Characterization of the Interface between \( \gamma \) and \( \epsilon \) Subunits of Escherichia coli F\(_1\)-ATPase*

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Chunlin Tang and Roderick A. Capaldi‡
From the Institute of Molecular Biology, University of Oregon, Eugene, Oregon 97403-1229

The interaction faces of the \( \gamma \) and \( \epsilon \) subunits in the Escherichia coli \( F_1\)-ATPase have been explored by a combination of cross-linking and chemical modification experiments using several mutant \( \epsilon \) subunits as follows: \( \epsilon \)S10C, \( \epsilon \)H38C, \( \epsilon \)T43C, \( \epsilon \)S65C, \( \epsilon \)S108C, and \( \epsilon \)M138C, along with a mutant of the \( \gamma \) subunit, \( \gamma \)T106C.

The replacement of Ser-10 by a Cys or Met-38 by a Cys reduced the inhibition of \( ECF_1 \) by the \( \epsilon \) subunit, while the mutation S65C increased this inhibitory effect. Modification of the \( \gamma \) subunit at position 10 with N-ethylmaleimide or fluorescein maleimide further reduced the binding affinity of, and the maximal inhibition by, the \( \epsilon \) subunit. Similar chemical modification of the \( \gamma \) subunit at position 43 of the \( \epsilon \) subunit (in the mutant \( \epsilon \)T43C) and a \( \gamma \) subunit at position 106 of the \( \gamma \) subunit (\( \gamma \)T106C) also affected the inhibition of \( ECF_1 \) by the \( \epsilon \) subunit.

The various \( \epsilon \) subunit mutants were reacted with TF-PAM3, and the site(s) of cross-linking within the \( ECF_1 \) complex was determined. Previous studies have shown cross-linking from the \( \gamma \) subunit at positions 10 and 38 with the \( \gamma \) subunit and from a Cys at position 108 to an N-terminal residue (Aggeler, R., Chicas-Cruz, K., Cai, S., X., Keana, J., F.W., and Capaldi, R.A. (1992) Biochemistry 31, 2956-2961; Aggeler, R., Weinreich, F., and Capaldi, R.A. (1995) Biochim. Biophys. Acta 1230, 62-68). Here, cross-linking was found from a Cys at position 43 to the \( \gamma \) subunit and from the \( \gamma \) subunit at position 138 to a \( \beta \) subunit. The site of cross-linking from Cys-10 of \( \epsilon \) to the \( \gamma \) subunit was localized by peptide mapping to a region of the \( \gamma \) subunit between residues 222 and 242. Cross-linking from a Cys at position 38 and at position 43 was with the C-terminal part of the \( \gamma \) subunit, between residues 202 and 286.

\( ECF_1 \) treated with trypsin at pH 7.0 still binds purified \( \epsilon \) subunit, while enzyme treated with the protease at pH 8.0 does not. This identifies sites around residue 70 and/or between 202 and 212 of the \( \gamma \) subunit as involved in \( \epsilon \) subunit binding.

As the name implies, \( F_1F_0 \) ATPases are made up of two parts, an extrinsic \( F_1 \) part that contains the catalytic sites and a membrane-intrinsic \( F_0 \) part that contains the proton channel. Cryo-electron microscopy shows these two parts separated by a stalk of 40-45 Å in length (Gogol et al., 1987; Lücken et al., 1990). In the bacterium Escherichia coli, the \( F_1 \) part (\( ECF_1 \)) is made up of five different subunits (\( \alpha \), \( \beta \), \( \gamma \), \( \delta \), and \( \epsilon \)) in the molar ratio 3:3:1:1:1. The \( F_0 \) part contains three different subunits (a, b, and c) in the ratio 1:2:9-12 (Senior, 1990; Fillingame, 1992).

The structure of the \( F_1 \) part in the enzyme from bovine mitochondria has been obtained recently to 2.8 Å resolution by x-ray crystallography (Abrahams et al., 1994). This shows three \( \alpha \) and three \( \beta \) subunits arranged in a hexagon, with the \( \gamma \) subunit extending the length of the cavity within the hexagon, projecting from one end of the structure into an extension that is part of the stalk. The \( \delta \) subunit of MF\(_1\), which is the equivalent of the \( \epsilon \) subunit in bacterial and chloroplast enzymes, is also in the stalk but, in the crystal form studied, is probably disordered and not resolved.

There is now considerable evidence that the \( \gamma \) and \( \epsilon \) subunits play an important role in coupling catalytic site events with proton translocation (e.g. Gogol et al., 1990; Nakamoto et al., 1993; Abrahams et al., 1994; Capaldi et al., 1994; Zhang et al., 1994) and that this coupling involves conformational changes and, probably, translocations of one or both subunits (Aggeler et al., 1992, 1993; Turina and Capaldi, 1994a, 1994b; Wilkens and Capaldi, 1994). It is important, therefore, to obtain the detailed structures of, and interaction sites between, these two subunits.

We have recently determined the structure of the \( \epsilon \) subunit in solution by NMR spectroscopy (Wilkens et al., 1995). Here, we describe experiments that identify the \( \gamma \) subunit binding site on the \( \epsilon \) subunit, and that locate regions of the \( \gamma \) subunit involved in the \( \epsilon \) subunit binding.

**EXPERIMENTAL PROCEDURES**

Enzyme Preparations—Mutants eS10C, eH38C, and eS108C have been described previously (Aggeler et al., 1992; Skakoon and Dunn, 1993; Aggeler et al., 1995a). The mutation eM138C was created in the unc operon-containing plasmid, pRA100 (Aggeler et al., 1992), for isolation of \( ECF_1 \) and \( ECF_1 \). Mutations T43C and S65C were introduced into the uncC gene on plasmid pEX2 (Skakoon and Dunn, 1993) for overexpression and isolation of the altered \( \epsilon \). Pure \( \epsilon \) subunit was isolated as described by Patel et al. (1990). \( ECF_1 \) depleted of \( \delta \) and \( \epsilon \) subunits (ECF\(_1\)*) was prepared by one passage through an anti-\( \epsilon \) subunit monoclonal antibody affinity column according to Dunn (1986) but in a buffer containing 0.2% N,N-dimethylformamide N-oxide. Trypsin-treated \( ECF_1 \) was prepared as described by Tang et al. (1994), with a trypsin digestion time of 1 h for pH 7.0 samples and 4 h for pH 8.0 samples, respectively, followed by one Sephacryl S200 sizing column to separate proteolytically treated \( ECF_1 \) from other components. Mutants \( \gamma S8C \), \( \gamma T106C \), and \( \gamma V286C \) are described by Aggeler and Capaldi (1992). \( ECF_1 \) isolated from these mutants was labeled with
TABLE I Characterization of ε mutants

| ε subunit | ATPase activity of F1 \* | ATPase activity of ECF1 \* + 9 x \* |
|-----------|--------------------------|----------------------------------|
| S10C      | 37 ± 3.3 (5)             | 12.9 ± 0.4 (4)                   |
| H38C      | 13 ± 0.8 (3)             |                                  |
| T43C\*    | 11.3 ± 1.2 (5)           |                                  |
| S65C\*    | 4.4 ± 0.2 (2)            |                                  |
| S108C     | 7.8 ± 0.2 (2)            |                                  |
| M138C\*   | 21 ± 0.4 (3)             |                                  |
| W T       | 10 ± 0.5 (5)             | 6.7 ± 0.4 (2)                    |

\* Units are μmol of ATP hydrolyzed per min.
\* In molar ratio.
\* The mutation has not been introduced into the unc operon.
\* The pure ε subunit has not been overexpressed.

RESULTS

A number of mutants were used in the present study. Mutants εS10C (Aggeler et al., 1992), εH38C (Skakoon and Dunn, 1993), εS108C (Aggeler et al., 1992), and εM138C (this study) were created in the unc operon containing plasmid pRA100 (Aggeler et al., 1992). These mutants each showed wild-type growth on limiting glucose. Mutants T43C and S65C were created in the plasmid pEX2 containing the uncC, i.e. ε subunit, gene (Skakoon and Dunn, 1993). The inhibitory effect of the ε subunit when bound to isolated ECF1 (Sternweis and Smith, 1980) was used as a convenient measure of the binding of the ε subunit to the core complex (α3β3γ). Table I summarizes the activity effects of the various ε subunit mutations. It includes data for ECF1 isolated from strains, in which the mutation was in the unc operon, and data from experiments in which wild-type ECF1 has been depleted of endogenous ε subunit and then reconstituted with an excess of the mutant ε subunit. The ATPase activity of wild-type ECF1 was 10 ± 0.5 units/mg (μmol of ATP hydrolyzed/min/mg of enzyme), under our assay conditions at pH 7.5, with 2 mM ATP and 5 mM Mg\(^{2+}\). The activities of ECF1 isolated from the mutants εH38C and εM108C were very similar to that of wild-type, while that of enzyme from mutants εS10C and εM138C were around 4- and 2-fold higher than the wild-type, respectively. The ATPase activity of α and ε-free ECF3 was 51 ± 3 units/mg, an activation of around 5-fold, which could be reduced to a basal of around 7 units/mg by addition of excess wild-type ε subunit. Addition of purified ε subunit carrying the H38C or S108C mutations had similar effects to the addition of wild-type subunits. In all cases, excess of the ε subunit inhibited activity below that of isolated ECF1, suggesting that there is some loss of ε subunit during isolation of the intact ECF1 complex. Rebinding of ε10C gave a minimal activity of 13 units/mg. Addition of a 9-fold excess of ε subunit with the mutation T43C also failed to inhibit the core ECF1 complex (α3β3γ) to levels found using wild-type, consistent with the mutant having a reduced affinity for the core complex. Addition of mutant εS65C gave a higher inhibition, giving a final ATPase activity that was only 70% of wild-type enzyme. The activity data, therefore, indicate that mutations S10C and T43C reduce, while the mutation S65C increases, the inhibitory effect of the ε subunit on the core ECF1 complex.

The ε mutants S10C and T43C were examined further in experiments in which the introduced Cys were reacted with various maleimides, and the effect of this modification on activity was monitored. Fig. 1 shows the concentration dependence of the inhibition of ATPase activity of the mutant εS10C before and after modification with different maleimides. It can be seen that both the concentration for half-maximal inhibition and the absolute extent of inhibition of this mutant were altered significantly by NEM and dramatically by modification.
with FM. Modification of the mutant T43C with FM had a much smaller, although significant, effect on the binding affinity and maximal inhibition (results not shown). These results indicate that the regions of the enzyme around residue 10 and, to a lesser extent, around residue 43 are important for interaction of the ε subunit with the core ECF1 and the resulting inhibition of enzymatic activity.

Cross-linking of the Mutant ε Subunit in the ECF1 Complex—Previous studies have shown that the ε subunit can be cross-linked to the γ subunit by reaction of the Cys introduced at positions 10 or 38, with the maleimide group of the photoaffinity reagent, TFPAM, and subsequent UV photolysis to activate the tetrafluorophenylazide (TFPA) group to a nitrene (Aggeler et al., 1992, 1995b). Using the same approach, the ε subunit in the mutant εS108C was found to cross-link to an α subunit (Aggeler et al., 1992). Fig. 2 shows a cross-linking experiment involving ECF1 from the mutant εM138C. Reaction of the mutant with TFPAM3 generated one major cross-linked product of Mr approximately 75,000, which monoclonal antibody blots (not shown) revealed to be a cross-linked product between the mutant ε subunit and a β subunit. As shown in Fig. 2, the yield of this cross-linked product was nucleotide dependent, highest with ADP in catalytic sites and low with ATP + EDTA (or AMP-PNP + Mg²⁺) bound.

The location of residues 43 and 65 were examined by reacting the ε subunit with TFPAM3 in the dark, removing excess reagent and then binding to ECFγ, treated to remove endogenous ε subunit with trypsin, followed by photolysis to activate the TFPA group. A cross-linked product between the ε and γ subunits was observed in ECF1 containing the T43C mutation (see below). No cross-link was observed with the S65C mutant.

Localization of the Sites of Cross-linking from Residue 10 of ε in the γ Subunit—The site(s) of TFPAM cross-linking from Cys-10 in the mutant εS10C to the γ subunit was determined by peptide mapping and protein sequencing. The cross-link was formed as described previously in ECF1 (Aggeler et al., 1992) and then the enzyme treated with trypsin (1:25) for 1 h at room temperature. Trypsin cleavage under these conditions has been used to generate two fragments of the γ subunit, labeled as γα and γδ (Tang et al., 1994). The protease treatment of the TFPAM-treated ECF1 from mutant εS10C (Fig. 3A) gives these same products by cleavage of non-cross-linked γ subunit and, in addition, generated a product of Mr approximately 19,000, which reacted with mAbs to the C-terminal part of the γ sub-
tentatively identifying it as a cross-linked product of the N terminus of \( \epsilon \) (via Cys-10) and the \( \gamma \)D fragment (residues 202–286) of the \( \gamma \) subunit.

The approximate M, 19,000 band was excised from polyacrylamide gels, protein was collected by electroelution, purified by HPLC, and then digested further with trypsin. The small fragments, so generated, were separated by high pressure liquid chromatography, and the peaks were resolved and then analyzed by N-terminal amino acid sequencing. One peak contained a peptide of \( \epsilon \) containing Cys-10, present in equimolar amounts with a fragment of the \( \gamma \) subunit identified by its sequence as including residues 222–242.

As shown in Fig. 4, the sequence of the \( \epsilon \) fragment continued beyond Cys-10, as expected, because the cross-link involves the side chain of the Cys, and Edman degradation is not prevented. In contrast, sequencing of the \( \gamma \) fragment stopped abruptly before tyrosine 228, consistent with cross-linking via the tetrafluorophenylazide into the backbone of the polypeptide between residues 227 and 228. We have seen a similar insertion of TFPAM into the backbone of the \( \beta \) subunit next to a trypto-
phan residue in experiments to identify the interaction site between γ and β (Aggeler et al., 1993). It appears that the TFP Amoëity tends to stack against aromatic residues, such as tyrosines and tryptophans, in an orientation that causes insertion of the reactive nitrene into the backbone.

Identification of the Sites of Cross-linking from Residues 38 and 43 of ε into the γ Subunit—Attempts to use the same general approach described above for εS10C to identify interaction sites on γ from the Cys at residue 38 or 43 of the ε subunit failed because of the low yield of cross-linking observed with these mutations, i.e. 10–20% compared with 50% for the εS10C mutant. Instead, the location of the cross-linking in these mutants was assessed by tagging different parts of the γ subunit for identification after the covalent linkage with the ε subunit.

An experiment involving the mutant εH38C is shown in Fig. 5. Isolated ε subunit containing the mutation H38C was reacted with TFPAM3 in the dark, and then the modified ε subunit reacted with ECF1 that had been treated with trypsin to remove endogenous δ and ε subunits and at the same time cleave γ to γA and γD. Two forms of ECF1 were used, one containing the mutation γS8C, the other γV286C (Aggeler and Capaldi, 1992). These sites were labeled with CM to identify the N-terminal γA fragment or C-terminal γD fragment on gels. As shown in Fig. 5, cross-linking from the Cys at 38 to the γ subunit with TFPAM3 gave a product of approximate Mr 22,000, which contained Cys-286 but not Cys-8 of the γ subunit and is, therefore, a covalent product of the ε subunit at position 38 with the C-terminal region 202–286 of γ.

Similar experiments were then used to localize the site of interaction of the ε subunit from residue 43 with the γ subunit. These show cross-linking of the ε fragment to the same C-terminal part of the γ subunit (results not shown).

Binding Sites for the ε Subunit on the γ Subunit from Protease Digestion Studies—As described above, trypsin digestion of ECF1 at pH 7.0 leads to generation of two fragments, γA and γD. Trypsin cleavage is more efficient at pH 8.0 when the γ subunit yields three fragments, γC involving residues 1 to ap-
proximately 70, γν from residues 71 to about 202, and γε, residues 212–286 (Tang et al., 1994). The fragment, or fragments, that includes 11 residues between 202 and 211 is not resolved on gels. Trypsin cleavage at pH 7.0 or 8.0 activates the EC_F, complex to the same level by removing the ε subunit (final activity of about 50 unit/mg, cf. 10 units/mg for untreated enzyme). Addition of a 6-fold excess of pure ε was found to fully inhibit the activity of EC_F, treated with trypsin at pH 7.0 (i.e. the activity was reduced to less than 10 units/mg). However, there was no inhibition of sample that had been treated with the protease at pH 8.0. The inability of enzyme cleaved by trypsin at pH 8.0 to be inhibited by isolated ε subunit resulted from its low affinity for the ε subunit, as shown by sedimentation studies in Fig. 6. Taken together, these data indicate that the cleavage of γ at residue 70 and/or cleavage of the subunit at 212 with probable removal of residues 202–211 alters ε binding to EC_F.

Chemical Labeling of the Mutant γT106C Places This Residue in the γ Subunit Binding Region—Insertion of a Cys for Thr at position 106 of the γ subunit has no effect on the activity of EC_F. This introduced Cys residue could be reacted with NEM without affecting the binding of, or the inhibition by, the ε subunit added to ε-free EC_F, from the mutant (γT106C). However, as shown in Fig. 7, reaction of the Cys at position 106 with the more bulky maleimide, CM, altered both the binding, as judged by the half-maximum concentration of ε for inhibition, and the absolute level of inhibition, obtained on binding wild-type ε subunit.

**DISCUSSION**

Studies presented here focus on the interaction faces of the ε and the γ subunits for each other. The results are summarized in Fig. 8, which shows the recently obtained structure of the N-terminal domain of the ε subunit involving residues 1–86 in part A (Wilkens et al., 1995) and the predicted folding pattern of the γ subunit in part B. Genetic studies have predicted that the ε subunit is a 2-domain protein (Kuki et al., 1988), and this is evident in the NMR structure determination of the isolated ε subunit in solution. Residues 1–86 form a 10-stranded β barrel, or sandwich, while the C-terminal 48 residues are arranged as an α-helix loop-α-helix structure. In solution, the helix-loop-helix domain binds back on the β barrel at one end.

The ε subunit interacts with the α and β subunits of the F₁ part through the C-terminal domain, as indicated by cross-linking from Ser-108 to Glu-381 in the DELESEED region of the β subunit by 1-ethyl-3-[3-dimethylamino)propyl]carbodiimide (Dallmann et al., 1992), by disulfide bond formation from a Cys at position 108 of ε to a Cys in place of Glu-381 in β (Aggeler et al., 1995b), and by disulfide bond formation between Cys at 108 of ε and a Cys at position 411 in the α subunit (this is the equivalent residue to Glu-381 of β). Finally, as shown here, there is cross-linking of the ε subunit to a β subunit by TFPAM3 from a Cys replacing Met-138 at the C terminus of ε.

In addition to interacting with α and β subunits, the ε subunit is now known to interact with the c subunits of the F₀ (Zhang et al., 1994, 1995; Watts et al., 1995), and this linkage is via the opposite end of the β sheet sandwich from which it binds the helix-loop-helix domain (Fig. 8A).

The ε subunit also binds to the γ subunit in EC_F₁ (Dunn, 1986; Aggeler et al., 1992; Skakoon and Dunn, 1993). The homologous subunit in chloroplasts (ε) also binds to the γ subunit in CF₁ (Suss, 1986; Soteropoulos et al., 1994). The results presented here identify Ser-10, His-38, and Thr-43 as close to, or involved in, this reaction based on mutagenesis to Cys and then cross-linking from these sites and/or by chemical modification of the introduced Cys residue and consequent steric effects on ε binding. These three residues are on one face of the β barrel and, as shown in Fig. 8, are close to a patch of hydrophobic residues, which may play a key role in the binding to the γ subunit.

The cross-linking results presented here indicate a role of residues between 202 and around 240 in ε subunit binding. TFPAM cross-linking from the Cys-10 of ε is with a tyrosine at residue 228 of the γ subunit. Cross-linking from Cys-38 or Cys-43 in the mutants εH38C and εT43C is within the region of γ from 202 to 286. The C terminus from residues 222–286 is organized as a long α helix that, from around residue 240 to the very C terminus, is intercalated within the cavity formed by the hexagonally arranged α and β subunits (Abrahams et al., 1994). Our data indicate that the ε subunit binds at, or close to, this α helix as it extends from the αβ, barrel through the stalk region and makes contact with the c subunits of the F₀ part somewhere between residues 202 and 229 (Watts et al., 1995).

The protease digestion data focus attention on the region of γ around residue 70, as well as on the region between residues 202 and 212 in binding the ε subunit. Residue 70 is in the epitope for a monoclonal antibody to the γ subunit, described by Dunn and colleagues, which reacts with EC_F, only when the ε subunit is first removed (Skakoon and Dunn, 1993). Residues 202–212 are close to the region of the γ subunit in which there is an insertion in the γ subunit of chloroplasts that contains two Cys residues, which regulate CF₁ activity by reduction and oxidation reactions involving the protein thioredoxin (reviewed by Soteropoulos et al. (1994)). The ε subunit has been shown to affect the oxidation and reduction reaction of these Cys residues and protect this region from proteolytic digestion in CF₁ (Schumann et al., 1985).

In addition to the region around residue 70 and the C-terminal part from residues 202 to around 240, our chemical modification studies also place Thr-106 of γ in the ε subunit binding site. The recent x-ray structure determination shows that the γ subunit has three segments in contact with α and β subunits, an N-terminal α-helix of residues 1–50, a short central α-helical residue region 83–99 in the numbering system of E. coli, as well as the C-terminal α-helix (Abrahams et al., 1994). It is noteworthy that the remainder of this subunit, including several of the sites identified here as in, or close to, the ε binding site, is predicted to be in the β sheet and turn structure. It seems likely, then, that the α helical part of the ε subunit binds in part to an equivalent structure formed by much of the region of the γ subunit, as well as binding to the extension of the C-terminal helix region.

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