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Studies on the pathogenesis of group A avian rotavirus infection in turkeys

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Group A 조류 로타 바이러스의 병원성에 관한 연구

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초록 : 조류 로타바이러스 감염 음성인 1일령, 7일령, 14일령 그리고 21일령된 칠면조에 조류 로타바이러스를 인공감염시킨 후 소장에서의 바이러스의 분포, 배출 그리고 소장의 흡수기능을 조사하였다. 1일령, 7일령 그리고 14일령의 칠면조는 조류 로타바이러스에 대한 혈중모체이행항체의 존재에도 불구하고 로타바이러스 감염에 민감하였다. 인공감염된 칠면조는 경미한 임상증상을 보였으며 십이지장, 공장 그리고 회장의 성숙된 융모상피세포에서 바이러스가 검색되었다. 병리조직학적인 소견으로서는 상피세포에 공포가 형성되었으며 호중구의 침윤이 관찰되었다. 조류 로타바이러스에 인공감염된 14일령 칠면조에서 감염후 1일과 3일에 D-xylose 흡수기능은 대조군과 비교하였을 때 유의성이 인정되었다.

Key words : avian rotavirus, pathogenesis, D-xylose adsorption, turkey.

Introduction

Rotaviruses infect a variety of animal species including avian species, where they have been associated with gastroenteritis in young animals.[1~5] Rotaviruses have been detected and isolated from turkeys and chickens with outbreaks of diarrhea.[6~9] Experimental infection of rotaviruses in gnotobiotic piglets, calves, lambs and dogs resulting in enteritis, diarrhea and decreased absorption of D-xylose has been reported.[10~14] The same authors showed that the virus infected primarily villous epithelial cells of the small intestine and destroyed the enterocytes resulting in shortening of the villi and reduction of villous mucosal disaccharidase enzyme. In avian species, experimental infection with avian rotavirus has been reported using specific-pathogen-free(SPF) chickens and SPF and conventional turkey poults.[15~16] Clinical signs observed were mild or subclinical and rotavirus antigens were detected in the mature epithelial cells of the small intestine. Colibacillosis seems to worsen in the presence of rotaviral infection.[17~22] The purpose of this study was to determine whether rotavirus infection would cause any gross or microscopic changes in the small intestine, secondly to see whether there is any correlation between serum maternal antibody and infection, finally whether mixed infection with rotavirus-*E coli* would more closely resemble the naturally occurring disease state. This paper describes the results of experimental infection of com-

mercial turkey poults infected with avian rotavirus and/or *E coli* orally at different ages.

Materials and Methods

Turkey poults : One-day-old Nicholas strain turkey poults were obtained from a commercial hatchery in Minnesota and were placed in Horsfall-Bauer units. All turkey poults were determined to be rotavirus-free. Cloacal swabs from birds were taken on arrival and placed in serum-free Eagle's minimum essential medium (EMEM). Cloacal swab suspensions were filtered through a 0.45 μm filter and inoculated onto confluent MA104 cell monolayers in roller tubes and incubated at 37℃. Inoculated roller tubes were examined for 7 days for virus growth. Pooled fecal samples from randomly sacrified turkey poults were examined by electron microscopy for the presence of virus particles.

Virus : Avian rotavirus AvRV-1 originally isolated in our laboratory from turkey poults with enteritis was used. This virus was identified as group A rotavirus by genome electropherotyping.[23] The virus was propagated in MA104 cells using EMEM supplemented with 10% fetal calf serum. The virus from 10th passage having a titer of $10^{7.0}$ $TCID_{50}/m\ell$ was used as inoculum. The virus was treated with trypsin(Type Ⅲ ; Sigma Chemical Co., St Louis, MO) at a final concentration of 10 $\mu g/m\ell$ and incubated at 37 C for 30 min before use.

Bacterium : *Escherichia coli* (serotype 078 : K80 : H9) originally isolated in our laboratory from turkeys with severe enteritis was used. *E coli* was streaked for purity on solid media and then grown overnight in tryptic soy broth at 37 C. The concentration of *E col* was adjusted to be $10^{7.0}$ colony forming units(CFU) per $m\ell$.

Virus isolation from fecal specimens : Ten percent suspension of fecal specimens were prepared in phosphate-buffered saline (PBS ; pH 7. 4) and were treated with trypsin as described previously.[24] Trypsin treated fecal suspension was filtered through a 0.45 μm filter and diluted 1 : 10 with serum-free EMEM. The diluted fecal suspensions were inoculated onto roller tube cultures on MA104 cells and incubated for 5 days at 37 C. Regardless of cytopathic effect (CPE) occurrence, inoculated culture tubes were frozen and thawed three times 6 days post-inoculation and treated with trypsin at a concentration of 10 $\mu g/m\ell$ and inoculated onto fresh confluent MA104 cell monolayers prepared on a microscopic slide for subsequent passage. MA104 cells were prepared on a teflon coated microscopic slide (Cell Line Associates Inc., Newfield NJ) having 10 rings each with a diameter of 7.0mm. Before inoculation, the cells were washed twice with serum-free EMEM. Fifty $\mu\ell$ of 1 : 10 dilution of trypsin-treated infected cell culture fluids were placed onto each ring. After 1 hr adsorption at 37 C in a humidified chamber, inoculum was removed and infected cells were washed once with serum-free EMEM and fed with 0.1$m\ell$ of maintenance medium containing 0.5$\mu g/m\ell$ of trypsin. The infected cells were incubated for 18～20 hr at 37 C in a humidified chamber. At the end of incubation periods, the infected cells were fixed for immunofluorescent antibody assay.

Enzyme-linked immunosorbent assay (ELISA) : Serum maternal antibody titer against avian rotavirus was determined by ELISA. Procedures for the ELISA were followed according to the method described with a few modifications.[25] Purified avian rotavirus was fragmented with chaotropic agent before absorption of the viral antigen to the solid phase. In brief, 150 $\mu\ell$ of 0.1M EDTA was added to 1.0$m\ell$ of purified avian rotavirus and the mixtures were incubated for 30 min at room temperature. After incubation, the mixtures were further treated with 1.5M sodium thiocyanate (NaSCN ; Sigma) for 15 min at room temperature. Antigens were diluted to a final concentration of 1 μg of protein per $m\ell$ with coating buffer and 0.1$m\ell$ was dispensed into microtiter plate per well. Following overnight incubation at 4 C, the antigen coated plates were washed three times with 0.01 M PBS containing 0.05% Tween 20 (PBS-tween ; pH 7. 4). One-tenth $m\ell$ of two-fold serial dilution of serum diluted with PBS-tween containing 1% bovine serum albumin (BSA) was added to each well. After 2hr incubation at room temperature, plates were washed three times with PBS-tween and then 0.1$m\ell$ of peroxidase conjugated rabbit anti-turkey IgG(Kirkegaard and Perry Laboratones Inc., Gaithersburg, MD) were added. The plates were again incubated for 1 hr at 37C. The plate were then washed and 0.1$m\ell$ of substrate was added. The substrate consisted of 0.012% hydrogen peroxide containing 0.04% orthophenylenediamine dihydrochloride (OPD ; Sigma) in citric acid-phosphate buffer, pH 5.0. After incubation for 30 min at room temperature, absorbance was

measured at 490nm by a microelisa autoreader(Dynatech Instruments Inc., Torrance, CA).

Immunofluorescence assay : Microscope slides infected with fecal specimens were fixed in acetone-ethanol mixture (1 : 1 ; v/v) for 10 min. Thirty $\mu\ell$ of rabbit anti-AvRV-1 serum[26] diluted 1 : 50 in PBS was added to each ring on the slide and allowed to react for 30 min at 37 C in a humid atmosphere. Cells were washed in four changes of PBS and air dried. After drying, fluorescein isothiocyanate labeled goat anti-rabbit IgG antibody (Kirkegaard and Perry Laboratories Inc.) was added for counter staining and the microscope slide was incubated for 30 min at 37 C. This was followed by washing the slide with PBS and rinsing in distilled water for 10 min. The slide was air dried and mounted in buffered glycerin, pH 8.5. Slides were examined with ultraviolet fluorescent microscopy.

Viral antigen : An indirect immunofluorescent antibody test was used to detect rotaviral antigen from tissue sections of duodenum, jejunum, ileum and cecum. Cryostat sections of tissues 4 to 6 μm thick were cut and fixed in acetone-ethanol mixture(1 : 1 ; v/v)for 10 min. The fixed tissue sections were treated as described above for immunofluorescence assay.

Histopathological studies : Tissue specimens were fixed in neutral buffered 10% formalin. Tissues were embedded in paraffin and sections were cut at 5 μm and stained with hematoxylin and eosin(H & E). All tissues were examined by light microscopy.

Experimental design : The pathogenesis of rotavirus infection was examined in turkey poults infected at 1, 7, 14 and 21 days of age. Turkey poults in each age group were divided into 6 groups of equal numbers and infected with rotavirus and/or *E coli* orally as shown in Table 1. Equal number of turkey poults were inoculated with PBS as controls. Each experimental group, consisting of 15 turkey poults, was housed in a Horsfall-Bauer negative pressure isolator. Two turkey poults from each group were sacrificed and examined each at 1, 3, 5, 7 and 9 days post-infection. Intestinal contents were collected from duodenum, jejunum, ileum and cecum and pooled for virus isolation. Intestinal tissue samples for viral antigen detection were collected in a glass container containing cold 2-methyl butane (Sigma) and immediately frozen in liquid nitrogen and stored at -70C until processed. Intestinal tissue samples were collected from 5 different areas. They included ; mid portion of the descending and ascending duodenum, proximal jejunum, distal jejunum, mid portion of ileum and mid portion of cecum.

D-xylose absorption experiment : Eighty 14-days-old turkey poults were equally divided into 4 groups. Birds in group 1 were given $10^{7.0}$ $TCID_{50}/m\ell$ of rotavirus per os. Birds in group 2 were given rotavirus and *E coli* (serotype 078 : K80 : H9) together per os. The dose of rotavirus given was the same as that of in group 1. The dose of *E coli* administered was $10^{7.0}$ CFU/$m\ell$ /bird. Birds in group 3 were given *E coli* alone per os. The serotype and the dose of *E coli* was the same as that of group 2. Birds in group 4 were given PBS as control. Birds in all groups were given feed and water ad libitum. At 1, 3, 5 and 7 days postinfection with rotavirus and/or *E coil* four turkey poults from each group were randomly selected, wing banded and transferred into a separate isolation unit for 12hr of fasting before the administration of D-xylose. Birds were individually weighed and a blood sample was taken. They were then given a 5% soultion (w/v) of D-xylose(Sigma) in deionized water (0.5g/kg of body weight) by intubation of the crop with a soft flexible rubber feeding tube. Blood samples were collected from each turkey poult at 30, 60, 90, 120 and 180 min post D-xylose intake. Plasma was separated and used for D-xylose estimation.

Determination of D-xylose absorption : Plasma D-xylose concentration was determined by the micromethod[27] recently modified by Goodwin et al.[28] In brief, 2$m\ell$ of color reagent which consisted of 0.5 g of phloroglucinol (1, 3, 5-trihydroxybenzene ; Sigma), 100$m\ell$ of glacial acetic acid and 10$m\ell$ of hydrochloric acid(37%), was added to a 20 $\mu\ell$ plasma sample in 12 $\times$75mm disposable test tube. All tubes were heated for 4 min at 100 C and then were cooled to room temperature. After cooling, absorbance was measured at 554nm with an automated continuous flow spectrophotometer system (Sepctronic 1001 split beam spectrophotometer ; Milton Roy Co., Rochester, NY).

Statistical analysis : Statistical significance of differences was determined with the Student's t-test.

Results

Clinical signs : Turkey poults infected with avian rotavirus manifested only mild clinical signs. They appeared depressed. The depression was not acute but most birds displayed inappetence usually persisting for 5 to 6 days. No mortality occurred throughout the period of observation. In turkey poults necropsied at 1 and 3 days post-infection, macroscopically, the cecum was abnormally distended with a liquid and gas mixture.

Maternal antibody in serum : Fig 1 shows the results of maternal antibody levels determined by ELISA. Maternal antibody against avian rotavirus was not detected from turkey poults at 21 days of age. However, maternal antibody was detected from turkey poults at 1, 7 and 14 days of age.

Virus isolation and distribution in tissue : Turkey poults infected at 1 day of age were susceptible to avian rotavirus infection despite of the presence of serum antibody to avian rotavirus. Virus was isolated from the infected group 1 day post-infection and isolated for 7 days post-infection. No virus was isolated from uninfected controls and poults infected with *E coli* alone throughout the experiment. Similar results as those of birds infected at one day of age were obtained with birds infected at 7 and 14 days of age(Table 2). However, turkey poults infected at 1 day of age seemed to be much less susceptible to infection compared to those infected at 1, 7 and 14 days of age. Table 3 shows the results of virus distribution in intestinal tissues as detected by FA from commercial turkey poults infected with avian rotavirus at different age. Viral antigens were mainly observed at 1 day post-infection, and were found predominantly in duodenum and jejunum. Sometimes viral antigens were detected in ileum. However, there were no differences in intensity of virus distribution between rotavirus alone and rotavirus and *E coli* infected groups. Viral antigens were located within the cytoplasm of the villous epithelial cells and infected cells were usually present on the tips of the villi. Sometimes infected cells were found sporadically on the lateral portion of the villi. No viral antigens were detected in crypt cells. None of the uninfected control and *E coli* infected groups showed fluorescing antigen when stained for avian rotavirus by FA technique. Viral antigens were detected from both rotavirus and *E coli* simultaneously infected group.

Table 1. Experimental design for mono and dual infections with avian rotavirus and/or *E coli* in commercial turkey poults

Group	No. of poultry	Sequence of inoculation	
		1st[A]	2nd[B]
1. Control	15	None	None
2. Rota	15	Rotavirus[C]	None
3. Rota/*E coli*[D]	15	Rotavirus/*E coli*	None
4. *E coli*	15	*E coli*[E]	None
5. Rota/*E coli*	15	Rotavirus	*E coli*
6. *E coli*/Rota	15	*E coli*	Rotavirus

[A]1st inoculation was done at 1, 7, 14 or 21 days of age.

[B]2nd inoculation to the same turkey poults was done 48 hours post-1st inoculation

[C]One mℓ of avian rotavirus(AvRV-1 : 10^7 $TCID_{50}$/mℓ) was given to each turkey poult.

[D]Rotavirus and *E coli* were inoculated simultaneously.

[E]One mℓ of *E coli*(serotype 078 : K80 : H9 : 10^7 CFU/mℓ)was given to each turkey poult.

Table 2. Viral isolation from feces of turkey poults infected with avian rotavirus(AvRV-1) at 1, 7, 14 and 21 days of age

Day[A]	Control					Rota					Rota/*E coli*[B]					*E coli*					Rota/*E coli*[C]					*E coli*/Rota[D]				
	1[E]	3	5	7	9	1	3	5	7	9	1	3	5	7	9	1	3	5	7	9	1	3	5	7	9	1	3	5	7	9
1	−	−	−	−	−	+	+	+	+	−	+	+	+	+	−	−	−	−	−	−	ND[F]	+	+	−	−	+	+	+	−	ND
7	−	−	−	−	−	+	+	+	−	−	+	+	+	−	−	−	−	−	−	−	ND	+	+	−	−	+	+	+	−	ND
14	−	−	−	−	−	+	+	+	+	−	+	+	+	+	−	−	−	−	−	−	ND	+	−	+	−	+	+	+	−	ND
21	−	−	−	−	−	+	+	−	−	−	+	−	−	−	−	−	−	−	−	−	ND	+	−	−	−	+	−	−	−	ND

[A]Day represents the age of turkey poults when they were inoculated with avian rotavirus (AvRV-1) and/or *E coli*.

[B]Rotavirus and *E coli* were inoculated simultaneously.

[C]*E coli* was inoculated 48 hrs post-inoculation with rotavirus.

[D]Rotavirus was inoculated 48 hrs post-inoculation with *E coli*.

[E]Days post-inoculation with rotavirus.

[F]Not determined.

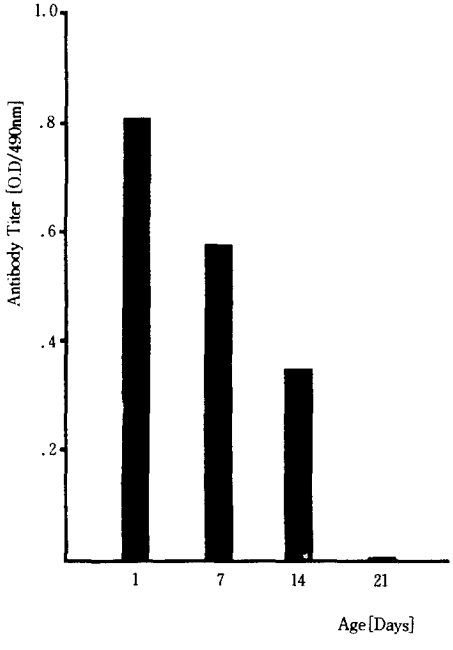

Fig 1. Serum maternal antibody level against avian rotavirus in commercial turkey poults at different ages.

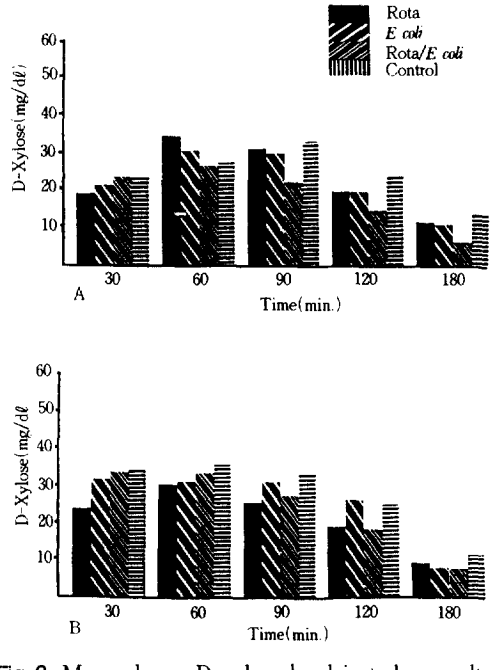

Fig 3. Mean plasma D-xylose level in turkey poults infected at 14 days of age with avian rotavirus and/or *E coli*.
A : 5 days post-infection with avian rotavirus.
B : 7 days post-infection with avian rotavirus.

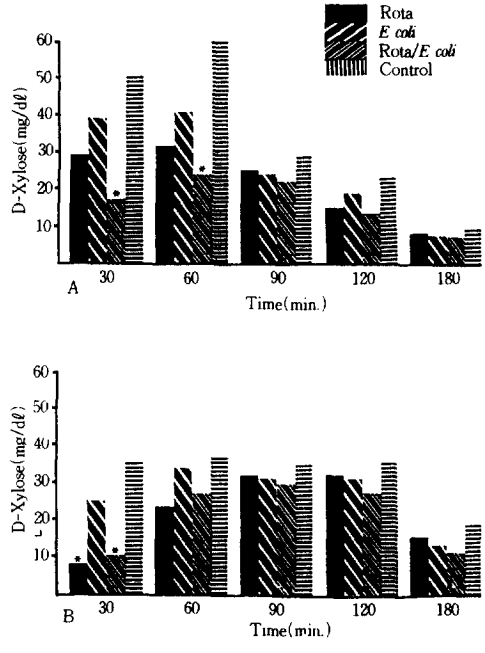

Fig 2. Mean plasma D-xylose level in turkey poults infected at 14 days of age with avian rotavirus and/or *E coli*.
A : 1 day post-infection with avian rotavirus.
B : 3 days post-infection with avian rotavirus.
Asterisks show the statistical significance at $p < 0.05$.

Histopathology : Histopathological lesions present were congestion and mild enteritis with sloughing of cells from the villous surface of epithelial cells. Infiltration of the lamina propria with heterophils was the major histopathological lesion in rotavirus and/or *E coli* infected turkey poults. Sometimes, eosinophilic infiltration was observed. Vacuolation of epithelial cells was also a remarkable lesion in infected turkey poults.

D-xylose absorption : Figs 2 and 3 show the results of comparison of mean plasma D-xylose levels absorbed for control and infected groups at 1, 3, 5 and 7 days postinfection with avian rotavirus. The rotavirus infected group absorbed less D-xylose compared to control group at 1 day post-infection, but it was not statistically significant. However, the differences in D-xylose absorption at 3 days post-infection between rotavirus infected and control group were statistically significant ($p < 0.05$). Both rotavirus and *E coli* infected group absorbed significantly less D-xylose compared to control group ($p < 0.05$) at both 1 and 3 days post-infection. There was no differ-

Table 3. Distribution of viral antigen in intestines from turkey poults infected with avian rotavirus (AvRV-1) at 1, 7, 14 and 21 days of age

Tissues	Control					Rota					Rota/*E coli*[B]					*E coli*					Rota/*E coli*[C]					*E coli*/Rota[D]				
	1[E]	3	5	7	9	1	3	5	7	9	1	3	5	7	9	1	3	5	7	9	1	3	5	7	9	1	3	5	7	9
1 Day[A]																														
D	−	−	−	ND[F]	ND	+	+	+	−	−	+	−	−	−	−	−	−	−	ND	ND	ND	+	−	−	−	+	−	−	−	ND
PJ	−	−	−	ND	ND	+	+	+	−	−	+	−	−	−	−	−	−	−	ND	ND	ND	+	−	−	−	+	−	−	−	ND
DJ	−	−	−	ND	ND	+	−	+	−	−	+	−	−	−	−	−	−	−	ND	ND	ND	+	−	−	−	+	−	−	−	ND
I	−	−	−	ND	ND	+	−	−	−	−	+	−	−	−	−	−	−	−	ND	ND	ND	−	−	−	−	−	−	−	−	ND
C	−	−	−	ND	ND	−	−	−	−	−	−	−	−	−	−	−	−	−	ND	ND	ND	−	−	−	−	−	−	−	−	ND
7 Days																														
D	−	−	−	ND	ND	+	−	−	−	−	+	−	−	−	−	−	−	−	ND	ND	ND	+	−	−	−	+	−	−	−	ND
PJ	−	−	−	ND	ND	+	+	−	−	−	+	−	−	−	−	−	−	−	ND	ND	ND	+	−	−	−	+	+	−	−	ND
DJ	−	−	−	ND	ND	+	+	−	−	−	+	−	−	−	−	−	−	−	ND	ND	ND	+	−	−	−	+	+	−	−	ND
I	−	−	−	ND	ND	+	+	−	−	−	+	−	−	−	−	−	−	−	ND	ND	ND	−	−	−	−	−	−	−	−	ND
C	−	−	−	ND	ND	+	−	−	−	−	−	−	−	−	−	−	−	−	ND	ND	ND	−	−	−	−	−	−	−	−	ND
14 Days																														
D	−	−	−	ND	ND	+	−	−	−	−	+	+	−	−	−	−	−	−	ND	ND	ND	+	−	−	−	+	+	−	−	ND
PJ	−	−	−	ND	ND	+	−	−	−	−	+	+	−	−	−	−	−	−	ND	ND	ND	+	−	−	−	+	+	+	−	ND
DJ	−	−	−	ND	ND	+	−	−	−	−	−	+	−	−	−	−	−	−	ND	ND	ND	+	−	−	−	+	+	+	−	ND
I	−	−	−	ND	ND	+	−	−	−	−	−	−	−	−	−	−	−	−	ND	ND	ND	−	−	−	−	+	+	−	−	ND
C	−	−	−	ND	ND	−	−	−	−	−	−	−	−	−	−	−	−	−	ND	ND	ND	−	−	−	−	−	−	−	−	ND
21 Days																														
D	−	−	−	ND	ND	−	−	−	−	−	+	−	−	−	−	−	−	−	ND	ND	ND	−	−	−	−	−	−	−	−	ND
PJ	−	−	−	ND	ND	−	−	−	−	−	+	−	−	−	−	−	−	−	ND	ND	ND	−	−	−	−	−	−	−	−	ND
DJ	−	−	−	ND	ND	−	−	−	−	−	+	−	−	−	−	−	−	−	ND	ND	ND	−	−	−	−	−	−	−	−	ND
I	−	−	−	ND	ND	+	−	−	−	−	−	−	−	−	−	−	−	−	ND	ND	ND	−	−	−	−	−	−	−	−	ND
C	−	−	−	ND	ND	−	−	−	−	−	+	−	−	−	−	−	−	−	ND	ND	ND	−	−	−	−	−	−	−	−	ND

* see footnote in Table 2.

ence in D-xylose absorption between infected and control groups at 5 and 7 days post-infection.

Discussion

Clinical signs and necropsy observations on the turkey poults in the present study were similar to those previously reported.[15,16] However, the clinical signs of experimentally infected commercial turkey poults were milder than might have been expected. It might have been due to a decrease in virulence of the rotavirus used. Turkey poults at 21 days of age had no detectable maternal serum antibodies, whereas those 1 and 7 days of age had maternal antibodies aganist avian rotavirus (Fig 1). Maternal antibodies in the serum of the turkey poults did not prevent infection. This observation is in agreement with other reports. Yason and Schat[16] reported that the presence of maternal antigodies did not prevent infection in maternal antibody-free SPF turkey poults. However, it was reported that maternal antibodies may delay the infection in chickens.[15] Studies showed that gnotobiotic calves, lambs and pigs were protected against rotavirus challenge when fed colostrum from orally or parenterally immunized dams.[29~31] However, the presence of high titered passively acquired, neutralizing antibodies in the serum was not capable of protecting aganist rotavirus gastroenteritis.[31,32] From these results we postulate that local immunity plays an important role in preventing rotavirus infection. The present study revealed that turkey poults 21 days of age were less susceptible to rotavirus infection compared to turkey poults infected at 1, 7 and 14 days of age. This observation is not consistent with other reports.[15,16] It is possible that age-dependent resistance was involved.

By indiredct immunofluorescent staining of cryostat sections of different portions of the intestinal tract of infected turkey poults, viral antigens were localized in villi of the duodenum and jejunum. Viral antigens were not observed distally in the intestine. The blunted, shortened, and focally fused villi with flattened enterocytes are

typical histopathological changes in the small intestine caused by rotavirus infection.[4,10,11] However, in the present studies, severe histopathological lsions were not observed from turkey poults infected with rotavirus and/or *E coli*. Histopathological changes were mild enteritis with some increased cell sloughing from the villous surface of the epithelial cells vacuolation of epithelial cells.

The D-xylose absorption test has been used as an indicator of intestinal absorptive function in rotavirus infected calves[33] and turkey poults.[16] The present study indicated that a significant difference in D-xylose absorption was observed between control and rotavirus infected groups at 1 and 3 days post-infection with avian rotavirus. However, there was no significant difference in D-xylose absorption 5 and 7 days post-infection. The difference in D-xylose absorption correlated with viral antigen distribution in the small intestine. Viral antigen were observed 1 and 3 days post-infection from turkey poults infected with avian rotavirus at 14 days of age(Table 3). One of the objectives of this study was to determine whether mixed infection with rotavirus and *E coli* would reproduce the naturally occurring disease. There are reports that the severity of outbreaks of rotavirus infection are related to concurrent infections with one or more other pathogenic bacteria, viruses and parasites.[17,18,20,22] In this study, no differences in clinical signs, gross lesions and histopathological changes were observed between the rotavirus alone infected and rotavirus and *E coli* infected groups. These results could be explained in two ways. First, it may be due to a decrease in virulence of the avian rotavirus used in this experiment. The avian rotavirus used was field isolate from turkey poults experiencing diarrhea and high mortality. The virulence may have been decreased during cell culture passages. Second, commercial turkey poults were used instead of SPF turkey poults. These turkey poults had serum maternal antibodies against avian rotavirus. The antibody titers against avian rotavirus in the small intestine were not examined. If antibodies were present in small intestine, they could modulate the rotaviral infection to some extent. Commercial turkey poults might also have had antibodies against the same *E coli* serotype used in this experiment.

In this experiment, it was observed that avian rotavirus infected turkey poults with mild clinical signs had impairment of D-xylose absorption. Further work will be needed to elucidate the relationship between conventional(group A) and non-group A avian rotavirus pathogenesis. Non-group A avian rotaviruses were prevalent in commercial turkey flocks in field and were commonly detected along with conventional avian rotaviruses from turkeys with enteritis.[23]

Summary

Commercial turkey poults not previously exposed to avian rotavirus were inoculated orally with the virus alone or in combination with *E coli* serotype 078 at 1, 7, 14 and 21 days of age. Turkey poults of 1, 7 and 14 days of age were susceptible to infection despite the presence of maternal antibodies against avian rotavirus in their serum. However, turkey poults at 21 days of age were less susceptible compared to those ages 1, 7 and 14 days. The clinical signs in poults of all ages were mild. Viral antigens were demonstrated in the mature villous epithelial cells of the duodenum, jejunum and ileum. Histopathological lesions were characterized by vacuolation of the epithelial cells and heterophil infiltration in infected turkey poults. A significant difference in D-xylose absorption was observed between control and rotavirus infected groups at 1 and 3 days post-infection in 14 days old turkey poults.

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