Heterosis or Hybrid Vigor

Heterosis, or hybrid vigor, refers to the phenomenon that progeny of diverse inbred varieties exhibit greater biomass, the speed of development, and fertility than the better of the two parents (Add figure of Brassica napus heterosis. This phenomenon has been exploited extensively in crop production and has been a powerful force in the evolution of plants. The genetic basis has been postulated for nearly a century ago(Shull, 1908; Bruce, 1910; Jones, 1917), but little consensus has emerged. With the advent of the genomic era, the tool to establish a molecular basis for heterosis exists. Previously any molecular difference between the parents and progeny have been attributed to the basis of heterosis. Due to the multigenic nature of heterosis, it has been considered as miserably complex, that's how some scientists let it off and that ultimately a combining principle will emerge. In this Article, We summarize the significant features of heterosis that a possible molecular marker essentially explains.Add figure hereHeterosis in Maize.

The classic quantitative genetic explanations for heterosis center on two concepts (Crow, 1948). The first is "dominance," which originally meant that heterosis results from the complementation in the hybrid of different deleterious alleles that were present in the inbred parental lines by superior alleles from the opposite parent. Over time, this term came to mean the degree to which the heterozygous genotype performs differently from the mean of the two homozygous classes. The second historical explanation for heterosis is "overdominance," which refers to the idea that allelic interactions occur in the hybrid such that the heterozygous class performs better than either homozygous class. Although these terms have developed a following in each case, they both now refer to nonadditive situations, differing in degree. These terms were coined before the molecular concepts of genetics were formulated and are not connected with molecular principles. Therefore, they are of diminished utility for describing the molecular parameters that accompany heterosisTwo extreme models can explain heterosis on their molecular level.IN Model one we can imagine that when two different alleles of the various gene are brought together in a hybrid it leads to a combined allelic expression.

IN Model two Different alleles combination produces an interaction that causes gene expression in the hybrid to deviate relative to the mid-parent predictions (i.e., by an upregulation of many housekeeping genes)The Models may be considered as the result of gene allelic interaction.In 2003 Song and messing provides evidence for altered regulatory effects in hybrids. The challenge in the development of molecular model for heterosis is to make the correct associations between phenotypes and causative molecular event that occurs in hybridsThe past century explains heterosis that, Slightly different and deleterious alleles exist at multiple loci in two inbred lines. In the hybrid produced all mutations are supplemented causing the progeny to

exceeds the parents. This hypothesis was criticized that if it is the correct explanation then it should be promising to produce an inbred line having all the superior alleles which shows minimum or no hybrid vigor, a condition that does not ascend.

The counter-argument specified that It would be impossible to gather all of the better alternatives into one line with so many genes involved in the linkage of deleterious alleles with the superior allele of the other gene. Though it is accurate that a deleterious allele can become homozygous in different inbred lines and that the hybrid would show complementation for these genes. This fact might be reasonable for hybrid being equivalent to the better of two parents for the effect of the individual gene. Instead, if the complementation of alleles in different genes were mounting in the phenotype it will result in heterosis. The Molecular question arises is that whether the simple complementation of slightly different and deleterious alleles causes a growth response that can lead to heterosis. Yet several observations related to heterosis proposes that the basic principle of heterosis is not confined to the simple complementation.Observation Ist. Though inbred lines have been improved greatly over the decades, the magnitude of heterosis has not diminished but has increased slightly. This observation suggests a basis other than simple complementation.East, 1936; Duvick, 1999If heterosis is supposed to cause by the complementation of deleterious alleles and inbred lines have been eradicated then the total sum of heterosis might be decreased. As Heterosis gives the appearance of being more resistant to artificial selection then the quality of inbred lines. More ever the quality of two inbred lines do not urge the amount of heterosis; this must be intent on a cross. This Observation suggests that rather replacing alleles of genes that modulate physiological processes important for heterosis, the slight increase in heterosis over the years might have occurred by selecting alleles at the right set of loci that make the best combination in hybrids to bring about heterosis.

Observation 2nd.Progressive heterosis in Tetraploids argues against simple complementation (Levings et al., 1967; Mok and Peloquin, 1975; Groose et al., 1989; Bingham et al., 1994). Two alleles of a gene can occur in an individual at diploid level However at higher ploidy level, Variety of allelic combinations are possible for a gene. In autotetraploids that are hybrid between two inbred lines But is potentially when there are three or four different alleles present at various loci. Also in allohexaploid wheat, where three different genomes contribute to the genetic constitution, hybrids between diverse varieties exhibit heterosis Briggle 1963. It appears that vigor advances as the greater number of distinct genomes exist. For simple complementation to explain progressive heterosis, each new step-wise combination of genomes would need to supply increasingly superior alleles to complement the preexisting rate-limiting alleles without introducing deleterious alleles at other loci. The probability of the said situation is very low. A release from negative dosage effects on vigor by identical alleles could account for progressive heterosis, which is elaborated further below.

Observation 3Inbreeding depression in tetraploids of many species proceeds faster than

expected based on homozygosis of alleles.(Randolph, 1942; Alexander and Sonnemaker, 1961; Busbice and Wilsie, 1966; Rice and Dudley, 1974)In a diploid, selling of a heterozygote (A/B) will produce half of the progeny that are homozygous at one locus and the other half that regenerate heterozygous condition. In an autotetraploid, the selfing of heterozygote (A/A/B/B)will produce homozygotes (A/A/A/A or B/B/B/B) at any locus in only ?1 of 18 offspring (depending on the degree of linkage to the centromere) In addition, A/A/B/B to heterozygotes being formed again, A/A/A/B and B/B/B/A heterozygotes are present in the population. Regardless this difference in the rate of progression to heterozygosity, the trajectory of inbreeding depression in tetraploid is often faster than expected and not very different from that in diploids. IN some species, tetraploids inbreeding depression proceeds faster than at the diploid level. As discovered by Randolph (1942), Tetraploid derivatives of maize lines are less vigorous than the diploid progenitor. Thus in this species the end product of inbreeding depression in tetraploid is less then that of diploids, though the genotype is identical (but differ in dosage)

One resolution of this finding is to suggest that allelic dosage plays a more important role in tetraploids for generating inbreeding depression than does complete homozygous itself, because the allelic dosage shifts more rapidly than the homozygosis during selling . The increasing number of identical alleles appears to have a negative dosage effect on vigor.If there is any contribution of dosage effect of alleles in polyploidy heterosis, this understanding is satisfying that the majority of Quantitative trait loci show some degree of semi-dominant behavior (Tanksley, 1993 indicating that the quantitative trait is largely affected by multiple loci that exhibit an allelic dosage effect. The results of aneuploidy studies suggest that quantitative characteristics are affected by multiple dosage dependant genes Lee et al., 1996). There is expected linkage between these two observations Guo and Birchler, 1994).

What is responsible for such dosage effects? It has been reasoned that these dosage effects are reflections of dosage-dependent gene regulatory hierarchies. (Birchler et al., 2001). Regulatory genes mostly genes reveal some measures of dosage dependence, As target housekeeping genes usually show greater dominance/recessive behavior between allelic alternatives (Birchler and Auger, 2003). A possible explanation for this partial dichotomy comes from an analysis of dosage-sensitive genes in yeast (Papp et al., 2003). In diploid yeast loci that tends to have a significant haplo insufficient effect on growth, encodes products that are involved in molecular complexes. Regulatory genes in multicellular organisms often function as part of complexes, so if the same rule applies, regulatory genes usually will exhibit some measures of dosage dependence whereas gene that encodes metabolic functions will be less likely to show a dosage effect. Empirical observations suggest that most regulatory genes do exhibit some kind of dosage response (Birchler et al., 2001). Accordingly a Quantitive will be controlled in large part by multiple dosage dependant regulatory loci. Following this background, One can be led to the idea that heterosis is the result of different alleles being present at loci

that contribute to the regulatory hierarchies that control quantitative traits.Gene expression in inbreds and hybrids suggests a shift in gene regulation in hybrids. Romagnoli et al. (1990), Leonardi et al. (1991), (Osborn et al., 2003) and Song and Messing (2003)their study suggests that the expression of many genes does not exhibit the expected mid-parent value.

If heterosis is due to the change in gene expression then which genes are involved and how do these changes compare with the alterations in gene expression that occur in aneuploids, which in most cases are detrimental to vigor? Aneuploidy also causes changes in gene expression typically within a twofold range (Birchler, 1979; Birchler and Newton, 1981; Guo and Birchler, 1994; Auger et al., 2001; Wanous et al., 2003). These changes can result from structural gene dosage effects, but more often they result from trans-acting effects that modulate the expression of most of the genome (Birchler et al., 2001; Matzke et al., 2003). It has been proposed that the reductions in gene expression that occur in both monosomic and trisomics are rate limiting on the phenotype and therefore act as underlying contributors to aneuploid syndromes (Birchler and Newton, 1981; Guo and Birchler, 1994; Birchler et al., 2001). It appears that the reductions in gene expression are detrimental to the vigor of the aneuploid plants. To date, these analyses have relied on a sampling of gene expression rather than a comprehensive examination of genome-wide expression patterns. A larger sampling might determine, for example, if heterosis, in general, is correlated with a majority of the increases in gene expression while aneuploidy leads to a significant number of reductions in gene expression in both monosomic and trisomies. A complete picture might elucidate this distinction if there is a meaningful comparison to be made.

What distinguishes the phenotypic consequences of the ups and downs of gene expression in aneuploids versus hybrids? One possibility is that the gene expression changes that foster increased biomass and fertility have been selected in hybrid states over long periods of time, whereas aneuploid situations usually are transitory and the result of laboratory manipulations.To formulate a molecular model of heterosis, simple broad alternatives need to be tested so that more refined and targeted hypothesis testing can focus on the detailed mechanism. One could argue that nothing less than defining how the genome interacts to create the phenotype is needed for an understanding of heterosis and that this understanding is too far in the future to attempt any examination of heterosis at present. Such a view is too agnostic and should not stand in the way of chipping away at alternatives. An eventual molecular explanation of heterosis will determine whether it can be manipulated for the benefit of agriculture and biotechnology