VERTEBRATE LECTINS

Comparison of Properties of β-Galactoside-Binding Lectins
from Tissues of Calf and Chicken

EVE BARAK BRILES, WALTER GREGORY, PAUL FLETCHER,
and STUART KORNFELD

From the Departments of Medicine and Biochemistry, Washington University School of Medicine,
St. Louis, Missouri 63110, and the Section of Cell Biology, Yale University School of Medicine, New
Haven, Connecticut 06510

ABSTRACT

β-Galactoside-binding lectins were isolated from various calf tissues and from
chicken hearts by affinity chromatography on asialofetuin-Sepharose, and were
compared with respect to biochemical characteristics, binding properties, anti-
genic cross-reactivity, and cellular localization. The lectins are all thiol group-
requiring, divalent cation-independent dimers, of apparent monomer mol wt
12,000 (calf lectins) or 13,000 (chicken lectin), and acidic pI. The calf lectins
appear essentially identical by dodecyl sulfate-polyacrylamide gel electrophoresis,
isolectric focusing, amino acid composition, and radioimmunoassay, while the
chicken lectin is distinctly different by these criteria. However, all of the lectins
competed for the same binding sites on rabbit erythrocytes, and could be inhibited
by the same saccharide haptenes (notably lactose and thiodigalactoside). Immuno-
fluorescence studies on several cultured cell lines revealed that the bovine and
chicken lectins had primarily an intracellular cytoplasmic localization. The β-
galactoside-binding lectins of vertebrates appear to be species-specific rather than
tissue-specific.

KEY WORDS lectins - vertebrate lectins - β-
galactoside-binding lectins - affinity chromatography

Although lectins from various plant and inverte-
brate sources have been known for many years
(for a comprehensive list, see reference 23), their
presence in vertebrate tissues has been investi-
gated only recently. Ashwell and Morell (3) have
described a hepatic binding protein which has
been implicated in the clearance of glycoproteins
from plasma; in mammals, this binding protein is
a β-galactoside-specific, integral membrane pro-
tein of large molecular weight, which can be
solubilized by detergents but not by hapten sac-
charides and which requires divalent cations for
binding activity. Subsequently, hepatic and retic-
uloendothelial cell-binding proteins which recog-
nize mannose, N-acetylglucosamine, and fucose
have been detected (1, 4, 6, 8, 18, 20, 29, 31,
32). A “lectin” from platelet plasma membranes
has been described by Gartner et al. (16) which is
inhibitable by free amino sugars and amino acids;
the large external transformation-sensitive
(LETS) protein or fibronectin also agglutinates
eythrocytes and is inhibitable by amines (35). In
addition to these membrane-bound lectins, several
Isolation of Lectins

Biochemical Characterization of Lectins

Sepharose, essentially as described by de Waard et al.

MATERIALS AND METHODS

was loaded onto a second column.

From adult chicken hearts. The calf lectins are all

similar to, yet distinctly from, the chicken

divalent cations for agglutinating activity. Al-

though some investigators have speculated that

these lectins might play a role in the specific

embryonic development (11, 15, 21, 28), the physio-

logic role of these lectins in vivo has yet to be

established.

In the present report, we compare the β-galac-

toside-specific lectins from various calf tissues and

from adult chicken hearts. The calf lectins are all

development (11, 15, 21, 28), the physiologic role of these lectins in vivo has yet to be

established.

MATERIALS AND METHODS

Isolation of Lectins

Fresh calf organs (liver, spleen, heart, thymus) and

chicken hearts were obtained from local slaughter-

houses. Soluble and particulate-associated lectins were

isolated by affinity chromatography on asialofetuin-

Sepharose, essentially as described by de Waard et al.

(12), with the following modifications: (a) The buffer

used throughout, SPB, contained 0.14 M NaCl, 0.02 M

KPO₄, pH 7.4, 8.5% wt/vol sucrose, 0.002% wt/vol

bromphenol blue, 1% wt/vol SDS, 35 mM 2-mercaptop-

ethanol, and 75 mM lactose) were incubated at 100°C

for 5 min before loading. Electrophoresis was carried

out at 45 mA (1.7 W) for 11 h. The gel was stained with

0.3% Coomassie Brilliant Blue R in 50% vol/vol meth-

anol containing 12% wt/vol TCA, and destained with

7% vol/vol acetic acid in 10% vol/vol methanol.

Gel permeation chromatography was performed on a

Bio-Gel P-60 (100- to 200-mesh) column (0.9 × 120

cm; Bio-Rad Laboratories, Richmond, Calif.), with SPB

as the eluting buffer; the column was calibrated using

ymyoglobin, cytochrome c, and ovalbumin as standards.

Slab isoelectric focusing was performed on an LKB

horizontal slab apparatus (LKB Instruments, Inc., Rock-

ville, Md.), using pre-poured pH 3.5–9.5 gels (LKB

PAG plate) which were sprayed lightly with 1.5 M 2-

mercaptopethanol just before use. The gel was fixed in

15% aqueous TCA for ½ h, rinsed with water, and then

stained with 0.09% Coomassie Brilliant Blue R in 8.8%

methanol-8% acetic acid, and destained with 10% acetic

acid-10% methanol. The pH gradient was determined

by measuring the pH of 1-cm segments cut from an edge

of the gel before fixation, after elution into a minimal

volume of water.

Protein was quantitated either by the method of Lowry et al. (24) on samples which were dialyzed free of

2-mercaptopethanol, or by the method of Bradford (7),

using bovine serum albumin (BSA) as the standard, or

by determining the optical absorption at 280 nm.

Radioiodination of Lectins

In early studies, the lectins were labeled with ¹²⁵I,

after dialysis to remove the mercaptopethanol, by the

chloramine-T method (17), using a 10-s exposure to the

chloramine-T. We observed, however, that this tech-

nique resulted in an unstable association of the label

with the lectin. The degree of instability tended to vary

widely from batch to batch, and some occasional prepa-

rations were actually stable over long periods of storage

time. To circumvent this problem, the ¹²⁵I-lectins were

either repurified by affinity chromatography immediately

before use, or the amount of ¹²⁵I label actually

bound to the lectin was determined by precipitation with

10% TCA at the time of the experiment. Alternatively,

the lectins could be conveniently and stably radioiodi-

nated with ¹²⁵I-N-succinimidyl-3(4-hydroxyphenyl) pro-

pionate as described by Bolton and Hunter (5), and this

became our method of choice for most of the experi-

ments described herein.

Lectin Binding to Rabbit Red Cells

Binding assays were performed in 1.5-ml plastic mi-
Radioimmunoassay

Rabbit antisera to soluble calf heart lectin and to purified chicken heart lectin were prepared as previously described (12). The initial immunoradiometric reactions were performed in 250 μl of SPB containing 150 μg of BSA, 30 μmol of lactose, competing agglutinins in varying amounts, 125I-labeled lectin probe (either 0.8 μg of calf heart particle-associated lectin, 0.16 μg of calf spleen particulate lectin, or 0.12 μg of chicken heart lectin), and either an amount of antiserum predetermined to complex 50% of the labeled probe in the absence of competitors or an equivalent volume of normal rabbit serum (control). The 125I probe was always added last. After incubation at 37°C for 2 h, the reaction mixtures were chilled, and 1.5 ml of ice-cold 25% wt/vol polyethylene glycol 6,000 in phosphate-buffered saline was added, to induce precipitation of antibody-antigen complexes as described by Creighton et al. (9). After overnight incubation at 0°C, precipitates were collected by centrifugation (Sorvall SS-34 rotor, DuPont Instruments, Wilmington, Del., 12,000 rpm x 30 min), and the precipitated radioactivity was determined in a Beckman gamma counter (Beckman Instruments, Inc., Fullerton, Calif.). In some experiments, up to 10 μl of additional normal rabbit serum was added to each assay tube as “carrier” to facilitate collection of precipitates.

Immunofluorescence Microscopy

Fluorescent antibody was prepared as follows: 800 μl of fluorescein isothiocyanate-conjugated goat anti-rabbit IgG (Gateway Immunosera Co., Cahokia, Ill.) was mixed with an equal volume of 50% washed packed mouse L-cells in normal goat serum (Gateway Immunosera Co.), incubated at 0°C for 15 min, and then centrifuged at 500 g for 5 min at 5°C. The supernate was reserved, and the L-cell pellet was resuspended with 2 ml of phosphate-buffered saline and recentrifuged. The two supernates were pooled, clarified by centrifugation for 15 min at 12,000 g at 5°C, and stored in 0.5-ml aliquots at -20°C.

Primary chick embryo fibroblasts were a gift from Dr. S. Schlesinger (Washington University), and were used at the second passage; primary bovine embryonic and postnatal kidney cells were purchased from Flow Laboratories (Rockville, Md.) and used at the third passage. The cells were seeded onto nitrous acid-precleaned, sterile glass microscope slides in 150-mm culture dishes, and cultured for 2 d before use in minimal essential medium-alpha medium without nucleosides (Flow Laboratories) supplemented with 50 U/ml penicillin, 50 μg/ml streptomycin, and 10% heat-inactivated fetal calf serum (Grand Island Biological Co., Grand Island, N.Y.).

For studies using intact cells, the slides were washed four times with Dulbecco's phosphate-buffered saline (13) containing 0.05% NaN₃, and drained. The area to be treated was marked by scoring a small circle on the underside of the slide with a diamond pencil. Then 50 μl of normal goat serum (NGS) diluted 1:20 was dropped onto the slide. 5 min later, 90 μl of either normal rabbit serum or antiserum, diluted 1:10, were added. After 30 min at 20°C, the slides were washed five times with buffer, drained, and 30 μl of 1:20 NGS was added. 5 min later, 90 μl of fluorescent antibody reagent (see above), diluted 1:8, was added. After 30 min at 20°C, the slides were washed five times, fixed in 2% paraformaldehyde in buffer at 20°C for 30 min, dehydrated in 90% ethanol for 15 min, and air-dried. Slides were viewed under 80% glycerine, 20% 0.2 M potassium borate, pH 8.5, in a Leitz Ortholux fluorescence microscope.

For studies using fixed cells, the slides were washed four times with buffer, fixed in 2% paraformaldehyde in buffer for 4 h, dehydrated with several changes of 90%...
ethanol, and air-dried. The dried slides were then re- 
wetted with 100 μl of 1:20 NGS, and then treated 
sequentially with serum and fluorescent antibody re-
agent, as described above.

**Amino Acid Analyses**

The amino acid composition was determined from 
duplicate protein samples hydrolyzed in 6 N HCl with 
0.5% phenol added for tyrosine protection (2) and 
individually sealed under vacuum as described by Moore 
and Stein (27). Hydrolysis was carried out at 110°C for 
20, 40, 72, and 120 h as described by Crestfield et al. 
(10) for extrapolating recoveries of individual amino 
acids to zero time for those labile amino acids, while 
those difficult to release were expressed as maximum 
values. Half/cystine content of these proteins was deter-
mimed as cystic acid according to the method of Moore 
(26). Amino acid analysis was carried out on the Durrum 
D-500 Amino Acid Analyzer (Durrum Instrument 
Corp., Sunnyvale, Calif.) modified for high sensitivity 
with the Mark II Data Analysis System.

**RESULTS**

**Lectin Yield and Characterization**

After homogenization of the various tissues, 
either all or substantially most of the lectin activity 
was recovered in the sedimentable fraction. The 
lectin could be released in soluble form by extrac-
tion with lactose, suggesting that the lectin was 
bound to the particulate material by its saccharide-
binding sites.

The yield of lectin per kilogram wet weight of 
tissue varied considerably among the tissues sam-
ples, as indicated in Table I; spleen was a particu-
larly rich source of the lectin.

The isolated lectins were judged to be highly 
purified on the basis of Coomassie Blue-stained 
alkaline disc polyacrylamide gel electrophoresis 
(not shown) and SDS-polyacrylamide slab gel 
electrophoresis (Fig. 1), except calf thymus lectin,

| Tissue          | Particle-associated (lactose-extractable) | Soluble         | Total |
|-----------------|------------------------------------------|-----------------|-------|
| Calf spleen     | 43                                       | 5               | 48    |
| Calf thymus     | 5.5                                      | none detected   | 5.5   |
| Calf liver      | 2.2                                      | none detected   | 2.2   |
| Calf heart      | 13                                       | 3               | 16    |
| Chicken heart   | 4.2                                      | none detected   | 4.2   |

* Yields are based on material specifically eluted from asialofetuin-Sepharose affinity columns, and are 
  expressed as milligrams of lectin protein per kilogram wet weight of tissue.

**TABLE I**

**Yield of Galactose-Binding Lectins from Various Tissues**

**FIGURE 1** SDS slab gel electrophoresis. Electrophoresis was performed as described in Materials and Methods. Lane 1, BSA; 2, ovalbumin; 3, myoglobin; 4, cytochrome c. Lanes 5, 6, 12, and 13, calf spleen soluble lectin; 7, calf spleen particle-associated lectin; 8, calf heart soluble lectin; 9, calf heart particle-associated lectin; 10, calf thymus lectin; 11, chicken heart lectin. Inset: Calibration curve used for estimating lectin molecular weights.

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which gave several bands. The subunit molecular weights of the calf lectins were found to be virtually identical (~12,000 daltons), while that of the chicken heart lectin was clearly larger (~13,000 daltons) (Fig. 1). Gel filtration on Bio-Gel P60 gave a single peak of agglutinin activity, corresponding to apparent mol wt of 24,000 in the case of the calf lectins and 26,000 in the case of the chicken heart lectin, suggesting that in each case the agglutinin is a dimer. The major protein peak on Bio-Gel P60 corresponded with the agglutinin peak, while a small, variable amount of protein eluted at a position corresponding to 12,000 in the case of the calf lectins and 13,000 in the case of the chicken lectin; the minor peak persisted after affinity-chromatography purification.

Analysis by isoelectric focusing revealed that both the soluble and lactose-extractable calf lectins from spleen and heart gave identical doublet (or, in other experiments, triplet) bands at pI ~4.8 (Fig. 2). The chicken heart lectin did not behave reproducibly on isoelectric focusing gels, but always focused in the acidic range (not shown).

The amino acid compositions of the purified lectins from chicken heart, calf heart, and calf spleen (lactose-extractable) are listed in Table II. The calf lectin compositions are remarkably similar to each other, while the chicken lectin is significantly different.

**Antigenic Relationships among the Lectins**

In a previous communication, de Waard et al. (12) demonstrated that soluble and particle-associated lectins from calf heart and lung all gave lines of identity on immunodiffusion plates. Similarly, calf spleen and thymus lectins gave lines of identity with the calf heart lectins on immunodiffusion plates (not shown), while the chicken heart lectin gave no band at all with anti-calf heart lectin serum. To further examine the antigenic relationships among these lectins, we performed radioimmunoassays. The results, illustrated in Fig. 3, demonstrate strong antigenic cross-reactivity among the calf lectins from heart, spleen, and liver (thymus was not tested). Since the anti-calf lectin serum used in these assays was directed against soluble heart lectin while the probes used were either spleen or heart particle-associated lectin, and since each antiserum was derived from a single rabbit, it is possible that some antigenic distinctions exist between the soluble and particle-associated forms of the lectin which our assays failed to pick up. Chicken heart lectin was distinctly different from the calf lectins. When chicken heart lectin was tested using anti-calf lectin serum and calf lectin probe, it was very weakly cross-reactive, while the reciprocal assay...
TABLE II
Amino Acid Compositions of Lactose-Extractable Lectins*

|       | Calf spleen | Calf heart | Chicken heart |
|-------|-------------|------------|---------------|
| Asp + AspNH₂ | 15.19       | 13.77      | 11.07         |
| Thr    | 2.04        | 2.18       | 5.15          |
| Ser    | 4.88        | 4.77       | 6.70          |
| Glu + GluNH₂ | 8.74       | 8.61       | 10.77         |
| Pro    | 4.39        | 6.03       | 5.63          |
| Gly    | 8.15        | 8.95       | 9.10          |
| Ala    | 9.43        | 9.54       | 3.93          |
| Cys 1/2 | 4.52        | 3.89       | 2.29          |
| Val    | 6.65        | 6.50       | 7.77          |
| Met    | 0.00        | 0.15       | 1.45          |
| Ile    | 3.39        | 3.51       | 3.80          |
| Leu    | 10.16       | 9.62       | 7.68          |
| Tyr    | 1.51        | 2.03       | 0.82          |
| Phe    | 7.17        | 6.79       | 7.48          |
| His    | 1.48        | 1.59       | 3.41          |
| Lys    | 5.79        | 5.50       | 6.65          |
| Arg    | 3.62        | 3.67       | 3.20          |
| Try†   | 2.89†       | 2.90†      | 3.08‡         |

* Expressed as No. of residues per 100 residues.
† Tryptophan values estimated.

using anti-chicken heart lectin serum and chicken heart lectin probe showed no cross-reactivity at all with calf lectins. Ricin, a plant lectin with similar saccharide-binding specificities (see below), showed no antigenic cross-reactivity with the vertebrate lectins in either assay system. Furthermore, neither lectin activity nor antigenically cross-reactive material could be found in fetal calf serum.

Lectin Binding to Rabbit Red Cells

Our binding studies demonstrate that the calf and chicken lectins have remarkably similar binding specificities. The apparent number of lectin-binding sites per rabbit erythrocyte for chicken heart lectin, calf spleen particle-associated lectin, calf heart particle-associated lectin, and calf heart soluble lectin is $2.5 \times 10^6$. As can be seen from Fig. 4, the various calf lectins and chicken heart lectin all compete with each other and with ricin for the same binding sites, but not with several other plant lectins tested. Furthermore, the order of efficacy of saccharide inhibitors is the same for both calf and chicken lectins (thiodigalactoside > lactose > $\alpha$-methylgalactoside > $\beta$-methylgalactoside) as shown in Fig. 5. We have no explanation at present for the seemingly contradictory observation that $\alpha$-methylgalactoside is a more potent inhibitor than $\beta$-methylgalactoside, while lactose and thiodigalactoside, both of which contain only $\beta$-linked galactosyl residues, are even more potent. Interestingly, however, the same

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FIGURE 4 Inhibition of 125I-lectin binding to rabbit cells by competing lectins. Inhibition assays were performed as described in Materials and Methods. Panel A: 125I-labeled chicken heart lectin. Panel B: 125I calf spleen particle-associated lectin. ○, ricin; □, calf spleen particle-associated lectin; ●, chicken heart lectin; ▲, *Agaricus bisporus* agglutinin; ●, *Phaseolus vulgaris* phytohemagglutinin E; ■, soybean agglutinin. Panel A only: ○, calf heart particle-associated lectin. Panel B only: ○, Concanavalin A.

FIGURE 5 Inhibition of 125I-lectin binding to rabbit red cells by saccharides. Inhibition assays were performed as described in Materials and Methods. Panel A: 125I-labeled chicken heart lectin. Panel B: 125I-labeled calf spleen particle-associated lectin. ○, thiodigalactoside; □, lactose; △, α-methylgalactoside; ○, β-methylgalactoside.

The order of efficacy has been described by Nowak et al. (28) for a lactose-extractable lectin from embryonic chick muscle tissue, and may thus be idiosyncratic for vertebrate galactoside-binding lectins in general.

Despite their remarkable similarities with respect to binding specificity, the calf and chicken lectins differ considerably in their apparent binding avidities. The observed $K_d$ of chicken heart lectin for rabbit erythrocytes was $4 \times 10^{-9}$ M, while that of the calf lectins (heart soluble and particle-associated and spleen particle-associated) was $8 \times 10^{-7}$ M. Neither trypsinization of the rabbit red cells nor varying the method of radioiodinating the lectins seemed to have any effect on the binding parameters; however, trypsinized rabbit red cells did agglutinate much more readily, and at lower lectin concentrations, than untreated cells.

**Subcellular Localization of the Lectins**

The tremendous antigenic cross-reactivity among the lectins from the calf tissues tested (see above) suggested that the lectin is ubiquitous, and that our anti-heart lectin antisera could therefore be used as reagents for the cellular localization of the lectin by immunofluorescence without necessarily using cells of heart origin.

Preliminary experiments using calf thymocytes teased from fresh thymus tissue demonstrated that most of the lectin was cytoplasmic but that some of it was on the extracellular face of the plasma membrane. However, many of the cells ruptured during the preparation of a single-cell suspension, and so it was possible that the cell surface lectin represented intracellular lectin from ruptured cells that had secondarily bound to surface receptor sites. We therefore performed immunofluorescence studies using cultured monolayers of calf kidney and chick embryo fibroblast cells, which could easily be manipulated without damaging the cells. Typical findings with the chick embryo fibroblasts are illustrated in Fig. 6. Fluorescence was uniformly distributed throughout the cytoplasm of fixed cells (but not in the nucleus), but no fluorescence was detected in unfixed cultures. These results indicate that the lectin is located primarily in the cytoplasm. Similarly, cellular fluorescence was observed in fixed bovine embryonic and postnatal kidney cells, but not in unfixed cultures, with one exception. Sometimes thin cytoplasmic strands were observed connecting two distal bovine cells, and these thin strands showed specific immunofluorescence in unfixed cultures (data not shown).

Some antigenic cross-reactivity was observed, in that chick embryo fibroblasts treated with anti-calf heart lectin serum fluoresced more brightly than those treated with control normal rabbit serum (Fig. 6c) but less brightly than those treated with anti-chicken heart lectin serum. This was consistent with our radioimmunoassay findings (see above).

**DISCUSSION**

Since the first report by Teichberg et al. (33), of the existence of β-galactoside-binding lectins in tissues of various vertebrate species, several laboratories have investigated such proteins. Lectins
FIGURE 6 Cellular localization of lectin by immunofluorescence microscopy. These experiments were performed as described in Materials and Methods. Each field was photographed using both fluorescence optics (left) and phase contrast (right). The original magnification was 500. (a) Fixed, ethanol-treated chick embryo fibroblasts (CEFs) plus anti-chicken heart lectin serum. (b) Fixed CEFs plus normal rabbit serum (control). (c) Fixed CEFs plus anti-calf heart lectin serum. (d) Unfixed, intact CEFs plus anti-chicken heart lectin serum.

with properties remarkably similar to those of the “electrolectin” isolated from electric eel electric organs (33) have been reported from sources as diverse as embryonic chick muscle (11, 28) and calf hearts and lungs (12). The properties which these lectins all appear to share include: a stringent requirement for thiol-reducing groups; an apparent specificity for terminal β-galactose resi-
dyes such as those of thiodigalactoside or lactose, yet a paradoxically marked preference for \( \alpha \)-methylgalactoside over \( \beta \)-methylgalactoside; an acidic pI; a dimeric structure comprised of two polypeptide chains ranging in apparent molecular size from 12,000 to 17,000 daltons; and an intracellular, cytoplasmic localization.

From the work of de Waard et al. (12) and the data presented in this report, it is clear that the lectins from various tissues of the calf (heart, lung, liver, spleen, and thymus) are very closely similar, and probably identical. They exhibit the same apparent size on gel permeation chromatography and SDS-PAGE; they all have the same binding specificity; they are antigenically cross-reactive, and give lines of identity on both immunodiffusion and radioimmunoeassay; and they show identical isoelectric focusing patterns. In addition, the heart and spleen lectins have remarkably similar amino acid compositions. In contrast, the lectin from chicken hearts, although similar in general characteristics, is clearly different from the calf lectin(s). It exhibits a slightly larger molecular size, and it is antigenically distinct. These findings suggest that the vertebrate lectins are species-specific rather than tissue-specific. This is in agreement with the finding by Kobiler et al. (22) that lectins from a number of embryonic chick tissues appear identical.

Our immunofluorescence studies revealed that the lectin is present primarily in the cytoplasm of cells. The question of whether any lectin is present on cell surfaces is of interest with respect to possible functions of the lectin. We did not observe any detectable lectin at the cell surface, except under circumstances where there was a strong likelihood that lectin was released from damaged cells and subsequently bound to cell surface receptors; however, this does not exclude the possibility that some lectin is normally present on the cell surface but in amounts too low to be detected by our methods. We did, however, observe the lectin on thin cytoplasmic strands which connected distal bovine cells; the significance of this is not clear. In a similar investigation, Nowak et al. (28) demonstrated that most of the \( \beta \)-galactoside-specific lectin of chick embryo myoblasts undergoing fusion in vitro is located in the cytoplasm; however, they did observe some lectin at the cell surface. Furthermore, Gremo et al. (18) examined dissociated tectal cells for surface lectin and found that fewer than half of the cells in their preparations were positive; however, they could not exclude the possibility that some lectin had leaked out of damaged cells.

On the basis of our data, we conclude that, in the intact cell, the lectin is located predominantly intracellularly. In tissue homogenates, the bulk of the lectin is associated with particulate material (probably membranous), and can be recovered in soluble form by extraction with lactose, Triton X-100 (12), or high ionic strength (11). We believe that this is most likely an artifactual situation resulting from the release of intracellular lectin during homogenization and the subsequent binding of the lectin to receptors on the membrane fragments. We would like to note that the distinctions reported previously from this laboratory (12) between the particle-associated and soluble forms of the calf heart lectin have not been reproducible in the present study.

It has been suggested by other investigators that these lectins might play a specific role in myoblast adhesion and fusion during embryonic development (11, 15, 21, 28). However, our findings, plus the remarkable ubiquity of these lectins, lead us to believe that the lectins may be responsible for some (as yet unknown) cellular or physiological function which is not specific for any particular organ or tissue.

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