Theor Appl Genet (2009) 118:1601–1608
DOI 10.1007/s00122-009-1008-3

ORIGINAL PAPER

Paraquat resistance in a *Lolium rigidum* population is governed by one major nuclear gene

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Received: 18 November 2008 / Accepted: 8 March 2009 / Published online: 24 March 2009

Abstract Paraquat resistance in an annual ryegrass (*Lolium rigidum* Gaud.) population (AFLR1) has been attributed to reduced paraquat translocation. Genetic inheritance of paraquat resistance in this population was investigated in the present study. The paraquat dose response of progeny from 8 F_1 families was more similar to that of the resistant than the susceptible parent, while the equivalent data for a further three families were intermediate compared to those of the parental populations. No significant differences in dose response were observed between reciprocal crosses of specific F_1 families. These results suggest that paraquat resistance in AFLR1 is inherited as a dominant or partially dominant nuclear-encoded trait. Pseudo-F_2 (ψ-F_2) generation seedlings were treated with multiple dose rates sufficient to control the susceptible parental population, and observed segregation ratios in all instances conformed to a 3:1 (resistant:susceptible) segregation ratio, and this ratio was further confirmed by individual phenotyping of cloned plant genotypes. A single major nuclear gene is hence apparently responsible for evolved paraquat resistance in AFLR1.

Communicated by M. Kearsey.

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Introduction

Paraquat is a non-selective, rapid-action bipyridyl herbicide that has been used commercially for over 40 years. Paraquat exerts a phytotoxic effect by catalyzing the transfer of electrons from photosystem I (PSI) in the chloroplast membrane to molecular oxygen, producing reactive oxygen species that cause lipid peroxidation and membrane damage (reviewed by Preston 1994). Paraquat is widely used for total weed control in agriculture and industry. A major use of paraquat is for pre-planting weed control in minimum- and no-tillage cropping systems. After more than four decades of use, there have been relatively few cases of evolved resistance to this herbicide compared to evolved resistance to other herbicides of different mode of actions, although biotypes of 24 weed species worldwide have evolved paraquat resistance (reviewed by Preston 1994; Heap 2008 online). In Australia, for example, evolved paraquat resistance has been documented in biotypes of four species: *Arctotheca calendula* (Powles et al. 1989), *Hordeum glaucum* (Powles 1986), *Hordeum leporinum* (Tucker and Powles 1991) and *Vulpia bromoides* (Purba et al. 1993b). In majority of the studied examples, herbicide resistance is determined by single, nuclear-encoded genes with partial or full dominance, with a few examples of recessive, multigenic or cytoplasmic control (reviewed by Darmency 1994). The inheritance of paraquat resistance in field-evolved resistant *Conyza bonariensis*, *Erigeron philadelphicus*, *E. canadenisis*, *H. glaucum*, *H. leporinum* and *A. calendula* plants has been determined as monogenic in nature (reviewed by Preston 1994). In contrast, recurrent selection for paraquat resistance in *Lolium perenne* suggested oligogenic control (Faulkner 1974). Additionally, paraquat resistance in the fern species *Ceratopteris richardii* was attributed to a nuclear-encoded recessive gene (Hickok and Schwarz 1989).

Lolium species are economically important agricultural weed species in several parts of the world. There are three well-recognized cross pollinated species within the *Lolium* genus: *L. perenne, L. multiflorum* and *L rigidum*. These three *Lolium* species freely cross pollinate and the hybrids are highly fertile. *L. rigidum* is an annual, obligate cross pollinated diploid species (2n = 14). Paraquat resistance has evolved in many South African populations of *L. rigidum* and the mechanism of resistance has been determined as reduced herbicide translocation (Yu et al. 2004, 2007). However, the molecular basis for the reduced translocation-based paraquat resistance in *Lolium* and other species remains unknown. The present study aimed to determine the genetic inheritance of paraquat resistance in one of these *L. rigidum* populations, so as to provide the basis for future molecular genetics-based resistance gene isolation strategies in *L. rigidum*.

Materials and methods

Plant material

Seeds of the *L. rigidum* AFLR1 paraquat-resistant population were originally collected from a vineyard situated near Stellenbosch in the Western Cape, South Africa (Yu et al. 2004). Seedlings grown from original resistant seed were treated with paraquat at a commercially relevant rate (200 g ha^{-1}), and survivors were grown to maturity and polycrossed (randomly inter-mated) to produce bulked seeds used for the experiments (hereafter referred to as the R population). This R population was further selected with the field rate paraquat and the survival rate was 100%. Seeds (about 50) of the R and a known susceptible (S) population (VLR1) were germinated in plastic trays (35 × 28 × 6 cm) containing potting mixture (50% sand and 50% peat) and grown in a glasshouse at 20/15°C day/night temperature under natural sunlight with regular watering and fertilization. At the three-leaf stage, the R seedlings were treated with 200 g paraquat ha^{-1}.

Generation of F_1 and pseudo-F_2 families

Before flowering, single paraquat surviving R and untreated S plants were paired and the two pots were enclosed in a 1.5-m high plastic enclosure to restrict external pollen movement. Plants were maintained in the glasshouse to maturity and seeds collected separately from R and S maternal parents within each cross. About 20 plants from each F_1 family were grown to the tillering stage and each individual was then divided into two clones. When the clones were growing vigorously, each clone was treated with one of the two paraquat rates (100 and 200 g ha^{-1}) to test for paraquat resistance segregation. The S parental plants were also included and treated the same way as controls. In the case of clear segregation at 100 g paraquat ha^{-1} within F_1 sib-ships, these families were discarded because they may derive from heterozygosity within the R base population, and the intention was to base the crossing structure on an RR × SS parental structure. Individual F_1 resistant plants from four F1 families were pair-crossed either within the same F_1 family (within-family pseudo-F_2: W-ψ-F_2) or across different F_1 families (between-family pseudo-F_2: B-ψ-F_2) to produce progeny sets. Seeds from each pair-cross were pooled.

Response of parents, F_1 and ψ-F_2 seedlings to paraquat treatment

Seeds were germinated in pots or trays containing potting mixture and maintained outdoors during the normal growing season. Seedlings from parent populations and F_1 families at the 2–3 leaf stage were treated with paraquat at rates 0, 12.5, 25, 50, 100 and 200 g ha^{-1} (about 40 seedlings per pot, two pots per treatment), and seedlings of ψ-F_2 families were treated at 100 and 200 g ha^{-1} (about 60–95 seedlings per tray per treatment depending on germination rate of 100 seeds). Paraquat was applied as a commercial formulation in 112 L ha^{-1} water, delivered in two passes at 200 kPa using a cabinet sprayer equipped with two flat fan nozzles. Treated plants were maintained outdoors during the normal winter growing season. Due to space limit, Experiment I was conduced with 11 F_1 and 9 ψ-F_2 families from April to May, and Experiment II with 7 ψ-F_2 families from June to July. Mortality was determined 2–3 weeks after herbicide application. Plants were recorded as dead if no new growth or active tillering was observed.

Phenotyping ψ-F_2 individuals using cloned plants

Individual plants from two ψ-F_2 families (#32 and 34) were allowed to grow to the tillering stage. Each individual was then separated into 6 ramet clones (the root and shoot was trimmed) and when growth was well established (about 1 week after cloning) each ramet clone was treated with one of the six paraquat rates (0, 100, 200, 400, 1,600 and 3,200 g paraquat ha^{-1}). About 30 individual plants from the S population were also included and treated similarly. Treated plants were maintained outdoors during the normal winter growing season (this experiment was conducted from June to September). The phenotype for each plant was determined 4–5 weeks after treatment according to its response to the six paraquat rates, in comparison to that of the S plants. The phenotype (S or non-S) of each progeny plant was accurately determined in comparison to the behavior of the parental S plant clones. Individuals whose

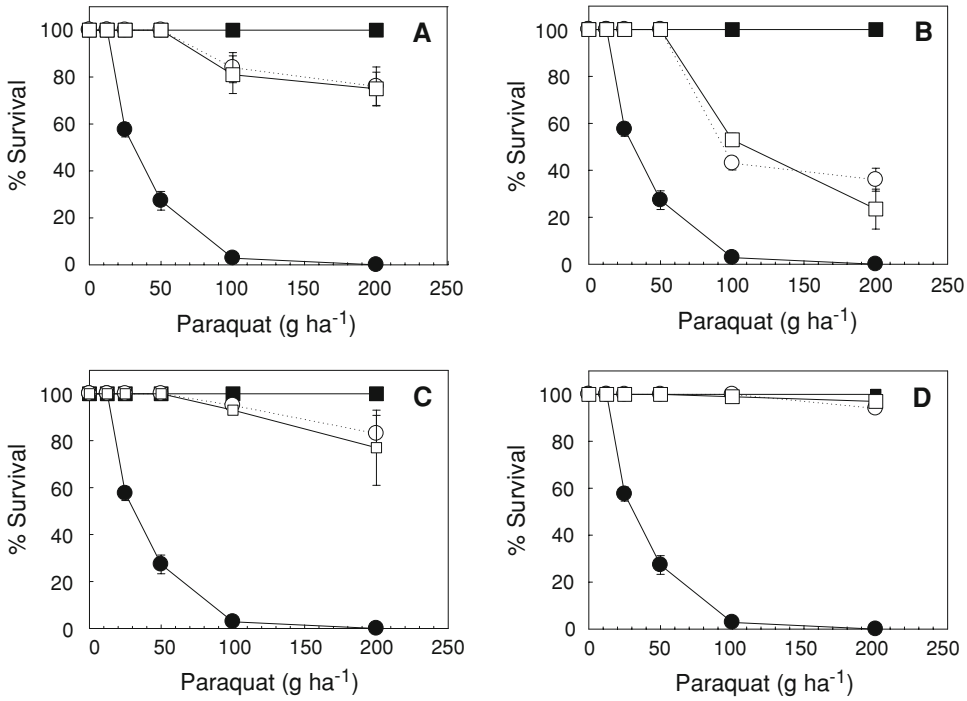

Fig. 1 Dose response to paraquat of the parental susceptible (*S*) (*filled circle*), parental resistant (*R*) (*filled square*), F_1 maternal S (*open circle*), and F_1 maternal R (*open square*) populations of *L. rigidum*. Data in **a** are the means of all 11 F_1 families and data in **b**, **c** and **d** are from single F_1 families representing intermediate, nearly-dominant and dominant responses to paraquat, respectively

clones were killed by any of the three rates (100, 200, 400 g paraquat ha^{-1}) was assigned as S, whereas individuals were classified as non-S if their clones survived all these three or higher rates. Any ambiguity was clarified by excising foliage of treated plants and after 5–7 days re-treatment at the same paraquat dose.

Statistical analysis

The herbicide rate causing 50% mortality (LD_{50}) of plants was calculated using Sigma Plot® (version 8.02, SPSS Inc. 233 South Wacker Drive, Chicage, IL) software capable of non-linear regression analysis (log-logistic model). Chi-square analysis of segregation of paraquat resistance in ψ-F_2 families was performed. The heterogeneity test was conducted to examine the variations among ψ-F_2 families in paraquat resistance. The mortality of the treated parental R and S and F_1 plants was also considered when calculating the expected mortality for each ψ-F_2 family from paraquat treatment at each rate.

Results

Paraquat resistance is inherited as a dominant or partially dominant trait

Dose response experiments were conducted on 11 F_1 families to determine the inheritance pattern and the level of dominance for paraquat resistance. Pooled data from all 11 F_1 families (Fig. 1a) demonstrated a resistance level intermediate between the R and S parental populations. A close-to-identical response was observed for the progeny sets harvested from the susceptible maternal parent [F_1(S)] and the resistant paternal parent [F_1(R)], indicating that the resistance trait in the population is pollen-transmitted, with no obvious indication of maternal inheritance. Some variation in response to paraquat in F_1 families was observed. A total of 27% (3/11) of the F_1 families (Fig. 1b) showed an intermediate response with a paraquat LD_{50} of 94–98 g ha^{-1} compared to 30 g ha^{-1} for the S and >200 g ha^{-1} for the R parental populations. In contrast, 45% (5/11) and 27% (3/11) of the F_1 families showed a response more similar (Fig. 1c) or nearly-identical (Fig. 1d), respectively, to the R parental population. These observations suggest that paraquat resistance in AFLR1 is nuclear-encoded and controlled by dominant or partially dominant genetic factors within the evaluated dose range.

Paraquat resistance is controlled by a single major gene

Response of ψ-F_2 seedlings to paraquat treatment

We chose resistant individuals from four F1 families which showed high-level paraquat resistance (belonging to types C and D dose response in Fig. 1) to produce sixteen segregating ψ-F_2 families. These ψ-F_2 families were examined to determine whether paraquat resistance in *L. rigidum* is controlled by a single or multiple genes. Seedlings from each of these families were treated with two different rates of paraquat (100 and 200 g ha^{-1}, respectively). The number of surviving and dead plants was recorded. Percentage mortality

Table 1 Chi-square analysis and heterogeneity test for goodness of fit of the observed segregation for paraquat resistance in ψ-F_2 families to the 3:1 (R:S) ratio expected from the single gene control model

ψ-F_2 family	Observed		Total	Expected		χ^2	Probability
	Dead	Alive		Dead	Alive		
Experiment I							
Within-F_1 family cross (W-ψ-F_2)							
#27	23	55	78	23	55	0.002	0.96
#31	16	50	66	19	47	0.80	0.37
#32	23	55	78	23	55	0.002	0.96
Between-F_1 family cross (B-ψ-F_2)							
#24	23	64	87	25	62	0.33	0.56
#28	22	67	89	26	63	0.88	0.35
#34	24	62	86	25	61	0.08	0.78
#35	25	59	84	25	59	0.011	0.92
#36	19	47	66	19	47	0.007	0.93
#39	18	59	77	22.5	54.5	1.28	0.26
Total	193	518	711	208	503	1.53	0.22
Heterogeneity test						1.87	0.99
Behavior of controls							
Parental S	70	2	72				
Parental R	0	74	74				
F_1(S), + F_1(R)	54	487	541				
Experiment II							
Within-F_1 family cross (W-ψ-F_2)							
#29	9	48	57	14	43	2.73	0.10
#41	19	50	69	17	52	0.19	0.66
Between-F_1 family cross (B-ψ-F_2)							
#25	19	70	89	22	67	0.72	0.40
#26	24	66	90	23	67	0.09	0.76
#30	22	68	90	23	67	0.03	0.86
#33	16	72	88	22	66	2.33	0.13
#38	25	66	91	23	68	0.24	0.63
Total	134	440	574	145	429	1.10	0.29
Heterogeneity test						5.20	0.52
Behavior of controls							
Parental S	77	0	77				
Parental R	0	60	60				
F_1(S), + F_1(R)	9	623	632				

Plants at the 2–3 leaf stage were treated with paraquat at 100 g ha^{-1}. Mortality was determined 2–3 weeks after herbicide application

of different phenotypic classes was also considered for paraquat treatment at each rate, assuming monogenic control. The observed numbers for mortality and survival for each ψ-F_2 sib-ship was found to be similar to the predicted values (assuming 3:1 segregation of resistant and susceptible individuals, $P > 0.05$) at the 100 or 200 g paraquat ha^{-1} rate (Tables 1, 2), consistent with a single gene effect. The heterogeneity test of pooled data was not significant ($P > 0.05$) at each paraquat rate (Tables 1, 2) indicating that all ψ-F_2 progenies behaved similarly. In general, goodness-of-fit to the predicted value was superior at 100 than at 200 g paraquat ha^{-1}. At the lower dose rate, the observed mortality in ψ-F_2#29, 33 and 39 was less than expected (although statistically not significant ($P > 0.05$): Table 1). At 200 g paraquat ha^{-1}, however, observed mortality was generally, and especially in ψ-F_2#31, 34 and 35, greater than expected (although $P > 0.05$; Table 2).

Phenotyping ψ-F_2 individuals using cloned plants

In order to confirm the 3:1 (R:S) segregation ratio observed in ψ-F_2 families at the seedling stage, about 200 individual plants from each of two sib-ships(#32 and 34) were grown to the tiller stage. Each individual was separated into 6 ramet clones and each ramet was treated with one of the multiple paraquat rates (see "Materials and Methods"). The

Table 2 Chi-square analysis and heterogeneity test for goodness of fit of the observed segregation for paraquat resistance in ψ-F_2 families to the 3:1 (R:S) ratio expected from the single gene control model

ψ-F_2 family	Observed		Total	Expected		χ^2	Probability
	Dead	Alive		Dead	Alive		
Experiment I							
Within-F_1 family cross (W-ψ-F_2)							
#27	27	59	86	26	60	0.08	0.78
#31	23	42	65	19.5	45.5	0.9	0.34
#32	31	59	90	27	63	0.85	0.36
Between-F_1 family cross (B-ψ-F_2)							
#24	25	49	74	22	52	0.5	0.48
#28	20	66	86	26	60	1.86	0.17
#34	30	57	87	26	61	0.83	0.36
#35	34	53	87	26	61	3.42	0.07
#36	18	32	50	15	35	0.86	0.36
#39	21	53	74	22	52	0.09	0.76
Total	229	470	699	210	489	2.54	0.11
Heterogeneity test						6.85	0.55
Behavior of controls							
Parental S	73	0	73				
Parental R	0	74	74				
F_1(S), + F_1(R)	54	486	540				
Experiment II							
Within-F_1 family cross (W-ψ-F_2)							
#29	16	41	57	16	41	0.009	0.92
#41	20	51	71	20	51	0.02	0.90
Between-F_1 family cross (B-ψ-F_2)							
#25	21	60	81	22	59	0.10	0.75
#26	25	69	94	26	68	0.04	0.84
#30	23	57	80	22	58	0.063	0.80
#33	31	54	85	23	62	3.43	0.06
#38	34	60	94	26	68	3.54	0.06
Total	170	392	562	155	407	2.13	0.14
Heterogeneity test						5.07	0.53
Behavior of controls							
Parental S	74	0	74				
Parental R	0	60	60				
F_1(S), + F_1(R)	62	551	613				

Plants at the 2–3 leaf stage were treated with paraquat at 200 g ha^{-1}. Mortality was determined 2–3 weeks after herbicide application

number of non-S and S plants observed in ψ-F_2#34 was consistent with predicted ratios based on the single gene hypothesis, while for ψ-F_2#32, the number of observed non-S and S plants was slightly but not significantly lower than predicted (Table 3). The test of heterogeneity between these two families was not significant ($P > 0.05$), demonstrating a similar response to paraquat treatment. Small variation in the response to paraquat between the two ψ-F_2 sib-ships is again likely to be due to genetic differences between F_1 families. The ψ-F_2#32 population is a within-F_1 family cross (as represented in Fig. 1d), while ψ-F_2#34 is a between-F_1 family cross (as represented in Fig. 1c, d). In total across families, the observed segregation ratio of non-S:S is consistent with the predicted ratio of 3:1 (R:S). These results confirm that a single major nuclear gene controls paraquat resistance in this *L. rigidum* population, while additional minor gene(s) may also contribute to variation.

Discussion

Our previous studies have demonstrated that paraquat resistance in South African *L. rigidum* populations is due to reduced paraquat translocation (Yu et al. 2004, 2007), and in this study it is clear that inheritance of paraquat resistance

Table 3 Phenotyping, Chi-square analysis and heterogeneity test for goodness of fit of the observed segregation for paraquat resistance in two ψ-F_2 families to the 3:1 (R:S) ratio expected from the single gene control model

Ψ-F_2 family	Observed		Total	Expected		χ^2	Probability
	S	Non-S		S	Non-S		
Within-F_1 family cross (W-ψ-F_2)							
#32	39	159	198	49	149	2.71	0.10
Between-F_1 family cross (B-ψ-F_2)							
#34	51	149	200	50	150	0.03	0.87
Total	90	308	398	99	299	1.09	0.30
Heterogeneity test						1.65	0.20

Individual plants were separated into 6 clones and each clone was treated with one of the 6 paraquat rates (0, 100, 200, 400, 1,600 and 3,200 g h^{-1}). Mortality was determined 4–5 weeks after herbicide application

in the AFLR1 population is controlled by a single nuclear gene. The level of dominance varied among the F1 families. While the majority of the F_1s displayed a nearly dominant resistance nature, a few (3 out of 11) families demonstrated a clear intermediate type of response to paraquat (Fig. 1b). This probably reflects genetic diversity between individual *L. rigidum* plants, or conversely, it may be possible that these 3 F_1 families are not based on a RR × SS cross structure but rather a back crosses (RS × SS) structure. Although efforts were made to ensure that the R parents of F_1s were homozygous by repeated selections with paraquat, it is still possible the heterozygous individuals were used in the pair crosses due to the dominant nature of the resistant trait. However, segregation in these 3 F_1 families, which was determined by small number of plants at 100 g paraquat ha^{-1}, was not apparent and therefore they were not excluded from the experiment.

In addition, the greater number of observed survivors (although statistically not significant) at the lower paraquat rate (100 g ha^{-1}; Table 1) may indicate the effect of one (or more) minor modifier gene(s). In contrast, the greater observed mortality (although statistically not significant) at the higher paraquat rate (200 g ha^{-1}; Table 2) is possibly due to the elimination of a proportion of heterozygous individuals at the higher dose rate. Response to paraquat treatment of intermediate-type (heterozygous) individuals in paraquat-resistant *H. glaucum* has been shown to be affected by environment conditions prevailing over the period following herbicide application (Islam and Powles 1988). A major factor affecting response to treatment in resistant populations would be temperature variations, as observed in our previous study with the same resistant *L. rigidum* population (Yu et al. 2004) as well as in paraquat resistant *H. glaucum* and *H. leporinum* populations (Purba et al. 1995). In field-evolved paraquat resistant biotypes of *C. bonariensis*, resistance was stated to be either due to enhanced activities of a number of antioxidant enzymes (Shaaltiel and Gressel 1986; Ye et al. 2000; Ye and Gressel 2000), or due to reduced paraquat mobility (or rapid sequestration) (Fuerst et al. 1985; Norman et al. 1994). However, activities of antioxidant enzymes (superoxide dismutase, ascorbate peroxidase and glutathione reductase) in F_1 were found as high as in the resistant parent and the higher activities co-segregated with paraquat resistance in F_2 (Shaaltiel et al. 1988). Therefore, a single, dominant, nuclear gene was suggested to be responsible for paraquat resistance in *C. bonariensis* through pleiotropic control of the levels of several antioxidant enzymes (Shaaltiel et al. 1988). In contrast, in paraquat resistant biotypes of *E. philadelphicus* and *E. canadensis*, paraquat resistance is not due to enhanced activity of antioxidant enzymes (Turcsanyi et al. 1998) but due to reduced paraquat movement (Tanaka et al. 1986). Paraquat resistance in these biotypes is also controlled by a single dominant nuclear gene (Itoh and Miyahara 1984; Yamasue et al. 1992). Similarly, in *H. glaucum, H. leporinum* and *A. calendula*, paraquat resistance is related to reduced paraquat translocation or penetration to the active site (chloroplast) (Bishop et al. 1987; Preston et al. 1992, 1994, 2005; Purba et al. 1995; Soar et al. 2003). Paraquat resistance in these populations is inherited as a single nuclear gene-encoded partially dominant trait (Islam and Powles 1988; Purba et al. 1993a). Therefore, all of these inheritance studies establish that field-evolved paraquat resistance, regardless of mechanism (reduced paraquat translocation/rapid sequestration, or enhanced activities of antioxidant enzymes, or both), is single nuclear gene endowed, and the level of dominance varies dependent on plant species, resistance mechanisms and paraquat rate range evaluated.

While paraquat resistance in this *L. rigidum* population is single gene endowed, the underlying molecular basis of the reduced paraquat translocation resistance mechanism remains unknown. Our ultimate objective is to identify the molecular genetic basis of paraquat resistance. The pair cross-derived population development, inheritance studies and population-based phenotyping provide the basis for

future detailed molecular genetic studies of paraquat resistance in *L. rigidum*. Although very limited molecular genetic marker-based mapping work has been so far performed for this species (Forster et al. 2005), the closely related pasture species *L. perenne* has been the subject of major developmental efforts (Forster et al. 2004, 2008). Detailed genetic maps incorporating restriction fragment length polymorphism (RFLP), amplified fragment length polymorphism (AFLP), simple sequence repeat (SSR) and single nucleotide polymorphism (SNP) markers have been developed for *L. perenne* (Jones et al. 2002a, b; Faville et al. 2004; Cogan et al. 2006). As a very high proportion of perennial ryegrass-derived SSR markers are directly cross-transferable and informative in *L. rigidum* (Jones et al. 2001; Forster et al. 2005), the ψ-F_2 sib-ships described here will be prioritized in future for bulked sergeant analysis-based screening to identify the genomic location of the paraquat resistance gene. Between-family ψ-F_2 populations will be given preference, due to the possible complicating effects of gametophytic self-incompatibility (SI) in cross-pollinated *Lolium* species.

In summary, paraquat resistance in *L. rigidum* population AFLR1 (due to reduced paraquat translocation) has been demonstrated in this study to be controlled by a nuclear-encoded gene. The resistance is inherited in a dominant or partially dominant fashion. Segregation patterns in ψ-F_2 populations are consistent with a monogenic trait, although other minor modifier gene(s) may play a role at low paraquat doses. This inheritance pattern resembles those identified for reduced translocation-based paraquat resistance in other weed species. The qualitative nature of the genetic control mechanism, together with the obligate cross-pollinated reproductive habit of *L. rigidum* populations, helps to explain the rapid increase in frequency and spread of resistance alleles under paraquat-based selection. The genetic inheritance studies and population-based phenotyping provide the basis for future molecular genetics-based resistance gene isolation strategies in *L. rigidum*.

Acknowledgments The Western Australian Herbicide Research Initiative (WAHRI) is funded by the Grains Research and Development Corporation of Australia (GRDC). This research is partially funded by the Australian Research Council through an ARC-Linkage Project (LP0669035). The authors wish to thank Dr. Noel Cogan for fruitful discussion.

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