Enteropathogenic *Escherichia coli* (EPEC) generally colonizes the intestine and adheres intimately to the enterocytes resulting in a characteristic effacement and cupping of the microvilli [6]. The term attaching and effacing *E. coli* (AAEC) has been used to designate *E. coli* that causes such lesions [8]. Infections with AAEC are common in humans [6], calves [3–5, 7, 9–13], rabbits [1, 8], pigs [2, 8] and have also been reported in cats [11]. In bovines, there are several reports on calf diarrhea associated with natural infection of AAEC [3–5, 9–12], but not in adult cows. In this paper, we report the pathological findings observed in an adult cow naturally infected with AAEC of serogroup O15.

A 9-month-old Holstein female manifested mucous-hemorrhagic diarrhea on July 16, 1992. She entered the public pasture in Hokkaido from June 28, 1992, and was kept on this pasture with 474 Holstein cows of similar age. Diarrhea was not seen in other cows from this herd. This particular cow was shedding coccidial oocysts (300 of oocysts per g) and was given sulfafoximine and Ringer’s solution intravenously from July 16 to July 22. However she didn't recover from diarrhea with subsequent dehydration, anorexia and depression. She was euthanatized by exsanguination on July 23, 1992.

At necropsy, the colon contained a large amount of bloody material mixed with mucus and there was diffuse petechial hemorrhage on the mucosal surface. No lesions were observed in other parts of the alimentary tract. Enlargement of the mesenteric lymph nodes and hepatization of the frontal lobes of the lung were seen.

Histologically, the epithelial layer of the colon was disrupted and irregular in outline. Numerous gram-negative bacilli were seen adhered to the eroded luminal surface of enterocytes. The lamina propria was congested or hemorrhagic, and infiltrated by neutrophils. The intestinal crypts were dilated with mucus, and colonic lumen was filled with necrotic enterocytes, mucus and erythrocytes (Fig. 1). A small number of coccidia was seen in the lamina propria of the colon. No lesion was observed in the alimentary tract other than the colon. Edema and infiltration of neutrophils and monocytes were seen in the medullary sinus of the mesenteric lymph node. Granuloma due probably to lungworm was seen in the lung.

*E. coli* antigen on paraffin sections of the colon was examined by the avidin-biotin complex immunoperoxidase technique (ABCIT: Vector Laboratories, Burlingame, U.S.A.). As the primary antibody, rabbit antisera against heat-inactivated antigen of *E. coli* strain F7902-41 (O15) was used at a dilution of 1:8192. As a result, *E. coli*
O15 antigen was seen to be distributed diffusely on the epithelial surface of the colonic lesion (Fig. 2).

Scanning electron microscopic examination of the colonic lesion showed that much of the mucosal surface was heavily colonized by rod-shaped bacteria (Fig. 3).

On transmission electron microscopy examination of the colon, numerous bacteria were found to be closely attached to the surface of the epithelial cells and the microvilli of some of these areas. The plasma membrane of the epithelial cell formed pedestals in the areas intimately associated with bacterial cell walls. Accumulations of dense fibrillar material were observed in the pedestals below the bacteria (Fig. 4).

On bacteriological culture of the rectal contents, E. coli isolate belonging to the O15 serogroup was not detected. However, E. coli and bovine viral diarrhea mucosal disease virus were not isolated from the rectal and other organ samples.

Although we failed to isolate E. coli O15 by culture, this attaching and effacing (AE) lesion is considered to be caused by E. coli O15 from the immunohistological finding. AECC organisms isolated from calves in previously reports belonged to serogroup O5, O8, O26 and O111 [3-5, 7, 9-13] and there is no report of AECC belonging to O15 in cows. E. coli O15, however, has been known as one of the causative organisms of AE lesion in rabbits and pigs [1, 2, 8]. Therefore this E. coli serogroup has the potentiality to induce AE lesion in cows.

It has been reported that another enteropathogen was concurrently involved with AECC [3, 5, 11]. In this case coccidia might aggravate the signs and lesions. All reports on AECC infection in bovines were confined to young calves [3-5, 9-12]. The AE lesions observed in this adult cow, however, were essentially similar to those reported for young calves.

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REFERENCES