Rotation of a Complex of the $\gamma$ Subunit and c Ring of Escherichia coli ATP Synthase

THE ROTOR AND STATOR ARE INTERCHANGEABLE*

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ATP synthase ($F_{0}F_{1}$) transforms an electrochemical proton gradient into chemical energy (ATP) through the rotation of a subunit assembly. It has been suggested that a complex of the $\gamma$ subunit and c ring ($c_{10-14}$) of $F_{0}F_{1}$ could rotate together during ATP hydrolysis and synthesis (Sambongi, Y., Iko, Y., Tanabe, M., Omote, H., Iwamoto-Kihara, A., Ueda, I., Yanagida, T., Wada, Y., and Futai, M. (1999) Science 286, 1722–1724). We observed that the rotation of the c ring with the c128T mutation (c subunit cIle-28 replaced by Thr) was less sensitive to venturicidin than that of the wild type, consistent with the antibiotic effect on the c128T mutant and wild-type ATPase activities (Fillingame, R. H., Oldenburg, M., and Fraga, D. (1991) J. Biol. Chem. 266, 20934–20939). Furthermore, we engineered $F_{0}F_{1}$ to see the $\alpha_{2}\beta_{2}$ hexamer rotation; a biotin tag was introduced into the $\alpha$ or $\beta$ subunit, and a His tag was introduced into the c subunit. The engineered enzymes could be purified by metal affinity chromatography and density gradient centrifugation. They were immobilized on a glass surface through the c subunit, and an actin filament was connected to the $\alpha$ or $\beta$ subunit. The filament rotated upon the addition of ATP and generated essentially the same frictional torque as one connected to the c ring. These results indicate that the $\gamma c_{10-14}$ complex is a mechanical unit of the enzyme and that it can be used as a rotor or a stator experimentally, depending on the subunit immobilized.

ATP is synthesized in chloroplasts, mitochondria, and bacteria by a ubiquitous ATP synthase ($F_{0}F_{1}$) coupled with an electrochemical proton gradient. The $F_{0}F_{1}$ of Escherichia coli consists of a catalytic sector, $F_{1}$ ($\alpha_{3}\beta_{3}\gamma\delta_{3}$), and a proton pathway, $F_{0}$ ($ab_{3}c_{10-14}$) (for reviews, see Refs. 1–6). The amino- and carboxyl-terminal $\alpha$ helices of the $\gamma$ subunit occupy the central space of the $\alpha_{3}\beta_{3}$ hexamer, as shown by the high resolution structure (7). The catalytic sites in the three $\beta$ subunits participate alternately in ATP synthesis and also in hydrolysis, as predicted from the binding change mechanism (6). The mechanism also proposes that the $\gamma$ subunit rotation plays a major role in the conformational transmission among the $\beta$ subunits.

The roles of the $\gamma$ subunit in energy coupling and catalytic cooperativity have been shown by extensive genetic studies (2). Mutation and suppression studies have suggested that the $\gamma$ subunit carboxyl-terminal domain and amino-terminal Met-23 interact through long range conformational transmissions involving the movement of the two helices (8, 9). $\gamma$ subunit rotation has been suggested by $\beta$–$\gamma$ cross-linking followed by dissociation and reconstitution (10) and the movement of a probe attached to the carboxyl terminus of the $\gamma$ subunit (11). Continuous rotation has been observed directly as the movement of an actin filament connected to the $\gamma$ subunit in the $F_{1}$ sector (12–14). The rotation of the $\epsilon$ subunit with the $\gamma$ subunit was also shown subsequently (15). Thus, this enzyme can be defined as a biological nanomotor carrying out rotational catalysis (16).

In ATP hydrolysis, the mechanical work done by the $\gamma$ subunit rotation should be transmitted to the $F_{1}$ sector for ATP-dependent proton transport. During ATP synthesis, proton transport should drive the $\gamma$ subunit rotation, which causes the $\beta$ subunit conformational change to release the product ATP. Therefore, it is essential to determine how the $\gamma$ subunit rotation is coupled to the proton transport through $F_{0}$.

Studies on the $F_{0}$ sector involving electron (17) and atomic force (18, 19) microscopy suggested that the $c$ subunits form a ring structure and that subunits $a$ and $b$ are located outside the ring. A ring formed from 12 $c$ subunits was proposed for the E. coli enzyme from the model of the solution structure (20, 21) and genetic fusion (22, 23), and the yeast structure formed from 10 monomers was observed by x-ray diffraction (24). A ring of 14 monomers was observed more recently for the chloroplast enzyme (25). The rotation of the $c$ ring with the $\gamma$ subunit has been proposed (26–29) and has been suggested by chemical cross-linking (30, 31). We recently provided the first direct evidence that the $c$ ring rotates continuously together with the $\gamma$ subunit in $F_{0}F_{1}$ (32); $F_{0}F_{1}$ was immobilized on a glass surface through His tags connected to the $\alpha$ subunits, and an actin filament was attached to the $c$ ring. Pänke et al. (33) have also demonstrated the $c$ ring rotation more recently using a different experimental system. The x-ray structure of the yeast enzyme showed the tight association between the $\gamma$ subunit and the $c$ ring, also suggesting that they can rotate together as an assembly (24).

As shown previously by Fillingame et al. (34), the membrane ATPase activity of the $c$ subunit cIle-28 (cI28) to the Thr (cT28) mutant was resistant to venturicidin, an effective inhibitor of the wild type. In this study, we observed that the $c$ ring rotation became less sensitive to venturicidin when the c128T mutation

* This work was supported in part by the Japanese Ministry of Education, Science, and Culture. 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 1734 solely to indicate this fact.

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was introduced into the c subunit. Furthermore, the αβ3 hexamer could rotate when F₀F₁ was immobilized through the c ring. These results indicate that a complex of the eγ subunit and the c ring is a mechanical unit of the nanomotor and can be a rotor or stator in an experimental system, depending on the subunit immobilized.

EXPERIMENTAL PROCEDURES

Plasmids, Bacterial Strain, and Growth Conditions—A plasmid (pBWU13) carrying the entire gene for F₀F₁ (35) was engineered for the rotation of the αβ3 hexamer. Codons for the (His)₉-Leu-His (His) tag were inserted between Met-1 and Asn-3 of the c subunit, and codons for the 123 (Val-18–Tyr-140) and 105 (Lys-20–Leu-124) amino acid residues of the transcarboxylase biotin binding domain (biotin tag) (including multilocating sites from the PinPoint Xa-1 vector (Promega)) were inserted, respectively, into the amino-terminal regions of the α and β subunits (both between the 1st and 2nd codons). Plasmid pBWU13 was also engineered for the c ring rotation, as described previously (32): a His tag, downstream of the initiation codon of the α subunit; cGlu-2 → Cys in the c subunit; and γCys-87 → Ala and γCys-112 → Ala in the γ subunit (32). The c28T (cIle-28 replaced by Thr) mutation was further introduced into the engineered F₀F₁ for the c ring rotation by a polymerase chain reaction-based method.

Recombinant plasmids were introduced into E. coli strain DK8 (Δψ) (which lacks the F₀F₁ gene (36)). Cells were grown at 37 °C in a synthetic medium containing 0.5% glycerol for enzyme purification, or in the same medium containing 0.5% succinate for testing growth as to oxidative phosphorylation (35). Biotin (2 mM) was purified from membranes (DK8/pBWU13) as described previously (35). Modified procedures (32) were used for the engineered enzyme with an engineered wild-type enzyme (Table I), except that the engineered F₀F₁-ATPase activity was assayed in 10 mM Hepes/NaOH (pH 7.2), 6 mM MgCl₂, and 5 mM Na₂Tris ATP (pH 8.0). The F₀F₁ was incubated with DCCD (10–40 μM) for 10 min at 20 °C before starting the reaction.

Other Procedures and Materials—Protein concentrations and the ATP-dependent formation of an electrochemical proton gradient were assayed as described previously (35). Fluorescently labeled biotinylated actin filament preparations were prepared as described (13). Venturicidin was kindly supplied by Dr. R. H. Fillingame. Triton X-100 was obtained from Nacalai Tesque (Kyoto, Japan). C₁₂E₈ was from Calbiochem. DNA-modifying enzymes were obtained from Takara Shuzo C., Ltd. or New England Biolabs (Beverly, MA). Other chemicals were of the highest grade commercially available.

RESULTS

Properties of the Engineered F₀F₁ for Rotation—Plasmids for two types of engineered F₀F₁ were constructed in this study: (a) the replacement of Glu-2 with Cys in the c subunit and the introduction of a His tag into the α subunit (αHis tag) with or without the c subunit Ile-28 to Thr (cI28T) mutation and (b) the introduction of a His tag into the c subunit and a biotin tag into the α (or β) subunit. The recombinant plasmids encoding these enzymes were introduced into E. coli strain DK8 lacking the entire F₀F₁ gene (36). The transformed cells could grow on succinate through oxidative phosphorylation similar to those harboring the control plasmid encoding the nonengineered F₀F₁ (Table I). These results indicate that the genetic modification for observing rotation did not affect the catalysis by or energy coupling of the enzyme.

Effects of DCCD and Venturicidin on the Purified Engineered F₀F₁—The engineered F₀F₁-ATP synthases could be purified by a method involving affinity chromatography and glycerol gradient centrifugation. They comprised eight subunits (Fig. 1) and showed substantial ATPase activities (30–50% of the non-engineered wild-type enzyme) (Table I), except that the engineered enzyme with an α subunit biotin tag was difficult to purify for an unknown reason and was obviously less pure than the other enzymes (Fig. 1, lane 3). The positions of the α and β subunits with a biotin tag and biotinylation of c subunit cE2C...
were confirmed by immunoblotting with streptavidin (data not shown). The c subunit with a His tag showed significantly stronger staining than that without the tag.

Similar to the nonengineered F₀F₁, the engineered F₀F₁ exhibited about 70–80% inhibition with 40 μM DCCD (assayed after a 10-min incubation without ATP). The enzyme with the c128T mutation exhibited reduced sensitivity (Table I), confirming the previous results for membrane ATPase with the same mutation (34). However, all of the F₀F₁ was unaffected by DCCD when assayed in the buffer used for rotation, although it showed low but significant sensitivity after a 30-min incubation (about 30% inhibition with 40 μM DCCD). This apparent low DCCD sensitivity may be attributable to the high protein concentration in the buffer, including serum albumin and the enzymes to regenerate ATP, or to the presence of a sulfhydryl agent. Therefore, it was not possible to examine DCCD for the rotation of an actin filament connected to the c ring because the inhibitor should be effective right after the addition to the rotating filament.

The inhibitory effect of venturicidin on the ATPase activity was examined; about 50% of the activity of the engineered F₀F₁ (cE2CαHis tag) as to the c ring rotation was inhibited by 8 μM venturicidin, whereas ~30% of the activity of c128T was inhibited (Fig. 2a), confirming the previous results for membrane enzymes (34). These enzymes became less sensitive when assayed in the buffer for rotation (Fig. 2b). However, this antibiotic can be considered as a specific inhibitor for the engineered F₀F₁ under the rotation conditions because it was less inhibitory for the enzyme with the c128T mutation and not inhibitory for the F₁, at all. It should be noted that specific inhibition was observed after a 5-min incubation, suggesting that venturicidin can be tested upon addition to the rotating enzyme.

The engineered enzyme with the β biotin tag/cHis tag for αβ c hexamer rotation was significantly less sensitive to venturicidin; only about 15% inhibition was observed with excess venturicidin (Fig. 2c). This result may be attributable to the introduction of the His tag into the c subunit. Therefore, the antibiotic could not be tested for the rotation of this enzyme.

Rotation of an Actin Filament Connected to the c Ring and Effect of Venturicidin—The rotation of actin filaments connected to the c ring was confirmed, and it was unlikely that the filaments were connected to the γ subunit of the contaminating F₁, as discussed previously (32). As a control, F₁ (with no cysteine in the γ subunit) was isolated from the engineered F₀F₁ to test whether the actin filament can bind to the Cys-less γ and rotate with the addition of ATP. We could find no rotating filament, indicating that the contaminating Cys-less γ F₁, if any, is not responsible for the rotation observed.

The rotation was also tested in the presence of venturicidin. On comparison of the filament rotation before and after the antibiotic addition, we found that venturicidin increased the pauses in the wild-type (c128) engineered F₀F₁ (Fig. 3a, b, c), confirming the previous results (32). On the other hand, the filament connected to the c ring with the c128T mutation showed apparently less frequent pauses after venturicidin addition; the ratios of events (pauses after venturicidin addition and pauses before venturicidin addition) were 2 for filaments with c128T and 4 for those without (Fig. 3d).

We examined the effect of the c128T mutation on the frictional torque generated by the c ring rotation. Torque can be determined from the filament rotation rate and viscous drag. Because the rotational rates of single filaments varied slightly and paused, as described above, we selected more than 20 linear segments with no pauses from the rotation time course of each filament and plotted the average rate (Fig. 3e). The c128T mutation showed no significant effect on the filament rotation or torque generation (Fig. 3e). Furthermore, venturicidin showed no effect on the torque generation (Fig. 3e, open and closed triangles).

Rotation of an Actin Filament Connected to the αβ Hexamer in F₀F₁—The results obtained for the c128T mutant strengthen the notion that a complex of the c ring and the γ subunit rotates during ATP hydrolysis, and this prompted us to examine a further possibility. If the γ subunit and c ring form a mechanical unit, the αβ c hexamer rotates when the c ring is immobilized on a glass surface (Fig. 4a). Because the rotational rates of single filaments varied slightly and paused, as described above, we selected more than 20 linear segments with no pauses from the rotation time course of each filament and plotted the average rate (Fig. 4a). The rotation direction of the hexamer (counterclockwise viewed from the F₀ side) was consistent with that of the c ring (clockwise from the F₁) in F₀F₁, immobilized on a glass surface through the α or β subunit. The rotation of the αβ hexamer generated essentially the same torque (~40 piconewton-nm) as that of the c ring. An effect of venturicidin on the αβ hexamer rotation was not apparent, which is consistent with its slight effect on the ATPase activity of the F₀F₁ engineered for αβ rotation (Fig. 2). As a control, we
purified F₁ with a biotin tag in the α subunit (but no His tag in the γ subunit) and connected an actin filament to it. We found no rotating filament among 10,000 attached nonspecifically to the glass surface, supporting the results described above.

**DISCUSSION**

As predicted by the binding change mechanism for ATP synthesis and hydrolysis, the continuous rotation of the γ subunit in the F₁ sector has been observed during ATP hydrolysis (12–14). The γ subunit rotation should be transmitted to the F₀ sector for coupling with proton transport. We provided the first evidence that the c subunit ring rotates with the γ subunit in F₀F₁ (32). Pänke et al. (33) have also demonstrated the c ring rotation more recently. The present study further confirmed the co-rotation of the γ subunit with the c ring.

Fillingame et al. (34) have shown that membrane ATPase
activity (attributable to F$_b$F$_1$) became less sensitive when the cI28T mutation was introduced. It should be noted, however, that this antibiotic was not a strong inhibitor even for the wild-type enzyme (maximum inhibition, about 60%). We observed lower but significant inhibition of the engineered F$_b$F$_1$ when ATPase activity was assayed under the rotation conditions. Consistent with the results for ATPase activity, this antibiotic did not have a strong effect on the rotation, such as immediate cessation upon its addition, but rather showed specific inhibition of F$_b$F$_1$. An actin filament connected to the c ring showed increased pauses upon the addition of venturicidin, and the cI28T mutation decreased the inhibitor sensitivity. As described above, this antibiotic had no effect on the ATPase activity or the $\gamma$ rotation of the engineered F$_1$. These results suggest that venturicidin inhibits F$_b$F$_1$ rotation by binding to the c ring and that a complex of the $\gamma$ subunit and c ring was rotating.

We tested a different engineered enzyme for rotation by introducing a His tag into the c ring and a biotin tag into the $\alpha_3\beta_3$ hexamer. Thus, F$_b$F$_1$ was immobilized on a glass surface and connected with an actin filament without using chemistry such as cysteine residue modification with biotin-maleimide (12–15, 32). Upon the addition of ATP, the actin filament connected to the $\alpha_3\beta_3$ hexamer rotated counterclockwise when viewed from the F$_1$ side and generated similar torque to the $\gamma$ rotation. These results strongly suggest that the $\gamma_{cI28T}$ complex is a mechanical unit and rotates during ATP hydrolysis and that the rotor and stator of the entire complex are inter-changeable experimentally, depending on the subunit immobilized.

One model for the interaction of the $\gamma$ subunit with the c ring during ATP synthesis and hydrolysis predicts that the $\gamma$ subunit interacts with the hydrophilic loop between the transmembrane helices of each c subunit one by one during rotation (discussed in Refs. 38 and 39). The rotation of the actin filament connected to the c ring indicates that this model will not work. However, it may be possible to argue that the c ring rotated with the $\gamma$ subunit as a part of the fluorescent probe (actin filament). The rotation of the filament connected to the $\alpha_3\beta_3$ hexamer finally excluded this possibility because the model predicts that the filament should not rotate if the c ring is immobilized and the $\gamma$ subunit moves on the ring.

Tsunoda et al. (40) have claimed that they could not connect an actin filament specifically to the c ring under their experimental conditions and criticized the experiments by Sambongi et al. (32) showing c ring rotation. The protocols used by the two groups are different, including the positions of Cys residues introduced into the c subunit (Sambongi et al. (32), Glu-2 $\rightarrow$ Cys; Tsunoda et al. (40), Cys inserted between Glu-2 and Asn-3), the conditions for maleimide modification (Sambongi et al. (32), 4 °C at pH 7.0; Tsunoda et al. (40), 25 °C at pH 7.5), and the detergent used for F$_b$F$_1$ preparation. Furthermore, in most experiments, Tsunoda et al. (40) reacted F$_b$F$_1$ with fluorescein-5-maleimide and then biotinylated anti-fluorescein IgG and the actin filament through streptavidin. On the other hand, Sambongi et al. (32) reacted F$_b$F$_1$ with biotin-maleimide and then connected an actin filament through streptavidin. Thus, the arguments raised by Tsunoda et al. (40), which are based on mostly negative observations, may reflect the differences in the experimental systems. As described in this study, the conclusion of Sambongi et al. (32) is supported by Pänke et al. (33) and the effect of venturicidin on the cI28T mutant, as described above. Furthermore, the actin filament connected to the $\alpha_3\beta_3$ hexamer in the immobilized F$_b$F$_1$ rotated, as discussed above.

In conclusion, we clearly showed that a complex of $\gamma$ and the c ring is a mechanical unit and that it rotates as an assembly. The preparations we used have all subunits of F$_b$F$_1$, and previous studies indicated that it is not easy to dissociate F$_b$F$_1$ even under conditions that release F$_b$ from membranes (41). Genetic (42) and reconstitution (43) studies have suggested that the three F$_b$ subunits (a, b, and c) are required for the formation of the F$_b$F$_1$ complex. However, we could not prove definitely that the rotating enzyme during video recording has the original integrity of F$_b$F$_1$. In this regard, Sambongi et al. (32) did not conclude that the rotating enzyme had all the subunits. Pänke et al. (33) stated more clearly that they might have observed the rotation of the c ring in incomplete F$_b$F$_1$. Although we accept this reservation as to the integrity of the rotating complex, we strongly suggest that the rotation of a complex of the $\gamma$ subunit and c ring is related to the fully functional F$_b$F$_1$. The obvious next step is to examine subunit rotation during ATP synthesis and to detect ATP synthesis when the c ring or $\gamma$ subunit is artificially rotated.

Acknowledgments—We thank Le Phi Ngia for technical assistance during the early stage of this work.

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FIG. 4. Rotation of an actin filament connected to the $\beta$ or $\alpha$ subunit. a, experimental system for observing the rotation of filaments connected to $\alpha$ subunits. b, time courses of rotating filaments (1.3 and 1.9 $\mu$m) connected to c, $\alpha$, and $\beta$ subunits. c, effect of the actin filament length on the rotational rate. The rotational rates of actin filaments connected to $\beta$ (open circles) or $\alpha$ (closed circles) subunits were plotted against filament length. The dotted line represents the calculated rotational rate of the filaments with a constant torque value of 40 piconewtonm.
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J. Biol. Chem. 2001, 276:15269-15274.
doi: 10.1074/jbc.M100289200 originally published online February 13, 2001

Access the most updated version of this article at doi: 10.1074/jbc.M100289200

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