The Formation or the Reduction of a Disulfide Bridge on the γ Subunit of Chloroplast ATP Synthase Affects the Inhibitory Effect of the ε Subunit*

Toru Hisabori‡§, Ken Motohashi‡, Peter Kroth¶, Heinrich Strotmann*, and Toyoki Amano‡

From the ‡Research Laboratory of Resources Utilization, Tokyo Institute of Technology, Nagatsuta 4259, Midori-ku, Yokohama 226–8503, Japan and the *Institut für Biochemie der Pflanzen, Heinrich-Heine Universität Düsseldorf, Universitätsstraße 1, D-40225 Düsseldorf, Germany

We have studied the change of the catalytic activity of chimeric complexes that were formed by chloroplast coupling factor 1 (CF1) -γ, α and β subunits of thermophilic bacterial F1 after formation or reduction of the disulfide bridge of different γ subunits modified by oligonucleotide-directed mutagenesis techniques. For this purpose, three mutant γ subunits were produced: γC199A, here 37 amino acids from Pro-194 to Ile-230 are deleted, γC199A, Cys-199 is changed to Ala, and γA200-204, amino acids from Asp-200 to Lys-204 are deleted. All of the chimeric subunit complexes produced from each of these mutant CF1-γ subunits and α and β subunits from thermophilic bacterial F1 lost the sensitivity against thiol reagents when compared with the complex containing wild-type CF1-γ. The pH optimum (pH 8.5–9.0) and the concentration of methanol to stimulate ATPase activities were not affected by these mutations. These indicate that the introduction of these mutations did not change the main features of ATPase activity of the chimeric complex.

However, the interaction between γ subunit and ε subunit was strongly influenced by the type of γ subunit itself. Although the ATPase activity of the chimeric complex that contained γC200-204 or γC199A was inhibited by the addition of recombinant ε subunit from CF1, similarly to complexes containing the reduced wild-type γ subunit, the recombinant ε subunit did not inhibit the ATPase of the complex, which contained the oxidized form of γ subunit. Therefore the affinity of the ε subunit to the γ subunit may be dependent on the state of the γ subunit or the ε subunit may bind to the oxidized form of γ subunit in a mode that does not inhibit the activity. The ATPase activity of the complex that contains γA194-230 was not efficiently inhibited by ε subunit. These results show that the formation or reduction of the disulfide bond on the γ subunit may induce a conformational change in the region that directly affects the interaction of this subunit with the adjacent ε subunit.

FεF1-ATP synthase synthesizes ATP from ADP and P1 at the expense of a proton-motive force (1–3). The enzymes consist of the membrane-embedded sector F1, responsible for proton translocation, and the extrinsic catalytic part F1. The architecture of F1 is very similar in various kinds of cells or organelles. The F1 part is composed of five different subunits designated as α, β, γ, δ, and ε, and their molecular stoichiometry is 3:3:1:1:1. Nucleotide binding sites reside on each of the α and β subunits, i.e., there are altogether 6 nucleotide binding sites per F1. The catalytic sites are located at the interfaces between α and β subunits. The high resolution x-ray structure of bovine heart mitochondrial F1 confirmed that most of the amino acid residues that form this site are provided from the β subunit (4). The α and β subunits, which have a similar three-dimensional structure, alternate in a hexagonal arrangement around a central cavity containing the γ subunit as already expected from previous electron microscopic studies (5). The crystal structure of an αβεδγ complex from the thermostable Bacillus PS3 was completely symmetric (6), but the incorporation of the γ subunit into this complex induced a functional asymmetry among the three catalytic sites (7).

Rotation of the γ subunit related with catalysis was suggested from kinetic results (8), the exchange of a disulfide bridge formed between γ and β subunits (9), and polarization anisotropy relaxation measurement of the fluorophore-labeled γ subunit of chloroplast F1 (CF1)3 (10). Recently, Noji et al. (11) directly observed that this γ subunit rotates in the central cavity formed by α and β subunits like a motor axis during the ATP hydrolysis reaction. This experiment clearly shows that the interaction between the part of coiled-coil of the γ subunit and the inner surface of the central cavity formed by α and β subunits is not tight, and the interaction between the γ subunit and one of α or β subunit can alternate step by step in one direction according to the catalytic reaction occurring sequentially at each of three catalytic sites.

The CFγCF1-ATP synthase of chloroplasts is regulated by the proton gradient, which activates the enzyme and by the reduction or the oxidation of a disulfide bridge in the γ subunit, which modulates activity. The latter regulation is known as thiol modulation (12). The structural basis for the thiol modulation is assigned to the sequence motif of 9 amino acids comprising two cysteines in the γ subunit (13). In vitro reduction can be achieved by dithiothreitol (DTT) or other dithiols, but the natural reductant is a reduced thioredoxin f (14), which was

* This work was supported by grants from Yamada Science Foundation (Japan) (to T. H.), by a Grant-in-aid for Science Research (to T. H.) (08640822 and 09044209) from the Ministry of Education, Science, Sports and Culture of Japan, and by Deutsche Forschungsgemeinschaft (SFB 189) (to H. S.). 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.

§ To whom correspondence should be addressed. Tel.: 81-45-924-200; Fax: 81-45-924-5277; E-mail: thisabor@res.titech.ac.jp.

1 The abbreviations used are: CFγ1, chloroplast-coupling factor 1; DTT, dithiothreitol; CFε(–ε), CF1 with the ε subunit removed; EFε, F1 from the plasma membrane of E. coli; TFε, F1 from the plasma membrane of thermophilic Bacillus PS3; γA194-230, γ subunit of CF1 with 37 amino acids from Pro-194 to Ile-230 deleted; γC199A, γ subunit of CF1 with Cys-199 changed to Ala; γA200-204, γ subunit of CF1 with the amino acids from Asp-200 to Lys-204 deleted; γε, recombinant γ and ε subunits of CF1 from spinach chloroplast; Tricine, N-(2-hydroxy-1,1-bis(hydroxymethyl)ethyl)glycine.
The Interaction between γ and ε Subunits of CF₁

reduced by the photosynthetic electron transport chain via ferredoxin. Recently, Ross et al. (15) have succeeded in constructing a mutant γ subunit with one or two cysteines substituted by serine in the green algae *Chlamydomonas reinhardtii*. By these substitutions, CF₁ became a DTT-insensitive enzyme. On the other hand, Gabrys et al. (16) selected a couple of mutants with chloroplast ATPase redox responses that were different from that of the wild-type plant by screening the *Arabidopsis* grown from the seeds that were previously treated with mutagen. They selected some mutant plants that might contain mutations within the γ subunit of CF₁.

The ε subunit of CF₁ is known as an intrinsic inhibitor protein. Recently, Cruz et al. (17, 18) produced recombinant mutant ε subunits and tested their effects on the ATPase activity of ε-deficient CF₁ (CF₁(ε⁻)). They determined that the most important part of the ε subunit as an inhibitor was the NH₂-terminal region. Similar experiments were reported for the ε subunit of F₁ from *Escherichia coli* (EF₁) (19). These studies suggested that about 15 amino acid residues from the ATPase activity, they estimated a

(20) reported the three-dimensional structure of the isolated ε subunit from EF₁ solved in solution by NMR spectroscopy. The structure showed that the NH₂-terminal 90 amino acids form so-called β-sandwich structure with two five-stranded β sheets. The C-terminal domain, on the other hand, is formed by two α-helices. Recently, Uhlin et al. (21) solved the crystal structure of this subunit at 2.3 Å resolution. They confirmed the β-sandwich structure reported by Wilkens et al. (20) and found that the C-terminal two helices arranged in an anti-parallel coiled-coil structure. From the three-dimensional structure and cross-linking experiments employing cysteine mutants of γ and ε subunits, it was concluded that the contact region of ε subunit to γ subunit is at one side of the β-sandwich structure (20, 22).

The results of Capaldi and co-workers (20, 22) show that about 40 amino acids from the NH₂ terminus of the ε subunit, which form one-half of the β-sandwich structure, represent the region where the subunit is in direct contact with the γ subunit. Recently Schulemburg et al. (23) also reported that the ε subunit of CF₁ can contact the γ subunit at the similar region. The characteristics of the interaction between γ subunit and ε subunit of CF₁ were investigated directly (24, 25) and indirectly (26). Andraloje and Harris (24) reported that the affinity of the ε subunit to CF₁(ε⁻) was decreased when the γ subunit of the complex was in a reduced state. From the inhibition of Ca²⁺-ATPase activity, they estimated a $K_D$ of 60 nM for the reduced CF₁(ε⁻) and 0.14 nM for the oxidized one. Duhe and Selman (25) also reported a stimulation of dissociation of the ε subunit from CF₁ of *Chlamydomonas* in the presence of DTT. They suggested that the dissociation of the ε subunit is an obligatory process in the DTT-induced unmasking of ATPase activity of soluble CF₁. Soteropoulos et al. (26) reported an activation of CF₁-ATPase by dilution of the enzyme. For the reduced CF₁₂, a half-maximal activation was obtained at a much lower dilution than with oxidized CF₁₂, and they estimated that the affinity for the ε subunit would be decreased about 20-fold by the reduction of CF₁₂.

Recently, we reported on the reconstitution of a chimeric complex from recombinant α and β subunits from F₁ of the thermophilic *Bacillus* PS3 (TF₁) and the recombinant γ subunit (γ₁) from spinach CF₁ (27). The complex had substantial ATPase activity and this activity was affected by the disulfide/dithiol state of the two regulatory cysteine residues on the γ subunits. That means that ε imposed redox control on the chimeric complex. Furthermore, the activity of the chimeric complex was suppressed by the addition of recombinant ε subunit from CF₁(ε₁), but not by the addition of the ε subunit from TF₁. These results suggest that the regulatory functions of γ and ε subunits of CF₁ may be linked to each other.

Here, we prepared three modified γ subunits of CF₁ by oligonucleotide-directed mutagenesis. We investigated the effects of the mutations on the enzyme activity and its regulation in chimeric complexes formed by these γ subunits, ε₁, and αβ₃ from TF₁. We found the region around the disulfide bridge of the γ subunit to be important for the regulatory interaction between the γ subunit and ε subunit.

**EXPERIMENTAL PROCEDURES**

**Materials**—Restriction endonucleases were obtained from Toyobo Inc., Tokyo, Japan. The Bradford protein assay system was from Bio-Rad Laboratories. DTT was purchased from Nacalai Tesque, Kyoto, Japan. DTT was from Sigma. Other chemicals were the highest grade commercially available.

**Construction of the Plasmids for the Mutant γ and Their Expression**—Recombinant plasmids carrying the gene for the γ subunit from spinach plastids (atpC) was previously constructed (27). Oligonucleotide-directed mutagenesis was carried out as described by Kunkel et al. (28). The oligonucleotide used to create the γ₁ with the additional amino acid stretch of the γ subunit of CF₁ (from Pro-194 to Ile-230) deleted (γ₁Δ₁₉₄₋₂₃⁰) was: 5'-ATCCACACCTACTCCCTTTAAGAAAGACGCAA-ACACCGACATT'T-3'. The one used to create the γ₁ with Cys-199 changed to Ala (γ₁Δ₁₹₉) was: 5'-ACCCTACTCCCTGAACGGAGAAATTGCCGCACTACAAATGAAAAAA-3'. To create γ₁ with the amino acid sequence from Asp-200 to Lys-204 deleted (γ₁Δ₂₀₀₋₂⁰⁴), the following oligonucleotide was used: 5'-ACCTACTCCCTTTAAGACCCGAAAGAGAAGAATTGGCTGTGTCGCACCGCAGAA-3'. Each of the genes was transferred to the expression vector pET23d (Novagen) and was transformed into the expression host *E. coli* strain BL21(DE3). Each of the γ₁ proteins was expressed yielding inclusion bodies and further purified by the methods described previously (27).

**Expression and Purification of the Recombinant ε₁**—The plasmid pSocB149 (28), which contains the gene for the subunit ε₁ of CF₁, from spinach (ε₁) was a generous gift from Dr. Whitfeld, R. P., Australia. The expression plasmid for ε₁ was constructed according to the method described by Hisabori et al. (27) and transformed into *E. coli* strain BL21(DE3). ε₁ was over-expressed by the method used for γ₁. The ε₁ inclusion bodies were first dissolved by the addition of 8 M urea, 40 mCrystal-Cl (pH 8.0), 0.4 mM DTT, and 0.8 mM EDTA and further purified by the method described previously (27).

**Preparation of α and β Subunits of TF₁**—The recombinant α and β subunits of TF₁ were expressed in *E. coli* strain DKS (bgIR, thi-1, rel-1, Hfr, P01, ∆uncB-uncCil::Tn10) and purified as described previously (30, 31).

**Reconstitution of the Chimeric Subunit Complex**—Reconstitution of the chimeric subunit complex was formed by the same method described previously (27). Briefly, each of the isolated γ₁ subunits was mixed with the α and β subunits from TF₁ in a ratio of 1:1 (w/v), and a solution containing 8 M urea, 1 mM EDTA, 0.5 mM DTT, and 50 mM Tris-Cl (pH 8.0) was added to yield a final urea concentration of 4 M. The solution was then dialyzed against 50 mM Tris-Cl (pH 8.0), 200 mM NaCl, 0.4 mM MgCl₂, and 0.4 mM ATP at 20 °C for 3 h. After the dialysis, the unsolved γ₁ subunits were removed from the solution by centrifugation and provided for the measurement of ATPase activity.

**Activation and Deactivation of the Complex and the Measurement of ATPase Activity**—To activate or deactivate the αβγ complexes by the formation or reduction of disulfide bridge on the γ subunit, formed complexes were incubated in the presence of 2 mM DTT or 50 μM CuCl₂ for 2 h at 30 °C. Then 10 μl of the complex solution (normally containing 2–3 μg of protein) was added to 90 μl of the reaction mixture containing 50 mM Tricine-KOH (pH 8.0), 2 mM ATP, and 2 mM MgCl₂ to initiate the reaction. The reaction was continued for 5–10 min, and then terminated by the addition of 100 μl of ice-cold 2.4% (w/v) perchloric acid. The activated or inactivated ε₁ complexes were analyzed by a celliminitigen for the ATPase activity was measured by the mixture of the chimeric complex and the monomer proteins, which were not incorporated into the celliminitigen, the specific activity of the ATPase was calculated based on the amounts of α plus β subunits that were used for the formation of the complex.

**RESULTS AND DISCUSSION**

**Responses of the Chimeric Complexes Containing Mutant γ₁ to Oxidation/Reduction**—In comparison to the γ subunit from thermophilic bacteria, the γ₁ subunit of CF₁ contains a stretch of
The Interaction between \( \gamma \) and \( \epsilon \) Subunits of CF1

![Amino Acid Aligments](image)

**FIG. 1.** Partial amino acid alignments of \( \gamma \) subunits. A, alignment of the protein sequences of the \( \gamma \) subunits of F1 complexes from spinach chloroplast (SPI) (13), E. coli (ECO) (38, 39), thermophilic Bacillus PS3 (PS3) (29), and bovine heart mitochondria (MIT) (40) was made by computer program CLUSTALW (41). The positions that are identical in three of the four sequences are marked by “\(*\)” and the identical positions in all of the sequences are marked by “\(*^*\)”. Gaps are marked by broken lines. Two cysteines (Cys-199 and Cys-204), which form the disulfide bridge, were shown as reversed capitals. Only the sequences from Leu-179 to Ile-263 of spinach CF1-\( \gamma \) subunit and the corresponding region of others are shown. B, the sequences from Leu-189 to Thr-234 of spinach CF1-\( \gamma \) subunit were engineered and expressed in E. coli. Amino acid positions that have been deleted are shown as broken lines.

an additional 37 amino acids comprising the two cysteines (Cys-199 and Cys-205), which take the thiol modulation (Fig. 1A). To characterize the differences of the properties between bacterial \( \gamma \) and chloroplast \( \gamma \), we designed three mutations of the \( \gamma \) subunit of CF1. These were \( \gamma_{194-230} \), which is lacking the CF1-\( \gamma \) specific additional amino acid stretch (see Fig. 1B). \( \gamma_{200-204} \), in which the space between the two regulatory cysteines is shortened, and \( \gamma_{199A} \), which cannot form the disulfide bridge involved in thiol modulation. The complexes containing these mutant \( \gamma \) subunits were not supposed to show any responses against oxidation/reduction. As shown in Fig. 2, the ATPase activity of these chimeric complexes was not altered by the incubation with DTT or with CuCl2, although the ATPase activity of the complex containing wild-type \( \gamma \) subunit was strikingly stimulated by the incubation with DTT and suppressed by the incubation with CuCl2 as reported previously (27).

**Subunit—\( \delta \) Subunit Complexes**—The Mg\(^{2+}\)-ATPase activity of the oxidized form of CF1-\( \gamma \) subunit was decreased by the mutation of \( \gamma \) subunit on the ATPase of EF1 expressed in E. coli (35). In a previous report, we found that the deletions of 6 amino acids from the C terminus of EF1 had no effect on the ATPase of EF1, whereas the deletions of 11 amino acids from the NH2 terminus decreased the inhibitory effect of this subunit on the ATPase of EF1 (36). Recently, Cruz et al. (17, 18) expressed the mutant \( \epsilon \) subunit of CF1 in E. coli and investigated their inhibitory effects on the CF1(\(- \epsilon\)). Similar deletion experiments were carried out for EF1 by Jouonouchi et al. (19). They reported that the deletion of the NH2-terminal 16 amino acids is strongly affecting the coupling between ATP hydrolysis and H\(^+\) translocation, but \( \delta \) subunit with an \( \epsilon \) subunit lacking the 15 amino-terminal residues could bind to F0 in a functionally competent manner. However, Cruz et al. (17) found that the deletions of 6 amino acids from the C terminus or the deletions of 11 amino acids from the NH2 terminus decreased the inhibitory effect of this subunit on the ATPase of CF1.

**Inhibitory Effects of the \( \epsilon \) Subunit**—The \( \epsilon \) subunit of F1 is an intrinsic ATPase inhibitor. However, this subunit may also be involved in H\(^+\) coupling of the ATPase (36). Recently, Cruz et al. (17, 18) expressed the mutant \( \epsilon \) subunit of CF1 in E. coli and investigated their inhibitory effects on the CF1(\(- \epsilon\)). Similar deletion experiments were carried out for EF1 by Jouonouchi et al. (19). They reported that the deletion of the NH2-terminal 16 amino acids is strongly affecting the coupling between ATP hydrolysis and H\(^+\) translocation, but \( \delta \) subunit with an \( \epsilon \) subunit lacking the 15 amino-terminal residues could bind to F0 in a functionally competent manner. However, Cruz et al. (17) found that the deletions of 6 amino acids from the C terminus or the deletions of 11 amino acids from the NH2 terminus decreased the inhibitory effect of this subunit on the ATPase of CF1.

The Mg\(^{2+}\)-ATPase activity of the oxidized form of CF1 is quite low or almost zero. However, the chimeric complex displayed remarkable Mg\(^{2+}\)-ATPase activity even in its oxidized form. Therefore it was possible to investigate the interaction between the \( \epsilon \) and \( \gamma \) subunits under the reduced and oxidized...
Andralojc and Harris (24) investigated the inhibition of Ca²⁺ by the chimeric complex with the oxidized γ subunit according to the method described under “Experimental Procedures.” A, the complexes with the wild-type γ subunit were formed and then incubated with 2 mM DTT (●) or 50 μM CuCl₂ (○) for 2 h at 30°C, and their ATPase activities were measured under the various concentrations of methanol indicated in the figure. B, the chimeric complex with γC₁₉₉₉₃₀−ₒ₀₀−₂₀₄ and γC¹₂₉₉₉₃₀ were formed, and their ATPase activities were measured.

conditions by measuring the inhibition of the ATPase. Surprisingly, the ATPase activity was less inhibited by the ε subunit in the chimeric complex with the oxidized γ subunit than with the reduced γ subunit (Fig. 4A) (27). The addition of methanol reduced the inhibitory effect of ε, for both the reduced and oxidized state complexes, indicating the stimulation effect of methanol on CF₁-ATPase can be partially attributed to the release of the ε subunit from the enzyme. A change of the responses of CF₁ against the ε subunit under the reduced or oxidized condition were already reported by Andralojc and Harris (24), Duhe and Selman (25), and Soteropoulos et al. (26). Andralojc and Harris (24) investigated the inhibition of Ca²⁺-ATPase activity. By adding the various amounts of isolated ε subunit to CF₁(−ε), they concluded that the oxidized CF₁(−ε) has a higher affinity for the isolated ε subunit than the reduced enzyme. Soteropoulos et al. (26) diluted CF₁ solution to nanomolar concentration and found a difference of the activation ratio by oxidation/reduction. Activation by dilution occurred for the reduced enzyme at higher than for the reduced enzyme. From their results, they concluded that the affinity of the ε subunit to the reduced CF₁ is about 20-fold lower than to the oxidized one.

It is difficult to explain why the apparent affinity of the ε subunit to the chimeric complex is lower when the γ subunit is in the oxidized state. Possibly the origin of the αβγ hexamer influences the interaction, too. On the other hand, we cannot be sure that the γ really has the same conformation in the chimeric complex as the authentic CF₁.

Relation between the Conformation of the γ Subunit and the Effect of the ε Subunit—If the additional amino acid stretch observed only on CF₁ is responsible for the interaction between the γ and ε subunits, a mutation of this segment might affect the inhibition of ATPase by the ε subunit. The sensitivity of the complexes that contain γC₁₉₉₉₃₀ or γC₁₂₀₀−₂₀₄ against the ε subunit was the same as that of the complex with the reduced form of the γ subunit (Fig. 4B), although we expected that the conformation of γC₁₂₀₀−₂₀₄ was similar to the oxidized form of the γ subunit.

On the other hand, the complex that contains γC₁₉₄−₃₀ was not inhibited by the addition of the ε subunit. From the report of Capaldi and co-workers (20, 22), the contact region between the ε and γ subunits is located around 40 amino acids from the NH₂ terminus of the ε subunit. The chemical cross-linking experiments carried out by using cross-linker-labeled ε subunit and CF₁(−ε) gave the same conclusion (23). The contact region on the γ subunit is very close to the position where the additional amino acid stretch is intercalated in CF₁-γ (13) (see Fig. 1A). Hence, the structure of this additional amino acid stretch might be very important not only for the redox regulation but also for the interaction between the γ and ε subunits.

Ross et al. (37) reported that mutation of the spacer region between the two regulatory cysteines (GEICDKD or AVDGK-D(CVDAA) diminished redox regulation of CF₁ from Chlamydomonas. They used C.reinhardtii strain atpC1, which lacks the gene for the γ subunit, and complemented the phosphorylation activity with mutated γ subunit genes. Thylakoid vesicles prepared from the mutant strain containing γD₁₁₉₉₉₅SDK₃₀ and γD₁₁₉₉₉₃₀ did not show a remarkable change of the photophosphorylation in the presence or the absence of DTT. Accordingly the authors concluded that the spacer region be-
between the disulfide bridge is also involved in redox regulation of CF$_1$-ATPase. This result corresponds with our result concerning the genes encoding the $\gamma_2$ subunit of spinach CF$_1$ (pSocB149). We also thank K. Saika, Dr. M.-H. Sato, and Dr. H. Noji for technical assistance. Thanks to F. Motojima for helpful discussion.

Acknowledgments—We thank Prof. R. G. Herrmann for providing us with a cloned gene for the $\gamma$ subunit of spinach CF$_1$ ( otpC gene), Dr. S. Werner-Grüne for preparing the otpC gene without transit sequence, and Dr. P. R. Whitfelt for providing us with a cloned gene for the $\gamma$ subunit of spinach CF$_1$ (pSocB149). We also thank K. Saika, Dr. M.-H. Sato, and Dr. H. Noji for technical assistance. Thanks to F. Motojima for providing us with the excellent program "Motojiman" to design the appropriate primer for the mutagenesis. Special thanks to Prof. M. Yoshida for helpful discussion.

REFERENCES

1. Futai, M., and Kanazawa, H. (1983) Microbiol. Rev. 47, 285–312
2. Strotmann, H., and Bickel-Sandköter, S. (1984) Annu. Rev. Plant Physiol. 35, 97–120
3. Senior, A. E. (1990) Annu. Rev. Biophys. Chem. 19, 7–41
4. Abrahams, J. P., Leslie, A. G. W., Lutter, R., and Walker, J. E. (1994) Nature 370, 621–628
5. Gogol, E. P., Agler, R., Sagermann, M., and Capaldi, R. A. (1989) Biochemistry 28, 4717–4724
6. Shirakihara, Y., Leslie, A. G., Abrahams, J. P., Walker, J. E., Ueda, T., Sekimoto, Y., Kambara, M., Saika, K., Kagawa, Y., and Yoshida, M. (1997) Structure ( Lond.) 15, 825–836
7. Kaibara, C., Matsui, T., Hisabori, T., and Yoshida, M. (1996) J. Biol. Chem. 271, 2433–2438
8. Boyer, P. D. (1993) Biochim. Biophys. Acta 1198, 215–250
9. Duncan, T. M., Bubgin, V. V., Zhou, Y., Hutcheon, M. L., and Cross, R. L. (1995) Proc. Natl. Acad. Sci. U. S. A. 92, 10964–10968
10. Sabbert, D., Engelbrecht, S., and Junge, W. (1996) Nature 381, 623–625
11. Noji, H., Yasuda, R., Kinoshita, K., Jr., and Yoshida, M. (1997) Nature 386, 299–302
12. Nalin, C. M., and McCarty, R. E. (1984) J. Biol. Chem. 259, 7275–7280
13. Miki, J., Maeda, M., Mukohata, Y., and Futai, M. (1988) FEBS Lett. 232, 221–226
14. Schwarz, O., Schürmann, P., and Strotmann, H. (1997) J. Biol. Chem. 272, 16924–16927
15. Ross, S. A., Zhang, M. X., and Selman, B. R. (1995) J. Biol. Chem. 270, 9813–9818
16. Gubers, H., Kramer, D. M., Crofts, A. R., and Ort, D. R. (1994) Plant Physiol. 104, 769–776
17. Cruz, J. A., Harfe, B., Radkowski, C. A., Dann, M. S., and McCarty, R. E. (1995) Plant Physiol. 109, 1579–1588
18. Cruz, J. A., Radkowski, C. A., and McCarty, R. E. (1997) Plant Physiol. 113, 1185–1192
19. Jounouchi, M., Takeyama, M., Nomi, T., Maeda, M., and Futai, M. (1992) Arch. Biochem. Biophys. 292, 87–94
20. Wilkens, S., Dahlquist, F. W., McIntosh, L. P., Donaldson, L. W., and Capaldi, R. A. (1995) Nat. Struct. Biol. 2, 961–967
21. Uhlin, U., Cox, G. B., and Guss, J. M. (1997) Structure ( Lond.) 15, 1219–1230
22. Tang, C., and Capaldi, R. A. (1996) J. Biol. Chem. 271, 3918–3024
23. Schulenberg, B., Wellner, F., Lill, H., Junge, W., and Engelbrecht, S. (1997) Eur. J. Biochem. 249, 134–141
24. Andraole, P. J., and Harris, D. A. (1990) Biochim. Biophys. Acta 1016, 55–62
25. Duhe, R. K., and Selman, B. R. (1990) Biochim. Biophys. Acta 1017, 70–78
26. Soteropoulos, P., Suss, K. H., and McCarty, R. E. (1992) J. Biol. Chem. 267, 10348–10354
27. Hisabori, T., Kato, Y., Motohashi, K., Strotmann, H., Kroth-Pancic, P., and Amano, T. (1997) Eur. J. Biochem. 247, 1158–1165
28. Kunkel, T. A., Bebenek, K., and McClary, J. (1991) Methods Enzymol. 204, 125–139
29. Munn, A. L., Whitfield, P. R., Bottomley, W., Hudson, G. S., Jans, D. A., Gibson, F., and Cox, G. B. (1991) Biochim. Biophys. Acta 1060, 82–88
30. Ohita, S., Yohda, M., Ichiraka, M., Hirata, H., Hamamoto, T., Otawara-Hamamoto, Y., Matsuda, K., and Kagawa, Y. (1988) Biochim. Biophys. Acta 953, 141–155
31. Ohtsubo, M., Yoshida, M., Ohita, S., Kagawa, Y., Yohda, M., and Date, T. (1987) Biochim. Biophys. Acta. Res. Commun. 146, 705–710
32. Yoshida, M., Sone, N., Hirata, H., and Kagawa, Y. (1977) J. Biol. Chem. 252, 2948–2945
33. Sakurai, H., Shinohara, K., Hisabori, T., and Shinohara, K. (1981) J. Biochem. (Tokyo) 90, 85–102
34. Selman-Reimer, S., Merchant, S., and Selman, B. R. (1981) Biochemistry 20, 5476–5482
35. Anthon, G. E., and Jagendorf, A. T. (1984) Biochim. Biophys. Acta 723, 358–365
36. Zhang, Y., Oldenburg, M., and Fillingame, R. H. (1994) J. Biol. Chem. 269, 10221–10224
37. Ross, S. A., Zhang, M. X., and Selman, B. R. (1996) J. Bioenegr. Biomembr. 28, 49–57
38. Kanazawa, H., Kayano, T., Mabuchi, K., and Futai, M. (1981) Biochim. Biophys. Res. Commun. 103, 604–612
39. Saraste, M., Gay, N. J., Eberle, A., Runswick, M. J., and Walker, J. E. (1981) Nucleic Acids Res. 9, 5287–5296
40. Walker, J. E., Fearnley, I. M., Gay, N. J., Gibson, B. W., Northrop, F. D., Powell, S. J., Runswick, M. J., Saraste, M., and Tybulewicz, V. L. J. (1985) J. Mol. Biol. 184, 677–701
41. Thompson, J. D., Higgins, D. G., and Gibson, T. J. (1994) Nucleic Acids Res. 22, 4673–4680