REVIEW

Molecular characterization of disease resistance in *Brassica juncea* – the current status and the way forward

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Brassica juncea (Indian mustard) is an economically important cultivated annual crop worldwide. However, there are a number of important diseases affecting *B. juncea*, including blackleg (*Leptosphaeria maculans*, *L. biglobosa*), sclerotinia stem rot (*Sclerotinia sclerotiorum*), white rust (*Albugo candida*), alternaria blight (*Alternaria brassicae*, *A.*

brassicicola, *A. raphani*), downy mildew (*Hyaloperonospora brassicae*), white leaf spot (*Neopseudocercosporella capsellae*), clubroot (*Plasmodiophora brassicae*), powdery mildew (*Erysiphe cruciferarum*), *Turnip yellows virus* (formerly *Beet western yellows virus*), *Cauliflower mosaic virus*, *Turnip mosaic virus*, and leaf blight (*Pseudomonas syringae*). This paper reviews what is currently known about disease resistance in *B. juncea*, including the mechanism of resistance and molecular markers associated with disease resistance, that can be used to develop improved *B. juncea* cultivars through marker-assisted selection (MAS). It also highlights how MAS, phenotypic selection, and transgenics provide pathways to validate candidate genes as functional resistance genes and new resources for breeding programmes for elite *B. juncea* cultivars. In addition, this review of disease resistance in *B. juncea*, together with the release of the *B. juncea* genome, will guide further discovery and identification of resistance genes and consequent substantial improvement in crop protection.

1 Introduction

Indian mustard (*Brassica juncea*) is an allotetraploid (AABB, 2n = 18), formed by hybridization between the A and B genome diploid *Brassica* species *B. rapa* and *B. nigra*, respectively. Yang *et al*. (2016) recently sequenced the *B. juncea* genome and reported a genome size of 954.90 Mb, providing the first *Brassica* B genome assembly. *B. juncea* is an important winter season oilseed crop (Kumar, 2012) that is also grown as a condiment crop and as a green vegetable (Rakow, 2004). It is cultivated worldwide, with India, Canada, Nepal, Russia, Ukraine, Pakistan, and Bangladesh all being major seed and oil producers (Skrypetz, 2007; FAO, 2016). *B. juncea* is an alternative oilseed crop to canola (*B. napus*) in lower rainfall regions, due to its greater tolerance to drought stress and high temperatures, and reduced pod-shattering (Oram *et al*., 2005; Gunasekera *et al*., 2006) . Canola-quality *B.*

juncea production began in 2002 in Canada (Potts *et al*., 2003) and later in 2007 in Australia (Burton *et al*., 2007), opening new opportunities for farmers and grain industry focusing on high quality food production.

Plant diseases adversely affect crop production worldwide. Plant resistance mechanisms have been extensively reviewed across different host–pathogen combinations, including rice (Dadley-Moore, 2006), wheat (Figueroa *et al*., 2017), and canola (Delourme *et al*., 2011). However, diseases of *B. juncea* have only been partially reviewed, and then only in a general sense (e.g., Saharan and Verma, 1992; Saharan, 1993; Saharan *et al*., 2016).

There are two types of disease resistance, qualitative and quantitative. Qualitative resistance is gene-for-gene, or race-specific, and provides a high level of resistance (Kushalappa *et al*., 2016). However, quantitative resistance is usually controlled by many race-nonspecific genes each with a small effect, resulting in the reduction of disease symptoms but not the complete arrest of pathogen growth, as can be the case with some qualitative resistances (St.Clair, 2010; Poland *et al*., 2009).

This review is the first to focus on disease resistances in *B. juncea*. In particular, we summarize the application of molecular mapping techniques, such as genomewide association mapping (GWAS) and quantitative trait locus (QTL) (linkage) mapping, on identifying molecular markers, which are mostly used to locate the chromosomal position of candidate resistance genes. We highlight how marker-assisted selection (MAS), phenotypic selection, and transgenics provide both the critical pathways to validate candidate genes as functional resistance genes, and the new resources for breeding programmes to develop elite *B. juncea* cultivars with resistances to the important *B. juncea* diseases, especially following the release of the *B. juncea* genome. We outline a guide for further discovery and

identification of resistance genes which can be used for substantial improvement in disease management for *B. juncea* worldwide.

2 Diseases affecting *B. juncea*

There are a number of important diseases affecting *B. juncea*, including blackleg (*Leptosphaeria maculans*, *L. biglobosa*), sclerotinia stem rot (*Sclerotinia sclerotiorum*), white rust (*Albugo candida*), alternaria blight (*Alternaria brassicae*, *A. brassicicola*, *A. raphani*), downy mildew (*Hyaloperonospora brassicae*), white leaf spot (*Pseudocercosporella capsellae*), clubroot (*Plasmodiophora brassicae*), powdery mildew (*Erysiphe cruciferarum*), *Turnip yellows virus* (TYV; formerly *Beet western yellows virus*), *Cauliflower mosaic virus* (CaMV), *Turnip mosaic virus* (TuMV), bacterial leaf blight (*Pseudomonas syringae*), and black rot (*Xanthomonas campestris*) (Kolte *et al*., 1987; Saharan and Verma, 1992; Saharan, 1993; Oram *et al*., 1999; Coutts and Jones, 2000; Sharma *et al*., 2001; Walsh and Jenner, 2002; 2006; Edwards *et al*., 2007; Saharan and Mehta, 2008; Coutts *et al*., 2010; Kehoe *et al*., 2010; Singh *et al*., 2010; Pradhan and Pental, 2011; Chattopadhyay *et al*., 2015; Nyalugwe *et al*., 2015a; 2015b; 2016a; 2016b; 2016c; van Leur *et al*., 2014; Van de Wouw *et al*., 2016; Saharan *et al*., 2016; Singh *et al*., 2016a; 2016b). While many of these diseases are present on *B. juncea* worldwide, geographic location can determine their relative importance. For example, sclerotinia stem rot and alternaria leaf blight are particularly severe on *B. juncea* in India (Chattopadhyay *et al*., 2015; Saharan *et al*., 2016), while white rust is particularly important in Canada (Saharan and Verma, 1992; Katiyar and Chamola, 2003; Pradhan and Pental, 2011), and India (Saharan and Verma, 1992; 2016; Singh *et al*., 2015). In Australia, blackleg and sclerotinia have been identified as the most destructive diseases for oilseed- and vegetable-type mustards (Murray and Brennan, 2012; Uloth *et al*., 2013; 2014; 2015a; 2015b;

Van de Wouw *et al*., 2016). However, there are several other diseases also identified as important in Australia, including TYV, downy mildew, white leaf spot, alternaria blight, bacterial leaf blight, and powdery mildew (Li *et al*., 2006a; 2007; 2008a; 2009b; Eshraghi *et al*., 2007; Murray and Brennan, 2012; Gunasinghe *et al*., 2014; Uloth *et al*., 2016; 2018; Van de Wouw *et al*., 2016; Guerret *et al*., 2017; Mohammed *et al*., 2017; 2018a; 2018b; 2019; Al-lami *et al*., 2019a; 2019b; 2019c; Murtza *et al*., 2019).

2.1 Fungal and oomycete diseases

2.1.1 White rust

White rust, caused by *Albugo candida*, affects *B. juncea* and other oilseed brassicas worldwide (Saharan and Verma, 1992; Saharan, 1993; Katiyar and Chamola, 2003, Barbetti *et al*., 2016; Mandiriza-Mukwirimba *et al*., 2016). Symptoms for white rust usually includes the development of white blister-like pustules on the underside of leaves, stems, and inflorescence of the plant, along with chlorosis on the leaf surface. In addition, infections cause malformation of inflorescences leading to “staghead” formation, which has been observed to cause a reduction of seed yield (Meena *et al*. 2002). White rust mainly affects *B. rapa* and *B. juncea*; however, some susceptible genotypes of *B. napus* have also been reported (Barbetti, 1981). While the germplasm belonging to the Indian gene pool is highly susceptible to the disease, the east European germplasm of *B. juncea* is highly resistant (Panjabi-Massand *et al*., 2010). In addition, varying levels of leaf and/or inflorescence (staghead) resistance have been reported across genotypes from Australia, China, and India, with China and Australian genotypes generally showing better resistance than those from India (Li *et al*., 2007; 2008b; 2009b; Kaur *et al*., 2011b). In Australia, yield losses from white rust of up to 20% in *B. juncea* have been estimated (Barbetti, 1981).

Variants of race 2 are the main *A. candida* types affecting *B. juncea*, particularly pathotype 2A (Gurung *et al*., 2007) and the more virulent 2V (Kaur *et al*., 2011c). Many studies have demonstrated resistance to *A. candida* in *B. juncea*, including specifically against race 2V (Li *et al*., 2007; 2008b; 2009b; Barbetti *et al*., 2016), and also against isolates sourced from non-*B. juncea* hosts (Pidskalny and Rimmer, 1985; Kolte *et al*., 1991; Dang *et al*., 2000; Kaur *et al*., 2008b; Li *et al*., 2008b; Ahmad *et al*., 2014). However, there is a lack of information in relation to resistance against other races, such as race 7. Furthermore, that *A. candida* isolates from *Capsella bursa-pastoris, B. oleracea, Raphanus raphanistrum,* and *R. sativus* are also pathogenic on *B. juncea* (Kaur *et al*., 2011c) highlights the need for further investigation and definition of races and pathotypes of *A. candida* that can attack *B. juncea*. For example, the study of Jouet *et al*. (2019), highlighting *A. candida* race diversity, ploidy, and host-associated microbes using DNA sequence capture on diseased plants in the field. It has also been reported that host susceptibility of *B. juncea* to white rust disease increases with preinoculation with *H. brassicae* (downy mildew) (Kaur *et al*., 2011d), highlighting a need for additional studies on the expression of host resistance to both downy mildew and white rust, as these two diseases commonly co-occur.

Kaur *et al*. (2011c) undertook a comparative proteomic study to explore molecular mechanisms in *B. juncea* to white rust. They showed 19 proteins with reproducible differences in abundance between a susceptible (RH 819) and a resistant (CBJ 001) variety and confirmed identities through Q-TOF MS/MS (Kaur *et al*., 2011a). Among these was a thaumatin-like protein (PR-5), a protein not previously associated with resistance of *B. juncea* towards *A. candida*.

White rust resistance has been shown to have monogenic dominance in *B. juncea* (Somers *et al*., 2002; Vignesh *et al*., 2009; 2011; Panjabi-Massand *et al*., 2010). Studies involving *B. juncea* 'Bio-YSR' showed control by a single dominant gene (Vignesh *et al*.,

2009; 2011). A subsequent study identified monogenic dominance for resistance using segregating populations from crosses with several resistant *B. juncea* lines, including Bio-YSR, BEC-144, and JM-1 (Yadava *et al*., 2012). A single dominant gene against *A. candida* race 2V has also been introgressed into *B. juncea* from *B. napus* (Chauhan and Sharma, 2001). In earlier studies there are also several examples of alternative genetic control and modes of inheritance, such as recessive or partial dominant resistance controlled by $Ac2a_2$ and a single dominant gene controlling for partial resistance (Bansal *et al*., 1999). More recently, Cevik *et al*. (2019) showed that resistance to an isolate of *A. candida* race 2 (Ac2V) can be explained by at least one of four genes encoding nucleotide-binding, leucine-rich repeat (NLR) immune receptors. Further, a broad-spectrum resistance to white rust was observed in transgenic *B. juncea* cultivars produced by insertion of the *WRR4* gene, a resistance gene from *Arabidopsis* that showed resistance to four races of *A. candida* (Borhan *et al*., 2010); Table 2).

Genetic mapping in *B. juncea* to identify molecular markers associated with white rust resistance has involved use of RAPD and/or RFLP markers (Cheung *et al*., 1998; Mukherjee *et al*., 2001; Panjabi-Massand *et al*., 2010). Mukherjee *et al*. (2001) reported a white rust resistance locus against race 2, *Ac2(t)*, linked to flanking RAPD markers $OPN01_{1000}$ and $OPB06_{1000}$. Subsequently, Varshney *et al*. (2004) developed a codominant and more precise CAPS marker from the linked RAPD marker $OPB06_{1000}$ and AFLP marker E-ACC/M-CAA_{350}. A resistance gene against race 2A, *Acr*, was mapped to linkage group 7 with three RFLP markers linked to *Acr*: X42, X83, and X140a (Cheung *et al*., 1998). A study by Prabhu *et al*. (1998) used an F_1-derived DH population of the same crossing to demonstrate that two RAPD markers, WR2 and WR3, were linked to a single dominant gene and the resistance locus, $Ac2a_1$.

Other studies have used several marker types, including AFLPs, intron polymorphic markers, and SSRs (Singh *et al*., 2015). Three resistance loci were identified from previous molecular mapping studies (Panjabi-Massand *et al*., 2010) and followed up by marker prediction/validation for resistance in known resistant cultivars (Singh *et al*., 2015). Two independent qualitative resistance loci positioned on different linkage groups were identified in two east European *B. juncea* lines by linkage (QTL) mapping: partially resistant AcB1-A4.1 and completely resistant AcB1-A5.1. Additional markers used to narrow the genetic interval for these loci were on chromosomes A4 and A5, respectively (Panjabi-Massand *et al*., 2010). Singh *et al*. (2015) identified the previous two loci, AcB1-A4.1 and AcB1-A5.1, linked to At5g41560 and At2g36360, respectively, along with a third locus in resistant *B. juncea* 'Bio-YSR', which was not linked to either marker. However, it was suggested that $_{Ac2(t)}$ and *AcB1-A4.1* were probably the same locus (Singh *et al*., 2015). More recently, Arora *et al*. (2019) showed the presence of a single CC-NB-LRR protein encoding *R* gene in *B. juncea* 'Donskaja-IV' could confer complete resistance to a range of isolates of *A. candida*. This was the first white rust resistance-conferring gene described from *Brassica* species and has been named *BjuWRR1* (Arora *et al*., 2019). Such a resistance gene, along with markers identified in the above studies, will assist the introgression of resistance gene(s) and/or resistance-conferring loci into otherwise susceptible varieties.

2.1.2 Blackleg

One of the most devastating diseases affecting brassica production is blackleg disease, also known as phoma stem canker, primarily caused by *Leptosphaeria maculans*, but also by *L. biglobosa*. Generally, there are different compositions of both pathogens but where disease is most severe, *L. maculans* predominates (Gugel and Petrie, 1992; Rimmer and van den Berg, 1992; Salisbury *et al*., 1995; Balesdent *et al*., 2005; Rouxel and Balesdent, 2005; Vincenot *et al*., 2008; Dilmaghani *et al*., 2009). Major symptoms of blackleg disease are leaf lesions

(phoma leaf spots) and stem canker (basal phoma stem canker). Stem canker, which appears as black/brown lesions on the stem, may lead to crop loss due to severance of the stem through plant lodging caused by the fungus girdling the stem (Khangura and Barbetti, 2001). Leaf lesions usually appear large pale brown/off-white with a clearly defined margin and tiny dark fruiting bodies (Khangura and Barbetti, 2001). Lesions caused by *L. maculans* may also occur on other parts of the plant, such as the stem and siliques. Infection of siliques may lead to abnormal seed development and/or seed infection. In addition, *L. maculans* can infect the roots, causing root rot.

In general, *B. juncea* shows a much higher level of resistance to blackleg diease compared with *B. napus* (Marcroft *et al*., 2002) and suffers much less yield loss from blackleg than *B. napus*, where it has long been a very devastating disease (Howlett *et al*., 2001). *B. juncea* canola, as a blackleg resistance alternative to *B. napus* canola, has been growing commercially in Australia since 2007. However, its inherent historic resistance to serious blackleg infection has been lost, and severe blackleg has been identified in *B. juncea* canola over recent years in Australia (Elliott *et al*., 2015). This report highlights the presence of isolates of *L. maculans* highly virulent to *B. juncea* and that such isolates are already widespread. This is unfortunate as stable introgression of the *B. juncea Rlm6* resistance into *B. napus* had been successful prior to 2008 (Chèvre *et al*., 2008), but now represents another lost single dominant resistance gene against *L. maculans*. Clearly, blackleg is now a major threat to *B. juncea* following this resistance breakdown.

Major gene resistance is effective across both seedling and adult plants, while quantitative resistance operates predominantly at the adult plant stage. It has been shown that *Brassica* species containing the B genome, that is, *B. juncea* and *B. carinata*, in general, have good seedling resistance to blackleg, in comparison with *B. napus* (Roy, 1984; Dolores Sacristan and Gerdemann, 1986). This B genome resistance has been successfully

214 introgressed (Roy, 1978; 1984) and stabilized (Chèvre *et al*., 2008) into *B. napus*. Genetic
215 studies for blackleg resistance in *B. juncea* have been conducted since 1999 using a variety of
216 molecular markers (Table 1) to understand its mode of inheritance and to discover new
217 sources of resistance for breeding programmes. An early study identified two resistance
218 genes in *B. juncea*, a dominant gene and a recessive gene, controlling blackleg resistance
219 (Keri *et al*., 1997). This is similar to findings from Rimmer and van den Berg (1992) who
220 also found two resistance genes, although in their case, the two genes were shown to have a
221 dominant recessive epistatic interaction where one gene was dominant and the other was
222 recessive. Later, Balesdent *et al*. (2002) also found that resistance in *B. juncea* (cultivars
223 Picra and Aurea) was controlled by two genes, *Rlm5* and *Rlm6* (*Rlm6* was previously referred
224 to as *Jlm1*). While *Rlm6* has been successfully introduced into *B. napus*, the durability of
225 *Rlm6* resistance is dependent on the presence of background quantitative resistance. In lines
226 lacking quantitative resistance, resistance in canola failed after three cropping seasons, but
227 remained effective for more than five years in lines containing quantitative resistance (Brun
228 *et al*., 2010). It is important to further investigate the interactions between quantitative and
229 qualitative resistances against blackleg in *B. juncea*. Christianson *et al*. (2006) used simple-
230 sequence repeats (SSRs) to map a dominant resistance gene, *LMJR1*, on linkage group J13
231 and a recessive resistance gene, *LMJR2*, on linkage group J18 (hypothesized to be located on
232 chromosomes B7 and B3, respectively; Fredua-Agyeman *et al*., 2014), from an F_2 population
233 (resistant parent AC Vulcan). However, there are likely to be additional qualitative resistance
234 genes present that have not been characterized to date.

235 Introgression lines have been commonly used to identify blackleg resistance in *B.*
236 *juncea*; a *B. napus–B. juncea* recombinant line (*Rlm6*) displayed a dominant mode of
237 inheritance (Chèvre *et al*., 1997). Complete dominant adult plant resistance in *B. juncea*
238 against blackleg was found to be controlled by three genes, Bl_1, Bl_2, Bl_3, working in a

complex interaction in *B. napus* lines (Pang and Halloran, 1996) with resistance introgressed from *B. juncea* (Roy, 1978; 1984). The presence of two resistance loci in *B. juncea* was indicated using *B. napus–B. juncea* recombinant lines, although it was also suggested that a third locus may be present (Struss *et al*., 1996; Plieske *et al*., 1998). *B. napus–B. juncea* recombinant lines, MX and MXS lines, were developed for identification and mapping of blackleg resistance genes from Australian interspecific *B. juncea–B. napus* lines, such as Roy 85-2 ($Onap^{JR}$), from a cross between *B. juncea* 'BJ168' and *B. napus* 'Cresus-O-Precose' (Roy, 1978; 1984). The resistance gene *Rlm6* was mapped to chromosome B8 of *B. juncea* from the analysis of *B. napus–B. juncea* recombinant lines (MXS lines), and linked to markers OPG02.800 and OPI-01.800 (Chèvre *et al*., 1997; Barret *et al*., 1998).

Additional resistance loci have been mapped, although it is difficult to determine whether they are the same or different loci, as different *B. juncea* genotypes were used in the crosses and different markers were used for genetic analysis. Possible genomic regions of interest for blackleg resistance genes in the *B. juncea* genome and other *Brassica* species were analysed by Plieske and Struss (2001). They used a *B. juncea–B. napus* line and a *B. carinata–B. napus* line where sequence-tagged site (STS) markers derived from an RFLP marker, pRP1513, were found to be more tightly linked to a blackleg resistance gene in the B genome than the AFLP-based STS marker, S7G4. The RFLP-based STS marker sequence was identical among *B. nigra* and the resistant genotypes of the *B. juncea* and *B. carinata* lines (Plieske and Struss, 2001). Struss *et al*. (1996) screened *B. juncea–B. napus* introgression lines using RAPDs and RFLPs and a RAPD marker, OPU9, was found to be linked to blackleg resistance. Genetic analysis was carried out in *B. juncea–B. napus* introgression lines to identify SCAR markers B5-1520, C5-1000, and RGALm linked to the monogenic recessive blackleg resistance gene, r_j*lm2* (Saal *et al*., 2004; Saal and Struss, 2005).

2.1.3 Alternaria blight

Alternaria blight, black spot, is a necrotrophic fungal disease caused by *Alternaria* species, primarily *A. brassicae*, *A. brassicicola*, and *A. raphani*, that threatens global *B. juncea* production. Alternaria infects aboveground parts of the plant, such as leaves, stem and/or pods. Lesions on leaves usually appear as target-like brown to black round necrotic spots, which are often surrounded by chlorosis (Van de Wouw *et al*., 2016). Siliques with necrotic spots usually have limited chlorosis during early stages of lesion development. Infection of siliques may lead to premature ripening and shattering, along with abnormal development of seeds, such as shrivelling, with a reduction of seed weight and oil content.

While alternaria blight has been reported in some parts of Europe and Asia, along with Canada and Australia (Saharan, 1993; Van de Wouw *et al*., 2016), it is particularly devastating in the Indian subcontinent (Saharan, 1993; Singh *et al*., 2009; 2010; Saharan *et al*., 2016), especially given that the rapeseed-mustard industry there predominately grows *B. juncea* and *B. rapa*. For example, alternaria blight has been reported in Indian mustard to cause yield losses of up to 57% in Nepal (Shrestha *et al*., 2005) and up to 35% in India on *B. juncea* (Kolte *et al*., 1987), including reductions in oil content and seed germination (Ansari *et al*., 1988; Shrestha *et al*., 2005; Meena *et al*., 2017). While *Brassica* species, including *B. juncea*, are generally more susceptible to *Alternaria* spp. compared to other Brassicaceae species from different genera, such as *Camelina* or *Erystimum* (Westman *et al*., 1999; Sharma *et al*., 2002), this is not always the case, as *A. brassicae* can cause serious disease on *Crambe abyssinica* (You *et al*., 2005). Alternaria blight is generally considered a major disease in India (Kolte *et al*., 1987) but a minor disease in Australia (Van de Wouw *et al*., 2016). In Victoria, Australia, a *B. juncea* advanced breeding line had shown resistance against *A. brassicae* (Van de Wouw *et al*., 2016), yet recent studies have shown that Australian *B. juncea* and *B. napus* varieties tested were all susceptible to *A. brassicae*, although some of

these same varieties did show good resistance to other *Alternaria* spp. such as *A. japonica* (Al-lami *et al*., 2019b). Other recent studies in Australia (Al-lami *et al*., 2019a) confirmed 10 species (*A. alternata, A. arborescens, A. brassicae, A. ethzedia, A. hordeicola, A. infectoria, A. japonica, A. malvae, A. metachromatica,* and *A. tenuissima*) as present on commercial rapeseed and pathogenic on both *B. juncea and B. napus*. That same study highlighted that alternaria leaf spot on canola across southern Australia is not solely caused by *A. brassicae*, but that a range of other *Alternaria* spp. are also involved to varying degrees, depending upon the year and the geographic locality.

Some resistance to alternaria blight exists within the *Brassica* species, with *B. juncea, B. napus,* and *B. carinata* considered as having more tolerance compared to *B. rapa* (Chadar *et al*., 2016). However, *B. juncea* along with *B. rapa* are generally more susceptible than *B. napus* or *B. carinata* (Skoropad and Tewari, 1977). Despite this, limited resistance does exist in some *B. juncea* genotypes, such as PHR-2, Rohini, and PAB-9511 (Upesh *et al*., 2011; Pratap *et al*., 2015, Gaur *et al*., 2016). Dang *et al*. (2000) analysed 36 Brassicaceae genotypes for several diseases, where *B. juncea* genotypes DIR-1507 and DIR-1522 were found to be resistant against alternaria blight, with stable resistance over several consecutive years that could be used in breeding programmes. However, molecular mapping of resistance gene loci or cloning of resistance genes has not been conducted for alternaria blight to date. In addition to resistance genes, identification and mapping of genes controlling other phenotypic characteristics, such as production of epicuticular wax layer, could be of interest. For example, the epicuticular wax layer of resistant somaclones of *B. juncea* reduced disease severity by reducing the spore germination rate and the germ tube length of *A. brassicae* (Gupta and Sharma, 2012). There are several examples that highlight how transgenic *B. juncea* lines, produced by insertion of different defence genes, show some resistance or tolerance to alternaria blight (Table 2) (Kanrar *et al*., 2002; Mondal *et al*., 2007; Chhikara *et*

al., 2012; Rustagi *et al*., 2014; Hada *et al*., 2015). Despite *B. juncea* varieties such as RC-781 and PHR 1 showing some field resistance across different years and locations (Saharan *et al*., 2016), reliably effective in-field resistance has been difficult to locate, and managing *A. brassicae* on *B. juncea* remains an ongoing challenge.

2.1.4 Sclerotinia stem rot

Sclerotinia sclerotiorum is the fungal pathogen responsible for the development of sclerotinia stem rot disease, with a large host range that includes more than 400 plant species, such as common bean, sunflower, alfalfa, soybean, peanut (Purdy, 1979; Boland and Hall, 1994) and all brassicas including *B. juncea* (Li *et al*., 2006a). Sclerotinia is an important disease found worldwide, including Brazil, Canada, China, India, Europe, and Australia (Saharan, 1993; Hind *et al*., 2003; Murray and Brennan, 2012). In India, sclerotinia stem rot threatens the sustainability of *B. juncea* production (Singh *et al*., 2008; Saharan and Mehta, 2008; Goyal *et al*., 2011). Sclerotinia stem rot can reduce seed yield losses by up to 92% (Sharma *et al*., 2001). Sclerotinia stem rot effects aboveground parts of the plant. Lesions on stems, branches, and pods usually appear enlarged, necrotic, and bleached (greyish white) where some lesions may have white cotton-like mycelial growth. Severely infected plants may wilt and die whereby the fungus has grown throughout the stem causing rot, and lesions appear followed by stem girdling (Li *et al*., 2006a).

The percentage of yield loss caused by this disease is affected by the timing of infection. Shukla (2005) reported that while infection starting at post-flower initiation stage or later reduced yield of *B. juncea* by 51%, if the infection started on or before flower initiation then yield losses could reach 100%. Quality parameters of *B. juncea* are also adversely affected by sclerotinia stem rot. For example, reductions of oil content and total protein content of up to 35% and 40%, respectively, have occurred. The timing of disease

appearance can also be related to disease severity, where early infections were more severe compared with late infections (Singh *et al*., 2009). In south-eastern Australia, *B. juncea* has suffered yield losses of more than 20% in approximately 10% of years (Oram *et al*., 2005).

B. juncea has displayed variable levels of sclerotinia resistance, including genotype-dependent and isolate-dependent responses (Ge *et al*., 2012; 2015; Uloth *et al*., 2013; 2014; 2015b; Barbetti *et al*., 2014; Gaur *et al*., 2016). In Australia, *B. juncea* genotypes have generally shown poor tolerance against most Australian *S. sclerotiorum* isolates (Garg *et al*., 2010b; Oram *et al*., 2005) with, at best, only limited resistance (Li *et al*., 2009a; Uloth *et al*., 2015b; You *et al*., 2016). However, recently, qualitative cotyledon resistance has been demonstrated in *B. juncea* genotypes Ringot I and Seeta (Ge *et al*., 2015) and also in some interspecific crosses involving *B. juncea* (Barbetti *et al*., 2014). Perhaps of most significance are the studies by Rana *et al*. (2017) and Atri *et al*. (2019) that demonstrated high levels of resistance in *B. juncea–B. fruticulosa* introgression lines, and the study of Rana *et al*. (2019) that demonstrated high levels of resistance in *B. juncea–Erucastrum cardaminoides* introgression lines. In India, some *B. juncea* genotypes show increased sclerotinia resistance under field conditions, such as EC 597328, BIOYSR, and PHR-2 (Gaur *et al*., 2016). In Australia, the *B. juncea* genotypes tested to date are all very susceptible to sclerotinia (Uloth *et al*., 2015b).

Introgression from other brassicas, such as *B. carinata* (Barbetti *et al*., 2014), three wild crucifers (*E. cardaminoides*, *Diplotaxis tenuisiliqua*, and *E. abyssinicum*; Garg *et al*., 2010a) and most recently *B. fruticulosa* (Rana *et al*., 2017; Atri *et al*., 2019) and again *E. cardaminoides* (Rana *et al*., 2019), has also been performed to develop resistant *B. juncea* cultivars by interspecific hybridization. In addition, a comparison of *Brassica* species found *B. oleracea* to be substantially more tolerant to sclerotinia (Mei *et al*., 2011), as are some *B. napus* varieties, compared with *B. juncea* (Uloth *et al*., 2015b). However, when testing

seedlings of selections of *B. carinata*, *B. incana*, *B. juncea*, *B. napus*, and *B. napus* introgressed with *B. carinata*, *B. nigra*, *B. oleracea*, *B. rapa* var. *rosularis*, *B. rapa* var. *chinensis*, *B. tournefortii*, *Raphanus raphanistrum*, *R. sativus*, and *Sinapis arvensis*, Uloth *et al*. (2014) also showed that the three most resistant genotypes were all from *B. oleracea* (viz., *B. oleracea* var. *italica* 'Prophet' and *B. oleracea* var. *capitata* 'Burton' and 'Beverly Hills'). More importantly, Uloth *et al*. (2013) showed very high-level quantitative resistance in one or more *R. raphanistrum*, *B. napus*, *B. carinata*, *R. sativus* genotypes and high resistance in one or more *B. incana*, *B. rapa* var. *rosularis*, *B. oleracea* vars *acephala* and *italica*, and *Sinapis arvensis*. Such resistances could be introgressed and bred into *B. juncea* cultivars.

Currently, there has only been a single genetic mapping study conducted to identify markers linked to sclerotinia disease resistance that examined the introgression of genomic segments with resistance from wild Brassicaceae, *B. fruticulosa*, into *B. juncea* introgression lines, where Rana *et al*. (2017) used association mapping to detect 10 significant marker trait associations. Subsequently, Atri *et al*. (2019) conducted GWAS to map resistance responses to stem rot on *B. fruticulosa*-into-*B. juncea* introgression lines. They identified 13 significant loci on seven *B. juncea* chromosomes (A01, A03, A04, A05, A08, A09, and B05) along with 20 candidate genes belonging to major disease resistance protein families, including *TIR-NBS-LRR* class, chitinase, malectin/receptor-like protein kinase, defensin-like (*DEFL*), desulfoglucosinolate sulfotransferase protein, and lipoxygenase. Rana *et al*. (2019) developed a set of *B. juncea–E. cardaminoides* introgression lines and identified six marker loci associated with resistance in both A and B genomes using SSR-based association mapping. Using GWAS, they highlighted an array of resistance mechanisms in terms of signal transduction pathways, hypersensitive responses, and production of antifungal proteins and metabolites; including LRR-RLK (receptor-like kinases) genes that encode LRR-protein kinase family proteins, genetic factors associated with pathogen-associated molecular

patterns (PAMPs), and effector-triggered immunity (ETI). Such studies not only provide critical new understanding of the defence pathway of these introgressed weedy Brassicaceae resistances, but marker trait associations (MTAs) identified will facilitate marker-assisted introgression of these critical resistances into new cultivars of *B. juncea* initially and, subsequently, into other crop *Brassica* species.

2.1.5 Clubroot

Clubroot, caused by *Plasmodiophora brassicae*, is a global soilborne disease shown to affect mainly Brassicaceae species (Oxley, 2007). Clubroot is a particular issue for regions with warm temperatures (20–24°C), acidic soil conditions, and poor drainage (Wellman, 1930). The most typical symptom of clubroot seen on infected mustard, canola, and other brassica plants is roots with swollen galls ("clubs") that appear firm and white at early infection to greyish brown at latter stages of infection where galls mature and decay. Severely affected plants may also show stunting, yellowing, wilting, and premature ripening, along with leaf lesions.

Infection in oilseed brassicas has been reported worldwide (Saharan, 1993; Bhattacharya *et al*., 2014; Řičařová *et al*., 2016; Xue *et al*., 2017), as well as in *B. juncea* vegetables in China (Xue *et al*., 2017) and *B. juncea* crops in India (Chattopadhyay and Bagchi, 1989; Bhattacharya *et al*., 2014). A major first epidemic of clubroot occurred in the early 1980s in India (Laha *et al*., 1985), where disease incidence ranged from 30% on *B. juncea* to 70% on *B. rapa* (Chattopadhyay and Bagchi, 1989). Strehlow *et al*. (2014) also reported up to 60% yield loss for oilseed rape crops in Canada. In China, *B. juncea* vegetables were found to show infection levels of up to 37% (Liu *et al*., 2017; Xue *et al*., 2017). In a comparison study among different germplasm of *Brassica*, *B. juncea* accessions showed the highest disease severity (Sharma *et al*., 2012).

While clubroot has caused significant widespread losses for Australian vegetable *Brassica* production, it is yet to prove to be a major issue for the rapeseed-mustard industry, due to Australian pathotypes causing disease mostly in warmer months (Hind-Lanoiselet and Parker, 2005), rather than during the cooler late autumn, winter, and early spring conditions when rapeseed is grown. However, there is a potential for this disease to be a threat to the rapeseed-mustard industry in Australia if crops are exposed to warmer than normal conditions during the growing season, or if there is a spread of pathotypes that more readily infect in cooler months or year-round. No resistant *B. juncea* genotypes have been identified among several studies, including in India and Europe (Chattopadhyay and Bagchi, 1989; Chattopadhyay *et al*., 2001; Zhang *et al*., 2014).

Using resistant cultivars is the most effective strategy for clubroot management (Zhang *et al*., 2014). Similar to other diseases, resistance can be achieved by combining both types of resistance (i.e., qualitative and quantitative) (Piao *et al*., 2009). Race-specific resistance genes against clubroot have been identified in *B. napus* and *B. rapa* (James and Williams, 1980) and different QTLs also have been found in *B. oleracea* (Chiang and Crête, 1970) and *B. rapa* (Suwabe *et al*., 2006). While there have been many studies on resistance genes and/or QTLs in *B. napus*, such is not the case with *B. juncea*. Although *B. juncea* generally does not show high resistance to clubroot, it commonly shows low or moderate resistance (Peng *et al*., 2014). However, an accession of *B. juncea* was found to be highly resistant to the local pathotype 4 under field conditions in China (Ren *et al*., 2016). In a recent study by Chang *et al*. (2019), a clubroot resistance gene was identified in the B genome, in *B. nigra*. This gene, named *Rcr6*, was identified in a region homologous to *B. rapa* chromosome A08. However, to date there is no available resistance available in mustard species (*B. juncea* and *B. carinata*). Interestingly, lines with effective resistance to a broad range of pathotypes of *P. brassicae* have been identified in ancestral diploid species (Jakir

Hasan *et al*., 2012; Peng *et al*., 2014), which could greatly broaden the genetic base of clubroot resistance in mustards such as *B. juncea*.

In general, whilst many studies have been conducted to understand resistance to clubroot in brassicas, these have been in species other than *B. juncea*. For example, Hejna *et al*. (2019) used SNP association analysis and identified two major loci (on chromosomes A2 and A3) controlling resistance in *B. napus*, along with seven minor loci, and a further 21 genes potentially involved in resistance using gene expression marker (GEM) analysis. They highlighted the potential for these results to be used for accelerated pyramiding of multiple clubroot resistance genes in new varieties, and there is potential for future transfer of such genes into *B. juncea*.

2.1.6 Hypocotyl rot

Rhizoctonia solani, a soilborne fungal pathogen, causes hypocotyl rot, damping off, and root rot in brassicas. Common hypocotyl symptoms caused by *Rhizoctonia solani* are dark brown watersoaked lesions, wire-like appearance, hypocotyl girdling, and pinching off, leading to loss of plants (Khangura *et al*., 1999). Affected plants may have leaves with an orange to purple colouration. Also, symptoms shown for taproot rot are usually dark brown lesions whereas lateral root rot lesions are pale brown to dark brown in appearance (Khangura *et al*. 1999). *B. juncea* has been shown to be susceptible to *Rhizoctonia* isolates from group ZG5 (AG2-1) and ZG1-1 (AG8) in Australia (Khangura *et al*., 1999) and in the USA (Babiker *et al*., 2013). While ZG5 causes severe disease, particularly a hypocotyl rot, on both *B. juncea* and *B. napus, B. juncea* is much less susceptible to ZG1-1 (Barbetti and Khangura, 2000). Whilst no studies are available to show yield loss in *B. juncea* due to *R. solani*, losses up to 30% have been reported in *B. napus* (Verma, 1996) and are probably similar in *B. juncea*, particularly with ZG2-1. Several resistant genotypes have been shown against *R. solani*

(Kluth *et al*., 2010) including against AG 2-1 and AG 4 from Canada (Kataria and Verma, 1992; Yang and Verma, 1992).

A *B. juncea* chitinase gene (pathogenesis-related protein), *BjCHI1*, identified by Zhao and Chye (1999), provides resistance against several fungal pathogens, including *R. solani*, along with a benefit of abiotic stress tolerance (Wu *et al*., 2009). Transgenic *Arabidopsis*, potato, and tobacco were developed with *BjCHI1* from *B. juncea* (Fung *et al*., 2002; Chye *et al*., 2005; Guan *et al*., 2008; Gao *et al*., 2014) to further demonstrate its role in defence.

2.1.7 Downy mildew

Downy mildew, caused by *Hyaloperonospora brassicae* (formerly *Peronospora brassicae* or *H. parasitica*), infects rapeseed-mustard in Canada, several Asian and European countries, including the UK and France, and also China and India (Saharan, 1993) and Australia (Ge *et al*., 2008). Downy mildew is an important disease in India, where the predominant oilseed brassica grown is *B. juncea*, and where it can cause serious economic loss even during the seedling stage (Thakur and Mathur, 2002). Downy mildew is seedborne, all aboveground parts of the plants are affected by *H. brassicae*, and plants die under severe infection at the seedling stage. Infection at the adult plant stage can also result in debilitation of plants and serious yield loss (Meena *et al*., 2014). Lesions are commonly chlorotic yellow or yellowish-brown on the upper leaf surface of plants, along with white powdery mycelial fungal growth on the under surface of the leaf (Van de Wouw *et al*., 2016). Other symptoms of downy mildew are large, irregularly shaped blotches developing from several necrotic spots joining together to cause the leaf to dry out and show a yellow-red colouration.

Downy mildew in *B. juncea* has caused reductions in seed yield of 42.6%–66.7%, depending on the cultivar grown, and causes an estimated annual economic loss of about 683 million Indian rupees (Meena *et al*., 2014). Between 37%–47% and 17%–32% reductions in

485 seed yield have been reported in *B. juncea* when exposed to downy mildew in the presence of
486 white rust (Bains and Jhooty, 1979). Preinfection of *B. juncea* with white rust suppresses
487 immunity to downy mildew, which could explain the very large yield reductions in such
488 situations (Bains and Jhooty, 1985; Cooper *et al*., 2008). A subsequent study by Kaur *et al*.
489 (2011d) showed that white rust symptoms were exacerbated on *B. juncea* by subsequent
490 infection with downy mildew. *B. juncea* cultivars resistant to both white rust and downy
491 mildew are an important requirement in many regions where both pathogens coexist (e.g.,
492 India and Australia). In Australia, downy mildew is a major disease affecting oilseed and
493 vegetable brassicas, particularly across southern and western Australian regions (Mohammed
494 *et al*., 2018a; 2018b). Although *H. brassica* has been present on Australian *B. napus* and *B.*
495 *juncea* crops for decades (Barbetti and Khangura, 2000), it shows increasing severity and
496 threat to commercial brassica crops in some regions of Australia, particularly since 1998
497 (Barbetti and Khangura, 2000). This may be in part associated with periods of less than ideal
498 rainfall in autumn–early winter and to the continued deployment of one or more highly
499 susceptible varieties, as both seem to foster development of downy mildew (Mohammed *et*
500 *al*., 2017). In addition, the diverse pathotypes and phylogenetic variation recently reported in
501 Australian *H. brassica* populations have been highlighted as key drivers of downy mildew
502 epidemics in Brassicaceae, including in *B. juncea* (Mohammed *et al*., 2018b).

503 Resistances that are effective across mixed pathotypes have been demonstrated. For
504 example, Mohammed *et al*. (2017) tested 131 diverse Brassicaceae varieties, including *B.*
505 *napus*, *B. juncea*, *B. carinata*, *B. nigra*, *B. rapa*, *C. abyssinica*, and *R. sativus*, and
506 highlighted excellent pathotype-independent highly resistant genotypes to downy mildew
507 across *R. sativus*, *B. carinata*, *B. napus*, *B. juncea*, and *C. abyssinica*. Several Australian *B.*
508 *juncea* genotypes have shown resistance in Indian field conditions for downy mildew, along
509 with resistance to several other diseases such as alternaria blight, white rust, and powdery

mildew (Singh *et al*., 2010). *B. juncea* accessions, susceptible to *H. brassicae* isolates from India, were shown to be resistant to isolates derived from rapeseed hosts in the UK; whereas rapeseed was able to withstand Indian isolates from a *B. juncea* host (Nashaat and Awasthi, 1995). Similarly, in Australia, where several varieties of *B. napus* were highly susceptible to *H. brassicae* isolates, several genotypes of *B. juncea* were highly resistant (Mohammed *et al*., 2017). These findings suggest that the degree of virulence of isolates depends upon different *Brassica* species grown in different geographical regions that allows *H. brassicae* to evolve and overcome resistance. In other studies by Mohammed *et al*. (2019), almost all *B. juncea* genotypes tested showed a high level of resistance, in contrast to *B. napus* genotypes that showed wide-ranging responses from high resistance to extreme susceptibility. This study by Mohammed *et al*. (2019) highlights the ready availability of very high levels of pathotype-independent resistance, not just for *B. juncea*, but also in other Brassicaceae such as *Raphanus sativus*, *B. carinata*, *B. oleracea*, and *C. abyssinica*. Nashaat *et al*. (2004) analysed the genetic basis for resistance against multiple isolates (IP04A, IP05B, IP00A, IP33A) where several dominant genes were identified in *B. juncea* cultivars RESBJ-190 and RESBJ-200. The resistance genes from the two *B. juncea* lines were shown to be independent to each other due to resistance against two different isolates, IP00A and IP33A. Qualitative resistance was also investigated using field screening (Dang *et al*., 2000) where several genotypes showed resistance (TMS-50, Zem-7, DIR-1522, and DIR-1507). In addition, a transgenic pearl millet line, which was transformed by insertion of *B. juncea* pathogenesis-related gene 1 (*BjNPR1*), was resistant to downy mildew (Ramineni *et al*., 2014). Such resistances identified can be used as sources of resistance not only for *B. juncea*, but also for rapeseed and vegetable Brassicaceae breeding programmes and/or directly deployed as new varieties where downy mildew is prevalent. Currently, although there have been several studies to

identify resistant *B. juncea* accessions and/or their mechanisms of resistance, there is no genetic mapping information available relating to *B. juncea*.

2.1.8 White leaf spot

White leaf spot, caused by the fungal pathogen *Neopseudocercosporella capsellae* (perfect stage *Mycosphaerella capsellae;* syn. *Pseudocercosporella capsellae*), can cause severe disease on *B. juncea* (Petrie and Vanterpool, 1978; Gunasinghe *et al*., 2014; 2016a). White leaf spot affects leaves, along with stems and pods, where lesions appear greyish-white to light brown with a granular surface and brown margins at lesion maturity. Lesions often merge together to form large irregular-shaped lesions and cause premature defoliation during severe infection. White leaf spot can be severe in North America, Europe, and Australia on a range of different *Brassica* species, including *B. juncea* (Petrie and Vanterpool, 1978; Barbetti and Sivasithamparam, 1981; Inman *et al*., 1999; Söchting and Verreet, 2004; Eshraghi *et al*., 2005; 2007; Gunasinghe *et al*., 2014, 2016d). However, to date, white leaf spot does not occur on *B. juncea* in India, in contrast to Australia where it does occur widely on *B. juncea* (Eshraghi *et al*., 2005; Van de Wouw *et al*., 2016). Gunasinghe *et al*. (2017b) showed Australian genotypes of *B. juncea* were highly susceptible to *N. capsellae*, whereby up to 58% of leaves showed disease incidence. The first report of *B. juncea* infected by white leaf spot in Australia was 2005 in Western Australia (Eshraghi *et al*., 2005). Murtza *et al*. (2019), in phylogenetic association studies among 2005 versus 2015–2016 *N. capsellae* isolates, highlighted how pathogenicity variations could be explained by geographically different *N. capsellae* pathogen populations. More importantly, Murtza *et al*. (2019) showed that *N. capsellae* populations are evolving rapidly, challenging management through host resistance by evolving populations overcoming previously resistant varieties, at a time of increasing incidence and severity of white leaf spot disease over the past decade.

There have been several studies that have identified various levels of resistance to white leaf spot in Brassicaceae, including *B. juncea*. For example, Gunasinghe *et al*. (2017b) showed high levels of resistance in *B. rapa* subsp. *oleifera*, *B. oleracea* such as *B. oleracea* var. *gongylodes* and *B. oleracea* var. *sabellica*, and *B. fruticulosa*, and while they found most *B. juncea* varieties were highly susceptible, there was a range in severity, with both Montara and VT 535 G showing useful resistance in this species. Gunasinghe *et al*. (2014) showed that the highest levels of resistance were in *B. carinata*, *C. abyssinica*, *Eruca sativa*, and *B. oleracea* var. *capitata*. In that study, the most resistant of the *B. juncea* genotypes tested were from China, the most susceptible from India, with those from Australia intermediate. These and earlier studies such as that of Eshraghi *et al*. (2007) highlight the general overall susceptibility of *B. juncea* and the necessity to either locate high level resistances in *B. juncea* and/or to introgress the resistances from other Brassicaceae into *B. juncea* to manage this disease in mustard crops if the significant yield losses due to high susceptibility in most *B. juncea* genotypes is to be curtailed (Eshraghi *et al*., 2007; Gunasinghe *et al*., 2017b). For example, the significant level of resistance found in *B. juncea* cultivars like Montara and VT 535 G (Gunasinghe *et al*., 2017b) could be more widely deployed into regions highly prone to severe white leaf spot. The moderately resistant *B. juncea* genotype Dune, a resistance associated with increased stomatal density (Gunasinghe *et al*., 2016d), along with highly susceptible Indian or Chinese *B. juncea* lines (Gunasinghe *et al*., 2014; 2016b; 2016d; 2017b), could be used as a basis to identify resistance genes or QTLs by developing populations from crossing, association mapping, and/or linkage mapping. Further, resistances associated with the critical role in white leaf spot development played by cercosporin production by *P. capsellae* (Gunasinghe *et al*., 2016b; 2016c), with unique infection structures (Gunasinghe *et al*., 2017a), and with spore disintegration, spore germination, germ tube impedance, and lower stomatal density such as on resistant *B. juncea* ‘Dune’, are all

583 factors that probably contribute towards resistance and could also be used as foci for defining
584 and understanding the resistance genes associated with resistance to this pathogen
585 (Gunasinghe *et al*., 2016b). Such an approach is needed, as currently there is a lack of
586 knowledge of resistance markers against white leaf spot, despite it being a highly problematic
587 disease for *B. juncea* crops.

588 *2.1.9 Powdery mildew*

589 Powdery mildew is caused by the fungal pathogen *Erysiphe cruciferarum*, which has a broad
590 host range, and threatens the quality, quantity, and marketability of brassica crops by
591 attacking leaves, stems, and pods, and can severely reduce yield (Uloth *et al*., 2016). It infects
592 *Brassica* crops, including *B. juncea*, in the USA (Saharan, 1993), Australia (Kaur *et al*.,
593 2008a; Uloth *et al*., 2016), and several Asian and European countries, including Germany and
594 France, Vietnam, South Korea, and India (Saharan, 1993; Tam *et al*., 2016). In 2014, 80% to
595 100% of the *B. juncea*-growing regions in Vietnam reported powdery mildew. In India,
596 significant disease incidence and yield loss reaching up to 70% can occur for *B. juncea* crops
597 (Kumar *et al*., 2017). In *B. napus*, epidemics are favoured by warmer seasonal conditions
598 such as occur in late winter/early spring that coincide with more susceptible and more mature
599 plants, and it is likely the same applies for *B. juncea* (Uloth *et al*., 2018).

600 Several studies have investigated the resistance status for *B. juncea* against powdery
601 mildew, with some genotypes from Australia and India having resistance against *E.*
602 *cruciferarum* when either exposed to only powdery mildew or in conjunction with another
603 disease (Singh *et al*., 2010). A recent study analysed resistance among *B. juncea* and *B. napus*
604 cultivars where it was found that *B. juncea* varieties Sahara CL and Xceed X121 CL were
605 able to provide resistance against leaf infection by powdery mildew (Uloth *et al*., 2016).
606 Also, in another study, a *B. juncea* genotype, UDN-11-32, was found resistant along with

several genotypes showing moderate resistance (Kumar *et al*., 2017). However, there is a lack of information regarding molecular mapping for resistance against powdery mildew.

2.1.10 Verticillium wilt

Verticillium wilt can be caused by different species of *verticillium*, with *V. longisporum* mainly targeting plants from the Brassicaceae while *V. dahliae* readily infects more than 200 plant species (Depotter *et al*., 2016). However, in 2017 *V. dahliae* was reported for the first time on canola in North America (Hwang *et al*., 2017). Verticillium wilt caused by *V. longisporum* causes serious canola yield losses in Europe, particular Sweden (Heale and Karapapa, 1999). It is almost always associated with *B. napus* and the situation for *B. juncea* has not yet been comprehensively studied. However, *B. juncea* can be affected by *V. dahlia* var. *longisporum*, and though less severely affected than *B. napus*, yield losses were still considerably high (Zeise and Buchmuller, 1997). A possible reason why severe verticillium wilt is not being reported on *B. juncea* is perhaps because *B. juncea* shows strong antifungal effects, especially on microsclerotia germination, as compared with some other *Brassica* species. However, this is unlikely to preclude verticillium wilt from being a future threat to production of *B. juncea* in regions where *V. longisporum* is prevalent. As fungicides are generally ineffective, the current focus for management of verticillium wilt is on identifying quantitative host resistance. However, no resistance genes have yet been identified against verticillium wilt (Depotter *et al*., 2016), but locating host resistance remains an overall focus for *Brassica* spp.

2.2 Bacterial diseases

2.2.1 Black rot

Black rot, caused by *Xanthomonas campestris*, is important across a range of oilseed brassicas, including *B. juncea*, and vegetables including *B. oleracea*, and occurs worldwide (Saharan, 1993; Mulema *et al*., 2012; Singh *et al*., 2016a; Akhtar *et al*., 2017). Black rot most commonly affects leaves of vegetable brassicas and makes them unsaleable, and severe cases involve total yield loss from internal soft rotting in stems. Black rot symptoms on leaves originate from the leaf margins. Vein blackening is initially observed, followed by V-shaped chlorotic lesions of interveinal leaf tissue that eventually progresses to desiccation of interveinal leaf tissue. Affected *B. juncea* plants have been reported to display dark-coloured streaks on stems leading to girdling, softening, and hollowing of the stem due to internal rotting, stem cracking, leaves displaying midrib cracking, wilting and rotting, and lodging of plants. In India, black rot can show up to 60% incidence on different *B. juncea* cultivars (Vir *et al*., 1973) where it can sometimes be very serious in some regions (Shukla *et al*., 2003). Generally, it is not a major issue for *B. juncea* crops where there is effective qualitative and quantitative resistance against the globally predominate *X. campestris* races such as R1 and R4 (Vicente *et al*., 2001; Taylor *et al*., 2002; Tonguç and Griffiths, 2004). However, *B. juncea* is susceptible to races R2, R5, and R6, but fortunately, these historically occur at a lower frequency in the field (Vicente *et al*., 2001). Despite several *B. juncea* cultivars having resistance against one or more prevailing *X. campestris* races (Rathaur *et al*., 2016a; 2016b; Singh *et al*., 2016b), this situation could deteriorate quickly if new races develop. Hence, monitoring for increased frequency of highly virulent and/or new races is essential in regions where *B. juncea* is an important crop, especially in regions where prevailing environmental conditions are conducive for this disease.

Neither genetic mapping nor cloning of resistance genes and loci from *B. juncea* have been published to date (Vicente and Holub, 2013). Fargier and Manceau (2007) postulated that the qualitative resistance genes *R1* (locus *Xca1*), *R5*, and possibly *R4* (locus *Xca4*) are

present in *B. juncea* where *R1* and *R4* are positioned on the B and A genomes, respectively (Vicente *et al*., 2002). Vicente *et al*. (2002) mapped the black rot resistance locus, *XcaI4*, to linkage group N5 in *B. napus* using RFLP markers, which corresponds to linkage group A05 in *B. rapa* (Soengas *et al*., 2007). The RFLP markers for *Xcal4* could be used for *B. juncea* because *R4*, or *Xcal4*, is possibly also present on the *B. juncea* A genome. Recently, Sharma *et al*. (2016) identified molecular markers linked to black rot race 1 resistance loci, *Xca1bc*, on the *B. carinata* B genome with monogenic dominant inheritance. *Xca1bc* was linked to *B. juncea*-derived intron length polymorphic (ILP) markers At1g70610 and At1g71865, and the SSR marker Na14-G02, which are positioned on *B. carinata* linkage group B7. However, analysis of the markers associated with *XcaI4* and *Xca1bc* have not been investigated in terms of their chromosomal position in *B. juncea*.

The absence of investigation into resistance loci *Xca1* (*R1*) and *Xca4* (*R4*), along with resistance gene *R5*, suggest further studies are required for direct identification of linked molecular markers in *B. juncea*. Identification of such genes could be performed using analysis of *B. juncea* genotypes inoculated with isolates of races 1, 3, 4, 7, 8, and 9 possessing avirulence genes *A1*, *A4*, and *A5*. The avirulence genes *avrXccC*/*xop AH* and *AvrBs2* have been identified as responsible for a resistant response in *B. juncea* (Ignatov *et al*., 2002; He *et al*., 2007). Development of isolates with a single avirulence gene allows further analysis by association mapping to identify their corresponding *R* gene, such as *avrXccC* corresponding to *Rc1* (*R1*) (He *et al*., 2007). In addition, quantitative resistance was suggested to be present in *B. juncea*, although no genetic mapping of that resistance has been conducted (Taylor *et al*., 2002).

2.2.2 Leaf blight

Pseudomonas syringae, causal bacterial agent of leaf blight, infects *B. juncea* in Australia (Peters *et al*., 2004) and the USA (Wechter *et al*., 2014; Zhao *et al*., 2000). It is generally considered a minor disease affecting a range of oilseed and vegetable brassicas. In the USA, leaf blight generally causes average yield loss of only 5% in brassicas, including *B. juncea*. However, there, the yield losses in brassicas, including *B. juncea*, can reach up to 100% in severely affected regions (Smith and Keinath, 2004). Symptoms have been reported on *B. juncea* showing brown to tan watersoaked necrotic spots surrounded by faint chlorosis along with multiple lesions coalescing together on leaves to cause extensive leaf necrosis. Typical leaf blight symptoms on other *Brassica* species were angular, watersoaked flecks on leaves that develop to become tan to grey, angular, necrotic lesions. These lesions are usually surrounded by chlorosis with watersoaked margins on the underside of leaves and irregular edges.

A recent study has shown that host resistance reduces disease severity more than pesticide usage, with a reduction in severity by 81% achieved using resistant vegetable-type *B. juncea* (Keinath *et al*., 2016). This highlights the need to maintain a strong focus on locating and using effective host resistance. Historical screening studies with *B. juncea* demonstrated bacterial leaf blight resistance to either American (Zhao *et al*., 2000) or Australian isolates of the pathogen (Oram *et al*., 2003). Oram *et al*. (2003) determined that resistance found in a *B. juncea* segregating population was controlled by two codominant alleles at a single locus. Further, field resistance against *P. syringae* has also been shown to be present in other *B. juncea* accessions (Wechter *et al*., 2007). However, several pathogen isolates sourced from the Florida region showed a highly virulent reaction against a *B. juncea* cultivar (Zhao *et al*., 2000), highlighting the variation in avirulence gene pattern between isolates from different geographic areas. A concern is the lack of available data on molecular mapping for resistance and very limited information on the mode of inheritance of resistance.

However, a related pathogen, *Pseudomonas cannabina*, which causes bacterial leaf and stem rot of hemp (*Cannabis sativa*), has been shown to display field resistance controlled by a single recessive gene, through analysis of segregating populations (resistant *B. juncea* × susceptible *B. juncea*), or control by two genes from the recessive BC (back cross) population (Wechter *et al*., 2012).

2.3 Viral diseases

Important viral diseases that infect various *Brassica* species include TuMV, TYV, and CaMV (Coutts and Jones, 2000; Walsh and Jenner, 2002; 2006; Coutts *et al*., 2010). TuMV and TuYV are prevalent worldwide in *Brassica* crops, including *B. juncea* crops (Hertel *et al*., 2004). In Australia, for example, *B. juncea* is seriously affected by TuMV (Kehoe *et al*., 2010; van Leur *et al*., 2014; Nyalugwe *et al*., 2015a; 2015b; 2016a; 2016b; 2016c). TuMV, a potyvirus, diminishes *B. juncea* plant and seed development, in which it causes up to 85% reduction in mature plant height and 84% in seed yield (Schwinghamer *et al*., 2014, references therein). By comparison, TYV, a polerovirus, causes up to 28% reduction in mature plant height and 12% reduction in seed yield in *B. juncea* (Schwinghamer *et al*., 2014, references therein). All three of these viruses are spread by aphid transmission in the field. A wide host range, for example with TuMV, makes control challenging as this can survive on a range of crop and/or wild species during and between growing seasons (Hughes *et al*., 2002; Schwinghamer *et al*., 2014; Nyalugwe *et al*., 2015a, 2015b).

Several TuMV resistance phenotypes occur in *B. juncea* and single gene resistance is present in this species (Fjellstrom and Williams, 1997; Kehoe *et al*., 2010; Nyalugwe *et al*., 2015a,; 2016a; 2016b; 2016c). Several TuMV resistance phenotypes and/or single gene resistances are found in *B. napus* and *B. carinata* (Nyalugwe *et al*., 2014; Walsh and Jenner, 2002; 2006). However, a strain of TuMV that overcame all TuMV resistance genes in *B.*

napus, failed to overcome dominant TuMV hypersensitive resistance gene *TuRBJU01*, found in *B. juncea* (Nyalugwe *et al*., 2016a; 2016b; 2016c; Guerret *et al*., 2017). *TuRBJU01* was identified by studying Mendelian segregation in a population arising from a cross that included TuMV-resistant *B. juncea* 'Oasis CL' as a parent (Nyalugwe *et al*., 2015a; 2016a). Although *TuRBJU01* has not been cloned to date, it has been shown to provide full dominant resistance in the homozygous condition, but the hypersensitive phenotype was less severe in the heterozygous condition (Nyalugwe *et al*., 2015a). This resistance was effective against most TuMV isolates (Nyalugwe *et al*., 2016a). When Nyalugwe *et al*. (2016c) studied the systemic hypersensitive resistance (SHR) caused by TuMV in *B. juncea*, they found phloem necrosis, xylem occlusion, lignification, and hydrogen peroxide accumulation were all associated with the SHR in plants carrying gene *TuRBJU01*. Phloem necrosis apparently acted as the primary cause of SHR and xylem occlusion as an important secondary cause (Nyalugwe *et al*., 2016c). Nyalugwe *et al*. (2016b) showed that TuMV isolates triggered morphological changes consistent with apoptotic-like programmed cell death and necrosis-like programmed cell death that depended upon isolate virulence and stage of infection reached, respectively. These findings are important as they highlight how quantification of dead cell deformation and electrolyte leakage offer a new understanding of compatible and incompatible plant responses to early virus infection in plants. Such studies provide a basis for further studies to determine the resistance locus involved in *B. juncea* for this disease. In addition, TuMV resistance was shown to be enhanced by an oxidase gene, *BjAOX1a*, which has been cloned from *B. juncea* (Zhu *et al*., 2012). Currently, resistance genes or loci have not been found in *B. juncea* for the other viral diseases, TuYV or CaMV, and further work is required with TuMV resistance gene *TuRBJU01*, such as identification of molecular markers and cloning the gene.

3 Discussion

There are many diseases that affect *B. juncea*. The severity of these diseases depends on several factors, including the environmental conditions of the regions under cultivation and cultivated genotypes. Cropping of *B. juncea* is increasing worldwide, due to its ability to tolerate higher growing season temperatures along with its ability to be productive under lower rainfall conditions compared with *B. napus* (Oram *et al*., 2005). Such conditions are becoming more frequent across at least some of the major oilseed brassica-growing regions; conditions that not only impact plant productivity, but also relative severities of different disease epidemics, and even the expression of host resistances to these brassica diseases (Barbettti *et al*., 2012). Across the different diseases, *B. juncea* and *B. napus* vary in their relative disease susceptibilities and resistances, both at an overall species level, and between different commercial cultivars within either species. As areas sown to *B. juncea* increase, the likelihood of increasing disease issues, including resistance breakdown of host resistances currently available within *B. juncea*, will also increase. Consequent to this, there is a need for renewed focus to identify and understand the underlying resistance mechanisms in *B. juncea* across different aspects, including host physiology and host and pathogen genetics, if future disease epidemics in *B. juncea* are to be curtailed.

There are several disease management strategies that can be applied to reduce the effect of the pathogen on yield loss. These include: (a) foliar fungicide application that is widely used on diseases such as blackleg and sclerotinia stem rot, but this is not consistently effective, nor in many instances cost-effective; (b) deployment of host resistance, including rotation of resistant genotypes having different types of resistance, such as is widely use against blackleg in order to curtail the build-up of pathogen isolates with ability to overcome host resistance; (c) manipulating the plant growth conditions, such as soil nutrition,

775 temperature, and humidity, which influence the severity of disease, as occurs with clubroot;
776 and (d) controlling seed-/soilborne diseases such as alternaria blight and downy mildew by
777 use of disease-free seed or application of seed fungicides (Kolte, 2009; Fry, 2012). While
778 growers would benefit most from a combination and/or integration of these strategies, using
779 host resistance as the basis is the only long-term cost-effective strategy for management of
780 diseases in *B. juncea*.

781 While there has been substantial breeding towards developing disease resistant *B.*
782 *juncea* and identification of host resistances to many of the diseases of *B. juncea*, in general,
783 characterization of resistance genes has been relatively little studied in *B. juncea*. Molecular
784 mapping has been predominantly conducted for blackleg and white rust, caused by *L.*
785 *maculans* and *A. candida*, respectively. Whilst multiple loci have been mapped against
786 blackleg and white rust, it is unknown whether all these are actually different genes, as
787 different markers and lines were used across these studies (Table 1). The resistance genes
788 against blackleg have mainly been dominant in their mode of inheritance (Saal *et al*., 2004;
789 Saal and Struss, 2005; Christianson *et al*., 2006). Most molecular mapping for white rust
790 resistance has involved using *B. juncea* lines from mapping populations, whereas identifying
791 resistance from *B. juncea* for blackleg disease primarily involved using *B. napus*
792 introgression lines using *B. juncea* for resistance (e.g., Christianson *et al*., 2006). Some of the
793 resistance loci detailed in Table 1 have been genetically mapped to linkage groups for *B.*
794 *juncea*. Most of the markers used for mapping were RAPDs and RFLPs. However, there has
795 been limited use of sequence-based markers, such as SSRs, SNPs, and IP markers
796 (Christianson *et al*., 2006; Panjabi-Massand *et al*., 2010). Three recent studies have focused
797 on molecular mapping of sclerotinia resistance in *B. juncea* that carried genomic segments
798 from wild Brassicaceae species. The first involved *B. fruticulosa*, and showed that these
799 possessed high levels of resistance against *S. sclerotiorum*, detecting 10 significant marker

trait associations (Rana *et al*., 2017). The second also involved *B. fruticulosa*, where 20 candidate genes were identified, including *TIR-NBS-LRR* class, chitinase, malectin/receptor-like protein kinase, defensin-like (*DEFL*), desulphoglucosinolate sulphotransferase protein, and lipoxygenase (Atri *et al*., 2019). The third involved a set of *B. juncea–E, cardaminoides* introgression lines, where six marker loci associated with resistance in both A and B genomes were identified, along with an array of resistance mechanisms, including LRR-RLK (receptor-like kinases) genes that encode LRR-protein kinase family proteins, genetic factors associated with pathogen-associated molecular patterns (PAMPs), and effector-triggered immunity (ETI). Except for blackleg, white rust, and sclerotinia resistance, there is no other molecular mapping conducted in *B. juncea* for other fungal, bacterial, or viral diseases mentioned in this review. However, the genetic basis of resistance has been investigated for blackrot, leaf blight, TuMV, hypocotyl rot, and downy mildew. Furthermore, *BjCHI1*, the *B. juncea* gene for *R. solani* resistance, is one of a limited number of *B. juncea* genes cloned for disease resistance, and functional in other plants.

Molecular mapping studies, such as genome-wide association mapping (GWAS) or QTL mapping, are used to identify important markers, especially SNPs, for resistance and to locate the chromosomal position of candidate resistance genes. However, except for the recent studies of Atri *et al*. (2019), who used SNPs to identify 20 candidate genes for *Sclerotinia* resistance in a set of *B. juncea* lines with introgression from *B. fruticulosa*, and that of Rana *et al*. (2019) who used SNPs to identify an array of *Sclerotinia* resistance mechanisms, use of SNP markers for molecular mapping of resistance in *B. juncea* remains a little-explored opportunity. SNPs are one of the preferred molecular markers for mapping of resistance due to being the most abundant molecular marker and simplest form of variation, providing a higher marker density (Varshney *et al*., 2014; Grover and Sharma, 2016). They are the preferred molecular marker for genomewide genotyping for identification of

associated genes, where allelic forms can be detected (Mammadov et al., 2012; Huang and Han, 2014). Although linkage groups for several molecular markers are known for some resistance loci, except for the recent studies by Rana *et al*. (2019) and Atri *et al*. (2019) on *Sclerotinia* resistance in *B. juncea*, there has been no analysis of chromosomal position for these resistance gene loci. This was probably due to absence of a reference genome until that recently published in 2016 by Yang *et al*. (2016). It is this reference genome that has enabled studies where genomic positions of previously mapped resistance genes can be located, through mapping of sequence-based markers. In addition, genome wide identification of resistance genes, which have already been identified in other *Brassica* species (in addition to those for *B. juncea* noted above; Rana *et al*., 2019; Atri *et al*., 2019), is now enabled (Alamery *et al*., 2018).

Marker-assisted selection can be used to identify important germplasm with desirable agronomic traits, such as disease resistance, or for crop improvement. MAS has been implemented in *B. juncea* for important traits such as seed colour, linolenic acid, oil content, yield-influencing traits, and disease resistance, including white rust, blackleg, clubroot, and alternaria blight (Ramchiary *et al*., 2007; Gupta *et al*., 2010; Singh *et al*., 2015; Wang, 2013). Next-generation sequencing (NGS) and genotyping by sequencing (GBS) can accelerate the use of MAS in breeding programmes (He *et al*., 2014) through rapid development and genotyping of thousands of markers.

Resistance could be improved in *B. juncea* through wider use of introgression lines. For example, interspecific hybrids were developed from crossing susceptible *B. juncea* and resistant *B. carinata* (Krishnia *et al*., 2000; Gupta *et al*., 2010), and wild resistant crucifer species may also be used for introgression of resistance into *B. juncea* (Sharma *et al*., 2002), such as introgression of *B. fruticulosa* into *B. juncea* and *E. cardaminoides* into *B. juncea* for *Sclerotinia* resistance (Atri *et al*., 2019; Rana *et al*., 2019). Gene editing and transgenics are

novel approaches for resistance improvement. Enhanced resistance in transgenic *B. juncea* lines against alternaria blight, white rust, and powdery mildew diseases has been shown in several studies (Table 2) (Mondal *et al*., 2003; 2007; Chhikara *et al*., 2012; Rustagi *et al*., 2014; Hada *et al*., 2015; Munir *et al*., 2016; Ali *et al*., 2017). These studies reveal that development of resistance transgenic lines would be an effective approach for *B. juncea* cultivation. However, such an approach might be more practical against some important diseases like alternaria blight caused by *A. brassicae*, where there is no identified source of effective resistance among *B. juncea* germplasm (Grover and Pental, 2003; Al-lami *et al*., 2019c).

The impact of climate changes cannot be ignored in resistance-improvement strategies. Such impacts include changes of temperature, rainfall, and humidity, the main drivers plant disease severity. For example, the expression of some *Brassica* resistance genes against pathogens are temperature dependent, including blackleg (Li *et al*., 2006b), TMV (Nyalugwe *et al*., 2014), *Sclerotinia* (Uloth *et al*., 2015c), powdery mildew (Uloth *et al*., 2018), and downy mildew epidemics in *B. juncea* and *B. napus* (Mohammed *et al*., 2018a). Li *et al*. (2006a) argued that resistance against sclerotinia stem rot may not be currently necessary for *B. juncea* due to its optimal cultivation in low rainfall regions, where the conditions are less favourable for this disease. Climate change will influence future changes in the distribution and challenge of oilseed brassica pathogen threats and corresponding changes in pathogen diversity (Barbetti *et al*., 2012). There is a need for further research into the specific implications of climate change for pathogens on *B. juncea*, particularly as the areas sown to *B. juncea* are expected to increase due it its greater adaptability to warming temperatures and declining seasonal rainfall.

Identification and characterization of resistance genes, along with investigation of quantitative resistance, will help us better understand how the plant defends itself against

these pathogens. In addition, such information can be used to speed up the breeding process and for rotation of resistance sources and types for maintaining effective disease management practices. Further, the availability of the *B. juncea* genome will permit increased understanding of the genetic-induced resistance within and between *Brassica* species, as well as with other genera in the Brassicaceae. An increase in knowledge and understanding about the role of physiological and molecular mechanisms controlling plant immunity, along with the identification of resistance genotypes preferentially with broad-spectrum resistance at both local and international scales, will pave the way for more effective and durable strategies to overcome pathogen attacks in *B. juncea* worldwide. While advances in genotyping and next-generation sequencing are rapidly progressing, the challenge now is to transfer this new knowledge from the laboratory into the field in order to enable better disease management for *Brassica* crops in general, and *B. juncea* in particular.

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Data availability statement

Data sharing is not applicable to this article as no new data were created or analysed in this study.

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