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A Monthly Newsletter Summarizing Important Advances in Transfusion

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Detection and replication of pathogenic prion in skeletal muscle

Several epidemiological studies tend to confirm that new variant Creutzfeld-Jakob disease, a fatal neurodegenerative illness affecting humans, is caused by consumption of bovine meat from an animal suffering from mad-cow disease. To this day, it was believed that the infectious agent, an aberrant form of the prion protein, is found mostly in neural and lymphoid tissues. However, recent results from Stanley B. Prusiner's group from the University of California at San Francisco, published in the 19 March issue of the *Proceedings of the National Academy of Sciences of the United States of America*, seem to suggest that infectious prions can be found in muscles. Moreover, the same study demonstrates replication of the infectious prion in certain muscles of experimentally infected mice.

Bosque, P. J., et al., **Prions in Skeletal Muscle**, Proc Natl Acad Sci USA 99 (6) : 3812-3817 (2002) (DOI : 10.1073/pnas.052707499).

Prevalence of parvovirus B19 among blood donors

Parvovirus B19 infection is generally asymptomatic in healthy individuals. However, this virus can cause certain forms of anemia in immunocompromised persons. The results of Hitzler and Runkel (Hospital of the Johannes Gutenberg-University, Mainz, Germany) on the prevalence of parvovirus B19, published in the November 2001 issue of *Vox Sanguinis*, reveal that of the 28 972 blood donations screened, 255 were positive, according to a viral antigen detection method. However, when these 255 samples were subjected to nucleic acid detection of the virus' genome, only four turned out to be positive. These results confirm the intermediate prevalence of parvovirus B19 among blood donors, and unveil the lack of specificity of the antigen-based screening test used.

Hitzler, W. E., and Runkel, S., **Prevalence of Human Parvovirus B19 in Blood Donors as Determined by a Haemagglutination Assay and Verified by the Polymerase Chain Reaction**, Vox Sang 82 (1) : 18-23 (2002).

Sickle-cell anemia : a promising animal model of allograft and immunotherapy

Sickle-cell anemia is a genetic disorder resulting from deleterious mutations in the gene encoding β-globin, one of the protein chains of hemoglobin. In an article published in the March 1 issue of *Blood*, a research team led by David R. Archer from Emory University (Atlanta, GA, USA) demonstrates the efficacy of bone marrow allograft, accompanied by a tolerance-inducing immunotherapy, to effectively cure sickle-cell anemia in a murine model of the disease.

Kean, L. S., et al., **A Cure for Murine Sickle Cell Disease through Stable Mixed Chimerism and Major Histocompatibility Complex-Mismatched Bone Marrow Transplantation**, Blood 99 (5) : 1840-1849 (2002).

An animal model of gene therapy for β-thalassemia

Similarly to sickle-cell anemia, β-thalassemia is a hereditary disease involving the β-globin gene. In the 15 March issue of *Blood*, Sadelain's team, from the Memorial Sloan-Kettering Cancer Center (New York, NY, USA), report on a successful gene therapy modality in a murine model of β-thalassemia.

May, C., et al., **Successful Treatment of Murine β-Thalassemia Intermedia by Transfer of the Human β-Globin Gene**, Blood 99 (6) : 1902-1908 (2002).

An additional study tends to confirm the plasticity of hematopoietic stem cells

The results of Körbling's team (University of Texas M.D. Anderson Cancer Center, Houston, TX, USA) suggest that hematopoietic stem cells from peripheral blood are capable of differentiating into various cell types, including liver and blood vessel–lining cells.

Körbling, M., et al., **Hepatocytes and Epithelial Cells of Donor Origin in Recipients of Peripheral-Blood Stem Cells**, N Engl J Med 346 (10) : 738-746 (2002).