GLOSSARY

CERVIDS

Any ruminant mammals, including deer, that belong to the family *Cervidae*; distinguished from *Bovidae* by the presence of deciduous antlers in males

Perinatal infection with neurotropic viruses is associated with cerebral palsy

Many viruses, including herpesviruses and enteroviruses, can cross the placenta and infect the fetus. The virus might then cause neuronal damage and could, directly or indirectly, lead to the development of cerebral palsy. A group in Australia has investigated associations between potentially neurotropic viruses and cerebral palsy.

The study included babies born with cerebral palsy to white mothers in South Australia from 1986–1999 ($n = 443$). The control group consisted of 883 babies, born to white mothers in the same period, who were without cerebral palsy or other birth defects and who survived the first year of life. Polymerase chain reactions (PCRs) were used to detect viral nucleic acids in newborn screening blood samples that had been taken within a few days of birth and held for up to 18 years.

Results were obtained for 414 (93.5%) cerebral palsy samples and 856 (96.5%) control samples, though not all virus tests were possible for each of the samples. Viral nucleic acids were found in 298/749 (39.8%) control samples. Detection of any viral nucleic acid, in particular herpes PCR group B virus nucleic acids (varicella zoster virus, human herpesviruses 6 and 7), was associated with increased risk of cerebral palsy across all gestational ages (odds ratio 1.30, 95% CI 1.00–1.67 for all viruses; 1.68, 95% CI 1.09–2.59 for herpes PCR group B viruses).

The authors conclude that perinatal exposure to neurotropic viruses is associated with increased risk of cerebral palsy, though other factors are almost certainly required for the manifestation of the condition.

Rebecca Ireland

Original article Gibson CS *et al.* (2006) Neurotropic viruses and cerebral palsy: population based case-control study. *BMJ* 332: 76–80

Cerebral microhemorrhage implicated in progression of Alzheimer's disease

Cerebral vessel disease is reported to occur in up to 90% of Alzheimer's disease cases. Heme-rich deposits (HRDs) are present in large numbers in the brains of patients with Alzheimer's disease and Down syndrome. Cullen *et al.* proposed that HRDs are indicative

of cumulative damage, and that cerebral microhemorrhaging is a direct cause of neurodegeneration in Alzheimer's disease. They therefore investigated the relationship between HRDs and β -amyloid deposits in Alzheimer's disease and Down syndrome.

Brain tissue was used from 20 individuals—14 cases of Alzheimer's disease (2 of whom also had Down syndrome) and 6 controls. HRDs were found to colocalize with β -amyloid deposits; furthermore, both HRDs and β -amyloid deposits were relatively densely distributed in the cortex and sparsely distributed in the white matter. β -Amyloid protein was also found to colocalize with the collagen of blood vessels.

The results suggest that β -amyloid deposits form around small blood vessels, often at branch points. Abnormal capillary morphology and endothelial abnormalities have been described in Alzheimer's disease and have been implicated in disease progression. The authors suggest that rupture of cerebral blood vessels might lead to the deposition of blood-borne β -amyloid protein and formation of amyloid plaques in Alzheimer's disease. They also speculate that high levels of β -amyloid in blood might contribute to vascular damage; these hypotheses will be tested in future research.

Jim Casey

Original article Cullen KM *et al.* (2005) Microvascular pathology in the aging human brain: evidence that senile plaques are sites of microhaemorrhages. *Neurobiol Aging* [doi: 10.1016/j.neurobiolaging.2005.10.016]

Could chronic wasting disease be transmitted by contact with meat from prion-infected deer?

Prions are infectious, proteinaceous agents that interfere with the normal structure of host proteins, leading to degeneration of the CNS. The transmission of prion diseases between different species has led to concern regarding transmission of chronic wasting disease (CWD)—a prion disease of North American CERVIDS—from animals to humans. Angers *et al.* compared the ability of CNS and muscle tissue from CWD-affected deer to transmit prion disease to susceptible mice.

Transgenic mice expressing the cervid prion protein were given intracerebral inoculations of either muscle or CNS extracts from deer with or without CWD, and the length of