Introduced species and their missing parasites

Mark E. Torchin*, Kevin D. Lafferty†, Andrew P. Dobson‡, Valerie J. McKenzie* & Armand M. Kuris*

** Marine Science Institute and Department of Ecology, Evolution and Marine Biology, University of California, Santa Barbara, California 93106, USA*
† US Geological Survey, Western Ecological Research Center, c/o Marine Science Institute, University of California, Santa Barbara, California 93106, USA
‡ Department of Ecology and Evolutionary Biology, Princeton University, Princeton, New Jersey 08544-1003, USA

Damage caused by introduced species results from the high population densities and large body sizes that they attain in their new location[1–4]. Escape from the effects of natural enemies is a frequent explanation given for the success of introduced species[5,6]. Because some parasites can reduce host density[7–13] and decrease body size[14], an invader that leaves parasites behind and encounters few new parasites can experience a demographic release and become a pest[4,15]. To test whether introduced species are less parasitized, we have compared the parasites of exotic species in their native and introduced ranges, using 26 host species of molluscs, crustaceans, fishes, birds, mammals, amphibians and reptiles. Here we report that the number of parasite species found in native populations is twice that found in exotic populations. In addition, introduced populations are less heavily parasitized (in terms of percentage infected) than are native populations. Reduced parasitization of introduced species has several causes, including reduced probability of the introduction of parasites with exotic species (or early extinction after host establishment), absence of other required hosts in the new location, and the host-specific limitations of native parasites adapting to new hosts.

On average, 16 parasite species were recorded from native populations of host species. Of these, an average of only three parasite species successfully accompanied an invader to its introduced range. In addition, an average of four new 'native' parasites colonized the introduced host. In sum, introduced populations had roughly half the number of parasite species of native populations. These differences in parasite species richness between introduced and native ranges were significant when species richness was standardized across studies (Figs 1 and 2a), and this effect was independent of sampling effort (Methods).

Introduced populations were also less heavily parasitized in terms of both average prevalence of each possible parasite species (4% in introduced versus 15% in native) and sum of the prevalences (71% in introduced versus 133% in native) of total parasite species per host population (where the prevalence of a parasite is the percentage of hosts that it infects in a population; Figs 1 and 2b, c). Average prevalence on a per-parasite-species basis (that is, parasites with zero prevalence were excluded from the calculation) did not differ between native and introduced populations (Figs 1 and 2d). In other words, parasites that invaded with their hosts achieved as high a prevalence in introduced populations (mean prevalence 28%) as in their native populations (mean prevalence 23%; Wilcoxon signed-rank test, $P_{\text{two-tailed}} = 0.18$). The parasite species left behind tended to be those that were less prevalent (mean prevalence 20%) in native populations as compared with those that did transfer (mean prevalence 27%; Wilcoxon sign-rank test, $P_{\text{one-tailed}} = 0.001$). For example, only the most prevalent of the seven reported trematode species that infect the snail *Batillaria cumingii* in its native range, Japan[16–18], has invaded the west coast of North America (M.E.T., J. Byers and T. Huspeni, manuscript in preparation). Native parasites that colonized introduced host populations (mean prevalence 29%) attained prevalences that were not significantly different from those introduced with the exotic host (mean prevalence 20%; paired $t = 0.31$, $P_{\text{two-tailed}} = 0.76$). Taken together, these findings suggest that there is nothing inherently different about the susceptibility of introduced populations versus native populations. Instead, parasites may be lost or 'filtered out' as a result of the invasion process.

Introduced populations are often derived from relatively small subsets of native populations (and sometimes from uninfected life-history stages), and this reduces the probability of introducing parasites along with a host species. Another potential limitation for the establishment of introduced parasites is that many parasites have complex life cycles requiring more than one host. If suitable hosts for all parasite life-cycle stages are not present, then the parasite will not become established. In addition, host population bottlenecks after introduction may break transmission of those parasites present in the founder population. For example, descendants of 100 adult European starlings, *Sturnus vulgaris*, released in New York City (1890–1891) spread over all regions of the United States[15,19,20]. Of the 44 parasite species that we report from European starlings, a random sample of 100 invading birds should have had

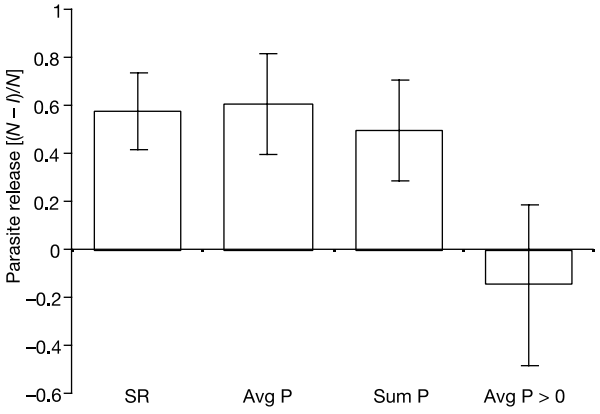

Figure 1 Parasite release experienced by introduced species. This release is represented by the proportion $(N - I)/N$, where N is the value for the native range and I is the value for the introduced range for standardized parasite species richness (SR), average prevalence (Avg P), summed prevalence (Sum P) and average prevalence on a per-parasite-species basis (Avg P > 0; that is, parasites with zero prevalence were excluded from the calculation). This analysis is carried out on all taxa combined (based on data in Fig. 2). Error bars show 95% confidence intervals.

only 28 parasite species (estimated by an iterative resampling of 100 starlings from an infinite population, $n = 10{,}000$ trials, range = 19–37, with 95% of the time values falling between 23 and 33 parasite species). The small size of the founding starling population and lack of appropriate intermediate hosts might have further reduced the 28 expected parasite species to the nine species that we recorded in North American starlings. This is consistent with a previous quantitative study examining the parasites of native and introduced populations of starlings and house sparrows[15].

Individuals arriving after an invader's population density increases could bring additional parasite species, and these would probably experience increased transmission efficiency at the higher host densities. For example, the black rat, *Rattus rattus*, was most certainly introduced repeatedly around the world. It is not surprising that, in our analysis, 38% (one of the highest values) of the rat's native parasites were also recovered from introduced populations.

Along with a complementary study of pathogens on introduced plant species[21], to our knowledge this is the first taxonomically broad quantitative support, using a standardized analytical procedure, for the hypothesis that introduced species lose their native parasites and that their colonization by new parasites does not make up for that loss. Although the hypothesis that release from parasites may contribute to the success of an introduced species in a new environment is rarely examined quantitatively[15], a study of the European shore crab, *Carcinus maenas*, shows that prevalences of parasitic castrators of the shore crab are negatively associated with demographic success (biomass and body size): introduced populations of *C. maenas* were not infected with these parasites and were significantly larger and had a greater biomass as compared with European populations[14]. Our results highlight the importance of evaluating the role of parasites when examining the invasive species problem. More generally, invasions provide several opportunities to assess how parasites regulate host populations. In addition, their absence from introduced pest species suggests that the full potential of biological control to mitigate invasive species has not been explored as yet. □

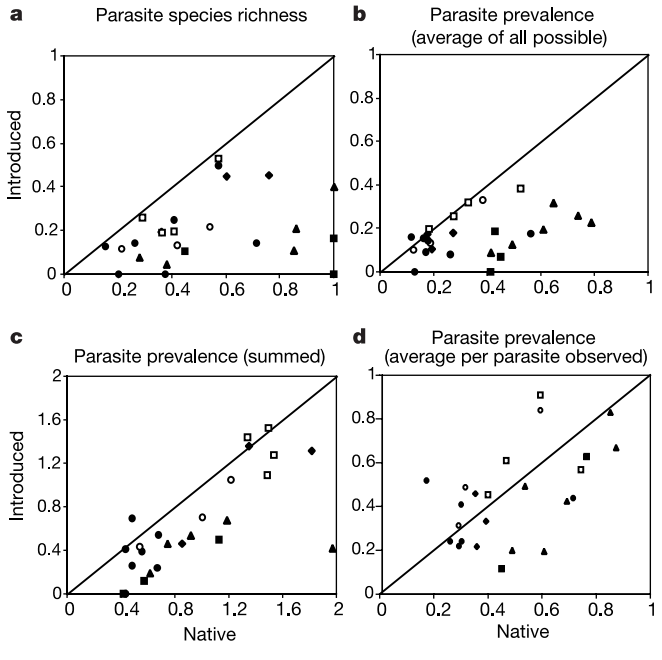

Figure 2 Parasitism in introduced and native populations. **a**, Standardized parasite species richness (estimated as $N_r = N_f/N_n$, where N_f is the number of parasite species found in the study and N_n is the total number of parasite species found in all studies in the native range of a given host species) in the native (*x* axis) and introduced (*y* axis) range. The diagonal line indicates no difference. **b**, Average prevalence (% of hosts infected, including parasites with zero prevalence). **c**, Summed prevalence (sum of prevalences of all parasite species). **d**, Average prevalence on a per-parasite-species basis (that is, parasites with zero prevalence were excluded from the calculation). In **a**–**d**, filled circles, molluscs ($n = 7$); filled squares, crustaceans ($n = 3$); filled triangles, fishes ($n = 6$); open circles, amphibians and reptiles ($n = 3$); filled diamonds, birds ($n = 3$); and open squares, mammals ($n = 4$).

Methods

Measures of parasitism

We analysed parasitological studies of 26 invasive species from seven taxa examined in their natural habitats (see Supplementary Information for full list of species and study selection criteria). To compare parasite measures across the diverse range of host taxa studied (which varied in their parasite richness), we standardized parasite species richness for each population of hosts in each study as a proportion relative to the total number of parasite species found in all studies in the native range of that host species. In addition, we compared both mean prevalence (averaged across parasite species for each host species and the summed prevalence (sum of all parasite species for each host species). The latter measure gives an indication of the unweighted cumulative extent of parasitism (or potential impact of parasitism on a host population) that each host experiences[14]. For each of these metrics, we estimated the proportional parasite release experienced by introduced species as $(N - I)/N$, where N is the value for the native range and I is the value for the introduced range of the above metrics.

Controls for potential confounds

We addressed two potential confounds of this approach. We expected to find a larger number of parasitological studies in native regions than in regions where the host had been introduced—an artefact that might lead to more comprehensive parasite lists in the native ranges and, therefore, could generate a spurious pattern with species richness consistent with our prediction (fortunately, prevalence is generally independent of sample size[22]). However, a detailed analysis of the association between parasite species richness and number of hosts examined (in host's native ranges) showed that there was no significant association ($P > 0.05$) for all but four of the 26 species (*Bufo marinus*, $P = 0.02$; *Lepidodactylus lugubris*, $P = 0.02$; *Perca fluviatilis*, $P = 0.01$; and *Poecilia latipinna*, $P = 0.01$). In addition, in a general linear model with invasion status (either native or introduced) and host species as main effects, sample size was not significantly associated with parasite species richness ($P > 0.05$) and there was a significant effect of invasion status and host species on parasite species richness ($P = 0.0001$ and $P = 0.0001$, respectively). We also considered that a positive association between a species' geographical range and the community of parasites that it supports could confound our comparisons if species had limited introduced ranges relative to their native ranges. We controlled for this by averaging standardized parasite species richness (instead of summing standardized parasite species richness) across sample sites. This enabled us to use sites of relatively similar areas as replicates in native and introduced regions for each host species. In addition, introduced ranges were, on average, five times larger than native ranges, indicating that if such a bias existed, it ran counter to our results.

Received 20 August; accepted 29 November 2002; doi:10.1038/nature01346.

1. Vitousek, P. M. Biological invasions and ecosystem processes: Towards an integration of population biology and ecosystem studies. *Oikos* **57,** 7–13 (1990).
2. Wilcove, D. S., Rothstein, D., Dubow, J., Phillips, A. & Losos, E. Quantifying threats to imperiled species in the United States. *Bioscience* **48,** 607–615 (1998).
3. Ruiz, G. M., Fofonoff, P., Hines, A. H. & Grosholz, E. D. Non-indigenous species as stressors in estuarine and marine communities: Assessing invasion impacts and interactions. *Limnol. Oceanogr.* **44,** 950–972 (1999).
4. Torchin, M. E., Lafferty, K. D. & Kuris, A. M. Parasites and marine invasions. *Parasitology* **124,** S137–S151 (2002).
5. Keane, R. M. & Crawley, M. J. Exotic plant invasions and the enemy release hypothesis. *Trends Ecol. Evol.* **17,** 164–170 (2002).
6. Shea, K. & Chesson, P. Community ecology theory as a framework for biological invasions. *Trends Ecol. Evol.* **17,** 170–176 (2002).
7. Crofton, H. D. A model of host–parasite relationships. *Parasitology* **63,** 343–364 (1971).
8. Anderson, R. M. & May, R. M. Regulation and stability of host–parasite population interactions I. Regulatory processes. *J. Anim. Ecol.* **47,** 219–247 (1978).
9. May, R. M. & Anderson, R. M. Regulation and stability of host–parasite population interactions II. Destabilizing processes. *J. Anim. Ecol.* **47,** 249–267 (1978).
10. Scott, M. E. Regulation of mouse colony abundance by *Heligmosomoides polygyrus* (Nematoda). *Parasitology* **95,** 111–129 (1987).
11. Gulland, F. M. D. The role of nematode parasites in Soay sheep *Ovis aries* L. mortality during a population crash. *Parasitology* **105,** 493–503 (1992).
12. Kuris, A. M. & Lafferty, K. D. Modeling crustacean fisheries: Effects of parasites on management strategies. *Can. J. Fish. Aquat. Sci.* **49,** 327–336 (1992).
13. Hudson, P. J., Dobson, A. P. & Newborn, D. Prevention of population cycles by parasite removal. *Science* **282,** 2256–2258 (1998).
14. Torchin, M. E., Lafferty, K. D. & Kuris, A. M. Release from parasites as natural enemies: Increased performance of a globally introduced marine crab. *Biol. Invas.* **3,** 333–345 (2001).
15. Dobson, A. P. & May, R. M. in *Ecology of Biological Invasions of North America and Hawaii* (eds Mooney, H. A. & Drake, J. A.) 58–76 (Springer, New York, 1986).
16. Shimura, S. & Ito, J. Two new species of marine cercariae from the Japanese intertidal gastropod *Batillaria cumingii* (Crosse). *Jpn. J. Parasitol.* **29,** 369–375 (1980).
17. Rybakov, A. V. & Lukomskaya, O. G. On the life cycle of *Acanthoparyphium macracanthum* sp.n. (Trematoda, Echinostomatidae). **22,** 224–229 (1988).

18. Harada, M. & Suguri, S. Surveys on cercariae in brackish water snails in Kagawa Prefecture, Shikoku, Japan. *Jpn. J. Parasitol.* **38,** 388–391 (1989).
19. Chapman, F. M. *Handbook of Birds from Eastern North America* (Dover, New York, 1966).
20. Hair, J. D. & Forrester, D. J. The helminth parasites of the starling (*Sturnus vulgaris* L.): A checklist and analysis. *Am. Midl. Nat.* **83,** 555–564 (1970).
21. Mitchell, C. E. & Power, A. G. Release of invasive plants from fungal and viral pathogens. *Nature* **421,** 625–627 (2003).
22. Gregory, R. D. & Blackburn, T. M. Parasite prevalence and host sample size. *Parasitol. Today* **7,** 316–318 (1991).

Supplementary Information accompanies the paper on *Nature*'s website (**http://www.nature.com/nature**).

Acknowledgements This work was conducted as part of the Diseases and Conservation Biology Working Group supported by the National Center for Ecological Analysis and Synthesis, a centre funded by the National Science Foundation (NSF), the University of California, and the Santa Barbara campus. We thank S. Altizer, S. Gaines, P. Hudson, H. McCallum, A. W. Miller, C. Mitchell and A. Power for discussion and comments; A. Dove and G. Ruiz for providing data; and L. Mababa for data collection. This research was supported by NSF through the NIH/NSF Ecology of Infectious Disease Program, and by the National Sea Grant College Program, National Oceanic and Atmospheric Administration (NOAA), US Department of Commerce through the California Sea Grant College System, and in part by the California State Resources Agency. The views expressed herein are those of the authors and do not necessarily reflect the views of NOAA or any of its subagencies. The US Government is authorized to reproduce and distribute for governmental purposes.

Competing interests statement The authors declare that they have no competing financial interests.

Correspondence and requests for materials should be addressed to M.E.T. (e-mail: torchin@lifesci.ucsb.edu).

The contribution of *Shaker* K^+ channels to the information capacity of *Drosophila* photoreceptors

Jeremy E. Niven*†, Mikko Vähäsöyrinki†‡, Mika Kauranen‡, Roger C. Hardie§, Mikko Juusola* & Matti Weckström‡

** Physiological Laboratory, University of Cambridge, Cambridge CB2 3EG, UK*
‡ Department of Physical Sciences, Division of Biophysics, University of Oulu, PO Box 3000, 90014 Oulun Yliopisto, Oulu, Finland
§ Department of Anatomy, University of Cambridge, Cambridge CB2 3DY, UK
† These authors contributed equally to this work

An array of rapidly inactivating voltage-gated K^+ channels is distributed throughout the nervous systems of vertebrates and invertebrates[1–5]. Although these channels are thought to regulate the excitability of neurons by attenuating voltage signals, their specific functions are often poorly understood. We studied the role of the prototypical inactivating K^+ conductance, *Shaker*[6,7], in *Drosophila* photoreceptors[8,9] by recording intracellularly from wild-type and *Shaker* mutant photoreceptors. Here we show that loss of the *Shaker* K^+ conductance produces a marked reduction in the signal-to-noise ratio of photoreceptors, generating a 50% decrease in the information capacity of these cells in fully light-adapted conditions. By combining experiments with modelling, we show that the inactivation of *Shaker* K^+ channels amplifies voltage signals and enables photoreceptors to use their voltage range more effectively. Loss of the *Shaker* conductance attenuated the voltage signal and induced a compensatory decrease in impedance. Our results demonstrate the importance of the *Shaker* K^+ conductance for neural coding precision and as a mechanism for selectively amplifying graded signals in neurons, and highlight the effect of compensatory mechanisms on neuronal information processing.

Insect photoreceptors have provided a model system for examining specific molecular mechanisms involved in information processing with graded voltage signals, including signal transduction (the phototransduction cascade)[10] and membrane filtering (the photo-insensitive membrane)[11]. Using these mechanisms, insect photoreceptors must compress the vast spatiotemporal range of light intensities to which they are exposed into voltage responses of limited amplitude and speed. In *Drosophila*, these mechanisms can be studied in relative isolation by patch-clamping dissociated photoreceptors, but *in vitro* photoreceptors do not survive prolonged light stimulation. By contrast, *in vivo* photoreceptors can be recorded intracellularly for more than an hour, and exposed to a full range of light intensities[12] (Fig. 1a). The photo-insensitive membrane of these cells contains three voltage-activated K^+ channels: a *Shaker* channel that generates an A-type current, a slow delayed rectifier and, in some cells, a fast delayed rectifier[9]. The contribution of the *Shaker* K^+ channel and its functional homologues (including vertebrate Kv channels)[2,3] to neuronal function remains unclear, although they are thought to attenuate the amplitude of graded potentials and back-propagated action potentials in dendrites[13–15], to influence the firing frequency of spiking neurons[16] and to determine the reliability of spike propagation[17]. The performance of a photoreceptor in coding a light signal can be described quantitatively by its sensitivity, signal-to-noise ratio and frequency response, allowing specific components of the signalling machinery, including ion channels, to be related to specific aspects of cellular

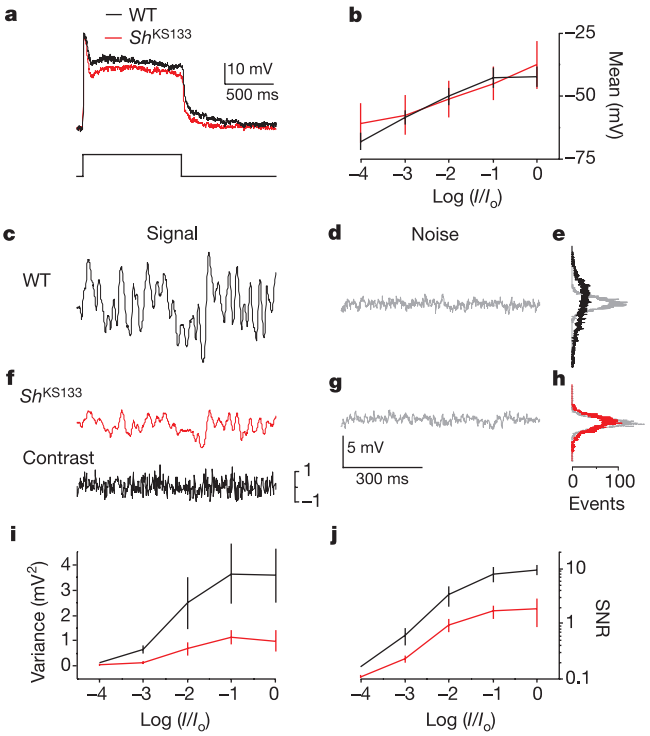

Figure 1 *Shaker* K^+ channels amplify photoreceptor voltage responses. **a**, Responses of wild-type (WT, black) and *Sh*^{KS133} (red) photoreceptors to a 1 s pulse of light. **b**, Mean (±s.e.m.) depolarization of WT (black) and *Sh*^{KS133} (red) photoreceptors to dynamically modulated light contrast at five light intensities ($n = 6$ for each photoreceptor type in all experiments presented here). I, given background light intensity; I_0, maximum background light intensity. **c**, **f**, Waveform of the average voltage signal of WT (black) and *Sh*^{KS133} (red) photoreceptors to noise-modulated light contrast at the highest light intensity. **d**, **g**, Corresponding voltage noise (grey) for the averages presented in **c** and **f**. **e**, **h**, Distributions of the signal (WT, black; *Sh*^{KS133}, red) and noise (grey) for **c**–**g**. **i**, **j**, The signal variance (**i**) and the signal-to-noise ratio (SNR, **j**) for WT (black) and *Sh*^{KS133} (red) photoreceptors at each adapting-light background.