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Short Communication

Digestive Troubles and Nutritional Toxicosis in the Laboratory Rabbits

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Troubles with digestion

Carbohydrates in rabbit feed can be divided into two groups: the readily available carbohydrates (RAC., e. g. sugars and starch) and those, participating in fibre structure. Feeding too much RAC increases the incidence of enterotoxaemia. This can be explained by the carbohydrate overload of the hind gut, which is in actual fact, a fermentative dysbiosis. The latter will develop, if the feed mixture contains high amount of starch (e. g. corn grain) and part of it cannot be digested in the small intestine and will serve as nutrient for the caecal bacteria. Too small (fine) particle size facilitates the process. Pathologic fermentation in the caecum favours the multiplication of pathogenic bacteria (among others for the iota toxin producing *Clostridium spiroforme*). The absorbed iota-toxin will cause lethal enterotoxaemia (hemorrhagic inflammation of the caecum, watery diarrhoeas causing exsiccation, paralysis of the central nervous system). Increasing the fibre level up to 12 to 15% minimized the occurrence of the disease. It is not clear, that the effect derives from the energy diluting (starch concentration decreasing) influence ("per se" effect), or from the specific characteristics of the fibre. Nevertheless, it is highly probable that it is not sufficient to minimize the presence of starch, but also important are the fibre fractions. (Efforts to prevent the enterotoxaemia by buffer - sodium bicarbonate, zeolites, MgO - supplementation had trifle or negative results).

The sufficient "indigestible fibre" is important not only to prevent diarrhoeas, but also in maintain optimum growth rate and to exclude fur chewing. It is highly important to differentiate the effect of fibre on the digestibility of nutrients and its general, positive dietetic influence on the whole organism. The sum of the dietetic effects (maintenance of the healthy peristalsis of the gastro-intestinal tract, adsorption of toxin in the gut lumen, assuring the natural feeling of fulfilment, the so-called feeding comfort, giving substrate for the microbial fermentation and volatile fatty acid production in the caecum) up to a certain fibre level (20% crude fibre) may be higher than the negative consequences of the decreased digestibility of nutrients. Over 22% of crude fibre the incidence of caecum impaction and constipation is higher.

Fibre deficient diets - especially at angora rabbits - facilitate the formation of gastric hair ball (zootrichobezoar) To prevent mortality due to diarrhoea the following minimum fibre levels are recommended: 15.3% ADF (acid detergent fibre), 11.7% crude fibre and 11.7% indigestible fibre. The required fibre level should be set by combining the natural ingredients or by adding straw meal.

Above 24.6% ADF concentration the live weight gain decreases. Each per cent ADF in the feed mixture decreases the digestibility of dry matter (-1.17% unit), organic matter (-1.32% unit) and crude protein (-0.64% unit). at higher fibre level even the digestibility of crude fibre will be lower. feed composition also influences the volume and mass of the stomach (parallel to the high fibre-low starch diet the intake will be larger); lower than 11.7% ADF causes the enlargement of the caecum, because caecotrophy is not practised. The starch has no separate dietetic effect: in case of a sufficient fibre supply even 25% starch has no harmful effect on the digestive processes. After fasting the normally 67 - 72 mmol/kg caecal content VFA level dramatically declines to 10 mmol/kg.

The pathogenesis of the diarrhoea of weaning rabbit may also be explained by the high alkali value of the solid feed. (Alkali value of a feed is equivalent to the amount of HCl (in mmol/kg) which is able to set the pH of 1 kg feed on pH 3). Too alkaline diet means that the

bicarbonate overload is increasing the blood. i.e. HCO^3-concentration, which, in turn, will be excreted into the caecum. The developed high caecal pH (6.5 instead of the 6.0 - 6.2) attenuates the local antibacterial effect. The final result may be the multiplication of pathogenic bacteria (e.g. *E. coli*), diarrhoea, death [1]. Instead of the optimum alkaline values most of the feeds have a value of 500 - 700. The bacterial imbalance in the caecum and the intake of feed high in protein may lead to the putrid dyspepsia.

The supplementation of the drinking water with 20 ml/l acetic acid, or 20 ml/l lactulose (Lactulose = 1,4-fructos-galactosid, a non-fermentable and indigestible disaccharide, able to bind the NH^3 in the gut lumen) may decrease mortality rates; the lactic acid proved to be ineffective. Animals, drinking water with acetic acid, had a lower than 6.5 to 6.2 pH in the caecum. The described protective effect was even sufficient in case of ampicillin provocation. Actually, the supplementation of drinking water with 18.75 mg/100 ml ampicillin (or other broad spectrum antibiotics) for some days will cause diarrhoea and mortality among the weaning rabbits. The intake of antibiotics was approximately 10 mg/rabbit/day. Ampicillin acts by upsetting the intestinal balance of the bacteria.

Feed toxicity

The pathogenesis of the peroral antibiotic-associated enterotoxaemia (ampicillin, lincomycin, clindamycin etc.) and the clinical signs like diarrhoea can be explained by the reduction of number of the predominantly Gram positive anaerobic bacteria (in the majority *Bacteroides*). Parallel to their lower number the concentration of the produced non-dissociated volatile fatty acids, and the antimicrobial effect falls. This antibacterial effect is the strongest in pH range between 6.0 - 6.5, with a volatile fatty acid concentration of 100-2000 mmol/kg caecal content. Thus, the use of antibiotics (especially penicillin family) can change the intestinal flora and disrupt their natural balance. Reduced competitive protection of indigenous intestinal flora permits the overgrowth of pathogenic *E. coli, S. typhimurium, Proteus, Shigella, Klebsiella* and *Clostridia*) and proliferation of their toxins, resulting in fatal enteritis and diarrhoea.

To the antibiotic narazin (ionophor type coccidiostatics) rabbits are extremely sensitive, the LD^{50}-value is low, 11.9 mg/kg LW [2]. In practice toxicosis may occur when poultry premix is added to the rabbit feed: After the compound is taken in, uncertain, uncoordinated movement and the flaccidity of the limbs develop. In some cases, neurological signs (tonico-clonico cramps, spastic contractions, salivation and the side-position of the head) appear and some of the animals will die. Necropsy reveals pale fields in the heart and muscles (Zenker-type myodegeneration, necrosis of the myofibrils) and lympho-histiocytic infiltration.

Mycotoxicosis

Mycotoxicosis are diseases, caused by toxins ("mycotoxins") produced by fungi (moulds) and ingested mostly by the feed. The best known toxins are the aflatoxins (B^1, B^2, G^1 and G^2), produced by the *Aspergillus flavus* and *A. parasiticus fungi*. Intake of feed, containing 350 ppb (ppb=g/kg) aflatoxin B^1 may cause chronic aflatoxicosis in growing rabbits. The decline in feed intake is 30, in weight gain 70%. After 3 weeks the first mortality cases occur, and at day 45 it reaches 20 - 30%. Sick animals lose weight, practise fur chewing and the number of leukocytes fall. Necropsy reveals hepatitis and splenomegaly. Subacute B1-alfatoxicosisra (0.06 - 0.09 mg/kg LW/day per os) can be characterized by diffuse hepatic necrosis, disturbed blood clotting and disseminated intravascular coagulation.

Ingestion of citrinin, produced by *Penicillium* and *Aspergillus* spp. Develops diarrhoea and nephrosis in rabbits. The T-2 toxin (and its metabolites) is produced by the *Fusarium sporotrichioides*, *F. poae* and *F. tricinctum* mould species. The damage of brain tissue is characteristic for the fusariotoxicosis of rabbit (and horse). Ingestion of feed of 0.024% vomitoxin (deoxynivalenol, DON) causes 100% embryonic mortality; T-2 toxin is excreted by the milk, causing death of suckling pups. VányI and co-worker [3] in acute and subacute peroral test have found the necrosis of lymphoid cells in the mucous membrane of the small intestine and the depletion of "B" and "T" dependent zones of lymphoid organs (spleen, lymph nodes, bone marrow, sacculus rotundus). The necrosis of MPS (mononuclear phagocyte system) cell in the liver is proportional to the amount of ingested toxin. The LD^{50}-value of T-2 toxin in rabbit is between 1 and 4 mg/kg LW. It means a high sensitivity towards trichotecen-structured mycotoxins, thus the rabbit may serve as a good model for their research. Long-lasting subtoxic peroral intake (1 mg/kg LW/day) attenuates the capacity of the liver to metabolize and the sensitivity of the organism increases. Feeding pellets of subtoxic (12.5 and 25 mg T-2 toxin/kg DM) decreases the voluntary feed intake by 60 to 70% (similar to the pig's feed refusal). The water content of the faeces from animals under toxin influence increases by 10%. The T-2 toxin (and its metabolites) concentration in the urine, faeces and caecotrophy are proportional to the intake; consequently, from the analysis of the mentioned biological samples the mycotoxin state of a group of animal can be evaluated [4]. By reingestion of the caecotrophy the toxin gets once more into the stomach of the rabbit, which may explain the high sensitivity of this species to the mycotoxins. The intake of feed with a very low amount

(0.19 mg/kg feed) of T--2 toxin during some weeks, in female rabbits will decline the progesterone production of the yellow bodies in the ovaries (and may also lead to the collapses of the immune functions [5-11].

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