Photonics

More than transparent

Roy Sambles

So you thought your microwave oven couldn't radiate through the mesh of metal on the windowed door? Experiments tell us that hardly any radiation penetrates a metal plate with holes of diameter smaller than the radiation's wavelength. But it appears, from new experiments reported by Ebbesen and co-workers on page 667 of this issue[1], that such metal grids may not be as impervious to radiation as we had believed. Silver is an excellent conductor of electricity and so should screen out radiation very effectively, yet Ebbesen *et al.* found that thin, perforated silver films deposited on quartz are remarkably transparent. There is strong and selective transmission of radiation with wavelengths greater than the hole diameter.

Why should this be? Crucially, the structure is not just a random array of holes in the silver film but a regular, periodic two-dimensional grating structure (Fig. 1). The holes were 150 nm in diameter, and from 0.6 to 1.8 μm apart, in both square and hexagonal arrays. For radiation perpendicular to the film, these structures constitute zero-order diffraction gratings (for which transmission is only possible with no deflection) for radiation of wavelength λ above 0.6 to 1.8 μm on the air side (0.88 to 2.6 μm on the quartz side). Remarkably, the 0.6-μm-spaced square-array grating transmits very strongly at 0.96 μm — with an efficiency greater than two. Increasing the thickness of the silver layer from 200 nm to 500 nm appears to *increase* the strength of this transmission peak.

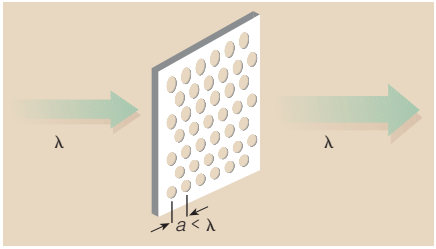

Figure 1 **The phenomenon described by Ebbesen *et al.*[1]. Holes in a metal screen with a diameter less than the radiation wavelength, arranged in an array with a periodicity that is also less than the radiation wavelength, can selectively transmit normal-incidence radiation.**

Selective reflection has been seen before in similar metal mono- and bi-gratings[2,3], but this seems to be the first observation of selective transmission. The explanation for this extraordinary behaviour appears to rest with the excitation of surface modes called surface plasmons. These are oscillating electromagnetic fields, strongly localized at the surface of a metal on which there are associated charge oscillations.

On a flat plate, an incident photon can only be converted to a surface plasmon (or vice versa) if it has the same momentum (equivalently, wave number, $1/\lambda$) parallel to the surface, and the same energy. So a normal-incidence photon cannot excite a surface plasmon. But scattering of plasmons from a periodic array of holes allows the excitation of the surface plasmon resonance, because diffraction allows the addition of multiples of the wave number to the in-plane wave number (for normal incidence this is zero).

The lowest-momentum surface plasmon (hence, lowest energy radiation, or longest wavelength) to be excited in this way has exactly the wavelength of the grid. Higher-momentum components demand higher-energy, shorter-wavelength radiation, which is readily transmitted by the holes in any case. Also, but more mysteriously, the grating geometry must somehow allow the air/silver surface modes to couple strongly with modes of different momenta on the quartz/silver interface, so energy can be transferred from one side to the other and re-radiated.

Although the theoretical analysis is incomplete, it seems clear that it is these grating-perturbed surface plasmons that allow the selective transmission of radiation. As would then be expected, the strong normal-incidence transmission occurs close in wavelength to the grating separation multiplied by the refractive index of the quartz substrate — that is, there is a small grating-induced perturbation of the surface plasmon having the highest available wavelength.

This could be a useful new way to geometrically filter electromagnetic radiation with no diffractive effects. By controlling the hole spacing, and perhaps overcoating the metal to modify the surface plasmon dispersion curve, a narrow, selectable, band of wavelengths may be transmitted over a wide angle. Combining this with voltage-controllable liquid crystals may then yield new displays or other active filter devices.

Will the phenomenon also happen at longer wavelengths? The answer might rest with the permittivity of the metal, as that dictates the momentum of the surface plasmon, at least on a planar interface. However, as the wavelength of the radiation is increased so, in general, the real part of the permittivity of most metals becomes more negative. This makes the surface-plasmon resonance sharper, and the band structure of the surface plasmons is then dictated almost entirely by the surface geometry, becoming largely

Ecology

Spring origami

Next time you pack for a scientific conference, consider how much more you can cram into a suitcase if your clothes are neatly folded. Problems of packing are common in biology —examples include DNA in chromatin, brain architecture and the arrangement of alveoli in the lung. An entertaining example from functional ecology is addressed by H. Kobayashi *et al.* in *Proceedings of the Royal Society* (265, 147–154; 1998). They have looked at the geometry of unfolding tree leaves from tightly packed buds.

The design of the leaves is reminiscent of man-made structures such as solar panels and the lightweight antennae of satellites, which have to be packed optimally to ensure fail-safe deployment. The authors have used techniques borrowed from engineering to model the leaves of two species of deciduous tree, the hornbeam *Carpinus betulus*, and the beech *Fagus synaticus*. These species have oval, corrugated leaves with a central midrib, which develop from scaled buds.

The leaf is modelled as a plain surface with straight parallel folds. Conveniently, the creases or folds of the leaves run along lateral veins, angled at 30–50° from the midrib of the expanded leaf. Using vector analysis, the authors calculate the effect of varying the 'vein angle' and leaf shape on packing efficiency and leaf expansion. Increasing the vein angle allows more compact folding within the bud, but only at the cost of energetically less efficient, slower leaf expansion. By getting the balance of design parameters right, natural selection may optimize the timing of leaf deployment, maximizing photosynthesis while minimizing damage caused by late frosts and leaf-eating larvae.

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metal independent. So an appropriately periodic surface with holes smaller than the radiation wavelength should also transmit radiation selectively for almost any metal.

Fortunately for the microwave user, the mesh period and the hole diameter are so much smaller than the radiation wavelength that there is no chance of leakage through this process. □

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Immunology

Unmasking the killer's accomplice

Marco Colonna

Class I molecules of the major histocompatibility complex (MHC) are polymorphic glycoproteins which, under normal circumstances, are expressed on the surface of almost every cell in the body. Upon viral infection or in tumour cells, however, MHC class I can be downregulated, and its absence is 'sensed' by natural killer (NK) cells in a process known as recognition of 'missing self'[1]. This is mediated by cell-surface receptors which, on binding class I molecules, transduce inhibitory signals that block NK-cell-mediated lysis. When class I expression is lost or reduced, NK cells are released from inhibition and can, therefore, rapidly identify and eliminate virally infected or tumour cells.

Intriguingly, some MHC class I NK-cell-receptor homologues promote — rather than inhibit — the activation and cytotoxicity of NK cells, by a hitherto unknown mechanism. But on page 703 of this issue, Lanier *et al.*[2] describe the key to this mystery. They have discovered a signal-transducing molecule called DAP12, which couples an MHC class I receptor on the surface of the NK cell to an activating signal-transduction pathway within that cell.

Natural killer cells monitor the expression of class I molecules using many cell-surface receptors. Molecular cloning of these receptors has revealed remarkable diversity[3,4]. Two receptor families have been identified: the C-type lectin molecules (including rodent Ly49 receptors and heterodimers of the human CD94 and NKG2 polypeptides); and molecules belonging to the immunoglobulin superfamily, known as human killer-cell inhibitory receptors (KIR).

Each receptor family includes many members that recognize different class I molecules. Heterogeneity has also been observed in the cytoplasmic domains of the receptors. Inhibitory receptors have a long cytoplasmic tail that contains immunoreceptor tyrosine-based inhibitory motifs (ITIMs). On recognition of class I ligands, ITIMs are phosphorylated, allowing recruitment and activation of a protein tyrosine phosphatase[5]. This results in dephosphorylation events that block NK-cell activation and, thus, the cytotoxic response. But, in each family, many NK-cell receptors (such as Ly49D, CD94/NKG2C and KIR2DS) have short cytoplasmic domains that lack ITIMs and do not inhibit the cytotoxic response. In fact, these truncated receptors transmit stimulatory signals to NK cells by activating protein tyrosine kinases and phospholipase C, leading to an increase in NK-cell-mediated cytotoxicity[6–9].

How do these receptors initiate NK-cell activation on binding class I molecules? Because activating receptors lack the sequence motifs that are implicated in positive signal transduction, they probably transduce signals through associated proteins. Use of a separate subunit to mediate signal transduction is not unusual among immunoreceptors — FcεRI and FcγRIII, for example, are oligomeric complexes in which signal transduction is mediated by associated protein subunits. The T- and B-cell receptors also use separate subunits, and all contain one or more immunoreceptor tyrosine-based activation motifs (ITAMs) in their cytoplasmic domain[10]. When the ligand-binding component of the receptor is engaged, the cytoplasmic ITAMs are tyrosine-phosphorylated by src-family tyrosine kinases. This leads to the recruitment of ZAP-70 and SYK protein tyrosine kinases[10], which trigger a cascade of intracellular phosphorylations that lead to cellular activation.

Biochemical studies[11] have shown that one activating NK-cell receptor is noncovalently associated with a protein (relative molecular mass, M_r, 12,000) that exists as a disulphide-linked dimer. Following a logical and insightful path, Lanier and colleagues[2] have now identified the complementary DNA that encodes such a molecule. They screened a database for cDNAs encoding unidentified ITAM-bearing proteins, and pulled out DAP12, which encodes a transmembrane protein (M_r 12,000) that strikingly resembles the γ-chain of the FcεRI and the ζ-chain of the T-cell receptor. The cytoplasmic tail of DAP12 contains a single ITAM that is tyrosine phosphorylated and can bind in this form to both ZAP-70 and SYK tyrosine kinases (Fig. 1).

The DAP12 protein associates with one activating receptor, KIR2DS2. The resulting complex, when expressed in pre-B or T cells, induces events that lead to cell activation. Moreover, in the hydrophobic transmembrane region of DAP12 is a negatively charged aspartic-acid residue that is thought to interact with the positively charged lysine residue in the transmembrane domain of the class I-binding subunit (Fig. 1). Finally, DAP12 has a very short extracellular domain that contains cysteine residues which, presumably, mediate interchain disulphide linkage.

Interestingly, DAP12 is expressed not only in NK cells, but also in other types of cell that are involved in the immune response, such as dendritic cells. So could DAP12 also be involved in the activation of these cells? Receptors bearing transmembrane and cytoplasmic domains similar to those in activating NK-cell receptors have been identified in myeloid and lymphoid cells[12], representing potential partners for DAP12. The murine pre-T-cell receptor also associates with a disulphide-linked dimer (M_r 12,000) that may correspond to DAP12 or to a related molecule[13].

Although the discovery of DAP12 is a breakthrough in our understanding of the mechanism by which activating MHC class I receptors transduce stimulatory signals, the biological function of these receptors is still

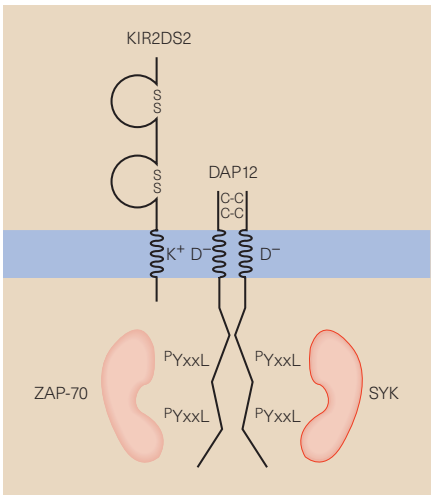

Figure 1 **Structure of the activating natural killer (NK)-cell receptor complex for major histocompatibility complex (MHC) class I molecules. The complex consists of a class I-binding component, the KIR2DS2 receptor, associated with the DAP12 disulphide-linked homodimer identified by Lanier *et al.*[2]. DAP12 contains a cytoplasmic immunoreceptor tyrosine-based activation motif that is tyrosine phosphorylated upon receptor ligation, and recruits ZAP-70 and SYK protein tyrosine kinases. These kinases trigger a cascade of phosphorylation events that lead to cellular activation. The KIR2DS2 receptor and DAP12 are non-covalently associated — complex formation is facilitated by interaction of transmembrane residues with opposite charges.**