IN THE NEWS

Give me that remote!
A new report published in *Science* seems to confirm something that many media critics have been trying to persuade us of for years — watching television makes us violent. This is the conclusion of a study by Jeffrey Johnson and colleagues at the New York State Psychiatric Institute, who showed that adolescents who watch television for more than one hour a day are significantly more likely to become violent than those who watch less.

Brad Bushman from the University of Iowa puts the problem into perspective: "The correlation between violent media and aggression is larger than the effect that wearing a condom has on decreasing the risk of HIV. It's larger than the correlation between exposure to lead and decreased IQ levels in kids. It's larger than the effects of exposure to asbestos. It's larger than the effect of secondhand smoke on cancer" (*Washington Post*, 29 March).

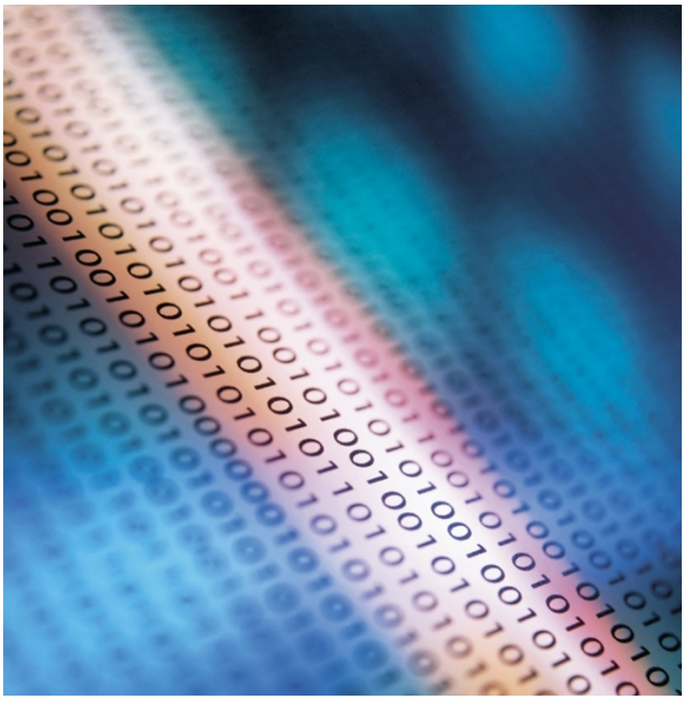
The study seems to indicate that the number of hours spent watching television, and not necessarily violent programme content, is the determining factor. However, as the *Guardian* (UK, 29 March) points out, "in the US, three to five violent acts occur in an average hour of prime time television, and 20 to 25 violent acts in an average hour of children's television".

Johnson believes that his findings have profound implications. He says "By decreasing exposure to media violence, we may be able to prevent millions of Americans from being raped and murdered" (*New York Times* 29 March). Others, such as Steven Goodman from the Johns Hopkins University, were less convinced: "The question is, what makes kids different who watch TV for many hours as compared to those who watch little? You have to be extremely careful about making any causal inferences here" (*New York Times*).

Heather Wood

COMPUTATIONAL NEUROSCIENCE

Quantifying synaptic efficacy



Synaptic efficacy is a basic concept in neuroscience for which we have an intuitive definition — the capacity of a presynaptic input to influence postsynaptic output. Similarly, we have an intuitive idea of the factors that affect synaptic efficacy. So, if release probability or the number of quanta increases at a given synapse, its efficacy will also increase. Although these ideas are useful, a more rigorous analysis of synaptic efficacy and the factors that influence it would be very valuable for deciphering what a synaptic input tells the postsynaptic neuron. There are several points of view on how to address this problem, each of which has different merits. London *et al.* now contribute to this field by introducing a new functional measure of the mutual information shared by the pre- and postsynaptic spike trains — the synaptic information efficacy (SIE) — and by evaluating the factors that influence it.

London *et al.* started by thinking about the postsynaptic output as a continuous string of '1's and '0's, depending on whether or not the cell fired a spike at any given point in

CORTICAL DEVELOPMENT

Ena/VASP-deficient neurons go too far

The Ena/VASP family of proteins have emerged as important regulators of cell motility, owing to their effects on the actin cytoskeleton. Three members of this family — Mena, VASP and EVL — have been identified in mammals, and it was suspected that they might control neuronal migration in the developing neocortex. However, this has proved difficult to study, largely because inactivation of the genes produces a lethal phenotype. Goh *et al.* have now solved this problem by inactivating the proteins in only a fraction of cortical progenitors, and their initial data, reported in *Current Biology*, seem to confirm that the Ena/VASP proteins help to determine the position of pyramidal neurons in the cortex.

The authors constructed a retroviral vector that targeted the expression of an Ena/VASP ligand motif to the surface of mitochondria. This allowed the Ena/VASP proteins to be sequestered onto mitochondria, thereby neutralizing their function. The retrovirus was injected into mouse embryos *in utero* at embryonic day 11.5, and was incorporated into a subset of dividing cortical cells.

The mammalian cortex develops in an inside-out manner, such that early-born neurons inhabit deeper layers than those born later in development. However, in injected brains that were allowed to develop to adulthood, many pyramidal cells that had incorporated the retrovirus were located nearer to the surface than would be expected. The morphology of the ectopically positioned cells was normal, indicating that inactivation of the Ena/VASP proteins affects only the positioning of the neurons and not their differentiation.

This result is consistent with the previously described role of Ena/VASP proteins in negatively regulating cell motility in fibroblasts. Future studies should address how the Ena/VASPs interact with other molecules and pathways that regulate cortical cell migration. Interestingly, Goh *et al.* showed that the Ena/VASP proteins are highly expressed in neurons that lie next to reelin-expressing cells. Reelin is thought to provide a stop signal for migrating neurons, so it is tempting to speculate that it exerts its action through regulation of the actin cytoskeleton by Ena/VASPs. This new technique for selectively inactivating the Ena/VASP proteins might help us to answer this question.

Heather Wood