AGRICULTURAL SCIENCE

Edited by **Godwin Aflakpui**

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Preface

The whole world needs agriculture because agriculture does not only feed the entire human race but also produces fibre for clothing, feed for livestock and bio-energy. In the developing world agriculture contributes significantly to the gross domestic product, leads the way in reducing poverty and accounts for the lion's share of employment opportunities especially for women. Agriculture also has one of the highest potentials for reducing carbon emissions and helping vulnerable people adapt to climate change.

The Food and Agriculture Organisation (FAO) of the United Nations and the World Bank have indicated that:

- 100% of the global population depends on agriculture for nutrition
- 40% of the global population relies on agriculture for employment
- 70% of worldwide withdrawal of water is for the purposes of agriculture
- 30% proportion of greenhouse gas emissions is related to agriculture
- 70% increment in food production is needed to feed a global population of 9 billion by 2050.

These figures by the FAO and the World Bank indicate that without agriculture, the world economy would not be what it has been and what it is today.

For agriculture to continuously contribute to food security, environmental sustainability and economic opportunities by driving the rural and national economic development with well targeted investments, it is imperative that the research and development agenda which is based on the science behind agriculture must be pursued vigorously. It is in this context that this book, Agricultural Science has been written with multiple authors compiling some important state-of-the art contributions on the subject in recent years.

The contributions of chapters in the book are divided into seven sections: Crop Improvement, Crop Production, Crop Response to Water and Nutrients, Crop Response to Temperature, Crop Protection, Agriculture and Human Health, and Animal Nutrition. The sections vary in the number of chapters which was largely due to the number of authors who contributed chapters to publish the book. The chapters in each section and in the book in general vary in scope and the way they attempt to

manipulate resources and variables to improve on productivity and also to link agricultural landscape to recreation and therefore human health, albeit remotely.

I acknowledge the authors for willingly contributing their chapters without which we could not have published this book. I am equally grateful to Ms Jana Sertic, the Publishing Process Manager for the able assistance she provided and to the Information Technology Department for providing the requisite framework that greatly enhanced the work of putting together the chapters in the book. The Technical Editors deserve commendation for preparing the online publication and print versions of the book.

Finally, I owe a debt of gratitude to the Scientific Board of the INTECH OPEN ACCESS PUBLISHER for the trust reposed in me to edit this book. I am most grateful to be of service to the scientific community.

Dr. Godwin Aflakpui
Rector, Wa Polytechnic, Wa, Upper West Region,
Ghana, West Africa

Section 1

Crop Improvement

1

Impact of Epistasis in Inheritance of Quantitative Traits in Crops

Bnejdi Fethi and El Gazzeh Mohamed
Laboratoire de Génétique et Biométrie Faculté des Sciences de Tunis,
Université Tunis, El Manar,
Tunisia

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. Theses 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.

Durum Wheat (*Triticum durum*)
Two crosses/two sites Number of head per plant , Spiklets per spike and Number of grains per spike (Bnejdi and El Gazzeh 2010b)
Four crosses/ one site Resistance to yellowberry (Bnejdi and El Gazzah, 2008)
Four crosses/ one site Resistance to yellowberry (Bnejdi et al., 2010a)
Four crosses/ Two sites Grain protein content (Bnejdi and El Gazzeh, 2010a)
Two crosses/ Five salt treatments Resistance to salt at germination stage (Bnejdi et al., 2011a)
Two crosses/ fifteen isolates Resistance to *Septoria tritici* (Bnejdi et al., 2011b)
Pepper (*Capsicum annuum* L.)
Two crosses/ Two isolates Resistance to *Phytophthora nicotianae* (Bnejdi et al., 2009)
Two crosses/ Six isolates Resistance to *Phytophthora nicotianae* (Bnejdi et al., 2010b)
Oates (Avena sp.)
Two crosses/ Two isolates Resistance to *P. coronate* Cda. f. sp. *avenae* 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 *Pf* or their female parent *Pm*), and the **mid-parent-offspring regression** (the offspring mean regressed on the mean of their parents, the mid-parent $MP = (Pf + Pm)/2$).

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

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

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

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

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

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

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

Assuming an absence of epistasis we have

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