

Photoluminescence intensity versus  $\text{SO}_2$  concentration for porous silicon samples irradiated at 400 nm and monitored at 650 nm. Inset, An argon sulfur dioxide mixture (890 p.p.b.  $\text{SO}_2$ ) flows through cell containing oxidized luminescent porous silicon. Emission intensity shows a significant drop on introduction of the  $\text{SO}_2$  dose (time, 600 s) and subsequent restoration after the  $\text{SO}_2$  flow is shut off (time, 1,400 s).

con. Sulphur dioxide is unique in several ways. It is a Lewis acid, and that alone makes it stand out from the other quenchers. Sulphur dioxide quenching does not require an acid to restore the loss of photoluminescence. To restore light emission to its original intensity, it is necessary only to remove the source of  $\text{SO}_2$ . The blueshift of the photoluminescence maximum present in Brönsted base quenching does not appear, rather an obvious redshift occurs during  $\text{SO}_2$  quenching<sup>5</sup>. Plots of photoluminescence intensity versus time during exposure to  $\text{SO}_2$  and to a Brönsted base show kinetics that are more rapid for quenching by  $\text{SO}_2$ <sup>5</sup>. The differences observed between base quenching and  $\text{SO}_2$  quenching indicate that the silicon surface- $\text{SO}_2$  interaction is distinct from the previously reported Brönsted base interaction.

We have used illumination from an argon-ion laser (458-nm line  $<5 \text{ mW cm}^{-2}$  intensity) to irradiate porous silicon in a

quartz cell. On flowing a  $\text{SO}_2$ /argon mixture through the cell, the photoluminescence intensity decreases. When we used  $>10\%$   $\text{SO}_2$ /argon doses, we clearly saw luminescence quenching by the naked eye. Using a spectrofluorimeter to detect smaller changes in photoluminescence intensity, we found the limit of detection of  $\text{SO}_2$  in argon is 440 parts per billion (p.p.b.). This concentration produces a reproducible 1.2% change in the observed photoluminescent intensity. Different instrumentation is expected further to reduce the limit of detection. We observed saturation above 70%  $\text{SO}_2$  atmosphere, giving a useful working  $\text{SO}_2$  concentration range from approximately 400 to 700,000 p.p.m. (see figure).

Exposure of the  $\text{SO}_2$ -active oxidized porous silicon surface to high concentrations of the typical atmospheric and stack gases ( $\text{O}_2$ ,  $\text{CO}$ ,  $\text{CO}_2$ ,  $\text{H}_2\text{S}$  and  $\text{NO}_x$ ) does not illicit a quenching response. This makes it an interesting candidate for  $\text{SO}_2$  monitoring with no pre-separation of samples necessary. At present, the surface reactivity toward  $\text{SO}_2$  lasts for several weeks. Over that period of time, the layer of oxidized silicon grows thicker and the quenching response grows weaker, until eventually exposure to  $\text{SO}_2$  results in no quenching. Experiments involving silyla-

tion of the surface show that porous silicon retains  $\text{SO}_2$  reactivity on covalent bonding of a siloxy layer. Because such treatments are known to inhibit the oxidation of silicon surfaces<sup>6</sup>, it is probable that chemistry of this type can be used substantially to increase sensor lifetime.

In summary, we have observed selective gas-phase quenching of porous emission by  $\text{SO}_2$ . The photoluminescence loss is reversible and the signal achieved is of analytical value. Preliminary experimentation with surface modification to lengthen the active lifetime of  $\text{SO}_2$  sensitivity is promising. The high selectivity and sensitivity of porous silicon for  $\text{SO}_2$  detection suggests possible applications in environmental monitoring. The mechanism for  $\text{SO}_2$  quenching is apparently different from the better understood base-quenching mechanism, and requires further study. Clarification of the  $\text{SO}_2$ -quenching mechanism is expected to lead to optimal sensitivity and lifetime, and may suggest new applications for porous silicon as a sensor.

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## Species fragmentation or area loss?

SIR — Tilman *et al.*<sup>1</sup>, extending a metapopulation model to include multi-species competition and habitat destruction, arrive at the paradoxical conclusion that, in a patchy habitat, the species most at risk to deterministic extinction when habitat is lost is the dominant competitor.

This result arises from the equilibrium between the dominant species' colonization of new sites occupied by inferior competitors and its mortality loss. The fraction of sites  $p$  occupied by the dominant competitor is described by the equilibrium relationship  $dp/dt = 0 = cp(1-p-D) - mp$ , where  $c$  and  $m$  are coloniza-

tion and mortality rates and  $D$  is the fraction of sites destroyed. The factor  $(1-p-D)$  thus represents the proportion of sites available for colonization by the dominant competitor.

In an undisturbed habitat ( $D = 0$ ), the initial equilibrium value of  $p$  is given by  $p_0 = 1 - m/c$ ; in the disturbed habitat,  $p = 1 - m/c - D = p_0 - D$ . Thus if  $D$  is equal to or greater than  $p_0$ , the dominant species is subject to deterministic extinction. A dominant species occupying 10% of an undisturbed habitat will be wiped out if as little as 10% of that habitat is destroyed. Habitat loss reduces effective colonization

by all competitors, but the dominant competitor most of all, as it relies on a trade-off between a low intrinsic colonization rate  $c$  and a large proportion of available sites to colonise,  $(1-p)$  in the undisturbed condition.

What may be overlooked, however, is that the result obtains only if the sites selected for destruction are randomly distributed throughout the habitat, so that the same proportionate loss  $D$  of available colonization sites takes place in the local vicinity of every site occupied by the dominant

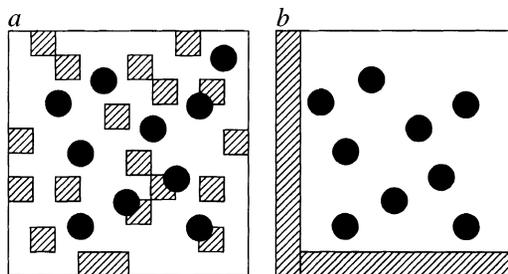


FIG. 1 a, Habitat destruction (shaded squares) randomly distributed throughout a patchy habitat proportionately reduces the sites open to colonization by the dominant competitors (black circles), reducing that species' effective colonization rate and, if the proportion of sites destroyed exceeds the proportion occupied by the dominant species, resulting in its eventual extinction. b, Destruction concentrated at the edge leaves the effective colonization rate unaffected.

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species (*a* in the figure). The analytical model of Tilman *et al.* implicitly assumes this spatial homogeneity.

Should the destruction of habitat be concentrated in a contiguous block along the perimeter, however, then the undestroyed sites occupied by the dominant species will each remain surrounded in their immediate vicinity by the same proportion of available colonization sites as before the habitat loss occurred (*b* in the figure), and the effective colonization rate will remain unchanged. Indeed, the very fact that the equilibrium condition and extinction predictions of the analytical model are independent of the absolute size of the original habitat force this conclusion. Without fragmentation, the equilibrium condition of a 1-million-acre forest will be unchanged from that of a geometrically similar, "intact" 10-million-acre forest.

Thus if destruction is concentrated at the perimeter, the analytical model would predict no deterministic extinctions through competitive effects. In such a case, extinctions would only occur when the absolute area of the habitat is reduced to the point that the populations become small enough to fall victim to stochastic extinction processes<sup>2</sup>.

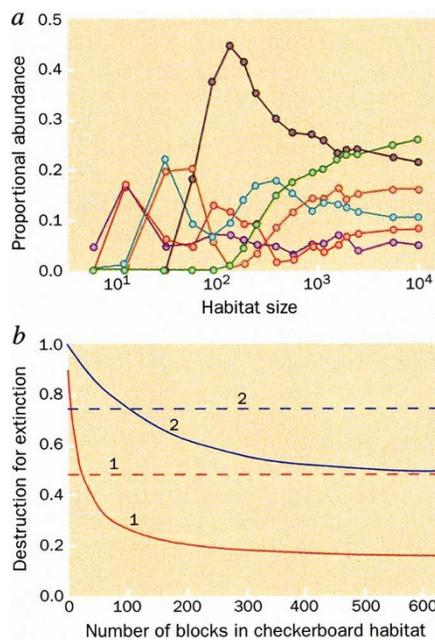
Extinction predictions based on species-area relations have been criticized<sup>3,4</sup> for assuming a functional relation between area loss and species loss where in fact none may exist. Metapopulation models offer a convincing mechanism to predict species loss in highly fragmented habitats, but offer no support for the notion that area loss *per se* results in extinction. Predictions of global extinction rates based on estimates of area loss alone seem to be unreliable, unless it can be shown that permanent habitat destruction (and not just temporary forest clearing) is occurring in a highly random fashion that fragments the habitat consistently on a very local scale.

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TILMAN *ET AL.* REPLY — Budiansky raises an important issue — that the quantitative relationship between the amount of habitat destroyed and the number of resulting species extinctions may depend on the spatial pattern of habitat destruction. Our analytical model<sup>1</sup> has no provision for habitat shape or size and cannot directly address this issue. However, a modified analytical model and extensive simulations of explicitly spatial versions of our model<sup>5</sup> reveal two important effects.

First, the novel prediction of our analytical model<sup>1</sup> holds: for all spatial patterns of habitat destruction we tried, destruction leads to the biased extinction of superior competitors<sup>5</sup>. Even when they originally were the most abundant species,



superior competitors become extinct at lower levels of destruction than inferior competitors when an otherwise pristine habitat is reduced in size, such as by the loss of edge (Fig. 2). This biased extinction is a deterministic effect, caused by poor dispersal abilities, not the stochastic effect alluded to by Budiansky.

For habitats as large as those containing 104 sites, loss of edge differentially harms the best competitor. As edge is lost, the best competitor goes extinct first (species 1; Fig. 2a) in habitats of the size that Budiansky (his Fig. 1) suggested would have no edge effects. Further reductions in habitat size causes progressive extinction of species in order of competitive abilities (Fig. 2a), as occurred with destruction in our analytical model<sup>1</sup>. Simulations show that better competitors, because of poorer dispersal, require larger areas for survival. The underlying reason for the robustness of our conclusions is the broad assumption that inferior competitors persist by virtue of greater dispersal ability and/or lower mortality rates.

It follows that better competitors, because of poorer dispersal, have larger minimal demands for habitat size. Furthermore, similar predictions of biased deterministic extinction of superior competitors arose when we modified our analytical model<sup>1</sup> by adding a term for the proportion of propagules lost to edges<sup>5</sup> (via decreases in colonization rate proportional to *S*<sup>-1/2</sup> for square habitats, with *S* being habitat size). Other simulations showed that patch shape also influenced extinctions: for patches of a given area, those with greater perimeters caused more extinctions, with extinctions again biased toward the best competitors.

Second, additional simulations showed that extinctions can occur at amounts of habitat destruction markedly greater than,

FIG. 2 a, Mean equilibril species abundance versus habitat size from replicate spatially explicit simulations of competition<sup>1</sup> among 6 species. Species 1 (green) is the best competitor but poorest disperser, species 2 (grey) the next best competitor and next poorer disperser, (species 3, 4, 5 and 6 are brown, blue, red and purple, respectively). Mortality was 0.05 yr<sup>-1</sup> and colonization was 0.076, 0.146, 0.2975, 0.601, 1.24 and 2.53 yr<sup>-1</sup> for species 1 to 6, with absorbing boundaries, annual reproduction, and propagules dispersed randomly across the neighbouring four rings of sites in a hexagonal array of *n* × (*n*-1) sites. b, Solid curves, amount of habitat destruction required to drive the best competitor (species 1) or the next best competitor (species 2) extinct when an explicitly spatial habitat was destroyed in a checkerboard pattern. Dashed lines, amount of habitat destruction required for extinction of species 1 and 2 as predicted by our analytical model<sup>1</sup>. Here, a 100 × 99 hexagonal habitat was divided into from 1 to 625 equal-sized blocks in a checkerboard pattern. Solid curves show the contiguous proportion of each block that had to be destroyed to cause extinction of species 1 or 2 for various numbers of blocks.

markedly less than, or similar to those predicted analytically<sup>1</sup>, depending on the spatial pattern of destruction (Fig. 2b). Extinctions occur at less destruction than analytically predicted if uniformly spaced blocks and/or small blocks of habitat are destroyed, and at more if large and/or clumped blocks are destroyed. Destruction of random small patches led to results like those predicted by our analytical model. In all cases, there is biased extinction of abundant, superior competitors.

These spatially explicit versions of our analytical model demonstrate that control of the spatial pattern of habitat destruction can be a significant management tool for minimizing extinctions. Equally as important, they show that habitat destruction can differentially harm abundant, superior competitors that are poor dispersers. This novel and alarming prediction of biased extinction was the central point of our paper. Its robustness, in response to many alternative spatial patterns of destruction, reaffirms our warning that the time-delayed extinction of originally abundant species may be an unexpected consequence of habitat destruction.

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