Limiting factors and population regulation

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The great debate over the meaning and relevance of population regulation lingers on under the perceived difference between limitation and regulation of animal numbers – what Murray (1999) calls the “population limitation hypothesis” versus the “density dependent regulation hypothesis”. Berryman et al. (2002) attempt to solve this dilemma by (1) arguing that the problem is partly caused by ambiguity and inconsistency in the use of the term density dependence, (2) deducing the necessary and sufficient conditions for population regulation from first principles, and (3) concluding that there is no real distinction between the concepts of population limitation and regulation. Apparently we were unconvincing. White (2004) criticizes our argument on empirical grounds -“the evidence from nature is that populations are not regulated; they are limited by the capacity of the environment to support them”- and insists that limitation and regulation “are fundamentally different processes”. In spite of the rhetoric, it seems to me that the latter issue is the crux of the problem because, if I can show that limitation and regulation are in fact and parcel of the same phenomenon, it should put the other matters to rest as well. In addition, the empirical evidence for population regulation has been addressed at length previously (Sinclair 1989, Harrison and Cappuccino 1995, Huffaker et al. 1999). Since our appeal to logic seems to have failed, I now try a more empirical approach. Figure 1 shows the dynamics of six Tribolium confusum populations growing in the laboratory on constant but different food supplies (Chapman 1928). Notice that the six populations grow at first but eventually stabilize at, or more correctly fluctuate to varying degrees around, an average density (I note that the population with the largest food supply may not have reached its steady-state). Since the level at which the populations stabilize is directly related to the quantity of food, White would probably say that these Tribolium populations were being limited by food, and few ecologists (including this one) would disagree with him (Berryman 1999). Stated another way, we could say that food acts as the limiting factor, an old idea in ecology (Liebig 1840, Blackman 1905, Allee et al. 1949, Odum 1971). However, some of us would add that the Tribolium populations were also being regulated by density-induced negative feedback (Huffaker et al. 1999, Berryman et al. 2002). On this point, however, White would undoubtedly disagree, claiming that “there is no need to evoke regulation by negative feedback loops.” I now try to clarify this difference of opinion by reference to Chapman’s experimental populations. It seems pretty obvious that the per-capita birth (B) and death (D) rates of a population at or near steady-state must, on the average, be equal. Defining the realized per-capita rate of change of a population over a given interval of time as $R = \ln(1 + B - D)$, then we can say that, on the average, $R = 0$ during the latter part of the Tribolium experiments, when the populations were at their highest. (See Berryman 1999, 2003 for the reasons why $R$ is expressed as a natural logarithm.) In contrast, births must have exceeded deaths, and consequently $R > 0$, at the beginning of the experiment when the populations were small. From this it seems logical to propose that $R$ must be inversely related to population size, $N$. Actually, since the Tribolium populations can only change through births and deaths, the value of $R$ over a sampling interval can be estimated by subtracting the logarithmic count at the first sampling date from that at the second; i.e. $R = \ln N_t - \ln N_{t-1}$, where $N_t$ is the size of the population sampled at time t. Plotting $R$ against the initial size of the population, $N_{t-1}$, gives us the $R$-functions for the populations growing on different food supplies (Fig. 2). Notice that all the functions have a negative slope over the complete range of the data, which satisfies both the necessary and sufficient conditions for population regulation (Berryman et al. 2002). As White (2004) finds no fault with the formal deriva-
tion of these conditions, it may be difficult for him to escape their conclusion, that the Tribolium populations are, by definition, regulated.

It is clear from Fig. 2 that food supply (the limiting factor) has little effect on the maximum per-capita rate of change (i.e. \( R_m = 3.6 \pm 0.12 \)) but has a profound influence on maximum numbers attained, or what is often called the “carrying capacity of the environment” (i.e. \( K = 1820 \pm 2043 \)). In fact, linear regression analysis indicates that food quantity explains almost all the variation (99.8%) in the observed carrying capacity (Fig. 3). It is interesting and significant that this model completely defines the qualitative dynamics of the Tribolium system (see Berryman 1981 for details about the “reproduction plane” depicted in Fig. 3). For example, if an experiment is begun with 1000 beetles in 80 grams of flour (the point x in Fig. 3), the model predicts that the population will grow and eventually stabilize at about \( K_{80} = 43.17 \times 80 = 3454 \) individuals. From this point of view I have to agree with White (2004), that knowledge about the limiting factor is all one needs to predict how many individuals can be supported by a given environment, and that, if this is all one requires, then the notion of regulation is indeed redundant. On the other hand, Fig. 3 cannot tell us how long it will take a population to reach its carrying capacity, nor can it explain how the population is kept at or near this level. To do this requires knowledge about the process of population regulation.

Perhaps I can use an analogy to illustrate this point. The temperature of a room is regulated at a given level (the set-point) by negative feedback between a thermostat and a heater and/or cooler. On the other hand, the set-point is determined by an external influence, the person who turns the thermostat dial to a particular setting. In this example the set-point is equivalent to the carrying capacity of the Tribolium system, and the person who sets it is analogous to the food supply. If one is only interested in the factor that determines the set-point, then this explanation may be sufficient (I set the thermostat, or food determines carrying capacity, and that’s that!). However, if one is interested in explaining how temperature (or the Tribolium population) is kept at (or near) this level, then it is necessary to understand the mechanism of temperature (or population) regulation by negative feedback. This information is obviously necessary to engineers, for without it they would be unable to control the temperature of buildings or the flight of missiles. It is equally important to those who deal with biological systems.

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**Fig. 1.** Growth of six Tribolium confusum populations in the laboratory (27°C) when provided with specific quantities of whole wheat flour at the time of sampling (data from Chapman 1928).

**Fig. 2.** Relationship between the logarithmic biweekly per-capita rate of change (\( R = \ln N_t - \ln N_{t-1} \)) and initial population size (\( N_{t-1} \)) for Tribolium populations grown on different amounts of food. \( R_m \) is the maximum value of \( R \), which occurs at the beginning of the experiment when \( N_{t-1} \) is small. \( K_F \) is the carrying capacity of a population provided \( F \) grams of flour at the time of sampling; i.e. \( K_F = K_{80} \) at the point where \( R = 0 \). Lines are fitted logarithmic R-functions. Data from Chapman (1928); I should note that Chapman did not sample his populations at constant intervals, so the rates were adjusted to a common two-week sampling period.

**Fig. 3.** Relationship between Tribolium carrying capacity, \( K_F \) (from Fig. 2) and the rate of food supply, \( F \) (grams of flour provided every 2 weeks), fit by the linear regression \( K_F = 43.17F; r^2 = 0.998 \). Arrows mark the direction of population change when an experiment is started at the coordinate x (80 grams of food and 1000 Tribolium adults).
ecologists who wish to understand, model and predict the dynamics of living populations.

Chapman (1928) discovered that the mechanism of population regulation in *Tribolium confusum* was cannibalism of immature stages (especially eggs) by adult beetles, noting that “the per cent of eggs eaten varied directly with the population of adults per gram of flour”. It is this negative relationship between the survival of immature stages and adult density that provides the negative feedback necessary for population regulation. It suggests that a model for the process of population regulation should be based on the density of individuals per gram of flour or, in a more general sense, on the ratio of population size to its limiting factor (Berryman 1999). When Chapman’s data are expressed as density per gram of flour, we find that they are defined by a common R-function (Fig. 4). With this model, one can compute the trajectory of any *Tribolium confusum* population growing under similar conditions, including those in which the food supply is varied continuously or randomly (Fig. 5). It not only predicts the eventual steady-state, but also the time it will take to reach carrying capacity from any starting density, and the pattern of fluctuations expected in constant or variable environments. In other words, it defines the complete dynamics of *Tribolium confusum* populations growing under the specified physical conditions.

It is now possible to see the connection between population limitation and regulation. The former focuses on the environmental factor(s) that sets the limit to population growth while the latter focuses on the process that keeps the population at or close to this limit (Berryman et al. 2002). It makes sense to talk about limiting factors, but I’m not so sure about regulating factors. It makes sense to talk about the process (or mechanism) of population regulation, but what about limitation? Actually the meaning of the word limitation is “being limited” and, therefore, it implies a mechanism that brings this about. From this point of view it is difficult to see the distinction between the notions of population regulation and limitation. However, regulation seems to be a more general and technically correct term since it can be applied to any negative feedback process, including those resulting from mutual interactions between consumers and their resources (Berryman 1999, Turchin 2003). On the other hand, it does not seem unreasonable to employ the term limitation for the special case in which populations are regulated by intra-specific interactions (e.g. competition, cannibalism) in response to some limiting factor (e.g. food), provided that the factor is uninfluenced by population density.

I hope I have been able to convince the reader that the concepts of population limitation and regulation are not separate paradigms for ecology, as claimed by Murray (1999) and White (2001), but are part and parcel of the
same fundamental process. If there is a weakness in my argument, it is probably that it is based on laboratory experiments, which can be claimed are not necessarily representative of real life. For example, although den Boer and Reddingius (1996) allow that regulation is possible in laboratory environments, it is not in nature because “a regulating mechanism can only exist in a deterministic world”. It is not clear to me where they get this idea from but it is obviously wrong. For one thing, a laboratory experiment is not completely deterministic since organisms are, by their very nature, variable. For another, we can easily make the laboratory environment more stochastic by randomly varying the supply of food (Fig. 5B). But doing this does not change the nature of the regulating processes, since cannibalism will still increase with population density, nor does it prevent us from predicting the dynamics of the beetle population. What Chapman’s experiments allow us to do is to see the connection between the limiting factor and the regulatory process, a connection that would be difficult or impossible to visualize if the limiting factor was continuously changing. Even more important, perhaps, is that they provide a test for the predictions of more general theories. But that is another story.

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References


Erratum

Our 2004 paper “Making eggs from nectar: the role of life history and dietary carbon turnover in butterfly reproductive resource allocation” (Oikos 105: 279–291) contains the following incorrect mathematical expression:

\[ \delta^{13}C = C_0^{13C} C_{larval \, c} + ((\delta^{13} C_{diet} + f_a) - \delta^{13} C_{larval \, c})(1 - e^{-rt}) (p_{max}) + C_0^{13C} C_{larval \, c}(1 - p_{max}) \]  

(4)

This equation should appear as

\[ \delta^{13}C = (\delta^{13} C_{larval \, c} + ((\delta^{13} C_{diet} + f_a) - \delta^{13} C_{larval \, c})(1 - e^{-rt})) (p_{max}) + (\delta^{13} C_{larval \, c})(1 - p_{max}) \]  

(4)

The parameters estimated by the model (cited as bold in the article text) are as follows:

- \( p_{max} \) = the maximum % of egg carbon to derive from the adult diet
- \( r \) = the fractional turnover rate of carbon from larval to adult sources
- \( f_a \) = the isotopic fractionation associated with incorporating adult dietary carbon into eggs
- \( \delta^{13} C_{larval \, c} \) = the isotopic composition of egg carbon deriving from the larval diet.

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