BEHAVIORAL GENETICS

Phenotype

Genotype

Gene

- locus
- allele
- mutation
- dominant/recessive
- homozygous/heterozygous

Structural Gene – transcribed into messenger RNA that are in turn translated into a protein

Regulator Gene – transcribed into RNA that in turn turns other genes on & off

Pleiotropy – one gene, many traits

Polygenic – many genes, one trait

Epistasis – action of one gene is modified by one or more other genes that assort independently (i.e., reside at different loci in the genome).

Epigenesis – the developmental process that includes sequential interacts between genes and their environment (nature + nurture) that leads to phenotypic expression.

Examples:

Regulator gene in fruit fly

Elaborate courtship ritual: male chases, licks, & strokes the female; vibrates his wings to produce a "love song". A single gene (Fruitless), 70X larger than most, regulates other genes, governs male sexual behavior, functions in only 1% of fly's brain cells - in clusters implicated in courtship

Mutations to this gene:

attracted to males

blocks song

prevents successful copulation

Single gene, pleiotrophic effects in rodents

"Knockout" mice that lack a gene for an enzyme (protein kinase C epsilon), **PKCε**, which diminishes the sensitivity of particular GABA receptors in the nervous system. GABA is an inhibitory neurotransmitter.

Mice without **PKCε** experience greater inhibition in particular GABA pathways, resulting in enhanced calmness and exploratory behavior when placed in stressful, unfamiliar mazes than controls. They also had lower blood concentrations of stress hormones.

Injections of a substance that obstructs GABA-receptor activity rendered the knockout mice as fearful and cautious in novel mazes as mice with intact **PKCε** gene. [GABA depletion is linked with anxiety disorders]

Small genetic change for speciation in Monkeyflower plants

species1 – pollinated by hummingbirds (large red flowers and much nectar)

species2 – pollinated by bees (small pale flowers and modest nectar)

Gene x Environment interaction

Major Depression

Affects 1 in 10 adults

Causes profound changes in:

- mood (aggression, suicide)
- energy levels (e.g., respiration, body temp, cardiovascular activity, metabolism)
- sleep patterns
- appetites (e.g. food and libido)
- interests (cognitive impairment)

Caused by low levels of serotonin, a neurotransmitter of a fundamental neural pathway that works as a neural modulator

Environment - Can be triggered by:

- chronic stress (e.g., long-term threat, loss, social separation, humiliation, defeat)
- changes in background levels of hormones (e.g., pregnancy, postpartum)
- changes in photoperiod

Genotype - functional polymorphism in the promoter region of the serotonin transporter gene (5-HTT)

Two forms (alleles) of the gene:

- Long allele (l) - higher transcriptional efficiency
- Short allele (s) - lower transcriptional efficiency

Using a large sample (1036 children) of long-term tracked (26 years) cohorts

Sample genotyped (ss = 17%; sl = 51%; ll = 31%)
Sample assessed for last five year and past year depression

Significant relationship between ‘s’ carriers, stressful events, and depression
non-significant relationship between ‘l’ carriers, stressful events, and depression

G x E interaction: ‘s’ carriers more susceptible to depression-induced stress.

(Caspi et al. 2003, Science 301, 386-389)

Treatment for Depression:

- Correct the genetic causation (gene replacement??)

- Correct the deficiency in serotonin
  - Selected Serotonin Reuptake Inhibitors (SSRIs)
    - e.g., prozac, paxil, zoloft, paroxetine
  - brain imaging shows
    - Chronically depressed patients have smaller hippocampi than non-depressed people
    - Antidepressants protect the hippocampus from shrinking

- Correct the behavioral manifestations
  - "Talk" therapy (e.g. cognitive behavioral therapy)
  - Brain imaging shows heighten front brain activity under the influences of CBT, whereas SSRIs appear to work at a more primitive level (e.g., hippocampus)

NATURE/NUTURE CONTROVERSY

HERITABILITY

That part of phenotypic variability which is genetically based (i.e., the capacity to be inherited)

How to measure heritability?

$\mathbf{V_T} = \mathbf{V_G} + \mathbf{V_E} + \mathbf{V_I}$

All phenotypic variance of a population = Variance due to genotype + Variance due to environment + Interaction of $V_G \times V_E$

$H^2 = V_G / V_G + V_E$

H^2 can vary between 0 (no genetic influence)
and 1 (complete genetic influence)

METHODS OF ANALYSIS

SELECTIVE BREEDING

(selectively increase homozygosity)

Low High

TRAIT (phenotype)

Mate lows to lows (low line)

and highs to highs (high line)

(selectively increase homozygosity)

Low High

TRAIT (phenotype)

What if both low & high line generated same curve?

INBREEDING (genetic homozygosity)

$V_G \approx 0$, so $V_T \approx V_E$

TWIN STUDIES

($V_G = 0$, so $V_T = V_E$)

CROSS-FOSTERING

($V_G \approx 0$, so $V_T \approx V_E$)

STRAIN DIFFERENCES

(V_G known, but hold $V_E \approx 0$)

MUTATION

(V_G known, hold or alter V_E)

Small genetic changes can yield RIM

Example: SIBLING SPECIES OF LACEWING

Chrysopa carnea (Cc)

Chrysopa downsei (Cd)

Cc - breeds all summer (constant, long daylength

stimulates breeding with no diapause

Cd - early spring breeder (short → longer daylengths necessary to avoid dispause. Under constant light cycle (16:8 L:D), Larvae go into diapause and no not metamorphose or reproduce

[what kind of reproductive isolating mechanism?]

In lab can manipulate light to crossbreed the species

F_1 Cc X Cd = 100% phenotype of Cc

F_1 X F_1 = 7% (males & females) Cd phenotype (≈ /16)

F_1 X Cd = 25% (males & females) Cd phenotype

[Genetic nature of controlling mechanism?]

Cc [AABB → AB gametes]; Cd [aabb → ab gametes]

Cc x Cd F_1 [AaBb → AB, Ab, aB, ab gametes]

Cross F_1 with F_1

	AB	Ab	aB	**ab**
AB				
Ab				1/16
aB				with Cd phenotype
ab			**aabb**	

Backcross F_1 with homozygous recessive Cd

	ab
AB	
Ab	1/4
aB	with Cd phenotype
ab	**aabb**

Must be homozygous recessive at BOTH loci

to express *C. downsei* phenotype

HYBRIDIZATION EXPERIMENTS

Bentley & Hoy used songs by two crickets:

Teleogryllus oceanicus

Teleogryllus commodus

Conclusion

There must be a genetic linkage between
song pattern generator and pattern detection.

Possible pattern detection mechanisms by female nervous system may be:

1. Neurons acting as FILTERS - receptors "tuned" and only pass on temporally patterned sensory input of a particular set of characteristics

2. CNS has a stored "template" against which the sensory input is compared (a recognition when a fit is achieved)

3. A combination of the two

Genetic Imprinting (where an allele at a locus silences the other)

Sexual conflict in mice

(Male fitness) paternal gene attempts to program female for large placenta and huge pups

(Female fitness) maternal gene attempts to cancel paternal effects where minimum energy investment per pup is best for her (< placenta and < pups)

lgf2 – growth factor (paternally active)
lgf2r – degrades growth factor (maternally active)

2 close species (1 polygynous, 1 monogamous):
polygynous males X monogamous females = 6x placenta size & huge pups

polygynous females X monogamous males = small placentas and pups