PROBIOTICS RESTORING THE PATTERN OF APICAL JUNCTION COMPLEX PROTEIN (AJC) EXPRESSION IN THE INTESTINAL MUCOSA OF DOGS WITH INFLAMMATORY BOWEL DISEASE

G. Rossi*, A.E. Jergens†, J.S. Suchodolski†, G. Pengo‡, A. Piccinini* and G.E. Magi*

**School of Veterinary Medical Sciences, University of Camerino, †Texas A&M University, College Station, Texas, USA and ‡Practitioner, Cremona, Italy*

Introduction: The apical junction complex (AJC) plays a significant role in regulating epithelial paracellular permeability.

Materials and Methods: We have studied by immunohistochemistry the distribution of tight junction components (claudin-2, occludin) and adherens junction (E-cadherin) proteins in the normal intestinal tract from three control dogs (CD) and from 10 dogs with IBD, before and after a probiotic treatment (VSL#3, VSL Pharmaceuticals, Inc; 450 billion lyophilized bacteria daily for 60 days).

Results: In the CD group, occludin-specific labelling was most intense at the epithelial cell AJC and appeared uniformly expressed throughout the epithelium of the small and large intestine, but in the IBD group a weak to absent expression was observed in luminal epithelium and in some intestinal glands of the small intestine. No differences in the distribution or labelling intensity of E-cadherin were observed between normal and affected dogs. In the CD group, claudin-2 was detected in the duodenal epithelium and glands and in the colonic crypt epithelium, decreasing in intensity from the distal to the proximal crypt and becoming barely detectable at the luminal surface of the colon. However, claudin-2 expression was increased in the proximal crypt and luminal epithelium of all dogs with IBD.

Conclusions: The observation that the expression and distribution of occludin and claudin-2 were restored after VSL#3 treatment may provide insight into the effects of probiotics on intestinal barrier function.

PATHOLOGICAL EFFECTS OF APPLICATION OF SILICIUM CARBIDE ENGINEERED NANOPARTICLES

T. Ristoski*, V. Vasilevska†, K. Mladenovska‡ and A. Grozdanov§

**Faculty of Veterinary Medicine, University 'Ss Cyril and Methodius', Skopje, †Clinic of Surgery 'St. Naum Ohridski', University 'Ss Cyril and Methodius', Skopje, ‡Faculty of Pharmacy, University 'Ss Cyril and Methodius', Skopje and §Faculty of Technology and Metallurgy, University 'Ss Cyril and Methodius', Skopje, Macedonia*

Introduction: Nanotechnology is an emerging science and the research on the new nanotechnologies has been extensive. In this study, the influence of silicium carbide (SiC) nanoparticles on haematological and histological parameters was analyzed.

Materials and Methods: The toxicity of SiC nanoparticles on 4-week-old female Wistar rats was investigated. The rats were divided into four groups: two control groups (one control, not-treated group and one vehicle-control group) and two experimental groups treated with different dose of nanoparticles (1 g/kg and 5 g/kg body weight). Animals were killed after 2, 7 and 14 days of exposure. Biochemical and histopathological examinations were performed.

Results: Loss of weight and a significant increase in platelet count occurred in both experimental groups, as well as significant changes in serum LDH. Histopathological investigations showed hepatocyte degeneration, stasis with microhaemorrhages, and lymphocyte and eosinophil infiltration in almost all tissues examined.

Conclusions: Toxicological effects are provoked by exposure to SiC nanoparticles.

THE EFFECT OF INTENSE PULSED LIGHT IN MOUSE SKIN

D. Rodrigues, A. Gama, M.J. Pires, B. Colaço and P.A. Oliveira

Department of Veterinary Sciences, UTAD, Vila Real, Portugal

Introduction: The use of intense pulsed light (IPL) devices has increased significantly and there are few studies about the side-effects of this type of treatment. The aim of this study was to understand the influence of IPL in normal mouse skin and in the development of precancerous skin lesions using an animal model of skin carcinogenesis.

Materials and Methods: Animal procedures were performed in accordance with the European Guidelines. ICR female mice were used and they were divided into: group I, exposed to the skin carcinogenesis initiator 7,12-dimethylbenz(a)anthracene (DMBA), without further treatment; group II, subjected to IPL treatment after DMBA initiation; group III, exposed to the promoter 12-o-tetradecanoylphorbol-13-acetate, after DMBA initiation; group IV, exposed only to IPL and group V, the control group. The experimental protocol lasted 25 weeks.

Results: At the end of the experimental protocol, 87 papillomas and seven squamous cell carcinomas were identified in group III (DMBA + TPA). Histologically, it was observed that IPL was not linked to neoplastic development, but dermal fibrosis was identified (groups II and IV). Nevertheless, 50% of the animals in group II developed epidermal focal hyperplasia, a result that may suggest IPL as a promoter of carcinogenesis.

Conclusions: Further studies are required in order to confirm these results and to verify the hypothesis that IPL is a promoter of carcinogenesis.

BRAIN DEVELOPMENT IN A MOUSE MODEL OF MUSCLE–EYE–BRAIN DISEASE

H.S. Booler, J. Williams and S.C. Brown

Neuromuscular Diseases Group, Department of Comparative Biomedical Sciences, Royal Veterinary College, London, UK

Introduction: Normal brain development is a complex, tightly regulated, multistep process, which requires the formation of a stem cell pool, division and differentiation of these progenitors into neurons and glia, and the ordered migration of post-mitotic neurons. Type II lissencephaly (also referred to as cobblestone lissencephaly) is a neuronal migration defect pathognomonic of a group of severe congenital muscular dystrophies, which are characterized by brain, eye and muscle defects. These disorders include Walker-Warburg syndrome (WWS) and muscle–eye–brain (MEB) disease, and show a range of substantial structural brain abnormalities associated with defects in neuronal migration. They represent the severe end of the phenotypic spectrum of a heterogenous group of muscular dystrophies called the dystroglycanopathies. These are characterized by the hypoglycosylation of alpha dystroglycan and vary in severity from the severe congenital muscular dystrophies (such as WWS and MEB) with substantial structural brain and eye defects, through to milder limb girdle muscular dystrophies. To date, more than 14 genes have been implicated in the glycosylation of alpha dystroglycan. Fukutin-related protein (FKRP) is one of these genes.

Materials and Methods: In this developmental study, we investigated the pathogenesis of brain lesions in the $FKRP^{KD}$ mouse, a model of MEB disease with an 80% knockdown in FKRP expression. Work from other groups involving mouse models of dystroglycanopathy has shown that the brain initially develops normally, but basement membrane defects begin to develop around E13.5 (Hu *et al.*, 2007).