Evolution in Four Dimensions

Chapters 7-10

Outline by John Protevi of
Eva Jablonka and Marion Lamb, *Evolution in Four Dimensions: Genetic, Epigenetic, Behavioral, and Symbolic Variation in the History of Life* (MIT, 2005)
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Chapter 7: Interacting Dimensions – Genes and Epigenetic Systems

I) Introduction
- A) Concept of genetic assimilation will be important
- B) Two ways epigenetic dimension can influence genetic dimension
  1) Generation of variation
  2) Selection bias

II) Effects of Epigenetic systems on generation of genetic variation
- A) Recap of previous points about DNA
  1) DNA is always packaged in chromatin, often with methyls
  2) Epigenetic factors influence gene expression (high methylation tends to turn off genes)
  3) Epigenetic marks reconstructed after DNA replication (e.g., mitosis in somatic cell lineages)
- B) Epigenetic marks affect probability that a region will undergo genetic change
  1) Types of change
     a) Mutation
     b) Recombination
     c) Transposons ("jumping genes")
  2) Change more likely in low chromatin / high gene activity regions (but there are exceptions)
- C) Significance of epigenetically-influenced genetic changes for development / evolution
  1) Development: cancer
  2) Generation of mutations in germ-cell lines
     a) McClintock and stress-induced transposons
     b) Transposons correlated with low methylation
  3) Are such changes adaptive?
     a) Or simply "parasitic" / "selfish" genes?
     b) Plants are able to take advantage of such stress-induced transposons due to modularity
  4) These transposons can lead to mutations by messing around with gene regulation
  5) But in general "we do not know full evolutionary significance of effects of EIS on mutations"

III) How EISs have molded evolution of development
- A) EIS role in cell memory is precondition for evolution of multicellulars
  1) w/o cell memory, uncontrolled cell type switches can hurt organism efficiency
  2) so you need evolution for EISs allowing flexibility and reliability
- B) Evolution of EIS role in development so gametes have neutral state allowing cell differentiation
  1) Locked-in cell types cannot become gametes, so strong EIS prevents danger that way
  2) Reason for early germ-line segregation in animals
  3) Reprogramming in meiosis and gamete production

C) Genomic Imprints and Gene Selection
    1) Parental marking on inherited chromosomes affect gene expression
    2) Originated as incidental by-products of different DNA packaging in gametes
    3) Developmental functions
        a) Sex determination
        b) "dosage compensation"
            i) Female inactivation of one X chromosome
            ii) In extraembryonic tissues, it's always father's X that is inactive
                (a) Haig's hypothesis to account for this phenomenon
                (b) Countering "greedy" embryos requires strong parental markers
        c) Possible means for fathers to influence daughters (sons receive only Y from fathers)

IV) Induced Epigenetic Variations and the Selection of Genes
A) Russian silver fox experiment:
    1) Domestication changes phenotype: behavior, hormones, morphology, and chromosomes
    2) Belyaev's interpretation
        a) Stressful situations allow "dormant" genes to become activated
        b) Phenotypic changes are due to epigenetic rather than genetic change
        c) JL: cf. McClintock, who posits genomic change (transposons) due to stress
    3) JP: stress makes unexpressed variation be now exposed to selection
        a) But what is "unexpressed variation"?
        b) It's variation at the level of networks of regulation of gene expression
        c) NOT at level of structure of DNA sequences (McClintock's transposons)
        d) So we have to think the "virtual" being of such "unexpressed" variation
B) Genetic Assimilation: How Interpretation Selects the Score
    1) Stress-induced epigenetic changes can guide selection of genetic variants
        a) Waddington provided a Darwinian explanation for Lamarckian IAC
        b) Background:
            i) Most development is robust, despite genetic / environmental variation
            ii) But mutants can exhibit great phenotypic variation
            iii) Because the hadn't been subject to NS which selects for developmental "canalization"
        c) Canalization presupposes "invisible" (= shielded from NS) genetic variation
        d) Stress reveals this variation (exposes it to NS,)by allowing it to produce new phenotypes
        e) So canalization has a double effect
            i) In normal circumstances it masks variation and allows it to accumulate
            ii) In stress it allows development of new phenotypes exposed to NS and thus new evolutionary potential
                (a) JP: but what exactly is "masked"?
                (b) It is "unexpressed genetic variation"
                    1. But is that structural genetic variation (different DNA sequences)?
                    2. Or is it functional genetic variation (networks of gene regulation)?
                        a. If the latter, then what's masked is the potential for forming new networks, which include cell conditions / enzymes, etc
                        b. E.g., JL p. 268: selection "brings together" previously hidden [and previously independent!] genetic variations
                            i. But what is ontological status of this potential to form new networks out of previously independent elements?
                            ii. Here I think Deleuze's virtual can help
    2) Waddington called his process "genetic assimilation"
        a) Recurrent X-gen environmental stresses produce recurrent epigenetic changes

b) Individuals with genes enabling quick and easy production of those epigenetic changes thrived and those genes spread, so that most members of the population now need only slight environmental input
c) Eventually, the phenotype is genetically assimilated
d) NB: West-Eberhard writes at 448 of her review that this is better version: "an induced epigenetic change that occurs repeatedly can guide selection on phenotypes that favor genes that bias development toward production of same phenotype"

3) So big question, post Molecular Revolution: what are mechanisms of genetic assimilation?

C) Genetic Assimilation Meets Molecular Biology
1) Revival of Waddington
a) Ecological biologists looking to phenotypic plasticity
b) Evo-devo interest in epigenetics and regulation of gene expression
c) Possible mechanisms for genetic assimilation
2) Rutherford, Linquist et al.
a) Protein folding sometimes needs "chaperones," like Hsp90
b) Dual function of Hsp90
i) Normal conditions: stabilizes proteins regulating development by helping them fold
ii) Stress conditions: helps protect / restore damaged proteins
c) Experimental results show Hsp90 is developmental buffer, masking genetic variation

D) A Revealing Yeast Prion

V) Epigenetic Revelations
A) Summary of main points
1) Belyaev: hidden genetic variation revealed by domestication
2) Waddington:
a) Developmental canalization through NS of buffering combinations
b) Stress pushes development out of canals and produces different phenotypes which expose previously hidden genetic variation to new round of NS
c) Genetic assimilation: induced phenotypes can be produced w/o environmental induction
3) Lindquist group posits Hsp90 as buffering mechanism
4) Rudner suggests heritable epigenetic variation
5) Prions may also be transmitted

B) Conclusions:
1) Epigenetic changes can reveal masked (due to developmental canalization) genetic variation
2) Genetic assimilation is Darwinian explanation of supposedly Lamarckian evolution
a) Lamarckianism requires IAAC (inheritance of acquired adaptive characters)
b) But not all environmentally induced phenotypic plasticity is adaptive
3) Inheritance of induced epigenetic changes: new dimension to evolution
a) Additional source of variation
b) Arise during changing conditions, when variation is most important
c) Reversibility of epigenetic variations is quicker and easier
d) Can do a "holding job" until genes catch up (W-E: genes are followers, not leaders)

VI) Dialogue

Chapter 8: Genes and Behavior, Genes and Language

I) Introduction
A) Enviroment's role in evolution
1) Traditionally: selection of [mutation / sexual recombination] variants
2) Generation of developmental variants [phenotypic / developmental plasticity]
a) Ch 7 considered morphology
b) Ch 8 looks at behavior

B) Niche-construction: organisms affect environment; thus selection pressures for self and X-gen

II) Genes, Learning, and Instincts
- A) How to explain evolution of instinct by NS?
  - 1) Learned behavior becomes innate
  - 2) Sexual selection works alongside NS
  - 3) Baldwin effect, as read through Waddington's genetic assimilation
    - a) Presupposes unexposed genetic variation (this time for learning, not morphology)
    - b) Environmental changes expose differential learning capacities
      - i) These learning capacities can be selected
      - ii) And thus the genetic components of that learning capacity can be assimilated
- B) Expanding the Repertoire: The Assimilate-Stretch Principle
  - 1) Previous treatment:
    - a) Selection for learning undermines learning by replacing it w/ instinct
    - b) But this presupposes a stable environment
  - 2) Changing environment: leads to selection for behavioral / learning flexibility
  - 3) Consequences of genetic assimilation:
    - a) Assimilate-stretch principle: adding behavior to instinct frees learning and creates possibility of cumulative sophisticated behavior
    - b) Categorization:
      - i) Full assimilation might lock in a specific identification
      - ii) Partial assimilation might allow for categorization

III) Cultural Niche-construction
- A) X-gen transmission of changed environment via learning co-implicates genes and behavior
- B) Durham and co-evolution of genes and culture
  - 1) Dairy practices and lactose / lactase
  - 2) Slash & burn agriculture and mosquitoes / malaria / sickle-cell
- C) Tay-Sachs
  - 1) Evolution of TB-fighting allele due to ghettoization of Jews?
  - 2) Now, the inverse: presences of Tay-Sachs allele changing cultural practices re: screening
- D) Problem
  - 1) If culture is constantly changing niche and hence conditions for genetic NS, is there time for genes to ever catch up?
  - 2) JP: maybe NS for genes for general intelligence / flexible learning / cortical plasticity?
  - 3) IOW, instead of assimilation of instincts, assimilation of loss of instinct?
  - 4) So human nature is to be open to culture as our second nature? (cf. Wexler)

IV) What is Language?
- A) Three schools of thought
  - 1) Chomsky and generative grammar / innate UG / "language organ"
  - 2) Functionalists: language as product of general cognitive mechanisms
  - 3) Daniel Dor: constrained communication / core set of semantic categories in all languages
- B) How Language Changed the Genes
  - 1) Dor and Jablonka: language = co-evolution of genetic and cultural inheritance systems
    - a) Niche-construction and genetic assimilation both play role
    - b) Adaptive innovations must meet two constraints
      - i) Good for communication (blend of ambiguity and precision)
      - ii) Easy to learn, remember, use
    - c) There will have to be genetic component for neural / vocalization changes
  - 2) Key for Dor and Jablonka: *partial* genetic assimilation (308)
  - 3) JP: at some point we have to acknowledge linguistic exuberance / excess / poetry!

V) Dialogue
- A) Moderate modularity, not "massive"

B) Selection for both plasticity and resilience / canalization / buffering
    1) Both involve masking / unmasking
    2) W-E: "gen. accommodation" = gen. stabilization via selection of new phenotypic responses
C) Culture / gene co-evolution of language
    1) "limited emergence" (vs. full blown sudden "language organ" appearance)
    2) Gossip and recursion

Chapter 9: Lamarckism Evolving: The Evolution of the Educated Guess

I) Introduction
    A) Recap of important points
        1) Multiple inheritance systems
        2) Variation can be targeted and constructed
        3) Adaptations can be re-purposing of existing mechanisms selected for other functions
II) The Origin and Genetics of Interpretive Mutations
    A) Two views on stress-induced mutagenesis
        1) Pathological, not adaptive
        2) Adaptive (shaped by NS)
    B) Mechanisms of stress-induced mutagenesis
        1) SOS response system
        2) E. coli: making appropriate guesses (which part of genome to allow mutations in)
        3) Hot spots in pathogenic micro-organisms
    C) Evolution of stress-induced "interpretive" mutagenesis
        1) By-products of emergency DNA repair systems
        2) Common and random DNA changes
III) The Origin of EISs and the Genetics of Epigenetics
    A) Role of EIS in ancient unicellulars
        1) Allows switches among heritable states
            a) Faster rate than genetic mutation
            b) Readily reversible
            c) Functional link to changing environment
        2) Adaptation to predictable aspects of environment: e.g., cycles
            a) Long-lived organisms can adapt physiologically / behaviorally
            b) Short-lived organisms can change genetically
            c) Mid-term organisms (ratio of generation to environmental cycle):
                i) Epigenetic inheritance / cell memory tailored to environmental cycle
                ii) NS can hone in on appropriate fit of epigenetic change to "" "" ""
    B) Four types of EIS
        1) Self-Sustaining Loops
            a) Gene expression and cellular conditions form distributed network
            b) Stuart Kauffman and self-organization of such networks
                i) Multiple stable configurations
                ii) Flexibility vs. resilience maximized at "edge of chaos"
        2) Structural Inheritance
            a) Lindquist lab and genetic basis of protein-templating
            b) Cavalier-Smith and "genetic membranes"
        3) Chromatin Marking
            a) Methylation:
                i) Today it is part of a "genomic immune system"
                ii) Probably originated for stabilizing gene regulation
                    (a) And was recruited for defense

(b) Only after development of protein marking
b) Protein markings
i) Originally transmitted by accident
ii) NS would select favorable changes
c) Repeated DNA sequences
4) RNA interference
a) Evolved as genomic immune system
b) But perhaps regulatory functions are even older?

IV) The Origins of Animal Traditions: Selection for Social Attention and Social Learning
A) With social learning, other animals are part of environment in which learning happens
B) Problems with asocial learning
1) High cost of mistakes
2) Difficulty in learning w/o help from others

V) What is needed for the Evolution of Communication through Symbols?
A) Bonobo research: learned symbol use (not training!)
B) Two conditions for evolution of hominid symbol use
1) Altered ecological / social environment: leaving forests for savannah
2) Anatomical / physiological changes allowing vocalization

VI) Transitions on the Evolution Mountain
A) This is a classic anthropocentric / progressive image, but authors are aware of this
B) Evolutionary changes via new types of information transmission
C) Comparison with Maynard Smith and Szathmáry
1) MS and S: focus on changes in genetic system; no room for instructive processes
2) JL: focus on new types of inherited information as crucial factors in evolution (EIS, BIS, SIS) and on "educated guesses" / "interpretive" variation

VII) Dialogue
A) Evolution of evolvability still controversial
B) Why not see all this just as temporally extended phenotypic plasticity of same genotype?
1) But recognizing relative autonomy of other ISs for full view of evolution is important
2) Phenotypic continuity preceded and probably formed basis of genetic system
a) Self-sustaining chemical cycles
b) Structural templating
C) Surplus! (351)

Chapter 10: A Last Dialogue

I) Four common objections
A) It's all genetic at base
B) Epigenetic inheritance is unimportant to both evolution and development
C) You haven't shown Lamarckism really works
1) Definitions:
a) Narrow definition: feedback from soma to genes
b) Broader definition: "soft inheritance"
2) Why not just avoid "Lamarckism" terminology?
D) Comparative question:
1) Two versions
a) Broad question: what is relative importance of each system
b) Narrow question: what is genes vs culture for humans
2) Discussion of "heritability"

II) Practical implications (364)
A) X-gen effects of nutrition

- B) Agriculture
- C) Cloning

III) Ecology and niche-construction
- A) Margulis
  1) Multicellulars as community
  2) Phenotypic continuity is essential to ecological webs
- B) Gaia

IV) Political / economic forces
- A) Behavioral / symbolic level: Sociobiology and memetics
- B) Spencer

V) Philosophical questions
- A) Allies
  1) DST
  2) Lewontin
- B) Reductionism and replicators
- C) Process-orientation

VI) Moral implications (379)