The Glutamine Residue of the Conserved GGQ Motif in 
Saccharomyces cerevisiae Release Factor eRF1 Is Methylated by the 
Product of the YDR140w Gene*

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Stop signals in mRNA are recognized by class I release factors (RFs)† (1). In eubacteria, two class I RFs with different 
but overlapping specificity for stop codon recognition perform this task. RF1 reads UAG and UAA, and RF2 reads UAA 
and UGA. However, in archebacteria and eukaryotes, a single class I RF, aRF1 and eRF1, respectively, recognizes all three stop 
codons. aRF1 and eRF1 are homologous to each other, and although they belong to a different structural family (2) and display 
different chain foldings (3, 4), one clear sequence motif alone is found in common between eubacterial RF1 and RF2, aRF1 and 
eRF1: a tripeptide motif Gly-Gly-Gln (GGQ) (5). In the crystal 
structure of human eRF1, this tripeptide is at the tip of domain 
2, which is rich in basic amino acids and projects from the bulk 
of the molecule (Fig. 1) (3). In a model of Escherichia coli RF2 
based on both the crystal structure (4) and on cryoelectron 
microscopy observations of the factor bound to ribosomes (6, 7), 
the GGQ tripeptide is positioned rather similarly (Fig. 1). This 
part of RFs from all of the kingdoms is thought to enter the 
peptidyltransferase center and trigger hydrolysis of peptidyl-
tRNA (5). The GGQ motif is essential to the function of both 
prokaryotic and eukaryotic RFs. Replacement of either Gln 
residue by Ala inactivates the factors (3, 5, 8, 9). Replacement 
of the Gln residue by certain amino acids in either human (9) or 
E. coli (8) RFs allows significant release activity to be retained 
in in vitro termination assays, but no substitution has been 
found for the Gln residue in Saccharomyces cerevisiae (3) or 
E. coli (8) RFs that supports cell growth, even at low levels. The 
three-dimensional structure of eRF1 in solution is similar if not 
identical to that in the crystal (10), whereas the crystal 
structures of both E. coli RF2 (4) and Thermotoga maritima RF1 
(11), which are thought to approximate to the structures of the 
 factors free in the cytoplasm, are much more compact.

An additional type of RF, referred to as a class II factor, 
participates in protein synthesis termination in eubacteria and 
eukaryotes. These factors, named RF3 and eRF3, respectively, 
are ribosome-dependent and class I RF-dependent GTPases (12). RF3 catalyzes the dissociation of class I factors from 
the ribosome following peptide release (13). eRF3 may perform a similar role in eukaryotes (1, 14), although this has not yet 
been demonstrated but has also been shown to interact with 
other cell components and may have other roles (15). eRF1 and 
eRF3 form a complex in the absence of ribosomes, which 
involve the C-terminal domain of each protein (16). No such 
interaction between bacterial RF1 or RF2 and RF3 has been 
observed in the absence of ribosomes, but when both are bound 
to the ribosome, they are thought to interact in a way that 
involve the N-terminal domain of RF1 or RF2, which would 
then be functionally analogous to the C-terminal domain of 
eRF1 (Fig. 1) (17, 18).

In E. coli, both RF1 and RF2 are post-translationally modi-
fied by methylation of the Gln residue in the GGQ motif to give 
N°-Me-Gln (19). The methyltransferase (MTase) involved has 
been identified and is coded in E. coli and in most bacteria by 
a gene, hemK (now named prmC), located immediately down-
stream of prfA, encoding RF1 (20, 21). In data bank 
annotations, PrmC has been classed among MTases methylating N°-

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ences. 

‡ The abbreviations used are: RF, release factor; AdoMet, S-adenosyl-
l-methionine; MTase, methyltransferase; PMSF, phenylmethyl-
sulfonyl fluoride; MALDI-TOF, matrix-assisted laser desorption ion-
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6-Me, methyl.
adenine or N⁴-cytidine in DNA because of the presence of S-adenosyl l-methionine (AdoMet) binding motifs and of an NPPY motif thought to be a hallmark of this class of MTases. It is now understood that the real specificity of the (D/N)PPY motif thought to be a hallmark of this class of MTases is for a nitrogen conjugated to a planar system, which may be an amide moiety or a nucleotide base (22). Pairs of apparent homologues to bacterial prmc, one member of which probably encodes a protein targeted to the mitochondrion, are present in the genomes of eukaryotic organisms including yeast, fly, mouse, and man (20). So far, no genetic or biochemical evidence has existed to ascribe a function to any of these supposed MTases.

The perfect conservation of the GGQ motif in RFs from all of the kingdoms and the widespread presence of genes in higher organisms potentially encoding homologues of prmc, one member of which probably encodes a protein targeted to the mitochondrion, are present in the genomes of eukaryotic organisms including yeast, fly, mouse, and man (20). So far, no genetic or biochemical evidence has existed to ascribe a function to any of these supposed MTases.

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were concentrated by ultrafiltration (Amicon Ultra 10 molecular weight cut-off, Millipore) and dialyzed against 10 mM Tris-HCl, pH 7.6, 50 mM KCl, 10 mM Mg acetate, 6 mM β-mercaptoethanol, and 0.1 mM PMSF, and 50% glycerol and stored at −20 °C. An analysis by SDS-PAGE and Western blotting using polyclonal anti-YDR140w antibodies showed the presence of YDR140w protein only in extracts from cells containing pYeVH407. Several protein impurities were visible in preparations of YDR140w but were present in the control extracts as well.

**Purification of His-tagged eRF1—** Cultures of strain yLM6 in 4 l of rich medium (YPD), 2% glucose (23) were grown to an optical density at 600 nm of ~5. The cells were resuspended in 20 ml of 50 mM sodium phosphate buffer, pH 7.5, 5 mM β-mercaptoethanol, and 0.1 mM PMSF with EDTA-free anti-protease at twice the concentration recommended by the manufacturer. The cells were broken by passage through a French press, and the supernatant after centrifugation for 1 h at 30,000 × g was made 8 M in urea by the addition of the solid product and readjusted to pH 7.5 with NaOH. The solution was loaded on Mono Q fast-flow (Amersham Biosciences). The column was washed with 50 mM sodium phosphate buffer, pH 7.5, 8 μM urea, and 50 μM PMSF (buffer B) and eluted at 2 ml/min with a 100-ml gradient from 0 to 0.6 M NaCl in buffer A. The fractions containing eRF1 were identified by SDS-PAGE and Western blot, pooled, and concentrated by ultrafiltration (Amicon Ultra 10 molecular weight cut-off, Millipore) to 1 M in 40% acetic acid with 0.5 mM 2-mercaptoethanol. The solution was loaded on a column of Mono Q fast-flow (Amersham Biosciences) according to the manufacturer’s instructions, and proteins were then fractionated by SDS-PAGE (10% gel), and His6-tagged eRF1 was located by Western blot performed on a lane containing ~5% of the preparation. A gel fragment containing the remaining amount of the identified protein was excised, washed, dehydrated, and resuspended in 20 μg of a trypsin solution (sequencing grade, Roche Applied Science, 15 ng/μl in 5 mM CaCl2, 25 mM HCHO2), and incubated overnight at 37 °C. Peptides were desalted on C18 Zip-Tips (Millipore) for analysis by mass spectrometry.

**Mass Spectrometry—** MALDI-TOF mass spectrometry analysis was performed with an Applied Biosystems Voyager-DE PRO spectrometer equipped with a pulsed nitrogen laser (337-nm, 3-ns pulse) at the Department of Protein Engineering and Metabolic Control (Institut Jacques Monod, Paris, France). Operating parameters for reflectron mass spectrometry were as follows: accelerating voltage (20 kV); grid voltage (75%); reflectron voltage (50%); and laser parameters (100,000 × g supernatant from E. coli cells overproduced in wild-type yeast strain W303–1B from the high-density filter paper). The source of PrmC was a 100,000-g supernatant from E. coli cells overproduced in wild-type yeast strain W303–1B from the high-density filter paper. The source of PrmC was a 100,000-g supernatant from E. coli cells overproduced in wild-type yeast strain W303–1B from the high-density filter paper.

**Identification of a MTase for eRF1 Methylation in Vivo—** A search of the S. cerevisiae genome revealed two genes, YDR140w and YNL063w, with significant homology to prmC in E. coli, going beyond the motifs that are known to be involved essentially in the binding AdoMet. The two genes probably encode a protein targeted to the mitochondrion (20). To determine whether the YDR140w gene might be required for eRF1 methylation, it was inactivated by insertion of a kanamycin-resistant cassette. The resulting strain (yVH11) was viable but grew slowly with a doubling time of 185 min in Hartwell complete synthetic medium containing 2% glucose as compared with 90 min for the parent strain (results not shown). The complementation of yVH11 by plasmid pYeVH407 carrying the YDR140w gene restored the growth rate to that of the wild type strain, yLM6. These results are consistent with the experiments of Niewmierzycka and Clarke (29) and the growth data obtained during systematic deletion studies in S. cerevisiae (30). When His-tagged eRF1 was prepared from the mutant strain yVH11, digested with trypsin, and analyzed by mass spectrometry, no fragment with a mass of 702.3 corre-
Methylation of S. cerevisiae eRF1 by YDR140w

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Summary

Here we show that eRF1 from S. cerevisiae is methylated on the Gln residue of the conserved GGQ motif, as in bacterial RFs, despite the absence of further sequence homology between these different protein families. Only two instances of Gln methylation have previously been described, both in eubacteria. The first case was the methylation of Gln-150 in ribosomal protein L3 in E. coli (31), which may be important for ribosome assembly. Gln-150 in L3 is not universally conserved in bacteria, but when present, the gene encoding the L3 MTase, prmB (or yfcB), also appears to be conserved. Methylation of bacterial RF1 and RF2 is the only other documented case of Gln methylation, and the MTase responsible, PrmC (or HemK), appears to be universal in eubacteria. Inactivation of prmC is lethal for E. coli K12 strains on minimal media and almost abolishes growth on rich media. However, this phenotype is peculiar to E. coli K12 laboratory strains and is related to the nature of the amino acid residue at position 246 in E. coli K12 RF2, very close to the Gln-252 residue methylated by PrmC. This residue is Thr, whereas in every other sequenced RF1 and RF2 gene in all eubacteria including E. coli strains other than K12, the residue...
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TABLE I
Methylation in vitro of RFs from S. cerevisiae and E. coli with MTase preparations

| MTase* | RF1 | eRF1(sc) | eRF1(sc) + eRF3 | eRF1(sc) + eRF3 + GTP | eRF1(sc) + eRF3 + GDP | eRF1(sc) + eRF3 + GTP |
|--------|-----|----------|-----------------|------------------------|-----------------------|------------------------|
| PrmC   | 12.5| ND       | ND              | <0.05                  | ND                    | <0.05                  |
| YDR140w| <0.05| <0.05   | <0.05           | 5.5                    | <0.05                 | 9.5                    |
| control| ND  | ND       | ND              | <0.05                  | ND                    | <0.05                  |

* MTases were the RF MTase PrmC from E. coli, partially purified His-tagged YDR140w protein overproduced in S. cerevisiae, or a control preparation made similarly except that cells were transformed with the parent plasmid pYeDP60 instead of plasmid pYeVH407.

Substrates were E. coli RF1, S. cerevisiae eRF1 (eRF1(sc)), human eRF1 (eRF1(h)) in the presence or absence of S. cerevisiae eRF3 and GDP or GTP.

FIG. 3. Analysis of products methylated in vitro. The products of the methylation reaction in vitro in the presence of S. cerevisiae eRF1 with a C-terminal His6 tag, eRF3 (N-terminally truncated and His6-tagged), GTP, and \([methyl-\text{\textsuperscript{3}}H]\)AdoMet were separated by SDS-PAGE on a 9% polyacrylamide gel and further analyzed by fluorography and Western blotting. Track 1, proteins stained with Coomassie Blue. Track 2, radioactivity revealed by fluorography. Track 3, Western blot using polyclonal anti-eRF1 antibodies. The arrows on the right show the positions of molecular mass standards (in kDa), and those on the left show the positions of His6-tagged S. cerevisiae eRF1 and His6-tagged N-terminally truncated eRF3.

FIG. 4. Analysis of amino acids from RFs labeled in vitro with [methyl-\text{\textsuperscript{3}}H]AdoMet as methyl donor. A, the products of in vitro methylation reactions of RFs in the presence of [methyl-\text{\textsuperscript{3}}H]AdoMet and S. cerevisiae eRF3 (N-terminally truncated and His6-tagged) and GTP in the case of yeast and human eRF1 were precipitated with trichloroacetic acid and digested with Pronase, and the amino acids were separated by TLC as described under “Experimental Procedures.” Triangles, S. cerevisiae His6-tagged eRF1; squares, E. coli RF1; circles, His6-tagged human eRF1. B, amino acid standards chromatographed in parallel and visualized with ninhydrin.

is Ala or Ser. In K12 strains, revertants to normal growth of prmc mutant strains are readily obtained and show the replacement of Thr-246 in RF2 by Ser or Ala. Either of the two circumstances, the presence of Thr rather than Ala/Ser or the lack of Gln methylation, reduces the termination efficiency of RF2 in vitro (19). The effect of the two factors is cumulative, and we interpret these observations to mean that the termination machinery can tolerate the effect either of the presence of Thr rather than Ala/Ser or the lack of Gln methylation but that the accumulated effect of both factors reduces termination efficiency at some stop codons to a level incompatible with cell growth. For reasons that remain to be determined, PrmC in Yersinia pseudotuberculosis is required for virulence, although not for growth in rich liquid media (32).

A previous analysis (20) of HemK-related proteins showed three clearly defined subgroups. The smallest family was typified by YfB (or PrmB) (31), which was identified biochemically as the N\textsuperscript{5}-Me-Gln MTase modifying ribosomal protein L3 in E. coli (20). The second subfamily included HemK, for which the gene is now renamed prmc, that was shown to be the N\textsuperscript{5}-Me-Gln MTase specific for eubacterial RFs. Included in the HemK subfamily are proteins from S. cerevisiae (YNL063w), Schizosaccharomyces pombe (SPAC29B12.05c), man (Q9Y5R), mouse (Q921L7), and Drosophila (CG9531) that diverge significantly from the main group and are predicted to be targeted to mitochondria (20). These proteins may be MTases specific for mitochondrial RFs, but no biochemical or genetic evidence is at present available to support this supposition. These two subgroups are classed together in data banks as bearing the InterPro family assignment of sequences from both the PrmC/HemK and HemK-related families that we characterize here is the first protein of this family to which biochemical experiments have allowed a function to be assigned. These proteins diverge in sequence from the HemK family in both the N- and C-terminal regions. A partial alignment of sequences from both the PrmC/HemK and HemK-rel-arch families is shown in Fig. 5. The crystal structure of PrmC/HemK from T. maritima has been solved and shows the protein to consist of two domains, an N-terminal domain that may contribute to the binding of the RF substrate and a C-terminal domain typical of class I AdoMet-dependent MTases (22). Fig. 3 shows that the residues that constitute the N-terminal do-
Methanococcus jannaschii
PYRAB
TrEMBL databases.
indicates the last residue shown and the total number of residues in the protein. Sequence data and accession numbers are from the Swiss-Prot/
METTH
(ATCC);
S. pombe
eubacterial sequences are derived from the crystal structure of
followed by the accession numbers for the sequence.

PrmC/HemK proteins. The helical regions (spirals), β-strands (arrows), and the limits of the N-terminal, linker, and catalytic domains are shown in the crystal structure of T. maritima PrmC/HemK (22). Boxes show the most conserved of the AdoMet binding motifs (m1) and the NPPY motif (m2) of the catalytic center common to both families of MTases. The abbreviations for the organisms are followed by the accession numbers for the sequence. A, eubacterial sequences. ECOLI, E. coli; YERPE, Yersinia pestis; VICHIO, Vibrio cholerae; SHONE, Shewanella oneidensis; THMAR, T. maritima. B, eukaryotic/archebacterial sequences. SULSO, Sulfolobus solfataricus; SCPOM, S. pombe; HUMAN, Homo sapiens; ECOLI, Drosophila melanogaster; SCERC, S. cerevisiae; CAEEL, Caenorhabditis elegans; GIALA, Giardia lamblia (ATCC); METTH, Methanobacterium thermoautotrophicum; METKA, Methanopyrus kandleri; THACI, Thermoplasma acidophilum; MATJA, Methanococcus jannaschii; PYRAB, Pyrococcus abyssi; HALSF, Halobacterium sp. The C-terminal residues are not shown. The final column indicates the last residue shown and the total number of residues in the protein. Sequence data and accession numbers are from the Swiss-Prot/TrEMBL databases.

FIG. 5. Partial sequence alignment of PrmC/HemK homologues from eubacteria and members of the HemK-rel-arch family from eukaryotes and archaebacteria, showing the absence in the HemK-rel-arch family of sequences corresponding to domain 1 in PrmC/HemK proteins. The helical regions (spirals), β-strands (arrows), and the limits of the N-terminal, linker, and catalytic domains are shown in the crystal structure of T. maritima PrmC/HemK (22). Boxes show the most conserved of the AdoMet binding motifs (m1) and the NPPY motif (m2) of the catalytic center common to both families of MTases. The abbreviations for the organisms are followed by the accession numbers for the sequence. A, eubacterial sequences. ECOLI, E. coli; YERPE, Yersinia pestis; VICHIO, Vibrio cholerae; SHONE, Shewanella oneidensis; THMAR, T. maritima. B, eukaryotic/archebacterial sequences. SULSO, Sulfolobus solfataricus; SCPOM, S. pombe; HUMAN, Homo sapiens; ECOLI, Drosophila melanogaster; SCERC, S. cerevisiae; CAEEL, Caenorhabditis elegans; GIALA, Giardia lamblia (ATCC); METTH, Methanobacterium thermoautotrophicum; METKA, Methanopyrus kandleri; THACI, Thermoplasma acidophilum; MATJA, Methanococcus jannaschii; PYRAB, Pyrococcus abyssi; HALSF, Halobacterium sp. The C-terminal residues are not shown. The final column indicates the last residue shown and the total number of residues in the protein. Sequence data and accession numbers are from the Swiss-Prot/TrEMBL databases.

main in PrmC/HemK proteins are absent from the HemK-rel-arch family and that the main region of sequence similarity lies in the center of the molecule, spanning the Gly-rich AdoMet binding motif VI/D/E(X)XG(G) (Fig. 5, m1) to the NPPY motif (Fig. 5, m2) of the catalytic domain.

The striking observation that the methylation of S. cerevisiae eRF1 requires the presence of eRF3 to be consistent with the fact that the proteins readily form a heterodimer in the absence of ribosomes. The need for eRF3 also adds a new perspective to the observation that yeast cells require eRF3 for viability. Whether this requirement for eRF3 is related to the role of the factor in termination or to some other role, such as messenger circularization (33) or the regulation of mRNA deadenylation (34), is uncertain. In either case, our results suggest that the process may be independent of a class II RF because no homologue of eRF3 appears to be present in any completely sequenced archaebacterial genome. Further studies will be required to determine how the two types of RF MTase recognize their respective substrates and to understand the role of Gln methylation in the process of translation termination.

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Protein kinase C βII regulates Akt phosphorylation on Ser-473 in a cell type- and stimulus-specific fashion.

Yuko Kawakami, Hajime Nishimoto, Jiro Kitaura, Mari Maeda-Yamamoto, Roberta M. Kato, Dan R. Littman, Michael Leitges, David J. Rawlings, and Toshiaki Kawakami

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The glutamine residue of the conserved GGQ motif in Saccharomyces cerevisiae release factor eRF1 is methylated by the product of the YDR140w gene.

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Dr. Merkulova-Rainon’s last name was misspelled. The correct spelling is shown above.

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