Advances in Crop Science and Technology

Mithila and Godar, Adv Crop Sci Tech 2013, 1:4
http://dx.doi.org/10.4172/2329-8863.1000115

Short Communication Open Access

Understanding Genetics of Herbicide Resistance in Weeds: Implications for Weed Management

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Abstract

Herbicide-resistant weeds pose a serious threat to weed management across the globe. Weeds evolve resistance to herbicides as a result of herbicide selection pressure. Under continuous herbicide selection, the resistant individuals dominate in a population. Understanding the genetics of herbicide resistance will help assess frequency and spread of herbicide resistance allele(s) in a population, which will assist in formulating prudent weed management practices to delay the evolution of resistance. Herbicide resistance, inherited by nuclear genes evolves quicker and spreads rapidly as the trait can be transmitted via pollen as opposed to maternally-inherited resistance. Dominant expression of the resistance allele also accelerates the process. Target-site resistance (TSR) is determined by a single gene, and is more likely a result of strong selection pressure. Although, non-target site resistance (NTSR) usually results under moderate selection pressure accumulating multiple alleles with minor effects imparting resistance, it may also involve a single gene when evolved under high selection pressure. Such monogenic resistances evolve and spread quickly, especially when the resistance is dominant and nuclear-inherited. Herbicide mixtures with different modes of action when applied at recommended doses can effectively delay the evolution of both TSR and NTSR.

Evolution of Herbicide Resistance

Herbicides have made significant contributions to modern agriculture by offering exceptional weed management in crops and also facilitate no-till crop production to conserve soil and moisture. However, repeated field applications of herbicides with the same mechanism of action resulted in selection of herbicide-resistant weeds. A total of 403 confirmed herbicide-resistant weed biotypes have been documented to date (129 dicots and 89 monocots) to 21 of the 25 known herbicide sites of action [1]. Weed Science Society of America (http//www.wssa.net) defines herbicide resistance as the inherited ability of a plant to survive and reproduce following exposure to a dose of herbicide normally lethal to the wild type. In the presence of continued selection pressure (application of herbicides with the same mode of action), the resistant plants increase in frequency over time (generations) [2], thereby transforming a population dominated by individuals resistant to that herbicide.

Several factors govern the rate at which the resistant individuals (alleles) become dominant in the population [3]. Genetic factors, such as the initial frequency of resistant alleles present in the population, the dominance relationships among the alleles and fitness cost of the resistance gene(s) can significantly influence evolution of resistance to herbicides. Other factors, such as biology of weed species (e.g. life cycle, seed production capability, mating system), the herbicide and target site properties (chemical structure, herbicide-target site interactions and residual activity), herbicide dose and application performance can impact dynamics of herbicide resistance evolution, as well. This review focuses on how genetic factors influence the evolution and spread of herbicide resistance in weeds and how herbicide use pattern can determine the genetics of herbicide resistance. Additionally, how this information can be useful in both proactive and reactive management of herbicide-resistant weeds is also discussed.

Monogenic Target Site Resistance and Polygenic Non Target Site Resistance

Mechanisms which confer resistance to herbicides can be categorized into: a) target site resistance (TSR) and non-target site resistance (NTSR). In TSR, mutation(s) in the target site of herbicide results in insensitive or less sensitive herbicide target protein [3], and in essence, involves alteration of the target-site gene (monogenic) [4]. NTSR results from non-target site alterations endowing reduced herbicide uptake/translocation, increased rates of herbicide detoxification, decreased rates of herbicide activation or sequestration of the herbicide [5]. NTSR, especially if involved herbicide detoxification by cytochrome P450 monooxygenases, is usually governed by many genes (polygenic) and may confer resistance to herbicides with other modes of action [4,6]. However, glutathione S-transferase (GST)-mediated NTSR to triazines in velvetleaf is inherited as a single nuclear gene [7].

Herbicide resistance conferred by a single gene with major effect can spread within a population much faster compared to resistance conferred by multiple genes. Polygenic resistance evolves as a result of additive effect of several alleles, each possessing individual minor effect [8]. Accumulation of these alleles in individuals may take several generations of sexual recombination, especially when the initial frequency of the alleles is low. Each individual allele may not necessarily contribute to the same mechanism of resistance [4].

Dominance Relationships among the Alleles

A key aspect in predicting the evolutionary trajectory of herbicide resistance and determining frequency distribution of herbicide resistance allele in a population is to understand dominance relationships among the alleles of the gene in question. Herbicide resistance, expressed by a recessive allele, evolves and spread relatively slowly as the susceptible heterozygotes and homozygous dominant phenotypes are eliminated by application of the herbicide to which resistance developed [9]. This is factual for cross-pollinating species, however, in highly self-pollinating species, the resistant alleles can spread quickly as selfing increases the frequency of homozygotes at the expense of heterozygotes [9,10]. Additionally, prolific seed production

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Received October 3, 2013; **Accepted** October 25, 2013; **Published** October 28, 2013

Citation: Mithila J, Godar AS (2013) Understanding Genetics of Herbicide Resistance in Weeds: Implications for Weed Management. Adv Crop Sci Tech 1: 115. doi:10.4172/2329-8863.1000115

may result in rapid increase of recessive alleles in a population [11]. The cases of recessive gene inheritance of herbicide resistance are rare and limited to few herbicide chemistries. Not surprisingly, these cases have been reported in primarily self-pollinating species (except picloram and clopyralid resistance in yellow starthistle), and the examples include trifluralin resistance in green foxtail [11], quinclorac resistance in false cleaver [12], picloram and clopyralid resistance in yellow starthistle [13], as well as triallate resistance in wild oats [14].

A dominant allele spreads faster in a population than a recessive allele [9]; because a dominant allele is expressed in both homozygous and heterozygous states and the selection continually eliminates the recessive alleles from the population. However, the level of dominance can influence the spread of the resistance allele [15].The level of resistance conferred by the resistant allele relative to herbicide dose determines the response to selection. If the heterozygote individuals fit equally under selection (under the use rate of the herbicide) as homozygous dominant plants, then the response to selection becomes slow and the population remains heterogeneous for longer generations, despite the continued selection pressure. In such conditions, complete dominance or semi-dominance expression of herbicide resistance cause similar response to the selection pressure. In contrast, if the heterozygotes display an intermediate response to the herbicide dose (between the two homozygotes), then the response to selection will be greater, quickly resulting in a homogeneous [for the resistance trait] population, i.e. allele fixation. Most commonly, evolved herbicide resistance is determined by alleles with complete or partial dominance [16]. For example, a single completely dominant allele determines dicamba, 2,4-D and picloram resistance in wild mustard [17,18]. However, resistance to dicamba in kochia is determined by a single allele with a high degree of dominance [19].

Nuclear-vs. Cytoplasm-Inherited Resistance

The target protein of most herbicides is encoded by the nuclear gene and the mutations in those target site genes can inherit via both male and female gametes. In such cases, enrichment of resistant alleles in the population can occur quickly and gene flow (spread) is more rapid as opposed to cytoplasm-inherited resistance. Herbicides that inhibit photosystem I and II (PS I and II) bind to proteins encoded by chloroplast genes. The TSR to triazines (PS II-inhibitors) is inherited only from female parent (maternal inheritance) [20], slowing down the evolutionary process.

Fitness Cost and Herbicide Resistance

Natural mutations resulting in herbicide resistance in populations may be associated with a fitness cost, either due to pleiotropic effects of the resistance gene itself [21], or due to linkage of the resistance gene with one or more other loci that impose the fitness cost. If fitness costs are directly associated with the herbicide resistance mutation then in the absence of selection, sensitive plants would be expected to replace the resistant types [9]. Furthermore, it has been emphasized that in order to understand the effect of a resistance mutation on potential for fitness reductions or fitness costs in plants, near-isogenic lines (NILs) that are herbicide-resistant or -sensitive must be compared [9]. Thus far, fitness studies using NILs of weed species have been performed only with triazine-resistant and -sensitive lines [22,23]. These studies showed that the mutation resulting in triazine resistance is associated with reduced fitness. However, recently, for the first time NILs with auxinic herbicide-resistant and -sensitive were developed in wild mustard [24]. Using these lines, the fitness costs associated with auxinic herbicide resistance were determined. The data demonstrated that resistant lines produced significantly less biomass and seed output compared with sensitive lines [24]. If a resistant population is less fit than the susceptible, and when there is no selection applied, the susceptible plants can outperform the resistant individuals; thereby, a population dominanted by resistant individuals may be switched to a population with more susceptible plants [i.e. reverse selection]. However, rate of reverse selection depends on the degree of fitness penalty associated with the resistance and the frequency of resistant alleles in the population.

Herbicide Resistance and Weed Management

Weed management strategies will continue to rely on herbicides. Despite the inevitable selection for herbicide-resistant weed biotypes, we must constantly investigate possible strategies to delay the evolution of resistance. Understanding the genetic basis of herbicide resistance can aid in formulating and recommending effective weed management strategies that can delay the evolution and spread of herbicide resistance. A proactive weed management strategy should incorporate as many of the following practices as possible: 1) crop rotations, 2) use of herbicides with different modes of action in mixture or in rotation, 3) application of the maximum labeled dose of the herbicide [25]. Use of recommended field rate of herbicides interfere accumulation of minor resistance alleles in the individuals in the population, thereby significantly reducing the risk for polygenic NTSR. Those minor alleles can be pre-existing in the population (genetic variation) or stress-induced due to sub-lethal effect of herbicide [25]. Therefore, factors other than herbicide doses need to be considered for maximum efficacy. Herbicide mixtures, when each herbicide component is highly effective, can effectually delay the evolution of resistance [4,26]. The chance of simultaneous occurrence of mutations in the multiple target sites in a single plant is a product of the individual probabilities; thus, the possibility of resistance evolution to multiple herbicides is much lower. Sub-lethal effects of such mixtures on individuals may lead to simultaneous evolution of NTSR to all components of herbicides in the mixture, especially when the herbicide mixtures target similar metabolic pathways in plants [8].

When resistance has already occurred in a field, reactive weed management strategies, focused towards restricting the spread of herbicide resistance need to be implemented. Herbicide resistant plants, especially when the resistance is conferred by enhanced metabolism of herbicides, can possess resistance to herbicides with different modes of action [27]. Such cases may limit the choice of alternative herbicides in the reactive herbicide resistance programs. Use of reduced-risk herbicide sites of action [reviewed in 27], thus, can be used to manage or delay herbicide resistance. Furthermore, use of integrated weed management strategies including herbicides combined with cultural practices such as tillage can be implemented. In some environments or geographies, cultivation may be a primary option for weed management because cultivation can significantly influence weed species composition and their germination and growth, which consequently affects the amount of seed present in the soil seed bank and thereby spread of resistant plants. Furthermore, use of herbicide mixtures and crop/herbicide rotations also reduce the spread of resistance [28,29]. Therefore, implementation of integrated weed management strategies coupled with educating growers of risks of evolution of weed resistances to herbicides; and disseminating awareness programs highlighting herbicide stewardship are highly warranted to sustain the use of herbicides as weed management tools and reduce spread of herbicide resistance.

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