Crystal Structure of Quinohemoprotein Amine Dehydrogenase from *Pseudomonas putida*

**IDENTIFICATION OF A NOVEL QUINONE COFACTOR ENCAGED BY MULTIPLE THIOETHER CROSS-BRIDGES**

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The crystal structure of a quinohemoprotein amine dehydrogenase from *Pseudomonas putida* has been determined at 1.9-Å resolution. The enzyme comprises three non-identical subunits: a four-domain α-subunit that harbors a di-heme cytochrome *c*, a seven-bladed β-propeller β-subunit that provides part of the active site, and a small γ-subunit that contains a novel cross-linked, proteinous quinone cofactor, cysteine tryptophylquinone. More surprisingly, the catalytic γ-subunit contains three additional chemical cross-links that encage the cysteine tryptophylquinone cofactor, involving a cysteine side chain bridged to either an Asp or Glu residue all in a hitherto unknown thioether bonding with a methylene carbon atom of acidic amino acid side chains. Thus, the structure of the 79-residue γ-subunit is quite unusual, containing four internal cross-links in such a short polypeptide chain that would otherwise be difficult to fold into a globular structure.

Recently, a number of modified amino acids have been identified in proteins that are generated by post-translational oxidation or non-oxidation processes (1, 2). Such a controlled modification of a specific amino acid residue forming part of the active site provides catalytic power to the protein. In the case of a certain class of amine-oxidizing enzymes, depending on the enzyme concerned, oxidation of a specific tyrosine or tryptophan residue leads to the generation of a redox-active quinone cofactor: 2,4,5-trihydroxyphenylalanine quinone (topaquinone) (3), lysine tyrosylquinone (4), or tryptophan tryptophylquinone (TTQ)1 (5). Together with several enzymes containing the first identified, non-proteinous quinone cofactor, pyrroloquinoline quinone (PQQ), the enzymes containing those cofactors constitute a quinoprotein family of enzymes (6).

Quinohemoprotein amine dehydrogenases (QH-AmDH) from Gram-negative bacteria represent a new type in the quinoprotein class of amine-oxidizing enzymes because they contain not only a quinone but also one or two hemes as a redox active group (7, 8) providing an opportunity for intramolecular electron transfer. Intermolecular electron transfer from QH-AmDH has been demonstrated with the natural electron acceptors azurin for the enzyme from *Pseudomonas putida* (7) and cytochrome c-550 for the enzyme from *Paracoccus denitrificans* (9). The structure of the presumed quinone cofactor in QH-AmDH remain to be settled, although biochemical and spectroscopic analyses have suggested the presence of a quinone group similar to, but not identical, with TTQ (7, 8).

To identify the quinone cofactor and its position in the protein, we (10) have recently determined the primary structure of the quinone-containing small subunit of QH-AmDH from *P. putida* using a combination of automated Edman degradation and mass spectrometry, and we have also cloned the genes coding for the three subunits of the heterotrimeric enzyme. Although we initially encountered difficulties in the interpretation of the chemical and mass data, the progress in elucidating the crystal structure of the enzyme enabled us to unequivocally identify a novel quinone cofactor, cysteine tryptophylquinone (CTQ) (naming is analogous with that for TTQ). Besides this novel “in situ-generated” quinone cofactor, the covalent structure reported in the preceding article (10) and the 1.9-Å crystal structure reported in the present article have also revealed a unique feature of the catalytic γ-subunit in which CTQ is encaged by three intra-chain cross-links. These links are unprecedented as they are thioether bonds between a cysteine sulfur atom and a methylene carbon atom of an aspartic or a glutamic acid residue.

**EXPERIMENTAL PROCEDURES**

Crystallization and Data Collection—QH-AmDH of *P. putida* was purified as described (7) and crystallized at 20 °C using the hanging-

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1 The abbreviations used are: TTQ, tryptophan tryptophylquinone; CTQ, cysteine tryptophylquinone; PQQ, pyrroloquinoline quinone; QH-AmDH, quinohemoprotein amine dehydrogenase; pNPH, p-nitrophenylhydrazine.
The crystal structure of the native QH-AmDH was determined at 1.9-Å resolution (Tables I and II), utilizing the DNA-based protein sequence (10). Consistent with the previous biochemical analysis (7), the enzyme is composed of three non-identical subunits: a 494-residue α-subunit (60 kDa) carrying two heme c groups, a 349-residue β-subunit (40 kDa), and a 79-residue γ-subunit (9 kDa) bearing the quinone cofactor. The γ-subunit is embedded in the crevice of the α-subunit with an extensive intersubunit interface with a contacting surface area of 4,640 Å², making the αγ pair look like a single subunit (Fig. 1A). When viewed in cross-section, the convex “bottom” of the heterodimer lies on the concave “top” surface of the β-subunit, with a contacting surface area of 4,913 Å² to build up the overall heterotrimetric structure. As a result, only 34% of the surface area of the γ-subunit is exposed to solvent.

The α-subunit comprises four distinct domains (I–IV). Domain I has a predominant helical structure and can be subdivided into two subdomains, each containing a heme c group covalently attached to a Cys residue through a thioether linkage, as in class I cytochromes c (21) (Fig. 1A). The overall structure of Domain I is similar to that of a di-heme cytochrome c (22–24), in which the two cytochrome c-like domains are related by a pseudo 2-fold axis, suggesting that the α-subunit harbors a di-heme cytochrome c forming an intramolecular electron transfer system. One of the two heme c groups (heme I) is encapsulated within the protein interior, facing the interface with the γ-subunit, whereas the other heme c group (heme II) is exposed to the solvent (Fig. 1A). The two heme planes are tilted by about 51° with each other and separated in an Fe–Fe distance of 15.8 Å. The two iron atoms of the heme groups have thermal factors after refinement were above 58 Å² (corresponding to 58 Å² (corresponding to the maximum thermal factor of the main chain atoms). Further model building and refinement cycles gave an Rfactor value of 21.1% and an Rfree value of 24.5% calculated for 87,307 reflections (Fobs > 2σ(Fobs)) observed in a 10.0–1.9-Å resolution range (see Table II). During the last step of the refinement, unambiguous water molecules were added including those with a temperature factor higher than 58 Å². The maximum temperature factor of the water molecules was 78 Å².

The same refinement procedure was applied to the pNPH-complexed enzyme but using the coordinates of the native QH-AmDH as an initial model. When the Rfactor value reached below 25%, the difference Fourier map clearly exhibited the residual electron density corresponding to the bound pNPH. Further model building and refinement cycles gave an Rfactor and an Rfree value of 21.1% and 25.0%, respectively, calculated for 74,658 reflections (Fobs > 2σ(Fobs)) observed in a 10.0–2.0-Å resolution range (see Table II). The maximum temperature factor of the assigned water molecules was 75 Å².

**Quality of the Structure**—The final models of the native enzyme and the pNPH complex both contained 909 amino acid residues and two heme c groups with 457 water molecules for the native enzyme and 359 water molecules plus one pNPH molecule (as the hydrase) for the pNPH complex. No interpretable electron density was observed for one N-terminal residue of the α-subunit, three N-terminal residues and residues 220–226 of the β-subunit, and two N-terminal residues of the γ-subunit. Both models had a good quality with 98.8% of residues falling in the most favorable and additionally allowed region and only 0.3% in the disallowed region, when the stereochemistry was assessed by PROCHECK (17) (see Table II). Structure diagrams were drawn with programs MOLSCRIPT (18), Raster3D (19), and BOBSCRIPT (20).

**RESULTS AND DISCUSSION**

### Overall Structure

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### Table I

| Data collection | Native | pNPH | Hg I | Hg II | Hg III | Hg IV | Pt |
|-----------------|--------|------|------|-------|--------|-------|----|
| Wavelength (Å)  | 1.00   | 1.00 | 1.00 | 1.00  | 1.00   | 1.00  |    |
| Resolution (Å)  | 1.9    | 2.0  | 2.2  | 2.6   | 3.1    | 2.2   | 2.2|
| Unique          | 89255  | 75744| 56513| 34618 | 19497  | 56425 | 56405|
| Completeness (%)| 99.9 (100.0) | 99.7 (99.7) | 99.0 | 99.0 | 96.1 | 99.3 | 98.8 |
| Rmerge (%)      | 11.0 (2.8) | 8.2 (2.7) | 11.3 | 11.6 | 6.2 | 12.2 | 13.0 |
| Rfree (%)       | 5.1 (26.0) | 6.7 (25.9) | 4.3 | 9.5 | 10.3 | 4.5 | 4.4 |
| Phasing power   | 10.1 | 18.0 | 12.1 | 9.6 | 10.6 |
| No. of sites    | 2 | 4 | 1 | 1 | 1 | 1 |
| Phasing power   | 0.55 | 0.96 | 0.38 | 0.31 | 0.64 |
| Figure of merit | 0.20 | 0.25 | 0.10 | 0.12 | 0.27 |

* a Heavy atoms derivative: Hg I and Hg II, Baker’s Dimercuric; Hg III, K2[HgI]3; Hg IV, ethylmercurichlorosalicilic acid; Pt, PtCl2(NH2CH2CH2NH2).

b The enzyme complexed with pNPH.

c Values in parentheses are for the highest resolution cells.

d Rmerge = \[ \sum_{hkl} \left| F_{hkl} - \langle F_{hkl} \rangle \right| / \sum_{hkl} F_{hkl} \], where \( F_{hkl} \) is observed intensity and \( \langle F_{hkl} \rangle \) is average intensity for multiple measurements.

e Rfree = \[ \sum_{hkl,i} \left| F_{hkl,i} - \langle F_{hkl,i} \rangle \right| / \sum_{hkl,i} F_{hkl,i} \], where \( F_{hkl,i} \) and \( \langle F_{hkl,i} \rangle \) are the derivative and native structure-factor amplitudes, respectively.

f Phasing power is the ratio of the root-mean-square (rms) of the heavy atom scattering amplitude and the lack of closure error.
His-18α and Met-46α in heme II, suggesting that they have different redox potentials.

Domains II–IV of the α-subunit consist mainly of β-structures with two short α-helices located on the surface side of domain IV. Domain II has a typical β-barrel structure with eight up-and-down antiparallel β-strands. Domain III is a pseudo barrel formed by three and four antiparallel β-strands. Domain IV is also a pseudo barrel of seven β-strands with an additional β-sheet of three strands on the N-terminal side and the C-terminal loop interacting with the γ-subunit. It is noteworthy that the barrel folds of domains III and IV resemble those of the first and third domains, respectively, of the monomeric galactose oxidase, which contains another type of posttranslationally generated redox cofactor, the Cys-Tyr adduct, in which Cys and Tyr are covalently coupled to each other by a thioether bond between the sulfur atom and the phenyl ring (25, 26).

The β-subunit is folded into seven motifs of four up-and-down antiparallel β-strands, which are arranged around a pseudo seven-fold axis, giving a seven-bladed propeller. The N- and C-terminal β-strands of each blade are located on the inner and outer side, respectively, of the β-propeller, and the C-terminal strand is connected to the N-terminal strand of the neighboring blade. The β-propeller fold has also been observed in galactose oxidase (25) and in quinoproteins such as TTQ-containing methylamine dehydrogenase (27) and PQQ-dependent glucose (28), methanol (29) and ethanol (30) dehydrogenases. A structure similarity search (31) showed that the large β-propeller subunit of methylamine dehydrogenase is quite similar to the β-subunit of QH-AmDH.

**Unique Structure of Catalytic Subunit**—The most surprising finding revealed by this work concerns the structure of the small γ-subunit. As deduced from the DNA sequence (10), this 79-residue subunit contains a total of four Cys and five Trp residues. However, neither free SH groups nor S-S bridges were detected in it by chemical analysis (10). Furthermore, during the initial stage of the structure determination by x-ray crystallography, modeling of the γ-subunit puzzled us with its main chain branching off at many points in the electron density map. Complete modeling of the γ-subunit became possible only

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**TABLE II**

**Refinement and model statistics**

|                          | Native      | pNPH       |
|--------------------------|-------------|------------|
| Resolution limit         | 10.0–1.9 Å  | 10.0–2.0 Å |
| Rfactor (%)              | 21.1 (23.7) | 21.1 (27.9) |
| Rfree (%)                | 24.5 (29.6) | 25.0 (29.3) |
| Mean B factors           |             |            |
| Main chain atoms (Å²)    | 18.7        | 19.8       |
| Side chain atoms (Å²)    | 19.5        | 21.0       |
| Water atoms (Å²)         | 30.6        | 30.4       |
| Deviations               |             |            |
| Bond length (Å)          | 0.009       | 0.009      |
| Bond angles (deg)        | 1.61        | 1.63       |
| Ramachandran plot        |             |            |
| Favored (%)              | 87.2        | 85.5       |
| Allowed (%)              | 11.6        | 13.3       |
| Generously allowed (%)   | 0.9         | 0.9        |
| Disallowed (%)           | 0.3         | 0.3        |

*Values in parentheses are for the highest resolution cells.

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**FIG. 1. Ribbon structural drawings of QH-AmDH from P. putida.** A, stereo diagram of the overall structure of the heterotrimer. The α-subunit (blue) consists of one α-helical domain (domain I) and three β-barrel domains (domains II–IV). Two heme c groups (green) are bound to domain I through a covalent bond. The seven-bladed β-propeller β-subunit (yellow) is viewed perpendicularly to the pseudo seven-fold axis. The γ-subunit (red) is placed in the space formed between α- and β-subunits. B, stereo diagram of the γ-subunit, shown with the two heme c groups attached in domain I of the α-subunit. CTQ and residues involved in internal cross-bridges (red) are shown in ball-and-stick representation.
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after the $2F_o - F_c$ electron density map was calculated at 1.9-Å resolution using the phases improved on the basis of the models of the α- and β-subunits. The quality of the omit electron density map reached the point at which the side chains were identified from the shape of the electron densities contoured at the 1-σ level. In this stage, the side chains of all the Cys residues in the γ-subunit were found to have unusually close contacts with a Trp, Asp, or Glu residue. For example, the Sγ atom of Cys-37γ was within the covalent bond distance with the indolyl C4 atom of Trp-43γ. Also, extra electron densities were found to protrude from the C6 and C7 positions of the indolyl group of Trp-43γ, which were assigned to oxygen atoms based on the previous finding that the γ-subunit contains a quinone group similar to, but not identical with, TTO (7). Consequently, the crystal structure has revealed that one (Cys-37γ) of the four Cys residues is covalently cross-linked to the side chain of Trp-43γ (at C4 of the indole ring), which is modified to an ortho-quinone, resulting in the novel quinone cofactor CTQ (Figs. 1B and 2A). Furthermore, the other three Cys residues are involved in thioether cross-bridges with an Asp or Glu residue, all in a hitherto unknown bonding with a methylene carbon atom of acidic amino acid side chains: Cys-7γ–Glu-16γ (Cγ) (Fig. 2B), Cys-27γ–Asp-33γ (Cγ) (Fig. 2A), and Cys-41γ–Asp-49γ (Cγ) (Fig. 2C). Crystallographic identification of these cross-linked structures greatly enabled us to interpret the results obtained by the chemical and mass spectrometric analyses of the γ-subunit, as reported recently (10). Besides the novel CTQ cofactor encaged by multiple internal cross-bridges, another intriguing feature is that the γ-subunit scarcely contains regular secondary structures as it has only two short α-helices with many turns and bends (Fig. 1B). A schematically drawn structure of the γ-subunit (Fig. 2D) reminds us of peptide antibiotics with an internal loop rather than of a protein. Indeed, in a structure similarity search using the DALI calculation (31), no structure with Z scores of higher than 2.0 was found, indicating that the polypeptide fold of the γ-subunit is unique. It thus appears that the multiple cross-links play at least a structural role in maintaining the globular structure of the small γ-subunit polypeptide.

The γ-subunit occupies the space formed between the interface of the α- and β-subunits with 11 N-terminal residues (Ala-3γ, Cys-7γ–Thr-11γ, Val-17γ–Gly-21γ) and 29 C-terminal residues (Met-51γ–Lys-75γ) exposed to the solvent on both sides of the molecule. The three cross-bridges of Cys to Asp or Glu contained in 48 N-terminal residues (Ala-3γ–Met-50γ), together with numerous internal hydroxide bonds, lead to a compact γ-subunit and an encaged CTQ. As compared with the C-terminal part, this region is rich in hydrophobic and acidic residues. CTQ resides in the vicinity of the interface between the β- and γ-subunits and near the pseudo seven-fold axis of the β-subunit, directing its C6 carbonyl group toward the interface. The topology of CTQ, heme I, and heme II (Fig. 1B) suggests that intramolecular electron transfer occurs from sub-
strate-reduced CTQ to heme II via heme I followed by intermolecular electron transfer from heme II to azurin (7). The fact that the quinone ring of CTQ is separated by about 7.8 Å from the edge of heme I with a dihedral angle of 19° with respect to each plane supports this view.

**Active Site Cavity and Presumed Catalytic Base**—On close inspection of the γ-subunit structure, the active site cavity seems to be located at the interface between the β- and γ-subunits, surrounded by CTQ 43γ, the side chains of Pro-13γ, Asp-33γ, Pro-40γ, and Trp-42γ from the γ-subunit, and the side chains of Leu-198β, Phe-200β, Thr-201β, Tyr-201β, Phe-258β, Tyr-298β, and Thr-341β from the β-subunit (Fig. 3A). Inside the cavity, two water molecules (W1 and W2) are found. A hydrogen-bonding network exists between the C6 carbonyl group of CTQ, the carboxylate group of Asp-33γ, W1, and W2, and the hydroxyl group of Tyr-298β. The N1 nitrogen and C7 carbonyl oxygen of CTQ are hydrogen-bonded to the main chain carbonyl oxygen of Thr-10γ and amide nitrogen atoms of Asp-12γ and Gly-14γ, respectively. To determine the catalytic site of CTQ, where the substrate amine reacts (either the C6 or the C7 carbonyl), forming a Schiff base intermediate similar to the proposed reaction mechanism of TTQ-dependent methylamine dehydrogenase (32). Because Asp-33γ is the sole residue close enough to the C6 carbonyl oxygen of CTQ and the Schiff base imine nitrogen, it could act as a catalytic base abstracting the α-proton from the CTQ substrate Schiff base. The hydrophobic environment around Asp-33γ could be helpful in raising the pKₐ of the CTQ substrate Schiff base, thereby enhancing its catalytic function. It should also be noted that the conformational flexibility of the carboxylate group of Asp-33γ is largely restricted by the cross-link from Cys-27γ to this residue.

**Implication for Cofactor and Cofactor Subunit Biogenesis**—The unique active site structure of QH-AmDH raises a number of questions: Why, when, where, and how are the CTQ cofactor and the multiple internal cross-links in the γ-subunit generated? In the absence of further information, these questions cannot be answered yet. However, sequencing of the gene cluster encoding QH-AmDH of *P. putida* has already provided a clue for how the biogenesis could take place (10). In the stretch containing the structural genes for the three subunits, a hypothetical 53-kDa protein was found whose sequence is homologous to that of “sulfatase-activating enzyme,” containing an iron/sulfur cluster and participating in the oxidative formation of the formylglycine cofactor in the active site of sulfatase (33). By analogy, the hypothetical 53-kDa protein could play a role in the post-translational formation of CTQ and/or the thiocysteine bonds in the proenzyme of QH-AmDH.

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