Movement of the Iron-Sulfur Subunit beyond the ef Loop of Cytochrome b Is Required for Multiple Turnovers of the bc₁ Complex but Not for Single Turnover Q₀ Site Catalysis*

Received for publication, August 19, 2001, and in revised form, November 12, 2001
Published, JBC Papers in Press, November 13, 2001, DOI 10.1074/jbc.M107974200

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Recent kinetics experiments using mutants of the bc₁ complex (ubihydroquinone-cytochrome c oxidoreductase) iron-sulfur subunit with modified hinge regions have revealed the crucial role played by the large scale movement of its [2Fe-2S] cluster domain during the activity of this enzyme. In particular, one of these mutants (+1Ala) with an insertion of one alanine residue in the hinge region is partially deficient in this movement. We found that this defect can be overcome by the appearance of a second mutation substituting the leucine at position 286 in the ef loop of cytochrome b with a phenylalanine. Detailed studies of these mutants and their derivatives revealed that the ef loop acts as a barrier that needs to be crossed for multiple turnovers of the enzyme but not for a single turnover ubihydroquinone oxidation site catalysis. These findings indicate that the movement of the iron-sulfur subunit is composed of two discrete parts: a "micro-movement" at the cytochrome b interface, during which the [2Fe-2S] cluster interacts with ubihydroquinone oxidation site occupants and catalyzes ubihydroquinone oxidation, and a "macro-movement," during which the cluster domain swings away from cytochrome b interface, crosses the ef loop, and reaches a position close to cytochrome c₁ heme, to which it ultimately transfers an electron.

Ubihydroquinone:cytochrome c oxidoreductase (the bc₁ complex) plays a crucial role in both respiratory and photosynthetic electron transfer chains (1–4). This enzyme catalyzes electron transfer from ubihydroquinone (QH₂) to a c-type cytochrome (cyt) and couples the energy thus released to proton transport across the membrane. The bc₁ complex is therefore a key component of cellular energy transduction systems and contributes to the establishment of an electrochemical gradient subsequently used for ATP production by the ATP synthase. A key step during the mechanism of the bc₁ complex, known as the Q cycle mechanism (5, 6), is the bifurcation of the electrons at the QH₂ oxidation (Q₀) site. Indeed upon QH₂ oxidation at this catalytic site, the two electrons released are conveyed to two different electron transport chains. One electron follows a high redox potential chain constituted of the [2Fe-2S] cluster borne by the iron-sulfur subunit and the cyt c₁ heme of the cyt c₁ subunit, whereas the other electron is transferred to a low redox potential chain constituted by two b-type hemes (low and high potential b-type heme) and reduces a ubiquinone (Q) or semiquinone radical in a second catalytic site (ubiquinone reduction site) located on the other side of the membrane (4).

During the past few years, major advances have been accomplished in our understanding of the structure and function of the bc₁ complex especially because of the resolution of its three-dimensional structure at atomic scale resolution (7–10). Comparison of various such structures in which the [2Fe-2S] cluster domain of the iron-sulfur subunit occupies different positions (3, 7–11) and various biophysical and biochemical data (3, 12–21) have led to the discovery of an unprecedented electron shuttle function for the iron-sulfur subunit of the bc₁ complex. Use of the phototrophic bacteria of Rhodobacter species as a model system has further advanced these studies especially via the engineering of mutants in the linker domain of the iron-sulfur subunit that acts as a hinge for the large scale domain movement (12, 13, 19–21). Remarkably, one such mutant (+1Ala) with a single alanine residue insertion in the hinge region considerably slowed down the movement and the associated electron transfer that is too fast to be time-resolved in the native enzyme (19). This finding allowed us for the first time to unveil the intra-enzyme complex domain motion kinetically and to define its lower and upper limits (4, 19). It also established that the motion is not rate-limiting for the function of the bc₁ complex because it is faster than the catalytic rate of this enzyme. Although convincing evidence has been accumulated over the past few years about the occurrence and importance of the domain movement during the catalytic cycle of the bc₁ complex, little is yet known about whether or not the movement is controlled during the Q₀ site catalysis and, if so, what are the regions of the enzyme that might be implicated in its control (4)?

Previous studies have demonstrated that an optimal combination of length and flexibility is required at the level of the iron-sulfur hinge domain to have a fully functional enzyme (12, 13, 16, 19–21). Additional studies have also revealed that the iron-sulfur subunit cluster domain interacts closely with the cd1 and cd2 loops of cyt b (14). In this work we present detailed studies of a revertant of the +1Ala mutant of the bc₁ complex (19) that regains its normal function upon the occurrence of a second mutation, substituting the leucine residue at position 286 of the ef loop of cyt b with a phenylalanine. The results reveal that the ef loop of cyt b is an important structural feature that interferes with the movement of the iron-sulfur subunit cluster domain and acts as a physical barrier that...
a single exponential equation (19, 20). They are expressed as a percentage of the values observed with a wild type amplitude of the EPR QH$_2$ oxidation.

We had noticed that under photosynthetic growth conditions rare Ps growth conditions on plates containing enriched medium supplemented with 2 mg/mL kanamycin or in anaerobic conditions in the same medium and under continuous light (19, 20). E. coli was grown in LB medium in the presence of 50 μg/mL kanamycin or ampicillin. All biochemical and physical techniques, including time-resolved light-induced absorption kinetics and EPR spectroscopy, are described in Refs. 19, 20, and 22 except that a dual wavelength instead of a single wavelength spectrophotometer (Biomedical Instrumentation Group, University of Pennsylvania, Philadelphia, PA) was used for some of the light-induced cyt c reduction or cyt b reduction kinetics obtained in this work.

**EXPERIMENTAL PROCEDURES**

*Rhodobacter capsulatus* strains were grown under semiaerobic conditions at 35 °C in the dark in enriched medium supplemented with 2 mM MgCl$_2$, 2 mM CaCl$_2$, and 10 μg/mL kanamycin or in anaerobic conditions in the same medium and under continuous light (19, 20). E. coli was grown in LB medium in the presence of 50 μg/mL kanamycin or ampicillin. All biochemical and physical techniques, including time-resolved light-induced absorption kinetics and EPR spectroscopy, are described in Refs. 19, 20, and 22 except that a dual wavelength instead of a single wavelength spectrophotometer (Biomedical Instrumentation Group, University of Pennsylvania, Philadelphia, PA) was used for some of the light-induced cyt c reduction or cyt b reduction kinetics obtained in this work.

**RESULTS**

**Genetic Analyses of the +1 Ala Revertant**—The +1 Ala mutant that contains an insertion of a single alanine residue in the hinge region of the iron-sulfur subunit grows under photosynthetic growth conditions noticeably slower (P$_{slow}$) than its wild type parent. This P$_{slow}$ growth is a result of its impaired bc$_1$ complex function caused by hindered movement of its iron-sulfur subunit cluster domain (19). We had noticed that under Ps growth conditions rare Ps colonies growing faster than the +1 Ala mutant readily appear on enriched medium containing plates. One such Ps colony was retained, purified extensively, and analyzed further. Appropriate subcloning and DNA sequencing experiments revealed that this Ps revertant contained a second mutation that substituted the leucine residue at position 286 of the ef loop of cyt b by a phenylalanine. To verify this, the L286F substitution in cyt b was solely responsible for the improved Ps phenotype of this revertant, the 920-bp Xmal-SfuI DNA fragment of the petABC operon encoding the bc$_1$ complex and containing this mutation was exchanged with its counterpart in a petABC operon carrying the +1 Ala mutation in the iron-sulfur subunit and yielded the mutant +1 AlaL286F. Moreover, to define the effect of the secondary mutation L286F alone, and also to probe its ability to suppress the Ps$^-$ defect of a +2 Ala mutant (19), the same DNA fragment was exchanged with its counterparts in the wild type petABC operon and in a petABC operon carrying the +2 Ala mutation (19) and yielded the single L286F and the double +2 AlaL286F mutants, respectively.

**Properties of the L286F, +1 AlaL286F, and +2 AlaL286F Mutants—**Ps growth properties of the newly obtained L286F, +1 AlaL286F, and +2 AlaL286F mutants were compared with those of the wild type, +1 Ala and +2 Ala mutants, respectively. The L286F mutation readily endowed Ps$^-$ growth ability to the +1 Ala mutant, but it was ineffective in the case of the +2 Ala mutant, whereas the L286F mutation alone did not exhibit any detectable Ps growth defect on plates containing enriched medium (Table I).

Next, whether or not the mutants assembled properly the iron-sulfur subunit into their bc$_1$ complexes was probed using SDS-PAGE and Western blot analyses (data not shown). The data indicated that a slight substoichiometry (<50%) was seen in the presence of the L286F mutation in all cases, and this effect was most visible when this mutation was alone. Moreover, the amplitude of the EPR g$_c$ signal that reflects the amount of the [2Fe-2S] cluster also indicated a similar defect (23, 24) (data not shown). In addition, the steady-state bc$_1$ complex activities measured using 2,3-dimethoxy-5-methyl-6-decyl-1,4-benzohydroquinone as a substrate analog indicated that the presence of the L286F mutation decreased the ability of the enzyme to reduce cyt c. This activity decrease was partly due to the substoichiometry of the iron-sulfur subunit in the mutants carrying the L286F mutation and possibly enhanced by the increased sensitivity to the detergent used to disperse the membrane preparations under the assay conditions (25). A

**TABLE I**

Properties of the +1 AlaL286F revertant and its derivatives

| Ps phenotype | Assembly | E$_{m/2}$(2Fe-2S)$_{1}$ | Steady-state activity | Electron transfer QH$_2$ to cyt $c^+$ | Electron transfer QH$_2$ to cyt $b^-$ |
|--------------|----------|------------------------|----------------------|--------------------------------------|--------------------------------------|
| Wild type    | Ps$^+$   | 100                    | 310                  | 100                                  | 100                                  |
| L286F        | Ps$^+$   | 100                    | 310                  | 100                                  | 100                                  |
| +1 Ala       | Ps$_{slow}$ | 140                    | 370                  | 120                                  | 125                                  |
| +1 AlaL286F  | Ps$^+$   | 100                    | 310                  | 100                                  | 100                                  |
| +2 Ala       | Ps$^-$   | 120                    | 410                  | 5                                    | 2                                    |
| +2 AlaL286F  | Ps$^-$   | 120                    | 410                  | 5                                    | 2                                    |

$^a$ Ps$^+$ and Ps$^-$ refer to photosynthetic growth ability or disability, respectively, and Ps$_{slow}$ indicates that the growth rate is significantly (about 2-fold) slower than that of the wild type.

$^b$ Assembly Fe-S subunit refers to the stoichiometry of the Fe-S subunit in respect to the cyt c$_1$ or cyt b subunits as determined by scanning of SDS-PAGE/immunoblots and expressed as a percentage of the wild type (20).

$^c$ Assembly [2Fe-2S] cluster refers to the relative amounts of the [2Fe-2S] cluster compared with that of the wild type, as determined by the amplitude of the EPR g$_c$ signal (20).

$^d$ The E$_{m/2}$ values were obtained after fitting the EPR g$_c$ signal amplitude during potentiometric dark titration of the [2Fe-2S] cluster (23, 24).

$^e$ Steady-state bc$_1$ complex activity refers to the 2,3-dimethoxy-5-methyl-6-decyl-1,4-benzohydroquinone: cyt c reductase activity expressed as a percentage of that of the wild type activity, which was approximately 3 μmol of cyt c reduced min$^{-1}$ mg of membrane protein$^{-1}$ (25).

$^f$ QH$_2$ to cyt c electron transfer rates were determined at 100 mV by recording cyt c reoxidation kinetics at 550–540 nm and fitting them to a single exponential equation (19, 20). They are expressed as percentages of the values observed with a wild type bc$_1$ complex, which was about 300 s$^{-1}$.

$^g$ QH$_2$ to cyt b electron transfer rates were determined at either 100 or 400 mV by recording cyt b reduction kinetics in presence of 5 μM antimycin A at 560–570 nm and fitting them to a single exponential equation (19, 20). They are expressed as percentages of the values observed with a wild type bc$_1$ complex, which were 500 and 60 s$^{-1}$ at 100 and 400 mV, respectively.

$^h$ ND, not determined.
similar effect has been encountered previously during the analyses of the iron-sulfur subunit hinge region deletion mutants (20). To further characterize these mutants, the effect of the L286F mutation on the redox midpoint potential \( E_m \) of the iron-sulfur subunit [2Fe-2S] cluster was also sought. Potentiometric dark titrations (22) of the EPR \( g \) signals were performed as previously (23, 24) (Fig. 1), and the redox midpoint potential at pH 7 \( (E_m) \) values thus obtained are shown in Table I. Clearly, the presence of the L286F mutation located in the ef loop of cyt b decreased the \( E_m \) of the iron-sulfur subunit [2Fe-2S] cluster by about 50 mV in all mutants as compared with their respective parent strains.

**Time-resolved cyt c Rereduction and cyt b Reduction Kinetics**—Next, detailed functional analyses of mutant bc1 complexes in L286F, +1AlaL286F and +2AlaL286F were undertaken, and light-induced time-resolved single turnover cyt c rereduction and cyt b reduction kinetics were recorded as previously (19) at 550–540 and 560–570 nm, respectively, in the presence of 5 \( \mu \)M antimycin A. First, both cyt c and cyt b kinetics were monitored at the potential \( E_m \) value of 100 mV where the quinone pool contains both Q and QH2, and the [2Fe-2S] cluster of the iron-sulfur subunit is oxidized, although subsequent reduction of cyt c heme by this cluster was seriously hampered because of its restricted motion.

**DISCUSSION**

**Location of the Suppressor Mutation of the Iron-Sulfur Subunit** (1Ala Insertion Mutant)—Available structural data for the mitochondrial bc1 complex show that the ef loop of cyt b sticks out of the membrane as a surface feature at the Qo site. Hence, it represents a conspicuous barrier on the trajectory of the iron-sulfur subunit that needs to be crossed during the movement of the [2Fe-2S] cluster domain from the Qo site stigmatically (8) and b positions2 to the cyt c1 position (8, 9). This loop appears to be highly flexible because it is located in a different position in comparison with the other structures of the bc1 complex (7, 8) when the iron-sulfur subunit cluster domain is in the intermediate position (9). Although this position of the ef loop may be artificial because of stabilizing crystal contacts, nonetheless it suggests that this loop could move.

Further, comparisons of various structurally defined positions of the iron-sulfur subunit indicate that its movement is composed of a translation and a rotation. Thus, a too long or too

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2 This structure has been solved by S. Iwata in absence of inhibitor, and the coordinates were provided as a privileged communication.
rigid hinge may lead the extrinsic domain of the iron-sulfur subunit to clash with the ef loop of cyt b. Indeed, as seen with the +1Ala and +2Ala mutants the longer the hinge is, the more blocked is the movement (19). The movement is also prevented when the hinge is too rigid as in the 6Pro mutant (i.e. when six amino acids of the hinge are substituted by six consecutive proline residues), yet it can readily occur when a rigid but shorter hinge is present (i.e. 3Pro/H9004 3 mutant) (20). Although second site suppressor mutations of the +1Ala revertant located on the ef loop of cyt b are not unexpected, substituting the Leu residue at position 286 (corresponding to 262 in bovine numbering) by a Phe with an even larger side chain may appear surprising at first. However, the side chain of Phe as an aromatic ring is planar unlike that of Leu, which is tetrahedral. Molecular modeling indicates that one of the most favorable rotamers of Phe at this position could place this side chain away from the iron-sulfur cluster domain to diminish any possible steric hindrance as illustrated in Fig. 4. Hopefully, resolution of the structure of R. capsulatus bc1 complex, currently in progress, will provide a better understanding of the impact of the L286F second site reversion on the interactions of the iron-sulfur subunit cluster domain with the flexible ef loop barrier.

### FIG. 2. cyt c rereduction kinetics in the presence of myxothiazol or stigmatellin in various mutants.

cyt c rereduction kinetics were triggered by flash activation of the photochemical reaction center and recorded at 550–540 nm using chromatophore membranes poised at 100 mV as described in Refs. 19 and 20. In each case, the traces obtained with no inhibitor (no), in the presence of myxothiazol (Myx) (no QH2 oxidation), or in the presence of stigmatellin (Stig) (no electron transfer to cyt c, heme) are shown. A–F correspond to the wild type strain and L286F, +1Ala, +1AlaL286F, +2Ala, and +2AlaL286F mutants, respectively.

### FIG. 3. cyt b reduction kinetics in various mutants.

cyt b reduction kinetics in the presence of 5 μM antimycin A were triggered by flash activation of the photochemical reaction center and recorded at 560–570 nm using chromatophore membranes poised at either 100 mV (A) or 400 mV (B), as described in Refs. 19 and 20. The traces obtained for the wild type strain and the L286F, +1Ala, +1AlaL286F, +2Ala, and +2AlaL286F mutant strains are shown in both panels. In B, similar traces obtained with Y147A mutant (26) are also shown.

Double Effect of the L286F Mutation—Interestingly, the L286F mutation alone affects slightly both the assembly of the iron-sulfur subunit to the bc1 complex and the Qo site catalysis. In particular, the slower cyt b reduction rates seen at 400 mV, an ambient redox potential at which cyt b kinetics are virtually independent of the movement of the iron-sulfur subunit (Table I), suggest that the L286F substitution in the +1Ala background improved mainly the difficulty of the iron-sulfur subunit to move rather than its ability to catalyze QH2 oxidation. However, this improvement is limited because in the case of the +2Ala mutant the L286F suppressor mutation has little, if any, effect because almost no electron transfer between the [2Fe-2S] cluster and cyt c1 can be observed with the +2AlaL286F double mutant (Fig. 2). Thus, if the hinge becomes too long the L286F substitution is not sufficient to allow the iron-sulfur subunit cluster domain to move across the ef loop barrier.

Dual Role of the ef loop of cyt b as a Shield for the Qo Site and as a Barrier for the Iron-Sulfur Subunit Motion—The effect of the L286F mutation is also seen on the [2Fe-2S] cluster E3 with a decrease in the wild type as well as the +1Ala and
+2Ala mutant backgrounds, suggesting that the environment of the cluster must have been changed. When the iron-sulfur subunit cluster domain is proteolytically cleaved off the bc1 complex into a soluble form or when the Qo site is occupied with myxothiazol that releases the iron-sulfur subunit cluster domain away from the stigmatellin position in the Qo site, the $E_m$ value of the [2Fe-2S] cluster decreases (11, 24). The lower $E_m$ values seen in the presence of the L286F mutation therefore strongly suggest that in these mutants the cluster must be in a more polar environment because of a more leaky shield provided by ef loop around the cluster. If indeed this is the case, then the L286F mutation must be overcoming the steric hindrance inflicted by a longer hinge at the expense of rendering the Qo site more solvent-accessible, in agreement with the slower QH$_2$ oxidation rates observed in the case of the +1AlaL286F mutant. Thus, although the L286F mutation per se is slightly harmful for Qo site catalysis, it improves the movement of the iron-sulfur subunit in the case of the +1Ala mutant, resulting in better growth of the +1AlaL286F double mutant. In the native enzyme, although this region acts as a barrier for the movement, it conceivably may hold the iron-sulfur subunit cluster domain in the Qo site in a position optimal for catalysis. The two opposing effects seen with the L286F mutation clearly reflect the dual role of the ef loop of cyt b providing a shield that is tight enough but not too much to not hinder the movement.\footnote{The conclusion that the ef loop is important both for the movement and for the Qo site catalysis is also supported by the analyses of additional mutations in the same region of cyt b, such as the Thr$^{268}$ substitutions and their revertants (E. Darrouzet and F. Daldal, manuscript in preparation).} Obviously, additional studies are needed to understand the crucial role played by the ef loop region and its interactions with the iron-sulfur subunit during the catalytic cycle of the bc1 complex. In particular, is this loop just a flexible domain pushed away by the iron-sulfur subunit during its motion, or does it play an active role in controlling the movement in response to specific redox events at the Qo site? In this respect, it is noteworthy that when one of the electrons provided by a QH$_2$ oxidation in the Qo site is blocked in the low potential cyt b chain by the presence of antimycin A in the ubiquinone reduction site, this electron cannot return back to the [2Fe-2S] cluster of the iron-sulfur subunit in a millisecond time scale, implying coordination of events occurring at the Qo and ubiquinone reduction sites. If this is the case, then could it be due to the ef loop prohibiting the iron-sulfur subunit to return back to the Qo site until the opportune time?

\textbf{Micro-movement and Macro-movement of the Iron-Sulfur Subunit Cluster Domain—Additional aspects of the bc1 complex mechanism of function, in particular the role of the different subdomains encountered at the Qo site, are also highlighted in this study. First, a comparison of the cyt b kinetics recorded at 400 mV with the cyt c$_1$ kinetics, especially using the +2Ala or +2AlaL286F mutant, clearly show that the rate of the cyt c$_1$ rereduction can be casually null, whereas cyt b reduction kinetics are quasi-wild type. Thus, the concerted electron bifurcation mechanism at the Qo site must indeed occur between the [2Fe-2S] cluster and cyt low potential b-type heme and not between the cyt c$_1$ and cyt low potential b-type heme, and careful analyses are needed when redox kinetics of these cofactors are considered, especially when the movement of the iron-sulfur subunit may be impaired. Second, the overall data indicate that the movement of the iron-sulfur subunit appears to have two discrete phases with a “micro-movement” at the interface of cyt b moving the cluster domain away from the stigmatellin subdomain of the Qo site and a “macro-movement” crossing the ef loop toward cyt c$_1$. In respect to the first step, data obtained using EPR spectroscopy (24) and proteolysis experiments performed in the presence of myxothiazol (21, 24) suggest that the micro-movement still occurs even when the macro-movement is prohibited and is enough to assure QH$_2$ oxidation. Indeed the +2Ala and +2AlaL286F mutants, which are blocked for the movement and hence cannot transfer electrons to cyt c$_1$, still catalyze QH$_2$ oxidation provided that the [2Fe-2S] cluster is initially oxidized. Moreover, the data also indicate that the macro-movement of the iron-sulfur subunit from the Qo site toward cyt c$_1$ is necessary for multiple turnovers of the bc1 complex. Finally, it is noteworthy that the displacement of the cluster domain, required for binding myxothiazol, is more difficult in mutants unable to perform the macro-movement. The +2Ala and 6Pro mutants, like the alanine mutants in yeast (16), exhibit increased resistance to myxothiazol because large excess of this inhibitor is required to
displace their iron-sulfur subunit out of the stigmatellin subdomain to obtain a pure \( g_x \) signal (24). Furthermore, cyt \( c \) re-reduction kinetics with the +1Ala mutant in the presence of myxothiazol are faster than with no inhibitor (19), and a similar phenomenon called “overshoot” has also been observed by Crofts and co-workers (27) with the \textit{Rhodobacter sphaeroides} native bc\(_1\) complex. These observations suggest that in the presence of Q the reduced iron-sulfur subunit needs to break some interactions to move away from the stigmatellin subdomain and that the energy required for it can be seen kinetically even with the native enzyme complex. Hopefully, future work will shed further light on these intriguing aspects of the bc\(_1\) complex mechanism of function.

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J. Biol. Chem. 2002, 277:3471-3476.
doi: 10.1074/jbc.M107974200 originally published online November 13, 2001

Access the most updated version of this article at doi: 10.1074/jbc.M107974200

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