The complete deduced primary structure of mouse brain testican has been established from cDNA cloning. The cDNA encodes a polypeptide of 442 amino acids belonging to the proteoglycan family. The mouse brain testican core protein is 95% identical to its human testicular counterpart. In situ hybridization investigations revealed that mouse testican mRNA is mainly present in a subpopulation of pyramidal neurons localized in the CA3 area of the hippocampus. An immunocytochemical approach, with antibodies directed against an overexpressed chimeric antigen, produced in bacterial systems, showed that testican is associated with the postsynaptic region of these pyramidal neurons. Testican includes several putative functional domains related to extracellular or pericellular proteins associated with binding and/or regulatory functions. On the basis of its structural organization and its occurrence in postsynaptic areas, this proteoglycan might contribute to various neuronal mechanisms in the central nervous system.

Proteoglycans (PGs)\(^1\) have been demonstrated to be involved in mechanisms such as cell adhesion, cell migration, and cell proliferation (Ruoslhti, 1989; Ruoslhti and Yamanouchi, 1991; Wight et al., 1992).

Proteoglycans of the nervous system have been reviewed recently (Margolis and Margolis, 1993; Lander, 1993; Oohira et al., 1994). Chondroitin sulfate and keratan sulfate proteoglycans are mainly found in the extracellular space, whereas heparan sulfate proteoglycans are predominantly located on the cell surface (Margolis and Margolis, 1989). A developmentally regulated expression of PGs in various areas of the central nervous system has been described (Krueger et al., 1992; Rauch et al., 1992; Maeda et al., 1992; Carey et al., 1992; Stipp et al., 1994). PGs play a pivotal role in the growth and morphology of neurons (Cole and McAbb, 1991; Oohira et al., 1991; Faissner et al., 1994). GAGs have been shown to modulate axonal outgrowth and dendrite elongation (Damon et al., 1988; Lafont et al., 1994). The protein cores of PGs have also been proposed to participate in these events (Perris and Johansson, 1990; Iijima et al., 1991).

We have recently characterized a human testicular PG, named “testican” on the basis of its first identification in testes (Alliel et al., 1993). The analysis of a mouse multiple tissue Northern blot with human testican cDNA probes revealed that hybridizing transcripts are restricted to mouse brain. In order to define a murine model suitable for functional characterization of testican in brain, the complete primary structure of mouse brain testican was deduced from cDNA cloning experiments. The human and mouse testican protein cores were compared, and their multidomain structure is discussed and connected with potential biological functions. In situ hybridization showed that testican transcripts are mainly detected in pyramidal cells of the hippocampus. Immunohistochemical localization studies further indicated that testican is localized in the postsynaptic area of these neurons. A contribution of testican to a postsynaptic complex of these cells is suggested.

**MATERIALS AND METHODS**

**Human Testican cDNA Probes—**Two probes, spanning coding regions and portions of the 5’ and 3’ ends of human testican cDNA, were used in this study (see Fig. 2a). Probe A is a 889-bp EcoRI/MscI restriction fragment encompassing the 5’ end noncoding region and encoding the 152 N-terminal amino acids. Probe B is an EcoRI/EcoRI restriction fragment encoding the 120 C-terminal amino acids and including 310 bp of the 3’ end noncoding region.

**Antibodies—**A restriction fragment of human testican cDNA coding for the 120 C-terminal amino acids was cloned at the EcoRI site in pGEX-2T expression vector (Pharmacia Biotech, Inc.). Plasmid clones were grown in E. coli strain CAG 1139. A clone, containing the inserted fragment and producing a fusion protein of M, 38 kDa with Schistosoma japonica glutathione S-transferase attached at its N-terminal end, was selected. This fusion protein was purified by affinity chromatography on gluthathione-Sepharose 4B (Pharmacia). A polyclonal antiserum, PTC9, directed against this hybrid protein was raised in rabbits and purified by three successive adsorptions on a pGEX-2T- wild clone protein extract conjugated to CNBr-activated Sepharose 4B.

**Northern blot Analysis—**A mouse multiple tissue blot (Clontech Laboratory Inc.) was used for Northern blot analysis. The blot was hybridized with the random labeled ([\(\alpha\)-\(32\)P]dATP/dCTP) human probe B (2.10\(^{10}\) cpm/ml) in a solution containing 5 \(\times\) SSPE, 2 \(\times\) Denhardt’s solution, 0.5% SDS, 100 \(\mu\)g/ml salmon sperm DNA (3 \(\times\) SSPE = 150 mM NaCl, 10 mM NaH\(_2\)PO\(_4\), 0.1 mM EDTA, pH 7.4) for 18 h at 42°C. The blot was then washed twice with a 2 \(\times\) SSC, 0.05% SDS solution at room temperature for 15 min and subsequently four times (15 min each) with a 0.1 \(\times\) SSC, 0.1% SDS solution at 50°C (1 \(\times\) SSC = 150 mM NaCl, 150 mM sodium citrate, pH 7.4). The blot was exposed to Hyperfilm-MP (Amersham Corp.) at \(-70°C\) for 72 h.

**Isolation of Mouse cDNA Clones—**An oligo(dT)- and random-primed adult Balb/c mouse brain \(\lambda\)11 cDNA library (Clontech Laboratory Inc.) was screened with human probe B. Approximately 2 \(\times\)10\(^8\) recombinant plaques were screened according to standard techniques. The hybridization with the digoxigenin-11-dUTP randomly labeled probe (20 ng/ml) was carried out in 50% formamide, 5 \(\times\) SSC, 0.02% SDS, 10% blocking solution at 42°C. The cDNA clones were sequenced and compared to their human counterparts.
Mouse Brain Testican, Cloning and Cellular Localization

0.1% lauroyl sarcosine, 2% blocking reagent (Boehringer Mannheim) for 18 h at 42 °C. The filters were washed twice (15 min) in 2 × SSC, 0.1% SDS at room temperature and two times in 0.1 × SSC, 0.1% SDS for 15 min at 50 °C. Positive clones were detected by anti-digoxigenin rabbit Fab fragments, conjugated with alkaline phosphatase, using 5-bromo-4-chloro-3-indolyl-phosphate/4-nitroblue tetrazolium chloride as substrates (Boehringer Mannheim). The DNA library was subsequently screened, under identical conditions, with 5′ and 3′ EcoRI fragments derived from isolated mouse cDNA clones.

Subcloning, Sequencing, and Computer Analysis—Phage DNA of positive clones was prepared from liquid cultures using the Sephaglas PhagePrep kit (Pharmacia). The EcoRI fragments were subcloned into the EcoRI site of plasmid pUC18 and used either as probes for further screenings or subcloned at the EcoRI site into M13 mp18 for sequencing experiments. DNA sequencing was performed by the dideoxy chain termination method (Sanger et al., 1977) using T7 DNA-polymerase Sequenase II (Amersham) with [α-35S]dATP. Sequencing compressions were resolved using dITP. Sequences were verified from overlapping clones, by sequencing both strands of DNA using M13 universal primers or cDNA specific primers.

Nucleic acid sequence comparison between mouse and human testicans was performed following the DIAGON program (Staden, 1982), using the CITI-2 (Paris, France) sequence analysis software.

In situ Hybridization—Sagittal cryostat sections (16 μm) of 15-day-old mouse forebrain (Swiss OF1 strain) were collected at −20 °C on 0.5% gelatin, 0.05% chrome alum-coated slides and fixed for 10 min in 4% paraformaldehyde dissolved in phosphate-buffered saline. The sections were pretreated and hybridized under the experimental conditions previously reported (Roussel et al., 1991). Mouse cDNA probes were labeled to high specific activity (0.6–0.8 × 106 dpm/μg) with 50 μCi (1.85 MBq) of [α-35S]dATP by the multiprime DNA labeling system (Boehringer Mannheim). After development of the x-ray film (5 days of exposure), the slides were dipped in Ilford K5 emulsion, exposed at 4°C for 21 days, and treated as usual (Roussel et al., 1991) for observation under bright or epipolarization light illumination with a LEICA DMRB microscope.

Optic and Electron Microscopic Immunocytochemistry—The procedure was performed essentially as described previously (Roussel et al., 1987). 100-μm-thick cross-sections, taken at different brain levels were incubated for 20 min in Tris-buffered saline containing 5% calf serum. Afterwards, an appropriate volume of polyclonal antisemur (1:500 final dilution) was added. The sections were incubated for 2 h and then washed exhaustively with Tris-buffered saline. They were then treated for 1 h with horseradish peroxidase-conjugated secondary antibodies (either goat anti-rabbit IgG or sheep anti-rabbit IgG) Fab fragments (1:500) diluted in Tris-buffered saline. After a final washing step, the peroxidase activity was revealed with hydrogen peroxide and either 4-chloro-1-naphtol (optical microscopy) or diaminobenzidine (electron microscopy) as chromogens. Irrelevant antibodies were used in each experiment. For electron microscopic examination, the sections were additionally fixed with 1% osmic acid in 0.1M phosphate buffer, pH 7.4, and then dehydrated in graded ethanol and embedded in Spurr’s resin as usual (Roussel et al., 1987). Ultrathin sections were observed with a Philips EM 420 microscope.

RESULTS

Northern Blot Analysis, Molecular Cloning, and Characterization of Mouse Brain cDNA

Hybridization of a mouse multiple tissue Northern blot with human testican cDNA probe B revealed both a minor 2.3-kb and a major transcript of 5.2 kb, restricted to the lane containing mouse brain poly(A)+ RNA (Fig. 1).

Three positive clones, MB4, MB6, and MB8, were selected during a first screening of the gkt11 mouse brain cDNA library, using human testican probe B. The two largest EcoRI restriction fragments from these mouse cDNA clones, which hybridized either with human probe A or B, were used as selective probes during a second screening of the gkt11 mouse brain library. Four independent clones (CSA4, CSA5, CSA6, and CSA10) were selected. The larger 3′ EcoRI restriction fragment in the CSA5 clone did not hybridize with any human probe used in this study; it allowed us to select two additional clones (TCSS-1 and TCSS-2) during a last screening (Fig. 2a).

Nucleotide and Deduced Amino Acid Sequences of Mouse Brain Testican

Comparison with Human Testican—The nucleotide sequence of a 4343-bp composite mouse brain testican cDNA, established from nine overlapping cDNA clones and coding for the complete deduced protein core of mouse testican, is shown in Fig. 2b. The sequence around the first ATG, encountered at position 134 in the longest open reading frame, fulfills the criteria of eucaryote initiation sites (Kozak, 1991). This putative initiation codon is followed by a nucleotide sequence coding for a stretch of hydrophobic amino acids consistent with a signal peptide (von Heijne, 1986), which is identical to the N-terminal end of human testican. However, 98 nucleotides upstream from the initiation codon, mouse testican cDNA differs from its human counterpart by a seven-nucleotide deletion that has been observed in four independent clones (MB4, CSA4, CSA6, and CSA10). These observations support the assumption that the putative initiator is used both in mouse and human testican transcripts. In addition, nucleotide 10 was identified as a G or C in CSA6 and CSA10, respectively.

The initiation codon is followed by a 1326-bp open reading frame, which codes for a polypeptide of 442 amino acids. The mouse nucleotide coding sequence and its deduced protein sequence are 90 and 95% identical to those of human testican, and 60% of the amino acid substitutions are conservative (Fig. 3a). Serine residues 386 and 391, whose counterparts are the GAG attachment sites in human testican, are conserved. The sequence around the initiation codon shows that the silver grains are mostly concentrated over the N-terminal end of human testican. However, 98 nucleotides upstream from the initiation codon, mouse testican cDNA differs from its human counterpart by a seven-nucleotide deletion that has been observed in four independent clones (MB4, CSA4, CSA6, and CSA10). These observations support the assumption that the putative initiator is used both in mouse and human testican transcripts. In addition, nucleotide 10 was identified as a G or C in CSA6 and CSA10, respectively.

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A dot matrix plot comparing human and mouse testican cDNAs indicated that transcript homologies extend beyond the coding sequence both in the 5′ and 3′ regions (Fig. 3b). A putative polyadenylation site (ATTAAA), conserved in both mouse and human transcripts, was identified at position 1953. The use of this polyadenylation site may give rise to minor mRNA, with a shortened 3′ region.

In situ Hybridization—Two EcoRI restriction fragments from the MB6 clone (Fig. 2a) were used as probes. MB6-M is a 472-bp fragment covering the middle part of the translated region. MB6-T is a 932-bp fragment that codes for the 120 amino acid N-terminal end of human testican. However, 98 nucleotides upstream from the initiation codon, mouse testican cDNA differs from its human counterpart by a seven-nucleotide deletion that has been observed in four independent clones (MB4, CSA4, CSA6, and CSA10). These observations support the assumption that the putative initiator is used both in mouse and human testican transcripts. In addition, nucleotide 10 was identified as a G or C in CSA6 and CSA10, respectively.

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In situ Hybridization—Two EcoRI restriction fragments from the MB6 clone (Fig. 2a) were used as probes. MB6-M is a 472-bp fragment covering the middle part of the translated region. MB6-T is a 932-bp fragment that codes for the 120 C-terminal amino acids and encompasses also the 3′ noncoding region of the clone. Serial parasagittal to sagittal brain sections (Fig. 4), hybridized with both testican cDNA fragments, clearly show that the silver grains are mostly concentrated over the CA3 area of the hippocampus. The CA1, CA2, and CA4 areas are less labeled or not labeled. The cortex, thalamus, and...
hypothalamus display a faint labeling, whereas the white matter areas (corpus callosum, fimbria) of the cerebrum and the striatum show only a background distribution of grains (Fig. 4, a–d). In the cerebellum, only the Purkinje cell layer shows a low accumulation of silver particles (not shown). At higher magnification, examination of the labeled CA3 area shows that the silver particles are mainly distributed over the cell bodies of pyramidal neurons (Fig. 4, i and j).
Immunocytochemistry—The spatial distribution of testican in 15-day-old mouse brain structures was investigated with antiserum PTC9 directed against the 120 C-terminal amino acids of human testican. Low magnification revealed that diffuse immunolabeling is present along the hippocampus, whereas the cerebral cortex, thalamus, and hypothalamus display only a faint punctate labeling, and the cerebrum white matter is nonreactive (not shown). High magnification of the hippocampus CA3 area allowed us to notice a diffuse cytoplasmic staining in the pyramidal cells and an intense punctate labeling, mostly located in the stratum lucidum, which contains synapses of the dendrites emanating from the stained neighboring pyramidal neurons (Fig. 5, a and b). In the cerebellum, a faint punctate labeling limited to the molecular layer is observed (Fig. 5c). Immunoreactive dots are evenly distributed over the cortex (Fig. 5d). Electron microscopic immunocytochemistry revealed a clear-cut postsynaptic localization of testican in the brain (Fig. 6). The labeling is concentrated on
**Fig. 4. Distribution of testican transcripts in brain, analyzed by in situ hybridization.** MB6-M (nucleotides 627-1098) and MB6-T (nucleotides 1099-2030) cDNA probes are EcoRI restriction fragments from MB-6 clone (see Fig. 2a). a–d, macroscopic images of testican mRNA distribution on serial parasagittal to sagittal brain sections of a 15-day-old mouse after hybridization with the 35S-labeled mouse MB6-M cDNA probe. Lane 1, cortex; lane 2, corpus callosum; lane 3, striatum; lane 4, thalamus; lane 5, hypothalamus; lane 6, hippocampus. e–j, in situ localization of testican mRNA on brain sections of a 15-day-old mouse brain at the cellular level using either the 35S-labeled MB6-M (e–h) or MB6-T (i–j) cDNA probes. e and f, the images correspond to the insert shown in b. Numerous silver grains are present on the neurons of the hippocampus CA3 area; in contrast the granule cells of the dentate gyrus (DG) are clearly not labeled. g and h, the images correspond to the insert shown in d. Intense labeling in the CA3 is observed; the proximal cortex (Co) is less labeled, and the corpus callosum (CC) shows a background distribution of silver grains. i and j, view of pyramidal cells in the CA3 hippocampal at higher magnification. Autoradiograms were photographed under epipolarization light (e, g, i) or bright field illumination (f, h, j) after thionine blue staining with a LEICA DMRB microscope. Scale bars in e and f, 25 μm; scale bars in h and j, 50 μm.
the postsynaptic densities. Some reactivity, likely due to testican molecules carried from their site of synthesis to their target postsynaptic site, is also present in the surrounding cytoplasmic fluid. The presynaptic area and the synaptic cleft of these synapses do not react with the antiserum.

**DISCUSSION**

The composite sequence, established from nine mouse brain cDNA clones, encodes a 442-amino acid polypeptide highly homologous to the human testican protein core. Structural conservation around the so far identified GAG attachment sites suggests that mouse brain testican may be substituted by chondroitin sulfate and heparan sulfate, as shown for testican from the human male reproductive organs (Bonnet et al., 1992). Neither any other serine-glycine pairs, matching the consensus sequence corresponding to GAG attachment sites (Bourdon, 1990), nor NXS or NXT N-linked glycosylation consensus sequences are present within mouse or human testican deduced protein sequences. The Northern blot analysis was indicative of a brain-restricted distribution of testican transcripts in mice; however, reverse transcriptase-polymerase chain reaction also allowed their identification in testes. Human transcripts appear to be more widely distributed, but, among tested tissues, brain is one of the three tissues where testican transcripts were mostly detected. 3

The high conservation of mouse and human polypeptidic structures led us to consider that functional elements reside within the testican’s protein core. Such conserved structures are encountered in proteins involved in basic cell biological processes. The distribution of the proline residues, which account for 5.4% of the total amino acids, is not random. The PCPCLPEEPLKP peptide contains 25% of the total proline residues clustered in a region corresponding to less than 3% of the protein. The human counterpart of this peptide, despite a variation in the proline residue distribution, shows the same features. The tetrapeptide GKSL is present at positions 172–175 and in the palindromic peptide LSGKGL (amino acids 326–332). In mouse testican, the supernumerary tripeptide 63EVE is inserted in a pseudopalindromic structure. 5

FRDEVEDDYFRNW. The corresponding peptide in human testican allows us to determine a WNRFXD(1–4)DXFRNW palindromic sequence (X is a nondefined amino acid, and 1–4 denotes the number of spacing amino acids) whose biological significance, if any, is not known yet. Testican contains a cluster of glutamic acid and aspartic acid residues, which account for 40% of the 63 C-terminal residues. This acidic region includes a stretch of 9 consecutive acidic amino acids. A similar stretch is also encountered in other PGs: in the β-amyloid precursor protein (Kang et al., 1987), in claudin (Burg and Cole, 1994), in versican (Zimmermann and Ruoslahti, 1989), and in brevican (Yamada et al., 1994). Such a stretch may be involved in the binding of cationic substances. The QKLSK pentapeptide, located in the upstream vicinity of the CWCV domain, is highly reminiscent of the NKSK sequence, which has been shown to be of importance for the binding of decorin to fibronectin (Schmidt et al., 1991).

The mouse brain testican, as its human testicular counterpart (Alliel et al., 1993), is a multidomain protein that shares substantial similarities with proteins involved in adhesion, migration, and cell proliferation. Some of these domains have been identified in proteins involved in neural development, synaptogenesis, and synaptic transmission. The counteradhesive neural molecules QR1 and SC-1, either associated with neurite outgrowth and synapse formation (Guermah et al., 1991) or expressed during neural postnatal development (Ohnston et al., 1990), contain testican’s osteonectin-like domains. A 45-amino acid sequence related to Kazal-type protease inhibitor domains (Apostol et al., 1993) exists as a single copy in QR-1 and SC-1, and is tandemly repeated in follistatin (Hemmati-Brivanlou et al., 1994). Besides its implication for the gonadal function, follistatin-activin interaction has been suggested to contribute to the maintenance of nerve survival (Schubert et al., 1990), to be involved in the control of neurosecretory neuron activity (Sawchenko et al., 1988), and to participate in neural differentiation (Hashimoto et al., 1992). A wide protease inhibitory effect has been demonstrated for a recombinant rat agrin (Biroc et al., 1993), a protein that contains nine Kazal-type domains (Ferns and Hall, 1992) and plays a role in acetylcholine receptors clustering around the neuromuscular junctions. Agrin, which can be substituted by GAG chains (Tsien et al., 1995), has been proposed to be an interactive partner of neural cell adhesion molecules that may regulate a variety of

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2 F. Bonnet, J.-P. Périn, F. Charbonnier, and P. M. Alliel, unpublished data.

3 F. Bonnet, J.-P. Périn, F. Charbonnier, and P. M. Alliel, unpublished observation.
cell adhesion processes, including synaptogenesis. A 46-amino acid motif, the CWCV domain, has been identified in various proteins associated to the basement membrane and epithelial cell surface (Alliel et al., 1993). It is of interest to notice that this interspecies-conserved domain in the IGF-BP family (Shimasaki et al., 1991) is assumed to contribute to growth factor binding (Huhtala et al., 1986). Heparin-like GAGs have been proposed to act as local regulators of IGF/IGF-BP complexes (Arai et al., 1994). IGFs are known to influence neurotransmission in the hippocampus and regulation of synaptic transmission occurs through modulation of transmitter receptors in the postsynaptic membrane (Araujo et al., 1989). Thus testican is a molecule whose structure contains peptidic motifs and can be substituted by polysaccharides encountered in contributors to extracellular matrix organization and in regulators of neuralization and neurotransmission.

The spatial immunolabeling distribution of testican in mouse brain correlates well with the in situ hybridization results. Glial cells appear to be devoid of testican. Among the neuron-enriched brain regions, testican is mainly distributed on the postsynaptic densities of neurons occurring mostly in the CA3 area. The staining is concentrated at the postsynaptic side of the plasma membrane of the dendrite. This observation and structural features of testican suggest that this PG is likely to interact with membrane-bound components at the postsynaptic nerve terminals. Testican contains neither an hydrophobic domain, except for the N-terminal putative signal peptide, nor a C-terminal sequence that could be glypiated (Gerber et al., 1992). The ability of the core protein of some brain PGs to interact with neurons via neural cell adhesion molecules has been recently proposed as a regulatory mechanism for cell adhesion and cell migration in the central nervous system (Grumet et al., 1993; Friedlander et al., 1994; Maurel et al., 1994; Grumet et al., 1994). The testican transcripts and protein core are mainly distributed within the pyramidal cells in the hippocampus CA3 region where glypican is also present (Karthikeyan et al., 1994). On the basis of its structure, mimicking a set of proteins sharing diverse binding abilities, it is...
neurotransmission. activity, neuromodulation, synaptic plasticity, or even region, is an argument for its potential contribution to receptor family. The spatial distribution of testican in a restricted area be considered as a new member of the brain proteoglycan can be structurally related to testican. This molecule can thus 

Apostol, I., Giletto, A., Komiyama, T., Zhang, W. L., and Laskowski, M., Jr. (1993) J. Biol. Chem. 268, 564–574

Biroc, S. L., Payan, D. G., and Fisher, J. M. (1993) J. Biol. Chem. 268, 20388–20393

Breese, P. M., Perin J-P., Jolles, P., and Bonnet, F. (1993) J. Biol. Chem. 268, 565–569

Bourdon, M. A. (1990) in Extracellular Matrix Genes (Sandell, L. J., and Boyd, C. M., ed) pp. 157–174, Academic Press, Inc., San Diego

Breese, R. L., D’Costa, A., Boxee, R. M., and Sonntag, W. E. (1991) in Molecular Biology and Physiology of Insulin and Insulin-like Growth Factors (Raizada, M. K., and Le Roth, D., ed) pp. 449–458, Plenum Press, New York

Burg, M. A., and Cole, G. J. (1994) J. Neurobiol. 25, 1–22

Carey, D. J., Evans, D. M., Stahl, R. C., Asundi, V. K., Conner, K. J., Garbes, P., and Clomez-Smith, G. (1992) J. Cell Biol. 117, 191–201

Cole, G. J., and McCabe, C. F. (1991) Neuron 7, 1007–1018

Damon, D. H., D’Amore, P. A., and Wagner, J. A. (1988) J. Cell. Physiol. 135, 293–300

Faisnser, A., Clement, A., Lootier, A., Streit, A., Mandi, C., and Schadner, M. (1994) J. Cell Biol. 125, 783–799

Fayer, N. A., Courtois, Y., and Jenney, J. C. (1992) J. Biol. Chem. 267, 1–13

Ferns, M. J., and Hall, Z. W. (1992) Proc. Natl. Acad. Sci. U.S.A. 89, 783–787

Fayein, N.-A., Courtois, Y., and Jenney, J. C. (1992) J. Biol. Chem. 267, 12168–12173

Grumet, M., Flaccus, A., and Margolis, R. U. (1993) J. Cell Biol. 120, 815–824

Grumet, M., Milev, P., Sakurai, T., Karihikeyan, L., Bourdon, M., Margolis, R. K., and Margolis, R. U. (1994) J. Biol. Chem. 269, 12142–12146

Guermah, M., Crisanti, P., Lauzier, D., Dezelee, P., Bidou, L., Pesac, B., and Calothy, G. (1991) Proc. Natl. Acad. Sci. U.S.A. 88, 4503–4507

Hashimoto, M., Nakamura, T., Inoue, S., Kondo, T., Yamada, R., Eto, Y., Sugino, H., and Maramatsu, M. (1992) J. Biol. Chem. 267, 7203–7206

Hemmati-Brivanlou, A., Kelly, O. G., and Melton, D. A. (1994) Cell 77, 283–295

Huhtala, M.-L., Koistinen, R., Palomäki, P., Partanen, P., Bohn, H., and Seppälä, M. (1986) Biochim. Biophys. Res. Commun. 141, 263–270

Iijima, N., Oohira, A., Mori, T., Kitabatake, K., and Kohsaka, S. (1991) J. Neurochem. 56, 706–708

Johnston, I. G., Paradiso, T., Gurd, J. W., and Brown, I. R. (1990) Neuron 4, 165–176

Kang, J. J., Lemaire, H.-G., Unterbeck, A., Salbaum, J. M., Masters, C. L., Grzeschik, K.-H., Muller, K., Beyerleuter, K., and Muller, H. (1987) Nature 325, 733–736

Karihikeyan, L., Flad, M., Engel, M., Meyerputtlitz, B., Margolis, R. U., and Margolis, R. K. (1994) J. Cell Biol. 125, 499–509

Kozak, M. (1991) J. Biol. Chem. 266, 19867–19870

Krueger, R. C., Jr., Hennig, A. K., and Schwartz, N. B. (1992) J. Biol. Chem. 267, 288, 199–207

Lafont, F., Prodhant, A., Valenza, C., Petitou, M., Pascal, M., Rouget, M., and Rousselet, A. (1994) Dev. Biol. 156, 453–468

Lander, A. D. (1993) Curr. Opin. Neurobiol. 3, 716–723

Maeda, N., Matsui, F., and Oohira, A. (1992) Dev. Biol. 151, 564–574

Margolis, R. U., and Margolis, R. K. (1989) in Neurobiology of Glycoconjugates (Margolis, R. U., and Margolis, R. K., ed) pp. 85–126, Plenum Publishing Corp., New York

Margolis, R. K., and Margolis, R. U. (1993) Experimental 49, 429–446

Marks, L. J., King, M. G., and Baskin, D. G. (1991) in Molecular Biology and Physiology of Insulin and Insulin-like Growth Factors (Raizada, M. K., and Le Roth, D., ed) pp. 409–470, Plenum Press, New York

Maurel, P., Rauch, U., Flad, M., Margolis, R. K., and Margolis, R. U. (1994) Proc. Natl. Acad. Sci. U.S.A. 91, 2512–2516

Oohira, A., Matsui, F., and Katoh-Semba, R. (1991) J. Neurosci. 11, 822–827

Oohira, A., Katoh-Semba, R., Watanabe, E., and Matsui, F. (1994) Neurosci. Res. 20, 195–207

Periss, R., and Johansson, S. (1990) Dev. Biol. 137, 1–12

Rauch, U., Karihikeyan, L., Maurel, P., Margolis, R. U., and Margolis, R. K. (1992) J. Biol. Chem. 267, 19536–19547

Roussel, G., Neskovic, N. M., Trifilieff, E., Artault, J. C., and Nussbaum, J. L. (1992) Dev. Biol. 152, 783–799

Roussel, G., Felix, J. M., Dautigny, A., Pham-Dinh, D., Hindelang, C., Jolles, P., and Nussbaum, J. L. (1991) Dev. Neurosci. 13, 98–103

Ruoslahti, E. (1988) J. Biol. Chem. 263, 13369–13372

Ruoslahti, E., and Yamaguchi, Y. (1991) Cell 64, 867–869

Sanger, F., Nicklen, R., and Coulson, A. R. (1977) Proc. Natl. Acad. Sci. U.S.A. 74, 5463–5467

Sawchenko, P. E., Pitsky, P. M., Pfeffer, S. W., Cunningham, E. T., Jr., Vaughan, J., Rivier, J., and Vale, W. (1988) Nature 334, 615–617

Schmidt, G., Hauser, H., and Kresse, H. (1991) Biochem. J. 280, 411–414

Schubert, D., Kimmel, C., LaRocque-Rood, G., Vaughan, J., Karr, D., and Fischer, W. H. (1990) Nature 344, 868–870

Shimasaki, S., Gao, L., Shimonaka, M., and Ling, N. (1991) Mol. Endocrinol. 5, 938–947

Staden, R. A. (1982) Nucleic Acids Res. 10, 2951–2961

Stipp, C. S., Litwack, E. D., and Lander, A. D. (1994) J. Biol. Chem. 269, 13447–13450

Tsen, G., Halfter, W., Kröger, S., and Cöle, G. J. (1995) J. Biol. Chem. 270, 3392–3399

Wight, T. N., Kinsella, M. G., and Qwarnstro¨m, E. E. (1992) Curr. Opin. Cell Biol. 4, 793–801

von Heijne, G. (1986) Nucleic Acids Res. 14, 4683–4690

Yamada, H., Watanabe, K., Shimonaka, M., and Yamaguchi, Y. (1994) J. Biol. Chem. 269, 10119–10126

Zimmermann, D. R., and Ruoslahti, E. (1989) EMBO J. 8, 2975–2981
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