of light waves able to propagate freely in the structure, for the hexagonal array of voids (presumably water-filled) in a chitin matrix. This is shown in Fig. 1b for $E_{||}$ polarization, indicating that the region of enhanced reflectance in the red for the spine corresponds exactly to a partial photonic bandgap of the hexagonal structure (in the range Γ–M) — a circumstance in which light cannot propagate in a narrow range of wavelengths in the structure. Note that for $H_{||}$ polarization of incident radiation with the magnetic vector perpendicular to the plane of Fig. 1a, reflectance is similarly enhanced in the red, although there is not a clear-cut partial bandgap.

We have seen that the sea mouse achieves brilliant narrow-band coloration of its spines through a remarkable piece of photonic engineering. The regularity of the structure shown in Fig. 1a and the strong narrow-band reflectance shown in Fig. 1c suggest that growing optical filters by molecular self-assembly is a technological goal worth pursuing. These structures may have application in photonic communications, where there is much interest in fabricating photonic crystal fibres[8] with similar morphology to that shown in Fig. 1a in order to improve bandwidth and nonlinear properties.

Andrew R. Parker*, Ross C. McPhedran†, David R. McKenzie†, Lindsay C. Botten‡, Nicolae-Alexandru P. Nicorovici†

**Department of Zoology, University of Oxford, South Parks Road, Oxford OX1 3PS, UK*
†*School of Physics, University of Sydney, New South Wales 2006, Australia*
e-mail: ross@physics.usyd.edu.au
‡*School of Mathematical Sciences, University of Technology, Sydney, New South Wales 2007, Australia*

1. Parker, A. R. in *Functional Morphology of the Invertebrate Skeleton* (ed. Savazzi, E.) 65–90 (Wiley, Chichester, 1999).
2. Land, M. F. *Progr. Biophys. Mol. Biol.* **24,** 75–106 (1972).
3. Parker, A. R. *Proc. R. Soc. Lond. B* **262,** 349–355 (1995).
4. Vukusic, P. *et al. Proc. R. Soc. Lond. B* **266,** 1403–1411 (1999).
5. Rouse, G. in *Invertebrate Zoology* (ed. Anderson, D. T.) 174–203 (Oxford Univ. Press, Melbourne, 1998).
6. Atkins, E. D. T., Dlugosz, J. & Foord, S. *Int. J. Biol. Macromol.* **1,** 29–32 (1979).
7. McPhedran, R. C. *et al. Aust. J. Phys.* **52,** 791–809 (1999).
8. Knight, J. C. *et al. Science* **282,** 1476–1478 (1998).

Vertical mixing in the ocean

The thermohaline circulation of the ocean results primarily from downwelling at sites in the Nordic and Labrador Seas and upwelling throughout the rest of the ocean. The latter is often described as being due to breaking internal waves. Here we reconcile the difference between theoretical and observed estimates of vertical mixing in the deep ocean by presenting a revised view of the thermohaline circulation, which allows for additional upwelling in the Southern Ocean and the separation of the North Atlantic Deep Water cell from the Antarctic Bottom Water cell. The changes also mean that much less wind and tidal energy needs to be dissipated in the deep ocean than was originally thought.

Previous calculations of vertical mixing based on the stratification of the deep ocean[1,2] assumed that a flux of 25 or 30 sverdrups (Sv) of water, made up of both deep and bottom waters, is injected at depths of about 4,000 metres and mixed upwards to depths near 1,000 m by turbulent mixing. Both reports conclude that the spatially averaged diapycnal (cross-density surface) mixing coefficient is 10^{-4} $m^2 s^{-1}$.

However, observations of turbulence[3] and dye diffusion[4] in the deep ocean indicate that there exists a background diapycnal diffusivity of only 10^{-5} $m^2 s^{-1}$, although much larger values are found in localized regions near rough topography[5]. The background value is consistent with mixing due to the background internal wave field, and the larger values are consistent with extra internal waves due to the interaction of currents with topography.

But it is not obvious that the latter is enough to raise diffusivity by an order of magnitude when averaged over the whole ocean. The extra power required to do this is also large[6]. If the efficiency is 20%, which is normally considered a maximum for the final stage of breaking internal waves, then the power required is 2.1 terawatts. This is just possible, given current estimates of the energy input from the wind and tides, but this figure does not allow for losses at other stages in the conversion process.

A contrasting view of the thermohaline circulation has come from low-resolution[7] and high-resolution[8] computer model studies of the ocean circulation. These show that between 9 and 12 Sv of deep water is brought to the surface by Ekman suction in the Southern Ocean. This is driven northwards in the surface Ekman layer and is reduced in density primarily by surface freshening. The model results also emphasize earlier observations[9] that in the primary regions of bottom-water formation around Antarctica, the near-surface water masses have the same density as North Atlantic deep water. It is therefore not necessary for the bottom water to be mixed through the whole depth of the water column, only up to the level of the deep waters.

Using this new view of the thermohaline circulation, we need only consider the vertical mixing of the main deep-water mass, North Atlantic Deep Water, whose flux is estimated to lie between 14 and 17 Sv (ref. 10). Taking the larger of these two values and the smaller of the two model-based estimates of upwelling leaves a maximum of 8 Sv to be mixed vertically within the ocean.

The 10^{-5} $m^2 s^{-1}$ background term can upwell 3 Sv, leaving 5 Sv to be upwelled by localized regions of intense mixing. If this view is correct, then the vertical mixing coefficient, averaged over the whole ocean, is less than 3×10^{-5} $m^2 s^{-1}$ and, assuming 20% efficiency, the total amount of extra energy required is less than 0.6 terawatts.

The revised values are consistent with existing observations of mixing within the ocean. They also emphasize again the importance of the Southern Ocean and imply that although further research is needed on the localized mixing in the deep ocean, such mixing does not control the thermohaline circulation.

D. J. Webb*, N. Suginohara†
**Southampton Oceanography Centre, Empress Dock, Southampton SO14 3ZH, UK*
e-mail: david.webb@soc.soton.ac.uk
†*Center for Climate System Research, University of Tokyo, 4-6-1 Komaba, Meguro-ku, Tokyo 153-8904, Japan*

1. Munk, W. H. *Deep-Sea Res.* **13,** 707–730 (1966).
2. Munk, W. H. & Wunsch, C. *Deep-Sea Res.* **45,** 1977–2010 (1998).
3. Gregg, M. C. *J. Geophys. Res.* **94,** 9686–9698 (1989).
4. Ledwell, J. R., Watson, A. J. & Law, C. S. *J. Geophys. Res.* **103,** 21499–21529 (1998).
5. Polzin, K. *et al. Science* **276,** 93–96 (1997).
6. Wunsch, C. *Nature* **405,** 743–744 (2000).
7. Toggweiler, J. R. & Samuels, B. *J. Phys. Oceanogr.* **28,** 1832–1852 (1998).
8. Döös, K. & Coward, A. C. *Int. WOCE Newslett.* **27,** 3–4 (1997).
9. Foster, T. D. & Carmack, E. C. *Deep-Sea Res.* **23,** 301–317 (1976).
10. Schmitz, W. J. *Rev. Geophys.* **33,** 151–173 (1995).

How wild are wild mammals?

In bacteria associated with humans, antimicrobial resistance is common, both in clinical isolates and in the less-studied commensal flora, and it is thought that commensal and environmental bacteria might be a hidden reservoir of resistance. Gilliver *et al.* have reported that resistance is also prevalent in faecal bacteria from wild rodents living in northwest England[1]. Here we test the faeces of moose, deer and vole in Finland and find an almost complete absence of resistance in enterobacteria. Resistance is thus not a universal property of enterobacterial populations, but may be a result of the human use of antibiotics.

Bacterial resistance to antimicrobial agents has become a serious problem in modern medicine and a debated evolutionary question[2]. The use — and misuse — of antibiotics is generally blamed, but it has also been claimed that there must be other reasons for the increase in resistance[3]. This question is important: if resistance increases independently of antibiotic use, restrictive policies would be unnecessary. One way to test the effect of human activities is to compare the resistance frequencies of popula-

brief communications

Table 1 Antimicrobial resistance in enterobacteria from the faeces of wild moose, deer and vole

Bacteria (no. of isolates)	*Percentage resistant (MIC$_{50}$; MIC$_{90}$)*						
	AMP ≥32*	AMC ≥32/16	CEX ≥32	CXM ≥32	CTX ≥64	ATM ≥32	IPM ≥16
Escherichia coli (98)	0 (4; 8)	0 (4; 4)	0 (8; 8)	0 (4; 4)	0 (0.06; 0.06)	0 (≤0.06; 0.1)	0 (≤0.2; ≤0.2)
Enterobacter agglomerans group (48)	NA (8; 16)	NA (4; 4)	NA (8; 16)	4 (4; 4)	0 (0.06; 0.1)	0 (≤0.06; 0.1)	0 (0.5; 1)
Yersinia spp. (29)	NA (32; 64)	NA (32; >64)	NA (>64; >64)	7 (4; 4)	0 (0.2; 0.5)	0 (0.5; 0.5)	0 (0.5; 1)
Serratia spp. (11)	NA (32; 128)	NA (8; >64)	NA (>64; >64)	82 (64; >64)	0 (0.5; 1)	0 (0.5; 0.5)	0 (0.5; 0.5)

Bacteria (no. of isolates)	*Percentage resistant (MIC$_{50}$; MIC$_{90}$)*							
	GEN ≥16	STR (≥32)	NAL ≥32	CIP ≥4	CHL ≥32	TET ≥16	TMP ≥16	SUL ≥512
Escherichia coli (98)	0 (0.5; 0.5)	1 (4; 4)	0 (2; 4)	0 (0.03; 0.06)	0 (4; 8)	0 (1; 2)	0 (0.2; 0.5)	0 (16; 32)
Enterobacter agglomerans group (48)	0 (0.2; 0.2)	0 (2; 2)	0 (1; 4)	0 (0.03; 0.1)	0 (≤2; ≤2)	0 (1; 1)	0 (≤0.06; 0.1)	0 (8; 16)
Yersinia spp. (29)	0 (0.2; 0.5)	0 (2; 4)	0 (1; 1)	0 (0.01; 0.03)	0 (8; 8)	0 (2; 2)	0 (1; 2)	0 (16; 64)
Serratia spp. (11)	0 (0.2; 0.5)	0 (2; 4)	0 (2; 2)	0 (0.06; 0.1)	0 (4; 16)	NA (2; 64)	0 (0.2; 0.5)	0 (16; 32)

MIC$_{50}$ and MIC$_{90}$ are the antibiotic concentrations (mg l^{-1}) at which 50 and 90%, respectively, of the tested population is inhibited from growing. AMP, ampicillin; AMC, amoxycillin/clavulanic acid; CEX, cephalothin; CXM, cefuroxime; CTX, cefotaxime; ATM, aztreonam; IPM, imipenem; GEN, gentamicin; STR, streptomycin; NAL, nalidixic acid; CIP, ciprofloxacin; CHL, chloramphenicol; TET, tetracycline; TMP, trimethoprim; SUL, sulphamethoxazole. NA, not applicable: most strains or species intrinsically resistant.
*Resistance breakpoint (mg l^{-1}) according to the National Committee for Clinical Laboratory Standards.

tions of the same bacterial species that have or have not been exposed to humans.

Fresh faeces from newly felled moose (*Alces alces*; $n = 16$) and white-tailed deer (*Odocoileus virginianus*; $n = 7$) were collected in the autumn of 1999 by hunters in two areas of Uusimaa, southern Finland. Faecal pellets were prepared from bank voles (*Clethrionomys glareolus*, $n = 23$) trapped in Ostrobothnia, western Finland[4]; these had been stored whole at −20° C for less than one year. Five bacterial colonies per sample, representing all different colonial morphologies present, were identified to at least genus level, and the minimum inhibitory concentrations (MICs) of 15 antibiotics were determined, as previously described[5].

The ungulate faecal flora was similar to human flora, with *Escherichia coli* as the main species. In vole faeces, *Enterobacter agglomerans* and *Yersinia* spp. dominated. Results are given only for genera represented by more than four isolates. The only resistance found was to cefuroxime (Table 1) and to streptomycin (in one sample of *E. coli*; this could be transferred by conjugation[6] to *E. coli* C600). Most of the cefuroxime resistance was, as judged from MIC profiles, most likely caused by a class A (Bush group 2e) cefuroximase similar to the chromosomal *Proteus vulgaris* enzyme[7], and was thus most probably indigenous. It was found not to be caused by the most common transferable class-A β-lactamases: the cerufoxime-resistant strains were tested by using the polymerase chain reaction for the presence of TEM and SHV[8], but only one strain contained a TEM-type enzyme and none carried SHV.

These results disagree with those from a study of enterobacteria from wild English rodents, where extremely high resistance was found[1]. The English study questions the usefulness of restricting antibiotic use, as these rodents are presumed to have had no contact with antibiotics. However, the overall load from antibiotic use in England is larger than in Finland: the mean number of inhabitants per square kilometre in Finland is 17, compared to 378 in England (see www.statistics.gov.uk and www.stat.fi); also, the load from agriculture is less in Finland — there are, for example, ten times fewer cattle and five times fewer pigs than in the UK (see www.stat.fi and www.maff.gov.uk). Since 1996, the use of antibiotic additives in animal feed has gradually been abandoned, but occasional contact cannot be ruled out. Our sampled populations almost certainly represent wild animal populations better.

In faecal flora, *E. coli* is the species showing the most resistance (resistance to streptomycin, to sulphamethoxazole, and to tetracycline is highest at 14–18%, even in healthy people)[5]. Resistance is known to increase with increased exposure to antibiotics and during hospitalization[5]. Our finding of an *E. coli* population that has never been exposed to humans and which is free of resistance to antibiotics strongly suggests that the widespread resistance found in all *E. coli* populations associated with humans must be caused by human activities. Antibiotic restrictions whenever feasible are still very much on the agenda.

Monica Österblad*, Kai Norrdahl†, Erkki Korpimäki†, Pentti Huovinen*
*Antimicrobial Research Laboratory, National Public Health Institute, Kiinamyllynkatu 13, FIN-20520 Turku, Finland
†Section of Ecology, Department of Biology, University of Turku, FIN-20014 Turku, Finland

1. Gilliver, M., Bennett, M., Begon, M., Hazel, S. & Hart, C. *Nature* **401**, 233–234 (1999).
2. Baquero, F. & Blázquez, J. *Trends Ecol. Evol.* **12**, 482–487 (1997).
3. Levy, S. B., Marshall, B., Schlueederberg, S., Rowse, D. & Davis, J. *Antimicrob. Agents Chemother.* **32**, 1801–1806 (1988).
4. Korpimäki, E. & Norrdahl, K. *Ecology* **79**, 2448–2455 (1998).
5. Österblad, M. *et al. Antimicrob. Agents Chemother.* **44**, 1479–1484 (2000).
6. Murray, B. E. & Hodel-Christian, S. L. in *Antibiotics in Laboratory Medicine* (ed. Lorian, V.) 453–501 (Williams & Wilkins, Baltimore, 1996).
7. Livermore, D. M. & Williams, J. D. in *Antibiotics in Laboratory Medicine* (ed. Lorian, V.) 502–578 (Williams & Wilkins, Baltimore, 1996).
8. Manninen, R. *et al. Clin. Microbiol. Infect.* **3**, 408–413 (1997).

Gilliver et al. *reply* — The study by Österblad *et al.* confirms the importance of understanding the role of commensal bacteria, particularly in wildlife, in the ecology of antibiotic resistance. The two studies combined suggest that the gut flora of wildlife populations with very little or no contact with either humans or anthropogenic antibiotics (Österblad *et al.*'s study) may have negligible levels of antibiotic resistance, whereas wildlife populations living in closer proximity to humans but still with no known direct contact with anthropogenic antibiotics (our study) may have much higher levels of antibiotic resistance.

These conclusions fit with earlier findings of a higher prevalence of antibiotic resistance among baboons living close to humans than in baboons in more isolated populations[1]. Questions that still need to be addressed concern the extent and frequency of antibiotic exposure necessary to generate significant resistance, what determines the dynamics of decline in resistance following restrictions in antibiotic use, and the nature and extent of any reservoir of antibiotic resistance that may exist in natural environments and which could undermine future attempts to manage resistance.

These questions can only be resolved by thorough spatial and temporal mapping of antibiotic resistance in natural environments. We inferred from our study that it would be unwise to assume that resistance would decline significantly as a consequence of restricted use of antibiotics. This suggestion still holds, because resistance has been maintained for over three years at our study site, over several generations of rodents, without any obvious exposure to antibiotics.

We agree with Österblad *et al.* that antibiotic restrictions should still be very much on the agenda, but that agenda must include concerted attempts to understand what the consequences of restrictions are likely to be.

Moira A. Gilliver, Malcom Bennett, Michael Begon, Sarah M. Hazel, C. Anthony Hart
Centre for Comparative Infectious Diseases, University of Liverpool, PO Box 147, Liverpool L69 3BX, UK

1. Routman, E., Miller, R. D., Phillips-Conroy, J. & Hartl, D. L. *Appl. Environ. Microbiol.* **50**, 749–754 (1985).