**SPECIES CONCEPT (part of the MODERN SYNTHESIS)**

**Main papers:**

Mayr, E. 1976. Evolution and the Diversity of Life. Harvard University Press, Cambridge, Massachusetts. 🡨 **Biological species concept** - defined by reproductive isolation

Suzanna White, John A.J. Gowlett, Matt Grove. 2014. The place of the Neanderthals in hominin phylogeny. Journal of anthropological Archaeology 35: 32-50. 🡨 tl;dr gene flow or hybridization between sapiens and Neanderthals != conspecificity.

Mayr, E., 1996. What is a species, and what is not? Philos. Sci. 63, 262–277. 🡨 **Mosaic evolution** – different traits evolve at different rates

Complications/challenges to the biological species

- **hybrids** between species can allow gene flow between species (violating reproductive isolation, e.g. sapiens and Neanderthals (White et al., 2014)

- **ring species** (like the herring gull and lesser black backed gull);

- **species complexes** (like the at least 6 treefrog species in the Hypsiboas calcaratus-fasciatus complex)

- **sibling species** (like the African forest elephant and the bush elephant)

- **asexually reproducing populations**.

Taxonomy of extinct species relies largely on morphology, despite an imperfect relationship between morphological and genetic differences (i.e., convergence; accumulation of genetic difference prior to morphological change). See White et al (2014, p. 34) for a list of species criteria applied to extinct forms and **Appendix A for a table of different species concepts and their dis/advantages**.

Holliday, T.W., 2003. Species concepts, reticulation, and human evolution. Curr. Anthropol., Anthropol., 653–660. 🡨 argues that **hybridization is not a challenge to species categorizations** if one defines species as lineages that ‘remain cohesive despite occasional genetic exchange.’

Jolly, C.J., 2001. A proper study for mankind: analogies from the papionin monkeys and their implications for human evolution. Yearbook Phys. Anthropol. 44, 177– 204. 🡨 **Genes acquired through hybridization have a better chance of being advantageous** than genes that result directly from mutation, as they have already gone through natural selection in their original genome

**MODERN SYNTHESIS:** Mendelian genetics meets Darwinian selection (1930s-40s)

Haldane, Fisher, and Wright: the forefathers of Neo-Darwinism

Haldane J.B.S. 1929. The orgin of life. The rationalist annu. 3, 3–10. 🡨 **Primordial Soup theory**; his work on population genetics in the early 20th century helped to create the modern synthesis of evolutionary theory (bridging genetics and natural selection).

**GENE-CENTERED VIEW:**

\*\*\*Natural selection helps those genes that help themselves\*\*\*

Kimura, Motoo. 1983. **The neutral theory of molecular evolution**. Cambridge University Press. 🡨 Kimura thought that, at the molecular level, most mutations that were retained in populations (and not selected out) were neutral with respect to fitness (partly because of synonymous codons), and that their frequency was largely determined by drift.

Does this theory have any consequences for the visible products of evolution?

**GROUP SELECTION AND ALTRUISM:**

Wynne-Edwards, Vero. 1962. Animal dispersion: in relation to social behavior. Edinburgh. Oliver and Boyd. **🡨** arguing for **group selection** as an explanation for the reproductive restraint observed in most species (e.g., why does a flicker only lay 6 eggs in a clutch, when experiments have shown that she is capable of laying 71 eggs if her eggs are removed every day?). But data on gene flow between local populations makes this explanation untenable – groups are not bounded in the way they would need to be for a self-damaging/group-benefitting allele to drift into high enough frequency.

Answer: reproductive restraint is an illusion. Lay more eggs and you don’t necessarily get more surviving offspring. The most common clutch size will therefore (because of the effects of natural selection) be the most successful clutch size. 🡨 Wilson, Margot and Daly, Martin. 1983. Sex, Evolution, and Behavior. Willard Grant Press.

Hamilton , W.D. 1964. The genetical evolution of social behaviour. Journal of Theoretical Biology. 7 (1): 1–16. 🡨 i**nclusive fitness** – the representation of an individual’s genes in the future gene pool, rather than the number of offspring born to that individual. This is the only direct measure of fitness – most measures of ‘fitness’ are degrees removed, even ‘mating success.’

Dawkins, Richard. 1976. The Selfish Gene. Oxford University Press 🡨 **Altruism can be explained by inclusive fitness**, the ‘gene’s eye view.’

**EVOLUTION OF SEX:**

**Why sex?**

Not just the **Red Queen**. Facultatively sexual species turn to sex when **the future is uncertain**; when offspring will disperse into other environments (according to George C. Williams). 🡨 Williams, George C. 1966. Adaptation and Natural Selection. Princeton University Press.

In some species, individual growth takes the place of cloning (as they expand into their environments, e.g. trees), and they only reproduce when they are about to send offspring further afield into uncertain environments – hence, sexual reproduction is always optimal and becomes canalized. This theory only works if most offspring do not survive, and only those few who are by chance well adapted to their territory survive and ultimately procreate – otherwise sexual reproduction does not have the advantage over asexual.

KOMODO DRAGONS ARE FACULTATIVELY ASEXUAL! (all offspring from asexual reproduction turn out male, unlike female whiptail lizards, who are obligate asexuals and produce only females. Fascinating. (Watts, Phillip C.; Buley, Kevin R.; Sanderson, Stephanie; Boardman, Wayne; Ciofi, Claudio; Gibson, 2006)

Advantage of sexual reproduction: Muller’s ratchet

Hermann Joseph Muller – mutations accumulate in asexual lineages, and sexual recombination breaks them up, avoiding the accumulation of fitness-lowering mutations at high frequencies in the population.

Evidence seems to be limited and mixed about the combined effects of multiple mutations – additive, synergistic epistasis (deterministic mutation hypothesis, Kondrashov – each mutation has an outsized effect b/c of its combination with other mutations), or each additional mutation may have a disproportionately small effect.

Geodakyan’s evolutionary theory of sex? Developed in Russia sometime between 1960-80, but only known in the ‘west’ after the internet. Suggests that males and females represent a phenotypic partitioning of the species, with males as the more variable, higher risk set of phenotypes and females as the stable phenotype of the species.

Repair and complementation theory? Maybe sex’s major function is the repair of double-stranded DNA breaks via meiotic recombination. The masking of deleterious recessive mutations would be an added benefit (outcrossing is necessary for this, as inbreeding is much closer to clonal replication and will amplify deleterious recessives), and genetic variation is a side effect of this.

Libertine bubble theory??? Maybe sex evolved in proto-eukaryotes to solve the problem of massive amounts of DNA (not a problem had by bacteria) – additive weak advantages of recombination, meiosis, gametogenesis and syngamy result in selection for sexual reproduction. Asexual eukaryotes may have evolved more recently, due to the conflict of interest inherent in anisogamy (parental investment conflict). Does this make any sense??

**SEX RATIOS, MATING SYSTEMS, SEXUAL SELECTION**

R.A. Fisher – **why a 1:1 sex ratio**? Females are the necessary sex, and multiple females can be mated by one male. Why produce so many males? B/c if you’re a mother, you might want your offspring to be male b/c that will maximize your inclusive fitness. But with so many males in the population, not every male gets to mate – so males are a high-risk, high-reward offspring for a mother. If either sex were rare, natural selection would favor it (b/c it gets more genes into the next generation) until ratios were equal again. Parents should, on average, invest equally in the production of each sex – an equal sex ratio results when males and females are equally costly. Sex (male/female) is subject to frequency-dependent selection in a population, generally converging on a 1:1 ratio.

Female mammalian **parental investment** = internal fertilization, gestation, placentation, lactation. This leads to different constraints on reproductive capacity between mammalian males (extra matings with fertile females) and females (time and calories).

Trivers, Robert and Willard, Dan. 1973. Natural selection of parental ability to vary the sex ratio of offspring. Science 179:10, 90-91. 🡨 natural selection should favor **parental ability to adjust the sex ratio** of offspring produced according to parental ability to invest. Data from mammals support the model: As maternal condition declines, the adult female tends to produce a lower ratio of males to females.

**Bateman’s principles**:

Bateman, A.J. 1948. Intra-sexual selection in Drosophila. Heredity 2: 349-368 🡨 drosophila experiments show differences in male/female mating strategies and reproductive variance:

1. Male RS increases with successive matings; female RS does not

2. RS will show greater variance for males than females

3. sexual selection will affect the sex with greater variance in RS more than the other sex.

\*replace ‘male’ with ‘fast sex’ (ugh, this is the ‘promiscuous male and choosy female’ argument)

**Here, let me fix that for you:**

Brown GR, Laland KN, Borgerhoff Mulder M. 2009 Bateman’s principles and human sex roles. Trends Ecol. Evol. 24, 297–304. 🡨 **humans are unlikely to conform to Bateman’s principles**, or indeed any one universal pattern of mating behavior, due to our adaptations for plasticity.

**MATING SYSTEMS:**

Weatherhead, Patrick J., and Robertson, Raleigh J. 1979. Offspring quality and the polygyny threshold: the “sexy son hypothesis.” American Naturalist 113:2. 201-208. 🡨 **Polygyny threshold**: monogamy is expected when mate quality does not vary or monopolizing more than one mate is not possible (maximally constrained polygamy). Polygyny is expected when mate quality/resource holding is variable, and sharing a high quality/wealthy mate is a better economic situation than monopolizing a poorer quality mate.

12 years after Origin of Species, Darwin published the theory of **sexual selection**. He distinguished it from natural selection (the environment) because the two forces could, theoretically, work in opposition to each other. But both ultimately select for reproductive success.

Zahavi, Amotz and Zahavi, Avishag. 1997. The Handicap Principle: A missing piece of Darwin’s puzzle. Oxford University Press. 🡨 The **handicap principle = costly honest signals** and the roles they serve (largely in service of sexual selection, sometimes (like stotting) for predator avoidance).

**LIFE HISTORY THEORY:**

Improves upon r-selected vs. K-selected models. r strategist: maximizes rate of population growth ( r ) whenever possible. E.g., mice. K strategist: maximizes survival/reproduction in situations at/near carrying capacity, when r is 0. Quality rather than quantity. E.g., elephants.

Trivers, Robert L. "**Parent-Offspring Conflict**". American Zoologist. 14 (1): 249–264. 🡨 ‘any adaptation that enables the child to succeed in this conflict by extracting extra parental investment cannot evolve if it is to the detriment of the parent’s inclusive fitness…. Any gene that helps a child to get more than its share of parental investment will then be passed on to that same child’s offspring, whereupon the child’s inclusive fitness will suffer as its mother’s did.’

**FROM GENE TO PHENOTYPE:**

**Heritability**: Low heritability may suggest canalization due to strong selection pressures; or, it could suggest that adaptive modification of the trait in response to environmental variables has been selected for. \*High heritability does not mean that environmental factors can have little effect.\* (Wilson and Daly)

Novel environment – what about invasive species? Are they not experiencing novel environments? Novel environments are not always by definition going to be detrimental to an organism, otherwise we create a theoretical tautology. Novel environments can uncover hidden variation, expand plasticity through exercising the range of a reaction norm, and encourage increases in intelligence by rewarding solutions to new problems.

However, over time, optimal becomes average; any time the needle moves and the environment is a little different, larger numbers of individuals will fall outside the optimal, and it takes time for selection to re-position the trait mean for the population.

**EVOLUTIONARY PSYCHOLOGY**:

Richerson, Peter J., and Boyd, Robert. 2004. Not by Genes Alone: How culture transformed human evolution. University of Chicago Press.

What is evolutionary psychology? Evoked vs. epidemiological culture.

Because a general-purpose learning mechanism will be inefficient (is this an assumption or can it be proven?), **special-purpose cognitive modules** will be favored for species that face the same adaptive obstacles for many generations. Evidence: small children appear to be born with preconceptions about physics, biology, and social dynamics, and these preconceptions influence how they pick up information about the world. E.g., language acquisition is only possible because children have an idea of how grammar and syntax should work, and can slot available data into an existing framework. Chomsky argued for an innate universal grammar – evolutionary psychologists think this sort of universal specialized module is present for many (most) domains. \*Personally I buy that the tabula is not rasa, but I think that a general-purpose brain that is specialized for plasticity and experiential learning will conquer most problems. Some ev psych ideas are worth examining. Most cannot be adequately tested. Proponents take the logic of their approach too far (but shhhh you can’t say any of this). \*

Jablonka and Lamb say it better anyway:

Ev Psych and Mental Modules:

‘culture as a colorful and thin veneer spread upon genetically selected, innate, human-specific, psychological mechanisms.’

Do we see human behavior as the result of naturally selected independent mental modules (‘mini-computers’) for behaviors that were adaptive in the Pleistocene? Or the result of selection for plasticity coupled with a system of symbolic communication? This puts more importance on ecological variation and social environments in shaping cultural variation.

How far can cultural transmission go without genetic change?

Do we have a “language module?” Do we have a “literacy module?” Langugage is universal and human specific. Its structure is complex. It is acquired early and without conscious effort. There are brain defects that can affect specifically language abilities. But we know that we do not have a “literacy module” – literacy is a cultural practice generally acquired during development that makes use of preexisting mental adaptations. Although such abilities may be the result of direct genetic selection, they do not need to be.

Universality of behavior does not need to mean genetics – it could mean universality of experience. E.g., Gilbert Gottlieb showed that ducklings vocalize while in the egg, learn the sound of their own voices, and so can recognize the calls of their own species – it is not a genetic ‘instinct.’ Perhaps the same is true for human universal behaviors?

Who is it good for? ev psychologists look for the individual (or gene); memeticists say it is the cultural traditions themselves. These authors (Lamb and Jablonka) think we must also ask how and why new variation is generated, how it develops, and how it is passed on, because all of these will inform the current picture of behavioral diversity and the potential patterns for future cultural evolution.

Neo-Darwinian explanations do not incorporate the importance of cultural networks for the meaning, invention, regeneration, and preservation of ideas.

We can talk about ‘cultural evolution’ despite this complexity (no good unit of inheritance, meaning only within social contexts, reconstruction affected by developmental environment) in the same way that we can talk about ‘biological evolution’ despite the complexity of gene networks/epigenetics – the frequency of behaviors/genes in a population do vary from one generation to the next.

But how does cumulative culture fit into a model that dismisses the role of the social transmission of information (beyond its role in filling the innate modules)?

“A Darwinian account of culture does not imply that culture must be divisible into tiny, independent gene-like bits that are faithfully replicated. Rather, the best evidence suggests that cultural variants are only loosely analogous to genes. Cultural transmission often does not involve high-fidelity replication; nor are cultural variants always tiny snippets of information. Nonetheless, cultural evolution is fundamentally Darwinian in its basic structure.”

**Forces of cultural evolution**: cultural mutation (e.g., misremembering); cultural drift (e.g., forgetting how to make pottery b/c pottery specialists die); guided variation (nonrandom changes); biased transmission – content-based bias (catchiness or adaptive value), frequency-based bias (conformity bias or rarity bias), and model-based bias (who are you copying? Success bias, prestige bias, self-similarity bias). Natural selection – for culture, this can occur at individual or group levels.

Since **cultural variants (alleles) do not sit at a locus, they need not be mutually exclusive.** People can learn, retain, and selectively employ multiple variants (oh hey, code switching!). Competition between variants is determined by the cost of learning each one (e.g., which PhD do you get? You’re probably only going to get one….).

**Culture has no recessive genes** – if it is not expressed as a behavioral phenotype, then it is not transmitted.

**DEFINITIONS OF CULTURE**:

‘the handing down of learned behaviors’

- non-human animals have this

- learned at what level? What about intra-uterine biasing of food preferences (rabbits with juniper or humans with carrot juice) – is that culture?

- examples: capuchin ritual interactions (hand sniffing, eye poking etc, as proof of trust and tolerance?); meerkats teaching scorpion hunting to their offspring

- what about behavioral imprinting? Is this culture? It is learned during a crucial period and can’t be unlearned (fly away home, are you my mama?)

Boyd/Richerson (Not by Genes Alone) say culture is any transmitted information.

Jablonka and Lamb (E4D) say it is ‘a system of socially transmitted patterns of behavior, preferences, and product s of animal activities that categorize a group of social animals.’

- not species-specific – non-human animals have this too.

- emphasizes the gestalt of the cultural system, rather than cultural memes

**Memes** (Dawkins) work from a gene’s eye view, but not from a network perspective (genetic networks are the next step after the ‘modern synthesis’) – with complex ideas, what is the unit of inheritance? (transmission vs. reconstruction)

Culture and biological reproduction are both forms of **information transmission.**

- is biological transmission unbiased?

-- meiotic drivers say no, not entirely

-- but mutations get copied regardless of their effects on phenotype

- is cultural transmission unbiased? Definitely not

**Transmission Biases**:

* Content bias: some aspect of the information makes it more memorable/likeable. Pascal Boyer (book= Religion Explained). Minimally counterintuitive ideas (magical realism?)
* Conformity biases: monkey see monkey do; do as others do
* Rarity/nonconformity bias: Do as others aren’t doing
* Self-similarity (in-group) bias: do what your friends do
* Prestige bias: do what famous people do
* Skill bias:
* Success bias:

Evolution is a process of information transmission. Reproduction is a transmission of information, somatic cell division is a transmission of information, breastfeeding is a transmission of information.

Claude Levi-Strauss – cultural anthropologist – ‘bricoleur’ = ‘tinkerer’ with the idea of cultural change (cultural evolution) – analogue to the bio-evolution metaphor of ‘evolution as tinkerer’

**Culture-gene feedback** – learning biases/preferences can influence cultural transmission, which can in turn influence certain genetic preferences/biases. A certain cultural environment can influence a reaction norm (life history strategy). E.g., a reaction norm responding to the harshness of the environment – others in the environment are choosing a fast life history strategy, that can serve as input for an individual developing their own life history strategy. Using another’s reaction norm as a cue/input for developing your own reaction norm.

Social Coordination Norm (Efferson et al., 2015)– leads to binary expressions of a trait across multiple populations with a ‘threshold tipping point’. When a trait shows intermediate frequencies, it may be more subject to frequency-dependent selection pressures. Frequency-dependent selection suggests that any individual shift in strategy/trait will have population-level effects.

**The Baldwin effect** – Genetic Assimilation?

Does genetic assimilation work best for evolved psychology? Initial abilities for plastic response to a changing environment allow the creation of a new behavioral phenotype. If this environment remains stable for a long period of time, a function-specific module may be more efficient to face this environmental challenge than pure plasticity…. But does selection for levels of plasticity operate faster than selection for more structural changes? I guess if environment remains stable, then selection for high plasticity may relax, and when environments become mutable again, individuals who retain high plasticity will be selected. Plasticity has a cost. If plasticity extends the phenotype in an adaptive direction and then a genetic underpinning arises to support that phenotype, plasticity can relax and diminish its own costs while the organism maintains optimal fitness.

This is not the most parsimonious thing though, and Jerry Coyne lambasts Dobbs for his belief in in it (David Dobbs mucks up evolution)

* Baldwin, J. Mark; [*A New Factor in Evolution*](http://www.brocku.ca/MeadProject/Baldwin/Baldwin_1896_h.html), The American Naturalist, Vol. **30**, No. 354 (June 1896), 441-451

**DARWIN’S THEORY OF NATURAL SELECTION**:

Darwin studied medicine and theology at Edinburgh and Cambridge, but we know him from his time as a naturalist on the HMS Beagle (1831-1836).

1. All species are capable of producing offspring faster than the food supply increases (from Malthus’ work on demography)
2. All living things show variations; no two individuals of a species are exactly alike
3. Because more individuals exist than can survive, there is competition for resources to sustain existence. Not everyone can survive. If those variations that exist between individuals affect the likelihood of survival, then some individuals are more likely to survive than others.
4. These favorable variations are inherited and tend to be more likely to be present in the next generation, just because their bearers are more likely to survive and reproduce.
5. Over long periods of geological time (from Lyelle, geologist), these successful variations produce great differences that result in new species.

\*make this into a diagram\*\*

The loops of the HPA Axis: Hypothalamus (in the brain) CRH signals the anterior pituitary (on other side of blood-brain barrier). AP releases ACTH which is read by the adrenal cortex, and adrenal cortex releases corticosteroids, which are read by the hypothalamus, which can then downregulate its production of CRH – Negative Feedback mechanism!! (i.e. thermostat) HOMEOSTASIS.

**EPIGENETICS:**

Epigenetics: heritable phenotypes not encoded in the genome, including microbiome, immune priming, cortisol diurnal curves….

Recognized mechanisms of epigenetic regulation in mammals include DNA methylation, post-translational modification of histones (nucleosome packaging), chromatin remodeling, microRNAs, and long noncoding RNAs

As we age we see not just methylation differences by cell type, but an accumulation of methylation markers. These vary by individual life course, and can vary quite a lot between monozygotic twins at later ages. Methylation is transmitted from cell to cell as they divide – this is part of why differentiated tissue stays differentiated. But methylation marks are more subject to copying errors than DNA bases are.

**Random epigenetic drift**: Cancer and epigenetics – cancer is uncontrolled cell replication – your own cells. Normally cell replication is limited by Concogenes and Tumor-suppressor genes. If these genes are methylated, hello cancer.

**Tissue Field Hypothesis**: Cells talk to each other to figure out when they should/should not divide. Interrupt this communication can lead to cancer; restoring it can cure cancer.

* Study: ‘Parental olfactory experience influences behavior and neural structure in subsequent generations.’ Mice conditioned to fear an odor – offspring fear this odor upon first exposure.
* Study: ‘Integration of maternal genome into the neonate genome through breast milk mRNA transcripts and reverse transcriptase’ (reverse transcriptase turns RNA back into DNA). Evidence that mothers are packaging up their own genes and passing them to offspring in vesicles. There is not evidence yet to tell whether these vesicles get integrated into the offspring genome or not, but it does present the possibility that this could happen.
* Microchimerism: breast milk contains pluripotent stem cells (Hassiotou et al 2012)
* Imprinting: epigenetics marks that signal whether a gene has come from mom or dad. E.g., **inter-genomic conflict – the different goals of maternal genes vs. paternal genes** that have been selected for. From dad’s POV, set up the offspring to take as many resources as possible from mom (assuming species where repeat paternity with the same partner is not a given/likely). Dad’s genes are going to try to upregulate those resource-gaining genes; mom’s genes are going to want to turn that off/down. Dysregulation, with both copies turned on or off result in misregulated in utero growth of the infant.
* Epigenetic systems are not copied as faithfully as genetic systems, so they have a higher degree of mutation – so higher rate of generation of novel variation that can be selected. Since it doesn’t change the structural proteins – usually just how much, and where, and how it’s regulated. They’re much less likely to result in catastrophic bad mutations – so higher chance of mutations that might be appropriate/neutral. Adaptation through selection of epigenetic variants might be quite rapid compared to evolution through selection of genetic changes.

E4D Ch. 4: The Epigenetic Inheritance Systems

* Cell differentiation is epigenetic (i.e., the result of changes during development), but differentiated cells produce daughter cells of the same tissue – they pass on their acquired characteristics, even though all somatic cells contain the same dna.
* **Self sustaining loops**: e.g., when one of the products of a gene is its own promoter
* Whereas the information in DNA is modular, the information in a loop (the productive region, the regulatory region, and the protein product) is holistic – the loop itself is the unit of heredity that is transmitted.
* **Structural inheritance**: cilia structure (Sonneborn et al., 1960s), changed by surgery, is heritable. Using structural inheritance to combat prions (disease organisms without dna/rna – made only of proteins).
* Prion example: kuru/mad cow disease (Fore people, New Guinea). Prions are abnormal proteins that can transform the normal form of the protein into the abnormal form as well, hence spreading the disease.
* Chromatin marks: methylation, acetylation, etc. These are disrupted with transcription, as the new strand of DNA does not carry chromatin marks, but marked positions seem to draw other copies of the same chemical groups to their matching loci on the new strand, seeding a similar pattern of methylation/acetylation in the daughter DNA.
* The RNAi system: micro RNAs, the cellular defense system, and effects on silencing mRNA in cells across the body

Changes through the epigenetic system

- occur more often than through the genetic system

* can occur several at a time
* are not blind to function (i.e., occur in genes that are turned on due to environmental input)
* = evolution through epigenetic inheritance is faster than evolution through genetic inheritance, and possibly more capable of building adaptations fast too.
* \*think about the importance of epigenetic changes in speciation – allopatric isolation, then reintroduction but inability to breed due to phenotypic differences (usually assumed to be due to underlying genetic differences – but maybe not! Maybe due to epigenetic differences)
* Incompatibility of epigenetic marks from mother and father may prevent an embryo from developing, even if individuals mate.

**Ch. 7 Interacting Dimensions – Genes and Epigenetic Systems**

* Genetic Assimilation: Does a popular version of a musical piece affect the selection of the version of the score, or the generation of variations in the score? Epigenetic systems could bias the generation of variations in DNA, or they could affect the selection of variants, or both. E.g., cancer – epimutation of regulatory genes leads to runaway mutation of genes, which could create mutation in chromatin producing genes, which could impact epigenetic reconstruction….
* Just like the risk of damage to your car is greater when you’re driving it than when it’s parked in the garage, active (unmethylated) genes mutate at higher rates than inactive genes.
* Epigenetic Inheritance Systems (EISs) allow cells to have memory and maintain their characteristics. This in turn allows for the evolution of complex development.
* In multicellular organisms, cells cooperate rather than compete, and mechanisms that stop them from competing (e.g., by keeping them differentiated into function-specific types) will be selected for because they result in a more efficiently functioning organism. Cells that disregard the needs of the body and divide selfishly may destroy the whole (e.g., cancer). The need for germ cells to be free of an epigenetic legacy may be the reason why many animals separate germ cells and somatic cells This way germ cells have little epigenetic memory to erase before the next generation, (because they divide infrequently throughout development) and less opportunity for epimutations to arise.
* Many aspects of development (e.g., efficiency of cell memory, stability of cell death among somatic cells, segregation of somatic and germ cells) are evolved mechanisms that prevent the inheritance of epigenetic markers that might destabilize the organism in the next generation.
* Genomic Imprints:
* Genes ‘know’ whether they come from the mother or the father, and respond to cellular signals differently (e.g. Angelman’s syndrome). This may be the result of difference in chromatin packaging marks (sperm are small and tightly packaged; eggs are larger, with diffuse chromatin and active genes).
* Dosage compensation:
* Females inactivate one of their X chromosomes (at random) to make up for the fact that Y chromosomes have much less protein-coding DNA. In embryo-derived tissues surrounding the fetus it is always the father’s X chromosome that is inactive – mechanism of fetal tolerance? Apparently parental imprints can be ignored in some tissues and exploited in others…. Enter Haig, fetal-maternal conflict. Alleles inherited from dad should have growth-enhancing marks; alleles from mom should have growth-suppressing marks. This has been observed in some (but not all) growth-related genes in mice and humans.
* Induced Epigenetic Changes:
* Belyaev’s domesticated fox experiments. He thought that the stress of domestication activated dormant genes, resulting in expression of greater variation in the population.
* C.H. Waddington (of epigenetic landscape fame): wild-type variants are canalized/buffered because of past selection for gene combinations that produce the same outcomes, so many genetic knockout experiments do not change the phenotype (any stable system has redundancies built into it). Mutant strains that have not faced natural selection to stabilize their development remain variable.
* Nature article, 1942 – Canalization of Development and the Inheritance of Acquired Characters – outlines genetic assimilation. Waddington also did the heatshock broken veined drosophila experiment, showing how a phenotypic response to environment could become a genetically encoded trait (selection for hidden variation in a population). Epigenetic events reveal hidden variation, and sexual recombination creates a new phenotype (a new reaction norm?).
* Epigenetics fell out of favor in the 60s/70s with the rise of molecular biology. (selfish gene) The new discovery of mechanisms made abstract biological theory seem unnecessary.
* Suzanne Rutherford and Susan Lindquist – HSP90 regulates protein folding (by being tolerant f variation in its target protein sequences), thus stabilizing development. Inadequate supplies of HSP90 allow for developmental trajectories to ‘wobble.’
* Moral of the story – once hidden genetic variation is exposed due to environmental effects, selection can act on the new phenotype.

**What is a gene?**

A gene is a unit of particulate inheritance. **It is a theoretical object**, not a physical object, because it contains DNA, regulatory elements, machinery for reading (mitochondria etc, the chefs of the recipe), epigenetic marks.

\*Glucocorticoids are probably key to many reaction norms, because they regulate responses to stressors and mobilize energy\*\*\*\*

**Epistasis**: genetic context clues.

‘To draw on P.Z. Myers' apt analogy, epistasis means that single genes often carry little more inherent significance than individual playing cards do in poker. In a poker hand, the significance and effect of a two of hearts — its ‘trait’ — depend so heavily on the other cards you’re holding that it’s almost meaningless to say the card has any replicable power on its own. It’s replicable in that it’s a two of hearts every time it’s dealt. But it can deliver the same effect in subsequent generations only if it’s dealt not just into the exact same handful of cards, but into a round in which all the other players at the table also hold the same cards as before — and happen to bet, hold, and fold in exactly the same way. Not something to count on.’ – from Die Selfish Gene Die, David Dobbs 2013

**Reaction Norms and Gene Expression:**

Selection on the Reaction norm (**evolutionary change through development**)

Does Domestication cause changes in growth reaction norms? A study of farmed, wild and hybrid Atlantic salmon families exposed to environmental stress.

* When you stress out wild salmon with crowding (by lowering water levels in a tank), they grow to much smaller sizes. Farmed salmon (which are larger than wild counterparts overall) show less shrinkage in stressed, crowded conditions – they’ve been bred to grow large even in crowded conditions. Size has been more canalized in farmed salmon – there’s no selection to maintain plasticity.

Lysenko’s ‘cold-resistant’ wheat: cold temperatures affect the VRN1 gene, which suppresses vegetative growth and promotes flowering – environmental input affects gene expression.

The evolution of ageing: mutation accumulation, antagonistic pleiotropy, and disposable soma theory.

**EXTENDED EVOLUTIONARY SYNTHESIS:**

Includes:

- multilevel selection

- transgenerational epigenetic inheritance

- niche construction

- evolvability

- OR evolutionary developmental biology (a different synthesis, b/c they felt the extended synthesis focused too much on genetics at the cost of developmental plasticity)

**JABLONKA AND LAMB – EVOLUTION IN 4 DIMENSIONS**

**Analogy for genetic and cultural inheritance**:

Genotype = musical score; Phenotype = each performance of the piece

Recording technology – allows new performances (phenotypes) to be partly influenced by old performances (phenotypes), in addition to the musician’s training, the instrument, the general musical culture etc.

Particularly popular performances (phenotypes) may even result in notated scores (genotypes) to allow the easier reconstruction of the popular performance (phenotype). But the way that past performances influence future performances is nothing like the way the musical score influences each performance.

Ch. 5 **The Behavioral Inheritance Systems**

Socially mediated learning = ‘a change in behavior that is the result of social interactions with other individuals, usually of the same species.’

1. transfer of behavior-influenceing substances (e.g. juniper berries and carrot juice)

2. socially mediated learning (e.g. tits opening milk bottles; Israeli black rats moving into pine forests and learning to strip pine cones)

3. imitation – e.g. songs of birds, whales, and dolphins are learned through individual imitation, resulting in regional ‘dialects’ through drift. Motor imitation is important in human learning, but has less evidence in other animals (maybe b/c sampling bias?). Unlike other forms of BIS, imitation transmits modular information, allowing for partial variants – but remember, copying of variants is not content-blind (and many variants are not functional).

\*these are not mutually exclusive\*

The transmission of information depends on both the transmitter AND the receiver – the receiver must be able to select, generalize, categorize, reconstruct, and adjust the behavior that it has observed. And the behavior must be displayed in order to be transmitted.

What about cumulative evolution in animals?

Koshima study – Japanese macaques, Imo and the sweet potato washing which led to a whole seaside lifestyle. The transition was complex, gradual, and cumulative, with behaviors in one domain influencing and reinforcing behaviors in another – but it did not lead to linear accumulation of complexity.

**Ch. 6 The Symbolic Inheritance System (the 4th dimension)**

Man the symbolic animal (Cassirer, 1944).

‘words act as symbols because they are part of a rule-governed system of signs that are self-referential.’ Signs have a one-to-one truth value. Symbols can be remixed and made metaphorical, refer to things that are present or absent, hypothetical or false. Symbols’ meanings depend both on the relations they have to the way objects and actions in the world are experienced by humans, and the relations they have to other signs in the system. A symbol cannot exist in isolation, because it is part of a network of references.

Images can do the same (e.g., Christ on the cross).

Symbols, like genes and unlike behaviors, can transmit latent information.

Why do humans engage in active teaching? Because the symbolic systems must be culturally acquired.

**Models of cultural evolution: ev psych vs memeticism**

Meme (Dawkins) – ‘the new replicators’ – “a unit of cultural inheritance, hypothesized as analogous to the particulate gene, and as naturally selected by virtue of its ‘phenotypic’ consequences on its own survival and replication in the cultural environment” (The Extended Phenotype, p.290

Memes are problematic b/c you can’t separate the replicative unit from its phenotypic development, so it’s hard to actually define any particular meme. Dawkins is fond of separating ‘replicators’ and ‘vehicles,’ but if the vehicle acquires traits through its development that it passes on, then it is no longer strictly a vehicle, and this compromises the definition of a meme.

E.g., the transmission/replication of a nursery rhyme depends on the contents of the rhyme, its melody, how many times we/others repeat it, when we learned it, our own and the child’s musical talent, the child’s motivation, etc. Transmission/acquisition is sensitive to the behavioral-developmental history of both the student and the teacher. Introducing a variation on the rhyme that you know results in a transmission of this ‘phenotypic’ variation – so even mechanical imitation of memes is not equivalent to gene transmission.

E.g., tits opening milk bottles – the phenotypic behavior is milk bottle opening, but this is reconstructed from a piece of information, milk bottle = food source. There is no ‘meme for milk bottle opening,’ just a reconstruction of a behavior based on ecological information.

E.g., Christ on the Cross – there is no ‘crucifixion meme’ because what is reconstructed from the symbol is a complex cultural-religious package that has to be learned through a developmental process involving several levels of organization (family, society, church), and the crucifixion references all of that, but does not hold all of that meaning for anyone who does not have that developmental/social background, so a ‘crucifixion meme’ is a gross and meaningless oversimplification.

**Final word on memes**: “The distinctive feature of human culture is its potent constructive power, which includes the ability to design and plan the future, and its coherence and internal logic. Symbolic communication allows humans to communicate ideas and artifacts that are constructed to deliberately shape their future within a very complex social and political system. Thinking of the spread of human habits and ideas in terms of the replication of selfish memes obscures these unique aspects of human evolution.”

Ch. 8 – **Genes and Behavior, Genes and Language**

\*Is behavioral assimilation an argument in favor of evolutionary psychology?

Does selection for fast learning lead to just instinctual behaviors, or instinctual behaviors and sophisticated behaviors layered on top of instinct (b/c of space freed up on the hard drive)? (Assimilate and Stretch)

HBE:

Production:

- When/why do men and women favor different productive tasks?

Bliege Bird et al 2009 – production inequalities among the Martu foragers of Western Australia result from stochastic variation in men’s foraging success on large prey vs women’s consistent production of small, low-variance resources.

Codding et al 2011 – when possible, men and women pursue low-risk, high-yield resources. But when there is a tradeoff between risk and yield, men pursue high risk high yield resources for provisioning other adults, while women focus on low risk resources in order to provision offspring. Tested with the Ache, Martu, and Meriam.

Hilton and Greaves 2008 – “It appears that the consistent and large returns of female foraging underwrite the large energetic effort of men’s hunting.”

Panter-Brick 2002 – Beware of overly simplistic gendered divide models. note that, with ‘the sexual division of labor,’ women are almost universally not big game hunters – BUT in the Agta of the Philippines women do engage in bow and arrow hunting of wild pig and deer.

**EEA** is, by definition, the set of circumstances to which we are adapted – abstract invariances - *The “environment of evolutionary adaptedness” (EEA) is not a place or a habitat, or even a time period. Rather, it is a statistical composite of the adaptation-relevant properties of the ancestral environments encountered by members of ancestral populations, weighted by their frequency and fitness-consequences.* – since it relies on invariance, parts of the environment that are variable do not fit into this model, and instead selection for plasticity may have triumphed. ‘Mismatch’ is not a necessary conclusion for behavior in these realms. 🡨 Bowlby J. (1969). Attachment. Attachment and loss: Vol. 1. Loss. New York: Basic Books.

Tooby, J. & Cosmides, L. (1992). The psychological foundations of culture. In J. Barkow, L. Cosmides, & J. Tooby (Eds.), The adapted mind: Evolutionary psychology and the generation of culture.New York: Oxford University Press.