Characterization of the genetic basis of resistance to (bio)pesticides in Cydia pomonella

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CHARACTERIZATION OF THE GENETIC BASES OF RESISTANCE TO (BIO)-PESTICIDES IN *CYDIA POMONELLA (L.)*

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BACKGROUND

Cydia pomonella (Lepidoptera) is the major pest in apple orchards worldwide

Emergence of resistances in field

Current methods of detection have drawbacks : cost, accuracy, duration (~1 year)

Need to develop more effective tools!

Known types of resistance in *C. pomonella*:

Target-site resistance	Detoxification
Simple and known	Complex and mostly unknown

What are the mechanisms involved in multi-resistance?

? **Are there specific or common mechanisms depending on the active substance?**

Is detoxification the main mechanism at work?

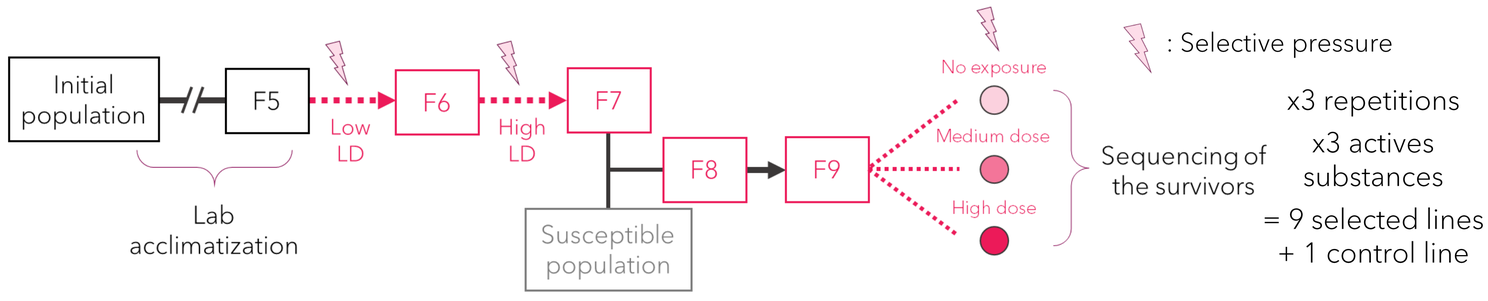
METHODS

One population sampled in the South-East of France, characterized as multi-resistant to different insecticides.

An 'evolve and resequence' experiment was conducted to detect genomic signatures of resistance from standing genetic variation.

It consists of several steps:

A) Selecting pesticide resistant strains through an experimental evolution



'Evolve and resequence' experiment design

3 strains were selected:
Spinosad-resistant (SPI), Deltamethrin-resistant (DEL) and Chlorantraniliprole-resistant (CHL).
Within each strain, a gradient of dose was applied: no exposure (C), medium dose (M-LD) and high dose (H-LD).

B) Phenotyping using bioassays to assess the resistance level of the strains

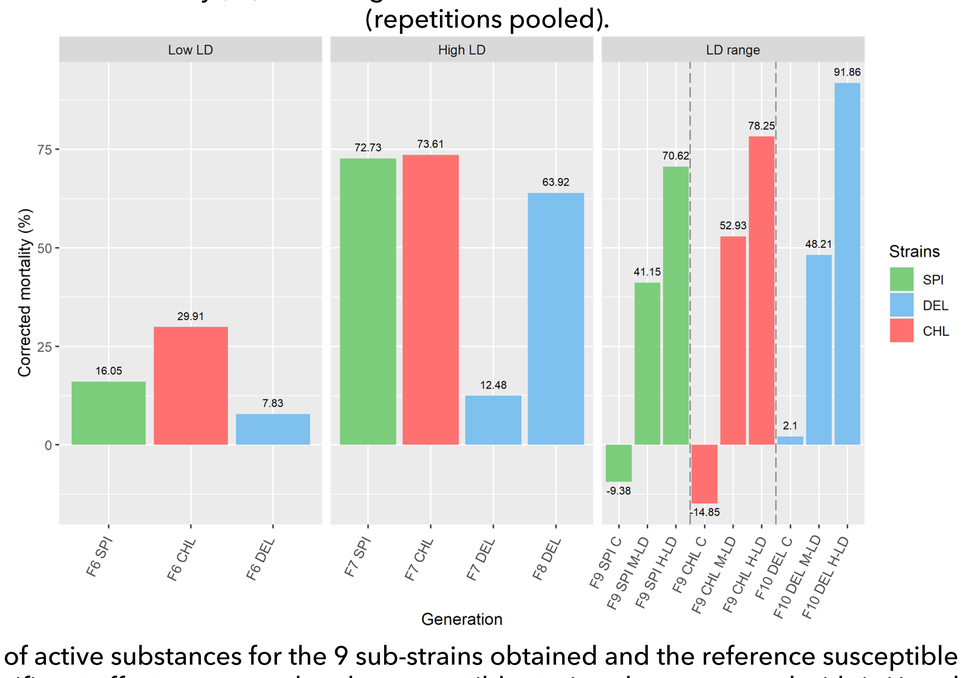
C) RNA-pool seq and DNA-pool seq of the F9 to identify the differential expression between reference genome and strains and variants through a filtering approach

$$F_{SNP\ (control)} \leq F_{SNP\ (F9\text{-}10\ no\ exposure)} \leq F_{SNP\ (F9\text{-}10\ medium\ dose)} < F_{SNP\ (F9\text{-}10\ high\ dose)} > 0.2$$

Criteria used to identify polymorphisms associated with resistance in our variant analyses

MAIN FINDINGS

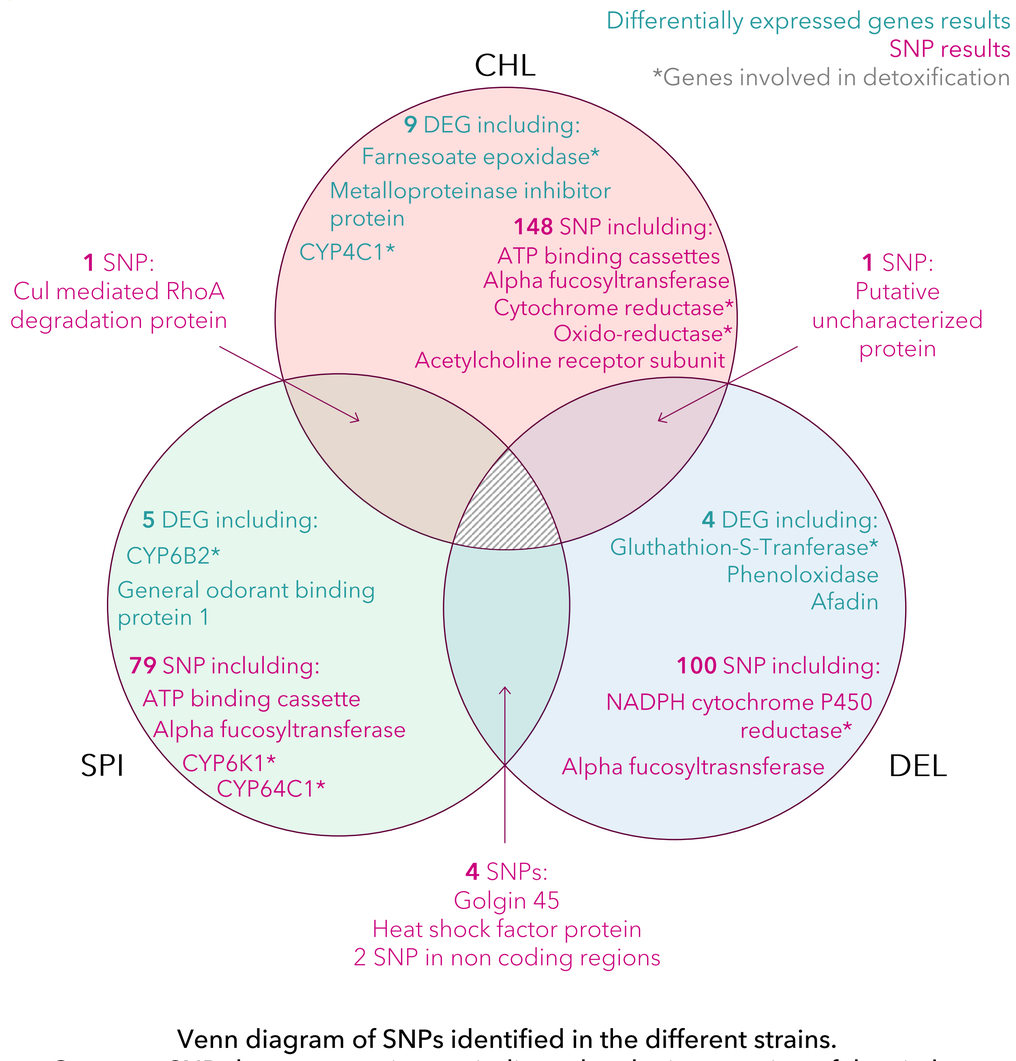
A) Corrected mortality (%) for each generation of selection of each of the strains created (repetitions pooled).



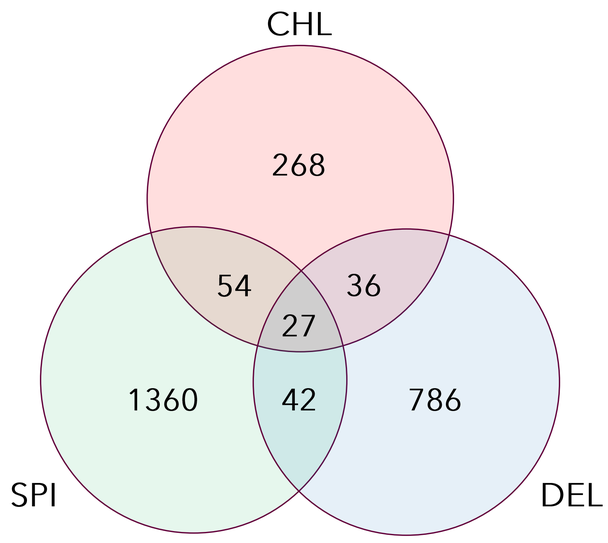
B) LD50 of active substances for the 9 sub-strains obtained and the reference susceptible strain. Significant effects compared to the susceptible strain values are noted with *, ** and ***.

Active substances	Strains			
	F9-10 No exposure strain	F9-10 Medium dose strain	F9-10 High dose strain	Susceptible strain
Spinosad	5.66 ns (±2.11)	7.53 *** (±1.288)	7.35 ** (±1.671)	2.84 (±0.963)
Deltamethrin	0.064 ns (±0.025)	0.111 ns (±0.036)	0.193 ns (±0.92)	0.083 (±0.027)
Chlorantraniliprole	0.517 ns (±0.053)	0.823 ns (±0.121)	0.75 ns (±0.111)	0.687 (±0.127)

C) Results from the differential expression and transcript polymorphisms analyses.



Venn diagram of SNPs identified in the different strains.
Common SNPs between strains are indicated at the intersection of the circles



CONCLUSIONS

- **Precise phenotype characterization is complex.**
  → Inconsistency between an effective selection in rearing and phenotype measurements.
- Rather than cross-resistance, it seems that an accumulation of mechanisms conferred resistance to our population, leading to **multiple resistance.**
- Detoxification is not the main mechanism at work, **other mechanisms of metabolic resistance** and **genes involved in other functions** are involved in resistance.
- **Several markers** were **identified as linked to resistance** phenotypes, providing a basis for the development of **new biomolecular tools for resistance monitoring** in *C. pomonella*.

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