Review

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Deciphering the evolution of herbicide resistance in weeds

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Resistance to herbicides in arable weeds is increasing rapidly worldwide and threatening global food security. Resistance has now been reported to all major herbicide modes of action despite the development of resistance management strategies in the 1990s. We review here recent advances in understanding the genetic bases and evolutionary drivers of herbicide resistance that highlight the complex nature of selection for this adaptive trait. Whereas early studied cases of resistance were highly herbicide-specific and largely under monogenic control, cases of greatest concern today generally involve resistance to multiple modes of action, are under polygenic control, and are derived from pre-existing stress response pathways. Although 'omics' approaches should enable unraveling the genetic bases of complex resistances, the appearance, selection, and spread of herbicide resistance in weed populations can only be fully elucidated by focusing on evolutionary dynamics and implementing integrative modeling efforts.

Herbicide-resistant weeds: a growing threat to food security

Arable weeds (see Glossary) have been the major biotic cause of crop yield losses since the origins of agriculture. Weeds result in 34% loss of crop yield, on average, worldwide [1]. In the USA alone, the annual cost of crop losses due to weeds is greater than 26 billion US$ [2]. Weeds are thus a major threat to food security. Early in agriculture, weed control was labor-intensive and only moderately effective until the first herbicides were marketed in the late 1940s [1]. Herbicides are by far the most effective weed control tools ever developed, killing 90 to >99% of the weeds targeted (e.g., [3]). Non-chemical weeding can achieve similar efficacies only by combining multiple methods, each generally far more labor-intensive than herbicide application [4]. Consequently, the arable surface treated and range of weed species targeted by herbicides increased rapidly worldwide after their development [1], and a diversity of herbicides are currently used by growers (Figure 1 and Table 1).

This golden age of herbicides was quickly cut short, however, by the detection of the first herbicide-resistant weeds in 1957 [5]. Today, herbicide resistance has been reported in 217 weed species in more than 670 000 fields worldwide (a conservative estimate). Moreover, the number of cases collated at http://www.weedscience.org is continuously rising. Resistance has been reported to all major known herbicide modes of action (see: http://www.weedscience.org), and no new mode of action has been marketed since 1991 [6].

Herbicide resistance is now widely recognized as the result of the adaptive evolution of weed populations to the intense selection pressure exerted by herbicides [7,8]. The least herbicide-sensitive individuals have a selective advantage in weed populations repeatedly treated

Glossary

Allogamy/autogamy: cross-fertilization/self-fertilization in plants. The degree of allogamy and autogamy varies among and within species; mating systems intermediate between complete autogamy and complete allogamy are termed mixed-mating systems.
Arable weed: plant growing unwanted in a cultivated field. Weeds include numerous, diverse, mostly annual short-lived plant species that thrive in highly disturbed agricultural ecosystems.
Constitutive (response/pathway): a response/pathway that is continually activated, as opposed to a response/pathway that is only activated in response to an environmental signal (induced response/pathway).
Cross-resistance: resistance to different herbicides caused by one gene or one mechanism (as opposed to multiple resistance: resistance to different herbicides caused by different genes or different mechanisms).
Effective population size: the size of an ideal population that experiences the same amount of genetic drift as the observed population. In this ideal population, all individuals have an equal probability of contributing to the next generation via reproduction. The effective population size is typically smaller than the actual number of individuals in a population (termed the census population size).
Epigenetic process: process altering gene regulation that does not involve changes in the DNA sequence (e.g., DNA methylation, histone protein modifications). These changes can persist through cell divisions and be transmitted to progeny.
Fitness cost: adaptation to a selective pressure endows a fitness cost when it results in a decrease in the ability of an organism to survive and/or reproduce in the absence of the selective pressure. Fitness cost can result from antagonistic pleiotropy.
Herbicide: synthetic organic molecule used to kill weeds. Herbicides act by lethally inhibiting the activity of proteins crucial for weed physiological processes.
Pleiotropy, antagonistic pleiotropy: phenomenon where one gene influences multiple phenotypic traits in an organism. Antagonistic pleiotropy occurs when selective pressure upon a given trait changes the mean value of a correlated trait that has a negative effect on fitness.
Selective sweep: the rapid increase in frequency of a beneficial allele due to selection that also reduces genetic variation at linked loci.
Soil seed bank: the natural stock of seeds in the soil, which are usually dormant but viable. The soil seed bank is generally the major source of weeds that infest crops.
Standing genetic variation: the presence of multiple alleles at a locus in a population. Genetic variation that is neutral or nearly neutral (i.e., that results from the opposing forces of mutation and genetic drift) may become beneficial after an environmental change.

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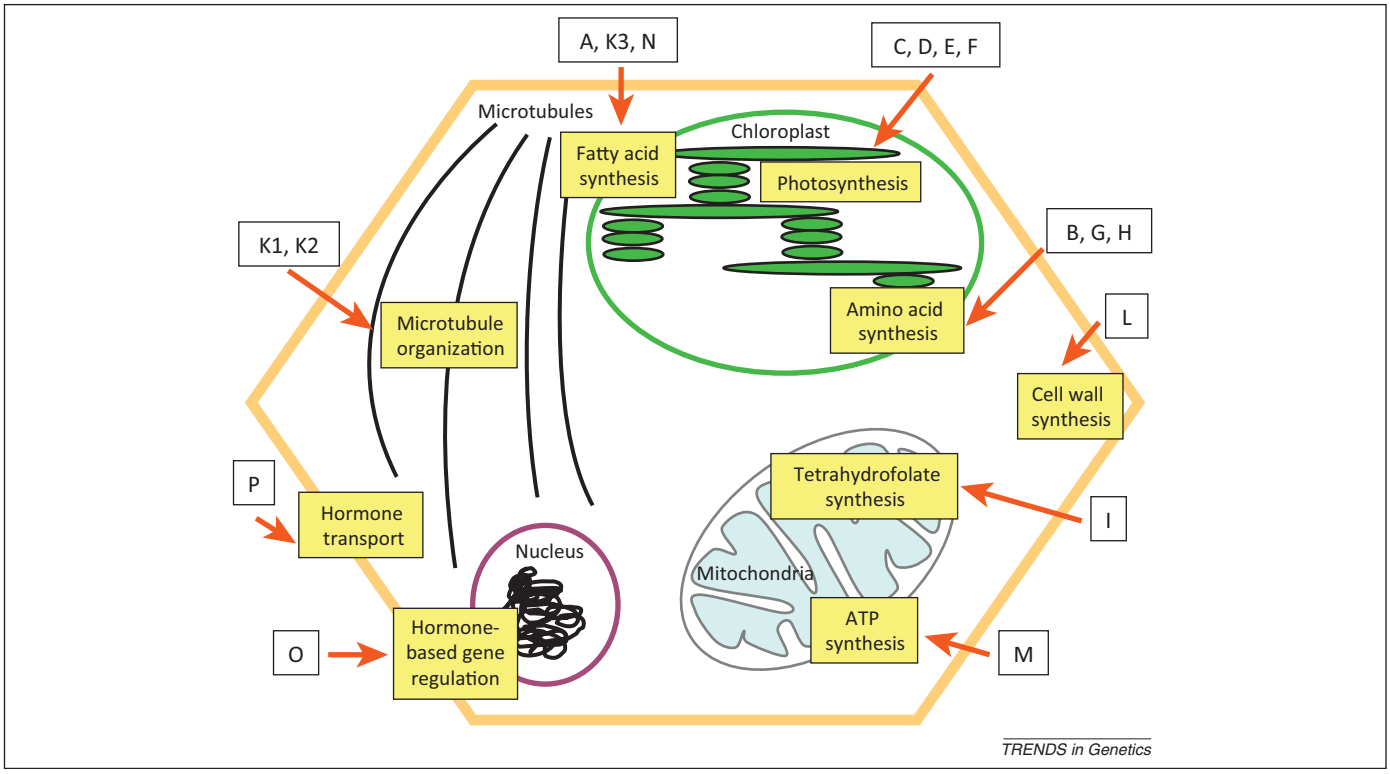

Figure 1. Cellular targets of herbicide action and herbicide classification by mode of action according to the Herbicide Resistance Action Committee (HRAC). Herbicides target only a few proteins or processes among the tremendous range present in plants.

Table 1. Herbicide modes of action

HRAC group[a]	Herbicide mode of action	Gene encoding target protein	Pathway or process targeted
A	Inhibition of acetyl-CoA carboxylase (ACCase)	Nuclear	*Fatty acid biosynthesis*
B	Inhibition of acetohydroxyacid synthase (AHAS, ALS)	Nuclear	*Amino acid biosynthesis* (Leu, Ile, Val)
C	Inhibition of photosystem II protein D1 (psbA)	Chloroplastic	*Photosynthesis* (electron transfer)
D	Diversion of the electrons transferred by the photosystem I ferredoxin (Fd)	Chloroplastic	*Photosynthesis* (electron transfer)
E	Inhibition of protoporphyrinogen oxidase (PPO)	Nuclear	*Photosynthesis* (heme biosynthesis for chlorophyll)
F	Inhibition of phytoene desaturase (PDS) or 4-hydroxyphenylpyruvate dioxygenase (4-HPPD) or of an unknown protein	Nuclear	*Photosynthesis* (carotenoid biosynthesis)
G	Inhibition of 5-enolpyruvylshikimate-3-phosphate synthase (EPSP synthase)	Nuclear	*Amino acid biosynthesis* (Phe, Trp, Tyr)
H	Inhibition of glutamine synthase	Nuclear	*Amino acid biosynthesis* (Gln)
I	Inhibition of dihydropteroate synthase	Nuclear	Tetrahydrofolate biosynthesis
K1, K2	Enhancement of tubulin depolymerization	Nuclear	Microtubule polymerization
K3	Inhibition of fatty acid synthase (FAS)	Nuclear	*Fatty acid biosynthesis*
L	Inhibition of cellulose-synthase	Nuclear	Cell wall biosynthesis
M	Uncoupling of oxidative phosphorylation	-	ATP biosynthesis
N	Inhibition of fatty acid elongase	Nuclear	*Fatty acid biosynthesis*
O	Stimulation of transport inhibitor response protein 1 (TIR1)	Nuclear	Regulation of auxin-responsive genes
P	Inhibition of auxin transport	Unknown	Long-range hormone signaling
Z	Unknown		

[a]The 18 groups of herbicides classified by mode of action according to the global Herbicide Resistance Action Committee (HRAC; http://www.hracglobal.com). The three most widely used groups are indicated in bold. Pathways or processes targeted by several herbicide groups are indicated in italic text.

with herbicide and thus increase in frequency until populations shift towards a predominance of herbicide-resistant individuals. Resistant weeds can survive herbicide application via a variety of mechanisms [9–11] that can be divided into two broad categories. Target-site resistance (TSR) mechanisms include increased expression of the target protein or structural changes to the herbicide binding site (Figure 2). Non-target-site resistance (NTSR) mechanisms include any other mechanism (see Figure 2 for mechanisms currently reported).