Translated from *Uspekhi Khimii* **60** 1777–1816 (1991) U.D.C. 577.15+591.818+547.963+612.118

Lectins in the investigation of receptors

V M Lakhtin, I A Yamskov
A N Bakh Institute of Biochemistry, Academy of Sciences of the USSR
Institute of Foodstuffs, Academy of Sciences of the USSR, Moscow

ABSTRACT. Problems of the purification and characterisation are considered for approximately 270 receptors (including cell surface and organelle enzymes), which are glycoconjugates (mainly glycoproteins) from animals, plants and microorganisms, using various lectins (mainly lectin sorbents). An analysis has been carried out of the stages of lectin affinity chromatography of receptors (choice of detergent, use of organic solvents, elution with carbohydrates, etc.). Examples are given of procedures for the purification of receptors, including the use of paired columns and combination chromatography on lectins. The possibility of separating sub-populations of receptors using lectins has been demonstrated. Examples are given of the use of lectins in the analysis of the oligosaccharide structure of receptors. Cases are recorded of the interaction of receptors with endogenous lectins and of receptor lectins with endogenous glycoconjugates. It has been shown that lectins, in combination with glycosidases and antibodies, may be useful in the investigation of receptors.

The bibliography contains 406 references.

Contents

I. Introduction 903
II. Interaction of immobilised lectins with receptors 903
III. Investigation of receptor glycans using lectin sorbents 915
IV. Interconnection between receptors, endogenous lectins and enzymes 915
V. Conclusion 916

I. Introduction

In recent years the study of receptors has been at the centre of investigators' attention due to their participation in biorecognition at various levels of the organisation of living systems.[1–9] It has been established that key (important in the metabolic sense) interactions between receptors and external agents (endogenous and exogenous, soluble and bound to cell surfaces or cell organelles) proceed in many cases by specific bonding between lectins (carbohydrate-binding proteins) and glycoconjugates.[10–22] The problems of purifying and characterising receptors with a selection of lectin sorbents or labelled lectins are therefore of interest to biologists and chemists. A large number of publications has appeared since the publication of the review by Lotan and Nicolson[23] on the purification of membrane glycoproteins by lectin affinity chromatography. These have extended our ideas on the possible use of lectins in the study of receptors and enzymes bound to cellular structures (see reviews[21,22,24–27] for example).

The aim of the present review is to examine the purification and characterisation of receptors of various types using lectins, chiefly lectin sorbents.

II. Interaction of immobilised lectins with receptors

Results are summarised in Tables 1 and 2 of the use of lectin affinity chromatography for the purification of receptor glycoproteins, including enzymes bound to cellular structures (see also reviews[22–24,26]). It is evident from Tables 1 and 2 that receptors possess an affinity for a wide range of lectins. Wheat-germ lectin (WL) is the most frequently used which is explained by the widespread distribution on its receptor glycoproteins of polyantenna sialylated glycans, branched residues of *N*-acetyl-D-glucosamine, *O*-glycoside-bound *N*-acetyl-D-glucosamine residues, hybrid oligosaccharides and glycans of the polylactosamine type (see below for investigations of receptor structure with lectins).

1. Results of the use of lectin affinity chromatography for the purification of receptors

It is evident from the data shown in Tables 1 and 2 that the use of wheat lectin enables 50–100-fold purification of Ca-channels, opioid receptors and glycoproteins of olfactory neurones.[47,61,74,90,117,193] Concanavalin A (Con A) is highly effective in the purification of phospholipase A_1 (165 times, based on enzyme activation[269,270]) and pea (*Pisum sativum*) lectin in the purification of fibroblast glycoproteins (58 times, by affinity HPLC [187]). Some lectins, which are relatively rarely used in lectin affinity chromatography, may also prove to be extremely useful, e.g. castor-oil plant (*Ricinus communis*) lectin (CL) in the purification of the glycoproteins induced by Epstein–Barr virus and of the cholecystokinin receptor [144 and 440 times respectively,[98,223] gorse (*Ulex europaeus*) lectin-I (GL-I) in the purification of the cholecystokinin receptor (300 times)[99]] and the lectin from seeds of *Vicia bungei* which is specific for α-mannose in the purification of opioid receptors (200 times[60]). Lectin affinity chromatography is the most efficient step in receptor purification in these and in other cases (see Tables 1 and 2).

Stabilisation of receptor preparations is achieved by lectin affinity chromatography. This is important for their preservation (for example, in the case of dopamine receptors[76]). This is evidently linked with the removal of contaminating hydrolases and oxidases.[92] Occasionally investigators have found activation of receptors in eluates from the lectin sorbent, as in the case of receptors of insulin,[91] phospholipase A_1 [269] or Ca^{2+}-dependent peptidase.[295] Activation of the glycoconjugates indicated above may occur both by the removal (separation in eluates) of inhibitors (glycoconjugates and proteins) and by the stabilisation of receptors in comparison with an unstable control (before purification). An appreciable

increase in the yield of receptors, up to 150% of the initial activity, is achieved in specially selected cases.[269]

Simple immobilisation of glycoconjugates (low yield at a high percentage of binding) with their simultaneous purification from contaminants is possible during affinity chromatography on lectins. This occurs in the case of α_1-adrenergic and lactogenic receptors, epitectin (Ca-antigen), receptors of thyrotropin and interleukin 4 and sulphotransferase.[65,108,112,163,214,256] As in other cases of oligomeric receptors, subunits with a predominant affinity for the given lectin (glycosylated subunits) are bound primarily with the lectin sorbent and provide subsequent directed assembly of the receptor (the addition, for example, of non-glycosylated subunits) as in the case of receptors of α-latrotoxin, fibronectin, insulin-like growth hormone, cGMP-channel, Na^+,K^+-ATPase and antigens of trypanosomes[43,123,134,201,235,302,303] and also receptor complexes.[51,126,202]

Table 1. Purification of receptors (R) on immobilised lectins (IL).

Receptors, sources, schemes of purification (sorbents)	Result of using IL (yield, %; purity, times in parentheses, notes)	Refs.
Receptors of cations, low molecular compounds, receptor complexes from animal tissues and cells		
Animal gut Na^+/glucose transporter, 75 kD	Con A (b)	[28]
Hexose transporter from hamster ovocytes deficient at Lec-1, 40 kD form 55 kD form	WL (10–20% b), Con A (80–90% b) WL (25–40 b) Con A (60–75 b)	[29]
GABA transporter from bovine brain, 80 kD	WL (b)	[30]
Serotonin transporter from chromaffin granules of bovine adrenal, 80 kD DEAE → HA → PC → WL	WL (b)	[31]
L-Glu transporter from rat brain	WL (50%; 12 times)	[32]
GABA transporter/L-Glu transporter from rat brain WL → HA → DEAE	WL (20%; 6 times)*	[33]
R for L–Glu from *Nephrops* muscle, sensitive to quisqualate, 74+65 kD Con A→Ultragel	Con A (57%; 178 times)*	[33a]
GP from Na^+-channel from locust brain neurones/saxitoxin R, 240–260 kD	WL (34%; 2.7 times) Con A (33%)	[34]
Na^+-channel from the eel *Electrophorus electricus*, 285 kD	Slug L better than WL (out of 11 lectins)	[35]
Na^+-channel from presynaptic rat brain membranes	WL (80% b)	[36, 37]
GP of K^+-channel from rat brain/dendrotoxin-binding R, DEAE → AC → WL	WL (44%; 6 times)	[38]
Na^+-Ca^{2+}-exchange GP from dog heart sarcolemma, 160 kD, DEAE → WL	WL (50%; 1.7 times)	[39]
Na^+-Ca^{2+}-exchange GP from the outer segment of bovine eye rods, 215 kD, DEAE → WL	WL (20%; 1.7 times) CL-2 (b)	[40, 41]
Ca^{2+}-binding GP from bovine heart sarcolemma	WL (> 60%)**	[42]
GP of the cGMP entry channel from bovine eye rod photoreceptors, 63+240 kD	Con A (63 kD b)** out of 5 L	[43]
Na^+/K^+-antiporter from rat renal brush border membranes	Binding with 5 biotinyl-L***	[44]
Complex of 1,4-dihydropyridine R and Ca^{2+}-channel from chick heart DEAE → Hexylamine → WL	WL (70%; 3 times)	[45]
Dihydropyridine R/Ca^{2+}-channel blockers from rabbit skeletal muscle, 142+56+30 kD, WL(1) → DEAE(HPLC) → WL(2)	WL(1) (70%, 24 times)* WL(2) (66%; 1.7 times)	[46]
Chick heart Ca^{2+}-channel sensitive to dihydropyridine, 140 kD, DEAE → Con A → WL	Con A → WL (42%; 132 times), including WL (88%; 97 times)*	[47]
Dihydropyridine R from rabbit skeletal muscle 53+160 kD, WL→DEAE→Dihydropyridine	WL (85%; 14 times)*	[48, 49]
Complex of dihydropyridine R and Ca^{2+}-channel from rabbit skeletal muscle, 340 kD, WL → DEAE	WL (38%; 39 times)*	[50]
Complex of dihydropyridine R and rabbit skeletal muscle dystrophin, 360 kD (36+43+52+61+94 kD) WL → DEAE	WL (100% b), affinity for α_2-SU of dihydropyridine R in the case of Con A and WL	[51, 51a]
Ca^{2+}-channel blocker R from rabbit skeletal muscle, 56+150 kD	WL (b)	[52]
Complex of dihydropyridine R and *L* type Ca^{2+}-channel from bovine adrenal, 185–190 kD	WL (b)	[53]
α_2-like component of Ca^{2+}-channel from chick heart muscle, 139 kD	WL (removal of component 197 kD)	[54]
Inositol triphosphate R from boovine aorta smooth muscle microsomes, 224 kD Heparin→WL	WL (46%, 2.4 times)	[55]
Inositol triphosphate R from rat cerebellum, 260 kD, DEAE → Heparin → Con A → GF; Heparin → Con A → Mono Q (HPLC)	Con A (60%; 5 times)	[56–58]
Opioid R from chick, rat, bovine and human brain	WL (40–60%; 30 times) best of 7 lectins	[59]
Opioid R from rat brain, 45 kD	*Vicia bungei Ohwi* L (200 times) better than WL	[60]
Complex of opioid R and rat brain guanine-binding protein sensitive to whooping cough toxin, 41+39 kD, WL → Phe	WL (20–50%; 100–40 times)	[61]
Opioid R from neuroblastoma–glioma hybrid NG cells 108–15, 58 kD	WL (b)	[62]
Opioid R from human placenta	WL (of 5 lectins)	[63, 64]
α_1-adrenergic R from mouse BC3HI and hamster DDT1 smooth muscle cells	WL (affinity for the hamster cell R was 6 times weaker than for the mouse cell R)	[65]
α_1-adrenergic R from rat brain and liver, Heparin → AC → WL	WL (45%; 8 times, brain) (48%; 2.6 times, liver) binding–100%	[66]
α_1-adrenergic R in a complex with guanidine-binding protein	WL (68%; 5 times)*, removal of unglycosylated guanidine-binding proteins	[66a]
α_1-adrenergic R from DDT1 and MF2 muscle cells, 80 kD, Antagonist → WL → GF (HPLC)	WL (70–90%; 10 times, binding > 90%)	[67]

Table 1 (contd.).

Receptors, sources, schemes of purification (sorbents)	Result of using IL (yield, %; purity, times in parentheses, notes)	Refs.
α_2-adrenergic R from human thrombocytes, 64 kD, AC → Heparin → WL → AC → Heparin	WL (74%; 5 times)	[68]
β-adrenergic R from mouse S49 lymphoma cells	WL, Con A (effect of swainsonine) binding > 75%, yield 50%	[69]
β_1-adrenergic R from turkey erythrocytes	6 L (differences from mammalian R)**	[70]
β_2-adrenergic R from hamster erythrocytes and lungs, 62 kD	WL, Con A (tissue specificity)**	[71]
Dopamine D1 R from bovine brain and parathyroid, 64 kD	WL, Con A (but not SL, AL)**	[72]
Dopamine D2 R from bovine brain caudate nuclei	WL (but not Con A)	[73]
Dopamine R from bovine brain caudate nuclei, 95 kD, AC → WL	WL (52%; 49 times)	[74]
Dopamine D2 R from bovine caudate nuclei and olfactory tubercle	WL (b 68%; yield 100%, separation of forms of R**	[75]
Dopamine D2 R from bovine striatum, WL → AC; AC → GF → WL	WL (80%; 10 times), stabilisation of R	[76, 77]
Cocaine R (dopamine transporter) from bovine striatum synaptosomes	Con A (b)	[78]
Dopamine transporter from rat brain caudate nuclei and striatum, SU 58 kD	WL (b)** Con A (no b)	[79]
Dopamine transporter from dog brain striatum	WL (95%; 10 times)	[80]
Histamine H1 R from mouse smooth muscle differentiated BC3H1 cells, 68 kD	Con A (30%; 25 times)**	[81]
Octopamine R/neurohormone R/adenyl cyclase activator R from luminescing organs of the glow-worm *Photinus Pyralis*, SU 75 + 79 kD	Affinity for Con A**(blotting)***	[82]
Muscarinic acetylcholine R from porcine atrium cordis	WL (55%; 20 times)	[83]
Acetylcholine R from bovine brain cortex, 73 kD	WL (b 90%; yield up to 65%)** Con A**	[84]
Acetylcholine R from rat muscle fibres	WL (b)	[85]
Acetylcholine R from rat brain cortex	WL better than Con A**	[86, 87]
Acetylcholine R from the electric organ of the eel, α + β + γ + δ (SU)	Labelled*** L-FGA (b δ- and γ-SU) WL (b δ-SU) Con A (b all SU)	[88]
Ryanodine R from rabbit brain and muscle	WL (no b) Con A (no b) LL (no b)	[51a, 88a]
Receptors of peptide and protein hormones and growth factors from animal tissues and cells		
Insulin R from human placenta WL → LL → CL-1	WL (98%; 22 times) PL (45%; 14 times) LL (30%; 15 times) Con A (20%; 5 times) CL-1 (15%; 7 times)	[89]
Insulin R from human placenta WL → Insulin; Con A → Insulin	WL (54%; 7.5 times) WL (40%; 5 times)	[90, 90a]

Table 1 (contd.).

Receptors, sources, schemes of purification (sorbents)	Result of using IL (yield, %; purity, times in parentheses, notes)	Refs.
Insulin R from human IM9 lymphocytes WL → CL-1 → PL	WL (70%; 21 times) CL-1 (50%; 50 times) CL-2 (30%; 50 times) PL (25%; 26 times) LL (20%; 15 times) Con A (10%; 5 times)	[89]
Insulin R from rat skeletal muscle	WL (80%; 70 times)	[90a]
WL → Insulin	WL (94 – 120%; 7 times)	[91]
Insulin R from rat brain and liver, SU 135 + 95 kD DEAE → Con A → Insulin → WL	Con A (93%; 36 times) WL (94%; 2.3 times; removal of insulinase)	[92]
Formyl-peptide R from rabbit peritoneal neutrophils, 50 – 70 kD (fMet-Leu-Phe) → WL	AC → WL (74%; 203 times)	[93]
Vasopressin V1 R from rat liver, 58 – 68 kD, GF → WL → HA → DEAE	WL (84%; 6 times)	[94]
Peptide transporter from rabbit small intestinal brush border membranes, 127 kD, WL → DEAE	WL (b)	[95]
Vasoactive intestinal peptide R (neurotransmitting hormone R) from rat liver, 80 + 56 kD	WL (b 50%;)**, but not Con A	[96]
Vasoactive intestinal hormonal peptide R from rat lung, 58 kD	WL (b)	[97]
Cholecystokinin R from rat pancreas, 80 – 90 kD, CL-2 → Affigel	CL-2 (about 100%; 440 times)	[98]
Cholecystokinin R from rat pancreas, 95 kD S-Sepharose → GL-1 → GF	GL-1 (47%; 300 times) better than WL	[99]
Cholecystokinin R from rat pancreas, 85 – 95 kD	WL (b)**	[100]
R from liver bile duct cells, 110 kD	WL (b)	[101]
R of luteinising hormone and choriogonadotropin from rat ovary, 73 kD or 90 – 93 kD, WL → Choriogonadotropin	WL (70%; 18 times)**	[102 – 104]
R of luteinising hormone and choriogonadotropin from porcine ovary Sepharose 6B → Con A	Con A (30 – 40%; 4 – 10 times)	[105]
Lutropin R from porcine ovary, 22 + 40 + 66 kD Hormone → Con A	Con A (50 – 75% b)	[106]
Folliotropin R from bovine testes, 240 kD (SU 60 kD) DEAE → WL	WL (92%; 3.7 times) two types of GP	[107]
Thyrotropin R from bovine thyroid, 316 + 115 + 54 kD Thyrotropin → GrL	Thyrotropin → GrL (3%; 808 times) GrL (84% b) WL (70% b) CL-1 (69% b) Con A (60% b)	[108]
Thyrotropin R from thyroids of patients with Graves' disease, 70 + 50 + 35 kD WL → Thyrotropin	WL (26%; 4.7 times; b more than 90%)	[109]
Somatostatin R from rat pancreas, 400 kD (100 + 56 + 21 kD, SU)	WL (75%; 7 times)	[110]
Somatostatin R from rat brain	WL (50% b; yield 67% of bound form)	[111]
Lactogenic R from mouse liver, SU 62 + 100 kD (in complex with human growth hormone 20 kD)	Con A (95% b; yield 30%) CL-1 (81% b; yield 69%) LL (52% b; yield 38%) WL (27% b)	[112]

Table 1 (contd.).

Receptors, sources, schemes of purification (sorbents)	Result of using IL (yield, %; purity, times in parentheses, notes)	Refs.
Lactogenic R from rat ovary Con A → Choriogonadotropin	Con A (yield 60%)	[113, 114]
Corticotropin releasing factor R from rat anterior pituitary and brain, SU 75 and 58 kD respectively	WL 20% (b)** Con A (b)	[115]
Growth β-transforming factor R from chick and human tissue, mouse 3T3 cells	WL (b)	[116]
Platelet growth factor R from porcine uterus WL → Mono Q → (Ab to phosphotyrosine)	WL (99%; 90 times)*	[117]
Fibroblast growth factor R from bovine brain, 125–165 kD DEAE → Mono Q → WL → (Growth factor)	WL (100% b), to remove heparin	[118]
Insulin-like growth factor I R from phaeochromocytoma PC12 cells and bovine adrenals	WL (Purification 36–72 times)	[119]
Insulin-like growth factor I R from human placenta	WL (b)	[120]
R for platelet-derived growth factor from cat brain, 145 kD	WL (b)**	[121]
Growth hormone R from cultured human lymphocytes	WL better than Con A, LL, CL-1, CL-2	[122]
Insulin-like growth factor II R from human lipid cells, 260 kD (SU 87+60 kD)	WL (b)	[123]
Heparin-binding growth factor R from human hepatoma Hep G2 cells, 130 kD	WL (b)**	[124]
Macrophage and granulocyte colony-stimulating factor R from human placenta, neutrophils and leukemic cells lines, 84 kD	WL (b)	[124a]
α-Interferon R from human lymphoblastoid *Namalva* cells	WL (47%; 13 times)*	[125]
Human β-interferon R, 110 kD WL → Ab	WL (90% b; yield 68%)	[126]
γ-Interferon R from human foreskin fibroblasts Interferon → CL-1	CL-1 (100%; 2.6 times)	[127]
γ-Interferon R from human placenta, 90 kD WL → Interferon	WL (b)**	[128]
Other receptors and animal antigens		
Complement CR1 R from human peripheral nerve myelin, 190 kD	LL (b)	[129]
Immunoglobulin Fc-fragment R from lymphocytes of patients with chronic leukemia LL → GF	LL (b)	[130]
α_2-Macroglobulin R from human placenta, 420 kD GF → Macroglobulin → Mono Q → WL	WL (b)	[131]
Mouse lactotransferrin R, 130 kD	Con A, L-FGA (b)**	[132]
Fibronectin R from rat hepatocytes and fibroblasts, 155+115 kD WL → (Fibronectin hexapeptide)	WL (b)	[133]
β-SU R of mouse sarcoma fibronectin, 135 kD LL → WL → AC → HA	WL (no b, purified by filtration)	[134]

Table 1 (contd.).

Receptors, sources, schemes of purification (sorbents)	Result of using IL (yield, %; purity, times in parentheses, notes)	Refs.
R of fibronectin/integrin from rat liver parenchymal cells, SU 110 kD WL → Fibronectin	WL (b)**	[135]
Antigen associated with integrins from plasma membranes of human placenta cells, SU 50 kD WL → γ-Interferon → B6H12 (Ab)	WL (b)	[136]
β_1-SU of integrin (R of fibronectin from larvae of amphibian *Pleurodeles waltl* GF → Con A	Con A (b)	[137]
Transferrin R from mouse NS-1 plasmacytoma cells, SU 90–95 kD	4 L (b GP)**	[138]
Transferrin R from chicken oviduct, 180 kD (SU 91 kD)	4 L (b GP)	[139]
Vitronectin R from human M21 melanoma cells	Con A, LL (b)	[140]
Vitronectin R/integrin $\alpha_v\beta_3$/fibronectin R from human placenta LM 609 (Ab) → WL	WL (b)	[141]
Integrin $\alpha_1\beta_5$ (adhesion R of vitronectin) from human placenta LM 6–9→MN142→WL	WL (b)	[142]
Fibrinogen R (GP-IIb/IIIa-like R) from human endothelial cell lines, 150+(110–125)+95 kD AC → WL → (Ab to GP-IIIa)	WL (9b 100%)	[143]
Laminin R (200/120 kD-integrin) of B50 neuroblastoma cells (integrin $\alpha\beta_1$) WL → Laminin	WL (b)	[144]
Adhesin R of endothelial cells of bovine embryo aorta and human umbilical vein (not a member of the cadherin family), 130 kD WL→Ab	WL (b)	[145]
Adhesin R of neural cells from C2 muscle cells	AL (b)	[146]
Adhesin R of substrate from mouse OTT6050 teratocarcinoma cells, 125 kD	CL-1 (b)	[147]
GP-Ib of human platelets/Willebrand factor R, 165 kD (SU 143+22)	WL (removal of actin and GP-IX)	[148]
GP-Ib of human platelets/adhesion R to vessel subendothelium WL → DEAE	WL (b)	[149]
GP-IIb/IIIa of human platelets Con A → Heparin → GF → WL	Con A (b GP-IIb/IIIa+thrombospondin+fibrinogen), WL (removal of fibrinogen)	[150]
GP-IV of human platelets LL → WL	LL (removal of GP-IIb/IIIa by binding), WL (b GP-IV, Ib)	[151]
GP-IV of human platelets (CD 36-like), 88 kD WL → WL → GF; DEAE → WL → GF	WL (b)	[152]
GP-V of human platelets Superose 12 → Mono Q → WL	WL (66%; 7 times)*	[153]
GP-IX of human platelets, 17 kD WL → (Ab to GP-Ib) → GF (HPLC)	WL (b)	[154]
Thromboxane A-2 R/prostaglandin H-2 R from human platelets GF → WL → Red agarose → GF (HPLC)	GF → WL (22%; 1620 times)*	[155]

Table 1 (contd.).

Receptors, sources, schemes of purification (sorbents)	Result of using IL (yield, %; purity, times in parentheses, notes)	Refs.
Glycocalicin of human platelets WL → Mono Q	WL, Con A, CL-1**, E-FGA	[156, 157]
Bean lectin R from porcine spleen lymphocytes	FGA (b)	[158]
Peanut lectin R from mouse immature thymocytes, 170–180 kD	AL (b)	[159]
GP Thy-1 (antigen) of mouse thymocytes Con A → (Ab to Con A) → GF	Con A (83%; 19 times)	[160]
Antigen Thy-1 of mouse thymocytes, less than 27 kD + 28 kD	Thorn apple lectin (<27 kD not b 50%; retarded 25%; 28 kD b and desorbed 25%)**	[161]
Mammalian interleukin 3 R, 140 kD	WL (b)	[162]
Interleukin 4 R from gibbon MLA 144 line T-lymphocytes, 130 kD CM → WL → Interleukin 4	WL (35%; 4.5 times) CM (filtration) → WL (10–15 times)	[163]
CDw44 antigen from T-lymphocytes of patients with chronic leukemia, 85 kD F10-44-2 (MAb) → DEAE → WL → CM → GF	WL (b)	[164]
Leukosialin (sialoformin, CD 43) from human leucocytes (MOLT-4 cells), 95 kD	WL and CL-1 (b 100%), Con A and LL (partial b); desorption with detergent (except for WL)**	[165]
Leukosialin from human line HL-60 promyelocytes WL → Ab	WL (b)	[166]
Leukosialin from human K562 erythroleukemic leukocytes, 105 kD	WL better than other lectins	[167]
Lymphocytic antigen from membrane, 28 kD	LL (b)**	[168]
GP (H-antigen) containing fucose from human erythrocytes DEAE → (aleuria lectin)	Aleuria lectin (b)**	[169, 170]
GP containing A-antigen from human blood group A (+) erythrocytes, 500 kD (SU 42–55 kD)	Snail lectin (b)	[171]
GP containing Cad-antigen from human blood Cad-group erythrocytes	Dolichos L better than snail L, WL, E-FGA, Maclura L	[172]
Glycophorin M from human blood St^a-group erythrocytes	LL (removal of glycophorin N)	[173]
Glycoprotein precursors of blood group N antigen with Thomson–Friedenreich (T) antigen activity from patients with malignant carcinoma of the ovary (*Vicia unijuga* L) → AL	*Vicia unijuga* L (b), AL (b)	[174]
Glycophorins A, B, N from human blood Dantu-group erythrocytes	LL (no glycophorin N b)	[175]
Glycophorins A, B, C, E from human erythrocytes WL (HPLC) → Cu^{2+}-chelate	WL (separation of A + B + C and E – no b	[176]
Glycophorin A from human MM- and NN-erythrocytes	WL (b 100% for MM and 70% for NN), CL-1 (b of asialoglycophorin)	[177–179]
Gonococcus R from human erythrocytes (band-3), 186 kD (SU 93 kD)	Con A (b)	[180]

Table 1 (contd.).

Receptors, sources, schemes of purification (sorbents)	Result of using IL (yield, %; purity, times in parentheses, notes)	Refs.
GP of adipocyte ghosts from porcine great omentum, 47 + 79 kD PL → DEAE → GF	PL (b)	[181]
Lipoprotein R from insect fat body, 120 kD DEAE → HA → Con A → AC	Con A (b)*	[182]
R of cytolytic (antitumour) activity of mouse peritoneal macrophages, 153 + 140 + 90 kD	WL (b)	[183]
R for the activation of cytotoxic activity from mouse macrophages, 460 kD (110 + 170)	*Eristalis* L (b)	[184]
R for *Mycoplasma pneumoniae* microbe from human lung MRC-5 fibroblasts	WL (b)	[185]
GP of type II alveolar cells of rabbit and rat lung, 230 and 200 kD respectively	Maclura L (b)**	[186]
GP from human diploid fibroblasts PL (HPLC), Con A (HPLC)	PL (purification 58 times) Con A (purification 6 times)	[187]
GP induced by aldosterone from toad bladder epithelial cells, 65 + 70 kD	WL (purification 20 times)** Con A (b)	[188]
Epidermal factor R from A-431 cells, 170 kD	Con A and LL (b OS)**	[189]
Unique kidney antigen in human renal disease, 98 + 105 kD	Thorn appleperoxidase L (b) out of 5 L***	[190]
GP-II from chromaffin granules of bovine adrenals, SU 80–100 kD	WL (b)	[191]
GP-1 associated with ovocyte lysosomal membranes from the chinese hamster, SU 135 + 115 kD	Bovine heart Ln (b), Tomato Ln (b)	[192]
Transmembrane GP from frog olfactory neurones, 95 kD	WL (50 times)	[193]
Antigen 2B8 from rat olfactory neurones, 215 kD	WL, CL-1 (b asialoGP)	[194]
GP of oligodendroglial cultured cells, 99 + 77 kD IEC → WL	WL better than Con A, LL (no b with CL-1 LL SL, dolichos L)	[195]
GP from bovine brain oligodendroglia, 99 + 77 kD IEC → WL	WL (yield 70%)	[196]
GP from human brain myelin and oligodendrocytes, 120 kD AL → AL	AL (b)	[197]
Antigen of mouse cerebellum Purkinje cells, 400 kD GF → Con A → GF	Con A (b)	[198]
GC binding hyaluronic acid from 8-week mouse brain (GC is more than 50% proteoglycans) PL → GF → DEAE	WL (89–100%; 22 times)	[199]
R for α-latrotoxin (neurotoxin) from bovine brain cortex, 280–320 kD (200 + 160 + 79 + 43) DEAE → WL → Ab	WL (70%; 4 times), b α-SU of toxin (200 + 160)	[200, 201]
Complex of scorpion α-toxin with rat brain Na-channel	WL (b β-SU of Na-channel)	[202]
GP of cows milk lipid droplets GP-1, 2, 3, 4, 5, 6, 7	Con A, WL GP-1, 2, 6 b GP-3, 4 retarded GP-7 not b	[203]

Table 1 (contd.).

Receptors, sources, schemes of purification (sorbents)	Result of using IL (yield, %; purity, times in parentheses, notes)	Refs.
R for endogenous factor–cobalamin from porcine and human ileum, 75+65+43 kD	WL, LL and Con A (b 45–79%); WL (b 75 kD); LL and Con A (b 75+65+43 kD)	[204]
Histocompatibility antigens H-2^b from mouse RBL-5 tumour cells GF → WL → Ab	WL (80%; 8 times)	[205]
Other class II histocompatibility antigens	5 lectins***	[206–208]
GP of mouse small intestine and teratocarcinoma cells Dolichos L → dolichos L	Dolichos L	[209]
GP binding collagen from mouse MDAY-D2 lymphoreticular tumour cells (collagen type II R), 110+130 kD WL → L-FGA → GF	WL (b)	[210, 211]
GP from human embryonic PA-1 carcinoma cells, 80–120 kD	Thorn apple L (b)	[212]
R for type I fimbriae (lectin) of *E. coli* from guinea pig erythrocytes, 65 kD	Fimbriae type I, Con A (b)**	[213]
SialoGP of Morris hepatoma 7777 cells	WL (b)	[178]
Epitectin (Ca-antigen) of human pharyngeal carcinoma cells, 1–1.5 MD (390+350 kD) Ab → WL	WL (irreversible binding)	[214]
Preneoplastic antigen in liver carcinogenesis from malignant nodes of rat liver, 145 kD (2 SU) GF → Con A	Con A (73%; 6 times)	[215]
GP of human malignant melanoma SK-Mel-37 cells, 110 kD Con A → WL	Con A → WL (b)	[216]
GP of rat hepatoma cells, 130 kD Con A → GF → (Ab to alkaline phosphatase)	Con A (b)	[217]
GP of rat liver and Morris hepatoma 7777 cells, 60+80+100 kD Con A (HPLC), Con A	Con A (HPLC) (b)	[218]
Antigen from Walker 256 rat carcinoma cells, 57 kD CM → WL → Phe; Con A	WL (removal of 98% contaminating protein)	[219]
GP of human embryonic PA-1 carcinoma cells, 80–120 kD	Thorn apple L (b)	[166]
GP of HM-1 pluripotent embryonal carcinoma cells, more than 90 kD	*Phytolacca* L (b 48%) Thorn apple L and lotus L (17% b), PTL (no b)	[220]
Dystrophin complex from rabbit dystrophic skeletal muscle, 156+50+43+35 kD	WL (removal of myosin and ATPase)	[51, 221]
Tumour antigen associated with *Herpes simplex* virus from human liver carcinoma, 70 kD GF → DEAE → GF → Con A	Con A (b)	[222]
GP of BNK-21 cells infected with *Herpes simplex* virus	Con A (b)	[203]
Major GP induced by Epstein–Barr virus, 320 kD DEAE → CL-2	CL-2 (100%; 144 times)	[223]

Table 1 (contd.).

Receptors, sources, schemes of purification (sorbents)	Result of using IL (yield, %; purity, times in parentheses, notes)	Refs.
Histocompatibility antigens HLA-DR and MB from human lymphoblastoid cells transformed by Epstein–Barr virus	CL-2 (b)	[224]
R for measles virus from *Vero* cells	LL (b) PL (b)	[225]
R for the minor group of rhinoviruses from human *HeLa* cells, 450 kD LL → GF → IEC	LL (yield 100%) CL-2 (yield 100%) Con A (yield 20%)	[226]
Transcript GP of nuclear pore membranes of monkey COS-1 cells, 62 kD	succWL (b)**	[227]
Glycoconjugates of inducers of human and animal diseases		
Recombinant GP-160 of HIV-1 from Vero cells infected with two vaccinia viruses LL → (Ab to GP-41) → mono Q → LL	LL (b)	[228]
Prion GP from brains of hamsters with virus-like infection, scrapie type of illness, 26+28 kD	LL, CL-1 (b asialoGP)** out of 6 lectins	[229]
Antigen of the pneumonia initiator, *Pneumocystis carinii* bacteria, from infected ferret lungs, 66–110 kD Superose-12 → Con A	Con A better than WL (SL and asparagus lectin no b)	[230]
Bacterial antigens (vaccines) of the *Bordetella* family	L (b)	[231]
GP from the coating of spores of *Bacillus sphaericus* 1593 (toxin for gnatworm) *Culex pipiens*	Con A (b)	[232]
GP of plasma membranes of *Spiroplasma citri*, 84 kD	Con A (b)	[233]
Antigens of promastigotes of 14 strains of *Leishmania*, 14–86 kD	Potential lectin sorbents (14 L)***	[234]
Surface antigens of protozoa *Trypanosoma brucei brucei* variant AnTat 1.1, 60+94 kD Con A → HPLC (RP)	Con A (separation of 94 and 60 kD)	[235]
Antigens of epimastigotes of protozoa *Trypanosoma cruzi* (72 kD) and specimens of the metacyclic phase (90+85+55 kD) CB → DEAE → WL → Con A → GF (72 kD) CB → DEAE → WL → HA → Con A (55 kD)	WL (different affinity for each antigen)	[236]
GP-85 of infectious tripomastigote form of *Trypanosoma cruzi*, including sialic acid and residues of fucose and α-1,3-galactose	WL → WL**	[237]
Protective Ag of ringed tick (vaccine), 86 kD WL → Con A → EP → WL → GF (HPLC)	WL and Con A (b)	[238]
Filarial surface antigens of adult nematode–parasites *Brugia malayi*, 29+(17–200) kD	Con A, WL, SL, lotus L	[239]
Tegumental glycoconjugate antigens of adult specimens of *Schistosoma mansoni*, 23–300 kD, 24 kD	Con A and LL** (out of 7 lectins)	[240–242]
GP of *Schistosoma mansoni* from infected mice, 170 kD	AL (b)	[243]
Virulence antigens of the 3rd stage of the nematode–parasite *Trichinella spiralis*	LL (b)	[244]

Table 1 (contd.).

Receptors, sources, schemes of purification (sorbents)	Result of using IL (yield, % purity, times in parentheses, notes)	Refs.
Glycoconjugates of microscopic fungi		
Adhesin R of cells of the slime fungus *Dictyostelium discoideum* at a late stage of development, 95+(40–50) kD	WL (b)	[245]
GP-34 specific for the 'stalk' stage of the slime fungus *Dictyostelium discoideum* NC4 (absent from spores)	WL (b)	[246]
Antigens of the mycelial fungi *Aspergillus fumigatus, Mucor pusillus, Petriellidium boydii, Trichophyton rubrum*	Con A (b)	[247]

*Lectin affinity chromatography was the most effective purification stage;

**Investigation of receptors in combination with their treatment with endo- and exoglycosidases;

***Affinity for labelled lectin.

Abbreviations used

Ab—antibody;
MAb—monoclonal antibody
AL—arachis lectin
Asn—asparagine
ATP—adenosine triphosphate
AC—affinity chromatography
b—binding
BSA—bovine serum albumin
CB—Cibacron Blue
cGMP—cyclic guanosine monophosphate
CL—castor oil plant lectin
CM—carboxymethyl
Con A—concanavalin A
DEAE—diethylaminoethyl
EP—electrophoresis
GF—gel filtration
GL—gorse lectin
GP—glycoprotein(s)
Gp—glycopeptides
GrL—griffonia lectin
HA—hydroxyapatite
HIV-1—human immunodeficiency virus type 1
HPLC (RF)—high performance liquid chromatography (reversed phase)
IEC—ion-exchange chromatography
L—lectin(s)
L-FGA, E-FGA—bean lectin (leuco and erythro forms)
LL—lentil lectin
OS—oligosaccharides
PL—pea lectin
PS—phosphocellulose
PTL—potato tuber lectin
SDS—sodium dodecyl sulphate
SL—soyabean lectin
SU—subunits
succWL—succinylated wheat-germ lectin
Receptor nomenclature from *Trends in Pharmacological Sciences*, 1991, Supplement, 35 pp

Purification of receptors or separation of their subpopulations is also possible by retardation of the glycoprotein during elution from the lectin sorbent without the use of desorbing carbohydrates (i.e. due to weak affinity interactions with the lectin).[161,203,284] Separation by retardation on elution has also been described for mixtures of glycopeptides when using immobilised bean or castor-oil plant (*Ricinus communis*) lectin which confirms the effect of glycans on elution behaviour.

The simplest procedures for purifying receptors comprise filtration through one lectin sorbent (absence of binding) and binding with another lectin sorbent (concentration of receptors) for example by the scheme: wheat-germ lectin (WL^-)→Con A^+ or Con A^-→WL^+, as in the case of trypanosome antigens or rat kidney ATPase.[236,301] Other purification schemes are also used, for example lentil (*Lens culinaris*) lectin (LL^+)→WL^+ (fibronectin receptor[134]), LL^+→Con A^+ (prothrombin precursor[317]), WL^+→LL^+→castor-oil plant lectin-1 ($CL\text{-}1^+$) (insulin receptor[89]), WL^+→bean leucoagglutinin ($L\text{-}FGA^+$).[210,211] The combination of Con A^+→WL^+ is used in the case of melanoma antigens, dipeptidase IV and Mg^{2+}-ATPase,[216,290,291,305] and WL^+→Con A^+ in the case of the protective antigens of arthropods.[238] The examples of combination chromatography on lectins indicated above enable the separation of multiple forms (subpopulations) of receptors and an increase in the overall yield of any receptor heterogeneous in the carbohydrate portion. An example of appreciably increasing yield is the purification of dipeptidyl-dipeptidase IV by affinity HPLC on paired columns of immobilised Con A and WL.[291] In several cases rechromatography of antigens and receptors on immobilised LL,[228] WL,[46,69] dolichos (*Dolichos biflorus*) lectin[209] or peanut (*Arachis*) lectin[197] is used.

As is evident from Tables 1 and 2 the maximum degree of purification of receptors when using a particular type of lectin is achieved in the majority of cases by varying the first stages of purification immediately after solubilisation of the receptors with detergent. However it must be remembered that chromatography of crude preparations may lead to irreversible contamination of the lectin sorbent and a reduction in its affinity capacity.[125,150,291] In addition preferential binding of some receptors at the expense of others to WL at the first stage of purification may be expected, for glycoproteins from thrombocytes for example.

Lectin affinity chromatography of receptors is frequently used in combination with other affinity sorbents. Paired columns of immobilised thyrotropin and griffonia (*Bandeiraea simplificifolia*) lectin-I (GrL-I) enable thyrotropin receptors to be purified 808-fold.[108] Immobilised heparin and WL are convenient, following

(Con A)-sorbent, for the removal of thrombospondin and fibrinogen from a preparation of thrombocyte glycoproteins IIb–IIIa.[150] Immobilised WL is convenient, following immobilised fibroblast growth factor (elution of heparin receptors), for the removal of heparin from the receptor preparation.[118] After chromatography of thymocyte Thy-1 antigen on (Con A)-sorbent immobilised antibody to Con A was used to remove lectin molecules partially washed out from the sorbent with desoxycholate.[160] When purifying carcinoma cell galactosyl transferase the filtrate from the (CL-1)-sorbent was applied to immobilised *N*-acetyl-D-glucosamine.[254]

Table 2. Purification of enzymes (receptors) bound to cell surfaces and organelles with immobilised lectins.

Enzymes, sources, purification schemes	Result of using IL (yield, %, purification, times, notes)	Refs.
Oxido-reductases		
L-Trp oxidase of field cabbage (peroxidase-like)	Con A (83%, 4 times)	[248]
Ribonucleotide reductase of rat liver, SU 89 kD	Con A (b)	[249]
Peroxidase from human placenta	Con A (90%, 20 times); removal of eosinophilic peroxidase, myeloperoxidase, catalase and cytochrome P-450	[250]
Dopamine-β-hydroxylase associated with the β-adrenergic R from bovine adrenal medulla Con A→DEAE→GF (HPLC)	Con A (66%; 120 times)*	[251]
Dopamine β-hydroxylase from rat adrenal, 300 kD (SU 75 kD) Con A → GF (HPLC) → IEC (HPLC)	Con A (removal of chromogranins)	[252]
Tyrosinase from hamster melanoma DEAE → GF → Con A	Con A (80%; 7 times)*	[253]
Transferases		
N-Acetylglucosaminide-β-1,4-galactosyl transferase of mouse F9 carcinoma cells IEC → XL–1 → Acetylglucosamine → α-Lactalbumin	CL-1 (no b)	[254]
Galactosyl transferase of the Golgi apparatus of the budding yeast *Schizosaccharomyces pombe*, 61 kD DEAE (HPLC) → UDP-hexanolamine → Con A → Mono Q	Con A (73%; times) GL-1 (b)	[255]
Tyr-protein sulphotransferase of the Golgi apparatus of bovine adrenal cells, 50–54 kD	WL (b 37%)**	[256]
N-Heparan-sulphate sulphotransferase of rat liver, 97 kD DEAE → Heparin → ADP → WL	WL (44%; 3 times)	[257]
Oligosaccharyl transferase, stabilised with phosphatidylcholine, of calf thyroid microsomes	Con A (b)	[258]
Tyr kinase of cat brain (R of colony stimulating factor type 1 or obtained from growth factor of thrombocytes), 145 kD	WL (b)**	[121, 259]
Tyr kinase of insulin R of rat brain and liver	WL (b)	[260, 261]
Protein kinase/interleukin-1 R from rat liver mesangial cells in culture (SU 48 kD)	WL (b)	[262]
Phospholamban kinase of mammalian heart sarcoplasmic reticulum cells, SU 56+53 kD	affinity for Con A	[263]

Table 2 (contd.).

Enzymes, sources, purification schemes	Result of using IL (yield, %, purification, times, notes)	Refs.
Arginine-specific ADP-ribosyl transferase of animal skeletal muscle microsomes, SU 39 kD DEAE→Con A→GF (HPLC)	Con A (88%; 50 times)* WL (no b)	[264]
Poly A polymerase of the nucelar membrane of rat liver cells, 64 kD	Con A (5 times)**	[265]
Esterases		
p-Nitrophenyl acetate esterase type A from rat liver microsomes, 61 kD Con A → GF → DEAE → HA	Con A (80%; 3 times)	[266]
Carboxy esterase E1 from rat liver microsomes, 175 kD (SU 59 kD)	Con A (b)**	[267]
Peptidase associated with acetylcholinesterase of the electric eel and ewe brain basal ganglia AC → GF → Con A	Con A (b)	[259]
Alkaline phosphatase of fly integument, 180 kD Con A → GF → GF	Con A (b)	[268]
Phospholipase A-1 of rat liver lysosomes Con A → Chromatofoc. → GF; (Orange + Red agarose) → Con A → Phe	Con A (156%; 165 times)*	[269, 270]
Acid phosphatase of promastigotes of Mexican and Brazilian Leishmanii (inhibited with citrate) GF → DEAE → Con A	Con A (72%; 14 times)*	[271]
3′-Nucleotidase/nuclease of promastigotes of *Leishmania donovani*, 43 kD	Contains Asn-OC, available for L**	[272]
5′-Nucleotidase of rat heart, 74 kD CM → Con A → DEAE → ADP	Con A (123%; 13 times)	[273]
5′-Nucleotidase of rat kidney, 69 kD Con A → ADP	Con A (94%; 17 times)	[274]
5′-Nucleotidase of bovine thyroid, 150 kD (2 SU) LL → AMP	LL (81%; 10 times)*	[275]
Cell wall ribonuclease of the fungus *Aspergillus oryzae*, similar to ribonuclease T-2-L in carbohydrate composition	Possible affinity for Con A	[276, 277]
Sphyngomyelinase of human placental lysosomes, 123 kD (SU 62 kD) Con A → Sphyngosylphosphorylcholine → Hexyl → IEC (HPLC)	Con A (100%; 21 times)	[278]
Nucleotide pyrophosphatase of human placenta CB → AMP → WL → ADP → GF → DEAE	WL (96%; 2 times)	[279]
Nucleoside diphosphatase type B from rat brain, 75 kD (thiamine pyrophosphatase associated with 1D4) DEAE → Con A → GF → CB → Cu^{2+} chelate → GF	Con A (48%; 6 times)*	[280]
Glycosidases		
α-Glucosidase of human placental lysosomes, 110 kD Con A → GF	Con A (99%; 12 times)	[281]
Glucosidase II-processing GP from cow mammary gland, 290 kD Con A → HA → DEAE	Con A (79%; 8 times)	[282]
Glucosidase II-processing GP from bean shoots and soya cell culture, 220 kD (2 SU) DEAE → HA → Con A → GF	Con A (73%; 10 times)	[283]

Table 2 (contd.).

Enzymes, sources, purification schemes	Result of using IL (yield, %, purification, times, notes)	Refs.
α-1,2-Mannosidase-processing GP from rabbit liver microsomes, 52 kD DEAE → Con A → GF → CB → GF	Con A (77%; 27 times)*	[284]
β-Fructofuranosidase from radish cell walls (similar to cytoplasmic)	4 L	[285]
Trehalase of the American cockroach, 110 kD Con A → GF	Con A (48%; 32 times)	[286]
Trehalase of the brush border of rabbit renal cortex Phe; Con A	Con A (b 20%)	[287]
Saccharase–isomaltase complex from a biopsy sample of human small intestine, 210+212+245 (SU 145+130 kD)	LL (all forms b), snail L (b 212 and 245 kD)	[288]
N-Acetylglucosaminidase from spermatozoids of the surface membrane of *Phallusia mammilata*, 158 kD (2 SU) DEAE → Phenyl → Con A → EP	Con A (69%; 2.5 times)	[289]
Proteases		
Dipeptidyl peptidase IV of rat liver Con A → WL → Arg; Con A (HPLC) → WL (HPLC)	Con A → WL (74%; 46 times), Con A (84%; 32 times)	[290, 291]
Carboxypeptidase N from human placenta, 280 kD HA → LL → Histargin	LL (27%; 2.5 times)	[292]
Carboxypeptidase M from human placenta, 62 kD	Con A (b)	[293]
Aminopeptidase N from rabbit intestine	WL (b)	[95]
Aminopeptidase N from porcine intestinal mucosa, 130 kD SU (Stilbenedisulphonate) → Con A	Con A (37% yield, removal of SU 90 kD)**; affinity for CL-1, WL, tomato L	[294]
Peptidase dependent on Ca^{2+} participating in the action of the pairing pheromone of yeast *Rhodosporidium toruloides* (type 'a' cells) DEAE → Con A	Con A (107%; 3.3 times)	[295]
Proteinase of the surface of promastigotes of various *Leishmanii* species, 63 kD SU	Con A and LL (b)	[296]
Trypsin-like proteinase/esterase specific for Arg from rat liver insulin R	Con A (b)	[297]
Cysteine proteinase/neutral gelatinase from human malignant LOX melanoma cells SU 170 kD Sephacryl → WL	WL (purification; 10 times; removal of metalloproteinase with SU 50 kD	[298]
Other hydrolases		
Lipoamidase of pig brain, 140 kD CM → DEAE → GF → WL	WL (purification 10 times)	[299]
Ampicillin acylase of bacteria *Pseudomonas melanogenum*, 146 kD (2 SU)	Contains 13% hexose with probable affinity for L	[300]
NAD-glycohydrolase of bovine thyroid (probable thyrotropin R) GF → LL	LL (b)	[301]
Na^+, K^+-ATPase of rat kidney cortex microsomes, 95+54 kD Con A → WL	Con A (no b), WL (b β-SU, 54 kD)	[302, 303]

Table 2 (contd.).

Enzymes, sources, purification schemes	Result of using IL (yield, %, purification, times, notes)	Refs.
β-SU, Na^+, K^+-ATPase of rat brain (adhesion molecule on brain glial cells) and porcine intestine, 60–80 kD	WL and CL-1**, better than Con A	[303–305]
Mg^+-ATPase of rabbit skeletal muscle, 107 kD Con A → WL → IEC (HPLC)	Con A, WL, LL (b)	[51, 306]
ATPase of dog kidney and human K562 cells (P-glycoprotein), 170 kD	WL, Con A and CL-1 (b), AL (no B)	[307–309]
ATPase 'P' type (forming phospho intermediate) from cholinergic synapses of cells of the electric organ of *Torpedo californica*, 110+104+98+60 kD HA → WL → GF (HPLC)	WL (71%; 2 times)	[309a]
GTPase of rat olfactory neurones, 55 kD	Con A-FITC (b)***	[310]
Lyases		
Carbonic anhydrase of ovine salivary gland, 540 kD (SU 45 kD) Sulphonamide → WL → GF	WL and Con A (b)**	[311]
Catalytic component of bovine brain adenylate cyclase sensitive to hormone, 120 kD	WL (30%; 2 times)	[312]
Catalytic component of the adenylate cyclase system of bovine brain cortex sensitive to calmodulin, 155 kD Calmodulin → WL	WL (52%; 12 times)	[313]
Atrial natriuretic factor R with and without cyclase activity (guanylate cyclase) from bovine adrenal glomerula cells GTP → WL	WL (55%; 1.3 times)	[314]
Particle-bound highly active guanylate cyclase of rat lung Con A → GTP	Con A (25%; 3–6 times)	[315]
Phosphorylated form of guanylate cyclase of sea urchin sperm, 160 kD GTP → DEAE → Con A	Con A (5 times)	[316]
Carboxylase from rat liver microsomes dependent on vitamin K	Con A (b 80%) LL (b 70%)	[317]

*Lectin affinity chromatography was the most efficient step in the enzyme purification

**investigation of the enzyme in conjunction with its treatment with endo- and exoglycosidases

***affinity for non-immobilised lectin

Abbreviations used (see also Table 1)

ADP—adenosine diphosphate
AMP—adenosine monophosphate
ATP—adenosine triphosphate
FITC—fluorescein isothiocyanate
GrL-I—griffonia lectin-I
GTP—guanosine triphosphate corr
RNA—ribonucleic acid
UDP—uridine diphosphate

Enzymes are named in accordance with the International Classification (Enzyme Nomenclature, Recommendations of the Nomenclature Committee of the International Union of Biochemistry, Orlando, Acad. Press, 1984, 646 pp).

Tandem chromatography has also been used in other combinations of a lectin sorbent with non-lectin sorbents. Paired columns with immobilised Con A and phenylalanine are effective in the purification of phospholipase A_1,[270] with Affigel S-145 and with immobilised WL in the case of the thromboxane A_2 receptor.[155] A technologically simple procedure is the filtration of partially purified receptors through carboxymethyl sorbents before and after immobilised WL for receptors of interleukin 4 and glycoprotein CDw 44.[163,164] The possibilities of tandem chromatography using a lectin sorbent are well illustrated for the thromboxane receptor which was purified 1620-fold on paired columns directly from the crude solubilised protein extract.[155] In addition paired combinations of lectin and non-lectin sorbents give an increase in the yield of receptors.[155]

The use of immobilised Con A, PL, WL and CL-1 in variants of affinity HPLC is promising for the purification of membrane-bound glycoproteins.[176,187,218,291]

2. Solubilisation of receptors and features of the lectin affinity chromatography of receptors

Many factors are important when carrying out lectin affinity chromatography. These are the solubilisation method to increase receptor solubility, buffer composition (detergent type, organic solvents, buffer types, pH, ionic strength, presence of metal cations or EDTA, the need to reduce disulphide bonds in proteins, protease inhibitors, phospholipids, etc.), method of application, washing and elution, times and temperatures. For each receptor purification it is necessary to select the optimum procedure for lectin affinity chromatography depending on the lectin sorbent chosen (lectin type, matrix, means of lectin immobilisation, capacity of sorbent for conjugated lectin or binding of model glycoconjugates, conditions for regeneration and storage for repeated use). The actual procedure for chromatography on a lectin sorbent under the conditions selected is a simple operation. The complexity of working with a lectin sorbent is seemingly determined by the presence in these proteins not only of carbohydrate-binding regions but also of regions of hydrophobic binding, binding of cations of divalent metals and by the presence of other domains.

Perhaps the most complex factor is the selection of the means of solubilising receptors (type of detergent or solubilisation by treatment with hydrolases).

Most frequently 0.5–1.0% Triton X-100 (TX-100) is used for the solubilisation and lectin affinity chromatography of receptors. TX-114 has also been used, for example in the case of glycoproteins of thrombocytes and kidney.[149,152,190] Carbohydrates are also used occasionally for solubilisation instead of TX.[202] Since high concentrations of TX-100 cause desorption (washing off) of lectin from the sorbent,[218] lectin affinity chromatography of receptors is carried out at concentrations of detergent lower than in the initial solubilisation in the crude extracts when using immobilised Con A,[218,255,259,286] WL [97,119,125,202,279] or dolichos lectin.[209] Extremely low TX-100 concentrations (0.01–0.02%) were effective in the purification of olfactory receptors.[107,309] The absence of TX-100 did not permit elution of receptors even at high carbohydrate concentrations (0.5–1.0 M) in the case of (Con A)-sorbent.[182,215,259] As a rule, it is important to control the ionic strength of the buffer when selecting the detergent concentration. The buffer ionic strength is usually increased during lectin affinity chromatography from 0.1–0.2 M to 0.4–0.5 and even to 1 M NaCl or KCl in the case of receptors of neurotoxin or bovine dopamine.[75,200] Chromatography of receptors on WL-sorbent is carried out as a rule in the presence of 5–10 mM Mg^{2+} ions[77,97,107,119,125,279] and/or 1–2 mM Ca^{2+} ions.[45,46,50,52,131,133,142–145,200] Ca^{2+} cations are also used in the chromatography of receptors on FGA- and (CL-2)-sorbents but in the case of Con A-, LL- and (pea lectin)-sorbents 0.5–1.0 mM manganese, potassium and magnesium chlorides are used together.

Various combinations of TX-100 and Na desoxycholate are also used,[34,210,211,239,289] although in some cases the preferential use of nonionic detergents (TX-100 and CHAPS) rather than cholate has been recorded, such as on solubilisation of the serotonin transporter, Na-channel, etc.[23,31,36] Cholate (0.5–1.5%) is used in the solubilisation of erythrocyte Cad-antigen, liver esterase, the antigens of nematode parasites and AIDS virus, 5′-nucleotidase and macrophage receptors.[172,184,239,266,275] During lectin affinity chromatography the cholate concentration may vary depending on the type of receptors being investigated. For Dopamine receptors 0.2–0.3% is used,[73–75] for tumour glycoproteins[210,211,216] and proteins of T-lymphocytes[160,164] and thrombocytes 0.4–0.5%,[154] for thyroid gland glycoproteins[275,289] 0.6% and for the L-Glu receptor 0.05%.[33a]

A 0.5–2% solution of 3-[3-(cholamidopropyl)dimethylammonium]-1-propanesulphonate (CHAPS) is widely used for the solubilisation of receptors.[57,77,94,111,112,248,265] It is seemingly promising to use fractionation of receptors with detergents. In the case of vasopressin receptors, contaminants are removed by a preliminary treatment with 0.5% CHAPS and the actual receptors are solubilised in high yield on subsequent treatment of the extract with 2% CHAPS.[94] When solubilising Ca-binding sarcolemma glycoproteins and receptors of corticotropin releasing factor 6–8 mM CHAPS is used.[42,115] The concentration of CHAPS varies in the following way during chromatography on WL-, Con A- or (CL-2)-sorbent: 20 mM for the glutamate receptor,[33] 0.1–0.8% for other transporter glycoproteins[28,41,52] and 1% for receptors of inositol triphosphate, the bacterial antigen of infected ferret lung cells and carboxypeptidase M.[55,230,293]

One more nonionic detergent, Nonidet P-40 (NP-40), is widely used for the solubilisation of receptors at concentrations of 0.5–2%.[72,129,143,159,161,212,224,283,295,303] Lower concentrations of NP-40 are used during chromatography on lectins, e.g. 0.1–0.2% for WL- and (Con A)-sorbents,[72,318] 0.4–0.5% for immobilised Con A,[258] LL,[168] WL,[100,145] AL [159] and thorn apple lectin.[212] The concentration of NP-40 was reduced from 0.4 to 0.07% at the stage of eluting receptors with carbohydrate.[258] The need to use 0.4% NP-40 for oligosaccharide transferase is explained by the fact that the glycans of this glycoprotein bound to asparagine are inverted inside the membrane vesicles and do not react with the lectin without previous treatment with a relatively high concentration of detergent.[258]

Digitonin (1–2%) is also used for the solubilisation of receptors.[45–47,50,51,53,54,59,61,64–66,68,76,79,80,93,98] Occasionally it is expedient to change the type of detergent. Thus, after solubilisation of receptor complexes with 1% digitonin (for example Ca channels) the components of the complexes may be separated successfully using 1% TX-100.[54,84] Digitonin (0.1%) is used in the chromatography of opioid receptors,[59,61,64] receptors of dopamine and cholecystokinin,[76,79,80,98] α_1-adrenergic receptors,[65,66] and receptors of dihydropyridine and Ca channels.[45–47,49–51,53] Chromatography is usually carried out in this way on WL-sorbent although there are also data for immobilised CL-2,[98] LL and Con A.[59,64,98,305]

n-Octyl-β-D-glucopyranoside (0.05–0.2 M) is also used for the solubilisation of receptors.[128,131,135,137,141,144,226] The octylglucoside (40–50 mM) is included at the stage of purification by gel filtration as in the case of β_1-integrin and sphingomyelin.[137,278] The octylglucoside (2 mM) is used during chromatography of glycoproteins of animal lung alveoli on immobilised *Maclura* lectin.[186] Yeast peptidase solubilised in NP-40 was chromatographed on (Con A)-sorbent in the presence of 0.3 mM octylglucoside.[295]

Sodium dodecyl sulphate (SDS: 2%) is also used for the solubilisation of receptors of slime mould antigen,[246] the cationic detergent DTAB for H^+,K^+-ATPase[303] and 8 M urea for the bacterial antigen of infected ferret cells.[230] Tween-20 or Tween-40, Renex-30 or Brij are used for lymphocyte receptors[130,160,164] Lubrol PX for adenylate cyclase and guanylate cyclase[312,313,315,316] and lysolecithin in combination with other detergents for Mg^{2+}-ATPase.[305] Replacement of Brij (0.5%) by cholate (0.5%) or Zwittergent (1%) is used in the purification of antigens on WL-sorbent.[164,238] Emulgen-109P (1%) is also used for the chromatography of fucosylated erythrocyte proteins on immobilised aleuria (*Aleuria aurantia*) lectin,[169] Zwittergent (0.5%) in place of TX-100 (0.5% for the chromatography of Purkinje cell antigen on (Con A)-sorbent,[198] Tween-60 (0.1%) in the purification of adenylate cyclase[312,313] and Lubrol (0.1–1%) in the purification of guanylate cyclase[315,316] on immobilised WL and Con A respectively.

Occasionally $C_{12}E_8$ [n-dodecylocta(ethyleneglycol) monoester] is used, 1% for solubilisation[307,309a] and 0.1% for lectin affinity chromatography.[294]

Traditionally SDS is used during lectin affinity chromatography, 0.01% at the stage of eluting with *N*-acetyl-D-glucosamine for WL-sorbent,[40,302] 0.1–0.5% for (Con A)-sorbent[203,235,318] or WL-sorbent,[246] 1% for the non-specific elution of receptors, particularly for immobilised SL, CL-1, Con A and LL.[165,195,318] The use of such a set of non-specific elutions will possibly be useful for regenerating lectin sorbents. However, it is necessary to keep in mind that SDS may be precipitated as a solid in the presence of metal cations,[203] and may wash out lectin from the sorbent and reduce the lectin capacity, as in the case of immobilised Con A.[235] In addition some proteins are able to form complexes (such as dimers) even in the presence of SDS as in the case of the opioid receptors.[62]

The multistage process of receptor chromatography on a lectin sorbent (application, different variants of washing the sorbent, elution of receptors) is characterised by a reduction in detergent concentration or by variation of combinations of different detergents and by the possible absence of detergent at the very last stage, viz. elution of receptors with carbohydrate. This is seen when purifying thrombocyte glycoprotein-Ib,[148] lymphocyte antigen CD 43,[165] dopamine receptors,[77] sarcolemma receptors,[42] yeast galactosyl transferase,[255] insulin receptors[92] and adrenal glycoproteins[191] using immobilised WL. The detergent itself (SDS) was ineffective even at high concentrations in comparison with *N*-acetyl-D-glucosamine in eluting the purified receptors from the WL-sorbent.[165] Desorption of receptors with carbohydate without a detergent (TX-100) did not occur for the (Con A)-sorbent (see above).

Treatment of extracts or purified membrane glycoproteins with a chloroform–methanol mixture was also carried out to solubilise phospholipid-containing glycoproteins (such as ligatin) or to remove lipid contaminants.[171,237,319] Treatment with organic solvent is seemingly also important when regenerating the lectin sorbents used in the first stages of purifying receptors because of the contamination of the sorbents with glycolipids.[291]

Apart from the means of solubilisation enumerated above there is undoubted interest in the data on the solubilisation of receptors with hydrolases since the soluble receptors obtained frequently do not even require the presence of detergents in the elutant during lectin affinity chromatography. Phospholipase C is used for the solubilisation of hepatoma glycoproteins,[217] trypsin for lysosomal enzymes,[280] trypsin in combination with TX-114 for CD 36 antigen,[152] Ca^{2+}- and thiol-dependent proteinase for thrombocyte glycoprotein-Ib,[148] papain for lymphocyte Fc-receptors solubilised with Tween and Renex and purified on a WL-sorbent[130] and lysozyme for the treatment of the high molecular form of fly integument alkaline phosphatase associated with chitin and purified on a (Con A)-sorbent.[268] Trypsin was also useful for the separation of the subunits of the saccharase–isomaltase complex from human intestinal membranes.[287]

Organic solvents are used together with detergents during receptor chromatography on immobilised lectins and improves the stabilisation of receptors while reducing hydrophobic interactions between receptor and lectin. Ethyleneglycol is used in chromatography on (Con A)-sorbent, 1 M for the purification of Thy-1 antigen,[160] 20% for phospholipase A_1 [269] and 10% (together with 25% glycerol) for guanylate cyclase.[316] The increase in concentration of ethyleneglycol used may be compensated by a reduction in the concentration of carbohydrate eluting the receptors,[160] which indicates the possibility of also using carbohydrate for reducing the hydrophobic binding of receptors with the lectin. Chromatography on immobilised GL-1 was carried out in the presence of glycerol (5%) but the concentration of glycerol was varied when using WL-sorbent from 10% [38,50,52,111,117,163] [for (Con A)-sorbent[310]], to 20% [155,246,257] [for (Con A)-sorbent[258]] or 30%.[107]

β-Mercaptoethanol (1 mM)[235,303] was used in the purification of the rat inositol triphosphate receptor and of trypanosome antigens on a (Con A)-sorbent and 10 mM β-mercaptoethanol in combination with 2% detergent in the isolation of the glycosylated subunits of ATPase on WL-sorbent.[56] Evidently other agents capable of reducing disulphide bonds in protein complexes may also be used.

Problems of eluting receptors, bound to lectin sorbents, with carbohydrates are important. Excess concentrations of carbohydrates are usually used (up to 0.5 M, more rarely 1 M monosaccharide) as in the following, α-methyl-D-mannopyranoside and α-methyl-D-glucopyranoside for Con A, LL- and PL-sorbents, *N*-acetyl-D-glucosamine for immobilised WL and thorn apple lectin, lactose or D-galactose for immobilised CL-1, CL-2, AL and SL, *N*-acetyl-D-galactosamine for SL and L-fucose for immobilised GL-1 and aleuria lectin. Occasionally stepwise elution with increasing concentrations of carbohydrate or a linear carbohydrate gradient is used, such as in the purification of membrane-bound oxidase[248] and histamine receptors[81] on (Con A)-sorbent or membrane-bound lipoamidase,[296] receptors of dihydropyridine and dopamine transporter[49,80] on WL-sorbent. Firstly this enables removal of contaminating glycoproteins with a lower affinity for the lectin, secondly, it permits separation of subpopulations of receptors close in physicochemical properties, thirdly, it enables removal from the sorbent of firmly bound glycoproteins with the aim of regenerating the immobilised lectin, fourthly, it affords a quantitative assessment of the strength of the binding of the purified receptors to the lectin in order that excessive concentrations of carbohydrate are not used for subsequent desorption. It was shown that lipoamidase was eluted at 60 mM *N*-acetyl-D-glucosamine,[296] dopamine transporter at 125 mM *N*-acetyl-D-glucosamine,[80] histamine

receptors at 80 mM α-methylmannoside,[81] but Trp oxidase was separated into forms eluted at 10, 20 and 50 mM α-methylglucoside respectively.[248] Before elution of receptors with a carbohydrate specific for the lectin it was useful to wash the lectin sorbent either with lower concentrations of the same carbohydrate (a variant of the stepwise elution with carbohydrate) as for LL- and (Con A)-sorbents[134,293,294] or with a carbohydrate non-specific for the lectin at a relatively high concentration (0.5 M) as for WL- and (griffonia lectin-I)-sorbents.[108,183,227] When working with immobilised bean lectin, the elution of lymphocyte receptors was carried out without using carbohydrate (monosaccharides were unsuitable for this lectin which is specific for oligosaccharides) by acidifying the elutant to pH 3 [158] as was used for the elution of eukaryote nuclear glycoproteins during chromatography on WL-sorbent.[227]

When working with immobilised WL, significantly more effective eluting agents are used in place of *N*-acetyl-D-glucosamine. These include chitotetraose,[192] 3–10 mM chitotriose[100,110–112,163] or 15 mM chitobiose (containing 10 wt.% chitotriose as contaminant) as for immobilised thorn apple lectin.[161] It is not excluded that contaminating chitotriose and chitobiose in commercial preparations of *N*-acetyl-D-glucosamine make an appreciable contribution to the specific desorption of the receptors indicated above.

Provision of a relatively long contact time for the immobilised lectin and the glucoconjugate solution (when applying it) and with the desorbing carbohydrate (when eluting glycoconjugates and regenerating the sorbent) is a feature of lectin affinity chromatography. The contact time depends on the temperature. For complete binding of receptors with a WL-sorbent incubation of the mixture was carried out for 16–70 h at 4 °C [93,107,154,155,239] or 1.5–3 h [53,110,111] under conditions of suspending the sorbent[53,107,110,154,239] or recycling application of the receptors onto the sorbent as a variant of the column chromatography.[79,91,92,111,155] Elution of receptors from WL-sorbent with carbohydrate was carried out overnight[93] or for 1.5–3 h at 4 °C,[53,75] 15–20 min at room temperature[194,342] or with a double treatment with carbohydrate for 30 min and longer.[92,107,163] Extended contact of receptors and sorbent was also used for immobilised thorn apple, lentil, soyabean and lotus lectins.[134,212,239] When working with (Con A)-sorbent, the application of receptors was carried out for 12–21 h at 4 °C [239,316] or for 4 h at room temperature.[265] Desorption was carried out with portions of carbohydrate after incubation for a period of several hours[47,92,269] and increasing the temperature to 37 °C with the aim of reducing the hydrophobic binding of receptors to the lectin.[25,280]

An important factor of lectin affinity chromatography is the affinity or sorption capacity of the lectin sorbent determined either by binding model standard glycoproteins or by the quantity of lectin immobilised in 1 ml gel (the companies manufacturing lectin sorbents usually indicate the capacity from the amount of lectin in 1 ml gel). Since the data shown in Tables 1 and 2 comprise lectins immobilised on agarose in an overwhelming majority, data on the capacity of agarose sorbents will be considered below. The commercial preparations of WL-agarose (Sepharose, Affigel) most frequently used have the following lectin capacities: 5 or 1–2 mg ml^{-1} gel (Pharmacia, Sweden[65,214]), 6–10 mg ml^{-1} (E.Y. Lab., USA[36]), sorbent manufactured by Miles–Yeda (Israel[320]), 1 mg ml^{-1} (Bio-Rad, USA[124]) etc. In some cases investigators have synthesised the sorbents themselves, for example WL-Sepharose 4B (6–20 mg ml^{-1} [35,48,117]), WL-Affigel-10 (5–10 mg ml^{-1} [83,196]) and WL-Reactigel-6X (5 mg ml^{-1} [109]). Comparison of the synthesised WL-Sepharose sorbents with various lectin capacities in the range 0.08–10 mg ml^{-1} showed that one glycoprotein (glycophorin A for example) was well bound on the low capacity sorbent (0.08 mg ml^{-1}), but for other glycoproteins (such as fetuin, ovomucoid and α_1-glycoprotein or orosomucoid) capacities of more than 3 mg lectin per ml gel were necessary.[176] Erythrocyte glycophorin receptors and receptors from hepatoma cell were successfully obtained from crude extracts in good yield and a high degree of purity by purification in one step on WL-Sepharose (0.08 mg ml^{-1}).[176] When purifying insulin receptors, it was shown that the maximum yield of receptors was achieved using Miles-Yeda WL-sorbent in comparison with similar sorbents from other companies.[320] WL-agarose with a capacity of 4 mg ml^{-1} or above is most frequently used for purifying receptors. Exceptions are hepatoma cell sialoglycoproteins[124,176] and α_1-adrenergic receptors.[320] It was noted that when applying an excess quantity of receptors to a WL-sorbent its sorption properties were increased and there was a corresponding increase in the yield of material bound.[83] The same effect may also be observed on increasing the contact time of receptor and lectin (see above).

In the majority of the cases shown in Tables 1 and 2 the WL-sorbents are used as sialo-specific reactants for binding sialoglycoproteins which may readily be checked by treating the receptors with sialidase.[256] Immobilised succinylated WL is used as a sorbent specific for *N*-acetyl-D-glycosamine, as in the purification of the nuclear pore protein of mammalian cells,[227] or unmodified WL-sorbent for the purification of oligodendroglial glycoproteins.[196] It is better to use immobilised slug (*Limax flavus*) lectin in place of WL-sorbent for the purification of glycoproteins (such as eel Na-channels) which contain polysialic acids [(NeuAc-α-2,8-)$_n$].[35] Among the other widely used lectinagarose sorbents those with relatively high capacities may be mentioned such as 10–16 mg ml^{-1} gel for Con A (Pharmacia, Sweden), 12 and 4 mg ml^{-1} gel for CL-1 and bean erythroagglutinin.[156] The capacity of LL-Sepharose is 2 mg ml^{-1} gel (Pharmacia, Sweden). (Con A)-Sepharose of low capacity (0.5 mg ml^{-1}) is used for the purification of yeast peptidase.[295]

The means of immobilising the lectin onto the agarose may influence the binding of receptors. A comparison of the various means of activating Sepharose for the subsequent immobilisation of Con A showed that the maximum capacity of the sorbent for lectin at minimum selectivity towards glycoproteins was achieved for activation with cyanogen bromide, a significant reduction in capacity at maximum selectivity was found for activation with divinyl sulphone and intermediate results were obtained with glutaraldehyde.[321] Lectin–agarose sorbents immobilised with a carbodiimide have also been used for the purification of receptors.[156] It was noted that irrespective of the means of activating agarose the selectivity of the lectin sorbent was increased when applying an excess amount of the glycoprotein being purified.[321] It is possible that the reason for the irreversible binding of part of the applied glycoprotein when using a lectin sorbent obtained from cyanogen bromide-activated Sepharose (such as LL-Sepharose) is the presence of unblocked residues of carbonate and imidocarbonate groups, which are eliminated by previous treatment of the activated agarose with 0.1 N HCl.[322] An additional treatment of the immobilised lectin with glutaraldehyde in the presence of the appropriate lectin carbohydrate is recommended for the stabilisation of lectin sorbents with the aim of preventing washing out of lectin and reducing the sorbent capacity particularly in the presence of detergent.[58,318]

III. Investigation of receptor glycans with lectin sorbents

A selection of lectins with a known oligosaccharide specificity may serve as an effective instrument for the structural analysis of receptor glycans (see reviews on the specificity of lectins towards glycans[323,324] or glycoconjugates[21]). Such an analysis frequently leads, with the aid of combined chromatography, to lectins of the desired glycoproteins or their glycopeptides and oligosaccharides (see examples in reviews[21,25,26]) sometimes combined with treatments of the glycoconjugates with glycosidases, with glycosoxidases, with chemical modification of the carbohydrate portion or with chemical deglycosylation. The choice of lectin may prove to be useful for the separation of the receptors obtained into sub-populations depending on their microheterogeneity in carbohydrate. For example, glycophorin A type N, in contrast to glycophorin A type M, does not have glycans bound to asparagine (Asn) which enables both types to be separated readily on immobilised LL [173,175] but not on a WL-sorbent.[177] Glycophorins A, B and C are readily removed from glycophorin E by their specific binding to immobilised WL.[176] The Thy-1 antigen does not have glycans bound to serine/threonine (Ser/Thr) and its separation into three sub-populations occurs due to the Asn-glycans 1) not reacting with the lectin fraction, 2) being retained (emerging in a large volume of eluent) and 3) bound (as a result of glycans of the polylactosamine type).[161] The neural cell adhesion molecules (N-CAM) of brain contain tissue-specific Ser/Thr-bound glycans with an affinity for immobilised AL,[146] and also polysialic acids possibly reacting with slug lectin.[35,325] The hexose transporter in defective cells has unmasked Asn-glycans of the oligomannose type but in normal cells these glycans are masked by Asn-glycans of complex type which reduces the affinity for Con A and increases binding with WL.[29] Tri- and tetra-antenna (poly-antenna) Asn-glycans of a complex type are detected in lactotransferrin and transferrin receptors and are particularly numerous in the transferrin receptor according to results from the interaction with L-FGA.[132,138,139] The varying ratio of Asn-bound glycans of various types is readily determined for various histocompatibility antigens and their α- and β-chains using combinations of lectin affinity chromatography of the oligosaccharides by the analysis of the three fractions from (Con A)-sorbent, the three fractions from (CL-1)-sorbent and the two fractions from LL-sorbent.[207,208]

The oligosaccharide structures of the erythrocyte β_1-adrenergic receptor and of β-fructofuranosidase[70,284] have been established with the aid of lectins, as has the common localisation in subunits of glycans of the oligomannoside and of complex types in dopamine receptors,[72] the distribution of poly-antenna sialylated oligomannoside glycans and of glycans with a branched residue of *N*-acetyl-D-glucosamine in the four non-identical sub-units of the acetylcholine receptor.[88] The presence has been revealed of glycans of the lactosamine type in the β-sub-unit of the thrombocyte Ib receptor and in the α_2-like component of the Ca-channel sensitive to dihydropyridine or in both sub-units of the intestinal peptide receptor,[54,96,149] and also in two sub-populations of the dopamine receptor, in the receptors of corticotropin releasing factor and in α_1-adrenergic receptors.[65,75,115] Differences between six of the seven glycoproteins of milk lipid droplet membranes were successfully detected with the aid of two immobilised lectins (Con A and WL) as a result of the retention of these glycoconjugates on elution.[203]

Chromatography on lectin is important not only for the assessment of glycan structure and correcting the carbohydrate microheterogeneity of receptors but also for obtaining functionally active sub-populations of receptors, such as in relation to the enzymic activity of receptors.[57,110,314] When purifying on lectins glycoprotein receptors with an established glycan structure it is possible to take the features of the interaction of a lectin with standard reagents into account for subsequent standardisation of the lectin specificity.

When studying receptors it is possible to use a set of lectins in combination with glycosidases and in combination with antibodies. In essence, the combined use of lectins and antibodies is foreseen in all antigen purification procedures since initially antigens are precipitated from extracts by immunoprecipitation (in Tables 1 and 2 several cases are shown of immunoaffinity chromatography jointly with a lectin sorbent). Since immunoprecipitates comprise a collection of polypeptides (such as sub-units) the localisation of glycans in antigen components by lectins enables one to speak of the intra-antigenic specificity of lectins. Often the carbohydrate structures in the glycoconjugates themselves emerge in the role of antigens important for biorecognition.[10,11,170,326] In addition carbohydrates may play an appreciable role in receptor function, as in the case of complement receptors, the transporter activity of Na^+/H^+-antiporter and of the glucose transporter.[44,129,327] In other cases glycans of a particular type are not important for receptor function, for example the receptor of lutropin or *E*-cadherin.[104,328] It is possible that glycans are important for the directed assembly of receptor components (see above) and also serve to anchor the lectins and lectin-like effectors. There is scope for the purification of antigens using lectins as is evidenced by the data on the antigenic specificity of lectins in relation to cells.[329–331]

IV. Interconnection between receptors, endogenous lectins and enzymes

The antigens of human leucocytes and their interaction with adhesion receptors and receptors of homing lymphocytes have attracted considerable interest from investigators.[4–9] As a rule the antigens of leucocytes are glycoproteins,[165] such as CD 36, CD 43, CD 44, CDw 44,[152,164,199,332,333] which react with immobilised lectins. In addition some antigens themselves display the properties of lectins (for example CD 14 binds lectin lipopolysaccharide[334]) or adhesins (such as antigens CD 8, CD 11, CD 19, etc.[335,336]). In addition antigens are frequently associated with enzymes. Leukemia antigen CD 13 is aminopeptidase N,[337] CD 45 is a protein-tyrosine-phosphatase,[338] C 73 is an exo-5′-nucleotidase[339] etc. Antigen 1D 4 from mammalian brain is not only associated with a type B nucleoside-diphosphatase but also reacts with lectins forming a triple complex.[340] Enzymes forming part of the so-called adhesin molecules on cell surfaces participate actively in the functioning of membrane channels (such as Na^+,K^+-ATPase of rat brain[304]) and serve as receptors of polydomain proteins of the extracellular matrix (for example, chicken muscle 5′-nucleotidase is a fibronectin receptor[341]). In addition a serine esterase is necessary for the development of monocyte adhesion due to IgG receptors (antigen CD 32) and carboxypeptidase N (kininase I) possibly functions with peptide hormone receptors (kinins).[292,342] An important role in cell metabolism is played by the membrane-bound enzymes of tyrosine metabolism, such as tyrosyl-protein sulphotransferase (which reacts with a sialo-specific lectin[256]), tyrosinase (which reacts with an

α-mannose-specific lectin[253]), tyrosine kinases in the composition of insulin receptors[260,343–345]), receptors of insulin-like growth factor type I,[120] receptors of type I colony stimulating factor or growth factor obtained from thrombocytes,[121,260] interleukin-3 receptors,[162] receptors of the Ca^{2+} antagonist or Ca-channel blockers,[52] and receptors of lysosomal enzymes.[346] Receptors with tyrosine kinase and other protein kinases react specifically with WL (see Tables 1 and 2) and the receptors of the lysosomal enzymes themselves display the properties of lectins specific for mannose-6-phosphate. It was also shown that the phosphorylation of proteins by kinases may be inhibited by initiation factor 2 reacting with a lectin specific for the *N*-acetylglucosamine residue,[347] although the activation of receptors containing kinases by binding with a sialospecific lectin is also possible.[91] The data presented indicate the close interaction of cellular antigens, enzymes and lectins forming a single complex functioning system. Other examples of modulation (inhibition or activation) of membrane enzymes by lectins have been shown previously.[25] The highly effective procedures listed in Tables 1 and 2 for the purification of receptors and membrane-bound enzymes of various classes of the International Classification (achieving a degree of purification of 50–440 times on a lectin sorbent) are additional proof of the possibly important role of the participation of endogenous lectins in the regulation of such receptor systems.

An important role in biorecognition is discharged by a receptor lectin[10,12,20,326,348] which has been well studied in the case of mammals and is a complicatedly organised oligomeric high molecular glycoprotein with a set of functionally different domains (collagen type, carbohydrate-binding portions, carbohydrate-containing portions as targets for other lectins, growth factors regulators of the complement system, etc.). Examples are the receptors of mammalian liver asialoglycoproteins,[16] receptors binding IgE or IgG (antigens CD 16, CD 32, CD 64),[336,342,349] homing receptors[350–356] and other adhesion proteins with lectin properties.[357,358] Each of the receptor proteins indicated may under definite conditions function according to one of the domains expressed, consequently it is occasionally difficult to determine whether the receptor is behaving as a lectin or as a donor of carbohydrates in a reaction with another glycoprotein. In such cases help is obtained from an analysis of the primary amino acid sequence or analysis of the nucleotide sequence with subsequent recognition of portions of homology with known lectins. Thus the similarity has been demonstrated for lectin domains of the Fc receptor for IgE and lectin-35, which contains RNA, and other soluble lectins,[349,359,360] IgE and IgG receptors,[349] a laminin-binding receptor of the non-integrin type and lectin-35,[361] ELAM-1 adhesion molecules and lectins,[358] insulin-like growth factor II and the mannose-6-phosphate receptor[362–364] and for elastin receptors and galactose-binding lectins.[365,366] A biochemical and immunological similarity has been shown for the type III complement receptor (antigens CD 11b and CD 18) with lectins and with conglutinin,[336,367] the β-glucan-specific receptor of monocytes (lectin) and the phagocytosis receptor of complement alternative route activators,[368] the receptor of circulating mannosyl glycoproteins from liver sinusoidal cells (lectin) and cerebellum lectin R1,[369] and also the erythrocyte lectin receptors, glucose transporters and band 3, 4 and 5 proteins,[370,371] carbohydrate transporter and nucleotide-binding protein.[372] Other receptors and antigens also participate in lectin–glycoconjugate interactions, such as the interaction of endogenous ceruloplasmin (ferroxidase) and human erythrocytes,[373,374] complex-formation between antigen CD 4 and the 160 kD coat glycoprotein of human immunodeficiency virus type 1,[375] antigens CD 4, CD 8, CD 2 and CD 3 and mitogenic exogenous (foreign) lectins of plants or microbial virulency factors.

Other examples of receptors similar to one another may be found in Table 1. In addition, a structural and functional similarity between the elastin and laminin receptors[380] and between the α_2-macroglobulin receptor and the low density lipoprotein receptor[381] has been shown.

Examples of receptor lectins and adhesins of various organisms (including microorganisms) may also be found in books.[26,376] Besides the lectins mentioned above with the usual (frequently met) specificity towards carbohydrates (mannose-, galactose- and fucose-specific) the specificity of new lectins as potential instruments for the analysis of glycoconjugates must be mentioned. Receptor ligatin is specific for mannose-6-phosphate in glycoprotein composition,[26,319] lectins of T-suppressors from fish roe are α-rhamnose-binding,[12,26] receptors of lymphocyte homing display sialospecificity and an affinity towards sulphated fucan,[353,354] the colonisation factors of bacteria possess an increased affinity towards sulphated glycolipid,[18] adhesive lectins of Protozoa (parasites, such as the stimulators of amoebic dysentry) are directed to a galactoso/*N*-acetylgalactosamine-specific protein,[377] receptor lectins of insect larvae (such as mosquitos) are *N*-acetylglucosamine-specific,[378] the sorption domains on polysaccharides of several microorganism glycosidases and receptors of macrophage phagocytosis are β-glucan-specific,[27,368] the core protein of cartilage proteoglycan behaves as if xylose-specific, type I fimbria of bacteria and lectins of several plant bulbs recognise glycans of the oligomannoside type[21] and the lectin receptors of hepatocytes are specific for the L-glycero-D-mannoheptose residue in lipopolysaccharides.[20,379] After solubilisation of such receptor lectins (with enzymes, low ionic strength, etc. see above) the preparations obtained may be used in a set with the other commercial lectins as described above.

V. Conclusion

Potential targets for lectins may be not only the glycoproteins listed above (see also Tables 1 and 2) but also proteoglycans bound to the cell surface (such as the hyaluronate-binding receptor or the high molecular form of insect alkaline phosphatase associated with chitin,[199,268] polysaccharides with various degrees of branching and bond type,[21,382] lipopolysaccharides,[20,21,379] lipophosphoglycans[298] and glycolipids[18,21]). Apart from the external and cell surface-bound glycoconjugates many intracellular glycoconjugates bound to organelles have been described, which react with a wide selection of lectins,[11] including poly A polymerase, ribonucleotide reductase and glycoprotein 62 of the nuclear membrane[227,249,265] galactosyl transferase of the Golgi apparatus,[255] phospholipase A_1, tyrosine kinase and lysosomal α-glucosidase,[269,280,346] eukaryotic ribosomal initiation factor-2,[347] glycosidases for processing glycoproteins of the endoplasmic reticulum[281–283] and others (see Tables 1 and 2). Procedures have been described for the purification and separation of proteoglycans, polysaccharides and unidentified glycoconjugates on lectin sorbents.[21,26,382,383] After solubilisation in 95% tetrahydrofuran in water glycolipids may be separated successfully on immobilised lectins, such as on (snail lectin)-sorbent.[384] Bacterial lipopolysaccharides may also in principle be separated by selective precipitation with lectins, particularly in the case of various Ra type lipopolysaccharides.

Different variants of qualitative and quantitative microanalysis using a set of lectins are used for the detection and identification of receptor glycoconjugates.[21,27] Lectin–peroxidase conjugates are widely applied in histochemistry particularly in cases of human cancer and other diseases.[385] Lectins labelled with peroxidase or biotin are most widely used in blotting variants of analysis, such as in the case of blood cell membrane glycoproteins,[387] histocompatibility antigens,[20] sarcolemma glycoproteins[42] and acetylcholine receptors.[88] Lectin conjugates of this same type are used with multiwell plates for immunoenzyme analysis in the study of receptors of native cells of proto- and eukaryotes,[388–390] plasma membrane receptors[391] or purified receptors, such as erythrocyte receptors for type I fimbriae of bacteria and the phosphomannosyl receptor.[213,392] In this way lectin analysis supplements the use of antibodies to a significant extent.[18,386,393]

Investigators have shown increased interest in receptors of microorganisms both in the composition of the cell surface of eukaryotes and the surface of protokaryotes (see Tables 1 and 2). This is linked with the recognition of types of lectin, i.e. glycoconjugates, in infection and immune processes,[13–15,376] with possible use of lectins in medical diagnostics,[394,395] and also when working with bacteria, yeasts and fungi (examples[393,395–401]).

Data on the interaction of lectins with glycans of soluble glycoproteins[24,26] may be used in the lectin analysis of forms of these glycoconjugates bound to cellular structures, since a significant affinity has been established for the carbohydrate portion of them as in the case of RNAase T_2-L of *Aspergillus oryzae*,[276] β-fructofuranosidase[284] and mammalian liver α-1,2-mannosidase,[283] and also of recombinant glycoprotein-160 of the AIDS virus.[228] Such affinity is explained by the general rules for the biosynthesis of glycoprotein glycans in the endoplasmic reticulum and Golgi apparatus of cells. In addition, in the process of glycoprotein secretion into the extracellular space, glycoproteins remain bound in several cases to the cell surface or are solubilised by endogenous hydrolases (see above on the solubilisation of receptors).

It must be noted that the possibilities of lectins (there is even a set of what would have seemed to be fairly well studied commercial lectins) are still far from being exhausted since our ideas on the specificity of lectins towards oligosaccharides, glycopeptides, neo-glycoproteins (more broadly neo-glycoconjugates) and natural glycoproteins with a known distribution of glycans on the protein surface are constantly being extended, particularly in recent times. The specificity of commercial lectins towards bacterial glycoprotein,[22] steroid glycoside[402] and carbohydrate-containing antibiotics, animal glycolipid and bacterial lipopolysaccharide[21] has not been investigated in practice. New types of carbohydrate–peptide bonds are being discovered, viz. Rha-Rha-Rha-Asn for bacilli, Gal-β-1,3-Ga1*N*Ac-Ser/Thr for fungi, Fuc-Ser/Thr,[403,404] Glc-α-1,6-Glc-β-1,6-Glc-α-1-Asn [405] and Glc*N*Ac-Ser/Thr [11] for mammals. The latter two structures react with immobilised Con A and WL respectively.[11,405]

The procedures for obtaining lectin preparations are therefore relatively simple,[382,385,406] and the list of highly purified lectins from microorganisms, invertebrates, plants and animals is growing rapidly.[26,376] The use of lectin sets in combination with glycosidases and antibodies as fine instruments for the analysis of glycoconjugates is extremely promising.

References

1. A D Strosberg (Editor) *Molecular Biology of Receptors: Techniques and Applications of Receptor Research*, 1987, 235 pp
2. C-H Heldin, B Westermark *Eur. J. Biochem.*, 1989, **184** 487
3. L Birnbaumer, J Abramowitz, A Yatani, et al. *Crit. Rev. Biochem. Mol. Biol.*, 1990, **25**(4) 225
4. V Horejsi, V Bazil *Biochem. J.*, 1988, **253**(1) 1
5. L M Stoolman *Cell*, 1989, **56**(6) 907
6. T A Springer *Nature (London)*, 1990, **346**(6283) 425
7. B T Sher, R Bargatze, B Holzmann, et al. *Adv. Cancer Res.*, 1988, **51** 361
8. T A Yednock, S T Rosen *Adv. Immunol.*, 1989, **44** 313
9. M W Kieran, V Blank, O Le Bail, A Israel *Res. Immunol.*, 1989, **140**(4) 399
10. R Schauer *Biochimie*, 1988, **70**(11) 1705
11. G W Hart, R S Haltwanger, G D Holt, W G Kelly *Ann. Rev. Biochem.*, 1989, **58** 841
12. M Monsigny, A C Roche, C Kieda, et al. *Cell to Cell Signals in Plant, Animal and Microbial Symbiosis* (Edited by S Scannerini, et al.), Springer-Verlag, Berlin, 1988, p. 237
13. D M Weir *FEMS Microbiol. Immunol.*, 1989, **47**(6–7) 331
14. N Sharon *Biochem. Soc. Trans.*, 1989, **17**(1) 11
15. N Sharon *Science*, 1989, **246** 227
16. K Drickamer *J. Biol. Chem.*, 1988, **263**(20) 9557
17. V I Mikhailov *Molekulyar. Biologiya*, 1989, **23**(3) 639
18. K-A Karlsson *Ann. Rev. Biochem.*, 1989, **58** 309
19. N V Lyubimova, V D Shcherbukhin *Prikl. Biokhimiya Mikrobiol.*, 1991, **27**(1) 3
20. J B Parent *J. Biol. Chem.*, 1990, **265**(6) 3455
21. V M Lakhtin *Lectins: Biology, Biochemistry, and Clinical Biochemistry, Vol. 8* (Edited by A Kallikorm, et al.,), Sigma Chemical Company, St. Louis, USA, 1991 (in press)
22. V M Lakhtin, N L Kalinin *Lectin-Microbe Interactions* (Edited by R J Doyle, M Slifkin), Marcel Dekker, New York, 1992 (in press)
23. R Lotan, G L Nicolson *Biochim. Biophys. Acta*, 1979, **559**(4) 329
24. V M Lakhtin *Biotekhnologiya*, 1985, **1**(5) 11
25. V M Lakhtin *Biotekhnologiya*, 1986, **2**(6) 66
26. V M Lakhtin *Itogi Nauki i Tekhniki. Ser. "Biotekhnologiya"* (Results of Science and Techniques. Biotechnology Series), Vol. 2, VINITI Izd., Moscow, 1987, 290 pp
27. V M Lakhtin *Biotekhnologiya*, 1989, **5**(6) 676
28. B E Peerce, R D Clarke *J. Biol. Chem.*, 1990, **265**(3) 1731
29. H C Haspel, J Revillame, O M Rosen *J. Cell. Physiol.*, 1988, **136**(2) 361
30. B I Kanner, S Keynan, R Radian *Biochemistry*, 1989, **28**(9) 3722
31. Y Stern-Bach, N Greenberg-Ofrath, I Flechner, S Schuldiner *J. Biol. Chem.*, 1990, **265**(7) 3961
32. A M Gordon, B I Kanner *Biochim. Biophys. Acta*, 1988, **944**(1) 90
33. N C Danbolt, G Pines, B I Kanner *Biochemistry*, 1990, **29**(28) 6734

33a. S R Gray, F R Batstone, N F Santiapillai, P J Richardson *Biochem. J.*, 1991, **273**(1) 165

34. D Gordon, H Moskowitz, E Zlotkin *Biochim. Biophys. Acta*, 1990, **1026**(1) 80
35. W M James, M C Emerick, W S Agnew *Biochemistry*, 1989, **28**(14) 6001

36. J S Weiner, B Rudy *Biochim. Biophys. Acta*, 1988, **944**(3) 521
37. J W Schmidt, W A Catterall *J. Biol. Chem.*, 1987, **262**(28) 13 713
38. H Rehm, M Lazdunski *Proc. Natl. Acad. Sci. USA*, 1988, **85**(13) 4919
39. K D Philipson, S Longoni, R Ward *Biochim. Biophys. Acta*, 1988, **945**(2) 298
40. D A Nicoll, M L Appleburg *J. Biol. Chem.*, 1989, **264**(27) 16 207
41. D M Reid, U Friedel, R S Molday, N J Cook *Biochemistry*, 1990, **29**(6) 1601
42. M Michalak, L Fliegel, K Wlasichuk *J. Biol. Chem.*, 1990, **265**(10) 5869
43. P Wohlfart, H Muller, N J Cook *J. Biol. Chem.*, 1989, **264**(35) 20 934
44. A N K Yusufi, M Szczpanska-Konkel, T P Dousa *J. Biol. Chem.*, 1988, **263**(27) 13 683
45. A Rengasamy, J Ptasienski, M M Hosey *Biochem. Biophys. Res. Commun.*, 1985, **126**(1) 1
46. V Flockerzi, H-J Oeken, F Hofmann *Eur. J. Biochem.*, 1986, **161**(1) 217
47. C L Cooper, S Vandaele, J Barhanin, et al. *J. Biol. Chem.*, 1987, **262**(2) 509
48. N M Soldatov *Eur. J. Biochem.*, 1988, **173**(2) 327
49. H Kalasz, C Horvath, P L Vaghy *Chromatographia*, 1990, **30**(9–10) 533
50. U Kanngiesser, P Nalik, O Pongs *Proc. Natl. Acad. Sci. USA*, 1988, **85**(9) 2969
51. K P Campbell, S D Kahl *Nature (London)*, 1989, **338** 259
51a. M Yoshida, E Ozawa *J. Biochem. (Tokyo)*, 1990, **108**(5) 748
52. B S Tuana, B J Murphy , Q Yi *Mol. Cell. Biochem.*, 1988, **80**(1–2) 133
53. B J Murphy, C A Rogers, R K Sunahara, et al. *Mol. Pharmacol.*, 1990, **37**(2) 173
54. A Yoshida, M Takahashi, Y Fujimoto, et al. *J. Biochem. (Tokyo)*, 1990, **107**(4) 608
55. C C Chadwick, A Saito, S Fleischer *Proc. Natl. Acad. Sci. USA*, 1990, **87**(6) 2132
56. S Supattapone, P F Worley, J M Baraban, S H Snyder *J. Biol. Chem.*, 1988, **263**(3) 1530
57. A B Theibert, S Supattapone, C D Ferris, et al. *Biochem. J.*, 1990. **267**(2) 441
58. R J Mourey, A Verma, S Supattapone, S H Snyder *Biochem. J.*, 1990, **272**(2) 383
59. T Gioannini, B Foucaud, J M Hiller, et al. *Biochem. Biophys. Res. Commun.*, 1982, **105**(3) 1128
60. F Wang, Z-Y Li, Z-G Chi, et al. *Acta Pharmacol. Sinica*, 1989, **10**(6) 481
61. Y H Wong, C D Demcliou-Mason, E A Barnard *J. Neurochem.*, 1989, **52**(4) 999
62. W F Simonds, T R Burke, K C Rice, et al. *Proc. Natl. Acad. Sci. USA*, 1985, **82**(15) 4974
63. A Valette, P Rouge, E Coulais, et al. *Life Sci.*, 1987, **40**(6) 535
64. B-L Ge, D-H Zhou, H-P Zhang *Acta Pharmacol. Sinica*, 1988, **9**(3) 221
65. B I Terman, J F Reece, R D Brown, P A Insel *Biochem. J.*, 1988, **253**(2) 363
66. B I Terman, R P Riek, A Grodski, et al. *Mol. Pharmacol.*, 1990, **37**(4) 526
66a. M-J Im, R M Graham *J. Biol. Chem.*, 1990, **265**(31) 18 944
67. J W Lomasney, L M F Leeb-Lundberg, S Cotecchia, et al. *J. Biol. Chem.*, 1986, **261**(17) 7710
68. J W Regan, H Nakata, R M DeMarinis, et al. *J. Biol. Chem.*, 1986, **261**(8) 3894
69. S T George, A E Ruoho, C C Malbon *J. Biol. Chem.*, 1986, **261**(35) 16 559
70. P Cervantes-Olivier, O Durieu-Trautmann, C Delavier-Klutchko, A D Strosberg *Biochemistry*, 1985, **24**(14) 3765
71. G L Stiles, J L Bonovie, M G Caron, R J Lefkowitz *J. Biol. Chem.*, 1984, **259**(13) 8656
72. K R Jarvie, G Booth, E M Brown, H BNiznik *Mol. Pharmacol.*, 1989, **36**(4) 566
73. W M Abbott, P G Strange *Biosci. Rep.*, 1985, **5**(4) 303
74. R A Williamson, S Worrall, P L Chazot, P G Strange *EMBO J.*, 1988, **7**(13) 4129
75. M N Leonard, R A Williamson, P G Strange *Biochem. J.*, 1988, **255**(3) 877
76. F J Bosker, F J Van Bussel, A P Thielen, et al. *Eur. J. Pharmacol.*, 1989, **163**(2–3) 319
77. Z Elazar, H Kanety, C David, S Fuchs *Biochem. Biophys. Res. Commun.*, 1988, **156**(1) 602
78. C J Cao, M M Young, J B Wong, et al. *Membrane Biochem.*, 1989, **8**(4) 207
79. R Lew, D Grigoriadis, A Wilson, et al. *Brain Res.*, 1991, **539**(2) 239
80. H B Niznik, R F Tyndale, F R Sallee, et al. *Arch. Biochem. Biophys.*, 1990, **276**(2) 424
81. M Mitsuhashi, D G Payan *Mol. Pharmacol.*, 1989, **35**(3) 311
82. J A Nathanson, L Kantham, E J Hunnicutt *FEBS Lett.*, 1989, **259**(1) 117
83. G S Herron, M I Schimerlik *Arch. Biochem. Biophys.*, 1984, **230**(2) 533
84. J J Rauh, M P Lambert, N J Cho, et al. *J. Neurochem.*, 1986, **46**(1) 23
85. G M Dmytrenko, M G Scher, G Poiana, et al. *Exp. Cell. Res.*, 1990, **189**(1) 41
86. D M Alperin, V P Idoyaga-Vargas, H Carminatti *J. Neurochem.*, 1986, **47**(3) 355
87. D M Alperin, C B Bouzat, F J Barrantes *Biochem. J.*, 1987, **251**(2) 657
88. H Nomoto, N Takahashi, Y Nagaki, et al. *Eur. J. Biochem.*, 1986, **157**(2) 233
88a. P S McPherson, K P Campbell *J. Biol. Chem.*, 1990, **265**(30) 18 454
89. J A Hedo, L C Harrison, J Roth *Biochemistry*, 1981, **20**(12) 3385
90. E Z Tchlian, I E Zhelezarov, T A Getova, M S Kojouharova *C. R. Acad. Bulg. Sci.*, 1990, **43**(5) 57
90a. C Wang *Biochim. Biophys. Acta*, 1986, **888**(1) 107
91. R H Whitson, G K Grimditch, E Sternlicht, et al. *J. Biol. Chem.*, 1988, **263**(10) 4789
92. M Azam, G Gupta, N Z Baquer *Biochem. Intern.*, 1990, **22**(1) 45
93. C-K Huang *J. Leukocyte Biol.*, 1987, **41**(1) 63
94. J B Fishman, B F Dickey, R E Fine *J. Biol. Chem.*, 1987, **262**(29) 14 049
95. W Kramer, F Girbig, U Gutjahr, I Leipe *J. Chromatogr.*, 1990, **521**(2) 199
96. T D Nguyen, J A Williams, G M Gray, *Biochemistry*, 1986, **25**(2) 361

98. S Patti, M Akong, G Velicelebi *J. Biol. Chem.*, 1987, **262**(32) 15740
99. J Szecowka, G Hallden, I D Goldfine, J A Williams *Regul. Peptides*, 1989, **24**(3) 215
100. L T Duong, E M Hadae, L J Miller, G P Vlasuk *J. Biol. Chem.*, 1989, **264**(30) 17990
100. R K Pearson, L J Miller, E M Hadac, S P Powers *J. Biol. Chem.*, 1987, **262**(28) 13850
101. J K Petell, M Diamond, W Hong, et al. *J. Biol. Chem.*, 1987, **262**(30) 14753
102. S Kusuda, M L Dufau *J. Biol. Chem.*, 1986, **261**(34) 16161
103. K C McFarland, R Sprengel, H S Phillips, et al. *Science*, 1990, **245**(4917) 494
104. K P Keinanen *Biochem. J.*, 1988, **256**(3) 719
105. J Wimalasena, P Moore, J P Wiebe, et al. *J. Biol. Chem.*, 1985, **260**(19) 10689
106. T A Yarney, M R Sairam, G N Bhargavi, S K Mohapatra *Biochemistry*, 1990, **29**(15) 3751
107. B Dattatreyamurty, S-B Zhang, L E Reichert *J. Biol. Chem.*, 1990, **265**(10) 5494
108. B C Kress, R G Spiro *Endocrinol.*, 1986, **118**(3) 974
109. P J Leedman, J D Newman, L C Harrison *J. Clin. Endocrinol. Metab.*, 1989, **69**(1) 134
110. S Knuhtsen, J-P Esteve, C Cambillau, et al. *J. Biol. Chem.*, 1990, **265**(2) 1129
111. H-T He, S Rens-Domiano, J-M Martin, et al. *Mol. Pharmacol.*, 1990, **37**(5) 614
112. O Lascols, J Capeau, G Cherqui, et al. *Mol. Cell. Endocrinol.*, 1989, **65**(1–2) 145
113. M Mitani, M L Dufau *J. Biol. Chem.*, 1986, **261**(3) 1309
114. M L Dufau, S Kusuda *J. Receptor Res.*, 1987, **7**(1–4) 163
115. D E Grigoriadis, E B De Souza *Endocrinology*, 1989, **125**(4) 1877
116. J Massaque *J. Biol. Chem.*, 1985, **260**(11) 7059
117. L Ronnstrand, M P Beckman, B Faulders, et al. *J. Biol. Chem.*, 1987, **262**(7) 2929
118. A Mereau, I Pieri, C Gamby, et al. *Biochimie*, 1989, **71**(7) 865
119. M K Dahmer, L Ji, R L Perlman *J. Neurochem.*,, 1989, **53**(4) 1036
120. T R Le Bon, S Jacobs, P Cuatrecasas, et al. *J. Biol. Chem.*, 1986, **261**(17) 7685
121. S Majumder, K Brown, F H Qiu, P Besmer *Mol. Cell. Biol.*, 1988, **8**(11) 4896
122. K Asakawa, J A Hedo, A McElduff, et al. *Biochem. J.*, 1986, **238**(2) 379
123. M K Sinha, C Buchanan, C Raineri-Maldonado, et al. *Am. J. Physiol.*, 1990, **258**(3), Part 1, E534
124. D DiSorbo, E-G Shi, W C McKeehan *Biochem. Biophys. Res. Commun.*, 1988, **157**(3) 1007
124a. J F DePersio, C Hedvat, C F Ford, et al. *J. Biol. Chem.*, 1991, **266**(1) 279
125. A Traub, S Feinstein, M Gez, et al. *J. Biol. Chem.*,1984, **259**(22) 13872
126. Z-Q Zhang, A Fournier, Y H Tan *J. Biol. Chem.*, 1986, **261**(17) 8017
127. D Novick, P Orchansky, M Revel, M Rubinstein *J. Biol. Chem.*, 1987, **262**(18) 8483
128. J Calderon, K C F Sheehan, C Chance, et al. *Proc. Natl. Acad.Sci. USA*, 1988, **85**(13) 4837
129. C A Vedeler, R Matre, E Fischer *J. Neuroimmunol.*, 1989, **23**(3) 215
130. G Gorni, G A Medgyesi, M Garavini, et al. *Biochem. J.*, 1987, **245**(1) 75
131. J D Ashcom, S E Tiller, K Dickerson, et al. *J. Cell. Biol.*, 1990, **110**(4) 1041
132. W-L Hu, J Mazurier, J Montreuil, G Spik *Biochemistry*, 1990, **29**(2) 535
133. S Johansson, E Forsberg, B Lundgren *J. Biol. Chem.*, 1987, **262**(16) 7819
134. G Tarone, P Mascarello, M Zibetti, F G Giancotti *Eur. J. Biochem.*, 1988, **172**(3) 713
135. S C Stamatoglou, G Rui-Chang, G Mills, et al. *J. Cell. Biol.*, 1990, **111**(5) 2117
136. E Brown, L Hooper, T Ho, H Greshman *J. Cell. Biol.*, 1990, **111**(6) 2785
137. T Darribere, K Guida, H Larjava, et al. *J. Cell. Biol.*, 1990, **110**(5) 1813
138. P A Gleeson, V Dias *Biochim. Biophys. Acta*, 1990, **1036**(1) 47
139. I Poola, J J Lucas *J. Biol. Chem.*, 1988, **263**(35) 19137
140. D A Cheresh *Prog. Clin. Biol. Res.*, 1989, **288** 3
141. I F Charo, L Nannizzi, J W Smith, D A Cheresh *J. Cell. Biol.*, 1990, **111**(6) 2797
142. J W Smith, D J Vestal, S V Irwin, et al. *J. Biol. Chem.*, 1990, **265**(19) 11008
143. S M Albelda, M Daise, E M Levine, C A Buck *J. Clin. Invest.*, 1989, **83**(6) 1992
144. M J Ignatius, L F Reichardt *Neuron*, 1988, **1**(8) 713
145. S M Albelda, P D Oliver, L H Romer, C A Buck *J. Cell. Biol.*, 1990, **110**(4) 1227
146. F S Walsh, R B Parekh, S E Moore, et al. *Development*, 1989, **105**(4) 803
147. M Ozawa, M Sato, H Muramatsu, et al. *Exp. Cell Res.*, 1985, **158**(1) 127
148. V A Canfield, J Ozols, D Nugent, G J Roth *Biochem. Biophys. Res. Commun.*, 1987, **147**(2) 526
149. A N Wicki, K J Clemetson *Eur. J. Biochem.*, 1987, **163**(1) 43
150. L A Fitzgerald, B Leung, D R Phillips *Anal. Biochem.*, 1985, **151**(1) 169
151. T Tsuji, T Osawa *J. Biochem. (Tokyo)*, 1986, **100**(4) 1077
152. N N Tandon, R H Lipsky, W H Burgess, G A Jamieson *J. Biol. Chem.*, 1989, **264**(13) 7570
153. T Shimomura, K Fujimura, S Maehama, et al. *Blood*, 1990, **75**(12) 2349
154. G J Roth, J Ozols, D J Nugent, S A Williams *Biochem. Biophys. Res. Commun.*, 1988, **156**(2) 931
155. F Ushikubi, M Nakajima, M Hirata, et al. *J. Biol. Chem.*, 1989, **264**(28) 16496
156. T Tsuji, T Osawa *J. Biochem. (Tokyo)*, 1987, **101**(1) 241
157. S A M Korrel, K J Clemetson, H Van Halbeek, et al. *Eur. J. Biochem.*, 1984, **140**(3) 571
158. G Dupuis, J-P Doucet, B Bastin, J Cardin *Biochem. Cell. Biol.*, 1985, **63**(9) 932
159. A De Maio, H Lis, J M Gershoni, N Sharon *FEBS Lett.*, 1986, **194**(1) 28
160. S R Carlsson, T I Stigbrand *Biochem. J.*, 1983, **211** 641
161. M H Morrison, R A Lynch, W J Esselman *Mol. Immunol.*, 1986, **23**(1) 63
162. A L-F Mui, P H B Sorensen, R J Kay, G Krystal *J. Cell. Biochem.*, 1989, Supplement, 13C, 29
163. J-P Galizzi, B Castle, O Djossou, et al. *J. Biol. Chem.*, 1990, **265**(1) 439

164. B F Flanagan, R Dalchau, A K Allen, et al. *Immunology*, 1989, **67**(2) 167
165. I Stefanova, I Hilgert, H Angelisova, V Horejsi *Folia Biol.*, 1988, **34**(4) 255
166. M Fukuda, S R Carlsson *Med. Biol.*, 1986, **64**(6) 335
167. S R Carlsson, M Fukuda *J. Biol. Chem.*, 1986, **261**(27) 12779
168. J S Danska, B W McIntyre, H O McDevitt *Intern. Immunol.*, 1990, **2**(9) 795
169. S Yazawa, K Furukawa, N Kochibe *J. Biochem.*, 1984, **96**(6) 1737
170. S Yazawa, N Kochibe, T Asao *Immunol. Invest.*, 1990, **19**(4) 319
171. J Estelrich, M T Montero *J. Biochem. (Tokyo)*, 1989, **106**(5) 745
172. J-P Cartron, D Blanchard *Biochem. J.*, 1982, **207** 497
173. O O Blumenfeld, A M Adamany, M Kikuchi, et al. *J. Biol. Chem.*, 1986, **261**(12) 5544
174. K Yanagi, K Ohyama, T Yamakawa, S Ohkuma *Int. J. Biochem.*, 1990, **22**(9) 1015
175. O O Blumenfeld, A J Smith, J J Moulds *J. Biol. Chem.*, 1987, **262**(24) 11864
176. D Corradini, Z E Rassi, C Horvath, et al. *J. Chromatogr.*, 1988, **458** 1
177. P Pinnaduwage, L Huang *Biochim. Biophys. Acta*, 1989, **986**(1) 106
178. K Furukawa, J E Minor, J D Hegarty, V P Bhavanandan *J. Biol. Chem.*, 1986, **261**(17) 7755
179. S-M Zhang, Z-M Yang, J-X Li *Acta Biochim. Biophys. Sinica*, 1987, **19**(2) 109
180. G M Wiseman, P McNicol *Can. J. Microbiol.*, 1982, **28**(2) 219
181. H Lei, S Ce *Acta Biochim. Biophys. Sinica*, 1990, **22**(3) 252
182. K Tsuchida, M A Wells *J. Biol. Chem.*, 1990, **265**(10) 5761
183. M Satoh, M Yamazaki *J. Pharmacobio-Dynamics (Japan)*, 1987, **10**(11) 608
184. Y Ohkuma, H Komano, S Natori *J. Biochem. (Tokyo)*, 1988, **103**(3) 402
185. S J Geary, M G Gabridge *Isr. J. Med. Sci.*, 1987, **23** 462
186. B C Marshall, M F Joyce-Brady, J S Brody *Biochim. Biophys. Acta*, 1988, **966**(3) 403
187. D Renauer, F Oesch, J Kinkel, et al. *Anal. Biochem.*, 1985, **151**(2) 424
188. H M Szerlip, M Cox *J. Steroid Biochem.*, 1989, **32**(6) 815
189. R D Cummings, A M Soderquist, G Carpenter *J. Biol. Chem.*, 1985, **260**(22) 11944
190. R Kittelberger, T J Neale *Biochem. Biophys. Res. Commun.*, 1990, **172**(2) 439
191. D L Christie, D J Palmer *Biochem. J.*, 1990, **270**(1) 57
192. K-Y Do, D F Smith, R D Cummings *Biochem. Biophys. Res. Commun.*, 1990, **173**(3) 1123
193. Z Chen, U Pace, D Ronen, D Lancet *J. Biol. Chem.*, 1986, **261**(3) 1299
194. W K Allen, R Akeson *J. Neurochem.*, 1985, **5**(2) 284
195. C H Schmelzer, S E Poduslo *Biochim. Biophys. Acta*, 1987, **903**(1) 103
196. C H Schmelzer, S E Poduslo *Biochim. Biophys. Acta*, 1986, **858**(1) 56
197. D D Mikol, K Stofansson *J. Cell. Biol.*, 1988, **106** 1273
198. N Maeda, M Niinobe, K Nakahira, K Mikoshiba *J. Neurochem.*, 1988, **51**(6) 1724
199. M S Marks, G Chi-Rosso, B P Toole *J. Neurochem.*, 1990, **54**(1) 171
200. A G Petrenko, I N Surkova, O G Shamotienko, et al. *Bioorg. Khim.*, 1990, **16**(2) 158
201. A G Petrenko, V A Kovalenko, O G Shamotienko, et al. *EMBO J.*, 1990, **9**(6) 2023
202. F J Tejedor, W A Catterall *Cell. Mol. Neurobiol.*, 1990, **10**(2) 257
203. C Kanno *Agric. Biol. Chem.*, 1986, **50**(12) 2997
204. O Jokinen, J L Gueant, H Schohn, R Grasbeck *Glycoconjugate J.*, 1989, **6**(4) 525
205. M L Rogers, E A Robinson, E Apella *J. Biol. Chem.*, 1979, **254**(21) 11126
206. Y U Katagiri, S Kijimoto-Ochiai, T Hatae, H Okuyama *Comp. Biochem. Physiol.*, 1989, **B-93**(2) 259
207. S Iturbe, S Narsimhan, M Letarte *J. Immunol.*, 1986, **136**(12) 4596
208. D Neel, B Merlu, E Turpin, et al. *Biochem. J.*, 1987, **244**(2) 433
209. Y Arita, S-I Ogata, M Ozawa, T Muramatsu *Differentiation*, 1985, **28**(3) 254
210. S Laferte, J W Dennis *Biochem. J.*, 1989, **259**(2) 569
211. S Laferte, J W Dennis *Cancer Res.*, 1988, **48**(17) 4743
212. M N Fukuda, A Dell, J E Oates, M Fukuda *J. Biol. Chem.*, 1985, **260**(11) 6623
213. C S Giampapa, S N Abraham, T M Chiang, E H Beachey *J. Biol. Chem.*, 1988, **263**(11) 5362
214. R Bardales, V P Bhavanandan, G Wiseman, M E Bramwell *J. Biol. Chem.*, 1989, **264**(4) 1980
215. J C Lin, L K Ligman, E Farber *Cancer Res.*, 1980, **40**(10) 3755
216. S-I Ogata, K O Lloyd *Arch. Biochem. Biophys.*, 1982, **217**(2) 665
217. Y Ikehara, Y Hayashi, S Ogata, et al. *Biochem. J.*, 1987, **241**(1) 63
218. D Josic, W Hofmann, R Habermann, W Reutter *J. Chromatogr.*, 1988, **444** 29
219. B L Valle.e, J F Riordan, R R Lobb, et al. *Experientia*, 1985, **41**(1) 1
220. H Muramatsu, T Muramatsu *J. Biochem.*, 1990, **107**(4) 629
221. J M Ervasti, K Ohlendieck, S D Kahl, et al. *Nature (London)*, 1990, **345**(6273) 315
222. R Cocchiara, G Tarro, G Flaminio, et al. *Cancer*, 1980, **46**(7) 1594
223. L F Qualtiere, R Chase, G R Pearson *J. Immunol.*, 1982, **129**(2) 814
224. J A Sachs, M C Kuo, A H Johnson, et al., *J. Immunol. Meth.*, 1984, **73**(2) 387
225. D L Krah *Virology*, 1989, **172**(1) 386
226. H Mischak, C Neubauer, E Kuechler, D Blaas *Virology*, 1988, **163**(1) 19
227. C M Starr, J A Hanover *J. Biol. Chem.*, 1990, **265**(12) 6868
228. N Barrett, A Mitterer, W Mundt, et al. *AIDS, Res. Human Retroviruses*, 1989, **5**(2) 159
229. T Haraguchi, S Fischer, S Olofsson, et al., *Arch. Biochem. Biophys.*, 1989, **274**(1) 1
230. F Gigliotti, L R Ballou, W T Hughes, B D Mosley *J. Infect. Diseases*, 1988, **158**(4) 848
231. H Stompe, M Drescher, S Mebel, S Rustenbach, German P. 3095875 (1989); *Ref. Zh. Biol.*, 1990, (1), Ref. Ts 13
232. M L Narasu, K P Gopinathan *Biochem. Biophys. Res. Commun.*, 1986, **141**(2) 756
233. P Simoneau, J Labarere *Arch. Microbiol.*, 1989, **152**(5) 488
234. R J Rossell, A F Stevens, M A Miles, A K Allen *Parasitol. Res.*, 1990, **76**(4) 294

235. F Boutignon, M Hublart, V Gomes, et al. *Biol. Cell.*, 1988, **64**(2) 131
236. G Harth, C G Haidaris, M So *Mol. Biochem. Parasitol.*, 1989, **33**(2) 143
237. A S Couto, M F Goncalves, W Colli, R M De Lodeskremer *Mol. Biochem. Parasitol.*, 1990, **39**(1) 101
238. P Willadsen, C A Risling, R V McKenna, et al. *J. Immunol.*, 1989, **143** 1346
239. R M Maizels, W F Gregory, G-E Kwan-Lim, M E Selkirk *Mol. Biochem. Parasitol.*, 1989, **32**(2–3) 213
240. E G Hayunga, M P Sumner *J. Parasitol.*, 1986, **72**(2) 283
241. E G Hayunga, M P Sumner *J. Parasitol.*, 1986, **72**(6) 913
242. S R Karcz, B J Barnard, R B Podesta *Mol. Biochem. Parasitol.*, 1988, **31**(2) 163
243. A N MacGregor, J R Kusel *Mol. Biochem. Parasitol.*, 1989, **34**(3) 237
244. N W T Clark, M Philipp, R M E Parkhouse *Biochem. J.*, 1982, **206**(1) 27
245. C L Saxe, M Sussman *Cell*, 1982, **29**(3) 755
246. Y Kubohara, K Okamoto *Biochem. Cell Biol.*, 1990, **68**(4) 699
247. W M Hearn, D W R Mackenzie *Mol. Immunol.*, 1980, **17**(9) 1097
248. J Ludwig-Muller, W Hilgenberg *Physiol. Plant*, 1988, **74**(2) 240
249. M Sikorska, L M Brewer, T Youdale, et al. *Biochem. Cell Biol.*, 1990, **68**(5) 880
250. J L Nelson, A P Kulkarni *Biochem. J.*, 1990, **268**(3) 739
251. R G L Shorr, M D Minnich, A Varrichio, et al. *Mol. Pharmacol.*, 1987, **32**(2) 195
252. F Richard, M Buda, F Legay *J. Neurochem.*, 1988, **51**(4) 1140
253. J Vachtenheim, J Duchon, B Matous *Compar. Biochem. Physiol.*, 1987, **B-87**(4) 709
254. T Suganuma, H Muramatsu, F Murata, T Muramatsu *J. Biochem. (Tokyo)*, 1987, **102**(3) 665
255. T G Chappell, G Warren *J. Cell Biol.*, 1989, **109**(6), Part 1, 2693
256. C Niehrs, W B Huttner *EMBO J.*, 1990, **9**(1) 35
257. E Brandan, C B Hirschberg *J. Biol. Chem.*, 1988, **263**(5) 2417
258. R J Chalifour, R G Spiro *J. Biol. Chem.*, 1988, **263**(30) 15 673
259. R Majumder, L D Jayanthi, A S Balasubramanian *Indian J. Biochem. Biophys.*, 1988, **25**(4) 303
260. W L Lowe, D LeRoith *Biochem. Biophys. Res. Commun.*, 1986, **134**(2) 532
261. N Cheng, N Sahyoun *J.Biol. Chem.*, 1990, **265**(5) 2417
262. M Martin, D H Lovett, K Resch *Biol. Chem. Hoppe-Seyler*, 1985, **366**(9) 822
263. A Molla, J G Demaille *Biochemistry*, 1986, **25**(1) 3415
264. J E Peterson, J S-A Larew, D J Graves *J. Biol. Chem.*, 1990, **265**(28) 17 062
265. R N Kurl, S C Holmes, E Verney, H Sidransky *Biochemistry*, 1988, **27**(25) 8974
266. S Natsuka, M Himeno, S Hase, et al. *J. Biochem. (Tokyo)*, 1988, **103**(6) 986
267. T Harano, T Miyata, S Lee, et al. *J. Biochem. (Tokyo)*, 1988, **103**(1) 149
268. C Psachoulia, K Bourtzis, V J Marmaras *Arch. Insect. Biochem. Physiol.*, 1989, **11**(4) 217
269. B-M Loffler, H Kunze *Biochem. Intern.*, 1989, **18**(1) 129
270. B-M Loffler, H Kunze *Biochim. Biophys. Acta*, 1989, **1003**(3) 225
271. J L Avila, D Hernandez-Morales, M A Polegre, J Convit, *Comp. Biochem. Physiol.*, 1989, **94**(2) 335
272. J B Sacci, T A Campbell, M Gottlieb *Exp. Parasitol.*, 1990, **71**(2) 158
273. Y Naito, J M Lowenstein *Biochemistry*, 1981, **20**(18) 5188
274. M L Hir, R Gandhi, U C Dubach *Enzyme*, 1989, **41**(2) 87
275. C Peeters, M De Wolf, G Dessel, et al. *Int. J. Biochem.*, 1988, **20**(4) 409
276. S Kanaya, T Uchida *J. Biochem. (Tokyo)*, 1981, **90**(2) 473
277. G Reddy, V Shankar *Appl. Biochem. Biotechnol.*, 1989, **22**(3) 237
278. L Zou, N Kojima, M Kito, K Yagi *Biotechnol. Appl. Biochem.*, 1989, **11**(2) 217
279. T Yano, I Funakoshi, Y Yamashina *J. Biochem. (Tokyo)*, 1985, **98**(4) 1097
280. S-L Sano, Y Matsuda, H Nakagawa *Eur. J. Biochem.*, 1988, **171**(1–2) 231
281. A Tsuji, Y Suzuki *Biochem. Biophys. Res. Commun.*, 1988, **151**(3) 1358
282. S Saxena, K Shailubhai, B Dong-Yu, I K Vijay *Biochem. J.*, 1987, **247**(3) 563
283. G P Kaushal, I Pastuszak, K-I Hatanaka, A D Elbein *J. Biol. Chem.*, 1990, **265**(27) 16 271
284. W T Forsee, C F Palmer, J S Schutzbach *J. Biol. Chem.*, 1989, **264**(7) 3869
285. L Faye, B Mouatassim, A Ghorbel *Plant Physiol.*, 1986, **80**(1) 27
286. A P Jahagirdar, R G H Downer, T Viswanatha *Insect Biochem.*, 1990, **20**(5) 511
287. K Yokota, Y Takesue *J. Biochem. (Tokyo)*, 1988, **103**(1) 132
288. H Y Naim, E E Sterchi, M J Lentze *J. Biol. Chem.*, 1988, **263**(15) 7242
289. A Godknecht, T G Honegger *Dev. Biol.*, 1991, **143**(2) 398
290. S Hartel, C Hanski, W Kreisel, et al. *Biochim. Biophys. Acta*, 1987, **924**(3) 543
291. D Josic, K Zeilinger, Y-P Lim, et al. *J. Chromatogr.*, 1989, **484** 327
292. Y Ito, S Mizutani, O Kurauchi, et al. *Enzyme*, 1989, **42**(1) 8
293. R A Skidgel, R M Davis, F Tan *J. Biol. Chem.*, 1989, **264**(4) 2236
294. H See, R A F Reithmeier *Biochem. J.*, 1990, **271**(1) 147
295. T Miyakawa, M Kaji, Y K Jeong, et al. *J. Bacteriol.*, 1987, **169**(4) 1626
296. E P Wright, E R M Amin *J. Bacteriol.*, 1989, **67**(9) 525
297. E C Vega-SaenzadeMiera, B Rubalcava *Biochem. Biophys. Res. Commun.*, 1988, **156**(1) 30
298. A Aoyama, W-T Chen, *Proc. Natl. Acad. Sci. USA*, 1990, **87**(21) 8296
299. J Oizumi, V Hayakawa *Biochem. J.*, 1990, **266**(2) 427
300. D J Kim, S M Byun *Biochim. Biophys. Acta*, 1990, **1040**(1) 12
301. M De Wolf, A Lagron, H J Hilderson, et al. *Investigation of Membrane-located Receptors* (Edited by E Reid, et al.), Plenum Press, New York, 1984, p. 233
302. M Akayama, H Nakada, K Omori, et al. *Cell Struct. Funct.*, 1986, **11**(3) 259
303. K Omori, S Takemura, K Omori, et al. *J. Biochem. (Tokyo)*, 1983, **94**(6) 1857
304. C T Okamoto, J M Karpilow, A Smolka, J G Forte *Biochim. Biophys. Acta*, 1990, **1037**(3) 360
305. S Gllor, H Antonicek, K J Sweadner, et al., *Experientia*, 1990, **46** Abstracts, 7
306. T C Kirley *J. Biol. Chem.*, 1988, **263**(25) 12 682

307. D M Lieberman, R A F Reithmeier, V Ling, et al. *Biochem. Biophys. Res. Commun.*, 1989, **162**(1) 244
308. H Hamada, T Tsuruo *J. Biol. Chem.*, 1988, **263**(3) 1454
309. H Hamada, T Tsuruo *Cancer Res.*, 1988, **48**(17) 4926

309a. S K Yamagata, K Noremberg, S M Parsons *J. Neurochem.*, 1989, **53**(5) 1345
310. E E Fesenko, V I Novoselov, M F Bystrova *FEBS Lett.*, 1987, **219**(1) 224
311. R T Fernley, J P Coghlan, R D Wright *Biochem. J.*, 1988, **249** 201
312. M D Smigel *J. Biol. Chem.*, 1986, **261**(4) 1976
313. F V Zaitseva, V M Petrov, E Yu Elistratova, B M Lipkin *Biol. Membrany*, 1988, **5**(11) 1125
314. R Takayanagi, R M Snajdar, T Imada, et al. *Biochem. Biophys. Res. Commun.*, 1987, **144**(1) 244
315. S A Waldman, L Y Chang, F Murad *Prep. Biochem.*, 1985, **15**(3) 103
316. C S Ramarao, D L Garbers *J. Biol. Chem.*, 1988, **263**(3) 1524
317. T Brody, J W Suttie *Biochim. Biophys. Acta*, 1987, **923**(1) 1
318. M G Scher, W G Resneck, R J Bloch *Anal. Biochem.*, 1989, **177**(1) 168
319. E R Jakoi, P E Ross, H P Ting-Beall, et al. *J. Biol. Chem.*, 1987, **262**(3) 1300
320. J Shemer, D Le Roith *Neuropeptides*, 1987, **9**(1) 1
321. Y-J Zhao, M Belew *Biotechnol. Appl. Biochem.*, 1986, **8**(1) 75
322. J C Tercero, T Diaz-Maurino *Anal. Biochem.*, 1988, **174**(1) 128
323. T Osawa *Pure Appl. Chem.*, 1989, **61**(7) 1283
324. J T Gallagher *HPLC of Macromolecules* (Edited by R W A Oliver), IRL Press, Oxford, 1989, p. 209
325. J C Paulson *Trends Biochem. Sci.*, 1989, **14**(7) 272
326. T W Rademacher, R B Parekh, R A Dwek *Ann. Rev. Biochem.*, 1988, **57** 785
327. J-P Feugeas, D Neel, A A Pavia, et al. *Biochim. Biophys. Acta*, 1990, **1030**(1) 60
328. Y Shirayoshi, A Nose, K Iwasaki, M Takeichi *Cell Struct. Funct.*, 1986, **11**(3) 245
329. A M Wu, S Sugii *Molecular Immunology of Complex Carbohydrates* (Edited by A M Wu, L C Adams), Plenum Press, New York, 1988, p. 205
330. T Osawa *Molecular Immunology of Complex Carbohydrates* (Edited by A M Wu, L C Adams), Plenum Press, New York, 1988, p. 83
331. H Lis, N Sharon *Lectins: Properties, Functions, and Applications in Biology and Medicine* (Edited by I E Liener, et al.), Acad. Press, Orlando, 1986, p. 293
332. A Pallant, M Fukuda, J G Frelinger *Eur. J. Immunol.*, 1990, **20**(7) 1423
333. A Aruffo, I Stamenkovic, M Melnick, C B Underhill *Cell*, 1990, **61**(7) 1303
334. S D Wright, R A Ramos, P S Tobias, et al. *Nature (London)*, 1990, **249**(4975) 1431
335. J E Hambor, D R Kaplan, M L Tykocinski *J. Immunol.*, 1990, **145**(6) 1646
336. T Majima, N Minegishi, R Nagatomi, et al. *J. Immunol.*, 1990, **145**(6) 1694
337. O Noren, E Dabelsteen, P E Hoyer, et al. *FEBS Lett.*, 1989, **259**(1) 107
338. N K Tonks, H Charbonneau, C D Diltz, et al. *Biochemistry*, 1988, **27**(24) 8695
339. M Massaia, L Perrin, A Biachi, et al. *J. Immunol.*, 1990, **145**(6) 1664
340. S-I Sano, S Ito, M Nakamura, H Nakagawa *J. Neurochem.*, 1990, **55**(4) 1252
341. U Stochaj, H Richter, H G Mannherz *Eur. J. Cell Biol.*, 1990, **51**(2) 335
342. J G J Van de Winkel, M Jansze, P J A Capel *J. Immunol.*, 1990, **145**(6) 1890
343. E Van Obberghen, H Gazzano, A Kowalski, et al. *Biochem. Soc. Trans.*, 1984, **12**(5) 762
344. J-J Feige, E M Chambaz *Biochimie*, 1987, **69**(4) 379
345. A K Srivastava, J-L Chiasson *Biochim. Biophys. Acta*, 1989, **996**(1–2) 13
346. K Panneerselvam, S Ramamoorthy, A S Balasubramanian *Biochem. Biophys. Res. Commun.*, 1987, **147**(3) 927
347. B Datta, M K Ray, D Chakrabarti, et al. *J. Biol. Chem.*, 1989, **264**(34) 20 620
348. N P Korolev *Itogi Nauki i Tekhniki. Ser. "Obshchie Problemy Fiziko-Khimicheskoi Biologii"* (Results of Science and Techniques. General Problems in Physicochemical Biology Series), Vol. 1, Izd. VINITI, Moscow, 1984, 351 pp
349. F-T Liu *Crit. Rev. Immunol.*, 1990, **10**(3) 289
350. M H Siegelman, M Van De Rijn, I L Weissman *Science*, 1989, **243**(4895) 1165
351. M Tavassoli, C L Hardy *Blood*, 1990, **76**(6) 1059
352. S T Pals, E Horst, G J Ossekoppele *Blood*, 1989, **73**(4) 885
353. Y Imai, D D True, M S Singer, S D Rosen *J. Cell. Biol.*, 1990, **111**(3) 1225
354. S D Rosen, M S Singer, T A Yednock, L M Stoolman *Science*, 1985, **228** 1005
355. T Matsuoka, M Tavassoli *J. Biol. Chem.*, 1989, **264**(34) 20 193
356. P E Di Corleto, C A De La Motte *J. Immunol.*, 1989, **143**(11) 3666
357. J L Marx *Science*, 1989, **243**(4895) 1144
358. M P Bevilacqua, S Stengelin, M A Gimbrone, B Seed *Science*, 1989, **243**(4895) 1160
359. J G Laing, M W Robertson, C A Gritzmacher, J L Wong *J. Biol. Chem.*, 1989, **264**(4) 1907
360. K Ikuta, M Takami, C W Kim, et al. *Proc. Natl. Acad. Sci. USA*, 1987, **84**(3) 819
361. H-J Woo, L M Shaw, J M Messier, A M Mercurio *J. Biol. Chem.*, 1990, **265**(13) 7097
362. R G MacDonald *Cytotechnology*, 1989, **2**(4) 287
363. R G MacDonald, S R Pfeffer, L Coussens, et al. *Science*, 1988, **239**(4844) 1134
364. W Kiess, G D Blickenstaff, M M Sklar, et al. *J. Biol. Chem.*, 1988, **263**(19) 9339
365. A Hinek, D S Wrenn, R P Mecham, S H Barondes *Science*, 1988, **239**(4847) 1539
366. L E Grosso, R P Mechan *Glycoconjugate J.*, 1989, **6**(3) 384
367. C D Ross, J A Cain, P J Lachmann *J. Immunol.*, 1985, **134**(5) 3307
368. J K Czop, K F Austen *J. Immunol.*, 1985, **134**(4) 2588
369. J P Zanetta, A Bingen, M Dontenwill-Kieffer, et al. *Cell Mol. Biol.*, 1987, **33**(4) 423
370. R G Langdon, V P Holman *Biochim. Biophys. Acta*, 1988, **945**(1) 23
371. A B Pawagi, C M Deber *Biochem. Biophys. Res. Commun.*, 1987, **145**(3) 1087
372. A Carruthers, A L Helgerson *Biochemistry*, 1989, **28**(21) 8337

373. E L Saenko, V V Basevich, A I Yaropolov *Biokhimiya,* 1988, **53**(8) 1310
374. E L Saenko, V V Basevich *Uch. Zap. Tartuskogo Un-ta. Vyp. 870. Izuchenie i Primenenie Lektinov* (Scientific Reports of Tartu University. Part 870. Study and Application of Lectins), Vol. 2, Izd. Tartuskogo Un-ta (Tartu University Press), Tartu, 1989, p. 48
375. M A Jabbar, D P Nayak *J. Virol.,* 1990, **64**(12) 6297
376. D Mirelman (Editor) *Microbial Lectins and Agglutinins,* J. Wiley and Sons, New York, 1986, 443pp
377. J I Ravdin, T F H G Jackson, W A Petri *Infect. Immun.,* 1990, **162**(3) 768
378. G Muthukumar, K W Nickerson *Appl. Environm. Microbiol.,* 1987, **53**(11) 2650
379. N Kawasaki, T Kawasaki, I Yamashita *J. Biochem. (Tokyo),* 1989, **106**(3) 483
380. R P Mecham, A Hinek, G L Griffin, et al. *J. Biol. Chem.,* 1989, **264**(28) 16 652
381. D K Strickland, J D Ashcom, S Williams, et al. *J. Biol. Chem.,* 1990, **265**(29) 17 401
382. I L Goldstein, R D Poretz *Lectins: Properties, Functions, and Applications in Biology and Medicine* (Edited by I E Leiner, et al.), Acad. Press, Orlando, 1986, p. 35
383. V M Lakhtin *Lectins: Biology, Biochemistry, Clinical Biochemistry* (Edited by J Kocourek, et al.), Sigma Chemical Company, St. Louis, USA, 1990, p. 417
384. B V Torres, D F Smith *Anal. Biochem.,* 1988, **170**(1) 209
385. A D Lutsik, E S Detyuk, M D Lutsik *Lektiny v Gistokhimii* (Lectins in Histochemistry), Vishcha Shk., Lvov, 1989, 144 pp
386. U Schumacher, P Trudrung, M Ruhnke, et al. *Histochem. J.,* 1990, **22**(8) 433
387. M D Lutsik *Author's Summary of Doctoral Dissertation in Biological Sciences,* Institute of Biochemistry, Academy of Sciences of the Ukrainian SSR, Kiev, 1989, 42 pp
388. K Graham, K Keller, J Ezzell, R Doyle *Eur. J. Clin. Microb.,* 1984, **3**(3) 210
389. S R Martinod *Prep. Biochem.,* 1985, **15**(3) 171
390. C Geveriaux, F Loor *J. Immunol. Meth.,* 1987, **104**(1–2) 173
391. H G C J M Hendriks, J F J G Koninkx, M Draijer, et al., *Biochim. Biophys. Acta,* 1987, **905**(2) 371
392. J J Distler, R Patel, G W Jourdian *Anal. Biochem.,* 1987, **166**(1) 65
393. I Flemming, C Flemming, K Laube, et al. *Proc. XI Int. Lectin Meet. (June 4–9, 1989, Tartu-Tallinn), Abstracts,* p. 18
394. M Slifkin, R Doyle *Clin. Microbiol. Rev.,* 1990, **3**(3) 197
395. V M Lakhtin, N I Guseva, N V Fedurkina, et al. *Zh. Mikrobiol. Epidemiol. Immunobiol.,* 1985, (1) 30
396. V I Brilis, M E Mikel'saar, R Kh Kasesalu, et al. *Uch. Zap. Tartuskogo Un-ta. Vyp. 870. Izuchenie i Primenenie Lektinov* (Scientific Reports of Tartu University. Part 870. Study and Application of Lectins), Vol. 2, Izd. Tartuskogo Un-ta, (Tartu University Press), Tartu, 1989, p. 230
397. I Flemming, C Flemming *Proc. X Int. Lectin Meet. (July 3–8, 1988, Prague), Abstract Book,* p. 31
398. V M Lakhtin, Z M Yakovleva *Izv. Akad. Nauk SSSR, Ser. Biol.,* 1987, (5) 792
399. N V Lyubimova, K P Shuvalova, V N Lakhtin, M L Davletshina *Uch. Zap. Tartuskogo Un-ta. Vyp. 870. Izuchenie i Primenenie Lektinov* (Scientific Reports of Tartu University. Part 870. Study and Application of Lectins), Vol. 2, Izd. Tartuskogo Un-ta, (Tartu University Press), Tartu, 1989, p. 62
400. N N Nutsubidze, V M Lakhtin, V A Demina, et al. *Prikl. Biokhimiya Mikrobiologiya,* 1991, **27**(2) 274
401. D J Klionsky, P K Herman, S D Emr *Microb. Rev.,* 1990, **54**(3) 266
402. S Medda, N Das, B K Bachhawat, et al. *Biotechnol. Appl. Biochem.,* 1990, **12**(5) 537
403. N Kawaguchi, T Ohmori, Y Takeshita, et al. *Biochem. Biophys. Res. Commun.,* 1986, **140**(1) 350
404. R J Harris, C K Leonard, A W Guzzetta, M W Spellman *Biochemistry,* 1991, **30**(9) 2311
405. S Shibata, T Takeda, Y Natori *J. Biol. Chem.,* 1988, **263**(25) 12 483
406. M D Lutsik, E N Panasyuk, A D Lutsik *Lektiny* (Lectins), Vishcha Shk., Lvov, 1981, 156 pp