Opinion is intended to facilitate communication between reader and author and reader and reader. Comments, viewpoints or suggestions arising from published papers are welcome. Discussion and debate about important issues in ecology, e.g. theory or terminology, may also be included. Contributions should be as precise as possible and references should be kept to a minimum. A summary is not required.

On host life-history response to parasitism

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While an increasing amount of empirical and theoretical work is now pointing to the ecological and evolutionary importance of parasites, host life-history responses to parasitism seem yet largely under-investigated. Among the few theoretical works devoted to this topic is a life-history model by Forbes (1993), aimed at predicting the optimal reproductive effort of parasitized hosts.

This model builds on the concept of trade-off between current and future reproduction (Fig. 1). Because natural selection maximises the sum of these two quantities (Williams 1966), the optimal strategy can be found graphically as the point at which the trade-off curve has slope minus one (Fig. 1a). And since the shape of the trade-off depends on the environment, so does also the optimal strategy. Parasites are an important environmental component of reproducing organisms, and as such are bound to affect this trade-off. As argued by Forbes, different parasites may have different effects: so-called Type-I parasites are defined as having the potential to greatly reduce current reproduction, while Type-II parasites act only on the future reproduction of their hosts. As a result, suggests Forbes, hosts should respond to Type-I parasites by decreasing their current reproductive effort (RE) and to Type-II by increasing it. These claims rest on the graphical argument illustrated in Figs 1b and c. Type-III parasites, finally, have potential effects on both current and future reproduction, and therefore should induce either a Type-I (decreased RE) or a Type-II (increased RE) response, depending on the relative values of these effects.

The present note is a critical comment to some aspects of Forbes's model, in particular its applicability to the issue of parental effort. We first discuss a counter-example, in which experimentally introduced Type-I parasites elicited a "Type-II" response (increased RE), thereby falsifying one central prediction. We then point to the reasons for failure, that lie in some critical assumptions of the model onto the way parasites affect reproduction. And we finally propose an extended model, more realistic in its assumptions, and that can account for the empirical counter-example presented.

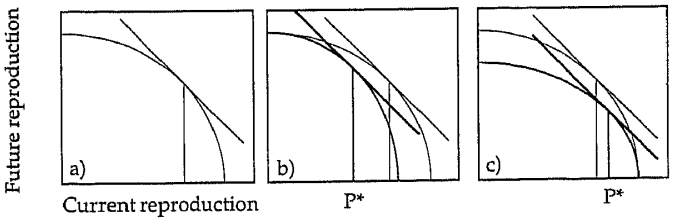

Fig. 1a). Trade-off between current and future reproduction. The evolutionary equilibrium is the point at which the trade-off has slope minus one. Thus, if a parasite affects the trade-off as illustrated in b), then the optimal current reproduction (P*) decreases. By contrast, if a parasite affects the trade-off as illustrated in c), then the optimal current reproduction (P*) increases.

Empirical counter-example

Our example stems from experiments recently performed around the campus of Lausanne University (Christe et al. 1996). The host under study was the great tit (*Parus major*), and its parasite the haematophagous flea *Ceratophyllus gallinae*. Fleas undoubtedly fit Forbes's Type-I category, since, like entomophilic mites, they "... develop [...] on their hosts for relatively short periods of time and then leave the host" (Forbes 1993). Indeed, *C. gallinae* has been shown to strongly affect nestling condition (Richner et al. 1993, Christe et al 1996), and thereby have the potential to greatly reduce current reproductive success.

The experiments consisted in infesting 14 out of 31 deparasitized nests with 60 fleas each, then comparing parental investment among the two treatments. This value of 60 fleas lies in the upper part of the natural range: the observed average was 37 fleas per nest (SD 28, range 3–85). The result most relevant to our present purpose is that adult males of parasitized nests increased their rate of feeding offspring by over 50% (those of females did not change significantly). This increase certainly was at the cost of their future reproductive success, since the amount of parental investment has been shown to increase the incidence of malaria in adult great tits, and thereby winter mortality (Richner et al. 1995). This result clearly opposes the predictions from Forbes's model, since the flea, categorized as Type-I parasite, elicited a Type-II response; i.e. an increase in reproductive effort, at the cost of lower residual reproductive value.

The question may arise whether fleas also have long-term effects on adults, in which case they would fit Type-III rather than Type-I. The fact is that we could not detect any change in adult weight, size or condition. Thus, if fleas have direct effects on adults, these must be lower than those on offspring, in which case Forbes's model would also predict a decrease in reproductive effort. Furthermore, and this is the central point of our note, there are good logical reasons to expect in fact such a Type-II response (and not a Type-I as predicted by Forbes) even in the complete absence of any direct and long-term effect of fleas on adult tits. These logical reasons we develop now.

The model's limitations

The failure of Forbes's model partly stems from a confusion between reproductive effort (RE) and current reproductive success (CRS): The x axis in Forbes's Figs 1 and 2, for instance, is correctly labeled as "current reproduction", but is referred to in the model as meaning reproductive effort. These, however, are different concepts, that represent different quantities. RE measures an investment in reproduction (time, energy, etc), while CRS measures the returns from this investment (i.e., benefits, in units of fitness). A given level of RE can result in strongly different CRS, depending on the environment. The effort of producing ten offspring, for instance, may be the same in environments A or B, but have quite different fitness results if all offspring survive to maturity in environment A, but only half of them in environment B. Thus, CRS is not equivalent to RE, but a function of it, which depends on the environment.

It is precisely on this function, and not on RE or CRS directly, that Type-I parasites like the fleas introduced in tit nests act: for a given RE, tits will get quite different fitness rewards depending on whether their

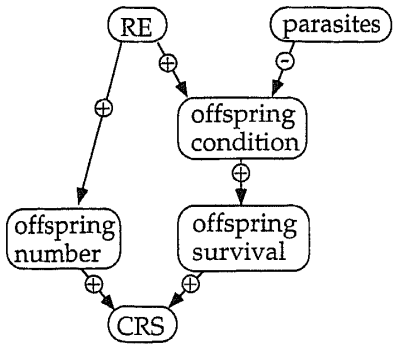

Fig. 2. The effect of parasites and reproductive effort (RE) on current reproductive success (CRS) is mediated by offspring number, condition and survival. The negative effect of parasites on offspring condition can be counterbalanced by an increased reproductive effort.

nests are parasitized or not. And, as we will show, Type-I parasites may affect this function in such a way that the host's optimal response is in fact to increase its current reproductive effort (as actually observed in tits). In this respect, the graphical argument developed by Forbes (see Figs 1b and c) is misleading: If Type-I parasites indeed changed the trade-off as shown in Fig. 1b, then obviously the optimal value of the current reproductive success (x axis) would be lowered, but this says nothing about the optimal reproductive effort, which might indeed increase. Furthermore, as we will show below, the actual effect of Type-I parasites (i.e. parasites detrimental to current offspring production) on this trade-off curve may differ quite drastically from what is illustrated in Fig. 1b.

Modified model

Our modified version of Forbes's model makes a clear distinction between RE and CRS. We reason as follows: the optimal decision at any age t is that which maximises the current reproductive value, v_t (Williams 1966):

$$v_t = p_o b_t + p_a v_{t+1} \quad (1)$$

where p_o is offspring survival, b_t is fecundity, p_a is adult survival until its next breeding cycle, and v_{t+1} its reproductive value at the start of the next breeding cycle. Eq. (1) makes explicit the fact that reproductive value is made of two components: the current reproductive success (not effort!), $p_o b_t$, and the residual reproductive success (RRS), $p_a v_{t+1}$.

The quantity $p_o b_t$ measures the CRS, so the way it depends on RE is of crucial importance for our argument. This dependence is mediated by both number (b_t) and survival (p_o) of offspring (Fig. 2). Offspring survival increases with condition, and condition in turn depends on both parental investment per offspring (which has a positive effect), and parasitism (with a negative effect). Thus, introducing 60 fleas in a nest will

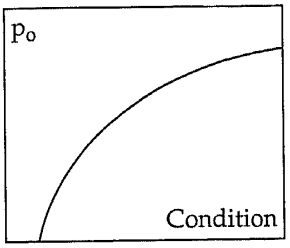

Fig. 3. Effect of offspring condition on survival (p_o). Survival probability is zero below some threshold condition level, and increases above it at a diminishing rate.

decrease offspring condition (and thereby survival), unless parents compensate for it by investing an additional RE (e.g., as argued by Johnson and Albrecht 1993).

At this point, assumptions have to be made as to the way parasites affect offspring condition, and thereby survival. The most plausible assumption in this respect (e.g., Smith and Fretwell 1974) is represented in Fig. 3: survival is zero below some critical condition value; it increases rapidly thereafter, then levels off at some asymptotic value. As a consequence (and for a given offspring number) CRS is also zero below some parental-investment value, increases rapidly above it, then levels off (Fig. 4a). It also follows from this assumption that a unit decrease in condition due to parasites has a much more drastic effect on survival if offspring are already in bad condition. This is shown in Fig. 4b, where Δ_{RE} represents the amount of additional parental investment that would be necessary to exactly compensate the negative effect of parasitism (i.e., achieve the same p_o level as unparasitized).

Now, since the optimal RE is the value for which the slopes of both current and residual reproductive success have the same absolute value (with opposite signs; Fig. 4a), it follows that adults should respond to such

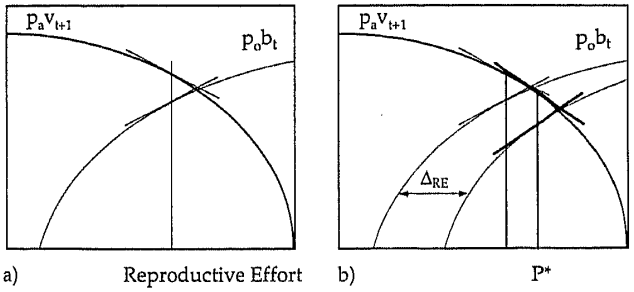

Fig. 4. a) Current reproductive success (p_ob_t) and future reproductive success (p_aV_{t+1}) as functions of reproductive effort; p_o is offspring survival and b_t offspring number; p_a is adult survival and V_{t+1} adult residual reproductive value. The optimal reproductive effort lies where the slopes are equal with opposite signs. b) The Type-I parasite under consideration is assumed to decrease offspring condition (and thereby survival) in such a way that an additional parental investment Δ_{RE} would be necessary to compensate for it (i.e., achieve the same survival as non-parasitized). The optimal RE (P*) is higher than that of non-parasitized, but not as much as Δ_{RE}, so that both current and residual reproductive success remain below the non-parasitized level.

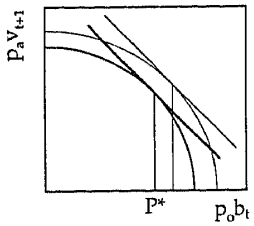

Fig. 5. Trade-off between current (p_ob_t) and future (p_aV_{t+1}) reproductive success; p_o is offspring survival and b_t offspring number; p_a is adult survival and V_{t+1} adult residual reproductive value. The thick line represents the effect of a parasite acting only on offspring survival probability (as in Fig. 4b). Future reproduction decreases for any level of current reproduction. The optimal response (P*) is an increase in current reproductive effort with, consequently, a decrease in both present and future reproductive success, relative to unparasitized birds.

parasites by increasing their parental investment (Fig. 4b). This arises because, at any RE value, parasites increase the slope of CRS vs RE, so that the equilibrium point shifts to the right. This outcome is understandable intuitively: parent tits that detect fleas should increase their parental investment, to make sure that nestling condition will allow them to withstand parasitism. Given that offspring are parasitized, parents gain more in investing a little bit more on them than a little less (as predicts Forbes's model), which would drastically decrease their current reproductive success.

This is clearly a Type-II response (according to Forbes's terminology) since hosts increase their reproductive effort in response to parasites. But, contrary to Forbes's model predictions, it occurs even though the RRS of adults is not directly affected. Thus, the modified model developed here correctly accounts for the experimental data presented above.

It is worth noting, however, that the additional reproductive investment predicted from the modified model is less than would be necessary to exactly compensate for the effect of parasites (Δ_{RE}). In other words, even though the RE of parasitized adults is higher, their resulting CRS is still lower than that of unparasitized (Fig. 4b). This prediction is corroborated empirically in the example discussed above, since parasitized nestlings showed a lower condition at fledging, in spite of the higher feeding rate (Richner et al. 1993, Christe et al. 1996).

Similarly, a prediction from our modified model is that, due to their increased parental investment, the residual reproductive success of adults is *indirectly* decreased by Type-I parasites (Fig. 4b). This must not be taken as a direct cost of parasitism, since it is mediated by the adaptive response of parents. A high feeding frequency seems indeed to increase the infection rate of adults with heamatozoa, which decreases their annual survival rate (Richner et al. 1995).

These two predictions are illustrated in Fig. 5 in the same representation as in Fig. 1 (i.e., in the RRS-CRS plane). The trade-off between current reproduction

($p_o b_t$) and future reproduction ($p_a v_{t+1}$) is modified by our Type-I parasite, although not in the same way as assumed by Forbes (compare to Fig. 1b). A change in the trade-off as shown in Fig. 1b might also result from some Type-I parasites, but under specific assumptions onto the way parasites affect reproductive success that obviously differ from ours. As appears in Fig. 5, under our assumptions the current reproductive success (P*) is indeed lowered at evolutionary equilibrium (as is also residual reproductive success). But the reproductive effort (which does not appear on this plane) is in fact higher.

We obviously do not claim that all Type-I parasites should elicit a Type-II response. There is strong empirical evidence that ectoparasites may also cause a decrease in reproductive effort. Ticks, for instance, can provoke complete nest desertion (the ultimate reduction in RE) by some seabirds such as brown pelicans (e.g. King et al. 1977), sooty terns (Feare 1976) and cormorants (Duffy 1983). House martins have been shown to reduce investment in response to hemipterans (Møller et al 1994), which may also induce nest desertion by cliff swallows (Emlen 1986, Loye and Carroll 1991).

Our point is that the relevant question to address in order to predict host response is not whether parasites potentially affect current reproduction more than future or vice versa. What matters is how parasites affect the shape of the relationship between reproductive effort and current reproductive success (or between maintenance effort and residual reproductive success). Depending on this, the optimal response may be either an increase, or a decrease in current reproductive effort. Thus, we are not rejecting Forbes's approach, but we insist that his model must explicitely include the relationship between reproductive effort and current reproductive success, for this relationship is likely to be strongly affected by parasites. That the optimal response to Type-I parasites might in some cases be an increase in reproductive effort logically derives from our argumentation, and is nicely illustrated by the empirical example of great-tit response to flea infection.

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