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# Impact of Epistasis in Inheritance of Quantitative Traits in Crops

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## 1. Introduction

Epistasis is the interaction between alleles of different genes, i.e. non-allelic interaction, as opposed to dominance, which is interaction between allele of the same gene, called inter-allelic or intra-genic interaction (Kearsey and Pooni, 1996). Statistical epistasis describes the deviation that occurs when the combined additive effect of two or more genes does not explain an observed phenotype (Falconer and Mackay, 1996).

The heritability of a trait, an essential concept in genetics quantitative, “certainly one of the central points in plant breeding research is the proportion of variation among individuals in a population that” is due to variation in the additive genetic (i.e., breeding) values of individuals:

$h^2 = VA/VP$  = Variance of breeding values/ phenotypic variance (Lynch and Walsh, 1998). This definition is now termed “heritability in the narrow-sense” (Nyquist, 1991). Estimation of this parameter was prerequisite for the amelioration of quantitative traits. As well as choosing the selective procedure, that will maximize genetic gain with one or more selection cycles. Various methods were developed in the past, Warner (1952), Sib-Analysis, Parent-offspring regressions etc. These methods considered that additive-dominant model is fitted, assuming epistasis to be negligible or non existent. Because of the complexity of theoretical genetics studies on epistasis, there is a lack of information about the contribution of the epistatic components of genotypic variance when predicting gains from selection. The estimation of epistatic components of genotypic variance is unusual in genetic studies because the limitation of the methodology, as in the case of the triple test cross, the high number of generations to be produced and assessed (Viana, 2000), and mainly because only one type of progeny, Half-Sib, Full-Sib or inbred families, is commonly included in the experiments (Viana, 2005). If there is no epistasis, generally it is satisfactory to assess the selection efficiency and to predict gain based on the broad-sense heritability. Therefore, the bias in the estimate of the additive variance when assuming the additive-dominant model is considerable. The preponderance of epistasis effect in the inheritance of quantitative trait in crops was recently reported by many geneticists (Pensuk et al., 2004; Bnejdi and El Gazzah, 2008; Bnejdi et al. 2009; Bnejdi and El-Gazzah, 2010a; Shashikumar et al. 2010). Epistasis can have an important influence on a number of evolutionary phenomena, including the genetic divergence between species.

The aims of our study were to determine the importance of epistasis effects in heredity of quantitative traits and their consequences in the bias of four methods of estimation of narrow-sense heritability.

2. Origin of data and genetic model

Nine quantitative traits with 88 cases of combination cross-site, cross-isolate or cross-treatment of six generations ( $P_1$ ,  $P_2$ ,  $F_1$ ,  $F_2$ ,  $BC_1$  and  $BC_2$ ) for three crops (*Triticum Durum*, *Capsicum annum* and *Avena sp*) were collected from different works realised in our laboratory. Crops, traits and origin of data are reported in Table 1. For each trait parents of crosses were extreme. Transformations (such as Kleckowski transforms (Lynch and Walsh, 1998)) were applied to normalize the distribution of data or to make means independent of variances for several traits.

<b>Durum Wheat</b> <b>(<i>Triticum durum</i>)</b>
<b>Two crosses/two sites</b> Number of head per plant , Spiklets per spike and Number of grains per spike (Bnejdi and El Gazzeh 2010b)
<b>Four crosses/ one site</b> Resistance to yellowberry (Bnejdi and El Gazzah, 2008)
<b>Four crosses/ one site</b> Resistance to yellowberry (Bnejdi et al., 2010a)
<b>Four crosses/ Two sites</b> Grain protein content (Bnejdi and El Gazzeh, 2010a)
<b>Two crosses/ Five salt treatments</b> Resistance to salt at germination stage (Bnejdi et al., 2011a)
<b>Two crosses/ fifteen isolates</b> Resistance to <i>Septoria tritici</i> (Bnejdi et al., 2011b)
<b>Pepper</b> <b>(<i>Capsicum annum</i> L.)</b>
<b>Two crosses/ Two isolates</b> Resistance to <i>Phytophthora nicotianae</i> (Bnejdi et al., 2009)
<b>Two crosses/ Six isolates</b> Resistance to <i>Phytophthora nicotianae</i> (Bnejdi et al., 2010b)
<b>Oates</b> <b>(<i>Avena sp.</i>)</b>
<b>Two crosses/ Two isolates</b> Resistance to <i>P. coronate</i> Cda. f. <i>sp. avenae</i> Eriks (Bnejdi et al., 2010c)

Table 1. Traits assessed in each crop and date of publication

## 2.1 Best genetic model

Weighted least squares regression analyses were used to solve for mid-parent [M] pooled additive [A], pooled dominance [D] and pooled digenic epistatic ([AA], [DD] and [AD]) genetic effects, following the models and assumptions described in Mather and Jinks (1982). A simple additive-dominance genetic model containing only M, A and D effects was first tested using the joint scaling test described in Rowe and Alexander (1980). Adequacy of the genetic model was assessed using a chi-square goodness-of-fit statistic derived from deviations from this model. If statistically significant at  $P < 0.05$ , genetic models containing digenic epistatic effects were then tested until the chi-square statistic was non-significant.

## 3. Phenotypic resemblance between relatives

We now will use the covariance (and the related measures of correlations and regression slopes) to quantify the phenotypic resemblance between relatives. Quantitative genetics as a field traces back to Fisher's 1918 paper showing how to use the phenotypic covariance to estimate genetic variances, whereby the phenotypic covariance between relatives is expressed in terms of genetic variances, as we detail below.

### 3.1 Parent-offspring regressions

There are three types of parent-offspring regressions: two **single parent - offspring regressions** (plotting offspring mean versus either the trait value in their male parent  $P_f$  or their female parent  $P_m$ ), and the **mid-parent-offspring regression** (the offspring mean regressed on the mean of their parents, the mid-parent  $MP = (P_f + P_m)/2$ ).

The slope of the (single) parent-offspring regression is estimated by

$$b_{o/p} = \frac{Cov(O, P)}{Var(P)}, \text{ where } Cov(O, P) = \frac{1}{n-1} \left( \sum_{i=1}^n O_i P_i - n \bar{O} \bar{P} \right)$$

Where  $O_i$  is the mean trait value in the offspring of parent  $i$  ( $P_i$ ) and we examine  $n$  pairs of parent-offspring. One could compute separate regressions using males ( $P_m$ ) and females ( $P_f$ ), although the later potentially includes maternal effect contributions and hence single-parent regressions usually restricted to fathers.

$$b_{o/p} = \frac{Cov(O, P)}{Var(P)}$$

$$Cov(O, P) = \frac{\sigma^2 A}{2} + \left( \frac{\sigma^2 AA}{4} + \frac{\sigma^2 AAA}{8} + \frac{\sigma^2 AAAA}{16} + \dots \right)$$

$$b_{o/p}^* = \frac{Cov(O, P)}{Var(P)} = \frac{\sigma^2 A}{2\sigma_p^2} + \frac{1}{\sigma_p^2} \left( \frac{\sigma^2 AA}{4} + \frac{\sigma^2 AAA}{8} + \frac{\sigma^2 AAAA}{16} + \dots \right)$$

$$b_{o/p}^* = \frac{Cov(O, P)}{Var(P)} = \frac{h^2}{2} + \frac{1}{\sigma_p^2} \left( \frac{\sigma^2 AA}{4} + \frac{\sigma^2 AAA}{8} + \frac{\sigma^2 AAAA}{16} + \dots \right)$$

Assuming an absence of epistasis we have

$$\begin{aligned} \text{Cov}(O, P) &= \frac{1}{2}\sigma^2_A, \text{ giving } b_{o/p} = \frac{\frac{1}{2}\sigma^2_A}{\sigma_p^2} = \frac{h^2}{2} \\ h^2 &= 2b_{o/p} \end{aligned}$$

### 3.2 Full-sib analysis

The covariance full-sib analysis is equal to:

$$\begin{aligned} \text{Cov}(FS) &= \frac{1}{2}\sigma^2_A + \frac{1}{4}\sigma^2_D + \frac{1}{4}\sigma^2_{AA} + \frac{1}{8}\sigma^2_{AD} + \frac{1}{16}\sigma^2_{DD} + \frac{1}{8}\sigma^2_{AAA} \dots \\ \frac{\text{Cov}(FS)}{\sigma_p^2} &= \frac{h^2}{2} + \frac{1}{\sigma_p^2} \left( \frac{1}{4}\sigma^2_D + \frac{1}{4}\sigma^2_{AA} + \frac{1}{8}\sigma^2_{AD} + \frac{1}{16}\sigma^2_{DD} + \frac{1}{8}\sigma^2_{AAA} \dots \right) \end{aligned}$$

So, when epistasis was considered negligible

$$\begin{aligned} \text{Cov}(FS) &= \frac{1}{2}\sigma^2_A \\ h^2 &= \frac{2\text{Cov}(FS)}{\sigma_p^2} \end{aligned}$$

### 3.3 Half-sib analysis

Based on half-sib analysis, narrow-sense heritability was calculated as:

$$\begin{aligned} \text{Cov}(HS) &= \frac{1}{4}\sigma^2_A + \frac{1}{16}\sigma^2_{AA} + \frac{1}{64}\sigma^2_{AAA} + \dots \\ \frac{\text{Cov}(HS)}{\sigma_p^2} &= \frac{h^2}{4} + \frac{1}{\sigma_p^2} \left( \frac{1}{16}\sigma^2_{AA} + \frac{1}{64}\sigma^2_{AAA} + \dots \right) \end{aligned}$$

So, epistasis was considered negligible and the narrow-sense heritability was determined as:

$$\begin{aligned} \text{Cov}(HS) &= \frac{1}{4}\sigma^2_A \\ h^2 &= \frac{4\text{Cov}(HS)}{\sigma_p^2} \end{aligned}$$

### 3.4 Method of Warner (1952)

Based on additive dominance model Warner in 1952 revealed that narrow-sense heritability could be estimated as:

$2\sigma_{F_2}^2 - (\sigma_{BC_1}^2 + \sigma_{BC_2}^2)$  Where  $\sigma_{F_2}^2$ ,  $\sigma_{BC_1}^2$  and  $\sigma_{BC_2}^2$  represented respectively the variance of generation  $F_2$ ,  $BC_1$  and  $BC_2$

In absence of epistasis

$$2\sigma_{F_2}^2 - (\sigma_{BC_1}^2 + \sigma_{BC_2}^2) = \frac{1}{2}a_A^2 = \sigma_A^2$$

$$\frac{2\sigma_{F_2}^2 - (\sigma_{BC_1}^2 + \sigma_{BC_2}^2)}{\sigma_{F_2}^2} = \frac{\sigma_A^2}{\sigma_{F_2}^2} = h^2$$

Therefore in presence of epistasis

$$2\sigma_{F_2}^2 - (\sigma_{BC_1}^2 + \sigma_{BC_2}^2) = 2(a + \frac{1}{2}ad)^2 + (d + \frac{1}{2}dd)^2 + \frac{1}{2}aa^2 + \frac{1}{2}ad + \dots\dots\dots$$

#### 4. Results and discussion

Separate generation means analysis revealed that the additive-dominance model was found adequate only for 18 cases. Therefore, the digenic epistatic model was found appropriate for 70 cases (Table 2). Additive and dominance effect were significant for all cases of combination. With regard to epistatic effects, the additive x additive effect was significant for 77 cases and the additive x dominance for 42 cases and dominance x dominance effects for 56 cases. Recent studies suggest that epistatic effects are present for inheritance of quantitative traits in many species. Examples are wheat (resistance to leaf rust, Ezzahiri and Roelfs 1989), wheat (resistance to yellowberry, Bnejdi and El Gazzah 2008), common bean (resistance to anthracnose, Marcial and Pastor 1994), barley (resistance to *Fusarium* head blight, Flavio et al. 2003), chickpea (resistance to *Botrytis cinerea*, Rewal and Grewal 1989), and pepper (resistance to *Phytophthora capsici*, Bartual et al. 1994).

To conclude for this part, the additive dominance model was rarely fitted and digenic epistatic model was frequently appropriate. Therefore epistasis is common in inheritance of quantitative traits and any model or methods assumed that epistasis was negligible were biased.

The comparison of the four methods is reported in Table 3. In absence of dominance and epistatic effect, the methods were not biased. Therefore, in presence of epistasis narrow-sense heritability based on the four methods was underestimated. Based in Full-Sib Analysis and Warner (1952) methods, bias was caused by dominance, interaction between homozygote loci, interaction between heterozygote loci and interaction between homozygote and heterozygote loci. Therefore based in Half-Sib Analysis and Parent-offspring regressions, bias was caused only with the presence of interaction between homozygote loci or fixable effect.

The result of generations means analysis indicate that digenic epistasis model were frequently appropriate. So the additive model in which many methods of genetic quantitative were based was rarely adequate. Based on the result, the methods of Half-Sib Analysis and Parent-offspring regressions were underestimated with additive x additive

effect (Table 3). Because additive x additive effect can be fixed by selection, estimation of narrow-sense heritability with theses methods was recommended and efficiency in crops breeding. Linkage disequilibrium and absence of epistasis are compulsorily assumed in almost all the methodologies developed to analyze quantitative traits. The consequence, clearly, is biased estimates of genetic parameters and predicted gains, as linkage and genetic interaction are the rule and not the exception Viana (2004). The prediction of gains from selection allows the choice of selection strategies. Therefore the gain from selection was estimated from narrow-sense heritability. Breeding strategies applied for plant breeding aimed to increase the favourable gene frequency. The efficiency of any methodology of selection was associated with the best estimated of the additive genetic effect value.

Best fit- model	Number of cases
M + A + D	18
M + A + D + AA	2
M + A + D+ AA + DD	26
M + A + D + AA + AD	13
M + A + D + DD + AD	3
M + A + D + AA + DD + AD	18
M + A + D + AA + DD + AD + C	8

M, mean; A, additive; D, dominance; AA, additive x additive; AD, additive x dominance; DD, dominance x dominance; C, cytoplasm effect.

Table 2. Best-fit models of nine traits with 88 cases of combinations Cross-site, cross-treatment and or cross-isolate for three crops.

In presence of epistasis effect, Parent-offspring regressions and Half-Sib Analysis were the best methods. In fact, these methods were biased only with interaction between homozygote loci represented by “additive x additive” effect. However, both the methods of Warner (1952) and Full-Sib Analysis were biased with dominance, additive x dominance, dominance x dominance and additive x additive effects. The interaction between the homozygote loci can be fixed by selection. But the fixation of interaction between heterozygote loci prerequisite maintain of heterozygote. Depending upon the methods, the bias in the estimation of narrow-sense heritability in presence of epistasis was more pronounced.

The presence of epistasis complicated the procedure of amelioration of quantitative traits and revealed the limitation of most quantitative studies based on the assumption of negligible epistasis. However, the exploitation of epistasis in the breeding program such as the superiority of heterozygous genotypes over their corresponding parental genotypes was of great importance.



Method	in absence of epistasis	in presence of epistasis
Parent-offspring regressions	$b_{o/p} = \frac{Cov(O,P)}{Var(P)}$ $Cov(O,P) = \frac{1}{2}\sigma^2 A$ $b_{o/p} = \frac{\frac{1}{2}\sigma^2 A}{\sigma_p^2} = \frac{h^2}{2}$ $h^2 = 2b_{o/p}$	$b_{o/p} = \frac{Cov(O,P)}{Var(P)}$ $Cov(O,P) = \frac{\sigma^2 A}{2} + (\frac{\sigma^2 AA}{4} + \frac{\sigma^2 AAA}{8} + \frac{\sigma^2 AAAA}{16} + .....)$ $b_{o/p}^* = \frac{Cov(O,P)}{Var(P)} = \frac{\sigma^2 A}{2\sigma_p^2} + \frac{1}{\sigma_p^2}(\frac{\sigma^2 AA}{4} + \frac{\sigma^2 AAA}{8} + \frac{\sigma^2 AAAA}{16} + .....)$ $b_{o/p}^* = \frac{Cov(O,P)}{Var(P)} = \frac{h^2}{2} + \frac{1}{\sigma_p^2}(\frac{\sigma^2 AA}{4} + \frac{\sigma^2 AAA}{8} + \frac{\sigma^2 AAAA}{16} + .....)$
Full-Sib Analysais	$Cov(FS) = \frac{1}{2}\sigma_A^2$ $h^2 = \frac{2Cov(FS)}{\sigma_p^2}$	$Cov(FS) = \frac{1}{2}\sigma_A^2 + \frac{1}{4}\sigma_D^2 + \frac{1}{4}\sigma_{AA}^2 + \frac{1}{8}\sigma_{AD}^2 + \frac{1}{16}\sigma_{DD}^2 + \frac{1}{8}\sigma_{AAA}^2.....)$ $\frac{Cov(FS)}{\sigma_p^2} = \frac{h^2}{2} + \frac{1}{\sigma_p^2}(\frac{1}{4}\sigma_D^2 + \frac{1}{4}\sigma_{AA}^2 + \frac{1}{8}\sigma_{AD}^2 + \frac{1}{16}\sigma_{DD}^2 + \frac{1}{8}\sigma_{AAA}^2.....)$
Half-Sib Analysais	$Cov(HS) = \frac{1}{4}\sigma_A^2$ $h^2 = \frac{4Cov(HS)}{\sigma_p^2}$	$Cov(HS) = \frac{1}{4}\sigma_A^2 + \frac{1}{16}\sigma_{AA}^2 + \frac{1}{64}\sigma_{AAA}^2 + .....)$ $\frac{Cov(HS)}{\sigma_p^2} = \frac{h^2}{4} + \frac{1}{\sigma_p^2}(\frac{1}{16}\sigma_{AA}^2 + \frac{1}{64}\sigma_{AAA}^2 + .....)$
Warner (1952)	$2\sigma_{F_2}^2 - (\sigma_{BC_1}^2 + \sigma_{BC_2}^2) = \frac{1}{2}a_A^2 = \sigma_A^2$ $\frac{2\sigma_{F_2}^2 - (\sigma_{BC_1}^2 + \sigma_{BC_2}^2)}{\sigma_{F_2}^2} = \frac{\sigma_A^2}{\sigma_{F_2}^2} = h^2$	$2\sigma_{F_2}^2 - (\sigma_{BC_1}^2 + \sigma_{BC_2}^2) = 2(a + \frac{1}{2}ad)^2 + (d + \frac{1}{2}dd)^2 + \frac{1}{2}aa^2 + \frac{1}{2}ad + .....)$

O, offspring; P, parent; A, additive; D, dominance; AA, additive × additive; AD, additive × dominance; DD, dominance × dominance; AAA, additive × additive × additive;

Table 3. Bias of four methods of estimation of narrow-sense heritability in presence of epistasis

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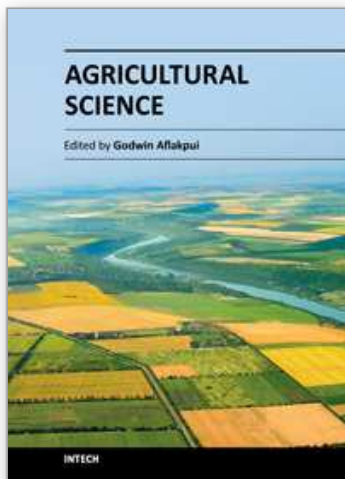
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This book covers key areas in agricultural science, namely crop improvement, production, response to water, nutrients, and temperature, crop protection, agriculture and human health, and animal nutrition. The contributions by the authors include manipulation of the variables and genetic resources of inheritance of quantitative genes, crop rotation, soil water and nitrogen, and effect of temperature on flowering. The rest are protecting crops against insect pests and diseases, linking agriculture landscape to recreation by humans, and small ruminant nutrition. This book is a valuable addition to the existing knowledge and is especially intended for university students and all professionals in the field of agriculture.

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