The formation of glutaminyl-tRNA (Gln-tRNA) in *Bacillus* chloroplasts, and mitochondria occurs in a two-step reaction. This involves misacylation of tRNA\(^{Gln}\) with glutamate by glutamyl-tRNA synthetase and subsequent amidation of Glu-tRNA\(^{Glu}\) to the correctly acylated Gln-tRNA\(^{Gln}\) by a specific amidotransferase (Schön, A., Kannangara, C. G., Gough, S., and Söll, D. (1988) *Nature* 331, 187-190). Here we demonstrate the existence of this pathway in green algae and describe the purification of the Glu-tRNA\(^{Glu}\) amidotransferase from *Chlamydomonas reinhardtii*. The purified enzyme showed an *M*\(_{r}\) of approximately 120,000 when analyzed by glycerol gradient sedimentation and gel filtration. An apparent *M*\(_{r}\) of 63,000 of the denatured protein was demonstrated by electrophoresis on sodium dodecyl sulfate-polyacrylamide gels. This indicates that the enzyme possesses an \(\alpha_2\) structure. The substrate for the purified enzyme is Glu-tRNA\(^{Glu}\) but not Gln-tRNA\(^{Glu}\). The enzyme requires ATP, Mg\(^{2+}\), and an amide donor for the conversion. Acceptable amide donors are glutamine, asparagine, and ammonia. Blocking of the glutamine-dependent reaction by alkylation of the protein with 6-diazo-5-oxonorleucine did not inhibit the ammonia-dependent reaction, suggesting that the enzyme has separate glutamine and ammonia binding sites. As suggested by Wilcox (Wilcox, M. (1969) *Eur. J. Biochem.* 11, 400-412) the amidation reaction may involve glutamyl-phosphate formation, since ATP is cleaved to ADP when the enzyme is incubated with Glu-tRNA\(^{Glu}\) and ATP. In common with other glutamine amidotransferases, the enzyme also possesses low glutaminase activity. The purified Glu-tRNA\(^{Glu}\) amidotransferase forms a stable complex with Glu-tRNA\(^{Glu}\) in the presence of ATP and Mg\(^{2+}\) but in the absence of the amide donor as determined by gradient centrifugation.

Two different metabolic pathways for the formation of Gln-tRNA

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Glutamine and ATP analogs were from Sigma. The C. reinhardtii cells were obtained from Dr. Y. Ishino, unpublished results.

**EXPERIMENTAL PROCEDURES**

All operations were performed at 0-4 °C unless stated otherwise.

**Materials**—Biochemicals were reagent grade from Sigma. [14C]Glu (285 mCi/mmol) was purchased from Du Pont-New England Nuclear. Dowex-1 (chloride form) was obtained from Sigma. DEAE-cellulose (DE52) and phosphocellulose (P11) were purchased from Whatman. The prepacked FPLC columns Superose 12, Mono S HR 5/5, and Mono Q HR 10/10 were obtained from Pharmacia LKB Biotechnology Inc. Glutamine and ATP analogs were from Sigma. The C. reinhardtii cell wall defective strain CC-400 cu-15 mt+ was a gift from Dr. Elizabeth H. Harris, Duke University.

**Buffers**—Buffer A, 10 mM Tricine-Cl, pH 8.3, 5 mM MgCl2, 3 mM DTT, 0.2 mM phenylmethylsulfonyl fluoride, 20% glycerol; buffer B, 20 mM Hepes, pH 7.0, 3 mM DTT, 0.2 mM phenylmethylsulfonyl fluoride, 20% glycerol; buffer C, same composition as buffer A but 10% glycerol and pH 7.5; buffer D, 20 mM Hepes, pH 7.5, 0.5 mM EDTA, 0.2 mM phenylmethylsulfonyl fluoride, 10% glycerol.

**AdT Assay**—During the purification procedure the AdT activity was followed by measuring the formation of [14C]Glu from precharged Bacillus subtilis [14C]Glu-tRNA+++, whereas the assays for the determination of the enzymatic properties of the pure AdT were carried out using C. reinhardtii [14C]Glu-tRNA+++. The reaction mixture contained 20 pmol of precharged [14C]Glu-tRNA+++ in a total volume of 50 µl of 100 mM Hepes, pH 7.5, 10 mM MgCl2, 10% glycerol, and 3 mM DTT with the indicated amounts of AdT in the presence of 1 mM ATP and 1 mM glutamine for 20 min at 37 °C. Reactions were stopped and the tRNAs were deacylated by the addition of 0.4 M NaOH for 20 min at 80 °C. The solution was neutralized with 81 µl of 100 mM Tricine-Cl, pH 7.0, 10 mM MgCl2, and 10% β-mercaptoethanol. Aliquots of each fraction were aminoacylated with homologous pure glutamyl-tRNA synthetase as described by Chen et al. (1990a).

**Column Chromatography of Chlamydomonas tRNA**—A complete description of the determination of the native molecular weight by gel filtration and rate zonal centrifugation is given by Chen et al. (1990a).

**RESULTS**

**Purification of the Glu-tRNA++ Amidotransferase from C. reinhardtii Cells**

Initial experiments showed that C. reinhardtii AdT can efficiently utilize B. subtilis [14C]Glu-tRNA+++ as substrate. As the B. subtilis tRNA was more readily available than C. reinhardtii tRNA we used the bacterial tRNA as substrate in the purification of AdT activity from C. reinhardtii. In the purification scheme we took care to enrich only the tRNA-dependent activity by checking in parallel assays for the conversion of free glutamate (instead of Glu-tRNA++) to glutamine. In addition, since it is known that glutamine synthetase can also carry out at low efficiency the conversion of Glu-tRNA++ to Gln-tRNA++, this enzyme (Lipmann and Tuttle, 1945) during the purification.

Purification of AdT from whole cell S-100 extracts was by four chromatographic steps (summarized in Table I). The first purification step was DEAE-cellulose chromatography to remove nucleic acids from the extract. Because of the high concentration of tRNA in the S-100 exact specific activity of AdT could not be determined. Assuming an overall enzyme recovery of 60% we estimate a 1-fold "purification" after this first step. The next purification by separation on phosphocellulose did not bind approximately 90% of the protein applied to the column. The AdT activity was recovered with elution by 250 mM KCl, yielding a 5-fold purification. At this step all glutamine synthetase activity was found in the flow-through of the column, separated from the AdT activity. After this step only tRNA-dependent Glu \rightarrow Gln conversion was detected in protein-containing fractions. Further purification was achieved on two different FPLC resins. Mono Q chromatography resulted in a 9-fold increase in specific activity, whereas the final step, Mono S chromatography, gave a 7-fold purification. The overall purification was at least 315-fold. The active fraction contained a single protein band as shown by SDS-polyacrylamide gel electrophoresis (Fig. 2).

**Physical Properties of Glu-tRNA+++ Amidotransferase**

We determined the molecular weights of the native and denatured enzyme. Two independent methods, rate zonal
AdT with micrococcal nuclease has no effect on enzyme activity (data not shown).

**TABLE I**

Purification of the Glu-tRNA\(^{\text{Gln}}\) amidotransferase from C. reinhardtii

| Purification step | Total volume | Total protein | Total activity | Specific activity | Yield | Purification factor |
|-------------------|--------------|---------------|----------------|-------------------|-------|--------------------|
| S-100             | 1,000        | 5,000         | 144            | 40                | 50\(^*\) | 1                  |
| DE52              | 270          | 1,100         | 44             | 40                | 50\(^*\) | 1                  |
| Qi                 | 95           | 105           | 21             | 200               | 23.8  | 5                  |
| Mono S            | 12           | 8             | 14.4           | 1,800             | 16.4  | 45                 |
| Mono S            | 2            | 0.2           | 2.5            | 12,500            | 2.8   | 315                |

* Not determined. The enzyme activity was not determined because of the presence of endogenous tRNA in the S-100 fraction.

* 50% recovery of enzyme activity was assumed for this step.

* One-fold purification was assumed.

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**FIG. 2.** SDS-Polyacrylamide gel electrophoresis of the purified AdT. 2 \(\mu\)g of the 100 mM KCl Mono S fraction were run on a 10% SDS-polyacrylamide gel followed by silver staining. The positions of the marker proteins (in kilodaltons) are indicated. They were taken and assayed for AdT activity. Carbonic anhydrase (31 kDa), ovalbumin (43 kDa), bovine serum albumin (66 kDa), \(\beta\)-galactosidase (118 kDa), and myosin (200 kDa) were sedimented as marker proteins on a parallel gradient; the positions in the gradient were determined by SDS-polyacrylamide gel electrophoresis and subsequent staining.

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**Fig. 3.** Determination of the native molecular mass and of the size of AdT. A, determination of the native molecular weight. 5 \(\mu\)g of the 100 mM KCl Mono S fraction was sedimented for 22 h at 4 °C through a 10-35% glycerol gradient. Fractions (0.4 ml) were taken and assayed for AdT activity. Carbonic anhydrase (31 kDa), ovalbumin (43 kDa), bovine serum albumin (66 kDa), \(\beta\)-galactosidase (116 kDa), and myosin (200 kDa) were sedimented as marker proteins on a parallel gradient; the positions in the gradient were determined by SDS-polyacrylamide gel electrophoresis and subsequent staining. B, estimation of the molecular size by gel filtration. 10 \(\mu\)g of the 100 mM KCl Mono S fraction was run through a Superose 12 column in the presence of 500 mM KCl. Fractions (0.5 ml) were collected and assayed for AdT activity. The molecular sieve column was precalibrated with the following marker proteins: cytochrome c (12.4 kDa), carbonic anhydrase (31 kDa), ovalbumin (43 kDa), bovine serum albumin (66 kDa), \(\beta\)-galactosidase (118 kDa), \(\beta\)-amylase (200 kDa), apoferritin (443 kDa). The void volume \((V_0)\) was determined with dextran blue.
Glu-tRNA\(^{\text{Glu}}\) Amidotransferase from C. reinhardtii

Reactions containing tRNA were further purified by ethanol precipitation and then deacylated by incubation with 20 mM Tris (pH 10) for 30 min at 60°C. A mixture of \[^{14}C\text{Glu}\] and \[^{14}C\text{Gln}\] (lane M) provided the reference markers. Lane 1, 5 \(\mu\)M \[^{14}C\text{glutamate}\] and 1 mM ATP (lane 1); lane 2, 5 \(\mu\)M \[^{14}C\text{glutamate}\], 1 mM ATP, and 1 mM glutamine; lane 3, \[^{14}C\text{Glu-tRNA}\] (30 pmol) and 1 mM ATP, and 1 mM glutamine; lane 4, \[^{14}C\text{Glu-tRNA}\] (30 pmol), 1 mM ATP, and 1 mM glutamine; lane 5, \[^{14}C\text{Glu-tRNA}\] (30 pmol) and 1 mM glutamine.

Amide donor and ATP requirement of Glu-tRNA\(^{\text{Glu}}\) amidotransferase

| Amide donor | NTP (or analog) | Glu-tRNA\(^{\text{Glu}}\) formation |
|-------------|----------------|-------------------------------|
| L-Glutamine | ATP            | 7.5                           |
| N-Acetyl-L-glutamine | ATP | 7.5                           |
| Ammonium chloride | ATP | 3.4                           |
| L-Asparagine | ATP            | 0.9                           |
| L-Leucineamide | ATP | 0.6                           |
| L-Valeramide | ATP            | 0.3                           |
| d-Glutamine | ATP            | 0.01                          |
| L-Glutamine | AMP-PCP         | 0.01                          |
| L-Glutamine | AMP-CPP         | 5.6                           |
| L-Glutamine | AMP-PCP         | 0.01                          |

cleavage occurs between the \(\beta\)- and \(\gamma\)-phosphate. This conclusion is also supported by the ATP hydrolysis experiment presented below. The activation is specific for ATP as GTP could not replace it (Table III).

Glu \(\rightarrow\) Gln Conversion Requires Activation by Phosphorylation

Earlier experiments (Wilcox, 1969) provided evidence that ATP is required in the reaction to activate the \(\gamma\)-carboxyl group of glutamate which is bound to tRNA. As a first step we wanted to investigate the site of cleavage of the phosphates of ATP. Therefore, we incubated AdT [\(\alpha-32P\)]ATP either alone or in presence of tRNA\(^{\text{Glu}}\) or Glu-tRNA\(^{\text{Glu}}\). The reaction was analyzed by TLC for the appearance of radioactive ADP and ATP indicating ATP conversion (Fig. 5). The enzyme did not bring about any ATP cleavage when incubated with ATP alone or in the presence of uncharged tRNA\(^{\text{Glu}}\) (Fig. 5, lanes 1 and 2). However, when charged Glu-tRNA\(^{\text{Glu}}\) was used, ATP was converted to ADP (Fig. 5, lane 3). This result is in agreement with the utilization of AMP-PCP, but not AMP-PCP, instead of ATP in the AdT reaction (see above).

ATP may be required in this reaction in order to provide a phosphorylated enzyme intermediate (for discussion see Buchanan, 1973). Therefore we performed an ATP-P, exchange experiment in the presence of large amounts of ADP and \[^{32}P\]orthophosphate and checked for the formation of radioactive ATP by TLC as described above. Although several...
Glu-tRNA<sup>Gln</sup> Amidotransferase from C. reinhardtii

concentrations of enzyme and substrate were used we were unable to detect labeled ATP. Thus, it appears that in the absence of tRNA or amide donor the enzyme cannot form a phosphorylated intermediate.

Are There Two Amido Donor Binding Sites on the Enzyme?

It is well known that some glutamine amidotransferases use glutamine and also ammonium chloride as amido donors (Zalkin, 1985). To distinguish between the two binding sites selective alkylation (of a cysteine residue) by the glutamine analog 6-diazo-5-oxo-L-norleucine (DON) has been used (Tso et al., 1982) which abolished the enzyme’s ability to use glutamine but not ammonia. We therefore preincubated AdT (Mono S fraction) without amide donor but in the presence of all other reaction components with different amounts of DON. After starting the reactions by the addition of the amide donors (glutamine or ammonium chloride), the amidotransferase activity was measured. Parallel reactions without inhibitor inactivation served as control. Fig. 6 shows that DON only affected the glutamine-dependent reaction. This result indicates the existence of separate binding sites for glutamine and ammonia on the AdT molecule. C. reinhardtii AdT differs in this respect from the B. subtilis enzyme, where DON cannot inhibit the glutamine-dependent activity in crude extracts (Strauch et al., 1988).

Glu-tRNA<sup>Gln</sup> Amidotransferase Has Low Glutaminase Activity

Since it is known that glutamine amidotransferases possess glutaminase activity which is sometimes stimulated by the products of the amidotransferase forward reaction we checked the glutaminase activity of purified AdT. Under the conditions of the normal AdT reaction the enzyme (Mono S fraction) was able to convert [<sup>14</sup>C]Gln to [<sup>14</sup>C]Glu. In the absence of added components, AdT was able to convert in 120 min 2.5% of input Gln (20 mM) into Glu. However, this low glutaminase activity was stimulated (to 7.5%) by the presence of ADP, P<sub>i</sub>, Mg<sup>2+</sup> ions, and pre-charged Glu-tRNA<sup>Gln</sup>. Without the addition of enzyme, almost no conversion was detectable.

Purified Glu-tRNA<sup>Gln</sup> Amidotransferase Forms a Stable Complex with Glu-tRNA<sup>Gln</sup>

As a first step towards the investigation of the reaction mechanism of the C. reinhardtii AdT, we wanted to examine the ability of the enzyme to form a complex with its tRNA substrate. Wilcox (1969) demonstrated complex formation of the B. subtilis AdT with its substrate by binding of pre-charged Glu-tRNA<sup>Gln</sup> to a protein out of a crude column fraction and isolation of the complex by gel filtration. Since we had previously achieved separation of charged tRNA from different tRNA binding enzymes under mild conditions with glycerol gradient sedimentation (Chen et al., 1990a), we applied this method to the analysis of the initial process of the enzyme reaction, the complex formation with the mischarged tRNA.

We assumed that stable complexes may be obtained in the absence of the amide donor so the enzymatic amide transfer by AdT can not proceed. As seen in Fig. 7D, AdT does form a stable complex with Glu-tRNA<sup>Gln</sup>, which is not present in the control gradients with Glu-tRNA<sup>Glu</sup> (Fig. 7B) and the enzyme alone (Fig. 7A). The complex did not form with uncharged tRNA<sup>abs</sup>, tRNA<sup>mus</sup>, and charged Glu-tRNA<sup>Gln</sup>, indicating a distinct substrate specificity for mischarged tRNA<sup>Gln</sup> (data not shown). Moreover, the absence of any complex without ATP in the assay demonstrated the dependence of complex formation on the presence of ATP (Fig. 7C).

DISCUSSION

The formation of Gln tRNA<sup>Gln</sup> in Gram-positive cubacteria, archaeabacteria, and in organelles (Fig. 1) requires the presence
of two enzymes, a glutamyl-tRNA synthetase which misacylates tRNA<sub>Glu</sub> with glutamate (Lapointe <em>et al.</em>, 1986; Chen <em>et al.</em>, 1990b) and a tRNA-dependent amidotransferase which forms the “correct” aminoacyl-tRNA. Since the presence of a functional glutamyl-tRNA synthetase could not be demonstrated in these organisms and organelles, this pathway provides the sole source of glutamine for protein biosynthesis. This situation contrasts from that found in the cytoplasm of eukaryotic cells and in Gram-negative eubacteria where free glutamine is acylated directly onto tRNA<sub>Glu</sub> by glutamyl-tRNA synthetase (see e.g. Schimmel and Söll, 1979). Given the occurrence of the different pathways in the various phyla it is plausible that they evolved independently, possibly to assure a greater degree of fidelity in protein synthesis. Conversely, the transamidation pathway may be the precursor of the direct aminoclaylation pathway, in which case AdT or the glutamyl-tRNA synthetase are evolutionary precursors of the cytoplasmatic glutamyl-tRNA synthetase. Interestingly, the AdT is in some ways similar to glutamyl-tRNA synthetase; both enzymes bind in a specific manner the same substrates, glutamine, ATP, and the tRNA<sub>Glu</sub> molecule.

In order to shed some light on these questions, we have purified from <i>C. reinhardtii</i> glutamyl-tRNA synthetase (Chen <em>et al.</em>, 1990b) and AdT (this paper) with the aim of a detailed structural analysis of both enzymes. Presumably both enzymes are located in the chloroplast of the organism (see Chen <em>et al.</em>, 1990b). The Glu-tRNA<sub>Glu</sub> amidotransferase has many aspects of other well known tRNA-independent glutamine amidotransferases; it utilizes ammonia as amide donor independently of glutamine binding, it can be inhibited by 6-diazo-5-oxonorleucine, it has low glutaminase activity, and it uses ATP as an energy source. In addition, it can discriminate between different Glu-tRNA species, using only mischarged Glu-tRNA<sub>Glu</sub> as its substrate. Thus, the enzyme may be a hybrid between an aminoclay-tRNA synthetase with its exquisitely RNA discrimination (Rould <em>et al.</em>, 1989; Perona <em>et al.</em>, 1989) and a glutamine amidotransferase with its conserved glutamine amide transfer domain (see e.g. Mei and Zalkin, 1989). Furthermore, the ATP cleavage by AdT to give ADP and P<sub>1</sub> resembles the amidotransferases and not the aminoclay-tRNA synthetases which produce AMP and PP<sub>1</sub> (Schimmel and Söll, 1979). The high substrate specificity of the C. reinhardtii AdT enzyme for Glu-tRNA<sub>Glu</sub> species from different organisms confirms the widespread evolutionary conservation of this mechanism.

How does the cell maintain accuracy in protein synthesis while coping with the mischarged tRNA? Clearly, the Glu-tRNA<sub>Glu</sub> must not be among the pool of aminoclay-tRNAs utilized on the ribosome as this would lead to errors in protein synthesis. A channelling mechanism (Srivastava and Bernard, 1986) might be possible where the mischarged tRNA would be transferred directly to the amidotransferase from the glutamyl-tRNA synthetase and only released when the correct aminoclay-tRNA is made. This may involve the formation of a tight complex between both enzymes. However, such a complex was not observed, even in the presence of Glu-tRNA<sub>Glu</sub> and ATP, in gel filtration and ultracentrifugation studies (data not shown). Another possibility is based on discrimination against the mischarged tRNA in the complex formation with the elongation factor EF-Tu or in the use on the ribosome. Experimental answers to these questions must be obtained in order to shed light on the evolution of the mechanisms maintaining accuracy in protein biosynthesis.

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