Behavioral Ecology
doi:10.1093/beheco/arj063
Advance Access publication 24 March 2006

# Habitat assessment by parasitoids: consequences for population distribution

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The ideal free distribution (IFD) is a stable distribution of competitors among resource patches. For equally efficient competitors, equilibrium is reached when the per capita rate of intake equalizes across patches. The seminal version of the IFD assumes omniscience, but populations may still converge toward the equilibrium provided that competitors 1) accurately assess their environment by learning and 2) remain for an optimal (rate-maximizing) time on each encountered patch. In the companion article (Tentelier C, Desouhant E, Fauvergue X. 2006. Habitat assessment by parasitoids: mechanisms for patch time allocation. Behav Ecol. Forthcoming), it is shown that the parasitoid wasp *Lysiphlebus testaceipes* adapts its exploitation of aphid host colonies based on previous experience, in a manner consistent with these two conditions. We therefore predicted that a randomly distributed population of initially naive wasps should converge toward the IFD. We tested this prediction by introducing 1300 *L. testaceipes* females into a 110-$m^2$ greenhouse containing 40 host patches. Just after introduction, the parasitoid rate of gain was positively affected by host number and negatively affected by parasitoid number but, as predicted, these effects vanished in the course of the experiment. Six hours after introduction, the expected rate of gain reached a constant. Surprisingly, this passage through equilibrium was not accompanied by a decrease in the coefficient of variation among gain rates or by a shift from a random to an aggregated distribution of parasitoids. These results challenge our understanding of the link between individual behavior and population distribution. *Key words:* aggregation, density dependence, ideal free distribution, interference, learning, *Lysiphlebus testaceipes*. *[Behav Ecol 17:522–531 (2006)]*

An increasing number of theoretical models describing population-level patterns in terms of the underlying individual processes are produced, as a means of deciphering population phenomena according to natural selection (Sutherland 1996). The ideal free distribution (IFD) described by Fretwell and Lucas (1970) is a classic example of such a model (reviews: Kacelnik et al. 1992; Lessells 1995; Tregenza 1995). According to the IFD model, the distribution of competitors among resource patches reaches a stable equilibrium when the per capita rate of gain is equal in all occupied patches. In the short term, this equalization of gain rates translates to spatial patterns concerning the distribution of competitors and the disappearance of resources, both of which depending on the nature and severity of competition (Lessells 1995; van der Meer and Ens 1997). These patterns may provide insight into the long-term dynamics of the populations involved (Sutherland 1983; Krivan 1997; Bernstein et al. 1999; van Baalen and Sabelis 1999).

Two types of IFD model may be distinguished according to how resources are declined and how competition affects consumers. The "continuous input model" addresses the simple case where resources arrive on the patches at a constant rate, may accumulate to an equilibrium standing crop, and are shared equally by the competitors. In this case, competition results entirely from exploitation—a decrease in intake with the decrease in resources (Lessells 1995). It results that 1) the per capita rate of intake is equal to the ratio of resource input rate to competitor density; 2) the equilibrium distribution is the "input-matching rule" (Parker 1978), according to which the proportion of competitors on a patch is equal to the proportion of resources arriving at that patch; and 3) the rate of resource disappearance is independent of the standing crop (Lessells 1995). The analysis becomes more complicated if consumers actively seek resources and competition also arises from interference—a decrease in the rate of intake with increasing competitor density. In this case, 1) the predicted per capita rate of intake may follow a variety of functional responses depending on modeling assumptions (van der Meer and Ens 1997); 2) the equilibrium distribution of competitors—although always consisting of a monotonous increase in the number of competitors with an increase in the standing crop of resources—depends to a much greater extent on modeling assumptions than the functional responses (van der Meer and Ens 1997); and 3) this is also true for the rate of resource disappearance and the subsequent dynamics of standing crops across patches (van der Meer and Ens 1997; echoed in Weber 1998). For these reasons and because continuous input situations are probably rare in the wild (Kacelnik et al. 1992), the equality of intake rates across patches may be the only robust prediction of the IFD model.

For gain rates to equalize across patches, individuals must have some knowledge about the available patches. In this respect, the IFD shares the common feature of most optimal foraging models. The assumption of omniscience, according to which competitors have perfect knowledge of the potential intake rate for all patches at all times, implicitly underlies the seminal IFD model (Fretwell and Lucas 1970). However, this

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Received 13 December 2004; revised 20 December 2005; accepted 18 February 2006.

assumption has since been relaxed and replaced by more biologically realistic assumptions. For example, foragers may acquire information about gain rates as they move between patches. This may lead to changes in behavior, resulting in the asymptotic convergence of the population distribution to the IFD. This idea was first formalized by Harley (1981) who defined a "learning rule for evolutionarily stable strategy (ESS)" as one that drives a population of initially naive individuals to an ESS in a single generation (see also Maynard Smith 1982; Houston and Sumida 1987). Envisaging the IFD as the steady state to contrast with, a variety of learning rules were investigated in simple continuous input systems including two patches (Harley 1981; Lester 1984; Regelmann 1984; Houston and Sumida 1987; Cézilly and Boy 1991; Thuijsman et al. 1995; Frischknecht 1996; Hakoyama 2003; Koops and Abrahams 2003). These models have a clear theoretical interest because they demonstrate that a set of decision rules based on simple cognitive processes can result in population distributions converging toward the theoretical equilibrium. Thus, omniscience is not necessary for populations to be ideal free distributed. These models may also be of value for studies of biological systems because some of their predictions are consistent with the existing data (Lester 1984; Regelmann 1984; Kacelnik and Krebs 1985).

In the wild, resource patches are much more variable than in continuous input systems. This is because natural systems include more than two patches, and the profitability of patches will vary both in space as a result of varying levels of interference and exploitation competition and in time as a result of resource depletion. The questions of patch assessment and of the subsequent spatial distribution of foragers in such a complex situation have been addressed by a single theoretical model, but to our best knowledge, not by experimental approaches. This model is an individual-based simulation model that we will henceforth refer to as the Bernstein, Kacelnik, and Krebs (BKK) model (Bernstein et al. 1988, 1991; but see also their "descendants" Beauchamp et al. 1997; Tyler and Rose 1997; Beauchamp 2000; Ward et al. 2000). It simulates individuals foraging randomly among 49 depletable patches over 40 time steps. The rule for patch departure is inspired from the frequency-independent optimality criterion of the marginal value theorem (MVT; Charnov 1976): each individual leaves its current patch when its instantaneous rate of gain falls below its personal estimate of the average environment-wide rate of gain. Individuals learn by weighting their own past gains against present gains by means of a linear operator (McNamara and Houston 1985). This model generates several predictions. Overall, if depletion is not more rapid than the learning process, competitor populations initially distributed at random should converge toward the IFD (Bernstein et al. 1988). If depletion occurs more rapidly or the environment is spatially structured, then the information gathered by individuals may not be representative of the entire environment and populations may remain further from equilibrium (Bernstein et al. 1988, 1991; see also Kacelnik et al. 1992; Krebs and Inman 1992).

In this theoretical context, the behavior of the parasitoid wasp *Lysiphlebus testaceipes* (Hymenoptera: Braconidae) is of considerable interest because it follows one of the main assumptions of the BKK model. Consistent with rate-maximizing models such as the MVT, *L. testaceipes* females behave in a time-limited manner as they must lay several hundred eggs during their life span of only a couple of days (van Steenis 1994). Host exploitation should be flexible (as defined by Piersma and Drent 2003) for two reasons. First, *L. testaceipes* is found on more than a hundred species of aphids and plants in the wild (Pike et al. 2000). Second, these aphids display high rates of population increase and density-dependent dispersal, so the abundance of aphid colonies within the habitat and the number of aphids within colonies fluctuates dramatically during the reproductive season of *L. testaceipes* (April–July in the south of France; unpublished data). *Lysiphlebus testaceipes* females adapt the time allocated to each patch on the bases of two complementary sources of information concerning the environment-wide abundance of aphids: 1) travel time between colonies and 2) the size of previously encountered colonies (see companion article; Tentelier et al. 2006). The use made of this information by *L. testaceipes* is consistent with the predictions of a version of the MVT based on learning (McNamara and Houston 1985) and, therefore, with the main assumption of the BKK model (Bernstein et al. 1988, 1991). On a given patch, host exploitation follows a type II functional response (Rochat 1997), but the level of interference is unknown. Finally, *L. testaceipes* females do not use long-range volatile cues other than the odors of host plants, whether infested with aphids or not (Lo Pinto et al. 2004). So, as assumed in the BKK model, females probably search randomly among patches.

We would therefore expect a population of initially naive and randomly distributed *L. testaceipes* to converge toward the IFD over time, even in environments containing more than two aphid colonies, provided that depletion is slow and that there is no spatial structuring of the number of aphids per colony. We report here a field test of this prediction, corresponding to the first explicit test of the BKK model and the first field test of the IFD for a parasitoid insect.

## THEORETICAL PREDICTIONS AND TESTS

The BKK model (Bernstein et al. 1988, 1991) defines convergence toward the IFD as convergence toward the equilibrium predicted by Sutherland's (1983) interference model for equal and omniscient competitors. The BKK model assumes the same type II functional response as Sutherland's model (1983), with searching efficiency affected by interference (Hassell and Varley 1969). By defining arbitrary values for each parameter of the functional response, Bernstein et al. (1988, 1991) calculated the equilibrium within-patch values for three different but interrelated measures: 1) the rate of gain, 2) the number of competitors, and 3) prey mortality. Equilibrium values for these three measures cannot be calculated for *L. testaceipes* because the effect of interference on patch exploitation is unknown. Thus, we must extract qualitative predictions from the BKK model that can be tested with no specific knowledge of the nature and severity of interference. Predictions for the rate of gain, the number of competitors, and prey mortality are therefore adapted from the BKK model. We assume a multipatch, host–parasitoid system with possible depletion. All variables are therefore denoted with the subscripts $i$, defining the patch, and $t$, defining the time.

### Parasitoid gain rates

The BKK model assumes that the instantaneous rate of parasitoid gain on a patch ($w_{it}$) increases with the standing crop of resources (here $h_{it}$, the number of healthy aphids) and either 1) is unaffected by the number of competitors (here $p_{it}$, the number of female parasitoids) if there is no interference competition or 2) decreases with the number of competitors if there is interference. Like other IFD models, the BKK model predicts that when the population converges toward equilibrium, all the per capita gain rates should converge toward a constant, common to all patches at a given time $t$ (Bernstein et al. 1988, appendix AI–1). This implies that when the population converges toward the IFD, the initial positive effect of the number of hosts and the negative

effect of the number of parasitoids on the rate of gain should cancel out. Consequently, at equilibrium, the rate of gain on any occupied patch should depend on neither the number of hosts on that patch nor the number of parasitoids.

In a multipatch environment, this prediction can be tested by fitting a statistical model to the measured rate of gain. This model should include, at least, time, host number, parasitoid number, and some of their interactions as explanatory variables:

$$w_{it} = \beta_0 + \beta_1 t + \beta_2 h_{it} + \beta_3 h_{it} t + \beta_4 p_{it} + \beta_5 p_{it} t + \varepsilon_{it}. \quad (1)$$

If the population converges toward the IFD, the effects of host number and parasitoid number should disappear, resulting in positive estimates for $\beta_2$ and $\beta_5$ and negative estimates for $\beta_3$ and $\beta_4$. At equilibrium, the instantaneous rate of gain should reduce to:

$$w^*_{it} = \beta_0 + \beta_1 t + \varepsilon_{it}. \quad (2)$$

The main effect of time should remain in the model to reflect the continuous process of patch depletion.

As a corollary, the coefficient of variation (CV) for $w_{it}$ should decrease over time. At IFD, the CV should converge toward zero if, as assumed, the only variables affecting $w_{it}$ are host number and parasitoid number (Bernstein et al. 1988, 1991). Of course, if attack rate is treated as a random variable, the CV will never reach zero.

### Parasitoid aggregation

The BKK model assumes that the foragers are initially naive and randomly distributed; the number of parasitoids on a patch is therefore independent of the number of hosts (Bernstein et al. 1988, 1991). As $w_{it}$ values converge toward a constant, the distribution of parasitoids converges toward the aggregative response predicted by Sutherland's (1983) model. The aggregative response is known to be sensitive to the functional response but always involves a monotonous increase in the number of competitors with the standing crop of resources (van der Meer and Ens 1997). Thus, one of the general predictions of the BKK model is that as $w_{it}$ tends to a constant, the initially random population distribution should become more aggregated, with the degree of aggregation depending on the nature and strength of interference (Sutherland 1983; van der Meer and Ens 1997).

This prediction can be tested statistically by linear modeling of the number of parasitoids in each patch at each time interval, with host number, time, and their interaction, at least, as explanatory variables:

$$p_{it} = \beta_0 + \beta_1 t + \beta_2 h_{it} + \beta_3 h_{it} t + \varepsilon_{it}. \quad (3)$$

If the population converges toward the IFD, the estimate of $\beta_2$ should not differ from zero and $\beta_3$ should be positive, reflecting the increasing effect of host number on parasitoid number.

### Host mortality

Host mortality depends on the number of parasitoids per patch and intake rate. Robust predictions of host mortality can therefore be made only if the functional and aggregative responses are known. In the absence of interference, host mortality at equilibrium should be density independent (Lessells 1995). With interference, mortality at equilibrium may be density dependent, density independent, or inversely dependent on density, according to the strength of interference (Kacelnik et al. 1992). Even doomed density dependence may be observed (Bernstein et al. 1991). It is therefore not possible to use predictions of host mortality to test the BKK model, unless analyses of gain rates have indicated an absence of interference.

## MATERIALS AND METHODS

These predictions were tested by releasing a population of naive *L. testaceipes* females in a patchy environment consisting of 80 cucumber plants (*Cucumis sativa* var. Carmen) in a 110-$m^2$ (20 × 5.5 m) greenhouse. Plants were about 1 m tall, had about eight leaves, and were arranged in four rows of 20 plants each.

We generated the spatial heterogeneity in host density required to test the theoretical predictions by manipulating the initial size of *Aphis gossypii* (Homoptera: Aphididae) colonies. Five days before parasitoid release, one leaf from each of 40 plants (every other plant in each row) was randomly infested with 5, 10, 20, or 40 apterous parthenogenetic *A. gossypii* females (10 replicates per level). This manipulation gave a spatially unstructured array of 40 colonies of 135–1736 healthy aphids at the time of parasitoid introduction. The distribution of instars was assumed to be constant for all colonies because all colonies were initiated simultaneously. We maintained the patchy nature of the environment by limiting aphid dispersal in three ways. First, the formation of alates during the 5 days of aphid reproduction was prevented by establishing colonies from wingless individuals reared at low density on the same cucumber variety in the laboratory. Second, during aphid reproduction before the release of parasitoids, colony-bearing leaves were enclosed in bags of polyester organdy. Third, just before the release of parasitoids, a ring of glue was applied on the petiole of each colony-bearing leaf, preventing the aphids from walking away from their colonies of origin. We henceforward refer to cucumber leaves bearing an aphid colony as host patches.

About 1300 naive *L. testaceipes* females were released into this patchy environment. The released females originated from a mass rearing over a large number of generations in the same trophic system (*A. gossypii* on cucumber plants). These females were released 1 h after their emergence, with conspecific males, in rearing cages. This 1-h period was assumed to be long enough to allow mating. For release, the females were transferred to a dozen smaller plastic vials. These vials were opened at random locations in the greenhouse, at 07:00 h, on November 18, 1997.

The variables estimated from sampling and observations are summarized in Table 1. We investigated changes in parasitoid distribution over time by counting the individual parasitoids on each of the 40 host patches every hour, from 8:00 to 16:00 h ($p_{it}$). We assessed changes in instantaneous parasitoid attack rate by recording the number of aphids attacked over a 4-min period by a focal female randomly selected on each patch, every 2 h from 10:00 to 16:00 h ($a_{it}$). The instantaneous reproduction rate on a patch ($w_{it}$, number of progeny produced in 4 min by an individual female) was obtained by multiplying the instantaneous attack rate by the average efficiency of an attack on that particular patch. The equation for $w_{it}$ is:

$$w_{it} = a_{it} \frac{(P_i/H_i)T_i}{135\overline{p_{i\cdot}}\,\overline{a_{i\cdot}}}. \quad (4)$$

The numerator of this equation describes the total number of progeny produced on patch $i$ estimated from a subsample of $H_i$ hosts. The denominator describes the total number of attacks across the 9 h of experiment (135 × 4 min) estimated from the average number of parasitoids and the average number of attacks observed on patch $i$ (see below and Table 1 for more details). We used simple populations dynamics to assess changes

**Table 1**
**Variables estimated from field sampling and focal observations**

| Variable | Description | Estimation |
|---|---|---|
| Time-independent variables | | |
| $T_i$ | Final number of (healthy + parasitized) hosts | Count at 16:00 h |
| $H_i$ | Number of hosts subsampled and reared | Count at 16:00 h |
| $M_i$ | Number of hosts that turned into mummies | Count 1 week later |
| $P_i$ | Number of emerging F1 parasitoids | Count 2 weeks later |
| $R_i$ | Per capita hourly risk of parasitism | $h_{i9} = h_{i0}\exp[(0.0146 - R_i) \times 9]$ |
| $A_i$ | Cumulative number of attacks over the 9 h (135 × 4 min) of the experiment | $A_i = \overline{p_{i\cdot}} \times \overline{a_{i\cdot}} \times 135$ |
| $W_i$ | Number of progeny produced per patch during the whole experiment | $W_i = \frac{P_i}{H_i} T_i$ |
| $S_i$ | Average success of an attack (number of progeny produced per attack) | $S_i = \frac{W_i}{A_i}$ |
| Time-dependent variables | | |
| $p_{it}$ | Number of parasitoids | Count |
| $p_{\text{tott}}$ | Total number of parasitoids | $p_{\text{tott}} = \sum p_{it}$ |
| $a_{it}$ | Instantaneous individual attack rate | Count of the number of attacks in 4 min by a focal parasitoid |
| $h_{i0}$ | Initial number of healthy hosts | $T_i = h_{i0}\exp(0.0146 \times 9)$ |
| $h_{i9}$ | Final number of healthy hosts | $h_{i9} = T_i\left(1 - \frac{M_i}{H_i}\right)$ |
| $h_{it}$ | Instantaneous number of healthy hosts | $h_{it} = h_{i0}\exp[(0.0146 - R_i) \times t]$ |
| $h_{\text{tott}}$ | Total number of hosts | $h_{\text{tott}} = \sum h_{it}$ |
| $w_{it}$ | Instantaneous individual reproduction rate (number of progeny produced in 4 min by a focal parasitoid) | $w_{it} = a_{it}S_i$ |

Uppercase letters indicate time-independent variables and lowercase letters indicate time-dependent variables. The subscript $i$ indicates the patch ($i = 1, \ldots, 40$) and $t$ indicates the time ($t = 0, \ldots, 9$; $t = 0$ at 7:00 h when parasitoids were released to $t = 9$ at 16:00 h when the observation period ended). $\overline{p_{i\cdot}}$ and $\overline{a_{i\cdot}}$ are the number of parasitoids and the attack rate averaged across time on each patch, respectively. 0.0146 $h^{-1}$ is the rate of increase of *Aphis gossypii* populations (Rochat 1997).

in host density, assuming that $h_{it} = h_{i0}\exp[(r - R_i)t]$. In this equation, $r$ is the estimated rate of increase in *A. gossypii* populations in the same conditions but in the absence of parasitoids ($r = 0.0146\ h^{-1}$; Rochat 1997), and $R_i$ is the estimated per capita risk of parasitism (also in $h^{-1}$) for each patch (Table 1). We counted all the aphids on each patch ($T_i$, healthy + parasitized) after the last observation at 16:00 h. This made it possible to calculate the initial number of healthy hosts per patch $h_{i0}$. We subsampled $H_i$ individuals (around 100) at random from the $T_i$ aphids and reared them in laboratory conditions for 2 weeks. We counted the aphids that turned into mummies ($M_i$) and those that produced a parasitoid ($P_i$). The proportion of mummified aphids in the subsample was used to estimate the number of healthy aphids remaining on each patch at the end of the experiment ($h_{i9}$). Finally, both $h_{i0}$ and $h_{i9}$ were used to estimate the hourly per capita risk of parasitism ($R_i$) and, in turn, all other $h_{it}$ values (Table 1).

We tested the theoretical predictions for the rate of gain and aggregation by estimating the parameters of Equations 1 and 3. We used generalized linear models because the three fitted variables ($a_{it}$, $w_{it}$, and $p_{it}$) do not meet the standard requirements of normality, homoscedasticity, and independence (as they are repeated over time). We therefore implemented these models with a Poisson distribution, a log link function for count data, and the generalized estimated equations (GEE) methods (Liang and Zeger 1986), making it possible to describe the correlation structure explicitly (and to estimate its parameter) from measurements made on the same patches at different times. These statistical models were fitted by maximum likelihood estimation of the parameters associated with the explanatory variables. For model selection, we used the backward procedure recommended by Crawley (1993), reducing maximal models (including all possible main effects and interactions) into minimal models (including only the significant effects and insignificant main effects that happened to be significant in interactions). The significance of parameters was assessed by means of score statistics (which converge toward the chi-square distribution; Boos 1992). These analyses were performed with Proc Genmod using SAS software (SAS Institute Inc. 1999). For other complementary analyses for measures not repeated over time, we used similar models with the correct distribution, but without the GEE option for repeated measures, and used likelihood ratio tests to evaluate the significance of estimated parameters.

We also fitted Sutherland's (1983) model to the data by means of a nonlinear iterative regression method and estimated the coefficient of interference $m$. The fitted model was:

$$\frac{p_{it}}{p_{\text{tott}}} = \left(\frac{h_{it}}{h_{\text{tott}}}\right)^{1/m}, \quad (5)$$

where $p_{it}$ and $h_{it}$ are as defined above, and $p_{\text{tott}}$ and $h_{\text{tott}}$ are the total number of parasitoids observed and the total number of hosts estimated to be present in the whole greenhouse at time $t$, respectively.

## RESULTS

### Instantaneous rates of gain

We used the rate of attack (the observed number of aphids stung per 4 min and per individual) and the rate of reproduction (the number of progeny produced per 4 min and per individual) as two complementary measures of instantaneous rate of gain. The rate of attack was affected only by aphid density (Table 2A). The parameter estimate for this variable

**Table 2**

**Most parsimonious (minimal) models for parasitoid instantaneous rate of attack (A) and instantaneous rate of reproduction (B)**

| | Estimate | SE | $\chi^2$ | $P$ |
|---|---|---|---|---|
| A—Rate of attack | | | | |
| $\beta_0$ intercept | 0.9444 | 0.1695 | | |
| $\beta_2$ host | 0.0006 | 0.0002 | 5.28 | 0.0216 |
| B—Rate of reproduction | | | | |
| $\beta_0$ intercept | 0.2052 | 0.3669 | | |
| $\beta_1$ time | −0.0386 | 0.0607 | 0.36 | 0.5469 |
| $\beta_2$ host | 0.0010 | 0.0004 | 1.99 | 0.1579 |
| $\beta_3$ host × time | −0.0003 | 0.0001 | 4.42 | 0.0355 |
| $\beta_4$ parasitoid | −0.7660 | 0.2604 | 6.77 | 0.0092 |
| $\beta_5$ parasitoid × time | 0.0890 | 0.0291 | 7.64 | 0.0057 |
| host × parasitoid | −0.0003 | 0.0001 | 4.16 | 0.0413 |

Parameter estimates (linear predictors) and their standard errors (SE) were estimated by fitting generalized linear models based on a Poisson distribution of errors, a log link function, and an unstructured correlation structure between repeated measures. Parameter significance was tested by means of score tests ($\chi^2$ and corresponding $P$ values with 1 degree of freedom). The β values correspond to the parameters of Equation 1 for which we had a priori predictions.

was significantly positive. Parasitoid attack rates were therefore higher on resource-rich patches than on resource-poor patches throughout the experiment. Attack rates were not affected by time, suggesting that decreases in the number of healthy hosts during the experiment had little effect on parasitoid foraging efficiency. If we consider the attack rate as a currency for fitness, then these initial results suggest that the population was not converging toward the IFD.

Very different results were obtained for the rate of reproduction, with almost all the explanatory variables of the maximal model significant (Table 2B). The effects of these variables on the rate of reproduction were similar to those expected for convergence toward the IFD (Equation 1). The rates of reproduction predicted by the statistical model are presented graphically in Figure 1. Overall, the rate of reproduction increased as the number of healthy hosts increased ($\beta_2 > 0$) and the number of parasitoids decreased ($\beta_4 < 0$), as assumed in the functional response of BKK and other models. However, consistent with the specific predictions of the BKK model, these effects gradually disappeared because the significant parameters for the interactions host × time ($\beta_3 < 0$) and parasitoid × time ($\beta_5 > 0$) had the opposite sign to the main effects (Table 2B). The expected rate of reproduction therefore became less variable over the observed range of host and parasitoid densities (Figure 1), suggesting convergence toward the IFD. In this analysis, the main effect of time on reproduction rate was not statistically significant, again indicating that the host depletion occurring during the 9 h of the experiment did not affect the efficiency of the foraging wasps.

As the interactions between time and host number or parasitoid number were significant, we carried out IFD-specific tests estimating the effects of host number, parasitoid number, and their interactions at each time step. At 2 h, parasitoid number had a negative effect, and host number had a positive effect via the host number × parasitoid number interaction (Figure 2). At 4 and 6 h, none of the parameters for each of the three explanatory variables was significantly different from zero, suggesting that the population had satisfied the conditions of Equation 2 and had reached the IFD. This conclusion is based on the acceptance of a null hypothesis ($H_0$: $\beta_i = 0$) with an unknown probability but is supported by the fact that the expected value for each parameter $\beta_i$ is very close to zero and that zero is well within the confidence intervals for these parameters (Figure 2). At 8 h, the significance of the host number × parasitoid number interaction suggests that there has been a significant deviation from equilibrium but not for the same reasons as at the beginning of the experiment. At 8 h, the expected rate of reproduction increased slightly with parasitoid density for high host density (Figure 1).

The CV for gain rates did not decrease with time (Figure 3). This finding conflicts with the results described above and with the predictions of the BKK model.

### Parasitoid aggregation

The number of parasitoids per patch was positively related to the number of healthy hosts ($\beta = 0.0011$; $\chi^2 = 11.12$, $P = 0.0009$) and time ($\beta = 0.1422$; $\chi^2 = 8.32$, $P = 0.0039$). These results suggest that, consistent with the predictions of IFD models, the distribution of the *L. testaceipes* population was host density dependent. The observed increase in the number of parasitoids per patch was probably due to individuals finding patches on plants. However, the number of parasitoids was not affected by a host number × time interaction ($\beta \cong 0.00$; $\chi^2 = 0.81$, $P = 0.3671$). This contrasts with the gradual shift from a random to an aggregated distribution predicted by the BKK model. Rather, the distribution of the introduced parasitoids seemed to be steadily host density dependent throughout the 9 h of patch exploitation (Fig. 4). When fitting Sutherland's (1983) model to the data, we obtained an estimate of $m$ that was almost constant and did not differ significantly from 1 (Figure 4).

### Host mortality and patch depletion

After the 9 h of exposure to parasitoids, about one-quarter of the aphids in each patch were parasitized (mean rate of parasitism and 95% confidence interval: 0.27 [0.25–0.28]). The rate of parasitism was density independent (effect of initial aphid density tested by means of logistic regression: $\beta = 0.00$; $\chi^2 = 1.45$, $P = 0.2289$). The hourly per capita risk of parasitism was estimated at 0.037 [0.030–0.045]. The equation for depletion, common to all patches, was $h_{it} = h_{i0}\exp(-0.0229t)$.

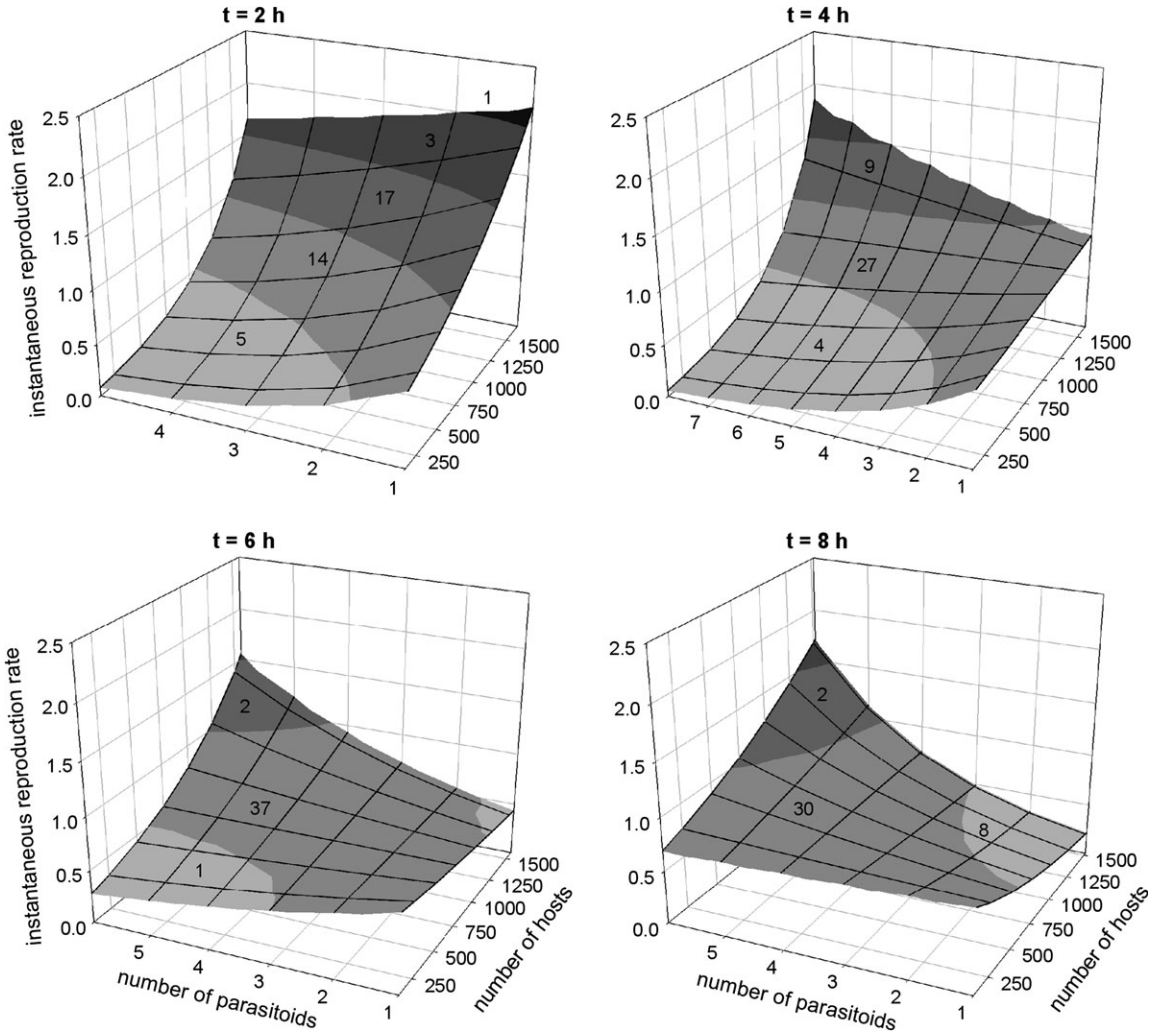


**Figure 1**
Expected number of progeny produced per individual in 4 min of direct field observation versus number of hosts and number of parasitoids on the patch. Expected values were obtained by fitting a log-linear model to the raw data with the number of hosts, the number of parasitoids, and time as explanatory variables (see text for details). The gray scale represents arbitrary classes of reproduction rates (every 0.5 progeny produced per 4 min), and the numbers shown correspond to the number of patches in each class. The scale of horizontal axes indicates the observed range for the number of hosts and number of parasitoids.

## DISCUSSION

### Field data and the BKK model

The aim of this study was to test the IFD (Fretwell and Lucas 1970) with an organism known to assess the profitability of its habitat by learning (see companion article; Tentelier et al. 2006). For this, we carried out a field experiment based on the theoretical predictions of the IFD model for learning foragers developed by Bernstein et al. (1988, 1991). When the distribution converges toward the IFD, these predictions are 1) the initially positive effect of host density and negative effect of parasitoid density on the rate of gain should decrease over time, resulting in a stable equilibrium in which these effects are no longer observed; 2) the CV of the rate of gain among patches should therefore decrease over time; and 3) simultaneously, the distribution of the parasitoids, which is initially random, should gradually become aggregated.

Our data for the parasitoid *L. testaceipes* are partly consistent with the first prediction, provided that the rate of reproduction rather than the rate of attack is used as a currency for fitness. Two hours after the introduction of parasitoids, the instantaneous rate of reproduction was found to increase with the number of hosts and to decrease with the number of competitors. This tendency is described as undermatching if there are fewer competitors (and hence, higher gain rates) than expected on the richest patches (Kennedy and Gray 1993). However, these effects vanished during the course of the experiment, such that the expected rate of reproduction approached a constant value 4 and 6 h after the introduction of parasitoids. Our results therefore demonstrate that a population of 1000 initially naive and randomly distributed parasitoids can converge toward the IFD, even in an environment consisting of 40 different host patches on 80 plants in a $5 \times 10^3$–$m^3$ greenhouse. However, this conclusion is tempered by the observed deviation from the IFD after 6 h. This deviation displayed a different pattern than that observed just after release, with gain rates increasing with increasing numbers of parasitoids at high host densities. Such an effect is not predicted by BKK models (Bernstein et al. 1988, 1991), not touched upon in the review of Kennedy and Gray (1993), and is therefore difficult to discuss in the light of the published literature on the IFD. Our hypothesis is that this

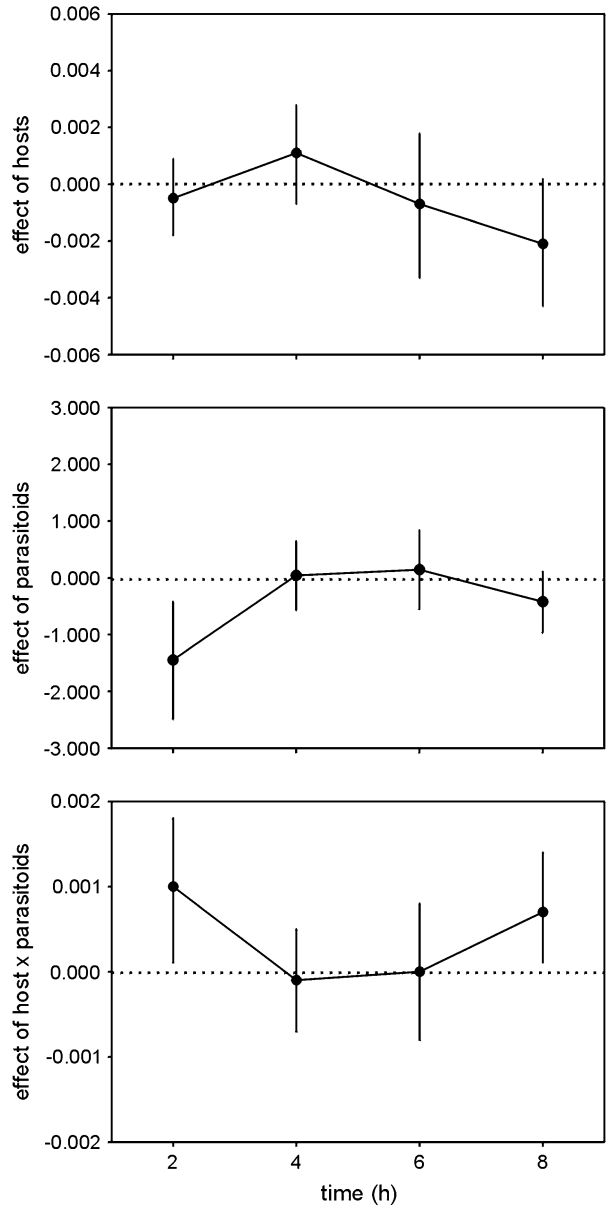


**Figure 2**
Parameter estimates for the effects of host number, parasitoid number, and their interaction on the rate of parasitoid reproduction. Estimates were obtained by fitting log-linear models to reproduction rates at each observation time. Dotted lines represent the equilibrium at which the rate of reproduction is not affected by host or parasitoid numbers. Vertical error bars indicate the 95% confidence intervals of the parameters.

observation 8 h after release reflects random fluctuations around equilibrium because in opposition with simulations, populations of insects like *L. testaceipes* never really reach a steady state in their natural environments. Longer time series are needed to test this hypothesis.

This passage through equilibrium contrasts with the results from the first and only other test of IFD in a parasitoid wasp (Tregenza et al. 1996). The authors argued that the parasitoid tested, *Venturia canescens*, would have been able to assess the environment profitability, but the distribution never reached the IFD. Our results are more consistent with studies suggesting that the distribution of a group of competitors (guppies, goldfish, or sticklebacks) can converge toward the IFD as a result of individual processes of patch assessment (Lester 1984; Regelmann 1984; Kacelnik and Krebs 1985; Abrahams 1989). Nonetheless, our data were obtained in a large and complex experimental environment, whereas the studies carried out on *Venturia* and fishes were carried out in more limited laboratory microcosms (6–24 wasps for 2 patches in a $4 \times 10^{-3}$ m$^3$ cage; 4–10 fishes for 2 patches in $10^{-2}$ to $10^{-1}$ m$^3$ aquaria). Hence, they demonstrate that the IFD is not restricted to simple and unrealistic experimental setups and may be applied to field populations (Wahlström and Kjellander 1995).

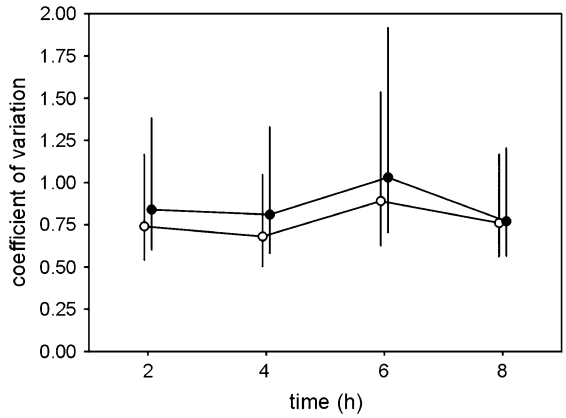


**Figure 3**
Coefficients of variation of attack rate (open circles) and reproduction rate (filled circles) over time since parasitoid introduction. Vertical bars indicate 95% confidence limits. Coefficients of variation and their confidence limits were calculated from log-transformed data.

Two other predictions of the BKK model were not confirmed by our data. First, the CV did not decrease as predicted (Bernstein et al. 1988, 1991). Second, the shift of the population from a random to an aggregated distribution and the convergence of reproduction rates toward a constant were not synchronous. We further discuss some possible reasons for these discrepancies between the data and the model.

### Variability among competitors

The BKK model, like many other IFD models, assumes that competitors have equal competitive abilities and resource consumption capacities. Consequently, only environmental variables such as the number of resource items and the number of competitors constrain the rate of gain on a patch (Bernstein et al. 1988, 1991; Kacelnik and Bernstein 1988; Lessells 1995; van der Meer 1997). For this reason, a direct consequence of gain rates being less affected by resource density and competitor density is a decrease in the CV of gain rates (Bernstein et al. 1988, 1991). The lack of such a decrease in this study calls this assumption into question and suggests that the reproduction rates of *L. testaceipes* females varied for reasons other than resource levels or competition severity.

The rate of gain may be intrinsically constrained by the phenotype, and phenotypes may not always be equivalent (Parker and Sutherland 1986; van der Meer 1997). Instead, they may differ in two ways (Parker and Sutherland 1986; Sutherland and Parker 1992): 1) efficiency to exploit resources and 2) efficiency to compete with others. Both types of variability are known to occur in parasitoids and to be genetically determined at least in part (Wajnberg et al. 1999, 2004). It is important to distinguish between these two sources of variability because the prediction of equal intake rate by the BKK

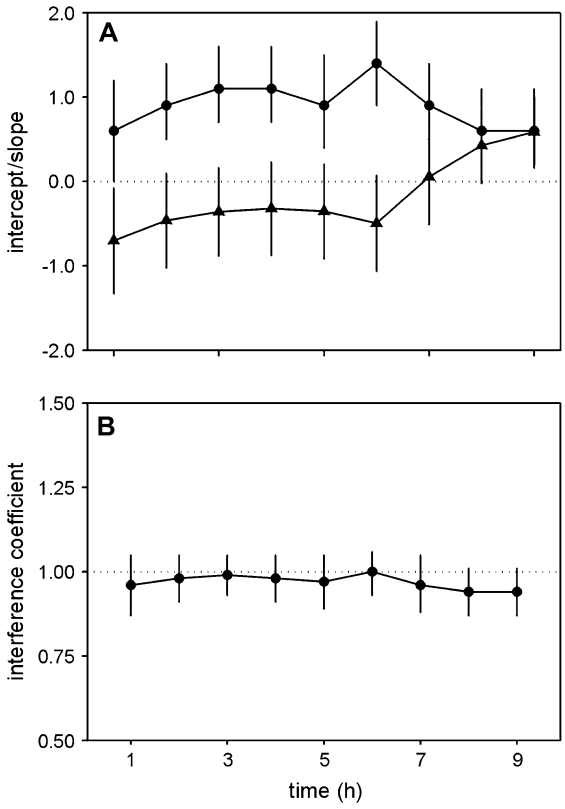


**Figure 4**
(A) Parameter estimates for the effect of the number of hosts on the number of parasitoids obtained by fitting log-linear models to the number of parasitoids at each observation time. Triangle: intercept; circles: slopes ($\times 10^{-3}$). (B) Estimated value of the interference coefficient $m$ obtained by fitting Sutherland's (1982) model to the data at each observation time via a nonlinear regression method. In both graphs, vertical error bars indicate the 95% confidence intervals of the estimates.

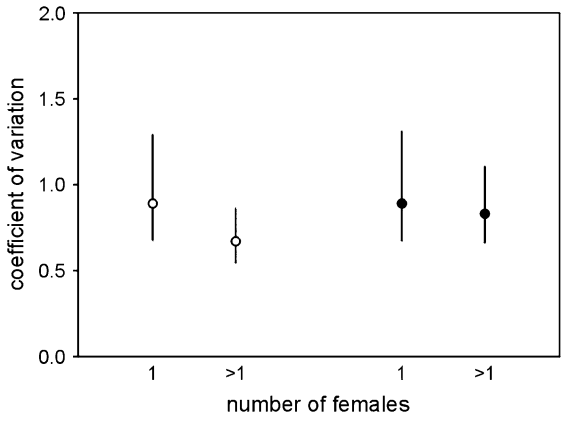


**Figure 5**
Coefficients of variation of attack rate (open circles) and reproduction rate (filled circles) for patches with one or more than one female. Vertical bars indicate 95% confidence limits. Coefficients of variation and their confidence limits were calculated from log-transformed data.

model, which is thought to be the only robust prediction of the IFD, holds for the first type of variability but not for the second (Parker and Sutherland 1986; Sutherland and Parker 1992; van der Meer 1997). These sources of variability can be distinguished by comparing the variability of intake rate in patches exploited by a single wasp to that in patches exploited by several wasps. If competitive ability is variable, then variation should increase with the number of competitors (Sutherland and Parker 1992). This was not the case for *L. testaceipes* (Figure 5), suggesting that the strong variability in rates of gain observed in this study resulted from variability in the ability of *L. testaceipes* females to exploit their hosts. Therefore, the equal gain rates observed presumably reflect IFD equilibrium.

### Information use and dispersal among patches

We tested the model of Bernstein et al. (1988, 1991) on *L. testaceipes* because some of the model's assumptions seemed to be consistent with two important aspects of the cognitive ecology of the parasitoid: 1) random movement among patches is consistent with the lack of odor-mediated orientation in *L. testaceipes* (Lo Pinto et al. 2004) and 2) optimal patch time allocation based on learning is consistent with the behavior described in the companion article (Tentelier et al. 2006). In the BKK model, the simultaneous equalization of intake rates and aggregation of competitors results from a combination of these two assumptions. We observed both phenomena in our data but not simultaneously. Individual intake rates equalized several hours after the immediate (<1 h) shift in population distribution from random to aggregated. Thus, the redistribution of parasitoids was not directly responsible for the equalization of gain rates across patches. A redistribution of hosts was also not responsible for this equalization as the relative abundance of healthy hosts on each patch remained constant due to density-independent host mortality.

It is unclear why the convergence of attack rates lagged behind the aggregation of parasitoids. The rapid host density–dependent aggregation of the parasitoid population does not at first sight seem compatible with the assumption of random walk and, instead, suggests that efficient orientation mechanisms were at work. Many parasitoid species, including aphid parasitoids, use the volatile compounds released by plants in response to herbivore damage to locate host patches, and the level of attraction may be positively related to host density (Du et al. 1998; Schmelz et al. 2003). One recent study found no evidence of odor-mediated orientation in *L. testaceipes* (Lo Pinto et al. 2004), whereas other studies have reported that *L. testaceipes* females are attracted to aphid-infested plants (Schuster and Starks 1974; Grasswitz and Paine 1993). The rapid aggregation of *L. testaceipes* in this study is consistent with effective attraction and suggests that the assumption of random search among plants was unrealistic. This is probably also true for many other organisms because natural selection generally results in animals being well equipped to find their resources. Interestingly, nonrandom movement was also identified as the most probable reason for the lack of fit between the data and model (the MVT) in the companion article (Tentelier et al. 2006). The BKK model could be improved by incorporating nonrandom orientation as well as learning. It would not be surprising that such implementations results in aggregation emerging quicker than equalization of gain rates.

### Exploitation and interference

Exploitation and interference competition are the two processes which constrain the functional response and, hence, link the equality of gain rates to the spatial distribution of competitors and the disappearance of resources (Sutherland 1983; Lessells 1995; van der Meer 1997). In this study, we obtained predictions from the BKK model (Bernstein et al. 1988, 1991) without specific assumptions concerning the effect of these two

processes on the exploitation of aphid colonies by *L. testaceipes*. However, a posteriori inferences from the data can be made.

One indirect approach to interference is to fit the data with Sutherland's (1983) model and estimate the coefficient of interference, *m*. The distribution of parasitoids and the mortality of aphid hosts were consistent with a coefficient close to one, which is considered to be high (Hassell and Varley 1969; but see also Arditi and Akçakaya 1990). However, this approach is not very informative because Sutherland's model is based on a functional response that may not match the foraging behavior of *L. testaceipes*. Closer examination of the data suggests that adult females do not display direct interference competition on the patches. If they did, the attack rates would have been affected by the number of interacting females. Consistently, focal observations of individual females exploiting the same aphid colony revealed no particular aggressive behavior. This contrasts with the results of one recent study showing direct interactions between some parasitoids in patches (Wajnberg et al. 2004). However, we found that parasitoid density had a significant negative effect on reproduction rate at the beginning of the experiment. This effect may be due to a lack of ability of *L. testaceipes* to discriminate between healthy and parasitized hosts (van Steenis 1994; Medrzycki et al. 2004). Poor discrimination may result in higher levels of superparasitism in patches exploited by several wasps simultaneously and, therefore, in lower per capita reproduction rates. This interpretation is supported by the observation that the initial negative effect of the number of parasitoids per patch on the per capita rate of reproduction was more severe on patches with few hosts than on patches with many hosts (significant host number × parasitoid number interaction). This finding highlights the importance of resources in the process of competition.

## CONCLUSION

Our results show that the BKK model of Bernstein et al. (1988, 1991) does not fit the observed change in behavior and distribution of *L. testaceipes*. The model predicted well the observed passage through an equilibrium characterized by reproduction rates depending on neither resource nor competition levels. However, the possible deviation from the IFD at the end of the experiment complicates the fit of the model to the data, and demonstrates the need for longer time series. Furthermore, the spatial redistribution of parasitoids occurred very rapidly and was therefore not directly responsible for the convergence toward equilibrium. In addition, *L. testaceipes* reproduction rates remained variable for reasons other than resource abundance and competition. Our data suggest that there was phenotypic variability in resource exploitation efficiency rather than in competitive ability. These findings suggest two possible ways of rendering IFD models based on patch assessment more realistic: 1) inclusion of nonrandom movements based on a priori information, such as volatile cues; this may better describe possible time lags between the spatial redistribution of the population and the equalization of gain rates, as observed in *L. testaceipes* and 2) use of differences between individuals. The second improvement has been made for simple continuous input systems with two patches (Regelmann 1984; Cézilly and Boy 1991; Koops and Abrahams 2003) but not for more complex environments in which foragers compete by interference and differ in their ability to exploit resources. This improvement is important because exploitation ability may reflect on the acquisition of information as well as the acquisition of resources, and the combination may lead to unexpected distributions (Koops and Abrahams 2003).

We very much thank Annie Géria, Emile Franco, and Arlette Marconi for technical assistance; Emmanuel Desouhant, Thomas Guillemaud, Thierry Spataro, and Cédric Tentelier for brain assistance; Jean-Michel Rabasse for financial assistance; and Hugh Moore for personal assistance. We are also grateful to Alex Kacelnik and anonymous referees for their contribution to the improvement of the manuscript.

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