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Does strong resistance to phosphine (PH_3) incur biological cost in *Cryptolestes ferrugineus* (Stephens): A two-way approach in dissecting the fitness cost?

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ABSTRACT

The rusty grain beetle, *Cryptolestes ferrugineus* (Stephens), is a cosmopolitan pest of stored grains that has developed very high levels of resistance to fumigant, phosphine (PH_3) (up to 1200×). Despite new fumigation protocols to manage *C. ferrugineus*, information on the development and spread of resistance remains unexplored. In particular, it is not known whether there is a fitness cost associated with phosphine resistance in *C. ferrugineus*. In this study, we adopted two complementary approaches to detect fitness cost directly linked to phosphine resistance in *C. ferrugineus*. These included (i) investigating inherent differences in developmental and reproductive traits in strains having isogenic background except for phosphine resistance genes, and (ii) determining the change in resistance allele frequency in populations segregating for phosphine resistance using gene specific DNA markers for one of the two genes needed for strong resistance (*cf_rph2*) at discrete generations. In both of these approaches, there was no selection pressure (i.e., phosphine fumigation) applied to experimental insects because selection could favour resistance traits.

Developmental traits showed no significant difference between the strain in which the phosphine resistance genes were introgressed into a susceptible genetic background, and the phosphine susceptible strain itself. The introgressed resistant strain showed a marginal delay of 3.6% in time to 50% emergence but the two strains produced similar numbers of progeny indicating at most a small fitness cost. Genotyping randomly selected individuals from the progeny of a genetic cross that were segregating for the resistance allele *cf_rph2* in four discrete filial generations (F_5, F_{10}, F_{15} and F_{20}) indicated a significant change in the proportion of *cf_rph2* genotypes (*rr, rs, ss*), especially with increase in homozygous resistant genotypes (*rr*) from F_5 to F_{15}. However, this increase in resistant homozygotes (*rr*) was not significantly reflected in phenotype assays. Thus, both the approaches indicated that phosphine resistance caused at most a small fitness cost that could otherwise be exploited to minimise resistance development in the field.

Keywords: Phosphine, Resistance, Numbers of progeny, Allele frequency, Fitness