ADP-dependent DNA Strand Exchange by the Mutant [P67G/E68A]RecA Protein

EVIDENCE FOR AN INVOLVEMENT OF ADP IN RecA PROTEIN-MEDIATED BRANCH MIGRATION*

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We have prepared a mutant RecA protein in which proline 67 and glutamic acid 68 in the NTP binding site were replaced by a glycine and alanine residue, respectively. The [P67G/E68A]RecA protein catalyzes the single-stranded DNA-dependent hydrolysis of ATP and is able to promote the standard ATP-dependent three-strand exchange reaction between a circular bacteriophage \( \phi X174 \) single-stranded DNA molecule and a homologous linear \( \phi X \) double-stranded (ds) DNA molecule (5.4 kilobase pairs). The strand exchange activity differs from that of the wild type RecA protein, however, in that it is (i) completely inhibited by an ATP regeneration system, and (ii) strongly stimulated by the addition of high concentrations of ADP to the reaction solution. These results indicate that the strand exchange activity of the [P67G/E68A]RecA protein is dependent on the presence of both ATP and ADP. The ADP dependence of the reaction is reduced or eliminated when (i) a shorter linear \( \phi X \) dsDNA fragment (1.1 kilobase pairs) is substituted for the full-length linear \( \phi X \) dsDNA substrate, or (ii) the Mg\(^{2+}\) concentration is reduced to a level just sufficient to complex the ATP present in the reaction solution. These results indicate that it is the branch migration phase (and not the initial pairing step) of the [P67G/E68A]RecA protein-promoted strand exchange reaction that is dependent on ADP. It is likely that the [P67G/E68A]RecA mutation has revealed a requirement for ADP that also exists (but is not readily apparent) in the strand exchange reaction of the wild type RecA protein.

The RecA protein of \( Escherichia coli \) (\( M_\text{s} 37,842; 352 \) amino acids) is essential for homologous genetic recombination and for the postreplicative repair of damaged DNA. The purified RecA protein promotes a variety of ATP-dependent DNA pairing reactions that presumably reflect \( \textit{in vivo} \) recombination functions. The most extensively investigated DNA pairing reaction is the three-strand exchange reaction, in which a circular ssDNA molecule and a homologous linear dsDNA molecule are recombined to yield a nicked circular dsDNA molecule and a linear ssDNA molecule. This reaction proceeds in three phases. In the first phase, the circular ssDNA molecule is coated with RecA protein, forming a helical nucleoprotein filament known as the presynaptic complex. In the second phase, the presynaptic complex interacts with the linear dsDNA molecule, the homologous sequences are brought into register and pairing between the circular ssDNA, and the complementary strand from the dsDNA is initiated. In the third phase, the complementary linear strand is completely transferred to the circular ssDNA by unidirectional branch migration to yield the nicked circular dsDNA and displaced linear ssDNA products (1, 2).

The x-ray crystal structure of the RecA protein indicates that the phosphate groups of the nucleotide cofactor, ATP, are bound by a loop consisting of amino acids 66-73 (3). The sequence of this loop (\( ^{66} \text{GPESSGKT}^{73} \)) corresponds to a variation of the well known phosphate binding loop (P-loop) consensus sequence (\( ^{XXXXXGK(T/S)}^{XXX} \)) found in many NTP-binding proteins (4). The invariant lysine and threonine/serine residues in the P-loop motif are generally found to interact directly with the \( \beta \) and \( \gamma \) phosphates of ATP and have been shown for the RecA protein (as well as many other proteins) to be directly involved in the catalysis of phosphoryl transfer (5). Interestingly, although the four variable residues (XXXX) in this sequence can differ widely in different classes of proteins, the specific sequence, \( \text{GPESSGKT} \), is highly conserved in over 60 different bacterial RecA proteins (1). As part of an investigation of the role of the P-loop in ATP hydrolysis, we prepared a mutant RecA protein in which the proline and glutamic acid residues at positions 66 and 67 were replaced by a glycine and alanine, respectively. The biochemical properties of the [P67G/E68A]RecA protein provide new insight into the role of ADP in the DNA strand exchange reaction and are described in this report.

EXPERIMENTAL PROCEDURES

Materials—Wild type RecA protein was prepared as described previously (6). ATP was from Sigma; dATP, \( [\alpha-^32\text{P}] \)ATP, and \( [\alpha-^32\text{P}] \)dATP were from Amersham Pharmacia Biotech. \( E. coli \) SSB was from Promega. Circular \( \phi X \) ssDNA \( ((+) \text{ strand}) \) and circular \( \phi X \) dsDNA were from New England Biolabs. Full-length linear \( \phi X \) dsDNA was prepared from circular \( \phi X \) dsDNA as described (7). The 1.1-kb \( \phi X \) dsDNA fragment was generated by digesting circular \( \phi X \) dsDNA with \( D_{\text{del}} \) (New England Biolabs) and was purified by agarose gel electrophoresis. Single- and double-stranded DNA concentrations were determined by absorbance at 260 nm using the conversion factors 36 and 50 \( \mu \text{g/}\text{ml} \times \text{A}^{\text{**260}} \), respectively. All DNA concentrations are expressed as total nucleotides.

Preparation of the [P67G/E68A]RecA Protein—The gene for [P67G/E68A]RecA, in which the nucleotide sequence coding for proline 67 and glutamic acid 68 was replaced with a nucleotide sequence coding for glycine and alanine, respectively, was produced using the Quick-Change™ protocol (Stratagene). The initial mutagenesis template consisted of a pET21a\((+) \text{ vector} \) (Novagen) containing the wild type \( recA \) gene cloned into a \( NdeI/HindIII \) site. The mutagenesis primers that

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1 The abbreviations used are: ssDNA, single-stranded DNA; dsDNA, double-stranded DNA; \( \phi X \), bacteriophage \( \phi X174 \); SSB, \( E. coli \) single-stranded DNA binding protein; kb, kilobase pairs; DTT, dithiothreitol.

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were used to introduce the E68A mutation were 5’-GAAATCTACGGA-
CCGGCGCTTCGGTAAACC-3’ and 5’-GGTTTACCCGAGAGCATCCGGTCTCCAGTTTCCGAGGATACG-3’ (the codon for alanine 68 is underlined, and the nucleotide mismatch is in bold). The resulting plasmid, pETRecA-
(E68A), was then used as a template for a second mutagenesis step. The mutagenesis primers that were used to introduce the P67G mutation were 5’-CGTATGCAGAATCTAGGGGGGggCTCTCCTGGTAAACC-3’ and 5’-GGTTTACCCGAGAGCATCCGGTCTCCAGTTTCCGAGGATACG-3’ (the codon for glycine 67 is underlined, the nucleotide mismatch is in bold, and the codon for alanine 68 is in lowercase). The entire gene for [P67G/E68A]RecA was sequenced to confirm that only the desired changes had been introduced during the mutagenesis procedure. The expression plasmid, pETRecA(P67G/E68A), was introduced into the E. coli recA strain, BLR(D3) (Novagen). The expression of the [P67G/E68A]RecA protein was induced by addition of isopropyl-β-thiogalactoside (1 mM final concentration) at 0.6, followed by a 3-h incubation at 37 °C. The [P67G/E68A]RecA protein was then purified to greater than 95% homogeneity by methods that have been described previously (6). The purified [P67G/E68A]RecA protein is shown in Fig. 1.

RESULTS

ssDNA-dependent NTP Hydrolysis Activity of the [P67G/E68A]RecA Protein—The [P67G/E68A]RecA protein was analyzed for ssDNA-dependent ATP and dATP hydrolysis activity at pH 7.5 and 37 °C. The reaction solutions contained 1 μM [P67G/E68A]RecA protein and 30 μM ϕX ssDNA; these conditions ensured that there was sufficient ssDNA to bind all of the [P67G/E68A]RecA protein present. The dependence of the rate of ssDNA-dependent ATP and dATP hydrolysis on NTP concentration is shown in Fig. 2, and the kinetic parameters for the hydrolysis of each NTP are summarized below.

The [P67G/E68A]RecA protein catalyzed the hydrolysis of both ATP and dATP (Fig. 2). The turnover number (V_max/ [E_total]) for hydrolysis was 6 min⁻¹ for both ATP and dATP, and the S₀.₅ values were 45 μM and 25 μM for ATP and dATP, respectively. Under the same conditions, the wild type RecA protein catalyzes ATP and dATP hydrolysis with turnover numbers of 20 min⁻¹ for ATP and 24 min⁻¹ for dATP, and with S₀.₅ values of 45 μM and 20 μM for ATP and dATP, respectively (8). Thus, the [P67G/E68A]RecA mutation lowers the rate of ssDNA-dependent hydrolysis of ATP and dATP by ~3–4-fold, but has little effect on the S₀.₅ values for these NTPs.

Three-strand Exchange Activity of the [P67G/E68A]RecA Protein—The DNA strand exchange activity of the [P67G/E68A]RecA protein was evaluated using the three-strand exchange reaction. In this reaction, a circular ϕX ssDNA molecule (5386 bases) and a homologous linear ϕX dsDNA molecule (5386 base pairs) are recombined to form a nicked circular dsDNA molecule and a linear ssDNA molecule; the substrates and products of this reaction are readily monitored by agarose gel electrophoresis (7). The strand exchange reaction that was promoted by the wild type RecA protein in the presence of ATP (3 mM) is shown in Fig. 3A. In this reaction, partially exchanged DNA intermediates are visible within the first 3 min, and the fully exchanged circular dsDNA product can be detected within 10 min. The strand exchange reaction appears to reach completion within 60 min. Similar results were obtained when dATP was used in place of ATP as the nucleotide cofactor (gels not shown). These results are consistent with previous results (8).

The [P67G/E68A]RecA protein was also able to promote strand exchange in the presence of ATP (3 mM) (Fig. 3A). Although partially exchanged DNA intermediates are visible within the first 6 min of the reaction, the fully exchanged circular dsDNA product is not detected until ~4 h after initiation of the reaction. The strand exchange reaction appears to reach completion after ~6 h. Similar results were obtained when dATP was used in place of ATP as the nucleotide cofactor (gels not shown). These results indicate that the [P67G/E68A]RecA protein is able to form initial pairing intermediates with an efficiency similar to that of the wild type protein, but that the formation of the fully exchanged circular dsDNA product is delayed significantly relative to that with the wild type protein.

The time course of the ATP hydrolysis reaction that occurred during the [P67G/E68A]RecA protein-promoted strand exchange reaction is shown in Fig. 3B. The hydrolysis of ATP followed a linear time course for ~4 h, and then terminated abruptly after ~80–90% of the ATP had been converted to ADP. A comparison of the time course of ATP hydrolysis (Fig. 3B) with the time course of strand exchange (Fig. 3A) indicates that the fully exchanged circular dsDNA product was just beginning to appear as the ATP hydrolysis reaction was ending.

The ATP hydrolysis reaction of the wild type RecA protein continued until ~50% of the ATP had been converted to ADP (Fig. 3B), and the formation of the fully exchanged product reached completion during the linear phase of ATP hydrolysis (Fig. 3A).
Properties of the [P67G/E68A]RecA Protein

Effect of ATP Regeneration on the Strand Exchange Activity of the [P67G/E68A]RecA Protein—The results in Fig. 3 indicated that the pool of ATP in the reaction solution was being depleted by the ATP hydrolysis activity of the [P67G/E68A]RecA protein before the strand exchange reaction could be completed. Therefore, two additional sets of strand exchange reactions were carried out. In the first set, the strand exchange reactions were carried out in the presence of an ATP regeneration system (creatine kinase/creatine phosphate), which phosphorylates the ADP generated by the ATP hydrolysis reaction to reform ATP. In the second set, the strand exchange reactions were carried out in the absence of an ATP regeneration system, but with a higher initial concentration of ATP.

As shown in Fig. 4, the formation of the fully exchanged circular dsDNA product by the [P67G/E68A]RecA protein was strongly inhibited when an ATP regeneration system was included in the reaction solution. Similar results were obtained when dATP was used in place of ATP as the nucleotide cofactor (gels not shown). By comparison, the ATP regeneration system had little effect on the ATP or dATP-dependent strand exchange activity of the wild type RecA protein (Fig. 4, dATP results not shown).

The strand exchange activity of the [P67G/E68A]RecA protein was also inhibited (in the absence of the ATP regeneration system) when the initial concentration of ATP in the reaction solution was increased. As shown in Fig. 5, when the initial concentration of ATP was increased from 3 to 6 mM, the time required for the appearance of the fully exchanged product increased from 4 to 6 h. Conversely, when the starting concentration of ATP was reduced from 3 to 1 mM, the time required for the appearance of the fully exchanged product decreased from 4 to 2 h. This same variation in ATP concentration had little effect on the strand exchange activity of the wild type RecA protein (Fig. 5).

These results indicated that the strand exchange activity of the [P67G/E68A]RecA protein was dependent not only on the presence of ATP, but also on the presence of ADP. Under standard reaction conditions (in the absence of the ATP regeneration system), the requisite ADP would be generated from ATP by the ATP hydrolysis activity of the [P67G/E68A]RecA protein, and consequently, the fully exchanged product does not form until a sufficiently high concentration of ADP has accumulated in the reaction solution. The inclusion of the ATP regeneration system prevents the accumulation of ADP and, therefore, inhibits the strand exchange reaction. Furthermore, the increase in the delay of the appearance of the fully exchanged product that was observed when the ATP concentration was increased (in the absence of the ATP regeneration system) suggests that the critical parameter for the [P67G/E68A]RecA protein may not be the absolute concentration of ATP (ADP is generated at approximately the same rate with a starting concentration of either 3 or 6 mM ATP), but rather the ratio of ADP to ATP that is present in the reaction solution.

Effect of ADP on the Strand Exchange Activity of the [P67G/E68A]RecA Protein—In order to explore the apparent dependence of the [P67G/E68A]RecA protein-promoted strand exchange reaction on ADP, a series of strand exchange reactions was carried out in which the total concentration of nucleotide cofactor (ATP + ADP) was kept constant (3 mM), but the percentage of ADP in the pool was varied from 0 to 100%.

As shown in Fig. 6, when the starting nucleotide pool contained 0% ADP, the fully exchanged circular dsDNA products were detected ~4 h after the reaction was initiated, consistent with the results described above (Fig. 3). As the percentage of...
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of both ATP and ADP. Similar results were obtained when dATP and dADP were used in place of ATP and ADP as the nucleotide cofactors (gels not shown). By comparison, the formation of the fully exchanged product by the wild type RecA protein was inhibited as the percentage of ADP in the starting nucleotide pool was increased from 0 to 30%, and was completely eliminated when the starting ADP percentage was 40% or higher (Fig. 6).

Strand Exchange Activity of the [P67G/E68A]RecA Protein Using a Shorter Linear dsDNA Substrate—Although the time required for the appearance of the fully exchanged circular dsDNA product in the [P67G/E68A]RecA protein-promoted strand exchange reaction was much greater than that for the wild type RecA protein, the partially exchanged DNA intermediates appeared to form at similar rates with the two proteins (Fig. 3). Furthermore, the formation of the partially exchanged intermediates by the [P67G/E68A]RecA protein did not appear to be inhibited by the ATP regeneration system (Fig. 4). It is thought that the partially exchanged intermediates are formed in an initial pairing reaction that is dependent on ATP but does not require ATP hydrolysis, and that the resulting nascent hybrid DNA intermediates (generally about 1 kb in length) are then extended in an ATP hydrolysis-dependent branch migration reaction to yield the fully exchanged circular dsDNA product (9).

As shown in Fig. 7, the [P67G/E68A]RecA protein was able to recombine the circular φX ssDNA with the 1.1-kb linear φX dsDNA in the presence of ATP. In this reaction, the fully exchanged gapped circular dsDNA product was detected within 5 min, and the reaction reaches completion after ~120 min. Moreover, in contrast to the results that were obtained with the full-length linear φX dsDNA substrate (Fig. 4), the inclusion of an ATP regeneration system in the reaction solution had no effect on the rate of the reaction with the 1.1-kb linear φX
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**DISCUSSION**

Under our standard reaction conditions (10 mM Mg\(^{2+}\)/3 mM ATP), the [P67G/E68A]RecA protein is able to recombine a circular \( \phi X \) ssDNA molecule with a linear \( \phi X \) dsDNA molecule to form a fully exchanged nicked circular dsDNA reaction product. Although it appears to form the initial pairing intermediates with an efficiency similar to that of the wild type RecA protein, the time required for the appearance of the fully exchanged product is much greater for the [P67G/E68A]RecA protein (4 h) than for the wild type protein (10 min). Since the turnover number for the [P67G/E68A]RecA protein-catalyzed ATP hydrolysis reaction (6 min \(^{-1}\)) is \( \sim 3\)–4-fold lower than that of the wild type RecA protein (20 min \(^{-1}\)), it might be expected that the rate of strand exchange would also be 3–4-fold lower, since we have shown (with a different mutant RecA protein) that the rate of strand exchange can be correlated with the rate of NTP hydrolysis (Ref. 8; see also Ref. 11).

The pronounced delay in the appearance of the fully exchanged product that is observed with the [P67G/E68A]RecA protein, however, is much greater than can be accounted for by this effect. Instead, our results indicate that, under standard reaction conditions, the strand exchange activity of the [P67G/E68A]RecA protein is dependent on the presence of both ATP and ADP, and that the fully exchanged reaction product does not form until the ATP hydrolysis activity of the [P67G/E68A]RecA protein has converted \( \sim 50\% \) of the starting ATP into ADP. Because of this requirement for ADP, the strand exchange reaction of the [P67G/E68A]RecA protein is inhibited when the starting ATP concentration is increased, since this increases the time required for the ATP hydrolysis activity of the protein to convert 80% of the ATP to ADP. Moreover, the formation of the fully exchanged product is completely prevented when an ATP regeneration system is included in the reaction solution because this prevents any ADP from accumulating in the reaction solution. By contrast, the strand exchange activity of the wild type RecA protein is not inhibited by high starting ATP concentrations or by the presence of an ATP regeneration system.

Consistent with the apparent requirement for ADP, the strand exchange activity of the [P67G/E68A]RecA protein is strongly stimulated when ADP is included in the starting reaction solution. The reaction appears to be most efficient when the starting nucleotide pool consists of \( \sim 90\% \) ADP and 10% ATP. Under these conditions, the time required for the [P67G/E68A]RecA protein to form the fully exchanged product is reduced from 4 h to \( \sim 1 \) h (close to the rate expected, based on a rate of NTP hydrolysis about 3–4-fold lower than for the wild type protein). By contrast, the wild type RecA protein-promoted strand exchange reaction is completely eliminated at the high ADP concentrations (90% ADP) that provide the maximal level of stimulation of the [P67G/E68A]RecA protein-promoted reaction. A modest stimulation of heteroduplex DNA formation by
the wild type RecA protein in the presence of low concentrations of ADP has been noted previously by Cox and co-workers (12). Although the mechanistic basis for this observation was not determined, it is likely that it is related to the pronounced ADP dependence of the [P67G/E68A]RecA protein-promoted strand exchange reaction.

Cox and co-workers have shown that the wild type RecA protein-catalyzed ATP hydrolysis reaction terminates when ~40–60% of the available ATP has been hydrolyzed to ADP, regardless of the initial ATP concentration. To account for this observation, they have proposed that the termination of ATP hydrolysis is due to a dissociation of the RecA protein from the DNA that occurs when the ADP/ATP ratio exceeds ~1:1; this dissociation is apparently due to a structural incompatibility of the ADP and ATP states of the recA-ssDNA filament (13, 14). The greater extent of ATP hydrolysis that was observed with the [P67G/E68A]RecA protein (90% ATP hydrolyzed) suggests that the [P67G/E68A]RecA-ssDNA complex may be more resistant to ADP-mediated dissociation than is the wild type RecA-ssDNA complex. Furthermore, the finding that the appearance of fully exchanged products in the [P67G/E68A]RecA protein-promoted reaction occurs when the ADP concentration reaches ~80% (and rate of ATP hydrolysis has begun to decrease sharply) suggests that an ADP-mediated destabilization of the [P67G/E68A]RecA-ssDNA filaments may be necessary in order for the initial pairing intermediates to be resolved into the fully exchanged product. Consistent with this idea, the ADP dependence of the [P67G/E68A]RecA protein-promoted strand exchange reaction appears to be reduced or eliminated when a shorter 1.1-kb linear φX dsDNA fragment is used in place of the full-length 5.3-kb dsDNA. The [P67G/E68A]RecA protein may be able to fully exchange 1.1 kb of DNA via the initial pairing step, without the need for an extensive branch migration reaction. Additionally, the ADP dependence of the full-length 5.3-kb strand exchange reaction is decreased when the free Mg$^{2+}$ concentration is reduced to 1 mM or less. The lower free Mg$^{2+}$ condition presumably disfavors the formation of discontinuous loop intermediates during the initial pairing step (9) and may allow the [P67G/E69G]RecA protein to form the fully exchanged reaction product more readily via the branch migration reaction.

We have recently prepared two additional mutant RecA proteins, one containing only the P67G mutation and the other containing only the E68A mutation. Although both of these mutations reduce the rate of ssDNA-dependent ATP hydrolysis ~2-fold (relative to the wild type RecA protein), neither the [P67G]RecA protein nor the [E68A]RecA protein exhibits the ADP-dependent strand exchange activity that is observed with the [P67G/E68A]RecA protein (data not shown). Thus, the effects that are described in this paper appear to depend on the presence of both the P67G mutation and the E68A mutation. The structural basis for the effect of the [P67G/E68A] mutation on the properties of the RecA protein is not yet clear. It is likely, however, that the [P67G/E68A]RecA mutation has not fundamentally altered the mechanism of the RecA protein, but rather has magnified a requirement for ADP during the branch migration phase of the strand exchange reaction that also exists (but is not as readily apparent) in the reaction of the wild type RecA protein.

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