

Epistasis and Plant Breeding

*James B. Holland**

U.S. Department of Agriculture—Agriculture Research Service,
Plant Science Research Unit, Department of Crop Science,
North Carolina State Univ., Box 7620, Raleigh,
North Carolina 27695-7620.

- I. INTRODUCTION
- II. GENE ACTION AND STATISTICAL EFFECTS
- III. EPISTASIS AND MOLECULAR INTERACTIONS
 - A. Molecular Interactions Lead to Epistasis
 - B. Duplicate Gene Interactions and Genetic Redundancy
 - C. Complementary Gene Interactions in Biochemical and Molecular Pathways
 - D. Synergistic and Antagonistic Gene Interactions
 - E. Compensatory Gene Interactions
- IV. COMPLEX MOLECULAR INTERACTIONS UNDERLIE QUANTITATIVE PHENOTYPES (SOMETIMES)
 - A. Models of Interactions in Metabolic Pathways
 - B. A Model of Gene Interactions in Developmental Regulation Pathways
 - C. Boolean Regulatory Networks as Models for Genetic Regulatory Pathways
 - D. Multiplicative Interactions: Phenotypic Epistasis as an Emergent Property of Additive Physiological Components
- V. BIOMETRICAL EVIDENCE FOR EPISTASIS
 - A. Outcrossing Plant Species
 - B. Self-Pollinating Species
 - C. Why Is There More Evidence for Epistasis in Selfing than in Outcrossing Species?
- VI. EVIDENCE FOR EPISTASIS FROM PLANT EVOLUTION STUDIES
- VII. MOLECULAR MARKER INVESTIGATIONS OF EPISTASIS
- VIII. WHY IS THERE MORE EVIDENCE FOR EPISTASIS FROM QTL EXPERIMENTS THAN FROM BIOMETRICAL STUDIES?
- IX. IMPLICATIONS OF EPISTASIS FOR PLANT BREEDING

*I thank Drs. Edwin Bingham, Charles Brummer, James Crow, Arnel Hallauer, Wyman Nyquist, and David Remington for their helpful comments on and corrections to a previous draft of this article.

Plant Breeding Reviews, Volume 21, Edited by Jules Janick
ISBN 0-471-41847-1 © 2001 John Wiley & Sons, Inc.

- A. Inbreeding Depression and Heterosis
 - 1. Inbreeding Depression
 - 2. Heterosis
 - 3. Multiplicative Epistasis and Heterosis
 - B. Epistatic Variance Can Be Transformed into Additive Variance After Bottlenecks
 - C. Temporary Response to Selection
 - D. Adaptive Landscapes
- LITERATURE CITED

I. INTRODUCTION

Epistasis is the interaction of alleles at different loci. The value of an allele or genotype at one locus depends on the genotype at other epistatically interacting loci, complicating the picture of gene action. A seemingly “favorable” allele at one locus may be an “unfavorable” allele in a different genetic background. There are well-defined cases of interactions occurring at the molecular level between gene products, but the relationship between molecular interactions and complex phenotypes is often not clear. Classical quantitative genetics methods relate observable phenotypic measures to the aggregate statistical effects of alleles and allelic combinations in specific populations. Genetic components of variance are population-dependent, often poorly estimated, and do not necessarily reflect the relative importance of different modes of gene action. DNA markers have simplified the direct estimation of gene action effects, and recent QTL and population genetics studies have revealed that epistatic gene action is more important for plant yield and fitness than was previously evident. Implications of strong epistasis for plant breeding include: (1) epistatic variance can shift to additive variance under drift or inbreeding; (2) epistatic variance contributes to “temporary” response to selection in outcrossing populations that can be captured as a form of heterosis using appropriate breeding procedures but may be otherwise squandered; and (3) fitness or yield is not a simple function of allele frequencies, resulting in rugged adaptive landscapes filled with local fitness optima on which breeding populations can become stranded. If epistasis is important, then genomics tools can be used to identify the nature and components of interacting genic systems and marker-assisted selection schemes can be designed to exploit epistasis.

The literature on theory of and empirical evidence for epistasis in crops is reviewed here. Explicit formulations for additive, dominance, and epistatic genetic effects and variances; inbreeding depression; heterosis; and response to selection are presented to unify the discussion