



### Volume 3, Number 1

#### **West Nile virus – infected birds: an early warning sign of upcoming human infections?**

To gain further insights on this emerging pathogen, Gupta et al. (U.S. Geological Survey, VA, USA) analyzed epidemiological data on the prevalence of infected birds and human infections per US county as a function of time in 2001 and 2002. Their results indicate that for a given area, the identification of infected birds early in the season when the virus is active increases the likelihood of human cases occurring.

Gupta, S. C., et al. (2003). **Early-season avian deaths from West Nile virus as warnings of human infection.** *Emerg Infect Dis* 9 (4): 483-484.

#### **Transmissible spongiform encephalopathies (TSE): Antibodies against prions are protective**

White et al., under the supervision of Simon Hawke (Imperial College, London, United Kingdom) demonstrate that prophylactic infusion of antibodies specific to the prion protein prevent encephalopathy in mice previously inoculated with an infectious prion in the abdominal cavity. However, treatment has to be started relatively early after infection, i.e., before infectious prions reach the central nervous system, in order to be efficacious.

White, A. R., et al. (2003). **Monoclonal antibodies inhibit prion replication and delay the development of prion disease.** *Nature* 422 (6927): 80-83. doi: 10.1038/nature01457.

#### **Hemolysis-induced vasoconstriction: Insights on the molecular mechanisms**

Sickle-cell disease, an inherited disorder affecting red blood cell hemoglobin, results in frequent peripheral vascular occlusions caused by the abnormal rigidity of sickled red blood cells. These episodes are often associated with hemolysis, that is, free hemoglobin in blood. A group led by Mark T. Gladwin (National Institutes of Health, Bethesda, MD, USA) presents results suggesting that nitric oxide inactivation by free hemoglobin plays a cen-

tral role in the peripheral circulation problems commonly encountered in sickle-cell disease patients.

Reiter, C. D., et al. (2002). **Cell-free hemoglobin limits nitric oxide bioavailability in sickle-cell disease.** *Nat Med* 8 (12): 1383-1389. doi: 10.1038/nm799.

#### **Are there bacteria in the blood of healthy individuals?**

Up until now, the vascular compartment of healthy individuals was thought to be devoid of bacteria. The results of a collaborative effort led by Eddie C. S. Chan (McGill University, Montreal) suggest that bacteria are found in low numbers in the blood of otherwise healthy individuals. Presumably harmless, the isolated bacteria are particularly difficult to grow, which may partly explain why they have remained elusive so far.

McLaughlin, R. W., et al. (2002). **Are there naturally occurring pleomorphic bacteria in the blood of healthy humans?** *J Clin Microbiol* 40 (12): 4771-4775. doi: 10.1128/JCM.40.12.4771-4775.2002.

#### **Gene therapy for anemia by controlled expression of erythropoietin (EPO)**

Two articles describing animal models of gene therapy designed for treatment of anemia have been recently published. Samakoglu, Bohl, and Heard (Pasteur Institute, Paris, France) succeeded in controlling EPO expression from a gene therapy vector using a pharmacological agent. Furthermore, Binley et al. (Oxford Biomedica (UK) Ltd., Oxford, United Kingdom) designed a gene therapy vector whereby EPO expression is auto-regulated in response to oxygen concentration in the tissue in which the vector is delivered.

Samakoglu, S., Bohl, D., and Heard, J. M. (2002). **Mechanisms leading to sustained reversion of beta-thalassemia in mice by doxycycline-controlled Epo delivery from muscles.** *Mol Ther* 6 (6): 793-803. doi: 10.1006/mthe.2002.0810.

Binley, K., et al. (2002). **Long-term reversal of chronic anemia using a hypoxia-regulated erythropoietin gene therapy.** *Blood* 100 (7): 2406-2413. doi: 10.1182/blood-2002-02-0605.