



#### **Efficacy of EPO to reduce blood transfusion requirements**

EPO (erythropoietin) is a biological product synthesized by specialized kidney cells in response to decreased blood oxygen concentrations. Its physiological effect is to stimulate red blood cell production by the bone marrow. Recombinant forms of EPO have been commercialized about fifteen years ago. Although primarily intended for the treatment of EPO insufficiency in renal failure patients, recombinant EPO is now increasingly administered to anemic patients undergoing chemotherapy treatments. Jerome Seidenfeld et al., from the Blue Cross and Blue Shield Association (Chicago, IL, USA) recently performed a meta-analysis of clinical trials assessing recombinant EPO treatment for chemotherapy-dependent anemia. The results suggest that EPO treatment slightly reduced red blood cell transfusion requirements in chemotherapy patients.

Seidenfeld, J., et al. (2001) **Epoetin treatment of anemia associated with cancer therapy: A systematic review and meta-analysis of controlled clinical trials.** J Natl Cancer Inst 93: 1204-1214.

#### **Contribution of IgG complexes to the therapeutic activity of IVIg**

Intravenous immunoglobulins (IVIg), the main therapeutic product derived from plasma fractionation, are essentially made up of immunoglobulin G (IgG). In addition to low quantities of IgM and IgA, IVIg often contain, especially after prolonged storage, measurable levels of IgG complexes. The relative contribution of these IgG complexes to the therapeutic activity of IVIg has recently been investigated by Wim K. Bleeker's team from Amsterdam University in the Netherlands. Using an animal model whereby platelet destruction is induced by infusion of an anti-platelet antibody, the authors of the study demonstrate that IVIg preparations containing up to 12% of dimeric IgG are more efficacious than IVIg preparations containing less than 4% dimers. Bleeker's group also provide data supporting the involvement of specific cell surface receptors found on white blood cells in the destruction of platelets and its inhibition by IVIg treatment.

Teeling, J. L., et al. (2001) **Therapeutic efficacy of intravenous immunoglobulin preparations depends on the immunoglobulin G dimers: Studies in experimental immune thrombocytopenia.** Blood 98: 1095-1099.

#### **Prion disease screening by a urinary test**

Bovine spongiform encephalopathy (BSE), or mad-cow disease, and its human form, variant Creutzfeldt-Jakob disease (vCJD), continue to be under active surveillance by public health authorities in Western world countries. Early diagnosis of these prion diseases remains a major challenge. In an article recently published in the Journal of Biological Chemistry, a team led by Ruth Gabizon, from Hadassah University in Jerusalem, Israel, report the identification of prion protein fragments in urine specimens of infected animals and CJD patients. In urine samples from infected animals, the presence of specific prion fragments preceded the onset of clinical symptoms. These results offers hope for the development of a relatively simple urinary test for prion disease screening.

Shaked, G. M., et al. (2001) **A protease-resistant prion protein isoform is present in urine of animals and humans affected with prion diseases.** J Biol Chem 276: 31479-31482.

#### **Prion disease treatment: A glimpse of hope?**

Transmissible spongiform encephalopathies (TSE), or prion diseases, remain incurable and are invariably fatal. The lack of treatment encouraged the research group headed by Stanley B. Prusiner (University of California, San Francisco, CA, USA) to screen a large number of pharmaceutical compounds that are normally used in the treatment of other ailments. This systematic approach led to the identification of two drugs that inhibit pathogenic prion protein replication *in vitro*. These results could soon lead to clinical trials assessing the efficacy of these drugs in human prion diseases.

Korth, C., et al. (2001) **Acridine and phenothiazine derivatives as pharmacotherapeutics for prion disease.** Proc Natl Acad Sci USA 98: 9836-9841.