Dependence of Initiation Factor IF-2 Activity on Proteins L7 and L12 from *Escherichia coli* 50 S Ribosomes*

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**SUMMARY**

The activity of initiation factor IF-2 from *Escherichia coli* strain MRE600 has been studied as a function of the presence or absence of the 50 S ribosomal proteins L7 and L12. Three specific aspects of initiation factor IF-2 function were examined: (a) the binding of radioactive initiation factor IF-2 to 70 S ribosomes; (b) the ribosomal binding of N-formylmethionyl transfer RNA which is dependent upon initiation factor IF-2; (c) 5′-guanosine triphosphate hydrolysis catalyzed by initiation factor IF-2 in the presence of 70 S ribosomes. The results show that the binding of initiation factor IF-2 and of N-formylmethionyl transfer RNA are both reduced 50 to 75% when 50 S particles deficient in L7 and L12 are employed. The 5′-guanosine triphosphate hydrolysis activity is far more drastically reduced with particles deficient in L7 and L12. The latter effect is similar to results found with the 5′-guanosine triphosphate hydrolysis activities associated with elongation factors EF-G and EF-Tu. The results suggest that proteins L7 and L12 are part of the binding site for initiation factor IF-2, but that they are more intimately involved in the 5′-guanosine triphosphate hydrolysis catalyzed by the factor. Furthermore, the results are consistent with a model in which the binding sites for all three factors, IF-2, EF-G, and EF-Tu, share certain common determinants, including the L7 and L12 proteins.

Three different protein factors required for protein synthesis possess GTPase† activity when they are bound to the 70 S ribosome: elongation factors EF-G (1) and EF-Tu (2), and initiation factor IF-2 (3). In all three cases, both the protein factor and the ribosome are required. The function of EF-G is associated with the translocation step of protein synthesis; those of EF-Tu and IF-2 primarily related to the binding of aminoaeryl-tRNA or fMet-tRNA, respectively. The functions of all three factors are coupled to their GTPase activities.

The GTPase activities of EF-G and EF-Tu have previously been shown to depend on the presence of two closely related acidic 50 S proteins, namely L7 and L12* (5–7). In the case of EF-G not only does its activity depend upon L7/L12, but cross-linking experiments have shown that the factor can be covalently attached to L7/L12 in the 50 S particle (8). Furthermore, it has been demonstrated that EF-G and EF-Tu cannot simultaneously bind to the ribosome (9–12). These results strongly suggest that the two factors occupy the same or closely overlapping sites on the 50 S ribosome, and that L7/L12 are constituents of the site(s).

Although the presence of both ribosomal subunits is necessary for the GTPase reaction catalyzed by IF-2 (13, 14), the specific ribosomal proteins which are involved have not been established. The investigation described here was designed to test the hypothesis that all three factors of protein synthesis which are linked to GTP hydrolysis share a common requirement for proteins L7/L12 for their activity. We describe here experiments which demonstrate that all the known activities of IF-2 associated with the 70 S ribosome, GTP hydrolysis, fMet-tRNA binding and the binding of IF-2 itself, are dependent upon the presence of proteins L7/L12.

Nakamoto and co-workers (15) made a preliminary statement indicative of some of the conclusions documented here. During the preparation of this manuscript similar results were published by Kay et al. (10). Moreover, an article by Lockwood et al. (17) reaches conclusions similar to our own.

**EXPERIMENTAL PROCEDURE**

**Materials**—The 30 S and 50 S ribosomal subunits of *E. coli* and initiation factors IF-1 and IF-2 were prepared as described by Fakunding and Hershey (18). 32P-IF-2 was prepared from purified IF-2 by using skeletal muscle protein kinase and [γ-32P]ATP

* The nomenclature of Wittmann et al. (4) is used for the designation of 50 S ribosomal proteins L7/L12. In the experiments reported here, no attempt has been made to separate or distinguish L7 from L12; hence, the designation L7/L12 will be used throughout. Subunits deficient in L7/L12 are referred to as [±L7/L12 50 S] particles; those to which L7/L12 have been added back as [±L7/L12 50 S] particles; and 50 S ribosomes reserved for the untreated subunits. The 70 S ribosomes formed by the association of normal 30 S subunits and [±L7/L12 50 S] particles are referred to as [±L7/L12 70 S] or [±L7/L12 70 S] ribosomes.
as described by Fakunding et al. (19). A-U-G was synthesized by the method of Bundararajan and Thach (20) and tMet-tRNA was prepared according to the method of Hershey and Thach (21). [γ-32P]GTP was prepared as described by Reimann et al. (22). The purified EF-G was a gift from Dr. James Bodley, University of Minnesota. Biochemical compounds were obtained as follows: GTP and pyruvate kinase from Calbiochem; Gpp(CH)₃p and polyuridylic acid from Miles Laboratories; dithiothreitol from Pierce Chemicals; [14C]phenylalanine from Schwarz-Mann; and glutaraldehyde 50%, biological grade, from Fischer Scientific. All other chemicals were reagent grade.

**Preparation of Deficient Subunits and L7/L12 Protein Fraction**

-Purified 50 S ribosomal subunits were extracted according to the procedure of Hamel et al. (15), except that 10 mM Tris-HCl, pH 7.4, was substituted for imidazole-HCl, and the subunits precipitated with ethanol were collected by centrifugation at 15,000 x g for 15 min.

The combined supernatant fractions containing L7/L12 were dialyzed overnight against three changes of 20 mM NH₄HCO₃, pH 7.6, lyophilized and dissolved in 50 mM Tris-HCl, pH 7.4, 100 mM NH₄Cl, 1 mM EDTA; 7 mM β-mercaptoethanol; 25% glycerol. The extracted ribosomal pellet, [-L7/L12 50 S] particles, was resuspended in 10 mM Tris-HCl, pH 7.4, 10 mM magnesium acetate; 100 mM NH₄Cl; 0.5 mM EDTA; 7 mM β-mercaptoethanol, and dialyzed against this buffer overnight. Both the L7/L12 protein fraction and the [-L7/L12 50 S] particles were stored at 3°.

Reconstituted [+L7/L12 50 S] particles were prepared by adding a saturating amount, 10 to 20 molar excess, of the L7/L12 protein fraction to the [-L7/L12 50 S] particles at 0°. These reconstituted particles were then added to appropriate reaction mixtures without further preincubation. The excess L7/L12 proteins had no effect on the various reactions tested. The concentrations of the [±L7/L12 50 S] particles used are indicated in the figure legends.

**IF-2 GTPase Assay**—“Coupled” IF-2 GTPase activity (dependent on fMet-tRNA binding) was assayed in a 50+1 reaction mixture containing: 50 mM Tris-HCl, pH 7.4; 12 mM MgCl₂; 0.2 mM dithiothreitol; 1 mM ATP, 0.3 mM GTP; 100 mM NH₄Cl; 275 μg of tRNA, 4.5 mM phosphoenolpyruvate; 40 μg of pyruvate kinase; 40 μg of polyuridylic acid; 0.05 mM [32P]GTP (activity, 260 Ci per mole); and 10 μM [γ-32P]GTP (activity, 150-300 cpm per mole). The amounts of purified IF-2, 50 S ribosomal subunits and any further additions are indicated in the appropriate figure legends or in the text. After incubation at 30° for 5 min, 40 μl of the reaction mixture were removed and added to a 15-m1 conical centrifuge tube containing 0.2 ml of 1 mM potassium phosphate, pH 7.2, and 0.2 mM of 1 M mercaptoacetic acid. The subsequent steps of the assay for the release of [32P]P were carried out as described by Kolakowski et al. (3). The “uncoupled” GTPase activity (GTP hydrolysis independent of fMet-tRNA binding) was assayed in an identical manner with the exception that A-U-G, [32P]Met-tRNA, and IF-1 were omitted from the reaction mixtures.

**Polypehylalanine Synthesis**—Each assay mixture (200 μl) contained: 50 mM Tris-HCl, pH 7.4; 12 mM MgCl₂; 0.2 mM dithiothreitol; 1 mM ATP, 0.3 mM GTP; 100 mM NH₄Cl; 275 μg of tRNA, 4.5 mM phosphoenolpyruvate; 40 μg of pyruvate kinase; 40 μg of polyuridylic acid; 0.05 mM [32P]phenylalanine (activity, 5 Ci per mole); and 5 μl of dialyzed supernatant free of ribosomes. After incubation for 15 min at 37°, the reaction was stopped by the addition of 1.5 ml of 5% trichloroacetic acid, and the tubes were heated to 90° for 20 min followed by filtration through glass fiber filters (Whatman GF/C). The filters were washed with 5% trichloroacetic acid followed by 95% ethanol, dried, and counted.

**Results**

**Characterization of [-L7/L12 50 S] Particles**

Polyacrylamide Gels—The isolated [-L7/L12 50 S] particles as well as the extracted L1/12 protein fraction were subjected to analysis by polyacrylamide gel electrophoresis in 8% urea at pH 4.5, according to the procedure of Truant (23). The gels of the [-L7/L12 50 S] particles revealed that at least 80 to 90% of stained material corresponding to the position of L7/L12 was removed. It was not established whether the remaining material represents residual L7/L12, or other minor 50 S proteins. The extracted protein fraction contained proteins L7/L12 as well as a contaminant of higher molecular weight (approximately 20% of the total protein) which was not considered to be a major ribosomal protein constituent. The gel patterns of isolated [+L7/L12 50 S] particles resembled that of 50 S subunits, indicating the reassociation of L7/L12 with the deficient particle.

**Association of [-L7/L12 50 S] Particles with 30 Subunits**—The function of L7/L12 previously reported as well as the experiments described here could be explained by the failure of [-L7/L12 50 S] particles to reassociate with 30 S subunits to form 70 S couples (24). We investigated this possibility by sucrose density gradient analysis and found that [-L7/L12 50 S] and [+L7/L12 50 S] particles both interact equally well in the subunit reassociation reaction to form what are designated [-L7/L12 70 S] and [+L7/L12 70 S] particles.

**Polyphenylalanine Synthesis**—The [-L7/L12 50 S] particles were tested for their activity in the synthesis of polyphenylalanine in the presence of 30 S ribosome and polyuridylic acid, as described by Nirenberg and Matthaei (25). Results similar to those of Hamel et al. (15) were obtained and are shown in Table I. The [-L7/L12 50 S] particles exhibited only 25% of the activity of control 50 S subunits. The addition of L7/L12 proteins to the [-L7/L12 50 S] particles restored 85 to 90% of the activity. Thus, protein synthesis with our [-L7/L12 50 S] particles, while not completely dependent on exogenous L7/L12 proteins, is stimulated 3- to 4-fold by their addition.

**EF-G Catalyzed GTPase Activity**—In order to confirm that the inhibition of protein synthesis activity was the result of a deficiency in the GTPase activity of the [-L7/L12 50 S] particle and to confirm that our [-L7/L12 50 S] particles were similar to those described in other laboratories, the uncoupled GTPase reaction of EF-G was measured (26). A 5-fold diminution in EF-G catalyzed GTPase activity was observed with [-L7/L12 50 S] particles as compared to [+L7/L12 50 S] particles. This result, in agreement with the reports of others (5, 7), confirms

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**Table I**

| Activity of [-L7/L12 50 S] particles in polyphenylalanine synthesis | Reaction conditions are described under “Experimental Procedure,” and contained 160 pmoles of 30 S ribosomal subunits. |
|---------------------------------------------------------------|---------------------------------------------------------------------------------------------------|
| **Additions**                                                  | **[32P]Phe incorporated** |
| None                                                          | 20                                                   |
| Unextracted 50 S particles (60 pmoles)                        | 1100                                                 |
| [-L7/L12 50 S] particles (50 pmoles)                          | 320                                                   |
| [+L7/L12 50 S] particles (50 pmoles)                          | 970                                                   |

* J. L. Fakunding, unpublished results.
that the presence of proteins L7/L12 are required for the EF-G catalyzed GTP hydrolysis activity. Since the [L7/L12 50 S] particles used in these experiments were similar to those previously reported with respect both to polyphenylalanine synthesis and EF-G catalyzed GTP hydrolysis, their activity in reactions associated with IF-2 was studied.

Reactions Associated with IF-2

GTP Hydrolysis—The rate of GTP hydrolysis catalyzed by IF-2 in the presence of all the components required for fMet-tRNA binding (i.e., GTPase coupled to fMet-tRNA binding) was assayed with [L7/L12 50 S] and [+L7/L12 50 S] particles. Details of the reaction conditions are given in the legend to Fig. 1. The results show that at concentrations of the 50 S subunit which were limiting with respect to 30 S subunits, [+L7/L12 50 S] particles catalyze the hydrolysis of GTP four to five times more rapidly than [L7/L12 50 S] particles. Furthermore, the hydrolysis of GTP is completely dependent on IF-2. The large stimulation obtained with L7/L12 shows that these proteins are required to obtain the maximum rate of the coupled GTP hydrolysis reaction catalyzed by IF-2.

The coupled and uncoupled IF-2 catalyzed GTPase reactions of [L7/L12 50 S] and [+L7/L12 50 S] particles were compared as a function of the amount of IF-2 added (Fig. 2). Both the coupled and the uncoupled GTPase reactions were substantially greater with the [+L7/L12 50 S] particle. Although even the uncoupled IF-2 GTPase activity is stimulated by the addition of L7/L12 proteins to [L7/L12 50 S] particles, a much larger stimulation is observed under coupled conditions.

fMet-tRNA Binding to 70 S Ribosomes—At limiting concentrations of IF-2, extensive fMet-tRNA binding to 70 S ribosomes requires GTP hydrolysis. The hydrolysis of GTP appears to stimulate the release of IF-2 from the 70 S initiation complex and thus allows the factor to act catalytically (18, 27, 28). Since [L7/L12 70 S] particles have a greatly reduced GTPase activity compared to [L7/L12 50 S] particles, the binding of both fMet-tRNA and IF-2 is comparably observed under coupled conditions.

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One standard method for determining fMet-tRNA binding to ribosomes is measurement of the fMet-tRNA retained on Millipore filters due to the tRNA-ribosome interaction (29). However, this assay does not distinguish between the binding of fMet-tRNA to 30 S subunits or to 70 S ribosomes. Since the object of these experiments was to examine the binding of fMet-tRNA to [L7/L12 70 S] particles, the reactions were analyzed by sucrose gradient centrifugation, where binding to 30 S and 70 S ribosomes is clearly differentiated.

The binding of [H]fMet-tRNA to 70 S ribosomes containing [L7/L12 50 S] or [L7/L12 70 S] particles was measured both with GTP and with the nonhydrolysed analogue, 5'-guanylyl methylenediphosphonate (Gpp(CH2)P). The gradient profiles are shown in Fig. 3. The amounts of [H]fMet-tRNA bound to the 70 S particles were calculated and are given in Table II. The results from these gradients show that [+L7/L12 70 S] particles bind fMet-tRNA about two times as well as [L7/L12 70 S] particles. The stimulation with L7/L12 proteins occurred under both conditions of GTP hydrolysis and also when GTP hydrolysis was prevented (i.e., with GMP-PCP). It is therefore clear that the L7/L12 proteins affect more than just the GTP hydrolysis functions of the 70 S ribosome.

$^{14}$P-IF-2 Binding to 70 S Ribosomes—The binding of $^{14}$P-IF-2 to 70 S ribosomes prepared with [L7/L12 50 S] and [L7/L12 70 S] particles in the presence of Gpp(CH2)P (Fig. 3, open circles) was measured by fixing the complexes with glutaraldehyde followed by sucrose gradient analysis (18). The [+L7/L12 70 S] ribosomes bound $^{14}$P-IF-2 about two times as well as the [L7/L12 70 S] ribosomes. In the presence of Gpp(CH2)P the binding of $^{14}$P-IF-2 and [H]fMet-tRNA was approximately equimolar (Table II), in agreement with our previous results (18). Therefore, the binding of both fMet-tRNA and IF-2 is comparably dependent on the presence of L7/L12 proteins.

DISCUSSION

The question posed in the experiments reported here is whether initiation factor IF-2 shares with elongation factors EF-G and
mixtures were incubated for 5 min at 30°C, a volume of 5% glucose, 4 pg of L7/L12 protein and 0.2 mM GTP or Gpp(CH2)p. The 0.8 pg of purified IF-1 (90 pmoles); 25-30 S ribosomal subunits; 30 pmoles of [-L7/L12 50 S] particles, [32P]-IF-2 (specific activity, 400 cpm per pmole); where indicated [3H]fMet-tRNA (specific activity, 2800 Ci per mole) ; 15 pmoles of [3H]fMet-tRNA bound in the 70 S region of the gradient (e.g. Fractions 5 to 12 in Panel A).

The amounts of IF-2 and fMet-tRNA bound to 70 S ribosomes were calculated from the profiles in Fig. 3 by summing the amounts of each species bound in the 70 S region of the gradient (e.g. Fractions 5 to 12 in Panel A).

Since, as previously demonstrated in this (18) and other laboratories (27, 28), GTP hydrolysis accompanies the release of IF-2 from the 70 S initiation complex, it might be anticipated that all conditions which blocked GTP hydrolysis would thereby enhance or stabilize the binding of IF-2. The results reported here (see Fig. 3) are contrary to this interpretation. When GTPase is inhibited by a deficiency in proteins L7/L12, a slight decrease, not an increase, in IF-2 binding is observed. The results are consistent with a dual function of L7/L12: (a) as participants in the binding site for IF-2; (b) as proteins required for GTPase activity. We interpret our results as indicating that, under the experimental conditions employed, the former effect of L7/L12 predominates with respect to the binding of IF-2.

Obviously if the absence of L7/L12 resulted in a complete loss of IF-2 binding to the 70 S ribosome, it would follow that the other related activities would also be abolished. However, we find that the absence of L7/L12 results in a reduction in, but not a complete loss of capacity for IF-2 to bind in the 70 S initiation complex. We estimate that the [-L7/L12 50 S] particles employed in these experiments are at least 80% to 90% deficient in L7/L12. Thus, the fact that the inhibition of IF-2 binding is only 50% cannot be explained by the presence of intact 50 S subunits in our preparation of [-L7/L12 50 S] particles. A more likely explanation is that, although L7/L12 is part of the binding site for IF-2, other proteins are also cooperatively involved in the site, and therefore the depletion of L7/L12 alone does not completely abolish IF-2 binding.

The results with the binding of fMet-tRNA are consistent with the above interpretation. Again fMet-tRNA binding is only partially reduced in the [-L7/L12 50 S] particles; moreover, this reduction is of the same order as that for IF-2 binding. Only when IF-2 is allowed to act catalytically does the amount of fMet-tRNA bound exceed the amount of bound IF-2.

By contrast to the results on IF-2 and fMet-tRNA binding, the experiments with IF-2 catalyzed GTPase show a much greater involvement of L7/L12; i.e. IF-2 catalyzed GTPase is inhibited to a far greater extent than either IF-2 binding or fMet-tRNA binding.

The results are consistent with a model in which L7/L12 are considered to play a more direct role in GTP hydrolysis than in the actual binding of IF-2. In the absence of L7/L12 IF-2 still binds, but with reduced efficiency; however, even though bound, its catalytic function in GTP hydrolysis is significantly impaired. A nonessential role for the L7/L12 proteins in the GTP hydrolysis reactions of EF-G and EF-Tu has been suggested recently by Hamel and Nakamoto (5). They reported that L7/L12 deficient particles, low in GTPase activity, were stimulated by making the reaction mixtures 20% in methanol. Their results indicate that the L7/L12 proteins do not themselves supply the amino acid residues responsible for the catalytic site of GTP hydrolysis. Instead the proteins were interpreted to play a role in maintaining a conformation of the 70 S ribosome necessary for efficient GTP hydrolysis and factor binding. However, Highland et al. (30) using antibodies to single pure 50 S proteins demonstrated that only anti-L7 and anti-L12 blocked the binding of EF-G. This, in addition to the demonstration by Acharya

![Figure 3. Analysis by sucrose gradient centrifugation of the effect of L7/L12 on the binding of fMet-tRNA and IF-2 to 70 S ribosomes. Reaction mixtures (0.1 ml) contained: 50 mM Tris-HCl, pH 7.4; 100 mM KCl; 5 mM Mg acetate; 2 mM dithiothreitol; 62 pmoles of 30 S ribosomal subunits; 30 pmoles of [-L7/L12 50 S] particles, 0.8 pg of purified IF-1 (90 pmoles); 25 µM A-U-G; 42 pmoles of [3H]fMet-tRNA (specific activity, 2800 Ci per mole); 16 pmoles of 32P-IF-2 (specific activity, 400 cpm per pmole); where indicated 4 µg of L7/L12 protein and 0.2 mM GTP or Gpp(CH2)p. The mixtures were incubated for 3 min at 30°C, 80% volume of 3% glutaraldehyde was added and the mixtures were cooled to 5°C. Each was then applied to a 7 to 25% sucrose gradient (4.5 ml) containing: 10 mM Tris-HCl, pH 7.4; 50 mM KCl; 5 mM magnesium acetate; and 2 mM dithiothreitol. The gradients were centrifuged for 145 min at 4°C at 40,000 rpm in a Beckman SW 56 rotor. Approximately 0.16-ml fractions were collected; 0.5 ml of water and 5 ml of Triton X-100 were added; 0.5 ml was aspirated by a Beckman LS-200 scintillation counter. See Table II for the components used for gradients A, B, C, D.

![Table II. Amounts of IF-2 and fMet-tRNA bound to 70 S ribosomes](http://www.jbc.org/)

| Figure panel | [3H]fMet-tRNA | 32P-IF-2 |
|--------------|--------------|----------|
| A (GTP: [-L7/L12 70 S]) | 4.2 | 0.8 |
| B (GMPPCP: [-L7/L12 70 S]) | 2.0 | 2.1 |
| C (GTP: [+L7/L12 70 S]) | 8.4 | 1.4 |
| D (GMPPCP: [+L7/L12 70 S]) | 4.2 | 1.5 |

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et al. (8) that EF-G can be crosslinked to L7/L12, suggests a
direct interaction of L7/L12 in the binding of EF-G.

The present results show that IF-2, like the two elongation
factors EF-G and EF-Tu, shares a requirement for L7/L12 for
its GTPase activity. We can infer that L7/L12 constitutes
part of the IF-2 binding site, but is more directly involved in
the GTPase activity. It seems likely that the binding site of IF-2
is identical with or overlaps the EF-G and EF-Tu sites, which
may be nearly identical. Experiments are in progress to deter-
mine whether IF-2, like EF-G, can be cross-linked to proteins
L7 and L12, and whether EF-G and EF-Tu compete with IF-2
for a common binding site.

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