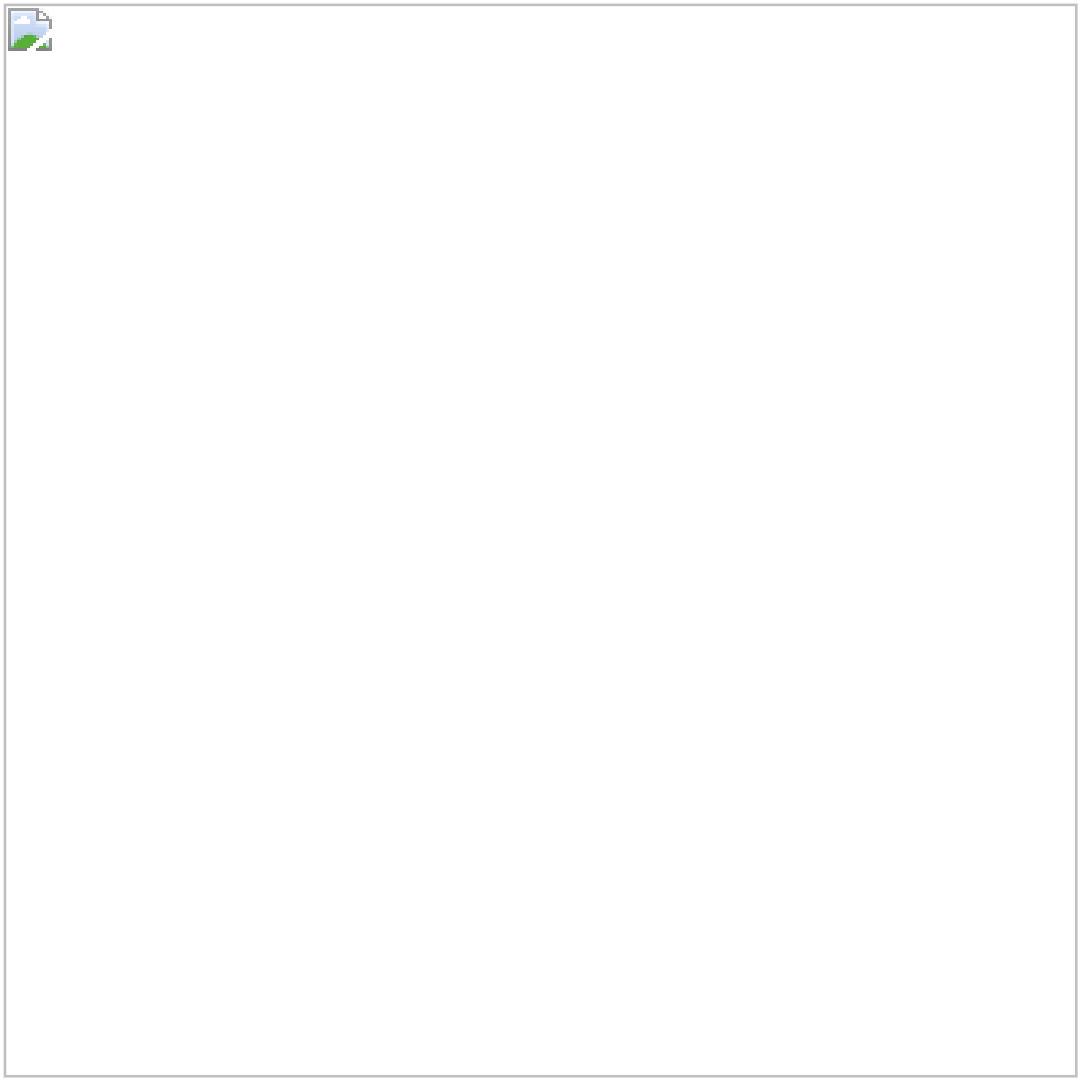

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Authors: Filin, Ido, Schmitz, Oswald J., and Ovadia, Ofer

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Consequences of individual size variation for survival of an insect herbivore: an analytical model and experimental field testing using the red-legged grasshopper

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IDO FILIN, OSWALD J. SCHMITZ AND OFER OVADIA

(IF, OO)Department of Life Sciences, Ben-Gurion University of the Negev, POB 653, Beer-Sheva 84105, Israel. Email: oferovad@bgu.ac.il
(OS)Yale University, School of Forestry and Environmental Studies and Department of Ecology and Evolutionary Biology, 370 Prospect Street, New Haven, CT 06511, USA.

Abstract

Variation in body size exists within any natural population. Moreover, variation in this fundamental physiologically based trait often translates into variation in demographic rates. Here we explore the effects of variation in the initial body size of individuals on the mean survival trajectory of a generalist herbivore living in a seasonal environment. We first present the results from an individual-based model, which provided expectations for the form of the relationship between mean survival and standard deviation in initial size. We then develop a heuristic analytical model that captures the essentials of the influence of initial-size variation on mean survival to end of season. Both theoretical formulations demonstrate that as initial body size variation increases, mean survival might initially increase; however, this initial positive effect is eventually reversed, causing mean survival to fall steeply as size variation becomes high. We then test these qualitative predictions in the field by manipulating the magnitude of initial size variation in experimental populations of the generalist grasshopper, *Melanoplus femurrubrum*. We show good qualitative congruence between model predictions and experimental results. Because herbivore survival is strongly linked to the strength of food-web interactions, we suggest that adopting such a combined theoretical and empirical approach can provide a profitable avenue toward a full understanding of the interplay between individual trait variation and higher-level dynamics.

Key words

body size, individual variation, seasonal environment, survival trajectory, univoltine lifecycle

Introduction

How different organizational levels interact to influence population dynamics remains a key research challenge in ecology (*e.g.*, Levin 1992). On one hand, ecological theory has provided substantial direction on choosing quantitative approaches to apply to such problems (*e.g.*, McCauley *et al.* 1993, Wilson *et al.* 1993, Durrett & Levin 1994, Abrams 1995, Bolker & Pacala 1997, Fahse *et al.* 1998, Grunbaum 1998, Pascual & Levin 1999). On the other hand, ecologists still lack the data needed to reliably choose the variables and organizational scales in natural systems that are causally linked to dynamics. The present study contributes toward choosing appropriate variables through a detailed examination of the effect of individual body-size variation on population-level demography and how that influences the trajectory of mean survival in the population.

Body size is one of the most important characteristics of any organism (*e.g.*, Peters 1983, Calder 1996). This fundamental trait can structure populations (*e.g.*, Caswell 2000) and has the potential to influence almost every aspect related to the function and performance of the individual organism, such as foraging (*e.g.*, Belovsky 1997), growth rate (*e.g.*, Pfister & Stevens 2002), survival (*e.g.*, Ovadia & Schmitz 2002) and trophic biology (*e.g.*, Maret & Collins 1994). Moreover, body size is especially important in seasonal environments. For example, individuals (hatchlings) initially smaller, may require a longer time to mature, and thus may have lower fitness than individuals who mature more rapidly because of an initial size advantage (*e.g.*, Rowe & Ludwig 1991, Abrams & Rowe 1996). Such differences in initial size may also influence the way different individuals trade-off avoiding predators and growth rate/foraging gains (*e.g.*, Abrams & Rowe 1996, Clark & Mangel 2000). This size-dependent trade-off behavior could result in differential direct effects of predators on the abundance of a given-sized prey.

Variation in body size exists within any natural population (*e.g.*, Uchmanski 1985), and this size variation often translates into variation in demographic rates (*e.g.*, Ebenman & Persson 1988). For instance, Wall & Begon (1987) have illustrated that a substantial variation in size-related life-history traits (*e.g.*, weight at maturity and time to maturity) exists within grasshopper populations. Such size variation might have important consequences for grasshopper population dynamics (Joern & Gaines 1990). Likewise, field experiments by Ovadia & Schmitz (2002) demonstrated that the survival trajectory of grasshoppers changes as a function of their initial body size. Subsequent theoretical work at the community level showed that herbivore size variation markedly influences the strengths of trophic interactions as mediated through mean herbivore survival (Ovadia *et al.* 2007). Therefore, both theoretical work and empirical studies emphasize that size variability is a fundamental property of natural populations, which has important ramifications for understanding the dynamics of ecological systems.

In this study, we explore the effects of variation in the initial body size of individuals on the mean survival trajectory of a generalist herbivore living in a seasonal environment. We first present the results of an individual-based simulation, which provided expectations for the form of the relationship between mean survival and standard deviation in initial size. Motivated by these outcomes, we then develop a heuristic analytical model that captures the essentials of the influence of initial-size variation on mean survival to the end of the season. Based on both simulation and analytical results, our main theoretical conclusion is that as initial body size variation increases, mean survival might initially increase as well; however, this initial positive effect is eventually reversed, causing mean survival to fall steeply as size variation becomes high. Moreover, the initial positive effect is expected to be small in magnitude, and in some cases it manifests itself by mean survival being initially unaffected by size variation.

We then test these predictions in the field by manipulating the magnitude of initial size variation in experimental populations of

the generalist grasshopper, *Melanoplus femurrubrum*. Our qualitative predictions are supported by the results from the field experiment, indicating that mean survival is initially insensitive to size variation, but as size variation further increases, it exerts a strong negative effect on the survival trajectory of *M. femurrubrum*.

Theoretical Framework

We consider the case of univoltine, seasonal life cycles, typical of many insects that live in seasonal environments, *e.g.*, temperate (Howard & Harrison 1984, Monk 1985) and arid (Antoniou 1978, Whitman 1988). In particular, we refer to a typical grasshopper life cycle (*e.g.*, Sibly & Monk 1987) in which eggs overwinter in a state of embryonic diapause, first instars emerge in early summer, and growth, maturation, and reproduction occur within a relatively short time window, often terminated by the seasonal onset of frosts. Such is a situation of nonoverlapping generations, *i.e.*, the population within each season is comprised of a single cohort.

We define initial size of individuals as their size at some standard early developmental stage (*e.g.*, size at hatching in the simulations and second instars in the field experiment). We denote body size here as body length, but emphasize that the analytical model described below is independent of the specific choice of a size measure (*i.e.*, it can also be used with body mass, or any other measure of body size). Variation in initial size, therefore, translates as variation (*e.g.*, variance, standard deviation, or coefficient of variation) in the body length of young individuals, all compared at the same developmental stage.

Expectations based on individual-based simulations.—Ovadia *et al.* (2007) used an individual-based model to investigate the effect of size variation among individual herbivores on population demography and on the strength of trophic interactions in a food web. The simulated food web consisted of two groups of plant resources: a herbivore that selects between the two resource groups providing different levels of nutrition and protection from predators, and a predator that preys on the herbivore. Using this bottom-up computational approach, Ovadia *et al.* (2007) could identify the form of the functions relating body size to demographic rate through simulations, which were driven, not by functions, but by assigning rule sets to individuals in the simulator at a fundamental level, *i.e.*, physiology and behavior.

The simulation experiments were conducted to quantify the relationship between initial body size of individual herbivores and their survival, using the following two scenarios: 1) No initial frequency dependence — populations of identical herbivores generated by systematically increasing individual initial size. Mean survival to the end of the season, for each of these populations, was obtained through simulations. A curve relating survival to initial size was constructed by linear interpolation (Fig. 1a). 2) Maximum initial frequency dependence — herbivore populations generated in which all initial sizes were equally represented (*i.e.*, uniform distribution). Individuals within each of these mixed populations were specifically tracked through the simulations to quantify the relationship between initial size and survival. In the same manner as above, a curve relating initial body size and survival was constructed (Fig. 1a). The difference between the two curves of survival *vs* initial size indicates to what extent frequency dependence (*i.e.*, effects of trait distribution on interaction strengths) influences demographic effects of body size.

The two curves of survival *vs* initial size (Fig. 1a) were used to calculate an envelope (*i.e.*, lower and upper bounds) for the relationship between mean survival and variation in initial size, which bound all other curves for intermediate levels of frequency dependence (Fig. 1b). The two curves are shown in Fig. 1b, which presents mean survival against the standard deviation of the initial body-size distribution. The width of the envelope measures the effect of frequency dependence on mean survival (*i.e.*, the wider the envelope the more important is frequency dependence). The envelope presented in Fig. 1b illustrates that frequency dependence can exert its strongest influence at low levels of size variation (*i.e.*, when standard deviation in initial body size is low). But as variation increases, the two curves converge (Fig. 1b), suggesting that frequency-dependent effects of size variation on mean herbivore survival may be evident only when individual size variation is relatively low.

Finally, simulation experiments were conducted to generate dynamics under different scenarios of initial size variation. Five different populations of herbivores were generated by systematically increasing initial size variation, while keeping mean initial size fixed. The results of these simulations (marked as "Observed" in Fig. 1b) indicate that when initial size variation is low, it tends to have a small positive effect on mean survival. But as variation further increases, the effect becomes strongly negative. Moreover, the results follow the 'no initial frequency-dependence curve' quite closely (Fig. 1b), suggesting that frequency-dependent effects on mean survival are weak. This is especially evident when we consider the range of low size variation, where frequency-dependent effects, if present, should exert their strongest influence (based on the maximal initial frequency-dependence curve of Fig. 1b).

A heuristic model using cumulative hazards.—Motivated by the above results from an individual-based simulation model, we now devise a relatively simple analytical model to capture the essentials of how variation in initial size affects mean survival within a cohort.

In general, survival from initial time $t = 0$ to some subsequent time $t = T$ is related to instantaneous mortality, $\mu(t)$, through the expression

$$S = \exp\left(-\int_0^T \mu(t)dt \right) \quad (1)$$

Mortality rate, $\mu(t)$, is sometimes referred to as hazard rate (*e.g.*, Ledder *et al.* 2004). Similarly, the expression within the parentheses in Equation 1 (*i.e.*, the time integral of instantaneous mortality) is sometimes referred to as the cumulative hazard (*e.g.*, Carey 2003, p.90), which we will denote by H. Hence, survival is given by $S = \exp(-H)$, where

$$H = \int_0^T \mu(t)dt \quad (2)$$

is the cumulative hazard.

Variation in mortality rates among individuals, will result in variation in cumulative hazards, and consequently, in survival. In the following, we incorporate variation in both mortality and individual size, in a similar manner to Bjørnstad & Hansen (1994), *i.e.*, by using a Taylor expansion. However, we focus here on the effect of variation on within-season survival, rather than on among-season population dynamics and stability (see also Filin & Ovadia 2007).

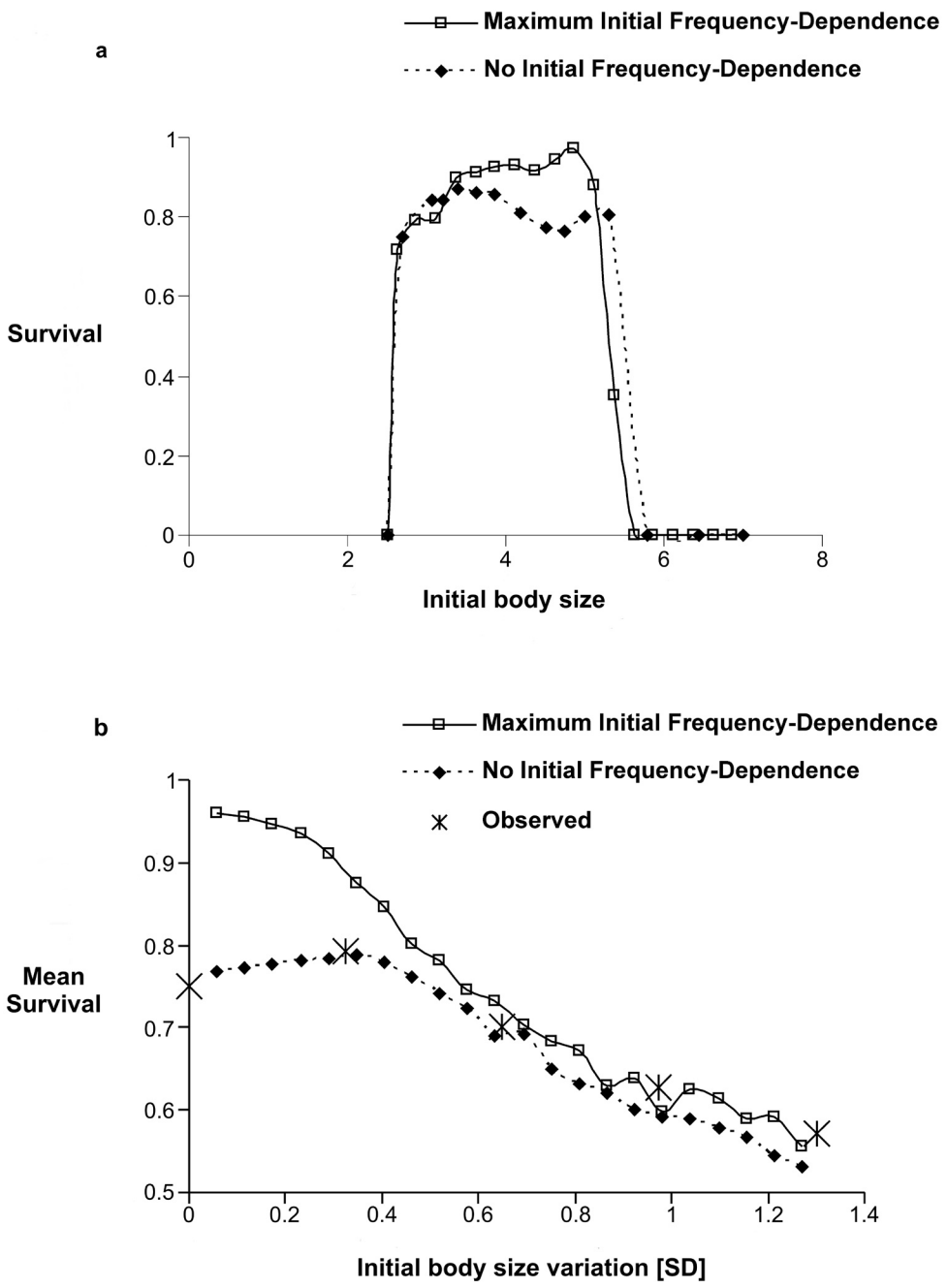

Fig. 1. Predictions obtained using an individual based model (Ovadia *et al.* 2007), for the effect of **a)** initial body size and **b)** body size variation on herbivore survival. (Units of initial body size are arbitrary.) **a.** The relationships between initial body size and survival to end of season were quantified using the following two scenarios: 1) No initial frequency dependence – homogenous populations of identically sized herbivores were generated by systematically increasing individual initial size. Survival to the end of the season, for each of these populations, was obtained through simulations; 2) Maximum initial frequency dependence – herbivore populations in which all initial sizes are equally represented (*i.e.*, uniform distribution) were generated. Individuals within each of these mixed populations were specifically tracked through the simulations to quantify the relationship between initial size and survival to the end of the season. Both curves relating survival to initial size were constructed by linear interpolation. **b.** An envelope (*i.e.*, lower and upper bounds) for the relationship between *variation* in initial size and *mean* survival generated using the above two curves of survival *vs* initial size. The width of the envelope measures the effect of frequency dependence on mean survival, *i.e.*, the wider the envelope the more important is frequency dependence. Observed simulation results (marked as "Observed") under different levels of initial size variation, overlaid on this envelope, indicate that when initial size variation is low, it tends to have a small positive effect on mean survival. But as variation further increases, the effect becomes strongly negative. Moreover, the results follow the 'no initial frequency-dependence curve' quite closely, suggesting that frequency-dependent effects on mean survival are weak.

Let $\overline{H}$ denote the mean cumulative hazard, and σ_H^2 the variance in cumulative hazards, within the population. Given that, mean survival can be approximated by

$$\overline{S} \approx e^{-\overline{H}}\left(1+\frac{1}{2}\sigma_H^2\right) \quad (3)$$

Equation 3 demonstrates that mean survival increases as the variance in cumulative hazards (σ_H^2) increases, while holding the mean cumulative hazard ($\overline{H}$) constant. That is because survival, given by $S = e^{-H}$, is a concave-up function of H. This is the essence of Jensen's inequality, which states that the mean of a nonlinear function is, in general, not equal to the value of this function evaluated at the mean. (For additional explanations of Jensen's inequality, see Bjørnstad & Hansen 1994, Kendall & Fox 2001, Lindström & Kokko 2002, Ovadia *et al.* 2007.)

Because mortality rates are expected to depend on size (denoted by z; *e.g.*, body length), the cumulative hazard H (Eq. 2) should depend on the range of sizes that an individual experiences, as it develops between time $t = 0$ and time $t = T$. Specifically, H should depend on the initial size of an individual, denoted by z_0. Because a smaller initial size means that it takes longer to reach adult size, and may also mean initially higher mortality rates, we expect the cumulative hazard to increase as the initial size decreases. This translates into the cumulative hazard being a decreasing function of initial size, *i.e.*, $H'(z_0) < 0$ (where H' is the derivative of the cumulative hazard with respect to z_0). Moreover, $H(z_0)$ may decrease with z_0 in either an accelerating or a decelerating manner, *i.e.*, the second derivative, denoted by $H''(z_0)$, may be either negative or positive, respectively. As we now proceed to demonstrate, these seemingly technical details regarding the relationship between cumulative hazard and initial size, have important consequences for the effect of size variation on mean survival.

Let $\overline{z}_0$ represent the mean initial size of individuals, and V, the variance in initial sizes, within the population. As demonstrated above (Eq. 3), increased variance in mortality rates (σ_H^2) translates into increased mean survival (given that mean cumulative hazard $\overline{H}$ is fixed). However, increasing the variance in initial sizes (V), while holding the mean initial size $\overline{z}_0$ fixed, may affect both the mean and variance in cumulative hazard, *i.e.*, both $\overline{H}$ and σ_H^2 in Eq. (3). Consequently, we cannot determine in an *a priori* manner, whether size variation increases or decreases mean survival. The sign of this effect of size variation on mean survival should depend on the specific relationship between cumulative hazard and initial size, *i.e.*, on the form of $H(z_0)$, as discussed in the previous paragraph.

To a first approximation (in terms of V), we obtain the following expression for mean survival (based on Eq. 3)

$$\overline{S} = S(\overline{z}_0)\cdot\exp\left(-\frac{1}{2}H''\cdot V\right)\cdot\left(1+H'^2\cdot V\right) + \ h.o.t \quad (4)$$

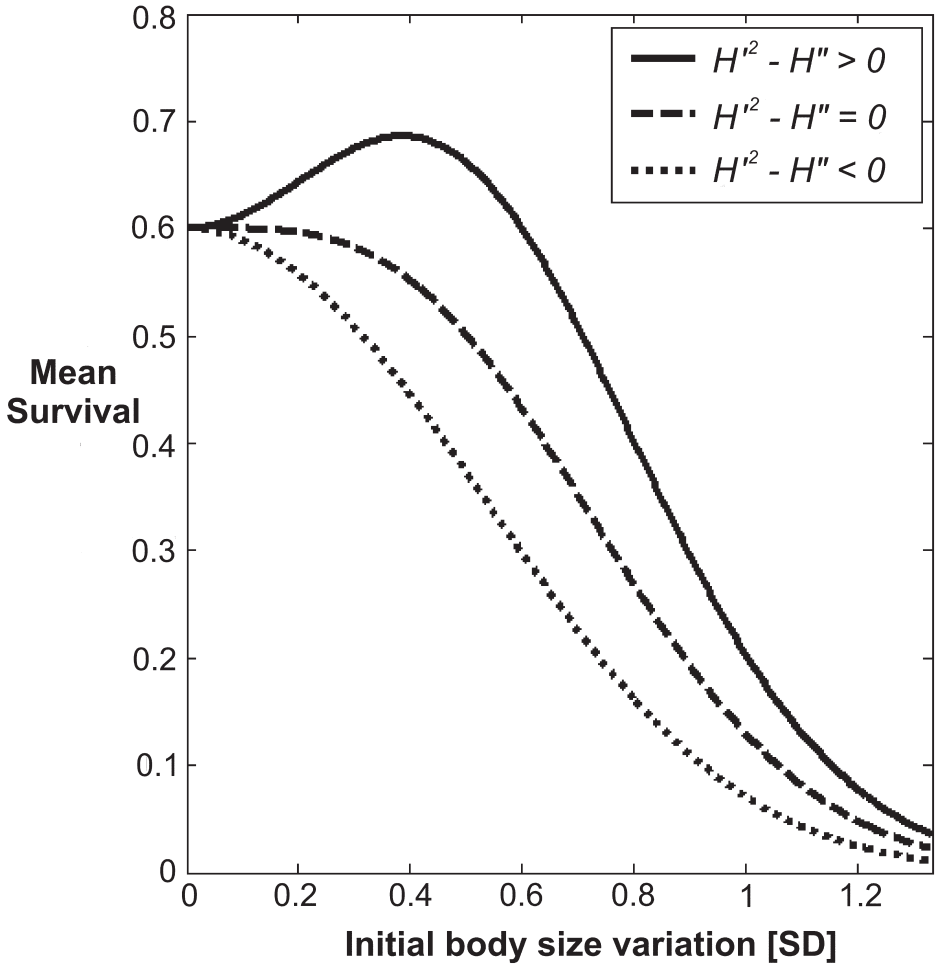

Fig. 2. Curves representing the relationship between mean survival and initial-size variation (measured as standard deviation of initial sizes), calculated using equation (3). Three cases have been distinguished according to the relative magnitudes of the size-variation effects mediated through both mean cumulative hazard (dependent on H'') and variance of cumulative hazards (dependent on H'^2). All curves conform to the case of positive second derivative ($H'' > 0$), which is the biologically more reasonable case (see text). Note that when $H'^2 - H'' > 0$, mean survival initially increases with size variation. When $H'^2 - H'' = 0$, the first-order effect of size variation on mean survival vanishes, and therefore, initially mean survival is not affected by increasing size variation (this is evident in the respective curve for $H'^2 - H'' = 0$. Parameter values used for the figure are: $S(\overline{z}_0) = 0.6$; $H'' = 5.76$; and H' received the values (-3.2), (-2.4), and (-1.5), representing the three curves. Units of initial body size are arbitrary.

where the derivatives H' and H'' are evaluated at the mean initial size $\bar{z}_0$, the term *h.o.t.* encompasses higher-order components (*i.e.*, terms involving higher powers of V), and $S(\bar{z}_0)$ is the survival of an individual with initial size equal to the mean of the population (*i.e.*, in terms of Jensen's inequality, it is the function of the mean, while $\bar{S}$ is the mean of the function). The term involving H'' in equation (4) represents the effect of size variation, V, on the mean cumulative hazard, $\bar{H}$, while the term involving H'^2 represents the effect on the variance of cumulative hazards, σ_H^2 (compare with Eq. 3).

We note that mean survival can both increase and decrease with size variation, depending on the values of H'^2 and H''. In fact, it is rather straightforward to demonstrate that, at least initially, when V is small enough so higher-order terms can be neglected (*h.o.t* in Eq. 4), mean survival either increases or decreases with size variation depending on the sign of $H'^2 - H''$. Three such examples are shown in Fig. 2, which presents typical curves of mean survival *vs* standard deviation in initial size (*i.e.*, $\sqrt{V}$), calculated using equation (4). In these examples we used a positive second derivative (*i.e.*, $H'' > 0$), which is the biologically more reasonable situation, because it means that the sensitivity of cumulative hazard to changes in initial size is higher for smaller individuals (this sensitivity is determined by the absolute value of H'). A positive second derivative is also the more interesting case, because it means that the effect of size variation on mean survival, mediated by $\bar{H}$, is negative, while that mediated by σ_H^2 is positive (see Eq. 4). Thus, mean survival is either reduced or raised, depending on which of the two effects has the upper hand (as presented in Fig. 2).

Whether initially increasing or decreasing with size variation, mean survival must ultimately decrease as size variation (V) further increases. There are mathematical reasons for that (involving, in Eq. 4, the negative exponent, given positive H'', as well as higher order terms; see also Fig. 2). But there is also a biological reason. As variation in initial size increases, there is an increasing fraction of chronically very small individuals that suffer zero (or negligible) survival. This clearly affects mean survival in a negative way. Such maladaptive, very high levels of initial size variation are presumably rarely attained in nature, though perhaps they can be achieved using experimental manipulations.

Therefore, this model predicts that the overall relationship between individual size variation and mean survival is nonlinear, and also not necessarily unidirectional (*i.e.*, monotonic). As Fig. 2 demonstrates, mean survival can initially be raised by increasing size variation, but is subsequently reduced by high levels of such variation in size. Interestingly, this relatively simple analytical model generates similar patterns to those of the much more complex individual-based model (previous section; Ovadia *et al.* 2007). Specifically, the slight initial increase of mean survival, which was evident in the results of the individual-based model (Fig. 1b), is also apparent in the predictions of the analytical model (Fig. 2). Nevertheless, the general prediction that arises from our model is that as size variation increases, mean survival in the population should eventually decrease.

This model is a "heuristic" tool because its main ingredient, the cumulative hazard, is ultimately only a summary variable that encompasses many biologically more meaningful details (*e.g.*, changes in mortality and growth rates along the developmental trajectory and /or during adult life as the season progresses), which we do not explicitly consider. These details enter only implicitly through the relationship between cumulative hazard and initial size. Additionally, higher order terms that become important when size variation is high, are also not considered explicitly (see Eq. 4). Nonetheless, the strength of the model is in its relative simplicity, in its ability to capture the essential effects of variation in initial size on mean survival, and in the predictions it can generate for the form of this relationship (*e.g.*, Fig. 2).

We now describe an experimental test of the prediction that mean survival should vary inversely with size variation.

Empirical field testing

Natural history.—This empirical research was completed in a meadow at the Yale-Myers Research Forest in northeastern Connecticut (Schmitz & Suttle 2001). The herbs *Solidago rugosa*, *Daucus carota*, *Aster novaeangliae*, and *Trifolium pratense*, and the grass *Poa pratensis* dominated this meadow. Using an enclosure experiment, we tested if the survival of the grasshopper herbivore *Melanoplus femurrubrum*, feeding on those plants, changes as a function of its initial body-size variation, in the absence and in the presence, of an important predator of the grasshopper — the sit-and-wait hunting spider *Pisaurina mira* (Schmitz & Suttle 2001).

Previous research has consistently shown that individual *M. femurrubrum* of the same developmental stage (2nd instar nymphs) vary in their body size (Ovadia & Schmitz 2002, Ovadia & Schmitz 2004a). Using this natural body-size distribution, Ovadia & Schmitz (2002) first designated three size classes: large individuals, the upper most 5% of the frequency distribution (*i.e.*, body length > 12 mm); small individuals, the lowest 5% of the frequency distribution (*i.e.*, body length < 9mm); and average individuals, the middle portion of the frequency distribution (*i.e.*, body length 9-12 mm). They then conducted a randomized blocks field experiment, in which grasshoppers of these three different body-size classes were stocked into cages with and without their spider predators. This experiment showed that, across all treatments, the survival of individual *M. femurrubrum* of the small size class was almost half that of the large size class (Ovadia & Schmitz 2002, Ovadia & Schmitz 2004a).

Study design.—Classical approaches view variation in body size as an emerging consequence of individuals facing different local levels of predation risk and forage availability in a community context (*e.g.*, Uchmanski 1985, Kimmel 1986). However, the recognition that the magnitude of variation in size distribution can have a crucial bearing on dynamics, means that field experiments must be designed to better understand the effect of initial conditions (initial size distribution) on population dynamics (Schmitz 2001). Such experiments involve creating populations of the same species (*e.g.*, grasshopper herbivore) that have different initial body-size distributions and then testing whether the magnitude of size variation is indeed causally linked to population dynamics.

We conducted an enclosure experiment in the field to test for the effect of initial body-size variation among individual *M. femurrubrum*, on their survival trajectory within a growing season in the absence and in the presence of their spider predators. The experiment was conducted in standard aluminum screen enclosure-cages measuring 0.25 m^2 (basal area) × 1 m (height). The protocol for cage construction and placement in the field has been presented elsewhere (Schmitz 2004). The cages were arrayed in a randomized-blocks design, separated by 1.5 m, and placed over natural vegetation in the field. This method of cage placement does not introduce bias in initial grass and herb composition in the cages (Schmitz 2004). Insects and spiders were removed from the cages by carefully hand-sorting through the vegetation and litter in each cage.

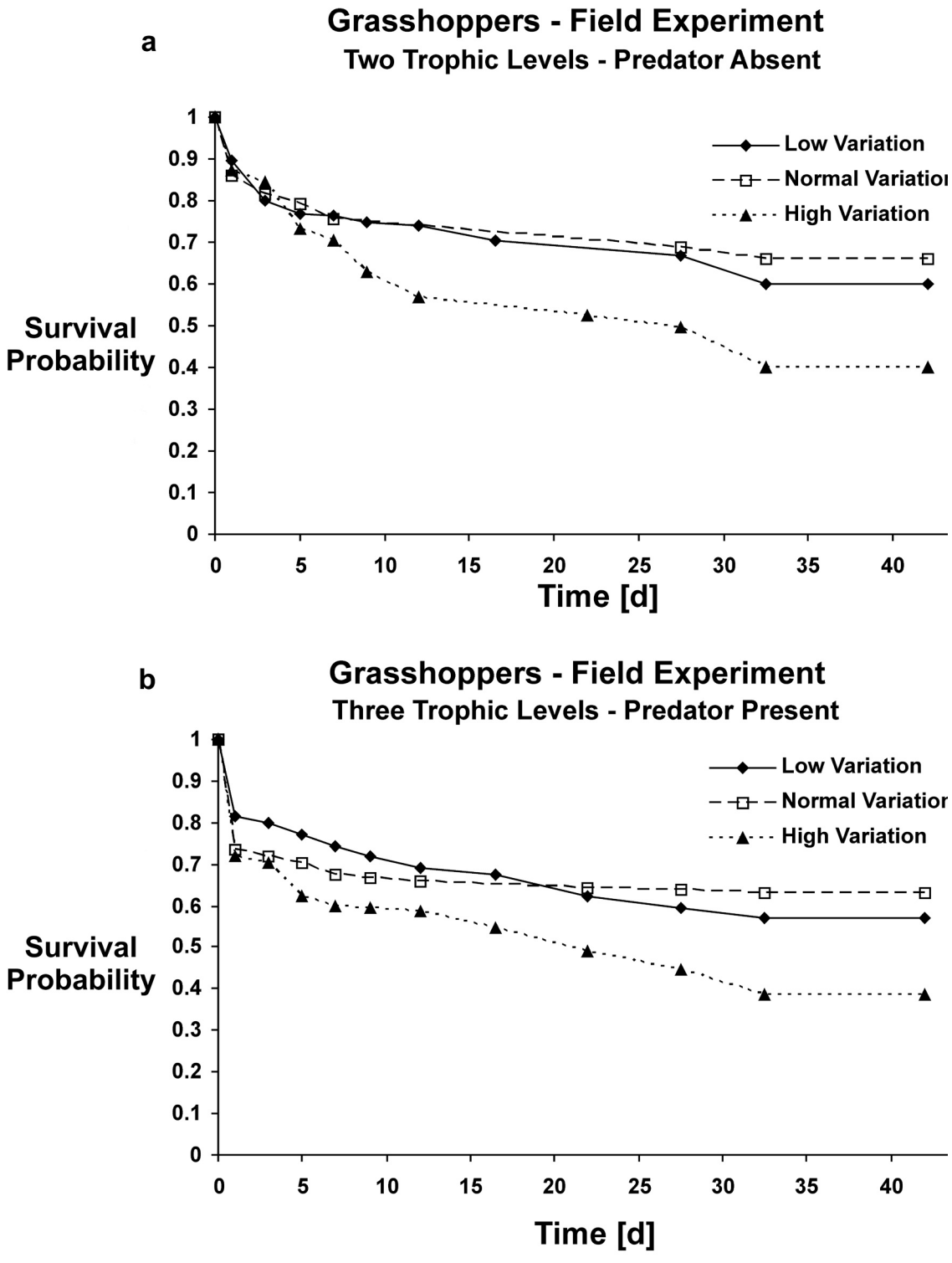

Fig. 3. The effect of initial variation in body size on the survival trajectory of grasshoppers in the a) absence and b) presence of spider predators, indicating that survival probability of low and normal size variation was significantly higher than that of high variation. Survival curves were estimated by using the Kaplan-Meier method (Kalbfleisch & Prentice 1980).

In mid-July 2003, we sampled 2nd instar *M. femurrubrum* nymphs using sweep nets. We first measured their body length (head to end of abdomen) and assigned them to one of the three size classes as defined by Ovadia & Schmitz (2002) (see above). We then used these size classes to assemble three different kinds of experimental populations by systematically increasing the variation in initial body size, while keeping mean initial size approximately the same. Each of these three populations consisted of nine individuals. The first experimental population consisted only of individuals pertaining to the average size class (*i.e.*, Low Variation Treatment). The second population consisted of five individuals of the average size class, and two individuals of each of the two other size classes (Normal Variation Treatment: similar to natural distributions in the field). The third population consisted of three individuals of each of the three size classes (High Variation Treatment).

In order to examine population and community-level consequences of initial trait variation, each population was randomly assigned to one of two treatments: with and without *P. mira* predators. Spiders stocked were large enough (16-20 mm) to capture and subdue all sizes of grasshopper prey (juveniles: 7-18 mm; adults 19-24 mm; Schmitz & Suttle 2001). The complete factorial design experiment included two treatments that varied the number of trophic levels in the community (2-level [plants and grasshoppers] and 3-level [plants, grasshoppers and spiders]) crossed with three different initial size-distribution grasshopper populations. Each treatment combination was replicated 15 times.

Enclosure densities of grasshoppers and spiders were censused over the course of the experiment. After initial stocking, the first three censuses were performed at two-day intervals to ensure that grasshopper populations did not go extinct due to artifacts of initial conditions. Thereafter, enclosures were monitored every five days until the termination of the experiment in early September. The experiment ran for the entire life-history development of the instar nymphs and terminated just before the seasonal onset of frosts that kill the active arthropod community and cause the herbaceous plant community to senesce.

Data analysis.—We tested for the effects of initial body-size variation on the survival of grasshoppers with and without their spider predators, using a Cox proportional hazard model (Hosmer & Lemeshow 1999) with initial body-size variation (Low Variation = 0, Normal Variation = 1, and High Variation =2), predation treatment (Predator Absent = 0, and Predator Present = 1), and the respective interaction term as covariates. This is a commonly used survival analysis method, which allows evaluating effects of different predictors (*i.e.*, covariates) on mortality rate, independent of the time-varying background mortality rate (Hosmer & Lemeshow 1999). To control for repeated measurements on a subject, which in our case, were individual cages that were repeatedly censused throughout the season, we used a robust jackknife variance estimator, grouped by observations per cage (Lin & Wei 1989).

Results

Survival analysis revealed that the average mortality rate of grasshoppers decreased significantly as initial size variation increased (Cox proportional hazard model, $z = 4.41$, $p < 0.001$; Fig. 3). Specifically, the relative hazard for grasshoppers in the high-variation treatment was about 1.8 times higher than that of grasshoppers in the normal and low-variation treatments. Consequently, their survival to the end of the season was reduced by 36%, compared to that of grasshoppers in the other two size-variation treatments. We could not detect significant differences in grasshopper survival between the predator and no-predator treatments (Cox proportional hazard model, $z = 1.55$, $p = 0.120$; Fig. 3). Additionally, the interaction term (Size variation × Predator treatment) was not significant (Cox proportional hazard model, $z = -0.03$, $p = 0.970$), indicating that this lack of numerical effect of spiders on grasshoppers was consistent among the three initial size-variation treatments (Fig. 3).

Discussion

We present here the results of an individual-based simulation and an analytical model whose aim is to predict the effect of variation in the initial body size of individuals on mean survival in a population. We find that mean survival might initially be raised by increasing size variation, but is subsequently reduced by high levels of such variation (Figs 1b, 2). We then test these qualitative predictions using data from field experiments, in which we manipulated the magnitude of initial size variation within experimental populations of the generalist grasshopper, *M. femurrubrum*. We demonstrate good qualitative congruence between model predictions and experimental results. As predicted by simulation and analytical model (Figs 1b, 2), mean survival in experimental populations is initially insensitive to increasing size variation (exhibiting a small initial increase that is consistent between predator treatments, but is not statistically significant; Fig. 3), but then it drops significantly as size variation becomes high (Fig.3). Therefore, high initial body-size variation has a strong negative effect on the survival trajectory of *M. femurrubrum*, irrespective of predator presence.

Assessing the generality of our theoretical and empirical results requires an understanding of their mechanistic basis. Many empirical studies have identified a U-shaped pattern of mortality *vs* size, where mortality first decreases with the size of individuals, but then begins to increase for larger individuals (*e.g.*, mammals: Caughley *et al.* 1988; insects: van Straalen 1985). In many other cases however, mortality often decreases monotonically with size, with the highest mortality occurring among small individuals (*i.e.*, type III survivorship curve, Deevey 1947). This type III curve is common in many aquatic (Keller & Ribi 1993) and terrestrial species (Boulton & Polis 1999), and was also evident in our study species *M. femurrubrum* (Ovadia & Schmitz 2002). Both mortality patterns can be accommodated by relatively simple models of demography in size-structured populations (*e.g.*, Kirkpatrick 1984). In addition, in both cases, mortality initially decreases with size. Thus, when considering variation in the initial size of individuals, we can safely assume a decrease of mortality with initial size.

Fundamental theoretical work on size-structured populations has found that survival depends on size via the ratio of size-specific mortality to size-specific growth (*i.e.*, $\mu(z)/g(z)$ where $g(z) = dz/dt$; Werner & Gilliam 1984). Consequently, given the knowledge of how mortality changes with size (as discussed in the previous paragraph), and given the growth trajectory of individuals in the population, it may be possible to obtain an explicit expression of survival (or cumulative hazard), as a function of initial size. By doing so, the heuristic analytical model becomes a full mechanistic model of survival, rooted in the processes of size-specific mortality and growth, which allows generating quantitative predictions, regarding the sign and magnitude of the effect of size variation on mean survival.

In this study we considered the effect of size variation on survival within a season. However, variation in the initial size of individuals may also have important consequences for population

dynamics and stability among years. For example, Schmitz (2001) has argued that the magnitude of variation in the state of individuals (*e.g.*, body size) has a crucial bearing on population dynamics and on the strengths of trophic interactions. He demonstrated his point using an individual-based modeling approach, in which he incrementally increased initial size variation, while keeping the mean trait value fixed (as we did in this paper). Population-density trajectories, based on the mean trait value, served as good approximation of actual dynamics (which includes variation in initial size of individuals) only when the magnitude of variation was relatively small. Uchmanski (1999, 2000) and Grimm & Uchmanski (2002) developed individual-based models of consumer-resource dynamics, to investigate the effect of variability in body size on population stability and persistence. They showed that initial body-size variation can enhance population stability and persistence, i.e., cause longer extinction times in comparison to homogenous populations.

A recent analytical model by Filin & Ovadia (2007) explored the effect of individual size variation on population dynamics and stability in a seasonal environment. The model shows that the effect of size variation on the population net reproductive rate varies in both magnitude and sign, depending on season length. Additionally, after calibrating the model with field data from *M. femurrubrum*, they show that under deterministic dynamics (fixed season length), size variation impairs population stability, given naturally occurring densities. However, in the stochastic case, where season length exhibits yearly fluctuations, size variation reduces the among-year variance in population growth rate, thus enhancing stability.

The model that we developed, although formulated to predict herbivore survival at the population level, may have some important implications for community-level patterns. For example, Ovadia & Schmitz (2004b) have shown that the survival trajectory of herbivores strongly influences the strengths of trophic cascades in an old field system. Specifically, because the presence of predators causes grasshoppers to forego feeding on the nutritionally superior grasses and to seek refuge in leafy herbs, then predator presence coupled with high within-season survival of grasshoppers results in high levels of damage to the herbs. In contrast, when survival was low, there were no detectable effects of the number of trophic levels on either grass or herb abundance (Ovadia & Schmitz 2004b). Using an individual-based model, Ovadia *et al.* (2007) demonstrated that increased size variation decreases mean herbivore survival. Moreover, because the strength of plant-herbivore interactions strongly depends on herbivore survival, they could accurately predict the plant biomass at the end of each growing season, based on the distribution of herbivore body size early in the season. In the study reported here we analytically described how body size variation translates into mean survival of the herbivore, and therefore, how it can be used to predict the impact of the herbivore on its plant resources, given initial density and body-size distribution.

In conclusion, our theoretical formulation and field experiment demonstrate that individual size variation is a key factor influencing the mean survival of a generalist herbivore in a seasonal environment. We suggest that the effect of such trait variation can also influence the strength of food-web interaction because of the high correlation between herbivore survival and the impact it inflicts on its plant resources (Ovadia & Schmitz 2004b, Ovadia *et al.* 2007). Therefore, trait distribution may be a major determinant of population and community dynamics. A complete description of these effects requires an understanding of the mechanistic processes that translate individual variation into population-level parameters, i.e., processes such as size-specific mortality and growth. It will also require an understating of the mechanisms that produce and maintain individual variation in physiological traits such as body size (*e.g.*, Uchmanski 1985, Kimmel 1986, Pfister & Stevens 2002, Peacor & Pfister 2006, Whitman & Ananthakrishnan 2008). We suggest that such a combined approach of simulation models, analytical models and field experiments provides a profitable avenue toward a full understanding of the interplay between individual trait variation and higher-level dynamics.

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