

However, the labour in European honeybee colonies is divided not only into within-nest and outside-nest tasks but also more finely to some extent, e.g. youngest workers clean cells, and second youngests care for broods and the queen and so on. While proximate explanations may be able to explain this pattern (*Social Evolution in Ants* by Bourke & Franks, Princeton University Press, 1995) and the generality of this pattern is still in dispute (*The Wisdom of the Hive* by Seeley, Harvard University Press, 1995), risk difference does not seem to explain the phenomena fully.

Schmid-Hempel proposes a new hypothesis that age polyethism is a colony-level adaptation to reduce the load of epidemic disease. If parasites invade a colony by first infecting a forager working outside the nest, limited interactions among workers, i.e. more contacts among members of the same task group but fewer contacts among different task groups, should impede disease transmission within the colony. Schmid-Hempel explains this using a conveyer belt analogy, i.e. older infected workers are replaced by young uninfected workers time after time. Often larvae of the honeybee are more sensitive to epidemic disease than adults, and furthermore infection of the queen is fatal to the entire colony. Larval care and queen attendance by the youngest workers, least likely to be infected, can be an effective antidisease strategy. Variation of lifespan of sterile workers, which had almost no logically convincing evolutionary explanation so far, can also be discussed using the conveyer belt analogy. A quick conveyer belt, i.e. short-lived workers, is an effective counter strategy for a high disease load in some situations, because a colony can more quickly 'metabolize' working force.

Importantly, many of these concepts have been analysed by explicit mathematical models (though most of them were basic and do not cover each special case of biological complexity) which have provided a number of testable predictions. The extremely wide scope of this book, however, may make it difficult for readers to organize their own studies to focus on a few relatively important issues. Therefore, I will try to suggest a few topics of general interest in social evolution. The first issue is colony size and infection risk. There has been no general consensus on the evolutionary explanation for inter- and intraspecific variation of colony size with an explicit biological mechanism. It is well known that larger colonies tend to have larger reproductive output, whereas per capita (per worker) reproductive output often decreases as colony size increases (reproductivity effect). Schmid-Hempel sets out a possible mechanism for this, namely that larger colonies will have a higher infection risk. His rationale seems logically robust; if there is a given fixed probability of new infection equal for all forager workers, there is a higher probability that a disease is imported into larger colonies. Although inter-specific comparisons presented in the book did not fully support the above idea, Schmid-Hempel uses the

assumption of positive correlation between colony size and disease load in order to deduce many other hypotheses. I feel that empirical and theoretical studies should pay careful attention to this topic, since colony size is one of the most important characteristics that affect many other traits of social insects (*J. Evol. Biol.* **12**: 245, 1999).

Secondly, disease and genetic variability particularly related to queen multiple mating. Since this topic has already attracted much attention, the current status is not only exploring theoretical models but also empirically testing of models predictions. While the genetic bet-hedging hypothesis as the cause of multiple mating is recently at a little discount for solitary organisms (*Tren. Ecol. Evol.* **13**: 246, 1998), in social insects genetic variation among worker progenies caused by queen multiple mating may enhance the mother queen's fitness. The key factor is the special population structures where related individuals frequently interact with each other, under which genetic variability among offspring can reduce the disease load by 'half sib-co-operation' (*Tren. Ecol. Evol.* **13**: 246, 1998). I believe interdisciplinary discussions on this theme among students of sexual selection, social evolution and life history strategy will be fruitful.

Finally, the evolution of virulence and resistance and the host-parasite population dynamics, discussed in Chapters 7 and 6, respectively, are also very fundamental topics. To advance further we particularly need empirical data, as Schmid-Hempel states. We should seriously seek for model organisms, like *Drosophila* in the biology of solitary animals, for experimental studies in laboratories and seminatural conditions. Honeybee colonies, the best known domesticated systems for disease issues, seem too bulky for this aim. Some ants and bumblebees could be the candidates for the host materials. We also have to choose corresponding parasites which are easily tractable.

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The concept of fitness and individuality revisited

A review by Csaba Pál and Eörs Szathmáry

Darwinian Dynamics: Evolutionary Transitions in Fitness and Individuality. By Richard E. Michod. Princeton University Press, New Jersey, 1999. \$45.00/£27.50. ISBN 0-691-02699-8.

Darwin himself considered fitness as a measure of the differential survival and reproduction of organisms. But are individuals the real targets of selection? Two problems emerged with the individual fitness concept. First,

under sexual reproduction, genes continuously reshuffle, and therefore offspring genomes are not identical copies of those of their parents. Second, when selection is occurring on several levels simultaneously, a fitness measure at any one level is a poor measure of the outcome of evolution. Therefore, the time is ripe for changes in concepts of fitness and individuality.

In his new book, Michod discusses the role of fitness concepts in evolutionary explanations. He gives special attention to multilevel selection conditions. As clarified in the first chapter, the book is written for two kinds of readers: those who are interested in the role of fitness in evolutionary explanations and those who wonder about causes of evolutionary transitions. We discuss these topics in turn. Darwinian dynamics relates to the frequency changes of those entities that satisfy the conditions of multiplication, heredity and variation. The author believes that the goal of the fitness concept is to describe the dynamic process of these entities. Because of the insufficiencies of the individual fitness function, he argues that a dynamically sufficient concept of fitness cannot relate to an overall property of organisms but instead must be associated with the dynamics of evolutionary change. According to this view, organisms are only temporary alliances of genes. Michod also argues that genetic transmission and survival are inextricably intertwined, and therefore it is not possible to separate selection and transmission of certain traits (as opposed to the usual procedure in quantitative genetics). We think that Michod's arguments on the fitness concept are well worth further discussion.

The questions mentioned above are especially relevant when the units of selection (such as genes, cells or organisms) are changing. An appropriate fitness concept must be able to handle the interplay between the dynamics of lower- and higher-level entities, and the evolutionary transitions between them. Michod argues – we think correctly – that in order to give a formal explanation for the emergence of higher-level entities, one should study the emergence of new levels of fitness. This study must be inherently related to the regulation of the selfish tendencies of the lower level entities.

Michod not only provides a detailed philosophical argument about fitness but also shows how to evaluate his concept in practice. The first field where he applies his concepts and techniques to is the origin of life, more specifically the transition from solitary genes to networks of genes. He provides a didactic case study, as it were, to introduce the reader to his modelling framework. Although well written from a technical point of view, it is unfortunate that the material on the particular subject matter is partially outdated and misleading. Michod wants to show how hypercycles emerge from unlinked genes. A molecular hypercycle is a network of replicators in which a member of the cycle catalyses the replication of the next one, and so on, until this higher level cycle is closed: the last member catalyses replication of the first.

There are serious doubts as to the prebiotic importance of hypercycles, naked or compartmentalized (Szathmáry, 1989). The main point is that naked hypercycles are neither necessary nor sufficient for the integration of genetic information, and that compartmentalized hypercycles are not necessary either. There are alternative models that solve the same problem: none of them is hypercyclical. Hypercycles seem to be too complicated, essentially because they double (or more than double) the energetic, mutational and parasite load on protocells. It is a mistake to think that these alternative models are all extensions or variations of Michod's (1983) model neighbour-modulated fitness model of prebiotic replicators. The stochastic corrector model is an explicit protocell model resting on group selection, where the groups are equivalent to the protocells (Szathmáry & Demeter, 1987). When Michod refers to multicompartment viruses as examples of contemporary hypercycles, he again ignores alternative models and the fact that it has been pointed out that there is no evidence for hypercyclical coupling among the covirus particles (Szathmáry, 1992a,b).

The transition from unicellular to multicellular organisms was one of the major transitions in life. For the organism to emerge as a new unit of evolution, within-individual change and interaction must be sufficiently controlled. Maynard Smith & Szathmáry (1995) argue that by often reproducing through a single-cell stage, organisms ensure close genetic relatedness among their component cells. They argued that close genetic relatedness is sufficient to preserve the integration of the organism and to reduce the selfish trends at the cellular level. Using a theoretical (population genetic) framework Michod queries this statement.

In his model multicellular organisms are considered as a group of co-operating cells that are clonal derivatives of a single zygote. Therefore, they are genetically identical except from the emergence of somatic mutations. During proliferation deleterious mutations could lead to the loss of cell functions. These mutations could theoretically produce somatic cells with different replication or survival rate. Organism fitness is proportional to the general level of co-operation among cells. Mutations that benefit the rate of cell replication at the expense of the fitness of the organism are of special interest because they are a source of conflict between the lower- and higher-level entities. By increasing the length of somatic cell-lineages the strength of within-organism selection increases, and hence defective cells are expected to displace co-operative cells. Hence, in the transition from unicells to multicells, organisms had to invent special regulatory mechanisms to mediate cellular conflicts.

We feel there are some conceptual ambiguities in this interpretation. This work provides neither a mechanistic argument nor the selective steps towards multicellularity. Rather, the focus is on how complex organisms mediate the selfish tendencies of their component cells. However,

because early, primitive multicellular organisms contained only a few clonally derived cells, it is unlikely that within-organism variation and selection of genetically different variants played a substantial role during the very first steps toward multicellularity. Genetic conflicts are expected to occur only rather later during the emergence of complex organisms. Accordingly, we do not think that this framework provides a scenario for the transition toward multicellularity.

Another problem is the total neglect of *epigenetic inheritance systems* (Jablonka & Lamb, 1995). During development, genes are turned on and off, and the expression patterns of differentiated cells are passed through during cell divisions. It is known that mis-expressions occur at a high rate, and sometimes these failures are mitotically heritable. It is reasonable to suspect that heritable epigenetic errors can change the replication rate of somatic cells, even at the expense of the organism. There is a growing acceptance that epimutations could be the first steps toward uncontrolled growth – cancer (Jones & Gonzalzo, 1997). Hence, variation and selection could arise even among genetically equivalent cells. Heritable epigenetic failures are important from two points of view. First, epigenetic errors further increase the potential for conflict. Second, different mechanisms are needed to reduce the effects of epigenetic and genetic failures. Michod believes that organisms are expected to develop several regulatory mechanisms to reduce the possibility of internal conflicts. He states that this can be achieved: (a) by policing the selfish tendencies of the cells, thereby reducing the benefits of cheaters; (b) through reducing the mutation rate; and (c) through the invention of the germ line.

Buss (1985, 1987) mentioned several policing mechanisms that may act to ensure replication fairness among cells. However, we do not know whether they were selected for that function. Another way to reduce selfish tendencies is by lowering the frequency of genetic or epigenetic failures. However, the mutation rate of the total coding DNA content of the genome per cell replication seems to be conspicuously constant in diverse metazoan taxa (Drak *et al.*, 1998), even if the number of cell divisions toward gametes is highly variable. Clearly, this fact does not support the theory: we would expect to find a reduced mutation rate in organisms with high somatic cell-turnover. How can the discrepancy be resolved? Based on previous work, we suspect that the rate of gene expression failures is higher than the mutation rate (Holliday, 1987; McAdams & Arkin, 1999). Probably, the effects of mutations are insignificant in the stream of epigenetic failures, especially at the time of origins with unreliable development. Accordingly, organisms are more likely to invent special gene silencing technologies in order to reduce the frequency of mis-expressions, rather than mutations, in somatic cells. One possible candidate for such innovation is DNA-methylation. Besides other functions, DNA-methylation is

known to be a highly effective gene suppressor mechanism. This would be especially beneficial in organisms with numerous somatic cell divisions. As a support of the theory, the presence of DNA-methylation in invertebrates correlates with the amount of somatic cell turnover (Regev *et al.*, 1998).

It has also been stated that by sequestering groups of cells early in development, the germ line provides a specific adaptation against defective cells. Gametes are the product of a cell lineage with a fewer number of cell divisions than the rest of the organism. Consequently, by sequestering gamete-producing cells from somatic ones, organisms reduce the possibility of transmitting defective cells for the following generations. Because the possibility of within-individual conflict is especially high in organisms with long cell lineages, Michod's theory would predict that a germ line is expected to be present in clades with extensive somatic cell turnover.

In Metazoa, early determination of the germ line is widespread but not universal. This provides a plausible basis for an adaptive scenario. However, Michod's theory is not supported by the fact that those phyla with constant cell number – and hence with relatively low number of somatic cell divisions – *always* segregate germ cells early during development (Table 1). Furthermore, there are clades which, in spite of extensive somatic cell turnover, develop by somatic embryogenesis (Table 1). This pattern cannot be explained by phylogenetic constraints: all groups in Table 1 are polyphyletic (e.g. Valentine, 1997).

Although we accept the logical coherence of this adaptive scenario for the germ line, we doubt its relevance in the light of the number of ambiguities. Michod endorses the idea of Davidson *et al.* (1995) that primordial Metazoan development was essentially identical to so-called type I development (essentially maximally mosaic,

Table 1 Phylogenetic distribution of the germ line and cell constancy. Adapted from Buss (1985).

Taxon	Cell constancy	Early germ line-soma segregation
Nematoda	+	+
Gastrotricha	+	+
Rotifera	+	+
Tardigrada	+	+
Arthropoda	+/-	+/-
Ctenophora	-	+
Acantocephala	-	+
Onychophora	-	+/-
Annelida	-	+/-
Mollusca	-	+/-
Echinodermata	-	+/-
Chordata	-	+/-
Cnidaria	-	-
Phoronida	-	-
Priapulida	-	-
Bryozoa	-	-

no cell movements, no germ line), found in some contemporary marine larvae. Larger organisms would have been possible only with the invention of 'set-aside' cells (Davidson *et al.*, 1995) for further differentiation and development of adult organs. Although we cannot go into the origin of metamorphosis here, we do agree with Lewis Wolpert (personal communication) that it possibly could not have evolved by any other means but subsequent intercalation of larval stages. Therefore, the invention of 'terminal' set-aside cells would have amounted to unprecedented evolutionary foresight. Although we are tempted to think that the germ line is an early invention in the Metazoa, its emergence was probably due to the fact that it is easier to push the RESET button (i.e. can be done more accurately) for gametogenesis if the cells do not undergo extensive prior differentiation (Maynard Smith & Szathmáry, 1999)

Nevertheless, we think that Michod's theoretical framework provides the best null hypothesis currently available to study within-individual variation and selection. Clearly, some important details are missing, but this book could be a good starting point for further investigations. In summary, we found the book's argument on the fitness concept and on the major transitions exciting, but we disagree when it comes to the origin of life and evolution of multicellularity. The book is better on evolutionary transitions in fitness than on scenarios for transitions in individuality.

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List of books received (not reviewed)

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