Crystallization and Characterization of the L-Arabinose-binding Protein of Escherichia coli B/r*

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SUMMARY

The L-arabinose-binding protein was purified by conventional methods from Escherichia coli cell paste and crystallized using 2-methyl-2,4-pentanediol. A molecular weight of 38,000 was determined using both ultracentrifugation and gel filtration. The amino acid composition revealed high concentrations of aspartic and glutamic residues and non-polar amino acids. The presence of 2 cysteine residues was also demonstrated.

The addition of L-arabinose did not produce changes in the circular dichroism spectra or sedimentation velocity coefficients, indicating that no gross structural alterations occur when the ligand is bound. Studies involving fluorescence spectroscopy and N-bromosuccinimide reactivity have shown that a change in the environment of some tryptophan residues occurs concomitantly with L-arabinose binding.

Many low molecular weight proteins have been isolated which have been implicated as elements of bacterial transport systems (1-7). Each protein has been found to have a high specificity for the ligands with which it will bind and each protein has been loosely designated "binding proteins." These binding proteins have been presumed to be located in the periplasmic space of the bacterial cell (8). This location is compatible with their possible function in transport and is based on the release of these proteins from the cell following osmotic shock or spheroplast formation. Recently, evidence has been presented which indicates that binding proteins serve as functional elements of transport systems (9, 10), although their exact role is still unknown. Escherichia coli B/r has been shown to have an L-arabinose-binding protein controlled by araC, the regulatory gene for the L-arabinose operon (2). This binding protein has been implicated in a high affinity uptake system in E. coli (11) by studies of competitive inhibitors which have shown that the high affinity L-arabinose uptake system and the in vivo binding of ligands via the

L-arabinose-binding protein have similar specificities. In addition, mutants lacking the high affinity uptake system have been isolated and several have been shown to lack detectable levels of the L-arabinose-binding protein.

This report describes the crystallization of purified L-arabinose-binding protein and a consideration of its chemical and physical properties. The purpose of this research was to determine the properties of the L-arabinose-binding protein at a molecular level with the intent of elucidating the role of this protein in the L-arabinose transport system and the mechanism of this process.

METHODS AND MATERIALS

Bacterial Strains—Escherichia coli B/r strain UP1041 (araA79) was used as a source of the L-arabinose-binding protein (2).

Chemicals—L-(1-3H)Arabinose was purchased from Calbiochem. The purity of radioactive L-arabinose was determined as reported previously (2).

Purification of L-Arabinose-binding Protein—L-Arabinose-binding protein was purified by a modification of the procedure of Hogg and Englesberg (2). A cell-free extract was prepared from 600 g of wet cell paste suspended in 1.800 ml of 10 mm potassium phosphate buffer (14.3 mM 2-mercaptoethanol, pH 7.6) by disruption in a French pressure cell followed by centrifugation at 23,300 × g for 90 min. Solid ammonium sulfate was added to the cell-free extract (≈30 mg of protein per ml) until 55% saturation was attained, and the solution was stirred at 0° for 60 min. Precipitated material was removed by centrifugation at 23,300 × g for 20 min and additional ammonium sulfate was added to 100% saturation. This solution was stirred for 2 hours at 0° and the precipitated material was collected by centrifugation at 23,300 × g for 30 min. The precipitate was redissolved in a minimal volume (approximately 100 ml) of 10 mm potassium phosphate buffer (14.3 mM 2-mercaptoethanol, pH 7.6), dialyzed against the same buffer, applied to a DEAE-cellulose column (4 × 90 cm), and eluted with 10 mm potassium phosphate buffer. Protein was monitored by absorbance at 280 nm. The first protein peak to elute was collected, and the solution was concentrated by vacuum dialysis to 40 ml, dialyzed 19 hours against 1 mm potassium phosphate buffer (14.3 mM 2-mercaptoethanol, pH 7.8), and applied to a DEAE-cellulose column (4 × 43 cm) equilibrated with the same buffer. This second DEAE-cellulose column was eluted with a 3-liter linear 0 to 20 mm potassium chloride gradient in 1 mM potassium phosphate buffer. Protein was monitored by absorbance at 280 nm. Two overlapping protein peaks were found to elute at concentrations of potassium chloride between 3 and 10 mm. All of the arabinose-binding activity was found in the first peak. The first half of this peak was pooled and the remaining half was concentrated and recycled through another DEAE-cellulose column to obtain material free from the second contaminating protein peak. Pools from both DEAE-cellulose columns were mixed, concentrated to 10 ml, applied to a Sephadex G-150 column.
(2.5 × 90 cm), equilibrated with 10 mM Tris buffer (14.3 mM 2-mercaptoethanol, pH 7.4), and eluted. The fractions containing the major protein peak were pooled.

**Crystallization of L-Arabinose-binding Protein**—Crystallization of the L-arabinose-binding protein was performed by a modification of the procedure of Pardee (12). Purified L-arabinose-binding protein, 10 mg per ml, was extensively dialyzed against 2 mM potassium phosphate, pH 6.5. 2-Methyl-2,4-pentanediol (Eastman) was slowly added until the solution became 65% (v/v). Distilled water was added until the solution cleared, and a few seed crystals were added. Crystals were visible after storage at 5°C for 4 hours; in the absence of seed crystals this process required extended time periods. The crystals were harvested by centrifugation and washed three times in 80% ethanol (95). Washed crystals were redisolved in 10 mM Tris-chloride (72 mM 2-mercaptoethanol, pH 7.4) containing 8 M urea and dialyzed against the same solution. The protein was next dialyzed against 10 mM Tris-chloride (14.3 mM 2-mercaptoethanol, pH 7.4). The urea treatment of the crystallized material ensured that all of the L-arabinose was removed from the protein (this treatment has been shown to remove all bound L-[14C]arabinose from L-arabinose-binding protein preparations). Urea-treated L-arabinose-binding protein retained the same specific activity (micromoles of L-arabinose bound per mg of protein) as material which was not treated with urea. Crystalline L-arabinose-binding protein which had been treated in this manner was diluted to a protein concentration of approximately 10 mg per ml and stored at -15°C. This solution was used for all subsequent experiments.

**Physical and Chemical Methods**—L-Arabinose binding was assayed by equilibrium dialysis as reported earlier (2).

Protein samples were prepared for amino acid analysis by the methods of Moore and Stein (13). Duplicate samples containing crystallized L-arabinose-binding protein (0.5 mg) were hydrolyzed in 6 N HCl for 24, 48, or 72 hours at 110°C and analyzed with a Beckman model 120 amino acid analyzer. Cysteine was determined as cysteic acid following performic acid oxidation (14, 15) and tryptophan was determined spectrophotometrically (16).

Sedimentation equilibrium experiments were performed with a Beckman model E ultracentrifuge using the meniscus depletion method as described by Yphantis (17). Samples of L-arabinose-binding protein were prepared in 50 mM potassium phosphate, pH 6.5, at 2.5, 1.0, and 0.5 mg of protein per ml. Each of these concentrations was centrifuged at 35,600, 29,500, and 24,600 rpm until equilibrium was attained, and photographic plates were prepared using interference optics. Fringe displacements were measured with a micro comparator and molecular weight calculations were performed for each protein concentration at each speed. Molecular weight determinations in the presence of 6 M guanidine hydrochloride were performed with L-arabinose-binding protein (1.0 mg per ml) in 50 mM Tris buffer (14.3 mM 2-mercaptoethanol-6 M guanidine hydrochloride, pH 7.0). L-Arabinose-binding protein solutions were centrifuged at 36,500 rpm at 20°C until equilibrium was attained. Sedimentation velocity coefficients were determined using a single sector synthetic boundary cell and schlieren optics. All sedimentation studies were conducted at 20°C at 50,780 rpm and photographs were taken at 4-min intervals.

Electrophoresis on polyacrylamide gels was performed as described earlier (2) or in the presence of sodium dodecyl sulfate (18).

All spectrofluorimetric measurements were made on a Webber spectrofluorimeter. Solutions of L-arabinose-binding protein (0.1 mg per ml) were prepared in 10 mM Tris buffer, pH 7.4. Ligands were added in 30-μl volumes to 3-ml samples as indicated.

Circular dichroism spectra were obtained with a Cary 60 spectropolarimeter. All measurements were made with a 2-mm path length at room temperature at a protein concentration of 0.32 mg per ml.

Antibody directed against crystallized L-arabinose-binding protein was prepared as described previously (19).

**RESULTS**

**Purification and Crystallization of L-Arabinose-binding Protein**—L-Arabinose-binding protein was purified by a modification of the procedure of Hogg and Englesberg (2) as described under "Methods and Materials." Approximately 500 mg of

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**TABLE I**

<table>
<thead>
<tr>
<th>Fraction</th>
<th>Total protein</th>
<th>Specific activity</th>
<th>Yield</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cell-free extract</td>
<td>48,000</td>
<td>N.D.*</td>
<td>100</td>
</tr>
<tr>
<td>55 to 100% (NH₄)₂SO₄ ppt.</td>
<td>12,800</td>
<td>1.9</td>
<td>98</td>
</tr>
<tr>
<td>DEAE effluent</td>
<td>3,120</td>
<td>7.8</td>
<td>74</td>
</tr>
<tr>
<td>DEAE gradient 1</td>
<td>860</td>
<td>21.4</td>
<td>70</td>
</tr>
<tr>
<td>DEAE gradient II</td>
<td>612</td>
<td>28.3</td>
<td>65</td>
</tr>
<tr>
<td>Sephadex G-150</td>
<td>560</td>
<td>28.3</td>
<td>65</td>
</tr>
</tbody>
</table>

* Conducted at saturating ligand concentrations.
* Not determined; high protein concentrations prevented accurate determinations of specific activity in crude extracts by equilibrium dialysis.
* Crystallization was carried out on 100-mg batches as required.
from the storage of L-arabinose-binding protein in the presence of 2-mercaptoethanol. Electrophoresis of pure protein preparations at either pH 6.0 or pH 3.8 did not resolve additional protein bands. In the presence of sodium dodecyl sulfate only a single band is observed. Double gel diffusion studies using the method of Ouchterlony (21) and immunoelectrophoresis have not resolved more than one antigenic species.

The binding protein was crystallized using 2-methyl-2,4-pentanediol as described under "Methods and Materials." Uniform hexagonal crystals (approximately 0.02 mm long) were obtained which appeared birefringent when observed under polarized light (Fig. 2). Larger crystals (0.5 mm long) could be obtained by dialysis of the protein against 55% 2-methyl-2,4-pentanediol in 2 mM potassium phosphate, pH 6.5. The crystallized L-arabinose-binding protein had a slightly higher specific activity for L-arabinose binding than noncrystallized material.

**Physical Properties**—A summary of the physical properties of the L-arabinose-binding protein is presented in Table II. A molecular weight of \( 38,100 \pm 1,300 \) was determined from sedimentation equilibrium studies in the ultracentrifuge. A partial specific volume of 0.73 was assumed in all calculations. The molecular weight values determined were not affected by the protein concentrations or the rotor speeds used. Linear plots were obtained from all determinations by graphing the ln of the relative fringe displacement _versus_ the square of the rotational radius, indicating molecular weight homogeneity of the sample. Molecular weight determinations conducted in the presence of 6 M guanidine hydrochloride did not reveal the presence of molecular weight species lower than 38,000. A molecular weight of \( 40,000 \pm 1,700 \) was determined by gel chromatography on a calibrated Bio-Gel P-100 column.

**Amino Acid Composition**—The amino acid composition of the L-arabinose-binding protein was determined as described under "Methods and Materials" (Table III). Values for each determination were standardized by assuming that the concentrations of lysine and glutamic acid remained unchanged throughout the hydrolysis. Corrections of this type did not result in changes greater than 10%. Half-cystine and tryptophan were determined by separate analyses as described under "Methods and Materials."

**Conformational Changes**—The mechanism involved in the transport of small molecules through the lipophilic environment of the membrane is largely unknown. One model suggests that proteins involved in the transport process undergo a conformational change on binding the transported ligand which facilitates transfer. In an attempt to detect such a conformational change for the L-arabinose-binding protein, a number of studies were undertaken.

Sedimentation velocity coefficients for the L-arabinose-binding protein were determined in the presence and absence of L-arabinose. Crystallized L-arabinose-binding protein (10 mg per ml in 10 mM Tris, pH 7.4) was diluted with various concentrations of L-arabinose to give final protein concentrations of 5 mg per ml. No significant changes of the sedimentation velocity coefficient were observed in the presence or absence of L-arabinose (Table IV), indicating that major changes in the tertiary structure of the binding protein probably do not occur when L-arabinose is bound.

The circular dichroism spectra were determined for the L-arabinose-binding protein in the presence and absence of L-arabinose as seen in Fig. 3. Measurements were made at 0, 0.1, and 10

<table>
<thead>
<tr>
<th>Amino acid</th>
<th>nmole/mg</th>
<th>moles/38,000 g</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lysine</td>
<td>0.78</td>
<td>29.6</td>
</tr>
<tr>
<td>Histidine</td>
<td>0.07</td>
<td>2.7</td>
</tr>
<tr>
<td>Arginine</td>
<td>0.22</td>
<td>8.4</td>
</tr>
<tr>
<td>Aspartic acid</td>
<td>0.00</td>
<td>34.2</td>
</tr>
<tr>
<td>Threonine</td>
<td>0.47</td>
<td>17.9</td>
</tr>
<tr>
<td>Serine</td>
<td>0.56</td>
<td>14.4</td>
</tr>
<tr>
<td>Glutamic acid</td>
<td>0.50</td>
<td>28.4</td>
</tr>
<tr>
<td>Proline</td>
<td>0.47</td>
<td>17.9</td>
</tr>
<tr>
<td>Glycine</td>
<td>0.87</td>
<td>33.1</td>
</tr>
<tr>
<td>Alanine</td>
<td>0.05</td>
<td>36.1</td>
</tr>
<tr>
<td>Valine</td>
<td>0.09</td>
<td>33.8</td>
</tr>
<tr>
<td>Methionine</td>
<td>0.50</td>
<td>11.4</td>
</tr>
<tr>
<td>Isoleucine</td>
<td>0.42</td>
<td>16.0</td>
</tr>
<tr>
<td>Leucine</td>
<td>0.02</td>
<td>23.6</td>
</tr>
<tr>
<td>Tyrosine</td>
<td>0.20</td>
<td>7.6</td>
</tr>
<tr>
<td>Phenylalanine</td>
<td>0.50</td>
<td>16.0</td>
</tr>
<tr>
<td>1/2 Cys</td>
<td>0.044</td>
<td>1.7</td>
</tr>
<tr>
<td>Tryptophan</td>
<td>0.01</td>
<td>7.0</td>
</tr>
</tbody>
</table>

* Calculated from the molecular weight and sedimentation velocity coefficient.
* Determined by dissolving dried, crystallized material in 0.01 M Tris, pH 7.4. Optical measurements were made with a Gilford spectrophotometer using a 1-cm path length.

![Fig. 2. Crystals of the L-arabinose-binding protein photographed using polarized light (× 400).](image-url)
FIG. 3. Circular dichroism spectra of L-arabinose-binding protein. Spectra of solutions of crystallized L-arabinose-binding protein (0.32 mg per ml in 10 mM Tris, pH 7.4) in 0, 0.1, and 10 mM L-arabinose were obtained using a Cary 60 spectropolarimeter with a 2-mm path length. The presence or absence of L-arabinose was not found to produce any alterations in the spectra.

TABLE IV

<table>
<thead>
<tr>
<th>L-Arabinose concentration</th>
<th>Sedimentation coefficient</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>3.28</td>
</tr>
<tr>
<td>10^{-4}</td>
<td>3.18</td>
</tr>
<tr>
<td>10^{-4}</td>
<td>3.38</td>
</tr>
<tr>
<td>10^{-3}</td>
<td>3.18</td>
</tr>
</tbody>
</table>

mm L-arabinose concentrations as described under “Methods and Materials,” and a comparison of spectra obtained did not reveal any significant differences, suggesting that gross changes in the peptide chain are not induced by the presence of L-arabinose. The presence of some β conformation in the protein backbone was revealed by the single trough between 215 and 220 nm. Spectral properties characteristic of α-helical structure were not detected in significant amounts, indicating that less than 10% of the peptide chain is in this form.

Fluorescence spectroscopy was used to reveal minor changes which would not have been previously detected. A 5-nm blue shift in the fluorescence maximum was observed when L-arabinose was present, although no significant difference in the fluorescence intensity was seen (Fig. 4). The spectral shift indicates that the local environments of some tryptophan residues become more hydrophobic when L-arabinose is bound. Similar fluorescent blue shifts were observed in the presence of 1 mM α-galactose, α-xylose, and α-fucose but not with 1 mM α-glucose. These results are consistent with the binding specificities previously determined for the L-arabinose-binding protein (11).

Titrations of fluorescence at 335 nm with N-bromosuccinimide (5) in the presence or absence of ligand was also used to detect changes in the local environments of tryptophan residues. Small volumes (less than 2% of the total volume in the cuvette) of 30 mM N-bromosuccinimide were added to L-arabinose-binding protein solutions in 0.1 M acetate buffer (pH 5.0) and the relative fluorescence at 335 nm was determined. The presence of L-arabinose or α-galactose lowered the relative reactivity of tryptophan residues, as can be seen in Fig. 5.

The binding activity of L-arabinose-binding protein treated with N-bromosuccinimide was also studied. The fluorescence at 335 nm of samples of L-arabinose-binding protein (0.1 mg per ml) in 0.1 M acetate buffer, pH 5.0, was determined using an excitation wavelength of 291 nm. N-Bromosuccinimide (0.03 M) was added in aliquots of 0.3% of the total volume and fluorescence was determined. O, L-arabinose-binding protein without substrate; □, L-arabinose-binding protein with either 10^{-4} M L-arabinose or 10^{-4} M α-galactose.

The binding activity of L-arabinose-binding protein treated with α-galactosucinimide was also studied. The fluorescence at 335 nm of samples of L-arabinose-binding protein (0.1 mg per ml in 0.1 M acetate buffer, pH 5.0) was determined using an excitation wavelength of 291 nm. N-Bromosuccinimide (0.03 M) was added in aliquots of 0.3% of the total volume and fluorescence was determined. O, L-arabinose-binding protein without substrate; □, L-arabinose-binding protein with either 10^{-4} M L-arabinose or 10^{-4} M α-galactose.

FIG. 4. Fluorescence spectra of L-arabinose-binding protein in the presence and absence of L-arabinose. Spectra were obtained using protein concentrations of 0.1 mg per ml in 10 mM Tris, pH 7.4. A, emission spectra, the excitation wavelength was 291 nm; ——, without L-arabinose; ·····, with L-arabinose. B, excitation spectra; the emission wavelength was 335 nm.

FIG. 5. Titration of L-arabinose-binding protein fluorescence with N-bromosuccinimide. The fluorescence of L-arabinose-binding protein (0.1 mg per ml) in 0.1 M acetate buffer, pH 5.0, at 335 nm was determined using an excitation wavelength of 291 nm. N-Bromosuccinimide (0.03 M) was added in aliquots of 0.3% of the total volume and fluorescence was determined. O, L-arabinose-binding protein without substrate; □, L-arabinose-binding protein with either 10^{-4} M L-arabinose or 10^{-4} M α-galactose.
TABLE V

The fluorescence at 335 nm of samples of L-arabinose-binding protein (0.35 mg per ml in 0.1 M citrate buffer, pH 5.0) either in the absence or presence of 10^{-3} M L-arabinose was determined using an excitation wavelength of 291 nm. N-Bromosuccinimide (15 mM) was added and the fluorescence was again determined. Samples from both before and after the addition of N-bromosuccinimide were assayed for L-arabinose binding by equilibrium dialysis.

<table>
<thead>
<tr>
<th>N-Bromosuccinimide added</th>
<th>Initial fluorescence</th>
<th>Initial specific activity</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>+Arabinose -Arabinose</td>
<td>+Arabinose -Arabinose</td>
</tr>
<tr>
<td>0</td>
<td>100% 100%</td>
<td>100% 100%</td>
</tr>
<tr>
<td>0.15</td>
<td>79% 74%</td>
<td>95% 72%</td>
</tr>
<tr>
<td>0.38</td>
<td>51% 32%</td>
<td>50% 50%</td>
</tr>
</tbody>
</table>

activity at low pH is similar to the pK values for the carboxyl groups of aspartic and glutamic acid residues (3.7 and 4.3, respectively). Under conditions of low pH (less than 4), where the L-arabinose-binding ability is significantly reduced, a 50% decrease in the fluorescence intensity at 335 nm also can be observed. These observations suggest that charged acidic groups are necessary to maintain a conformation favorable for binding. Ionic strengths varying from 0 to 1 M KCl (in 0.01 M Tris, pH 7.4-14.3 mM 3-mercaptoethanol) were found to have no effect on L-arabinose binding. The insensitivity of binding to pH and ionic strength indicates that charge interactions probably do not contribute significantly to the binding forces.

Preparations of crystallized protein were dialyzed against buffers containing various concentrations of L-arabinose. Normal hyperbolic binding with a single binding site per molecule was observed. The dissociation constant was determined to be 2.8 \times 10^{-7} M (Fig. 7). This dissociation constant, obtained using crystallized preparations, is lower than that obtained in earlier studies using crude preparations (2). A dissociation constant of 4.0 \times 10^{-7} M was determined for d-galactose binding by the L-arabinose-binding protein. The L-arabinose-binding protein was found to bind 1 mole of either of these ligands per mole of protein at saturating ligand concentrations.

DISCUSSION

The data presented indicate that the L-arabinose-binding protein shares physical properties common to the previously described binding proteins of bacterial origin. The L-arabinose-binding protein has a low molecular weight with a secondary peptide structure containing predominantly \( \beta \) conformation and a very small amount of \( \alpha \) helical structure. All binding proteins which have been previously characterized have been found to be low molecular weight proteins (\( \approx 20,000 \) to 40,000) and some have been shown to contain secondary peptide structures which are predominantly \( \beta \) conformation (22).

Amino acid analyses reveal that this protein contains a high content of aspartic and glutamic residues and nonpolar amino acids. Similar compositions have been reported for the galactose-, leucine-, and glucose-binding proteins (5, 23, 24). The apparent similarities in the pK values for acidic amino acids and the binding process of the L-arabinose-binding protein suggest that these charged groups are necessary to maintain the molecule in an active conformation. The high concentration of nonpolar amino acids observed may serve to facilitate the interactions between this molecule and the hydrophobic environment of the cell membrane. The observed presence of 2 cysteine residues is contrary to the concept that cysteines are present only in the membrane bound permeases and not in the osmotically shockable binding protein components.

Sedimentation velocity and circular dichroism studies indicate that no gross structural changes occur when L-arabinose is bound. Circular dichroism studies of other binding proteins also have not revealed major alterations when ligands were bound (4, 22). Fluorescence spectroscopy and N-bromosuccinimide reactivity data show that a change in the environment of some tryptophan residues occurs when ligands are bound which can be attributed to either the presence of tryptophan residues in the binding site which interact with the ligand, or alterations of the L-arabinose-binding protein which change the environmental conditions of tryptophan residues not present in the binding site. The presence of tryptophan residues in the binding site of the L-arabinose-binding protein is consistent with the loss of binding activity as tryptophan oxidation occurs and the relative insensitivity of binding to pH and ionic strength changes. Other binding proteins which have been well characterized have also been shown not to undergo major structural alterations when ligands
were bound. Boos et al. (22) have demonstrated alterations in the fluorescence spectra of the galactose-binding protein when galactose was bound which are similar to those observed for the L-arabinose-binding protein; however, conformational changes which have been observed in previously studied binding proteins and those reported here for the L-arabinose-binding protein seem to be limited to changes in the environments of tryptophan residues.

Models which involve ligand-induced conformational changes of transport proteins to facilitate transport cannot be eliminated by these results since very little is known about how binding proteins are associated with the cell membrane. Structural changes which cannot be readily detected in purified protein preparations in vitro may become more apparent when the binding protein is properly oriented within the cell membrane. An alternate role of binding proteins in transport may be as recognition units in a multiple component system where other proteins are involved in the actual transport process. Conformational changes induced by ligand binding would not be required in such a model. The high affinity histidine permease of Salmonella typhimurium has been shown to require a protein, designated the P protein, along with the histidine-binding protein for function (25). In this system the P protein may serve as the actual transport protein and the histidine-binding protein may serve as a recognition protein. At the present time it would appear that insufficient evidence exists to assign binding proteins to either role nor can it be expected that all binding proteins should function in the same manner.

Acknowledgments—We wish to thank Dr. Kenneth Neet for his helpful discussions throughout this work. We are also grateful to Mrs. Joan Sprague for her technical assistance.

REFERENCES

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