within *S. ruminantium* populations. The rumen of herbivorous animals is a strongly competitive ecosystem. If similar variability, as seen in *S. ruminantium*, is observed in other ruminal bacteria, competition between bacterial species will be markedly affected by competition and diversity within the competing species.

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Lack of surface receptors rather than possession of a restriction-modification system determines F4 phage resistance in *Streptococcus bovis* II/1. I Štyriak, P Pristaš, P Javorský *(Institute of Animal Physiology, Slovak Academy of Sciences, Šoltésovej 4-6, Košice, Slovakia)*

The resistance of *Streptococcus bovis* II/1 strain, which produces the *Sby*I restriction endonuclease, to F4 phage infection was demonstrated by the double agar layer method. Although restriction endonuclease *Sby*I is able to cleave F4 phage DNA to numerous fragments in vitro, evidence obtained from adhesion experiments in vivo suggests that inhibition of adsorption is the most important defence mechanism in phage resistance of *S. bovis* II/1. Electron microscopy of phage-host mixtures showed many phage particles to be present on the surface of a phage-sensitive control strain *S. bovis* 47/3, whereas no phage particles were seen on the surface of cells of the phage-resistant strain *S. bovis* II/1.

ANTI-NUTRITIONAL FACTORS

Biotransformation of toxic substances by rumen microbial ecosystem. P Javorský[1], P Pristaš[1], A Lauková[1] J Legáth[2] *([1]Institute of Animal Physiology, Slovak Academy of Sciences, Šoltésovej 4-6, 04001 Košice, Slovakia; [2]University of Veterinary Medicine, Komenského 73, 04001 Košice, Slovakia)*

The liver and kidney are generally regarded as main tissues for detoxification of toxic substances absorbed by animals. In ruminants, all potential poisonous substances enter the rumen before their passage to the lower parts of the digestive tract or before direct transport into the blood via the rumen wall or via the rumeno-hepatic circulation. The chemical composition of toxic substances following entry into the rumen can be very variable. Toxic substances which could enter the rumen through feedstuffs or by water include plant toxins and fungal or bacterial toxins which are a part of the natural microflora of grains. Farm and wild ruminants are, however, at the risk of exposure to inorganic and organic environmental pollutants, including pesticides, especially in countries with intensive industrial and agricultural production. The risk for exposure of wild ruminants to pesticides increases with the intensive chemical protection of forests.

We have paid particular attention to the mechanism of ruminant intoxication by the insecticide supermethrin, frequently used in agriculture against various species of insects, and the effect of PCBs presented in the silage pit coatings on the metabolism of silage lactogenic inoculants and, subsequently, on the mixed populations of

ruminal bacteria.

The effect of the pyrethroid insecticide supermethrin on the growth and cyanogenic activity of eight ruminal bacterial species *Streptococcus bovis* AO 24/85, *S. xylosus* 310, *Enterococcus faecium* 2, *Lactobacillus plantarum*, *Megasphaera elsdenii* 4MJ, *Selenomonas ruminantium* A17, *Fibrobacter succinogenes* 16J and *Bacteroides ruminicola* 3/3 in pure culture was examined in this study. Bacteria grown in two concentrations of supermethrin (0.66 and 6.6mg ml^{-1}) showed a similar growth rate and resistance to supermethrin. Production of $cyan^-$ from supermethrin occurred in all strains of ruminal bacteria examined, but the enzymatic activity varied considerably both with species and with the concentration of supermethrin.

In our experiments with the lactogenic strains *L. plantarum* S20 and S15, we found no effect of Delor 105 in concentrations 0.2, 2.0 and 20μg ml^{-1} on growth and lactic acid production. The mixed ruminal microorganisms cultured in RGC medium were resistant to 0.2 and 2μg ml^{1} Delor 105. The concentration of total volatile fatty acids were not significantly changed. Although the PCB concentrations detected in silage did not affect the silage preservative lactogenic bacteria and mixed ruminal bacteria, their cumulative toxic effects on the animal and, through the food chain, on humans, are still of considerable importance.

Survival in the face of tannins - a microbial perspective. JD Brooker, IK Skene, L O'Donovan, J McCarthy (*Animal Science Department, University of Adelaide, Waite Campus, Glen Osmond, SA 5064, Australia)*

Tannins represent a complex group of anti-nutritional plant phenolic substances that inhibit effective utilisation of many potentially valuable forages by browsing and grazing animals, especially in the arid regions of Australia. These compounds form indigestible tannin-protein complexes, cause the loss of minerals by chelation, inhibit rumen microbial growth and may have direct toxic effects on the rumen epithelium. However, some wild ruminants have developed a unique resistance to tannins that enables them to utilise woody shrubs and other forages. Domestic ruminants do not enjoy this advantage. Experimentally, crude inoculations of total rumen bacteria from feral goats to domestic ruminants have shown that this capacity is transferable, but more detailed information is needed on individual bacterial species and the molecular mechanisms of tannin resistance.

From feral goats browsing Acacia, we have isolated and identified two rumen microbial species than can tolerate high levels of tannins and may derive energy for growth from metabolism of these compounds. One species, *Streptococcus caprinus*, hydrolyses protein tannin complexes and produces pyrogallol from the decarboxylation of gallate, the major component of hydrolysable tannins. This species also produces large quantities of extracellular polysaccharide (EPS) in the presence of tannins. Tannin induction of EPS biosynthesis in *S. caprinus*, and identification of key genes involved is currently under investigation.

The other ruminal species, *Selenomonas ruminantium* K2 expresses an esterase activity that has been identified as tannin acyl hydrolase (TAH) (EC.3.1.1.20). This hydrolyses the ester