Evolutionary genetics of life-history traits in a haplodiploid mite

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Citation for published version (APA):
How do the results of this thesis contribute to understanding the role of natural selection in shaping the genetic architecture of life-history traits of *Tetranychus urticae*? Natural selection favors individuals that mature the fastest, produce the most offspring and live the longest. Yet, there is relatively high genetic variance for some life-history traits of *T. urticae*. Mechanisms that may explain this variance involve the structure of a trait (*Selection vs. structure across traits*), genetic trade-offs and balancing selection (*The type of selection on individual traits: directional or balancing*) and gender (*Directional dominance variance and the strength of selection*).

In this thesis I focused on the oviposition rate as a trait, which in two independent studies (see Chapter 2 and 4) was found to harbor more genetic variation (additive and directional dominance variance) than most other examined life-history traits. A likely reason for its high directional dominance variance – and for similar findings in other haplodiploid species – is discussed in *Directional dominance variance and the strength of selection*. The high additive variance was more difficult to explain and is discussed in *The type of selection on individual traits: directional or balancing* and *Deleterious variation for oviposition rate and the strength of selection*.

**Selection vs. structure across traits**

A central assumption in quantitative genetics is that the importance of a trait for fitness determines the strength of selection acting on that trait and that this selection strength shapes the amount of additive variance. Thus, the higher the importance for fitness, the higher the selection strength will be and the lower the levels of additive variance – and consequently also the lower $h^2$ and $CV_A$. However, a process with a conflicting influence on the amount of additive variance has been found: traits that are further down the gene-to-fitness pathway (i.e., that have a higher integration level) compound more variance than traits with a lower integration level (see Chapter 1 p. 9). The integration level influences both the amount of additive and residual variance, but has a disproportionally large effect on the residual variance. This influence of the integration level on the degree of variance compounding of a trait is defined here as the influence of ‘structure’.

Life-history traits have a higher integration level than morphological traits and have been found to contain on average more additive variance than morphological
traits (see, for example, Figure 7.1). Although the amount of additive variance is on average higher for life-history traits, the narrow-sense heritability is usually lower, which is due to an even larger effect of structure on the residual variance [since $h^2 = V_A / (V_A + V_R)$] (e.g., Houle 1992; Kruuk et al. 2000; Merilä and Sheldon 2000; McCleery et al. 2004; Coltman et al. 2005). Thus, structure appears to shape the variance patterns across life-history and morphological traits. In contrast, when examining behavioural traits, both structure and selection appeared to have left their signature (see the meta-analysis by Stirling et al. 2002).

Do these results imply that selection plays merely a minor role in shaping the genetic architecture of life-history traits? In Chapter 2, I argued that the relative importance of selection probably depends on the range of trait classes taken into account. Within the class of life-history traits a signature of selection may be more readily detected, since the range of integration differences between traits is narrower. Some exploratory work in a natural population of *T. urticae* showed a clearly positive relation of heritability to additive variance, whereas no relation to residual variance could be detected across the life-history traits (Chapter 2). This pattern is in agreement with a significant influence of selection on the heritability of these traits of the population under study. Teplitsky et al. (2009) found an influence of both structure and selection across life-history and behavioural traits, in a wild population of red-billed gull. But concerning other studies (e.g., Kruuk et al. 2000; Merilä and Sheldon 2000; McCleery et al. 2004), perhaps the lack of a signature of selection over life-history and morphological traits can be explained by an overwhelming influence of structural differences between the two classes of traits. Therefore, I re-examined the data of three studies that have been central in this debate: the red deer data of Kruuk et al. (2000), the flycatcher data of Merilä and Sheldon (2000) and the great tit data of McCleery et al. (2004). These studies contain accurate heritability estimates of a wide range of life-history traits of animals in the wild. Figure 7.1 shows the relationship of heritability to $CV_A$ as well as to $CV_R$ across life-history or morphological traits per study. The following pattern can be seen. First, the amount of additive and residual variance is markedly higher for the life-history traits (black dots) than for the morphological traits (grey dots). This confirms that the level of variance compounding is highly different between these two classes of traits. One can subsequently compare the relation of heritability to the two $CV$s across all traits (grey line) with that across the life-history traits only (black line). The relation of heritability to residual variance does not change dramatically: heritability is always negatively correlated with residual variance. This implies that the process that involves the structure of a trait remains important, whatever the range of trait classes taken into account. The relation of heritability to additive variance, however, changes sign in all three datasets: across morphological and life-history traits, heritability has a
negative relation to additive variance, but this changes to a positive relationship when considering life-history traits only. Also, the degree of fit of the regression of $h^2$ and $CV_A$ increases markedly in all three cases, if only life-history traits are taken into account. For the data on great tits, the relations of heritability with additive vari-

**Figure 7.1** Scatterplots of $h^2$ against $CV_A$ and $CV_R$, for three studies (great tit data from McCleery et al. 2004, flycatcher data from Merilä and Sheldon 2000 and red deer data from Kruuk et al. 2000). Grey dots are the morphological traits, black dots are the life-history traits. Grey lines represent regressions across all traits, black lines across life-history traits only. The degree of fit of the linear regression across all data (‘All: $r^2$’) and across the life-history data (‘LH: $r^2$’) are shown in the top right corner of each panel. Measures of total fitness were not taken into account in this analysis.

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 ance and those with residual variance are even of similar strength. These results sug-
gest that the strength of selection may be an important co-factor in shaping the heri-
tability within the class of life-history traits.

The type of selection on individual traits: directional or balancing
A next question is whether a signature of directional selection can be found for indi-
vidual traits of *T. urticae*. Strong directional selection is expected to reduce additive
variance. In Chapter 2, however, the oviposition rate of *T. urticae* revealed an amount
of additive variance that can be interpreted as high for a life-history trait. In first
instance this does not seem to comply with an important role for selection. But if there
are genetic relationships with other fitness trait, one may ask whether overall selec-
tion is perhaps balancing instead of directional. Although individual life-history traits
are under directional selection, perhaps negative genetic correlations with other com-
ponents of fitness cause balancing selection on a trait to dominate. In Chapter 3 I
showed that another population of *T. urticae* could not evolve toward higher oviposi-
tion rates (within six generations of strong artificial selection), while the evolution
towards lower oviposition rates proceeded readily. Moreover, no evidence for genetic
trade-offs with other life-history traits was obtained. The conclusion for this popula-
tion was that the additive variance for oviposition rate is dominated by alleles with an
overall negative effect on fitness. This does not comply with an important role for bal-
ancing selection. Thus, selection has probably been directional and the genetic vari-
ability is caused by a balance between mutation (leading to input of deleterious vari-
ation) and directional selection (leading to removal of deleterious variation). This
implies that the effect of selection was not strong enough to reduce the additive dele-
terious variation to a lower level. This finding leads to two questions: (1) Why was
selection on oviposition rate not stronger? (2) What does this finding mean for the
selection working on oviposition rate in other populations? The first question is con-
sidered in the next two sections, while the latter is discussed here.

The additive variance (*h*² and *CV*_A*) for oviposition rate was found to be even
higher in another population of *T. urticae* (see Chapter 2). Can we exclude a domi-
nant impact of balancing selection and genetic trade-offs in this population too? Not
directly. In this thesis I have worked with two very different types of populations: one
was maintained in the laboratory for many years (in Chapter 3) and the other was col-
lected directly from a natural population (in Chapter 2, 4, 5 and 6). The population
that was examined in Chapter 3 was a long-established laboratory population for
which many environmental conditions (especially the abiotic conditions) were more
stable than in the wild. This means that the potential for balancing selection via envi-
ronmental fluctuations was smaller than in the natural population. Thus, we cannot
rule out balancing selection and genetic trade-offs in the natural population. The
additive variance in the natural population was higher, albeit not significantly different (see the standard errors of the $h^2$ estimates in Chapter 2 and 3). This could be explained by balancing selection playing an additional role in the natural population. However, we found no indication of genetic trade-offs for oviposition rate, since there were only positive genetic correlations with other life-history traits and web production (see Chapter 3) nor was there an indication of a genetic trade-off with ambulatory dispersal (see Chapter 5). Note, however, that the absence of genetic trade-offs with other life-history traits concerns the laboratory population and may differ for the natural population. Also, correlated responses to artificial selection do not necessarily arise if genetic trade-offs are present (Pease and Bull 1988; Charlesworth 1990). One relatively straightforward way to investigate an additional role of balancing selection in more natural environments would be to maintain subsets of the same population under different levels of environmental heterogeneity and examine the long-term effect on the level of additive variance.

**Discussion: Selection on life-history traits**

Why do populations of *T. urticae* contain so much additive variance for oviposition rate, i.e., a trait pivotal to the fitness of this species? Can this be explained by weak effects of selection? As found in Chapter 4, a large amount of directional dominance variance was also present for the oviposition rate of the natural population. Here, I first discuss the role of selection strength in explaining directional dominance variance and then, in the next section, extrapolate this information to examine potential mechanisms explaining the overall deleterious genetic variation for oviposition rate.

In Chapter 4, I investigated the directional dominance variance for various life-history traits in *T. urticae*. Theory suggests that the strength of selection on a recessive deleterious allele depends on the gender in which a gene is expressed. A recessive deleterious allele can hide in heterozygous females, which decreases the efficiency of purifying selection. This shelter from selection is not available to the hemizygous males. Thus, the efficiency of purifying selection, and consequently the amount of directional dominance variance, for a life-history trait may be related to the degree to which the trait is controlled by genes with female-limited expression. Oviposition rate is a female trait and thus likely controlled by many such genes. In agreement with this hypothesis, oviposition rate was found to be negatively affected by inbreeding, contrary to juvenile survival and longevity. Can this pattern of gender-related dominance variance also be detected in other studies on haplodiploid species? I performed a literature search for studies that determined the effect of inbreeding on life-history traits in haplodiploid species. The question to be answered was whether there is a difference in the impact of inbreeding between traits related to female fecundity and life-history traits found in both genders (generic traits), such
as maturity rate, survival rate and longevity. Only this contrast was taken into consideration because there are clear potential differences in degree of female-control between female and generic traits. Data were compared within studies, because experimental set-up (such as inbreeding regime) and population characteristics can modify the effects of inbreeding, thereby hampering comparison across studies. Literature was gathered via two routes. I used the literature list of Henter’s meta-analysis (Henter 2003) and for the more recent literature I searched through Web of Science for studies on inbreeding depression in haplodiploid species. Within this list of articles I used the following criteria to select relevant data:

- Only data on basic life-history components of survival, reproduction, longevity and development are accepted. Composite life-history traits that incorporate other life-history traits are ignored. For example, reproductive productivity is an often measured trait but combines fecundity and juvenile survival.
- Per experiment, at least one trait is related to female fecundity and at least one generic life-history trait is measured. (Note that no data on other purely female or male life-history traits were available in the examined studies.)
- Only experiments which measured traits in experimentally manipulated settings are accepted. I.e., only experiments that compared experimentally inbred lines to either experimentally outbred lines or to a base population.
- Only experiments that created the experimental lines by consecutive mating, followed by direct measurement are accepted. Data concerning long established inbred lines are ignored.
- Articles are only accepted when the index for inbreeding depression RP can be calculated (for the definition of RP see Chapter 4).
- Experiments using long established laboratory populations are ignored.
- Only experiments using populations without a (known) preference for inbreeding and without a (known) history of small population sizes are accepted.

Only four studies complied with this suite of conditions (see Table 7.1). The only suitable study before 2003 was by Hoy (1977). This study found no significant inbreeding depression in any trait, but had very small sample sizes (n = 9 per treatment), making statistical power low. Comparing the RP’s in this study (Hoy 1977), the effect of inbreeding was lowest on fecundity, which contradicts my hypothesis. With regard to the other studies (published in 2004-2009), three out of four showed significant inbreeding depression with regard to fecundity (Perrot-Minnot et al. 2004; Gerloff and Schmid-Hempel 2005 and this thesis). The fourth study (Bilde et al. 2005) found no significant effect in any life-history trait, but the only positive RP value (i.e., negative effect of inbreeding) was found for fecundity. Only our work in addition showed significant inbreeding depression in another life-history trait (which
An overview of literature concerning the effect of inbreeding across life-history traits of haplodiploid species. Experimentally inbred lines were either compared to the base population or to experimentally outbred lines. ‘Comparison’ for the inbred lines was either with the base population (‘base’) or with simultaneously outbred lines (‘outbred’). Inbreeding scheme (‘Scheme’) concerns the number of generations of brother-sister (BS) or mother-son (MS) mating. ‘F’ = the inbreeding coefficient. ‘RP’ = an index for the degree of inbreeding depression, ranging from –1 (total outbreeding depression) to 1 (total inbreeding depression). ‘P<0.05’ is depicted ‘yes’ if the authors found significant differences between the inbred and outbred lines.

<table>
<thead>
<tr>
<th>Reference</th>
<th>Species</th>
<th>Comparison</th>
<th>Scheme</th>
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<th>Trait category</th>
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is likely a maternally controlled trait and thus in this respect also female-limited. See Chapter 4). Thus, the studies published after 2003 suggest that within a haplodiploid species a negative effect of inbreeding is more likely to be found in fecundity than in generic life-history traits. The number of studies is too low to draw firm conclusions, but the data available point at a relation between the amount of directional dominance variance and gender. This implies that the strength of selection on directional dominance variance is related to gender.

Deleterious variation for oviposition rate and the strength of selection

Large amounts of deleterious genetic variation were found for oviposition rate in both the laboratory population (with regard to additive variance) and the natural population (with regard to additive and directional dominance variance). The directional dominance variance is probably caused by relatively weak effects of selection on traits controlled by genes with female-limited expression. But how can we explain the large amount of deleterious additive variance? Why is selection not stronger on this central component of fitness? There are three lines of thought that might be relevant to this question. First, perhaps the haplodiploidy of *T. urticae* plays a role: is selection strength lower on additive variance expressed in females than that expressed in males? As a simplified case, consider individual genes with purely additive gene action. Selection will be less ‘tailor-made’ in the females than in the males: selection acts on the average effect of both alleles in a heterozygote female and each allele is thus shielded by its counter-allele from the most effective selection. A hemizygous allele is not. If a new deleterious additive allele is added to the population, the time this allele remains in the population will subsequently be longer if it is only expressed in females. The mutation-selection balance concerning additive variance may thus be higher for traits controlled by genes with female-limited expression – as the mutation-selection balance concerning directional dominance variance probably is. Second, the effect of female-limited selection on oviposition rate may have played a role in general (instead of specifically for haplodiploids). If both a generic and a female-limited trait are hit by a mutation with similar effects on fitness, selection on the generic trait can take place in all individuals whereas a female-limited trait can only be selected on in the female subsection of the population. This also may influence the mutation-selection balance for additive variance. Third, oviposition rate contains both additive and recessive variation that is deleterious. Since this variation is ultimately caused by mutations, and mutations are usually partially recessive, the directional dominance and additive variances within a population are probably partly caused by the same underlying genes. Does the shared origin of recessive and additive variation perhaps influence the selection pressures on either? It would be very interesting to investigate whether these mechanisms play a role in explaining
the high additive variance for oviposition rate in *T. urticae*. In conclusion, I suggest the female-limitation of oviposition may be a central aspect in explaining the high genetic variation for this life-history trait.

**References**


