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A lectin histochemistry comparative study in human normal prostate, benign prostatic hyperplasia, and prostatic carcinoma

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The partial oligosaccharide sequences of glycoconjugates and the nature of their glycosidic linkages were investigated in normal human prostate, benign prostatic hyperplasia (BPH) and prostatic carcinoma by means of lectin histochemistry, using light microscopy and Western blot analysis. The labeling pattern of BPH differed from that of normal prostate in having more intense staining with DSA, HPA, UEA-I and AAA, and in showing lesser staining with WGA and SBA. Prostatic carcinoma differed from normal prostates in displaying the more intense labeling with PNA, DSA, SBA, DBA, UEA-I and AAA, and in having lesser labeling with WGA. The main differences in labeling pattern between prostatic carcinoma and BPH were that the latter specimens showed more marked staining with PNA, DSA, DBA, SBA, UEA-I and AAA, and lesser staining with WGA and HPA. The staining patterns of SNA, MAA, ConA, LCA and GNA were similar in all three groups of specimens. For most of the lectins studied, including those showing a similar immunohistochemical staining in the three groups of specimens studied, the Western blot analysis showed differences in the banding pattern among normal, hyperplastic, and carcinomatous prostates. Present results suggest that the glycosylation of proteins was modified in both BPH and prostatic carcinoma. In BPH a strong expression of N-acetylgalactosamine residues occurred, while in prostatic carcinoma an increase of sialic acid, galactose and fucose residues was observed. No changes in mannose residues were detected.

Keywords: **lectins, Western blot, normal prostate, benign prostatic hyperplasia, prostatic carcinoma.**

Introduction

The human prostate contains three major glandular regions [1]: (1) the central zone, which comprises approximately 25% of the glandular prostate, is a conical region radiating from the verumontanum to the base of the prostate along the course of the ejaculatory ducts. Acini in this zone are large and irregular, and their epithelium shows a granular cytoplasm; (2) the peripheral zone, that comprises about 70% of the mass of the glandular prostate, contains small round acini lined by cells that have a pale, non-granular cytoplasm; and (3) the transition zone, which represents only the 5% of the glandular tissue, consists of two independent small lobes located at the mid level of the prostate. Glands of this zone are identical to the peripheral zone acini, but are less numerous and are surrounded by a more dense stroma. The importance of these morphologic differences has been highlighted by retrospective studies that have shown that adenocarcinoma arises mainly in the peripheral zone, whereas the central zone appears relatively refractory to the development of this neoplasm [2].

The biological importance of glycoconjugates derives from the fact that an enormous variety of glycans can be generated from a relatively limited number of monosaccharides. Thus, they offer a high capacity for carrying biological information [3–4]. Glycoconjugates are located both intracellularly and extracellularly and also at the cell surface, where they can be secretory or structural. Cell surface glycoconjugates undergo modification in association with certain cellular phenomena such as cell differentiation, adhesion, proliferation, morphological changes, functional modulation of cells, and under pathological conditions [5]. In the male accessory sex glands, complex glycans are important for the functional activities of these organs [6].

Several lectin histochemical studies have been carried out in human prostates in normal, hyperplastic or carcinoma-

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tous conditions [7]. Binding to Concanavalin A and wheat germ agglutin have been described in the normal and hyperplastic epithelia, and also in well-differentiated carcinomata [8]. Peanut agglutinin binding in human prostatic epithelium was present in the normal gland, but it was absent prepubertally and decreased in advanced age [9]. In another study, PNA binding was demonstrable in prostatic hyperplasia, but only after removal of sialic acid by neuraminidase digestion [10]. In benign prostatic epithelium, the *Ulex europaeus* binding site is predominantly glycolipid-based, but, following malignant transformation, becomes predominantly glycoprotein-based [11]. Drachenberg and Papadimitriou [12] have performed a lectin binding study in low and high grade prostatic intraepithelial neoplasia. In these studies, the only enzymatic treatment reported was neuraminidase [10,13–15], and neither Wester-blot analyses nor quantitations of staining intensities have been carried out.

The aim of the present report was to perform a comparative, lectin-histochemical study of normal, hyperplastic, and carcinomatous prostates, using a battery of 13 lectins, in combination with enzymatic and chemical treatments, Western blot analysis, and a semiquantitative study, to investigate the differences in oligosaccharide sequences of glycoconjugates and the nature of the glycosidic linkages among the three groups of specimens.

Material and methods

Materials

The prostates from 30 men (between 55 and 85 years of age) were obtained from (a) transurethral resections in 15 men who were clinically and histopathologically diagnosed as having benign prostatic hyperplasia (BPH); and (b) radical prostatectomies in 15 men who had prostatic adenocarcinoma and showed neither metastasis nor lymph node infiltration at the time of surgery. In addition, 10 prostates from 20- to 50-year-old men without reproductive, endocrine and related diseases were obtained between 8 and 10 h after death in autopsies. Five of these men showed BPH and the other five men presented histologically normal prostates. The samples were processed for light microscopy and Western blotting analysis.

Light microscopy

The tissues were fixed in 10% (v/v) formaldehyde in phosphate buffered saline pH 7.4, for 24 h at room temperature. The samples were routinely processed, embedded in paraffin wax, and 5 μm sections were obtained. Sections were deparaffinized in xylene and hydrated in a graded ethanol series. The lectin binding pattern was established using horseradish peroxidase (HRP)-and digoxygenin (DIG)-conjugated lectins.

For histochemical staining using HRP-conjugated lectins, endogenous peroxidase was previously blocked with 0.3% (v/v) hydrogen peroxide in Tris-buffered saline (TBS), and the sections were incubated for 90 min at room temperature with the following HRP-conjugated lectins (Table 1): PNA (25 μg/ml), Con A (20 μg/ml), LCA (20 μg/ml), WGA (10 μg/ml), SBA (18 μg/ml), UEA-I (20 μg/ml), HPA (6 μg/ml), and DBA (15 μg/ml). Peroxidase was developed with 0.05% (w/v) 3,3′-diaminobenzidine and 0.015% (v/v) hydrogen peroxide in TBS. Peroxidase-labeled lectins were purchased from Sigma, Barcelona, Spain.

For labeling with lectin-DIG conjugates, a two-step technique was applied. In brief, endogenous peroxidase activity was destroyed by a 30 min treatment with 0.3% (v/v) H_2O_2 in TBS. Sections was rinsed in TBS, covered with 1% (w/v) BSA (bovine serum albumin) in TBS for 10 min, and incu-

Table 1. Carbohydrate binding specificity of lectins

Lectin	*Abbreviation*	*Carbohydrate binding specificity*
Wheat germ agglutinin	WGA	$(GlcNAc)_n$
Sambucus nigra agglutinin	SNA	Neu5Acα2,6Gal; Neu5Acα2,6GalNAc
Maackia amurensis agglutinin	MAA	Neu5Acα2,3Galβ1,4GlcNAc
Arachis hypogaea agglutinin	PNA	Galβ1,3GalNAc
Datura stramonium agglutinin	DSA	Galβ1,4GlcNAc
Dolichos biflorus agglutinin	DBA	GalNAc
Glycine max agglutinin	SBA	GalNAc
Helix pomatia agglutinin	HPA	GalNAcα1,3GalNAc
Canavalia ensiformis agglutinin	Con A	αMannose> αGlucose
Lens culunaris agglutinin	LCA	Mannose/Fucose
Galanthus nivalis agglutinin	GNA	$(Mannose)_{1-3}$
Ulex europaeus agglutinin	UEA-I	L-Fucose
Aleuria aurantia agglutinin	AAA	L-Fucose

GlcNAc: N-acetylglucosamine. Neu5Ac: sialic acid. Gal: galactose. GalNAc: N-acetylgalactosamine. Fuc: Fucose.

bated in the following lectin-DIG conjugates for 90 min at room temperature: DSA (10 μg/ml), MAA (10 μg/ml), AAA (20 μg/ml), SNA (15 μg/ml), and GNA (60 μg/ml) (Table 1). After two rinses for 5 min in TBS, sections were incubated with peroxidase-conjugated anti-DIG Fab fragments (0.6 U/ml) (Boehringer-Mannheim, Mannheim, Germany) in TBS supplemented with 1% (w/v) BSA for 60 min at room temperature. Peroxidase activity was visualized with 0.05% (w/v) DAB and 0.015% (v/v) H_2O_2. Sections were counterstained with Harris hematoxylin. Digoxygenin-conjugated lectins were purchased from Boehringer-Mannheim.

Chemical treatments

To expose those carbohydrates that could be masked by sialic acid residues, acid hydrolysis was carried out by immersing the paraffin sections in 0.1 M HCl for 2–3 h, at 82 °C.

Removal of sulphate esters without modification of the oligosaccharide chains in the glycoproteins, was carried out by the desulfation technique, which consists of a sequential methylation-saponification processes. Paraffin sections were first treated with 0.15 N HCl in absolute methanol (5 h at 60 °C), and thereafter with 1.8% (w/v) $Ba(OH)_2$ in aqueous solution (1 h at 0–4 °C).

To eliminate O-linked oligossacharides (β-elimination reaction), the paraffin sections were incubated with 0.5 N NaOH in 70% (v/v) ethanol, at 4 °C, for 5 days [16].

Enzymatic treatments

Hydrolysis of N-linked oligosaccharides was carried out by enzymatic treatment with Endo-β-acetylglucosaminidase F/peptide N-glycosidase F (Endo F/PNGase F) (Boehringer-Mannheim). After incubation in 0.1 M Tris, 150 mM NaCl, 2.5 mM EDTA (pH 9) buffer containing 1% (w/v) BSA for 10 min, and a brief washing in the buffer without BSA, the sections were incubated with the enzyme, at a dilution of 6 U/ml, for 3 days.

Elimination of terminal glucose residues was carried out by the enzyme glucose oxidase from *Aspergillus niger* (Sigma). After incubation in 0.2 M sodium acetate buffer (pH 5) for 5 min, the sections were incubated with the enzyme, which oxidizes glucose to glucuronic acid, and H_2O_2, at a dilution of 50 U/ml, for 12 h at 37° C.

Histochemical controls

Two types of controls were used: (1) substitution of conjugated and unconjugated molecules by the corresponding buffer; and (2) preincubation of the lectins with the corresponding hapten-sugar inhibitor, used at a range of concentration from 0.025 to 1 M. The inhibitors were: D-Gal for PNA; methyl-α-mannoside for Con A, LCA and GNA; D-N-acetylgalactosamine for HPA, DBA and SBA; D-N-acetylglucosamine for WGA; N-acetyllactosamine for DSA; L-fucose for AAA, LTA, and UEA-I; and Neu5Ac for SNA. The inhibitor used for MAA was 0.1M Neu5Ac (α 2,3) lactose. The inhibitor sugars were purchased from Sigma. An additional control for SNA and MAA was pretreatment of the sections with 1 U/ml of *Vibrio cholerae* sialidase, diluted in acetate buffer (pH 5), for 3 h at 37 °C to remove sialic acid residues from tissue sialoglycoconjugates.

Quantitative study

A quantification of staining intensity in normal, hyperplastic, and neoplastic prostates was performed for each of the thirteen lectins. Of each prostate, six histologic sections of each region (central, transitional and peripheral) were selected at random, and the staining intensity of the epithelium and stroma was measured with an automatic image analyzer (MIP4 version 4.4, Consulting Image Digital, Barcelona, Spain) in five light microscopic fields using the ×40 objective. Delimitation of both types of surface areas (epithelium and stroma) was carried out manually using the mouse of the image analyzer. The labeling intensity of each cell type (basal cells, columnar cells, and stromal cells) could not be estimated because it was not possible to determine the borders of each individual cell. The staining intensity was measured by using a gray level scale (0: white; 255: black) with a negative image.

Western blot analysis

Western blotting analysis was separately carried out for each of thirteen lectins at described by Towbin et al. [17]. The prostates were homogenized in 0.5M Tris-HCl buffer (pH 7.4) containing 1 mM EDTA, 12 mM 2-mercapto-ethanol, and 1 mM phenylmethylsulphonyl fluoride (PMSF). The homogenates were centrifuged at 10000 g for 30 min. After boiling for 2 min at 98 °C, aliquots of 200 μg of protein were separated in SDS-polyacrylamide (9%, w/v) slab minigels, according to the procedure of Laemmli [18]. Separated proteins were transferred for 4 h to 0.25A to nitrocellulose paper and, thereafter, the nitrocellulose sheets were soaked in blocking solution (1 mM glucose, 1% (w/v) BSA, 0.5% (v/v) Tween-20, 10% (v/v) glycerol in PBS, pH 7.3) overnight at 37 °C and then incubated with HRP-conjugated lectins or lectin-DIG conjugates at 1:500 dilution in blocking solution for 2 h. After extensive washing with PBS-Tween-20 (for lectin-DIG conjugates) the sheets were incubated with a peroxidase-conjugated anti-DIG Fab fragments (0.6 U/ml) (Boehringer-Mannheim, Mannheim, Germany) in blocking solution. The filters were developed with an enhanced chemiluminescence (ECL) Western blotting analysis, following the procedure described by the manufacturer (Amersham).

Results

The results of the semiquantitative study on histological sections are summarized in Table 2. Western blot analysis is showed in Figure 1.

Table 2. Lectin histochemistry in human normal prostate, benign prostatic hyperplasia and prostatic carcinoma

	Columnar cells			*Basal cells*		*Stroma*			
Lectins	*NP*	*BPH*	*PC*	*NP*	*BPH*	*NP*	*BPH*	*PC*	*Results of treatments*
WGA	4	3	2	2	2	2	2	2	Staining decreased with β-elimination or Endo-F digestion
SNA	2	2	2	2	2	1	1	1	Staining disappears with β-elimination and increases with Endo-F digestion
MAA	2	2	2	2	2	1	1	0	Staining disappears with Endo-F digestion
PNA	1	1	3	1	1	2	2	2	Staining increases with desulfation or desialylation and disappears with β-elimination
DSA	1	2	3	0	2	1	2	2	Staining increases with desulfation or desialylation and disappears with Endo-F digestion
DBA	0	0	2	0	0	0	0	1	Staining increases with desulfation and desialylation (in PC) and disappears with β-elimination
SBA	2	1	4	2	1	2	1	1	Staining increases with desulfation or desialylation (in PC) and disappears with β-elimination
HPA	1	4	1	1	3	1	2	1	Staining increases with desulfation and desialylation (in PC) and disappears with β-elimination
Con A	3	3	3	1	2	1	1	1	Staining disappears with Endo-F digestion
BLCA	1	2	2	2	2	2	2	1	Staining disappears with β-elimination
GNA	3	3	3	3	3	1	1	1	Staining disappears with Endo-F digestion
UEA-I	1	2	3	1	2	2	1	1	Staining decreases with β-elimination or Endo-F digestion
AAA	1	2	3	1	3	2	1	1	Staining decreases with β-elimination or Endo-F digestion

NP: normal prostate; BPH: benign prostatic hyperplasia; PC: prostatic carcinoma. 0: no staining (grey level <60); 1: slight staining (grey level: 60–85); 2: moderate staining (grey level: 85–110); 3: intense staining (grey level: 110–140); 4: strong staining (grey level: >140).

Reactivity to WGA was observed in the apical cytoplasm and cellular limits of epithelial cells. The least intense staining was observed in prostatic carcinoma, and the most intensity in normal prostate (Figs. 2 and 3). The stroma showed a moderate reactivity in the three groups of specimens. Western blot analysis revealed some differences in the banding pattern among the three groups of specimens: reactivity in the bands of 34, 177 and 207 kDa was higher in prostatic carcinoma than in normal and hyperplastic prostates; the band of 88 kDa was weaker in BPH and prostatic carcinoma than in normal prostates; and a 38 kDa band was only observed in BPH.

In the three groups of specimens, SNA showed a moderate reaction in the apical cytoplasm of epithelial cells and a slight reaction in the stroma. A 42 kDa protein band, present in normal and hyperplastic prostates, disappeared in prostatic carcinoma.

MAA also labeled the apical cytoplasm of epithelial cells in the three groups of specimens. The stromal reaction was weak in normal and hyperplastic prostates, and negative in carcinoma. Western blot analysis revealed that in prostatic carcinoma the expression of a protein of 40 kDa was increased, while the staining of protein bands between 60 and 90 kDa decreased.

PNA showed a weak reaction in epithelial cells in normal and hyperplastic prostates. In contrast, most epithelial cells showed an intense reaction in prostatic carcinoma. The stroma stained moderately in three groups of specimens. The results of the Western showed that new bands with molecular weight of 37, 42, 45 and 53 kDa appeared in prostatic carcinoma.

Epithelial and stromal reactions to DSA were weak in normal prostates and moderate in the hyperplastic glands. In prostatic carcinoma, labeling was also moderate in stroma and very intense in the epithelium. DSA blots showed the appearance of a new glycoprotein of 32 kDa in adenocarcinoma.

DBA did not show reaction in normal and hyperplastic prostates. A moderate reaction was found in the epithelium and stroma of adenocarcinoma specimens. The blot analysis revealed a low molecular weight band in the carcinoma sheets.

SBA showed a moderate staining in the epithelium and stroma of normal prostates. In hyperplastic glands, the reaction only appeared in isolated epithelial and stromal cells. In prostatic carcinoma labeling was very intense in the epithelium and weak in the stroma (Figs. 4 and 5). With Western blot analysis, the reactivity in the bands of 38, 43

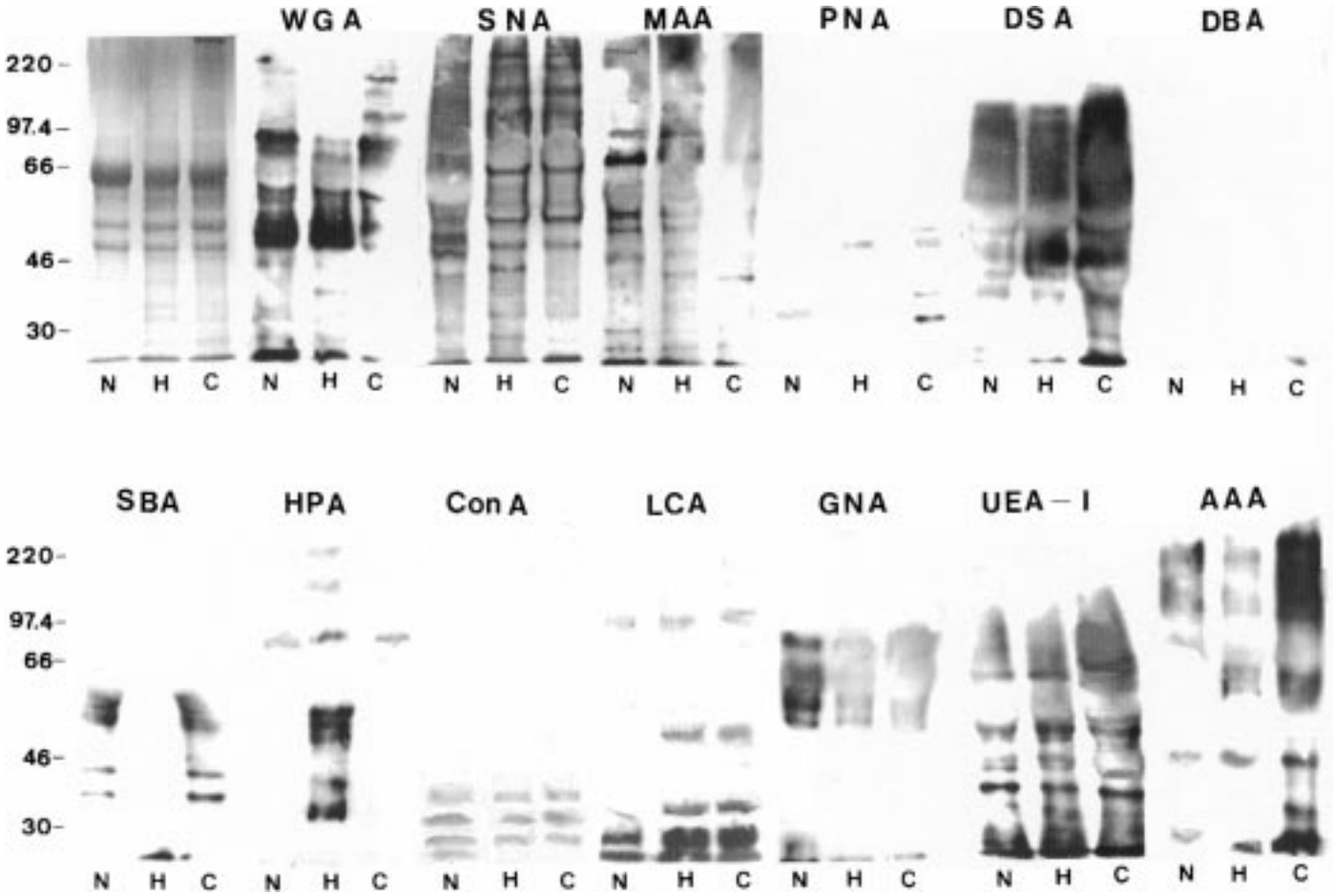


Figure 1. Lectin staining of Western blots from SDS-PAGE gels of human prostate. N: normal prostate. H: benign prostatic hyperplasia. C: prostatic carcinoma. Numbers on the left margin are molecular weight in kDa. The first group of columns is a gel stained with Coomassie Blue.

and 63 kDa observed in both normal and neoplastic prostates disappeared in BPH.

Epithelial and stromal reaction to HPA was weak in both normal prostate and carcinoma, and intense in BPH (Figs. 6 and 7). Western blotting showed bands at 35, 50, 122 and 195 kDa in BPH, but not in normal and carcinomatous prostates.

Reaction to ConA was intense in the epithelium of normal prostate, BPH, and adenocarcinoma. In the three groups of specimens, the stromal reaction was weaker. The Western blot pattern was also similar in the three groups.

Epithelial reaction to LCA was slight in normal prostate and between moderate and intense in BPH and carcinoma. Stromal reaction was moderate in normal and hyperplastic prostates, and slight in carcinoma (Figs. 8 and 9). The bands labeled with LCA showed that reactivity in most of the bands was more intense in both benign prostatic hyperplasia and carcinoma than in normal prostates.

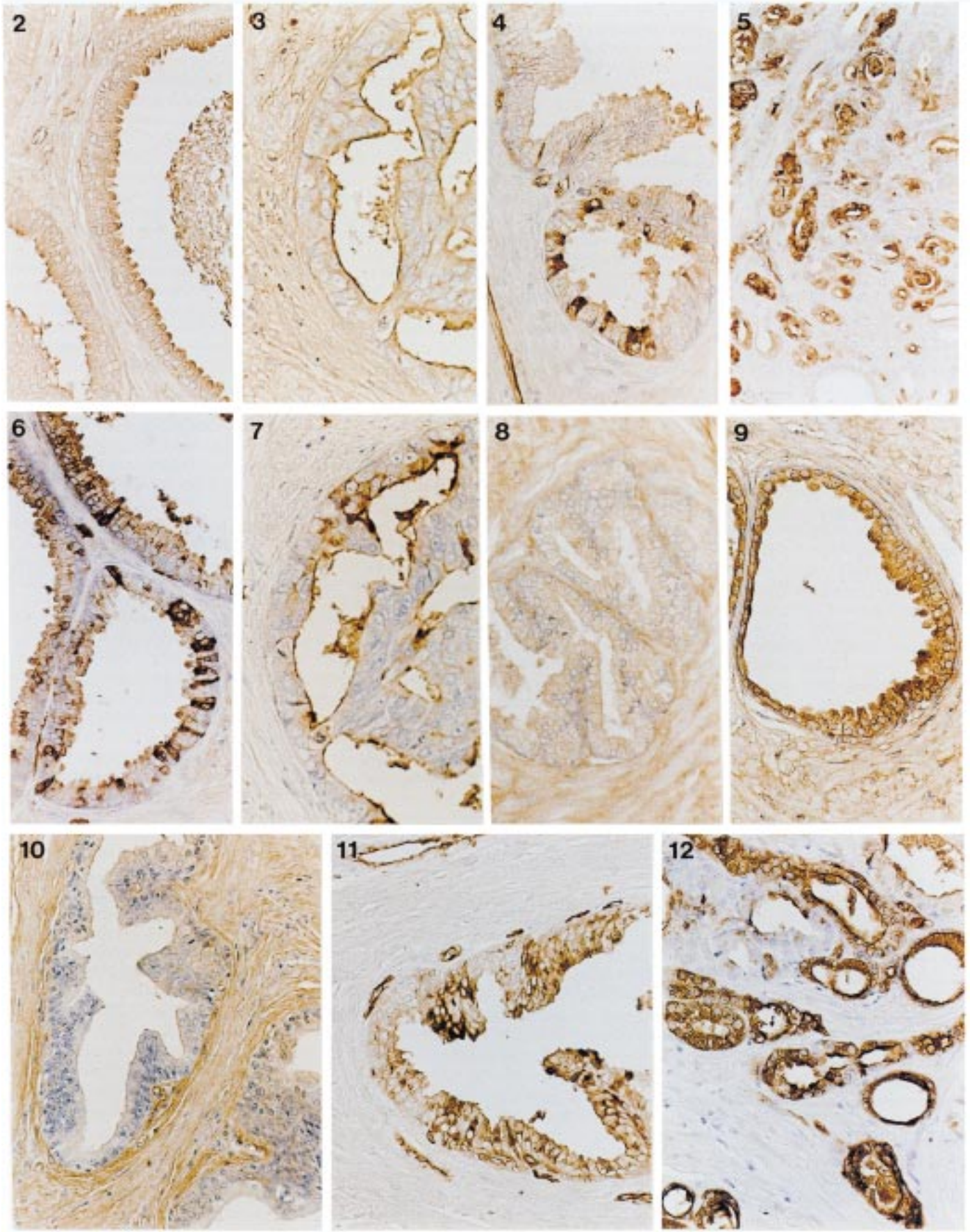
In the three groups of specimens, reaction to GNA was slight in the stroma and very intense in the glands. The Western blot analysis showed that the label location was similar in all three types of prostatic tissue, although in the normal prostate bands, the band of 87 kDa appeared more intensely stained than in BPH and carcinoma.

Epithelial reaction to both UEA-I and AAA was weak in normal prostates, moderate in BPH, and intense in carcinoma. Stromal reaction was moderate in normal prostate, and weak in BPH and carcinoma (Figs. 10 to 12). With Western blot analysis, the different tissue extracts showed similar banding patterns, but in prostatic carcinoma, the bands showed a stronger reaction.

The results of enzymatic and chemical treatments are also shown in Table 2.

Discussion

Jansen et al. [7] have observed that the expression of PNA increases from BPH through prostatic intraepithelial neoplasias to cancers. These authors also established a significant positive correlation between the numbers of PNA acceptors and those of prostatic specific antigen. In the present study, PNA expression of epithelial cells was in-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creased in prostatic carcinoma. Since the PNA labeling disappeared with the β-elimination reaction and was not modified with Endo F/PNGase F digestion, it may be deduced that the PNA-labeled residues belonged to O-linked oligosaccharides. The modification of the labeled sequences by sialic acid and sulfate groups revealed an increase of negative charges. Since the reactivity to SNA was also localized in O-linked glycoproteins, we could assume that these sialic acid residues are of the type Neu5Acα2,6-. In addition, Neu5Acα2,3 residues that are N-linked to Galβ1,4GlcNAc chains are probably present, because the reactivity to DSA lectin increased after the desialylation process. From these findings it can be concluded that the development of prostatic carcinoma is associated with an increase in galactose residues and that some of these residues are masked by sialic acid and sulfate groups.

The presence of GalNAc residues has been investigated with DBA, SBA and HPA. Loy et al. [19] reported positive staining for SBA in BPH, atypical hyperplasia, and prostatic carcinoma. It has also been reported that HPA binding is associated with biologically aggressive tumors [20]. In human prostatic carcinoma, Shiraishi et al. [21] observed that HPA expression is more common in metastatic than in non-metastatic tumors. We have encountered an increased expression of both DBA and SBA in prostatic carcinoma, although no differences in HPA staining were found between normal prostate and prostatic carcinoma. This agrees with the absence of metastasis in the prostatic carcinoma patients studied here. The fact that the reactivity of prostatic carcinoma to HPA differs from that to both DBA and SBA might be attributed to the residues that are labeled: whereas DBA and SBA each label certain oligosaccharides terminating in GalNAc residues, HPA labels a wide range of smaller glycans. Since the reactivity with the three lectins was abolished with the β-elimination reaction in the three groups of specimens, the labeled residues belong to glycoproteins with O-glycosidic linkage to proteins.

Altered sialylation of glycoproteins and glycolipids is a common feature of malignancy [22,23]. Increased negative charge on the surface of tumor cells might be a possible cause of reduced intercellular adhesion and hence increased metastatic potential [24]. However, the present results concerning SNA (which labels Neu5Acα2,6 residues), indicate that the amounts of sialic acid residues in prostatic carcinoma are similar to those found in normal prostate and BPH. In prostatic epithelial cells, the residues recognized by SNA belong to O-linked glycoproteins while the Neu5Acα2,3 residues, labeled by MAA, are present in N-linked glycoproteins. This sialic acid recognized by MAA in prostatic carcinoma is probably linked to the galactose residues that are labeled by DSA. Both stainings disappeared with Endo-F digestion.

McNeal et al. [25] failed to find staining with ConA and LCA. However, we have visualized a moderate staining with LCA, and a more intense reaction to both Con A and GNA. The mannose residues recognized by these lectins (glucose oxidase digestion did not modify the reactivity to these lectins) posses different linkages to glycoproteins. GNA stained exclusively N-linked mannose residues. The reactivity to Con A disappeared from the epithelium when the sections were subjected to Endo F/PGNase digestion, which suggests that these mannose residues are N-linked to glycoproteins. The mannose residues labeled by LCA belong to glycoproteins with O-linkage glycosidic, because they are eliminated with the β-elimination reaction.

Figure 2. Hyperplastic glands stained with WGA lectin. The epithelial cells show an intense reaction in their apical cytoplasm. The lumen is also stained. ×250.

Figure 3. Prostatic carcinoma stained with WGA lectin, the epithelial cells show a moderate reaction in their apical cytoplasm. The stroma is slightly stained. ×250.

Figure 4. Hyperplastic glands stained with SBA lectin. Reaction is very intense in several epithelial cells and in some stromal cells. ×250.

Figure 5. Prostatic adenocarcinoma showing intensely SBA labeled microglands. ×250.

Figure 6. Hyperplastic glands labeled by HPA. Some of the epithelial cells reacts intensely. The stroma shows a slight reaction. ×250.

Figure 7. Weak reaction to HPA in prostatic adenocarcinoma. ×250.

Figure 8. Human normal prostate labeled by LCA. The staining is homogeneously distributed and it is more intense in the stroma than in the epithelium. ×250.

Figure 9. Moderate staining with LCA in the epithelium of hyperplastic glands. ×250.

Figure 10. Normal prostate stained with UEA-I lectin. The epithelium shows a slight reaction, the reactivity is higher in the stroma. ×250.

Figure 11. Hyperplastic gland showing a moderate reaction to UEA-I lectin in the epithelium, and a weak reaction in the stroma. ×250.

Figure 12. Prostatic carcinoma with a intense reaction to UEA-I in the microglands. No staining is seen in the stroma. ×250.

Using UEA-I, which is a marker for the H antigen, McNeal et al. [15] found positive staining in the central zone of the prostate, but not in the peripheral zone. In contrast, Perlman and Epstein [26] observed an increased expression of H type-2 in prostates showing dysplasia or less-differentiated adenocarcinomata. With UEA-I and AAA, we have observed a moderate staining pattern in normal prostate, intense in hyperplasia and strong in carcinoma. These fucose residues belong to N- and O-linked glycoproteins (both β-elimination reaction and Endo-F digestion disminished the staining).

In human prostate, Quemener et al. [27] have purified the enzyme testosterone 5α-reductase 2. This seems to be a O-glycosylated sialoglycoprotein with oligosaccharide side chains composed of mannose, fucose and N-acetylgalactosamine, which is recognized by ConA, DBA, UEA-I and RCA. It is possible that modifications in the enzyme glycosylation, occurring in benign hypertrophy and cancer, are involved in the regulation of the enzymatic activity.

From the Western blot analysis it can be concluded that the expression of glycosylated proteins differs between normal prostate, BPH and prostatic carcinoma. In BPH a strong expression of GalNAc residues occurred, while in prostatic carcinoma an increase of sialic acid, galactose and fucose residues was observed. These increased reactions could be related to the increased cell proliferation; however, the histochemical study revealed that staining was increased in each individual cell and that the intracellular staining distribution also changed for some lectins. These changes in lectin reactivities could be related to changes in glycosylation patterns in order to regulate the activity of different enzymes. With the techniques used here, it cannot be detected if the different reaction intensities are also due the appearance of new glycosylated proteins.

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