineage of *Phytophthora infestans* in Uganda and Kenya. *Plant Pathology* **49**, 531–9.

Were HK, Kabira JN, Kinyua ZM *et al.*, 2013. Occurrence and distribution of potato pests and diseases in Kenya. *Potato Research* **56**, 325–42.

Widmark AK, 2010. *The Late Blight Pathogen,* Phytophthora infestans. *Interaction With the Potato Plant and Inoculum Sources*. Uppsala, Sweden: Swedish University of Agricultural Sciences, PhD thesis.

Supporting Information

Additional Supporting Information may be found in the online version of this article at the publisher's web-site.

Table S1. The different SSR multilocus genotypes identified in KE-1 and US-1 lineages of *Phytophthora infestans* isolates in Kenya and Uganda.