Research News

Monkey think, monkey do

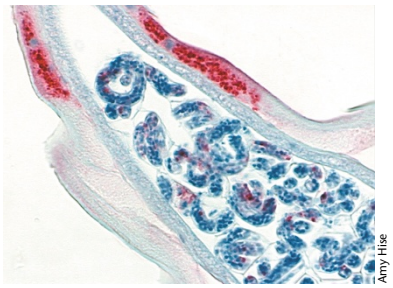
Monkeys with electrodes hooked up to only a few neurons in their brains can move a cursor on a computer screen by just thinking about it, using a new system devised by Mijail Serruya *et al.* and reported in the 14 March issue of *Nature*. To enable this feat, the researchers first took recordings from between 7 and 30 neurons that control hand movements, while trained monkeys moved their hands. The monkeys connected a cursor with a point on a screen and performed similar motor tasks. After providing the computer with information that associates neural signaling with cursor position, the researchers switched control of the apparatus to the brain. The monkeys were able to move the cursor almost as efficiently with their minds as they had with their hands. Moreover, the monkeys moved the cursor faster than predicted given inaccuracies in the algorithm translating neural signals into cursor movement, suggesting that the monkeys could compensate for these inaccuracies. The system has the potential to aid individuals with paralysis and other health problems that limit movement.

Non-Hodgkin lymphoma linked to SV40 virus

Two studies implicate the monkey polyomavirus SV40 in the development of non-Hodgkin lymphoma (NHL). In the March 9 *Lancet* Vilchez *et al.* report the presence of viral DNA in 42% of NHL, and in the same issue Shivapurkar *et al.* arrive at a similar percentage, 43%. Neither group detected SV40 in healthy individuals or saw a strong association between SV40 and epithelial cancers. Vilchez *et al.* also drew a link between SV40 in NHL and what may be a large source of SV40 in humans—SV40-infected polio vaccine. Three of ten viral samples showed high DNA sequence similarity to a strain first detected in a sample of contaminated vaccine from 1955. The new data also corroborate previous observations of other routes of exposure to SV40, as some of the individuals with NHL in the study were born after the use of contaminated vaccine was ended in 1963. These new studies indicate that SV40 likely plays a role in tumorigenesis in NHL, consistent with previous studies that show that SV40 can induce tumors in animals and is associated with some human brain and bone tumors.

Bacteria eyed in river blindness

River blindness strikes after small parasitic nematodes invade the eye. However, new research suggests that the primary culprit in vision loss is not the nematode, but a bacterium that lives inside it. The bacterium is the near-ubiquitous *Wolbachia,* which takes up residence in many arthropods and nematodes. In *Onchocerca volvulus*, the nematode that inflicts river blindness, *Wolbachia* serves as an essential endosymbiont (shown in red inside its host in this picture). In fact, recent research has shown that treatment of river blindness with antibiotics sterilizes the parasites and aids in treatment of the disease, which afflicts over 17 million people. In the in the March 8 issue of *Science*, Saint André *et al.* examined the source of vision loss in a mouse model in which nematode extracts are injected into the cornea. Extracts of *O. volvulus* lost much of their blinding power if the worms were first treated with antibiotics. Previous studies have indicated that blindness occurs when the body mounts an inflammatory response against the nematodes after they die and begin to degrade. The researchers found, however, that the inflammatory response required the Toll-like receptor 4, a host protein essential for the response to bacterial lipopolysaccharides. The researchers conclude that it's the host's immune response to *Wolbachia* that accounts primarily for vision loss.



Amy Hise

Nuclear transplantation gets therapeutic

Researchers have for the first time corrected a genetic disorder using a combination of gene therapy and 'therapeutic cloning', also known as 'nuclear transplantation'. Jaenisch and colleagues corrected mice mutant for the *Rag2* recombinase gene, required for the production of mature B and T cells, they report in *Cell* online on 8 March. To treat the defect, the researchers first generated embryonic stem cell lines by transplanting nuclei from tail tip cells of $Rag2^{-/-}$ mice into oocytes. They then introduced a wild-type copy of *Rag2* into these embryonic stem cells using homologous recombination, and induced hematopoietic stem-cell differentiation. These engineered cells restored partial immune system function when transplanted into $Rag^{-/-}$ mice. But the results were not flawless. The researchers found they needed to compensate for low expression of major histocompatibility complex in the hematopoietic progenitor cells by depleting natural killer cells in the host. Despite these and other barriers, the drawbacks were not attributable to the processes of gene therapy or nuclear transplantation themselves. That's only good news for researchers aiming to use the procedures for other disorders such as diabetes and Parkinson disease.

Arthritis reveals innate qualities

New data from a mouse model of the autoimmune disease rheumatoid arthritis (RA) indicate that disease development depends critically on the innate immune system. The end stages of RA include neutrophil and macrophage recruitment and cytokine release within inflamed joints, but the events preceding this scenario have been difficult to establish. Benoist *et al.* demonstrate that components of the innate immune system are essential for the RA that develops in wild-type mice following serum or immunoglobulin G (IgG) transfer from a mouse RA model (the transgenic T-cell receptor strain, K/BxN). The researchers report in the February *Immunity* that disease depends on the IgG-binding Fc receptors (FcRs), as well as mediators of the innate response, complement C5, C5a and the C5a receptor. Previously, it was thought that the FcR and innate arms of the immune system acted separately in RA, but these new data indicate they function in concert. In a reversal of standard thinking in which the innate (complement) immune system recruits the adaptive (IgG) response, in this case adaptive immunity recruits an innate immune response leading to autoimmune disease in RA. These insights offer new clues to potential therapies for the debilitating effects of RA.

Charlotte Schubert, Charlotte Wang & Paul Morin