Verméglio and Joliot (1) have emphasized their view that the components of the *Rhodobacter sphaeroides* chain are organized as supercomplexes. This model was developed to explain the low equilibrium constant between cytochrome (cyt) c₂ and oxidized reaction center (RC) observed in kinetic experiments (2). We have suggested an alternative view, that electron transfer requires diffusion of the ubiquinone and cyt c₂ couples between separate complexes (3-6). We were able to explain their original observations in terms of effects arising from the statistical distribution of components between small chromatophore vesicles, which would produce the same apparent change in equilibrium constant as they observed, without the need for supercomplexes (5).

It is an essential part of the supercomplex hypothesis that, on a time scale long compared with the ~1 ms turnover, cyt c₂ is not able to diffuse from the supercomplex (2). We showed that in chromatophores, contrary to this expectation, QH₂ or cyt c₂ can rapidly visit ~9 bc₁ complexes (approximately the number expected in a chromatophore vesicle) (4). We also demonstrated that in mutant strains in which the components are expressed in stoichiometric ratios that differ from those needed for the supercomplex, all bc₁ complex, cyt c₂, and reaction center (RC) that interact rapidly following excitation by a short (5 µs) actinic flash, showed the behavior expected from the diffusional model (5, 6, and Hong, S.J. and Crofts, A.R., unpublished).

Verméglio and Joliot (1) discuss the structures of the ordered arrays of C-shaped particles observed in tubular membranes in *Rb. sphaeroides* strains deficient in light-harvesting complex (LH) 2 (7), and claim that these support the supercomplex hypothesis. The structures likely represent the dimeric LH1-RC complex of Francia et al. (8), but in that work, no bc₁ complex was found in the dimer fraction. Although Jungas et al. (7) reported the presence of bc₁ complex in their tubular membranes at the stoichiometry expected, they showed no structural evidence for association between bc₁ and LH1-RC complexes. Verméglio and Joliot (1) suggest that “the electron density localized outside these structures could be attributed to the bc₁ complex”, or “(a)ternatively, the bc₁ complex could be localized between the two C-shaped structures”. However, in the Jungas et al. analysis (7), the electron density was almost completely accounted for by RC and LH1, and any unaccounted density would have to include the 1 PufX per RC found by Francia et al. (8). The mitochondrial bc₁ complex is dimeric, and requires dimeric association to bind the iron sulfur subunit (cf. 9); the complex from *Rb. sphaeroides* also appears to be dimeric (10). Such a dimeric complex would have about the same volume as that of an LH1-RC monomer, but there is no room for such a volume in the array, either within or outside the LH1-RC dimer. Indeed, there is no room for even a bc₁ complex monomer between the C-shaped structures. No immediate external location seems likely, since the bc₁ complex (1 per LH1-RC dimer) would impose an asymmetry in the array (whether dimer or monomer), but none is apparent. Furthermore, any external location would require a substantial diffusional pathway between binding sites on RC and bc₁ complex, because of the thickness of the LH1 ring. This would be difficult to reconcile with the tight binding required of the supercomplex mechanism (2).

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References