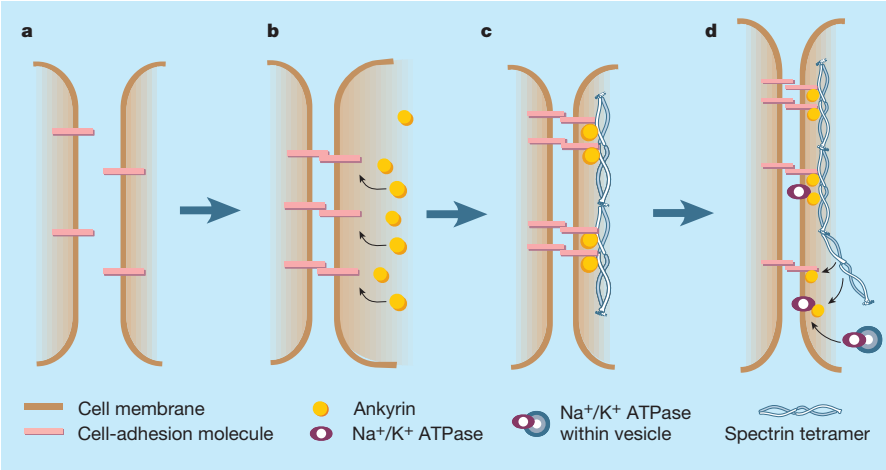

Figure 2 **Model for the accumulation of membrane proteins at points on the cell surface specified by cell-adhesion molecules. The new work[1–3] is consistent with the following model.** a, **Two cells interact through adhesion molecules.** b, **Ankyrin is recruited to ligand-bound cell-adhesion molecules. Other ankyrin-binding molecules, such as Na^+/K^+ ATPase, do not accumulate in the absence of spectrin.** c, **Spectrin tetramers are recruited to membrane-bound ankyrin/cell-adhesion-molecule complexes, crosslinking these complexes and stabilizing the regions of cell–cell contact.** d, **Further spectrin molecules are recruited into a submembrane skeleton crosslinked by the cytoskeletal protein actin. Binding sites on spectrin trap ankyrin bound to other molecules such as Na^+/K^+ ATPase, and promote their stable incorporation into the membrane domain.**

ankyrin-binding sites. One ankyrin molecule, recruited to a cell-adhesion molecule, binds to a spectrin tetramer (Fig. 2). This tetramer then has another ankyrin-binding site free to connect to other ankyrin-containing complexes, such as that consisting of ankyrin and Na^+/K^+ ATPase. If spectrin is missing, this ion pump cannot be captured at the membrane. So spectrin traps and stabilizes proteins at specific points on cell surfaces first specified by ligand-bound cell-adhesion molecules (Fig. 2). The results of Dubreuil *et al.*[3] support this theory as far as the Na^+/K^+ ATPase goes. And the results of Hammarlund *et al.*[1] and Moorthy *et al.*[2] indicate that spectrin may also trap and stabilize key muscle or nerve proteins at specific points on cell membranes as specified by cell-adhesion molecules.

There are more human genes encoding spectrins than there are worm or fruitfly spectrin genes. So, different spectrin isoforms may have evolved other tasks in vertebrates — for example, in Golgi function. This might account for some of the discrepancy between past and present results. But the new results[1–3] may also bear on human biology. It is interesting that, in *C. elegans*, a mutation that causes a loss of muscle contraction suppresses the problems that arise from a lack of β-spectrin. This result hints at the idea that any muscle that is in continual use (for example, the heart or diaphragm) might be the most vulnerable to the effects of spectrin mutations, and some unexplained human muscle and heart disorders may have their origins in mutated spectrin genes.

The new work might apply even more generally. Dystrophin is a protein that is mutated in Duchenne and Becker muscular dystrophies in humans and is a member of the protein superfamily to which spectrin belongs. *C. elegans* with the *dys-1* mutation lack a functional form of dystrophin, but do not show any obvious muscular degeneration. Instead, they lose the enzyme acetyl-cholinesterase, presumably from its anchoring points in muscle-cell membranes[10]. Dystrophic *mdx* mice, which also lack functional dystrophin, lose nitric oxide synthase from the plasma membrane of muscle cells[11]. Perhaps dystrophin and other members of this superfamily emulate spectrin's protein-accumulating activity. ■

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Daedalus

Pulses of the mind

The human brain, which works by processing discrete nerve impulses, is often regarded as a digital computer. Daedalus wants to measure its processing power in operations per second. Modern magnetic-resonance imaging can detect a region of high brain activity by its increased metabolic rate, but cannot deduce the corresponding digit rate. Daedalus now has an improvement.

He points out that the basic processing unit of the whole nervous system is the nerve pulse. At each point it involves a radial movement of sodium ions lasting about a millisecond. Sodium nuclei have spin and magnetic moment. In a suitable magnetic field, a resonating radio-frequency can elevate them to an excited state, from which they will later relax back to the ground state. Suppose that radio-frequency is 1 kilohertz, corresponding to a time constant of a millisecond. Then relaxation should be decidedly stimulated if the nuclei are being vibrated with the same time constant — as by being in a nerve along which a pulse is passing.

The magnetic field for which sodium ions resonate at 1 kHz is a modest 0.9 microtesla. Daedalus's nuclear magnetic psychometer places the subject's head in such a field and irradiates it at 1 kHz. It measures the relaxation times of the sodium ions in the various regions of his brain. The greater his mental activity, the faster the ions will relax; or more exactly, the stronger the peak of their relaxation-time spectrum within the 1 ms region. Once properly calibrated, the instrument will give the total rate of working for the subject's brain, in pulses per second.

Psychology will at last have a sound numerical basis. Daedalus expects that subjects with a high IQ will show a greater processing rate than those of low IQ. But asked to solve a problem, their rate of working will rise less — their algorithms will be more efficient. Yet intuitive types with an unexceptional IQ may still show high firing rates, from their active imaginations. As the subject learns a new skill, the firing rate in the relevant brain region will rise, and then decline as he automates his new ability. Skilled meditators may be able to drive their brain activity right down; but even a sleeping or drugged subject will still show the processing needed for the brain's steady 'housekeeping' — maintaining heart rate, temperature, peristalsis and so on. All the values should exceed a billion pulses per second, showing up modern computers for the primitive devices they are. **David Jones**