A key issue for hydrogen storage materials is that the hydrogenation and dehydrogenation processes should be sufficiently fast. The formation reaction of bulk hydrates is generally slow — owing to diffusion limitations — taking days to weeks. Lee *et al.* have taken a first step towards speeding up this process by dispersing the hydrate on silica beads with a large surface area. This reduces the times for loading and discharge to a matter of hours.

Other hydrate structures are also known, so it may even be possible to discover stable hydrogen hydrates with higher storage capacities or to find more efficient stabilizers than THF. The sII hydrates investigated by Lee *et al.* have among the highest free volumes in known clathrate phases, but the compositional tuning that they make possible using auxiliary guest species may lead to the discovery of phases with even more attractive properties. ■

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Environmental science

Germ theory for ailing corals

Stephen R. Palumbi

Human activities damage coral reef ecosystems. Application of the 'germ theory', proposed more than a century ago for human diseases, could foster action on global environmental ailments such as this.

In 1876, Robert Koch was struggling to convince the world that germs cause disease. Today, environmental degradation is a pervasive planetary disease, but the causes remain shrouded in the same popular murk that made diseases mysterious before the work of Koch and Louis Pasteur. For environmental issues, such as the decline of coral reefs, sceptics demand detailed evidence — we must know the exact cause and show that any proposed cures will work. This is a tall order. Writing in *Science*, however, Pandolfi *et al.*[1] chart the decline of reefs using data from previous work[2] and some new evidence, and provide a prescription to begin a planet-wide cure.

Some demand that we rigorously prove the cause before acting. Koch faced this dilemma in the form of scepticism about the germ theory of disease. He responded by deploying the following postulates[3] for showing that particular bacteria cause a disease:

- The bacteria must be present in every case of the disease.
- The bacteria must be isolated from the host with the disease and grown in pure culture.
- The specific disease must be reproduced when a pure culture of the bacteria is inoculated into a healthy, susceptible host.
- The bacteria must be recoverable from the experimentally infected host.

These postulates do not always apply, even in medicine[3], but they emphasize the critical role of correlational[1,4] and experimental data[2,3] in untangling complex problems. These approaches are nearly ubiquitous in the environmental sciences and an environmental version of these rules might be termed 'planetary postulates':

- The cause must be present in every case of the environmental condition.
- The cause must be isolated and known to act by itself to harm the ecosystem.
- The specific condition must be reproduced when the cause is experimentally introduced into the environment.
- The cause must again be verifiably present and active in the affected environment.

Action to address serious environmental issues need not wait until all of these postulates are fulfilled. However, Pandolfi *et al.* meet most of them by showing that reef degradation is a disease of human overuse, as opposed to part of natural cycles. First, fully degraded reefs are always affected by people, because reefs with easier access or longer habitation are more likely to be degraded[1]. Second, some human activities harm reefs (Fig. 1) — dynamite fishing, dumping sewage or sediment, dredging corals for cement and unsustainable fishing are well documented[4]. When people are excluded from reefs, fish and invertebrates tend to recover and reef decline tends to reverse[5]. Third, when people are reintroduced to reefs — usually through the collapse of marine reserve enforcement — the condition of over-exploitation returns[6]. Fourth, once people return to reefs, they can be demonstrably shown to be harming them again[7].

Although the planetary postulates may be met for coral reef damage, human impacts are complex. The culprit may be sedimentation from terrestrial runoff[8], excessive nutrient input from sewage[9], the introduction of foreign species or disease-causing organisms[10], overfishing[2] or global warming[11]. Analysing any one of these causes in isolation would not survive the strictures of the planetary postulates. But combining all of them, and their ultimate — human — driver seems to fulfil the postulates.

The value of this exercise should be that

MASA USHIODA/IMAGE QUEST

Figure 1 **Net effect — one manifestation of deleterious human impact on coral reefs. Abandoned nets trap fish and diving birds, and abrade the coral.**

diagnosis paves the way for a solution. Pandolfi *et al.* do not know exactly how to cure coral reefs of human overexploitation — no more than Koch, labouring in advance of the discovery of antibiotics, knew how to cure tuberculosis. Instead, they treat the problem as Koch might have done: keep the patients alive by alleviating the symptoms, and reduce exposure to the problem so that they can cure themselves.

To alleviate the symptoms, the most severe impacts on reefs must be reduced. This may mean investment in sewage treatment and abatement of run-off from land; reducing fishing intensity; establishing fully protected reserves; or building buffer zones with limited development near reefs. In the long term it may mean reducing global warming. In the meantime, to keep the patient alive, it is necessary to establish large coral reef parks, such as the Great Barrier Reef Marine Park, as well as small reserves to act as local seed sources.

Finally, the patient needs to heal. Here the sea is ready to help. Marine species have prodigious reproductive abilities — many female fish and invertebrates produce millions of eggs a year. Some corals are virtually immortal and can fragment to produce hundreds of clonal offspring. Movement of tiny larvae can transport species from healthy to damaged reefs. Reefs have recovered from hurricanes, floods, tsunamis and volcanoes. They can also recover from us.

Critics will say that Pandolfi and colleagues' proposed solutions are not socially or technically feasible. Koch faced similar problems. The requirements of his postulates outstripped the abilities of the microbiology and medicine of his day. But Koch did not worry that he could not culture every disease organism, nor that disease germs could not necessarily be killed once identified. Then, as now, progress is attained in steps, and the identification of causes is a key step.

Pandolfi *et al.* make a final plea — don't demand a perfect solution. As an initial goal, it is enough that the health of a reef stops declining and begins to improve. The next step is urgently to develop the science of global ecology to provide a toolbox of more lasting solutions. ■

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Developmental biology

Reproduction in clusters

François Spitz and Denis Duboule

Homeobox genes have some quirky features: they huddle together and tend to be expressed in the order that they appear in their cluster. A new cluster, specific to reproductive development, has now been discovered.

The survival of animal species depends upon the proper development of germ cells: oocytes and sperm. In mammals, this process is tightly coordinated with the development of non-reproductive cells nearby, such as Sertoli cells, which nourish developing sperm cells[1]. Changes in the delicate interactions between reproductive and non-reproductive cells are often a source of decreased fertility, so the appropriate regulation in time and space of the underlying genetic determinants must be essential. Writing in *Cell*, MacLean *et al.*[2] describe how they identified a new set of these genetic determinants — a cluster of homeobox genes that are expressed during the development of germ cells. The clustering of these genes may help to determine their spatial and temporal regulation.

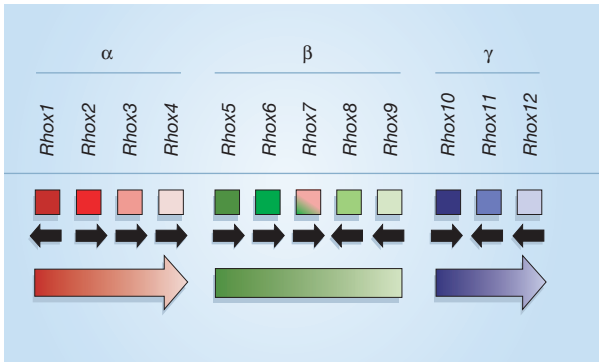

Figure 1 **Sperm and gene clusters. MacLean *et al.*[2] have identified the Rhox gene cluster on the mouse X chromosome. This cluster is organized into three subclusters, α, β and γ. The genes of each subcluster show collinear expression during sperm differentiation — they are expressed in the order that they occur on the chromosome. They are also expressed to different degrees and, in the α and γ subclusters, at different times (graded coloured arrows), such that the first genes in each subcluster are expressed earlier and to a higher maximal level than the next ones. Black arrows denote the direction of gene transcription. The pink shading for the *Rhox7* gene shows that, in sequence terms, it is more closely related to the α than the β subcluster.**

Homeobox genes are so called because they contain a characteristic DNA sequence, the homeobox. They are found in all animals, from mammals to fruitflies, and they are essential for numerous aspects of embryonic development. Until now, however, no homeobox gene cluster had been shown to be directly required for oocyte or sperm maturation, and none was known to be located on the sex chromosomes.

But MacLean and colleagues[2] have identified a previously unnoticed group of 12 homeobox genes within a small region on the mouse X chromosome. The authors call these genes 'reproductive homeobox X-linked' (Rhox) genes, and show that they define a new homeobox subfamily. Further sequence analyses reveal that the genes fall into three subgroups, which also correspond to their physical alignment along the X chromosome and therefore define three genomic subclusters (α, β and γ; Fig. 1). The only exception is the *Rhox7* gene, which is located in the β subcluster but whose sequence has more in common with the α subcluster. MacLean *et al.* have also found that the Rhox genes are specifically expressed in male and female reproductive tissues and in the placenta. Within the testis, they are mainly expressed in Sertoli cells, some under the control of androgen hormones.

Interestingly, during sperm differentiation, a striking correlation is observed between the genomic organization of the subclusters and the expression of the genes within them. Specifically, as one moves from the start to the end of each subcluster, the genes display a progressive decrease in their efficiency of expression (Fig. 1). In addition, in both the α and the γ subclusters, genes located at the start of the subcluster are activated earlier than genes further along.

These waves of gene activation correlate with changes in the nature of Sertoli cells and their associated germ cells, suggesting that different sets of Rhox genes might control the transition between different stages of maturation. Accordingly, MacLean *et al.* found fewer round and elongated spermatids in mice with mutations in the *Rhox5* gene. So the authors propose that the progressive maturation of sperm is probably controlled by Rhox genes, through their coordinated expression in Sertoli cells.

The regulatory strategy implemented by the Rhox genes is reminiscent of that mediated by other homeobox genes. In mammals, for instance, genes from the Hox family are also clustered and show a temporal sequence