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inoculated with a rotavirus of low virulence

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Table 1: Cause of death

Neoplasia	15	
Salpingo-peritonitis	12	
Chronic hepatopathy	11	
Cardiac failure	5	
Uricosis	2	
Marek's disease	1	
Coligranulomatosis	1	
Tuberculosis	2	
Aspergillosis	- 1	
Intestinal volvulus	1	

Table 2: Percen	tage of lesions	of the different	apparatuses

8	TT
Digestive tract	93%
Cadiovascular system	84%
Urogenital system	82%
Endocrine and Lymphatic system	45%
Musculo-skeletal system	32%
Respiratory tract	24%

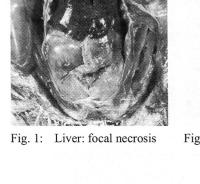




Fig. 2: Liver: Hepatoma

animals showed lesions of the cardiovascular system and 51% of the birds inspected were affected by atherosclerosis, mainly localized to the abdominal part of the aorta. The urogenital system showed an high degree of changes (82%), but most of these consisted of atrophy of the ovaries an oviducts, which could be considered as almost physiological changes due to the aging. Differently from what has been well established in poultry raised under intensive conditions, the pathology of the respiratory tract does not seem to play a major role in aged poultry (24%).

One of the most interesting observations standing out from this study is represented by the high rate of the non viral tumours (22%), which shows a considerable discrepancy with the values reported by diffe-

rent authors about the percentage of neoplastic lesions observed in broilers at slaughtering (.08 to 3%) (Hemsley, 1966; Bergmann et al., 1984)

Therefore our data show a direct correlation between aging and the development of non viral tumours in poultry, a phenomenon otherwise well established in mammals.

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# COMPARISON OF LESIONS IN ONE- AND TEN-DAY-OLD GNOTOBIOTIC CALVES INOCULATED WITH A ROTAVIRUS OF LOW VIRULENCE

G. A. Hall, J. C. Bridger, K. R. Parsons, K. C. Varshney
Calves usually experience infection with rotavirus in the first few
weeks of life, but many of these infections are subclinical (McNulty
& Logan, 1983). Fully virulent bovine rotaviruses are pathogenic in
calves of any age, whereas strains of low virulence only cause diarrhoea in day-old calves (Bridger, 1988). Explanations for this variation
in virulence have been sought by studying pathogenesis in one-dayold and ten-day-old calves inoculated with a strain of low virulence.

### **Material and methods**

Three, one-day-old gnotobiotic calves were inoculated orally with  $10^6$  TCID<sub>50</sub> of a cloned rotavirus of low virulence (strain C3-160) and three were inoculated similarly at ten-days-old. Two groups of three calves were inoculated with virus-free medium and were age-matched controls. Calves were examined at least twice daily for clinical

signs of disease and faeces were collected and the virus content assayed by inoculation of MA-104 cells. Calves were killed within 24 hours of peak virus excretion. They were inoculated with colchicine prior to induction of terminal anaesthesia and small intestinal tissue was taken for measurement of villus height and crypt cell production rate (MacDonald & Ferguson, 1978; Hall et al., 1988a), for histological examination (Hall et al., 1988b) and for immunoperoxidase detection of rotavirus antigen (Parsons et al., 1984). The area of immunostaining in the epithelium was measured with an image analyser and expressed as the mean percentage of total epithelial area.

## **Results**

Control calves and ten-day-old calves inoculated with rotavirus C3-160 remained healthy. Calves inoculated at one-day-old became depressed and developed explosive watery diarrhoea approximately

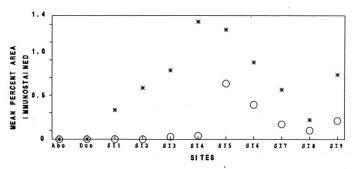


Fig. 1: Mean percent area immunostained for rotavirus antigen in the epithelium of the abomasum, duodenum and 9 small intestinal sites of one-day-old (\*) and ten-day-old (o) gnotobiotic calves.

24 hours after inoculation, when the peak titre of virus in faeces  $(10^{8.8}-10^{9.2})$  occurred. The peak titre of virus in faeces of calves inoculated at ten-days-old  $(10^{5.7}-10^{7.9})$  was detected between 48 and 72 hours after inoculation.

The mean percent area of epithelium immunostained in the small intestine was greater in calves inoculated at one-day-old than in the calves inoculated at ten-days-old; immunostaining was present throughout the small intestine in one-day-old calves but absent from the upper small intestine of calves inoculated at ten-days-old (Fig.). In the calves inoculated at one-day-old, enterocytes were exfoliated and villi stunted and fused and enterocytes remaining in the epithelium were cuboidal. Villus height was reduced to 50% of control

values in the mid and distal small intestine. Foci of enterocyte exfoliation and cuboidal enterocytes in the epithelium were seen in calves inoculated at ten-days-old, but villus height was not affected. Crypt cell production rates were increased in both groups of calves, compared with controls, in the proximal, mid and distal small intestine.

#### **Discussion**

This study explains why the outcome of rotavirus infection differs in calves of different ages. In calves of both ages, the rotavirus used here infected and killed enterocytes but the inability of this strain to cause diarrhoea in ten-day-old calves appeared to be related to its inability to cause severe intestinal damage; it was not able to kill enterocytes faster than they could be replaced. There were differences in susceptibility of the gut to damage by this rotavirus in the two age-groups of calves; these host differences require further investigation.

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# LIGHT- AND ELECTRONMICROSCOPICAL EFFECTS OF ATRAZINE IN RAINBOW TROUT (ONCORHYNCHUS MYKISS) AFTER SUBCHRONIC EXPOSITION

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Atrazine has become one of the most frequently used ingredients of herbicides in the last decade. It enters the aquatic environment by run-offs from agriculturally used areas. Morphological investigations of fish with atrazine have primarily been performed with very high concentrations. These high concentrations, however, normally occur in the environment only after accidents for a short period. To achieve more information on this water pollutant and its impacts on fish in sublethal concentrations, rainbow trout (*Oncorhynchus mykiss*) were used to assess the effects of various atrazine concentrations.

Atrazine was dissolved to a saturated mixture, filtered and added constantly to aquaria with a capacity of  $36\,l$  by mixing the original solution with tapwater by means of a dispenser. Atrazine concentration was measured regularly by a gaschromatograph at the beginning, during and at the end of the experiment to ensure the constant test concentration. Water temperature was  $10.5^{\circ}C$  at a flow rate of 2 1/h, pH 7.6 and conductivity was  $744~\mu$ S/cm. *Chronic exposure* to atrazine was performed at concentrations of  $5,\,10,\,20,\,40$  and  $80~\mu$ g/l for 28~days and *acute exposure* was done at concentrations of 1.4~and~2.8~mg/l for 96~h. For each concentration two tests with 10~fish each were performed simultaneously. Additionally, for each test two control

groups each with ten fish were examined. The mostly affected organs were gills and kidney.

Chronic exposure resulted in gills in hypertrophia as well as hyperplasia of chlorid cells. A low percentage of them had first signs of degeneration in lower concentrations (5–20  $\mu$ g/l) whereas in higher concentrations (40–80  $\mu$ g/l) degeneration was more prominent than hyperplasia. Acute exposition induced an immense proliferation of chlorid cells which were degenerated in a high percentage.

In kidney both the excretory as well as the hemopoietic parts were affected. At chronic exposure, podocyte proliferation, mesangial proliferation and thickening of glomerular basement membrane took place indicating a membrano-proliferative glomerulonephritis. Furthermore, a periglomerular fibrosis was seen in higher concentrations. Acute exposure induced an obliteration of Bowman's space due to proliferated mesangial cells.

The tubular system was especially affected at acute exposure showing extensive vacuolar degeneration.

In the hemopoietic tissue an increase of mitotic figures was the result of low concentrations. Higher concentrations at chronic exposure as well as acute intoxication resulted in a degeneration of sinus endothelia and hemopoietic cells.