Who controls cytoplasmic incompatibility? Host genetic effects on *Wolbachia*-induced sterility in *Drosophila suzukii*

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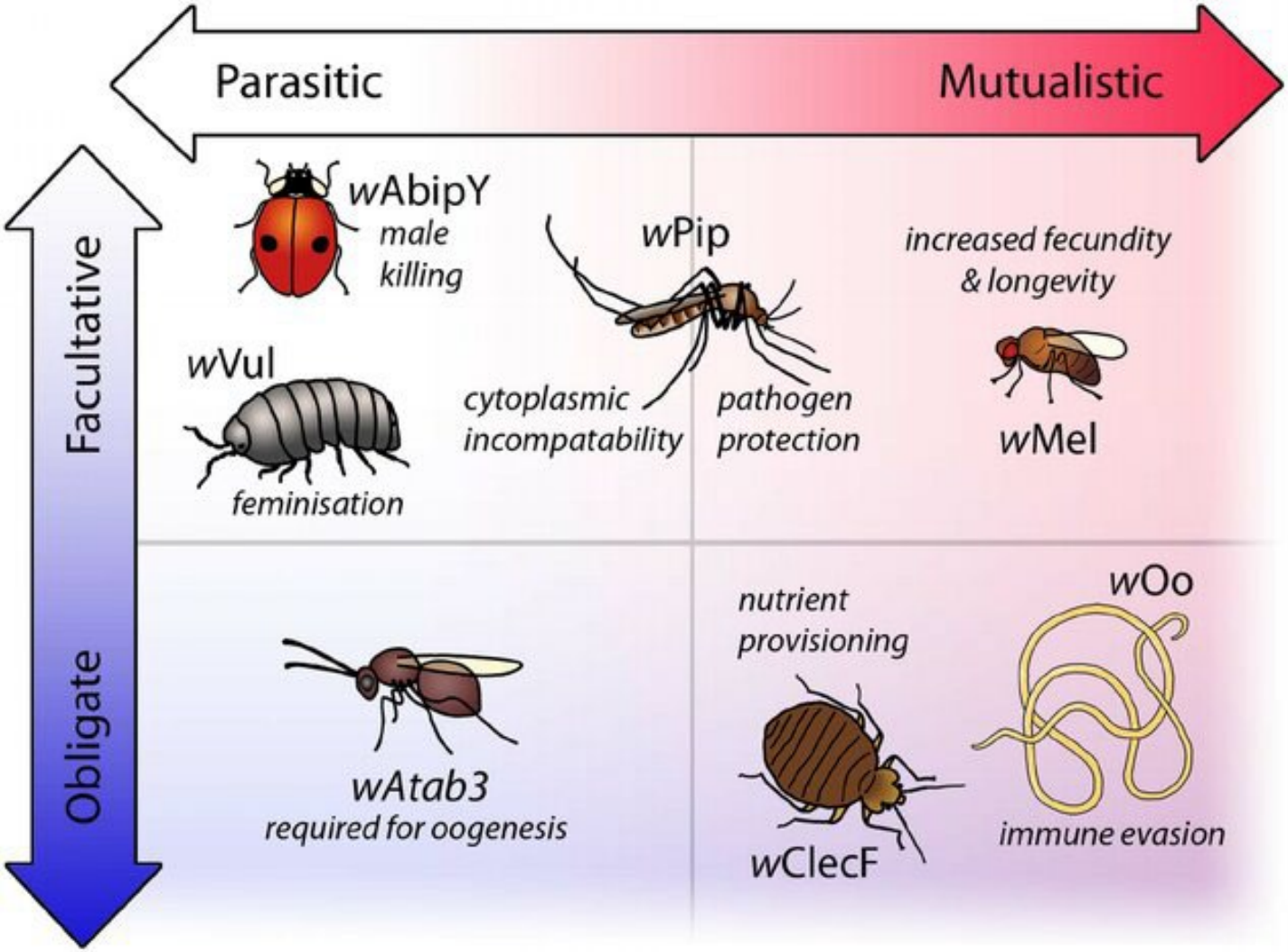
CBGP Seminar, 24 March 2026

Wolbachia are the most prevalent symbiotic bacteria in the world!

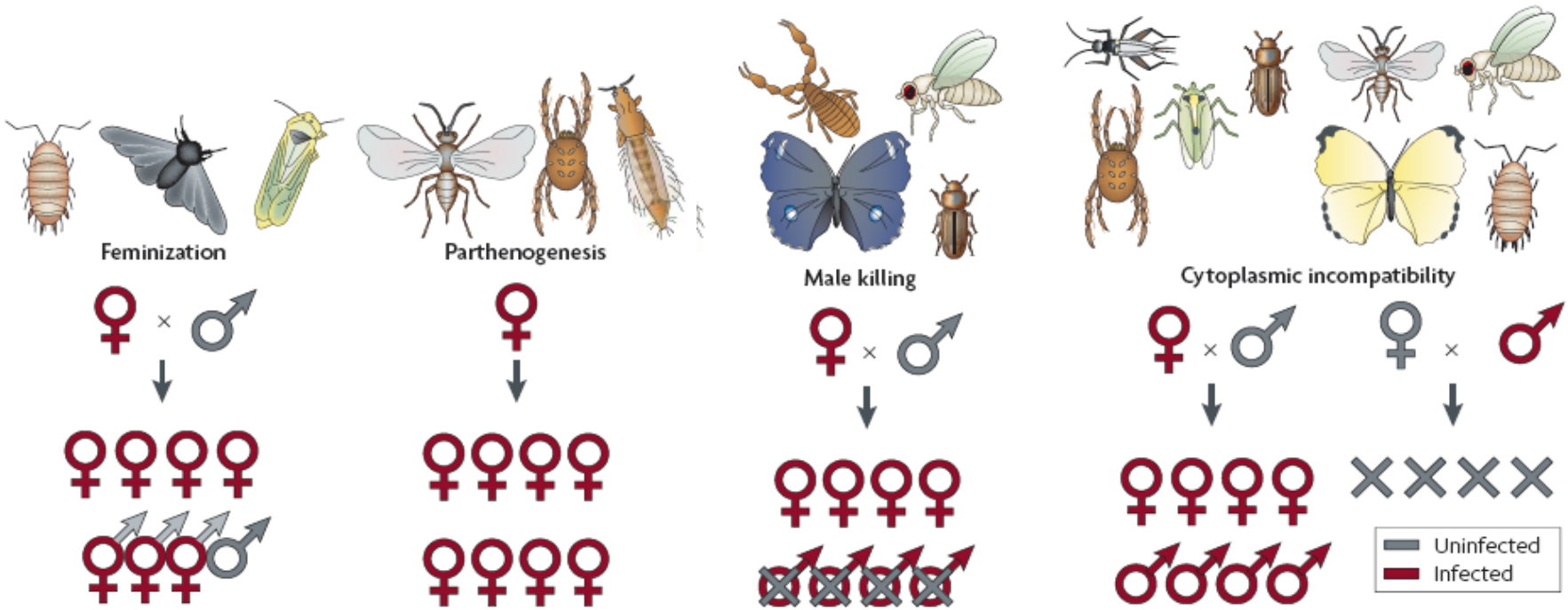
Wolbachia infect ~ 50% all insect species

Examples of *Wolbachia* phenotypes



(Gill et al, 2014)

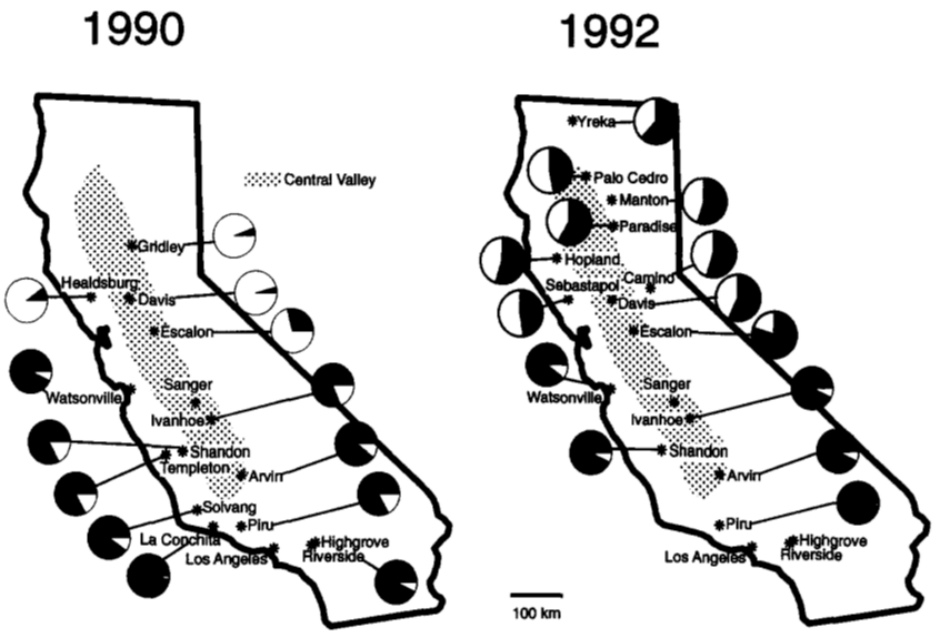
Reproductive manipulations induces by *Wolbachia*



Cytoplasmic incompatibility in 25% of all insect species

Werren et al, 2008

Rapid spread of CI-inducing *Wolbachia* in populations of *Drosophila simulans*



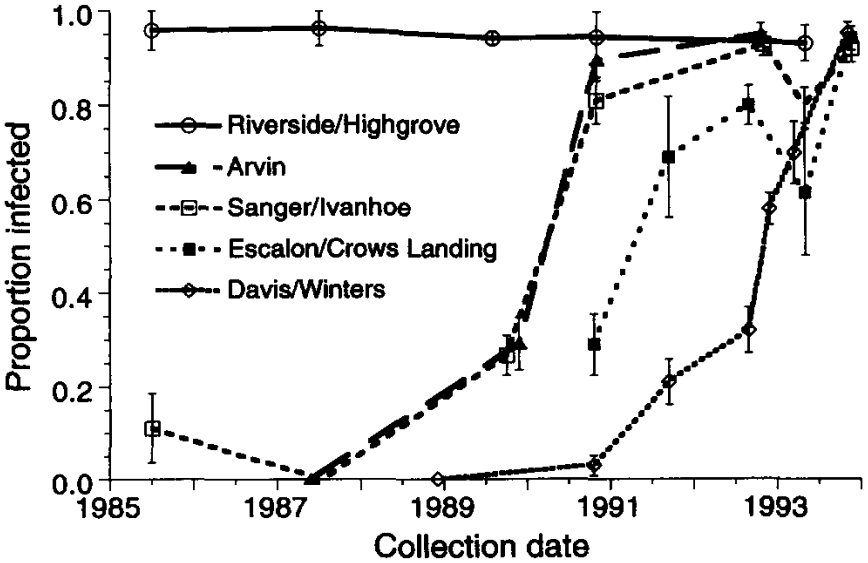
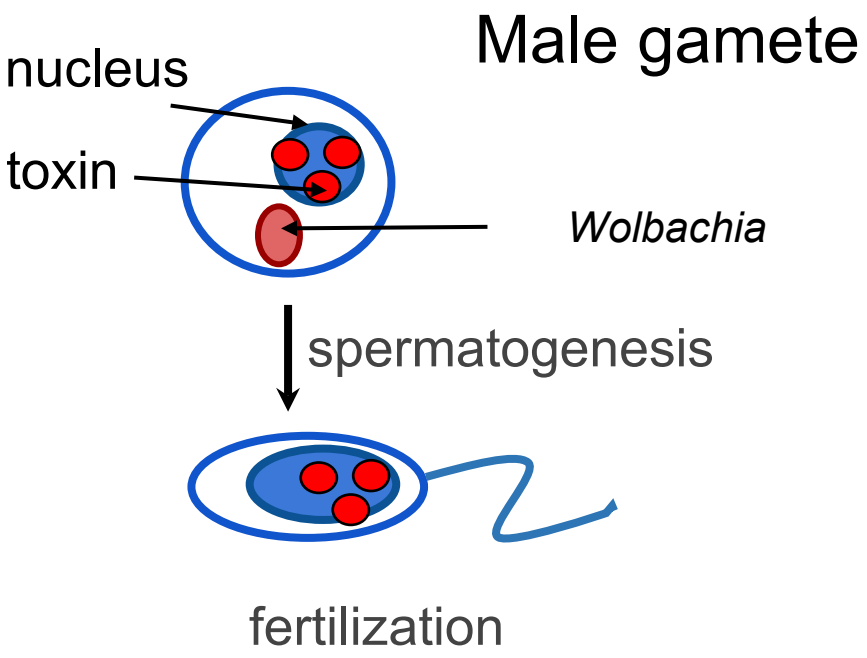


FIGURE 2.—Infection frequency at five sites in California over time. See Figure 1 for approximate locations.

Turelli and Hoffmann, 1995

Molecular mechanism of cytoplasmic incompatibility

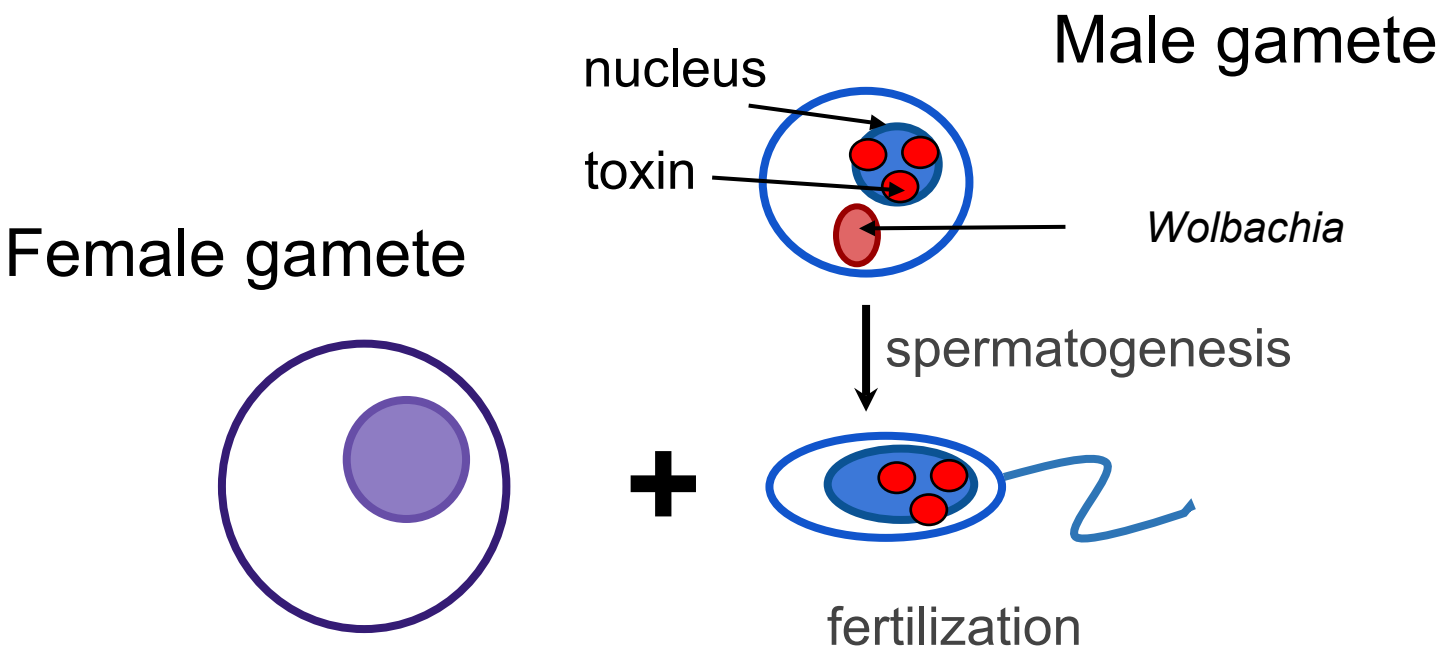
Toxin-antidote model



Beckmann et al., 2019

Molecular mechanism of cytoplasmic incompatibility

Toxin-antidote model



Beckmann et al., 2019

Molecular mechanism of cytoplasmic incompatibility

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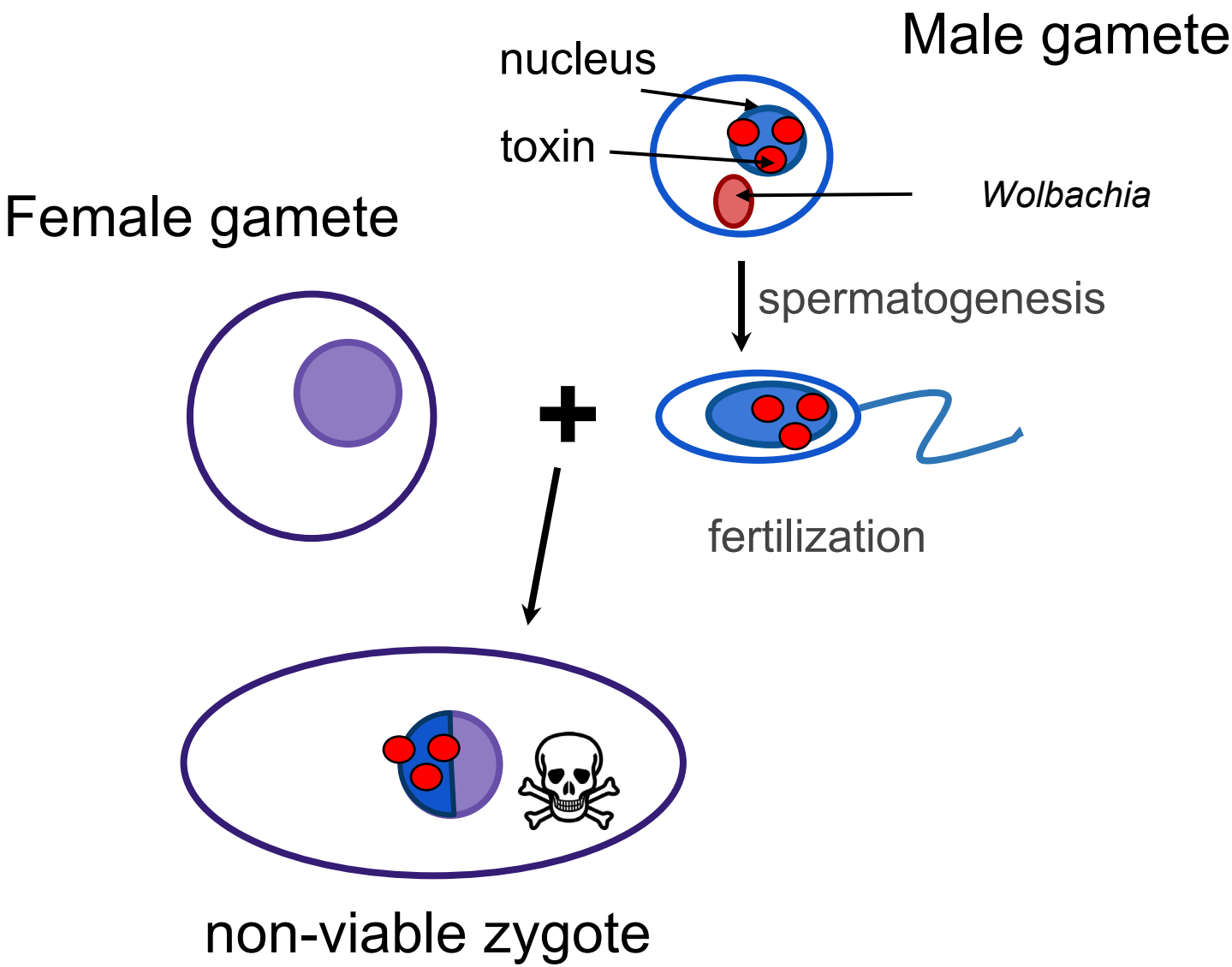
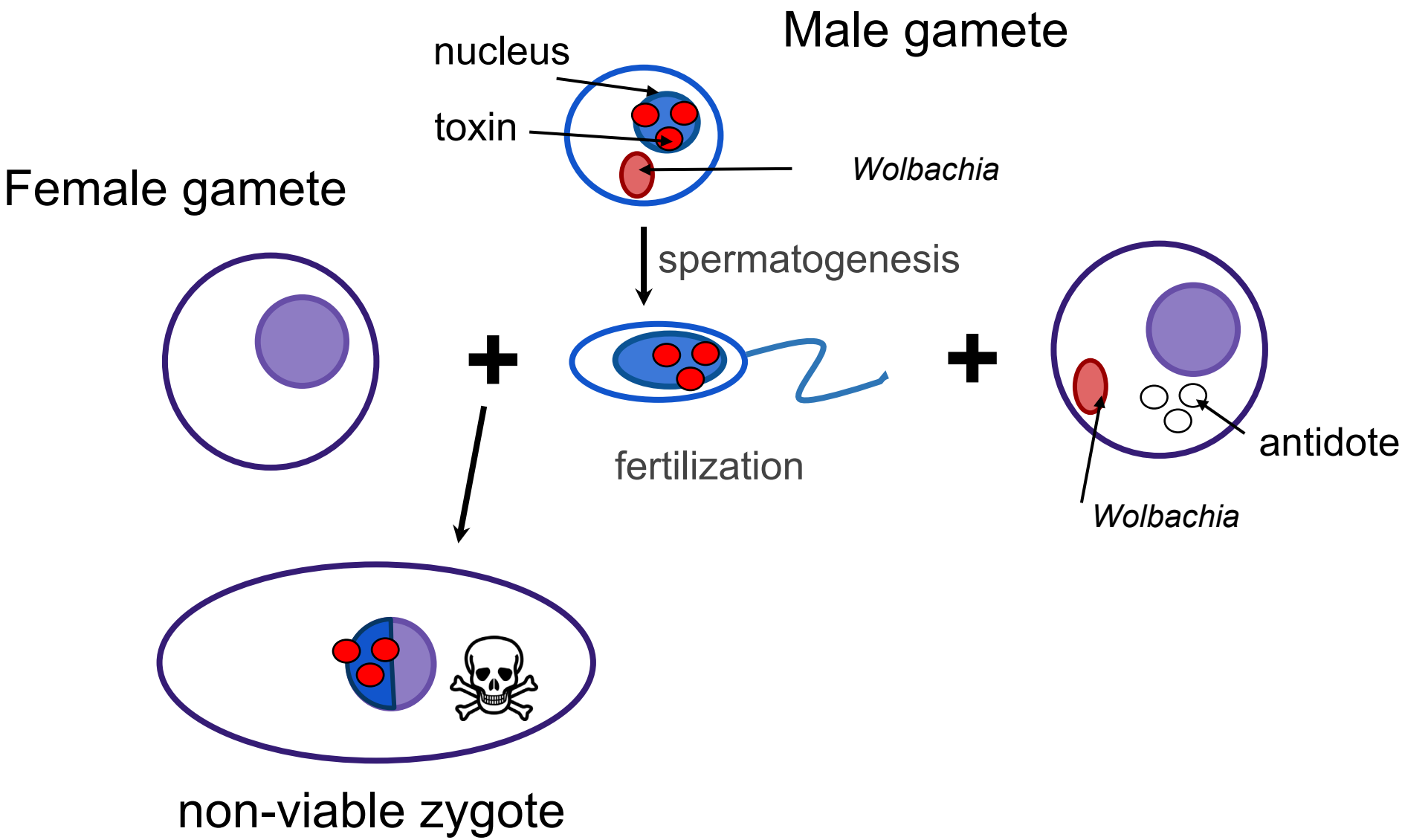


Beckmann et al., 2019

Molecular mechanism of cytoplasmic incompatibility

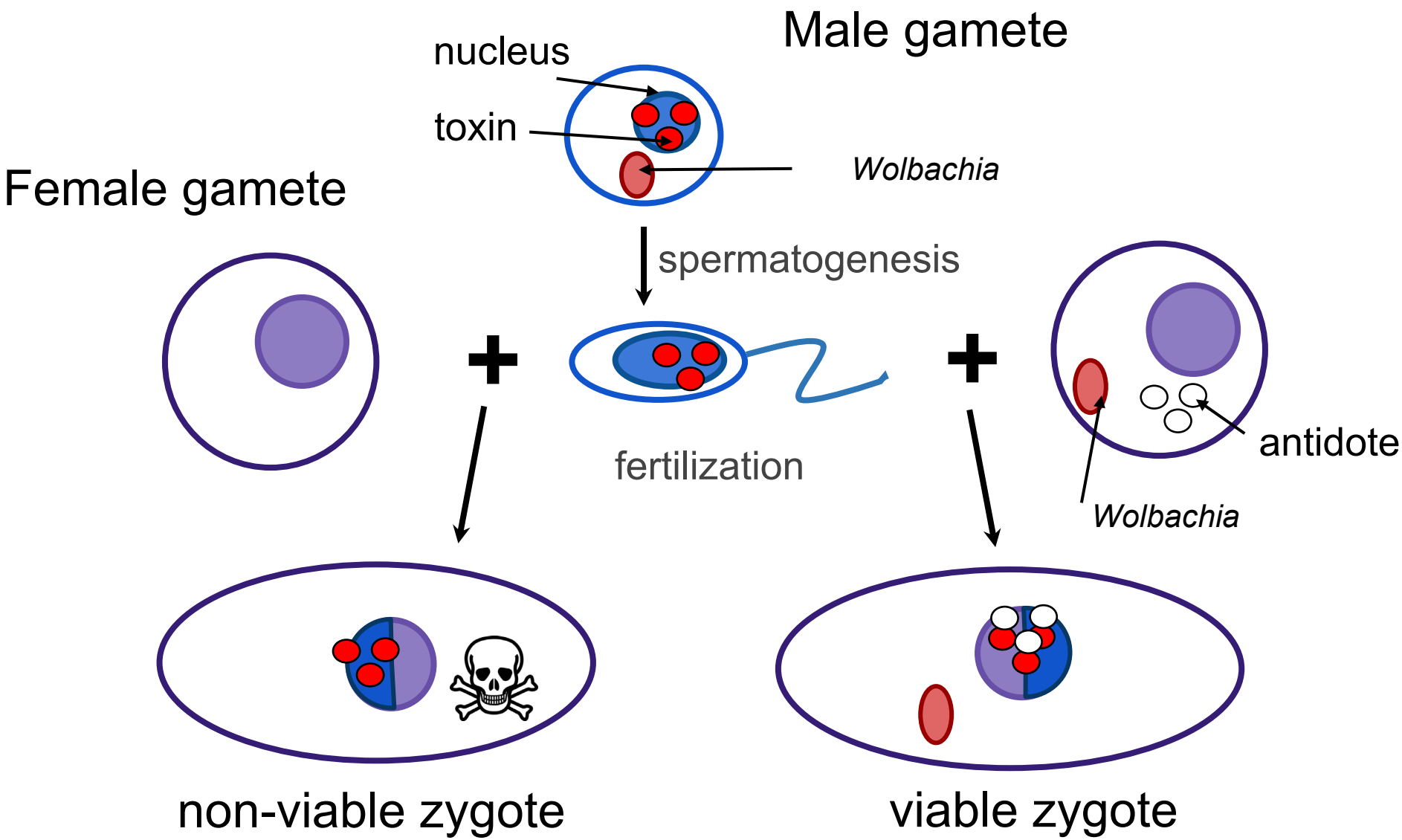
Toxin-antidote model



Beckmann et al., 2019

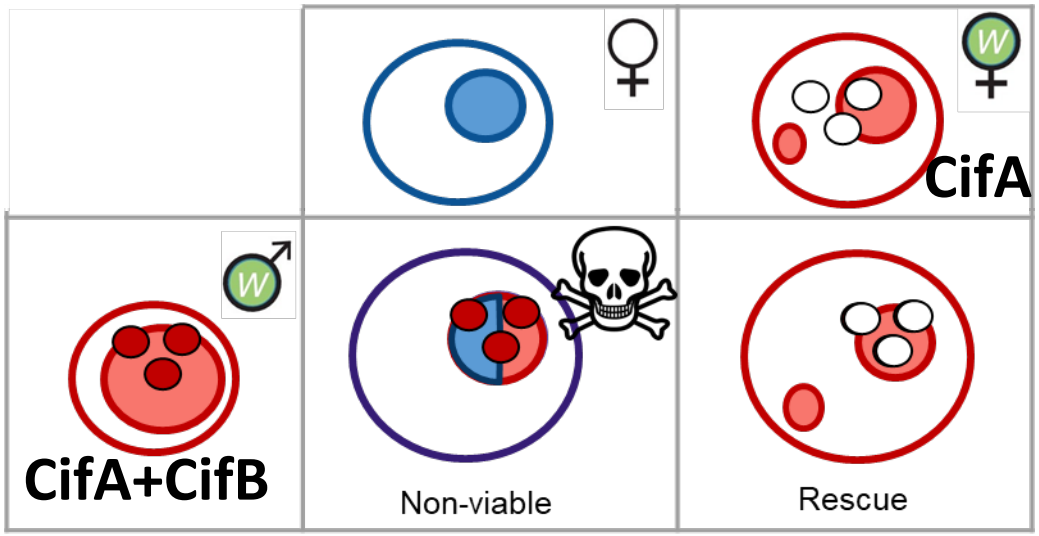
Molecular mechanism of cytoplasmic incompatibility

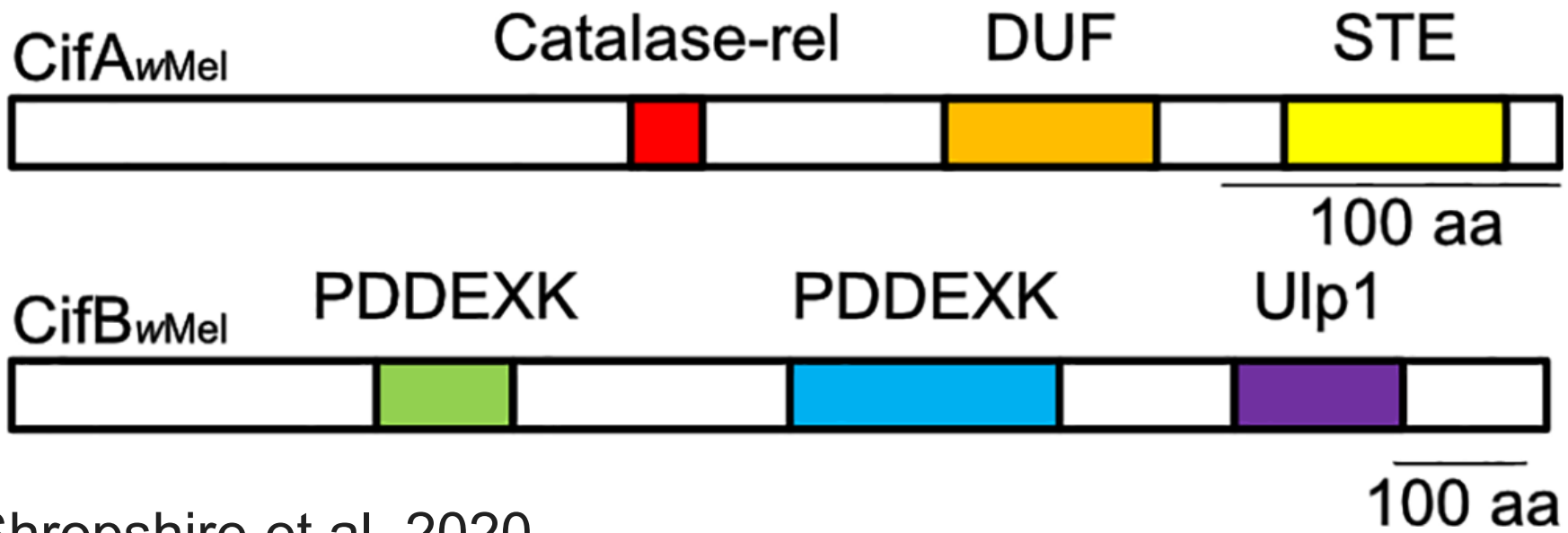
Toxin-antidote model



Beckmann et al., 2019

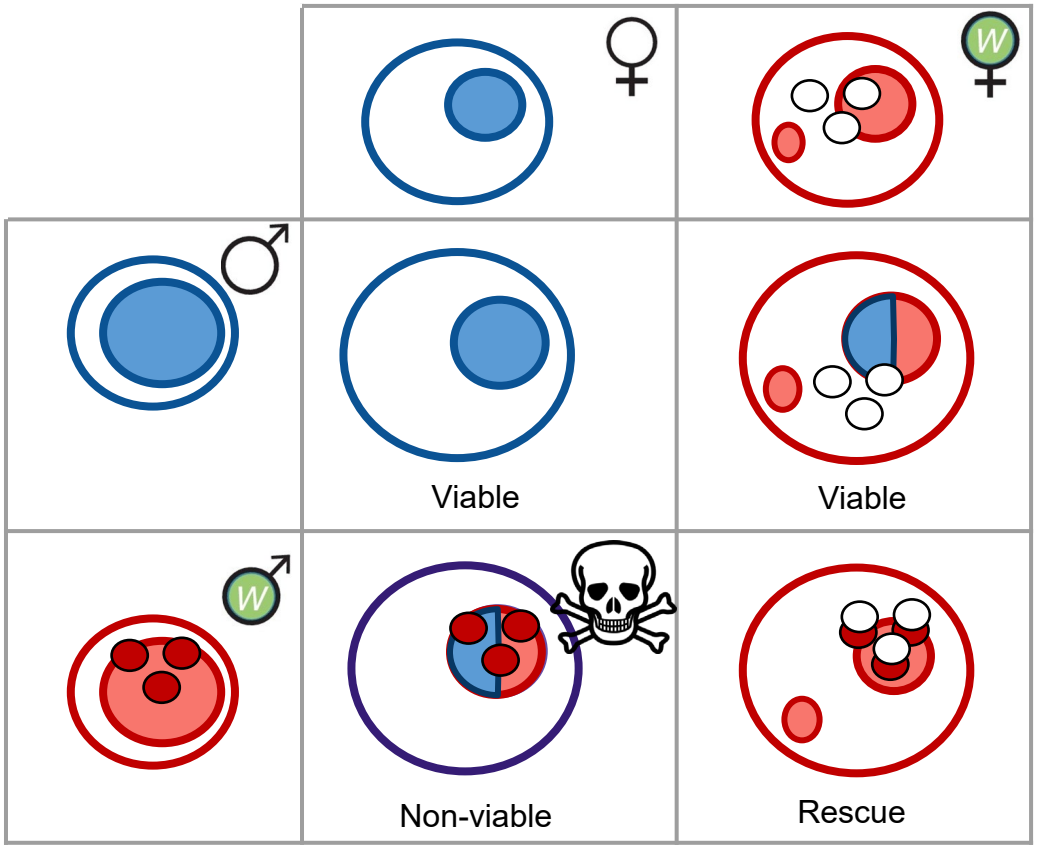
cif genes in *Wolbachia* genome are responsible for CI





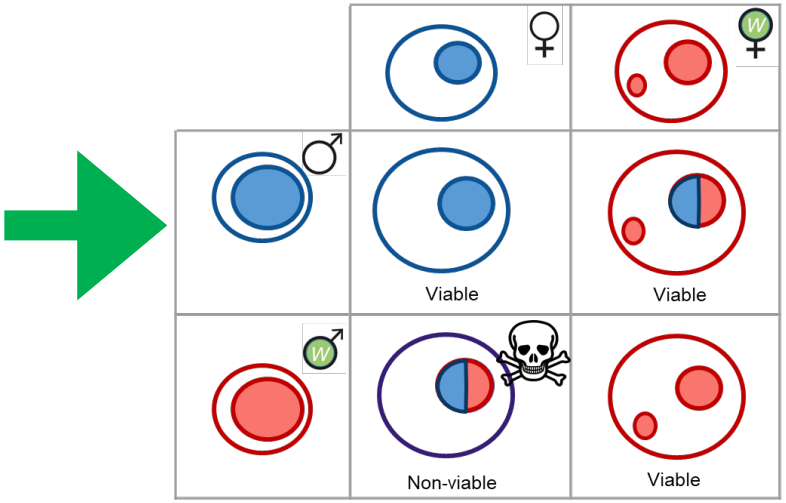
Shropshire et al, 2020

Toxin-antidote model of cytoplasmic incompatibility

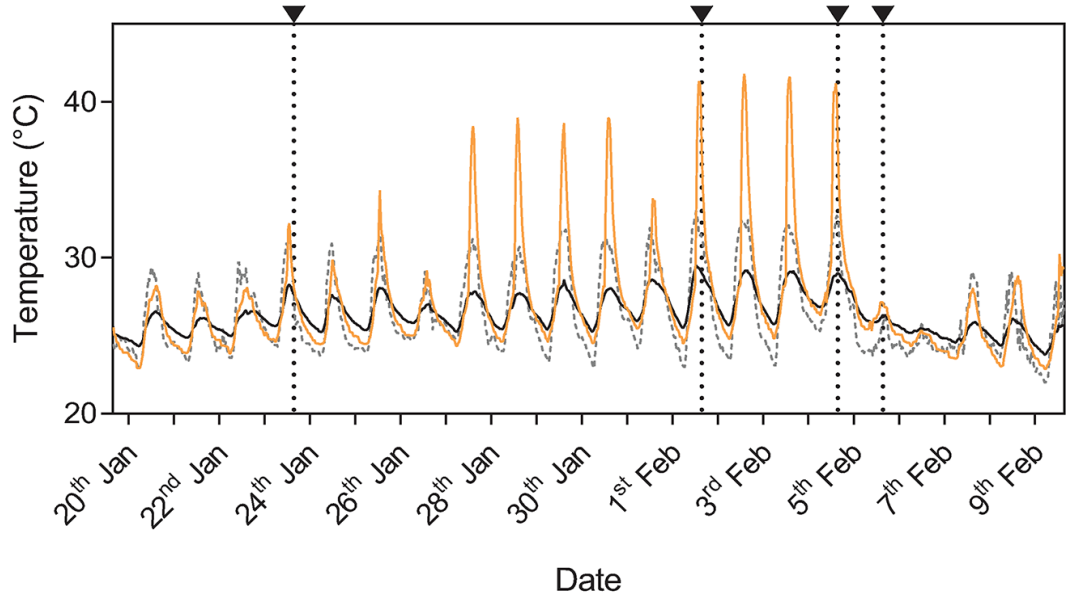


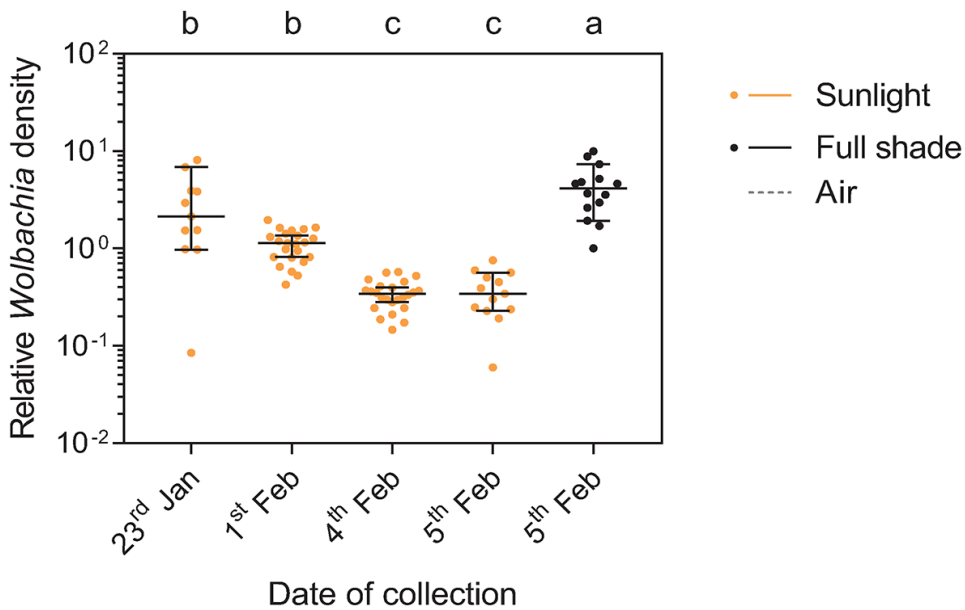
Factors affecting CI level

Environmental factors



Wolbachia density changes with temperature regimes in *w*Mel infected *Aedes aegypti*

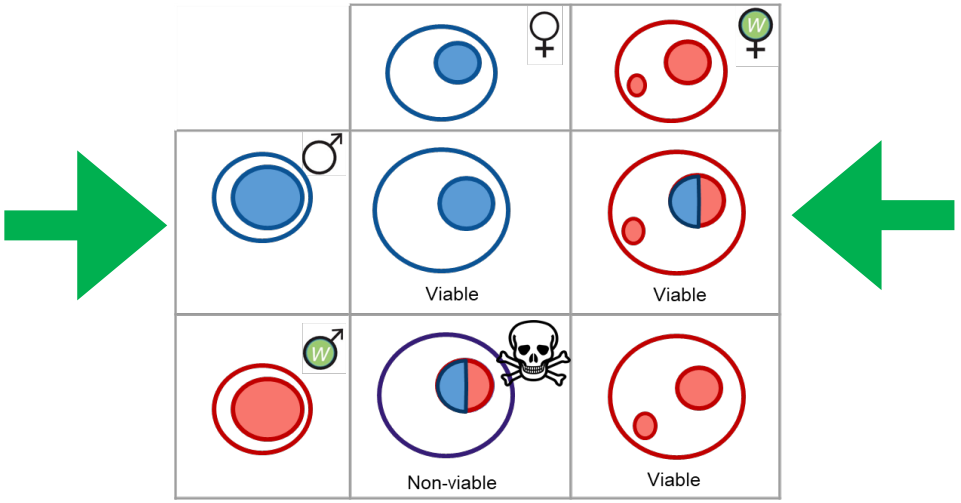




Ross et al, 2020

Factors affecting CI level

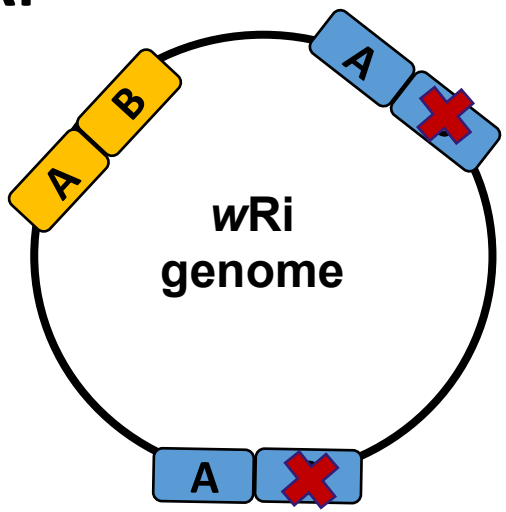
Environmental factors



Wolbachia genotype

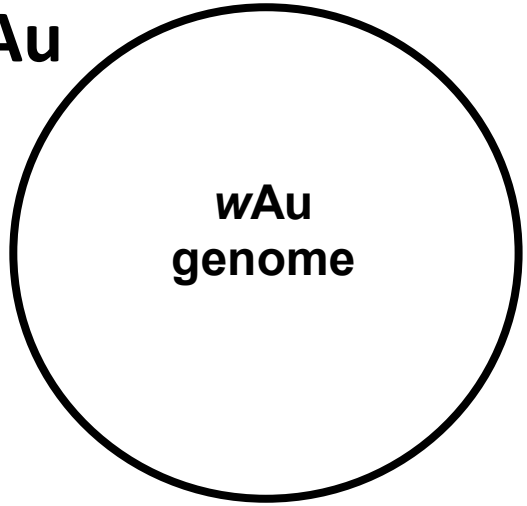
Differential CI induction phenotypes in two *Wolbachia* strains in *D. simulans: w*Ri *vs w*Au

***w*Ri**



90-100% of embryo mortality

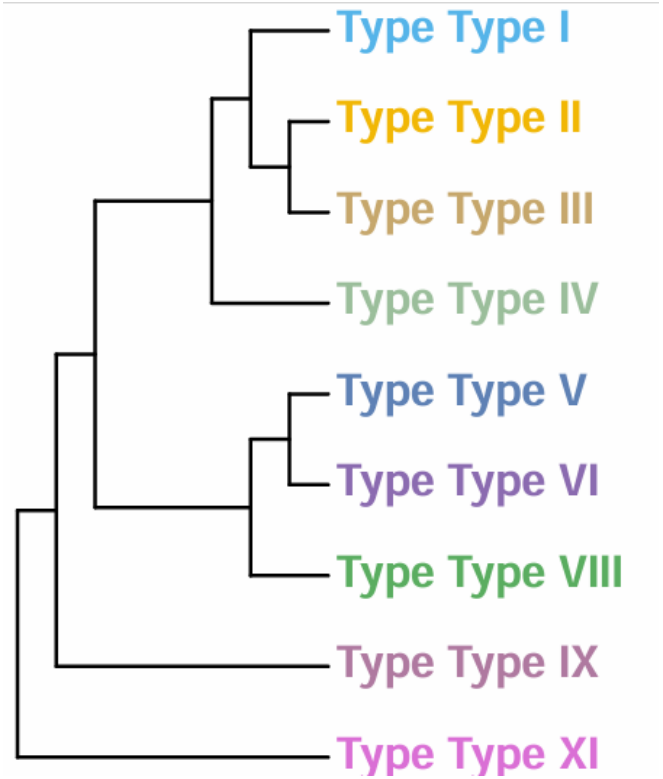
***w*Au**



Absence of embryo mortality

Type I *cif* gene

Type II *cif* gene

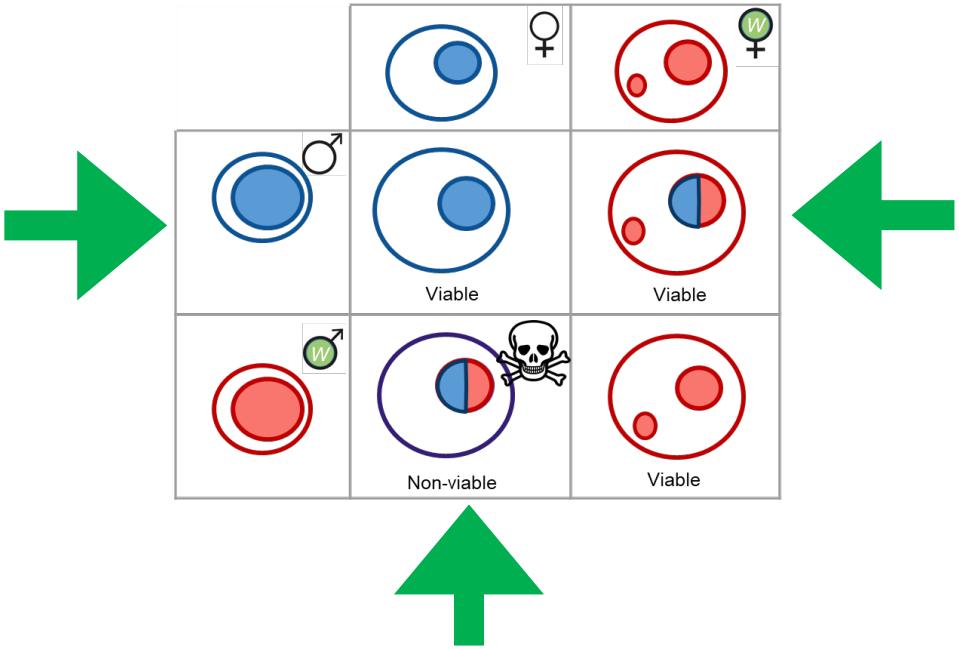


Tan et al, 2024;
Amoros et al, 2025

Turelli and Hoffmann, 1995
Hoffmann et al, 1996

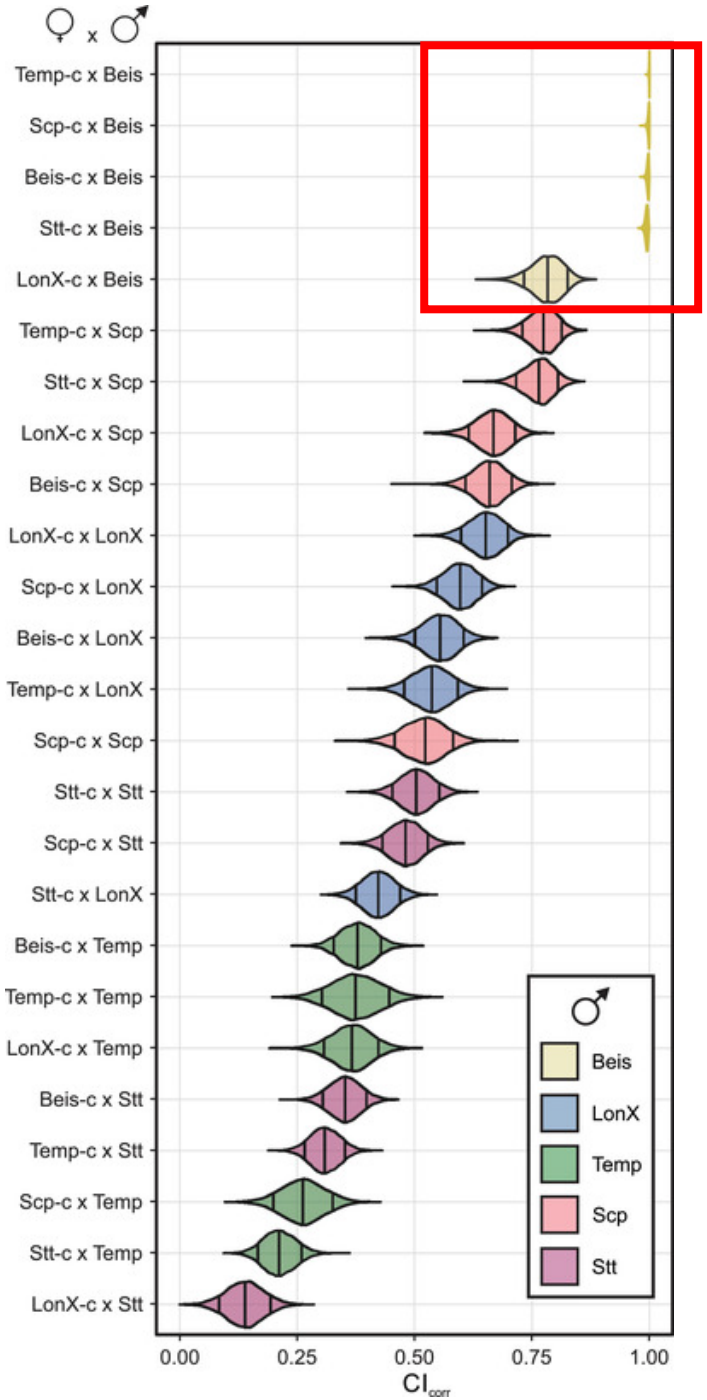
Factors affecting CI level

Environmental factors



Wolbachia genotype

Host genotype

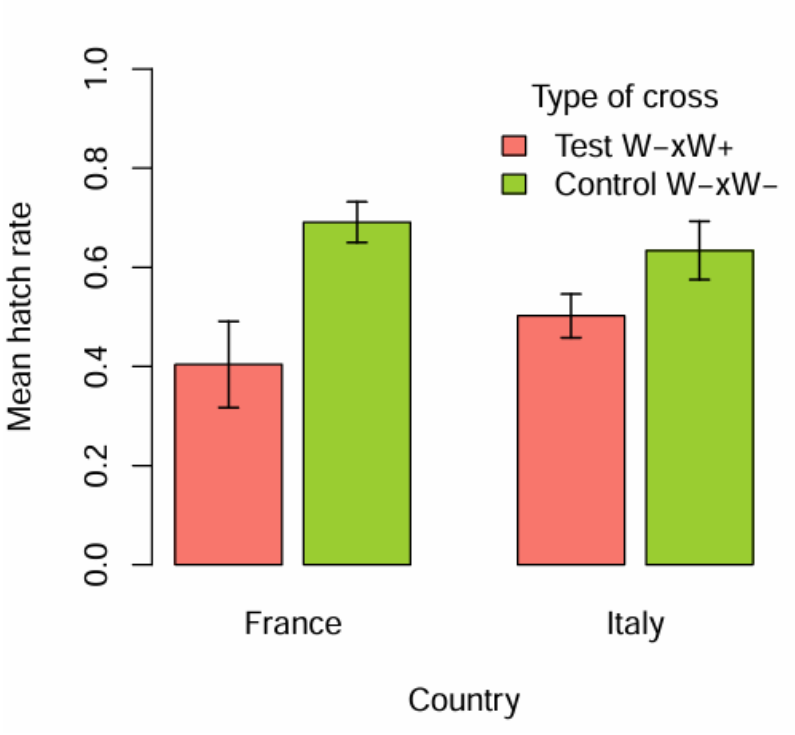


Host-modifier systems control CI strength in a *Tetranychus* spider mite

Nuclear modifier systems of CI that are expressed in males and interact with the female genotype.

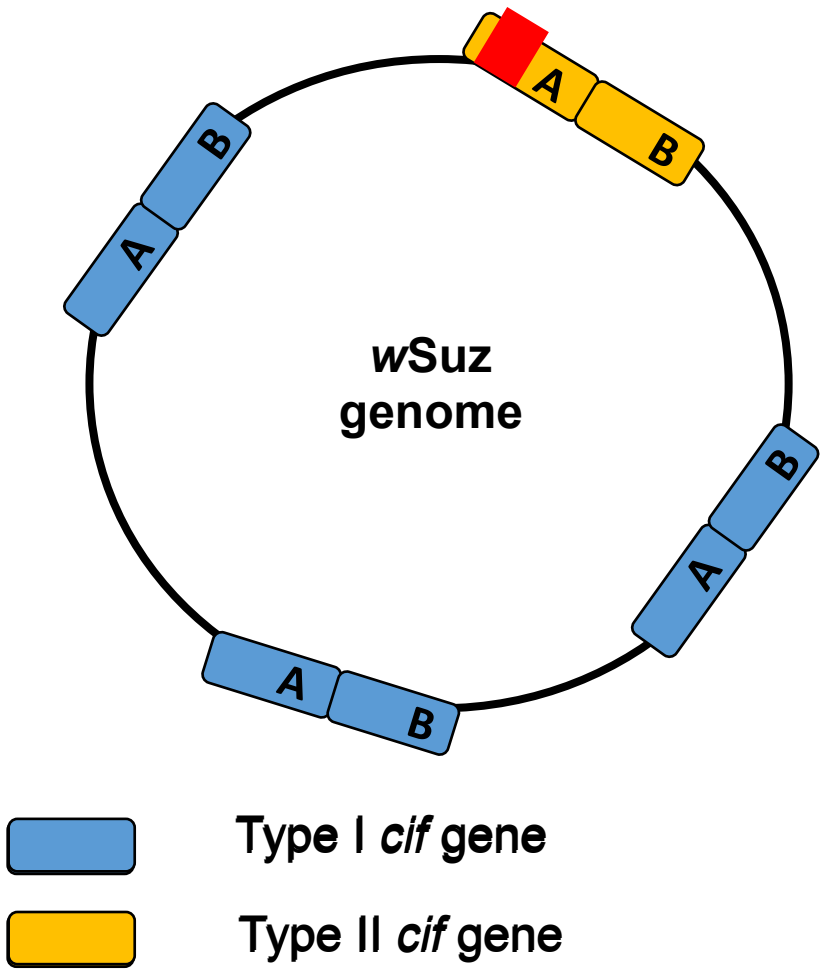
(Wybouw, Mortier, and Bonte 2022)

Drosophila suzukii or spotted-wing drosophila



Cattel et al., 2016

The puzzle of *w*Suz: presence of *cif* genes but no CI



3 copies of Type I *cifA* and *cifB* genes

1 copy of Type II *cifA* and *cifB* genes (IS element insertion at position 323 bp of *cifA*)

Conner et al, 2017

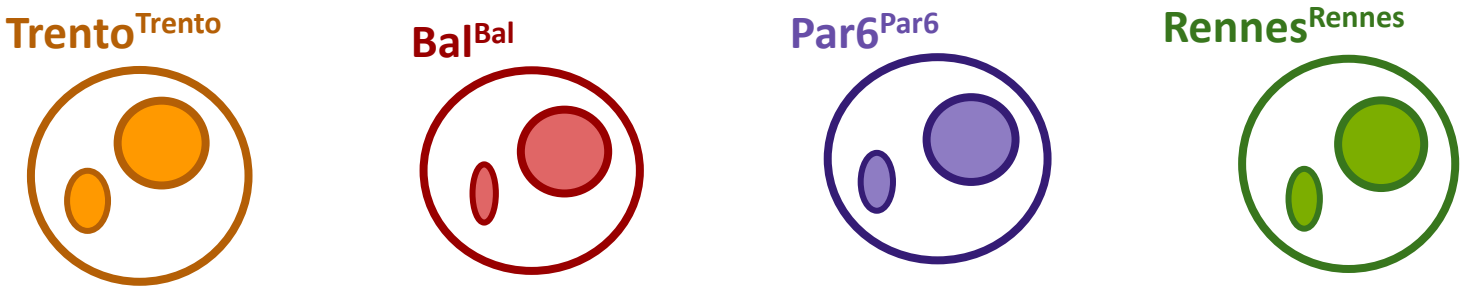
Research questions

Research questions

- How variable is cytoplasmic incompatibility across males from different lines of *D. suzukii*?

- What is the relative role of *Wolbachia* and host genotypes in this variation?

Cytoplasmic incompatibility assay



	♀	♀ (w)
♂	**Control cross 1** **W- × W-** Normal development	**Control cross 2** **W+ × W-** Normal development
♂ (w)	**Test cross** **W- × W+** Embryonic lethality	**Control cross 3** **W+ × W+** Normal development

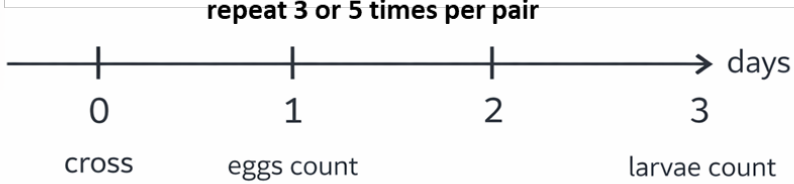
Collection of males and virgin females

Cross: 5 day-old females x up to 2-3 day-old males

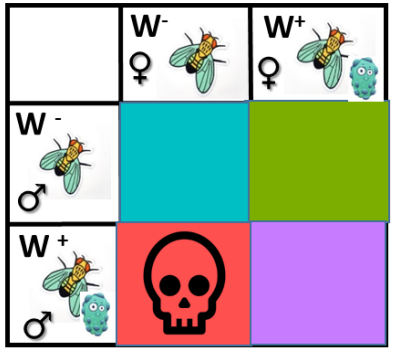
13-76 pairs per type of cross

Hatch rate estimation for each pair separately

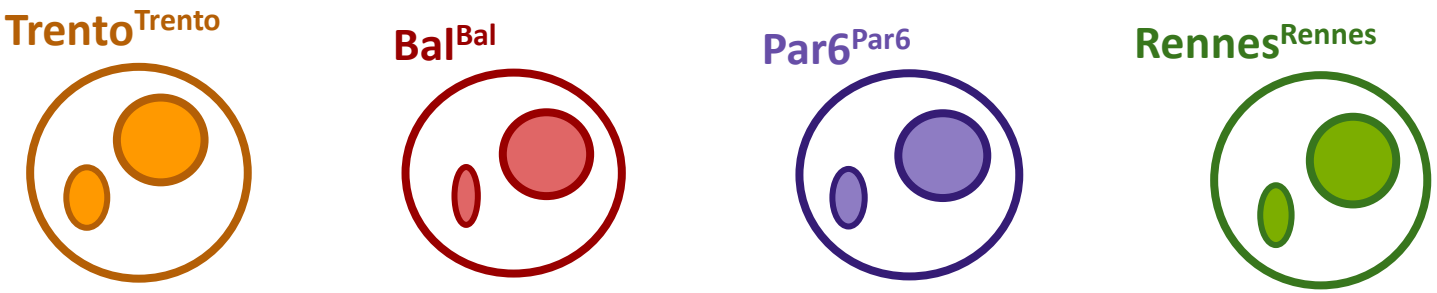
repeat 3 or 5 times per pair

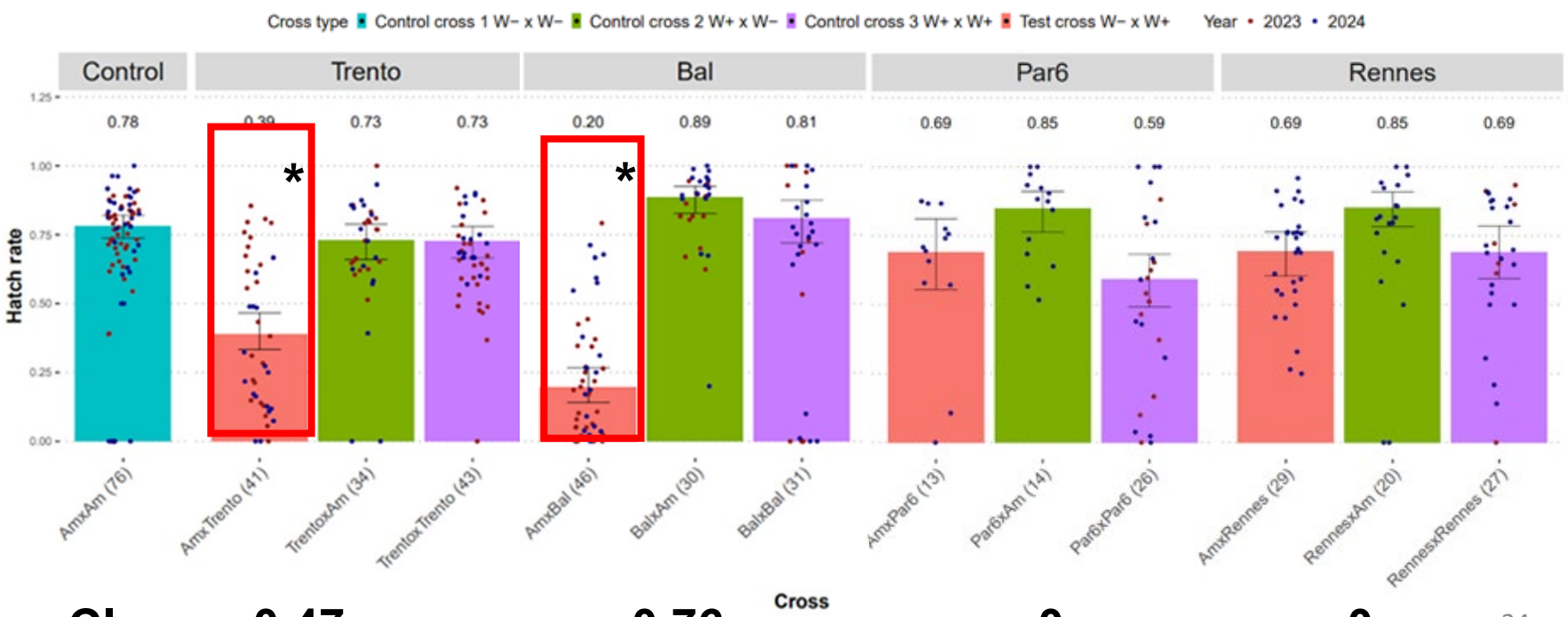


Hatch rate = eggs count/ larvae count



Cytoplasmic incompatibility in *D.suzukii*

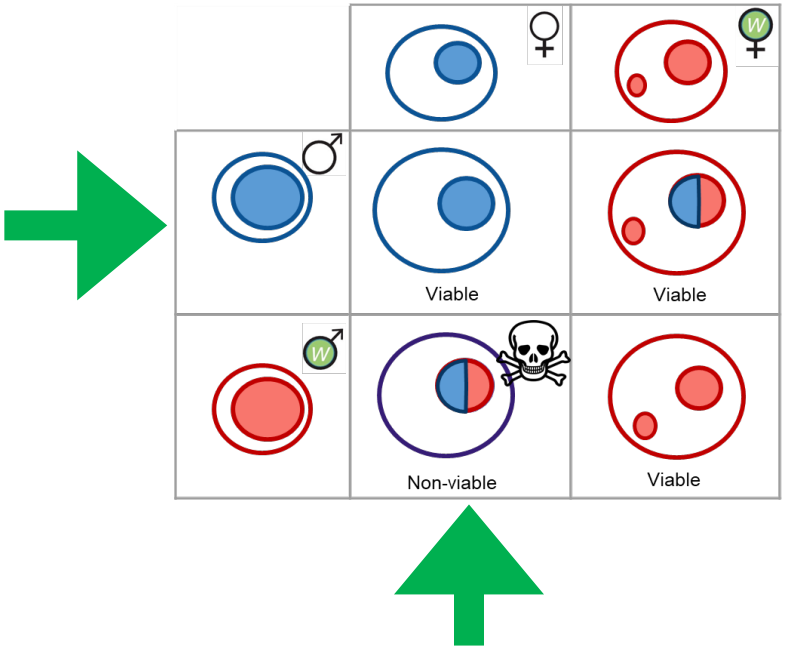




CI	**0.47**	**0.76**	**0**	**0**

Role of the different factors in this variation in CI?

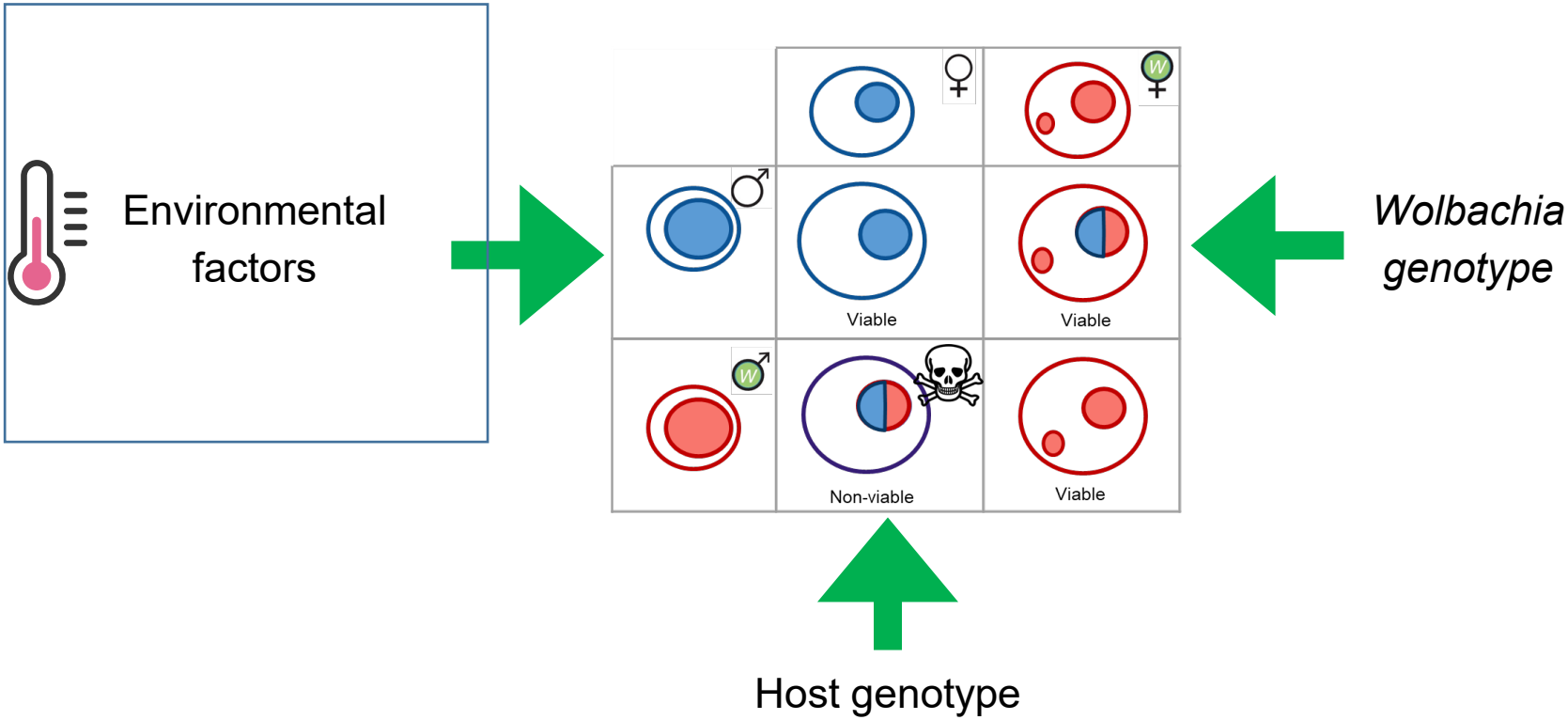
Environmental factors



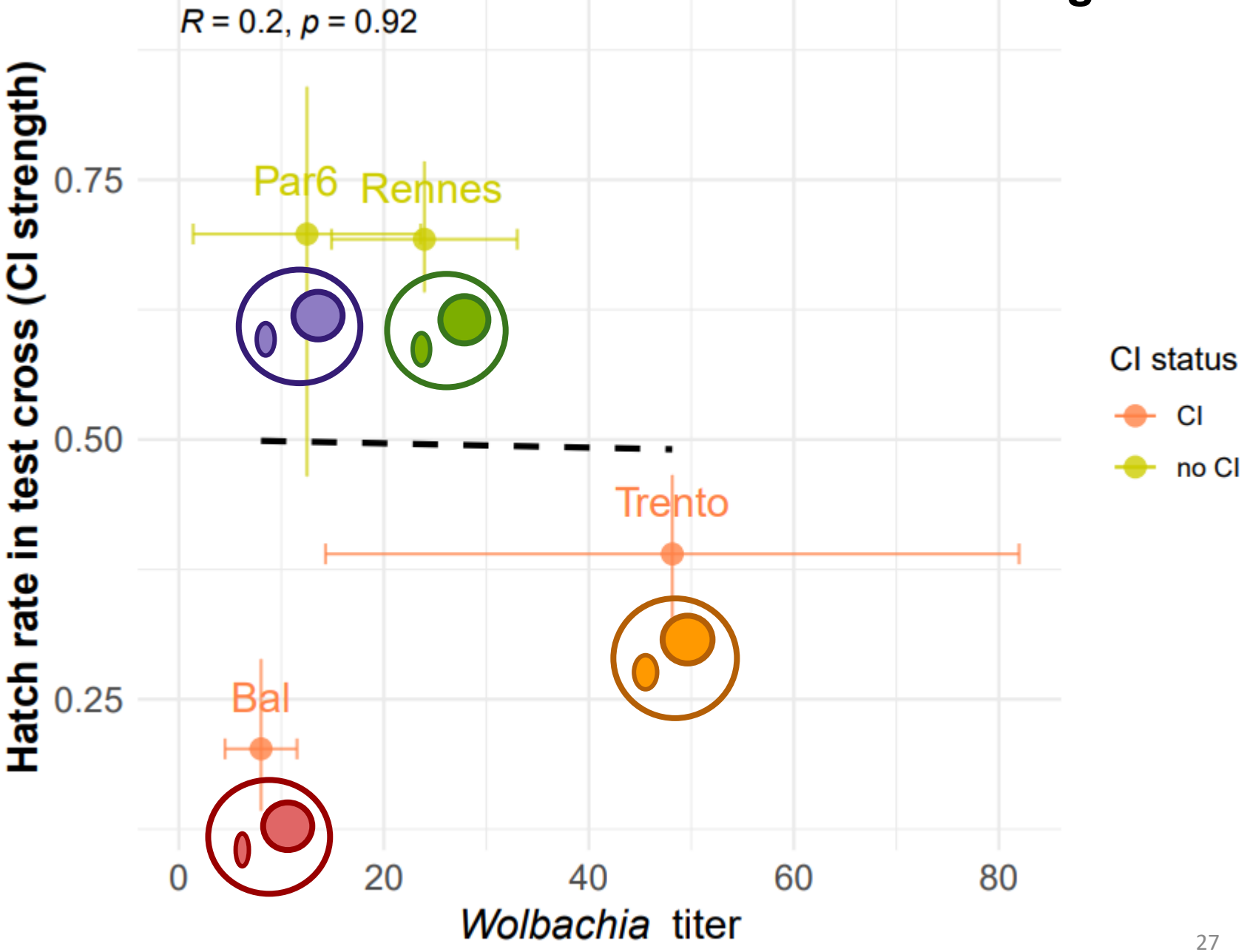
Wolbachia genotype

Host genotype

Role of the different factors in this variation in CI?

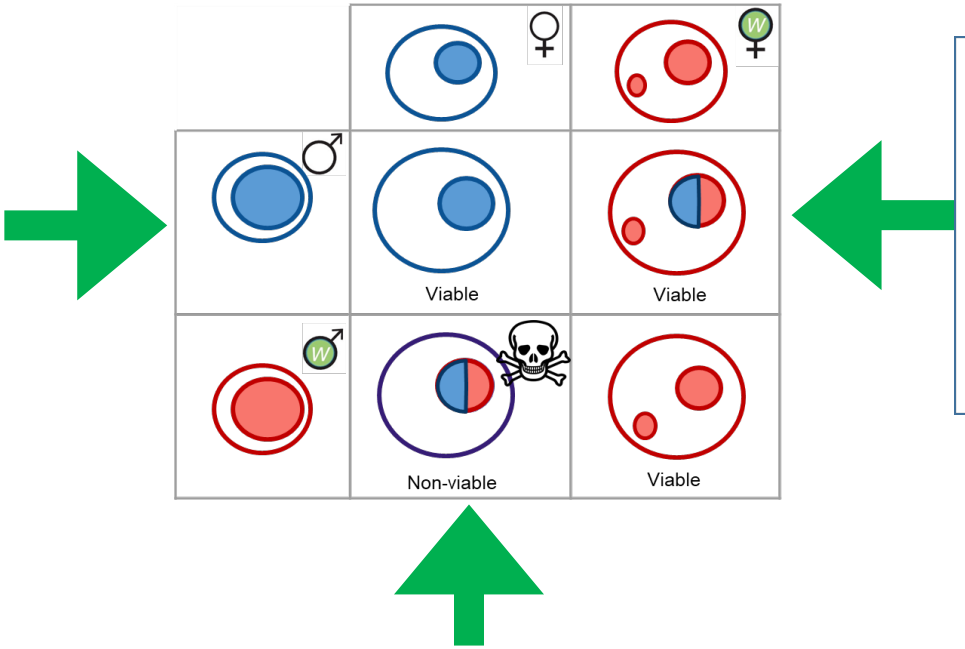


Correlation between *Wolbachia* titers and CI strength?



Role of the different factors in this variation in CI?

Environmental factors



Wolbachia genotype

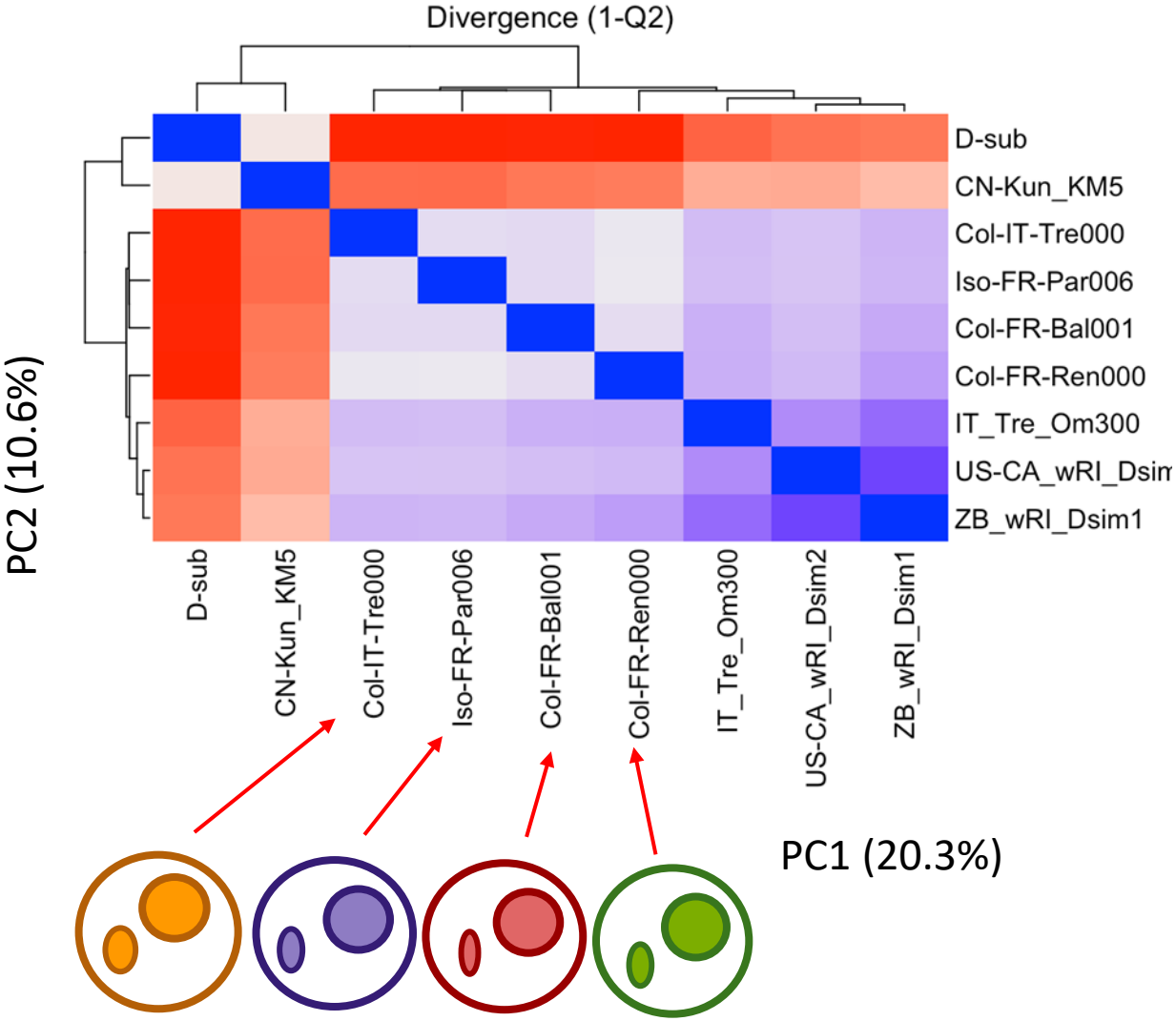
Host genotype

Genetic differences among *Wolbachia* isolates?

Illumina NovaSeq
25 pooled pairs of ovaries
from each strain,

Mapping to *w*Ri genome

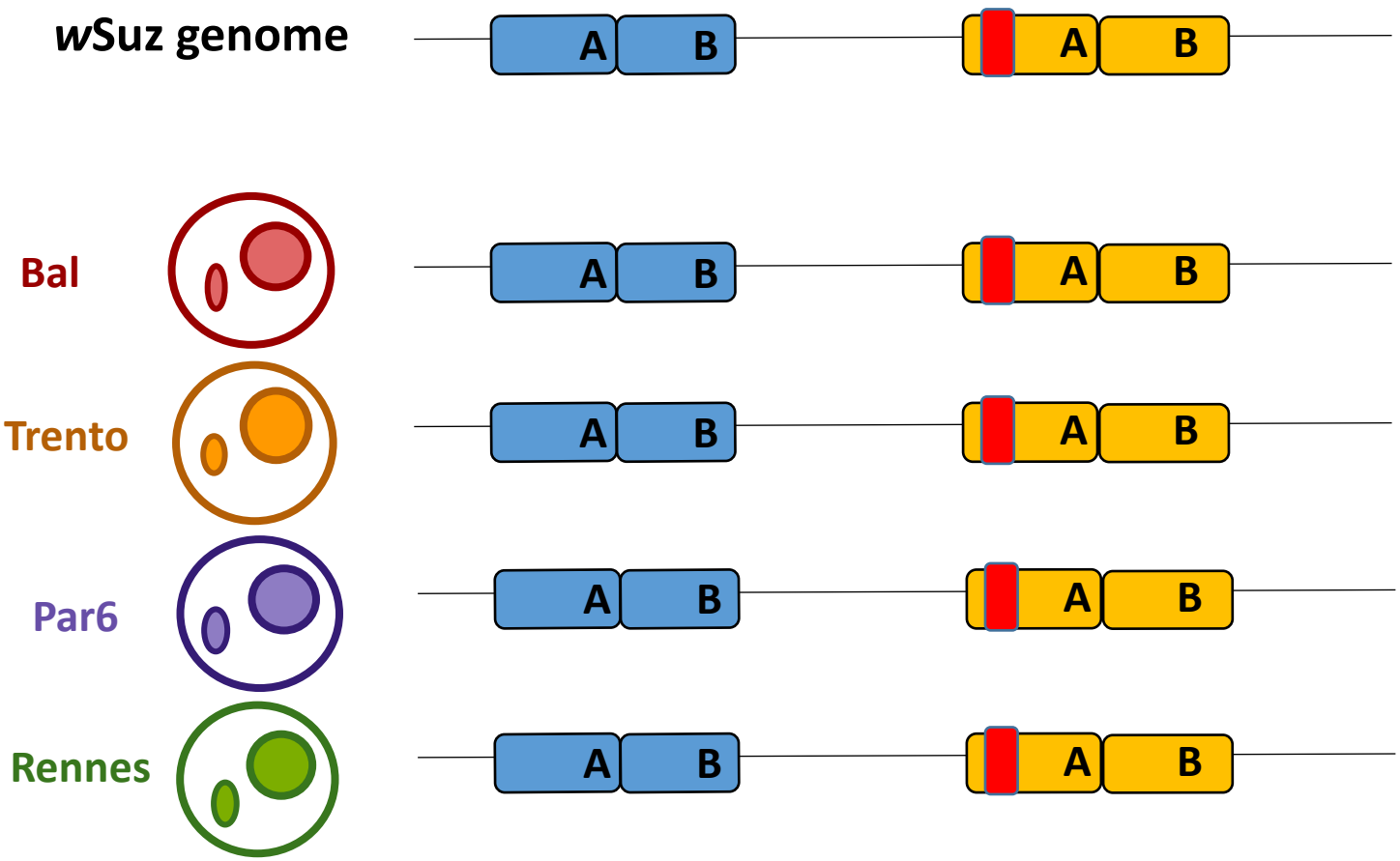
Genetic differences among *Wolbachia* isolates?



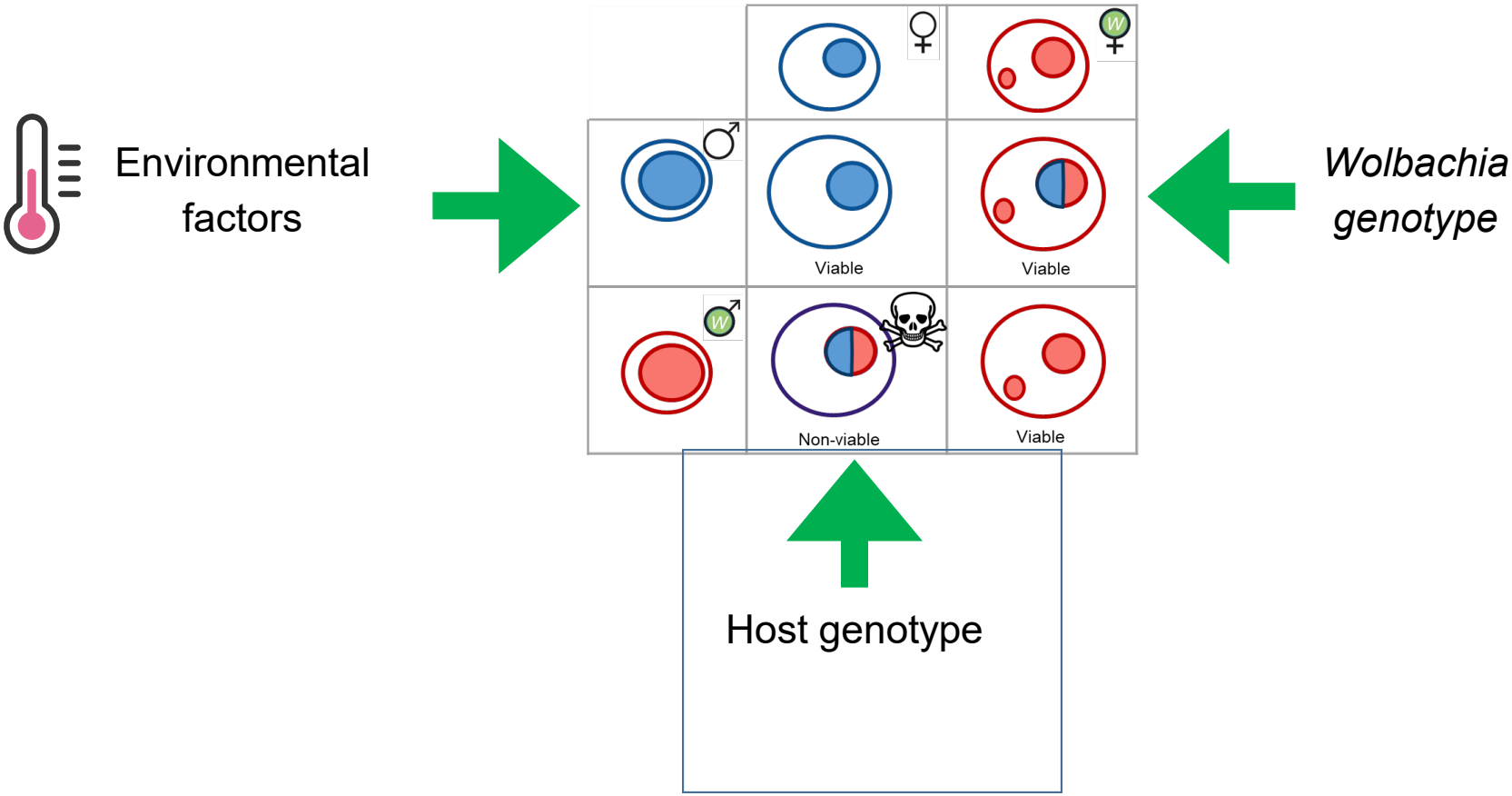
Illumina NovaSeq
25 pooled pairs of ovaries from each strain,

Mapping to *w*Ri genome

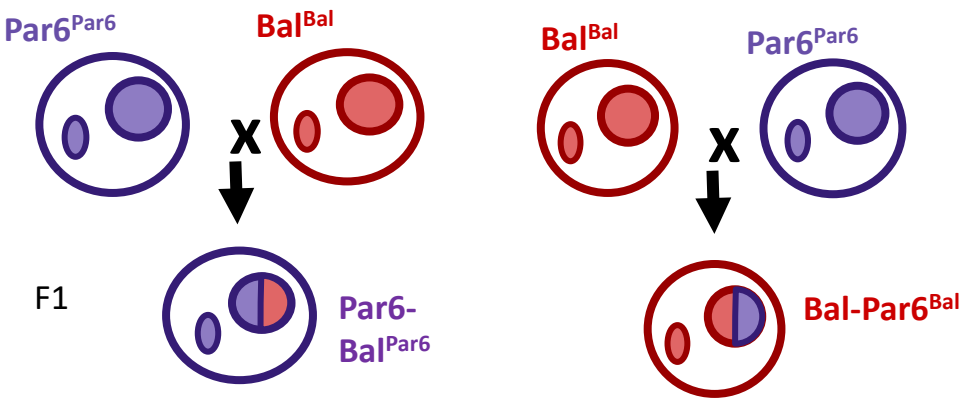
Variation in *cif* genes in genomes of *Wolbachia* isolates?

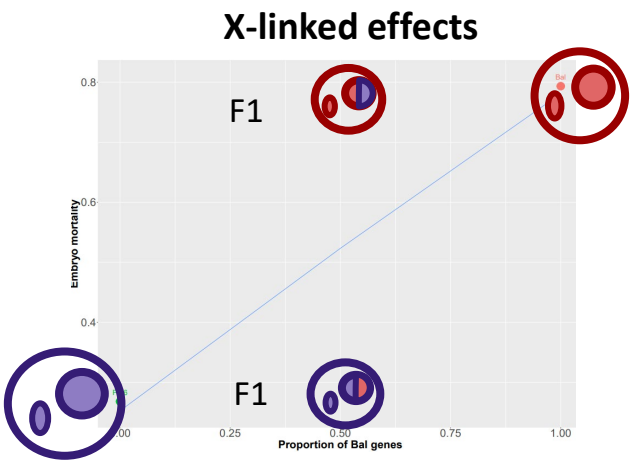


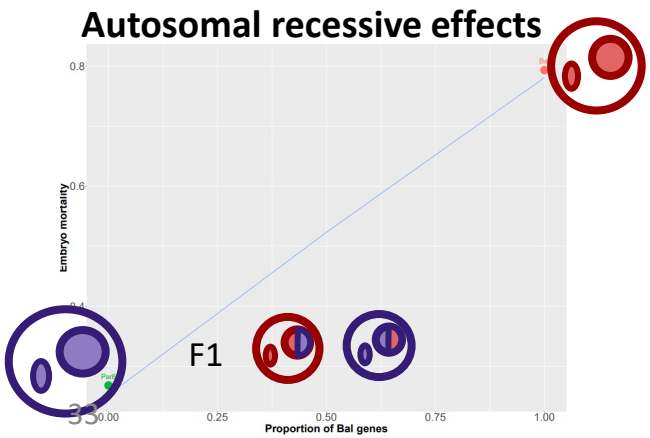
Role of the different factors in this variation in CI?



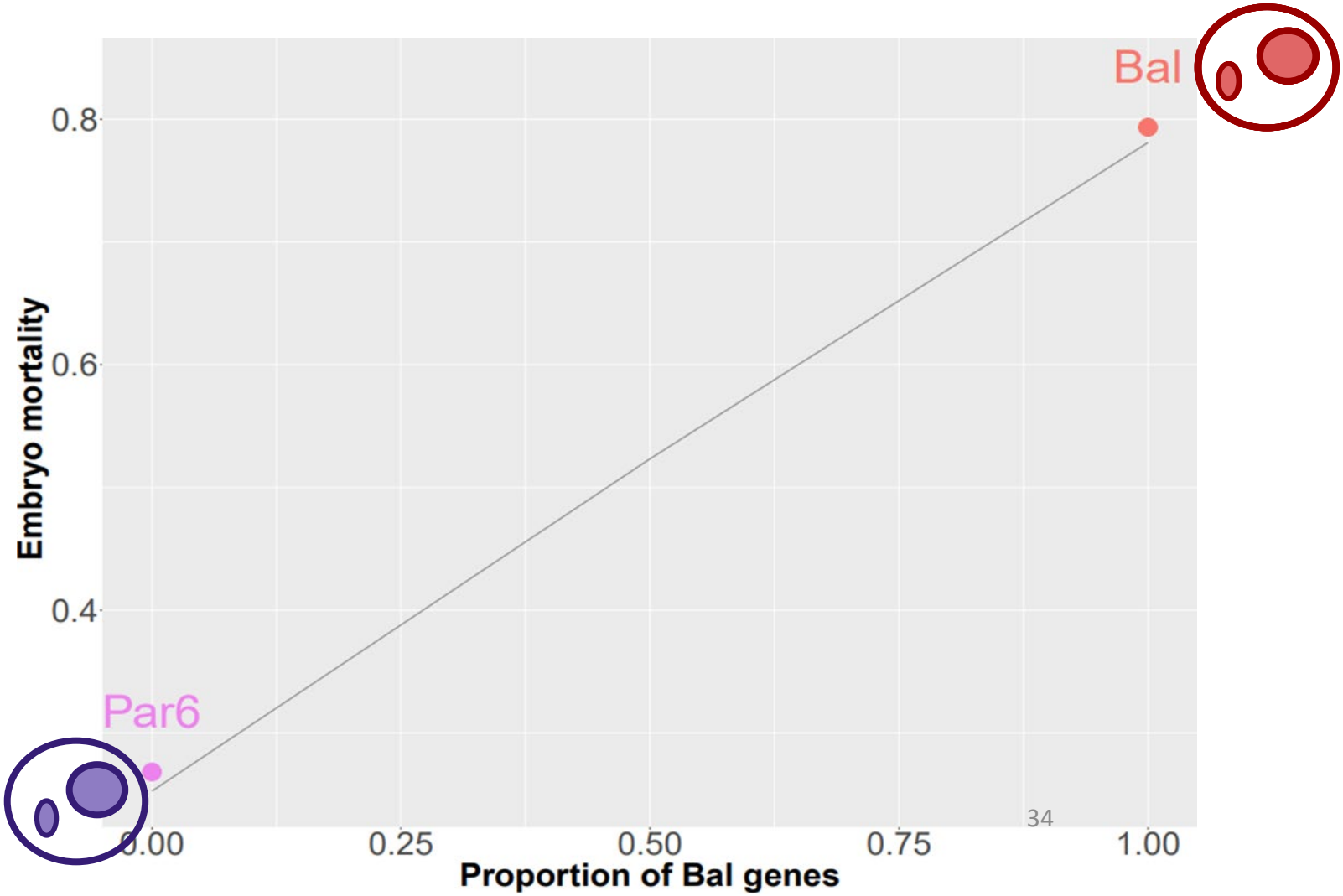
Role of male host genetic background in CI induction?



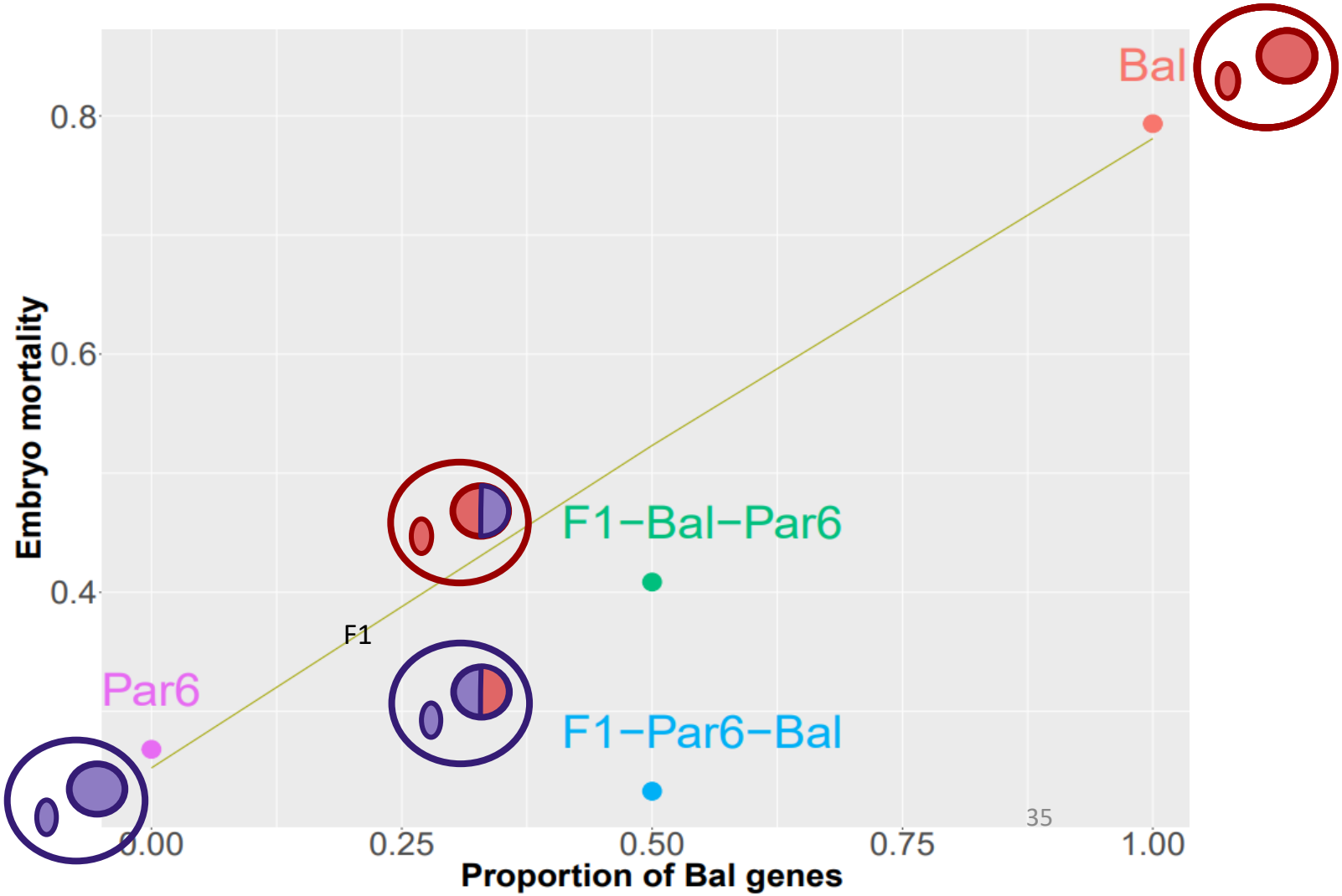




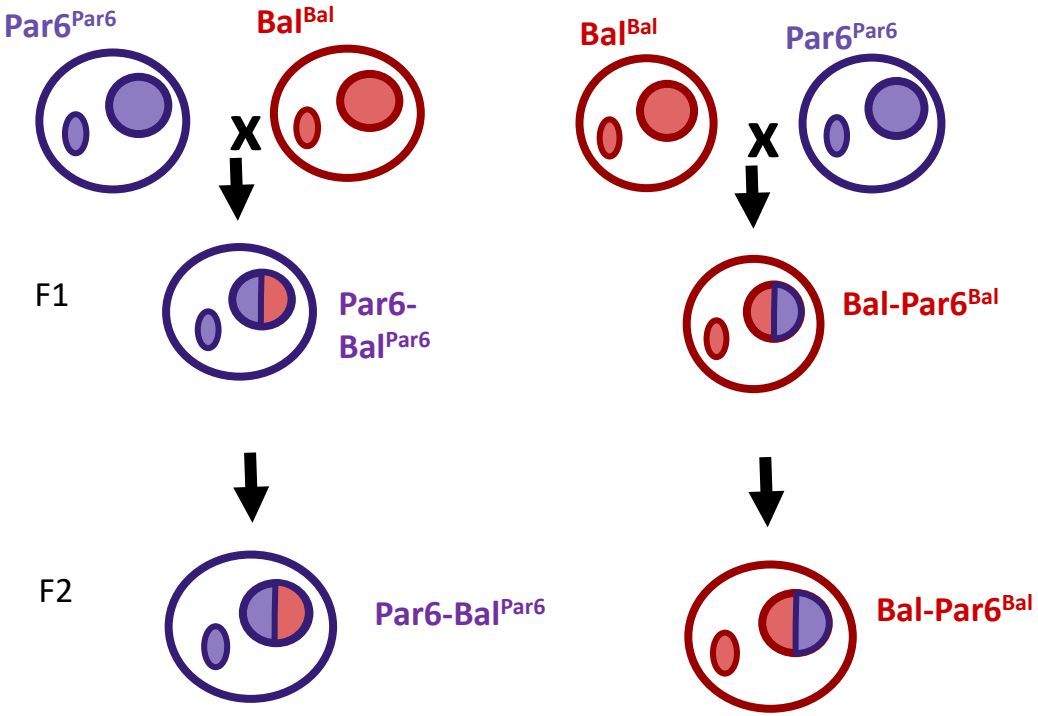
Results

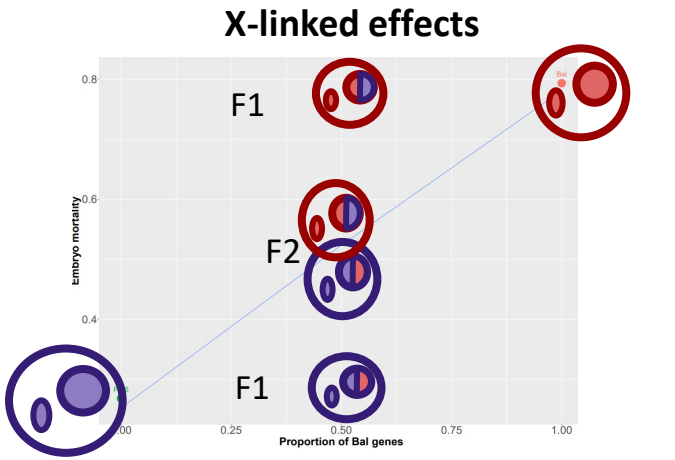

Bal
Par6
0.8
0.6
0.4
Embryo mortality
0.00
0.25
0.50
0.75
1.00
Proportion of Bal genes

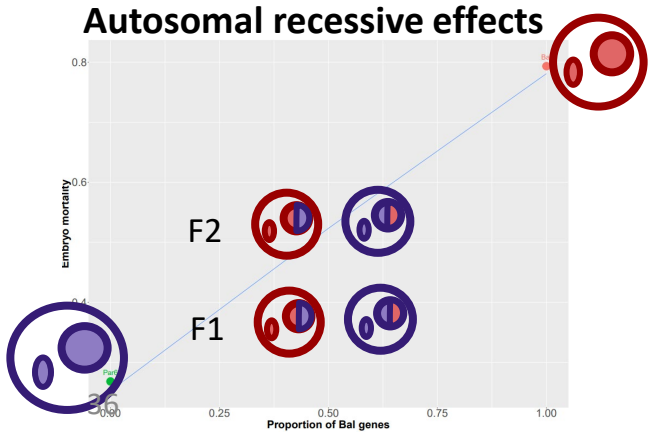
Results


Bal
F1−Bal−Par6
F1
Par6
F1−Par6−Bal
0.8
0.6
0.4
Embryo mortality
0.00
0.25
0.50
0.75
1.00
Proportion of Bal genes

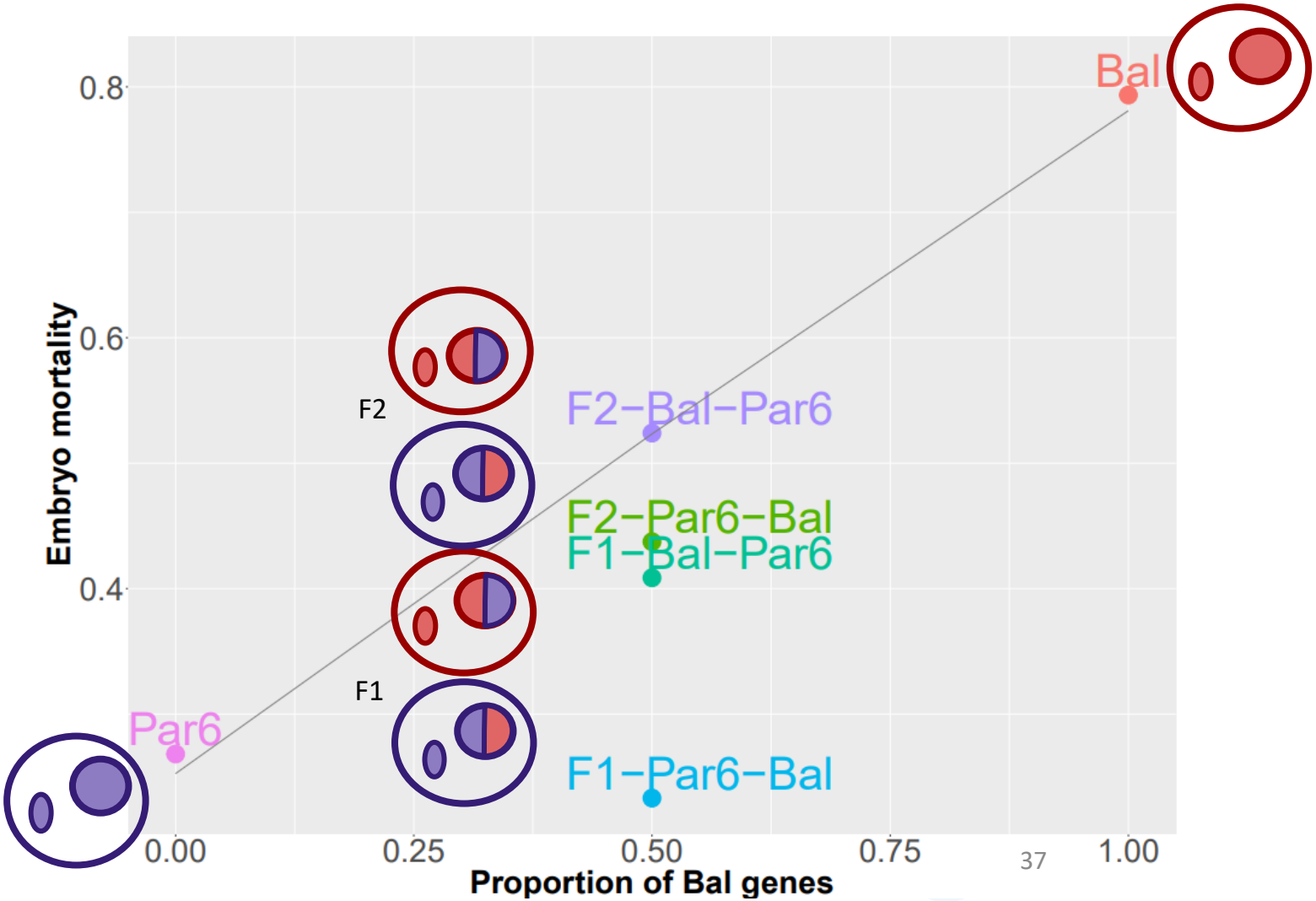
Role of male host genetic background in CI induction?







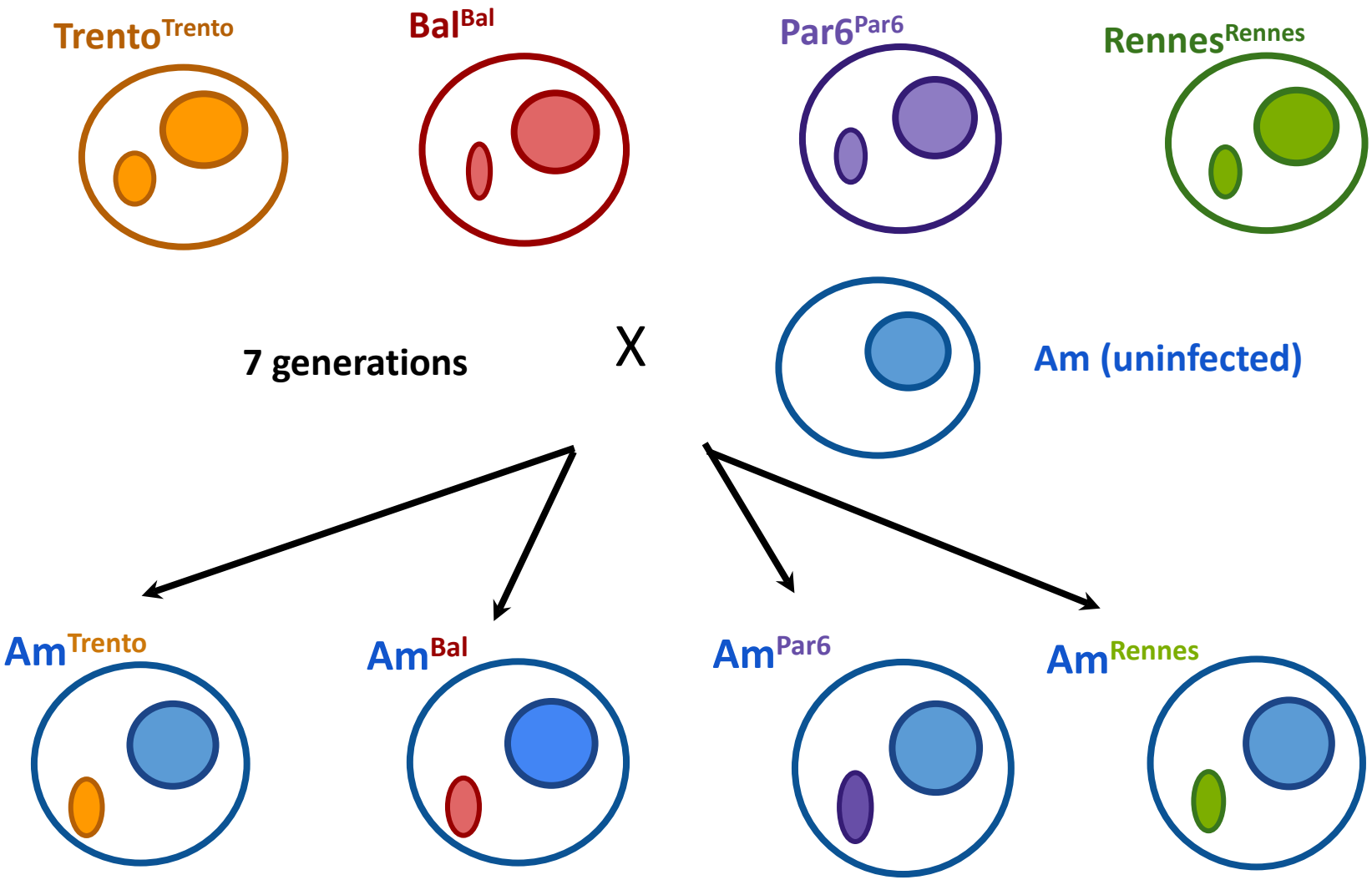
Results


Bal
F2
F2−Bal−Par6
F2−Par6−Bal
F1−Bal−Par6
F1
Par6
F1−Par6−Bal
0.8
0.6
0.4
Embryo mortality
0.00
0.25
0.50
0.75
1.00
Proportion of Bal genes

Genetic architecture of host genetics determination of *Wolbachia*-induced male sterility?

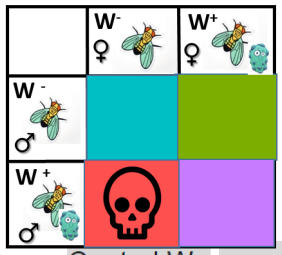
Model	Binomial part of the model	AICc
m5	Additive autosomal effect+Dominance autosomal effect	617.042936
m7	X effect+Additive autosomal effect+Dominance autosomal effect	618.23833
m6	Cytoplasmic effect+Additive autosomal effect+Dominance autosomal effect	618.622225
m2	Additive autosomal effect	619.519412
m4	Cytoplasmic effect+Additive autosomal effect	620.987928
m3	X effect	623.09379
m1	Cytoplasmic effect	623.228921
m0	Generation x cross direction	623.395526

Wolbachia isolate (cytoplasmic) effects on CI?



CI assay

Are there any effects of *Wolbachia* isolate (cytoplasmic effect) on CI?



Year • 2023 • 2024 Cross type • Control cross 1 W− x W− • Test cross W− x W+ • Control cross 2 W+ x W− • Control cross 3 W+ x W+

Natural lines

Control W−: Am/Am (0.78)

Italy: Am/Trento (0.39), TrentoxAm (34) (0.73), TrentoxTrento (43) (0.73)

France: Am/Bal (0.20), AmxBal (46) (0.89), BalxAm (30) (0.81), Am/Par6 (0.70), AmxPar6 (13) (0.87), Par6xAm (14) (0.64), Par6xPar6 (26), Am/Rennes (0.69), AmxRennes (29) (0.85), RennesxAm (20) (0.69), RennesxRennes (27)

Hatch rate

Introgressed lines

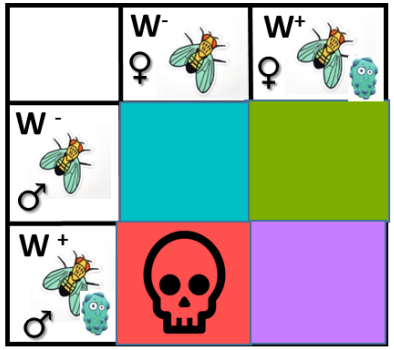
Am^{Trento}: 0.75, 0.67, 0.83, 0.70

Am^{Bal}: 0.71, 0.76, 0.66

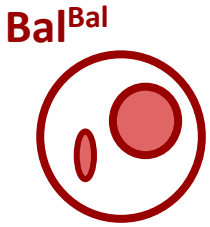
Am^{Par6}: 0.80, 0.84, 0.79

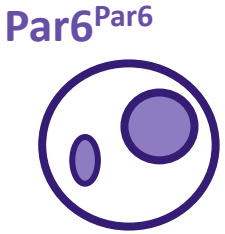
Am^{Rennes}: 0.77, 0.81, 0.82

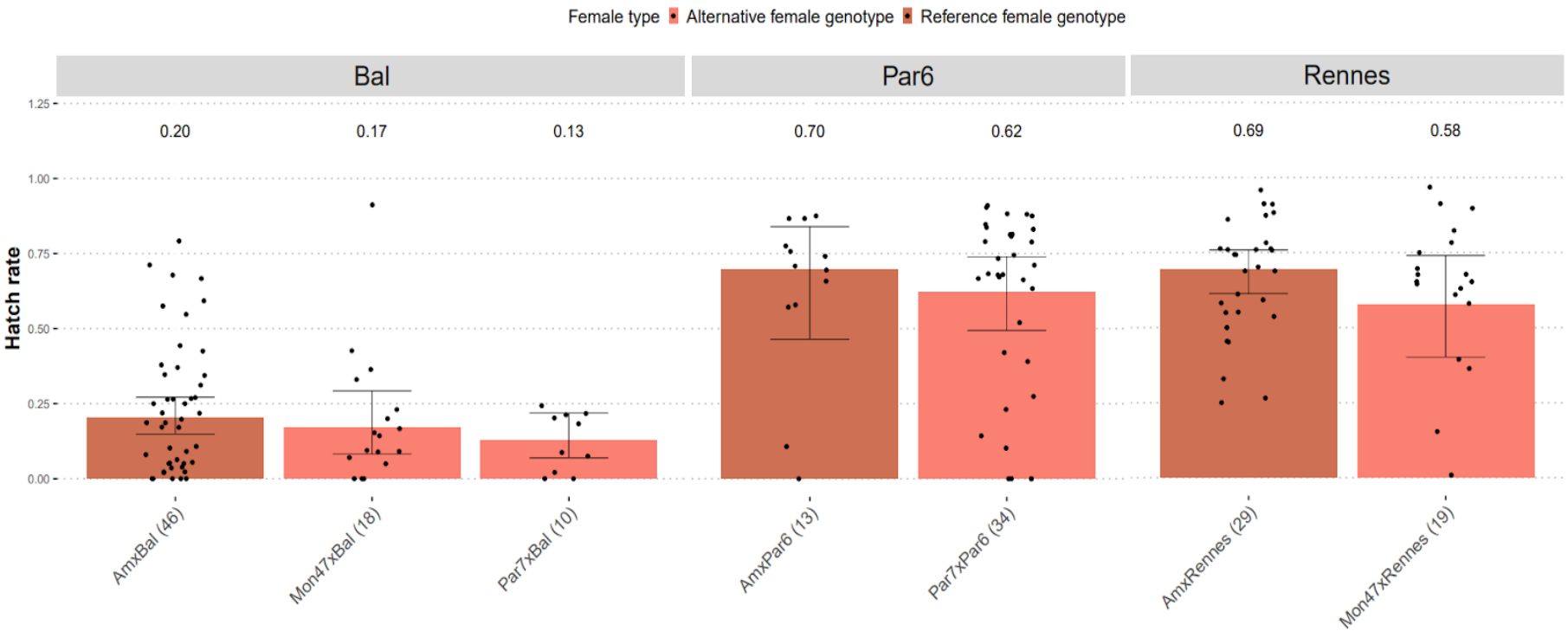
AmxAm (76), Amx Am^{Trento} (21), Am^{Trento} xAm (21), Am^{Trento} x Am^{Trento} (25), Amx Am^{Bal} (20), Am^{Bal} xAm (20), Am^{Bal} x Am^{Bal} (19), Amx Am^{Par6} (20), Am^{Par6} xAm (19), Am^{Par6} x Am^{Par6} (16), Amx Am^{Rennes} (21), Am^{Rennes} xAm (21), Am^{Rennes} x Am^{Rennes} (23)



Role of female host genetic background in CI induction?







Conclusions

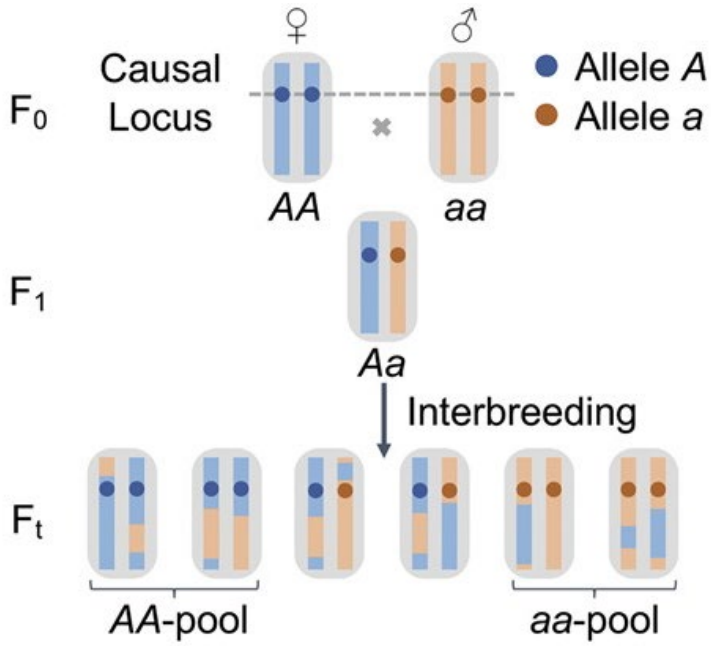
- High male genetic variation for *Wolbachia*-induced cytoplasmic incompatibility
- No *Wolbachia* genetic differences or variation in *cif* genes in strains with different CI levels
- Genetic architecture includes additive and recessive effects of Bal autosomal genes with possible contribution of X linked gene(s)

Perspectives

Identify the loci responsible for susceptibility to *Wolbachia*-induced CI

- Recombinant inbred lines between susceptible (e.g. Bal) and resistant (e.g. L6) genotypes
- Bulked segregant analysis (BSA)

“susceptible” genotype **“resistant” genotype**



High CI **No CI**

Perspectives

Identify the loci responsible for susceptibility to *Wolbachia*-induced CI

- Use Drosophila Phenotyping Panel (DPP) which contain 182 *Drosophila melanogaster* lines from nine natural populations
- Measure cytoplasmic incompatibility
- Perform Genome-wide association study (GWAS)

Acknowledgments

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