psbA mutation (Ala251 to Val) in *Chenopodium album* resistant to triazinones

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Summary The triazinone herbicides metamitron and metribuzin are inhibitors of photosynthesis at photosystem (PS) II. Like the chemically related s-triazines, they belong to Group C1 of the Herbicide Resistance Action Committee classification of herbicides according to mode of action. Metamitron is a key herbicide in European sugar beet whereas metribuzin is selective to a variety of crops.

So far, four point mutations in the *psbA* gene, encoding the D1 protein of PS II, conferring resistance to triazines have been identified in higher plants from the field. In fat hen (*Chenopodium album* L.), a cosmopolitan weed species, only the most frequent mutation of Ser264 to Gly within the QB binding site of the D1 protein has been reported before. This mutation is responsible for target site resistance to atrazine accompanied by target site cross-resistance to triazinones. In recent years, sugar beet growers in several European countries are experiencing problems with the control of fat hen. Seeds of fat hen populations were collected from sugar beet fields with unsatisfactory control located in Belgium, France and Sweden. The foliage fresh weight response of these populations to metamitron, metribuzin and atrazine was compared to that of two susceptible standards and one atrazine resistant standard from maize carrying the Ser264 to Gly mutation. A limited number of discriminating doses (from one to three), derived from earlier dose response information with triazine resistant fat hen, was used in these greenhouse bioassays. Resistance to metamitron, metribuzin and atrazine was detected in almost all of the Belgian and French 'suspected' populations. However, the one Swedish population displayed resistance to both triazinones but susceptibility to atrazine.

Given these contrasting resistance profiles, it was decided to carry out a DNA sequence analysis of the *psbA* gene using the method described by Naber *et al.* (1990). This method sequences only this part of the D1 protein where all known mutations causing resistance to PS II inhibitors are located, i.e. between amino acid residues Phe211 and Leu275. In five Belgian populations from sugar beet with resistance to triazinones and atrazine and in the atrazine resistant standard, plants surviving a discriminating dose of metamitron and/or atrazine were used for DNA sequence analysis. With the Swedish population, survivors of metamitron treatment were taken. Susceptible populations (two standards and one Belgian susceptible type from sugar beet) had no herbicide treatment. All three susceptible accessions were wild type in their herbicide-binding region. The Ser264 to Gly mutation, known in the atrazine resistant standard, was also confirmed in the five Belgian populations from problem sugar beet fields. However, in the Swedish biotype with the aberrant resistance profile, a different *psbA* point mutation (Ala251 to Val) was identified. This mutation has been previously reported only in unicellular algae such as *Chlamydomonas* and as part of a double mutation in cell cultures of *Chenopodium rubrum* L. To our knowledge, this is the first report of a higher plant from the field with this substitution at position 251 and resistance to PS II inhibitors.

The results recorded with the Swedish mutant type demonstrate that resistance to these triazinone herbicides may evolve in an environment having no history of triazine herbicide use but with selection pressure from the triazinones themselves.

Keywords *Chenopodium album*, *psbA* gene, mutation, resistance, triazinones, photosystem II.

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