The abundance and morphology of mitochondria in enterocytes of chickens exposed to necrotic enteritis and treated with a probiotic

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The Problem

The avian small intestine is lined by a single layer of columnar epithelium, which acts both as a permeability barrier between the gut lumen and the internal milieu, and as an important mediator for nutrient digestion and absorption. The intestinal epithelium is composed of different types of cells each with a distinct function. Enterocytes have important roles in nutrient transport, metabolism, and epithelial barrier integrity maintenance. Ultrastructural examination of enterocyte tight junctions, microvilli and organelles (i.e. mitochondria) can reveal alterations in cellular function. Changes of mitochondrial morphology occur in response to cellular stress. In mice, intestinal infections cause disruptions of mitochondrial structure and function. Similar mitochondrial changes are likely to occur during gut infections in poultry. No previous studies have examined this issue.

How we investigated or researched the problem

A subclinical necrotic enteritis (NE) model was developed in broiler chicks to identify mechanisms involved in the pathogenesis, and evaluate the efficacy of a probiotic [*Bacillus amyloliquefaciens* H57 (H57)]. Ten-day old chicks were exposed to *Eimeria* vaccine via drinking water, and 5 days later to an administration of *Clostridium perfringens* cultures in feed. Transmission electron microscopy (TEM) of the ileum was employed to explore ileal ultrastructural changes associated with NE and H57.

Results

The exposure of broilers to subclinical NE caused lesions in the small intestine, which occurred at significantly higher degree in NE-challenged birds when compared to non-challenged H57 ($P<0.001$), and NE-challenged H57 ($P<0.05$) birds. TEM examination showed that most mitochondria were located apically within enterocytes. Preliminary results suggest that mitochondria are more abundant ($P<0.001$) in H57 treated birds than in all other groups, including controls. Birds in NE-challenged H57 treatment also appear to have higher ($P<0.05$) mitochondrial density (75/100 µm2) than NE-challenged birds (54/100 µm2). In control, H57, and NE H57 birds, most mitochondria were round or elongated, with mild or no structural damage. In NE-challenged birds, mitochondria more often were irregular in form, containing electron-lucent regions of matrix, and with indistinct, swollen or damaged cristae.

Implications / Conclusions

The role of mitochondria in avian gut health needs to be established. Mitochondria can undergo dynamic alterations to meet changing cellular needs or in response to cellular stress. These changes may result in lowered ATP production and an increase in ROS exposure. Evaluation of mitochondria within the enterocyte in birds with gut health problems will allow for a more accurate assessment of cellular metabolic efficiency and functionality. The preliminary results of this study suggest that the ultrastructure, distribution and density of mitochondria within enterocytes may be related to treatment group. It appears that dietary addition of H57 increased mitochondrial density and probably efficiency, as these birds had reduced clinical signs of NE.