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The European Clubroot Differential series (ECD) - review of the lasting legacy

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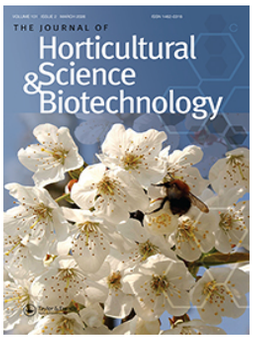

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REVIEW

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The European Clubroot Differential series (ECD) – review of the lasting legacy

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ABSTRACT

Clubroot, caused by *Plasmodiophora brassicae*, is a major disease of brassicas. The European Clubroot Differential (ECD) set developed in 1974 harmonises classification of variation in pathotypes across *Brassica rapa*, *B. napus*, and *B. oleracea*. This review traces the origin, evolution, and global legacy of the ECD. Early studies revealed pathotype variability even within single fields, highlighting the need for reliable differential hosts. The ECD triptych of 15 hosts provides a foundation for international collaboration and research. Despite initial limitations in reproducibility due to mixed pathotypes and host variability, the ECD system enabled comparative pathotyping across continents. Nationally tailored differential sets, including French, Japanese, Korean, Chinese (Sinitic), and Canadian versions, emerged addressing local cropping systems and resistance sources. The Canadian Clubroot Differential (CCD) set, derived from the ECD and expanded with *B. napus* cultivars, provided critical insights into emerging pathotypes and resistance erosion in canola. Tools, such as molecular markers and genome-wide association studies, enhanced pathotype discrimination and opportunities for resistance breeding. Region-specific sets remain essential until genomic tools enable comprehensive standardisation. The ECD's enduring contribution lies in shaping both historical and contemporary approaches for sustainably managing one of the most economically damaging diseases of brassica crops worldwide.

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Introduction – pathogen life cycle

Clubroot disease of brassicas is caused by the protist soilborne pathogen *Plasmodiophora brassicae* (Figure 1). Scientifically, *P. brassicae* was first identified and described by the Russian microbiologist Michael Woronin (Woronin & Chupp, 1878, Chupp, 1934). It is characterised by very persistent small resting spores (<5 µm diameter) that lay dormant in the soil for extended periods (Dixon, 2006). Germination is stimulated by the presence of brassicaceous (cruciferous) plants (Liu et al., 2020). The motile primary zoospores are attracted towards and penetrate the root hairs of cruciferous (and some non-cruciferous) species (Mattey & Dixon, 2015; Suzuki et al., 1992). Penetration is described as a physical process by Aist and Williams (1971). Resting spore germination and biflagellate zoospore movement in soil is the earliest, and possibly riskiest, life cycle stage when *P. brassicae* is exposed to the edaphic environment. There are suggestions that secondary zoospores may exit and re-enter the host during colonisation (Ingram & Tommerup, 1972). Re-exposure to the soil environment is, however, an unlikely risk for a pathogen as well evolved as *P. brassicae*. Within root hair cells, *P. brassicae* plasmodia undergo mitosis and possibly meiosis yielding secondary zoospores or zygotes as identified by Liu et al. (2020), which ultimately allow invasion more deeply into root cortical cells. Here, following further propagation and disruption of host metabolism that leads to clubroot symptom expression, resultant generations of robust pathogen resting spores are produced and shed back into the soil. The occurrence and economic impact of *P. brassicae* and clubroot disease are discussed by Dixon (2009b). Global incidence of clubroot disease is mapped by Adhikari et al. (2025). Recent studies have estimated the genome of *P. brassicae* to be approximately 25 Mb in size and consisting of 20 chromosomes (Javed et al., 2024; Stjelja1 et al., 2019). As a result of nuclear reassortments, there are ample opportunities during the life cycle of the pathogen for genetic variation that can alter its ability to cause disease in brassica cultivars, wild types, and breeding lines.

Variation in pathotypes and the development of differential sets

Initial studies of variation in host-pathogen relationships were reviewed by Walker (1959), which commenced with Biffen's (1905) original connection between Mendel's Laws and the management of cereal rust diseases. The first experimental evidence of variation in the pathogenicity of *P. brassicae* was provided by Honig (1931). He used three differential hosts and identified three pathotypes. Similarly, MacFarlane (1955)

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Figure 1. Serious clubroot disease symptoms caused by *Plasmodiophora brassicae* infecting the fibrous roots of Chinese cabbage taken from a field crop in Norway.

distinguished three pathotypes on a series of 11 hosts. The first differential series classifying pathotypes of *P. brassicae* was drawn up by Ayers (1957). Six differential hosts were used to identify six different virulence patterns. Adding an extra differential host to the series resulted in a seventh pathotype being recognised by Seaman et al. (1963). Williams (1966) proposed a series of four hosts, three of which had been included in the Ayers (1957) series as modified by Seaman et al. (1963). In New Zealand, Lammerink (1964, 1965) used seven *Brassica* species as differential hosts and identified six pathotypes of *P. brassicae*. In the Netherlands, Tjallingii (1965) suggested that, with some exceptions, populations of *P. brassicae* were generally specific for the host types commonly cultivated on a site from which the collection originated. The concept of a unified set of *Brassica* differential lines was originally developed by Ir. Hille Toxopeus, who was a plant breeder working at the Institut de Haaf, Wageningen, the Netherlands. Toxopeus researched aspects of the interactions between *P. brassicae* and *Brassica rapa*.

Development of the European Clubroot Differential (ECD) series provided a rational means for understanding these variations in pathogenicity. The ECD aimed at unifying separate series of differentials, allowing comparisons of virulence on *Brassica rapa*, *B. napus*, and *B. oleracea*. The ECD was established in 1974 and continues in current use, providing over fifty years of continuing value for the clubroot research and development communities. The ECD series contains selected genotypes from Williams (1966), Toxopeus and Janseen (1975), and Johnston (1968). Proposals for this unified approach were discussed initially at a Eucarpia meeting held at the Scottish Horticultural Research Institute (SHRI, which became the Scottish Crop Research Institute, SCRI, and now the James Hutton Institute), Mylnefield, Invergowrie, Dundee, in 1974. Fortunately, Professor Paul Williams from the Plant Pathology Department, University of Wisconsin – Madison, attended and provided his perspective on interactions between *P. brassicae* and *B. oleracea*. Present also was Dr Tom Johnson from the Welsh Plant Breeding Station (WPBS), now part of the Institute of Biological, Environmental and Rural Studies of Aberystwyth University. He contributed to understanding the interactions between *P. brassicae* and *B. napus*. The three major economic *Brassica* species affected by clubroot disease were used in developing suitable components of the draft ECD series triptych. The modes of inheritance of resistance postulated for these differentials varied by species. In turnips and Chinese cabbage, both belonging to the 20 chromosome *B. rapa* group, resistance was generally dominant and either monogenic or digenic and was located on the *B. rapa* A-genome. In *B. oleracea* (18 chromosomes), resistance was typically recessive and polygenic. In contrast, resistance in *B. napus* was characterised as dominant and monofactorial.

The Williams (1966) series consisted of: *B. oleracea* cv. Jersey Queen (universally susceptible); *B. oleracea* cv. Badger Shipper (resistant sauerkraut cabbage); *B. napus* cv. Laurentian rutabaga (Canadian selection); and *B. napus* cv. Wilhelmsburger green-skinned swede with variable field resistance. This series has been consistently used in the USA and elsewhere (Crute et al., 1980). The *B. rapa* differentials resulted from studies of variation in pathogenicity in *P. brassicae* in the Netherlands (Tjallingii, 1965; Wit & van der Weg, 1964) and by Toxopeus and Janseen (1975). Principally, they had studied pathogenicity towards stubble turnip (*B. rapa* [previously, *campestris*] var. *rapifera*). Pathotype variation was identified within and between populations of *P. brassicae* from

three sites using the differential series of four hosts published by Toxopeus and Janseen (1975). *Plasmodiophora brassicae* samples collected from the same cultivar in the same field were often heterogeneous, containing mixtures of pathotypes. Johnston (1968) proposed a series of five *B. napus* differential hosts, and these identified seven pathotypes (Buczacki & Humphrey, 1973; Johnston, 1968, 1970). The origins of the accepted differentials were as follows: *B. oleracea* cvs. Septa (white cabbage), Bindsachsener (white cabbage), and Verheul (borecole) were standard cultivars used by Nieuwhof (1969) for research at the then Vegetable Research Station (IVT), in Wageningen, the Netherlands. American contributions included Badger Shipper, a resistant cultivar from the University of Wisconsin – Madison, and Jersey Queen, a susceptible cultivar. *Brassica napus* Laurentian, a Canadian 'rutabaga' type cultivar, and Wilhelmsburger, a green-skinned swede from the Wisconsin differential set proposed by Williams (1966), were maintained in Sweden at Svalöf by Jönsson. Johnson contributed DC 101, DC 119, DC 128, DC 129, and DC 130 as the differential set described in Johnston (1968). A *B. rapa* ssp. *rapifera* differential set was used by Toxopeus and Janseen (1975) at SVP Wageningen. This set included Bicolour, aaBBCC, AAbbCC, AABBcc, AABBCC, the universally susceptible Chinese cabbage cv. 'Granaat' and Siletta (a *Raphanus sativus* var. *oleifera* fodder radish cultivar).

The use of differentials with defined genetic resistance enabled comparisons of the responses of *Brassica* species to different *P. brassicae* populations. Over time, accumulating data from these interactions have provided clear evidence for the multiplicity and variability of pathotypes (physiological races), as well as insights into their biology and biogeography.

Following initial discussions at the Eucarpia meeting in 1974 and subsequent consultations, the complete set of differential hosts comprising the ECD series was proposed (Buczacki et al., 1975). These included three groups (*B. rapa*, *B. napus*, and *B. oleracea*), each consisting of five hosts, for a total of 15 differential genotypes (Table 1). The *B. rapa* group was provided by Toxopeus, the *B. napus* group originated from Johnston, and the *B. oleracea* group came mainly from Williams and incorporated breeding lines developed there by Professor J.C. Walker. The cultivar Bindsachsner probably came from Dr Peter Mattusch at the Vegetable Research Station in Cologne, Germany.

Initial research using the ECD

The application of the ECD series was evaluated by Jones (1980), Dixon et al. (2020), Dixon (2020), and Jones et al. (1982a & 1982b). These studies utilised collections of *P. brassicae* clubs taken from UK cultivar field trials at Myerscough (Lancashire), Rosemaund (Shropshire), Trawsgoed (Ceredigion, Dyfed), and Seale Hayne (Devon). Replicate tests made with the ECD series, however, did not always yield consistent results; even when inoculum was prepared from different clubs within a single collection, passaging through Granaat (ECD 05) could result in changes to pathogenicity (Table 2). The Trawsgoed and Rosemaund populations each contained multiple pathotypes, and even different clubs from the same cultivar harboured distinct pathotypes. Three single-spore isolates produced from the Trawsgoed population differed with respect to pathogenicity when tested against the ECD series. Interactions between spores of two *P. brassicae* populations demonstrated that infection by one population could be restricted by the presence of spores from the other. These findings

Table 1. European Clubroot Differential (ECD) series.

Differential number	Differential host	Notes
Brassica rapa group		
01	subspecies *rapifera* line aaBBCC	
02	subspecies *rapifera* line AAbbCC	
03	subspecies *rapifera* line AABBcc	
04	subspecies *rapifera* line AABBCC	
05	var. *pekinensis* cv. Granaat	Universally susceptible
Brassica napus group		
06	var. napus cv. Nevin	
07	var. *napus* cv. Giant Rape	Selected as universally susceptible
08	var *napus* selection ex Giant Rape	Morphologically similar to 07 but carries resistance traits
09	var. *napus* New Zealand clubroot resistant rape	
10	var. *napobrassica* cv. Wilhemsburger swede	
Brassica oleracea group		
11	var. *capitata* cv. Badger Shipper	
12	var. *capitata* cv. Bindsachsner	
13	var. *capitata* cv. Jersey Queen	
14	var. *capitata* cv. Septa	
15	var. *acephala* subvar. *lacinata* cv. Verheul	

After Buczacki et al. (1975).

Table 2. European Clubroot Differential (ECD) codes of *Plasmodiophora brassicae* populations before and after host passage through *Brassica rapa* var. *pekinensis* cv. Granaat (universally susceptible).

Source population from UK trials centres	Original ECD code	After passage through cv. Granaat
Myerscough	16/2/12	16/11/30
Rosemaund	16/31/31	16/0/20
Seale-Hayne	16/27/13	16/19/4
Trawscoed	16/14/6	16/14/12

Source: G.R. Dixon.

showed that there is substantial differential pathogenicity within single collections of clubs, even where these are drawn from the same trial site and individual cultivars.

Clubs from three cultivars of *B. napus* var. *napobrassica* (swede), collected at the Myerscough site, revealed significant variation in pathotype virulence as classified using the ECD system (Dixon, 1980). Inoculum from cv. Acme produced an ECD code of 16/2/0, whereas spores extracted from clubs on cvs. Marian and Wilhelmsburger Prime registered as ECD code 16/22/0. At the Trawsgoed site, ECD codes of 16/14/06 and 16/14/- were recorded, while at the Rosemaund site, a code of 16/31/31 was observed. The latter pathotype could cause clubroot disease across a range of *B. napus* (swede) cultivars (Dixon, 1977b). This work established the existence of virulence variation between pathotypes affecting cultivars grown at a single site, as well as across geographically separated locations.

Studies by Tinggal (1980) and Tinggal and Webster (1981) examined populations of *P. brassicae* collected from crops grown in the UK's South Western counties of Devon and Cornwall. This is an area with a long history of intensive brassica production, particularly overwintered cauliflowers and other vegetable brassicas. The region is heavily infested by *P. brassicae*. The most prevalent pathotype identified was ECD 16/31/31, reflecting the dominant cropping of *B. oleracea* and *B. rapa* (swede) cultivars, with less frequent occurrence of ECD 22/31/31. The latter may have also suggested persistent cultivation of stubble turnips (*B. rapa*) and the erosion of their resistance. Results also indicated that spores extracted from individual galls often contained mixtures of pathotypes.

There were indications that soil type influenced the occurrence of different *P. brassicae* pathotypes, and that spore load affected the intensity of disease in the various ECD genotypes (Tinggal, 1980; Tinggal & Webster, 1981). These studies also suggested that brassica weeds may serve as inoculum reservoirs. Weeds examined included Shepherd's Purse (*Capsella bursa-pastoris*), Charlock (*Sinapis arvensis*), White Charlock (*Raphanus raphanistrum*), Field Penny Cress (*Thlaspi arvense*), Wavy Bittercress (*Cardamine flexuosa*), Pepperwort (*Lepidium campestre*), and Garlic Mustard (*Alliaria petiolate*). Among these, Shepherd's Purse, Charlock, and Field Penny Cress were found to be naturally infected with clubroot in the field and carried the ECD 16/31/31 pathotype, which was also dominant in associated cultivated crops. Ornamental wallflowers (*Cheiranthus cheiri* and *C. allionii*) were also considered as reservoirs of *P. brassicae*, predominantly harbouring the ECD 16/31/31 pathotype. Scottish field trials using land with an ECD code 31/31/31 searched for forms of resistance in cabbage (*B. oleracea* var *capitata*) (G.R. Dixon & Robinson, 1986) and calabrese (*B. oleracea* var *italica*) (G.R.W. Dixon et al., 1986).

International collaboration and analysis of populations

An international effort aimed at determining the distribution and predominance of differing strains of *P. brassicae* was coordinated by Toxopeus et al. (1986). Researchers requesting supplies of ECD seed were asked to send back information describing the frequency and geographical distribution of ECD codes (Tables 3 and 4). The extent of pathotype variation is emphasised by Table 4. At this time, there were more than 80 active researchers ('Clubrooters') worldwide. Data submitted by researchers using the ECD series yielded 477 test results, which were subsequently digitised. Results from tests that used fewer than 10 plants per differential were discarded. For inclusion in the final analysis, each test was required to include at least 30 plants per host and a minimum of 200 plants overall, with at least one host (usually cv. Granaat) exhibiting a disease index of 95% or higher. Based on these criteria, 170 tests were disqualified, leaving 307 using 15 hosts, resulting in a total of 4605 individual test results. Of these, 697 results (15%) were classified as

Table 3. Most frequently reported European clubroot differential (ECD) pathotype codes from a global survey of *Plasmodiophora brassicae* populations (after Toxopeus et al., 1986.)

16/2/15	16/22/31
16/2/30	16/23/31
16/2/31	16/30/15
16/3/30	16/30/31
16/3/31	16/31/14
13/6/30	16/31/15
16/6/31	16/31/30
16/7/31	16/31/31
16/14/31	20/31/31
16/15/31	

More recently, Lüders (2017) detected 33 distinct ECD triplet codes in European *P. brassicae* populations, with 16/14/31, 16/31/31, and 17/31/31 most frequently reported. In Germany, Zamani-Noor (2017) identified 16/14/30, 16/14/31, and 16/31/31 as dominant. In Czechia and Poland, 16/14/15 or 16/15/15 were most common, depending on the disease severity threshold used to distinguish susceptible from resistant reactions (Řičařová & Kaczmarek, 2016).

Table 4. European Clubroot Differential (ECD) codes reported more than once, classified according to the origin of the initial *Plasmodiophora brassicae*-infected galled root material.*

Original Source					
Brassica rapa	*Brassica napus*		*Brassica oleracea*		Soil
16/3/13	16/2/15	18/31/30	16/0/30	16/15/12	16/2/30
16/2/29	16/2/29	18/31/31	16/0/31	16/15/14	16/6/30
16/2/30	16/2/31	20/31/28	16/2/13	16/15/15	16/14/13
16/2/31	16/6/31	16/31/29	16/2/14	16/15/30	16/14/15
16/6/14	16/7/31	20/31/30	16/2/15	16/15/31	16/14/29
16/6/30	16/15/30	20/31/31	16/2/24	16/18/33	16/14/31
16/22/31	16/15/31	21/31/31	16/2/26	16/18/31	16/15/31
16/23/31	16/22/8	22/31/29	16/2/28	16/19/30	16/22/13
16/31/30	16/22/10		16/2/29	16/19/31	16/22/15
16/31/31	16/22/12		16/2/30	16/23/31	16/22/28
20/31/31	16/22/13		16/2/31	16/30/12	16/22/29
	16/22/14		16/3/14	16/30/14	16/22/30
	16/22/15		16/3/15	16/30/15	16/22/31
	16/22/29		16/3/26	16/30/30	16/23/31
	16/22/30		16/3/28	16/30/31	16/30/15
	16/22/31		16/3/30	16/31/14	16/30/31
	16/30/12		16/3/31	16/31/15	16/31/30
	16/30/14		16/6/14	16/31/22	16/31/31
	16/30/28		16/6/15	16/31/30	20/31/31
	16/30/31		16/6/28	16/31/31	
	16/31/14		16/6/30		
	16/31/15		16/6/31		
	16/31/29		16/7/12		
	16/31/30		16/7/31		
	16/31/31		16/14/14		
	17/31/31		16/14/30		
	18/3/29		16/14/31		

*Note: Not all experiments supplied this information; after Toxopeus et al. (1986).

debatable, meaning that they showed inconsistent resistant/susceptible responses.

Analyses of the results showed that, at a micro-geographical level, there was considerable variation in the occurrence of different *P. brassicae* pathotypes. In contrast, on a macro-geographical scale, similar pathotypes were found both nationally and internationally. Additionally, the movement of *P. brassicae* from Europe to other continents was detectable (Toxopeus et al., 1986). The studies also indicated that different cultivars at the same location could be infected with distinct pathotypes. Newsletters circulated amongst active 'Clubrooters' reported ongoing discussions regarding the interpretation of susceptible and resistant reactions. Symptom evaluation keys were published by Dixon (1977a), and Dixon (1976) also reported on an international survey of methods used for the culture and inoculation of ECD plants. Variation in clubroot disease intensity was demonstrated by Dixon (1977b), based on multi-year field assessments of disease index values on the swede cv. Wilhelmsburger. Recorded disease indices during the period 1969–1975 ranged from 7.6 to 40.0.

A study of the importance of clubroot internationally, conducted across 18 countries cultivating a combined total of 6 million ha of brassica crops, reported a mean infection rate of 10%. Infection levels ranged from 1% in Sweden to 48% in Scotland. The crops surveyed included *B. oleracea*, *B. napus*, *B. rapa* (*B. campestris*), *Raphanus sativus*, and *Sinapsis alba*. This survey was later updated by Dixon (2009).

Variation within populations of P. brassicae

The differential pathogenicity of *P. brassicae* populations cannot be adequately classified by a single test of bulked samples, regardless of how carefully those samples are collected, as highlighted by Crute et al. (1980) and Crute et al. (1983) in their analyses of host resistance. This is because there may be many pathotypes in each population. Individual pathotypes can be identified only by testing individual samples and, ideally, producing single-spore-derived isolates, which in turn requires the development of more reliable and repeatable methods for obtaining such isolates.

The discovery that spores of different pathotypes can interact further complicates pathotype classification. It is not sufficient merely to determine the relative proportions of different pathotypes within a population; it is also necessary to understand the minimum proportion of spores of a given pathotype required to cause infection. Several 'Clubrooters' observed differences in disease development among cultivars obtained from different seed suppliers, most notably in the swede cv. Wilhelmsburger. Additionally, it was noted that using highly

susceptible hosts to 'passage' inoculum of populations before ECD testing does not necessarily produce more reliable results. Inoculum is likely to contain multiple pathotypes, and some ECD hosts require greater concentrations of spores to achieve consistent symptom expression. During passaging through a susceptible host, *P. brassicae* may have undergone both mitotic and meiotic divisions, potentially altering its virulence characteristics.

Conserving and distributing the ECD seed

The conservation of ECD seed was eventually transferred in 1985 from SVP (Foundation for Agricultural Plant Breeding) in Wageningen, where it had been maintained for 10 years (1974–1984), to the UK Vegetable Gene Bank (UKVGB) at Warwick University Crop Centre (formerly National Vegetable Research Station, Wellesbourne). This arrangement has been successful for 40 years and continues currently. The UKVGB specialises in the conservation of genetic diversity in vegetable crops, with a focus on root and leafy vegetables. *Brassica* form a significant proportion of its collections, accounting for around 6000 of its samples. Alongside the main collection, the UKVGB manages research collections which have high importance to particular research communities, for example the *Brassica* S-allele collection (Ockendon, 2000). The ECD set, as a specialist research collection of high importance to the 'Clubrooters' research community, therefore, fits completely within the overall remit of the UKVGB collections (Trinder, 2024). Seeds were received in 1985 and underwent a round of propagation via protected mass pollination in polytunnels during this decade. Seed production managed in this way ensured that sufficient seed stocks were available for the longer term. Seed at the UKVGB is stored under optimal conditions for maximum longevity; and is dried to 5% moisture content by weight and kept at −20°C (Anon, 2014). Under these conditions, seed lifespan can be measured in decades and indeed the ECD seed currently distributed comes from the batches reproduced in the 1980s. In 2020, the ECD set was formally incorporated into the UK's National Inventory listing of seed samples managed under the Multilateral System of the International Treaty on Plant Genetic Resources. This designation further safeguards the ECD series' longevity as a protected collection.

Seed of the ECD series continues to be distributed to researchers worldwide, ensuring that the originators' goal of a unified set of differential lines for international comparison endures. The ECD set remains a highly requested resource; since 2010, when UKVGB request records were digitised, more than 60 requests for seed have been made. The geographic origin of these requests strongly reflects countries with high concentrations of research into clubroot disease, with many originating from Canada and Europe. Previously, between 1987 and 2011, there were 105 requests for seed. Requests predominately came from the United Kingdom, Germany, the Netherlands, Australia, and the United States, with smaller numbers from Belgium, Denmark, France, Ireland, Italy, Portugal, Turkey, New Zealand, Russia, China, South Korea, Taiwan, Japan, the Philippines, and South Africa (C. Allender, personal communication). A systematic review of the use of plant genetic resources from the UKVGB collections by Davies and Allender (2017) found that use of the ECD set accounted for 17 publications citing use of the UKVGB material, while further work by Trinder (2024) identified 142 publications using the ECD. This highlights the significance of the ECD series and more broadly emphasises the importance of maintaining specialist research collections that remain easily and consistently available to researchers over long periods of time, benefiting both the research and plant breeding communities.

International adaptations of the ECD set

The originators of the ECD series requested that users would report results obtained from its application, and, where appropriate, modify the differentials used or establish new sets of differentials. This was highlighted by Hille Toxopeus (personal correspondence, 1989) and has largely occurred, as summarised in Table 5 and discussed below. The initial ECD series was developed for use in Western Europe, the United States, and Australasia. Early adopters provided a substantial body of information, culminating in an international analysis (Toxopeus et al., 1986). Results suggested that *P. brassicae* spread from Europe via the colonisation of the USA and Canada. The rapid expansion of oilseed rape (*B. napus*) cultivation in Europe led to increasing reports of clubroot disease, mirroring the already well-established incidence of the disease in vegetable brassicas across Europe and North America. Studies in Australia (Donald et al., 2006) generated 23 triplet codes from 41 collections harvested from the main vegetable brassica cropping areas. The most common codes were 16/3/12, 16/31/31, and 16/19/31. Australian populations were more similar with those found in the USA than those in Europe, possibly indicating an original spread from the USA to Australia.

The presence of multiple pathotypes within a single gall collected from canola (oilseed rape; *B. napus*) crops in Canada has been confirmed by numerous studies (Askarian et al., 2021; Fu et al., 2020; Xue et al., 2008), supporting earlier findings. The conventional use of the European Clubroot Differential (ECD) series has been reported by

Table 5. International adaptations of the European Clubroot Differential (ECD) series.

System	Origin	Host No.	Species Composition	Notes	Reference
French differential set	France	3	*Brassica napus*	First system developed to adapt the ECD series to country-specific clubroot populations	Somé et al. (1996)
Japanese differential set	Japan	7	Six clubroot-resistant *B. rapa* lines and one susceptible Chinese cabbage	Developed to improve resolution of pathotypes affecting Chinese cabbage in Japan	Osaka et al. (2008)
Canadian Clubroot Differential (CCD) set	Canada	13	Nine *B. napus* genotypes (including key canola/oilseed rape types), two *B. rapa* (including ECD 05 as the universally susceptible check), and two *B. oleracea*	Developed to characterise *P. brassicae* pathotypes from canola; incorporates eight ECD hosts and all hosts from the Williams (1966) and Somé (1996) systems, enabling simultaneous classification	Strelkov et al. (2018)
Korean differential set	South Korea	4	Three clubroot-resistant *B. rapa* cultivars and one susceptible cultivar	Developed to differentiate pathotypes prevalent in Korean crops	Kim et al. (2016)
Sinitic Clubroot Differential (SCD) set	China	9	Eight inbred *B. rapa* lines carrying clubroot resistance genes and one universally susceptible check	Developed to improve pathotyping of *P. brassicae* populations affecting Chinese cabbage, where clubroot has become a major constraint	Pang et al. (2020)

Zeng et al. (2024), who examined clubroot outbreaks in China and variations in pathotype virulence. The predominant pathotypes identified were ECD 16/15/31 and 16/31/31, with the latter showing a strong ability to cause disease across all *B. napus* and *B. oleracea* differentials, reflecting the widespread cultivation of oilseed rape and Brassica vegetables in the region. A recent comprehensive study of European oilseed rape crops by Zamani-Noor et al. (2022), which analysed 84 *P. brassicae* isolates collected from Czechia, Germany, Poland, and Sweden, reported that 16/31/31 was the most common ECD pathotype. Virulence analyses using the ECD series further distinguished the isolates into 42 pathotypes. Similarly, Lüders (2017) identified 33 distinct ECD triplet codes in European *P. brassicae* populations, with 16/14/31, 16/31/31, and 17/31/31 reported most frequently. In Germany, dominant pathotypes included 16/14/30, 16/14/31, and 16/31/31 (Zamani-Noor, 2017), while in the Czechia and Poland, 16/14/15 or 16/15/15 were most common, depending on the disease severity threshold used to distinguish susceptible from resistant reactions (Řičařová & Kaczmarek, 2016). Collectively, these findings support the observations of Toxopeus et al. (1986) that *P. brassicae* pathotypes vary widely within regions but are dominated by a limited number of aggressive variants at a macrogeographical scale.

Among the differentials, ECD 01 and 04 consistently exhibited high levels of resistance to most Chinese pathotypes, demonstrating the utility of the ECD set in discerning pathotype diversity. However, isolates designated 16/15/31 and 16/23/31 on the ECD system were found to be highly virulent against the widely grown oilseed rape cv. Huashuang 5 R, highlighting the potential risk of overreliance on a single cultivar and the ability of *P. brassicae* to erode host resistance.

French differential set

One of the earliest attempts to tailor the ECD series for country-specific clubroot issues, in a country that had previously collaborated in its development, was undertaken in France, as reported by Somé et al. (1996), following studies of *P. brassicae* populations beginning in 1985. Variations in virulence were examined amongst 20 field collections of *P. brassicae* from Brittany, northern France, and southern France. The differentials initially used included *B. napus* cv. Nevin (ECD 06); a selection from Giant Rape (ECD 08); New Zealand Resistant Rape (ECD 09); all fodder rapes; the swede cv. Wilhelmsburger (ECD 10); rutabaga/swede cv. Laurentian; spring oilseed rape cv. Brutor; winter oilseed rape cv. Darmor; and the cabbage cvs. Badger Shipper (ECD 11) and Jersey Queen (ECD 13). Giant Rape (ECD 07) and cv. Granaat (ECD 05) were included as universally susceptible controls. These potential differentials were tested against *P. brassicae* populations isolated from European cabbage, Chinese cabbage, kale, radish, oilseed rape, and the weed *Sisymbrium officinale* (hedge mustard).

The differential properties of this assemblage were, however, found to be of limited value for distinguishing French populations of *P. brassicae*, highlighting the need to develop a differential set tailored to local requirements. Of the 10 brassica lines tested, seven showed differential responses to inoculation. Notably, two oilseed rape cultivars exhibited previously unreported differential reactions. Some of the differential lines used in earlier studies to classify pathotypes were susceptible to all collections, suggesting that French *P. brassicae* populations may differ significantly from those reported elsewhere. The refined differential set included the cvs. Nevin (ECD 06), Wilhelmsburger (ECD 10), and Brutor (Somé et al., 1996).

French studies confirmed the heterogeneity of field collections of *P. brassicae*, consistent with previous

reports by Tinggal and Webster (1981), Jones etal. (1982), and Scott (1985). They also corroborated the findings of Toxopeus et al. (1986), who observed that similar pathotypes could be found in different localities, while different pathotypes could coexist within the same field. Finally, studies of single-spore-isolates by Somé et al. (1996) demonstrated the rapid development of pathotype variation within *P. brassicae* populations. The ability to fractionate multiple distinct pathotypes from one spore suspension further confirmed the genetic heterogeneity of field populations.

Japanese differential set

The occurrence of variation in the virulence of *P. brassicae* populations affecting susceptible and resistant Chinese cabbage cultivars was reported by Tanaka et al. (1991) using the Williams (1966) differentials. More recently, some of the ECD hosts showed intermediate and fluctuating responses to Japanese populations of *P. brassicae* (Kuginuki et al., 1999), making clear pathotype classification difficult. To address this issue, 18 hosts (including clubroot-resistant (CR) F_1 hybrid cultivars of Chinese cabbage and *B. rapa* lines) were used for pathotype identification. The responses of some CR F_1 cultivars were very distinct, allowing for the differentiation of four field population groups of *P. brassicae*. The study concluded that using genetically uniform F_1 cultivars as differentials yielded clearer and more reliable results. Some *P. brassicae* pathotypes were also identified using the Williams (1966) differentials and ECD 01 to ECD 05. Kuginuki et al. (1999) suggested that CR F_1 cultivars, when used as differential hosts, improve the international classification and understanding of pathotype distribution.

The responses of the differential hosts suggest that several major clubroot resistance genes are present in *B. rapa*. The authors proposed that pyramiding these resistance genes could support the development of cultivars with more durable resistance, helping to delay the erosion of clubroot resistance in Chinese cabbage. This strategy was previously employed by Toxopeus (personal communication) in developing the clubroot-resistant stubble turnip ECD 04. More recently, Osaka et al. (2008) suggested that six clubroot-resistant *B. rapa* lines, along with a susceptible Chinese cabbage cultivar (cv. Nozaki Nigo), could serve as differential hosts to provide a clearer definition of *P. brassicae* pathotypes affecting Chinese cabbage in Japan. Classifying *P. brassicae* isolates by using single sequence repeats, Kubo et al. (2017) identified the presence of two pathotypes within a single collection site and found a close genetic relationship between them.

Canadian differential set

The outbreak of clubroot disease in Canadian canola (*B. napus*) was first identified in the early 2000s around the city of Edmonton in central Alberta. This epidemic, affecting the most valuable field crop in the Canadian Prairies, has driven the largest and most intensive research efforts into the biology and management of *P. brassicae*, including the search for and deployment of resistance genes. Testing facilities and an example of an ECD test are shown in Figures 2 and 3. Studies by Cao et al. (2009) characterised populations of *P. brassicae* using the ECD set as well as the differential systems of Williams (1966) and Somé et al. (1996). The predominant pathotype identified from canola in central Alberta was identified as ECD -/15/12, Williams pathotype 3, and Somé et al. pathotype P_2.

By contrast, two *P. brassicae* populations obtained from southern Alberta were nearly avirulent on the rutabaga cv. 'Laurentian,' a member of the Williams differential set that is highly susceptible to populations

Figure 2. Pathotyping of *Plasmodiophora brassicae* field populations collected from canola (*Brassica napus*) in western Canada using the Canadian Clubroot Differential (CCD) set, which includes, in part, the European Clubroot Differential (ECD) hosts ECD 02, ECD 05, ECD 06, ECD 08, ECD 09, ECD 10, ECD 11, and ECD 13. Assays are conducted in the greenhouse using a commercial potting medium. Plants are maintained under high moisture for 1 week after inoculation, then watered and fertilised (20N:20P:20K) as needed with slightly acidified water (pH 6.5), at ~20°C under a 16-h photoperiod with supplemental lighting.

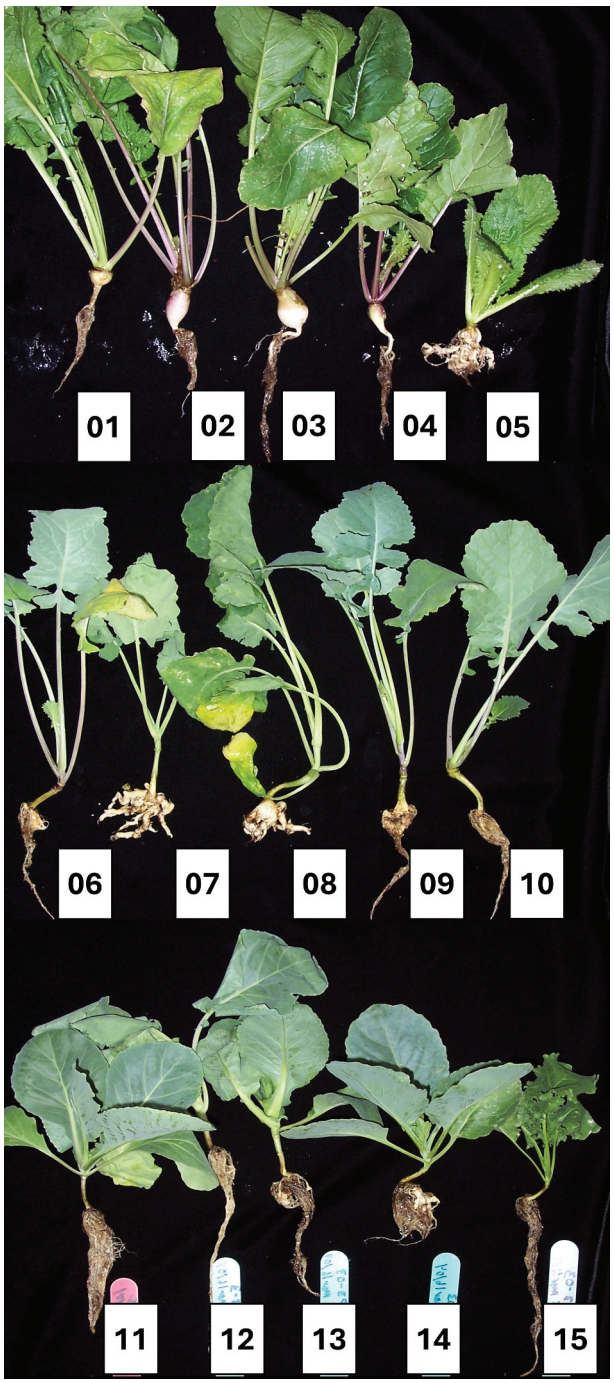

Figure 3. Reactions of the European Clubroot Differential (ECD) series inoculated with a field population of *Plasmodiophora brassicae*. The hosts include the *brassica rapa* group (top panel): *B. rapa* subsp. *rapifera* line aaBBCC (ECD 01), *B. rapa* subsp. *rapifera* line AAbbCC (ECD 02), *B. rapa* subsp. *rapifera* line AABBcc (ECD 03), *B. rapa* subsp. *rapifera* line AABBCC (ECD 04), and *B. rapa* var. *pekinensis* cv. Granaat, Chinese cabbage (ECD 05); the *B. napus* group (middle panel): *B. napus* var. *napus* cv. Nevin (ECD 06), *B. napus* var. *napus* cv. Giant rape (ECD 07), *B. napus* var. *napus* selection ex giant rape (ECD 08), *B. napus* var. *napus* New Zealand clubroot-resistant rape (ECD 09), and *B. napus* var. *napobrassica* cv. Wilhemsburger Swede (ECD 10); and the *B. oleracea* group (bottom panel): *B. oleracea* var. *capitata* cv. Badger shipper, cabbage (ECD 11), *B. oleracea* var. *capitata* cv. Bindsachsner, cabbage (ECD 12), *B. oleracea* var. *capitata* cv. Jersey Queen, cabbage (ECD 13), *B. oleracea* var. *capitata* cv. Septa cabbage (ECD 14), and *B. oleracea* var. *acephala* subvar. *lacinata* cv. Verheul kale (ECD 15).

from central Alberta. These southern populations also showed reduced virulence on several ECD hosts, resulting in distinct pathotype designations across all three differential systems used. On the ECD set, these populations were characterised as -/6/8 and -/4/0, which were unusual pathotypes for the province (Strelkov et al., 2006, 2007). It remains unclear whether these distinct virulence patterns reflect independent pathogen introductions or simply variation within a broader population; however, restriction-site associated DNA sequencing (RADseq) later revealed two genetically distinct *P. brassicae* populations in Alberta, suggesting multiple introductions (Holtz et al., 2018).

Pathotypes identified in Québec and Manitoba also indicated increased diversity while retaining the ability to cause clubroot disease on most brassica host differentials, including various species, subspecies, and varieties. Pathogen populations from central Alberta exhibited the widest host range, with the ability to cause disease on at least one genotype of each of the species/subspecies/varieties tested, except for kale (ECD 15). By contrast, a *P. brassicae* population found in Ontario caused disease only on vegetable differentials. This population was virulent only on the universally susceptible cv. Granaat (ECD 05) and on two cabbage differentials (ECD 13 and ECD 14). This limited virulence may reflect the long-term predominance of cruciferous vegetable production in Ontario and the corresponding selection pressure placed on *P. brassicae*.

Extensive information on pathogen variation and resistance in canola was reported by Strelkov et al. (2018). Challenges in accurately characterising *P. brassicae* populations infecting CR canola cultivars, particularly in cases involving multiple pathotypes, prompted the development and adoption of the Canadian Clubroot Differential (CCD) set. These challenges are understandable, as earlier differential systems were designed for other crop types: the Williams (1966) set for sauerkraut cabbage, the Somé et al. (1996) set for oilseed rape and vegetable brassicas, and the Toxopeus et al. (1986) set for *B. rapa* (stubble turnips). Within the original ECD set, only the *B. napus* differentials were designed for studying *P. brassicae* on rape crops, and even those were intended for fodder and forage types rather than oilseed cultivars. In the context of the current clubroot epidemic and its associated evolutionary pressures, it is evident that *P. brassicae* can generate distinct virulence phenotypes on oilseed rape (Strelkov et al., 2016; Strelkov & Hwang, 2014).

The CCD set incorporates host differentials from several sources, including Williams (1966), Somé et al. (1996), and eight entries from the ECD set. It also includes the CR winter oilseed rape cv. Mendel, the spring canola cultivars Westar and Brutor, and the CR

hybrid canola 45H29. From the ECD set, the following hosts are included: stubble turnip line AAbbCC (ECD 02), the universally susceptible Chinese cabbage cultivar Granaat (ECD 05), the resistant fodder rape cv. Nevin (ECD 06), Giant Rape selection (ECD 08), New Zealand resistant rape (ECD 09), the rutabaga cv. Wilhelmsburger (ECD 10), and the cabbage cvs. Badger Shipper (ECD 11) and Jersey Queen (ECD 13).

Pathotypes in the CCD system are designated using a combination of letters and numbers (Askarian et al., 2021; Strelkov et al., 2018). Recent studies have evaluated brassica genotypes to refine the CCD set, including the replacement of the hybrid canola 45H29, whose seed supply is becoming limited, with a doubled haploid *B. napus* line carrying the same resistance source (Hollman et al., 2025). This study emphasised the long-standing value of ECD 05 (Granaat) as a universally susceptible cultivar.

Strelkov et al. (2018) initially identified 17 distinct pathotypes using the CCD set, compared with only five using the Williams (1966) differentials and two with the Somé et al. (1996) system. As such, the CCD provides significantly greater discriminatory power, an essential feature for managing the severe clubroot epidemic affecting Canada's economically important canola crop. Ongoing surveillance of pathotype evolution and the erosion of resistance in canola cultivars across the Canadian Prairie Provinces has been described and compared with international experiences (Storfie et al., 2025). The incorporation of ECD differentials into the CCD framework has enhanced its resolving power, enabling more precise detection and classification of emerging pathotypes.

More recently, the development of six spring-type *B. napus* single-gene lines, each carrying a distinct clubroot resistance gene (*Rcr1*, *Rcr3*, *Rcr5*, *Rcr8*, *Rcr9*, or *Rcr10*), has further strengthened race characterisation efforts in Canada (Zhang et al., 2026). Developed in a Canadian canola background, these lines were used to differentiate 35 *P. brassicae* field strains from western Canada into 24 races, revealing substantial variation in avirulence allele frequencies and demonstrating that none of the individual CR genes conferred universal resistance. Compared with existing differential systems, these spring-type single-gene lines may provide enhanced precision for race profiling and represent a complementary tool to the CCD for monitoring pathogen diversity and guiding gene-stacking strategies aimed at achieving durable clubroot resistance.

Pathogen virulence on the ECD lines included in the CCD have been reported in detail. Notably, all or nearly all *P. brassicae* isolates were avirulent on ECD 02 and ECD 11. In contrast, virulence varied among isolates, and fluctuated over years, for ECD 06, ECD 09, ECD 10, ECD 13, and Laurentian (Storfie et al., 2025). These findings suggest that *P. brassicae* populations are becoming increasingly diverse. Similarly, recent studies of clubroot disease in canola crops grown in North Dakota (Marino et al., 2023) revealed significant pathotype variation. These pathotypes were identified using the CCD set, further underscoring the continued relevance of the ECD lines. The identification of virulence patterns using the CCD is also facilitating the development of resistant *B. napus* cultivars through molecular approaches, including genome-wide association mapping (Dakouri et al., 2021). Recently, Karim and Yu (2025) identified quantitative trait loci (QTL) for resistance to aggressive Canadian strains of *P. brassicae* in the rutabaga (green skinned swede) cv. Wilhelmsburger (ECD 10).

Korean differential set

Clubroot disease is a significant problem for Chinese cabbage (*B. rapa*) crops in Korea. Initial studies by Kim et al. (2016) used the Williams (1966) differential set to distinguish pathotypes of *P. brassicae*. Although these differentials were incorporated into the ECD set, they alone did not provide sufficient resolution to characterise the variation in Korean *P. brassicae* populations.

Following a survey of Chinese cabbage cultivars from Korea, China, and Japan, Kim et al. (2016) selected three CR cultivars (CR Cheongrok, Degao CR1016, and Akimeki) and one non-CR cultivar (Noranggimjang) to differentiate *P. brassicae* pathotypes isolated from Korean crops. This differential set is expected to support breeders and growers in managing clubroot disease in Chinese cabbage.

Chinese (Sinitic) differential set

The development of the ECD set was a response to serious crop losses caused by clubroot in Europe and North America during the 1950s; at the time, China was reportedly free of the pathogen (P. Williams, personal communication). China, however, has widespread production and consumption of Chinese cabbage as a staple vegetable and is also the world's largest producer of oilseed rape for domestic use. As a result, clubroot has become a major endemic disease in China as emphasised by Xu et al. (2025) in a review which discusses the use of ECD analyses as part of sustainable disease control.

The epidemic has led to the development of a specialised Sinitic Clubroot Differential (SCD) set (Pang et al., 2020), consisting of eight inbred lines of Chinese cabbage containing clubroot resistance genes, along with one universally susceptible line. The SCD set was designed to identify the pathotypes of *P. brassicae* in the region (Pang et al., 2020). Differences between pathotypes are ultimately

determined by variations in the presence, absence, or differential expression of certain pathogenicity-associated genes (Zhang et al., 2015). More recently, Zheng et al. (2019) identified six genes specifically associated with one *P. brassicae* variant, labelled P4, which predominates in the Chinese pathogen population.

The CR hosts used in the SCD system carried either known (e.g. *CRa, Crr1, Crr3*, and *CRd*) or novel resistance genes. Several CR hosts (H04, H05, and H06) showed differential responses to some pathotypes despite sharing the *CRa* gene, suggesting the presence of additional, unidentified resistance loci. Theoretically, the SCD system could distinguish up to 256 *P. brassicae* pathotypes; in practice, 16 different pathotypes were identified by Pang et al. (2020).

Analysis of differential sets

A critical analysis of current differential host sets by Zamani-Noor and Jedryczka (2024) examined all major systems, including the Williams (1966), the ECD set (Buczacki et al., 1975), the differentials of Somé et al. (1996), the CCD set (Strelkov et al., 2018), the Korean set (Kim et al., 2016), the SDC set (Pang et al., 2020), and the Japanese set (Osaka et al., 2008), using a broad spread of *P. brassicae* populations collected internationally. Their review highlighted that each set is tailored to the dominant crop hosts in the region where it originated.

The authors suggest the potential value of establishing a globally interpretable differential set, an idea that aligns with the original intent of the ECD, as outlined by Toxopeus et al. (1986). In practice, however, most researchers have found greater utility in developing region-specific systems. A more unified understanding of pathotype variation may eventually be achieved through the generation of a global pangenome of *P. brassicae* (Zamani-Noor & Jedryczka, 2024).

It has been suggested that resistance-eroding isolates and the proliferation of *P. brassicae* are increasing on a global scale. The early international studies of Toxopeus et al. (1986) anticipated this trend, proposing that such changes could occur in response to crop intensification and the widespread use of resistant cultivars and numbered breeders' lines.

Molecular pathotyping

Early efforts to complement phenotypic pathotyping of *P. brassicae* with molecular approaches began with the work of Manzanares-Dauleux et al. (2000), who demonstrated that random amplified polymorphic DNA (RAPD) markers could discriminate among French pathotypes (Somé et al., 1996) derived from single-spore isolates. In their study, RAPD profiles from 37 isolates representing seven pathotypes revealed a marker ($OPL14_{1200}$) consistently associated with highly aggressive pathotypes capable of infecting all differential hosts, highlighting the potential of DNA-based markers for the rapid identification of virulent strains. These early studies provided a proof of concept that genetic variation in *P. brassicae* could be linked to pathotype-specific virulence.

Subsequent research has expanded molecular pathotyping beyond RAPD to more robust and reproducible platforms, including SNP-based assays, quantitative PCR, and allelic discrimination technologies. Yang et al. (2018) identified extensive DNA sequence dimorphisms across multiple *P. brassicae* genes that distinguished 'old' pathotypes from newly emerged virulent populations capable of overcoming clubroot resistance and used these dimorphisms to develop an RNase H-dependent PCR (rhPCR) assay that differentiated resistance-breaking populations and revealed distinct pathogen lineages. Focusing specifically on resistance-breaking strains, Zhou et al. (2018) developed highly sensitive PCR- and TaqMan-based assays targeting the 18S–ITS region, enabling rapid and specific detection of pathotype 5-like populations (as defined on the Williams, 1966 differentials) directly from infected roots or soil, including at early stages of infection. More recently, genome-informed marker development has enabled SNP-based discrimination of *P. brassicae* CCD pathotype clusters using rhPCR and SNaPshot single-base extension assays, providing robust, high-throughput tools applicable to both single-spore isolates and field samples (Tso et al., 2022). In parallel, genetically distinct *P. brassicae* populations in Canada have been identified using RAD-seq, facilitating the development of population-specific molecular markers (Holtz et al., 2021). The increasing availability of *P. brassicae* genomic resources (e.g. Javed et al., 2024; Li et al., 2025; Sedaghatkish et al., 2025; Stjelja1 et al., 2019) is expected to further accelerate marker development for molecular pathotype identification across regions. Collectively, these approaches offer faster and more scalable alternatives to traditional bioassays and are particularly valuable for monitoring aggressive and resistance-breaking populations in regions where clubroot-resistant cultivars are widely deployed (Schwelm & Ludwig-Müller, 2021; Tso et al., 2021).

Challenges and future directions

Clubroot disease caused by *P. brassicae* is a critical concern in the cultivation of oilseed, brassica vegetable, fodder and forage, condiment, and ornamental crops, posing both agricultural and biological challenges (Dixon, 2014) and resulting in substantial economic losses (Dixon, 2009). The ECD continues to play a foundational role in the global understanding and management of *P. brassicae* pathotypes and has

done so for nearly five decades. While its original tripartite structure offered an effective framework for international collaboration and baseline pathotype comparison, subsequent adaptations, tailored to local crops, resistance sources, and production systems, have expanded its relevance and utility. The evolution of region-specific differential sets, such as the CCD in Canada and the Sinitic and Japanese systems in Asia, reflects the pathogen's dynamic nature and the agricultural contexts in which it thrives. Although calls for a standardised global differential set have resurfaced, the increasing integration of molecular and genomic tools suggests that future pathotype classification may shift from phenotypic to genetic platforms (Zhou et al., 2018). Nevertheless, the legacy of the ECD endures, not only in its scientific impact but also in its ongoing influence on breeding strategies, disease surveillance, and international cooperation in the fight against clubroot disease, which destroys brassica crops worldwide. Use of the ECD series has resulted in the identification of resistance genes in *Brassica* species as outlined by Dixon and Wells (2024).

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No potential conflict of interest was reported by the author(s).

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Author contributions statement

GRD: conceptualised the review, conducted the literature review; synthesised information, drafted the manuscript, and revised it based on feedback from the co-authors
SES: drafted the manuscript and its revision
ST: promoted the initial suggestion of a 'Lasting Legacy' and contributed to the manuscript
CA: contributed to the manuscript

Data availability statement

No data were generated or analysed during this study.

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