ATPase Activity of the Terminase Subunit pUL56 of Human Cytomegalovirus*

Received for publication, September 17, 2001, and in revised form, November 13, 2001
Published, JBC Papers in Press, December 13, 2001, DOI 10.1074/jbc.M108984200

Jae-Seon Hwang and Elke Bogner‡
From the Institut für Klinische und Molekulare Virologie, D-91054 Erlangen, Germany

Herpesviral DNA packaging is a complex process resulting in unit-length genomes packed into preformed procapsids. This process is believed to be mediated by two packaging proteins, the terminase subunits. In the case of double-stranded DNA bacteriophages, the translocation of DNA was shown to be an energy-dependent process associated with an ATPase activity of the large terminase subunit. In the case of human cytomegalovirus it was not known which protein has the ability to hydrolyze ATP. In this study we expressed human cytomegalovirus terminase subunits, pUL89 and the carboxyl-terminal half of pUL56, as GST fusion proteins and purified these by affinity chromatography. ATPase assays demonstrated that the enzymatic activity is exclusively associated with pUL56. The characterization of the ATP hydrolysis showed that the enzymatic reaction is a fast process, whereas the spontaneous ATP decay followed slow kinetics. Interestingly, although pUL89 did not show any ATPase activity, it was capable of enhancing the UL56-associated ATP hydrolysis. Furthermore, a specific association of in vitro translated pUL89 with the carboxyl-terminal half of GST-UL56C was detected. This interaction was confirmed by co-immunoprecipitations of infected cells. Our results clearly demonstrated that (i) both terminase subunits interact with each other and (ii) the subunit pUL56 has an ATPase activity.

Human cytomegalovirus (HCMV) DNA replication results in the formation of large head-to-tail DNA concatemers. The subsequent maturation into unit-length molecules involves site-specific cleavage at sequence motifs (pac motifs) located within the α sequence of the terminal and internal repeat segments (1–4). Unit-length genomes are then encapsidated into preassembled capsids. DNA-packaging is a complex biological process. While it is commonly accepted that ATP hydrolysis is the driving force behind it, the molecular mechanisms of DNA translocation and genome packaging remain unclear. In general, the following five steps are involved. (i) The recognition of concatameric DNA by a specific protein (complex) able to (ii) bind and cut the DNA at specific sequence motifs (packaging signals, e.g. pac1 and pac2), (iii) translocation of the DNA-protein complex into the procapsid, (iv) packaging of one unit-length genome, and (v) completion of the packaging process by cutting off excess DNA at the portal region (5–7).

Recently, we have demonstrated that the HCMV pUL56 gene product (pUL56), the homolog of the HSV-1 open reading frame UL28, is associated with sequence-specific binding of DNA containing packaging motifs leading to the suggestion that pUL56 plays a key role in DNA packaging (7–9). Comparable results were reported of the HSV-1 pUL28, demonstrating a direct interaction of pUL28 with DNA containing the pac1 motif (10). Furthermore, by the use of viral mutants it was demonstrated that the deletion of pUL28 leads to nuclear accumulation of naked nucleocapsids and uncleaved concatemeric DNA (11, 12). In addition, it has been noted that the HCMV UL89 gene product, the HSV-1 UL15 homolog, exhibits homology to the bacteriophage T4 gp17 terminase subunit, implying a similar function in DNA packaging (13). However, this homology was only due to the ATP binding motifs. DNA packaging of double-stranded DNA bacteriophage requires the terminase, a complex of two proteins (14–16). Most bacteriophage terminases are hetero-oligomers with each subunit carrying a different function (17–20). The large subunit catalyzes the ATP-dependent translocation of genomic DNA into the bacteriophage procapsids and the small unit binds and cleaves concatenated DNA (21, 22). Mutations in any of the encoding genes lead to an accumulation of empty procapsids (proheads) and DNA concatemers (23). Rao et al. (24) demonstrated that the ATPase activity is associated with the large terminase subunit gp17 of bacteriophage T4.

Here we address the question which component of the HCMV terminase has the ability to hydrolyze ATP. Exclusively, pUL56 exhibited an ATPase activity, defining this protein as the central function in DNA packaging of HCMV. Interestingly, we demonstrated that pUL89 and pUL56 interact.

EXPERIMENTAL PROCEDURES

Bacteria, Plasmid Construction, and Oligonucleotides—Escherichia coli strain BL21 codon plus was used as the host strain for UL56- and UL89-carrying pGEX-5X-1 constructs. The UL89 cDNA was generated by reverse transcription and PCR amplification. PCR primers amplified DNA segments from –17 to +2306 bp of the HCMV (strain AD169) UL89 open reading frame sequence except that a single nucleotide 5' of the initiation codon was deleted to generate an NdeI site. This fragment was cloned into the pCRII vector (Invitrogen, Karlsruhe, Germany) to generate construct pUB8. A BamHI-to-XhoI fragment from pUB8 was cloned into the corresponding sites of the vector pcdNA3.1/His C (Invitrogen) to generate pcDNA-UL89.

Plasmid GST-UL56C was generated by digestion of pRCCMV-UL56 (7) and pGEX-5X-1 (Amersham Biosciences) as described previously (23). The 1.5-kb fragment encoding the carboxyl-terminal half of HCMV pUL56 (amino acids 446–850) was ligated in-frame into pGEX-5X-1, yielding pGEX-UL56C. For construction plasmid GST-UL89, plasmid pGEX-5X-1 was digested with EcoRI and XhoI prior to insertion of a fragment encoding the entire HCMV pUL89. The respective fragment was obtained using plasmid pDNA-UL89 as a template for PCR and the following pair of synthetic oligonucleotides (restrictions sites are

* This study was supported by the Johannes and Frieda Marohn Foundation. The costs of publication of this article were defrayed in part by the payment of page charges. This article must therefore be hereby marked “advertisement” in accordance with 18 U.S.C. Section 1754 solely to indicate this fact.
‡ Recipient of a Habilitationstipendium from the Deutsche Forschungsgemeinschaft. To whom correspondence should be addressed: Institut für Klinische und Molekulare Virologie, Schlossgarten 4, 91054 Erlangen, Germany. Tel.: 49-9131-8522104; Fax: 49-9131-8526493; E-mail: eebogner@viro.med.uni-erlangen.de.
§ The abbreviations used are: HCMV, human cytomegalovirus; HSV, herpesvirus simplex.
underlined): 5’-CCGGAATTCATGGTCAGGCAGACTGCGCC-3’ (forward nucleotide) and 5’-CCGTCGAGCTAGCTGACCCTGCACGAAAGCGGATC-3’ (reverse nucleotide).

Protein Purification—A fresh overnight culture of E. coli BL21 carrying the plasmid pGEX-UL56C or pGEX-UL89 or pGEX-5x-1 was diluted 1:100 in LB medium (1 liter) containing 50 μg/ml ampicillin and incubated at 37 °C. After the cells reached an A600 of 0.5 the GST protein expression was induced by addition of isopropyl-1-thio-

β-galactosidase to a final concentration of 0.1 mM and incubated for 2 h at 37 °C. Sedimented cells were lysed in 40 ml of binding buffer (20 mM sodium phosphate, 0.15 mM NaCl, pH 7.4) containing 250 μl of 1 mM MgCl2, 25 μl of 1 mM MnCl2, 40 μl of DNase I (10 mg/ml), and 400 μl of lysozyme (10 mg/ml) incubated on ice for 30 min and sonicated on ice. Undissolved material was sedimented at 5000 × g at 4 °C and passed through a 0.2-μm filter. The purification was performed with a 1-ml GSTrap™ column by using a AKTA™ prime machine (Amersham Biosciences) according to the instructions of the manufacturer. The column was washed with three bed volumes binding buffer prior to loading the proteins. Elution was performed with 10 bed volumes elution buffer (50 mM Tris-HCl, 10 mM glutathione, pH 8.0), and ten fractions were collected. The fractions were analyzed by SDS-PAGE. Fractions containing the proteins were stored at −80 °C.

In Vitro Translation—Plasmid pcDNA-UL89 (0.5 μg) and plasmid pHM123 (0.5 μg), encoding HCMV IE1, was incubated with [35S]methionine (10 μCi/ml) and 20 μl TNT™ Quick Master Mix (Promega, Madison, WI) in a final volume of 30 μl for 1 h at 30 °C. Translation products were analyzed by SDS-PAGE.

Antibodies—HCMV pUL56-specific human polyclonal antibody pabUL62 (7), which was purified from high titer human serum by column affinity chromatography (Affi-Gel 10/15/π-UL56). Affinity purification of the anti-UL56 antibody was done as follows. The GST fusion protein, GST-UL56, was coupled to activated immunoaffinity supports Affi-Gel 10 and Affi-Gel 15 (1:2 Affi-Gel 10/Affi-Gel 15; Bio-Rad Laboratories) as described by Bio-Rad, yielding Affi-Gel 10/15-pUL56. Unspecific binding sites were blocked by incubation with buffer B (1 mM ethanolamine-HCl, pH 8.0, 0.02% NaN3) for 1 h. The resin was equilibrated with start buffer (150 mM NaCl, 50 mM Tris-HCl, pH 8.0, 0.02% NaN3). High titer human convalescent serum was incubated overnight with the purified antigen. After the matrix was washed twice with 10 bed volume of ice-cold start buffer, the bound antibody was eluted in 200 mM glycine-HCl, pH 2.5. The purified anti-pUL56 antibody, pabUL62, was neutralized with 100 mM Tris-HCl, pH 8.0.

PAGE and Western Blot Analysis—The fractions of the purified GST fusion proteins were separated on 15% (w/v) polyacrylamide gel, transferred to nitrocellulose sheets, and subjected to Western blot analysis as described previously (7). The primary antibodies used were pabUL62 antibody (1:10; 25) specific for pUL62 and pabUL89 antibody (1:10) specific for pUL89.

Co-immunoprecipitation—For immunoprecipitation total cell extracts were prepared from HCMV-infected cultures 72 h post infection by solu-
**ATPase Activity**—Purified GST fusion proteins GST-UL89, GST-UL56C, or the control proteins GST (each 1.2 μg) and Apyrase were incubated with 1 μCi of [γ-32P]ATP (specific activity 2500 Ci/mmol), [α-32P]ATP (specific activity 3000 Ci/mmol), [α-32P]dATP (specific activity 3000 Ci/mmol), or [γ-32P]GTP (specific activity 3000 Ci/mmol) in 30 μl of nuclease buffer. The samples were incubated at 37 °C for 1 h. The reaction was terminated by adding EDTA up to a final concentration of 50 mM. A 1-μl aliquot of the reaction mixture was applied onto a 20-cm-long PEI-F cellulose strip (Merck Eurolab, Darmstadt, Germany) and dried. The chromatogram was developed in 1 M formic acid and 0.5 M LiCl according to Guo et al. (26). The run was stopped when the solvent front reached 1 cm from the upper edge, dried, and autoradiographed on Kodak BIOMAX MR. Radioactive signals were quantified graphed on Kodak BIOMAX MR. Radioactive signals were quantified.

**RESULTS**

**Purification of GST-UL56 C and GST-UL89**—For this purpose E. coli BL21 cells expressing GST-UL89 or GST-UL56C fusion proteins were harvested and purified by GSTrap™ column chromatography. Aliquots of every fraction were assayed for protein content by SDS-PAGE followed by Coomassie staining (Fig. 1, A and C) or transfer to nitrocellulose. The nitrocellulose filters were reacted with the affinity purified antibodies against UL56 and pUL56 (Fig. 1B) or against pUL89 and pabUL89, (Fig. 1D). Fraction 2 containing GST-UL56C (Fig. 1, A and B) or GST-UL89 (Fig. 1, C and D), respectively, was used for further analysis.

**ATPase Activity of GST-UL56C**—To identify the terminase subunit that has the ability to hydrolyze [γ-32P]ATP, experiments with purified GST fusion proteins were performed. Hydrolysis of radiolabeled ATP of purified terminase subunits was assayed following chromatographic separation of ATP and ADP + P on PEI-F cellulose. Addition of GST-UL56C to the reaction mixture resulted in generation of [32P]Pi (Fig. 2B, lane 5) comparable with the reaction with the Apyrase, an ATPase and ADPase from *Solanum tuberosum* (EC 3.6.1.5; Fig. 2B, lane 2). GST-UL89 did not show a high hydrolysis of ATP (Fig. 2B, lane 4). GST alone did not show a significant ATP hydrolysis (Fig. 2B, lane 3). These experiments lead to the suggestion that hydrolysis of ATP is mainly associated with the terminase subunit pUL56.

**Analysis of the ATP Cleavage Site**—To characterize the position of the enzymatic cleavage site, experiments were performed with [α-32P]ATP, [α-32P]dATP, or [γ-32P]GTP. The reaction with GST-UL56C yielded a more slowly migrating radiolabeled product corresponding to ADP (Fig. 3A, GST-UL56C), which results from the hydrolysis of the β-γ phosphodiester bond. After the incubation with Apyrase, AMP was still detectable because this enzyme has also an ADPase activity (Fig. 3A, Apyrase). The buffer control (Fig. 3A, buffer) as well as the reaction with GST (Fig. 3A, GST) or GST-UL89 (Fig. 3A, GST-UL89) did not show a specific reaction. Experiments with dATP demonstrated that GST-UL56C could hydrolyze dATP (Fig. 3B, GST-UL56C), but the amount of converted dATP was reduced up to 7-fold. The reaction was specific for ATP since GTP was not hydrolyzed (Fig. 3C, GST-UL56C). These results demonstrated that GST-UL56C hydrolyzes the β-γ phosphodiester bond of ATP, generating ADP and Pγ.

**Kinetic of the ATPase Activity**—To further investigate ATP hydrolysis, the time course of the reaction was analyzed. The ATPase activity of GST-UL56C was already observed after 10 min of incubation (Fig. 4B, lane 5). In contrast GST-UL89 needed an incubation time of 60 min (Fig. 4A, lane 8). The control GST protein did not show an increase of ATPase activity over time (Fig. 4C). Hydrolysis of radiolabeled ATP into ADP and Pγ was measured by incubation with Apyrase (Fig. 4, lane 2). The buffer control is shown in lane 1. These experiments support the notion that the terminase subunit pUL56 is the main ATPase.

**Saturation of the ATPase Activity**—To determine whether the enzymatic reaction could be saturated, experiments were performed with increasing protein amounts. ATP hydrolysis was verified by incubation with Apyrase (Fig. 5, lane 1). Interestingly, a concentration of GST-UL56C of 0.6 μg/ml was defined as the point where the reaction is in the log phase. This amount of protein converted 65% of ATP into Pγ (Fig. 5B, lane 4). In contrast GST-UL89 still showed almost no enzymatic activity (Fig. 5A). GST as well as buffer were used as control reactions (Fig. 5C, Fig. 5, A–C, lane 1). Our results demonstrated that the hydrolysis of ATP is saturated at a concentration of about 0.6 μg/ml GST-UL56C.

**Influence of GST-UL89 on the GST-UL56 ATPase Activity**—To further investigate whether GST-UL89 could enhance...
the ATPase activity of GST-UL56C, experiments under limiting concentrations of GST-UL56C (0.3 \mu g/ml) were performed. GST-UL56 (0.6 and 3.0 \mu g/ml) was added to the ATPase reaction mix of UL56 (0.6 \mu g/ml) and incubated for 1 h. In the presence of GST-UL89, the UL56 enzymatic activity was stimulated up to 30% (Fig. 6, lane 3–4) while acetylated bovine serum albumin had no influence (Fig. 6, lane 5). Apyrase was used for verification of the position of Pi (Fig. 6, lane 2) GST as well as the buffer was used as control reactions (Fig. 6, lane 7, lane 1). Thus pUL89 stimulates pUL56 ATPase activity.

Interaction of the Terminase Subunits pUL56 and pUL89—GST pull down assays were carried out using the carboxy-terminal half of pUL56. To this end, the GST-UL56C containing the carboxyl-terminal portion or GST itself were immobilized on glutathione-Sepharose beads and incubated either with in vitro translated [35S]methionine-labeled pUL89 (in vitro UL89; Fig. 7, lane 3) or [35S]methionine-labeled IE1 (in vitro IE1; Fig. 7, lane 6). The amount of bound material was analyzed by SDS-PAGE and autoradiography. As shown in Fig. 7, pUL89 interacted specifically with GST-UL56C (lane 3) but not with GST alone (lane 1). In vitro translated IE1, used as a negative control, did not interact with GST-UL56C (Fig. 2, lane 5). To estimate the intensity of the pUL56-pUL89 interaction, quantification was performed using bioimaging analysis of the fluorographs. Binding of pUL89 to GST-pUL56C was 25× stronger than that of GST alone to pUL89. These experiments demonstrated for the first time a direct interaction of pUL89 with pUL56.

To further investigate a direct interaction between both proteins co-immunoprecipitation prior to Western blot analysis was performed. For co-immunoprecipitation the antibody...
pabUL89 was used. The following Western blot analysis using pabUL56 detected pUL56 as the full size protein of 130 kDa in HCMV-infected cell extracts and co-immunoprecipitates (Fig. 8, lanes 3 and 4). No detection was observed in mock-infected cell extracts or co-immunoprecipitates (Fig. 8A, lanes 1 and 2). Co-immunoprecipitation with an antibody against HCMV pUL112/113, mab M23, was used as a control. The resulting co-immunoprecipitates were not detected by pabUL56 (Fig. 8B, lanes 3 and 4). The protein recognized by the antibody pabUL89 was demonstrated in Western blots of mock- and HCMV-infected cell extracts (Fig. 8, C and D). In HCMV-infected cell extracts the 75-kDa pUL89 was detected. These experiments implicate a direct interaction between HCMV pUL56 and pUL89.

**DISCUSSION**

Terminase subunits are the only proteins known to be involved in packaging of viral DNA and have, so far, been described for double-stranded DNA bacteriophages only (2, 14, 26, 27). These enzymes are normally hetero-oligomeric complexes with multiple functions (17–20). Although the exact mechanism of DNA translocation is not known, it is commonly accepted that this process is energy-dependent. Studies by Guo et al. (26) and Shibata et al. (9) demonstrated that hydrolysis of one ATP provides energy to translocate two base pairs under in vitro conditions.

It has been noted that the HCMV UL89 gene product, the homolog of herpesvirus simplex type-1 (HSV-1) pUL15, exhibits homology to the bacteriophage T4 gp17 terminase subunit (13). Therefore we investigated whether either or both HCMV proteins exhibited an ATPase activity. Interestingly, the ATPase activity was only associated with pUL56, which hydrolyzed the β-γ phosphodiester bond, generating ADP and P_i. Other nucleosides were not hydrolyzed by pUL56. Preincubation of the recombinant protein with the specific antibody could reduce the enzymatic activity to ~50%, while unspecific antibodies had almost no effect (data not shown). Although pUL89 did not have an obvious enzymatic activity, it enhanced the pUL56-associated ATPase activity by about 30%. Comparable observations were reported by Leffers and Rao (28) with the bacteriophage T4 terminase subunits. The large subunit gp17 exhibited an ATPase activity and that the gp17 activity was enhanced by gp16. This bacteriophage terminase subunit has two ATP-binding sites in the central and a metal-binding motif in the C-terminal portion of the protein (28). A mutation of either the metal-binding motif or the ATP-binding site resulted in the loss of gp17 function (22). Furthermore we showed that both proteins interact under in vitro conditions as well as in infected cells. This could be an explanation for the enhancing of the pUL56-associated ATPase activity by pUL89. Therefore, it is possible that HCMV pUL56 and pUL89 are the functional homologues to the T4 gp16 and gp17 subunits, respectively.

ATP hydrolyzing enzymes contain a Walker box: box A, GKT; box B, DE. Both regions are found in pUL89 and its homologs. Therefore the presumption was that pUL89 would have an ATPase activity. However, under the conditions we used pUL89 did not have an enzymatic activity. A motif in pUL56 that could theoretically constitutes a partial Walker box A is GKQ (amino acids 714–716). Recently it was demonstrated that a mutation from GKT to AKT in HSV-1 UL15, the homolog of pUL89, was unable to complement a UL15 mutant virus (29),
thus leading to the presumption that ATPase activity is important for herpesvirus maturation. Based on our observations we postulate that the energy for translocation of the concatemeric DNA to the preformed procapsids is provided by the pUL56-mediated ATP hydrolysis. Recently, a new class of anti HCMV agents (30), benzimidazole ribonucleosides, were shown to inhibit HCMV DNA maturation via interaction with the viral UL89 (31) and UL56 (32) gene products. In addition, analysis of mutants of pUL56 homologs of HSV-1, pUL28, and pseudorabies virus demonstrated that the deletion of the protein leads to nuclear accumulation of naked nucleocapsids and concatemeric DNA (10, 11, 33). However, nothing was known to date about the function of pUL56. Our findings could now give an explanation for the prevention of packaging after deletion of the gene in pseudorabies virus and HSV-1. Protein deletion will lead to the loss of energy for translocation of the concatemers to the procapsids. In consequence the next step of packaging into the capsid and cleavage into unit-length genomes will not occur. This resulted in the accumulation of concatemeric DNA and naked capsids in the nucleus.

Together with our former study (8) we suggest that pUL56 (i) mediates the specific binding to pac sequences on the concatemers and (ii) provides energy for the translocation of the DNA into the procapsids. The role of the terminase subunit pUL89 during packaging of unit-length genome remains to be determined.

Acknowledgments—We thank Thomas Stamminger and Helmut Fickenscher for critical reading the manuscript, Mark Underwood (GlaxoWellcome) for kindly providing the plasmid pUB8, and Thomas Stamminger for providing the plasmid pHM128 expressing HCMV IE1. E. B. thanks B. Fleckenstein for kind support.

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J. Biol. Chem. 2002, 277:6943-6948.
doi: 10.1074/jbc.M108984200 originally published online December 13, 2001

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