an increase in local population density, and $\beta SI/N$, with I, S and N representing numbers will not model mass action.

The issue of estimating local population density is indeed not trivial [4]. However, all standard transect-, quadrat- or line-based population estimation methods (and most indices) return an estimate of local population density, rather than of population size. Mark–recapture methods aim to estimate population size, but measure local density unless applied to a spatially constrained population. The real issue, which we discussed at length, is determining the appropriate 'local' scale on which transmission occurs, and how variation in local density can cause transmission to differ from what might be predicted from mean population density over a larger area.

De Jong *et al.* suggest that most of the variant transmission models that we describe are empirical, but mechanistic derivations have been proposed. These largely involve spatial patchiness of disease, leading to a transmission term using 'mean crowding' rather than mean density [5]. This is the mean field approximation to a spatially heterogeneous process, and leads directly to other approximations, such as the negative binomial, as discussed in Ref. [1]. The approximations are equally appropriate for any heterogeneity in risk of infection between individuals [6]. Because of this mechanistic link, we prefer the term 'phenomenological' to 'empirical'; the models have a functional form that is intended to represent more complex processes.

One of our main conclusions was that spatially explicit mechanistic models of the transmission process and of the contact structures are required. However, simple models that represent transmission with a few parameters will continue to be necessary and the correspondence between the two is an important area for future investigation.

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Development and selection in adaptive evolution

In their recent exchange in *TREE*, Beekman [1] and Crespi [2] replay, with reference to social evolution, one of the oldest debates in evolutionary biology: does selection or a developmental mechanism explain the form of complex traits? Beekman's point, that complex form can arise via self-organizing processes whose details do not require explanation in terms of selection, echoes the 1894 argument of William Bateson [3], who showed that complex developmental variants can occur within species, without cumulative detailed change mediated by selection. Indeed, Darwin's emphasis on gradualism was linked to his emphasis on stepwise selection as the primary architect of form, and it is for this reason that saltatory developmental change has long been regarded as a challenge to darwinian selection theory. Other modern manifestations of the development versus selection controversy include the idea that developmental constraints can direct evolution, and the 'spandrel' argument [4] that traits can be nonadaptive developmental side effects of adaptive traits.

An important point not made by Beekman, Crespi or many previous discussants of adaptation versus development [3–8] is that all evolutionary novelty must originate because of some initially unselected developmental mechanism or response that effects a more or less complex reorganization of the phenotype before the occurrence of selection. Then, to persist or spread, the novel form (even if initially established because of drift) must eventually pass a test of selection. That is, it must have a net neutral or positive effect on fitness relative to other variants that arise. In other words, both an initially unselected mechanism and a selectively neutral or positive result must characterize every observed widespread and persistent characteristic in nature.

It is therefore misleading to engage in an either/or debate about whether selection or developmental mechanism (e.g. self-organization) explains an observed form. Both do, inevitably. It is also not enough, as in Crespi's response [2] and previous discussions of this issue [6–8], to reiterate the distinction between proximate (developmental) and ultimate (selective) factors as explanations of form. Both developmental mechanism and selection should be regarded as ultimate (evolutionary) factors in discussions of evolutionary adaptation, because a novel developmental mechanism must produce a new phenotypic variant before selection can affect its persistence and spread. The interesting feature of self-organization is not that it can produce novel form without selection (all persistent developmental mechanisms have done that), but that every separate detail of structure need not be explained in terms of a separate mechanism with selection operating step-by-step to gradually assemble a complex and coherent whole.

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