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Spreading and global pathogenic diversity of sunflower downy mildew – Review

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Abstract: Since almost a century, sunflower cultivation is endangered by *Plasmopara halstedii* (Farlow) Berlese & de Toni, a biotrophic oomycete causing downy mildew symptoms. The pathogen has conquered four of the five continents, and through high genetic plasticity recurrently avoided being reliably controlled by the introduction of resistant host cultivars in sunflower production. This paper attempts to retrace the historic routes of sunflower downy mildew spreading from its North American origin into Europe, South America, Asia and Africa. An update of the global diversity of pathotypes will be provided and critically discussed. Finally, the limits of the currently applied bioassay-based techniques for diversity assessment are pointed out and an alternative for continuous and area-wide monitoring is discussed.

Keywords: *Plasmopara halstedii*; sunflower downy mildew; pathotype diversity

Sunflower downy mildew (SDM) is one of more than 30 severe diseases attacking *Helianthus annuus* Linnaeus, one of the world's most important oil crops (Zimmer & Hoes 1978; Virányi 1992; Gulya *et al.* 1997). Despite intensive research, resistance breeding and fungicide application over more than 50 years, the responsible pathogen has not been brought under control. The disease is easily recognized by phenotypic symptoms such as severe stunting of plants, chlorotic appearance of infected leaves and white layers of sporangia protruding from the stomata of the lower leaf surface. It is caused by a highly specialized biotrophic oomycete of the Peronosporomycetidae, usually known as *Plasmopara halstedii* (Farlow) Berlese & de Toni. However, it should be mentioned that the taxonomic validity of this name has yet to be approved, as the original name was given by Farlow (1883) to a downy mildew pathogen initially found on *Eupatorium purpureum* Linnaeus and not on sunflower [for details see review of Virányi and Spring (2011)]. The first infection of a downy mildew pathogen on annual sunflower *H. annuus* was reported in 1888 (Farlow & Seymour 1890) and it was not until the 1920s that *P. halstedii* became a serious threat to sunflower cultivation in the U.S. (Henry & Gilbert 1924; Young & Morris 1927). Despite the taxonomic uncertainties, the name *P. halstedii* will be used here for the pathogen causing SDM. However, it will be used in the sense of a narrow species concept which excludes downy mildew pathogens on other genera of Asteraceae.

Origin, spreading and global distribution

There is a general consensus that the SDM pathogen originated from North America where its host *H. annuus* is a native floral element (Sackston 1992). However, reports on the history of spreading of the pathogen into the world's sunflower cultivation areas are fragmentary and inconsistent. This is due to contradictory species concepts for *P. halstedii* pursued in the early compilations published by Leppik (1962, 1966) and Novotelnova (1966). While Leppik (1966) – in contrast to many taxonomists and to the current state of art (Index Fungorum, http://www.indexfungorum.

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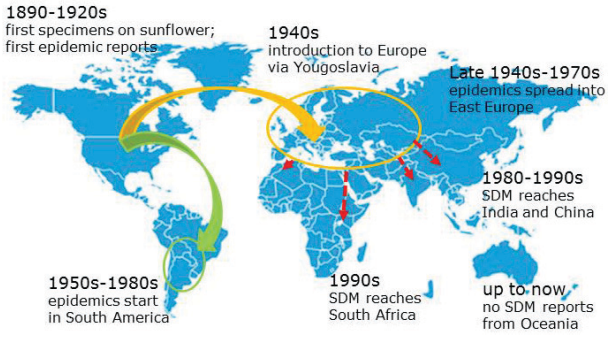

Figure 1. Origin and current distribution of sunflower downy mildew

org/Names/Names.asp) – propagated an extremely broad species concept which included downy mildew pathogens on a host of over 80 different Asteraceae genera, Novotelnova (1966) pled for a separation of the pathogen on *H. annuus* as new species under the name *Plasmopara helianthi* Novotel'nova. Another problem is that many early records of infection are scientifically imprecise with respect to locality, sampling date and pathogen identification. Tracing reports on such "observations" in literature is sometimes very difficult and often, no specimens were deposited for checking the records. This impedes the reconstruction of routes of distribution by means of genetic fingerprints as has been achieved, for example, for the late blight of potato, *Phytophthora infestans* (Montagne) de Bary (Yoshida *et al.* 2014).

Regarding the distribution of downy mildew on cultivated sunflower, it appears undisputed that a first wave of migration (Figure 1) occurred in the 1940s – for refs. see Novotelnova (1966), when the pathogen was recognized in sunflower fields of former Yugoslavia (1946 in Croatia, 1946 in Serbia). Afterwards, it rapidly invaded other Eastern European countries such as Romania (1946), Bulgaria (1947), Hungary (1949), Russia (1951) etc. (Table 1). Adjacent countries in Central and South Europe, Asia and North Africa were affected in the 1970s–1990s, when sunflower started to become a major oil crop in these areas as well, but precise data on first occurrences are often difficult to access.

A second route of long distance dispersal occurred in South America, where *P. halstedii* started to endanger sunflower production in Chile (Sackston 1956) and Argentina (Pontis *et al.* 1959) in the 1950s. Outbreaks in adjacent countries such as Uruguay, Brazil and Paraguay followed.

Meanwhile, SDM has been reported from over 50 countries (Virányi 2018) including four of the five continents. The number of reports is highest in Europe (26 countries), followed by Asia (13), Africa (8), South America (5) and North America (3). Downy mildew pathogens on Asteracea species of *Arctotheca* J.C. Wendland, *Arctotis* Linnaeus and *Sphagneticola* O. Hoffmann in Australia and New Zeeland which, according to the former broad species concept would have been also merged in *P. halstedii*, have recently been classified as independent species *Plasmopara majewskii* Constantinescu & Thines (Constantinescu & Thines 2010) and *Plasmopara sphagneticolae* McTaggart & R.G. Shivas (McTaggart *et al.* 2015). Indeed, no SDM infection has been reported from sunflower cultivation of Oceania to date.

The long distance dispersal of SDM almost certainly occurs through the exchange of oospore-contaminated seeds (Cohen & Sackston 1974; Spring 2001). Such seed import was either used by farmers to achieve higher yields with new cultivars or by breeders to broaden their gene pool for breeding. In particular, the option to propagate two generations per year when translocating newly developed cultivars from the northern to southern hemisphere and back significantly accelerated seed exchange from field to field. Hence, it is not surprising that introductions of SDM are not singular events, but became regular processes in the context of global markets. Delmotte *et al.* (2008) showed with molecular genetic methods that *P. halstedii* in France resulted from at least three independent introductions. Possibilities to avoid introduction of *P. halstedii* or phenotypes of new virulence of the pathogen via seed transmission with the implementation of contamination testing are limited (Virányi & Spring 2011). Either such tests are too labour intensive and time consuming or not sensitive enough (Ioos *et al.* 2007). Only Australia and New Zeeland, employing rigorous import restrictions, could so far keep their sunflower production free of SDM.

Global pathogenic diversity of *P. halstedii* in cultivated sunflower

With respect to resistance breeding and quarantine measures, it is essential to know the virulence diversity within the pathogen population of SDM. First differentiation started in the 1970s, when resistance from wild sunflower was implemented in the sunflower line RHA266 after the first resistance gene *Pl1* was defined (Vranceanu & Stoenescu

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Table 1. Reported global distribution of *Plasmopara halstedii* (Farlow) Berlese & de Toni on cultivated sunflower

Continent	Country	Date	Source
Africa	Egypt	~1988	CMI (1988) (doubtful source according to EPPO global database: https://gd.eppo.int/taxon/PLASHA/distribution/)
	Ethiopia	~1986§	Weldekidan (1986)
	Kenya	~1992	EPPO global database (1992)
	Morocco	1971	Achbani *et al.* (2000)
	South Africa	1993	Viljoen *et al.* (1997)
	Tunisia	~2014	Virányi (2018)
	Uganda	1988	EPPO global database (1992)
	Zimbabwe	1988	Gulya (1996)
North America	Canada	1893	[Ellis Collection # 2758952 NY Botanical Garden], Bisby *et al.* (1938)
	Dominican Republic	*	Leppik (1962)
	Mexico	~1976	Fucikovsky-Zak (1976)
	USA	1888	Farlow and Seymour (1890)
South America	Argentina	1958	Pontis *et al.* (1959)
	Brazil	1982	Ferreira *et al.* (1983)
	Chile	1954	Sackston (1956)
	Paraguay	~1993	EPPO global database (1993)
	Uruguay	1956	Sackston (1957)
Asia	Azerbaijan	~2014	EPPO global database (2014)
	China	1985	Yang *et al.* (1988)
	Georgia	1972	Machavariani (1972)
	India	1986	Mayee and Patil (1986)
	Iran	1973§	Rahmani and Madjidieh-Ghassemi (1975)
	Iraq	~1988§	EPPO global database (1992)
	Israel	~2011§	EPPO global database (2011)
	Japan	~1988	EPPO global database (1992)
	Jordan	~1960§	Leppik (1962)
	Kazakhstan	~1988	Mikhailova (1988)
	Korea	1991	Shin (1991)
	Myanmar	~2015	EPPO global database (2015)
	Pakistan	~1992§	EPPO global database (1992)
Europe	Albania	~1980	Kola (1980)
	Austria	~1993	EPPO global database (1993)
	Bosnia and Herzegovina	~1973	Batinica *et al.* (1973)
	Bulgaria	~1947	Novotelnova (1966)
	Croatia	~1946	Novotelnova (1966)
	Czech Republic	~1954	Novotelnova (1966)
	Estonia	~1992	EPPO global database (1992)
	France	1966	Delanoe (1972)
	Germany	1986	Spring *et al.* (1991)
	Greece	1991	Thanassoulopoulos and Mappas (1992)
	Hungary	~1949	Novotelnova (1966)
	Italy	~1983	Zazzerini (1983)
	Moldova	~1948	Novotelnova (1966)

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Table 1. to be continued

Continent	Country	Date	Source
	Montenegro	~1992	Masirevic (1992)
	Netherlands	1997	EPPO global database (2018)
	Poland	~1976	Kucmierz (1976)
	Portugal	2016	EPPO global database (2017)
	Romania	~1951	Savulescu and Savulescu (1951)
	Russia	~1951	Novotelnova (1966)
Europe	Serbia	~1947	Novotelnova (1966)
	Slovakia	~1994	EPPO global database (1994)
	Slovenia	~2017	EPPO global database (2017)
	Spain	~1982	Melero-Vara *et al.* (1982)
	Switzerland	1997	Gindrat *et al.* (1997)
	Ukraine	~1949	Bogovik (1953)
	Turkey	~1958	Karel (1958)

~ date of first literature report, no exact date of first observation, defined by locality or specimen deposition; § – presence dated before 1960 according to a seed screening trial in Iowa mentioned by Leppik (1962); *a report of sunflower downy mildew from Dominica by Gomez-Menor (1936) could not be verified so far

1970). This allowed, for the first time, differentiation between the "old" European race (= US race 1) and the new US race 2 (red river race = later on French race D) (Zimmer 1974). Successive discovery of new resistance genes allowed further differentiation of the pathogenic diversity in populations of *P. halstedii* and until 1990 this bioassay-based system using hosts with defined resistance afforded 11 virulence phenotypes in the US and 4 in France (Table 2).

Table 2. Virulence phenotypes according to different classification systems according to Sackston *et al.* (1990), Gulya *et al.* (1991b), and Tourvieille de Labrouhe *et al.* (2000)

US system	French system	International pathotype system
Race 1 (former European race)		pathotype 100
Race 2 (former Red River race)	French race D	pathotype 300
Race 3	French race C	pathotype 700
Race 4		pathotype 730
Race 5		pathotype 770
Race 6		pathotype 310
Race 7		pathotype 330
Race 8	French race A	pathotype 710
Race 9		pathotype 330
Race 10	French race B	pathotype 703
Race 11		pathotype 711

However, the methodology for the infection bioassays was not uniform and comparability of testing results was problematic. This was overcome when an initiative of scientists suggested a new system for virulence phenotyping. It was based on standardized inoculation and evaluation methods (Gulya *et al.* 1991a) and included the use of homozygous sunflower lines with defined *Pl* genes (Gulya 1995; Gulya *et al.* 1998). Susceptibility or resistance of nine sunflower genotypes (differentials) in three test sets (each with 3 differentials) resulted in a three digit coding system for so-called pathotypes [virulence phenotypes; for definition of the terms and differentiation against races see Spring *et al.* (2018)] ranging from virulence value 100 (infection on first differential in set I) to 777 (infection on all three differentials in each of the three sets). This system, although now slightly modified and expanded to 15 genotypes (Tourvieille de Labrouhe *et al.* 2012), is generally used and has allowed assessment of the pathogenic diversity of SDM in different regions of the world.

Within the past 20 years, the three digit pathotyping has been applied to field isolates of nearly 20 countries. Two reviews have previously summarized the global pathotype inventory up to the year 2006 (Gulya 2007) and 2014 (Virányi *et al.* 2015), respectively. Because some of the published data are incongruent (e.g. pathotypes 500–560) and several new highly aggressive pathotypes have been reported since then, an update of the current pathotype diversity is provided in Table 3.

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Table 3. Global pathotype diversity of *Plasmopara halstedii* (Farlow) Berlese & de Toni on cultivated sunflower according to literature reports before 2007 [x – Gulya (2007)], 2015 [y – Virányi *et al*. (2015)] and new reports after 2015 (z)

Type	North America		South America	Africa		Asia			Europe										
	Ca	US	Ar	Mo	SA	Ch	In	Ir	Bg	Cz	Fr	Ge	Hu	It	Ro	Ru	Sp	Se	Tu
100	y	xy		x	x	x	x	x	x		x	x	xy	x		xy	xy	xy	
120	y																		
300	xy	xy	xy	x	x	x	x		xy		x	x		x		xy	xy		x
304											xy								
307											xy								
310	x											xy				xy	xy		
314		y									xy								
320	y											y							
321	y																		
323	x																		
330		x	xy	x	x							xy	xy			x	y		x
331	y																		
333	x																		
334	y	y									xy					z			
354												z							
500					x														
502	x																		
560	x																		
563	x																		
700	xy	xy			x				xy	y	x	x	xy	xy		xy	xy	xy	x
702	xy																		
703	xy	x			x						xy	(x)		xy			xy	x	x
704		y								y	xy		y	xy					
705										z							z		
707											xy								
710	x	xy	xy		x					y	xy	xy	xy			xy	xy	x	x
713		y														z		x	x
714		y								y	xy								
715										z									
717											xy								
720	y											xy							
721									y										
722	xy																		
723	xy				x														
724													z						
730	xy	xy	xy	x	x				x	y	xy	xy	xy			xy	xy	xy	x
731	x								y										
732	xy	xy																	
733	xy	xy														z			
734		y														z			
735		y																	
737		y																	

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Table 3. to be continued

Type	North America		South America	Africa		Asia			Europe										
	Ca	US	Ar	Mo	SA	Ch	In	Ir	Bg	Cz	Fr	Ge	Hu	It	Ro	Ru	Sp	Se	Tu
740		xy																	
743	x																		
750												y						x	
754		y																	
770	xy	xy	xy		x					y	y	(xy)	(x)				(x)y		x
772	xy	xy																	
773		y																	x
774		y									y								

So far reported only once in italic; first report after 2015 in bold; ( ) not identified from field isolates in Germany (Spring: personal observation), Hungary and Spain (Virányi *et al*. 2015); Ca – Canada; US – United States of America; Arg – Argentina; Mo – Morocco; SA – South Africa; Ch – China; In – India; Ir – Iran; Bg – Bulgaria; Cz – Czech Republic; Fr – France; Ge – Germany; Hu – Hungary; It – Italy; Ro – Romania; Ru – Russia; Sp – Spain; Se – Serbia; Tu – Turkey

The number of reported pathotypes constantly increased from 35 in 2006 (Gulya 2007) to 41 in 2014 (Virányi *et al*. 2015) and 50 now in 2018. However, in view of the difficulties that may influence results of the bioassay-based classification (Trojanová *et al*. 2017) this number might be seen critically. It should be taken into account that about one third of the pathotypes have so far only been reported once and should be considered with caution until they have been confirmed independently. This accounts, for instance, for some pathotypes from Canada (e.g. 321–333 and 502–563) which never appeared elsewhere, not even in the adjacent US areas of sunflower production where regular surveys have been conducted. On the other side, it is noteworthy to mention that the data compilations in 2007, 2015 and here in Table 3 are mainly based on reports of relatively few countries in Europe (e.g. Bulgaria, Czech Republic, France, Germany, Hungary, Romania, Russia, Serbia, Spain) and North America (Canada, USA) where broad sampling of field isolates was conducted over many years to assess the diversity of SDM, whereas vast areas of intensive production in Asia (with together ca. 2.7 million ha of sunflower production in China, India, Kazakhstan, Myanmar, and Pakistan – FAO Statistics Database: http://www.fao.org/faostat/en/#data/QC), Africa (with together 2.1 million ha in South Africa, Sudan, Uganda, Tanzania, and Zambia) or South America (with together 2 million ha in Argentina, Bolivia, Brazil, Paraguay, Venezuela) contributed only few or no records. In other words, our knowledge on the global pathotype diversity of *P. halstedii* is still rather patchy. This will most likely not change until much faster and cheaper methods of molecular-based identification are available for world-wide and continuous screening.

Based on the available information, Gulya (2007) as well as Virányi *et al*. (2015) recognized a clear shift towards pathotypes of higher virulence in many regions. The early low virulent pathotypes such as 100, 300 or 700 were gradually replaced in the 1990s by pathotypes 310, 330, 710 or 730 which overcome the Pl_{PMI3} and Pl_5 resistance genes of sunflower [(for resistance genes in differential lines see Gascuel *et al*. (2015)]. This tendency continued with the occurrence of the first *P. halstedii* isolate which was able to break the Pl_6 resistance. Since the first identification of the new pathotype 304 in France (Tourvieille *et al*. 2000), no less than 16 additional pathotypes with this ability were reported, four of them (354, 705, 715, 724) within the past four years (Sedlářová *et al*. 2016; Bán *et al*. 2018; Drábková Trojanová *et al*. 2018; Spring & Zipper 2018). A clear pattern explaining the origin and distribution of these XX4, XX5 and XX7 pathotypes is missing, as they have been reported from South-West, Central and North-East Europe to North America within just over 10 years. The absence in Asia, Africa and South America is perhaps only due to the lack of widespread testing.

There are controversy theories to explain the observed increase in genetic diversity of *P. halstedii*. A former point of view was that the high diversity has existed for a long time and that the introduction of resistant cultivars in commercial sunflower production imposed the selection pressure to the pathogen population which favoured genotypes of increasing virulence so that their relative abundance increased

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(Gulya 2007). This selection theory may certainly explain the shifted dominance towards more virulent pathotypes in given areas (e.g. France) over time. However, it seems unlikely that pathotypes which overcome the Pl_6 resistance remained undetected over decades of bioassay testings with differential line HA335 (carrier of Pl_6) and now suddenly appear in the SDM population of many countries within a few years. Therefore, another explanation would be that new pathotypes evolved recently by means of genetic recombination in which new effector combinations may lead to new virulence patterns. Hundreds of putative effector genes have been identified recently in the genome of *P. halstedii* (Sharma *et al.* 2015), thus lending the pathogen an almost infinite arsenal of options to overcome resistance. The possibility for sexual recombination appears restricted for a homothallic oomycete like *P. halstedii* (Spring 2000), but parasexual recombination between different genotypes of the pathogen has been reported (Spring & Zipper 2006, 2016) and could significantly and quickly (several rounds of mitotic propagation happen per season) contribute to the genetic diversity in the population.

CONCLUSION

The first spreading of SDM from its presumed origin in North America most likely followed two major events of seed exchange after World War II to former Yugoslavia and to Chile in the 1950s. The further dissemination was fueled by natural propagation in the local areas, but also by intensified seed exchange over long distances. The search for well-preserved and documented specimens in combination with molecular techniques could help to trace at least some of the assumed routes. Moreover, it could help to shed light onto the still undefined classification of the pathogen on sunflower and its potential host range. The introduction of a uniform test system to assess the virulence diversity of SDM in the 1990s was a milestone which allowed the identification of almost 50 pathotypes. However, the system reaches its limits and the expansion of the sunflower differential sets from 3 × 3 to 5 × 3 (Tourvieille de Labrouhe *et al.* 2012) appears to be only a transient measure to cope with the increasing number of SDM pathotypes as it makes bioassay-based pathotyping even more cumbersome and complicated (Trojanová *et al.* 2017). Meanwhile, a total of 22 resistance genes (Pl_1–Pl_{22}, Pl_{Arg}) against SDM have been identified in sunflower [for details see Spring *et al.* (2018)] and their localization in the genome has been identified by QTL mapping (Gascuel *et al.* 2015). Together with the genome sequences of resistance genes in sunflower which are now available (Hübner *et al.* 2019), it is foreseeable that bioassay-based pathotyping could be replaced by a faster, cheaper and more precise virulence classification with molecular markers. A recent study by Gascuel *et al.* (2016) shows that a breakthrough in this direction could be imminent.

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