

Qrc reduces menaquinone with electrons from periplasmic hydrogen or formate oxidation, and is essential for growth in both substrates. The *qrc* genes are present in deltaproteobacterial sulfate reducers that have periplasmic hydrogenases and/or formate dehydrogenases that lack a membrane subunit for direct quinone reduction, and Qrc forms a supercomplex with the [NiFe] hydrogenase and Tplc3 in the membranes of *D. vulgaris*. Thus, Qrc links the periplasmic cytochromes *c* to the membrane menaquinone pool. Qrc is a striking example of how a different physiological function can be achieved with a minimal modification of subunits, a strategy that forms the basis for the diversity and flexibility of bacterial energy metabolism.

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Inhibitor titration of the cytochrome *bc*₁ complex of *Rhodobacter capsulatus* by myxothiazol and pyraclostrobin: Evidence for a binding change mechanism

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The cytochrome *bc*₁-complexes (hereafter *bc*₁), are proton-translocating quinol: cytochrome *c* oxidoreductases [1]. According to the Mitchell's Q-cycle mechanism [2], after the oxidation of each ubiquinol molecule in the catalytic center *P*, one electron is transferred to the [Fe₂S₂] cluster-carrying domain of the Rieske protein (hereafter the FeS domain), whereas the other electron is transferred across the membrane, via the two hemes of cytochrome *b*, into the other catalytic center *N*, where additional ubiquinol molecules could be formed. After it was found that the *bc*₁ is an intertwined dimer [3], a dimeric Q-cycle, where the two monomers could exchange electrons and were allosterically coupled, was suggested to explain the kinetic data [1, 4].

Myxothiazol is a specific center *P* inhibitor that does not block the mobility of the FeS domain and helps to separate the reactions which follow the oxidation of ubiquinol in center *P* from the preceding steps of the catalytic cycle. Here we show that pyraclostrobin, a synthetic analog of myxothiazol, behaved as myxothiazol when tested as an inhibitor of *bc*₁ with membrane vesicles (chromatophores) from *Rhodobacter capsulatus* and did not affect the electrical properties of the membrane. Furthermore, we have found that sub-saturating amounts of pyraclostrobin and myxothiazol retarded not the reduction, but the oxidation of cytochrome *b* in response to a flash of light under slightly alkaline conditions (pH about 8.5). We attribute this kinetic behavior to the enzyme complexes which contained only one molecule of inhibitor per *bc*₁ dimer before the flash. We interpret our results as an evidence for the binding change mechanism in the *bc*₁, in support of our earlier suggestions in [4].

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12P12

Two types of quinol: Periplasmic e-transporter oxidoreductase. Comparison of their operons variations and distribution through bacterial phyla

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During the progress of life different proteins emerged at different times. It seems possible to find out the point of the emergence of a protein and its subsequent evolution comparing its dendrogram with total phylogeny based on the dendrogram for universal molecular clocks. Usually 16s-rRNA is used as such a clock. Here, concatenated amino acid sequences of ten universal proteins not prone to Lateral Gene Transfer are used to construct a tree for a representative set of prokaryotes. The tree contains the same bacterial and archaeal phyla as on the 16s-rRNA tree, but it defines a short period with irresolvable branching order instead of the unreliable order in a one-clock-tree. It looks like a burst of branching.

Main bacterial phyla (Proteobacteria, Actinobacteria, Bacilli, Cyanobacteria) and some minor ones contain *bc*₁-complexes. The structure of the clusters on the dendrogram for these complexes is congruent to the structure of the phyla on the total phylogenetic tree. Operons encoding the complexes have the simple structure with little variations. But there are some mixed clusters that comprises the complexes from several bacterial phyla. The hosts of such complexes belong to the members of some orders of d-Proteobacteria and some sparsely occupied phyla (Acidobacteria, Planctomycetes, and Verrucomicrobia). The *bc*₁-operons of the members of these clusters are rather various and may possibly contain additional genes.

There are alternative complexes with the same function as the *bc*₁ one but homologous to three-subunit molybdopterin-containing complexes, one of which can be considered as a precursor. In the genomes of several representatives of δ-Proteobacteria there are operons that gradually acquire genes encoding additional subunits: five-heme cytochrome *c*, one-heme cytochrome *c*, duplicate of the membrane subunit, and one more membrane subunit. Each intervening type of these operons forms a subcluster within the δ-Proteobacteria-cluster on the dendrogram for the complex. Operons containing all indicated genes can be found in the genomes of such bacterial phyla as Flavobacteria, Sphingobacteria, Acidobacteria, Chloroflexi, Planctomycetes, Verrucomicrobia and others. Almost in all cases each cluster on the dendrogram for the complex corresponds to a phylum or a branch on the total tree.

The points of the emergence of the two types of oxidoreductases and their modes of evolution and inheritance – vertical and horizontal – are discussed.

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