MiaB IS AN IRON-SULFUR PROTEIN*

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The product of the miaB gene, MiaB, from Escherichia coli participates in the methylation of the adenosine 37 residue during modification of tRNAs that read codons beginning with uridine. A His-tagged version of MiaB has been overproduced and purified to homogeneity. Gel electrophoresis and size exclusion chromatography revealed that MiaB protein is a monomer. As isolated MiaB contains both iron and sulfide and an apoprotein form can chelate as much as 2.5–3 iron and 3–5 sulfur atoms per polypeptide chain. UV-visible and EPR spectroscopy of MiaB indicate the presence of a [4Fe-4S] cluster under reducing and anaerobic conditions, whereas [2Fe-2S] and [3Fe-4S] forms are generated under aerobic conditions. Preliminary site-directed mutagenesis studies suggest that Cys157, Cys161, and Cys164 are involved in iron chelation and that the cluster is essential for activity. Together with the previously shown requirement of S-adenosylmethionine (AdoMet) for the methylation reaction, the finding that MiaB is an iron-sulfur protein suggests that it belongs to a superfamily of enzymes that uses [Fe-S] centers and AdoMet to initiate radical catalysis. MiaB is the first and only tRNA modification enzyme known to contain an Fe-S cluster.

The chemistry of the biological reactions leading to the synthesis of sulfur-containing biomolecules remains largely unknown. However, this research field has recently received renewed attention with the study of the enzymology of important metabolic pathways such as those leading to iron-sulfur clusters, biotin, molybdothionin, thiamin, and isopenicillin (1–5) for examples. tRNAs also depend on the presence of sulfur-containing bases for activity. Conversion of uridine to 4-thiouridine by Thl protein is one of such sulfur atom insertion reactions into tRNAs (6). Another tRNA modification leading to 2-methylthio-N-6-isopenetyl adenosine requires a chemically difficult aromatic C–H to C–S bond conversion, which has not been investigated yet. ms2i6A-37 is found at position 37 next to the anticodon on the 3′-position in almost all eukaryotic and bacterial tRNAs that read codons beginning with U except tRNA1.5V Ser (7). The postulated pathway for the synthesis of ms2i6A-37 and the genes involved are as indicated in Fig. 1 (8–10).

Genetic studies have shown that, in Escherichia coli, the first step of the biosynthesis of ms2i6A-37 is the addition of the isopenetyl group to the N-6 nitrogen of adenosine, a reaction catalyzed by the well studied tRNA-isopenetylpyrophosphate transferase enzyme encoded by the miaA gene (11–13). The second step, which requires iron, cysteine, and S-adenosylmethionine (AdoMet), consists of both sulfur insertion and methylation at position 2 of the base moiety, but it is still unknown whether each step is catalyzed by a specific or by the same enzyme (14–16). On the other hand tRNAs from mutant strains lacking a functional miaB gene have been shown to contain only i6A-37, the product of the first step of the pathway suggesting that the MiaB protein is involved in C–S bond formation (17). Deficiency in methylation leads to a decreased efficiency of the corresponding tRNAs and an increased spontaneous mutation frequency (18, 19).

To gain insight into the mechanisms for sulfur atom insertion into biological molecules in general, we have focused our interest on the product of the miaB gene, which has been recently cloned (17). Here we report the first characterization of a purified MiaB protein and demonstrate that it is an iron-sulfur protein. MiaB is the first Fe-S enzyme shown to participate in tRNA modification.

EXPERIMENTAL PROCEDURES

All DNA manipulations were as described previously (20). Enzymes, oligonucleotides, and culture media were purchased from Invitrogen. T4 polynucleotide kinase, T4 DNA ligase, and the Wizard Genomic DNA Purification kit were from Promega, Inc. Bacterial alkaline phosphatase and plasmid DNA purification kit Fexiprep were from Amersham Biosciences. DNA fragments were extracted from agarose gel and purified with High Pure PCR Product Purification kit (Roche Molecular Biochemicals); DNA sequencing was performed by Genome Express (Grenoble, France). Ni-NTA Superflow was purchased from Qiagen (Hilden, Germany). E. coli strain DH5α was used for cloning and plasmid propagation. E. coli strain TX3436 miaB− was used for the in vitro activity tests (17). E. coli strain BS341(DE3)pLyS8 was used for gene expression.

Cloning of the miaB Gene and Construction of Plasmids Overexpressing MiaB and the N-terminal Hexahistidine-tagged MiaBH—Genomic DNA from E. coli DH5α was purified from 100 ml of culture grown in LB following a procedure recommended by the supplier. The MiaB-encoding gene was amplified by polymerase chain reaction using this genomic DNA as a template. The following primers were used: 5′-gcgaagcgacagcatATGcacaacac-3′ (NdeI site underlined, ATG codon in uppercase) hybridized to the noncoding strand at the 5′ terminus of the gene and 5′-ggaaaggccggagcctgccataagag-3′ (HindIII site underlined) hybridized to the coding strand −25 bp downstream of the transcription termination signal (17). PCR products were run on a Stratagene RoboCycler Gradient 40 machine as follows. Genomic DNA (2–4 µg) of E. coli was

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1 The abbreviations used are: ms2i6A-37, 2-methylthio-N-6-isopenetyl adenosine; AdoMet, S-adenosylmethionine; i6A-37, N-6-isopenetyl adenosine 37; Ni-NTA, nickel-nitritolatriacetic acid; MIABH, MiaB with a His6 tag at the N terminus; HPLC, high performance liquid chromatography.

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denatured for 4 min at 94 °C in the presence of the primers (0.5 μM each). The Pgo DNA polymerase (2 units) and deoxynucleotide mix (0.2 mM each) were added, and 25 cycles (1 min at 94 °C, 1 min at 55 °C, and 3 min at 72 °C) were then performed followed by a 10-min elongation step at 72 °C. The PCR product was digested with NdeI and HindIII and cloned in pT7 digested with the same enzymes. The cloned gene was completely sequenced to verify that the PCR had not introduced any errors. The pT7-derived plasmid containing the whole miaB gene was named pT7-miaB.

The hexahistidine linker sequence was introduced by using two oligonucleotides: His1 (5′-tagcagacttacatcatac3′), which anneals to the His2 (5′-tagttgatggtgaggtgca-3′) and forms a cassette containing a 5′ and 3′ NdeI-digested site. The two primers were previously end-phosphorylated with ATP by using T4 polynucleotide kinase as recommended by the manufacturer. This cassette was ligated into pT7-miaB digested with NdeI and dephosphorylated. The proper orientation of the His tag cassette was selected by PCR screening experiment and confirmed by sequencing the purified plasmid. A plasmid containing the His tag cassette was selected by PCR using the primers end-phosphorylated with ATP by using T4 polynucleotide kinase and confirmed by DNA sequencing.

Site-directed Mutagenesis—Mutagenesis was carried out on plasmid pT7-miaB with QuickChange™ Site-Directed Mutagenesis kits from Stratagene according to the manufacturer’s protocol. Mutations were confirmed by DNA sequencing.

overexpression and Purification of MiaBH—The pT7-miaB plasmid was used to transform E. coli B834(DE3)pLysS, which was grown at 37 °C in Luria Broth supplemented with 100 mg/liter ampicillin and 35 mg/liter chloramphenicol. When A600 reached 0.4 the production of the MiaBH was induced by addition of 50 μM isopropyl-1-thio-β-D-galactopyranoside, and the incubation was subsequently carried on overnight at 15 °C. Induction of protein overexpression in the culture was monitored by SDS-PAGE on portions of a 1-ml sample withdrawn at various times. Cells were collected by centrifugation at 4000 rpm at 10 °C, resuspended in 50 mM Tris/HCl, pH 8, and stored at −70 °C until use.

The MiaBH protein was purified aerobically at 4 °C as follows. The frozen cells were thawed, broken by sonication, and centrifuged at 220,000 × g at 4 °C for 1 h. The cell-free extract was loaded onto a 4×15-cm column of Ni-NTA Superflow previously equilibrated with buffer A (50 mM Tris/HCl, pH 8, 10 mM imidazole). The column was subsequently washed thoroughly with buffer A, and the protein was eluted with a linear gradient of buffer B (50 mM Tris/HCl, pH 8, 50 mM NaCl, 500 mM imidazole). Fractions containing the protein were pooled and concentrated in an Amicon cell fitted with a YM30 (Spectrapor) membrane.

Preparation of the Apoprotein—The apo form of MiaBH protein was prepared as follows. Protein-bound iron was removed by chelation during reduction of the protein aerobically at 0 °C in the presence of 10 mM EDTA and 10 mM sodium dithionite in 50 mM Tris-Cl, pH 8 buffer. The solution became colorless after 1 h incubation, and the solution was loaded onto a Sephadex G-25 column (P10) equilibrated and eluted with the same buffer. The apoMiaBH was then concentrated by ultrafiltration using Centricron 30 devices (Amicon).

Reconstitution of ApoMiaBH—All steps of the reconstitution procedure were completed anaerobically inside a glove box containing less than 2 ppm O2. The apoprotein was incubated with an 8-fold excess of ferrous iron and sodium sulfide under anaerobic conditions as described under “Experimental Procedures.” After anaerobic desalting on a Sephadex G-25 column, MiaBH was then found to contain 2.5–3 iron and 3–3.5 sulfur atoms per polypeptide chain.

The purified protein has a reddish-brown color in agreement with the light absorption spectrum in Fig. 3 (spectrum 1), and the analysis for labile iron and sulfide suggested the presence of a protein-bound iron-sulfur cluster. However, iron content was substoichiometric with regard to MiaBH (0.7 mol/mol), and the protein contained sulfide in slight excess with regard to iron (1.0–1.2 mol/mol), probably as a consequence of loss of the cluster during purification. Consequently to find how much iron and sulfide can be chelated by MiaBH, the protein was converted to the apoprotein form (Fig. 3, spectrum 2) and then reconstituted with an 8-fold excess of ferrous iron and sodium sulfide under anaerobic conditions as described under “Experimental Procedures.”

The electronic absorption spectrum of the as isolated MiaBH (Fig. 3, spectrum 1) displays absorption bands at 330, 416, and 460 nm, and a shoulder at around 560 nm, compatible with a [2Fe-2S]²⁺ center (27). The spectrum of the reconstituted MiaBH displays absorption bands at 330, 416, and 460 nm and a shoulder at around 560 nm, compatible with a [2Fe-2S]²⁺ center (27).

RESULTS

MiaB Is an Iron-Sulfur Protein—MiaBH protein is derived from MiaB by addition of a tag of six histidines at the N terminus. It was obtained from an E. coli overproducer and purified aerobically using a Ni-NTA column that specifically retains proteins containing cluster of histidines. The purity of the protein was checked by SDS-PAGE (Fig. 2). The purified protein was analyzed by Edman degradation and gave the six histidines and first 10 amino acids of the N-terminal sequence of MiaB (17). MiaBH behaves as a monomer with an apparent molecular mass of 60 kDa (Mₐ = 53,600 calculated from the amino acid sequence) during gel chromatography on Superdex200 by comparison with various molecular weight protein standards.

The purified protein has a reddish-brown color in agreement with the light absorption spectrum in Fig. 3 (spectrum 1), and the analysis for labile iron and sulfide suggested the presence of a protein-bound iron-sulfur cluster. However, iron content was substoichiometric with regard to MiaBH (0.7 mol/mol), and the protein contained sulfide in slight excess with regard to iron (1.0–1.2 mol/mol), probably as a consequence of loss of the cluster during purification. Consequently to find how much iron and sulfide can be chelated by MiaBH, the protein was converted to the apoprotein form (Fig. 3, spectrum 2) and then reconstituted with an 8-fold excess of ferrous iron and sodium sulfide under anaerobic conditions as described under “Experimental Procedures.” After anaerobic desalting on a Sephadex G-25 column, MiaBH was then found to contain 2.5–3 iron and 3–3.5 sulfur atoms per polypeptide chain.
MiaBH protein is slightly different (Fig. 3, spectrum 3) and more consistent with a [4Fe-4S] cluster (27). During anaerobic reduction of reconstituted MiaBH with a 10-fold molar excess of dithionite, a bleaching of the solution and a loss of the visible absorption bands were observed (data not shown). The protein was analyzed by EPR spectroscopy, both in the as isolated and reconstituted (Fig. 4) forms. In both cases it exhibited an isotropic EPR signal centered at $g = 2.01$ (Fig. 4, A1 and B1). The relaxation properties of this signal were characteristic for the $S = 1/2$ ground state of a [3Fe-4S]$^{1+}$ cluster.

This species accounted for 15–20% of total iron in the aerobically purified MiaBH protein (Fig. 4A1) but never exceeded 5% in the reconstituted sample (Fig. 4B1). Upon reduction by dithionite both samples gave rise to a new $S = 1/2$ species, characterized by an axial EPR signal with $g$ values at 2.06 and 1.93, accounting for 40–50% of total iron. The signal became broader at 30 K and could not be detected anymore above 40 K. Such a temperature dependence of the EPR signal and its microwave power saturation properties (data not shown) are characteristic for the $S = 1/2$ ground state of [4Fe-4S]$^{1+}$ clusters (28). Furthermore, the $g$ value of the low field feature is more consistent with a [4Fe-4S]$^{1+}$ rather than with a [2Fe-2S]$^{1+}$ cluster. Partially purified preparations of MiaB, with no His tag, proved to display spectroscopic properties similar to those of MiaBH (data not shown).

**MiaB Is Functional**—The functionality of the MiaB protein was assayed in vivo using the miaB$^{-}$ TX3346 E. coli strain transformed with control pT7-7 (A), pT7-miaBH (B), and pT7-miaBHC157A (C). The identification is based on UV-visible spectra (data not shown) and retention times (Fig. 5). With pT7-miaB, results are as in B, and with pT7-miaBHC161A and pT7-miaBHC164A, results are as in C. AU, arbitrary units.

**Fig. 3.** UV-visible absorption spectra of purified MiaBH. Spectrum 1, as isolated MiaBH; spectrum 2, apoMiaBH; spectrum 3, reconstituted MiaBH. The protein concentrations were 2 mg/ml, and the solvent was 50 mM Tris, pH 8.0, 50 mM KCl. The optical path length was 1 cm.

**Fig. 4.** Electron paramagnetic resonance spectra of 200 µM as isolated MiaBH (A1) and MiaBH after reconstitution (B1). Both samples have been reduced with 5 mM sodium dithionite anaerobically (spectra A2 and B2, respectively). The spectra were recorded under the same conditions: temperature, 10 K; microwave frequency, 9.655 GHz; microwave power, 0.15 milliwatts; modulation amplitude, 10 G.

**Fig. 5.** HPLC chromatograms of tRNA hydrolysates from miaB$^{-}$ TX3346 E. coli strain transformed with control pT7-7 (A), pT7-miaBH (B), and pT7-miaBHC157A (C). The identification is based on UV-visible spectra (data not shown) and retention times (A' elute at about 77 min and ms A' at about 91 min). With pT7-miaB, results are as in B, and with pT7-miaBHC161A and pT7-miaBHC164A, results are as in C. AU, arbitrary units.
strain showed an accumulation of i6A-37 with no evidence of ms2i6A-37 (Fig. 1), those from both MiaB-expressing strains showed the formation of ms2i6A-37 (Fig. 5B). These results demonstrated that the iron-sulfur protein MiaB (or MiaBH), product of the miaB gene, is functional during i6A-37 to ms2i6A-37 conversion.

Whether the iron-sulfur cluster was required for activity was investigated by studying site-directed mutants in which the three conserved cysteines of the conserved Cys
\[ \text{Cys}^{157}, \text{Cys}^{161}, \text{Cys}^{164} \] sequence have been changed to alanine. The above cysteine triad is the signature of a whole class of iron-sulfur proteins and provides the cysteines for iron chelation (29).

Using the in vivo assay described above, we observed that the miaB E. coli TX3346 strain transformed with pT-miaBHC157A, pT-miaBHC161A, and pT-miaBHC164A, each expressing a different mutant MiaB with an alanine in place of cysteine in positions 157, 161, and 164 respectively, was unable to produce the ms2i6A-37 modified nucleoside (Fig. 5C). This provides a strong evidence that the cluster is required for activity. Further characterization of the mutants is in progress.

**DISCUSSION**

The results presented here show that MiaB, the enzyme participating in the methylation of the base moiety of adenosine in some tRNAs (17), is a monomeric iron-sulfur protein. The type of iron-sulfur cluster in MiaB is not definitively identified here since MiaB seems to be able to assemble [4Fe–4S], [3Fe–4S], and [2Fe–2S] center forms as shown by UV-visible and EPR spectroscopy. Cluster lability is furthermore shown from the loss of iron and sulfide during purification. However, the present results indicate that, anaerobically, MiaB can assemble a [4Fe–4S] cluster with both 2+ and 1+ redox states. [3Fe–4S]1+ and [2Fe–2S]2+ clusters accumulated in aerated solutions of the protein indicating that the [4Fe–4S] cluster degrades into clusters of lower nuclearity under exposure to air. This transformation seems reversible since the latter can be converted back to a [4Fe–4S]1+ cluster upon anaerobic reduction. Whether oxygen sensitivity or cluster lability is responsible for incomplete cluster assembly in vitro remains unclear at this stage. Further experiments are required to optimize reconstitution procedures and to make conclusions as far as the iron content is concerned.

The presence, in MiaB, of an oxygen-sensitive iron-sulfur cluster with such unique properties and of essential cysteines within a Cys-Xaa-Xaa-Cys-Xaa-Xaa-Cys sequence (Cys
\[ \text{Cys}^{157}, \text{Cys}^{161}, \text{Cys}^{164} \]) as shown from activity assays reported here together with the requirement of AdoMet for the conversion of i6A-37 to ms2i6A-37 (15) strongly suggests that MiaB is a member of a superfamily of enzymes that utilize the combination of a labile Fe-S cluster and AdoMet to initiate radical catalysis (29, 30). Well studied members of this family are lysine aminomutase, biotin synthase, and the activating components of ribonucleotide reductase and pyruvate-formate lyase (25, 31–33), in which the conserved Cys-Xaa-Xaa-Cys-Xaa-Xaa-Cys sequence has been shown to provide cysteines for iron chelation. It is thus very likely that also in MiaB these cysteines play a similar role and that the MiaB iron center is essential for activity. We suggest that the methylation reaction proceeds through radical activation of the tRNA substrate by a 5′-deoxyadenosyl radical, which is generated by iron-catalyzed reduction of AdoMet. MiaB is the first characterized metalloenzyme and the first Fe-S protein involved in tRNA modification. It is thus very likely that the dramatic effects of iron starvation on ms2i6A-37 synthesis in E. coli cells are a consequence of the requirement of MiaB for iron (14, 16). The availability of pure MiaB now provides the opportunity to investigate whether one or two enzymes are required for i6A-37 to ms2i6A-37.

The MiaB protein catalyzes a C–H to C–S bond conversion. This type of reaction has been recently the subject of intense biochemical and spectroscopic investigations, and a complete understanding of the mechanisms used by enzymes such as biotin synthase or lipoic acid synthase is presently one of the most challenging and fascinating problems in bioinorganic chemistry. MiaB protein provides a new interesting tool to study the mechanistic and structural aspects of that reaction.

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