The filament (shown in Fig. 3A as a linear structure for simplicity) is more likely to be compacted in a solenoid in which the DNA wraps through multiple turns about a protein core, as was suggested by the observed changes in linking numbers upon formation of a SopB-DNA complex (19).

Prokaryotic centromeres are characterized by the presence of arrays of sites to which one of the partition proteins bind. There are 6 sites in p1 (Fig. 1A), 12 in F, 10 in R1, and 12 in pTAR (20). Whereas these plasmid-borne binding sites are tightly clustered, the several chromosomal binding sites for the Bacillus subtilis partition (and sporulation) protein Spo0P are distributed over a region spanning many kilobases (3). The centromere may serve as a handle that is used to tether or orient a large structure, with its several binding sites facilitating a steady grip or the formation of an intermediate that is appropriately paired for partitioning. Evidence for paired intermediates in the partitioning of plasmid R1 and involving its cognate ParB analog has recently been obtained (21). Like the proteins of heterochromatin that spread from centromeres or telomeres of eukaryotic chromosomes, the ParB that spreads from the P1 plasmid centromere can silence genes. In each case, this capacity for gene silencing may be incidental to a primary structural role that is associated with DNA segregation or movement.

References and Notes
13. The par5-bearing plasmid pMLO6 (12) could not be maintained in the presence of a source of ParB. By moving par5~180 from its position in pMLO6, a plasmid was created that, in the presence of ParB, no longer conferred spectinomycin resistance but had been shown to be the correct position in pMLO6. The altered distance of par5 from the DNA lori that are involved in replication and in the expression of antibiotic resistance can account for the difference in phenotypes.
15. O. Rodionov and M. Yarmolinsky, data not shown.
18. K. Gerdes, unpublished material.
28. Constructs O, I, and II were derived from a recA56 MC1061 (22) by the integration of par5, lacZ, and cat into attL. (23). The lacZ gene under control of the P1 repA promoter (par5), accompanied upstream by four rmb1 transcription terminators, was from pP112 (24). The source of par5 was a 298–base pair Eco RI fragment from pB145 (9). The cat gene was from pST52 (25). A mutant version of construct I that is constitutive for phoA expression was used for the alkaline phosphatase assay. Gene orientations and the absence of duplicate insertions were determined by restriction mapping.
29. To construct pOAR32, we cloned the par5 gene under tac promoter control in a derivative of pMMB67EH (26) in which the kanamycin-resistance gene of transposon (Tn) 903 had been inserted so as to disrupt the bla gene of the vector. For assays, cultures were grown in LB broth (27) with kanamycin (25 mg/ml) and, subsequently, for about six generations, with isopropyl-β-D-thiogalactopyranoside (IPTG) at several concentrations before the measurement of enzyme specific activities. The ParB concentration in sonicated cell extracts was estimated by the ECL protein immunoblotting analysis system (Amersham) with highly purified ParB as standard.
32. Template DNAs were purified with a high-purity PCR template preparation kit (Boehringer Mannheim) and, unless otherwise indicated, used at a dilution of 1:850. PCR reactions were performed with 100 pmol of each primer and 0.25 units of Taq polymerase in 1:8 buffer (Promega) in 50-μl volumes as follows: An initial denaturation at 95°C for 2 min was followed by 25 cycles with denaturation for 30 s at 95°C, annealing for 30 s at 61°C (at 54°C with primers directed against P1 DNA), polymerization for 1 min at 72°C, and a final 2-min extension at 72°C.
33. Single-letter abbreviations for the amino acid residues are as follows: D, Asp; E, Glu; C, Gly; I, Ile; K, Lys; L, Leu; P, Pro; Q, Gln; R, Arg; T, Thr; and V, Val.
36. M. Lobocka, M. Rusin, A. Samojedy, unpublished material.
37. We thank D. K. Chattoraj for valuable discussions, bacterial strains, plasmids, and antibody to RepA, and, with R. A. Weisberg, for critical readings of the manuscript; K. Gerdes for permission to cite unpublished material; B. E. Funnell for a sample of highly purified ParB; and D. C.-H. Lin for the cross-linking immunoprecipitation protocol that he developed in the A. D. Grossman laboratory. O.R. and M.L. were supported by Fogarty postdoctoral fellowships.

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LC neurons were recorded in four *Cynomolgus* monkeys performing a visual discrimination task (2). This task required the monkey to respond to infrequent visual target stimuli but not to frequent distractors (3). In many of our recordings, LC neurons changed levels of tonic discharge several times (Fig. 1A), in association with alterations in task performance. We divided behavioral performance into epochs of “good” and “poor” performance, on the basis of the frequency of false alarm (FA) errors produced [as described previously (2, 4)]. Signal detection sensitivity ($d'$) was substantially higher in epochs of good compared with those of poor performance, so the difference between these cannot be explained by a simple change in response criterion (4). Furthermore, although mean lever response times (RTs) were not systematically different between the two levels of performance, there was a significant narrowing of the distribution of lever release latencies during the good epochs (4) (Fig. 1B).

As shown in Fig. 1A, epochs of poor performance were associated with significantly higher tonic LC activity than were epochs of good performance ($3.0 \pm 0.3$ spikes/s compared with $2.0 \pm 0.2$ spikes per second $P < 0.001$; FA frequencies were $7.6 \pm 0.9\%$ compared with $1.0 \pm 0.2\%$ of trials; $P < 0.01$; $n = 30$ cells; paired $t$ tests). Similar results were obtained in an additional 37 multicell LC recordings. Thus, in addition to our previous finding of a close relation between phasic LC discharge and behavioral responses (2), we also found a close relation between the level of LC tonic activity and behavioral performance. We refer to the lower level of tonic LC activity during epochs of good performance as “intermediate,” to distinguish it from the low (near zero) level typically associated with drowsiness or sleep (2, 5).

We also found that sensory-evoked LC responses varied with the level of tonic LC activity and task performance. The phasic responses that LC neurons exhibit selectively for target stimuli in this task occurred almost exclusively during epochs of intermediate tonic LC activity and good task performance (Fig. 1, C to F). For the 30 single-cell recordings described above, response magnitudes to target stimuli during epochs of good performance were significantly greater than during epochs of poor performance ($2.7 \pm 0.4$ compared with $0.8 \pm 0.2$; $P < 0.001$; paired $t$ test). Thus, increased tonic LC discharge was associated with decreased responsivity of LC neurons to target stimuli as well as decreased task performance. This three-way association of tonic LC activity, LC phasic responses to target stimuli, and level of task performance was observed consistently across our recordings.

These results suggest that there is a precise relation between LC activity and behavioral performance. To elucidate the mechanisms that might underlie this relation, we developed a computational model of LC function and its effect on performance in this task.

The model is a hybrid, with two primary components: an LC network and a stimulus discrimination (behavioral) network (Fig. 2A). The LC network is relatively fine-grained and designed to simulate physiological mechanisms underlying LC function, whereas the behavioral network is the simplest capable of simulating performance in the visual discrimination task. Although the use of such a hybrid model that combines components at different levels of abstraction may be unusual, this is justified by the correspondence between each component of the model and the

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level of the phenomena it addresses.

The LC network is a population of 250 spiking neurons, each of which is a leaky integrate-and-fire cell (6, 7) that exhibits temporal dynamics similar to those in compartmental models (8). In the model, LC cells interact with each other in two ways. First, lateral inhibition simulates the effect of local NE release (9, 10). Second, a voltage-dependent interaction among LC units simulates the effects of hypothesized electrotonic coupling among LC neurons (11). In addition, each LC cell receives input from the behavioral network (see below), as well as noise that is responsible for a spontaneous firing rate of about 1 spike/s [as observed in vivo (2)].

The behavioral component of the model is a simple connectionist network, consisting of two input units (one for target and one for distractor stimuli), two corresponding decision units, and one response unit (Fig. 2A). Connections between units in different processing layers are excitatory (reflecting information flow), connections within a layer are inhibitory (competition), and the activity of units is subject to small random variations (noise) (12). Each input unit has a strong weight to the corresponding decision unit and a weaker projection to the other decision unit. The target decision unit has a positively weighted connection to the response unit and to the LC network (13). Finally, consistent with previous simulation work, we assume that NE release has the effect of increasing the gain of the activation function for units in the decision and response layers [see below and (14, 15)].

A task trial was simulated by activating the input unit corresponding to the current stimulus, which resulted in the spread of activation to the competing units in the decision layer and then to the response unit and LC. Characteristic dynamic responses of different units in the behavioral network after presentation of each type of stimulus (in the absence of modulation by LC) are displayed in Fig. 2B.

The simulated pattern of LC firing with and without electrotonic coupling, after target and distractor stimuli, is shown in Fig. 3. Target stimuli evoke a transient, synchronized LC response as a result of input from the target decision unit to LC cells. The target-evoked response is terminated by NE-mediated collateral inhibition within the LC. Electrotonic coupling among LC neurons has two main effects. First, coupling causes a stronger response of the LC population to target inputs, as a result of the reinforcement of spike-induced depolarizations in each individual neuron by similar, simultaneous depolarizations in other LC cells within the population. Second, coupling reduces the spontaneous (tonic) firing rate of LC cells by mutually shunting the effect of uncorrelated noise on each cell’s membrane potential (16).

These simulation results closely resemble the patterns of monkey LC discharge observed during epochs of intermediate (versus high) tonic activity and good (versus poor) behavioral performance (Figs. 1 and 3).

As noted above, the output of the target decision unit provides input to the LC net-

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Fig. 2. (A) Architecture of the model of task performance. Arrows represent excitatory links and small circles represent inhibition. There is a moderate positive bias on the response unit, which captures the observation that monkeys in this task make many FAs but very few misses (2). (B) Dynamic trajectories of the target, distractor, and response assemblies (as indicated), in response to targets (top) or distractors (bottom). Stimulus presentation is at time zero. Solid lines, target unit; dashed lines, distractor unit; dotted lines, response unit. In response to each stimulus, there is partial activation of both target and distractor decision units due to their overlapping connections with the input. However, because of mutual competition, after about 100 ms, the decision unit corresponding to the activated stimulus typically prevails, and the competing unit is suppressed. When the target unit prevails, the activity of the response unit is driven above threshold, and a response is recorded. FAs occur because of noise in the response unit, which interacts with transient activation of the target decision unit by a distractor stimulus to produce a response. A threshold is set for activation of the response unit (0.6).

Fig. 3. (A to D) PSTHs for the simulated data. (A and B) Response to targets. (C and D) Response to distractors. (A and C) Coupling among LC neurons. (B and D) No coupling among LC neurons. PSTHs are normalized for 100 trials, as for the empirical data (see Fig. 1). (E) Response time distributions for model responses (response unit activations) after targets. Solid line, distribution during simulated coupling among LC neurons; dashed line, distribution during no coupling among LC neurons. The difference of about 150 ms between the latencies of empirical (Fig. 1B) and simulated behavioral responses (E) is consistent with a residual sensory or motor latency.
work, whereas LC activity modulates the gain of units in the decision and response layers of the behavioral network. Unlike in previous models, where the effect of catecholamines on cognitive performance was modeled as a fixed gain parameter throughout a simulation (15), here the value of the gain was determined dynamically by the output of the LC network. Thus, the synchronized, transient responses to target stimuli during epochs of high coupling (Figs. 1, C to F, and 3, A to D) resulted in a temporally modulated process. The effect of LC on the performance of the behavioral network can be seen by comparing the activation of the response unit under conditions of high and low coupling among LC neurons. Increased coupling among LC neurons produced a reduction in FAs (from 12 to 2%), without an increase in misses, and a significant narrowing of the RT distribution, without a change in the mean (Fig. 3E). Thus, a change in coupling among LC neurons in the model reproduces the changes in LC activity, behavioral performance, and the relation between these that is observed empirically.

Changes in electrotonic coupling produce the associated changes in behavior for several reasons. First, increased coupling reduces tonic LC activity, reducing NE release in the behavioral network and thereby lowering the responsivity of those units. For the response unit, this is equivalent to raising its threshold (15), which reduces the number of FAs and anticipatory responses. Ordinarily, raising the response threshold would also increase the number of misses and lengthen mean RT. However, increased coupling enhances evoked LC responses to target stimuli. The enhanced LC response produces a transient reduction in threshold specifically and shortly after target stimuli, which compensates for the overall increase in response threshold and potentiates the processing of target stimuli. This averts an increase in misses or RT (17). This temporal modulation of processing, with maximal gain occurring shortly after a target stimulus, is consistent with an attentional window reported in the cognitive literature (18) and also with recently proposed mechanisms for attentional modulation based on neural synchrony (19). Moreover, this mechanism has the combined effect of eliminating anticipations and of speeding up slow responses, explaining the observed narrowing of the RT distribution during the good behavioral epochs (Figs. 1B and 3E). Thus, a change in a single parameter (an increase in coupling within LC) can account for the reduction in tonic LC activity, the enhanced target-evoked phasic responses, and the association of this pattern of LC activity with a reduction of FAs and a tightening of the RT distribution in behavioral performance.

The model makes the prediction that improved performance is associated with increased electrotonic coupling and therefore should also be associated with greater synchrony in the spontaneous firing of LC neurons (Fig. 4B) (20). We tested this prediction by comparing cross correlograms generated for pairs of simultaneously recorded LC neurons during epochs of good and poor performance. Consistent with our prediction, we found that 18 of 23 pairs of recorded neurons exhibited a central peak in cross correlograms during epochs of good performance that was not present for the same neurons during poor performance (Fig. 4, A and B). Quantitative analyses of correlograms for these 23 pairs of cells indicated that the central peak during good performance was significantly greater than during poor performance.

Our simulation results suggest that electrotonic coupling may be an important mechanism underlying patterns of LC activity and may play a role in regulating behavioral performance. Strong evidence for coupling within the LC of neonatal rats has been reported (21). Although electrotonic coupling appears to decrease postnatally, recent studies indicate that coupling may persist in the LC of the adult rat (22, 23). However, the presence of such coupling in the adult primate has not yet been empirically demonstrated. The model we have developed, together with the data regarding synchronization of LC activity, support this possibility and indicate that modulation of electrotonic coupling may produce potent effects on behavioral performance (24).

One important question concerns the adaptive advantage of the changes in behavior that are produced by changes in LC activity. In our model, intermediate tonic LC activity (due to increased coupling) facilitates a state of selective responding. This state is beneficial in a stable environment such as in our experimental task, where the source of reward is predictable and the behaviors relevant for acquiring it are known and consistent. However, what are the advantages of high tonic LC activity, which is associated with impaired performance in our experimental task? One possible answer is that heightened selectivity may at times be disadvantageous, such as in an uncertain or stressful environment, in which unexpected but imperative stimuli occur (for example, prey suddenly faced with a predator), or when previously reinforced responses lose their reward value (for example, satiety). Such circumstances require reevaluation of the sensory environment and abandonment of current behaviors in the search for more adaptive ones. This ability may also be critical for normal developmental and learning processes, as suggested by recent findings indicating that the best predictor of success in acquiring a new skill is not the speed with which the correct behavior is first discovered but the number of alternatives that are initially explored (25). According to our model, high tonic LC activity (as a result of low coupling) can provide a mechanism for sampling new stimuli and behaviors by reducing attentional selectivity and increasing behavioral responsiveness to unexpected or novel stimuli.

These considerations suggest that a tension exists between optimizing performance in a stable environment and favoring more flexible behavior in a changing or unfamiliar environment or when current rewards lose their value. This is a fundamental trade-off, which has been recognized in computational theories of reinforcement learning that distinguish between states that favor "exploitation"
of existing behavioral routines versus “explo-
ration” of new ones (26). The mechanisms
responsibility for shifting between such states
have not been specified. Our model indicates
that changes in the mode of LC functioning
produced by alterations of electrotropic cou-
pling) may provide a neural mechanism for
mediating such shifts. This hypothesis also
helps to integrate previously proposed roles
for LC function (27). Future research is need-
ed to directly test this hypothesis (28) and
to characterize the neural system or systems
providing input to the LC that are responsible
for monitoring the current behavioral context
and altering coupling among LC neurons
when shifts of state are appropriate. It will
also be important to determine the relation of
the LC-NE neuromodulatory system to oth-
ers, such as the dopamine system, that are
thought to regulate behavior based on expec-
tations about future events (29).

References and Notes
3. Training and experimental recording sessions took place in an acoustically insulated, electrically
shielded metal chamber (IAC, Bronx, NY). Monkeys
were trained to depress a lever and to stably
place in an acoustically insulated, electrically
insulated, electrically

4. Typically, epochs of poor performance contained
more than seven times the frequency of FA errors as
epochs of good performance. The hit rates varied
only slightly between these periods, remaining either
constant or declining slightly during poor perfor-

5. For the three weights analyzed, the d' values in poor compared with good periods in-
creased from 2.9 to 5.1, 3.7 to 4.7, and 3.7 to 5.1. The response criterion b also increased during the good
periods (0.36 to 2.92, and 0.06 to 1.11, respectively.
For these monkeys, the standard deviations of
RTs were 55, 55, and 46 ms, respective-
ly, during poor periods and 35, 33, and 35 ms,
respectively, during epochs of good performance
(P < 0.001; Levene test of variances).
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8. Each LC cell integrates its input current (see below) and fires when its voltage at time t, V(t), reaches
threshold (Vth) after it is artificially reset to
rest (V = 0) and remains refractory until its voltage
begins to rise again. We chose a refractory period
of 10 ms, to mimic the afterhyperpolarization that
follows individual LC spikes, V(t + Δt) = λV(t) + b,
where λ is related to the membrane integration constant. If input current t = 0 that depends
additively on the activity of the target cell assem-
blage x, the total amount of NE, and the hypothe-
sized gap-junction current (f) (see below) and is also affected by CNG channel noise. The
input current f(t) = ∑ j i = 1 netinput(t,i) on each LC unit is proportional to the sum of the ohmic currents
contributed by the other LC units (which depend
on the differences in voltage); for spiking neurons,
Vspike = SVc.
Res. 136, 570 (1977); T. M. Egan, G. Henderson, R. A.
North, T. Willis, E. Foote, J. Exp. Physiol. 63, 477 (1983); M.
11. Lateral inhibition occurs with a rise time of about 25
ms after LC cell firing and a decay of 250 ms [S. L. Foote, E. Foote, J. Exp. Physiol. 63, 844 (1983)]. This collateral NE release regulates
the firing rate of the LC population: After each target-
evoked, synchronized response of the population (see below), a slightly delayed inhibitory effect appears
(as reflected in PSTH histograms: Figs. 1C
and 3A).
12. Electrotropic coupling is consistent with observa-
tions of gap junctions among LC neurons in neo-

cultural level. Rather, it is intended to simulate tasks for which the activity of the other unit
is transiently activated by the target unit, either directly or after a delay. The length of this delay
is determined by the gain parameter (consistent with physiological data
on the differences in voltage); for spiking neurons,
Vspike = SVc.
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cultural level. Rather, it is intended to simulate tasks for which the activity of the other unit
is transiently activated by the target unit, either directly or after a delay. The length of this delay
is determined by the gain parameter (consistent with physiological data
on the differences in voltage); for spiking neurons,
Vspike = SVc.
Light-Gap Disturbances, Recruitment Limitation, and Tree Diversity in a Neotropical Forest

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Light gap disturbances have been postulated to play a major role in maintaining tree diversity in species-rich tropical forests. This hypothesis was tested in more than 1200 gaps in a tropical forest in Panama over a 13-year period. Gaps increased seedling establishment and sapling densities, but this effect was nonspecific and broad-spectrum, and species richness per stem was identical in gaps and in nongap control sites. Spatial and temporal variation in the gap disturbance regime did not explain variation in species richness. The species composition of gaps was unpredictable even for pioneer tree species. Strong recruitment limitation appears to decouple the gap disturbance regime from control of tree diversity in this tropical forest.

When a tree dies in a closed-canopy forest, it creates a “light gap,” a local disturbance that sets in motion a mini-successional sequence called gap-phase regeneration, which culminates in the replacement of the original canopy tree by one or more new trees (1). A widely accepted generalization in community ecology is that localized disturbances, such as treefall gaps, promote the coexistence of species having different resource use strategies and dispersal and competitive abilities—a hypothesis known as the intermediate disturbance hypothesis (2). A well-documented physiological and life-history trade-off exists in pioneers versus shade-tolerant mature forest trees in their degree of dependence on light and light gaps for germination, growth, and survival (3). At issue here is whether such life-history trade-offs exist or whether pioneers have an absolute requirement for gaps. The question is whether spatial and temporal variation in the gap disturbance regime is actually predictive of stand-to-stand variation in tree species richness and composition in particular tropical forests. If not, then the role of light gap disturbances in maintaining local tree diversity may need to be re-evaluated.

We tested the intermediate disturbance hypothesis in a 50-ha plot of old-growth tropical moist forest on Barro Colorado Island (BCI), Panama (4). All woody plants (excluding lianas) with a stem diameter of ≥1 cm dbh (diameter at breast height) have been tagged, measured, mapped, and identified to the species level (>300,000 stems comprising 314 species). Complete censuses have been conducted in 1982, 1985, 1990, and 1995 (5). From 1983 to 1996, we measured canopy height and gaps annually on a complete 5-m grid of 20,301 sample points (1, 6). From these data and the distribution of each species, we classified species into three regeneration niche guilds: strongly gap-dependent pioneer species, shade-tolerant species, and intermediate species (7). Through 1995, we monitored changes in 1983 sapling communities (stems 1 to 3.9 cm dbh) in all 1983 gap sites (canopy height 25 m) and nongap control areas. Control areas comprised the 28.1% of the 50-ha plot that remained in undisturbed high canopy (≥20 m) mature forest for the entire 13-year period. Because stem density increases in gap areas, we normalized species richness by dividing by number of stems. We compared species richness per stem in all 20 m by 20 m quadrats containing a gap in 1983 with nonoverlapping quadrats from control areas. We also tested for a relationship between the frequency of canopy disturbance and the 1995 species richness in the sapling community (8). Using a gap-focused method, we tested for an effect of gap size on species richness (9). In 1985, 1990, and 1995, we analyzed the sapling communities in same-aged (2-year-old) gaps created in 1983, 1988, and 1993. We analyzed the species richness and composition of sapling assemblies as a function of gap size for the three regeneration niche guilds. The disturbance regime in the BCI forest produces frequent but small light gaps from the death of one to several canopy trees (Fig. 1A). There are no records of severe disturbances such as hurricanes ever striking central Panama or BCI. Gaps varied over a 46-fold size range from 25 m² to the largest gap of 1150 m². Light gaps markedly increased sapling stem densities relative to nongap, mature forest control sites (P < 0.001). Gaps of 25 m² were legitimately included in the analysis, because pioneer species successfully germinated, survived, and grew in them (Table 1). As predicted by the intermediate disturbance hypothesis, quadrats containing light gaps had substantially more species than did quadrats in nongap, mature forest sites (P < 0.001, Komolgorov-Smirnov test) (Fig. 2A).