

Evolutionary Developmental Biology

Ron Amundson, University of Hawaii at Hilo

Routledge Encyclopedia of Philosophy Online. 2008. General Editor: Edward Craig.
<http://www.rep.routledge.com> Routledge is an imprint of the Taylor and Francis group.

This is a facsimile of the text in that online article.

Evolutionary developmental biology is the study of evolutionary change (called phylogeny) as it is revealed through the embryological development of individual organisms (called ontogeny). On this approach, the understanding of ontogeny contributes to our understanding of phylogeny, and vice versa. Evolutionary thinkers of the nineteenth century almost all held what may be called the core doctrine of evolutionary developmental biology: that in order to achieve a modification in the adult form, evolution must modify the embryological processes responsible for that form, so that an understanding of evolution requires an understanding of development.

But this principle was rejected during most of the twentieth century. This was the time during which the Modern Synthesis held sway. Synthesis-based evolutionary theory has no theoretical need for developmental views of evolution. Beginning around 1990 a series of discoveries and theoretical innovations in developmental genetics led to the reinvigoration of developmental approaches to evolution. Evolutionary Developmental Biology ('evo-devo' as it is now called) was inaugurated as a Division of the Society for Integrative and Comparative Biology in the year 2000.

- 1 The nineteenth century: ontogeny and phylogeny
- 2 The Modern Synthesis
- 3 The 1990s: Evo-devo rises again
- 4 Philosophy

- 1 The nineteenth century: ontogeny and phylogeny

Traditional histories of science often describe biology in the nineteenth century as centred on the debate between evolution and special creationism, the view that God created individual species. Those who pay close attention to embryology, however, see a broader dispute, the dispute between *structure* and *function* (Russell 1916, Ospovat 1981). Functionalists - advocates of the primacy of function, adaptation, or fitness - claim that the traits we see in organisms are just those that the organism needs to thrive (survive and reproduce) in its environment. Structuralists claim that some autonomous structural principles must be understood in order to comprehend organic nature, and function is merely the putting-to-use of traits that exist for autonomous structural reasons. For a simplified version of this dispute, consider the question of why mammals all have four limbs rather than some other number. Functionalists would say that four limbs function better than some other number for organisms like mammals. A structuralist would say that mammals are vertebrate tetrapods, descended from the earliest amphibious tetrapods, tetrapods are all built on a body plan (*Bauplan*) that involves four legs, and this is why mammals have four legs. The structure/function dichotomy is independent of a belief in evolution. The pre-evolutionary debates between structuralists and functionalists are eerily similar to those that would later take place between evolutionists.

The most influential pre-Darwinian structuralist was Richard Owen, who is known for formalizing the anatomical distinction between analogy and homology. *Analogy* is a similarity in body parts between species that is required by the similar adaptive needs that the part serves in the different species. For example, the wings of butterflies and birds, and the fins of flying fish are analogous. They are similar because they *need to be* in order to achieve the function of flight. *Homology* is a similarity that derives from the fact that the compared body parts are actually *the same body part under modification* (Owen 1848). As a structuralist, Owen was impressed with the importance of homology. Detailed sets of homologies could be seen throughout large taxonomic groupings. Owen showed that all vertebrate limbs were built with the same set of bones, even when they functioned in very different ways (the wings of bats, the forelegs of horses, the paddles of porpoises). In fact this was true of the entire skeleton of vertebrates, whatever their species. Vertebrate bodies could be seen as variations on an archetype, a sort of

generalized skeleton. Functionalists, in contrast, emphasized the importance of adaptive diversity among organisms rather than the sameness of homology. Sameness, when it could be found, was probably due to analogy.

From the very beginning, studies of embryonic development had tended towards structuralism. In the 1820s Karl Ernst von Baer established a series of laws of embryonic development. When one compared the developing embryos of related species of organisms (species of vertebrates or molluscs for example) one found that the early stages of embryos were simple in form and very similar, but as the embryos developed and became more complex, they diverged from the early similarity (Russell 1916, p.125 *ff.*; Amundson 2005, p.59 *ff.*). Recall that Owen defined homology in terms of 'same parts'. Modern readers might ask how pre-Darwinians like Owen defined 'sameness'. Von Baer's laws gave one criterion. Traits are 'the same' when they correspond in their embryonic origins. Similarities between organisms that are unrelated or distantly related - analogies - can be attributed to function (adaptation) rather than the structural sameness of embryonic origin. Birds and butterflies each have wings, but their wings are analogous, not homologous. Birds and butterflies belong to different *types* of organisms; types are defined simultaneously by shared patterns of development and (correspondingly) shared patterns of adult morphology.

In 1859 Charles Darwin published *On the Origin of Species* (Darwin 1859). The *Origin* introduced two basic ideas: descent with modification (the Tree of Life) and natural selection as the impetus behind evolutionary change. The Tree of Life doctrine was based in large part on structuralist conclusions about the *reality* of taxonomic relatedness, and Darwin gave full credit to the work of structuralists like Owen. But natural selection produces adaptation (improved function), and this was the motor of evolutionary change. Most biologists of Darwin's era were convinced by his argument for descent with modification, but they rejected natural selection as the long-term cause of evolutionary change. The majority were structuralists, followers of Richard Owen's approach. They devised a study called *evolutionary morphology* that was an early version of evo-devo (Bowler 1996).

Evolutionary morphologists all accepted the core doctrine: to achieve a modification in the adult form, evolution must modify the embryological processes responsible for that form, so that an understanding of evolution requires an understanding of development. Embryology was one focus of their research. Another primary focus was phylogenetic reconstruction, the attempt to reconstruct the Tree of Life. These two studies were deeply intertwined. Embryology (it was hoped) would help to show what the common ancestors of different species must have been like. Ancestors were conceived not just as adult organisms, but as ontogenies - as entire lifespans that developed from embryos into reproducing adults. Phylogenetic reconstruction and embryology together had a common purpose: understanding organic form. Where did the major body plans (vertebrates, molluscs) come from? How was form built during ontogeny, and how was form modified during phylogeny? The two questions could not be answered separately - they must be understood together (Amundson 2005).

Evolutionary morphology had died by the beginning of the twentieth century. The biological science of the time simply did not have the tools necessary to understand the mystery of embryological development. Today, in the first decade of the twenty-first century, those tools are only beginning to be fashioned. In the meantime, around 1930, a new theory of evolution replaced the proto-evo-devo of evolutionary morphology. It was called the Modern Synthesis (see MODERN SYNTHESIS, THE).

2 The Modern Synthesis

Why were nineteenth century evolutionists so sceptical about natural selection? The most important reason was that natural selection had never been proven to be consistent with what were then seen as the facts of heredity. In hindsight, we can see why. The nineteenth century concept of heredity was very different from the modern concept. A 'theory of heredity' had to do two things. First, it had to explain the similarities between parents and offspring, just like today. But second, it had to do that by showing how the traits that were similar between the two generations *actually developed in each*

generation. Heredity began in the germs of the adult, and continued through the embryological construction of the following generation. In other words, the nineteenth century concept called 'heredity' was a combination of (what we would call) heredity plus embryological development (Churchill 1980). A comprehensive theory of *this* concept of heredity would have to wait until development was fully understood. Until it was, natural selection was treated as an unproven hypothesis. The mystery of development had been the downfall of evolutionary morphology, and it was also a stumbling block for natural selection.

One way to solve a problem is to change its terms. For heredity, this took the form of separating the study of parent-offspring similarities from the study of embryological development. Only the first was now called 'heredity'. The idea of studying heredity-without-development may first have come to Gregor Mendel in 1865 (see GENETICS §1). It was modernized into a chromosomal theory by Thomas Hunt Morgan, and was widely accepted by the 1920s (see Morgan et al. 1915). Mendelian genetics ignores development. It merely keeps track of the co-occurrence of (mostly adult) traits between parents and offspring. The traits are tracked through hybridization, the crossbreeding of organisms that had differing traits. The method of hybridization has two drawbacks. First, it does not allow the comparison of genes between organisms that do not crossbreed, so it is almost entirely restricted to the study of genes within species. Second, because development is carefully ignored, Mendelism encourages the oversimplification that individual genes are directly responsible for the creation of the particular adult traits that they are associated with. This view came to be called *genetic determinism*. However, Mendelian genetics also produced a wonderful side effect. It made heredity consistent with natural selection.

It happened like this: Mendelian genetics was given a mathematical formulation called population genetics. Population genetics concerns the statistical behaviour of genes (associated with traits, as Mendelism specifies) in populations of breeding organisms through successive generations. Evolution is conceived as changes in frequencies of the various genes within a population through time. Population genetics (together with

paleontology, animal breeding, and other fields of biology) was built into the Modern Synthesis. The Synthesis finally proved that natural selection could be a long-term cause of evolutionary change. The only mechanisms of evolutionary change that were acceptable within the Synthesis were those that could be expressed in terms of (Mendelian) population genetics. These included mutation, migration, and genetic drift, but natural selection was soon accepted as the dominant cause of evolution.

Synthesis thinkers rejected the outmoded theories of the evolutionary morphologists, with all their talk of homologies, archetypes, and body plans. Like Darwin, most Synthesis evolutionists saw homologies as nothing other than left-over residue from common ancestors. The study of phylogenetic reconstruction lost much of its interest. Theodosius Dobzhansky, one of the architects of the Synthesis, commented that the only scientific purpose served by tracing evolutionary ancestries was to convince the public that evolution had occurred. He claimed that phylogenies had nothing to do with evolutionary *causation*. Evolutionary causation is a matter of populational genetics, including such things as natural selection (Dobzhansky 1937). This is an example of how contrasting scientific orientations have distinct concepts even of simple terms like causation. Structuralists had considered evolutionary causation to involve the modifications of ontogeny (see, again, the core doctrine), a very different thing from populational processes like natural selection.

The Synthesis included a large number of scientific fields, but one was left out: embryology. From the structuralist standpoint, it seemed that Mendelian genetics was shielding embryology from the eye of natural selection - or at least from the attention of Synthesis evolutionists. Developmental views of evolution came into open conflict with the Synthesis in the 1970s. The debate was once again structure versus function. As with the pre-Darwinian debates, adaptationists believed that adaptation is the most important fact about organisms, and that the best explanation of a phenomenon is the one that depicts it as a consequence or by-product of natural selection. Structuralists believed that the causal mechanisms of ontogeny placed important constraints on adaptive change, and that many phenomena were better explained as consequences of the mechanisms of

development rather than adaptation itself.

Here is a simplified version of a debate of the 1980s and 1990s between adaptationists and structuralists. Several species of salamanders have independently evolved a miniaturized form, because of known adaptive influences. These species, but not their larger relatives, have almost all evolved reduced numbers of toes. How do we explain this pattern of digit reduction among miniaturized, but not ordinary-sized, species? An adaptationist explanation would identify something in the environment of each miniaturized species that made reduced digit numbers selectively advantageous. In contrast, a proposed structuralist explanation was that embryonic salamanders develop their digits in a way that depends on the number of cells within the precursor of each digit. Digits develop one at a time, with each successive digit precursor beginning growth after the preceding digit has reached a given number of cells. Under the selective pressure of miniaturization, the developing digits have fewer cells to work with (because miniaturization occurs by reduction in the number of cells, not in the size of each cell). Fewer cells mean that fewer digits will be produced within the embryo of the miniaturized salamander. Digit reduction occurs by shared structural constraints of how digits are built during embryonic development, not adaptation to external circumstances. These competing explanations might each be true, and careful research could determine which was best. But adaptationists and structuralists had very different assumptions about which was preferable, and almost no one studied adaptation and structural constraint with equal fervour. The two sides were virtually at a stalemate, each considering the other's methods to be flawed. (See Amundson 2005, p. 233 *ff.* for details of this debate.)

One final point regarding Mendelian genetics will illustrate the Synthesis view that embryological development is not important for the understanding of evolution. Recall that Mendelian genetics can only examine genes *within* a species; it cannot compare genes in different species. Synthesis advocates did not see this as a problem. As adaptationists, they were not much interested in commonalities and shared structure, including shared ('homologous') genes. In fact, at least two major Synthesis theorists, Theodosius Dobzhansky and Ernst Mayr, claimed that homologous genes should be

expected *not to exist* except within closely related species (Dobzhansky 1955; Mayr 1966). The view makes perfect sense from their perspective. Adaptation was a force for divergence, and distantly related species have been diverging for hundreds of millions of years. Because homologies were thought to be mere leftovers, dust in the closet of common ancestry, there is no reason to expect them to persist after all that time. This prediction was a dramatic failure. Its fall proved to be the rebirth of evo-devo.

3 The 1990s: Evo-devo rises again

In retrospect, the tensions between adaptationist and structuralist evolutionary approaches were largely caused by the limitations of Mendelian biology. Homologous genes were invisible to Mendelian methods. So the adaptationists, who saw little value in homologies anyhow, were happy to ignore them. But another approach to genetics was slowly being developed (see GENETICS §2). Molecular genetics is able to identify genes by their molecular sequence. Hybrid crosses are no longer necessary to reveal genes. This led to an extensive reinterpretation of what genes 'really are' (Beurton et al. 2000). The ability to identify genes by their molecular sequence (instead of the phenotypic markers that show up in hybrids) allows a great expansion in the study of genetics. First, geneticists can now directly study the effects of genes on development. Second, because of the release of the requirement for hybridization, we can now compare genes *between species*, something that the Mendelian methods did not allow. If the structuralists were right, and ontogeny is an important aspect of evolution, then the new genetics may be able to prove it in a way that Mendelian genetics never could.

During the 1990s the data started coming in. The first discoveries involved genes that had originally been identified by ordinary Mendelian hybrid experiments within species. The new methods allowed comparison across species. The adaptationist prediction that homologous genes would only be found in closely related species was dramatically falsified. Homologous genes were found between species that had been separated by hundreds of millions of years of evolution. Not only that, the homologous genes were developmental triggers for very early developmental processes. The first results involved

a gene called *Pax-6* in mice that was necessary for eye development. Geneticist Paul Gehring recognized that *Pax-6* had molecular similarities to a gene with similar effect in fruitflies, called *eyeless*. So he transplanted *Pax-6* into the embryonic bodies of fruit-flies (Halder et al. 1994). The mouse genes triggered the growth of eyes in the flies' bodies. But the eyes they grew were fly-eyes, not mouse-eyes. The gene was not a gene 'for' a particular adult trait (a mouse eye), but a gene that stimulated a developmental process that involved many other genes and developmental resources in the fly. The trigger (*Pax-6* and its homologues) was shared, but later developmental resources (used in the building of different kinds of eyes) varied between the species (Carroll 2005).

Mice and flies are separated by 600 million years of evolution. Gehring's discovery was just a part of a flood of genes found to perform similar tasks in very early embryonic stages of the developing bodies of distantly related organisms, including triggering the development of hearts, limbs, and body segments. An important category of genes were called *Hox* genes, which occurred in clusters of up to ten repeats (with variations) along a single chromosome. These genes controlled the anatomical nature of successive sets of segments along the bodies of insects - the segmented head, three thorax segments, and abdominal segments. Homologous *Hox* genes were discovered to control the identities of *vertebrate* segments, including human bodies. Anatomists had long recognized vertebrate segmentation. But only the most speculative evolutionary morphologists had the nerve to suggest that vertebrate and insect segmentation was homologous. Now we have genes that seem to demonstrate that the segmentation of our bodies is *the same* (in the homological sense) as the segmentation of insect bodies. The ancestor to insects and mammals was segmented, its body was structured by a sequence of *Hox* genes, and the traits of segmentation and *Hox* control have been inherited by us and our mosquito companions.

These discoveries and methods constituted the modern rebirth of evolutionary developmental biology, evo-devo. They also brought about a reconceptualization of the genotype-phenotype relationship. In the early days of Mendelism and the Synthesis, genes were conceived as active agents that regulated development, and therefore were

causally responsible for phenotypic traits (Morgan et al. 1915, p.209). This was the view called genetic determinism. As developmental genetics progressed it became clear that genes did not regulate development at all. On the contrary, genes *were regulated* during development - they were regulated by a vast network of interactions with other genes (including a new category of regulatory genes) and other factors that came from within the nucleus, and also from the cytoplasm and the extra-cellular environment. The old genetic determinist strategy of treating individual genes as directly determining phenotypic traits lost credibility. Genes came to be viewed as responders rather than controllers. Genes responded to the developmental processes around them. The entire process of development, not individual genes, was responsible for adult traits.

The genetic determinism of the early Synthesis evolutionists had made development seem irrelevant to evolution. If genes directly caused phenotypic traits, evolution could be seen as the mere shuffling of genes. But if genes cause phenotypic traits only indirectly, by complex interactions with other developmental factors, then the core doctrine is vindicated. To understand evolution, we must understand *how ontogeny can be changed*. With the erosion of genetic determinism, the way was cleared for development again to be seen as a factor in evolution.

The rise of evo-devo has led to major scientific advances in two areas, both of which were uninteresting to most Synthesis evolutionists. One is the origin of evolutionary novelties. Novelties are new characters of major groups that arose from ancestors that apparently had no characters that could have been modified into those novelties. Examples are the vertebrate limb and jaw, the mammalian molar tooth, and the avian feather (Müller and Wagner 1991). The evo-devo explanation of these novelties takes two steps. First, identify the ancestral ontogeny that existed before the origin of a novelty by comparison between ontogenies of related organisms. Second, show how modifications of the ancestral ontogeny would lead to the development of the novelty. The case of the vertebrate limb required reduplication of a cluster of *Hox* genes followed by the co-option of the extra cluster to pattern the limb. This style of explanation is quite distinct from adaptationist explanation. It refers to ancestral ontogenies rather than

ancestral traits, and usually pays no attention to populations or to variable fitness.

The second area of major advances is phylogenetic reconstruction, in which evo-devo work is assisted by new taxonomic methods and molecular phylogenies. The result has been a great deepening of knowledge of the commonalities of life. Knowledge of deep ancestry has gone far beyond the level of phyla such as Chordata (that category that includes vertebrates) and Arthropoda. A new category of animal is now the Bilaterian -- the bilaterally symmetrical animal. This includes virtually all modern animals except jellyfish and sponges. The classification of the commonality of such diverse groups into one was based on discoveries of how our bodies are built in earliest ontogeny. We ('we' meaning us vertebrates, molluscs, insects, worms, sea squirts ... all animals more highly structured than jellyfish) are descended from the Urbilaterian ancestor, an organism whose ontogeny has been reconstructed by evo-devo researchers to include a number of regulatory genes such as *Pax-6*, which triggered the development of a light sensitive proto-eye in the ancestor. The ontogeny of Urbilateria included known developmental genetic triggers for the development of the heart, neural system, appendages, and other body parts. These body parts have been discovered to share their earliest ontogeny. As far as we can tell, there is only one way to develop an eye, a heart, an appendage, and so on; all animals on earth share these basic developmental resources. Research is ongoing into how these basic, shared, embryological processes are modified to give rise to the variety of life. After a long period of disrepute, evo-devo is back in business (Carroll 2005).

4 Philosophy

Philosophers of biology have worked extensively with evolutionists of the Modern Synthesis, and their philosophical ideas have been adjusted to the science. This was largely due to Synthesis architect Ernst Mayr, and his interest in philosophy. However, many of the standard philosophical concepts that arose from Synthesis ideas do not fit as well with evo-devo. Several dichotomies, in particular, are biased against the relevance of development for evolution. An example is the genotype/phenotype distinction (which was invented prior to the Synthesis, but modified to fit within it). Genotype and

phenotype seem exhaustively to classify all aspects of an organism. But where does embryological development fit into the dichotomy? Is it genotype or phenotype? It is neither. Some authors speak of development as the transformation of the genotype into the phenotype. But even this is a distortion. The genotype is the set of *all* genes in a genome. Developmental genetics is not the study of *all* genes, but of *which* genes are expressed in different parts of the embryo at different times during its development, and how those genes are caused to be expressed at that time and place. The genotype/phenotype distinction is a useful shorthand for thinking as a Mendelian or a population geneticist, but it is inadequate for thinking as a developmental biologist or an evo-devo evolutionist.

Another of Mayr's dichotomies is the distinction between *proximate* and *ultimate* (evolutionary) causation. Ordinary day-to-day events are proximate causes, and natural selection is the primary example of an ultimate, evolutionary cause. The question, as with genotype/phenotype, is: where does development fit? The development of an individual organism is certainly not a case of ultimate causation. But if we call development a matter of proximate causes, then it seems irrelevant to evolutionary change. A third important dichotomy is *typological thinking* versus *population thinking*. The distinction was intended to classify non-populational thought (like the thought of embryologists) as inappropriate to evolutionary topics. This leaves philosophers who are sympathetic to evo-devo with the problem of showing either that evo-devo can be accommodated to population thinking (populations of ontogenies?), or that it can have its own legitimacy independent of population thinking. At the present time it seems uncertain which of these routes is correct (see Amundson 2005 Ch.11, for discussion of the dichotomies).

Each of the previous dichotomies requires philosophical attention if evo-devo is to be integrated into contemporary philosophical thought in the way that the Modern Synthesis was integrated into the philosophical thought of the middle of the twentieth century.

Ron Amundson

See also: EVOLUTION, THEORY OF; GENETICS; MODERN SYNTHESIS, THE;
NATURAL SELECTION AND ADAPTATION

References and further reading

Amundson, R. (2005) *The changing role of the embryo in evolutionary biology: roots of evo-devo*. Cambridge UK: Cambridge University Press. (Provides the historical and philosophical background to the topics in this entry.)

Bowler, P. J. (1996) *Life's splendid drama*. Chicago, IL: University of Chicago Press. (History of the late-19th century field of evolutionary morphology.)

Buerton, P., Rheinberger, H-J., and Falk, R. (eds.) (2000) *Concept of the gene in development and evolution: historical and epistemological perspectives*. Cambridge: Cambridge University Press. (Anthology of historical and philosophical papers on the changing concepts of the gene.)

Carroll, S. B. (2005) *Endless forms most beautiful: the new science of evo devo*. New York: Norton. (Popular and accessible introduction to contemporary evo-devo.)

Churchill, F. B. (1980) The modern evolutionary synthesis and the biogenetic law. *The evolutionary synthesis*. eds. Mayr, E. and Provine, W. 112-22. Cambridge MA: Harvard University Press. (Article on the concepts that had to be changed before the Modern Synthesis could get underway.)

Darwin, C.R. (1859) *On the origin of species*. London: John Murray. (Darwin's main work on evolution.)

Dobzhansky, T. (1937) *Genetics and the origin of species*. New York: Columbia University Press. (One of the founding volumes of the Modern Synthesis by one of its "architects".)

Dobzhansky, T. (1955) *Evolution, genetics, and man*. New York: John Wiley & Sons. (Relating evolution to modern human concerns.)

Halder, G., Callaerts, P. and Gehring, W. (1994) Induction of ectopic eyes by targeted expression of the eyeless gene in *Drosophila*. *Science* 267, no. 1788-1792. (Article reporting Gehring's transplantation of *Pax-6*.)

Horder, T. J. (1989) Syllabus for an embryological synthesis. In *Complex organismal functions: integration and evolution in vertebrates*. Wake, D.B. and Roth, G. (eds.) 315-48. Chichester: John Wiley and Sons. (Anticipating the arrival of evo-devo; source of the 'core doctrine'.)

Mayr, E. (1966) *Animal species and evolution*. Cambridge, MA: Harvard University Press. (Volume summarizing Mayr's evolutionary thought.)

Morgan, T. H., Sturtevant A.H., Muller H.J. and Bridges C.B. (1915) *The mechanism of Mendelian heredity*. New York: Henry Holt. (The original publication of the Mendelian chromosomal theory of heredity, now called *classical genetics*. It contains the first argument that heredity is a separate process from embryonic development.)

Müller, G.B., and Wagner, G.P. (1991) Novelty in evolution: restructuring the concept. *Annual Review of Ecology and Systematics* 22: 229-56. (Article arguing that evo-devo can explain innovation in a way that the Modern Synthesis cannot.)

Ospovat, D. (1981) *The development of Darwin's theory*. Cambridge: Cambridge University Press. (History showing that Darwin was influenced by embryologists and morphologists like Owen.)

Owen, R. (1848) *The archetype and homologies of the vertebrate skeleton*. London: J. van Voorst. (One of Owen's important works on homology.)

Pennisi, E. (2000) An Integrative Science Finds a Home. *Science* 287, no. 5453: 570-572. (On the inauguration of Evolutionary Developmental Biology as a Division of the Society for Integrative and Comparative Biology.)

Raff, R.A. (1996) *The shape of life*. Chicago: University of Chicago Press. (Excellent introduction to the origins of modern evolutionary developmental biology as it appeared when the new molecular discoveries were only beginning to take shape.)

Russell, E.S. (1916) *Form and function*. London: John Murray. (History of the structure-versus-function debate as seen in the period before the Modern Synthesis.)

Author Information: Ron Amundson is Professor of Philosophy at the University of Hawaii at Hilo. His research is in history and philosophy of biology, and disability studies.