hens.

5) Five one-day-old chicks from an AE susceptible flock succumbed to oral inoculation with a wild strain of AEV \(10^{6.9} \text{CID}_{50}\) isolated from the pipped embryos when the flocks were infected with AEV. However, 7 or 14-day-old chicks from the susceptible flock and 1-, 7-, or 14-day-old chicks from AE infected flocks resisted the same challenge.

6) The chicks of 1-, 7-, 14-days old, from the same flock did not develop the symptoms. On the other hand, 2 out of 5 one-day-old chicks with a significant level of maternal antibody succumbed to the same challenge. The chicks of 7-, 14-, 21-, 35-days old from the AE infected flocks resisted the challenge irrespective of whether they maintained the significant level of the maternal antibody or not.

7) The immunological responses in producing a neutralizing antibody in the progenies from the AE infected flock was weaker than that produced in the chicks from the AE susceptible flock after the challenge.

8) From the results described in 5) and 6), it was reconfirmed that one of the resistance factors to AEV infection is the age of the chickens.

**ON THE ADENOID OR ADENOMATOID METAPLASIA OF THE MUCOSA IN THE RENAL PELVIS AND URETER OF THE HORSE**

*Isao Narama*

*Department of Comparative Pathology*

*Faculty of Veterinary Medicine*

*Hokkaido University, Sapporo, Japan*

Formal pathogenesis of the adenoid or adenomatoid structures of the mucosa in the renal pelvis and ureter of the horse was considered. Materials investigated were obtained from 50 horses. They consisted of one fetus in the 6th month of pregnancy, 2 fetuses in the 9th month of pregnancy and 47 horses at various ages from 30 days to 21 years old. Five cases out of the above 50 consisted of one case with chronic cystitis and 4 cases with chronic interstitial nephritis.

No adenoid alveoli were found in the lamina propria of the renal pelvis of the fetuses and some of the foals. Four cases out of 8 of which the ureters were fully investigated extending through their whole length, had the adenoid alveoli in the lamina propria of the ureters, and in 2 out of the 4 cases the adenoid alveoli were formed extending over the whole length of the ureters.
In considering the formal pathogenesis of the adenoid or adenomatoid structures, edematous processes (disturbances of water metabolism) in the lamina propria were especially taken seriously. The epithelium was desquamated frequently on account of the accumulation of edema fluids just beneath the epithelium. These edematous processes were considered due to microvascular alteration (edematous loosening and swelling of the wall of the small blood vessels). The degree of the adenoid alveolus formation had no relation to the existence of chronic cystitis or chronic interstitial nephritis.

Under the edematous tissue condition resulting from the microvascular alteration, the foamation of the adenoid or adenomatoid structures was regarded as "an epithelial regeneration in an abnormal way" (simplification of faculty of cell-division, conversion of direction of cell-division, inconstancy of velocity of cell-division, etc.), namely, a metaplasia (adenoid or adenomatoid metaplasia).

**THE KINETIC AND LYTIC INFECTION OF HAMSTER CELLS**

**BY INFECTIOUS CANINE HEPATITIS VIRUS**

Takeshi Nishi  
*Department of Hygiene and Microbiology*  
*Faculty of Veterinary Medicine*  
*Hokkaido University, Sapporo, Japan*

In the first part of this paper, the multiplication of ICHV in the hamster cells was studied using 2 strains, FD and Woc-4, and 4 types of cells - whole hamster embryo cells, baby hamster kidney cells, adult hamster kidney cells and the cells established from ICHV-induced hamster tumors. Fairly good multiplication was observed when hamster cells were infected with strain FD, and the maximum infective titers obtained were sometimes as much as those obtained from dog kidney cells (DKC). In contrast, the multiplication of strain Woc-4 in hamster cells was smaller than that of strain FD. In particular, no multiplication of strain Woc-4 was observed in BHK21 (W-12) cells established from the baby hamster kidney cells, nor in HT-7 and HT-8 cells which were established from the ICHV-induced hamster tumors.

The second part dealt with in vitro transformation of hamster cells by ICHV. Transformed foci were found in the cultures inoculated with 9 virus strains out of 11 used. The foci appeared about 37 days after virus inoculation and, as reported in oncogenic human adenovirus type 12 - hamster cell system, were observed as colonies of small epithelioid cells against the background of normal cell sheets chiefly consisting of fibroblastic cells. By the fluorescent antibody technique,