

in paramutation is strongly supported by work from Vicki Chandler's group showing that paramutation requires an RNA metabolizing enzyme that is involved in other epigenetic phenomena¹³.

Rassoulzadegan and colleagues' model has yet to be validated, and several points need to be clarified. Notably, the mechanism by which aberrant RNAs from *Kit^{tm1Alf}* heterozygotes induce spotting is not known. Does it involve bona fide siRNAs or miRNAs emanating from the mutant allele? Also, are aberrant RNAs causing mRNA degradation or epigenetic modifications at the wild-type allele? Are these effects mediated by RNAi or by other pathways influenced by small RNAs? If siRNAs or miRNAs do emanate from the paramutagenic *Kit^{tm1Alf}* allele, how do they arise, and do similar RNAs arise in other paramutation models? A particularly intriguing possibility is that such RNAs regulate other non-genetic modes of inheritance, such as metabolic or behavioural imprinting. These have far greater consequences for human development than for

spotty mice and maize, but we may learn about such mysterious processes by studying those mouse tales. ■

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NANOMATERIALS

Display of flexibility

László Forró

Treated the right way, carbon nanotubes can be moulded into large, flexible electron-emitting sheets. The material is one half of what's needed for an electronic display you could fold up and slip in your pocket.

As far as their electrical properties are concerned, the long, cylindrical carbon molecules known as carbon nanotubes are notoriously Janus-faced: picking out a nanotube from a newly fabricated batch, one cannot know whether it will be a conductor or an insulator. In principle, both types are useful, and the basic transport, mechanical and optical properties of each kind are more or less known. But not knowing how to tune the synthesis to yield just one type is a considerable handicap to the use of nanotubes as, for example, components in integrated circuits. Because of this, attention has concentrated on applications for which this uncertainty is irrelevant: nanoswitches¹, motors², actuators³ and yarns for composite materials⁴, to name but a few.

One particularly promising application of carbon nanotubes is the flat-panel display. Such displays exploit the fact that, owing to quantum-mechanical tunnelling effects, nanotubes are very efficient emitters of electrons when placed under electric fields. They thus act as a source of electrons that is similar to the cathode-ray tube used in conventional monitors, but one that is just a few millimetres thick and operates at a fraction of the power. The Samsung Advanced Institute of Technology in South Korea has recently reported the

fabrication of a 30-inch flat television screen, based on field emission from carbon nanotubes, that is close to commercialization⁵. Now, writing in *Nano Letters*, Yung Joon Jung and colleagues⁶ report the assembly of the field-emission part of a screen using carbon-nanotube electrodes embedded in a polymer matrix. Their simple idea could be a big step towards the implementation of large-area displays that are not only flat, but flexible too.

The authors' method consists of patterning islands of catalytic particles on a surface of silicon dioxide. Using the technique of chemical vapour deposition, they synthesize vertically aligned carbon-nanotube pillars 500 micrometres in diameter and 100 micrometres high. This large area of nanotube pillars is impregnated with dimethylsiloxane, a polymer precursor. Heating the resulting matrix to 100 °C polymerizes it, and the nanotube-polymer composite can be peeled off (Fig. 1). These embedded nanotube pillars show excellent emission characteristics, with a field-enhancement factor (defined as the ratio of the electric field at the tip of the pillar to the applied electric field) of 10,000.

The originality of the work lies in the polymer precursor, which perfectly wets the individual nanotubes within the pillars. Without



Figure 1 | Flexible friend. Jung and colleagues⁶ nanotube-polymer composite, with its evenly spaced carbon pillars clearly visible.

this wetting, surface tension would have shrunk the pillars, and the mutual screening of the electric field at tips of neighbouring nanotubes would have damped down the exceptional field-enhancement factor. Crucially, the nanotubes preserve their emission properties even if the matrix is severely bent.

Although this highly flexible nanotube-polymer composite could prove to be a central part of a future foldable flat-screen display, there is still a long way to go. A second, essential component will be a flexible screen that, as in a conventional display, fluoresces when struck by electrons sent out by the field-emitting part. This screen must be kept at a constant separation from the field-emitting composite, even when the display is bent. Constructing such a component will be difficult, but is not an insurmountable task.

The potential applications of flexible displays are many. One could conceive of a flexible electronic newspaper, the pages of which are reloaded using wireless network technology, and which you could bend or roll up after reading. More whimsically, projecting a car's surroundings onto its body with such a display would make it invisible — allowing James Bond to die another day. Perhaps one could even envisage a time when the opera diva changes her dress between scenes by simple reprogramming.

Composites of carbon nanotubes and polymers are not the only candidates to inspire such unbridled imagination. Organic light-emitting diodes are another, but these have their own problems, suffering from short lifetimes owing to air sensitivity, fatigue and the like. Thus, contributions such as that of Jung and colleagues⁶ play an important part in ensuring the emergence of the flexible display from the realm of science fiction to that of science fact. ■

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IMMUNOLOGY

Adaptable innate killers

Peter Parham

Natural killer cells are versatile white blood cells that act in the innate immune system. Quite how adaptable they can be in the absence of other, more specialized, immune cells comes as a surprise.

Contact dermatitis is an occupational hazard for many people. This disruptive immune reaction can be caused by contact with an odd assortment of items, including plants, drugs, chemicals and even money¹. In each case, small, reactive molecules from the item penetrate the skin and modify proteins within the tissue. Thus altered, the skin proteins are seen as foreign by the immune system, which then endeavours to destroy them. This dermatitis is caused by an adaptive response²: the reaction occurs only if that specific antigen has been encountered before, and it is mediated by the archetypal white blood cells, or lymphocytes, of adaptive immunity — activated antigen-specific $\alpha\beta$ T cells. In contact sensitivity, the mouse experimental model of contact dermatitis, three other types of lymphocyte are implicated in the reaction: B-1 B cells, NK T cells and $\gamma\delta$ T cells². These cells are part of the fast-acting defences of innate immunity, which do not strengthen with successive exposures to the antigen. Notably absent from this list is the natural killer cell. So the report by O'Leary *et al.* in *Nature Immunology*³ is unexpected. They find that in mice lacking all other lymphocytes, natural killer cells play all the roles necessary to produce contact sensitivity.

Natural killer cells are different from other lymphocytes because during cell maturation they do not need to rearrange the genes for their antigen receptors (T-cell receptors or immunoglobulins). In mutant mice lacking the *Rag2* gene (*Rag2*⁻ mice), such gene rearrangement is blocked, so B cells, T cells and NK T cells do not exist, and natural killer cells are the only circulating lymphocytes. Unexpectedly, contact-sensitivity reactions against the chemical dinitrofluorobenzene (DNFB) (Fig. 1) are almost as strong in *Rag2*⁻ mice as in normal mice³. In exploring this observation, O'Leary *et al.* show that, in the absence of other lymphocytes, natural killer cells are necessary for the contact-sensitivity reaction. They also find the reaction to be antigen-specific, so that *Rag2*⁻ mice primed with DNFB respond to further contact with DNFB but not to a first

contact with a different reaction chemical.

Not too long ago, natural killer cells were considered to be all exactly the same and lacking any antigen specificity. But O'Leary *et al.* add to a body of evidence showing how specific subpopulations of natural killer cells are activated in response to bone-marrow transplants^{4,5}, viral infections⁶, tumours⁷ and, now, as part of contact-sensitivity reactions to chemically modified skin tissue³. Natural-killer-cell subpopulations are distinguished by their differential expression of a variety of activating and inhibitory receptors, of which the Ly49 receptors are notably diverse and variable among individuals⁸. Contact sensitivity to DNFB is mediated by the subpopulation of natural killer cells expressing Thy1, an activation marker, and either the Ly49C or Ly49I receptor. These cells concentrate in the liver³, where DNFB is probably detoxified.

The experiments of O'Leary *et al.* touch upon immunological memory, the property that has most clearly distinguished innate from adaptive immunity. Surviving a childhood infection with mumps or measles virus provides immunity from the disease for the rest of a person's life. In current thinking, this protection is provided by 'memory' B and T cells, as part of adaptive immunity, that emerge during the immune response to the infection and thereafter ensure a quick, strong and overwhelming response to further infection.

After priming *Rag2*⁻ mice with DNFB, O'Leary *et al.* show that a secondary challenge, made either 5 or 28 days later, gave a strong response (Fig. 1). Their interpretation, that priming resulted in a persistent memory mediated by natural killer cells, is provocative and should stimulate investigation. For example, the difference between 5 and 28 days is not a particularly demanding test of persistent memory, even for a laboratory mouse with a lifespan of around two years. And, in O'Leary and colleagues' experimental design, it is possible that the primary response to DNFB had not truly subsided by the time of the secondary challenge. Elimination of DNFB and of DNFB-mediated tissue components from the

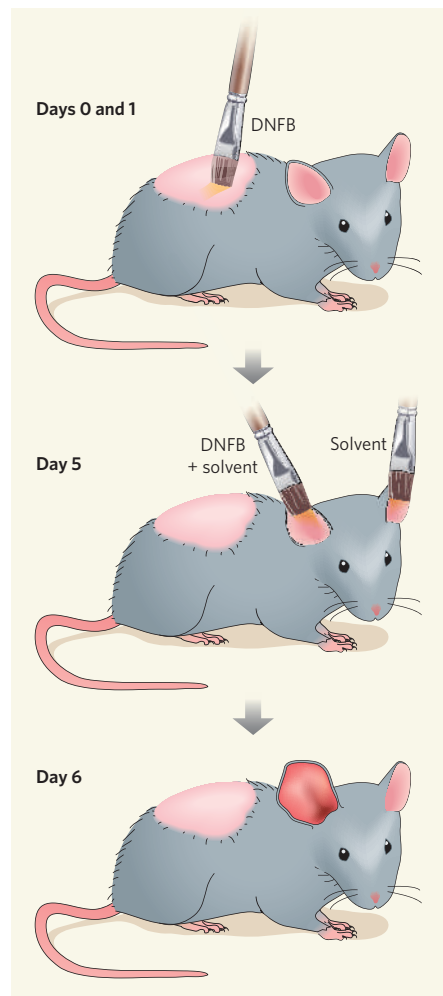


Figure 1 | Measuring contact sensitivity. The shaved back of a mouse is painted with a reactive chemical, such as dinitrofluorobenzene (DNFB). This first contact is the priming stage, in which the adaptive immune response to DNFB-modified tissue is induced. The second contact is made five days later, when one ear of the mouse is painted with DNFB and the other, the control, is painted with the solvent in which the DNFB is dissolved. One day after this secondary challenge the immunological reaction is measured by comparing the thickness of the two ears, as the ear painted with DNFB is enlarged by inflammation caused by infiltrating immune cells and plasma. O'Leary *et al.*³ found that the strength of the response to DNFB in *Rag2*⁻ mice, which have natural killer cells but no other lymphocytes, was comparable in strength and specificity to that in normal mice.

mouse's painted back could take considerable time, resulting in persistent stimulation, rather than the development of memory, throughout the experiment's duration. This sort of persistent stimulation is what causes occupational contact dermatitis in humans, for instance when sensitized supermarket clerks daily handle nickel-containing coins.

In the course of evolution, lymphocytes became more diverse and more specialized. But dependency accompanies specialization, as witnessed in the *Rag2*⁻ mouse — B cells, NK T cells and T cells are so reliant on their