› NEWS UPDATES

Novel antibody fights HIV in monkeys

Two papers published in the June issue of the *Journal of Virology* report a promising new approach to the production of antibodies efficacious against multiple HIV strains, a potential step in developing lifesaving immunotherapies against the virus that causes AIDS.

In the past, primary HIV strains have shown resistance to neutralization with laboratory-produced antibodies. Even antibodies that have demonstrated effectiveness against HIV have been limited by their specificity for a single HIV strain.

Now, by sequentially immunizing mice with synthetic peptides derived from the V3 region of six different strains of HIV, a group led by Mitsuo Honda of the National Institute of Infectious Diseases (Tokyo, Japan) was able to produce an antibody, dubbed KD-247, that cross-reacts with different HIV strains. Honda and colleagues showed that high levels of KD-247 protect male cynomolgus monkeys, *Macaca fascicularis*, from infection with a highly pathogenic HIV-derived simian immunodeficiency virus. Moreover, KD-247 even suppressed generation of some HIV strains in cells cultured from HIV-infected individuals.

Sheep lend further insight into prion transmission

New research in sheep raises interesting questions about prion transmission that could have important implications for understanding how mad cow disease is passed on to humans.

Prions are infectious, abnormally folded protein particles that cause bovine spongiform encephalopathy (mad cow disease) in cattle and Creutzfeldt-Jakob disease in humans. In sheep, prions cause scrapie, a fatal neurodegenerative disease that is known to be horizontally transmissible between sheep by ingestion of contaminated materials, a route very similar to the one implicated in cases of human infection with prion-contaminated beef. Not all sheep succumb equally to prions; some seem to have an inborn resistance to prion disease.

Hoping to shed more light on how prions enter the body from the digestive tract, Martin Jeffrey of the Veterinary Laboratories Agency-Lasswade (Scotland) and colleagues used a novel surgical procedure to introduce prion material directly into the small intestine of three different breeds of sheep (*J. Pathol.*, May). After sacrificing the sheep, the scientists examined them histologically for the presence of prions at various time intervals.

Notably, the prion-susceptible and prion-resistant sheep did not differ in prion uptake or prion localization after uptake. Moreover, *in vitro* experiments in which the prion-containing material was incubated with digestive enzymes resulted in prion degradation.

In combination these results suggest that prion infection may occur even before the contaminated food enters the stomach, perhaps even in the mouth. Another possibility is that genetic differences in digestive enzymes may be responsible for the differences in susceptibility to prion infection among sheep. In either case, further investigation into these possibilities could yield better treatment and prevention of prion diseases in humans.

Environmental estrogens linked to prostate cancer

Prenatal exposure to environmental estrogens may increase a man's likelihood of developing prostate cancer later in life, according to a new study in rats.

Estrogenic compounds, including bisphenol A (BPA), a chemical used in the manufacture of plastics and epoxy resins, are common environmental pollutants. BPA seeps out of polycarbonate plastic food and beverage containers and can be detected in the serum and in placental and fetal tissues; it may therefore affect developing reproductive tissues.

To investigate the effects of environmental estrogens on future prostate health, a research group led by Gail S. Prins of the University of Illinois at Chicago exposed neonatal male rats (at a developmental stage corresponding to the second and third trimester of human *in utero* development) to low-dose BSA or high- or low-dose estradiol (a natural estrogen). All three types of exposure led to a significantly increased incidence of adult-onset precancerous and cancerous prostate lesions (*Cancer Res.*, 1 June). Exposure to estrogenic compounds caused permanent changes in DNA methylation patterns, suggesting that estrogen imprinting may form the basis of this connection.