BIS101 F2013 Lecture 3: Gene Interaction

Questions

Reading

Skip 4.3,4.4

Allele effects

dominant/recessive

- bad mutations often but not always recessive — dominant negative examples in book
- ? **null mutation** which producess a non-functioning copy (not all mutations are null!)
- Figure 6-1 and 6-2 nice drawing explaining haplosufficiency

incomplete dominance (white/pink/red, draw genotypes)

- intermediate phenotype — mixing on a quantitative scale
- remember this is from phenotype perspective -- here looks like mixing
- also not the same as a **quantitative trait** e.g. human height is not incomplete dominance! because >1 locus is involved

codominance — both alleles are expressed

- blood types (ignoring the Rh factor +/- here)
    - 3 alleles, I^A, I^B, i
    - I^A / (A or i) A blood type
    - I^B / (B or i) B
    - I^A / I^B AB type
    - i/i O type
- at phenotypic level looks different from incomplete dominance
- at genetic level both are pretty much the same
- reality we treat dominance as a variable, where 0 is recessive and 1 is dominant
- phenotype can be e.g. 0, a*d, a (don't need to know, and notation not really correct)

lethal

- can be dominant or recessive — ? why is recessive more common?

pleiotropy — has more than one effect

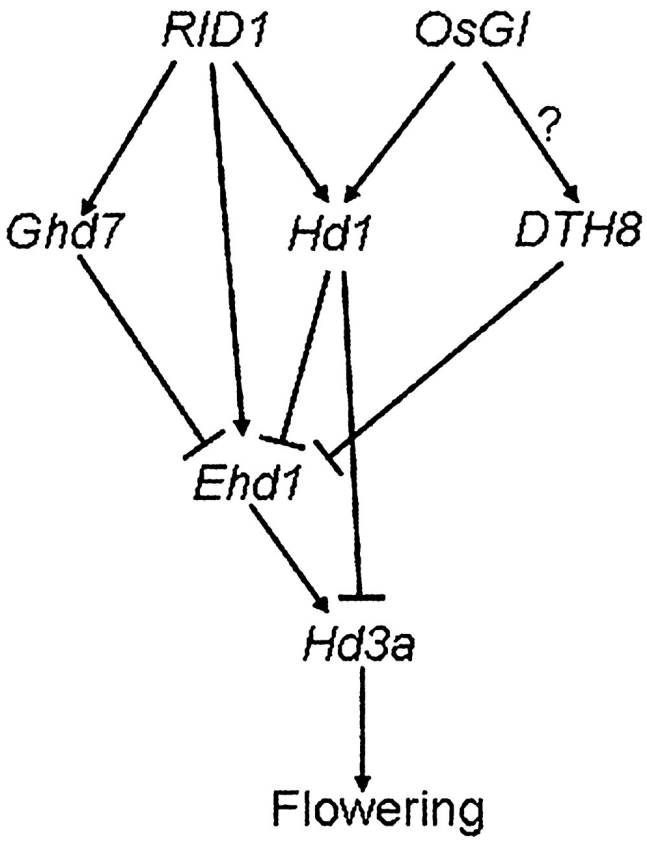
- can be silly ? what other effects might an allele that increases height have?
- "real" pleiotropy is traits that are less obviously correlated. perhaps an enzyme is ne5eded for pathway to flower and to make leaf hairs. a mutant in the gene for that enzyme would have two phenotypes

Pathways

genes not in vacuum and not just make one product that does it's thing

we've considered effects of alleles at each gene independent of others

- e.g. effects of alleles at the round/wrinkled locus not change effects of alleles at the yellow/green locus
- usually or often not the case Example: oversimplification of flowering time pathway in rice



Simple pathway example

- precursor1-> (enzyme A from gene A)-> precursor2->(enzyme B gene B)->red fruit (wt) - DRAW (leave on board) with enzymes as looped arrows not in pathway
- make recessive homozygote lines of two recessive mutants (so now a<A and b<B), cross to make double mutants
- **complementation** test
- draw out two possibilities (separate loci, same locus)
- ? If I cross two whites -> red 1 or 2 loci (what are genos)?
    - aaBB x AAbb (both white) = ? AaBb (red)
- ? why use recessive and not dominant mutants? (because no complementation even if in different genes)
- ? how do you know if your mutant is recessive? (make F1 and self!)
- what if I take two white plants w/ recessive mutants cross and = white?
    - perhaps aaBB x a'a'BB = aa'BB (fail to complement b/c just two mutations of same gene)

fig. 6-12 in the book rad

Epistasis

section 6.3 on inferring gene interaction is good.

9:3:3:1 and 3:1 ratios are for independent genes. (don't memorize)

What about interaction among genes? like in these pathways?

epistasis "stand upon"

- statistical epistasis is observation of an interaction, but unknown gene(s)
    - late flowering allele may make you flower 3 days later in one plant, but only 1 day later in another or 2 days earlier in a third
    - an allele that always made all plants flower 3 days later, regardless of genotype —> no epistatic interactions
- biochemical epistasis -> known pathway & functions

back to drawing (precursors):

precursor-> (enzyme A makes pigment)-> yellow pigment->(enzyme B turns yellow red)->red fruit (wt)

aaBB (white); aabb (white); AAbb (yellow); AABB (red); AABB (red);

mutant a is epistatic to b —> nonfunctioning mutation at locus A gives white regardless of locus B (always white)

? Is the same true of B? B depends on genotype at A (either yellow or white)

the epistatic locus is upstream in the pathway, changing precursor or action of other genes downstream.

that's an example of recessive epistasis

dominant is possible too! so now Aa - - would also be white! (a > A)

Epistasis vocab (know definition, but not worry about crosses on test) * **suppressor** (mutant that suppresses effects of other mutant making it wt) * **synthetic lethals** (two mutants @ different genes each nonlethal together lethal) * **penetrance** (% individuals with the phenotype) - e.g. mutant that makes you inefficient at getting water from soil will show low penetrance in an irrigated system * **expressivity** how much expressed (little effect on drought tolerance or lots) varies among individuals

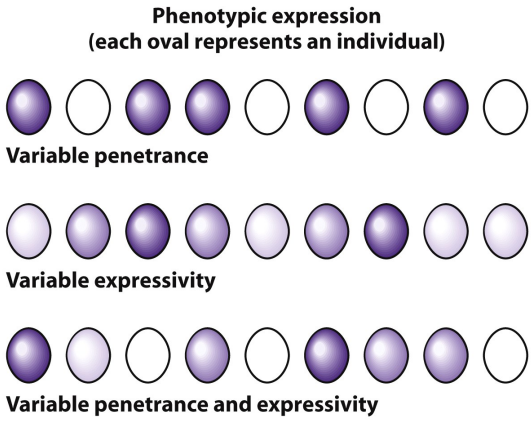


Figure 6-22
Introduction to Genetic Analysis, Tenth Edition