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**Artikel:** The nomenclature of E. Coli : a problem for the pathologist

**Autor:** Pearson, G.R. / Wray, C. / McLaren, I.

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*Institute of animal pathology, University of Berne*

## DISECDYSIS IN SNAKES: NON-INFECTIOUS AND INFECTIOUS AETIOPATHOGENESIS, WITH SPECIAL REFERENCE TO THE FIRST VIRAL DERMATITIS

*O. Pagan, M. Müller*

Disecdysis – disorder in skin shedding – is a world wide disease complex of reptiles in captivity. Little is known about the aetiopathogenesis in snakes; a summary will be given. Non-infectious: – environmental disorders; – nutrition deficiency; – trauma; – neoplasia. Infectious disecdysis: – bacterial; – fungal; – viral: a myxovirus-like dermatitis will be described for the first time.

Knowledge of physiological skin shedding (sloughing) is very important for successful dermatology of snakes. Healthy snakes shed the epidermis periodically during their life; complete as a tube, often only partially (Boidae). The shedding starts always at the lips. The sloughing cycle is divided in three periods: during the period of inactivity, the epidermis is composed of the stratum germinativum, the stratum intermedium, and the stratum corneum; the synchronized mitosis in the stratum germinativum during the period of renewal results in a inner and an outer epidermal generation; the actual sloughing period is characterized by a split in the stratum corneum of the inner epidermal generation and the stratum intermedium of the outer one; the consequence of proteolytic enzymes and a reptile-specific lymphatic fluid. Any abnormal function in one of the sloughing periods or destruction of the skin cause a partial or generalized disecdysis.

A non-infectious etiopathogenesis in disecdysis is very frequent: a) disorders in care: low relative humidity or inadequate bathing facilities (low elasticity of the skin), low temperature (low metabolic activity) as well as lack of a suitable substrate (no scrubbing facilities) are well known factors. b) Nutrition deficiency: disecdysis often arises during importation, transport and maladaptation in cachectic animals (low vitellogenin, a lipo-protein). Amino-acids and vitamins are essential for collagen and keratin metabolism. Spontaneous rupture of the skin in Boidae (neck, thorax) is considered a vitamin C deficiency. c) Trauma: burns, usually from poorly protected heat sources, and bites by rodents (food) are common. Dermal fibrosis and cicatrice involve a disecdysis for life. d) Neoplasia: In a saw-scaled viper the dermis was infiltrated by a rhabdomyosarcoma (thoracal, dorsal) and superficial ulceration in a boa constrictor the giant cell sarcoma of the upper lip prevented the disengagement of the «old skin».

Primary or secondary infectious disecdysis: Pseudomonas and aeromonas were often isolated in exsudative, ulcerative pyodermatitis («Scale rot» of snakes). Intra- and subcutaneous granulomas and abscesses are common. Aspergillus spp. and Candida spp. were isolated in generalized necrotizing dermatitis. The complex «maladaptation-unhygienic environment-dermatitis-disecdysis» is of great importance.

For the first time we observed virus associated skin lesions in snakes (boa constrictor 2, ball python 1, anaconda 1). Clinical signs were segmental massive disecdysis, multiple adhered outer epidermal layers and a thin inner epidermal generation. By handling, the skin had ruptured. Histologically, spongiosis, ballooning degeneration and basophilic cytoplasmic inclusions of different size in the stratum germinativum with little lymphocytic infiltrations suggested a viral infection. Ultrastructural examination revealed a large number of filamentous and spheroidal extracellular mature virus particles of 140 to 320nm in diameter, which were enclosed in a cell membrane-derived envelope. Budding on the cell surface was very prominent. Cytoplasmic inclusions condisted of accumulated nucleocapsid strands (7–8nm in width). The irregular shape was a typical sign for myxovirus. It is not yet proven that this myxovirus infection corresponds with the classical paramyxovirus which causes proliferative broncho-pneumonia in snakes.

*Dep. Pathology, University of Bristol School, Bristol (United Kingdom)*

## THE NOMENCLATURE OF E. COLI: A PROBLEM FOR THE PATHOLOGIST

*G. R. Pearson, C. Wray, I. McLaren, E. F. Logan*

Until relatively recently it was believed that *E. coli* in the neonatal calf did not produce microscopic changes in the small intestine (Fisher & Martinez, 1976; Moon, 1978). However it was recognised that certain strains of *E. coli* produce enterotoxins which result in fluid secretion to the intestinal lumen (Smith & Halls, 1967; Nagy et al., 1979). Thus the recognised strains of *E. coli* up to the late 1970's were known as enterotoxigenic *E. coli* or ETEC and were not thought to be associated with pathological changes. However, in 1978, Pearson et al. identified pathological changes in the distal small intestine of anaesthetised calves challenged with an enterotoxigenic strain of *E. coli*. These findings were later confirmed by Bellamy & Acres (1979) and Hadad & Gyles (1982). Thus it was shown that some strains of enterotoxigenic *E. coli* were also enteropathogenic. These strains of *E. coli* were adherent to the mucosa but separated from the microvilli of the enterocytes by a gap of 200–300 nm when viewed in the transmission electron microscope (Pearson & Logan, 1982).

In the USA, *E. coli* were found associated with the colonic mucosa of calves with naturally occurring diarrhoea. The mucosa with adherent bacteria had a characteristic ultrastructural appearance, and the associated bacteria were called «attaching and effacing» *E. coli* (AEEC) (Moon et al., 1979). They were intimately attached to the enterocyte surface by 'cup and pedestal' arrangements of the cell cytoplasm, and microvilli were effaced. Similar organisms were also identified in the United Kingdom (Hall et al., 1985). These became known as enteropathogenic *E. coli* (EPEC) from similar organisms

identified in rabbits (Takeuchi et al., 1978) and humans (Rothbaum et al., 1983). Similar *E. coli* were identified in the samll intestine of naturally infected calves (Pospischil et al., 1987; Pearson et al., 1988) and experimentally inoculated calves (Wray et al., 1989). In addition some of these strains produce a toxin which is toxic to vero cells (Verocytotoxin (VT)) (Konowalchuk et al., 1977) and are now known as VTEC; some produce haemorrhage in the large bowel (Riley et al., 1983) and are termed enterohaemorrhagic *E. coli* (EHEC), and may also produce VT.

With this proliferation of names the description *E. coli* diseases has become extremely complex. Thus it is suggested that. *E. coli* associated with enteric disease should be termed either enterotoxigenic (ETEC), with the understanding that some of these strains may be associated with pathological changes, and «attaching and effacing» *E. coli* (AEEC). Unfortunately both types require ultrastructural examination for a definitive pathological diagnosis.

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*Institute of Pathology, Hannover Veterinary School, West-Germany*

# ALTERATIONS OF THE GUT ASSOCIATED LYMPHOID TISSUE (GALT) IN HARBOR SEALS (PHOCA VITULINA VITULINA) DURING THE EPIDEMIC IN 1988

*G. Pohlmeyer, J. Pohlenz, E. Liebler*

In 1988 the seal population of the northern European coasts was severely decimated by an epidemic associated with distemper-like clinical symptoms. At necropsy bronchopneumonia, depletion of lymphoid organs and degenerative or inflammatory changes of the liver were found (Breuer et al., 1988; Kennedy et al., 1989; Friedhoff & Pohlenz, 1989). As cause infection with a Morbillivirus (PDV = Phocine Distemper Virus) and with several secondary infectious agents is considered (Osterhaus & Vedder, 1988; Liess et al., 1989; Kirchhoff et al., 1989). Since GALT has been recognized as a major pathway for entry of infectious agents into the host (Owen, 1983), it was the objective of this work to examine involvement of the intestine in the epidemic by morphological investigation of gut mucosa, especially of GALT.

## Material and methods

Intraluminally fixed intestines or intestinal specimens from 33 diseased seals and from one healthy control animal were examined macroscopically and/or by light microscopy (LM), scanning electron microscopy (SEM) and transmission electron microscopy (TEM). Distribution and amount of aggregated and solitary lymphoid follicles were determined. LM, SEM and TEM were performed on specimens from jejunal and ileal Peyer's patches and from solitary and aggregated lymphoid follicles of colorectum.

## Results

In phocine small intestine about 20 Peyer's patches and an individually varying number of solitary lymphoid follicles are present. In large intestine solitary follicles are distributed irregularly, patch-like aggregations occur in the middle of colorectum.

Paramyxovirus and Reovirus each were detected in the intestinal epithelium of three animals. Trematodes, Cryptosporidium and bacteria were present as single or combined infections in several animals. Infiltration of lamina propria with granulocytes was found in the intestines of most animals. In several seals villus atrophy and/or crypt abscesses were observed.

Compared to the control animal lymphoid follicles in small intestines of infected seals were characterized by varying degrees of depletion and reduced numbers of intraepithelial cells associated with M cells in the FAE. In four animals, three of which had Paramyxovirus inclusions in gut epithelial cells and intraepithelial cells, dome epithelium consisted mainly of immature epithelial cells. Findings in the large intestine were similar. Depleted lymphoid follicles were demarcated indistinctly and epithelium was relapsed in varying degrees. FAE was frequently composed of cuboidal immature epithelial cells.

## Discussion

Whereas symptoms of gastrointestinal disease were not reported to be clinically prevalent, our examinations revealed alterations of intestinal mucosa and of gut associated immune system and the presence of several microorganisms, some of which known to be pathogenic. Main finding was a depletion of GALT of varying degree. An infection with PDV is most likely to be the cause. In the course of Canine Distemper inflammation of the intestinal tract (Potel, 1951; Cornwell et al., 1965; Appel, 1970) as well as depletion of lymphatic tissue and necrosis of lymphatic cells are well known to occur (Stevens & Osburn, 1976; Krakowka et al., 1980). A preexisting immunosuppression of any other origin might have promoted an infection with PDV as well as with other pathogens. The presence of predominantly immature dome epithelial cells in