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SÉMINAIRE

On the genetic architecture of Cytoplasmic Incompatibility: inference from phenotypic data

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In the absence of a clear understanding of its genetic basis, Cytoplasmic Incompatibility (CI) has so far been conceptualized using a simple toxin-antitoxin model. Under this model, a symbiont's 3compatibility type2 is determined by the specific interaction between two components: a modification factor (mod, expressed in sperm) and a rescue factor (resc, expressed in the eggs). Here we confront this model to a well studied, complex and puzzling CI study system: the mosquito Culex pipiens. We show that a more elaborate model is required to account for the observed pattern, including multiple mod and resc genes, and possibly quantitative variation in gene products. We develop such a model and fit its parameters to the data using a parsimony approach. We thereby produce explicit predictions with regards to the genetic architecture of CI; namely, we infer that at least five mod / resc pairs are required to explain the data. These predictions provide a starting point for future genetic and genomic analyses of CI and will hopefully contribute to the decryption of its molecular basis.

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