Folia Neuropathologica

Lectin binding pattern in meningiomas of various histological subtypes

Anna Taraszewska, Ewa Matyja

Department of Experimental and Clinical Neuropathology, Mossakowski Medical Research Centre, Polish Academy of Sciences, Warsaw, Poland

Folia Neuropathol 2007; 45 (1): 9-18

Abstract

Altered tumour cell glycosylation in relation to cellular heterogeneity in human brain tumours remains relatively unexplored. It has been reported that meningiomas express variability in glycosylation properties; however only limited meningioma subtypes have been studied with lectins histochemistry.

The aim of this study was to compare the binding pattern of biotinylated lectins in seven subtypes of histologically benign intracranial meningiomas (meningothelial, transitional, fibroblastic, psammomatous, secretory, microcystic and angiomatous types). The study was performed on biopsy material of 30 cases of meningiomas with different lectins: Peanut agglutinin (PNA), Soybean agglutinin (SBA), Dolichos biflorus agglutinin (DBA), Wheat germ agglutinin (WGA), Cocanavalin A (Con A) and Ulex europaeus agglutinin 1 (UEA-1).

The expression of lectin-binding glycoconjugates exhibited differences between certain subtypes of meningiomas. WGA with affinity for GlcNAc and neuraminic acid labelled the cells of all meningiomas but most intensely those of fibroblastic type. Staining with PNA, SBA and DBA, which are GalNAc specific, varied from negative to strongly positive. Enhanced PNA reactivity reflected mainly cytoarchitectural pattern of tumour growth, such as syncytial lobules, whorled formations or trabecular arrangements of meningioma cells. DBA labelled the majority of cellular nuclei. SBA showed binding to psammoma bodies, while pseudopsammoma bodies were stained with PNA, WGA, Con A, and to a lesser extent with SBA and DBA. The secretory meningiomas exhibited strong and heterogeneous lectins reactivity within pseudopsammoma bodies whereas the neoplastic cells were only occasionally stained. The selective reactivity of UEA-1 with endothelial cells of blood vessels resulted in a specific visualisation of the vascular network in all histological subtypes of meningiomas. These results documented the heterogeneous glycosylation pattern in different subtypes of meningiomas and indicate the usefulness of lectins in the evaluation of pluripotential differentiation of meningioma cells.

Key words: *lectin histochemistry, benign meningiomas, tumour cell heterogeneity*

Communicating author:

Anna Taraszewska, MD, Department of Experimental and Clinical Neuropathology, Mossakowski Medical Research Centre, Polish Academy of Sciences, Pawińskiego 5, 02-106 Warsaw, Poland, tel.: +48 22 608 65 41, fax: +48 22 668 55 32, Email: tara@cmdik.pan.pl

Introduction

Lectins are carbohydrate-binding proteins with high affinity to different types of carbohydrate terminal moieties of cellular glycoproteins in plant and animal tissues [17,41,42]. Endogenous lectins are implicated in the function of the intercellular recognition system and play an important role in a variety of biological processes, including regulation of cellular growth, differentiation and morphogenesis [51]. On the other hand, a multitude of isolated plant lectins have been found useful in identification of glycoconjugates by means of lectin histochemistry [15,27]. Based on the carbohydrate-binding specificity, biotinylated plant lectins are widely used as markers of particular mono- and oligosaccharides and might be used to study the changes in carbohydrate structures of cellular glycoproteins and glycolipids in various physiological and pathological conditions [41,42]. In particular, the expression of lectin-binding glycoconjugates has been extensively investigated in the processes of cellular differentiation and maturation during development [11,18] and after neoplastic transformation [15]. It has been known that tumour cells frequently express a glycosylation pattern different from those present on their normal counterparts. Numerous studies have shown that in malignant epithelial tumours some forms of glycosylation changes correlated with metastatic properties, grading and progression of the tumour [7,8,26]. Altered glycosylation pattern of cells has been also demonstrated in pathological processes in the central nervous system, including ischaemia [43,44], Alzheimer's disease [45] and brain neoplasms [10,30,39,40]. Studies performed in variable human [13,47] and experimental gliomas [14] have indicated that lectins may be useful as markers of neoplastic cell differentiation rather than of tumour malignancy. The use of biotinylated lectins for diagnostic or prognostic purposes is notably limited because of great variation in the expression of glycoconjugates in tumour cells. Nevertheless, lectin histochemistry has been considered to be a valuable method for the study of tumour-associated alterations of cellular glycosylation and tumour cell heterogeneity [8,15,41]. Lectin histochemistry in respect of cellular heterogeneity of human brain tumours remains relatively unexplored.

In this study we examined the expression of specific lectin-binding glycoconjugates in tumour cells of several histologic subtypes of meningiomas. These tumours are typically benign, slow growing and characterised by a variety of histomorphological features [5,21]. The neoplastic cells exhibit a wide range of differentiation and/or metaplasia [25,31,32,46] and might produce fibroblastic, epithelial, secretory, angiomatous, lipomatous, cartilaginous and even bone elements. The distinct predominance of one of these components allows the meningiomas to be classified according to WHO classification into 9 histological subtypes of I grade malignancy [28].

The meningioma cells display a large amount of glycoproteins and proteoglycans, which are intrinsic constituents of the cell cytoplasm, basement membranes and extracellular matrix and may be involved in the mechanism of neoplastic cell transformation [9,12,49]. In previous studies differences between some histological subtypes of meningiomas in the expression of both cell surface carbohydrate-containing molecules [3,4,23,29,37] and carbohydrate-binding endogenous lectins [2] were detected. However, only limited meningioma subtypes were included in the study of lectin histochemistry.

The aim of this study was to compare the binding pattern of biotinylated lectins in seven histopathological subtypes of meningiomas of I grade malignancy, including uncommon secretory, microcystic and angiomatous variants (Table I).

Materials and methods

The study was performed on the archival material of surgical specimens of intracranial meningiomas derived from patients operated on in neurosurgical departments. A series of 30 cases of histologically benign meningiomas of different subtypes, diagnosed according to the WHO guidelines of classification and grading of brain tumours [28], was included for the study (Table I).

For immunohistochemical staining six biotinylated lectins, obtained from Vector Laboratories (Burlingame, CA, USA), were used: Peanut agglutinin (PNA), Soybean agglutinin (SBA), Dolichos biflorus agglutinin (DBA), Wheat germ agglutinin (WGA), Cocanavalin A (Con A) and Ulex europaeus agglutinin I (UEA-1); their specific glycoconjugates are presented in Table II. Before incubation with lectins, the deparaffinized sections were washed in

0.1M phosphate buffered saline (PBS) pH 7.4 and treated with 0.05M NH4Cl in PBS for 30 min, with 3% H_2O_2 in PBS for 5 min and with 1% bovine serum albumin in PBS for 20 min. The sections were incubated with biotinylated lectins, diluted in PBS to a concentration of 10 µg/ml for 1h. Then, they reacted with avidin-biotin peroxidase complex reagent (ABC Kit) for 1 h and with 0.05% 3'3'diaminobenzidine solution (DAB, Dako) for 10 min. Finally the sections were rinsed in tap water and distilled water, counterstained with haematoxylin and embedded in Permount.

Table I. Histological subtypes of meningiomas

Meningioma subtype	No. of cases
Meningothelial	4
Transitional	5
Fibroblastic	3
Psammomatous	2
Secretory	7
Microcystic	4
Angiomatous	5

Results

The distribution and intensity of lectin binding sites revealed distinct differences between particular examined meningioma subtypes.

Meningiomas of the menigothelial type demonstrated a relatively weak staining of tumour cells in syncytial cell arrangements with the majority of lectins. In some areas, particularly in the central parts of tumour lobules, intense reactivity of both cytoplasm and surface membranes was seen with PNA (Fig. 1). Labelling of tumour tissue was moderately uniform with WGA and very slight or negative with Con A. Some areas of slight positive staining of tumour cells were noted for SBA (Fig. 2) and DBA. Moreover, DBA often labelled cellular nuclei and perinuclear cytoplasm of many cells (Fig. 3), and this pattern was found in every type of examined meningiomas. Regardless of the meningioma subtype, binding of SBA to blood-derived leucocytes and macrophages and vessel walls was also observed (Fig. 2). In addition, SBA displayed marked reactivity with the psammomatous bodies, mostly within their central core (Fig. 4). Staining of the psammomatous bodies with other lectins was usually negative (Fig. 5).

The neoplastic cells of psammomatous meningiomas showed reactivity with the lectins most similar to those seen in transitional meningiomas.

The transitional type of meningiomas exhibited enhanced binding of the lectins to neoplastic cells arranged in whorls, accompanied by weak or negative immunolabelling in areas of syncytial cell arrangements. The cell processes and bodies of closely wrapped cells were strongly stained by PNA (Fig. 6) and to a smaller extent by SBA, Con A and DBA (Fig. 5). Labelling with SBA was seen only in some selected whorls dispersed within unstained or weakly stained areas of the tumour. WGA showed a more uniform staining of cell cytoplasm through the whole tumour tissue with enhanced reactivity of cell surface membranes in the whorls (Fig. 7).

The fibroblastic meningiomas displayed more prominent lectin immunolabelling within the bundles of elongated cells as compared with cells of meningothelial meningiomas. The cell cytoplasm, processes and surface membranes reacted intensely with WGA (Fig. 8) and PNA and to a lesser degree with Con A and SBA. Reactivity of interfascicular collagen

Table II. Characteristics of the lectins used for study

Lectin names	Abbreviations	Preferable sugar specificities
Peanut Agglutinin	PNA	β-D-galactose (1-3)N-acetylgalactosamine/Gal(β1-3)GalNAc/
Soybean Agglutinin	SBA	N-acetylgalactosamine (α1-3)galactose/GalNAc (α1-3)Gal/
Dolichos Biflorus Aggl.	DBA	α-N-acetylgalactosamine/α-GalNAc/
Wheat Germ Agglutinin	WGA	N-acetylglucosamine/GlcNAc (β1-4)/and neuraminic acid
Concanavalin A	ConA	α-D-mannose and β-D-glucose
Ulex Europaeus Aggl. 1	UEA-1	α-L-fucose

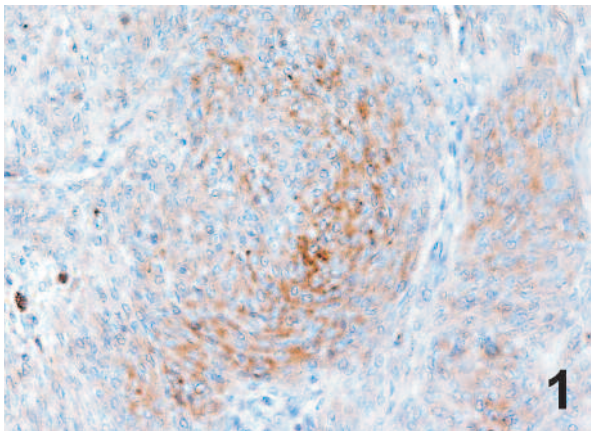

Fig. 1. Meningothelial meningioma. Not uniform, weak to intense reaction of tumour cells with PNA. x 200

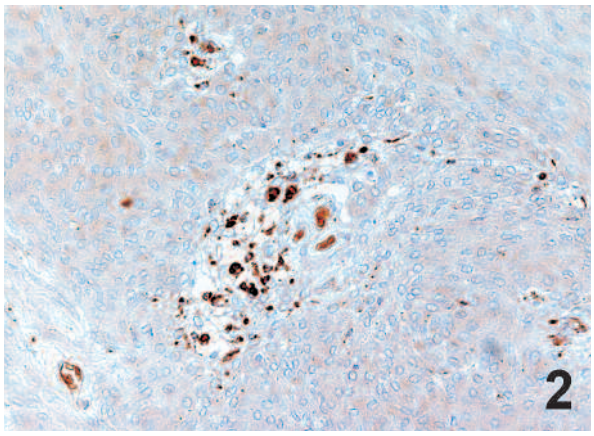

Fig. 2. Meningothelial meningioma. Slight, diffuse staining of tumour cell cytoplasm and strong labelling of blood vessels and blood derived cells by SBA. x 200

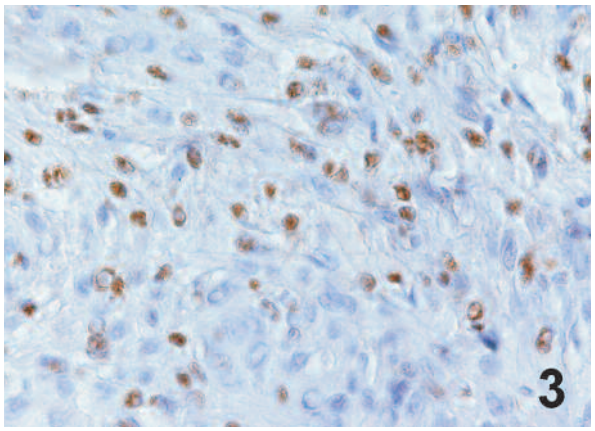

Fig. 3. Meningothelial meningioma. Cellular nuclei labelled by DBA. x 400

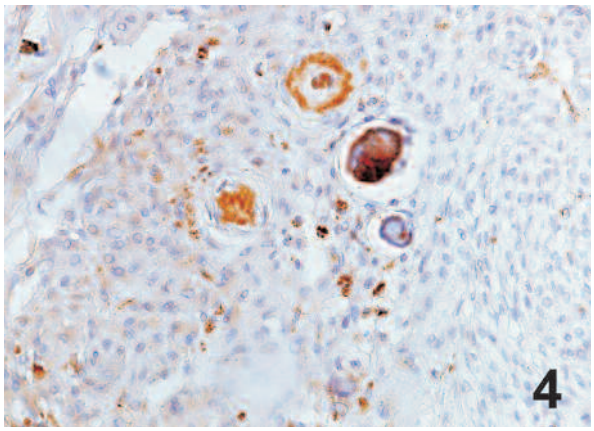

Fig. 4. Meningothelial meningioma. SBA binding pattern to psammoma bodies. x 200

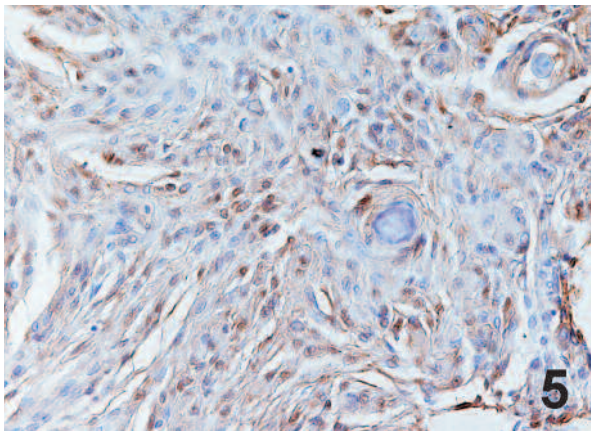

Fig. 5. Transitional meningioma. Staining of nuclei and moderate reaction of surface membranes and cytoplasm with DBA whereas psammoma bodies are unreactive. x 200

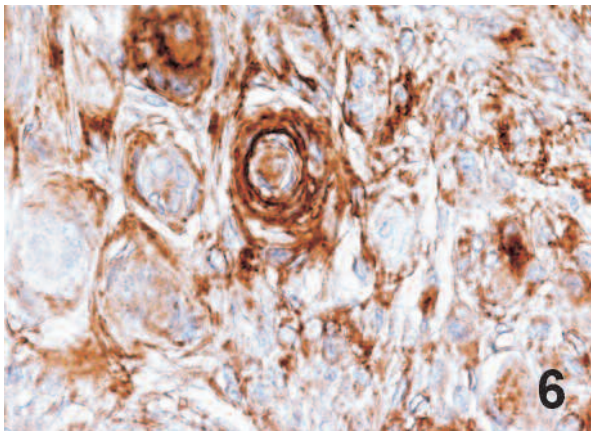

Fig. 6. Transitional meningioma. Increased binding of PNA to cytoplasm and processes of tumour cells wrapped in whorls. x 200

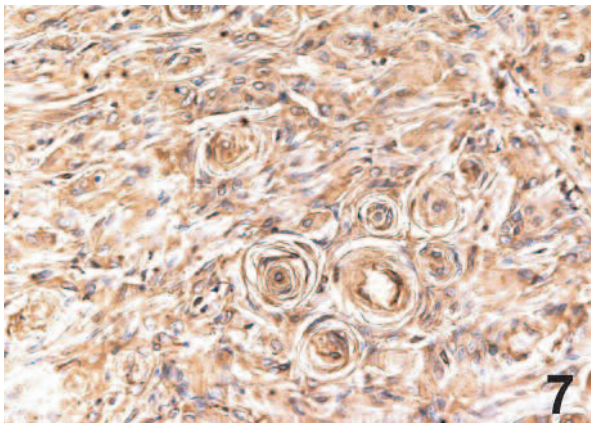

Fig. 7. Transitional meningioma. Diffuse staining with WGA of tumour cell cytoplasm and increased reactivity of surface membranes within whorled formations. x 200

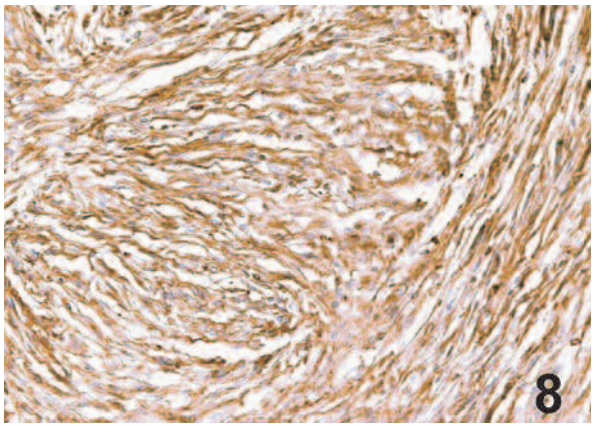

Fig. 8. Fibroblastic meningioma. Intense uniform reactivity of tumour cell cytoplasm and surface membranes for WGA. x 200

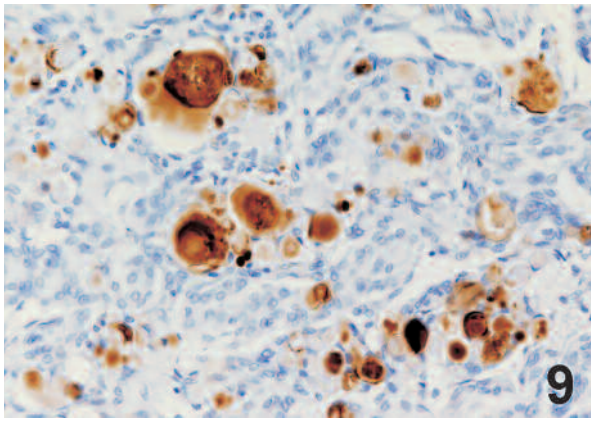

Fig. 9. Secretory meningioma. Strong binding of PNA to pseudopsammoma bodies within area of totally unreactive meningioma cells. x 200

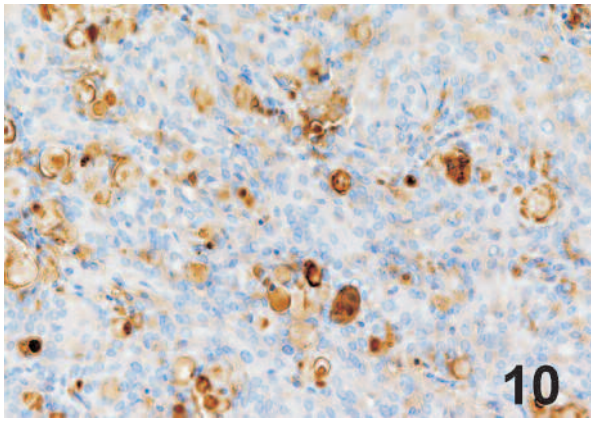

Fig. 10. Secretory meningioma. Heterogeneous staining of pseudopsammoma bodies and meningioma cells with WGA. x 200

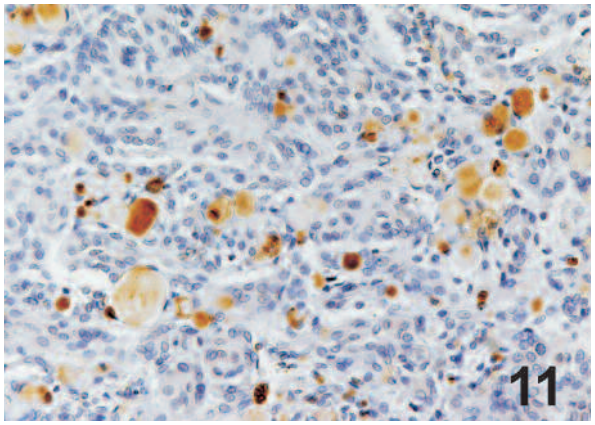

Fig. 11. Secretory meningioma. Staining of pseudopsammoma bodies with SBA ranging from positive to negative. x 200

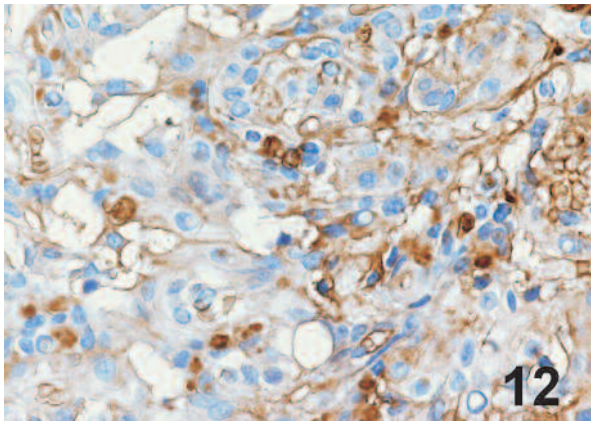

Fig. 12. Microcystic meningioma. Intense binding reaction for WGA in cell membranes and patchy staining of cytoplasm within microcystic areas. x 400

was weak with WGA and failed with Con A, whereas PNA showed diffuse moderate staining of tumour cells and intercellular collagen. DBA staining was seen in the collagen fibres and surface membranes.

The secretory meningiomas were characterised by intense reaction of pseudopsammoma bodies with PNA, WGA, SBA, DBA and Con A and slight or negative reactivity of lectins with tumour cell cytoplasm. Pseudopsammoma bodies exhibited a heterogeneous pattern of lectin binding. Both large and small pseudopsammoma bodies were either unevenly or very strong labelled or were entirely unstained by the same lectins (Figs. 9-11). Numerous bodies reacted with PNA (Fig. 9) and Con A, whereas WGA (Fig. 10) and SBA (Fig. 11) revealed a great heterogeneity of their immunolabelling. DBA stained intensely only a few pseudopsammoma bodies.

The microcystic meningiomas exhibited strong lectin labelling of intracytoplasmic vacuoles and cell surfaces or cellular processes surrounding the microcysts (Figs. 12-14). This staining pattern was most pronounced with WGA (Fig. 12) and PNA (Fig. 13). It was also distinct with Con A in the trabecular arrangements of cells (Fig. 14), while with DBA and SBA it was faint. WGA and SBA also labelled the luminal surface of blood vessels, often abundant within the microcystic areas. In contrast, PNA and Con A (Figs. 13,14) as well as DBA mostly failed to mark the lumina of blood vessels. The distinction between vacuolar or microcystic changes and small blood vessel lumina was difficult in the labelling pattern of these lectins. For this reason, additional staining with UEA-1 that specifically labelled endothelia of blood vessels was applied for evaluation of tumour vasculature in highly vascularized microcystic and angiomatous meningiomas. In the microcystic meningiomas staining with UEA-1 visualised numerous small and larger vessel lumina within the trabecula of unreactive meningioma cells (Figs. 15, 16).

In the angiomatous meningiomas, UEA-1 intensely labelled the rich vascular network among unstained meningioma cells (Fig. 17). The vascular endothelial cell linings of both thin-walled and thick hyalinized blood vessels of different size were labelled. A less distinct vascular pattern was observed with SBA staining. Other lectins, such as WGA, showed unspecific, often diffuse staining of tumour tissue, including both blood vessels and tumour cells. Con A failed to bind to blood vessel walls.

Moreover, selective UEA-1 staining of blood vessels evidenced great variability of tumour vascularization in particular subtypes of meningiomas (Fig. 18).

Discussion

Alterations of glycosylation are frequently observed in tumours; however, studies on the changes of cellular glycosylation in meningiomas are scarce. In this study, the immunohistochemical patterns of binding sites for six lectins were compared and analysed in various histological subtypes of benign meningiomas.

The lectins used for the study differ in their fine specificity and affinity for related mono- and oligosaccharides. Three of them, namely SBA, PNA and DBA, are classified as galactose/N-acetylgalactosamine (GalNAc) specific, but with differential binding sites [16,17,41]. PNA, which is β-D-galactose specific, does not bind NAc in its primary site but combines with the terminal disaccharide Galβ1→3GalNAc much more strongly than with galactose, so the combining site of this lectin is an extended one. SBA binds N-acetylgalactosamine (NAc) much more strongly than galactose, without preference for oligosaccharides containing this sugar residue, and its combining site is most likely small and open. In contrast to PNA and SBA, binding of DBA preferentially demonstrated terminal α-GalNAc. WGA binds to N-acetylglucosamine (GlcNAc) and N-acetylneuraminic acid (NeuAc) residues and thus shows affinity to glycoconjugates containing sialic acid at their terminals. Con A is preferentially specific for mannose-containing oligosaccharides and UEA-1 is an L-fucose-binding lectin.

This study evidenced some differences in lectin binding properties between histological subtypes of meningiomas. Among conventional subtypes of meningiomas, the fibroblastic type with obvious mesenchymal features exhibited more intense reactivity with lectins than transitional and meningothelial types. Similar results concerning fibroblastic meningioma were obtained in previous studies [29,37]. Increased staining for WGA of the cell cytoplasm, particularly observed in the fibroblastic type, and also in other meningiomas, might suggest that meningioma cells appeared to be rich in glucose residues and glycoconjugates containing sialic acid terminals. It has been demonstrated that different organelle-specific proteins are glycosylated with

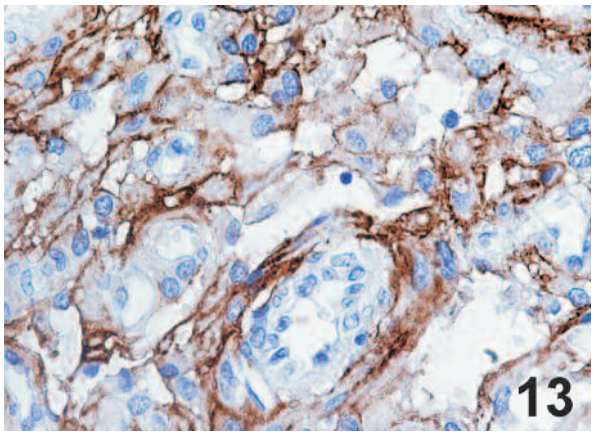

Fig. 13. Microcystic meningioma. Strong staining with PNA of surface cell membranes and cytoplasm surrounding microcystic and vacuolar changes. Loss of reactivity in luminal surface of vascular endothelia. x 400

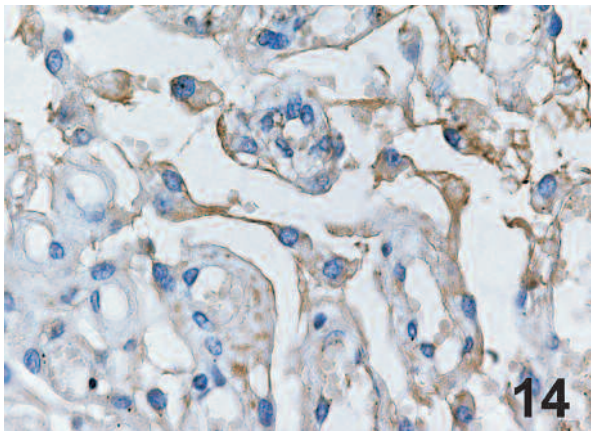

Fig. 14. Microcystic meningioma. Slight to moderate staining with Con A of cytoplasm and surface membranes of cells in trabecular arrangements. x 400

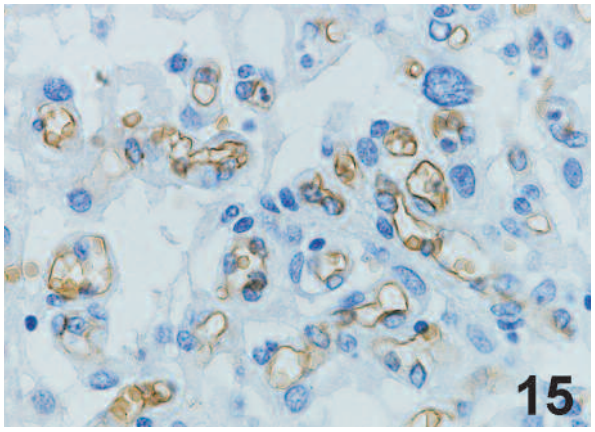

Fig. 15. Microcystic meningioma. Positive reaction for UEA-1 in endothelia of capillaries and lack of reactivity in meningioma cells. x 400

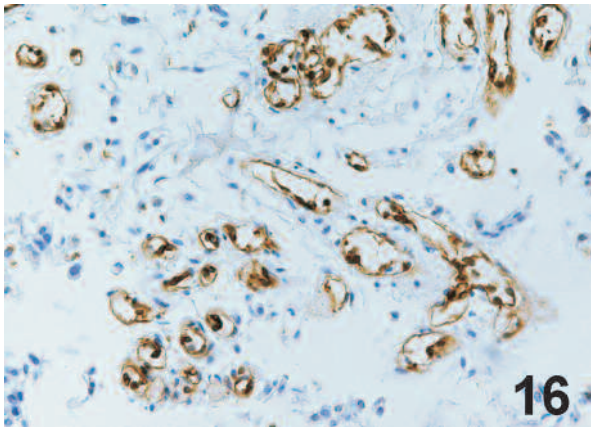

Fig. 16. Microcystic meningioma. Strong labelling by UEA-1 of blood vessels within unstained trabeculae of meningothelial tumour cells. x 200

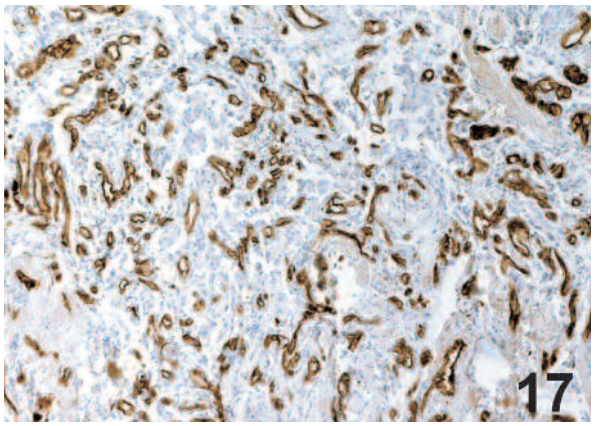

Fig. 17. Angiomatous meningioma. High density of blood vessels labelled by UEA-1. x 100

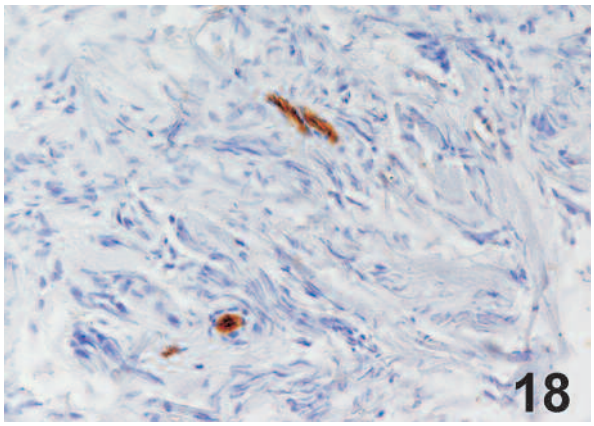

Fig. 18. Fibroblastic meningioma. Low vascular density seen with UEA-1 staining. x 200

O-linked GlcNAc residues and that significant amounts of glycoproteins bearing GlcNAc-terminated N-linked oligosaccharides are localised in rough and stripped microsomes and nuclear envelope of normal mammalian cells [19].

As regards GalNAc binding lectins, their staining pattern varied from negative to strongly positive within tumour cells of various types of meningiomas. For example, the enhanced reactivity with PNA reflects mainly the specific cytoarchitectural pattern of tumour growth, including syncytial lobules, whorled formations or trabecular arrangements of meningioma cells. The slight staining of many cells of meningothelial meningioma may be in part due to their similarities to normal arachnoid cells [49]. Salmon et al. [37] have emphasised the low level of membrane and cytoplasmic glycans in aneuploid benign meningiomas as the result of a degenerative phenomenon linked to tumour ageing. On the other hand, increased expression of PNA binding sites within tumour cell cytoplasm was reported for anaplastic meningioma [29] and in gliomas of high grade [39].

DBA labelled a minority of tumour cells, regardless of histological subtypes of meningiomas, but unlike other lectins it displayed an affinity for cell nuclei. The reaction to DBA in nuclei and/or the perinuclear area of meningioma cells has not been previously reported. It has been evidenced that DBA, which identifies terminal α-GalNAc [17], shares binding reactivity of galectin-3 to the A type histoblood group epitope. In squamous stratified epithelia the expression of galectin-3-binding sites and also DBA-reactive glycoligands correlates with increased differentiation and/or cessation of proliferation [36]. Nuclear expression of galectin-3 has also been reported in transitional and benign meningiomas [9], suggesting similar features of differentiation-dependent glycosylation in meningioma cells.

The presence of GalNAc terminal structures recognised by SBA was restricted. SBA has been found to bind with fibrous stroma in meningioma [23] and selectively labelled blood vessels in human skeletal muscle [35]. In our study, SBA stained the blood vessels walls and blood derived cells, the meningioma cells slightly, and it was the only lectin that markedly labelled psammomatous bodies.

The glucose and mannose binding lectin Con A showed relatively weak reactivity with almost all neoplastic cells in examined meningiomas, probably because most glucose- or mannose-containing oligosaccharides of cellular glycoproteins are sialylated at their terminals [33]. However, Con A showed enhanced staining of intracytoplasmic and surface membranes in cells of microcystic meningiomas and moderate to strong staining of pseudopsammoma bodies in secretory meningiomas.

The secretory meningiomas are characterised by accumulation of PAS-positive material in the form of pseudopsammoma bodies, which are usually located within intracellular cytoplasmic lumens lined by microvilli. The appearance of microvilli is thought to represent distinct properties of these tumour cells, associated with epithelial and glandular-like differentiation of meningothelial cells as it has been previously documented by immunohistochemical, ultrastructural and lectin histochemistry studies [1,6,22,24]. Positive Con A staining of pseudopsammoma bodies in secretory meningiomas was previously reported [24]. The present study showed intense reaction of pseudopsammoma bodies also with other lectins, particularly with PNA, WGA, and to a lesser extent with SBA and DBA. It should be noted that secretory meningiomas showed considerable paucity of lectin binding sites within tumour cell cytoplasm contrasting with strong and heterogeneous reactivity of the pseudopsammoma bodies.

Microcystic meningiomas were characterised by peculiar microcystic changes within spindle- and cobweb-shaped tumour cells and often a rich network of small blood vessels [34]. Abortive tumour cell differentiation to arachnoidal cells regulating cerebrospinal fluid circulation between blood vessels and subarachnoid space has been suggested on the basis of immunohistochemical and ultrastructural studies [50]. In this connection obvious enhancement of binding of differential lectins, Con A, PNA and WGA, to cell surface and vacuole-associated membranes and to cytoplasm of cells around the microcystic changes could be interpreted as abnormal tumour cell activity in production of membrane glycoproteins.

UEA-1 was a lectin reacting selectively with vascular endothelial cells and it failed to bind to any meningeal tumour cells [38,48]. As UEA-1 labels exclusively endothelial cells, mostly in capillaries [20], the use of this lectin offers simple assessment of the tumour vasculature in various histological subtypes of meningiomas. In particular, UEA-1

provides clear visualisation of the vascular network and distinction of meningioma cells from cells forming vessel lumina in microcystic and angiomatous meningiomas.

In conclusion, the results of this study indicate that changes in distribution of lectin-binding glycoconjugates in benign meningiomas are associated with variations of their architectural and histological pattern. The extent of glycosylation of tumour cell surface membranes, cytoplasm and intercellular matrix seems to be important for phenotypic diversity of neoplastic meningeal cells. The presented lectin histochemistry shows that use of lectins may contribute to a better characterisation of the mechanisms of pluripotential differentiation of meningioma cells and supports the previous reports [23,29,37] that meningiomas are heterogeneous in their glycosylation pattern.

References

1. Alguacil-Garcia A, Pettigrew NM, Sima AA. Secretory meningioma. A distinct subtype of meningioma. Am J Surg Pathol 1986; 10: 102-111.
2. Bardosi A, Dimitri T, Gabius HJ. (Neo)glycoproteins as tools in neuropathology: histochemical patterns of the extent of expression of endogenous carbohydrate-binding receptors, like lectins, in meningiomas. Virchows Arch B Cell Pathol Incl Mol Pathol 1988; 56: 35-43.
3. Beltrao EI, Medeiros PL, Rodrigues OG, Figueredo-Silva J, Valenca MM, Coelho LC, Carvalho LB Jr. Parkia pendula lectin as histochemistry marker for meningothelial tumour. Eur J Histochem 2003; 47: 139-142.
4. Bertolotto A, Giordana MT, Orsi L, Orsi R, Schiffer D. Biochemical, histochemical and immunohistochemical study of glycosaminoglycans in human meningiomas. Basic Appl Histochem 1989; 33: 239-249.
5. Bruner JM, Tien RD, Enterline DS. Tumors of the meninges and related tissues. In: Binger DD, McLendon RE, Bruner JM (eds.). Russell and Rubinstein's Pathology of Tumors of the Nervous System. Sixth Edition, Arnold, London 1998; pp. 67-112.
6. Budka H. Hyaline inclusions (Pseudopsammoma bodies) in meningiomas: immunocytochemical demonstration of epithelial-like secretion of secretory component and immunoglobulins A and M. Acta Neuropathol (Berl) 1982; 56: 294-298.
7. Carter TM, Brooks SA. Detection of aberrant glycosylation in breast cancer using lectin histochemistry. Methods Mol Med 2006; 120: 201-216.
8. Damjanov I, Sobrinho-Simoes M. Lectin histochemistry of human thyroid tumors. In: Gabius N-J, Gabius S (eds.). Lectins and cancer. Springer-Verlag, Berlin, Heidelberg 1991; pp. 41-51.
9. Das A, Tan W-L, Smith DR. Expression of extracellular matrix markers in benign meningiomas. Neuropathology 2003; 23: 275-281.
10. Davidsson P, Karlsson B, Svennerholm L. Glycoprotein pattern in human brain tumors studied using lectin binding after sodium dodecyl sulfate-gel electrophoresis and protein blotting. Brain Res 1987; 412: 254-260.
11. DeGrauw TJ, Liwnicz BH. Lectins are markers of neuronal migration and differentiation in rat brain. Dev Neurosci 1986; 8: 236-242.
12. Figarella-Branger D, Roche PH, Daniel L, Dufour H, Bianco N, Pellissier JF. Cell-adhesion molecules in human meningiomas: correlation with clinical and morphological data. Neuropathol Appl Neurobiol 1997; 23: 113-122.
13. Figols J, Madrid JF, Cervos-Navarro J. Lectins as differentiation markers of human gliomas. Histol Histopathol 1991; 6: 79-85.
14. Figols J, Kroh H, Cruz-Sanchez, Cervos-Navarro J. Lectin histochemistry of ethylnitrosourea-induced oligodendrogliomas in the rat. J Neurooncol 1997; 31: 227-232.
15. Gabius N-J, Gabius S (eds). Lectins and cancer. Springer-Verlag, Berlin, Heidelberg 1991.
16. Gabius HJ, Unverzagt C, Kayser K. Beyond plant lectin histochemistry: preparation and application of markers to visualize the cellular capacity for protein-carbohydrate recognition. Biotech Histochem 1998; 73: 263-277.
17. Goldstein IJ, Hayes CE. The lectins: carbohydrate-binding proteins of plants and animals. Adv Carbohydr Chem Biochem 1978, 35: 127-340.
18. Gotz W, Fischer G, Herken R. Lectin binding pattern in the embryonal and early fetal human vertebral column. Anat Embryol (Berl) 1991; 184: 345-353.
19. Holt GD, Hart GW. The subcellular distribution of terminal N-acetylglucosamine moieties. Localization of a novel protein--saccharide linkage, O-linked GlcNAc. J Biol Chem 1986; 261: 8049-8057.
20. Holthofer H,Virtanen I, Kariniemi AL, Hormia M, Linder E, Miettinen A. Ulex europaeus I lectin as a marker for vascular endothelium in human tissues. Lab Invest 1982; 47: 60-66.
21. Kepes JJ. Meningiomas. Biology, pathology and differential diagnosis. Masson, New York 1982.
22. Kepes JJ. The fine structure of hyaline inclusions (pseudopsammoma bodies) in meningiomas. J Neuropathol Exp Neurol 1975; 34: 282-294.
23. Kleinert R, Radner H. Lectin binding in meningiomas. Neuropathol Appl Neurobiol 1987; 13: 263-272.
24. Kuratsu J, Matsukado Y, Sonoda H. Pseudopsammoma bodies in meningotheliomatous meningioma. A histochemical and ultrastructural study. Acta Neurochir (Wien) 1983; 68: 55-62.
25. Lach B, Kanaan I. Granular cell meningioma. A case report. Folia Neuropathol 2007; 45: 19-22.
26. Lazaris AC, Chatzigianni EB, Paraskevakou H, Tseleni-Balafouta S, Davaris PS. Lectin histochemistry as a predictor of dysplasia grade in colorectal adenomas. Pathol Oncol Res 2000; 6: 265-271.
27. Lis H, Sharon N. Lectins as molecules and as tools. Annu Rev Biochem 1986; 55: 35-67.
28. Louis DN, Scheithauer BW, Budka H, von Deimling A, Kepes JJ. Meningiomas. In: Kleihues P, Cavenee WK (eds). Tumours of the nervous system. World Health Organisation Classification of Tumours. IARC Press, Lyon 2000; pp. 176-184.

29. Marafioti T, Barresi G, Batolo D. Lectin histochemistry of human meningiomas. Histol Histopathol 1994; 9: 535-540.
30. Matsumura K, Nakasu S, Nioka H, Handa J. Lectin histochemistry of normal and neoplastic peripheral nerve sheath. 2. Lectin binding patterns of schwannoma and neurofibroma. Acta Neuropathol (Berl) 1993; 86: 559-566.
31. Matyja E, Grajkowska W, Lazarczyk M, Marchel A, Czernicki T. Chordoid meningiomas of a different histopathological pattern: a report of two cases. Folia Neuropathol 2006; 44: 34-41.
32. Matyja E, Naganska E, Zabek M, Jagielski J. Meningioma with the unique coexistence of secretory and lipomatous components: a case report with immunohistochemical and ultrastructural study. Clin Neuropathol 2005; 24: 257-261.
33. Montreuil J. Spatial conformation of glycans and glycoproteins. Biol Cell 1984; 51: 115-131.
34. Nishio S, Takeshita I, Fukui M. Microcystic meningioma: tumors of arachnoid cap vs trabecular cells. Clin Neuropathol 1994; 13: 197-203.
35. Pena SD, Gordon BB, Karpati G, Carpenter S. Lectin histochemistry of human skeletal muscle. J Histochem Cytochem 1981, 29: 542-546.
36. Plzak J, Holikova Z, Smetana K Jr, Dvorankova B, Hercogova J, Kaltner H, Motlik J, Gabius HJ. Differentiation-dependent glycosylation of cells in squamous cell epithelia detected by a mammalian lectin. Cells Tissues Organs 2002; 171: 135-144.
37. Salmon I, Camby I, Remmelinck M, Rombaut K, Pasteels JL, Brotchi J, Kiss R, Danguy A. Lectin histochemistry, ploidy level and proliferation indices in meningioma subtypes. Neuropathol Appl Neurobiol 1996; 22: 68-76.
38. Schelper RL, Olson SP, Carroll TJ, Hart MN, Witters E. Studies of the endothelial origin of cells in systemic angioendotheliomatosis and other vascular lesions of the brain and meninges using ulex europaeus lectin stains. Clin Neuropathol 1986; 5: 231-237.
39. Schwechheimer K, Schnabel P, Moller P. Immunohistochemical localization of peanut lectin binding sites on human brain tumors as determined by peroxidase-antiperoxidase technique in paraffin sections. Acta Neuropathol (Berl) 1983; 61: 21-26.
40. Schwechheimer K, Weiss G, Moller P. Concanavalin A target cells in human brain tumours. J Neurol Sci 1984; 63: 393-401.
41. Sharon N. Molecular basis of lectin-carbohydrate interactions. In: Gabius N-J, Gabius S (eds.). Lectins and cancer. Springer-Verlag, Berlin, Heidelberg 1991; pp. 1-12.
42. Sharon N, Lis H. The structural basis for carbohydrate recognition by lectins. Adv Exp Med Biol 2001; 491: 1-16.
43. Szumanska G. Lectin histochemistry in the rats brain in experimental postresuscitation syndrome. (Early and late changes). Folia Neuropathol 1996; 34: 76-86.
44. Szumanska G, Gadamski R, Victorow IV, Barskow IV, Wojda R, Krzywicka J. Changes in the distribution of sugar receptors in blood vessels and cell elements of rat brain caused by photochemically induced focal ischaemia. Folia Neuropathol 2002; 40: 193-202.
45. Szumanska G, Vorbrodt AW, Mandybur TI, Wisniewski HM. Lectin histochemistry of plaques and tangles in Alzheimer's disease. Acta Neuropathol (Berl) 1987; 73: 1-11.
46. Taraszewska A, Bogucki J. A case of cystic form of angiomatous meningioma with prominent microvascular pattern mimicking haemangioblastoma. Folia Neuropathol 2001; 39: 119-123.
47. Wang XC, Kochi N, Tani E, Kaba K, Matsumoto T, Shindo H. Lectin histochemistry of human gliomas. Acta Neuropathol (Berl) 1989; 79: 176-182.
48. Weber T, Seitz RJ, Liebert UG, Gallasch E, Wechsler W. Affinity cytochemistry of vascular endothelia in brain tumors by biotinylated Ulex europaeus type I lectin (UEA I). Acta Neuropathol (Berl) 1985; 67: 128-135.
49. Yamashima T. On Arachnoid Villi and Meningiomas: Functional Implication of Ultrastructure, Cell Adhesion Mechanisms, and Extracellular Matrix Composition. Pathol Oncol Res 1996; 2: 144-149.
50. Yamazaki K, Eyden B. An ultrastructural and immunohistochemical study of microcystic meningioma with emphasis on matrix proteins and connexin 26 type gap junctions. Ultrastruct Pathol 2004; 28: 247-253.
51. Zanetta JP, Kuchler S, Lehmann S, Badache A, Maschke S, Thomas D, Dufourcq P, Vincendon G. Glycoproteins and lectins in cell adhesion and cell recognition processes. Histochem J 1992; 24: 791-804.