Is Somatic Selection an Evolutionary Force?[1,2]

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Progress in the evolutionary half of biology is to a considerable extent the evolution of concepts, as Mayr (1982) has remarked. Another form of progress is the explanation, or even the recognition of a need for explanation, of phenomena previously taken for granted, such as the remarkable unravelling of the consequences of patch selection which Wilson (1980) has given us.

Buss's earlier work has often been in this exemplary but rare tradition. He has continued this approach in his book, which unfortunately fails in its central argument. Conceptual change is not necessarily progress.

I am glad he wrote the book, though, even apart from the various and often insightful comments it makes on more or less peripheral subjects. He has succeeded in giving us a major set of partly new problems for which we lack adequately substantiated answers. There are also partly new approaches to research. The argument itself even fails in an interesting way.

Somatic selection

By somatic selection I mean selection within an individual at any level above DNA. The term is not new; e.g., Breese, Hayward, and Thomas (1965). (Part of the ambiguity of the term "individual" persists in Buss's book. He defines it [p. viii] as "a physiologically discrete organism", but in cases where criteria disagree he uses it consistently in the first part of the book for what botanists now refer to by Harper's term "genet". Later he consistently uses it in a third way, as only a multicellular physion [term defined below].)

In this context I realize that my earlier list of definitions (Van Valen, 1987) was incomplete despite its length. We need to be able to refer unambiguously and generally to the smallest naturally bounded unit which contains all the structures and functions needed for continuing normal life. For such a unit I propose the term **physion**. A worker bee, a pterobranch colonoid, a bacterium, and a lichen are all physions.

Somatic selection has been known since the Nineteenth Century, at least by plant breeders, but it has always been rather a curiosity. Yes, B chromosomes sometimes multiply disproportionately. Yes, unused branches of axons often degenerate. Yes, tumors have a short-term fitness different from that of the rest of the body. So what? Such phenomena may be significant for the cases themselves, and even for defenses which may evolve against the repeated occurrence of some of them, but it seems farfetched to claim that they can be of much overall importance in evolution.

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[1]Contribution 78, Lothlorien Laboratory of Evolutionary Biology.

[2]**The Evolution of Individuality. Leo W. Buss**. 1988 (copyright 1987). Princeton: Princeton Univ. Press. xv + 203 pp. ISBN 0-691-08468-8 hardbound, $40.00; ISBN 0-691-08469-6 softbound, $12.95.

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The argument

Buss claims that they caused much of what we see in the living world around us. "Evolutionists, even today, seek to understand how development will illuminate patterns in evolution, not how evolution will illuminate details of the developmental process" (p. 65). I applaud his viewpoint, and note that others have shared it. For the nature of his own original argument we can look at his most developed example, the sequestration of the germ line.

It is possible, in suitable organisms like sponges, for there to be somatic mutations in which the carriers become sexually reproductive cells. They thereby compete with the normally differentiated germ cells for access to nutrients and other requisites for successful gametogenesis. Most such variants will be deleterious, as with germinal mutations, although mutations advantageous at the individual level should be no less rare in somatic cells than in germ cells. There is even a likely example of one (p. 76). Furthermore, competition among cell clones within individuals is common.

However, if there is only a single germ line and it is determined early, the likelihood that the usually deleterious somatic variants will invade this germ line is reduced. It is eliminated entirely in organisms like Drosophila in which the maternal control of early development extends past the time of germ-line determination. (Buss implies incorrectly that maternal control ends abruptly and synchronously across the genome.)

Thus both germ-line sequestration and early maternal influence are explained as defences against somatic selection of selfish cell clones.

Competition within the body: a critique

I have reconstructed the argument in a more plausible form than is actually given. A theme which occurs again and again, here and elsewhere, is that the cells of a physion are ordinarily in competition with each other. "While germ-line sequestration is a selfish innovation, it should perhaps be emphasized that it is a selfish act on the part of somatic cells, not the germ line. Germ-line sequestration arose through cells pursuing increased replication within the somatic environment. The germ line is a mitotically inactive lineage, a loser in cell-lineage competition. The selfish cells here are the somatic cells, which abandoned a function of significance to the individual [i.e.,reproduction of the individual], in return for further replication" (p. 180). "Why, then, should any cell in a dividing embryo become ciliated, or otherwise differentiated, in a fashion which limits its own capacity for increase? It should not" (p. 53).

Such statements can be made only by ignoring both (1) the effective time scales of natural selection and (2) the degree of relatedness among the cells in an ordinary multicellular physion. The cells of a single physion usually have effectively the same genotype. There will be somatic mutations here and there, but unless they affect the behavior of the cells in a way related to the cells' proliferation they are irrelevant. Even if selection does occur on their carrier cells their change in frequency is only a byproduct of this selection. And their long-term fitness is effectively that of the physion itself.

In the simplest case, consider how a neuroblast can best maximize the expected number of its alleles in the next generation or in a hundred years. Obviously this is by being a good little neuroblast and differentiating into a proper neuron, even though by doing so it gives up any possibility of further replication itself. Similarly, although cell death is sometimes imposed by other cells (and not just by competition, as implied on p. 95) it is often enough (e.g., nematodes) autogenous, caused by a developmental program in the cell itself.

Buss regards lymphocytes as a good example of his clonal selection. Indeed, some clones do proliferate greatly. But their doing so is part of a more inclusive

program selected at the individual level: when genuinely selfish lymphocyte clones proliferate they do so outside of this program and we call the result leukemia.

The normal result of selfish behavior by cells is a tumor. Consider what would happen if a cell clone succeeded in invading a germ line or establishing a new one. All the cells of its offspring would be like that (ignoring irrelevant complications of biparental inheritance). The cell in the offspring more or less homologous to the initiator of the successful clone in its parent will now have no advantage and the development of the offspring will thus differ from that of the parent unless there is a fortuitous congruence of the somatic mutation and the local inductive environment. Buss does not mention this latter possibility but it may very occasionally occur if everything else somehow works. Otherwise there is no way in which this process provides for the origin of the inductive and other programmed controls which organize normal development. To say that "the history of its origin from the original lineage and its interaction with the original lineage may be replayed as an epigenetic interaction in development" (p. 116) is to ignore the origin of regulatory control, the foundation of most development. Buss claims that mutations in the germ line will be too sparse and ineffective ("extraordinarily improbable": p. 102), but with that alternative there is a way to build on what is already present rather than invoking magic. Reciprocal induction is not basically the mutual control of competing cell lineages (p. 95); its results are subject to selection at the individual level and the program itself unfolds earlier. The extraordinary statement is actually made (p. 82) that "the genome need only encode the relative competitive advantages of cell lineages."

"The fact that metazoans develop via a complex of epigenetic interactions between cell lineages is prima facie evidence that the principal modes of metazoan development arose as variants in the course of ontogeny" (p. 78). Hardly. What it does support is merely the modification of phylogenetically earlier programs. In the vertebrate immune system, some T-cells are selected for clonal amplification which do not perform the normal cytotoxic function. Buss says that if the development of the imune system "arose as anything but a consequence of interactions between cell lineages in the course of ontogeny, such functionless variants would hardly be expected" (p. 88). Does he think that adaptations must be perfect rather than merely improvements, and that perfection entails no cost?

The viewpoint which I have discussed in this section is actually the major emphasis of the book. It does make a few apparently corroborated predictions. However, the existence of correct predictions by an incoherent theory (remember Velikovsky, or even the Gaia Hypothesis?) means merely that there must be some other theory, which we may not yet have found, which predicts about the same consequences.

Defenses against somatic selection

But what of the sanitized proposal? Briefly, this is that somatic selection should be advantageous to individuals only rarely, so there is selection for defenses against it. Germ-line sequestration and maternal control are examples of such defenses.

I think that this general argument is unexceptionable, but it is also unoriginal in its general form. There are very diverse defenses against cancers. The critique in the preceding section, if correct, disposes of the central theme of the book. We should now examine whether the argument best explains the two specific phenomena mentioned.

Both these phenomena are predicted by other approaches.

The genome changes during development. The best-known widespread change is in the degree and detailed pattern of methylation (Cedar, 1988); a methylated gene must be demethylated before it can be transcribed. Also, the fact that the germ line does not age (otherwise an egg would have the physiological age of its mother, cumulative over generations) implies a genomic difference of some sort, if only again in pattern of transcription. Sequestration of the germ line permits the

genome in the cells of the rest of the developing organism to undergo changes which are not easily if at all reversed. The sooner the sequestration happens, in determination rather than differentiation, the sooner somatic tissues can go their own way. This is presumably more efficient in information use, with perhaps also a lower probability of error, and it permits more rapid development. Buss thinks the latter can't be important because plants have a similar advantage for rapid development but lack early determination. However, early determination seems inconsistent with arborescent and meristematic growth.

(Weismann knew this general argument on early determination. In a historical section which is poor overall, Buss repeats the usual view that Weismann proposed the continuity of specific germ-cell lineages over generations. However, this was an earlier view which Weismann argued explicitly and strongly against; his germ-plasm was informational and intracellular. Buss actually quotes Weismann's correct statement that the continuity of the germ-plasm doesn't itself entail the noninheritance of acquired characters; he then later repeatedly says that it does.)

Early maternal control is mostly a result of the provision of large amounts of mRNAs in the egg. These are not translated until after fertilization; in some organisms the mother continues to provide them even to the very early embryo. More than mRNAs are of course provided, but these (together with the putative control of early-embryonic gene transcription) are what make the early-embryonic genome more or less impotent. Much mRNA means that protein synthesis can proceed rapidly; the same result is often achieved later by such means as amplification of part or all (polyteny) of the genome or by duplicate genes. And the egg is a very large cell. It is easier for an embryo to produce enough mRNA by its own genome when the cells get smaller by cleavage.

Rapid development is particularly advantageous to an embryo which is more vulnerable when smaller, as is usually the case for free-living embryos, but even for mammals it affects the time to placentation and is part of a major fitness component, generation time. Maternal control of early development is not restricted to gonogens, as the somatic-selection hypothesis would suggest. It is characteristic also of somatogens and therefore occurred before germ-line segregation, because all gonogens are descended from somatogens.

Other aspects

The book bristles with ideas; I will comment on only a few.

Partly because the number of cell divisions in the germ line is usually a small proportion of those in somatic development, Buss thinks that most evolutionary innovation should occur in the latter before being incorporated into the germ line by somatic selection. Apart from other problems, though, an average somatic cell is separated from the zygote by about the same number of mitoses as is a germ cell, even disregarding the numbers of gametes sometimes produced. The discrepancy is thus merely in number of cell lineages. He notes that the combination of relative impotence of germline mutations and defenses against somatic selection should inhibit major evolution, as has perhaps been the case since the Cambrian. (He does not note that gonogens have not evolved less than somatogens in this interval.) He notes that in groups where "maternal predestination closely coincides with germ-line sequestration" (p. 108), like the Rotifera, Mesozoa, and Chaetognatha. there has apparently been little morphological or cladogenetic evolution. (He does not note this prediction on p. 116, where he says that in holometabolous insects germline sequestration precedes the end of maternal control.) Referring to the latter, he says that "there is no opportunity during the entire life cycle of Drosophila for any cell to influence its own fate [in later generations] by products of its own making" (p. 16). Because it is part of a body selected for cooperation among cells, it does so best by the whole body's influence on later generations.

Buss thinks that there are no organisms with cellular differentiation which are not at least primitively sexual, and no clonates with germline sequestration. He

seems to have forgotten bluegreen algae with their heterocysts, and aphids, for example. However, the correlations are real enough. He then uses somatic selection to explain the persistence of sex: if one as usual ignores the overwhelming selection for cooperation, cell clones which do not contribute directly to clonal offspring will at some time be selected against, and the organism will eventually lose its cellular differentiation.

More positively, there is an interesting hypothesis for the origin of gastrulation. It is based on Margulis's observation that cells with a euflagellum (a term which I prefer to "undulipodium" and which contrasts with the prokaryotic proflagellum) and a single microtubule-organizing center must lose their euflagellum before cell division. Such cells are supposed to include all metazoan cells and their near ancestors, but mammalian oocytes have multiple centers (Maro, 1985). I wonder how many other exceptions exist and what the nature of the apparent restriction on their evolutionary origin may be.

Anyway. a primitive free-living marine blastula has ciliated cells so that it can swim. These cells can't divide, and those that can must find a place in which to do so. The only place which will not hinder locomotion, Buss says, is the interior. (But later larvae have regions without cilia; why not start this pattern early?) Therefore gastrulation occurs. One can extend this sort of argument, without any implication of somatic selection needed, to the blastula, where an advantage of a blastocoele is the removal of all cells to the surface, where they can help in locomotion. Do early embryos use dissolved organic material, and if so does its patchiness of concentration affect development?

The received explanation for the relatively little evolution we see before gastrulation is the Markovian nature of development, each stage building on those before. Buss argues that it is rather because of functional constraints on a free-swimming embryo, together with the historical constraint of ciliation preventing mitosis. Gastrulation is modified and even abandoned when the embryo doesn't swim, and early determination (especially mosaic development) then also becomes the rule, although he doesn't note the exceptions.

I conclude that somatic selection still has no known general evolutionary significance except for defenses against it. A synthetic consideration of such defenses would be valuable. I do agree with Buss that many otherwise unexplained phenomena, such as the synchronous division of the nuclei in fungal coenocytes, or the clamp connections which characterize basidiomycetes, may have had their origin and continued maintenance in this way.

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