Determining herbicide resistance by molecular means

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Herbicide resistance is a steadily increasing problem for farming worldwide. Resistance to specific active ingredients or herbicide mode of action is a consequence of frequent and biased herbicide use. But not all herbicide modes of action are affected similarly. At the moment, most weed species exhibit resistance to herbicide actives belonging to the groups of ACCase inhibitors and ALS inhibitors. Additionally, weed species do not have the same inherent risk to develop herbicide resistance. In Germany, the most critical weeds are the grasses Alopecurus myosuroides (ALOMY) and Apera spica-venti (APESV). Another weed species that is also gaining attention for its potential to develop resistance is the scentless mayweed Tripleurospermum perforatum (MATIN). To manage existing herbicide resistance on fields and to prevent further development and spreading, a deep understanding how herbicide resistance works is required. Two major mechanisms are known: target-site resistance and non-target-site resistance. Target-site resistance is characterized by the alteration of the target protein structure so that the herbicide cannot bind to its target anymore. This alteration is due to an amino acid substitution in the amino acid sequence of the target protein. Several of these substitutions can occur in one protein leading to resistance against different active ingredients. Examples for this are the ALS protein of APESV where 7 different amino acid substitutions were observed to evoke resistance to different ALS inhibitors or the ACCase protein of ALOMY with 5 different amino acid substitutions involved in resistance to ACCase inhibitors. All amino acid substitutions result from single nucleotide polymorphisms (SNPs) in the coding DNA sequence. Different molecular methods, such as PCR Amplification of Specific Alleles (PASA), derived Cleaved Amplified Polymorphic Sequences (dCAPs) or pyrosequencing were described to determine SNPs in literature. In the herbology lab at the JKI in Braunschweig, these methods were adapted for specific SNPs in the ACCase gene of ALOMY and in the ALS gene from ALOMY and MATIN. So, target-site resistance was successfully determined in field samples of ALOMY and in MATIN plants grown in the greenhouse.

Non-target site resistance describes a collection of plant protective measures against herbicide action, such as an enhanced metabolism of the herbicide in the weed or a spatial separation of the herbicide and its target. In contrast to target-site resistance, the understanding and diagnosis of non-target-site resistance is still in its beginnings. Only a few actors were identified by now. Additionally, some genes were shown to be up-regulated in non-target-site herbicide resistant plants. They provide interesting candidates for the future research.