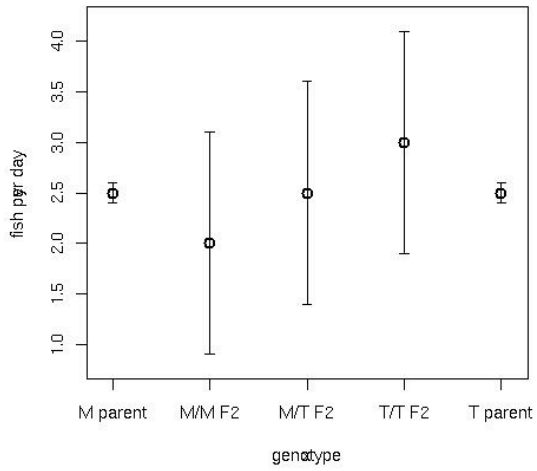
1. We learned in class that most common human diseases are controlled by many loci, each of which has a very subtle effect on disease. What is the value of actually mapping one of these loci? Name two ways that knowing the identity of a subtle causal locus associated with a disease might someday impact human health.

2. Imagine that you are studying fishing success in kingfisher birds, a quantitative trait. You have two birds, one deriving from a wild bird caught in Micronesia and the other deriving from a Tokyo Bay isolate, that are each inbred to complete homozygosity. You mate them to produce a large family of F2's. From this population you map two additive and incompletely dominant loci that completely explain variation in fishing success between birds in the cross. One locus is on chromosome II and the other is on chromosome X. The effect of the chromosome II locus is shown below, where M stands for Micronesia and T stands for Tokyo:



(a) What is the effect on phenotype (in units of fish per day) among the F2's of gaining a single T allele at the chromosome II locus?

(b) If you made a similar diagram comprising all the possible F2 **genotypes** plus the parents, how many columns (*x* values) would there be?

(c) The “error bars” in this plot don’t all reflect error per se. What do they represent in the middle three data points and why are they wide relative to the parents?

(d) What must the phenotype be of F2’s that are homozygous for the Micronesian allele at both the chromosome II locus and the X locus? (Hint: look at the first column of the figure and remember that the loci act additively.)

(e) Given this, what can you say about the average phenotype be of F2’s that are homozygous for the Micronesian allele at the chromosome II locus and homozygous for the Tokyo allele at the chromosome X locus?