Knitting a finer net for photons

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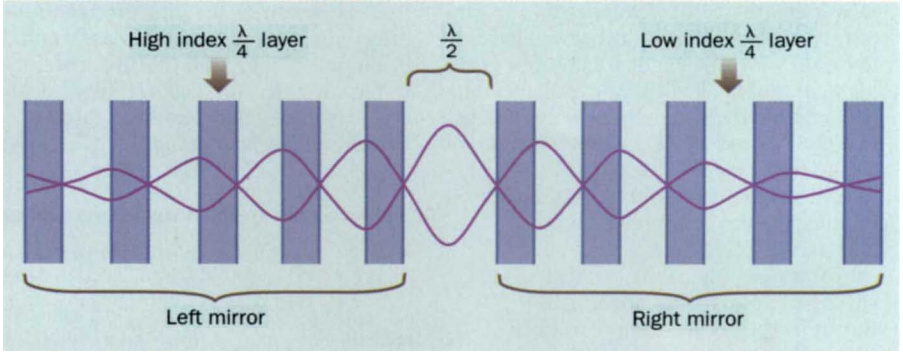
By etching an array of tiny holes in a semiconductor crystal, one can prevent light of certain wavelengths from propagating. Now these photonic bandgaps are approaching the technologically fertile optical regime.

SURPRISINGLY, science has never found a way to control optical waves in all three directions of space. Electromagnetic waves are like gelatine — squeeze in one direction and they leak out a different way. Until recently, we could control only one direction at a time, as in the face-to-face mirrors of laser cavities. But squeezing the gelatine in all three directions at once was beyond our imagination, and we needed a lesson from nature.

On page 699 of this issue[1], Krauss, De La Rue and Brand present the latest development in the field of photonic-bandgap structures, or photonic crystals. Photonic crystals are engineered three-dimensional structures that are an attempt by scientists to control electromagnetic radiation, to bottle it up and to trap it in the tiniest possible volume, or indeed to prevent atoms from emitting light in the first place. Such exquisite control would mean that we could make lasers much smaller and more efficient than the ones we have now — a step in the ongoing miniaturization revolution of both electronics and optoelectronics.

The lesson was that nature had already solved the problem, not for electromagnetic waves, but for electron waves in natural semiconductor crystals. Semiconductors possess the celebrated 'forbidden bandgap', the basis for the electronics, information and communications revolution. Try as it might, an electron wave of the wrong energy in a natural semiconductor-crystal structure can find every possible propagation direction forbidden, blocked by diffraction off one plane of atoms or another.

So the challenge for us is to make an artificial three-dimensional dielectric structure, but for electromagnetic waves rather than electron waves. This has led to an epic search for artificial crystal structures that would have such a 'photonic bandgap' or PBG. After many false starts, experimental and computational trials, and premature declarations of success, this search ended a few years ago[2,3].



A periodic stack of plates can confine light, but in only one dimension, leaving it free to leak out at the sides. Structures with two-dimensional periodicity can be devised to confine light to a line, and three-dimensional periodicity can forbid propagation in any direction.

Crystal geometries are now known that have a photonic bandgap, both in two and three dimensions, and which have been tested using microwaves in large-scale models.

This has led to world-wide competition to make these tiny structures at the scale of optical wavelengths, where the communications and optoelectronics industries work. Although the ultimate aim is three-dimensional structures, most researchers around the world have opted to first address the easier problem of making two-dimensional photonic crystals.

The paper by Krauss and colleagues[1] shows that they currently lead in the race to produce two-dimensional PBG structures at optical wavelengths. Using a combination of electron-beam lithography and plasma etching, they punched a honeycomb lattice of 100-nm holes in a crystal of AlGaAs, to produce a crystal with a bandgap in the 800–900-nm range — the shortest-wavelength gap to date in a semiconductor material. They have also measured the transmission characteristics of their crystal using a waveguide arrangement, demonstrating an impressive bandgap rejection of up to 20 dB (that is, an attenuation of a factor of 100 for light in the forbidden wavelength range). Although their structure has a gap for only one component of polarization (with the magnetic field parallel to the pillars of air), these results are an important step towards complete photonic-bandgap structures at optical wavelengths.

Several other groups have also produced short-wavelength photonic crystals, but much of the work has been done in lead-oxide glass, drawn out into fibres. Various structures have been made in this way, one with a near-infrared gap[4], and others with gaps spanning the visible range[5] down to 350 nm. Unfortunately, lead glass has a refractive index of only 1.65, which is not enough to produce a simultaneous gap for both electromagnetic polarizations. For the favourable geometry of a triangular lattice of air-filled rod-shaped holes embedded in a dielectric, a polarization-independent gap requires a minimum refractive index[6] of 2.66, which is easily achieved in semiconductors, but out of reach of most glasses. In two-dimensional optical fibres, however, it may be possible to use photonic-crystal periodicity instead of refractive-index guiding to confine the light[7].

Porous silicon is also an impressive photonic-crystal material, the first in which a polarization-independent, two-dimensional gap has been achieved[8], albeit centred at the infrared wavelength 4.9 μm. The inverse structure — a lattice of silicon pillars — has also been studied, but a bandgap has not been achieved[9].

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Some III–V semiconductor photonic crystals have likewise been made[10,11], but their transmission properties have not been measured.

As optoelectronic technology becomes ever finer, we will be able to make tiny lasers half a wavelength on a side. They may operate on new principles, like the single-mode light-emitting diode that would emit spontaneously but have many of the advantages of lasers as well. It would be reliable and insensitive to temperature, and would not require a threshold current. At the opposite extreme, macroscopic photonic crystals show much promise for microwave communications.

Krauss and colleagues have made important progress by synthesizing and testing a semiconductor-based two-dimensional photonic crystal with a gap centred at a record short wavelength. By concentrating on the semiconductor alloy AlGaAs, they have the necessary refractive index to eventually produce a gap for all electromagnetic polarizations. This race is far from over, and we will undoubtedly soon hear of more progress from other groups. Furthermore, the challenge of creating fully three-dimensional photonic crystal nanostructures has hardly begun. □

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SPONGIFORM ENCEPHALOPATHIES

A suspicious signature

Adriano Aguzzi and Charles Weissmann

THE infectious agent that causes transmissible spongiform encephalopathies — the prion — is the subject of one of the most passionate controversies of contemporary biology. The questions regarding the nature of this agent touch on a grave issue of public health: the possible link between bovine spongiform encephalopathy (BSE) and vCJD, a new variant of Creutzfeldt–Jakob disease (CJD) which has mainly affected young people in the United Kingdom (see table)[1]. Such a link has been shown to be plausible because a variety of animals can acquire BSE through the oral administration of brain tissue from BSE-infected cattle. Now on page 685 of this issue[2], John Collinge and co-workers describe an exciting new approach for tracing the passage of individual prion strains within and between species, and they suggest that vCJD resembles BSE rather than acquired or sporadic CJD.

Transmissible spongiform encephalopathies are spread by an agent which, unlike any other known agent of disease, apparently lacks informational nucleic acids[3]. Along with an impressive body of genetic evidence[4–6], this observation led to the development of the 'protein only' hypothesis — that the prion is simply the modified form of a normal protein named PrP^C, which is encoded by a single-copy host gene[7]. The surmised infectious form of PrP has been designated PrP*(ref. 8), and an abnormal form of PrP^C, called PrP^{Sc} (or PrP^{res}, for protease-resistant), has been equated with PrP*. PrP^{Sc} accumulates during the advanced stages of most cases of transmissible spongiform encephalopathies, and it is characterized by its partial resistance to proteolytic digestion and by its tendency to aggregate.

PrP^C and PrP^{Sc} seem to be chemically identical, so they may represent conformational isomers. PrP^{Sc} is thought to propagate by interacting with PrP^C and causing its conversion into PrP^{Sc}. As predicted by the 'protein only' hypothesis, PrP^C is essential for the propagation of infectivity[9], for clinical disease[9] and for brain pathology[10] But this hypothesis cannot account for the occurrence of distinct prion strains that can be propagated indefinitely in hosts that are homozygous with regard to their PrP genes. Prion strains are identified by the disease incubation time or by the pattern of brain lesions[11]. Stable prion strains could be easily explained if the infectious agent were virus-like: strains would then be encoded by the viral genome. However, if the prion is a conformational isoform of PrP^C, then all of its phenotypic characteristics should be determined by the host PrP genes alone and any prion strain should, after a few passages in hosts of the same species, become monomorphic.

Differences between sporadic and variant CJD

	Sporadic CJD	New variant of CJD (vCJD)
Typical age of onset	55–70 yr	19–39 (median 28) yr
Presenting features	Dementia, myoclonus	Behavioural changes, ataxia, dysaesthesias
Clinical course	Rapidly progressive	Insidious onset, prolonged course
PRNP genotype (codon 129)	Predominantly homozygous	Met/Met homozygosity in 100% so far
PrP^{Sc} deposits	Synaptic deposits, rarely plaques	Prominent florid plaques
PrP^{Sc} banding pattern	Type 1 and type 2*	Type 4 (similar to experimental BSE in mice, macaques and other species)

* Type 3 in iatrogenic cases with intramuscular inoculation

How can the existence of different strains be explained by the 'protein only' hypothesis? Three possibilities have been suggested. First, PrP* or PrP^{Sc} could be associated with an accessory molecule, such as a small nucleic acid[12], which modulates the phenotype of the prion without being essential for infectivity. However, there is no evidence for a nucleic acid of this kind. Second, PrP* could undergo secondary modifications, such as glycosylation. Certain polysaccharide residues could then specifically target the molecule to cells which in turn glycosylate PrP in an identical fashion. This is known as the 'target-cell hypothesis' (K. H. Meyer, personal communication). Finally, PrP* or PrP^{Sc} could exist in various different conformations (at least one for each prion strain), with each type of prion being capable of imparting its own conformation to the PrP^C molecule with which it interacts. More than one protein chemist has declared this idea to be insane — and yet this is precisely what is implied by a growing number of studies.

Western blot analysis of PrP from extracts of normal or prion-infected brains reveals three major bands, corresponding to PrP that has two, one or no polysaccharide chains attached to the amino terminus. In normal brain extracts, treatment with proteinase K degrades all of the PrP; however, in prion-infected brain, protease treatment leads to increased mobility of the three protein species because of amino-terminal truncations. Presumably, the changed conformation of PrP^{Sc} protects a large part of the molecule against degradation. Bessen and Marsh[13] showed that when two strains of PrP derived from mink were propagated in inbred Syrian hamsters, they gave rise to PrP^{Sc} molecules that had distinct and heritable migration patterns following proteinase K treatment. This difference in mobility was shown to be due to cleavage at distinct sites.

The most common form of prion disease in humans, Creutzfeldt–Jakob disease, occurs in four forms: a familial form, linked to mutations in the PrP gene; an iatrogenic or acquired form, due to inad-