disease or Alzheimer's-type neuropathological changes, we suppose that the investigation of the catabolic system of Aβ is important for four reasons. Firstly, it links to elucidation of the mechanism of accumulation of Aβ. As NEP is thought to be a main peptidase which accounts for the degradation of Aβ in the brain,[1] it is necessary to examine the influence of the NEP gene on the severity of the senile plaques and dystrophic neurites to search for a role of clearance of Aβ in the deposition of Aβ. Secondly, this research contributes in clarifying a role for senile plaques and dystrophic neurites in the development of Alzheimer's disease. Thirdly, the detection of key molecules in the degradation of Aβ might directly lead to the treatment of Alzheimer's disease. Fourthly, recent analyses disclosed that families with late onset Alzheimer's disease are linked to genetic markers near the insulin degrading enzyme gene, which is thought to be one of the catabolic enzymes of Aβ.[6] Genes of the degradating enzymes of Aβ such as the NEP gene still remain potential risk factors for sporadic Alzheimer's disease. The examination of other polymorphisms in the NEP gene or multivariate analysis taking in the related gene except ApoE which modifies the processing of Aβ might detect potential correlation of the NEP gene with Alzheimer's disease.

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Human T lymphotropic virus type I (HTLV-1) associated myelopathy acquired through a liver transplant

Subacute myelopathy (HAM/TSP) is the main neurological manifestation of human T lymphotropic virus type I (HTLV-1) infection.[1] This virus is endemic in central Africa, Caribbean countries, and Japan. It is transmitted through sexual contacts, during lactation, and by blood transfusions. The risk of seroconversion after blood transfusion is 40%–60%. Around 5% of the carriers will develop clinical manifestations; only 0.3% of them will have a myelopathy. Inmunosupresion enhances the risk of infection, reduces the latency, and accelerates the clinical pictures. We are reporting the first documented case of HTLV-1 infection through an organ transplantation in a western country. In another organ recipient the vehicle of the virus was the blood transfused during the surgical procedure.[2]

A 44 year old woman developed alcoholic cirrhosis and hepatocarcinoma. On 5 October 1998, she received a liver transplant followed by cyclosporin treatment (175 mg/day). The donor was an apparently healthy young man who died after brain injury. Eighteen months later, the patient complained of progressive weakness in her legs. In the next 3 months a rapidly evolving paraparesis with a T6 sensory level, pyramidal signs, and bladder dysfunction became evident. She was admitted to another hospital. The CSF contained 37 white cells/ml, 93 mg/ml protein, and 43 mg/ml glucose. Serological tests for neurotropic virus were negative. On T2 weighted MRI a diffuse hypersignal of the cervicothoracic spinal cord was seen. The rest of the data from an extensive investigation were non-contributory. She was transferred to our institution on 3 August 2000. Other than a complete paraplegia no neurological abnormalities were found. Somatosensory evoked potentials after median nerve stimulation were normal but they were abolished after posterior tibial nerve stimulation. In the CSF there were 9 white cells/ml, 133 mg/ml protein, and 43 mg/dl glucose. Serological tests for HTLV 1 (enzyme linked immunosorbent assay (ELISA) and western blotting) were positive in blood and CSF, and the polymerase chain reaction was positive in blood. Tests were negative for HTLV 2 and VIH. The patient received a pulse of intravenous methylprednisolne (1g/day/5 days) and a course of α-interferon (3 MU/day/1 month)[3] without any improvement in her neurological status.

We have conducted a retrospective serological survey for HTLV 1 antibodies in archival blood samples from the patient before the transplantation, from the liver donor, and from the blood donors. All the samples were negative except those from the liver donor. He was a multiorgan donor (both kidneys, liver, heart, and corneas). A follow up of all the recipients is in progress.

The prevalence of HTLV-1 infection in the endemic areas is between 3% and 30%. In western countries it is less than 1%.[1] Despite of this low prevalence, several European countries (France, Holland, Sweden, Denmark, Luxembourg) and the United States have introduced a systematic search for HTLV-1 antibodies in their blood banks. Furthermore, in France the test for HTLV-1 infection is mandatory in all organ donors. In Spain, a serological survey conducted among 23 000 blood donors in 1992 detected only one suspected, subsequently not proved, carrier. Consequently, a routine test for HTLV-1 was not implemented. However, an ad hoc national registry reported 24 cases in Spain up to 1994.[4] Since then, three further cases have been found (V Soriano, personal communication). In Japan, Nakamura *et al*[5] reported that 15 out of 153 recipients of renal transplants were HTLV-1 positive. They did not develop HAM/TSP or any HTLV-1 related disorder during a follow up of 1 to 10 years. By contrast, the case we are reporting here indicates that HTLV-1 infection may have devasting consequences for some immunocompromised organ recipients. This emphasises the necessity for a systematic survey of its antibodies in all potential donors despite the low current prevalence of HTLV-1 infection in western countries.

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Relation between the high production related allele of the interferon-γ (IFN-γ) gene and age at onset of idiopathic Parkinson's disease in Japan

Although the pathogenesis of progressive degeneration of nigrostriatal dopaminergic neurons in Parkinson's disease remains uncertain, cytokines are thought to contribute to the development of the disease.[1] Interferon (IFN)-γ is one of the Th1 cell derived multifunctional cytokines and seems to influence neuronal differentiation and to increase in inflammatory and neurodegenerative diseases.[2] Immunohistochemical studies showed an increase of IFN-γ expression in nigral astrocytes of patients with Parkinson's disease.[3] This increase of IFN-γ concentration may be a trigger for the disease or a compensatory response. It was reported that IFN-γ producing capacity in whole blood cultures of untreated parkinsonian patients decreased compared with sex and age matched healthy controls.[4] This supports the idea that IFN-γ may increase in Parkinson's disease as a compensatory response. Concerning genetic polymorphisms in the IFN-γ gene, the production of IFN-γ measured in peripheral blood mononuclear cell cultures may correlate with dinucleotide CA repeat polymorphism in the first intron of the IFN-γ gene.[5] In vitro production of IFN-γ is higher in