

## Supplementary Abstracts

### Disseminated Intravascular Coagulation

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ALTERED SUSCEPTIBILITY IN THE CHEDIAK-HIGASHI SYNDROME (ALEUTIAN) MINK TO THE GENERALIZED SHWARTZMAN REACTION. T.G. Bell, W.E. Carter, R.J. Aulerich, J.C. Mattson and G.A. Padgett. Departments of Pathology and Animal Science, Michigan State University, East Lansing, MI.

The Aleutian mink is known to have many characteristics similar to other species with Chediak-Higashi Syndrome (CHS) and is considered to have a form of CHS. The CHS mink is partially characterized by defective platelets that lack nucleotide-serotonin dense body storage and by impaired leukocyte function where chemotaxis, degranulation and bacterial killing are decreased. A normalization of CHS leukocyte microbicidal activity *in vitro* has been demonstrated following the addition of normal platelets. The influence of these platelet-leukocyte defects on the endotoxin induced generalized Shwartzman reaction (GSR) has never previously been examined.

A study was undertaken to determine differences in the endotoxin dose required for GSR induction and in clinical, gross and histopathologic findings in normal and CHS mink. Twenty mink, half normal and half CHS were administered 55:B5 *Escherichia coli* endotoxin or saline intraperitoneally followed by a second identical injection 21 hrs. later. Forty-eight hrs. later or at the time of death all animals were necropsied and sampled for histopathologic examination. The results revealed that CHS mink were more susceptible to lethal effects of endotoxin and had different signs and lesions. The dual dosage ranged from 200 µg to 25 mg/kg. CHS mink were symptomatic at 400 µg/kg and were killed at 12.8 mg/kg or greater. Normal mink were symptomatic at 800 µg/kg and died at 25.6 mg/kg. Splenohepatomegally with extravasation was present at a dose of 1.6 mg/kg in CHS while it was not a lesion of normal mink or control CHS mink. Renal tubular necrosis was present in CHS mink. Thus, CHS mink were more susceptible to GSR induction which may be the result of CHS platelet/leukocyte dysfunction manifest after endotoxin induced vascular injury.

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ENDOTOXIN AND BLOOD COAGULATION IN PATIENTS WITH TRAUMATIC-HAEMORRHAGIC AND BACTERIOTOXIC SHOCK. A. Stemberger, F. Straßer, G. Blümel, B.v. Hundelshausen\*, S. Jelen\*, O. Schmid\*, G. Tempel\* Institute of Experimental Surgery and \*Institute of Anaesthesiology of the Technical University Munich, FRG

Blood coagulation and fibrinolysis was carefully monitored in patients suffering from traumatic-haemorrhagic shock. Parameters like antithrombin III (AT III), factor X (IIa), plasminogen, fibrin degradation products, antiplasmin and fibronectin were measured. It could be demonstrated that a severe haemorrhagic shock correlated with an activated blood coagulation and stabilisation of circulation was followed by normalisation of blood coagulation.

However in patients developing septic complications new disorders in blood coagulation were observed. The appearance of endotoxins was measured by a recently developed limulus amoebocyte lysate (LAL). Best results were obtained by diluting heat treated heparinized plasma samples followed by an incubation step with the X a substrate S-2222. Detection of less than 0.2 ng endotoxin/ml plasma could be achieved. The available data of 30 patients with traumatic-haemorrhagic and bacteriotoxic shock showed that the appearance of endotoxin mostly correlated with a decrease of AT III and fibronectin.

In conclusion, AT III, fibronectin and endotoxin may serve as sensitive markers in the early diagnosis of sepsis.