LECTIN HISTOCHEMISTRY OF THE NORMAL HUMAN ENDOMETRIUM AND ENDOMETRIAL ADENOCARCINOMA

TANRI SHIOZAWA^{1,2}, YOSHIHARU TSUKAHARA¹, JUN NAKAYAMA²
AND TSUTOMU KATSUYAMA²

*Department of Obstetrics and Gynecology¹, Department of Laboratory Medicine²,
Shinshu University School of Medicine 3-1-1 Asahi, Matsumoto, 390*

Received for publication October 22, 1990 and in revised form January 16, 1991

Lectin stainings were performed to characterize the properties of glycoconjugates expressed in normal and malignant endometrial tissues. The specimens examined included 21 cases of normal endometrium and 20 cases of endometrial adenocarcinoma. The effect of prior neuraminidase digestion (Neu) upon the lectin staining was examined as well. Nine lectins were used such as *Griffonia simplicifolia* agglutinin-I-B4 (GS-I-B4), peanut agglutinin (PNA), *Ulex europaeus* agglutinin-I (UEA-I), wheat germ agglutinin (WGA), soybean agglutinin (SBA), *Dolichos biflorus* agglutinin (DBA), *Maclura pomifera* agglutinin (MPA), *Griffonia simplicifolia* agglutinin-II (GS-II), and *Limax flavus* agglutinin (LFA). The results obtained indicated that the lectin reactivities were classified into three groups; those detected in normal as well as neoplastic endometrium (Neu-GS-I-B4, WGA, SBA, LFA), those altered depending upon menstrual phases (PNA, SBA; proliferative phase dominant, WGA, DBA, MPA; secretory phase dominant), and those found only in carcinoma tissues (UEA-I, Neu-PNA). GS-I-B4 showed a unique staining pattern, since the reactivity of this lectin appeared only after neuraminidase digestion. This appears to indicate that sugar chains reactive to Neu-GS-I-B4 might be a tissue specific antigen, as such a reactivity was not demonstrated in other organs.

Recent advances in sugar biochemistry have revealed that structural alterations of glycoconjugates take place in response to differentiation, maturation (24) and further, oncogenic transformation (9). The human endometrium is known to show characteristic cyclic changes under controls of steroid hormones. These morphological changes of normal endometrium suggest the changes of sugar structures which may reflect the functional roles such as nidation or sperm transport. Further, in spite of many reports concerning the alterations of sugar structures corresponding to the malignant transformations in various organs, such alterations in endometrial carcinoma have not been explored sufficiently. In the present study, we employed 9 species of lectins in order to detect the terminal sugar residues of glycoconjugates expressed in the normal and neoplastic endometrial tissues. In addition, the effect of neuraminidase digestion upon the affinities for the lectins were likewise investigated (12).

MATERIALS AND METHODS

Endometrial samples

The samples used consisted of 21 cases of normal adult endometrium and 20 cases of endometrial invasive carcinoma. Normal endometrial tissues were obtained from surgically removed uteri at Shinshu University Hospital, which were resected due to carcinoma in situ of the uterine cervix or uterine leiomyoma. The normal tissues were then histologically classified into six groups; early, middle and late proliferative phases and early (ovulatory), middle and late secretory phases. Blood types of these cases are shown in Table 1. Endometrial carcinomas were classified according to FIGO classification (22), and 8 cases were G1 (well differentiated), 7 cases G2 (moderately differentiated) and 5 cases of G3 (poorly differentiated). All the surgically removed tissues were fixed for 24h in a chilled 4% paraformaldehyde solution, buffered with 0.1 M phosphate (pH 7.4). After fixation, each tissue was sliced at intervals of 5 mm in thickness to make tissue blocks, and were dehydrated through a graded alcohol series, cleared in

Footnote: all correspondence should be addressed to Tsutomu Katsuyama.

TABLE 1. *Blood types of specimens examined*

Blood types	Normal endometrium		Endometrial adenocarcinoma		
	PP	SP	Histological grading		
			G1	G2	G3
A	5	5	3	3	0
B	1	2	2	0	3
H (O)	3	2	2	4	0
AB	2	1	1	0	2
Sum	11	10	8	7	5

Abbreviations used: PP; proliferative phase, SP; secretory phase, G1; well differentiated, G2; moderately differentiated, G3; poorly differentiated

TABLE 2. *Lectins used, their sugar specificities and concentrations of lectins for stainings*

Lectins	Sugar specificities	Concentrations
<i>Griffonia simplicifolia</i> agglutinin-I-B4 (GS-I-B4)	α -D-Gal	20 μ l/ml
peanut agglutinin (PNA)	β -D-Gal	30 μ l/ml
<i>Ulex europaeus</i> agglutinin-I (UEA-I)	α -L-Fuc	30 μ l/ml
wheat germ agglutinin (WGA)	β -D-GlcNAc	5 μ l/ml
soybean agglutinin (SBA)	α -D-GalNAc	10 μ l/ml
<i>Dolichos biflorus</i> agglutinin (DBA)	α -D-GalNAc	10 μ l/ml
<i>Maclura pomifera</i> agglutinin (MPA)	α -D-GalNAc	5 μ l/ml
<i>Griffonia simplicifolia</i> agglutinin-II (GS-II)	D-GlcNAc	10 μ l/ml
<i>Limax flavus</i> agglutinin (LFA)	NeuAc	20 μ l/ml

xylene and embedded in paraffin. In each case for this study, two or three tissue blocks were selected. Serial sections of a thickness of 3 μ m were prepared for the following staining experiments.

Lectins used

Nine species of lectins used, and their sugar specificities together with dilutions are shown in Table 2. All of these lectins were labeled with horseradish peroxidase (HRP), and were purchased from E-Y Laboratories, San Mateo, California, USA.

Lectin staining

Two paraffin sections from each tissue block were prepared for individual lectin stainings, one for ordinary lectin staining and the other for the lectin staining in combination with prior neuraminidase digestion. Briefly, paraffin sections were dewaxed and placed in 100% methyl alcohol containing 0.3% hydrogen peroxide for 30 min to abolish endogenous peroxidase activity. We confirmed that this treatment did not influence the lectin affinity of tissues. After rinsing in running water, one of two sections from each tissue block were incubated in a solution containing neuraminidase (Nakalai Tesque, Kyoto, Japan) at a concentration of 1 unit/ml in 0.05 M phosphate

buffer (pH 7.0), for 18 hr at 37°C, whereas the other section was placed in the buffer. They were then rinsed in 0.1 M phosphate buffer (pH 7.4), containing 0.2 M NaCl and 0.1% bovine serum albumin for 3 min and incubated in lectin solutions in the same buffer for 18 hr at 4°C. After rinsing three times in the same buffer, lectin-peroxidase complexes were visualized in a solution containing 0.01% 3,3'-diaminobenzidine tetrahydrochloride (DAB) (Wako Pure Chemical, Tokyo, Japan) and 0.001 M hydrogen peroxide in 0.05 M phosphate buffer (pH 7.4) for 5 min. Tissue sections were finally counterstained with hematoxylin or methyl green. In all the lectin stainings, inhibition experiments for binding specificities were performed by adding 0.1 M specific sugars (as shown in Table 2) to the lectin peroxidase solutions.

RESULTS

The results obtained are summarized in Table 3. In this study, "positive" was defined to contain more than 1% of stained cells in each section. No correlation of the reaction was noted among different blood types of the specimens. The effect of the

TABLE 3. *Lectin reactivities of normal endometrial tissues and carcinoma tissues examined*

Lectins	Normal endometrium					Endometrial adenocarcinoma					
	Glandular		epithelium		Stromal cell	G1		G2		G3	
	PP		SP			ap	cp	ap	cp	ap	cp
	ap	cp	ap	cp							
GS-I-B4	—	—	— ~ ±	—	—	—	—	—	—	—	—
Neu-GS-I-B4	++	±	+++	+	—	++ ~ +++	+	++	+	+	+
PNA	++	—	+	—	±	+	—	+	—	—	—
Neu-PNA	++	—	+	—	±	++	+	+	±	+	—
UEA-I*	±	—	—	—	—	++ ~ +++	++ ~ ++	++	++	+	+
WGA*	+++	—	+++	+	+++	++	—	+	—	+	—
SBA*	++	—	+	—	±	++ ~ ++	±	+	—	—	—
DBA*	±	—	++	++	—	±	—	—	—	—	—
MPA*	±	—	++	—	—	—	—	—	—	—	—
GS-II*	—	—	—	—	+	—	—	—	—	—	—
LFA	+++	—	+++	+	—	++ ~ +++	+	++ ~ ++	+	+	—
Neu-LFA	±	—	+	—	—	—	—	—	—	—	—

Abbreviations used: PP; proliferative phase, SP; secretory phase, ap; apical surface, cp; cytoplasm, Neu; prior neuraminidase digestion, —; negative, ±; trace (less than 1% of stained cells), +; weakly positive (1–10% of stained cells), ++; moderately positive (10–50% of stained cells), +++; strongly positive (over 50% stained cells). Reactivities were evaluated only by the number of stained cells in this study.

* indicates that neuraminidase digestion did not significantly alter lectin reactivities.

neuraminidase digestion upon the reactivity was not evident in UEA-I, WGA, SBA, DBA, MPA and GS-II staining.

GS-I-B4: Normal as well as neoplastic endometrial tissues lacked almost any affinity for this lectin (Figs. 1a, 2a, 3a, 4a), even though glandular epithelia at secretory phase occasionally revealed weak reactivities on their apical surface. Digestion with neuraminidase imparted reactivity to most of the glandular epithelia in normal endometrium (Fig. 1b). The reaction product was consistently found on the apical surface and was revealed in the Golgi regions in 70% of the cases examined. The strongest reaction was detected in the secretory phase. The carcinoma cells likewise revealed reactivity only following the neuraminidase digestion. The reaction was visualized on the apical surface in 85% of the cases examined and in the Golgi regions in 65% of them. The G1 and G2 group of endometrial carcinoma exhibited stronger reactivity than the G3 group (Figs. 2b, 3b, 4b). Stromal cells did not stain with this lectin whereas vascular endothelia revealed the reactivity only after the neuraminidase digestion.

PNA: In normal endometrium, a weak reactivity was noticed on the apical surface of the glandular epithelia both in the proliferative and secretory phases (Fig. 5a). They were more intense in reactivity in the proliferative phase. The effect of the digestion with

neuraminidase upon the reaction was not evident (Fig. 5b). Stromal cells stained only faintly, irrespective of menstrual cycles and neuraminidase digestion. In only four cases of endometrial carcinoma positive reaction was noted on the apical surface of the epithelia (Fig. 6a), however the neuraminidase digestion imparted the reactivity in 80% of the cases (Fig. 6b). A similar effect of the enzyme digestion upon the reaction was found in the epithelial cytoplasm in 25% of the cases. Well differentiated carcinoma cells tended to exhibit relatively stronger reactivities.

UEA-I: In normal endometrium, the glandular epithelia of the proliferative phase and surface epithelial cells of the late secretory phase occasionally exhibit a weak reaction. Other endometrial components, except for vascular endothelia lacked the reactivity for this lectin (Fig. 7a). In 75% of the cases of the carcinoma tissues, the apical surface and cytoplasm of the epithelia showed positive reaction. The G1 and G2 groups showed stronger reactions than the G3 group (Fig. 7b, 7c, 7d), and the carcinoma cells characteristically revealed diffuse cytoplasmic reactions. No marked differences were noted in intensity of the staining among different portions of the carcinoma tissues.

WGA: In normal endometrium, WGA exhibited a consistent reaction on the apical surface of the glandular epithelia (Fig. 8a). One of the characteristic

features of WGA was its affinities to stromal cells irrespective of the menstrual phases. In about 55% of the cases of endometrial carcinoma, the epithelial apical surface showed distinct reactivities (Fig. 8b). Poorly differentiated carcinoma cells tended to exhibit weak reactions (Fig. 8c).

SBA: Weak to moderate SBA reactivities of the apical surface of the glandular epithelia were noted in normal endometrium. In the proliferative phase (Fig. 9a), the glandular epithelia exhibited a slightly more intense reaction than that detected in the secretory phase. In 13 cases of endometrial carcinoma, the

Figs. 1, 2, 3 and 4 show the distribution of GS-I-B4 reactive sites.

Figs. 1a, b. The proliferative phase.

- a. GS-I-B4 reactivity is negligible without neuraminidase digestion. $\times 140$
- b. Neuraminidase digestion results in intense GS-I-B4 reactivity on the apical surface of glandular epithelia. $\times 140$

Figs. 2a, b. Well differentiated adenocarcinoma (G1).

- a. Without neuraminidase digestion, the carcinoma cells do not exhibit any reactivity. $\times 105$
- b. Prior neuraminidase digestion yields intense reactivity on the apical surface. $\times 105$

Figs. 3a, b. Moderately differentiated adenocarcinoma (G2).

- a. GS-I-B4 reactivity is not observed without neuraminidase digestion. $\times 75$
- b. Neuraminidase digestion yields strong reactivity on the apical surface and cytoplasm. $\times 75$

Figs. 4a, b. Poorly differentiated adenocarcinoma (G3).

- a. Without neuraminidase digestion, GS-I-B4 reactivity is negligible. $\times 80$
- b. Neuraminidase digestion also imparts the GS-I-B4 reactivity which is less intense than that of G1 and G2 groups. $\times 80$

SBA reactivity was visualized on the apical surface of the carcinoma cells (Fig. 9b).

DBA: In normal endometrium, a weak to moderate reaction was noticed in the apical surface of the glandular epithelia in the both phases. In the secretory

phase, the epithelia showed slightly more intense reactivity than those in the proliferative phase (Fig. 10). In only 4 cases of endometrial carcinoma the apical surface of the carcinoma cells showed weak reactivities.

MPA: In normal endometrium, a moderate re-

Fig. 5 and 6 show the distribution of PNA reactive sites.

Figs. 5a, b. The early secretory phase.

- Without neuraminidase digestion, the apical surface of glandular epithelia shows rather weak reactivity. $\times 120$
- Neuraminidase digestion does not alter the reactivity significantly $\times 120$

Figs. 6a, b. Well differentiated adenocarcinoma (G1).

- Without neuraminidase digestion, PNA reactivity is found only in scattered patterns. $\times 94$
- Neuraminidase digestion imparts intense reactivity to the apical surface of the carcinoma cells. $\times 94$

Fig. 7 shows the distribution of UEA-I reactive sites.

Figs. 7a-d. a. The proliferative phase. Most of the glandular epithelia lack reactivity. $\times 80$

- Well differentiated adenocarcinoma (G1). The carcinoma cells are provided with a diffusely reactive cytoplasm. $\times 80$
- Moderately differentiated adenocarcinoma (G2). The carcinoma cells show diffuse cytoplasmic reactions. $\times 120$
- Poorly differentiated adenocarcinoma (G3). The carcinoma cells also reveal cytoplasmic reactivities, which is less intense than those of G1 and G2 groups. $\times 100$

Fig. 8 shows the distribution of WGA reactive sites.

Figs. 8a-c. a. The secretory phase. The apical surface shows the WGA reactivity. Stromal cells reveal the reactivity as well. $\times 80$

b. Well differentiated adenocarcinoma (G1). The apical surface of the carcinoma cells shows intense WGA reactivity. $\times 80$

c. Poorly differentiated adenocarcinoma (G3). The carcinoma cells show weak cytoplasmic reactivities. $\times 70$

Fig. 9 shows the distribution of SBA reactive sites.

Figs. 9a, b. a. The proliferative phase. The apical surface of the epithelia shows the SBA reactivity. $\times 70$

b. Well differentiated adenocarcinoma (G1). The apical surface of the carcinoma cells react moderately. $\times 100$

Fig. 10. The secretory phase. In the glandular epithelia, the apical surface as well as cytoplasm react moderately for DBA. $\times 40$

Fig. 11. The secretory phase. The apical surface of the epithelia react moderately for MPA. $\times 80$

Figs. 12 and 13 show the distribution of LFA reactive sites.

Figs. 12a, b. The secretory phase.

a. Without neuraminidase digestion, the apical surface and cytoplasm of the epithelia show the reactivity. $\times 80$

b. Prior neuraminidase digestion eliminates most of the LFA reactivity. $\times 80$

Figs. 13a, b. Well differentiated adenocarcinoma (G1).

a. Without neuraminidase digestion, the apical surface of the carcinoma cells reacts intensely. $\times 80$

b. Prior neuraminidase digestion abolishes the reactivity. $\times 80$

activity was detected on the apical surface in the secretory phase (Fig. 11). In endometrial carcinoma, the reactivity for this lectin was not distinct.

GS-II: In all the cases examined, no significant reactivity was noted except for stromal cells, which exhibited only a faint but definite reactivity.

LFA: In nearly all of the cases of normal endometrium, the apical surface of the epithelia showed reactivity (Fig. 12a). The neuraminidase digestion diminished the reactivity in the normal endometrium (Fig. 12b). In endometrial carcinoma, the apical surface of the carcinoma cells revealed an intense reaction, which was removed easily by prior neuraminidase digestion (Figs. 13a, b).

DISCUSSION

In the present study, the results obtained indicated that various glycoconjugates were expressed in normal as well as neoplastic endometrial tissues. In the normal endometrium, the lectin reactivities were mostly confined to the apical surface of the glandular epithelia. This tissue site is known to be consistently rich in glycoconjugates in most epithelial cells of other organs (10) and was stained for differentiation markers and tumor antigens (9). In the present study, the variation of lectin reactivities with menstrual phases and neoplastic changes was noted in this site as well.

GS-I-B4 binds the membrane glycoconjugates after prior neuraminidase digestion regardless of menstrual phases. This lectin could possibly recognize cell-specific antigens, since neuraminidase engendered GS-I-B4 reactivity has not been reported in other organs. Organ- or cell-specific antigens are not available in the endometrium, and therefore, the neuraminidase engendered GS-I-B4 reactivity would be useful for identifying endometrial tissues. West and Cope likewise noted the neuraminidase-engendered GS-I reactivity of endometrial epithelia (25) and results shown in their report were consistent with the present ones. GS-I is a tetramer and there are two types of subunit, A and B (14). Commercially available GS-I consist of A4 or B4, or might be any combination of A and B subunits. The results of the present study demonstrated that the GS-I affinity for the glandular epithelia depends on the B subunit, which is an excellent marker for blood B antigen (7, 20) and selectively binds terminal α -Gal. Sialic acid is known to bind with one of three sugar residues, sialic acid, β -Gal or β -GalNAc (6), and therefore, sialic acid responsible for masking α -Gal for GS-I-B4 would possibly occupy the terminal position of the adjacent oligosaccharides.

The lectin reactivities of the glandular epithelia were altered in response to their cyclic phases, and were more intense in the proliferative phase in PNA and in the secretory phase in both DBA and MPA. Such results are in agreement with previous reports (2, 4, 25). Since PNA possesses an affinity for terminal β -Gal, and DBA and MPA possess affinities for α -GalNAc (8), these results suggest the alternative appearance of terminal Gal and GalNAc, or the addition of GalNAc to oligosaccharides with terminal Gal as in the type I (3Gal β 1-3GlcNAc β 1) or type II (3Gal β 1-4GlcNAc β 1) core chain. DBA is, on the other hand, well known to show a selective bindability to blood group A antigens (5). Endometrial epithelia nearly lacks blood group antigens (11), and the DBA reactivity of the endometrium was not well correlated with the blood group of the tissue donors. Thus, this lectin (DBA) possibly binds to glycoconjugates other than blood group antigens.

UEA-I and PNA, in particular, following the neuraminidase digestion, preferentially bind neoplastic epithelia.

UEA-I bound carcinoma cells from most cases of endometrial carcinoma, and this result is consistent with previous reports (3, 13, 19, 23, 25). Since UEA-I reactivity was reported in colon cancer (18), it is not specific to endometrial carcinoma. Inoue et al. (11) and Ogasawara (21) reported the expression of blood type H antigens in malignant endometrial tissues regardless of ABO status of the patients. These findings were not inconsistent with ours, as H antigen is well known to be the receptor for this lectin (17).

PNA is a lectin known to bind with β -Gal and T antigen (16). It showed an affinity for carcinoma tissues, especially after the neuraminidase digestion, suggesting that sialic acid occupied the terminal position adjacent to penultimate β -Gal. These findings seem to indicate that endometrial tissues contain T antigens, and terminal sialylations take place in the course of malignant transformations. West and Cope similarly reported the elevated PNA reactivity both in the secretory phase and neoplastic endometrium after neuraminidase digestion (25). However we could not confirm the effect of neuraminidase digestion upon the reactivity in normal endometrium.

All the materials employed in the present study were fixed in paraformaldehyde and embedded in paraffin wax. Thus present results did not necessarily disclose all the glycoconjugates showing affinities to lectins, in as much as glycolipids could be lost during the course of dehydration and rehydration of tissue specimens (1, 15).

REFERENCES

1. Alroy, J., Ucci, A. A. and Pereira, M. E. A.: Lectins: histochemical probes for specific carbohydrate residues, In "Advances in Immunohistochemistry", ed. by R. A. DeLellis, Masson Publishing USA, New York, 1984, p. 67.
2. Aoki, D., Kawakami, H., Nozawa, S., Udagawa, Y., Iizuka, R. and Hirano, H.: Differences in lectin binding patterns of normal human endometrium between proliferative and secretory phases. *Histochemistry* 92; 177-184, 1989.
3. Aoki, D., Nozawa, S., Iizuka, R., Kawakami, H. and Hirano, H.: Differences in lectin binding pattern of normal endometrium and endometrial adenocarcinoma, with special reference to staining with Ulex europeus agglutinin and Peanut agglutinin. *Gynecologic Oncology* 37; 338-345, 1990.
4. Duncan, D. A., Mazur, M. T. and Younger, J. B.: Glycoconjugate distribution in normal human endometrium. *Int. J. Gynecol. Pathol.* 7; 236-248, 1988.
5. Etzler, M. E. and Kabat, E. A.: Purification and characterization of a lectin (plant hemagglutinin) with blood group A specificity from Dolichos biflorus. *Biochemistry* 9; 869-877, 1970.
6. Goldstein, I. J.: Glycoconjugates. Elsvbier, New York, 1964, p.246.
7. Goldstein, I. J., Blake, D. A., Ebis, S., Williams, T. and Murphy, L. A.: Carbohydrate binding studies on the Bandeiraea simplicifolia I isolectins. *J. Biol. Chem.* 256; 3890-3897, 1978.
8. Goldstein, I. J. and Poretz, R. D.: Isolation physicochemical characterization, and carbohydrate-binding specificity of lectins. In "The Lectins: Properties, Functions and Applications in Biology and Medicine," ed. by F. E. Liemer, N. Sharon and I. J. Goldstein, Academic Press, Orlando, Florida, 1986, p. 35.
9. Hakomori, S.: Tumor-associated carbohydrate antigens. *Ann. Rev. Immunol.* 2; 103-126, 1984.
10. Hirano, H., Parkhous, B., Nicholson, G., Lennox, E. S. and Singer, S. J.: Distribution of saccharide residues on membrane fragments from a myeloma-cell homogenate: Its implication for membrane biogenesis. *Proc. Natl. Acad. Sci. USA.* 69; 2945-2949, 1972.
11. Inoue, M., Sasagawa, T., Saitou, J., Shimizu, H., Ueda, G., Tanizawa, O. and Nakayama, M.: Expression of blood group antigens A, B, H, Lewis-a, Lewis-b, in fetal, normal and malignant tissues of uterine endometrium. *Cancer* 60; 2985-2993, 1987.
12. Katsuyama, T., Ono, K., Nakayama, J. and Kanai, M.: Recent advances in mucosubstances histochemistry, In Gastric Mucous Secreting Cells, ed. by K. Kawai, Excerpta Medica, Amsterdam, 1985, p. 3.
13. Kluskens, L. F., Kluskens, J. L. and Bibbo, M.: Lectin binding in endometrial adenocarcinoma. *Am. J. Clin. Pathol.* 82; 259-266, 1984.
14. Lamb, J. E. and Goldstein, I. J.: A structural comparison of the A and B subunits of Griffonia simplicifolia I isolectins. *Arch. Biochem. Biophys.* 229; 15-26, 1984.
15. Leatham, A. and Atkins, N.: Lectin binding to formalin-fixed paraffin sections. *J. Clin. Pathol.* 36; 747-750, 1983.
16. Lotan, R., Skutelsky, E., Danon, D. and Sharon, N.: The purification, composition and specificity of the anti-T lectin from peanut (Arachis hypogaea). *J. Biol. Chem.* 250; 8513-8523, 1975.
17. Matsumoto, I. and Osawa, T.: Specific purification of eel serum and Cytisus sessilifolius anti-H hemagglutinin by affinity chromatography and their binding to human erythrocytes. *Biochemistry* 13; 582-588, 1974.
18. Matsushita, Y., Yonezawa, S., Nakamura, T., Shimizu, S., Ozawa, M., Muramatsu, T. and Sato, E.: Carcinoma-specific Ulex europaeus agglutinin-I binding glycoproteins of human colorectal carcinoma and its relation to carcinoembryonic antigen. *JNCI.* 75; 219-226, 1985.
19. Memon, R. S., Yonezawa, S., Hasui, K. and Sato, E.: Expression of blood group antigens and lectin binding in adenocarcinoma of the endometrium. *Acta Pathol. Jpn.* 40; 509-516, 1990.
20. Murphy, L. A. and Goldstein, I. J.: Five alfa-D-galactopyranosyl-binding isolectins from Bandeiraea simplicifolia seeds. *J. Biol. Chem.* 252; 4739-4742, 1977.
21. Ogasawara, T.: Immunohistochemical study in expression of blood group antigens on endometrial carcinoma—with emphasis on H antigen—. *Adv. Obstet. Gynecol.* 39; 223-234, 1987.
22. Pettersson, F.: Annual Report gynecological cancer F. I. G. O., Vol. 20, International Federation of Gynecology and obstetrics, Stockholm, 1988, p. 76.
23. Soederstroem, K. O.: Lectin binding to formalin-fixed paraffin sections of human endometrium. *Int. J. Gynecol. Pathol.* 6; 55-65, 1987.
24. Tanaka, K. and Hirano, H.: Changes in soybean agglutinin (SBA) and peanut agglutinin (PNA) binding pattern in the epidermis of the developing chick embryo. *Dev. Growth. Diff.* 25; 299-305, 1983.
25. West, K. P. and Cope J. L.: The binding of peroxidase-labelled lectins to human endometrium in normal cyclical endometrium and endometrial adenocarcinoma. *J. Clin. Pathol.* 42; 140-147, 1989.