HERBICIDE RESISTANCE

Kreiner et al 2019

Multiple modes of convergent adaptation in the spread of glyphosate-resistant *Amaranthus tuberculatus*

BACKGROUND

HERBICIDE RESISTANCE

Herbicides are used across the world to control weeds

Strong selective pressure on weeds to evolve resistance

>500 unique cases of resistance known (Heap 2021)

How does resistance evolve?

Population genetics may help predict and prevent future resistance (Kreiner et al 2018)

GLYPHOSATE

Glyphosate is the active ingredient in Roundup Ready technology

Ubiquitous (e.g., >80% of soybean fields in USA)

Resistance is widespread (weedscience.org)

AMARANTHUS TUBERCULATUS

Historically a riparian species

There are now many agriculturally associated populations

Resistance first arose in Missouri in 2005, now in 19 states

Resistance in Ontario ~2010

QUESTIONS

How does resistance evolve?

- what is its genetic basis?

How many times has resistance evolved?

- one mutation that spread via gene flow, or multiple mutations?

METHODS

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Seeds from 163 individuals from 19 fields

- Missouri, Illinois, and Ontario (Essex and Walpole)

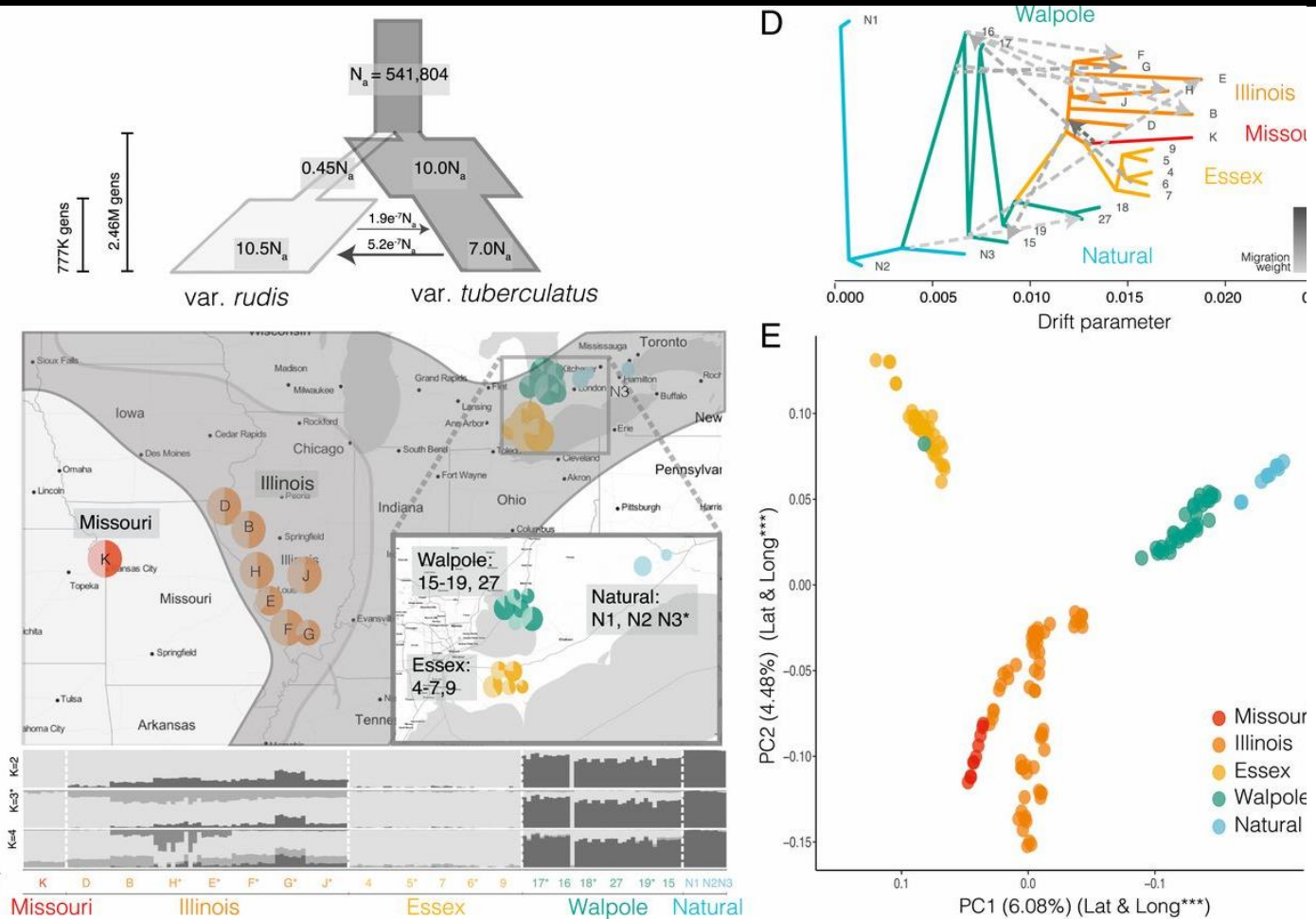
Seeds from 10 individuals in non-agricultural setting (Ontario)

Built genome, resequenced all individuals

Scored resistance in the greenhouse

RESULTS

DEMOGRAPHY



Used computer programs (STRUCTURE, dadi, treemix, and PCA) to infer demography of the population from genomes

- 2 varieties
- lots of gene flow
- resistance everywhere

GENETIC BASIS OF RESISTANCE

Compared genomes of Walpole individuals (some resistant) with non-agricultural individuals (not resistant) to look for difference that might explain resistance

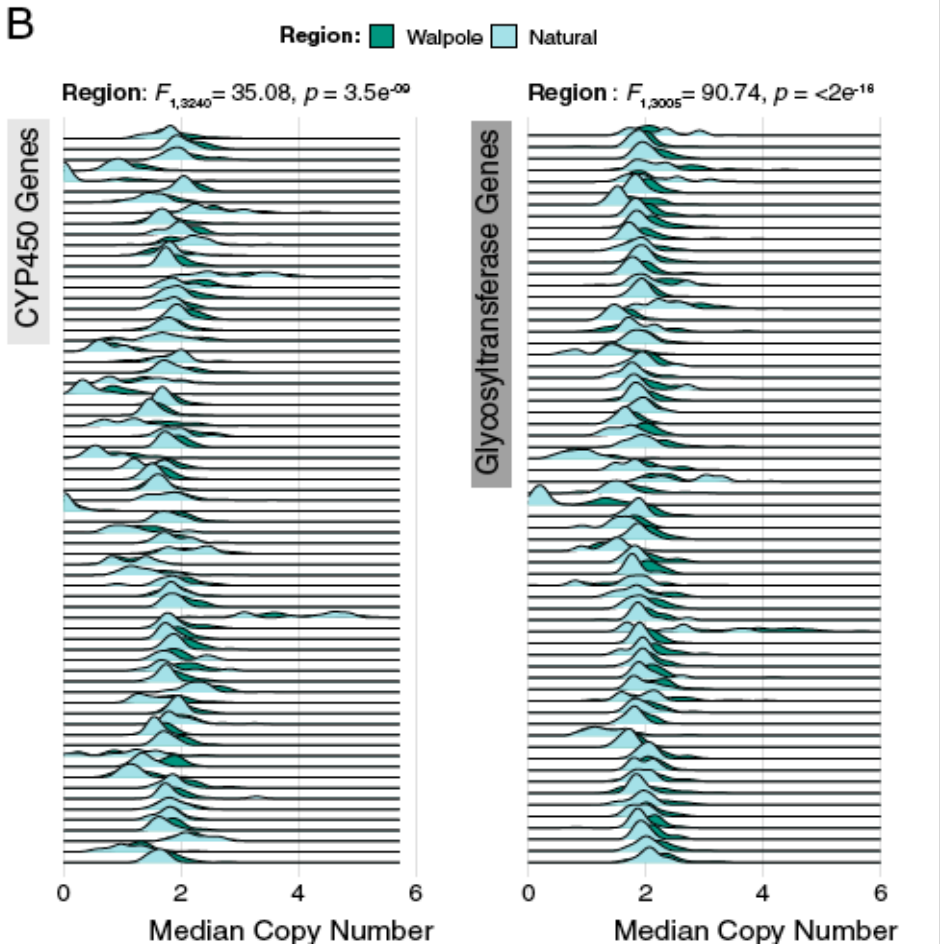
- 2 families of genes involved in detoxifying herbicides have increased copy number in Walpole

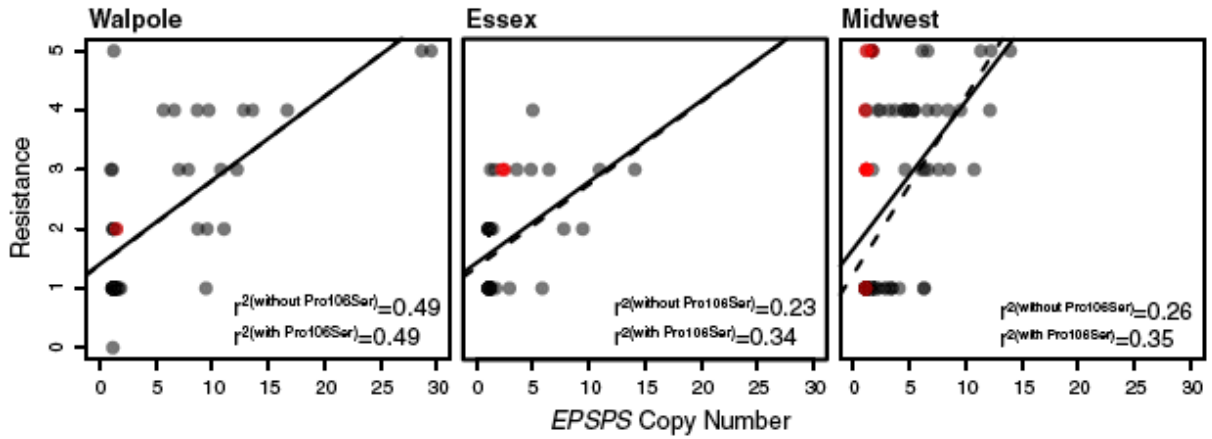
- but did not correlate with resistance in the greenhouse, suggesting more factors involved

A

Molecular Function	Fold Enrichment	raw P value	FDR
alkane 1-monooxygenase activity	46.08	0.00000682	0.021
oxidoreductase activity	40.32	0.0000101	0.0156
Biological Process			
histone lysine methylation	16.8	2.50E-05	4.92E-02
peptidyl-lysine methylation	14.23	8.73E-06	5.16E-02
peptidyl-amino acid modification	3.95	1.15E-05	3.40E-02
Protein Class			
DNA methyltransferase	15.12	0.00153	0.0531
ribonucleoprotein	6.3	0.00158	0.0459
transfer/carrier protein	4.55	0.00119	0.0516
dehydrogenase	2.92	0.000743	0.0431
oxidoreductase	2.42	0.0000143	0.00249
hydrolase	1.78	0.00164	0.0409

B

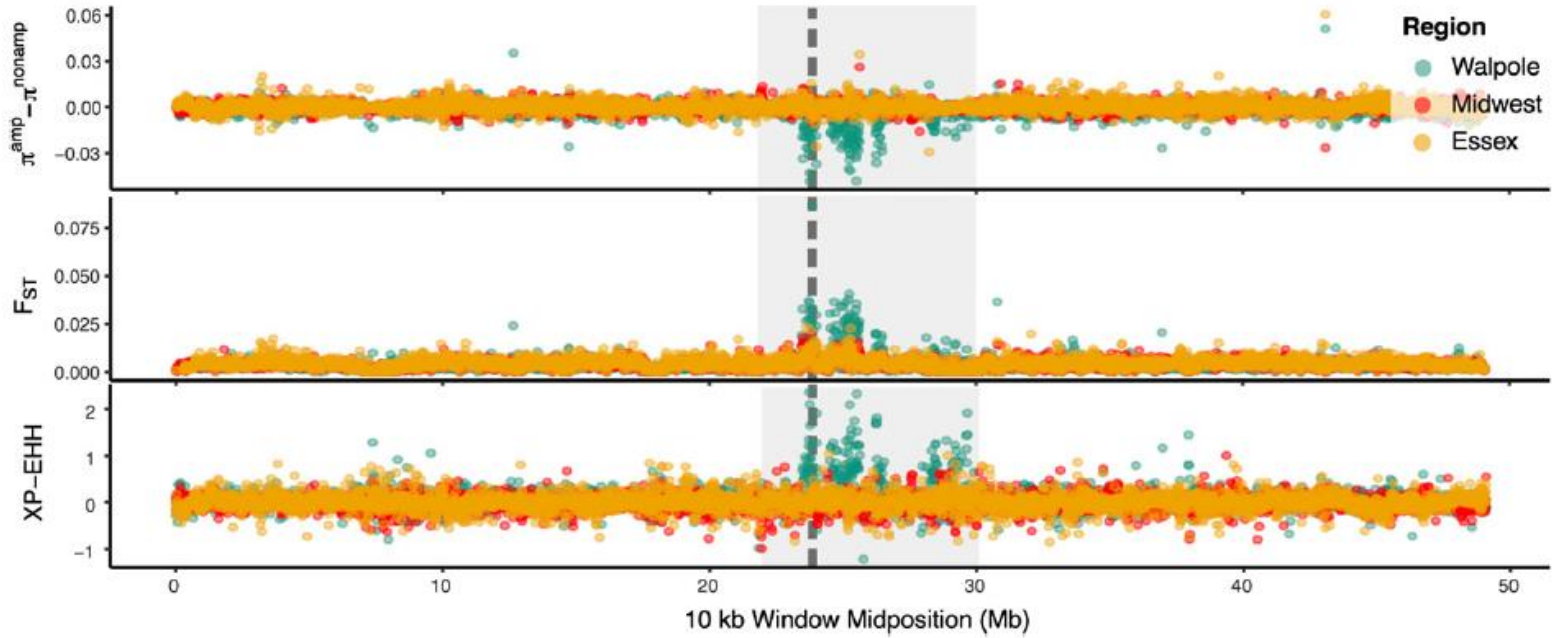




GENETIC BASIS OF RESISTANCE

Suspected EPSPS gene might be involved in resistance, so compared copy number of EPSPS and mutations in EPSPS with greenhouse resistance

- together explained ~30-60% of variation in resistance

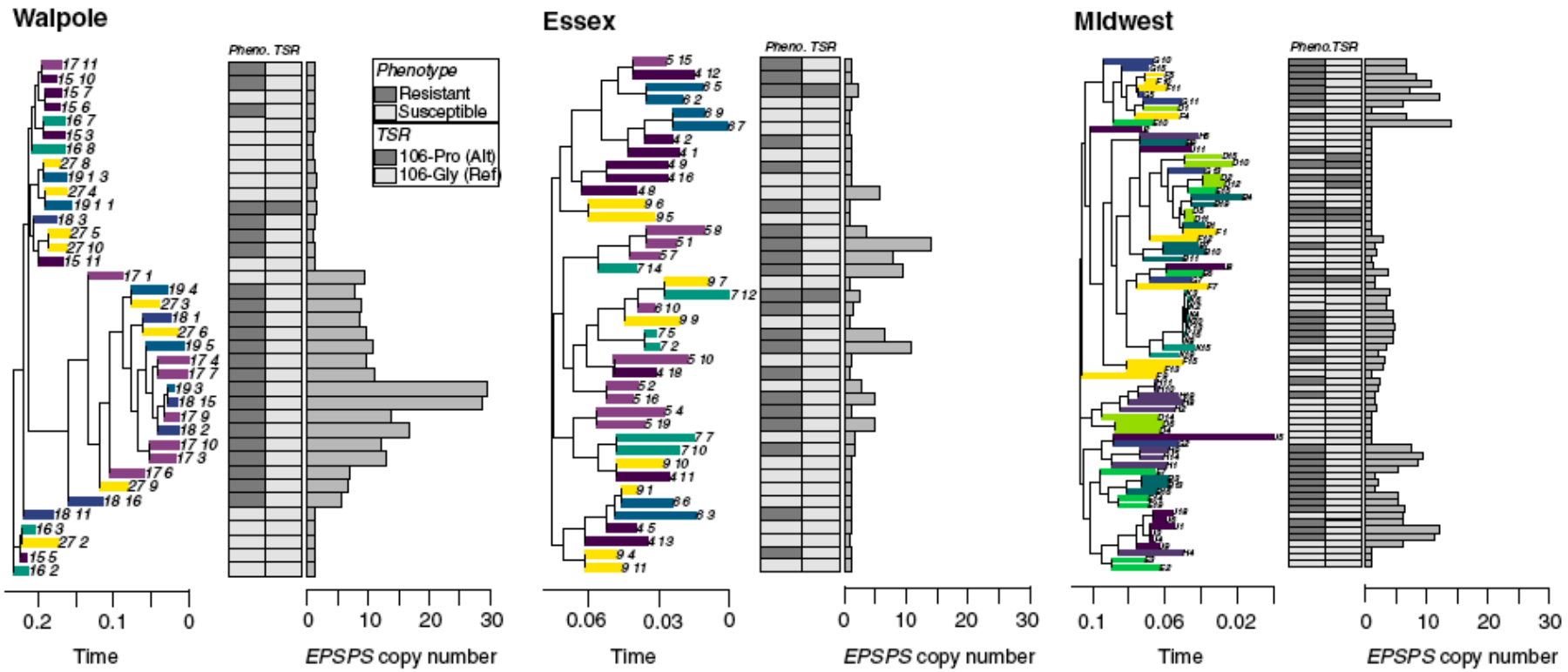


GENETIC SIGNALS OF SELECTION

Looked at the EPSPS region of the genome for signatures of natural selection

- found evidence for strong selection in Walpole

HOW MANY TIMES HAS RESISTANCE ARISEN?

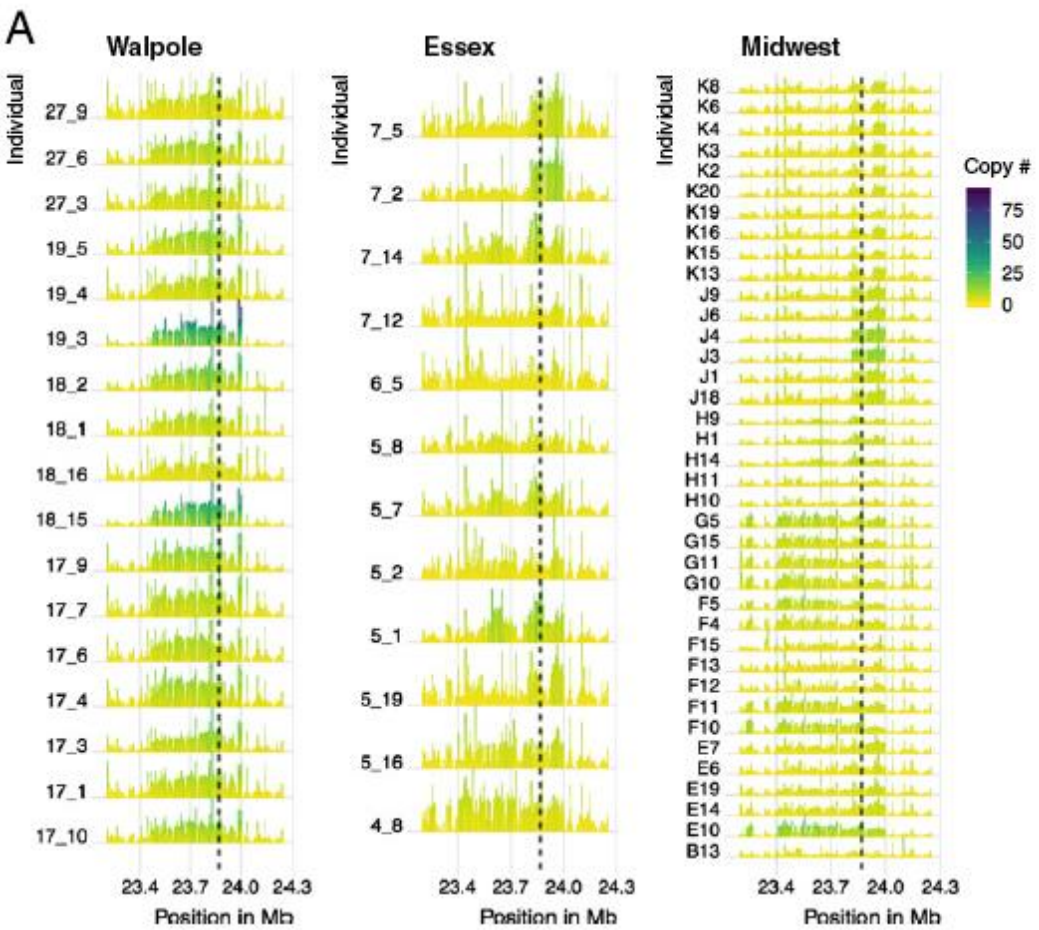


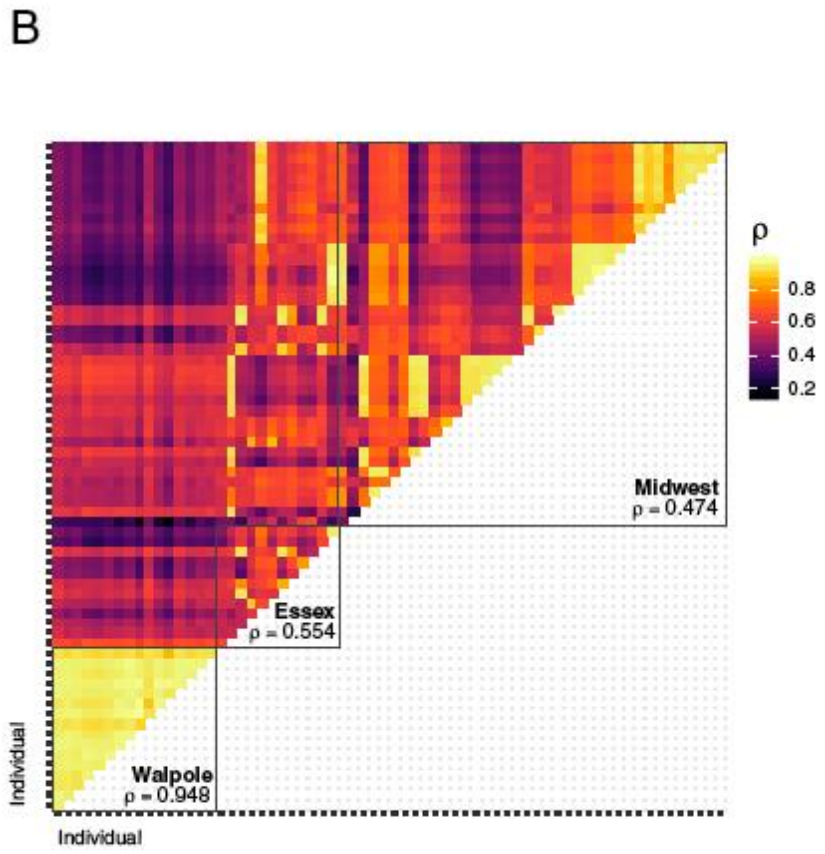
Mapped resistance and EPSPS copy number onto a phylogenetic tree to count occurrences

- 1 mutation in Walpole

- >1 elsewhere

HOW MANY TIMES HAS RESISTANCE ARISEN?





Compared EPSPS copy number profile across individuals

- 1 group in Walpole
- >1 elsewhere
- Essex and Midwest share some groups

CONCLUSIONS

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Resistance widespread

Resistance involves multiple genes (EPSPS, CYP450s, …)

Resistance has rapidly evolved multiple times (at least 3?)

Resistance genes also appear to have spread via gene flow

So perhaps resistance can be slowed by reduced gene flow, but seems inevitable that resistance will arise in this system?

OUTSTANDING QUESTIONS

1. Where did resistance in Walpole arise from? Was it new mutations on the non-agricultural var. *tuberculatus* background? Or was it introgression from an unsampled var. *rudis* population? How to test for this?

2. How could we prevent resistance arising from sensitive populations in the future? On/off herbicide regimes (crop rotation)? More herbicides? Different herbicides?

3. Why is the number of copies of a gene so important in this case? Why isn't resistance determined more by mutations in the gene copies that are already there?