Eur J Plant Pathol (2011) 131:529–537
DOI 10.1007/s10658-011-9828-9

Aggressiveness of eight *Didymella rabiei* isolates from domesticated and wild chickpea native to Turkey and Israel, a case study

Hilal Ozkilinc · Omer Frenkel · Dani Shtienberg · Shahal Abbo · Amir Sherman · Abdullah Kahraman · Canan Can

Accepted: 20 June 2011 / Published online: 7 July 2011

Abstract Ascochyta blight, caused by *Didymella rabiei*, affects both domesticated chickpea and its congeneric wild relatives. The aim of this study was to compare the aggressiveness of *D. rabiei* isolates from wild and domesticated *Cicer* spp. in Turkey and Israel on wild and domesticated hosts from both countries. A total of eight isolates of *D. rabiei* sampled from *C. pinnatifidum*, *C. judaicum* and *C. arietinum* in Turkey and Israel was tested on two domesticated chickpea cultivars and two wild *Cicer* accessions from Turkey and Israel. Using cross-inoculation experiments, we compared pathogen aggressiveness across the different pathogen and host origin combinations. Two measures of aggressiveness were used, incubation period and relative area under the disease progress curve. The eight tested isolates infected all of the host plants, but were more aggressive on their original hosts with one exception; Turkish domesticated isolates were less aggressive on their domesticated host in comparison to the aggressiveness of Israeli domesticated isolates on Turkish domesticated chickpea. *C. judaicum* plants were highly resistant against all of the isolates from different origins except for their own isolates. Regardless of the country of origin, the wild isolates were highly aggressive on domesticated chickpea while the domesticated isolates were less aggressive on the wild hosts compared with the wild isolates. These results suggest that the aggressiveness pattern of *D. rabiei* on different hosts could have been shaped by adaptation to the distinct ecological niches of wild *vs*. domesticated chickpea.

H. Ozkilinc · C. Can
Department of Biology, The University of Gaziantep,
Gaziantep 27310, Turkey

O. Frenkel · S. Abbo
The Levi Eshkol School of Agriculture,
The Hebrew University of Jerusalem,
Rehovot 76100, Israel

O. Frenkel · A. Sherman
Genomics Department, The Volcani Center,
Agricultural Research Organization,
Bet Dagan 50250, Israel

D. Shtienberg (✉)
Department of Plant Pathology and Weed Research,
The Volcani Center, Agricultural Research Organization,
Bet-Dagan 50250, Israel
e-mail: danish@volcani.agri.gov.il

A. Kahraman
Faculty of Agriculture, Department of Field Crops,
Harran University,
Sanliurfa 63100, Turkey

Present Address:
H. Ozkilinc
Department of Plant Pathology,
Washington State University,
Pullman, WA 99164, USA

Keywords Ascochyta blight · Disease severity · Host adaptation · Incubation period · Wild *Cicer*

Introduction

Chickpea (*Cicer arietinum* L.) was first domesticated in southeastern Turkey (Ladizinsky and Adler 1976; Lev-Yadun et al. 2000), where it is grown sympatrically with its wild progenitor *C. reticulatum* and a number of other annual *Cicer* spp. including *C. pinnatifidum, C. bijigum* and *C. echinospermum* (Ladizinsky and Adler 1976; Berger et al. 2003; van der Maesen et al. 2005). Following the Neolithic agricultural revolution, the chickpea crop spread in all directions throughout the east Mediterranean and reached the southern Levant within one millennium (Gopher et al. 2001) where it became sympatric with wild *C. judaicum* (Ben-David et al. 2006). *C. pinnatifidum* and *C. judaicum* are closely related but have an allopatric distribution (Ladizinsky and Adler 1976). *C. pinnatifidum* grows wild in Turkey (mainly in southeastern Turkey), Syria and Lebanon while *C. judaicum* grows in Israel and Jordan (van der Maesen et al. 2005). The two wild species grow in rocky habitats (of limestone or igneous bedrock) and are part of the annual elements of dwarf shrub formation in the hilly parts of the east Mediterranean (Ladizinsky and Adler 1976; Berger et al. 2003; Ben-David et al. 2006). Natural ecosystems have greater environmental heterogeneity, higher species diversity and lower host density than agricultural systems (Burdon et al. 2006; Stukenbrock and McDonald 2008). Often, the different ecological characteristics of natural and agricultural ecosystems impose different selection pressures on the host plants and their respective plant pathogens (Burdon et al. 2006; Abbo et al. 2007; Stukenbrock and McDonald 2008).

Ascochyta blight caused by *Didymella rabiei* (anamorph: *Ascochyta rabiei*) affects all aboveground parts of both domesticated chickpea (Nene 1984; Akem 1999) and its wild relatives *C. pinnatifidum* (Can et al. 2007) and *C. judaicum* (Frenkel et al. 2007). Under environmental conditions that favour development of the pathogen, domesticated chickpea crops are destroyed and yield losses can reach 100% (Nene 1984; Akem 1999; Muehlbauer and Chen 2007). In contrast, Ascochyta blight in natural ecosystems occurs rarely and usually does not devastate its wild *Cicer* hosts (H. Ozkilinc, *unpublished*; Frenkel et al. 2007). Hence, the destructive pathogens affecting domesticated chickpea may have evolved from an ancestral population infecting wild congeneric and conspecific host species and have become more aggressive under domestication (Harlan 1976). This may have happened due to differences in plant density, the genetic structure of the cultigen, seasonal profiles of wild *vs.* domesticated chickpea, environmental and/or ecological characteristics of agricultural and natural ecosystems (Abbo et al. 2003, 2007).

Differences in genetic markers, lack of gene flow and differences in in vitro temperature responses of colony hyphal growth have been documented among *D. rabiei* sampled from domesticated and wild *Cicer* spp. (Frenkel et al. 2010; Ozkilinc et al. 2010). This was interpreted as evidence for the selective role of the distinct ecologies of wild *vs.* domesticated chickpea systems in shaping the adaptive and aggressiveness pattern of *D. rabiei* (Frenkel et al. 2010; Ozkilinc et al. 2010). Recently, Frenkel et al. (2007) showed that *D. rabiei* isolates sampled from Israeli *C. judaicum* are capable of infecting a number of annual wild and domesticated *Cicer* species under laboratory conditions. However, several of the wild annual *Cicer* species tested are native to Turkey and do not occur naturally in Israel. Indeed, Turkey is an important centre of diversity for *Cicer* spp. (Berger et al. 2003). As such it also may be the centre of diversity of its pathogens (Leppik 1970), suggesting that different patterns of *Cicer* spp.-*D. rabiei* interactions may be seen in this area. Frenkel et al. (2008) also showed that on a local scale in Israel, *D. rabiei* isolates sampled from sympatric wild *C. judaicum* and domesticated chickpea hosts were more aggressive on their original host. In most cases parasite populations are expected to have higher mean fitness on their sympatric hosts than on allopatric hosts (Kaltz and Shykoff 1998, Gandon and Michalakis 2002; Laine 2005). For example, Andrivon et al. (2007) showed that French and Moroccan *Phytophthora infestans* populations were better adapted to their local potato cultivars than to allopatric potato cultivars. Therefore, it is advisable to investigate the aggressiveness traits of *D. rabiei* isolates on a broader regional scale, including wild and domesticated hosts (sympatric and allopatric *Cicer* spp.) to better understand the pathogen adaptation profile and its specific-

ity across the sympatric wild-domesticated Near Eastern system.

In this work we studied a limited number of host-pathogen combinations aimed at gaining empirical evidences towards answering the following two questions: 1) what are the aggressiveness patterns of *D. rabiei* isolates from different *Cicer* species hosts on their sympatric and allopatric / wild and domesticated hosts; and 2) do isolates sampled from wild *Cicer* or from domesticated chickpea exhibit a similar level of aggressiveness regardless their country of origin? We tested the aggressiveness of *D. rabiei* isolates sampled from both wild (*C. judaicum* and *C. pinnatifidum*) and domesticated chickpea (*C. arietinum*) in Turkey and Israel on susceptible *C. pinnatifidum/C. judaicum* accessions and on susceptible domesticated chickpea cultivars grown in Turkey/Israel.

Materials and methods

Fungal isolates

D. rabiei was sampled from infected wild and domesticated chickpea hosts in Turkey and in Israel (Frenkel et al. 2010; Ozkilinc et al. 2010). The sample included two isolates from each of *C. pinnatifidum* and *C. arietinum* in Turkey, and two isolates from each of *C. judaicum* and *C. arietinum* in Israel (Table 1). Chosen isolates were collected from areas where wild *Cicer* and domesticated chickpea grow side by side. We preferred to use the term "domesticated isolates" for the isolates sampled from domesticated chickpea and "wild isolates" for the isolates sampled from wild *Cicer* spp. hosts.

The isolates were maintained as single-spore colonies in Petri dishes containing potato dextrose agar medium and were incubated in a growth chamber at 19±1°C under alternating cycles of 12 h of light/darkness. Isolates from wild *Cicer* spp. were transferred to chickpea meal agar medium to enhance sporulation (Wilson and Kaiser 1995; Can et al. 2007; Frenkel et al. 2010). Conidia suspensions of fourteen-day-old colonies were used for inoculations in the aggressiveness experiments.

Plant material

Two domesticated chickpea cultivars and two wild *Cicer* spp. accessions from Turkey and Israel were used in this study. Cultivar Cagatay was bred by The Black Sea Agricultural Research Institute in Samsun, Turkey, and, is known to be susceptible to *D. rabiei* isolates sampled from the southeastern region of Turkey (H. Ozkilinc, *unpublished*). Cultivar Spanish White is highly susceptible to *D. rabiei* (Frenkel et al. 2008) and has been used in Israel for more than half a century. *C. pinnatifidum* accession Cp2 from Adiyaman,Turkey, (37°42′ N and 37°58′ E) and *C. judaicum* accession Cj64 from Nahal Arava, Israel, (31°54′ N and 34°59′ E), chosen from the collection of Ben-David and Abbo (2005), are also susceptible to *D. rabiei* (Frenkel et al. 2008; Ozkilinc 2010). 2–3 seeds were planted in 0.5-l pots and plants were maintained in a greenhouse at 15–25°C under natural light. Wild *Cicer* spp. seeds were scarified to enhance germination. Domesticated chickpea plants were grown for three weeks and wild *Cicer* spp. plants were grown for four weeks under the same conditions prior to inoculation.

Table 1 *Didymella rabiei* isolates sampled from *Cicer* spp. in Turkey and Israel

Country	Province	Latitude	Longitude	Host	Year	Isolate Code
Turkey	Kahramanmaras	37°48′	37°29′	*Cicer arietinum*	2006	Myp3.2
	Adiyaman	37°78′	37°61		2006	Ak4.7
	Kahramanmaras	37°53′	37°35′	*Cicer pinnatifidum*	2005	Cp1.05
	Adiyaman	37°66′	38°32′		2006	Cp2.06
Israel	Judean foothills	31′ 41′	34′ 59′	*Cicer arietinum*	2004	Natif
	Judean foothills	31′ 49′	34′ 55′		2005	Bakoa
	Northwestern Samaria	32′ 34′	35′ 04′	*Cicer judaicum*	2005	Yw15
	Northwestern Samaria	32′ 31′	35′ 08′		2007	Fahm5

Aggressiveness of *D. rabiei* isolates from wild and domesticated hosts

Aggressiveness of the fungal isolates sampled from domesticated chickpea and from wild *Cicer* spp. was tested on all of the host plants. Plants were sprayed with conidial suspension of the pathogen (3×10^5 spores ml^{-1}) with an air pressure hand sprayer to run-off. Water-sprayed plants of each cultivar/accession were used as controls. Then, the plants were immediately covered with two polyethylene bags to maintain moisture and placed in a growth chamber at 19±1°C under the same light conditions described above. Bags were removed 24 h later and the plants were kept in the same growth chamber for the duration of the experiment. Each treatment [*i.e.*, *D. rabiei* isolate (sampled from wild or domesticated chickpea plants) × domesticated chickpea cultivar or wild *Cicer* spp. accession] was repeated three times (three replicates) and the experiment was conducted twice.

The plants were examined 4, 5, 6, 7, 8, 9, 12, 16, and 19 days after inoculation. Two aggressiveness measures were used: the incubation period and the relative area under the disease progress curve (RAUDPC). The appearance of first disease symptoms was used as an estimation for the incubation period (in days). All the aerial plant parts (stems, leaflets and petioles) of each plant in each pot were inspected and disease severity (*i.e.*, the proportion of affected plant area, in %) was determined visually. For example if the affected plant area is ¼ of the total plant material, the score is 25%; with nearly dead plant (virtually no green parts visible) but still with a green stem, the score is 90%. Because the last disease assesments were carried out on day 16 for domesticated chickpea cultivars and on day 19 for wild *Cicer* spp. accessions, RAUDPC was calculated. The area under the disease progress curve (AUDPC in % × days) was calculated using all disease-assesment records. Then, RAUDPC was calculated as "AUDPC/16×100" for domesticated chickpea cultivars and "AUDPC/19×100" for wild *Cicer* spp. accessions. RAUDPC (in %) represents the intensity of the disease over time. Data were analyzed by ANOVA (analysis of variance) using JMP 5.0 software for windows (SAS Institute, Cary, NC). To enable analysis of variance, the disease severity values were normalized by the arcsine square-root transformation (Ahrens et al. 1990). The following ANOVA model was used:

$$\begin{aligned} Y = \mu &+ CI + PO + CP + CO + CI \times PO + CI \\ &\times CP + CI \times CO + PO \times CP + PO \times CO + CP \\ &\times CO + CI \times PO \times CP + CI \times PO \times CO + CI \\ &\times CP \times CO + PO \times CP \times CO + CI \times PO \times CP \\ &\times CO + \text{pathogen isolate}\,[CI, PO] + \text{experiment}; \end{aligned}$$

where Y represents the aggressiveness measure variable (*i.e.*, incubation period or RAUDPC); μ represents the true mean; CI is the 'country of isolate (isolates from Turkey or Israel)' effect; PO is the 'pathogen origin' effect (isolates from wild *Cicer* or domesticated chickpea); CP is the 'country of host plant' effect, it relates to the growth country (Turkey or Israel) of the cultivars/accessions used in the study; CO is the 'cultivar / accession origin' effect (wild *Cicer* or domesticated chickpea host); the following terms are their respective interaction effects; and pathogen isolate (CI, PO) is the individual pathogen isolate effect nested within country of isolate and pathogen origin. Multiple comparisons of the means were done by the Tukey-Kramer HSD test (α=0.05). Experiment effect was insignificant for both incubation period (P=0.90) and RAUDPC (P=0.38) (Table 2). Therefore, the data from both experiments were pooled and the results of both experiments are shown in the present report.

Results

Typical Ascochyta blight symptoms developed on petioles and stems of all the tested domesticated chickpea cultivars and wild *Cicer* accessions from both countries. No morphological differences were observed between symptoms caused by isolates from the different host/country origins. Disease symptoms appeared 4–6 days after inoculation on the domesticated chickpea plants and 5–10 days after inoculation on the wild chickpea plants (Fig. 1). Disease severity ranged from 48.3 to 92.4% on domesticated chickpea plants and from 34.6 to 71.2% on wild chickpea plants (Fig. 1).

The linear ANOVA model used to compare aggressiveness of *D. rabiei* isolates from different origins was highly significant for both incubation

Table 2 Analysis of variance of aggressiveness measures (incubation period and RAUDPC) of *Didymella rabiei* isolates collected from domesticated and wild chickpea from Turkey and Israel. Isolates' aggressiveness was evaluated on Turkish and Israeli domesticated chickpea (*C. arietinum*) cultivars and wild *Cicer* spp. (*C. pinnatifidum* & *C. judaicum*) accessions

Source	Incubation period	RAUDPC
ANOVA		
Model	<0.0001	<0.0001
R^2	0.557	0.670
P values of each effect		
Country of isolate (CI)	0.001	<0.0001
Pathogen origin (PO)	0.173	0.043
Country of host plant (CP)	<0.0001	<0.0001
Cultivar / accession origin (CO)	<0.0001	<0.0001
CI×PO	0.858	0.134
CI×CP	0.533	0.024
CI×CO	0.018	0.539
PO×CP	0.010	0.206
PO×CO	<0.0001	<0.0001
CP×CO	<0.0001	<0.0001
CI×PO×CP	0.047	<0.0001
CI×PO×CO	0.395	0.012
CI×CP×CO	0.043	<0.0001
PO×CP×CO	0.082	0.629
CI×PO×CP×CO	0.019	0.0007
Pathogen isolate [CI, PO]	0.405	0.104
Experiment	0.903	0.382

period ($P<0.0001$) and RAUDPC ($P<0.0001$). The model explained 56% and 67% of the variation in incubation period and RAUDPC, respectively (Table 2). The individual 'pathogen isolate' effect nested within 'country of isolate' and 'pathogen origin' was not significant for aggressiveness measures (Table 2), so average values of the isolates from each country/host origin (*e.g.*, Turkish wild, Israeli wild, Turkish domesticated and Israeli domesticated isolates) were used in the analyses. The interaction "country of isolate × pathogen origin × country of host plant × cultivar/accession origin" had a significant effect on both studied measures and this interaction explained the aggressiveness patterns of the isolates considering the pathogen's and host's origin (Table 2, Fig. 2).

Significant differences in incubation period were mostly observed for the interaction of *D. rabiei* with *C. judaicum*. Israeli isolates from *C. judaicum* had the shortest incubation period (mean=5.2 days) on their own host (Fig. 2). Wild Turkish isolates had long incubation periods (mean=9 days) on *C. judaicum* plants. Both Turkish and Israeli isolates from domesticated chickpea had the longest incubation period on *C. judaicum* plants (mean=11 and 10 days, respectively).

The most severe infection developed when Israeli domesticated chickpea plants were challenged with Israeli domesticated isolates (mean RAUDPC=51%, Fig. 2). In comparison to Israeli domesticated isolates, isolates from Turkish domesticated chickpea, *C. pinnatifidum* and *C. judaicum*, were less aggressive on Israeli domesticated chickpea (mean RAUDPC= 39%, 33%, 30%, Fig. 2). These mean RAUDPC differences were found to be statistically significant using a Student's *t* test (data not shown).

Interestingly, Turkish domesticated isolates caused the lowest level of disease (mean RAUDPC=15%) on their original host compared with the Israeli domesticated, Israeli and Turkish wild isolates on the Turkish domesticated chickpea plants (mean RAUDPC=32, 22 and 19%, respectively).

Turkish and Israeli wild isolates were highly aggressive on their own hosts (*C. pinnatifidum* and *C. judaicum*, respectively) (mean RAUDPC=28% and 31%, respectively) (Fig. 2). While Israeli wild isolates were quite aggressive on *C. pinnatifidum* (mean RAUDPC=17%), Turkish wild isolates exhibited a significantly lower level of aggressiveness on *C. judaicum* plants (mean RAUDPC=8%) (Fig. 2). Domesticated isolates from both Israel and Turkey had significantly low levels of aggressiveness on both *C. pinnatifidum* (mean RAUDPC=14% and 9%, respectively) and on *C. judaicum* (mean RAUDPC= 13%, 10%, respectively) (Fig. 2).

Discussion

All of the tested isolates infected both wild and domesticated chickpea plants, but were generally more aggressive on their original hosts, suggesting a certain degree of host adaptation (Fig. 1). Similarly, Frenkel et al. (2010) found that isolates of *D. rabiei* sampled from sympatric wild and domesticated chickpeas in Israel were better adapted to their original hosts. In our study, we found one exception

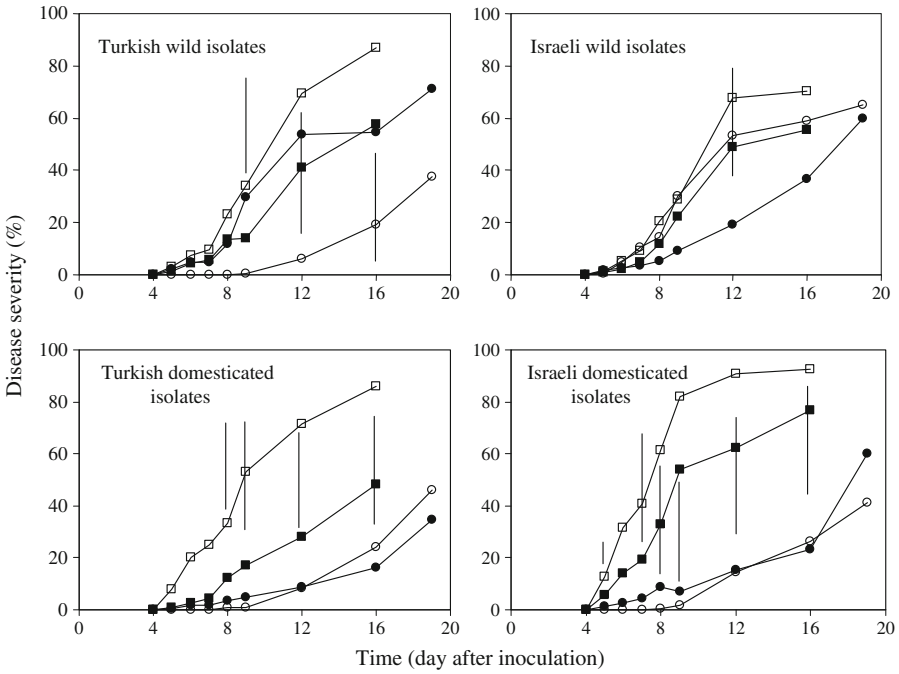

Fig. 1 Disease progress curves of *Didymella rabiei* isolates from different host/country origins (Turkish & Israeli / wild & domesticated *Cicer* spp.) on: the Turkish chickpea cultivar Cagatay (■); Israeli chickpea cultivar Spanish white (□-); Turkish *C. pinnatifidum* accession Cp2 (●); Israeli *C. judaicum* accession Cj64 (○-). The average disease severity values of the two isolates randomly sampled from each host/country origin (Turkish wild, Israeli wild, Turkish domesticated and Israeli domesticated isolates) represents the average values of 6 replicates. Vertical lines represent the least significant differences for the examined dates where differences among hosts were significant at $P=0.05$, as determined by the Tukey-Kramer HSD test

to the pattern of higher aggressiveness on the original host of isolation; on the Turkish cultivar Cagatay, Israeli domesticated isolates were more aggressive than Turkish domesticated isolates (Fig. 1). However, this finding needs to be treated with caution due to the low number of isolates tested. Interestingly, Ozkilinc et al. (2010) showed that Israeli domesticated isolates had higher hyphal growth rates in vitro compared with Turkish domesticated isolates. Therefore, it is tempting to assume that the higher growth rate of Israeli domesticated isolates in vitro is associated with higher aggressiveness in vivo. A positive correlation between in vitro radial growth and pathogenicity was also observed in the Dutch elm disease fungus, *Ophiostoma ulmi* (Brasier and Webber 1987). However, in vitro growth rate does not always correlate with aggressiveness (Thrall et al. 2005; Pariaud et al. 2009).

The wild isolates were quite aggressive on domesticated chickpea cultivars from both countries while the domesticated isolates regardless of the country of origin were less aggressive on the wild hosts. This observation is consistent with the hypothesis that *D. rabiei* infecting wild *Cicer* spp. gave rise to Ascochyta blight on domesticated chickpea at an early stage of the crop's evolutionary history (Abbo et al. 2003). The selection acting on *D. rabiei* populations on domesticated chickpea, has presumably selected isolates better adapted to domesticated chickpea and less adapted to wild chickpea hosts (Abbo et al. 2003, 2007). This hypothesis is supported by the findings of Frenkel et al. (2008) showing that, on a more resistant Israeli cultivar Yarden, the Israeli domesticated isolates had higher aggressiveness when compared with the Israeli wild isolates. Our past work has shown that isolates from domesticated chickpea are better adapted to higher temperatures (Frenkel et al. 2008, 2010; Ozkilinc et al. 2010). This finding supports the hypothesis that chickpea summer cropping has been a traditional practise across the Levant to avoid the winter conditions favouring Ascochyta blight (Abbo et al. 2003). As a result, it is possible that the isolates

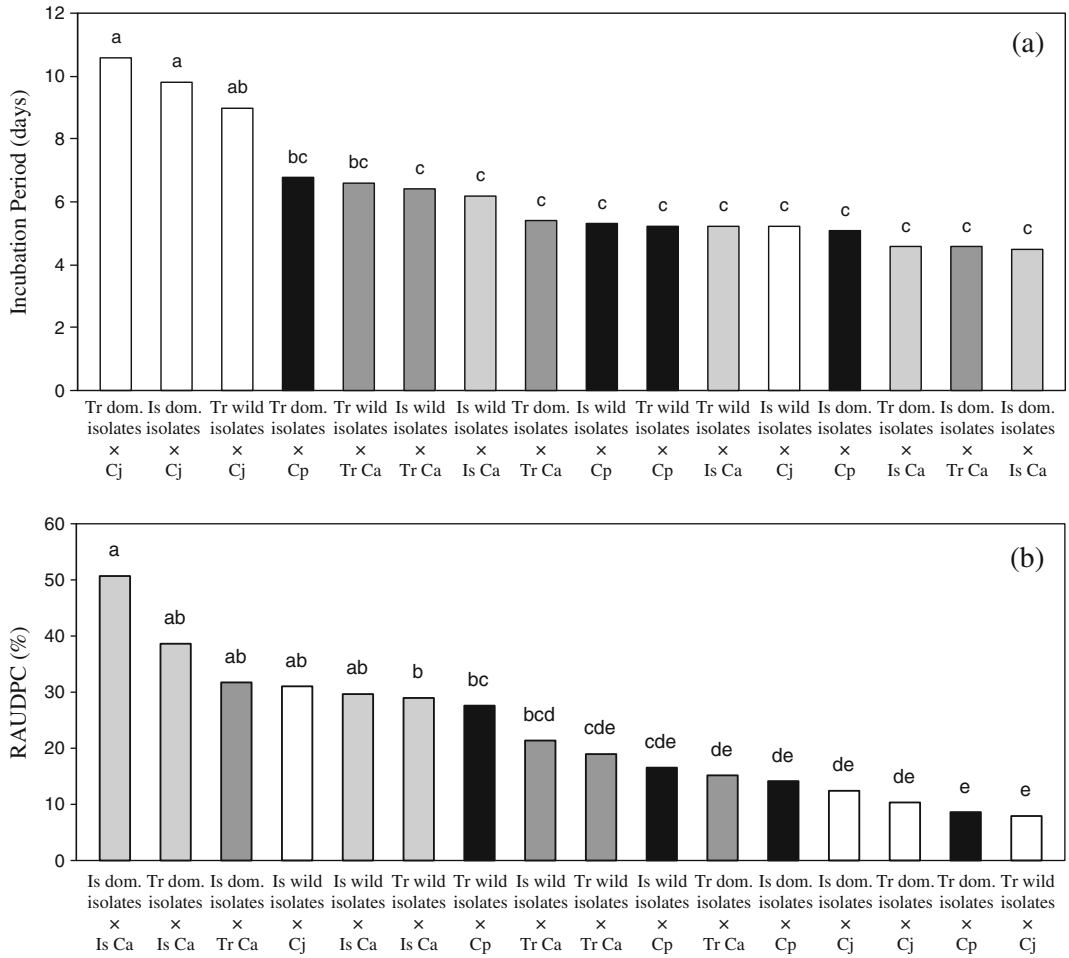

Fig. 2 **a** Incubation period and (**b**) RAUDPC values of each pathogen-isolate combination (from Turkish (Tr)/Israeli (Is) domesticated (dom.)/wild host origin) on each host origin (Turkish/Israeli domesticated and wild *Cicer* spp.). Columns were coloured according to the host type in the interaction [empty/*C. judaicum* (Cj), black/*C. pinnatifidum* (Cp), dark grey/ Turkish *C. arietinum* (Tr Ca), light grey/Israeli *C. arietinum* (Is Ca)]. Values in the same column followed by the same letter are not statistically different (P=0.05) according to the Tukey-Kramer HSD test

from domesticated chickpea have evolved to become better adapted to hotter temperatures and are therefore better competitors and colonizers on the cultigen in its man-managed habitats. Still, both wild and domesticated host plants may serve as a potential source of inoculum for each other *via* pathogen migration during the season, especially (but not exclusively) in sympatric systems. Very limited gene flow was observed between *D. rabiei* infecting wild *C. judaicum* and domesticated chickpea (Ozkilinc et al. 2010). Gene flow may hinder local/host adaptation, but effective gene flow depends on both the performance of the immigrants and the fitness of their offspring and later-generation descendants (Kawecki and Ebert 2004). Interestingly, a significant decrease in pathogenic fitness was detected among the progeny of crosses between Israeli *D. rabiei* isolates sampled from wild and domesticated hosts in laboratory conditions (Frenkel et al. 2010), thereby suggesting that the restricted gene flow between the sympatric wild and domesticated *D. rabiei* isolates does not prevent the slow evolution of host specificity in the domesticated chickpea-wild *Cicer* spp. system (Frenkel et al. 2010). Such a phenomenon was also recorded in other pathosystems such as the *Heterobasidion annosum* species complex in California, where pathogen hybrids were less adapted to the host species compared with their parental isolates (Garbelotto et al. 2007).

Interestingly, *C. judaicum* plants were resistant to all of the isolates from different origins, but not to their own isolates. Isolates from *C. judaicum* had shorter incubation periods and higher RAUDPC values compared with the isolates sampled from other sources on *C. judaicum* plants. Frenkel et al. (2008) also showed higher aggressiveness of *D. rabiei* isolates from *C. judaicum* on their own host compared with the isolates from domesticated hosts. It is interesting to speculate on the mechanisms driving higher aggressiveness of *C. judaicum* isolates on *C. judaicum* compared with the isolates from other hosts. One hypothesis is that *C. judaicum* has had a long evolutionary history of geographic (and ecologic) isolation from other congeneric *Cicer* species and possibly also their *D. rabiei* pathogens. A number of wild annual and perennial *Cicer* spp. are known from the Balkans, Turkey, Syria, Lebanon, Iraq, Iran and in Central Asia. *C. judaicum* however, is known only from Israel and Jordan and no other wild *Cicer* spp. are native to these two countries. *C. judaicum* is found in drier and hotter environments compared with the above-mentioned taxa (Berger et al. 2003). Host–parasite co-evolution processes are tightly coupled with parasite adaptation to the abiotic environment, thereby affecting strength, and even direction, of speciation and local adaptation to temperature (Laine 2008). In the southern Levant, similar effects may have selected for wild *D. rabiei* pathogens with environmental requirements closer to that of domesticated chickpea. A larger sampling from annual and perennial wild *Cicer* species and their *Ascochyta* pathogens is required for a deeper understanding of the *C. judaicum-D. rabiei* interaction. At present, we have no information on the genetic control of aggressiveness in the pathogen nor on the genetic basis of resistance in the wild hosts. Advances in understanding host-pathogen co-evolutionary interactions requires integrating knowledge of the molecular basis of host resistance and pathogen virulence (Burdon and Thrall 2009).

Our results have shown that aggressiveness patterns differ among *D. rabiei* isolates from wild and domesticated hosts and suggest that the distinct ecology of wild *vs*. domesticated chickpea shapes the aggressiveness pattern of *D. rabiei* resulting in differential adaptive (phenotypic) and genetic structure across the wild-domesticated pathosystem. This is in line with the reports of Frenkel et al. (2010) and Ozkilinc et al. (2010) on different ecologically adaptive traits and the molecular divergence of *D. rabiei* from wild and domesticated hosts. While these results provide some clues about the *Cicer-D. rabiei* pathosystem in wild and domesticated / sympatric and allopatric formations, a larger number of isolates should be studied to obtain deeper understanding of aggressiveness and host/local adaptation patterns of the pathogen and its wild and domesticated hosts.

Acknowledgements The authors would like to thank Profs. Avigdor Cahaner and Yonathan Elkind (Institute of Plant Science, Hebrew University, Rehovot, Israel) for their valuable statistical advice and Dr. Tobin Peever (Department of Plant Pathology, Washington State University, Pullman, USA) for his valuable comments. We would like to thank Mr. Haim Vintal for technical assistance. The work of Dr. H. Ozkilinc in Drs. Sherman and Shtienberg laboratories was made possible by a research scholarship granted to H. Ozkilinc by the Israeli Ministry of Foreign Affairs.

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