Structural Asymmetry of F1-ATPase Caused by the γ Subunit Generates a High Affinity Nucleotide Binding Site*

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The αβγγ and αβ3 complexes of F1-ATPase from a thermophilic Bacillus PS3 were compared in terms of interaction with trinitrophenyl analogs of ATP and ADP (TNP-ATP and TNP-ADP) that differed from ATP and ADP and did not destabilize the αβ3 complex. The results of equilibrium dialysis show that the αβγγ complex has a high affinity nucleotide binding site and several low affinity sites, whereas the αβ3 complex has only low affinity sites. This is also supported from analysis of spectral change induced by TNP-ADP, which in addition indicates that this high affinity site is located on the β subunit. Single-site hydrolysis of substoichiometric amounts of TNP-ATP by the αβγγ complex is accelerated by the chase addition of excess ATP, whereas that by the αβ3 complex is not. We further examined the complexes containing mutant β subunits (Y341L, Y341A, and Y341C). Surprisingly, despite very weak affinity of the isolated mutant β subunits to nucleotides, low affinity ATP binding sites are mostly on the α subunits whereas noncatalytic nucleotide binding sites are mostly on the γ subunits. This subunit has at least three stretches of α helices, and two of them form a coiled coil structure that penetrates the central cavity of the αβ3 structure.

Penevsky and his colleagues showed that bovine MF1 has a single high affinity ATP binding site (Grubmeyer et al., 1982; Cross et al., 1982). ATP added at substoichiometric amount binds rapidly to this site and is hydrolyzed slowly (single-site or "uni-site" catalysis). This slow hydrolysis is greatly accelerated by the addition of excess ATP (chase acceleration). However, in the case of F1-ATPase from a thermophilic Bacillus PS3 (TF1), the single-site hydrolysis of ATP is much faster than that of MF1, and only a very poor chase acceleration is observed (Yoshida and Yoshida, 1987). Nonetheless, steady state ATP hydrolysis by TF1 does not obey simple Michaelis-Menten type kinetics but shows apparent cooperativity (Wong et al., 1984; Yokoyama et al., 1989). The attenuated incorporation of water oxygen into the product P_i during hydrolysis of increasing concentrations of ATP by TF1 also provided a support for cooperative nature of the catalysis (Kasho et al., 1989). We found that TF1 hydrolyzed a substoichiometric amount of trinitrophenyl adenosine triphosphate (TNP-ATP) slowly, and this slow hydrolysis was accelerated by the chase addition of excess ATP (Hisabori et al., 1992). These observations have led us to the conclusion that TF1, as well as F1 from other sources, has a functional asymmetry among the three catalytic sites, although TF1 molecules, different from F1 from other sources, do not contain any endogenously bound nucleotide. Moreover, from the analysis of the nucleotide binding of the TF1-ADP 1:1 complex, we suggested the functional asymmetry among the three catalytic sites (Hisabori et al., 1994).

TF1 is unique in its ability to reconstitute from each isolated subunit. Complexes with various combinations of subunits, such as αβγγ and αβ3 (Yoshida et al., 1977a; Yokoyama et al., 1989) and αβ3 (Miwa and Yoshida, 1989; Kagawa et al., 1989) have been characterized. Most recently, the minimum catalytic unit, whose most probable subunit composition was αβγ3, was reconstructed on the solid support (Saika and Yoshida, 1995). As expected, this αβγ3 complex shows simple Michaelis-Menten type kinetics when it hydrolyzes ATP. Therefore a critical question has arisen: is the kinetics of the αβγ3 complex cooperative or not? Because the structure of this complex should be a perfect 3-fold symmetry, all three catalytic sites of ATP and ADP, respectively; PIPES, piperazine-N,N’-bis(2-ethanesulfonic acid); Tricine, N-(2-hydroxy-1,1-bis(hydroxymethyl)ethyl)glycine; HPLC, high pressure liquid chromatography.

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The abbreviations used are: MF1, F1-ATPase from mitochondrial inner membrane; TF1, F1-ATPase from thermophilic Bacillus strain PS3; TNP-ATP and TNP-ADP, the 2’3’-O-(2,4,6-trinitrophenyl) derivatives (Abrahams et al., 1994) revealed that the α and β subunits have a similar fold alternating in a hexagonal arrangement around a central cavity containing the γ subunit as expected from previous electron microscopic studies (Gogol et al., 1989; Fujiyama et al., 1990), and catalytic nucleotide binding sites reside mostly on β subunits whereas noncatalytic nucleotide binding sites are mostly on α subunits. The γ subunit has at least three stretches of α helices, and two of them form a coiled coil structure that penetrates the central cavity of the αβ3 structure.
Role of the \( \gamma \) Subunit in Function of F\(_1\)-ATPase

EXPERIMENTAL PROCEDURES

Preparation of the Subunit Complexes—The \( \alpha \) and \( \beta \) subunits of F\(_1\) were expressed in Escherichia coli strain OK9 (bgK, thi-1, rld-1, HfrP01, \( \Delta (\mu kD\text{-uncC}) \) IIv\( \text{Tn10} \)) (Ohta et al., 1988) and purified as described previously (Ohtsubo et al., 1987). To isolate the \( \alpha \)\( \beta \) complex, each subunit was precipitated by ammonium sulfate, dissolved in the minimum volume of 50 mM Tris-SO\(_4\) buffer (pH 8.0), and mixed. After incubation at 30°C for 30 min, the solution was loaded on a gel filtration HPLC column (TSK G3000SWXL) equilibrated with 10 mM PIPES and 0.2 M Na\( _2\)SO\(_4\) (pH 7.0) (PIPES-Na\(_2\)SO\(_4\) buffer), and fractions containing pure \( \alpha \)\( \beta \) complex were collected (Kaibara et al., 1993). The wild-type and mutant \( \alpha \)\( \beta \)\( \gamma \) complexes were over-expressed as the complex in E. coli strain MJ103uncB-D and purified as described previously (Matsui and Yoshida, 1995). Expression vectors for mutant complexes were made from that of the wild-type complex by exchanging a SalI site in the promoter obtained from wild-type \( \beta \) subunits (Odaka et al., 1990). The purified \( \alpha \)\( \beta \)\( \gamma \) complexes contained no or very little bound nucleotide (<0.1 mol/mol). The concentrations of proteins of the wild-type and mutant complexes were determined by measuring absorbance at 280 nm. The factor 0.45 at 280 nm as 1 mg/ml (Yoshida et al., 1977a) was used. The effect of replacement of Tyr-341 by other residues on the above factor was small and neglected.

Materials—TNP-ATP and TNP-ADP were synthesized and purified according to (Hiratsuka and Uchida, 1973; Hiratsuka, 1982). The purity was checked by absorption spectra (\( \epsilon_{260} = 25,000 \text{ cm}^{-1}\text{M}^{-1}\text{cm}^{-1} \) and \( \epsilon_{280} = 26,400 \text{ cm}^{-1}\text{M}^{-1}\text{cm}^{-1} \) at pH 8.0) (Hiratsuka and Uchida, 1973) and by reverse phase HPLC (Hisabori et al., 1992).

Stability of the \( \alpha \)\( \beta \) Complex—10 \( \mu \)l of the \( \alpha \)\( \beta \) complex (3.2 \( \mu \)M) were preincubated for 1 min at 25°C and subjected to gel filtration HPLC (TSK G3000SWXL). The solutions used for preincubation, equilibration, and elution of the HPLC column were all the same: PIPES-Na\(_2\)SO\(_4\) buffer containing indicated concentrations of nucleotide and MgSO\(_4\). The flow rate was 0.5 ml/min, and elution was monitored by measuring the absorbance at 280 nm. The maximum concentration of TNP-ATP (or TNP-ADP) tested was 50 \( \mu \)M due to high baseline absorbance.

Measurement of the Nucleotide Binding—The binding capacity of nucleotides on the subunit complexes were measured directly by equilibrium dialysis and indirectly by difference absorption spectrum induced by the interaction between the complex and nucleotides. Equilibrium dialysis was carried out as described by Hisabori and Sakurai (1984). Sample cells contained 0.02–0.5 \( \mu \)M \( \alpha \)\( \beta \)\( \gamma \) complex in 20 mM Tricine-NaOH (pH 8.0) or 0.36–0.81 \( \mu \)M \( \alpha \)\( \beta \) complex in the PIPES-Na\(_2\)SO\(_4\) buffer (Hisabori et al., 1986). Difference spectra were measured 5 min after mixing.

RESULTS

Stability of \( \alpha \)\( \beta \) Complexes in TNP-ATP(D)—The \( \alpha \)\( \beta \) complex of F\(_1\) dissociates into \( \alpha \)\( \beta \) complexes in the presence of ATP (>6 \( \mu \)M) and ADP (>30 \( \mu \)M) (Harada et al., 1991).

TNP-ATP did not induce dissociation of the \( \alpha \)\( \beta \) complex. As shown in Fig. 1A, when the \( \alpha \)\( \beta \) complex was incubated with TNP-ATP and analyzed on a gel filtration HPLC column equilibrated with the buffer containing the same concentration of TNP-ATP, the retention times of the protein peaks remained unchanged, suggesting that no dissociation took place. TNP-ADP also did not induce dissociation (Fig. 1B). When the \( \alpha \)\( \beta \) complex was preincubated with 20 \( \mu \)M TNP-ATP at first and then applied to a HPLC column equilibrated with 10 \( \mu \)M ATP, most of the \( \alpha \)\( \beta \) complex remained intact (Fig. 1C). However, complete dissociation was observed at 20 \( \mu \)M ATP for the complex preincubated with 20 \( \mu \)M TNP-ATP. Thus, different from ATP, TNP-ATP (or TNP-ADP) is a “safe” nucleotide that does not induce destabilization of the \( \alpha \)\( \beta \) complex. Taking this
advantage, following experiments were carried out.

Binding of TNP-ADP to the $\alpha_3\beta_3\gamma$ and $\alpha_3\beta_3$ Complexes—It is clear from the results of equilibrium dialysis that the $\alpha_3\beta_3\gamma$ complex has at least two classes of binding sites for TNP-ADP with different affinity; a high affinity binding site with a $K_d$ value in the low $\mu$M range and several low affinity sites with $K_d$ values in the $\mu$M range (Fig. 2, closed circles). On the contrary, the $\alpha_3\beta_3$ complex has only low affinity binding sites with $K_d$ in the $\mu$M range, and no high affinity binding site was observed (Fig. 2, open circles). Therefore, the $\gamma$ subunit in the $\alpha_3\beta_3\gamma$ complex is responsible for generating a single high affinity nucleotide binding site.

Subunit Location of the High Affinity Nucleotide Binding Site—It has been known that the difference absorption spectra induced by the interaction between TNP-ATP (or TNP-ADP) and the isolated $\alpha$ or $\beta$ subunit are characterized with a trough at 450 nm and a peak at 510 nm ($\alpha$ subunit) or a trough at 395 nm and a peak at around 420 nm ($\beta$ subunit), respectively (Hisabori et al., 1992). We can determine the subunit location of the TNP-ADP binding site of the complex by this means. When TNP-ADP at a 0.25 molar ratio was added to $\alpha_3\beta_3\gamma$ complex, the shape of the induced difference spectrum agreed almost the same as the one observed for the isolated $\beta$ subunit, indicating that the binding of TNP-ADP occurred to the site on one of three $\beta$ subunits in the complex (Fig. 3, top traces and the trace that is second from the top). For the $\alpha_3\beta_3$ complex, the shape of the difference spectrum was not the one typical for the $\beta$ subunit, and the magnitude of the spectrum was far smaller than that of the $\alpha_3\beta_3\gamma$ complex under the same condition (data not shown). This small magnitude of the spectrum agrees with a weak affinity of the $\alpha_3\beta_3$ complex to TNP-ADP indicated from equilibrium dialysis (Fig. 2). When we measured a series of the difference spectra induced by each step-wise addition of TNP-ADP to the $\alpha_3\beta_3\gamma$ complex, the shapes of the spectra induced by each addition were similar to the one observed for the isolated $\beta$ subunit until the concentration of added TNP-ADP reached a 1.25:1 molar ratio to the complex (Fig. 3). When the molar ratio exceeds 1.5:1, the shape of spectra induced by each addition of TNP-ADP becomes similar to the one observed for the isolated $\alpha$ subunit, a peak at 510 nm. These results clearly show that TNP-ADP binds first to a high affinity site on the $\beta$ subunit, and after this site is filled it starts to bind to a site on the $\alpha$ subunit.

Single Site Hydrolysis of TNP-ATP by the $\alpha_3\beta_3\gamma$ and $\alpha_3\beta_3$ Complexes—The $\alpha_3\beta_3\gamma$ complex hydrolyzes TNP-ATP, which is added in a stoichiometric molar ratio to the complex (Fig. 4A, closed circles). When ATP was chase-added to the reaction mixture on the progress of this single-site reaction, the hydrolysis of TNP-ATP was strongly accelerated. Even 1 $\mu$M ATP (Fig. 4A, open circles) is as effective as 3.3 mM ATP (Fig. 4A, open squares) for inducing acceleration of TNP-ATP hydrolysis. As observed for MFe$_3$(Grubmeyer and Penefsky, 1981), rapid binding of TNP-ATP is followed by slow hydrolysis, which then is accelerated by the occupation or hydrolysis of ATP at the second nucleotide binding site.

The $\alpha_3\beta_3$ complex also catalyzes TNP-ATP hydrolysis under the single-site hydrolysis condition (Fig. 4B, closed circles), but this hydrolysis is not accelerated by the chase incubation for 5 s with the addition of 1 $\mu$M ATP (Fig. 4B, open circles). The lack of the chase acceleration at 1 $\mu$M ATP for the $\alpha_3\beta_3$ complex cannot be attributed to the dissociation of the complex. As described above, nucleotide-induced destabilization of the $\alpha_3\beta_3$ complex did not occur under this condition. In addition, Sato et al. (1995) reported that the dissociation of the $\alpha_3\beta_3$ complex into the $\alpha_3\beta_1$ complexes induced by ATP proceeds fairly slowly; at 1 mM ATP, only very little dissociation was observed in 20 s, and it took about 100 s for the complete dissociation. We measured the amounts of ATP and ADP in the reaction tubes of the experiment of Fig. 4B after termination of the chase reaction and confirmed that added ATP was almost completely hydrolyzed in 5 s (data not shown). Thus, lack of chase acceleration for the complex may not be due to inability of ATP at 1 $\mu$M to interact with the complex, even though a possibility remains that ATP preferentially binds and is hydrolyzed by the complexes without bound TNP-ATP. To be sure, we measured the effect of chase addition of 1 mM ATP, and as expected no acceleration was observed (data not shown).

Single Site Hydrolysis of TNP-ATP by the $\alpha_3\beta_3\gamma$ and $\alpha_3\beta_3$ Complexes Containing Mutant $\beta$ Subunits—Tyr-341 of the T$\gamma_2$ subunit is located at a catalytic site and involved in binding of ATP (or ADP) (Bullough and Allison, 1986; Cross et al., 1982; Xue et al., 1987; Jault et al., 1994; Abrahams et al., 1994). Replacement of this residue by other residues such as Leu, Ala, and Cys resulted in a drastic decrease of binding affinity of the isolated $\beta$ subunit to ATP, and $K_m$ values for ATP of the mutant $\alpha_3\beta_3\gamma$ complexes increased in a parallel manner (Table I) (Odaka et al., 1994). To know the relation between the nucleotide binding site on the $\beta$ subunit and a single high affinity site of the $\alpha_3\beta_3\gamma$ complex, hydrolysis of TNP-ATP by the $\alpha_3\beta_3\gamma$ complexes containing mutant $\beta$ subunits (Y341L,
Y341A, or Y341C) were examined. Surprisingly, \( \alpha_3 \beta_3 \gamma \) complexes containing these mutant \( \beta \) subunits exhibited almost the same kinetics in single-site hydrolysis of TNP-ATP and in chase acceleration by 3.3 mM ATP as those of the wild-type complex (Fig. 4, C, E, and G, closed circles and open squares).

However, when 1 mM ATP was chase-added, the \( \alpha_3 \beta_3 \gamma \) and \( \alpha_3 \beta_3 \) complexes exhibited smaller extent of acceleration, and the \( \alpha_3 \beta_3 \gamma \) complex did not show chase acceleration (Fig. 4, C, E, and G, open circles). ATP concentrations necessary for half-maximal chase acceleration were measured for each of the mutant complexes, and it was shown that higher concentrations of ATP, compared with the wild-type complex, were required for acceleration of the mutant complexes (Table I). For the wild-type \( \alpha_3 \beta_3 \gamma \) complex, less than 1 mM ATP was sufficient to induce half-maximal acceleration of single-site TNP-ATP hydrolysis. However, nearly 40 mM of ATP was required for the \( \alpha_3 \beta_3 \gamma \) complex. Chase-added ATP should bind to the second (or third) nucleotide binding sites of the complex whose first site is already occupied by TNP-ATP. Although the result of the \( \alpha_3 \beta_3 \gamma \) complex is somehow not exactly parallel with the nucleotide binding affinity of the isolated \( \beta \) subunit, the affinity of the second (or third) site to ATP seem to reflect intrinsic nucleotide binding affinity measured for the isolated \( \beta \) subunit.

Different from the \( \alpha_3 \beta_3 \gamma \) complex, single-site hydrolysis of TNP-ATP by mutant \( \alpha_3 \beta_3 \) complexes proceeded more slowly than that by wild-type \( \alpha_3 \beta_3 \) complex (Fig. 4, D, F, and H, closed circles). The hydrolysis was not accelerated by the chase addition of 1 mM ATP (Fig. 4, D, F, and H, open circles). The rate of...

### Table I

| \( K_d \) for ATP | \( K_m \) | \( K_{50} \) | \( t_{1/2} \) |
|------------------|---------|---------|---------|
| mM               | mM     | mM     | s       |
| Wild-type \( \beta \) | 0.015   | 0.26    | <1      | 15      |
| \( \beta \)341L | 0.75    | 0.39    | <1      | 20      |
| \( \beta \)341A | >3      | >8      | ~1      | 60      |
| \( \beta \)341C | >3      | 5.3     | ~40     | 60      |

* Affinity of the isolated \( \beta \) subunit to ATP expressed as dissociation constants. The values were adapted from a reference (Odaka et al., 1994).

* \( K_m \) values of ATPase activities of the \( \alpha_3 \beta_3 \gamma \) complexes containing listed \( \beta \) subunits. The values were adapted from a reference (Odaka et al., 1994).

* The concentration of chase-added ATP necessary for 50% of the maximum acceleration of the single-site TNP-ATP hydrolysis by the \( \alpha_3 \beta_3 \gamma \) complexes containing listed \( \beta \) subunits.

* The time required for hydrolysis of 50% of the TNP-ATP by the \( \alpha_3 \beta_3 \gamma \) complex under the single-site hydrolysis condition. The values were estimated from Fig. 4.
single-site hydrolysis of TNP-ATP by each of mutant αβ3 complexes shows a parallel relation with the nucleotide binding affinity of each isolated mutant β subunit to ATP (Table I).

When the same experiments of difference spectra induced by the step-wise addition of TNP-ADP as shown for the wild-type complex (Fig. 3) were repeated for the αβ3γ complexes containing mutant β subunits, very similar transition of the spectra with increasing TNP-ADP were observed, that is, β subunit-type spectra (molar ratio < −1.5) at first and then α subunit-type spectra (molar ratio > −1.5) (data not shown). Therefore, in agreement with the results of single-site catalysis, a high affinity site is generated on one of the mutant β subunits when the mutant β subunits are assembled into the αβ3γ complex in spite of the fact that the isolated mutant β subunits have only very weak affinities to nucleotides (Odaka et al., 1994). For the mutant αβ3 complexes, the magnitude of difference spectra were very small, and clear transition as described above was not observed (data not shown).

**DISCUSSION**

The Role of γ Subunit in Generation of a High Affinity Nucleotide Binding Site on the β Subunit—It has been known that the affinity of the isolated TF γ subunit to TNP-ADP (apparent $K_d = 4.5 \mu M$) greatly increases when it is assembled in the form of TF 1 ( $K_d = 22 \mu M$), and only a single β subunit out of three β subunits in a TF 1 molecule bears this high affinity site (Hisabori et al., 1992). Here, using TNP-ATP (or TNP-ADP) as safe nucleotides that do not induce destabilization of the αβ3 complex (Fig. 1), we compared nucleotide binding characteristics and kinetics of the αβ3 and αβ3γ Complexes. The main differences between these two complexes are: 1) a single high affinity nucleotide binding site exists on the αβ3γ complex but not on the αβ3 complex (Fig. 2) and 2) an acceleration of the single-site TNP-ATP hydrolysis by the chase addition of ATP is observed for the αβ3γ Complex but not for the αβ3 complex (Fig. 4). Thus, the γ subunit is responsible for both generation of a single high affinity catalytic site and communications among catalytic sites in the αβ3γ Complex. This conclusion may not be restricted in TF γ. Gao et al. (1995) reported recently that F1-ATPase from chloroplast thylakoid membrane lacking α and ε subunits, designated as CF 1(-ε), whose subunit composition should be αβ3γ, retained about four nucleotides (2 ADP and 2 ATP) after passage through two centrifuge gel filtration columns, whereas the αβ3 complex, whose subunit composition should be αβ3, retained about one ATP. Although some ambiguity remained because of the retention of one ATP on the αβ3 complex, the results suggested that binding of the γ subunit to αβ3 structure induces a three-dimensional conformational transition that is necessary for high affinity asymmetric nucleotide binding.

The importance of the γ subunit in coupling of ATP hydrolysis and proton translocation was pointed out a long time ago (Yoshida et al., 1977b), and recent studies using cross-linking and fluorescent probes have provided evidence for movement or large conformational change in the γ subunit during catalysis (Turina and Capaldi, 1994; Aggeler et al., 1995). Because the αβ3γ complex should have a structure with a perfect 3-fold symmetry and it contains essentially no endogenously bound adenosine 5’-diphosphate (Aloise et al., 1991).
Role of the γ Subunit in Function of F₁-ATPase

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