Effects of microwaves on cells and molecules

SIR—For a paper stating that "the literature on biological effects of low-level microwave energy remains shot through with ambiguity", Foster and Pickard's commentary on the risks of microwaves[1] is wide of the mark and unreasonably denigrates the efforts of the many who have established structural and functional substrates for essential aspects of these interactions at cellular and subcellular levels[2]. They chose as examples of current knowledge three unrelated observations that are not now, nor were ever, in the mainstream of studies of mechanisms of low-level radiofrequency (r.f.) and microwave biological effects: the auditory effect; rate changes in the isolated frog heart; and altered permeability in the blood–brain barrier.

Foster and Pickard ignore a wealth of findings that relate to the role of cell membranes in transductive coupling of electrical and chemical signals from the outside to the inside of the cell[3]; in interactions at cell surfaces between weak electromagnetic fields (extremely low-frequency (ELF) fields and r.f./microwave fields amplitude- or pulse-modulated at ELF frequencies) and the gamut of natural and artificial chemical molecules that stimulate cell surfaces at specific receptor sites; in the occurrence of these interactions only in certain bands of modulation frequencies and at certain field intensities, constituting 'windowed' interactions[4]; and in the clear evidence that these processes involve enormous amplification between initial energies of the first binding triggers and their ultimate actions within cells[5]. Beyond the evidence for these modulation-dependent effects, further research is needed to evaluate the nature and extent of possible resonant interactions between biomolecules and millimetre microwave fields.

Evidence for cell membrane interactions with modulated r.f./microwave fields is firmly based on modern cell and molecular biology. Of prime importance is the demonstration that these fields sensitively modify calcium binding at cell surfaces and modulate a host of calcium-dependent intracellular enzyme mechanisms[6]. These intracellular markers of events initiated at cell surfaces regulate the flow of intracellular messages (protein kinases), cell metabolism and cell growth.

In less than a century, the frontier of our understanding has moved from tissues to cells to molecules. The use of imposed fields will now reveal the essential importance of biological organization at the atomic rather than the molecular level, and in physical rather than chemical terms; with coherent states between adjacent molecular electric charges and enormous cooperativity in energy release by very weak triggers as the physical essence of living matter[7,8].

Studies of chemical tumour promoters are providing new knowledge about their disruption of normal communication between cells with onset of unregulated growth[9]; bioelectromagnetic research is producing clear evidence of joint cell-surface actions of tumour promoters with ELF and modulated r.f. fields[10]. The union of these two disciplines has initiated the first significant new approach to tumour formation in 75 years — an approach that directs attention to dysfunctions in inward and outward streams of signals at cell membranes, rather than to damaged DNA in cell nuclei[11].

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Training the brain using neural-network models

SIR—Recent discussions in *Nature*[1,2] and elsewhere[3] highlight the potential of parallel distributed processing (PDP) for modelling various aspects of cognition. The PDP approach, however, is circumscribed by the need of a network to be informed of its output error so as to modify hidden units through back-propagation. Without this performance, feedback training of the network cannot occur. Consequentially, there exists a paradox within PDP; for a network to learn anything it must in some way already possess a means of knowing what is and is not a correct output — but the ability to do this is precisely what the network has been created to explain.

This paradox does not affect present-day running of PDP models as realized on computers. PDP experimenters train them with thousands of example inputs with correction feedback. But connectionist models are claimed to be able to explain actual brain processes. This raises a question: what takes the place of the training provided by PDP experimenters for the brain's neural networks?

One possibility is that the brain tutors its own neural networks through a bootstrapping process. Cognition could involve development of easily acquired tutoring processes of limited efficiency and effectiveness which in turn tutor later and cognitively more effective and efficient processes.

The development of reading may provide such an example. Children pass through several stages in learning to read English. Initially — often before entering school — children recognize some frequently occuring words using visual 'logographic' characteristics[4,5]. Most children then quickly learn to use the phonological information present in written words[5]. Later, the use of phonological information loses importance as a more abstract non-phonological letter sequence[5] information takes its place. The limited vocabulary of logographically recognizable words could provide the examples needed to train the phonological neuronal network. This relationship could occur between the phonological and orthographic neuronal networks with the phonological decoding of written words functioning not only to identify them but also to tutor the development of hidden units in the orthographic network. This might explain why phonological decoding skills predict reading success[6] even though mature reading in English is not itself phonological.

PDP has been called behaviourism in computer's clothing[7]. While this analogy is misleading, it is true that the PDP account of learning requires a kind of conditional reinforcement in the form of adjustment of hidden units by back-propagation. The circumstances which could optimize the availability of information for this reinforcement — its 'tutorability' — is an aspect of PDP that so far has received little attention. But it could be important to our understanding of the role of PDP models in the development of human cognition.

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