laughing. The innuendo? Constrain Crandon and she can't produce. It takes Fontana to inform us that "Mina apparently produced phenomena while enclosed in the special fraud-proof box designed by Houdini in which she sat for three other sittings, and which only left her head and hands free. Houdini remained silent about this ... He also remained silent about those séances with Mina when impressive phenomena had been produced and when, along with the other members of the *Scientific American* committee, he had signed a statement affirming that the controls were perfect." Fontana's take on Duncan is equally more balanced and informative than Roach's. After looking carefully at the evidence, Fontana suggests both mediums may have resorted to trickery at times, but he concludes that genuine phenomena also took place. It's not the neat, easy answer the lazy reader may want, but it's what the evidence suggests. Fontana isn't fishing for laughs; he's fishing for the truth.

In fairness, Roach isn't competing with Fontana. Her book targets the average reader with a layman's curiosity about the afterlife question, and she delivers a decidedly delightful evening's read. If you're in the mood to skip the broccoli and proceed directly to dessert, *Spook* is a tasty treat.

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Evolution in Four Dimensions by Eva Jablonka and Marion J. Lamb. MIT Press Books, 2005. 472 pp. $34.95 (paper). ISBN 0-262-10107-6.

This engaging book takes the reader through a tour of evolution—the process by which novelty appears in the bodies and behavior of living things. The main claim here is that evolution is best understood when considered more widely, and not restricted to traditional changes in gene sequences. Using evidence from molecular biology and a wide range of species, the authors discuss four elements of heredity: genetic, epigenetic, learning, and symbolic communication. The first two of these "dimensions" concern processes that build the organism. The genetic view concerns the evolutionary effects of changes within gene sequences, and the authors rightly point out that even this basic level contains ample opportunity for enlarging our perspective, such as by focusing on population frequencies of gene interaction networks rather than of specific alleles (p. 7). The second dimension describes a number of interesting biological mechanisms by which cells with identical gene content are able to become different and pass on these characteristics to offspring cells. The third dimension concerns the modification of species' evolutionary trajectories by individuals' behaviors and preferences, as modified by learning. The final dimension concerns the effects of symbolic communication and culture on shaping evolutionary paths. These are truly "dimensions" in the sense that they are orthogonal—they form a conceptual space within which change occurs along each parameter simultaneously and often independently, making for a very rich phase space of possible trajectories for species as they change in time. After discussion of each aspect and detailed examples, part III integrates these threads back into a unifying vision of change in the biosphere.

One of the nice things about this book is the integration of cell, molecular, and developmental biology. The discussion consists of straight text and dialogs, with the most technical parts separated out. Many interesting and subtle points are covered, but the main thrusts of the logic are very clear. It contains sufficient historical perspective to place important ideas in context for the reader. For example, it is pointed out that Lamarckism, which is commonly thought to be antithetical to Darwinism, had much in common with Darwin's view and does not in fact contradict his original formulation. Importantly, though, one bold claim of modern Darwinism (an empirical hypothesis which may be true or false) is that inheritance of acquired characteristics is not necessary—that the observed adaptive changes can be the result of selection acting upon genetic changes that are random with respect to the fitness of the organism. This minimalist position strains credulity at first, but is powerful in its elegance and simplicity, and may well be true.

The discussion of inheritance of acquired characteristics has a central place in this book. The authors rightly point out that the ability to inherit changes does not, counter to what NeoDarwinists sometimes claim, require reverse translation. This violation of the "Central Dogma", wherein genes direct production of proteins (via RNA) but never the reverse, is not required because many "acquired characters" do not depend on changes in protein primary structure at all. Such examples are amply illustrated in this book, and also in the authors' recent work, *Epigenetic Inheritance and Evolution.* However, there is an even deeper issue here: even if proteins could be reverse-translated into DNA, the real problem is knowing *how* proteins have to be changed to result in the particular character to be changed. Most interesting characters come about as the result of a complex dynamical system of interactions of many proteins. Given the complexity and non-linearity of the pathways that together define the length of the giraffe's neck, exactly how must the component proteins be altered to make it longer? As the authors point out, the effects of genes on the fitness of the individual are often non-additive (p. 77).

This problem is isomorphic to a number of situations where the final outcome is a product of recursive local interactions of many components, including the Game of Life and other cellular automata (Poundstone and Wainwright, 1985; Wolfram, 2002), and creating fractals such as the Mandelbrot set belonging to a function (Flake, 1998). In the former case, one can ask what rules and initial configuration can give rise to a specified pattern? In the latter case, one wants to be able to identify a function whose Mandelbrot set matches a pre-defined image. For such dynamical systems, of which embryonic development is surely one, solving the inverse problem (running the system in reverse) to determine the trajectory leading to a given outcome is computationally intractable. The complex networks of interactions among components ensures that small changes made in the activity of one gene will have many repercussions elsewhere, making it very difficult to determine how the rules must be changed to ensure a desired outcome. For the general case, it's not likely to be possible to determine how to change protein sequences to result in a specific "acquired change", making translation back into RNA/DNA a moot point. That is probably a deeper fundamental reason why the inheritance of arbitrary acquired characteristics (not just some special cases, but the whole range necessary to explain the complexity of the biosphere today) is not possible. This, however, does not affect weaker claims to the effect that genetic variation is not an unregulated (random) process (p. 80).

The Central Dogma of developmental biology parallels that of molecular biology but is more fundamental and extends beyond the DNA/protein distinction. It is that morphogenetic information flows in one direction: from the operation of the genetic apparatus to the final body which results from the embryonic interactions of the myriad of components specified by the genetics—not in reverse. In keeping with this, the mechanisms revealed by developmental biology have not been "master plans" of the final structure but rather local instructions to be followed by subcellular components whose activity results in the finished product as a superb example of emergence.

Having said that, it's interesting to consider a case that seems to counteract the primacy of local rules over global planning. It would have been interesting for the authors to have commented on "Trophic Memory" (Bubenik and Pavlansky, 1965; Bubenik and Bubenik, 1990), as it provides a striking example of the kinds of mechanisms with which this book is concerned. Deer antlers are shed and re-grow every year. Remarkably, if a cut is made in a particular place on a set of antlers, resulting in a small outgrowth as the bone heals, a bigger growth is noted in the *same place* for several years afterwards, as new sets of antlers grow. This demonstrates that the location of the injury is communicated to the growth center at the scalp, which is able to alter next year's growth in precisely the way necessary to demonstrate inheritance of the acquired morphological change. The mechanism is unknown, but is likely to represent a useful component in the evolutionary toolbox since it illustrates both epigenetic and environmental influences on pattern formation. This example of "instructive" inheritance in a mammalian species provides a fascinating addition to similar properties that have been ascribed to metabolic pathways in lower organisms. There is another organism that is likely to shed light on these problems. Planaria are flatworms with a well-developed brain and 3 body layers. Remarkably, as little as 2% of the worm is able to regenerate every other tissue and becomes a complete (but very small) worm; the worms can also reproduce by fissioning in half. In this case, changes in somatic cells (neoblast adult stem cells) can indeed be passed on to the next generation (p. 149).

This book is quite comprehensive and often very thought-provoking. It is recommended highly, as are the authors' other works. The discussion ranges over prions, RNAi, embryonic mechanisms, and a wide variety of organisms. The authors "prefer to think about the selection of heritable phenotypic traits, rather than genes." (p. 280) This is indeed a deeper, richer perspective for thinking about evolution, and is well-captured by the four dimensions of this enjoyable work.

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Vaccine A: The Covert Government Experiment That's Killing Our Soldiers, and Why GI's Are Only the First Victims by Gary Matsumoto. Basic Books, 2004. 362 pp. $25.00 US, $34.95 Canada (hardcover). ISBN 0-465-04400-X.

After the first war in Iraq, in the early 1990s, returning veterans complained of a mysterious malady, whose symptoms included headache, memory loss, joint and muscle pain, fatigue, sleep disorders, and intestinal and respiratory ailments. Their illness often persisted and became debilitating. This sickness came to be called Gulf War Syndrome (GWS), although medical and military authorities never agreed that it is a single disease. Estimates of how many veterans suffered from GWS vary widely; the official Pentagon estimate is that about 1 percent of Gulf War veterans, or about 7,000 soldiers, have had illnesses with no other confirmed diagnosis.

What caused GWS was the subject of much speculation. Some suggested that affected soldiers might have been exposed—without anyone detecting it at the time—to nerve gas released when Iraqi weapons were destroyed, or that GWS was a reaction to the stress of battle. Neither theory is particularly consistent with available evidence, and the cause of GWS remains officially unknown.

In *Vaccine* A, investigative journalist Gary Matsumoto offers his own provoc-