Powles Plain English

Making crops and weeds research interesting, understandable, and accessible to all.

Colleagues,

For your information please find attached an AHRI paper by Yu, Hamdani, Han, Christoffers & Powles, just published in the journal Heredity (2013) entitled "*Herbicide resistance-endowing ACCase gene mutations in hexaploid wild oat (Avena fatua): Insights into resistance evolution in a hexaploid species*". Vol 110, 220-231.

Here in AHRI we are particularly proud of this paper, which is the culmination of a great deal of work. We believe this paper sheds light on the complexities of resistance evolution in a polyploidy species, in this case hexaploid Avena. Hexaploid Avena consists of three genomes!

We found three unlinked resistance-endowing ACCase gene mutations that have been previously identified in resistant grass species: Ile-1781-Leu, Asp-2078-Gly and Cys-2088-Arg.

We found that individual plants could express one of these mutations or an individual could express two mutations and **one plant expressed all three mutations**!

Each of these three ACCase gene mutations segregated independently, showing each mutation was on a different genome within the same plant.

Importantly, **ALL three ACCase gene mutations were expressed**, meaning that that ALL three ACCase gene copies are expressed respectively in three genomes. Sometimes, tetraploids operate as functional diploids but this was not the case with these hexaploid Avena populations in which all copies of a gene on three genomes are expressed!

An important evolutionary learning and a clear difference from diploids became evident in that resistant hexaploid Avena homozygous for only one resistant ACCase gene (thus resistance only in one of the three genomes) displayed only relatively low level resistance to ACCase herbicides. This is in marked contrast to much higher resistance in a homozygous diploid plant expressing the same mutation.

Obviously, in hexaploid Avena if only one ACCase resistance mutation is present (even if homozygous for resistance on only one genome) then resistant ACCase is only 1/3rd whereas the individuals still has 2/3rds normal herbicide susceptible ACCase. **Thus the hexaploid plant carrying only one homozygous resistance mutation shows only low level resistance to ACCase herbicides.** This contributes to relatively slow dynamics of resistance evolution in hexaploid Avena in contrast to a diploid in which even a heterozygous resistant individual has 50% resistant ACCase and 50% susceptible and can be strongly resistant. Only as hexaploid resistant

Avena plants are homozygous for resistance on one genome and resistance mutations are also present on other genomes do the plants attain a high level of ACCase herbicide resistance. This helps explain that resistance evolves more slowly in a hexaploid species than in unrelated diploid species. This confirms our field observations which always show that in the same field in which Avena and Lolium co-exist and are under ACCase herbicide selection we see that ACCase herbicide resistance evolves much faster in diploid Lolium than in hexaploid Avena. Of course, the above explanation is for ACCase gene mutations, which are mostly semi dominant in nature.

While in this paper we have focussed on the target site ACCase gene mutations in hexaploid Avena It must also be noted that in addition to the three ACCase resistance mutations that non target site enhanced metabolism resistance (likely P450) was evident in the resistant Avena populations.

We believe that this paper in hexaploid Avena sheds light on resistance evolution in showing that resistance evolution can be much slower in a hexaploid than in diploids.

Thank you,

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