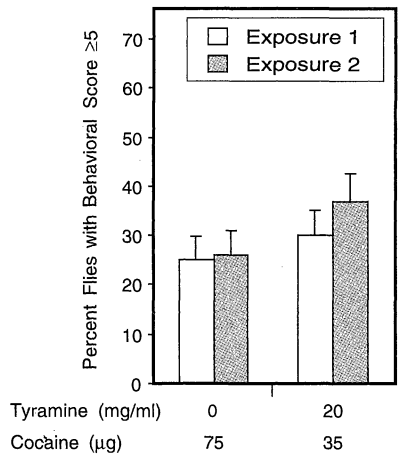

Fig. 4. Tyramine feeding increases initial cocaine responsiveness of *per*^{o} flies but does not restore sensitization. *per*^{o} flies were fed on instant food (Carolina Biologicals, Burlington, NC) with or without tyramine (20 mg/ml) for 2 days (0 mg/ml tyramine, n = 81, 73; 20 mg/ml tyramine, n = 73, 57). Flies were exposed to the indicated amounts of volatilized cocaine and assayed as in Fig. 3.

product is required for this regulated release.

Similar to *inactive* (*25*), tyramine increases initial cocaine responsiveness in *per*^{o} flies. Exposure of tyramine-fed *per*^{o} flies to 35 μg of cocaine induced behaviors normally seen in control flies exposed to 75 μg (Fig. 4). Thus, although long-term increase of tyramine levels can affect initial cocaine responsiveness, it is not sufficient for sensitization in flies lacking normal *per* function.

A unifying feature of most genes that regulate circadian rhythmicity in *Drosophila* and vertebrates is the PAS dimerization domain, common to a subset of basic helix-loop-helix transcription factors (*26*, *27*). Within the circadian cycle, CLOCK/CYCLE heterodimers activate *per* transcription, whereas PER/TIM heterodimers inhibit the activity of CLOCK/CYCLE (*28*–*30*). We find that mutations in *per*, *clock*, and *cycle* share the same cocaine phenotype: a deficiency in the ability to sensitize after one or more drug exposures. This similarity leads us to suspect that as in circadian behaviors, these genes are functioning in a common pathway.

In contrast to the above mentioned genes, the *tim*^{o} mutant showed normal cocaine responses. The implication of this finding is twofold. First, there must be an as yet unidentified PER binding partner that is specifically involved in regulation of drug responsiveness. Second, drug responsiveness is likely regulated by *per* expression in a set of cells distinct from those involved in circadian function. In *tim*^{o} mutants, PER levels are constitutively low (*19*, *20*); if the same TIM-containing cells were involved in circadian and cocaine responses, *tim*^{o} flies should not sensitize.

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13. A failure to sensitize, as is seen with the circadian mutants, is a rare phenotype among the mutants that have been screened in this laboratory, having been seen previously only for the mutant *inactive* (*25*). A number of behavioral mutants have been screened, including *dunce*, *rutabaga*, *shibire*, and several alleles of *amnesiac*, and mutants in cell signaling pathways including *Gprk2*, *trp*, and *Nf1*. None of these lines shows significant aberrations in cocaine responsiveness or sensitization (C. McClung, J. Walman, J. Hirsh, unpublished data). A number of WT strains, including the background strains from which the circadian mutants were isolated, similarly show only minor variations in initial responsiveness to cocaine, with no variation in the ability to sensitize. In addition, forward-based screens show a phenotype of failure to sensitize at extremely low frequency.
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31. Flies were exposed to 75 μg of volatilized cocaine three times over the course of 2 days and decapitated 4 hours after the last exposure as described (*24*). A 2 mM solution of the dopamine D2-like agonist quinpirole was applied to the cut end of the nerve cord, made up in 3% green food coloring to assess diffusion into the nerve cord. Locomotion after drug application was video-recorded for 2 min.
32. Hand-dissected *Drosophila* adult brains (four brains in 12 μl) were homogenized in 50 mM tris (pH 7.5), 1 mM phenylthiourea. Forty-eight microliters of assay mix [0.1 M sodium phosphate buffer (pH 6.8), 0.1 mM pyridoxal phosphate, 0.1 mM EDTA, and [^{3}H]tyrosine (20 μCi/ml)] was added to 12 μl of brain homogenate and incubated for 10 min at 31°C. Conversion to tyramine was linear over the 10-min incubation time. Reactions were stopped by addition of 150 μl of chloroform containing 0.1 M diethylhexylphosphoric acid, followed by addition of 400 μl of 0.05 M sodium phosphate buffer (pH 6.8). After brief vortexing, tubes were centrifuged to separate phases, and the aqueous phase was discarded. Aqueous washing of the organic phase was repeated two times, after which the organic phase was pipetted into scintillation vials, dried, and counted.

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Dynamical Role of Predators in Population Cycles of a Forest Insect: An Experimental Test

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Population cycles occur frequently in forest insects. Time-series analysis of fluctuations in one such insect, the southern pine beetle (*Dendroctonus frontalis*), suggests that beetle dynamics are dominated by an ecological process acting in a delayed density-dependent manner. The hypothesis that delayed density dependence in this insect results from its interaction with predators was tested with a long-term predator-exclusion experiment. Predator-imposed mortality was negligible during the increase phase, grew during the year of peak population, and reached a maximum during the period of population decline. The delayed nature of the impact of predation suggests that predation is an important process that contributes significantly to southern pine beetle oscillations.

Ecologists have been trying to solve the puzzle of population cycles for at least three-quarters of a century (*1*). One class of ecological system that seems particularly prone to population oscillations is insects attacking forest trees (*2*, *3*). Because these insects cause

widespread economic damage, the causes of their outbreaks have been a focus of intensive research. Despite this effort, however, the biological mechanisms that drive oscillations are not yet well understood even in the best-studied systems (*2, 4*). Here we present results of a long-term field experiment designed to test the hypothesis that cycles in one forest insect, the southern pine beetle (SPB) *Dendroctonus frontalis*, are driven by the beetle's population interaction with its predators (we use the term "predators" in the broad sense that includes parasitoids, but not pathogens).

During the 1980s, SPB outbreaks in pine forests of the southern United States were thought to be driven by exogenous (density-independent) factors, namely, fluctuations in climate (*5, 6*). However, our analysis of SPB activity in eastern Texas, USA, during 1957 to 1987 did not reveal any statistically significant effects of climatic variables on the rate of population change (*7*). Time-series analysis indicated that SPB fluctuations were driven primarily by endogenous (density-dependent) factors: ~80% of the variance in the rate of population change was explained by a joint action of current and lagged population densities. The evidence for second-order dynamics [that is, delayed density dependence; see (*8*) for the definition of process order] was strong, because regression of the rate of population change on lagged density was highly significant ($P < 0.0001$) and it alone explained 55% of the variance (*7*). First-order endogenous factors (those that act in an undelayed manner) or exogenous influences are not unimportant; the former may prevent oscillations from getting out of hand, whereas the latter add stochastic irregularity. However, to understand why SPB populations oscillate, we should look to those mechanisms that act in a delayed density-dependent manner, because theory states that lags in regulation promote the possibility of cycles (*9*).

Several ecological mechanisms can generate second-order dynamics: maternal effects (*10*), food quantity (*11*) or quality (*12*), pathogens (*13*), and specialist predators or parasitoids (*14, 15*). Although time-series analysis cannot distinguish between these alternatives, it suggests how to formulate rival hypotheses in quantitative and testable terms [the predictions of the experiment described below were published in (*7*)].

The question we addressed experimentally was, what is the dynamical role of predation in the SPB cycle? A demonstration that predators inflict substantial (or even overwhelming) mortality at any particular point in time does not tell us whether predators are responsible for the oscillation or not. We need to determine how the predator impact changes with time, or more precisely, with cycle phase. Three broad outcomes can be distinguished, corresponding to the hypotheses that predators are (i) an exogenous, (ii) a first-order endogenous, or (iii) a second-order endogenous factor (Fig. 1).

In the first case, there is no dynamical feedback between prey density and the predation impact. The average predator-induced mortality may be very high and still predators would have no dynamical impact, simply reducing the intrinsic rate of population increase to a lower value. Fluctuations in predator-imposed mortality will affect prey density in a stochastic manner, but they cannot drive a regular oscillation. In the second case, predators respond to changes in prey population without a significant lag time. The dynamical role of predators, therefore, is stabilizing rather than causing oscillations (*16*). Generalist predators may act in this manner, reducing the amplitude of oscillations or preventing diverging oscillations. Only in the third case, when acting in a delayed density-dependent manner, are predators actually causing the oscillation. Note that the three scenarios represent extremes of a continuum, because it is possible for the predator community to act in a mixed manner (for example, a mixture of generalist and specialist predators would act in a manner intermediate between cases 2 and 3).

To determine which of the three scenarios (or some combination of them) characterizes

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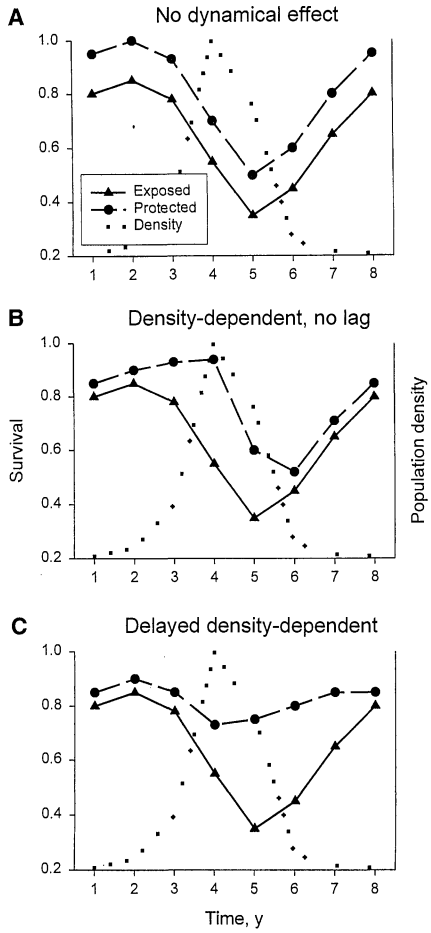

Fig. 1. Possible dynamical effects of predation. In all graphs, the dotted line indicates SBP population density during the course of a single oscillation, peaking in year 4. The solid line indicates the survival rate that determines the course of the oscillation (for simplicity, we assumed fecundity to be constant). The broken line indicates the survival rate when predators are excluded, and the separation between the solid and broken lines measures the predation impact. (**A**) The expected or mean predation impact does not vary with density. If predator impact has a large stochastic component, then predators will act as an exogenous factor; if predation impact does not vary with time, then predators are a null factor. (**B**) Predation acting as a first-order process, with the greatest impact occurring during the peak year. (**C**) Predation acting as a second-order process, with the greatest impact occurring during the period of population collapse. If predation were the dynamical factor completely responsible for population change, then the broken line in (C) would be completely flat.

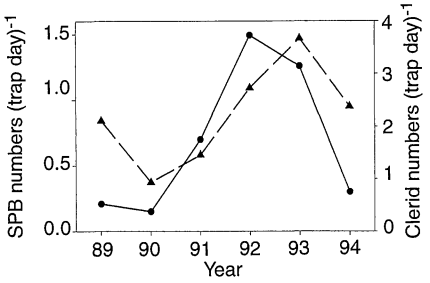

Fig. 2. Population numbers of the SPB (circles, solid line) and one of its important natural enemies, the clerid beetle *Thanasimus dubius* (triangles, dashed line), during 1989 to 1994, as measured by a network of pheromone-baited traps within Kisatchie National Forest.

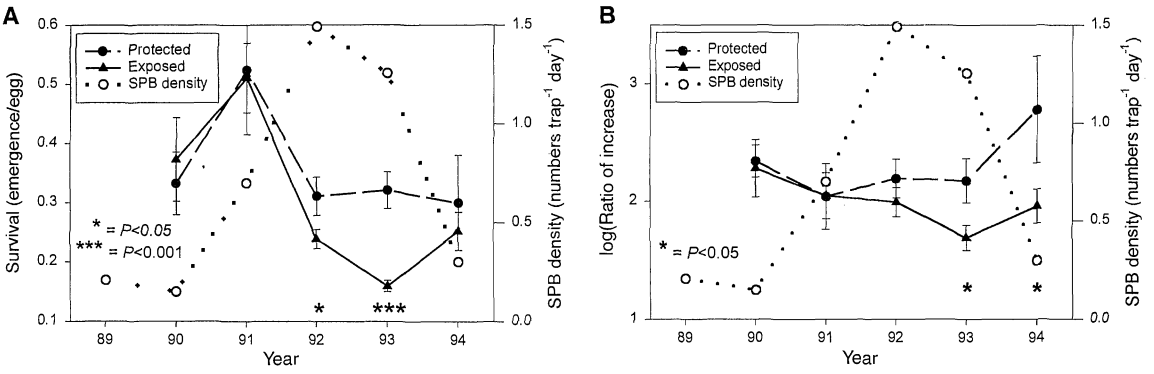

Fig. 3. (**A**) Survival of bark beetles, measured by the proportion of eggs surviving to become emerging adults: protected from predation (inside cages, broken line) and exposed to predation (outside cages and on control trees, solid line). The dotted line indicates the course of the outbreak (from Fig. 2). (**B**) Natural logarithm of the SPB ratio of increase, defined as the number of emerging adults divided by the number of attacking adults (the previous generation). Same notation as in (A). For statistical tests, see (*18*).

the predation impact in the SPB system, we performed a long-term study that measured predation impact by experimentally excluding all natural enemies of the SPB (*17*). The 5-year-long study covered a complete increase-peak-decrease cycle (Fig. 2). In 1990 and 1991 the survival of SPB brood inside cages did not differ from that outside cages (Fig. 3A), indicating negligible predation impact during the increase phase (*18*). Predators imposed detectable mortality during the peak year (1992), but numerically the strongest effect of predation was observed during the first year of decline, 1993 (*19*). We observed a qualitatively similar pattern in the effect of predators on the SPB ratio of increase (Fig. 3B); but this measure of predation was statistically significant during both decline years, and not during the peak year. Thus, both measures indicate that the predator complex acts primarily as a second-order (that is, delayed) process, with perhaps an admixture of a weaker first-order impact. The second-order effect is probably due to arthropod natural enemies, including several species of parasitoid wasps and predacious beetles (*20*). One predator, the clerid beetle *Thanasimus dubius*, appears to be a particularly promising subject for further investigation. This predator is a specialist on bark beetles, capable of inflicting significant mortality on SPB (*21*), and its densities exhibit oscillations that are phase-shifted with respect to those of SPB (Fig. 2). A particularly interesting feature of this predator is its tendency to go into an extended diapause (*22*). It is known that long developmental delays can have a destabilizing effect on dynamics (*23, 24*).

Our finding that predators in the SPB system act as a second-order process should be tempered by two caveats. First, our results do not preclude the possibility that other mechanisms (for example, maternal effects, food quantity or quality, and pathogens) also contribute to the delayed density-dependent pattern of SPB dynamics. Nevertheless, given the consistent and forcible impact of predators (50% decrease in survival and 50 to 70% decrease in the ratio of increase), it is clear that they play an important role in driving SPB oscillations. A twofold survival differential per generation translates into a 32- to 64-fold differential per year (because there are five to six SPB generations per year).

Second, our experiment was designed to determine the dynamical role of the whole predator complex. Thus, we do not yet know which particular enemies play an especially important role in causing SPB oscillations. Currently, our results implicate *T. dubius* as a particularly numerous and effective predator of the SPB. However, the SPB is a native "pest" of pines, and there is a diverse predator community associated with it (*20*). Only continuing empirical work coupled with modeling can yield quantitative estimates of the relative importance of different SPB predators.

Ecologists have used three general approaches to investigate potential mechanisms that can explain population cycles: general ecological theory based on mechanistic models (*25, 26*), analyses of time-series data (*8*), and field experiments (*27, 28*). No single approach in isolation can resolve the issue of why a particular population exhibits density oscillations. As our study and another recent study (*28*) illustrate, greatest progress may be achieved when all three approaches are used synergistically in investigations of population cycles.

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16. First-order predation acting in an overcompensating manner may cause cycles, but such first-order cycles are characterized by short periods (for example, two-point cycles; even four- or eight-point first-order cycles are dominated by a period of 2) as opposed to multiannual (or multigenerational) second-order cycles with which we are concerned in this report. Note that SPB oscillations are characterized by a period of 6 to 8 years (*7*) or 30 to 48 generations, assuming five to six SPB generations per year (*29*).
17. The cage design and experimental protocol are similar to previous work (*30*) and only briefly summarized here. At the beginning of each experiment, we located stands of loblolly pine in the Kisatchie National Forest, Louisiana, USA, and selected trees of the same diameter (25 to 30 cm) separated by 100 m. On some ("cage") trees we installed 2-m-tall cylindrical exclosures made from polyethylene screening, and other trees were used as controls. The cage was divided into a central 1-m experimental area with 0.5-m buffer zones above and below, which acted as barriers to the movement of insects into the experimental area. (Some predators managed to enter the exclusion zone, however, and thus our results are probably conservative with respect to measuring predation impact.) The trees were baited with SPB aggregation pheromone to induce attack, and then adult beetles (2000 total) were added to the experimental area of the cage in installments, generating an attack pattern and density that mimicked exposed areas. The densities of successful attacks and eggs inside and outside the cage were estimated by taking bark samples (*30*) after egg laying was complete. When brood development was complete, sections of the trunk were cut from inside and outside the cage and placed in individual rearing cans, and the number of emerging SPB recorded. Apart from having no cage installed, the control trees were treated exactly as the cage trees. In addition to using replicate trees at each location, we replicated the study spatially (two locations separated by at least 3 km) and seasonally (early summer versus fall), for a total of four studies each year. The complete data set over the 5-year course of the study consisted of 56 cage and 64 control trees. We quantified predation impact in two ways: by calculating the survival (emerging adults divided by eggs) and ratio of increase (emerging adults divided by successful attacks) both inside and outside cages. The densities of successful attacks and eggs were statistically indistinguishable inside compared with outside the cages, except for 1994, when egg density was significantly lower outside cages. This does not affect our main result, however, because lower egg density elevated SPB survival outside cages, resulting in a conservative estimate of predation impact.
18. The statistical significance of the effect of predator

exclusion was assessed by t tests, treating each year as a separate comparison. The survival on control trees versus outside cages on experimental trees was not significantly different (apart from fall of 1992), so we treated these replicates as a single category, "exposed." Our main result was not affected by this data pooling, because in 1993, the first year after the peak, survival inside cages was significantly higher than survival both outside cages on experimental trees and on control trees ($P < 0.001$ for both comparisons). The comparisons in the ratio of increase were performed in the same manner, except the data were first log-transformed to stabilize the variance.

19. The difference in survival inside and outside of the cages was not statistically significant in 1994, the second decline year. This was probably because of lower intraspecific competition outside of the cages due to lower egg density there (*17*). There was a significant negative effect of egg density on survival inside exclusion cages (linear regression: $F_{1,51} = 9.38$, $P < 0.0035$). When we corrected survival rates by taking intraspecific effects into account using this relation, we found a significant difference in survival, suggesting that the predation impact on survival was still detectable 2 years after the peak.
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Common Dynamic Structure of Canada Lynx Populations Within Three Climatic Regions

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Across the boreal forest of Canada, lynx populations undergo regular density cycles. Analysis of 21 time series from 1821 onward demonstrated structural similarity in these cycles within large regions of Canada. The observed population dynamics are consistent with a regional structure caused by climatic features, resulting in a grouping of lynx population dynamics into three types (corresponding to three climatic-based geographic regions): Pacific-maritime, Continental, and Atlantic-maritime. A possible link with the North Atlantic Oscillation is suggested.

Periodic population fluctuations of the Canada lynx (*Lynx canadensis*) have greatly influenced both ecological theory and statistical time series modeling [(*1*, *2*); see (*3*) for a summary]. Recent analyses have focused on the extent of synchrony in population fluctuations, assessing the importance of external abiotic factors (such as weather) and internal biotic factors (such as dispersal among populations) in causing spatial patterns (*4*). Such empirical and theoretical approaches have, however, assumed that the populations were structurally similar [that is, the density-dependent relationships are identical among populations (*5*)]. This assumption has never been thoroughly evaluated. To do so requires determining whether the lynx populations display the same phase- and density-dependent structure (*3*) and then searching for similar underlying causes of the observed dynamics. Using new statistical methods developed for this purpose (*6*), we ask to what extent the time series on the Canada lynx (Fig. 1) compiled by the Hudson Bay Company for the period 1821 to 1939 (*7*) and the corresponding more modern time series compiled by Statistics Canada for the period 1921 to present (*8*), taken together, are structurally similar. Specifically, we ask whether the phase- and density-dependent structure of changes in lynx abundance cluster into groups defined according to ecological-based features (*9*) or according to climatic-based features (*10*, *11*).

The available time series (Fig. 1A) cover two ecosystems (referred to below as ecological regions): the northern, open boreal forest (Fig. 1B) and the southern, closed boreal forest. In western Canada, the mountainous topography adds complexity. Additionally, the series cover three climatic regions defined by the spatial influences of the North Atlantic Oscillation (NAO) [Fig. 1C; see (*12*)], which may contribute to spatial differences in trophic interactions (*13*).

Previously, we fitted a piecewise linear autoregressive model (*14*) to each of the series (*3*). A general hare-lynx model (*3*, *15*) may be expressed as an equivalent model in delay coordinates of the lynx (the species for which we have data). Here we check whether all the time series, or some subsets of these, display the same underlying phase- and density-dependent structure. For this purpose we use a piecewise linear model (*14*, *15*):

$$y_{s,t} = \begin{cases} \beta_{s,1,0} + \beta_{s,1,1} y_{s,t-1} + \beta_{s,1,2} y_{s,t-2} + \varepsilon_{s,1,t} & y_{s,t-d} \le \Theta_s \\ \beta_{s,2,0} + \beta_{s,2,1} y_{s,t-1} + \beta_{s,2,2} y_{s,t-2} + \varepsilon_{s,2,t} & y_{s,t-d} > \Theta_s \end{cases} \quad (1)$$

where $y_{s,t}$ is the log-transformed abundance of lynx at site s and for year t [that is, $y_{s,t} = \log(Y_{s,t})$ where $Y_{s,t}$ is the abundance of lynx at site s and in year t, and where s = 1, 2, . . . , represent the sites corresponding to the individual time series; see Fig. 1A]; $\beta_{s,i,j}$ are the statistical parameters that determine the phase- and density-dependent structure of the system (i = 1 and 2 correspond to the lower and the upper regimes of the model; j = 0, 1, 2 correspond to the constant term, the first lag, and the second lag, respectively) at site s; $\varepsilon_{s,i,t}$ is normally distributed, time-independent noise [$N(0,\sigma^2_{s,i})$]; and θ_s is the threshold applicable to the log-transformed density d years earlier.

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