investing time in study of $CD4^+$ cells, however, Tarleton has been building a case for the involvement of $CD8^+$ T cells. Previously, he showed that depletion *in vivo* of $CD8^+$ T cells with anti-CD8 monoclonal antibody rendered mice susceptible to *T. cruzi*, the infected mice dying within a month. $CD8^+$ T cells seemed to contribute to the establishment of protective immunity but not its long-term maintenance, because depletion of $CD8^+$ cells from chronically infected mice compromised neither control of the infection nor the capacity to resist reinfection[14].

The β_2m-negative mouse lacks $CD8^+$ T cells and thus provided Tarleton *et al.*[5] with a new system to assess their importance for defence against *T. cruzi*. The experiment was simple — infect animals with *T. cruzi* and see if they die — and the results were unequivocal: the β_2m-negative mice all die whereas their heterozygous and wildtype litter mates survive. As with other experiments on transgenic or 'knocked-out' mice, the interpretation is not so clear cut. It is safe to conclude that β_2m or β_2m-associated class I molecules contribute to resisting *T. cruzi*, but to invoke $CD8^+$ T cells means assuming that the only effect of β_2m is in the selection and function of $CD8^+$ T cells. This is a brave premise, and one that Tarleton *et al.* acknowledge is a simplification. In particular, class I MHC molecules affect the specificity and function of natural killer (NK) cells as well as $CD8^+$ cells[15], and NK cell development is compromised in β_2m-negative mice[16,17]. Natural killer cells are important during the early phases of many infections; as a good number of them express the CD8 molecule, one can make a case that the results of Tarleton and coworkers[5,14] are consistent with resistance to *T. cruzi* depending upon NK cells, as has already been suggested[11].

Resolution of this and other possible explanations will require attempted rescue of β_2m-negative mice from *T. cruzi* attack with infusions of defined cell populations. That $CD8^+$ cytotoxic T cells contribute to defence against *T. cruzi* is certainly plausible, given the parasite's life cycle. Amastigote replication takes place within the host cell's cytoplasm, the precise cellular compartment from which peptides presented by class I MHC molecules are commonly derived (see figure). Such presentation of *T. cruzi* antigens could stimulate $CD8^+$ cytotoxic T cells, leading to destruction of infected cells and interference with parasite replication.

Contrasting with the finesse with which the β_2m-negative mouse disposed of influenza virus[4], it seems to have just given up in the face of *T. cruzi*. On the evidence of Tarleton *et al.*, the mice became completely overrun with parasites before their defences could fire off a significant shot. Immune activities seen in normal mice seem to be attenuated or running at slower tempo in their β_2m-negative siblings: the induced immunosuppression was less, parasite numbers were higher and acute inflammatory reactions in muscular tissues much reduced. The incontrovertible message from Tarleton *et al.* is that, despite a façade of laboratory vigour, β_2m-negative mice collapse on confrontation with a hardened Third World parasite.

Despite success in identification of components of the immune system, immunologists are still a long way from a precise description of events following trespass of an infectious organism on mammalian territory. The black box standing between insect bite and heart failure in Chagas' has its equivalent in every infectious and autoimmune disease. Since the first description of the β_2m-negative strains, mice knocked out for an increasing number of immune components have been produced and the general result is viable, healthy, fecund mice. With judicious breeding of these mice, the immune system can gradually be pared away until the 'minimal mouse' — lacking immunoglobulins, T-cell receptors, MHC molecules, co-receptors, lymphokines and all the other weaponry of immunity — is left. The availability of such mice will create a new order in the analysis of host–pathogen relationships, in that they will provide a system in which the immune response can be reconstituted using purified proteins, cloned cells and one's chosen pathogen. □

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DAEDALUS

Pinched diamonds

DIAMOND is the most tantalizing of materials. It is uniquely strong, stiff, thermally stable and thermally conductive, and is transparent over an amazingly wide spectral bandwidth. Yet to synthesize it from graphite is absurdly difficult, usually needing clever catalysts and desperate extremes of heat and pressure. A recent synthesis has formed diamond as tiny fibres. Daedalus now plans to make diamond fibre by the kilometre.

His process exploits the pinch effect, which compresses a current-carrying conductor in its own magnetic field. A large current passed briefly through a catalyst-doped carbon fibre should heat it so strongly, and pinch it to such a pressure, that it would collapse to diamond. For a fibre 0.01 mm across, a mere five thousand amps should do the trick. Carbon fibre passed through two sets of rollers with a suitable potential between them could be continuously 'pinched' into diamond fibre, and wound on a receiving drum. The transformation is martensitic and very rapid; so the process could be run fast enough to make diamond fibre a bulk commodity.

Diamond fibre will be a wonderful new material. Plastics reinforced with it will be many times stronger and stiffer than rival composite materials. On diamond-fibre cables, suspension bridges and power lines will soar many kilometres in a single span. Wide-band diamond optical fibres will carry thousands of trashy TV channels simultaneously. On a more intimate scale, it will be woven into marvellous silky lingerie, sheer, lustrous, utterly glamorous — and also snag-resistant, stain-rejecting, indigestible to moths, impervious to heat, and bullet-proof. Indeed, diamond-fibre fabrics will revolutionize the design of flak-jackets, military uniforms, fire-blankets and protective clothing generally.

Variants on the basic product will also be possible. By running fine carbon tube through the pinch-mill, diamond hypodermic tubing should result. Similarly, a carbon-coated wire feedstock would give diamond-plated wire. With its marvellous mechanical strength, electrical insulation and unprecedented heat-transfer properties, it should be widely welcomed by the electrical and electronics industries. Furthermore, diamond-pinching could easily be scaled up. A few million amps could pinch a thick cylindrical graphite feedstock into diamond rod several centimetres across. It could be made into all sorts of novel diamond lenses, crucibles and cutting tools. But probably not jewellery. Bulk pinched diamond will be so cheap that nobody will want to advertise their poverty by wearing the stuff. David Jones