# Mixing with latitude

Chris Garrett

Measurements over a range of latitudes support a theory relating ocean turbulence to internal waves. The upshot should be easier mapping of ocean mixing, and eventually better climate models.

Anyone who looks at the sky can see that clouds have a big effect on weather and climate. But global computer models cannot deal explicitly with individual clouds, let alone the water droplets in them. Phenomena that mix the ocean are likewise important but too small to be resolved in models of ocean circulation. These 'subgrid-scale' processes need to be mapped, in both the atmosphere and the ocean. They also need to be understood so that their effects can be 'parametrized' — that is, represented in terms of larger-scale features that are dealt with explicitly in the models. Only in this way can we correctly allow for changes in such processes and their effects in a changing world. As they describe on page 513, Gregg and colleagues[1] have produced an exciting advance in ideas about how both mapping and parametrization in the ocean might be accomplished.

The overall oceanographic context for their observations is as follows. Below a uniform surface layer that is typically some tens of metres thick, and directly stirred by wind and surface cooling, the ocean is stratified according to density, with lighter water above denser water. This gives it stability, which from time to time is disrupted by bursts of turbulence and mixing. The mixing helps to redistribute ocean properties, influencing circulation and heat transport, and hence climate[2]. Mixing also redistributes nutrients and dissolved gases, thus affecting biological productivity in the oceans.

Most of the turbulence and mixing is caused by an 'overturning instability', which has a vertical scale that varies from a fraction of a metre to many metres and is associated with the vertical gradient, or shear, of transient horizontal currents. These currents are a manifestation of internal waves (Fig. 1) — fluctuating motions that resemble waves at the ocean surface in relying on restoring buoyancy forces associated with a vertical change in density, but having much longer periods (typically tens of minutes to many hours). Some of the internal-wave energy originates as internal tides[3], which are waves of tidal period generated by the main tidal currents flowing over bumps on the sea floor. Another source is the 'inertial' waves that are generated as the ocean responds to currents driven at the sea surface by the winds of fast-moving storms[4]. These waves have a period, related to the Coriolis force resulting from Earth's rotation, of 12 hours at the poles and much longer at low latitudes.

As the internal waves propagate into the ocean interior, some energy spreads into waves with other periods and generally smaller vertical scales, causing a greater vertical shear and hence more likelihood of wave-breaking, turbulence and mixing (Fig. 1). This cascade to smaller scales is reminiscent of what occurs in turbulent motions in unstratified water, with big eddies tearing one another apart and giving rise to ever smaller eddies, and it is also described by nonlinearities in the governing equations. The processes are, however, subtle and not fully understood. Nonetheless, a plausible model[5,6] suggests that the cascade is not only more vigorous in places where internal waves are more energetic, but is also a function of latitude, with a slower cascade as one approaches the Equator.

It is this prediction that is so nicely confirmed by Gregg *et al.*[1]. They have simultaneously measured the energy level of internal waves (as largely represented by the shear of currents on a vertical scale of the order of ten metres) and the turbulent mixing at scales of a centimetre or less. They reinforce their earlier confirmation[7], from a limited range of latitudes, that turbulence depends on the internal-wave energy, but they now have enough data from low latitudes to show that, as predicted, a given energy level at low latitudes causes much less mixing.

This confirmation of theory is as if a cloud physicist had shown that it is no longer necessary to study individual droplets, but only the larger-scale features of clouds. In the ocean we may no longer need to undertake the technically demanding measurement of the turbulence itself, but can predict it from motions at much larger scales. To be sure, the theoretical underpinnings of the formula are not completely robust, and it is known to break down in some locations[8]. We need more theoretical studies[6] and numerical simulations[9] of the energy cascade of internal waves. But it does seem that we have a good provisional basis for indirectly mapping the global distribution of ocean mixing, and of any seasonal or longer-term variations.

Such mapping would still require vertical profiles of horizontal current. This is a more sophisticated measurement than is routinely made. Alternatively, improvements in measuring and analysing profiles of temperature and salinity alone, from ships or profiling floats[10], could provide a simpler approach based on analysis of, say, the overturning instability itself[11].

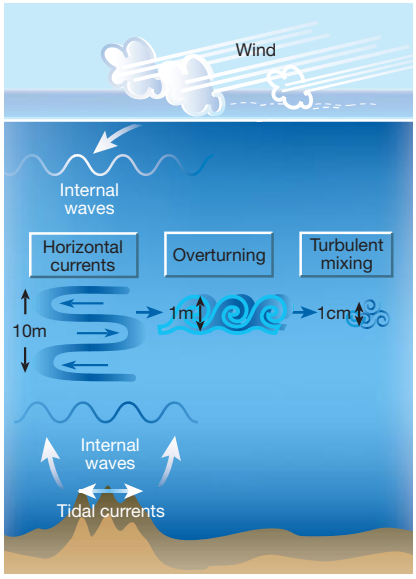


Figure 1 **Internal waves and scales of ocean mixing. Waves produced at the sea surface and sea floor propagate into the ocean interior, generating small-scale mixing processes that affect circulation, heat transport and nutrient distribution (and so biological productivity). In confirming theory, two conclusions emerge from the observations of Gregg and colleagues[1]. First, that the vertical gradient of the horizontal currents with vertical scales of tens of metres is a good guide to overturning at a scale of a metre or less and turbulent mixing at the centimetre scale. Second, that the connection is strongly dependent on latitude. Note that the scale of this graphic is distorted to show processes at scales of metres and less in an ocean that is about 4,000 m deep.**

Even with a confirmed parametrization of mixing, predictive models of ocean circulation will still need information on the internal-wave field, probably requiring a model to run in parallel. In a windier world, for example, internal waves might be more energetic but lose more of their energy closer to their source, changing the strength and spatial patterns of mixing rates. This kind of feedback on circulation and climate will have to be allowed for. Finally, although mixing from internal-wave-breaking is a key

phenomenon in the deep ocean, particularly over rough topography, it may actually be less important for changing water properties in the top kilometre or so — there, some combination of air–sea interaction and stirring by eddies, with scales of tens or hundreds of kilometres[12], may dominate.

There is still much to do before we can claim to have simple operational techniques for monitoring ocean mixing rates, and a full understanding of the mechanisms and effects of mixing. But the connections between motions with scales from millimetres to thousands of kilometres are becoming clearer, and we are closer to having the parametrization of small-scale processes that is needed if models are to have predictive capability. ■

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Microbiology

# Gut defence

Tomas Ganz

The severity of salmonella infections depends in part on how effectively the invaders are destroyed. Incisive experiments now show that host defence in the intestine centres on the aptly named defensins.

Cells that are engaged in the antimicrobial defence of mammals and birds produce defensins, a family of structurally related antimicrobial molecules[1,2]. The production of similar small proteins is also often induced by infections in invertebrates and plants[3,4]. Because of their abundance in infected tissues and their ability to kill a variety of microbes under laboratory conditions, defensins are thought to function as natural antibiotics. Until now, however, the evidence for their contribution to antimicrobial defence in living mammals was entirely circumstantial. That changes with the appearance of the paper by Salzman *et al.* on page 522 of this issue[5]. The authors report that they performed a genetic transplant of human defensin 5 (HD-5) into mice and observed a dramatic improvement in the resistance of the mice to intestinal infection with *Salmonella typhimurium*.

Salmonella bacteria cause several types of disease in humans and animals[6]. A diarrhoeal illness contracted by eating contaminated food is the most common consequence of *S. typhimurium* infection in humans. In this disease, the bacteria are confined to the lumen and absorptive surface of the small intestine, where they cause inflammation but do not spread through the blood to other organs. However, when *S. typhimurium* infects mice, or the related bacterium *S. typhi* infects humans, they cause typhoid fever, a much more serious illness, in which the bacteria spread from the intestine to other organs. The severity of the disease depends to a large extent on the ability of the infected host to restrict the multiplication of the bacteria and to prevent them from penetrating the intestinal wall.

The intestinal tract is normally inhabited by a variety of resident bacteria growing at low density in the small intestine but abundantly in the colon. Paneth cells[7] located in crypts, tiny pits throughout the small intestine (Fig. 1), release defensins and other antimicrobial substances and contribute to the ability of fluid in the small intestine to prevent the growth of invading bacteria. Previous studies had shown that *S. typhimurium* is much more sensitive to human defensin HD-5 than to the defensins produced by mouse Paneth cells.

Salzman *et al.*[5] reasoned that if defensins function as natural antibiotics, then transgenic mice constructed to make HD-5 in their Paneth cells (HD-5 mice) should have increased resistance to infection with *S. typhimurium*. They orally infected normal and HD-5 mice with these bacteria, and observed that all HD-5 mice survived infection with doses of bacteria that killed all the normal mice within 2 days. Moreover, by 24 hours after infection, the bacterial counts in the faeces or intestines of HD-5 mice were a thousand times lower than those in normal mice, and the spread to other organs was also greatly decreased. The protective effect of HD-5 was detectable very early, with obvious differences between the HD-5 and normal mice only 6–12 hours after infection. The site of HD-5 activity was clearly in the intestine,

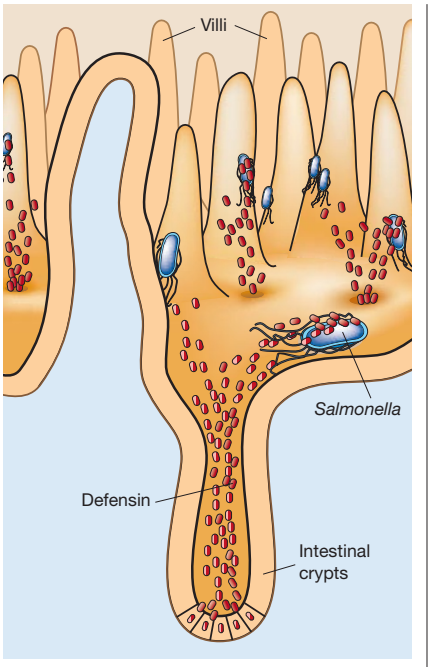


Figure 1 **Bacterial invasion and the gut response. Salmonella bacteria attach to villi of the intestine but are attacked by defensins secreted from Paneth cells located in the intestinal crypts. Defensins are stored in large cellular granules, and are activated during or after release by the removal of a 'propiece' by trypsin, an enzyme released by Paneth cells. In the new work, Salzman *et al.*[5] provide solid experimental evidence that defensins act as species-specific antibiotics *in vivo*. (Redrawn from artwork by Dave Schumick, Cleveland Clinic Foundation.)**

because there was no difference in the survival of HD-5 mice and normal mice when the bacteria were injected into the abdominal cavity, bypassing the intestinal secretions.

Finally, the authors also documented that the concentrations of human defensin in HD-5 mice were comparable to the concentrations of native mouse defensins, so that the observed effects were not due to unrealistically high levels of human defensins in the transgenic animals. The presence of comparable amounts of normal mouse Paneth-cell defensins, and the rapid time course of the effect of HD-5, also suggest that HD-5 acts as an antibiotic and not by indirect mechanisms such as increased recruitment of host defence cells to the infection area or priming of other immunological responses. More detailed examination of HD-5's mechanism of action in this setting should clarify this point.

Several previous studies support the idea that defensins and other antimicrobial peptides contribute to antibacterial defence in mammals. Mice that lack matrilysin, an enzyme that activates several mouse intestinal defensins, also show impaired resistance to intestinal infections[8]. However, matrilysin