# Radiation risk

SIR—There is a significant increase of ultraviolet levels in the Antarctic during the presence of the 'ozone hole'[1], and consequently there is concern for Antarctic life forms which may have developed only minimal defences against this radiation[2,3]. We have previously pointed out[4] that the problem is potentially most acute under the vast sea ice cover surrounding Antarctica, for the sea ice is particularly transparent when the ozone hole occurs. Because we did not know the ultraviolet properties of the ice, our argument relied
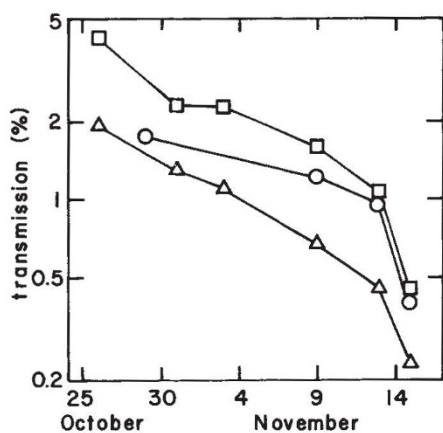
Seasonal development of the transmission of sea ice at wavelengths of 300 nm (ultraviolet-B, triangles) 350 nm (ultraviolet-A, squares) and 650 nm (mid-visible, circles).

on an extrapolation from data in the visible part of the spectrum. Here we report measurements showing that the transmission of sea ice shows an even larger seasonal variation in the ultraviolet than in the visible spectrum.

The experiments were performed on 1.7-m-thick first-year sea ice in McMurdo Sound, using a technique described elsewhere[5]. The measured development of the transmission of the ice at three wavelengths is shown in the figure. All three curves show a substantial decrease during the spring, resulting from the development of a turbid surface layer left behind after brine pockets in the ice drain[6]. Surprisingly, the development is stronger in the ultraviolet than in the visible, for which we do not yet have a satisfactory explanation.

Algae residing near the ice–water interface are the first major biological popula-tion to experience the radiation entering the ice. These algae have been estimated to account for up to 30% of the primary food production in the ice covered regions of the southern ocean[7], and they are known to be sensitive to ultraviolet radiation[2]. We have estimated the level of flux falling on these algae as the product of the sea ice transmission of the figure and the surface flux measured at Palmer Station during the 1988 spring[1]. In the latter half of October, the under-ice flux rises as high as 10^{-3} W m^{-2} in a 10-nm band centred on the more damaging ultraviolet-B region, up from about 0.3×10^{-3} W m^{-2} before the appearance of the ozone hole. The 1988 ozone depletion was rather weak, and for the stronger depletions of 1987 and 1989 the October under-ice flux in the same band[8] would rise to about 5×10^{-3} W m^{-2}. Later in the season, the turbid surface layer shields the ice, so that in the height of summer the flux in this band is very much lower, about 5×10^{-5} W m^{-2}. Thus the yearly dose at the base of the ice is overwhelmingly dominated by the October flux, with the consequence that the ice algae will have experienced more than a tenfold increase of ultraviolet-B dose in recent years compared to the pre-1975 levels.

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Cortical maps

SIR—Durbin and Mitchison[1] suggest an explanation for cortical maps and their distortions. If their purpose is to explain the biological conditions for the formation of two-dimensional brain maps, a theoret-ical description of the lateral interactions between neural cells would require the use of spatio-temporal partial differential equations[2,3]. But to implement powerful artificial 'neural' computing structures (which can form dimensionality-reducing mappings and abstractions), the self-organizing process is better described by computational algorithms that simultan-eously update sets of neighbouring neural units[4].

Contrary to what Durbin and Mitchison claim, the equation they use (see their legend to Fig. 2) is not based on Hebb's law of synaptic plasticity; the first term would need a more lengthy justification[5]. Their second term was introduced "to make neighbours have similar receptive fields", but they did not explain what kind of biological adaptive process would account for this effect. This term in fact seems to stem from the physical analogy of stress–strain relations in an elastic ring or net[6]. In the other works cited by Durbin and Mitchison, on the other hand, lateral interaction has been used to activate neighbouring cells to learn from the same input, which is a different and natural assumption.

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DURBIN AND MITCHISON REPLY—As Kohonen points out, self-organizing mod-els have previously been used to simulate cortical maps. We did not intend to sug-gest the contrary, and we did, in fact, cite Kohonen's book and Swindale's work. However, we should perhaps have spelt out more clearly the ancestry of our simu-lation. Of course, the main goal of our paper was to consider wiring minimization as an organizing principle for maps of many variables, and to relate this to self-organizing models. We believe this is a new approach to cortical mapping.

We are surprised at Kohonen's rather critical comments about the self-organizing model we used, since it oper-ates in a similar fashion to his own. Both work at a fairly abstract level, which we believe was appropriate for our paper. As he remarks, the first term in our equation, though not directly hebbian, can be derived from Hebb-like interactions. Our second term can be justified in a similar way, by averaging neighbour interactions of the sort he describes; the 'elastic net' analogy is only intended to give intuitive guidance on the way the model works. The resulting equation has the advantage of a cleaner mathematical form which can be analysed in detail[7]. Empirically, it gives results similar to his and other self-organizing models, and in our hands is certainly no less computationally effective.

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