

Pathogen profile

***Puccinia coronata* f. sp. *avenae*: a threat to global oat production**ERIC S. NAZARENO¹, FENG LI¹, MADELEINE SMITH², ROBERT F. PARK³, SHAHRYAR F. KIANIAN⁴ AND MELANIA FIGUEROA^{1,5,*}¹Department of Plant Pathology, University of Minnesota, St. Paul, MN 55108, USA²Department of Plant Pathology, University of Minnesota-Northwest Research and Outreach Center, Crookston, MN 56716, USA³Plant Breeding Institute, The University of Sydney, Narellan, NSW 2567, Australia⁴Cereal Disease Laboratory, US Department of Agriculture-Agricultural Research Service, St. Paul, MN 55108, USA⁵Stakman-Borlaug Center for Sustainable Plant Health, University of Minnesota, St. Paul, MN 55108, USA**SUMMARY**

Puccinia coronata f. sp. *avenae* (*Pca*) causes crown rust disease in cultivated and wild oat (*Avena* spp.). The significant yield losses inflicted by this pathogen make crown rust the most devastating disease in the oat industry. *Pca* is a basidiomycete fungus with an obligate biotrophic lifestyle, and is classified as a typical macrocyclic and heteroecious fungus. The asexual phase in the life cycle of *Pca* occurs in oat, whereas the sexual phase takes place primarily in *Rhamnus* species as the alternative host. Epidemics of crown rust happens in areas with warm temperatures (20–25 °C) and high humidity. Infection by the pathogen leads to plant lodging and shrivelled grain of poor quality.

Disease symptoms: Infection of susceptible oat varieties gives rise to orange–yellow round to oblong uredinia (pustules) containing newly formed urediniospores. Pustules vary in size and can be larger than 5 mm in length. Infection occurs primarily on the surfaces of leaves, although occasional symptoms develop in the oat leaf sheaths and/or floral structures, such as awns. Symptoms in resistant oat varieties vary from flecks to small pustules, typically accompanied by chlorotic halos and/or necrosis. The pycnial and aecial stages are mostly present in the leaves of *Rhamnus* species, but occasionally symptoms can also be observed in petioles, young stems and floral structures. Aecial structures display a characteristic hypertrophy and can differ in size, occasionally reaching more than 5 mm in diameter.

Taxonomy: *Pca* belongs to the kingdom Fungi, phylum Basidiomycota, class Pucciniomycetes, order Pucciniales and family Pucciniaceae.

Host range: *Puccinia coronata sensu lato* can infect 290 species of grass hosts. *Pca* is prevalent in all oat-growing regions and, compared with other cereal rusts, displays a broad telial host range. The most common grass hosts of *Pca* include cultivated hexaploid oat (*Avena sativa*) and wild relatives, such as bluejoint grass, perennial ryegrass and fescue. Alternative hosts include several species of *Rhamnus*, with *R. cathartica* (common

buckthorn) as the most important alternative host in Europe and North America.

Control: Most crown rust management strategies involve the use of rust-resistant crop varieties and the application of fungicides. The attainment of the durability of resistance against *Pca* is difficult as it is a highly variable pathogen with a great propensity to overcome the genetic resistance of varieties. Thus, adult plant resistance is often exploited in oat breeding programmes to develop new crown rust-resistant varieties.

Useful website: <https://www.ars.usda.gov/midwest-area/st-paul-mn/cereal-disease-lab/docs/cereal-rusts/race-surveys/>.

Keywords: avirulence factors, buckthorn, crown rust, fungicides, genetic resistance, oat.

INTRODUCTION

Oat (*Avena sativa*) is the sixth largest cereal crop based on worldwide production [Food and Agriculture Organization (FAO), 2014], with Russia, Canada, the European Union, Australia and the USA as the major producers [US Department of Agriculture, Foreign Agricultural Service (USDA-FAS), 2017]. Although oat is primarily grown as a livestock feed crop, *A. sativa* ranks fourth amongst the cereals after wheat, rice and corn based on human consumption. Wholegrain oat products are usually attractive to consumers because of their impact on cholesterol levels in humans and their preservative-free properties, as well as high nutritional and fibre content (van den Broeck *et al.*, 2016; Burnette *et al.*, 1992).

Unfortunately, the global prevalence of *Puccinia coronata* f. sp. *avenae* (*Pca*) hinders oat production and reduces the economic value of the grain, as infection affects the grain yield, kernel weight and groat percentage (Doehlert *et al.*, 2001; Holland and Munkvold, 2001; Humphreys and Mather, 1996). Moreover, crown rust infection weakens straw production and causes oat plants to lodge (Endo and Boewe, 1958). The earliest documented account of *P. coronata* was made in 1767, as described by Tozzetti (1952), and, since then, several subdivisions have been suggested,

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including splitting into physiological forms (*formae speciales*), varieties and subspecies. Numerous classifications based on spore morphology, as well as telial and aecial hosts, have been described to reflect variations within *P. coronata sensu lato* (Brown, 1937; Cummins, 1971; Eriksson, 1908; Eriksson and Henning, 1894; Peturson, 1954; Urban and Markova, 1994); however, such assignments fail to represent genetic differences between isolates or true host ranges (Anikster *et al.*, 2003; Eshed and Dinooor, 1980). Nonetheless, some of these designations remain in use to indicate the major grass hosts, as in the case of *Pca*. Most recently, the implementation of molecular analysis has shown that *P. coronata* encompasses multiple species (Szabo, 2006) and suggests the division of the complex into seven species (Liu and Hambleton, 2013). For the purpose of this review, we refer to *Pca* as a pathogen affecting primarily cultivated and wild oat, as well as some grasses, such as *Lolium*, and which, in previous publications, has been denominated as *P. coronata* var. *avenae* f. sp. *avenae* (Liu and Hambleton, 2013) or *P. coronata* var. *avenae*, PcSP2 (Szabo, 2006). In this review, we summarize the current knowledge on this pathogen, including the economic importance and life cycle, as well as perspectives on rust virulence, disease management and future research directions.

ECONOMIC IMPORTANCE

Historical reports of damage to oat crops caused by *Pca* first appeared in the late 1800s. Crop failures as a result of crown rust infection were first reported in Europe (Cornu, 1880) and the Baltics (Sivers, 1887), just shortly before the pathogen was noted in the USA (Thaxter, 1890). Since then, oat crown rust epidemics have continued to affect oat production, resulting in 10%–40% yield losses (Behnken *et al.*, 2009; Martinelli *et al.*, 1994; Simons, 1970). Throughout the 20th century, crown rust epidemics have occurred intermittently in different regions of the world. Severe damage caused by this pathogen has been reported in South America (Gassner, 1916), Portugal (D'Oliveira, 1942), Australia (Waterhouse, 1952), Israel (Wahl and Schreiter, 1953), southeastern Europe (Kostic, 1959) and the USA (Sherf, 1954). Since the 1990s, epidemics of crown rust have occurred almost annually in Brazil and Uruguay (Leonard and Martinelli, 2005; Wahl and Schreiter, 1953). More recently, *Pca* has been reported to pose a serious risk to oat production in Tunisia (Hammami *et al.*, 2010) and Canada (Chong *et al.*, 2008).

In years in which the weather is not conducive to infection or inoculum pressure is not high, yield losses caused by crown rust can be as low as 5%. According to records at the US Department of Agriculture, Agricultural Research Service, Cereal Disease Laboratory (USDA-ARS CDL), oat crown rust epidemics remained relatively mild (less than 10% losses) in the USA from 1981 to 2013, with the exception of 1991 and 1993, when losses reached 15% in North Dakota, and, in 1997, when Minnesota and Louisiana

reported 20% losses (Long and Hughes, 2000). Severe losses occurred in the USA in 2014, when over 13 million bushels, equivalent to 18.7% of the country's oat production, were lost as a result of damage by *Pca*. During this epidemic, yield losses in the two major oat-producing states, Minnesota and South Dakota, were 50% and 35%, respectively (USDA-ARS CDL, 2014).

LIFE CYCLE AND INFECTION PROCESS

Puccinia coronata f. sp. *avenae* possesses five infectious stages that are associated with either sexual or asexual reproductive phases in its life cycle (Fig. 1) (Simons, 1970). In the Middle East, Europe and North America, where alternative hosts grow in close association with oat, both sexual and asexual stages of *Pca* exist (Dinooor, 1977; Simons, 1985; Wahl, 1970). In contrast, alternative hosts are uncommon or absent in East Africa, South America, Australia and New Zealand, and the pathogen is probably limited to a repetitive asexual stage in these regions (Harder and Haber, 1992; Simons, 1985). The asexual infection phase (telial stage) occurs entirely in oat, whereas sexual reproduction (aecial stage) takes place in alternative hosts (Dietz, 1926; Simons, 1985). The asexual phase involves repeated cycles of infection and sporulation mediated by urediniospores that can repeat as quickly as every 2 weeks (Fig. 2). At this stage, *Pca* is dikaryotic, with each single-celled urediniospore containing two haploid nuclei. The urediniospores germinate on the adaxial and abaxial leaf surfaces under suitable conditions (i.e. mild temperatures, adequate moisture and short exposure to light). Once germinated, these spores form appressoria and, subsequently, a penetration peg, which allows the fungus to penetrate the stoma and gain access to the mesophyll space of the leaf. A substomatal vesicle is formed in the stomatal cavity, from which infection hyphae originate, and hyphal tips elongate to produce specialized haustorial mother cells that develop into haustoria—structures essential for nutrient uptake (Staples and Macko, 1984). The intercellular branching of the infection hyphae proceeds until a fungal colony is formed in the surrounding leaf tissue, which, after 7–10 days, gives rise to sporulating uredinia that produce a new set of urediniospores. The uredinia emerge as bright orange–yellow oblong pustules (Fig. 2) that constitute the characteristic symptom of infection (Harder and Haber, 1992; Jackson *et al.*, 2008; Simons, 1970; Staples and Macko, 1984).

The sexual phase of *Pca* involves both oat and the alternative host, common buckthorn (*Rhamnus* spp.) (Fig. 1). Late in the cropping season, as the plant starts to senesce, rust infection sites differentiate teliospores (Fig. 2). These dikaryotic, thick-walled, survival structures germinate in the spring and undergo meiosis to produce haploid basidiospores, which subsequently infect growing buckthorn leaves (Mendgen, 1984; USDA-ARS CDL, 2017). Once in buckthorn, *Pca* completes additional developmental processes, resulting in the spermatial stage. At this stage, pycnia are formed on the adaxial surface of the leaf and produce pycniospores,

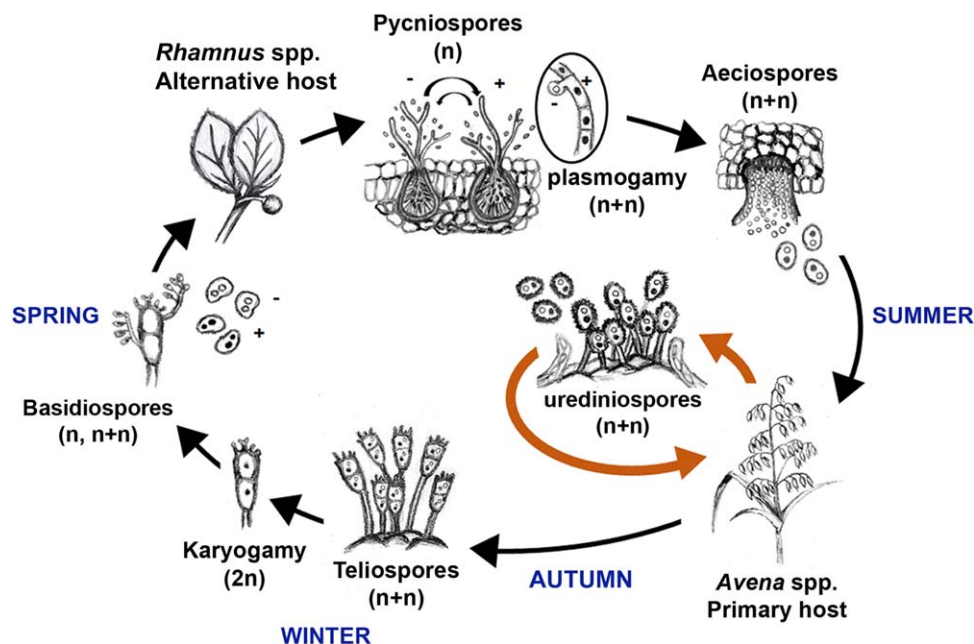


Fig. 1 Life cycle of *Puccinia coronata* f. sp. *avenae*. The asexual phase of the cycle (orange arrows) is completed in domestic and wild oat species during summer as multiple rounds of infection. The sexual phase of the cycle begins with the differentiation of teliospores in late summer or autumn to withstand cold winter temperatures. Teliospores undergo karyogamy and complete one meiotic event to produce basidiospores in the spring. Basidiospores carry either the (–) or (+) mating type and probably undergo mitosis prior to germination and infection of buckthorn (*Rhamnus* species). In buckthorn, the fungus differentiates pycniospores, which come into contact with neighbour hyphae from the opposite mating type, enabling plasmogamy. Aeciospores then form and infect oat to re-initiate the asexual cycle. Drawing by M. Figueroa.

which act as gametes to fuse with receptive hyphae and re-establish a dikaryotic stage in the fungus (Fig. 2) (Harder and Haber, 1992; Simons, 1985). After plasmogamy, aecium formation occurs on the abaxial surface, and these cylindrical-shaped structures produce aeciospores that re-infect the grass host (Figs 1 and 2) (Harder and Haber, 1992). Each of the haploid nuclei contributed by the two gametes remains in the aeciospores (dikaryotic), and therefore represents a complete haplotype genome inherited from a single parent.

Urediniospores and aeciospores are wind transmitted and can travel long distances (Jackson *et al.*, 2008). For instance, during early summer in North America, urediniospores from infected oat in Mexico and the southern USA may be blown northward to the north-central region of the USA and the provinces of Manitoba and Saskatchewan in Canada, a route known as the *Puccinia* pathway (Harder and Haber, 1992). During autumn, the wind may carry urediniospores south to infect winter oat (Forbes, 1939). However, in the northern USA, where buckthorn is abundant, inoculum from the alternative host probably plays a more important role in crown rust outbreaks than do urediniospores carried through the *Puccinia* pathway (USDA-ARS CDL, 2017). The northward movement of crown rust inoculum also occurs in Europe (Klenová-Jiráková *et al.*, 2010; Simons, 1985). Moreover, migrating birds have been shown to play a role in both the northward

and southward dispersal of spores during spring and autumn, respectively, and even across continents (da Silva *et al.*, 2016; Warner and French, 1970).

PATHOGENICITY AND POPULATION BIOLOGY OF *Pca*

The development of rust diseases results from a failure of the host plant's immune system to recognize the pathogen and activate defence responses. As a first line of defence, plant immunity involves the recognition of conserved pathogen components, such as chitin in fungi, known as pathogen-associated molecular patterns (PAMPs) (Dangl *et al.*, 2013; Dodds and Rathjen, 2010; Hogenhout *et al.*, 2009; Toruño *et al.*, 2016). However, adapted pathogens have evolved mechanisms to suppress or prevent the activation of these plant basal defences, often through the action of secreted effector proteins. Thus, as a second line of defence, plants have also evolved mechanisms to detect such effectors (Dangl *et al.*, 2013; Dodds and Rathjen, 2010; Petre and Kamoun, 2014; Toruño *et al.*, 2016). This latter plant recognition system (refers to Effector-Triggered Immunity, ETI) was initially characterized at the genetic level by H. H. Flor, who developed the gene-for-gene hypothesis based on interactions in the flax rust disease system (Flor, 1971). In this model, dominant resistance (*R*) genes in the host plant confer

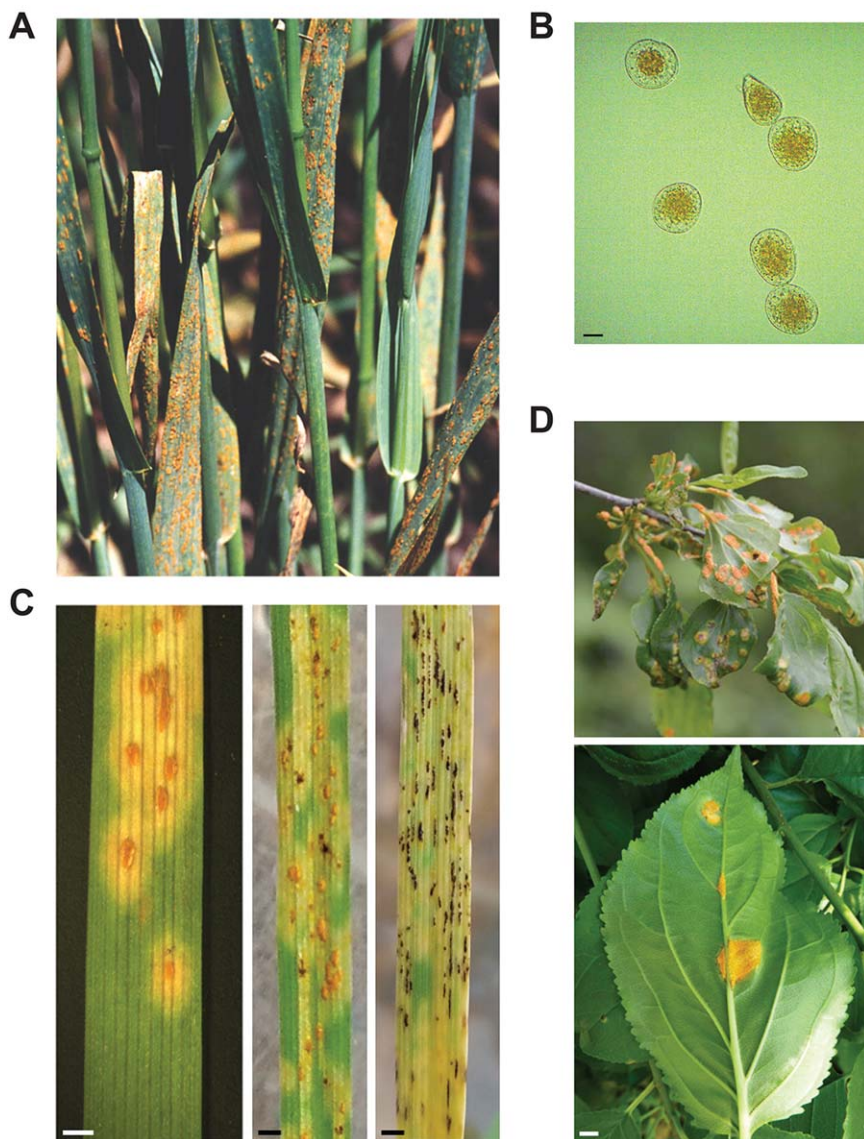


Fig. 2 Symptoms of oat crown rust infection. (A) Infection of oat by *Puccinia coronata* f. sp. *avenae* in the field. A high density of pustules (clusters of urediniospores) is shown in the leaves. Photograph by R. Caspers. (B) Micrograph of urediniospores; scale, 10 μ m. (C) Close-up image of pustules in susceptible oat variety *Marvelous* (left) and two stages of teliospore formation (middle and right); scale, 2 mm. (D) Photographs of crown rust in common buckthorn (*Rhamnus cathartica*). Symptoms are shown in the leaves and petioles. Pycnia are present on the adaxial surface of the leaf (top), whereas aecia form on the abaxial surface (top and bottom); scale, 2 mm. Courtesy photographs by R. Caspers and E. Byamukama.

recognition of specific avirulence (*Avr*) genes from the pathogen. This model has now been verified at the molecular level, with plant *R* genes found to encode intracellular immune receptor proteins mostly belonging to the nucleotide-binding leucine-rich repeat (NB-LRR) class (Dodds and Rathjen, 2010; Jones and Dangl, 2006). The *Avr* proteins recognized by these receptors are typically effector proteins that are delivered into host cells. Work in the flax rust system demonstrated that rust haustoria can deliver effector proteins to the host plant cell, which, if recognized by the corresponding *R* proteins, trigger immune responses and resistance (Dodds *et al.*, 2004, 2006; Garnica *et al.*, 2014). The relationship between crown rust resistance in oat and *Pca* virulence displays typical gene-for-gene characteristics, with resistance conferred by multiple single dominant resistance genes, each displaying different specificities towards

pathogen isolates (Chong *et al.*, 2000, 2008). To date, more than 100 crown rust resistance (*Pc*) genes have been described in oat (Table S1, see Supporting Information) (Gnanesh *et al.*, 2014; Graichen *et al.*, 2010; Tan and Carson, 2013; USDA-ARS CDL, 2016), although the lack of molecular information makes it difficult to determine whether some of these genes may be the same or are alleles at a common locus. In contrast with this wealth of *R* genes, no effectors have yet been identified in *Pca*, and the knowledge of rust pathogen *Avr* proteins remains limited to *Melampsora lini*, the causal agent of flax rust, and *Hemileia vastatrix*, the causal agent of coffee leaf rust (Maia *et al.*, 2017; Ravensdale *et al.*, 2011). Nonetheless, it is evident that virulence profiles can vary greatly amongst isolates of *Pca*, and this can only be explained by the existence of distinct effector/*Avr* gene repertoires in the pathogen.

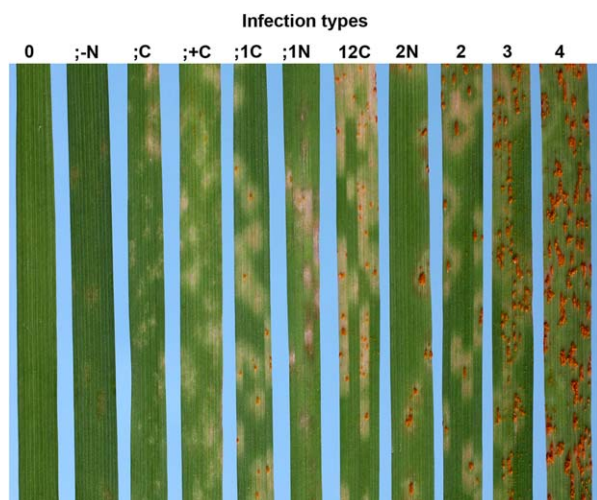


Fig. 3 Examples of infection types (ITs) on the disease rating scale as developed by Murphy (1935). ITs are used to score seedling resistance and to conduct physiological race assignments of *Puccinia coronata* f. sp. *avenae*. Incompatible reactions include the following: 0, no urediniospores; fleck (;), presence of flecks; 1, few small pustules; 2, small pustules, presence of green islands; all of these may be accompanied by necrosis (N) and/or chlorosis (C). Compatible reactions include the following: 3, large pustules surrounded by chlorotic halos; 4, large pustules, often coalescing. Those illustrated here (photographs by R. F. Park) were scored as, left to right: 0, ;-N, ;C, ;+C, ;1C, ;1N, 12C, 2N, 2, 3 and 4.

As documented by national and international surveys, *Pca* is one of the most pathogenically diverse cereal rust fungi, exhibiting the rapid emergence of new virulent isolates (Carson, 2011; Chong and Kolmer, 1993; Klenová-Jiráková *et al.*, 2010; Park, 2008; Simons, 1985). Pathogen characterization is conducted on a set of oat genotypes carrying one or more *Pc* genes in various genotypic backgrounds to define pathogenicity based on compatible (virulent) or incompatible (avirulent) interactions, each assigned to a physiological race (Chong *et al.*, 2000; Leonard *et al.*, 2004; van Niekerk *et al.*, 2001). The response of each differential genotype to an isolate is recorded using a 0–4 infection type (IT) scale (Fig. 3), with the cut-off between compatible and incompatible usually at either IT 3 or >3. Simons and Michel (1964) reported about 400 physiological races of *Pca* worldwide (Harder and Haber, 1992), which clearly demonstrates the pathogenic variability of the pathogen. The number of *Pc* genes represented in the differential sets used to identify pathotypes (races) in surveys varies among countries, and has been updated over time in response to the rise of new virulences (Chong *et al.*, 2000). For instance, in the USA, the USDA-ARS CDL utilizes a set of 40 oat differentials. There is no universal set of differentials used across research institutions, although there is some overlap of both *Pc* genes and oat differentials (Table 1).

The deployment of race-specific seedling genes has been a method of choice to mitigate oat crown rust damage in the field.

Unfortunately, this strategy has not delivered resistance durability and has resulted in boom-and-bust cycles as pathogen populations evolve virulence to previously resistant varieties over time (Chong and Seaman, 1997; Chong and Zegeye, 2004; McCallum *et al.*, 2007; McDonald and Linde, 2002; Park *et al.*, 2000). These shifts occur as a result of the selective pressure exerted by the limited genetic diversity in the crop on the rust population, leading to a change in virulence frequencies within populations. Thus, the evolutionary capacity of the pathogen should be considered in disease management strategies. Alternative hosts play an important role in the epidemiology and evolution of pathogenicity in some rust species, as they act as a reservoir for the pathogen and allow sexual recombination, which leads to a more diverse population (Zhao *et al.*, 2016). Given that buckthorn is prevalent in North America, high rates of sexual recombination probably account for the extremely polymorphic *Pca* populations and the rapid evolution of new virulent pathotypes (Leonard, 2002).

Studies in North America have shown that race diversity is greater amongst isolates collected on *Rhamnus carthartica* or in oat found in close proximity to *R. carthartica* (Fleischmann, 1965; Murphy, 1935), suggesting that the alternative host may play a role in generating the genetic diversity of *Pca*. Nevertheless, high levels of pathogenic variation in *Pca* are not exclusive to sexual populations, as asexual populations in regions in which the alternative host is absent or rare, such as Australia, are also highly polymorphic (Park, 2013; Simons, 1985). In Australia, surveys from 1998 to 2010 identified more than 100 *Pca* races, much higher than found for the same period of time in other cereal rust species that are also not sexually active (Park, 2008; Park and Wellings, 2012). These observations raise questions about the role of buckthorn in the pathogenic diversity of *Pca* and whether other factors, such as repeated introductions, high mutation rates or somatic hybridization and recombination, are also important contributors to the diversity and virulence in *Pca* populations (Bartos *et al.*, 1969; Klenová-Jiráková *et al.*, 2010; Park and Wellings, 2012; Simons, 1970). It is possible that the genetic diversity of *Pca*, resulting from the re-assortment of pathogenicity factors through sexual reproduction, can be further enhanced by multiple molecular mechanisms that introduce variability into asexual populations (Simons, 1985).

The characterization of the genetic diversity of *Pca* at the molecular level is limited. A preliminary analysis of 12 Australian *Pca* isolates using DNA amplification fingerprinting markers detected two pathogen subpopulations, indicating two distinctive exotic rust isolate introductions (Brake *et al.*, 2001). Klenová-Jiráková *et al.* (2010) used an amplified fragment length polymorphism (AFLP) analysis to evaluate the genetic diversity of 40 *Pca* isolates collected from seven different countries (Israel, Czech Republic, Belarus, Estonia, Hungary, Austria and Serbia), which confirmed the high genetic diversity of the species, as each isolate

Table 1 Oat differential genotypes used in the USA, Australia, Canada and Brazil to assign pathotypes of *Puccinia coronata* f. sp. *avenae*.

Gene(s)	Australia	USA	Brazil*	Canada†
<i>Pc1</i>	X			
<i>Pc3c, Pc4c, Pc6c, Pc9</i>	X			
<i>Pc4, Pc5</i>	X			
<i>Pc6, Pc7, Pc8, Pc21</i>	X			
<i>Pc6d</i>	X			
<i>Pc14</i>		X	X	
<i>Pc15, Pc16, Pc17</i>	X			
<i>Pc35</i>		X	X	
<i>Pc36</i>	X	X	X	
<i>Pc38</i>	X	X	X	X
<i>Pc38, Pc39, Pc52</i>	X			
<i>Pc39</i>	X	X	X	X
<i>Pc39, Pc61, PcBettong</i>	X			
<i>Pc40</i>		X	X	X
<i>Pc45</i>	X	X	X	X
<i>Pc46</i>	X	X	X	X
<i>Pc48</i>	X	X	X	X
<i>Pc48+</i>	X			
<i>Pc48, Pc56</i>	X			
<i>Pc50</i>	X	X	X	X
<i>Pc50+</i>	X			
<i>Pc51</i>	X	X	X	X
<i>Pc52</i>	X	X	X	X
<i>Pc53</i>		X	X	
<i>Pc54</i>		X	X	X
<i>Pc55</i>	X	X	X	
<i>Pc56</i>	X	X	X	X
<i>Pc56+</i>	X			
<i>Pc57</i>		X	X	
<i>Pc58</i>	X	X	X	X
<i>Pc59</i>	X	X	X	X
<i>Pc60</i>	X	X	X	
<i>Pc60, Pc61</i>	X			
<i>Pc61</i>		X	X	
<i>Pc61+</i>	X			
<i>Pc61, PcBettong</i>	X			
<i>Pc62</i>	X	X	X	X
<i>Pc63</i>	X	X	X	
<i>Pc64</i>	X	X	X	X
<i>Pc67</i>		X	X	
<i>Pc68</i>	X	X	X	X
<i>Pc70</i>		X	X	
<i>Pc71</i>	X	X		
<i>Pc91</i>	X	X		X
<i>Pc92</i>	X			
<i>Pc94</i>	X	X		X
<i>Pc96</i>		X		X
<i>Pc97</i>				X
<i>Pc98</i>				X
<i>Pc101</i>				X
<i>Pc103-1</i>				X
<i>Pc104</i>				X
<i>PcH546</i>			X	
<i>PcH548</i>	X			
<i>PcMortlock, PcCulgoa</i>	X			
<i>PcWIX1, PcWIX2</i>	X			
Unknown (Bondvic)	X			

Table 1 *Continued*

Gene(s)	Australia	USA	Brazil*	Canada†
Unknown (X716)	X			
Unknown (Marvelous)		X		
Unknown (H548)		X		
Unknown (IA B605X sel.)		X		
Unknown (WI X4361-9)		X		
Unknown (TAM-O-405)		X		
Unknown (Belle)		X		
Unknown (HiFi)		X		
Unknown (Leggett)		X		
Unknown (Stainless)		X		
Total	42	40	28	24

*Leonard and Martinelli (2005); Vieira *et al.*(2007).†Chong *et al.* (2000, 2011); Menzies *et al.* (2015).

presented a unique AFLP molecular pattern, although genetic similarity was found for those isolates from the Czech Republic, Austria and Serbia. To our knowledge, simple sequence repeat-expressed sequence tag (SSR-EST) markers have only been developed to study the genetic diversity of *Puccinia coronata* f. sp. *lolii* (pathogen of ryegrass) (Dracatos *et al.*, 2006), but not for *Pca*. Similarly, the development of single nucleotide polymorphism (SNP) markers to conduct genetic studies in *Pca* has not been implemented, as EST or genomic sequences are not yet available for this pathogen.

DISEASE MANAGEMENT APPROACHES

Management measures to prevent crown rust outbreaks include the use of biocontrol agents, removal of the alternative host, development of varieties with resistance and application of fungicides (Fig. 4) (Hoffman *et al.*, 2006; McCallum *et al.*, 2007; Simons, 1970).

Common buckthorn is a shrub native to Europe, northwest Africa and western Asia, and was first introduced to the USA in the mid-1800s as an ornamental plant and windbreak. The species quickly spread through the upper midwestern and northeastern regions of the USA and has now reached Saskatchewan and the Maritime provinces of Canada (Knight *et al.*, 2007). The invasiveness of buckthorn in forests, prairies and savannas has led to its classification as a restricted noxious weed in parts of both the USA and Canada [US Department of Agriculture, Natural Resources Conservation Service (USDA-NRCS), 2017], and has awakened interest in the launch of stringent eradication programmes to reduce its ecological impact. However, a significant challenge to these measures is to successfully prevent the re-emergence of buckthorn, particularly in remote areas. The USA, together with other countries, including Canada, Kenya, Latvia, Estonia and Russia, where buckthorn has become a problem, have passed legislation to prevent the dissemination and encourage the eradication of this invasive weed (Sherf *et al.*, 1956), which has resulted in a

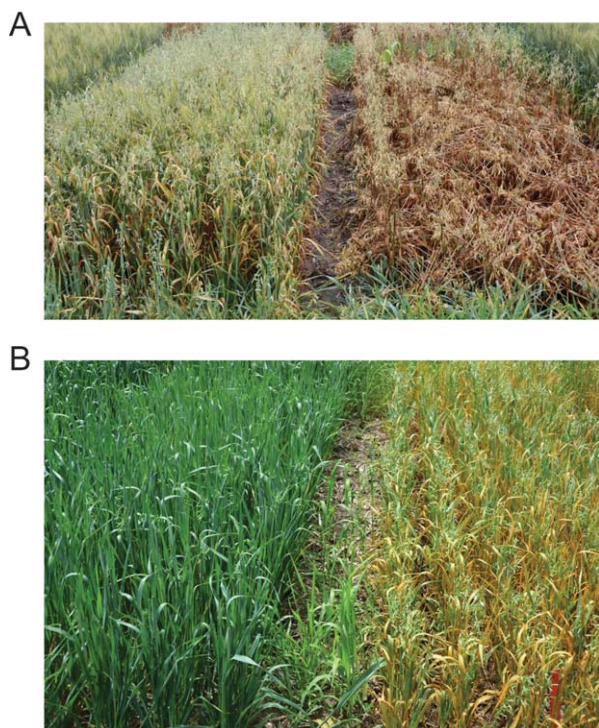


Fig. 4 Crown rust management strategies. (A) Effect of fungicide treatment in an oat field. Right section of the field depicts damage of the pathogen in the absence of fungicide treatment on a susceptible variety, in contrast with the left side of the field that was treated with fungicide. (B) Effect of genetic resistance in the field. Photograph illustrates the positive effect of using the oat variety Deon versus a variety that does not contain genetic resistance. Deon was selected from the cross Sesqui \times 2/Bettong/MN02108 and was released by the Minnesota Agricultural Experiment Station in 2012. Courtesy photographs by R. Caspers and E. Byamukama.

great reduction in buckthorn in places such as Iowa (Simons, 1985). Currently, the biological control of buckthorn using insects is also being explored in North America and Europe [Centre for Agriculture and Bioscience International (CABI), 2015].

Oat crown rust can reduce grain yield by as much as one-half in susceptible varieties, which may be mitigated by fungicide application (Martinelli *et al.*, 1994; Picinini and Fernandes, 1994). May *et al.* (2014) demonstrated that fungicide application improved both the yield and grain quality in crown rust-susceptible varieties, when sprayed after the flag leaf had fully emerged, but the more resistant variety, 'Leggett', showed no yield response to fungicide application. The decision to apply fungicide is often influenced by cost–benefit analysis, dictated by grain value and fungicide and application costs. For instance, in Minnesota, where oat is largely grown for feed, the grain has a lower value compared with that used for food and milling, most often making chemical intervention uneconomic. Nevertheless, in 2015, oat crown rust infections in Minnesota became so severe that, in the following growing season, many farmers applied

fungicide to the crop for the first time. The number of fungicides that control crown rust is limited, not just for lack of effective chemistry, but because some fungicides are not labelled for use in oat in some regions. Products containing pyraclostrobin or azoxystrobin (alone or in combination), or a combination of propiconazole and trifloxystrobin can control the pathogen (Behnken *et al.*, 2009). However, some of these products have restrictions that prevent application 30–40 days prior to harvest, making it difficult to time the application for disease control without encroaching on the mandated labelling regulations.

The high evolutionary capacity of *Pca* and the increasing use of fungicides in oat raise the prospect of fungicide insensitivity in this pathogen. A recent evaluation by Oliver (2014) argues for the classification of rust fungi within the high-risk group of pathogens to evolve fungicide insensitivity. The polycyclic and airborne nature of rust fungi strongly resembles that of pathogens which have developed fungicide insensitivity in the past. Reduced sensitivity to demethylation inhibitor and quinone outside inhibitor fungicides has been detected in *Puccinia triticina*, the wheat leaf rust pathogen (Arduini *et al.*, 2012). Similarly, the monitoring of fungicide effectiveness for more than a decade in *Phakopsora pachyrhizi*, the causal agent of Asian soybean rust, demonstrated a reduction in fungicide sensitivity over time (Godoy *et al.*, 2016).

It is worth noting that, although fungicides can reduce the impact of rust, their use comes with environmental and economic costs, as well as risks to human health. Research on the impact of fungicide applications on yield, grain quality, milling characteristics and plant development is still required. Finally, oat occupies an important niche in organic production systems, where chemical treatments are not permitted, and thus there is increasing market pressure to avoid the use of fungicides and to employ alternative approaches.

GENETIC RESISTANCE TO OAT CROWN RUST

Oat crown rust resistance may be mediated by race-specific or non-race-specific mechanisms (Carson, 2011; Leonard, 2002; Periyannan *et al.*, 2017). Race-specific resistance is underpinned by the concept of the gene-for-gene interaction, which has been fundamental in the breeding of disease-resistant crop varieties (Dodds and Rathjen, 2010; Flor, 1971), and has been extensively exploited in oat breeding programmes to control crown rust since the early 1900s (Ohm and Shaner, 1992). This type of resistance depends on highly heritable single genes that typically elicit a hypersensitive response, which either completely or partially inhibits rust sporulation (Ohm and Shaner, 1992). The use of seedling resistance against *Pca* was intensified in the USA in the late 1920s with the introduction of the highly resistant oat varieties 'Victoria' from Uruguay and 'Bond' from Australia (Ohm and Shaner, 1992; Simons, 1985). As oat with the Victoria-type resistance (*Pc2* and *Pc11*) showed strong protection against crown rust

(Coffman, 1977; Simons, 1970), breeding efforts were rapidly undertaken to transfer the resistance from Victoria to US-adapted oat germplasm. The first varieties carrying the Victoria-derived resistance were released in 1940. Unfortunately, such success was short lived as these varieties were quickly devastated by Victoria blight caused by *Helminthosporium victoriae* in 1946 (Murphy and Meehan, 1946), as *Pc2* had the pleiotropic effect of making lines with this gene highly susceptible to Victoria blight (Lorang *et al.*, 2007). Varieties carrying the Bond-derived resistance (*Pc3* and *Pc4*), as well as other varieties, such as 'Landhafer', 'Ukraine' and 'Santa Fe', replaced Victoria-derived varieties after the epidemic of *H. victoriae*. The resistance of these varieties was eventually overcome with the emergence of new crown rust races (Simons, 1985). Learning from these lessons, oat breeding programmes have been reluctant to use single sources of resistance since the mid-1960s (Simons, 1985). Nevertheless, the field durability of crown rust-resistant varieties continues to be challenged in the USA (Carson, 2011), Canada (McCallum *et al.*, 2007), Australia (Park, 2008), and Brazil and Uruguay (Leonard and Martinelli, 2005).

There are many examples that illustrate the limited durability of *Pc* genes. For instance, *Pc38* and *Pc39* were released in the early 1980s and were used together; however, by the end of the decade, virulence to these genes, individually and in combination, was reported (Chong and Seaman, 1997; McCallum *et al.*, 2007). Similarly, *Pc48*, which was first deployed in the 1990s, was defeated in 2001 (Chong and Zegeye, 2004). Two *R* genes that have been fairly effective are *Pc68* and *Pc91* (McCallum *et al.*, 2007; McCartney *et al.*, 2011; Rooney *et al.*, 1994). Varieties carrying *Pc68* were released in the early 1990s in Canada and remained effective until 2001. Later, *Pc68* was combined with *Pc94* in the variety 'Leggett', which was released in 2004 (McCallum *et al.*, 2007). However, virulence to *Pc94* has now been reported (Chong *et al.*, 2011; R. F. Park, unpublished). Only a few races have been identified in the USA that overcome *Pc91* and, for this reason, there is interest in the use of this gene in combination with other *Pc* genes (Chong *et al.*, 2008). However, *Pc91* lacked durability when deployed in Australia, with virulence detected 6 years after its release in the variety Drover (Park, 2013). Two years later, two further virulent pathotypes had been detected and approximately 35% of isolates analysed from eastern Australia were virulent for *Pc91* (R. F. Park, unpublished). Notwithstanding, gene pyramiding is seen as a valid strategy for the use of race-specific genes to more effectively extend the durability of *R* genes. The effectiveness of this approach will probably depend on the genes, the number of genes in use and whether or not such virulence combinations already exist in the pathogen population. For example, virulence on the variety 'AC Assiniboia', which contains three genes (*Pc38*, *Pc39* and *Pc68*), was detected only 4 years after its release in North America (Leonard, 2007;

McCallum *et al.*, 2007). The same variety was released in Australia in 1999, under the name 'Graza 68', and virulence was detected just 2 years later (R. F. Park, unpublished). Thus, a priori knowledge of the pathogenic diversity of the pathogen population may be helpful to better inform decisions about gene pyramiding schemes to maximize resistance durability.

In addition to race-specific resistance, oat breeding programmes have benefited from non-specific quantitative resistance, often known as adult plant resistance (APR) or partial resistance (Ohm and Shaner, 1992). APR does not typically manifest at the seedling stage, and crop protection comes from the reduction in fungal sporulation or delay of symptom appearance (Díaz-Lago *et al.*, 2003; Jones, 1978; Lin *et al.*, 2014; Portyanko *et al.*, 2005). As such, APR is usually effective against all or at least a wide range of rust genotypes, a characteristic that is highly attractive in cases such as crown rust where so many pathotypes exist. The identification and characterization of APR genes in oat against *Pca* has been difficult because of the complexity of inheritance or the presence of race-specific resistance within a single oat variety (e.g. Victoria, Santa Fe and Ukraine) (Klos *et al.*, 2017; Loarca *et al.*, 2016; Upadhyaya and Baker, 1960). In other instances, inheritance of APR can be less complex; for example, Victoria/Garry oat contain only two genes, *Pc27* and *Pc28*, contributing to the overall APR phenotype (Simons *et al.*, 1978; Upadhyaya and Baker, 1960). The Canadian *A. sterilis* accession 1387 is a case in which a single partially dominant gene, *Pc69*, controls APR (Harder *et al.*, 1984). Currently, six genes have been designated to be APR-conditioning genes in oat, namely *Pc27*, *Pc28*, *Pc69*, *Pc72*, *Pc73* and *Pc74* (Table S1) (USDA-ARS CDL, 2016), and more than 25 quantitative trait loci (QTLs) associated with APR have been mapped in oat (Acevedo *et al.*, 2010; Babiker *et al.*, 2015; Barbosa *et al.*, 2006; Jackson *et al.*, 2007; Lin *et al.*, 2014; Portyanko *et al.*, 2005; Zhu and Kaeppler, 2003).

In contrast with race-specific resistance, which often does not remain effective for more than 5 years in the field (Carson, 2011), APR can provide rust protection for extended periods of time. One example that illustrates such resistance durability is the variety 'Red Rustproof' (*A. byzantina* K. Koch), an oat introduced into the USA in the 1860s, which served as a progenitor of most winter oat in the country (Stanton, 1955). Red Rustproof oat have several strains that were widely used in the field for many years as some carried consistently effective crown rust resistance for more than 100 years (Luke *et al.*, 1972). The resistance in Red Rustproof oat is thought to be primarily associated with APR because both late-rusting (delayed symptom development) and slow-rusting (low disease severity) traits are manifested. According to genetic studies of the variety Red Rustproof-14, these resistance traits are controlled by a few genes with high heritability (Luke *et al.*, 1972). However, Red Rustproof oat also carry race-specific resistance, as hypersensitive-like responses to some races of *Pca*

occurs (Luke *et al.*, 1972). Such race specificity could perhaps be explained by the presence of uncharacterized race-specific genes, in addition to the gene *Pc1*, which was isolated in Red Rustproof oat and shown to be linked to *Pc2* (Davies and Jones, 1927; Dietz and Murphy, 1930) (Table S1).

A second example of durability is provided by the oat line MN841801, which exhibits APR and has remained partially resistant to crown rust infection for over 30 years (Leonard, 2002). MN841801 was developed by the oat breeding programme at the University of Minnesota in the 1960s, and was the result of a cross between 65B663 (a selection from Florad/58-7, originally from Coker Pedigreed Seed Company) and 65B1362, both highly resistant lines (Leonard, 2002). Over the years, the durable resistance of MN84801 has attracted much attention and several studies conducted by independent groups to map the QTLs associated with the resistance phenotype reached different conclusions. The inheritance of APR in MN841801 was examined by Chong (2000), who used recombinant inbred lines (RILs) from the cross AC Assiniboia/MN841801, and found two complementary APR genes effective against a specific pathotype of *Pca*. In contrast, in a cross between MN841801-1 (a reselection of MN841801) and 'Noble-2' (a reselection of 'Noble'), molecular markers allowed the identification of four major and three minor QTLs that control crown rust resistance (Acevedo *et al.*, 2010; Portyanko *et al.*, 2005), whereas another study using 6K oat SNPs and Kompetitive allele-specific PCR (KASP) found only one major QTL in the Assiniboia/MN841801 RIL population (Lin *et al.*, 2014). Efforts to determine the underlying mechanism of APR in MN841801 still continue and have extended to comparative transcriptome analysis of MN841801 and Noble-2 (Loarce *et al.*, 2016). Such transcriptome profiling comparisons reveal an interesting list of defence-related genes that are up-regulated during crown rust infection, including those for signal perception and transduction, hormone production and cell wall modification, amongst others.

Another source of APR that has been studied is 'TAM O-301', a variety developed from a cross between a Texas-adapted *A. sativa* and the *A. sterilis* accession PI295919 (Hoffman *et al.*, 2006). TAM O-301 carries the *R* gene *Pc58* (Simons *et al.*, 1978) and, according to results using restriction fragment length polymorphism (RLFP) and AFLP markers in a 'Ogle'/TAM O-301 RIL mapping population, the resistance from TAM O-301 is also controlled by two additional genes, probably responsible for the APR phenotype (Jackson *et al.*, 2007, 2008; Portyanko *et al.*, 2001). However, TAM O-301 could also contain more than one seedling resistance gene (R. F. Park, unpublished). As exemplified by QTL identification in the oat lines MN841801 and TAM O-301, the detection of genes for quantitative resistance is expedited by new marker technologies that are proving advantageous in the alleviation of mapping complexities. Newly developed high-throughput genetic marker assay systems, such as SNP, KASP and diversity

arrays technology (DART), are of benefit in oat breeding, with the construction of dense genetic maps enabling the discovery of additional resistance QTLs against crown rust in different oat crosses and RILs (Babiker *et al.*, 2015; Barbosa *et al.*, 2006; Chaffin *et al.*, 2016; Jackson *et al.*, 2007; Zhu and Kaeppeler, 2003). In parallel, such systems are also used for genome-wide association study (GWAS) as an alternative route to locate crown rust resistance QTLs (Klos *et al.*, 2017; Montilla-Bascón *et al.*, 2015). Apart from their application in marker-assisted selection, molecular markers are essential for map-based cloning of resistance genes (Cabral *et al.*, 2014) as a basic step towards the determination of the mechanisms by which these genes exert their function. In this context, recent advances in genotyping by sequencing (GBS) and RNA sequencing (RNA-seq) in oat (Gutierrez-Gonzalez *et al.*, 2013; Huang *et al.*, 2014) have provided essential resources to enable functional studies of candidate genes.

FINDING NEW SOURCES OF RESISTANCE AGAINST OAT CROWN RUST

The discovery of diverse resistance genes in *A. sterilis* certainly made important contributions towards the exploitation of multiple gene resistance in breeding programmes (Leonard, 2007; McCallum *et al.*, 2007; Simons, 1985). Indeed, more than 45 effective *Pc* genes were obtained from *A. sterilis*, including *Pc38*, *Pc39*, *Pc68* and the majority of the APR genes. Several other wild oat species have been screened and used to identify new resistance genes (Aung *et al.*, 2010; Cabral and Park, 2016; Cabral *et al.*, 2011; Mitchell Fetch *et al.*, 2007; Rooney *et al.*, 1994). Recent sources of both race-specific and APR include *A. strigosa*, *A. glabrota*, *A. trichophylla*, *A. longiglumis*, *A. magna* and *A. murphyi* (Cabral and Park, 2014; Sowa *et al.*, 2016; USDA-ARS CDL, 2016); some of these sources originated from Moroccan and Israeli collections (Dinoor, 1970; Tan and Carson, 2013; Wahl, 1970). Carson (2010) screened accessions of *A. barbata* from the Mediterranean region and found some additional sources of resistance for both seedling and adult stages, including a new type of 'blotchy' reaction in adult plants. Thus, it is unlikely that resistance resources from wild species have been exhausted, and systematic evaluations of seed banks could enhance current management approaches. In a similar framework, non-host resistance holds promise to provide quantitative and durable disease resistance against a broad spectrum of pathogens (Bettgenhaeuser *et al.*, 2014; Figueroa *et al.*, 2015; Heath, 2000). *Brachypodium distachyon*, a temperate grass that belongs to the Pooideae subfamily, is closely related to oat (Gutierrez-Gonzalez and Garvin, 2011; Kellogg, 2001) and has been shown to harbour resistance against multiple wheat cereal rusts (Ayliffe *et al.*, 2013; Dawson *et al.*, 2015; Figueroa *et al.*, 2013). Thus, it is worthwhile to examine its potential against crown rust to unravel genes or loci that are associated with either disease resistance or susceptibility using modern biological techniques such as genome editing.

FUTURE RESEARCH DIRECTIONS AND PERSPECTIVES

To date, no seedling or APR resistance gene has been cloned from oat. However, current efforts to assemble genome references for *A. sativa* and *A. strigosa* will deliver improvements in molecular and genetic tools that will facilitate the identification and mapping of known resistance genes or QTLs. Furthermore, new approaches, such as mutagenesis resistance gene enrichment (MutRenSeq), developed to rapidly clone disease resistance genes in wheat (Steuernagel *et al.*, 2016), offer promise for the cloning of oat resistance genes. Such efforts will lead to valuable tools for the introgression and/or pyramiding of resistance genes into elite oat germplasm and help to develop new varieties with enhanced resistance against crown rust.

Wild relatives are a known reservoir of genetic diversity that has been extremely advantageous to oat breeding programmes in the past, and there should be further efforts to screen germplasm collections. Nevertheless, we must keep in mind that the introgression of traits from alien germplasm can be difficult and extremely laborious. Lack of synteny between species could translate into suppressed recombination, failure to achieve chromosome pairing and hybridization incompatibility, amongst other challenges (Wulff and Moscou, 2014). However, these can be overcome by the employment of special techniques, such as embryo rescue, the development of synthetic hexaploids and colchicine treatment coupled with backcrossing to improve fertility (Loskutov and Rines, 2011; Rines *et al.*, 2007). The generation of resistance gene cassettes is one option to stack alien resistance genes, but this would require the development of genetically modified (GM) oat varieties. Like other GM crops, such an approach would face significant hurdles in terms of public acceptability, especially given that oat occupies a health food niche in the market. Thus, as we conceive viable paths towards sustainable oat production and health, gene editing should be considered as a powerful tool to access untapped genetic sources and make advances in the management of this devastating disease. The identification of susceptibility factors may provide opportunities to engineer broad-spectrum resistance by the modification of gene alleles in oat. Three naturally occurring examples that support the feasibility of this approach are the *mlo* resistance in barley against powdery mildew (Humphry *et al.*, 2006), as well as *Lr34*- and *Lr67*-mediated resistance in wheat against rust and powdery mildew (Krattinger *et al.*, 2009, 2011; Moore *et al.*, 2015). In the case of *mlo* resistance, recessive *mlo* resistance alleles of the *Mlo* locus are responsible for non-race specific resistance, and the simultaneous modification of all three *Mlo* homoeoalleles in wheat resulted in broad-spectrum resistance to *Blumeria graminis* f. sp. *tritici* (Wang *et al.*, 2014). Likewise, *Lr34* and *Lr67* illustrate how simple amino acid changes in a putative adenosine triphosphate-binding cassette transporter and a hexose

transporter, respectively, can confer broad-spectrum resistance (Dodds and Lagudah, 2016; Krattinger *et al.*, 2011). Current work to make precise genome edits using Clustered Regularly Interspaced Short Palindromic Repeat/CRISPR Associated protein 9 (CRISPR/Cas9), coupled with studies to determine susceptibility factors in oat, will open up doors to control crown rust at a global scale.

Little is known about the mechanisms that drive genetic variability in *Pca*; however, efforts are underway to generate high-quality reference genomes for this species to enable comparative genomics and effector gene discovery. Once these resources are in place, population genomics studies will shed light into the genetic diversity of *Pca*. Such studies may determine the contribution of sexual recombination and other factors to the rapid evolution of this pathogen. An understanding of the diversity of *Pca* is essential in staying ahead of the pathogen's ability to overcome *R* genes and in the prevention of large-scale epidemics, which will negatively impact oat growers. Thus, monitoring of the population dynamics and virulence shifts of *Pca* should be an important component to design strategies to control crown rust.

CONCLUSIONS

In the past few years, the management of oat crown rust has become more difficult as the pathogen has evolved virulence to most of the *R* genes deployed in the field (Carson, 2008, 2011; Park, 2008). The future release of a genome reference for *A. sativa* and other relatives will be key to the acceleration of the identification of novel disease resistance genes and will allow us to design more effective disease management strategies. Although there might not be a silver bullet to achieve durable resistance, we must consider the importance of protecting current research and development investments by avoiding the deployment of single *R* genes in the field. This will require strategic planning of effective gene combinations, and tailored deployment of resistant varieties amongst geographical regions. Ongoing developments in molecular genetics and genomics tools are expected to have a tremendous impact in improving our understanding of the genetic basis of resistance to crown rust in oat, pathogenicity in *Pca* and the host–pathogen interaction. The high genetic variability of *Pca* makes this pathogen an ideal model to investigate virulence evolution in rust fungi.

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SUPPORTING INFORMATION

Additional Supporting Information may be found in the online version of this article at the publisher's website:

Table S1 Oat resistance genes against *Puccinia coronata* f. sp. *avenae*.