Evolution in Four Dimensions

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)
LSU French Studies
www.protevi.com/john
www.protevi.com/john/Morality/evolution4dimensions.pdf
protevi@lsu.edu

Chapter 1: The Transformations of Darwinism

JL = Jablonka and Lamb
Evolution = change in historical series of entities
NS = natural selection (mechanism of evolution)
ENS = evolution by natural selection
IAC = inheritance of acquired characteristics
EIHV = environmental induction of heritable variation
CM = chromosomal material

I) Intro: controversies in science and in biology
  A) Darwin had little to say about nature and causes of hereditary variation
  B) JL will trace history of gene-centered versions of Darwinism
II) Darwin's Darwinism
  A) Laws of Evolution by Natural Selection (ENS)
    1) Darwin's version of ENS
      a) Reproduction
      b) Inheritance
      c) Variability
      d) Struggle for Existence
    2) Maynard Smith's generalization of ENS
      a) Multiplication
      b) Variation
      c) Heredity
      d) Competition
    3) With Maynard Smith's generalization
      a) No need to know anything about
        i) Processes of heredity and multiplication
        ii) Origins of heritable variation
        iii) The nature of the entity that evolves through NS
      b) Thus it can in theory be applied to many systems (warning: many debates here!)
        i) Cosmology
        ii) Economics
        iii) Culture
  B) Darwin and heritable variation via "effects of life on organism and 'use and disuse'"
    1) Sounds like Lamarck
      a) Inheritance of acquired characteristics (IAC)
      b) Shown to be wrong by Darwin's theory of NS

2) But the usual story about Lamarck is wrong
    a) What's wrong about it?
        i) Lamarck was not a simpleton
        ii) Lamarck did not invent idea about IAC
        iii) Darwin had a role for "use and disuse" in his theory
        iv) Darwin's theory of NS did not displace IAC
    b) What's the real story?
        i) Lamarck was a sophisticated thinker and didn't solely focus on IAC
        ii) Almost all biologists believed in IAC throughout 19th C
        iii) Darwin believed in IAC
        iv) NS was not the reason IAC was displaced

C) Darwin's theory of heredity: pangenesis
    1) Gemmules as units of heredity and development
        a) Tiny particles spread throughout body
        b) In sexual beings, gemmules accumulate in reproductive organs
            i) Gemmules join in sperm and egg before development
            ii) Offspring are a blend of parental characters
            iii) What is inherited is the actual character
            iv) Some gemmules are dormant and awake in later generations
            v) "inheritance = form of growth"
    2) But what accounts for variation, since with blending, you should get uniformity?
        a) The theory: environmental change can "induce" variation
            i) Change in nutrition / climate affects growth
                (a) Alters proportion of gemmules in reproductive organs
                (b) Awakens dormant gemmules
            ii) Changed conditions change gemmules themselves
        b) The result: Darwin's theory allows for IAC

D) Conclusion by JL: EIHV does NOT weaken evolution by NS
    1) In fact, EIHV increases
        a) Amount of variation
        b) Scope of NS
    2) Generality of theory of evolution by NS is not limited to
        a) Any one theory of mechanism of heredity
        b) Any one theory of causes of variation

III) Weismann's Neo-Darwinian Theory: Acquired Characteristics Discarded

A) Three great advances in mid-19th C biology
    1) Cell theory (Virchow)
    2) Evolution by NS (Darwin)
    3) Refutation of spontaneous generation (Pasteur)

B) Linking cell theory to heredity / development / evolution (Weismann)
    1) Once chromosomal material was identified as hereditary / developmental substance
    2) Then mitosis must be distinguished from meiosis
        a) Mitosis: division of ordinary [eukaryotic] cells:
            i) Doubling then division of chromosomes
            ii) Splitting of cell into two daughter cells
            iii) Each daughter cell
                (a) Inherits one half of the doubled material
                (b) That is, a full batch of chromosomal material (CM)
            iv) But that creates problems for reproduction
                (a) If sperm and egg are produced by mitosis
                (b) Then each have full complement of CM

(c) And the fertilized egg would have double amount of CM!
b) Meiosis: "reduction division" in production of sperm and egg
i) Each daughter cell (sperm or egg) receives only half CM
ii) So the fertilized egg will have the normal full complement of CM

C) Weismann rejects IAC
1) No way for properties of somatic cells to be transmitted to sperm or egg cells
2) Thus we have the continuity of the germ plasm
a) Segregation of germ plasm early in development for separate production of sperm / egg
b) Partial inheritance of "determinants" as answer to cell differentiation
i) Each embryonic cell receives a different part of nuclear material
ii) So the nuclear material should be getting simpler as cell differentiation proceeds
iii) Only the germ plasm in germ line retains full complement of determinants

D) Source of variation for Weismann
1) Sexual reproduction involves mingling germ plasm from parents
a) Which means there's a long history of mingling from ancestors
b) Now half of CM eliminated in meiosis is not same for every sperm / egg
c) Thus we have rich source of variability from mingling and meiotic "reshuffling"
2) But what was original source of variation?
a) Random accidents alter determinants
b) Leading to "germinal selection" via changes in nutrition / temperature etc.
i) Environment has heritable effects via direct action on germ plasm
ii) Unit of selection issue:
(a) Not just individual organism
(b) But determinants in germ plasm
(c) Cells w/in a tissue
(d) Groups

E) Summary: differences btw Weismann and Darwin
1) Weismann gives NS an exclusive role, rejecting IAC
2) Weismann had different hereditary theory (determinants vs gemmules)
3) Weismann source of hereditable variation via effects on determinants in germ line
4) Weismann focused on sexual reproduction as producing heritable variation

IV) Doubts about Darwinism
A) Neo-Lamarckians:
1) Progressive / goal-directed evolution
2) Herbert Spencer: evolution in many developmental processes / belief in IAC
3) Lacked a good theory of heredity

B) Gradualism vs discontinuous evolution
1) Can Darwin's notion of gradual evolution through selection of small variation account for continuously varying traits or must we think in terms of saltatory "sports"?
2) What about speciation? There doesn't seem to be continuity here, but gaps!
a) De Vries proposes "mutation" as accounting for sudden, discontinuous speciation
b) So many evolutionary biologists at this time didn't bother with either Lamarck or Darwin

V) The Modern Synthesis: Development Vanishes
A) Synthesis of Weismann's ultra-Darwinism and Mendlian genetics
B) Theory of heredity in the Modern Synthesis
1) Gene as "hereditary unit of biological information" determining development
2) Alleles: different versions of each gene (inherited from each parent)
3) Mendel's laws
a) Two alleles separate in formation of gametes in same condition as entering parent body
b) Alleles belonging to different pairs segregate independently
4) Dominants and recessives: hybrids do not show intermediates

5) Genes were located on the chromosomes, likes beads on a string

C) Mendelian genetics based on analysis of (visible) differences
1) At first, genetics seemed to reinforce non-Darwinian discontinuous evolution
2) But later it was shown genes can account for continuous variation
a) When characters are controlled by many genes
b) Each having a small effect

D) Mendelian genetics has no explanation for development
1) Genes located only in nucleus: ignored role of cytoplasm
2) Morgan and *Drosophilia*
3) Johansen
a) Phenotype / genotype distinction
i) All individuals in a pure line have same genotype
ii) Phenotype depends on interaction of genotype and environment
b) Genes
i) Pass on "potential for characters"
ii) Unit of information about potential phenotype
iii) Unaffected by use of that information in development
iv) Very stable, though open to occasional mutation

E) ENS according to the Modern Synthesis
1) Heredity =
a) Transmission of germ-line genes
b) Located on chromosomes (nuclear focus)
c) Discrete unit of information about character
2) Variation =
a) Consequence of random combinations of alleles through sexual reproduction
b) Each allele has small effect on characters
c) Mutations in genes are result of accidents
d) Genes are not affected by their use in development
3) Selection
a) Occurs at phenotype level
b) Alleles accumulate in population through phenotypic selection

F) Complaints by embryologists and plant biologists
1) Nuclear genetic material is not the only important thing
2) Cytoplasm is important for both heredity and development

VI) Molecular Neo-Darwinism: The Supremacy of DNA

A) Triumph:
1) DNA steps to the fore as mechanism of heredity and development
a) Hereditary gene = nucleotide string
b) Developmental gene = protein synthesis (via mRNA)
2) "Central dogma" = unidirectional information flow

B) Needed nuances
1) Non-nuclear inheritance in cytoplasmic organelles (mitochondria / chloroplasts)
2) Breakdown of one-to-one allele / protein relation
a) Many allelic variations can produce same protein
b) And many differences in amino acid strings in proteins persist in population
c) Thus many differences in proteins and alleles are "selectively equivalent"
3) "Junk DNA" or non-coding DNA
a) Some non-coding DNA is regulatory (controls gene expression) but not all!
b) Hereditary information becomes genetic program, not just genes as discrete units

C) ENS according to the Molecular Revolution
1) Gene = unit of heredity = DNA string

2) Inheritance = DNA replication
3) Cytoplasmic organelles have own DNA
4) Mutations = changes in DNA sequence via
    a) Mistakes in DNA replication
    b) Chemical / physical damage to DNA w/ improper repait
    c) Movement of DNA itself
    d) Mutagens do not increase adaptive variations
    e) So induced variation is blind / random

VII) Selfish Genes and Selfish Replicators: unit of selection controversy

A) Altruism
1) Explanation through group selection
2) Attack on group selection by mathematical arguments
    a) At first successful
    b) But then others claim math proof of possibility of group selection
3) Kin selection: Hamilton

B) Dawkins and the "gene's eye view" or "selfish gene"
1) Genes are units of inheritance *and* selection (bcs of stability and permanence)
2) Replicators vs vehicles
3) Replicators can unify biology, bcs they are units of
    a) Heredity
    b) Variation
    c) Selection
    d) Evolution
    e) Development
4) They can even unify biology and sociology / psychology if we accept "memes"

C) Attacks on Dawkins
1) Defenders of other targets of selection (individuals / kin / groups) still are gene-centered
2) In that they agree that hereditary genes are in control of development
3) Gould: gene-centered evolution is just "bookkeeping"
    a) Individuals / groups / species are targets of selection (bcs they survive / reproduce)
    b) NS is not the only agent of evolution
        i) Historical events (climate changes)
        ii) Accidents affecting genetic variation
        iii) Evolution is constrained by development (if organism isn't viable, it can't reproduce)
        iv) Side effects or "spandels" are possible (adaptationist debate)
4) Gould and Dawkins agree
    a) Gene-centered heredity relevant to non-human organisms
    b) No such thing as IAC

VIII) The Transformations of Darwinism

IX) Dialogue

A) A-Life discussion: "limited heredity systems"

B) Dawkins
1) Unit of heredity / selection:
    a) Dawkins: replicators must have high replication fidelity, which individuals don't have
    b) Response: no one said individual was unit of heredity and selection: it's the trait
2) Relation of inheritance and development
    a) Dawkins: assumes unidirectional influence from replicators to vehicles, but not inverse
    b) Response: development does impinge on heredity
3) Unit of heredity
    a) Dawkins: the gene is only biological hereditary unit
    b) Response: there is epigenetic inheritance

C) Molecular revolution
    1) It's progress to be able to speak at the molecular level
    2) But there are physiological and behavioral levels of heredity as well (not just cytoplasmic)
D) Ideology
    1) Surely the Modern Synthesis isn't ideological like Lysenko?
    2) Of course, but "ideology" can be "assumptions" from "socio-political general worldview"

Chapter 2: From Genes to Characters

I) Intro
    A) Relation of genes and development is very important today
    B) JL forecast:
        1) Epigenetic inheritance is important too
        2) Question of information in different inheritance systems:
            a) What kind of info is transmitted
            b) Mechanism of info transmittal
            c) Extent and fidelity of info transmittal
            d) Effects of transmitted info
II) From DNA to Proteins
    A) Heredity: DNA replication is a property of the cellular system, not of DNA alone
    B) Function: DNA codes for proteins
        1) Transcription = splitting of double helix and production of primary RNA transcript
        2) Splicing = introns and exons
        3) Transport = exit from nucleus and entry into cytoplasm
        4) Ribosome = site of protein production
        5) Translation = formation of a polypeptide chain (amino acids)
    C) Noncoding DNA
        1) Regulation of gene activity
        2) But the cell is involved in this regulation process, which means environment contributes too
III) Digression: What is Information?
    A) Two types of DNA info
        1) Coding for proteins
        2) Attachment sites for regulatory molecules
    B) JL definition of information:
        1) Correlation of changes in receiver's functional state with form and organization of source
        2) This is "interpretation"
        3) Source remains unchanged by such interpretation by receiver
    C) DNA information
        1) Characteristics:
            a) Linear and modular
            b) Replication is insensitive to content (vs. learning)
        2) Consequences:
            a) A lot of raw material for NS can be generated
            b) But "nonsensical" DNA can also be generated and transmitted
IV) Genes, Characters, and Genetic Astrology
    A) Monogenic diseases (one-to-one gene / trait determinism)
        1) Are quite rare
        2) But are a popular model for *all* gene action in development (= "genetic astrology")
    B) Correlation is not causation in complex systems
V) The Tangled Web of Interactions
    A) Example of APOE gene and coronary artery disease

- B) Conclusions
  - 1) Cannot just add average effects of genes in a population and predict individual profiles
  - 2) Defeat of genetic determinism
    - a) Developmental plasticity = many phenotypes from "same" genotype
    - b) Canalization = same phenotype from "different" genotype
- C) Waddington and the epigenetic landscape
  - 1) Developmental canalization
  - 2) Multiple genetic effects
- D) Knockout gene experiments often produce no phenotypic differences
  - 1) Reasons:
    - a) Duplicate genes
    - b) Functional replacements
    - c) Dynamic regulatory networks can adapt
  - 2) Results
    - a) Demonstrates structural and functional redundancy in genome
    - b) Developmental canalization
    - c) Selectively neutral alleles
    - d) It's the "evolved network of interactions" that accounts for canalization
      - i) NB: genetic regulatory network (includes epigenetic factors) is unit of evolution
      - ii) Not the hereditary gene as DNA sequence

VI) Genes in Pieces

- A) Splicing:
  - 1) Excision of introns and splicing of exons
  - 2) The limits of introns and exons are not fixed
  - 3) The decisions are made by the network
    - a) Developmental (cytoplasmic & intrasomatic) & environmental (extrasomatic) conditions
    - b) Regulatory genes
- B) Results: can no longer identify hereditary gene as locus of developmental control

VII) Changing DNA in Development

- A) This is "natural genetic engineering"
- B) Discussion of Weismann and chromatin diminution
- C) This discussion has been only about development of somatic cells
  - 1) Though it's fascinating that development can change DNA ("recipe")
  - 2) What about evolution?
    - a) Are there developmentally induced heritable changes to DNA in the germ line?
    - b) IOW, are there directed changes in variation for NS? This would be Lamarck's revenge!

VIII) Dialogue

- A) Language and DNA have same structure
  - 1) Modular
  - 2) Content-indifferent replication
  - 3) Encoded information
  - 4) Allows unlimited heritable variation
- B) Population averages mask individual variation, but organisms live as individuals!
- C) Problems with prediction:
  - 1) Hyper-astronomical number of permutations
    - a) Genetic interaction
    - b) Gene – environment interaction
  - 2) Cannot define "environment"
  - 3) Social environments are "partially constructed by individuals"
- D) Must shift focus to dynamic / flexible / fuzzy networks
- E) Focus on DNA alone is politically loaded

F) DNA focus allows fight against monogenic diseases
G) Effects on our thinking about evolution from focus on relation of genes / development
    1) Channels and limits our thought about evolutionary mechanisms so that we focus on "selection for the developmental, physiological, and behavioral stability and flexibility of genetic and cellular networks."
        a) Controversy over genetic determinism / selectionism
            i) Genetic determinism = thesis about development
            ii) Genetic selectionism = thesis about evolution
        b) Plasticity and canalization = network properties, not gene properties
    2) DNA can change in response to environmental cues
        a) Systems of nonrandom DNA change
        b) Formation of heritable DNA variation
        c) Transmission of such variation

Chapter 3: Genetic Variation: Blind, Directed, Interpretive?

I) Intro: John Cairns reintroduces question of directed (i.e., non-random) mutation
    A) In retrospect, Cairns was wrong with his example, but opened door to research on mutation
    B) JL will now claim that not all mutation is random, as previously believed
        1) Variation through sex
        2) Variation through mutation
        3) [NB: Lynn Margulis proposes symbiogenesis as major source of variation for evolution; mutations only modulate these major changes.]
II) Genetic variation through sex and sexual reproduction
    A) Sources
        1) Mixing genes from 2 nonidentical parents
        2) Meiosis: assignment of which chromosomes to gamete is random and independent
        3) Recombination of genes during "cross-over"
    B) What is the evolutionary advantage of sexual reproduction?
        1) Prevent accumulation of bad mutations
        2) Faster evolution in changing and selective / competitive environments
    C) Spectrum of modes of sexual and asexual reproduction: subject to evolution via NS
    D) Sex as genetic exchange is not always tied to reproduction (e.g., bacteria)
III) Variation through mutation
    A) Tradeoff between reliability and flexibility of DNA
    B) NS for "DNA-caretaker genes" for proof-reading and editing / correcting of DNA replication
IV) Randomness questioned
    A) Traditionally assumed that mutations are non-adaptive "mistakes," most of which are harmful
    B) Now, we have question of "directed mutation"
        1) We have seen developmental changes in DNA
        2) McClintock proposed genome as "organ of cell" that "responds … by restructuring genome"
        3) This is "stress-induced mutagenesis": affecting the maintaining / repairing system for DNA
    C) Thought experiment: three types of response to change
        1) Conservative:
            a) Heuristic strategy: Always and only try the traditional response
            b) Biological method: use physiological response and wait for lucky mutation at natural rate
            c) Prospects for success: works in slight changes, but in radical changes not very helpful
        2) Exploratory:
            a) Heuristic strategy: try whatever is imaginable
            b) Biological method: increase rate of random mutation
            c) Prospects for success: depends on population size

3) Interpretive:
    a) Heuristic strategy: stick to tradition when you can; cautiously interpret when you must
    b) Biological method: non-random, but not-precisely-directed mutation
    c) Prospects for success: seems to be good, as this strategy is selected for in many cases [?]

V) Acquired, Required, Interpretive Mutations
- A) Four types of non-random mutation
    1) Induced global mutation
    2) Local hypermutation
    3) Induced local mutation
    4) Induced regional increased mutation
- B) These fit into a spectrum between "blind" and "developmentally regulated" genetic change

VI) Evolved genetic guesses
- A) Can no longer clearly distinguish instruction (development) and selection (evolution)
    1) E.g., immune system changes are developmental and selective (Edelman)
    2) Some evolutionary change (e.g., bacterial) are instructive
    3) So Lamarck is back in the picture
- B) Development, heredity, and evolution are intertwined

VII) Dialogue
- A) Stress-induced increase in recombination in specific regions of chromosomes
- B) Central dogma outlawing of backtranslation (changes from proteins to DNA)
    1) Backtranslation is not necessary for many types of IAC, which do not involve amino acids
    2) Most cell responses to changed conditions target regulatory and not coding sequences
    3) Most altered proteins due to changes in splicing / translation, not in coding sequences
- C) So, most genetic change affecting gene expression
    1) Alter number of copies of genes
    2) Control sequences
    3) Gene location on chromosome
- D) Why not more directed mutation? Because educated guesses are better than pure instruction.
- E) How are induced adaptive changes possible, as they require phenotypic feedback?
    1) In bacteria, this is easy to imagine
    2) In complex multicellulars, it's very unlikely to have mechanisms for directed genetic change
        a) Bcs. of complex interactions, such genetic change = "random" phenotypic effect
        b) But not all inheritance is genetic!
        c) So we can have IAC in other inheritance systems
            i) Epigenetic
            ii) Behavioral
            iii) Symbolic

Interlude: Transition to Part II

I) Two problems
- A) Genetic system is thought to be sufficient
- B) Properties of genetic system are attributed to other systems

II) Analogy: genotype = score; phenotype = performance
- A) Transmission:
    1) Transmission of genotype: copying
    2) Transmission of phenotype: reproduction of interpretations of score
- B) Direction of effect

1) Usually only changes in score affect performance
2) But sometimes changes in performance (phenotype) affects score (genotype)

C) Consequences
1) Transmission systems have different technologies
2) Other systems complement but do not replace genetic system
3) Phenotypic variation / evolution can proceed independent of genetic change

Chapter 4: Epigenetic Inheritance System (EIS)

I) Cell differentiation must have a memory system, not just a genetic triggering system

II) Thought experiment: evolution via EIS w/o any genetic change: asexual reproduction

A) Double role of EISs allows for "directed or interpretive variation"
1) Response system
2) Transmission system

B) Discussion of thought experiment
1) Different use of same DNA: regulation of gene expression
2) Variants w/in each network of regulation of gene expression
a) NB: *not* a "regulatory gene network"
b) Bcs that implies it's only genes that do the regulating
c) Rather it's the distributed cellular system that regulates gene expression
3) EIS transmit
a) Interpretations of DNA information
b) Phenotypes rather than genotypes

III) Four types of EIS (all of which work together and interact: see p. 137)

A) Self-sustaining loops: memories of gene activity
1) Stability / instability
a) Simple loops can switch easily to alternate states (small threshold for perturbation)
b) Complex loops can be resilient / stable (large threshold for perturbation)
2) Type of information
a) Loop is unit of heritable variation, so information is "holistic" / nondecomposable
b) Versus the modularity of DNA
3) Amount of variation depends on number of interacting loops
a) A single isolated loop has two state: on or off
b) But with networks of loops we have lots of permutations / variation

B) Structural inheritance: architectural memories: cell structures
1) Ciliates: templates: variation in organization, not components
2) Cavalier-Smith and the "membranome" in early evolution
a) First true cells
b) Bacterial groups
c) Eukaryotic cell
3) Prions: self-templating proteins:
a) Diseases: kuru / CJD / BSE
b) Adaptive roles in multicellulars?
4) Information and replication

a) Holistic information
b) No special / content-insensitive replication: only specific reconstruction

C) Chromatin marking systems: chromosomal memories
1) DNA is tightly wound via "chromatin packaging"
2) Chromatin marks allow inheritance of pattern of gene expression
a) DNA methylation
i) Do not affect protein coding, but only regulatory sites, hence probability of transcription
ii) Part of heredity system transmitting epigenetic info in cell lineage
(a) Hitchhike on DNA replication during mitosis (= somatic cell lineages)
(b) But what about meiosis (= generation of gametes)?
iii) Content-insensitive copying / modular information
b) Protein complexes
c) Histone marks

D) RNA interference: Silencing of the Genes
1) Complex mechanism
2) Basic idea: part of cellular genomic immune system
3) Variations can have big developmental effects

IV) Examples of Epigenetic inheritance
A) Controversy: EIS between generations, not just in somatic cell lineages? (see W-E review)
1) Single celled eukayrotes: all 4 mechanisms in
2) Bacteria: Methylation marks
3) Multicellulars:
a) Asexual reproducers: no theoretical problem for X-gen EIS
b) Sexual reproducers: Fertilized egg must allow cell differentiation, so it must be neutral
i) So it was assumed epigenetic history was wiped clean before gamete production
ii) So "parental genomic imprinting" was big surprise:
(a) Different sexes produce different chromatin marks
(b) But these are transient: erased when chromosome passes to other sex
(c) So it doesn't seem good candidate for evolution

B) Examples of persistent X-gen EIS
1) Methylation
a) Silencing of transgene by inherited heavy methylation
b) Modification of patterns of ordinary gene activity:
i) Peloric plants: stable and transmitted "epimutation": different methylation of a gene
ii) Yellow mice: variation in methylation pattern on extra DNA from transposon
2) What about X-gen EIS by RNA interference?

C) Conclusion
1) EIS = another source of variation = another dimension of evolution
2) Epigenetic variation (EV) vs genetic variation
a) Occurs much faster, especially under changed environment
b) Several EVs may occur at same time
c) May not be blind to function, and hence increases chance of adaptive variation
3) Role in speciation?

4) EIS not just cellular: e.g., uterine environment among Mongolian gerbils

V) Dialogue
1) Non-DNA heredity may have preceded DNA system
2) Why include all 4 systems together? They all transmit "information"
3) These systems seem more developmental than evolutionary
a) These systems are both heredity and regulatory systems
b) Thus evo / devo / physio are linked
4) Organism level advantages are needed from evolution (target of selection)
5) Epigenetic variants passed to gametes?
a) Variant somatic cells can develop into gametes
b) Information exchange between somatic cells and germ line (RNA interference)
6) Variant somatic cell types as basis for X-gen development?
a) Gametes must have full developmental (cell differentiation) potential
b) But this doesn't exclude chromosome marks in egg that bias only some development
7) Cloning uses [already differentiated] somatic cells
a) Their specialization must be erased to allow full cell differentiation
b) Don't forget there are a lot of developmental errors in cloning
8) Reliability of epigenetic inheritance
a) Admittedly less than for genetic variations
b) But this low reliability can be supplemented by stable environmental induction
9) What exactly is transmitted by EIS?
a) P. 151: "components of an activity or a state that biases the reconstruction of same activity or state in next generation."
b) Thus we have to think in terms of reconstruction rather than copying (Very Important!)
i) Most likely with chromatin marks or RNA interference
ii) Other routes between soma and germ line: aren't they contra the central dogma?
(a) No, CD only forbids info from protein to DNA / RNA (reverse translation)
(b) Going from RNA to DNA (reverse transcription) is not a problem
(c) Info bypassing germ line (e.g., substances in mother's milk) is no problem
10) No direct evidence of adaptive X-gen epigenetic variation

Chapter 5: Behavioral Inheritance Systems (BIS)

I) Thought experiment: evolution via BIS w/o genetic or epigenetic change
A) Info must be transmitted and acquired so that behavior is reconstructed
B) Culture = "system of socially transmitted patterns of behavior, preferences, and products of animal activities that characterize a group of social animals"
C) Cultural evolution = "change, through time, in nature and frequency of socially transmitted preferences, patterns, or products of behavior in a population"
1) Mostly independent of genetic evolution
2) But the two systems can intersect

II) Transmitting information through social learning: 3 forms of BIS
A) Transfer of behavior-influencing substances
1) Means of transmittal

a) Uterine environment
b) Milk
c) Saliva / breath
d) Feces

2) Enables avoidance of trial-and-error learning
3) Information
   a) Holistic
   b) Transferred substance is building block enabling reconstruction of mother's behavior
4) Two further properties
   a) Non-parental transfer is possible (adoption)
   b) Usually non-blind variation:
      i) Info via acquisition and testing by mother
      ii) Variations due to her development and learning

B) Non-imitative learning: observation allowing reconstruction
1) Non-material information transfer: requires interpretation by recipient
2) Imprinting: usually there is a relatively early window for learning
   a) Behavioral imprinting
   b) Sexual imprinting
3) Attention is drawn to features of environment / outcome of behavior (goal, not method)
4) Information:
   a) Must be displayed (no latent info, as with genetic system)
   b) Holistic
   c) Never a random or blind variation (requires interpretation / reconstruction)
   d) Not limited to parental transmission
   e) Not many variants concerning any one behavior

C) Imitative learning
1) E.g., vocal imitation, as in birds, whales, dolphins
2) Information
   a) Must be displayed
   b) Modular, not holistic
3) Variants not blind to function
   a) Needs "internal filter" to identify potentially useful variants
   b) Variations are targeted and culturally constructed
      i) Simple rules to organize perceptions, emotions, learning (categories)
      ii) Type of info is structured by evolutionary history of its lineage
4) Recipients play active role:
   a) "niche construction": animal activity changes environment / selection pressures
   b) So one generation's actions in constructing a niche will bias info transmission / reception

III) Traditions and cumulative evolution: evolving new lifestyles
A) Obviously, we have to consider genetic basis of capacity for culture
B) There's no basis for assuming animal culture is limited in scope / complexity
1) There hasn't been a lot of research done here
2) Question of funding
   a) Molecular biology / biotech / corporation / grants system

b) Basic research vs applied research
c) Bayh-Dole Act and shift to "public-private partnerships" in biotech research
d) Public funding of infrastructure ("risk")
i) Buildings
ii) Education
e) Private appropriation of success ("profit")

C) Evidence of cumulative cultural evolution among nonhuman animals: Japanese beach monkeys

IV) Dialogue

A) Early experience is important, but not absolutely determining
B) Genetic predispositions vs learned preferences
1) Probably no visible difference in behavior
2) X-gen effects
a) Genetic info can be latent: it is passed on even w/o environmental inputs
b) Learned preferences only transmitted w/ environmental input
i) Thus the preference can easily die out
ii) What can account for cultural tradition then?
(a) Need stable environment for continued tradition (i.e., transmission of variants)
1. This can lead to "environmental addiction"
2. And degradation of genes for that preference
a. This is called "masking": stable environment hides genetic variation
b. IOW, you get successful phenotype even w/ degraded genome
(b) And / or variant is stabilized by links to other behaviors and / or environment
C) Targeted variations can be stabilized by "functional fidelity"
1) With genes, you need highly reliable replication of structural genes to conserve adaptations
2) But with other inheritance systems, you just need whatever works:
a) "content" doesn't need to be the same
b) Effects just need to be as good or better
D) Role of BIS in speciation
E) Altruism and NS
F) Horizontal transmission allows for bad behaviors to spread
G) Animals are not passive recipients of BIS!
H) Inseparability of heredity / evolution and development
I) Insect traditions?

Chapter 6: Symbolic Inheritance System (SIS)

I) Intro: discussion of symbols as key to human uniqueness
II) Thought experiment: Crusoe and the parrots
A) Similarities of parrot calls and human language
1) Signs are arbitrary
2) Signs are referential
3) Signs are conventional
B) Differences between parrot calls and human language
1) Parrots have poor repertoire

2) Sign system is rigid; each sign is a unit
    a) They don't generalize the properties of words and apply to new items
    b) They don't grasp relation between words

C) Humans: words are symbols bcs part of rule-governed system of self-referential signs
1) Can transfer truth-value, emotional value and action value to sentence level
2) Can not only go from situations to words, but from word combinations to situations
3) Rule-bound generation of variants

D) General properties of symbols
1) Interpreted w/in shared cultural framework of practices w/in which symbols function
2) Meaning of symbols depends on
    a) Relations of symbols to way culture experiences objects / actions in world
    b) Relations of symbols to other signs in the cultural system
3) Thus we have a "shared imagined reality"

E) JP: I would add that this treatment underplays the affective dimension:
1) Cultural practices teaching meaning of symbols w/in a system
2) Also shape the affective aspect of the "body politic"
    a) Patterns, triggers, thresholds of basic and social emotions
    b) This historically developed body politic is what generates moral intuitions

III) Symbolic communication as inheritance system
A) Shared structure w/ genetic system: transmission of latent information
B) Difference from genetic system: unlimited translatability into different media
C) New symbolic information is targeted
1) Fit with tradition
2) With an eye to innovation and readiness for future

D) Structure
1) Language is modular
2) Picture / dance tends to holism, but retains some modularity
3) Hierarchical organization

E) Many transmission paths: vertical and horizontal
F) Requires active instruction

IV) Cultural evolution and symbolic communication
A) Symbols permeate all aspects of human culture
1) Even acquired behaviors have symbolic association
2) JP: as do practices forming "body politic" / affective cognition

B) Many cultural evolution models are problematic
1) They assume copying vs reconstruction (= active acquisition / interpretation)
2) JP: for me, "information" is problematic too
    a) Even if reconstructed, rather than copied
    b) I'd rather focus on affective cognition / body politic formation

V) Competing Theories
A) "Selfish Memes"
1) Presuppositions:
    a) Meme = unit of information embodied in neural circuits
    b) Relies on memes as replicators and organism – cultural products as vehicles

2) Criticisms:
    a) Vehicles cannot transmit acquired variations
    b) But heritable variation in behavior and ideas (allegedly, "memes")
        i) Are reconstructed (by the alleged "vehicles") in next generation's development
        ii) Hence can transmit variations, bcs learning is function / meaning sensitive process
    c) Reconstruction of a behavior ties together transmission and the transmitted variant
        i) The developmental consequence (= neural circuits for the behavior)
        ii) Come not by copying but by reconstruction of a social / environmental network
            (a) So transmission is not simply copying of the transmitted unit
            (b) But is a property of a distributed system

B) Evolutionary psychology (EP) and mental modules
1) JP: Preliminary distinction (not offered by authors)
    a) Sociobiology: explananda = behaviors
    b) EP: explananda = psychological modules producing tendencies for behavior
2) Characteristics of EP
    a) Gene-centered: culture is veneer
    b) Massive modularity of mind / embodied in specific neural networks
    c) Selection during Pleistocene
    d) Possible contemporary mal-adaptivity of inherited module (which *was* an adaptation)
3) Alternate explanations
    a) Neural plasticity (especially in cortex)
        i) Thus I can accommodate some modules, esp. for basic emotions
        ii) But deny "massive" modularity, esp. for complex social situations
    b) Culture as productive
        i) Culture is not simply a modification of underlying structure
        ii) IOW, cultural practices produce body politic via neural plasticity
            (a) Cultural differences cannot be decoded to reveal universal human nature (UHN)
            (b) I believe in such a UHN (I'm not a pure social constructivist)
                1. But UHN is limited to patterns basic emotions
                2. While triggers and thresholds are plastic and constructed by culture
4) Thought experiment: "literacy module"
    a) Evidentiary issues:
        i) Reasons to be tempted to posit evolved literacy module
            (a) Complex behavior
            (b) Yet easily acquired at early age by almost every child
            (c) And disrupted by neurological anomalies
        ii) Can also be explained by combination of two factors
            (a) Pre-existing cognitive adaptations
                1. But not for "literacy"!
                2. But for pattern recognition, etc.
            (b) Wide spread and continuously reproduced environmental inducers
5) Methodological issues with EP
    a) No neurological data to back up EP (on the contrary, cortical plasticity is rule)
    b) EP relies on inferences from psychological tests

c) Overlooking of more plausible cultural explanations
    i) E.g., sexual selection argument for greater male creativity
    ii) Parsimony of explanation would argue for cultural explanation
6) Major difficulties with central planks EP
    a) Universality and invariance
        i) Explanation by means of gene-based psychological module
        ii) May be premature bcs social universals haven't been identified
    b) Ease of acquisition (Chomsky vs Deacon)
        i) Chomsky: poor quality input but impressive competence anyway = language organ
        ii) Deacon: coevolution of language and neural capacities = ease of learning
            (a) Cultural evolution can adapt to capacities of brain
            (b) Cultural niches can then exert selection pressure on brain

VI) From evolution to history
A) EP and memetics are Darwinian:
    1) Cultural competition is only a selection factor for mysteriously generated variations
    2) Which are quite strongly constrained by inherited modules anyway
B) JL are Lamarckians:
    1) Culture also generates directed variation via capacity for future planning
    2) And culture operates so as to consolidate and create mutually reinforcing life-styles
        a) Thus it's impossible to isolate a "meme" or "module" for independent selection
        b) Because "selection, generation and transmission" of cultural variants
            i) Are linked together
            ii) And embedded in economic, legal and political systems
C) Example of change in punishment for stealing sheep

VII) Dialogue
A) Relation of language to other symbolic systems
    1) Language transformed gesture into symbolic system
    2) Language co-evolved with other symbolic modes (Merlin Donald)
B) Culture as emergent
    1) Are cultural products autonomous replicators merely inhabiting robotic vehicles
    2) No; even though culture is independent of any one individual, humans are agents
        a) "Men make history, but not on conditions of their own choosing"
        b) Need to see diachronic and not just synchronic emergence
            i) Synchronic: part / whole leads to impossible problems
                (a) Individual / society
                (b) Structure / agency
            ii) Diachronic:
                (a) Highlights issues of X-gen reconstruction
                (b) And criteria for identifying "crisis zones / periods" for innovation
C) Latent cultural information can be transmitted: potential for variants to be created later
D) Is "cultural evolution" really a useful term?
    1) Can you give a rigorous definition?
    2) Why not just give thick descriptions of cultures (Geertz)?
    3) David Hull's definition of Lamarckianism vs Mayr and "soft inheritance"

E) EP and UHN
F) JL's final statement: agency and the "tangle of construction"

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)