A Novel Role of the Mammalian GSPT/eRF3 Associating with Poly(A)-binding Protein in Cap/Poly(A)-dependent Translation

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The mammalian GSPT, which consists of amino-terminal (N) and carboxyl-terminal (C) domains, functions as the eukaryotic releasing factor 3 (eRF3) by interacting with eRF1 in translation termination. This function requires only the C-domain that is homologous to the elongation factor (EF) 1α, while the N-domain interacts with polyadenylate-binding protein (PABP), which binds the poly(A) tail of mRNA and associates with the eukaryotic initiation factor (eIF) 4G. Here we describe a novel role of GSPT in translation. We first determined an amino acid sequence required for the PABP interaction in the N-domain. Inhibition of this interaction significantly attenuated translation of capped/poly(A)-tailed mRNA not only in an in vitro translation system but also in living cells. There was a PABP-dependent linkage between the termination factor complex eRF1-GSPT and the initiation factor eIF4G associating with 5′ cap through eIF4E. Although the inhibition of the GSPT-PABP interaction did not affect the de novo formation of an 80 S ribosomal initiation complex, it appears to suppress the subsequent recycle of ribosome. These results indicate that GSPT/eRF3 plays an important role in translation cycle through the interaction with PABP, in addition to mediating the termination with eRF1.

The process of eukaryotic protein biosynthesis is divided into three steps: initiation, elongation, and termination. Among them, termination had been the least investigated aspect. However, the identification of two releasing factors, eRF1 (1) and eRF3 (2), provided a breakthrough in understanding the terminating with eRF1. These results indicate that GSPT/eRF3 plays an important role in translation cycle through the interaction with PABP, in addition to mediating the termination with eRF1.

EXPERIMENTAL PROCEDURES

Plasmids—For production of N-terminally GST-fused GSPT2 mutants, PCR products were inserted to pGEXt1 (Amersham Biosciences). To produce full-length GSPT2 and its deletion mutants fused with N-terminally GST and C-terminally His6 epitope, respectively, the SalI-Ncol fragment of pGEXex1 vector (Amersham Biosciences) was ligated with the synthetic adaptor HIS (HIS5‘ plus HIS3‘ as described below) to make pGP6. PCR products encoding the corresponding sequences (amino acid sequence 1–632, 1–204, 58–204, and 80–204) of GSPT2 were inserted to pGP6, resulting in pGP2-full, pGP2-1, pGP2-58, and pGP2-80. To produce the amino acid 45–204 of eIF4G I fused
with GST at the N terminus, a PCR product was inserted to pEGEX2p1 to make pEGEX2p1a. To express PABP in mammalian cells, human PABP I DNA was inserted to pFlag-CMV-2 (Eastman Kodak Co.) to make pFlagPABP. PCR products encoding the N-domain or C-domain of GSPT2 were also inserted to pFlag-CMV-2 to produce pFlagGSPT2pN and pFlagGSPT2pC, respectively. To construct pcDNA3/GSPT2a/aa 1–204–His6, GSPT2a/aa 1–204–His6 cDNA excised from pGL3-T7 was inserted to pcDNA3 (Invitrogen). pcDNA3/GSPT2a/aa 19–204–His6, aa 36–204–His6, aa 58–204–His6, aa 80–204–His6, and aa 1–204–65–71–His6 were created from pcDNA3/GSPT2a/aa 1–204–His6 by the Kunkel method. The plasmid to express N-terminally FLAG-tagged GSPT2 and eRF1 was previously described (4). To construct a luciferase reporter gene, the BglII-NcoI fragment of pGL3-Basic vector (Promega) was ligated with the synthetic adenovirus T7 (T75 plus T73 as described below), which encodes T7 promoter to make pGL3-T7. The XbaI-BamHI fragment of the pGL3-T7 was then ligated with the synthetic adaptor pA55 (pA55 plus pA23 as described below) to make pGL3-T7-PA. To construct pUC18-T7-R-luc-HCV IRES-F-luc, T7 promoter, Renilla luciferase (R-luc), hepatitis C virus IRES, and firefly luciferase (F-luc) were placed in this order in the multicloning site of pUC18. The synthetic oligonucleotides used were: HIS5’-TCG ACC ATC ATC ATC ATC ATC ATC ATC ATT GGC CCG CCA ATG ATG ATG ATG ATG, pT75’-GAT CCT AAT ACG ACT CAT TAT AGG CCT ACG TCT GTA GAC, pT73’-CAT GGT CGA CAA GCT TAG GCC TAT ACG TCG TAT A, pA55’-CTA GA55G, and pA23’-CAT GGT CGA CAA GCT TAG GCC TAT ACG TCG TAT A.

Production of Recombinant Proteins—Proteins were induced by the addition of 0.1 mM isopropyl-1-thio-

β-D-galactoside to 37°C for 3 h in Escherichia coli JM109. The cells were resuspended in buffer A consisting of 50 mM Tris-HCl (pH 8.0), 1 mM EDTA, 150 mM NaCl, 1% Nonidet P-40, 5 µg/ml aprotinin, 100 µM phenylmethylsulfonyl fluoride, and 2 µg/ml of leupeptin. After incubation with 1 mg/ml lysosyme at 4°C for 30 min, the cell lysate was sonicated for 3 min on ice. The supernatant after centrifugation at 100,000 × g for 60 min was subjected to glutathione-Sepharose 4B (Amersham Biosciences) and/or Ni-NTA-agarose (Qiagen). If necessary, GST was removed using PreScission™ Protease (Amersham Biosciences). The purified proteins were dialyzed against buffer B consisting of 20 mM Tris-HCl (pH 8.0), 500 mM NaCl, and 1% Nonidet P-40. PABP I was purified as described previously (10).

Cell Culture, DNA Transfection, and in Vivo Translation Assay— COS-7 and HeLa cells were cultured in Dulbecco’s modified Eagle’s medium (Invitrogen) containing 10% fetal calf serum and maintained at 37°C in 5% CO2. Transfections were performed with Lipofectin

(Amersham Biosciences). HeLa cells that had been transfected with pcDNA3/GSPT2 mutants and a reporter pUC18-T7-R-luc-HCV IRES-F-luc were incubated for 40 h and infected with vaccinia virus VTF-3 (25) for 4 h. Dual luciferase activities were measured using Stop & Glo luciferase assay system (Promega).

In Vitro Binding Assay—Recombinant GST-fused proteins were incubated with glutathione-Sepharose 4B for 30 min at 4°C. After removal of the unbound fraction, the resin was mixed with recombinant PABP in buffer B and further incubated at 4°C for 30 min. The resin was washed with buffer B and incubated with synthetic peptides or recombinant proteins at 4°C for 60 min. After washing with buffer B, proteins were eluted from the resin with SDS-polyacrylamide sample buffer and subjected to SDS-PAGE and immunoblot analysis.

Immuno precipitation and Ni-NTA Pull-down Assay—The transfected cells were lysed in buffer C consisting of 20 mM Tris-HCl (pH 8.0), 50 mM NaCl, 1% Nonidet P-40, 1 mM dithiothreitol, 10 µg/ml bovine serum albumin, 100 µM phenylmethylsulfonyl fluoride, 2 µg/ml of aprotinin, and 2 µg/ml of leupeptin. After centrifugation at 15,000 × g for 30 min, the supernatant was incubated with 4°C for 30 min with anti-FLAG IgG-agarose (Sigma) or Ni-NTA-agarose, and then the resin was washed with buffer C. As the need arose, recombinant proteins or synthetic peptides were added, and the resin was further incubated at 4°C for 30 min. The supernatant was subjected to SDS-PAGE and immunoblot analysis. Immuno precipitation from nucleosorb-treated rabbit reticulocyte lysate (RRL, Promega) was performed in the same manner as described above using an anti-GSPT polyclonal antibody and protein A-agarose 4B (Amersham Biosciences).

In Vitro Translation Assay— Luciferase mRNAs containing poly(A) tail or not were synthesized with T7 RNA polymerase after linearization of pGL3-T7-PA with BamHI or XhoI, respectively. When capped mRNAs were synthesized, m2GpppG (Stratagene) was used. In vitro translation reaction was performed as described below. Nuclease-
appear to be independent of RNA tethering because cell extracts had been treated with RNase. Thus, these experiments show that GSPT associates with PABP and eRF1 via its N-domain and C-domain, respectively, in living cells, and consequently GSPT mediates the association between eRF1 and PABP.

Identification of the Site Critical for PABP-Binding in the N-domain of GSPT2—To identify a PABP-binding sequence in the N-domain of GSPT, a co-precipitation assay was performed using COS-7 cells expressing deletion mutants of the N-domain (Fig. 2A). As shown in Fig. 2B, deletion mutants starting from amino acid positions 1, 19, 36, and 58 interacted with PABP (lanes 7–11), while a mutant starting from the 80th amino acid (lane 2) did not associate with PABP (lane 12). Since the expression of this mutant (aa 80–204) was rather low in COS-7 cells, we performed a binding assay using recombinant proteins. GSPT2/aa 58–204 associated with PABP, but aa 80–204 did not (Fig. 2C). Furthermore, GSPT2/aa 1–79 but not aa 78–141 was sufficient for the PABP binding (Fig. 2D). Thus, the amino acid sequence 58–79 of GSPT2 (see Fig. 2A) was identified as a critical region for PABP binding. This sequence is conserved well between GSPT1 and GSPT2 in mice and humans (4).

The significance of the identified sequence was further investigated using a synthetic peptide corresponding to amino acid 58–75 (Fig. 3A). As shown in Fig. 3, B and C, the GSPT-PABP interaction was progressively inhibited with increasing amounts of the whole N-domain or the synthetic peptide but not with GST or a control peptide consisting of the same amino acid composition in a scrambled order (see Fig. 3A). The complete inhibition of the interaction by the synthetic peptide supports the notion that the sequence aa 58–75 of GSPT2 constitutes a critical site for the PABP-binding. However, since the half-maximum inhibition by the synthetic peptide was observed at about 100 μM, which is almost three orders of magnitude higher than that of the whole N-domain (Fig. 3C), we cannot exclude the possibility that other regions might also be involved in the interaction.

Involvement of the Interaction between the N-domain of GSPT and PABP in Cap/Poly(A)-dependent Translation—PABP was reported to regulate translation in a cap/poly(A)-dependent manner by mediating the interaction between the cap-binding complex eIF4F and the poly(A) tail of mRNA (21–23). To investigate whether the interaction between GSPT and PABP is involved in cap/poly(A)-dependent translation, we utilized nuclease-treated RRL as a cell-free translation system. It was previously reported that the synergistic stimulation by cap and poly(A) was observed in the RRL system with partial removal of ribosome and the associated initiation factors (26, 27). However, such a synergistic stimulation was observed only by changing the concentrations of MgCl2 and KCl (Fig. 4A). In this system, cap-dependent translation was markedly stimulated by the simultaneous presence of the poly(A) tail, while mRNA containing only the poly(A) tail had little activity. The N-domain of GSPT2 fused to GST markedly inhibited the cap/poly(A)-dependent translation (Fig. 4B, closed triangles). Interestingly, the N-domain was also capable of inhibiting translation of capped and non-poly(A)-tailed mRNA (closed circles). In contrast, GST alone had little effect on any of the mRNAs (open
circles and triangles). The effect of the synthetic peptides was also investigated. The synthetic peptide aa 58–75 inhibited translation not only of capped/poly(A)-tailed mRNA but also of capped mRNA (Fig. 4C). In accordance with the results in Fig. 3, the concentration of the synthetic peptide required for translation inhibition was much higher than that of the whole N-domain of GSPT2. Thus, both the GSPT-PABP interaction and the cap/poly(A)-dependent translation are inhibited by the synthetic peptide aa 58–75 and the whole N-domain in a similar concentration-dependent manner. In addition, the results presented here suggest that the interaction between GSPT and PABP may also be involved in poly(A)-independent translation (Fig. 4, B and C). Although the exact mechanism is still unclear, it is noteworthy that PABP was reported to stimulate translation, which is in sharp contrast to the results obtained with capped/poly(A)-tailed mRNA. These results suggest that the GSPT-PABP interaction is not involved in translation termination or elongation.

No Involvement of Paip1 in the Inhibition of Cap/poly(A)-dependent Translation by the N-domain of GSPT—In addition to GSPT, two other proteins that interact with PABP have been reported. One is Paip1 identified as a translation activator and the other is Paip2 identified as a translation repressor. In this study, we used RRL immunodepleted of Paip1 by anti-Paip1 antibodies. As shown in Fig. 5A, Paip1 completely depleted, but PABP and GSPT were little affected. Under these conditions, significant effect was not observed in cap/poly(A) synergy (Fig. 5B), and the N-domain of GSPT2 had still inhibitory effect on translation (Fig. 5C). These results indicate that the inhibitory effects of the N-domain of GSPT are independent of Paip1.

The N-domain of GSPT Inhibits Cap/poly(A)-dependent Translation in Living Cells—To confirm that the GSPT-PABP interaction is indeed involved in cap/poly(A)-dependent translation in living cells, we examined the effect of overproducing ribosome (28), and if the elongation step is inhibited, luminescence would also diminish. As shown in Fig. 4D, the N-domain of GSPT2 had no inhibitory effect on the cap/poly(A)-independent translation, which is in sharp contrast to the results obtained with capped/poly(A)-tailed mRNA. These results suggest that the GSPT-PABP interaction is not involved in translation termination or elongation.
the N-domain of GSPT2 on translation by monitoring the synthesis of R-luc and F-luc from the bicistronic construct T7-R-luc- HCV IRES-F-luc was illustrated. B and D, HeLa cells that had been transfected with pcDNA3/GSPT2 mutants and the reporter plasmid were infected with vaccinia virus to express T7 RNA polymerase. The cells were assayed for dual luciferase activities. Results are averages of three independent assays with standard deviations from the means as percentages of the value obtained with pcDNA3. The ratios of Renilla luciferase/firefly luciferase are illustrated with bars, and closed circles show the firefly luciferase activity. C, this experiment was performed as described in Fig. 2B. Asterisks indicate the position of the GSPT2 mutants.

FIG. 6. The N-domain of GSPT inhibits cap/poly(A)-dependent translation in living cells. A, a reporter mRNA expressing Renilla and firefly luciferases from the bicistronic construct pUC18-T7-R-luc- HCV IRES-F-luc was illustrated. B and D, HeLa cells that had been transfected with pcDNA3/GSPT2 mutants and the reporter plasmid were infected with vaccinia virus to express T7 RNA polymerase. The cells were assayed for dual luciferase activities. Results are averages of three independent assays with standard deviations from the means as percentages of the value obtained with pcDNA3. The ratios of Renilla luciferase/firefly luciferase are illustrated with bars, and closed circles show the firefly luciferase activity. C, this experiment was performed as described in Fig. 2B. Asterisks indicate the position of the GSPT2 mutants.

FIG. 7. GSPT and eIF4G form the complex mediated through PABP. A, RRL was incubated with the anti-GSPT antibody (lane 3) or preimmune serum (lane 2) immobilized to protein A-agarose. Proteins that associated with the resins (lanes 2 and 3) and the lysate (lane 1) were analyzed by SDS-PAGE and immunoblot with anti-eIF4G (upper), anti-PABP (middle), and anti-GSPT (lower) antibodies. Asterisks indicate the position of GSPT. B, GST-fused eIF4G/aa 45–204 (lanes 3–5) or GST (lane 2) was immobilized to glutathione-Sepharose and incubated with the purified PABP and/or GSPT2-His6. Proteins that associated with the resin (lanes 2–5) were resolved by SDS-PAGE and immunoblotted with anti-His (upper), anti-PABP (middle), and anti-GST (lower) antibodies. Lane 1 shows the purified GSPT2-His6 and PABP (marked by asterisks) used in the pull-down assay.
GSPT Interacts with PABP through a Site in Its N-domain

We previously presented evidence that GSPT interacts with PABP in in vitro experiments (9). This conclusion was confirmed and further extended by our present experiments. First, the interaction between the N-domain of GSPT and PABP was observed with cell extracts (Figs. 1, 2) and cycloheximide (50 μM) at 30 °C for 15 min in the presence of 4 μM GST-GSPT2(aa 1–204-His)₆ (closed circles) or GST alone (open circles). 20-μl aliquots of the mixture were analyzed on 5 ml of 15–30% linear sucrose gradient. A, a luciferase mRNA (50 ng) containing cap plus poly(A) was used in the translation assay in the presence of 4 μM GST (open circles) or the GST-fused N-domain of GSPT2 (closed circles).

DISCUSSION

GSPT Interacts with PABP through a Site in Its N-domain

We previously presented evidence that GSPT interacts with PABP in in vitro experiments (9). This conclusion was confirmed and further extended by our present experiments. First, the interaction between the N-domain of GSPT and PABP was observed with cell extracts (Figs. 1, 2B, and 7A) and with purified proteins (Figs. 2C and 7B). Moreover, we identified a possible PABP-binding sequence in the N-domain (Figs. 2 and 3). The GSPT2-PABP interaction is mediated at least through the amino acid sequence aa 58–75 of GSPT2, since the synthetase peptide completely inhibited the association (Fig. 3).

In addition to GSPT, Paip1 and Paip2 have been reported to interact with PABP. PABP-binding sites in Paip1 and Paip2 are similar to the sequence aa 58–75 of GSPT2, and this motif is important for their interactions with the C-terminal domain of PABP (19, 20, 30). Thus, GSPT may compete with Paips for PABP binding. However, the relationship between GSPT and Paips may not be so simple, since Paips interact with both the N- and C-terminal regions of PABP (19, 20, 29, 30, 33). In contrast, GSPT interacts only with the C-terminal site (10). A rabbit reticulocyte lysate, which we used in this study, has a much smaller amount of Paips than a rabbit liver lysate when compared with the amount of PABP (data not shown). Thus, it is possible that Paips may be the factors modifying the function of PABP on the requirement of each tissue.

A Novel Role of GSPT/eRF3 in the Eukaryotic Translation System—It is generally believed that the function of GSPT was solely to facilitate the release of completed peptide chains from ribosome as a GTP-dependent stimulator of eRF1. However, the present study reveals that GSPT associates with eIF4G through PABP (Fig. 7) and that the GSPT-PABP interaction is involved in the multiple rounds of translation (Figs. 4, 6, and 8). The synergistic enhancement of translation by cap and poly(A) is explained by the circularization of mRNA, which is mediated through a complex consisting of poly(A)-PABP-eIF4F-cap (21–23). This fact suggests the hypothetical model that a translation-terminating ribosome may be recruited to the next translation initiation. However, this idea is unsatisfactory since translation is terminated at stop codons that are not always close to the poly(A) tail of mRNA. Therefore, some factors are likely to mediate the physical coupling between the terminating ribosome on the stop codon and the poly(A) tail. The fact that GSPT interacts with eRF1 and PABP at the same time (Fig. 1B) suggests that GSPT may be the bridging protein to connect the stop codon with the poly(A) tail. In this hypothesis, a 3′-untranslated region, which locates between a stop codon and a poly(A) tail, could be looped out, and the terminating ribosome could be passed to the 5′ cap structure through the novel protein bridge consisting of eRF1, GSPT, PABP, and eIF4F (Fig. 9).

In addition to the role of PABP in translation, several lines of evidence suggest that PABP might affect translation in a poly(A)-independent manner (18, 20, 34, 35). Furthermore, this function appears to be independent of its binding to eIF4G (24). The results presented here suggest that the GSPT-PABP interaction may also be involved in poly(A)-independent translation (Fig. 4, B and C), though the exact mechanism is still unclear.

It is well established that PABP has another function; it prevents mRNA degradation by protecting the poly(A) tail. In general, mRNA degradation, an important aspect of gene expression, is a strictly regulated process that is often linked to translation (12, 36, 37), and translation-dependent deadenylation is an important step of this mechanism in which PABP is probably involved. Moreover, several reports show that GSPT is involved in nonsense-mediated decay, a mechanism by which mRNAs containing a premature termination codon are rapidly degraded (38, 39). These mechanisms are not well understood, but it is conceivable that they are linked to the translation...
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termination. Further studies on GSPT/eRF3 should be important for the understandings of not only translation machinery but also mRNA-decay mechanism.

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