Book reviews

Simulating Ecological and Evolutionary Systems in C. Will Wilson. Cambridge University Press, Cambridge. 2000. Pp. 301. Price £18.95, paperback. ISBN 0 521 77658 9.

What is not clear from the title of this book is that it tries to act as a guide to learning C, building from quite simple concepts in the early chapters – 'while' loops, 'if' statements and simple mathematical operators – to more powerful features of C such as pointers, structures, headers and arrays. I don't know how many times a student or colleague has expressed a desire to learn some programming, or frustration that they can't try out a simple idea with a few lines of code. These potential programmers will be very interested by this book because instead of being subjected to exercises matching names of cats with their owners in order to learn the language, the examples are all biological and right from the start you feel that your code is doing something interesting.

I am sure that C is the right programming language to learn for most theoretical biologists, considering the investment of time. It might not be the most modern of languages but it is very widely available, excellent at the number crunching characteristic of ecological modelling, is compatible (mostly) with C + + to give access to visual programming packages and object orientation, and is quicker than Java.

Wilson uses a Unix environment to execute his C code and some things, usually well flagged, will be specific to Unix systems, or less useful if you are using a PC environment. One example is that quite a few pages are dedicated to explaining how to write PostScript files. This is quite a good method if you don't have access to a graphics package to visualise your results but in the context of this book I felt it was not really appropriate.

Other aspects of the book should be applauded. The discussion on the use of random numbers is extensive and vital. Any model with a stochastic element must have a good random number generator or the results are meaningless. This is very well illustrated by using three different random number generators in the same simple model when the short repeat cycle in the numbers from the weakest random number function is exposed. At the end of each chapter there is a useful set of exercises to carry out. Often these take the form of ideas to expand the program, collect more data or add a new twist.

There is a heavy bias to ecological rather than evolutionary systems, although there is a whole chapter dedicated to two models on the maintenance of gynodioecy. In the absence of a book devoted to evolutionary modelling or population genetics, I think this volume is going to give anyone using it ideas, even if they don't have a strong interest in ecology.

My biggest criticism with the book is the very large content of maths. In the introduction the author makes it clear that the book grew from a taught course from which students without a strong background in maths were excluded. Be warned, if you don't have a good grasp of mathematics at a high level the discussions of the results of the simulation models and many of the rationales for using particular approaches will be indecipherable to you.

I am certain that this book will sell well and if used as a companion to another C programming book will be useful to many. However, it is not helpful enough to be used as a course in C on its own and with the very heavy maths content I suspect that only the dedicated or the maths graduate will work through to the end.

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Epistasis and the Evolutionary Process. Jason B. Wolf, Edmund D. Brodie III and Michael J. Wade (eds). Oxford University Press, Oxford. 2000. Pp. 330. Price £62.50, hardback. ISBN 0 19 512806 0.

A plane in flight that loses one of its two engines should at least be able to land safely. Loss of both engines, however, has a decidedly greater effect than two times the loss of the one. Looked at this way, its seems perfectly perverse that we should assume, when modelling evolution, that the relative effect of a given mutation is independent of the context. But this is what for the most part is done. That is, we assume there is no epistasis.

Consider a simple two locus haploid model. The wild type alleles we shall call A and B with alternative alleles a and b. An aB individual has fitness $1-s$ and an Ab individual has fitness $1-t$. What is the fitness of ab? We usually assume it to be $(1-t)(1-s)$, or, in the symmetrical case $(1-s)^2$. But this is the same as assuming that flying on no engines is only a little worse than flying on one. More precisely then, we should suppose the fitness to be $(1-t)(1-s) + \varepsilon$, where ε can be positive or negative. If ε is not zero, we have epistasis. The situation is yet more perverse when one realizes that when we discuss alleles at a locus in the diploid we typically do not make this simplifying assumption. Rather than writing fitness as 1, $1-s$ and $(1-s)^2$, for AA, Aa and aa, we instead make our uncertainty clear and write them as 1, $1-hs$ and $1-s$. By incorporating the h parameter we are in effect saying that we realize that the simpler assumptions need not apply. Most of the time they do not (e.g. if there is dominance).

So where did our strange assumption come from, is it at all defendable and if not generally true, does it really matter? While the first of these questions is not central to this book, it is touched on by many of the authors. All ascribe it to Fisher's influence, as he was the first to consistently make the assumption. Why did Fisher do this? Brodie suggests that Fisher, when analysing adaptive evolution, considered a model in which

mutants at a locus went through to fixation so fast that no interacting allelic variants at other loci were around to disrupt the process. This defends the use of single locus models but does not explain why the assumption was made. Most consider that Fisher was imagining a model in which, because of recombination, the background that any given allele would sit in was constantly churning so the average effect was all that we needed to consider. The average might well approximate to multiplicativity if ε is greater then zero, as often as it is less than zero.

I reckon a further reason needs to be given, namely ease of modelling. If one allows for epistasis then the parameters of your model go up, which, while possibly more realistic, can very easily lead to models of horrendous complexity. Just think about the number of possible sorts of interactions when positive and negative mutations are allowed and more than two loci are involved, and you will very shortly have enormous sympathy for those who prefer the simpler assumption. Attempting to make sense of the horrid terminology, discussed by some in the book, for the sub-categories of interactions leaves one wishing that $\varepsilon = 0$, so it could all go away.

Whatever the reason, the authors of this book make plain that epistasis should probably not be shoved under the carpet. Phillips, Otto and Whitlock make, I think, a most telling point: even if Fisher was right that on average ε is about zero, the little studied variance in ε still matters. This they illustrate by discussion of the evolution of recombination. They show how the parameter space within which the mutational deterministic model works shrinks dramatically when we assume $\varepsilon = 0$ on average but that there is also some non-zero variance.

For the most part the other authors simply point to examples where epistasis has been shown, or is likely, and appeal to our better instincts as biologists, in an unspoken plea that we should respect what the data says. Case histories where epistasis can not be ignored (e.g. two locus segregation distorters, discussed by Palopoli, and supergene complexes more generally, discussed by Kelly) aim to strengthen the case, but one always is left wondering about exceptions and rules.

What then more generally is going on? Is epistasis everywhere? Many studies say not. But here Templeton provides possibly the most incisive of the chapters in arguing that standard modes of analysis have such a baggage of Fisherian assumptions that it is actually biased against finding epistasis, it simply being what is left over when all other sources of variance are removed.

Even if epistasis were common, this still doesn't demonstrate that it is of any importance. Many chapters discuss the possible role in peak jumping after bottlenecks. Wright's view, antithetical to that of Fisher, was that epistasis was important because after population sub-division, the epistatic effects would allow different phenotypes in different sub-populations, without needing to evoke selection and local adaptation. Importantly, the finding that additive variation increases after bottlenecking, reviewed separately by Meffert and Goodnight, is strong support for Wright's position (or at least some version of it). This result is quite the opposite of what the simple Fisherian models would predict. This finding has been the spur to much of the research in this field. The Wrightian ancestry of many involved in epistasis research also goes a long way to explaining why all but one of the 24 authors is at a North American institute (and the other is an American abroad). At least two other areas have all but independently come around to thinking that epistasis is of importance. The problem of speciation and the evolution of hybrid incompatibility is one. Johnson, for example, provides a good overview of the possible role of epistasis in explaining Haldane's rule. Many also touch on the importance of epistasis in the maintenance of sex. Peters and Lively's chapter is an unusually clear account of this problem.

But all is not always clarity and light. The definition of epistasis I have given above is just one definition. The term non-additive is used regularly throughout the book but it is not always clear just what is meant. The multiplicative fitness assumption that I defined above is not additive, it only becomes so if we log transform the fitnesses $[\text{Log}(W_{ab}) = \text{Log}(1 - t) + \text{Log}(1 - s)]$. Likewise, not all authors talk about fitness. Many concentrate on gene interactions in determining phenotype. Rice, for example, makes a well considered case that the evolution of development cannot really be considered without thinking about epistasis of some variety. But, as Brodie makes clear, effects that are additive for phenotype can be non-additive for fitness and *vice versa*. So is a situation that shows epistasis for phenotype but not for fitness epistasis or not? If one defines epistasis broadly enough I am sure it is quite possible to find it anywhere and everywhere.

Confusion over just what epistasis might be is not then entirely abolished, although the chapter by Phillips, Otto and Whitlock goes a long way to unscrambling the problem. This chapter alone, for its sheer clarity, should be recommended reading for anyone interested in the problem but confused by the terminology. The introductory chapter by Brodie attempts to do the same graphically but leaves more problems than answers.

The only strong criticism I would make, however, is that the book seems confused as to the prospective audience. The stated intention is to act as a primer. But here I think the book fails. I assume that Brodie's introduction was designed with advanced undergraduates in mind, but many chapters assume at least some working knowledge of quantitative genetics. Personally I found Cheverud's chapter on methods to detect epistasis among QTLs completely impenetrable.

Nonetheless, without exception the chapters are well considered and reach guarded conclusions. Indeed, in this context it was rather fun to see Wade cite a particularly strong and unguarded assertion from Brian Charlesworth. In a letter to Norm Johnson he posits that 'this relentless and futile search for intraspecific epistasis needs to be abandoned!'. The book convinced me that Charlesworth is wrong on this one. With the new methods outlined in the book and masses of data from genome projects, now is the time we can start to ask seriously whether epistasis is the exception rather than the rule.

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