# Chapter 15
# Herbicide Resistance in *Setaria*

**Henri Darmency, TianYu Wang, and Christophe Délye**

**Abstract** The four documented cases of field selection for herbicide resistance in weedy *Setaria* are described in this chapter. In each case, weed control failure was observed in practice in the field. In all cases, resistance was target-site-based resistance and was due to single nucleotide mutations causing amino-acid substitutions at codon 264 of psbA (photosystem II inhibitors), codons 136 and 239 of α2-tubulin (tubulin polymerization inhibitors), codon 1781 of acetyl-CoA carboxylase (acetyl-CoA carboxylase inhibitors), or codons 653 or 654 of acetolactate-synthase (acetolactate-synthase inhibitors). The heredity of resistance in these cases was maternal, nuclear recessive, nuclear dominant, or partially dominant, respectively. Pleiotropic effects of the mutant alleles were observed on seed production for the herbicide-resistant alleles Gly-264 of psbA and Ile-239 of α2-tubulin (22 % yield reduction for both alleles), but not for the Leu-1781 acetyl-CoA carboxylase allele. These alleles were introgressed in foxtail millet (*S. italica*) to develop herbicide-resistant genetic resources and germplasm with the aim to produce and release elite varieties of foxtail millet. This material was also used to study pollen dispersal and possible gene flow between weedy *Setaria* and cultivated foxtail millet.

**Keywords** *Setaria* • Millet • Foxtail • Weed • Herbicide • Resistance • Gene flow

## 15.1 Introduction

From the middle of the nineties, weed control in arable fields, roadsides, urban and industrial areas has most often relied upon herbicide spray. In various places where the same herbicide was continuously used, herbicide-resistant plants were selected and have caused trouble to farmers (Beckie and Tardif 2012; Délye et al. 2013). Globally, there are 245 species that have evolved resistance to 22 of the 25

H. Darmency, M.Sc., Ph.D. (✉) • C. Délye
Agroécologie, AgroSup Dijon, INRA, Univ. Bourgogne Franche-Comté,
F-21000 Dijon, France
e-mail: henri.darmency@dijon.inra.fr

T. Wang
Institute of Crop Sciences, Chinese Academy of Agriculture Sciences, Beijing, 100081 China



A. Doust, X. Diao (eds.), *Genetics and Genomics of Setaria*, Plant Genetics and Genomics: Crops and Models 19, DOI 10.1007/978-3-319-45105-3_15

known herbicide modes of action, in 85 crops and in 66 countries (Heap 2015). Different mechanisms are responsible for resistance, an adaptive response of weeds to the herbicide selection pressure: (1) escape of the spraying period via modified phenology; (2) reduction in herbicide penetration through modified cuticle properties; (3) altered translocation of the herbicide toward its target site; (4) sequestration of the herbicide away from its target site; (5) enhanced degradation of the herbicide; (6) mutation at the herbicide target site; (7) overproduction of the herbicide target site; and (8) compensation for deleterious effects of the action of the herbicide (Délye et al. 2013).

The *Setaria* genus also demonstrates herbicide resistance. While one species (*S. italica*, foxtail millet) is a staple crop in Asia and Africa, the most widespread *Setaria* species are serious arable weeds, possibly because their original natural habitats became cultivated or managed by human activities after the onset of agriculture: species adapted to highly disturbed arable fields are indeed offered vast surfaces as potential habitat (Dekker 2004). Weedy *Setaria* include *S. viridis* (L.) P. Beauv. (green foxtail), *S. verticillata* (L.) P. Beauv. (bristly foxtail), *S. faberi* F. Hermann (giant foxtail), *S. pumila* (Poiret) Roemer & Schultes (syn. *S. glauca*) (yellow foxtail), and *S. parviflora* (Poir.) Kerguélen S. (syn. *S. geniculata*) (knotroot foxtail). As weeds, these species have been subjected to repeated selection by herbicides over broad areas and consequently have evolved herbicide resistance. Understanding the evolution of herbicide resistance is of scientific relevance because of the impact of this resistance in agriculture and because research addressing herbicide resistance mechanisms and evolution allows considerable insight into plant physiology and response to selection. In the first part, we review herbicide resistance cases reported in *Setaria* species, and their underlying genetics, mechanisms, and biological consequences. We analyze in particular the fitness cost, estimated by comparing resistant and susceptible material sharing a common origin (see Vila-Aiub et al. 2009 for a review). In the second part, we summarize the efforts implemented to transfer genes responsible for herbicide resistance to cultivated varieties of *Setaria* in order to facilitate weed control in those crops. Indeed, developing herbicide-resistant cultivars in "minor" crops could be a way to maintain the global diversity of cropping systems. The alternative would be laying aside these crops because they are often not considered profitable enough by agrochemical companies to foster the development of selective herbicides, and the cost and time required for mechanical or hand weeding render them unattractive to growers. As far as we know, introgressing herbicide resistance is one of the rare cases of using wild *Setaria* in foxtail millet breeding programs.

## 15.2 Herbicide Resistance in Weedy *Setaria*

The high variability described for the *Setaria* genus prompted early researchers in the field of herbicide-based weed control to investigate natural variation in sensitivity to herbicides. Variation in efficacy of the herbicide dalapon, a lipid synthesis inhibitor (Herbicide Resistance Action Committee (HRAC) group N), was observed

among various accessions of *S. pumila* and *S. faberi*, which could explain the reported variable efficacy in controlling these species in the fields (Santelmann and Meade 1961). Although they observed a lack of control for some populations that eventually produced seeds, the authors did not use the term "resistance" but referred to "variation of dalapon susceptibility." Similarly, Oliver and Schreiber (1971) observed a differential efficacy of the two photosynthesis inhibitors herbicides atrazine and propazine (HRAC group C1) among *S. viridis* forms, including various forms corresponding to spp. *pycnocoma* (Steudel) Tzvelev. A relationship between variation in the capacity to metabolize herbicides and variation in sensitivity observed among the different *S. viridis* forms was subsequently established (Thompson 1972). Small (two- or three-fold) intraspecific differences in sensitivity to atrazine among populations were subsequently confirmed in *S. viridis*, *S. adhaerens*, *S. verticillata*, and *S. pumila* (De Prado et al. 1990; Wang and Dekker 1995). Similar resistance ratios were also observed for metolachlore, a cell division inhibitor (HRAC group K3) in *S. viridis* and *S. pumila* (Wang and Dekker 1995). Intraspecific variation in herbicide detoxification could be at the root of these differences. However, all these cases of variation in sensitivity to herbicides were not related to a documented specific and repeated herbicide use of the herbicide and did not lead to weed control failure. As such, they did not fall within the definition of herbicide resistance in weeds (Heap 2015) and were considered representing the standing variation in sensitivity of the *Setaria* species. The four documented cases of resistance in *Setaria* are reported below in chronological order (Table 15.1). Rapid biological tests at the seed germination and seedling stage, as well as molecular tools, were set up to identify the resistant mutants (Fig. 15.1). Typical evolution of herbicide resistance had also been observed in response to a long and repeated use of acetochlor, a cell division inhibitor (HRAC group K3), but this case was not further investigated (Baeva 2007).

**Table 15.1** Summary of the characteristics of the four documented herbicide-resistance cases in *Setaria viridis* and date of the first field record

| Herbicide | Date | HRAC group | S/R plant | S/R target site | Field rate | Inheritance | Gene | Codon substitution | Fitness |
|---|---|---|---|---|---|---|---|---|---|
| Atrazine | 1980 | C1 | >50 | 1000 | 5× | Maternal | psbA | Ser264-Gly | −22 % |
| Trifluralin | 1987 | K1 | 7 | ND | 0.6× | Nuclear, recessive | α2-tubulin | Leu136-Phe | −20 % |
| | | | | | | | | Thr239-Ile | |
| Sethoxydim | 1990 | A | 2980 | 700 | >2× | Nuclear, dominant | ACCase | Ile1781-Leu | = |
| Imazethapyr | 2001 | B | 182 | 260 | >2× | Nuclear, ND | ALS | Ser653-Thr/Asn/Ile | ND |
| | | | | | | | | Gly654-Asp | |

The R/S resistance factors (R/S ratio of the herbicide doses which cause 50 % mortality of a plant population or 50 % inhibition of growth or other vital physiological function) show the values expressed at the whole-plant level and at the target site (chloroplast or enzyme activity) recorded for the most resistant accession
*ND* not determined

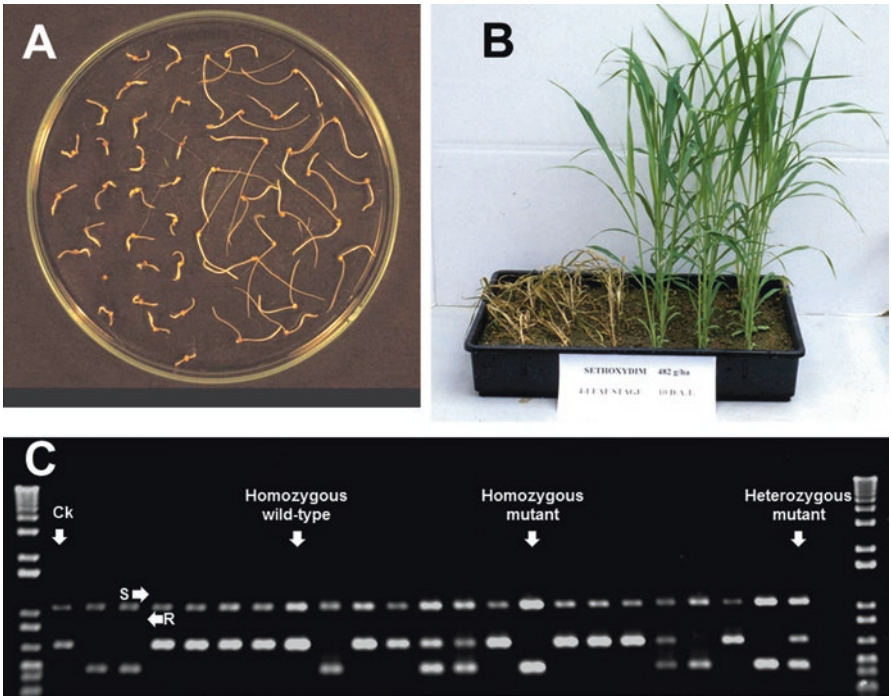


**Fig. 15.1** Examples of identification of herbicide-resistant plants using different types of assays. (**a**) Petri dish assay showing sensitive (*left*) and resistant (*right*) seedlings growing on a medium containing a tubulin polymerization inhibitor (HRAC group K1). Growth of sensitive seedlings is reduced with distorted shoot and roots, while growth of resistant seedling is unaffected. (**b**) Whole-plant spraying assay showing sensitive (*left*, killed) and resistant (*right*, unaffected) seedlings 10 days after application of a commercial formulation of an acetyl-CoA carboxylase inhibitor (HRAC group A) at the 3–4 leaf stage. (**c**) Genotyping assay showing the detection of Leu-1781 ACCase mutant alleles using allele-specific PCR as described in Délye et al. (2002) (Ck, internal positive control (1087 bp); S, amplicon specific for ACCase alleles carrying a wild-type 1781 codon (Ile-1781, 677 bp); R, amplicon specific for ACCase alleles carrying an herbicide-resistant, mutant 1781 codon (Leu-1781, 448 bp) [(**c**) reproduced with permission of Springer]

### *15.2.1 Resistance to Photosystem II Inhibitors*

The triazine herbicide family (HRAC group C1) inhibits electron transfer at the photosystem II (PSII) level. Triazines were massively and repeatedly used to control weeds in maize monoculture during the seventies. Although *Setaria* species display some detoxification capacity against triazines (Gimenez-Espinosa et al. 1996), triazine applications generally resulted in a nearly total control of these weeds. The first case of resistance to triazines in *Setaria* was observed in a maize monoculture in France, where plants surviving three-fold the atrazine field rate evolved in a *S. viridis* population that had been sprayed with this herbicide for 7 years (Gasquez and Compoint 1981). Similar resistance evolution was subsequently observed in

Spain (De Prado et al. 2000) and Yugoslavia (Konstantinovic 2001) in *S. viridis* and in France in *S. viridis* spp. *pycnocoma* (Darmency and Pernès 1985). Other *Setaria* species were also involved: *S. pumila* in eastern Canada and Spain (Stepenson et al. 1990; De Prado et al. 1989), and *S. faberi* in the USA and Spain (Ritter et al. 1989; De Prado et al. 2000). All these confirmed resistance cases evolved in fields grown with maize and with a long history of atrazine applications. Resistant *S. viridis* have also been found in French vineyards continuously treated with triazine herbicides (Darmency, unpublished). Resistant plants withstood up to ten times the herbicide field dose, a rate at which the maize crop is killed. Further investigation showed that photosynthetic electron transport was unaffected by the herbicide in the resistant plants, with a resistance factor (i.e., R/S ratio of the herbicide doses which cause 50 % mortality of a plant population or 50 % inhibition of growth or other vital physiological function) ranging from 300 to 1000 as evaluated at the chloroplast activity level. A rapid and simple fluorescence test allowed easy resistance diagnosis (Gasquez and Compoint 1981; Ritter et al. 1989; De Prado et al. 1989, 2000).

Today, resistance to triazines has been reported in 72 weed species (Heap 2015). Resistant plants showed target site-based resistance resulting from a mutation at the herbicide-binding site, a chloroplast 32 kDa polypeptide called D1 and encoded by the chloroplastic gene psbA (see Tian and Darmency 2006 for a review). In most cases, this modification was a Ser-to-Gly substitution at amino-acid residue 264 protein D1. This results in an altered conformation of the herbicide-binding site on protein D1 causing a drastic reduction in herbicide binding. The Gly264 psbA allele was identified in the French accessions of *S. viridis* exhibiting resistance to triazines (Tian and Darmency 2006; Jia et al. 2007). Being chloroplast encoded, triazine resistance is expected to be maternally inherited. This was confirmed by interspecific crosses between the sexually compatible and closely related species *S. viridis* and foxtail millet (*S. italica*): only hybrid progeny derived from *S. viridis* resistant mother-plants inherited resistance, and resistance did not segregate in the $F_2$ generation (Darmency and Pernès 1985). However, maternal inheritance was not absolute. Analysis of >750,000 hybrid plants produced using a male sterile foxtail millet variety crossed with triazine-resistant *S. viridis* and confirmed as hybrids by reciprocal markers revealed pollen-mediated transfer of the chloroplast resistance gene: the sensitive female parent produced 0.03 % resistant progeny.

The herbicide-resistant Gly264 psbA allele entails physiological consequences: a less efficient electron transport through the PSII and a series of functional and anatomical alterations of the chloroplast, which cause a strong fitness cost (Arntz et al. 2000). Fitness cost was not directly investigated in weedy *Setaria*. Analysis of a series of backcrossed progeny derived from a cross between triazine-resistant *S. viridis* carrying Gly264 psbA and foxtail millet showed that resistant $BC_2$ progeny had a lower rate of photosynthesis ($CO_2$ fixation) than their sensitive counterparts (i.e., the reciprocal $BC_2$) at 27 °C, a normal growth temperature for this summer growing plant. No difference was observed at lower temperatures (Ricroch et al. 1987). This could be due to a chlorophyll a/chlorophyll b ratio that was 10 % lower in the resistant *S. viridis* plants than in the sensitive plants

(Darmency et al. 1992). In field experiments, seed production of the resistant *S. viridis* plants was 22 % lower than that of the sensitive counterparts (Darmency and Pernès 1989). In greenhouse experiments, high plant density conditions further decreased the relative productivity of the resistant *S. viridis* plants up to 65 % (Reboud and Till-Bottraud 1991).

### *15.2.2 Resistance to Tubulin Polymerization Inhibitors*

The dinitroaniline herbicide family (HRAC group K1) inhibits cell division. At the end of the eighties, resistance to dinitroaniline herbicides evolved in several populations of *S. viridis* in the Canadian prairies that received at least 3–5 applications of these herbicides in 10 years (Morrison et al. 1989). *S. viridis* was the only *Setaria* species reported to have evolved a resistance to dinitroaniline leading to practical control failure. A rapid Petri dish bioassay allowed clear discrimination of resistant and sensitive young *S. viridis* seedlings on the basis of inhibition of radicle growth (Beckie et al. 1990). Segregation studies identified a 3:1/sensitive:resistant ratio in the $F_2$ generation derived from crosses between dinitroaniline-resistant and sensitive *S. viridis* plants, showing that the resistance was under the control of a recessive nuclear locus (Jasieniuk et al. 1994). Resistance factors were moderate (c.a. seven-fold), but the resistant plants were cross-resistant to all dinitroaniline herbicides (Beckie and Morrison 1993a, b) as well as to other unrelated tubulin-destabilizing drugs. This suggested that the resistant plants may contain an altered protein that stabilizes microtubule formation (Smeda et al. 1992). Tubulins are dimeric proteins consisting into one α and one β subunit that polymerize into microtubules (Breviario et al. 2013). Four tubulin genes (two α and two β) were identified in *S. viridis* (Délye et al. 2004). A Leu136-Phe or a Thr239-Ile substitution in the gene encoding the α2-tubulin was found responsible for resistance (Délye et al. 2004). Occurrence of two mutant α2-tubulin alleles was necessary to confer resistance, confirming the recessive status of this resistance (Jasieniuk et al. 1994). Tridimensional modelling shed light on the stereochemical organization of the α2-tubulin region involved in herbicide binding and tubulin polymerization (Délye et al. 2004). Allele-specific polymerase chain reaction (PCR) assays were set up to allow quick discrimination of the different α2-tubulin alleles (Délye et al. 2004, 2005).

Resistance to dinitroanilines was the first demonstrated case of recessive control of a resistance to herbicides in weeds. Recessive control is not favorable to the establishment of a resistance in the field. However, *S. viridis* is highly self-fertilized and produces huge number of seeds, a feature facilitating the emergence of homozygous resistant plants. The frequency of dinitroaniline-resistant plants did not vary after 7 years with no dinitroaniline application, suggesting there was no substantial fitness penalty associated to this resistance (Andrews and Morrison 1997). However, the persistence of resistance could also be due to the long-term persistence of the herbicide in the soil and/or to a large resistant soil seed bank established during the

years *S. viridis* was not controlled. Indeed, comparison of dinitroaniline-resistant (Ile239 allele) and sensitive nearly isogenic *Setaria* material identified a reduction in seed production of about 20 % in the resistant lines (Darmency et al. 2011). This fitness cost was similar to that found for resistance to triazines and was confirmed by field experiments, where Ile239-dinitroaniline- and triazine-resistant lines tested together showed similar seed production (Wang et al. 2010a).

### *15.2.3 Resistance to Acetyl-CoA Carboxylase (ACCase) Inhibitors*

The herbicides inhibiting ACCase (HRAC group A) specifically disrupt fatty acid biosynthesis in the Gramineae. Resistance to ACCase inhibitors evolved in the early nineties in several populations of *S. viridis* (Heap and Morrison 1996) and *S. faberi* (Stoltenberg and Wiederholt 1995) following the repeated (around seven times) use of these herbicides during one decade. Resistance increased rapidly in *S. viridis* in Canada. It was detected in 6 % of the fields sampled in a large survey in 2001–2003, and in 27 % of those sampled in 2007–2011 (Beckie et al. 2013). It was later found in Spain (De Prado et al. 2004). The resistant *Setaria* plants showed cross-resistance to the majority of ACCase inhibitors with various resistance factors, with particularly high resistance levels to the herbicide sethoxydim (resistance factors ranging from 20 to 2900: Heap and Morrison 1996). The ACCase enzyme extracted from resistant *S. viridis* or *S. faberi* plants was much less sensitive to ACCase inhibitors than that from sensitive plants (Marles et al. 1993; Shukla et al. 1997; Volenberg and Stoltenberg 2002a; De Prado et al. 2004). Subsequent segregation studies using hybrid progenies obtained by crossing *S. viridis* and foxtail millet showed that resistance to the ACCase inhibitor sethoxydim was due to a single, completely dominant, nuclear locus (Wang and Darmency 1997b). Similarly, a single, nuclear, co-dominant locus controlled the response to the ACCase inhibitor fluazifop in *S. faberi* (Volenberg and Stoltenberg 2002b).

A point mutation causing an Ile1781-Leu substitution in the carboxyltransferase domain of the nuclear gene encoding the plastidic ACCase isoform was demonstrated to be a cause for resistance to the ACCase inhibitors sethoxydim resistance in *S. viridis* (Délye et al. 2002). Other mutations that confer different patterns of cross-resistance have subsequently been identified in other grass weed species (Délye 2005; Beckie and Tardif 2012). Observation of different patterns of cross-resistance among *S. viridis* populations (Heap and Morrison 1996; Beckie et al. 1999) most likely denotes the presence of mutant, herbicide-resistant ACCase alleles different from the Leu1781 allele or from other resistance mechanisms in this species. Complete proof that the Leu1781 allele does encode for resistance was provided by the transfer of the mutated gene sequence to maize through genetic engineering resulting in herbicide-resistance expression (Dong et al. 2011).

Using nearly isogenic plant material derived from interspecific crosses between *S. viridis* and foxtail millet, more vigorous juvenile growth in the field,

earlier flowering, a higher number of tillers and grains were recorded on the resistant plants carrying Leu1781 ACCase allele than on their sensitive counterparts (Wang et al. 2010b). The differences were exacerbated when both genotypes were grown in mixture. The seeds of the Leu1781 ACCase plants were lighter than those of the sensitive plants although more abundant (Wang et al. 2010b). Fitness of both genotypes over the whole life cycle was not different in a 3-year experiment when plots of mixed populations were left unmanaged, but an excess of Leu1781 ACCase plants was found in plots where low doses of trifluralin herbicide (HRAC group K1, not related to the mode of action of ACCase inhibitors and ACCase resistance) created stressing conditions (Wang et al. 2010b). Therefore, fitness neutrality or benefit of the Leu1781 ACCase allele (or of a closely linked gene) exists and was triggered by the habitat conditions (Wang et al. 2010b). This predicts a long and successful persistence of plants carrying Leu1781 ACCase in the field.

### *15.2.4 Resistance to Acetolactate-Synthase (ALS) Inhibitors*

The herbicides inhibiting ALS (HRAC group B) disrupt the biosynthesis of branched-chain amino acids. Resistance to these herbicides evolved in several populations of *S. viridis*, *S. pumila*, and *S. faberi* in Canada and the USA at the end of the nineties after one or two yearly applications of these herbicides during 4–9 years (Volenberg et al. 2001, 2002; Heap 2015). Dose-response experiments on whole *S. faberi* plants showed resistance factors of about 10–20 that varied according to population and the herbicide (Volenberg et al. 2001). In vitro ALS enzyme assay showed that the enzyme of resistant *S. faberi* plants was resistant to herbicides. Resistance segregation in *S. faberi* showed a 1:2:1 sensitive:intermediate:resistant segregation in $F_2$, thus indicating that resistance was controlled by a nuclear semidominant locus (Volenberg et al. 2001). Since *S. faberi* is an allotetraploid (Benabdelmouna et al. 2001), it is likely that only one locus is involved here with simple disomic inheritance. Similar results were obtained for *S. viridis*, with resistance factors varying with the herbicide tested (Volenberg et al. 2002).

In other *S. viridis* populations, resistance was demonstrated to be due to mutant alleles encoding ALS enzymes carrying amino-acid substitutions that modified the herbicide-binding site (Laplante et al. 2009). Different substitutions were identified: Ser653-Thr, Ser653-Asn, Ser653-Ile and Gly654-Asp. They conferred different cross-resistance patterns to ALS inhibitors (Laplante et al. 2009). Recently, the first case of *S. viridis* with nontarget-site resistance has been identified for a population in maize in France (Délye, unpublished).

No report has been published to date for *Setaria* species on the possible consequences on plant fitness of one mutant ALS allele. A few indications may be inferred from "herbicide-tolerant" crop cultivars carrying natural or induced similar mutations: an Asn653 ALS allele is present in maize, rice, oilseed rape and wheat culti-

vars of the brand Clearfield® and no any deleterious effect has been recorded on yield; similarly, there was no effect of this mutation on seed production in *Arabidopsis* (see Darmency 2013 for review).

## 15.3 Herbicide-Resistant Crop Varieties of Foxtail Millet (*S. italica*)

Breeding for herbicide resistance (sometimes referred to as "tolerance") is a very recent trend in plant breeding. Demonstrating the single-gene control of resistance to triazines, i.e., a single gene controlling a drastic change in phenotype, was certainly a major incentive to this approach: breeders realized that a single gene could make herbicides selective for hitherto sensitive crop varieties, as illustrated for triazine-resistant oilseed rape (Beversdorf et al. 1980). This approach is particularly attractive considering the current lack of new herbicides released by the industry (Duke 2012). In addition, few herbicides selective for foxtail millet are marketed because this crop does not represent a market profitable enough to justify the expenses pertaining to herbicide development and commercial release. Accordingly, weed control remains one of the major issues when growing foxtail millet (Shanxi Academy of Agricultural Sciences 1987; Zhou et al. 2013). Herbicide-resistant foxtail millet cultivars were thus bred to overcome this situation. Since no herbicide-resistant germplasm was available for foxtail millet (Shanxi Academy of Agricultural Sciences 1987), herbicide-resistant genotypes of the sexually compatible and closely related weed *S. viridis* described in the preceding sections were used as a source for the resistance trait. At the same time, breeding herbicide-resistant foxtail millet aroused considerable concern about the potential dispersal of the genes endowing herbicide resistance back to weedy *Setaria* populations.

### *15.3.1 Introgression of Herbicide Resistance Genes*

Triazine-resistant foxtail millet germplasm was first generated from an interspecific cross with *S. viridis* with the weed as the female in order to retain the chloroplast encoded Gly264 psbA allele conferring triazine resistance (Darmency and Pernès 1985). Two backcross generations combined with morphological selection were enough to eliminate weedy traits and generate resistant foxtail millet germplasm (Naciri et al. 1992). High-yield foxtail millet lines were ultimately derived from these crosses (Ji et al. 2006; Shi et al. 2008) despite the fitness cost associated to Gly264 psbA.

Dinitroaniline-resistant germplasm was then generated. The recessive nature of resistance associated with the causal Ile239 α2-tubulin allele complicated the breeding scheme. In addition, segregation distortion against homozygous mutant hybrid

progeny was observed with on average 15 % homozygous resistant progeny plants instead of the expected 25 % (Wang et al. 1996b). The segregation distortion was attributed to linkage of the Ile239 α2-tubulin allele with a modifier gene whose expression was only observed in the interspecific hybrids (Tian et al. 2006). It was inferred from alignment of the rice and millet genetic maps that the α2-tubulin gene belongs on linkage group IX, a chromosome that showed distorted segregation in an independent RFLP study (Wang et al. 1998). The linkage of the resistance gene with the putative gametophyte gene resulting in 69 % gamete viability could be broken in advanced backcross progeny (Tian et al. 2006). Resistant foxtail millet lines were further selected and a resistance pattern was observed that was similar to that observed for the original *S. viridis* genotype (Wang and Darmency 1997a). As the resistance factor obtained in the foxtail millet germplasm was not high enough to enable fully satisfactory weed control in the field, the Ile239 α2-tubulin foxtail millet germplasm was not used for commercial release, but it was helpful to facilitate hybrid seed production as described below (Wang et al. 1996a).

Foxtail millet germplasm obtained by introgressing Leu1781 ACCase from *S. viridis* was used in a breeding program intended to release a series of commercial, herbicide-resistant cultivars. The resistance pattern observed in the *S. viridis* parent was transferred to the derived foxtail millet lines (Wang and Darmency 1998). Before the point mutation was elucidated, AFLP markers were developed to identify the mutation throughout the breeding scheme (Niu et al. 2002). The higher seed production associated to the Leu1781 ACCase allele is a desired herbicide-resistant resource for breeding, and was combined with a restorer for seed size in further crosses (Wang et al. 2000, 2010c).

Based on this research, Wang's team at the Chinese Academy of Agricultural Science established a nation-wide cooperation network in China for the breeding of herbicide-resistant varieties in foxtail millet. A cooperative group distributed new and improved herbicide-resistant materials and breeding techniques to local breeders to help further advance adoption of varieties. Millet breeders in different ecological areas used existing commercial varieties that exhibited strong performance as the recurrent parent to improve herbicide-resistant materials for high yield and high herbicide resistance, and to generate lines that showed an aggregation of desirable traits. These methodologies effectively accelerated the breeding process of new varieties and hybrids, and their application to the field. There are now presently 30 novel herbicide-resistant millet varieties/hybrid varieties registered at the national or local level in China. These new varieties are now being widely used in all three major millet-producing regions of China. Resistant varieties employed in the Mid-northern region of China include SR3522, Jigu 24, Jigu 25, Jigu 29, Changgu 2, and zhangzagu 3, those employed in the West-northern region include zhangzagu 3, zhangzagu 5, zhangzagu 6, zhangzagu 9, Bagu 214, and Longgu 11, and the East-northern region relies heavily on zhangzagu 3 zhangzagu 5, Chizagu 1, and Jigu 24. Since this is a new technology, it is necessary to use a new way of promotion. Zhangjiakou Academy of Agricultural Sciences and allied teams established a novel system that connects millet breeding, planting, processing and marketing, which has achieved considerable positive social and economic influence (Li et al. 2014; Song et al.

2014). To date, all new herbicide-resistant millet varieties have demonstrated characteristics such as efficient herbicide resistance, high and stable yield and strong adaptability. Since 2001, the newly-bred varieties/hybrids have been grown on more than 0.8 million hectares (customarily, foxtail millet acreage in China is 1–1.5 million hectares each year). Some varieties were also trialled in a large area in Ethiopia and other Africa countries (Liu and Zhao 2012; Hao et al. 2013).

During the process, different types of herbicide resistance have been developed to secure the selection and production of hybrid varieties expressing heterosis and potentially having higher yield (Wang et al. 2000). The resistance traits to triazine (cytoplasmic inheritance) and dinitroaniline herbicides (nuclear recessive) were transferred to male sterility lines. Using these triazine-resistant male sterility lines and corresponding herbicides it is possible to simplify seed production procedures and improve seed production yield. In addition, the use of dinitroaniline-resistant male sterility lines would help to maintain line purity and to reduce outcrossing so that there would be less need to isolate the reproduction field. The resistance traits to herbicides inhibiting ACCase (nuclear dominant) combined with a restorer provides a mechanism to efficiently eliminate false positive hybrids (mixed mother parent seedling and weeds) (Wang et al. 2000, 2010c; Tian et al. 2010). The successful breeding of various herbicide-resistant millet varieties has been instrumental in pursuing these objectives.

### *15.3.2 Gene Flow*

Although *Setaria* species are primarily self-pollinated, pollen can move several dozen meters from the source plants and fertilize male-sterile as well as male-fertile plants (Wang et al. 1998, 2001). *S. viridis* ssp. *pycnocoma* is considered to be the result of ancient hybridization between *S. viridis* and foxtail millet (Darmency 2004). These species constitute a dynamic and evolving "weed-crop complex" (Darmency 2004), and spontaneous crosses could have occurred reciprocally (Till-Bottraud et al. 1992). Thus, when *S. viridis* grows close to foxtail millet, gene flow is unavoidable. Hybridization of other weedy *Setaria* species with foxtail millet is far less likely (Darmency and Dekker 2011). Average outcrossing rate for *S. viridis* plants planted 0.25 m apart was 0.74 % (Till-Bottraud et al. 1992), and 0.48 % for *S. faberi* plants planted 0.36 m apart (Volenberg and Stoltenberg 2002b). Around 0.2 % interspecific hybrids were produced by *S. viridis* plants because of pollination by foxtail millet in field (row) conditions (Darmency et al. 1987; Till-Bottraud et al. 1992), and up to 3 % hybrids were recorded when plants of the two species were grown in close mixture (De Wet et al. 1979). Under commercial field conditions, the rate of hybrid produced by *S. viridis* pollinated by foxtail millet was lower, ranging from 0.039 % within the foxtail millet field to 0.002 % 20 m from the field (Shi et al. 2008). After 6 years of testing, results show that the gene flow from *cv.* to wild population effectively occurs in production condition but is manageable when the herbicide selection stops so that it can be controlled through crop and herbicide rotation.

*In Natura*, hybrids of *S. viridis* and foxtail millet are expected to suffer a fitness penalty due to a mix of antagonistic wild and domesticated characters (i.e., flowering synchrony, seed shedding, seed size, seed dormancy). Although no direct estimate of the relative fitness cost of hybridization has ever been carried out, we indirectly calculated from our own experiments that $F_1$ hybrids of *S. viridis* and foxtail millet produces 15–30 times less viable seeds than a *S. viridis* plant (Darmency, unpublished). However, in the subsequent generations, seed fertility is rapidly restored by back-cross with *S. viridis*, although the number of tillers remains low compared to *S. viridis*. In field experiments with a foxtail millet cultivar carrying the dominant Leu1781-ACCasa allele, only a slow increase in herbicide-resistant progeny ($F_1$ and hybrid descendants of *S. viridis*) was observed during 4 years of herbicide-free cultivation of the resistant cultivar (0.1 % after 4 years) (Shi et al. 2008). This proportion decreased rapidly in the absence of the resistant foxtail millet cultivar to reach 0.01 % within 2 years (Shi et al. 2008). Using herbicides to which resistance has been introgressed in foxtail millet is obviously expected to facilitate the selection of the resistant progeny of the hybrids, which would jeopardize the herbicide-resistant cultivar strategy. For this strategy to be efficient, it is clearly necessary to closely monitor the increase in frequency of the resistance genes in weedy *Setaria* populations and, if possible, to alternate growing foxtail millet cultivars with resistance to different herbicide modes of action in a given field.

## 15.4 Perspectives

Although weedy *Setaria* are widespread weeds, few *Setaria* populations evolved herbicide-resistance in comparison to other grass weed genera (e.g., *Lolium, Alopecurus, Echinochloa, Poa*: Heap 2015). Only four herbicide modes of action are affected by resistance in *Setaria* species. Non-target-site-resistance that is a major cause for resistance in other grasses (Beckie and Tardif 2012; Délye et al. 2013) has not been identified to date in *Setaria*, although some studies identified the potential for this non-target-site based resistance to evolve in *Setaria* (Santelmann and Meade 1961; Oliver and Schreiber 1971; Thompson 1972; De Prado et al. 1990; Wang and Dekker 1995). This situation may be due to non-target-site based resistance having been overlooked by researchers that were more focussed on target-site-based resistance. Non-target-site based resistance is largely considered to evolve by accumulation of genes with additive effects in a same plant via sexual reproduction (Délye et al. 2013). The strong autogamy of the *Setaria* species may thus also be a reason for the absence of report of non-target-site based resistance in these species. However, multiple resistance to ACCase and microtubule inhibitors was detected in some locations in Canada, which may confirm the potential for further evolution in response to environmental conditions (Beckie et al. 1999). In contrast to *S. viridis* and *S. faberi* for which resistance cases have been reported, there is an absence of reported resistance cases in *S. adhaerens* and *S. verticillata,* which may be due to moderate herbicide use in the distribution areas of these species, i.e., warmer and more tropical

zones. Genome-based differences also could contribute to this difference because *S. viridis* (diploid genome A) and *S. faberi* (tetraploid genomes A and B) share in common the A genome while *S. adhaerens* has the B genome. However, both *S. faberi* and *S. verticillata* are allotetraploid and carry genomes A and B, thus casting some doubt on a genome-mediated effect. Introgression of the resistance genes into foxtail millet has proven to be an efficient strategy to generate herbicide-resistant cultivars. However, such cultivars must be used with care because of the high risk for the transfer by gene flow of the herbicide-resistant allele back to weedy *Setaria* species. We have already paid attention to this topic, especially in view of releasing future genetically engineered lines into production. Fortunately, we have not found the problem in the fields of foxtail millet, as well as in other crops up to now. Perhaps this is due to the fact that above-mentioned herbicides, especially ACCase inhibitors, are not utilized in field production so much. In addition, it could be due also to field scouting and hand weed control of remaining weeds since Chinese farmers usually deal with small-scale fields by hand. In any case, a more in-depth understanding of the genetic relationships between *S. viridis* and foxtail millet may be necessary to be able to correctly assess the risk for interspecific herbicide resistance gene flow at both field and landscape levels and to devise more appropriate recommendations for the use of herbicide-resistant foxtail millet cultivars.

## References

Andrews TS, Morrison IN. The persistence of trifluralin resistance in green foxtail (Setaria viridis) populations. Weed Technol. 1997;11:369–72.

Arntz AM, De Lucia EH, Jordan N. From fluorescence to fitness: variation in photosynthetic rate affects fecundity and survivorship. Ecology. 2000;81:2567–76.

Baeva G. Resistance study of Setaria glauca PB to the herbicide acetochlor. In: Proceedings of the 14th EWRS symposium, Hamar, Norway; 17–21 June 2007. p. 146.

Beckie HJ, Morrison IN. Effect of ethalfluralin and other herbicides on trifluralin-resistant green foxtail (Setaria viridis). Weed Technol. 1993a;7:6–14.

Beckie HJ, Morrison IN. Effective kill of trifluralin-susceptible and -resistant green foxtail (Setaria viridis). Weed Technol. 1993b;7:15–22.

Beckie HJ, Tardif FJ. Herbicide cross resistance in weeds. Crop Prot. 2012;35:15–28.

Beckie HJ, Friesen LF, Nawolsky KM, Morrison IN. A rapid bioassay to detect trifluralin-resistant green foxtail (Setaria viridis). Weed Technol. 1990;4:505–8.

Beckie HJ, Thomas AG, Légère A. Nature, occurrence, and cost of herbicide-resistant green foxtail (Setaria viridis) across Saskatchewan ecoregions. Weed Technol. 1999;13:626–31.

Beckie HJ, Lozinski C, Shirriff S, Brenzil CA. Herbicide-resistant weeds in the Canadian Prairies: 2007 to 2011. Weed Technol. 2013;27:171–83.

Benabdelmouna A, Shi Y, Abirached-Darmency M, Darmency H. Genomic in situ hybridization (GISH) discriminates between the A and B genomes in diploid and tetraploid Setaria species. Genome. 2001;44:685–90.

Beversdorf WD, Weiss-Lerman J, Reickson LR, Souza-Machado V. Transfer of cytoplasmically-inherited triazine resistance from bird's rape to cultivated rapeseed (Brassica campestris L. and B. napus L.). Can J Genet Cytol. 1980;22:167–72.

Breviario D, Giani S, Morello L. Multiple tubulins: evolutionary aspects and biological implications. Plant J. 2013;75:202–18.

Darmency H. Incestuous relations of foxtail millet (Setaria italica) with its parents and cousins. In: Gressel J, editor. Crop ferality and volunteerism: a threat to food security in the transgenic era. Boca Raton: CRC Press; 2004. p. 81–96.
Darmency H. Pleiotropic effects of herbicide-resistance genes on crop yield: a review. Pest Manag Sci. 2013;69:897–904.
Darmency H, Dekker J. Setaria. In: Kole C, editor. Wild crop relatives: genomic and breeding resources. Millet and grasses. Berlin: Springer; 2011. p. 275–96.
Darmency H, Pernès J. Use of wild Setaria viridis (L) Beauv to improve triazine resistance in cultivated S. italica (L) by hybridization. Weed Res. 1985;25:175–9.
Darmency H, Pernès J. Agronomic performance of a triazine resistant foxtail millet (Setaria italica (L) Beauv). Weed Res. 1989;29:147–50.
Darmency H, Zangre GR, Pernès J. The wild-weed-crop complex in Setaria: a hybridization study. Genetica. 1987;75:103–7.
Darmency H, Chauvel B, Gasquez J, Matejicek A. Variation of chlorophyll a/b ratio in relation to population polymorphism and mutation of triazine-resistance. Plant Physiol Biochem. 1992;30: 57–63.
Darmency H, Picard JC, Wang T. Fitness costs linked to dinitroaniline resistance mutation in Setaria. Heredity. 2011;107:80–6.
De Prado R, Dominguez C, Tena M. Characterization of triazine-resistant biotypes of common lambsquarters (Chenopodium album), hairy fleabane (Conyza bonaeriensis), and yellow foxtail (Setaria glauca) found in Spain. Weed Sci. 1989;37:1–4.
De Prado R, Diaz MA, Tena M. Differential tolerance to atrazine in four Setaria species. In: Proceedings of the EWRS symposium on integrated weed management in cereals, Helsinki, Finland; 4–6 Jun 1990. p. 61–8.
De Prado R, Lopez-Martinez N, Gonzalez-Gutierrez J. Identification of two mechanisms of atrazine resistance in Setaria faberi and Setaria viridis biotypes. Pest Biochem Physiol. 2000;67:114–24.
De Prado R, Osuna MD, Fischer AJ. Resistance to ACCase inhibitor herbicides in a green foxtail (Setaria viridis) biotype in Europe. Weed Sci. 2004;52:506–12.
De Wet JMJ, Oestry-Stidd LL, Cubero JI. Origins and evolution of foxtail millets (Setaria italica). J Agri Tradit Bot Appl. 1979;26:53–64.
Dekker J. The evolutionary biology of the foxtail (Setaria) species-group. In: Inderjit, editor. Weed biology and management. Dordrecht: Kluwer Academic; 2004. p. 65–113.
Délye C. Weed resistance to acetyl coenzyme A carboxylase inhibitors: an update. Weed Sci. 2005; 53:728–46.
Délye C, Wang T, Darmency H. An isoleucine-leucine substitution in chloroplastic acetyl-CoA carboxylase from green foxtail (Setaria viridis L Beauv) is responsible for resistance to the cyclohexanedione herbicide sethoxydim. Planta. 2002;214:421–7.
Délye C, Menchari Y, Michel S, Darmency H. Molecular bases for sensitivity to tubulin-binding herbicides in green foxtail. Plant Physiol. 2004;136:3920–32.
Délye C, Menchari Y, Michel S. A single polymerase chain reaction-based assay for simultaneous detection of two mutation conferring resistance to tubulin-binding herbicides in Setaria viridis. Weed Res. 2005;45:228–35.
Délye C, Jasienuk M, Le Corre V. Deciphering the evolution of herbicide resistance in weeds. Trends Genet. 2013;29:649–58.
Dong Z, Zhao H, He J, Huai J, Lin H, Zheng J, et al. Overexpression of a foxtail millet acetyl-CoA carboxylase gene in maize increases sethoxydim resistance and oil content. Afr J Biotechnol. 2011;10:3986–95.
Duke SO. Why have no new herbicide modes of action appeared in recent years? Pest Manag Sci. 2012;68:505–12.
Gasquez J, Compoint JC. Observation de chloroplastes résistants aux triazines chez une panicoidée, Setaria viridis L. Agronomie. 1981;1:923–6.
Gimenez-Espinosa R, Romera E, Tena M, De Prado R. Fate of atrazine in treated and pristine accessions of three Setaria species. Pest Biochem Physiol. 1996;56:196–207.
Hao HB, Cui HY, Li MZ. Germplasm innovation of herbicide resistant and colored summer millet. J Hebei Agric Sci. 2013;17:1–2.

Heap I. The international survey of herbicide resistant weeds. http://www.weedscience.org (2015). Accessed 12 Feb 2015.
Heap I, Morrison I. Resistance to aryloxyphenoxypropionate and cyclohexanedione herbicides in green foxtail (Setaria viridis). Weed Sci. 1996;44:25–30.
Jasieniuk M, Brûlé-Babel A, Morrison I. Inheritance of trifluralin resistance in green foxtail (Setaria viridis). Weed Sci. 1994;42:123–7.
Ji G, Du R, Hou S, Ceng R, Wang X, Zhao X. Genetics, development and application of cytoplasmic herbicide resistance in foxtail millet. Sci Agric Sin. 2006;39:879–85.
Jia X, Yuan J, Shi Y, Song Y, Wang G, Wang T, et al. A Ser-Gly substitution in plastid-encoded photosystem II D1 protein is responsible for atrazine resistance in foxtail millet (Setaria italica). Plant Growth Regul. 2007;52:81–9.
Konstantinovic B. Determination of triazine resistant biotypes of Setaria viridis. In: Proceedings of the BCPC conference: weeds, Brighton, UK; 12–15 Nov 2001. p. 607–12.
Laplante J, Rajcan I, Tardif FJ. Multiple allelic forms of acetohydroxyacid synthase are responsible for herbicide resistance in Setaria viridis. Theor Appl Genet. 2009;119:577–85.
Li MZ, Liu GR, Song K, Cui HY, Hao HB, Wang XM. Development status and analysis of hybrid millet seed industry. J Hebei Agric Sci. 2014;18:1–3.
Liu JJ, Zhao ZH. Cultivation technology and extension experiences of hybrid millet in Ethiopia. J Hebei Agric Sci. 2012;16:9–12.
Marles MAS, Devine MD, Hall JC. Herbicide resistance in Setaria viridis conferred by a less sensitive form of acetyl coenzyme A carboxylase. Pest Biochem Physiol. 1993;46:7–14.
Morrison IN, Todd BG, Nawolsky KM. Confirmation of trifluralin-resistant green foxtail (Setaria viridis) in Manitoba. Weed Technol. 1989;3:544–51.
Naciri Y, Darmency H, Belliard J, Dessaint F, Pernès J. Breeding strategy in foxtail millet, Setaria italica (LP Beauv), following interspecific hybridization. Euphytica. 1992;60:97–103.
Niu Y, Li Y, Shi Y, Song Y, Ma Z, Wang T, et al. AFLP mapping for the gene conferring sethoxydim resistance in foxtail millet (Setaria italica L Beauv). Acta Agric Sin. 2002;28:359–62.
Oliver LR, Schreiber MM. Differential selectivity of herbicides on six Setaria taxa. Weed Sci. 1971;19:428–31.
Reboud X, Till-Bottraud I. The cost of herbicide resistance measured by a competition experiment. Theor Appl Genet. 1991;82:690–6.
Ricroch A, Mousseau M, Darmency H, Pernès J. Comparison of triazine-resistant and -susceptible cultivated Setaria italica L (PB): growth and photosynthetic capacity. Plant Physiol Biochem. 1987;25:29–34.
Ritter RL, Kaufman LM, Monaco TJ, Novitzky WP, Moreland DE. Characterization of triazine-resistant giant foxtail (Setaria faberi) and its control in no-tillage corn (Zea mays). Weed Sci. 1989;37:591–5.
Santelmann PW, Meade JA. Variation in morphological characteristics and dalapon susceptibility within the species Setaria lutescens and S. faberii. Weeds. 1961;9:406–10.
Shanxi Academy of Agricultural Sciences. The cultivation of foxtail millet in China. In: Gu S, editor. Beijing: Agriculture Press; 1987.
Shi Y, Wang T, Li Y, Darmency H. Impact of transgene inheritance on the mitigation of gene flow between crops and their wild relatives: the example of foxtail millet. Genetics. 2008;180:969–75.
Shukla A, Leach GE, Devine MD. High-level resistance to sethoxydim conferred by an alteration in the target enzyme, acetyl-CoA carboxylase, in Setaria faberi and Setaria viridis. Plant Physiol Biochem. 1997;35:803–7.
Smeda RJ, Vaughn KC, Morrison IN. A novel pattern of herbicide cross-resistance in a trifluralin-resistant biotype of green foxtail [Setaria viridis (L.) Beauv.]. Pest Biochem Physiol. 1992;42:227–41.
Song XZ, Wang YW, Li HX, Tian G, Li ZH, Liu X. Breeding and the key cultivation techniques in herbicide-resistant millet Jingu 56. J Shanxi Agric Sci. 2014;42:115–8.
Stepenson GR, Dykstra MD, McLaren RD, Hamill AS. Agronomic practices influencing triazine-resistant weed distribution in Ontario. Weed Technol. 1990;4:199–207.
Stoltenberg DE, Wiederholt RJ. Giant foxtail (Setaria faberi) resistance to aryloxyphenoxypropionate and cyclohexanedione herbicides. Weed Sci. 1995;43:527–35.

Thompson Jr L. Metabolism of chloro s-triazine herbicides by Panicum and Setaria. Weed Sci. 1972;20:584–7.
Tian X, Darmency H. Rapid bidirectional allele-specific PCR identification for triazine resistance in higher plants. Pest Manag Sci. 2006;62:531–6.
Tian X, Délye C, Darmency H. Molecular evidence of biased inheritance of trifluralin herbicide resistance in foxtail millet. Plant Breed. 2006;125:254–8.
Tian BH, Wang JG, Li YJ, Zhang LX. Study on selection of suitable herbicide for hybrid millet. J Hebei Agric Sci. 2010;14:46–7.
Till-Bottraud I, Reboud X, Brabant P, Lefranc M, Rherissi B, Vedel F, et al. Outcrossing and hybridization in wild and cultivated foxtail mil-lets: consequences for the release of transgenic crops. Theor Appl Genet. 1992;83:940–6.
Vila-Aiub MM, Neve P, Powles SB. Fitness cost associated with evolved herbicide resistance alleles in plants. New Phytol. 2009;184:751–67.
Volenberg D, Stoltenberg D. Altered acetyl-coenzyme A carboxylase confers resistance to clethodim, fluazifop and sethoxydim in Setaria faberi and Digitaria sanguinalis. Weed Res. 2002a;42:342–50.
Volenberg DS, Stoltenberg DE. Giant foxtail (Setaria faberi) outcrossing and inheritance of resistance to acetyl-coenzyme A carboxylase inhibitors. Weed Sci. 2002b;50:622–7.
Volenberg DS, Stoltenberg DE, Boerboom CM. Biochemical mechanism and inheritance of cross-resistance to acetolactate synthase inhibitors in giant foxtail. Weed Sci. 2001;49:635–41.
Volenberg DS, Stoltenberg DE, Boerboom CM. Green foxtail (Setaria viridis) resistance to acetolactate synthase inhibitors. Phytoprotection. 2002;83:99–109.
Wang T, Darmency H. Dinitroaniline herbicide cross-resistance in resistant Setaria italica lines selected from interspecific cross with S. viridis. Pest Sci. 1997a;49:277–83.
Wang T, Darmency H. Inheritance of sethoxydim resistance in foxtail millet, Setaria italica (L). Beauv. Euphytica. 1997b;94:69–73.
Wang T, Darmency H. Cross-resistance to aryloxyphenoxypropionate and cyclohexanedione in foxtail millet (Setaria italica). Pest Biochem Physiol. 1998;59:81–8.
Wang RL, Dekker J. Weedy adaptation in Setaria spp. II. Variation in herbicide resistance in Setaria spp. Pest Biochem Physiol. 1995;51:99–116.
Wang T, Du R, Chen H, Darmency H, Fleury A. A new way of using herbicide resistant gene on hybrid utilization in foxtail millet. Sci Agric Sin. 1996a;29:96.
Wang T, Fleury A, Ma J, Darmency H. Genetic control of dinitroaniline resistance in foxtail millet (Setaria italica). J Hered. 1996b;87:423–6.
Wang ZM, Devos KM, Liu CJ, Wang RQ, Gale MD. Construction of RFLP-based maps of foxtail millet, Setaria italica (L.) P. Beauv. Theor Appl Genet. 1998;96:31–36.
Wang T, Xin Z, Shi Y, Darmency H. A creation, evaluation and utilization of the new crop germplasm: herbicide resistance foxtail millet (Setaria italica). J China Agric Sci Technol. 2000;2:62–6.
Wang T, Zhao Z, Yan H, Li Y, Song Y, Ma Z, et al. Gene flow from cultivated herbicide-resistant foxtail millet to its wild relatives: a basis for risk assessment of the release of transgenic millet. Acta Agric Sin. 2001;27:681–7.
Wang T, Shi Y, Li Y, Song Y, Darmency H. Population growth rate of Setaria viridis in the absence of herbicide: resulting yield loss in foxtail millet Setaria italica. Weed Res. 2010a;50:228–34.
Wang T, Picard JC, Tian X, Darmency H. A herbicide-resistant ACCase 1781 Setaria mutant shows higher fitness than wild type. Heredity. 2010b;105:394–400.
Wang YW, Li HX, Tian G, Shi QX. Study on innovation and application of highly-male-sterile line with high outcrossing rate in millet. Sci Agric Sin. 2010c;43:680–9.
Zhou HZ, Hou SL, Song YF, Zhao Y, Dong L, Jia HY, et al. Impacts of monocotyledon weeds on millet yield loss in foxtail millet field. Chinese Agric Sci Bull. 2013;29:179–84.