A Spatial Model of the Chromatophore Vesicles of *Rhodobacter sphaeroides* and the Position of the Cytochrome bc_1 Complex

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ABSTRACT The photosynthetic apparatus of purple bacteria is generally considered a well-studied and understood system. However, recent atomic force microscopy images of flattened chromatophore vesicles from *Rhodobacter sphaeroides* restarted a debate about the stoichiometry and positions of the membrane proteins, with the interpretations of the observed images only partly being in agreement with earlier models. The most puzzling observation from the recent images is that the Cytochrome bc_1 complex, which is a central part of the photosynthetic apparatus, seems to be missing on the chromatophore vesicles, even when these were extracted from photosynthetically grown bacteria. From the available information on the geometry of the vesicle and of the proteins we reconstructed here a three-dimensional model vesicle at molecular resolution. Its central feature, also determining its diameter of ~45 nm, is an equatorial array of LH1 dimers, lined by a region of LH2 rings. This naturally puts the Cytochrome bc_1 complexes and the ATPase at the vesicle's poles. This spatial model may explain why the vesicle's endcaps with the bc_1 complexes are lost during the preparatory steps of the imaging process together with the ATPase and are therefore absent from the available images.

INTRODUCTION

Purple bacteria like *Rhodobacter* (*Rb.*) *sphaeroides* can grow photosynthetically. Being an evolutionarily old species, their photosynthetic apparatus is built comparatively simple. Thanks to a relatively easy handling of the bacteria and due to decades of intense experimental work, it can now be considered a well and extensively studied system. Even the three-dimensional structures of the proteins of the photosynthetic apparatus of the purple bacteria have been resolved by x-ray crystallography down to atomic resolution and the last uncertainties of their internal mechanistic details are about to be solved.

In addition to its relative simplicity and the large body of experimental observations, there is one more important feature of the photosynthetic apparatus of *Rb. sphaeroides*, which makes it a very attractive model system for theoretical studies: it is located on so-called chromatophore vesicles, small lipid vesicles differentiated to host only the photosynthetic apparatus. These vesicles are closed units, separated from their environment. When aiming at developing systems biological techniques or molecular simulation techniques of complete functional subunits of a cell, each of these vesicles can be treated as a naturally isolated unit—which is of computationally manageable size and complexity at a molecular resolution.

These small closed vesicles are only found in purple bacteria that also express an additional protein, PufX, like *Rb. sphaeroides* or *Rb. capsulatus*. In the other species lacking PufX, and also in PufX$^-$ mutants of *Rb. sphaeroides*, the membranes with the photosynthetic apparatus form, e.g., tubular or lamellar structures (1,2).

Recently, images of ruptured chromatophore vesicles from atomic force and cryo-electron microscopy (AFM and EM) have significantly augmented our knowledge about the vesicle structure by revealing the relative spatial arrangement of the membrane-bound proteins. If these data were complete, a three-dimensional model vesicle could be built thus helping to end an old debate about whether supercomplexes of the proteins exist or not. The spatial arrangement of the transmembrane proteins on the vesicle is, however, still debated, especially the relative frequency and position of the Cytochrome bc_1 complexes. This is because these AFM and EM studies only revealed the native arrangement of the light-harvesting complexes and the reaction centers but failed to show the bc_1 complex (1–6). Contrary, older biological observations found one bc_1 complex for every two reaction centers (7,8), a ratio at which the bc_1 complex ought to show up with only little uncertainty.

As mentioned above, these chromatophore vesicles are an ideal model system for molecular simulations which prompted us to collect the available information on the chromatophore vesicles of *Rb. sphaeroides*. The findings from stoichiometries, throughputs, and kinetic rates allowed us to build up a kinetic process-view model of the vesicle, which is presented in the accompanying article (9). Here, we show that the information from the recent microscopy images together with findings on the mysterious PufX protein plus some kinetic considerations may be used to reconstruct a three-dimensional spatial model of a chromatophore vesicle. This model predicts the positions of all transmembrane proteins on the vesicle, including the Cytochrome bc_1

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complex, and allows us to argue why the bc_1 does not show up on the microscopy images.

This article is organized as follows: the following section reviews the photosynthetic apparatus of purple bacteria with an emphasis on the spatial organization of the chromatophore vesicles of *Rb. sphaeroides*. The next section, PufX and the Size of the Chromatophore Vesicles, collects and explains the findings why PufX should be responsible for the correct formation of the vesicles. Then the constraints on the possible positions of the bc_1s are laid out in Constraints on the bc_1 Position. They come from the diffusion of the electron carriers inside and of the protons outside of the vesicle. The section Proposing a Model Vesicle then presents the three-dimensional model of a chromatophore vesicle from *Rb. sphaeroides* reconstructed from the arguments given in this article. This reconstruction process is finally summarized and put back into the context of molecular simulations in Summary and Conclusions.

SPATIAL VIEW OF BACTERIAL PHOTOSYNTHESIS

The following short overview over the photosynthetic apparatus of *Rb. sphaeroides* relates to Fig. 1, which depicts the geometric and functional relations of the vesicle and the involved proteins. For more details we refer the reader to the reviews of, e.g., Schulten and co-workers (10,11). In purple bacteria, light is absorbed by the light-harvesting complexes

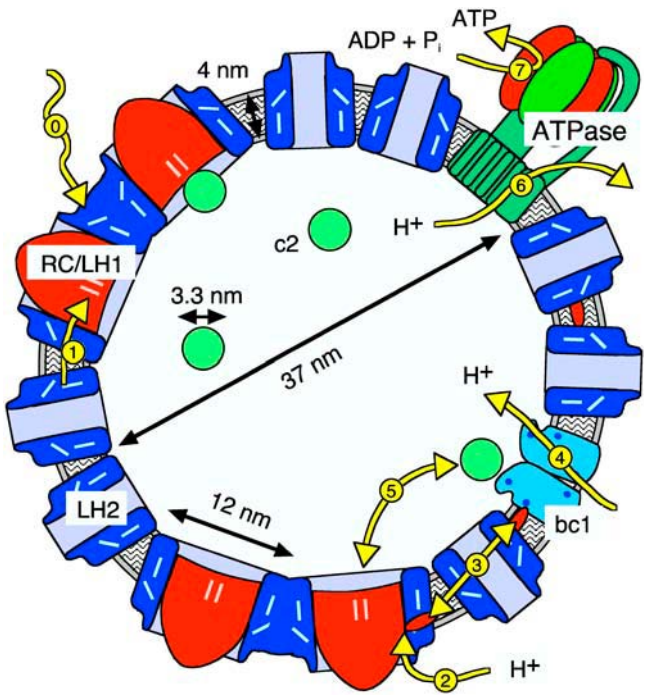

FIGURE 1 Cross section drawn to scale through a chromatophore vesicle from *Rb. sphaeroides* with a typical outer diameter of 45 nm. For a description of the processes (*yellow arrows*) and abbreviations, see text. The light-blue disks illustrate the positions of bacteriochlorophylls.

of types 1 and 2 (LH1 and LH2, *process 0* in Fig. 1) (12,13), is then funneled to the reaction center (RC), and converted there into an electron proton pair (*processes 1* and *2*) (14,15). Two of these pairs are then transported by one of the membrane-bound quinone molecules to the Cytochrome bc_1 complex (bc_1, *process 3*). There, the two protons are released to the interior of the vesicle, while the energy of the electrons is used to pump another two protons into the vesicle (*process 4*) (16,17). The electrons are returned to the RC by the water-soluble protein Cytochrome c_2 (c_2, *process 5*), which is confined to the interior of the vesicle. The proton gradient across the vesicle membrane is finally used by the F_0-F_1-ATP synthase (ATPase) to synthesize ATP (*processes 6* and *7*) (18). Further kinetic details of this conversion process of light energy into its chemical equivalent, stored in ATP, are laid out in the accompanying article on the process-view of bacterial photosynthesis (9).

For the geometric considerations of this article, a spatial view of the vesicle and of the involved proteins is more appropriate than this process-view. The photosynthetic chain of *Rb. sphaeroides* is located on small lipid vesicles with an average diameter of 45 $\pm$ 15 nm (19). The largest objects on these vesicles are the light-harvesting complexes of type 1. In most purple bacteria these antennas of the reaction centers form closed rings, each encircling an RC. In *Rb. sphaeroides* and *Rb. capsulatus*, however, an additional protein, called PufX, induces a dimerization of the LH1s (20). This small protein, which is expressed together with the subunits of the RC, has a single transmembrane helix and is similar to the α-subunit of the LH1. These LH1 dimers, which are well resolved in the recent AFM and EM images (1–6), cover an area of $\sim 20 \times 13$ nm^2, i.e., their length equals the radius of the chromatophore vesicle. For every LH1 dimer, the vesicle carries approximately six of the smaller auxiliary light-harvesting complexes of type 2, as can be seen on the AFM images of reference (3). Their closed rings have a diameter of $\sim$7 nm. Each vesicle contains on average a single ATPase (19), which synthesizes ATP by utilizing the proton gradient between the interior and the outside of the vesicle. The ATPase is built from two subunits. The transmembrane F_0 part occupies roughly the same membrane area as an LH2 ring, while the F_1 part sits above the membrane like a huge mushroomlike structure (19,21). One can therefore expect that the ATPase can be torn from the membrane relatively easily. It is unclear how the Cytochrome bc_1 complex would look like in an AFM image. Its crystal structure was determined recently (17) and it is known that the bc_1 always occurs as a homodimer (22). Although such a dimer occupies roughly the same membrane area as an LH2 ring, it should be much harder to visualize by AFM techniques than those, as the x-ray structure shows that it is essentially level with the vesicle membrane on the cytosolic side.

Little was known for sure until recently about the relative arrangement of the transmembrane proteins. The most controversial subject still is whether the RCs and the LH1 would or would not form supercomplexes with the bc_1s (see,

e.g., (8) versus (23)). The debate about the spatial composition of the photosynthetic apparatus was restarted by Jungas et al. (1) in 1999, as their EM-reconstructed pictures of tubular membranes from an LH2-lacking mutant of *Rb. sphaeroides* showed periodic arrays of S-shaped structures. Theses were interpreted as supercomplexes of two RC/LH1 units and one bc_1 between them. Their positioning of the bc_1 complexes had to be modified, though, due to the results of subsequent imaging experiments (2,3,24). Most troubling, the new experiments did not show the bc_1s or the ATPase at all. While the ATPase had been seen in earlier AFM images (21), the missing bc_1 complex led to considerable confusion, as the observed membrane patches seem to be unable to perform photosynthesis due to the lack of a key component.

Is this proton-pump hiding amid the partly scattered and partly organized light-harvesting complexes (LHCs) seen in the AFM images (3)? Before addressing this question, we present some arguments that both the existence of the vesicles and their specific size are a consequence of the PufX-induced dimerization of the LH1s.

PufX AND THE SIZE OF THE CHROMATOPHORE VESICLES

The recent AFM experiments, which triggered this quest for the bc_1 complex, were performed on manipulated chromatophore vesicles. For the AFM imaging, these spherical vesicles have to be ruptured and then fixated to a flat surface. This resulted in images similar to these of Bahatyrova et al. (3) or Hu et al. (11): small, flat torn-out patches of lipid membranes densely covered with LHCs and RCs.

Fig. 1 highlights how small the spherical chromatophore vesicles are compared to the LHCs located on them. On a vesicle with an average diameter of 45 nm, the LH1 dimers, which have a length comparable to the vesicle radius, consequently have to be bent by an angle of ~26° at the joint where the two barrel-shaped LH1 rings meet. Scheuring et al. (4) report that lipid vesicles of ~50 nm diameter also form spontaneously when LH1 dimers are reconstituted in lipids—the very same size as the naturally occurring chromatophore vesicles, which again requires a bend in the LH1 dimers of the above-mentioned angle.

Now, there could be two opposite reasons for this observed bend of ~26°:

1. It may either be forced upon the LH1 dimers by the curvature of the supporting lipid membrane, or
2. The bend is built into the LH1 dimers, which then force the membrane to follow their curvature.

There are another two independent arguments against the first and for the second case, i.e., that the LH1 dimers are rather stiff, with an intrinsic bend, and consequently determine the size of the vesicles.

The first indirect argument comes from an observation of Barz et al. (25). They found that the chromatophore vesicles of a PufX-lacking mutant of *Rb. sphaeroides* were not closed, which permitted exchange of the Cytochrome c_2 and of the protons between the interior and exterior of the vesicles. Consequently, due to this shortcut, the vesicles were nonfunctional and this PufX$^-$ mutant was not able to grow photosynthetically. The main difference between these nonfunctional vesicles from the PufX$^-$ mutant and the observed closed vesicles of the PufX$^+$ wild-type is the formation of closed LH1 rings (monomers) for PufX$^-$ and Z-shaped LH1 dimers for PufX$^+$. If the lipids were responsible for closing the chromatophore vesicles, one would expect closed vesicles with the smaller LH1 monomers, i.e., without PufX, and open vesicles when the twice-as-large LH1 dimers are present. This would contradict the observations of Barz et al. (25).

Secondly, the bend of the LH1 dimers was directly observed in a completely different setup, too. Scheuring et al. (4) reported that LH1 dimers reconstituted into planar membranes form periodic corrugated structures with alternating rows of S- and Z-type dimers, as sketched in the upper panel of Fig. 2. In fact, a height-scan perpendicular to the rows (Fig. 4 *D* of (4), and indicated in the *upper panel* of Fig. 2, this article, by the *broken line*) is consistent with an arrangement of LH1 dimers with a bend of the above-mentioned 26° with their faces up and down alternatingly, as shown in the lower panel of Fig. 2. The S-type dimers are consequently dimers seen from the inside. If the LH1 dimers were flexible enough to adapt to a highly curved lipid membrane, then they should be able to adapt to a flat surface, too. Then one would not

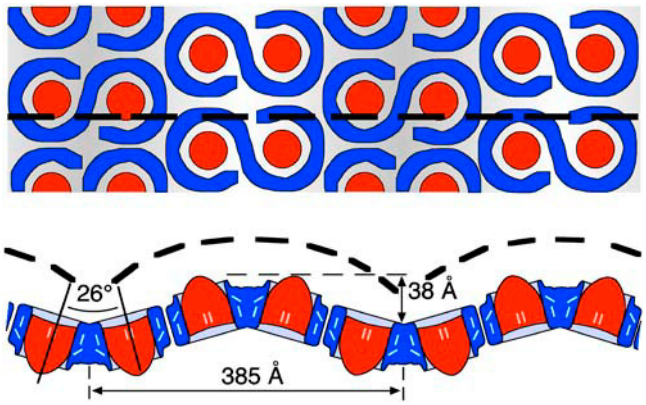

FIGURE 2 The upper panel of the figure is drawn after the AFM images of Scheuring et al. (4) of LH1 dimers reconstituted into planar lipid membranes. The lower panel shows an interpretation how alternatingly-oriented LH1 dimers may explain the observed height scan indicated by the dashed lines. The color-coding of the proteins is the same as in Fig. 1. The Z-shaped dimers are seen from their cytoplasmic side. The average height of the membrane is indicated in the upper panel by the shaded level of the background. Scheuring et al. (4) measured the distances between the minima of the AFM height scan to be 385 Å and the distance between the lowest and the highest parts to be 38 Å. These values are nicely reproduced by the proposed arrangement of the LH1 dimers, when one assumes that they are stiff enough to retain the bending angle of 26° that they would have on a spherical vesicle of 45-nm diameter and taking into account the length of a single LH1 dimer of ~195 Å.

expect the observed corrugated long range structure with almost crystalline ordering of the LH1 dimers, but extended patches of dimers that all present the same cyto- or periplasmic side. On the other hand, the observed corrugated ordering is most easily explained by chains of bent dimers that strongly attach to each other with their long axes parallel forming the chains of S- and Z-type dimers. To cancel the curvature of the LH1 dimers these chains then align upside down to the neighboring chains. The resulting arrangement of the LH1 dimers, which reproduces the observed AFM images, is sketched in the lower panel of Fig. 2 as the cross-sectional view.

These observations by Barz et al. (25) and by Scheuring et al. (4) consequently indicate that the LH1 dimers are indeed responsible for the correct formation and size of the chromatophore vesicles via their intrinsic bend induced by PufX. In vivo, the lipids on their own have no apparent reason to form buds and, finally, vesicles of this particular size, but the built-in curvature of the LH1 dimers, which are the largest objects on the vesicle, easily explains the observed size of the chromatophore vesicles.

CONSTRAINTS ON THE bc_1 POSITION

Arguments from inside the vesicle

After showing how the size of the chromatophore vesicles may be explained by the association of the bent LH1 dimers, the position of the bc_1 complexes needs to be determined.

In the accompanying article on the process-view of the vesicle, we estimated the transport capacities of the electron carriers c_2 and ubiquinone (Q). For both, the time necessary to diffuse across the vesicle was much smaller than the times for docking and undocking at the RC and the bc_1, respectively. Consequently, the electron carriers do not impose any constraint on the positions of the bc_1s on the vesicle: even for the extreme cases when the RC and the corresponding bc_1 are located diametrically across the vesicle, the diffusion times—which depend on the protein placement—make up only a small contribution to the transport capacities of the c_2s and of the Qs.

Actually, most of the dynamical data, including the diffusion of the electron carriers, is compatible with any setup ranging from super complexes, where the bc_1s are attached to the LH1/RC units, to a wide separation between the bc_1s on the one hand and LH1/RCs on the other hand. However, there is one experimental observation regarding the kinetics of photosynthesis that favors a spatial separation between the RCs and the bc_1s: for dark-adapted chromatophore vesicles of *Rb. sphaeroides*, a delay of $\sim$1 ms was found between the onset of illumination and the onset of the activity of the bc_1s (26). This delay is compatible with a diffusion of the reduced Qs from the RCs to the bc_1s over a distance of approximately the vesicle radius, i.e., with a setup where the bc_1s are as far away from the RCs as possible on the small spherical vesicles.

The recent images observed with different techniques also favor a setup in which the bc_1s are separated from the LH1/RC dimers. In particular, the observations of Scheuring et al. (4) point to a very strong association of the LH1/RC dimers, leaving no room for interspersed bc_1s. Even more, their experiments suggest that the mutual association of the LH1 dimers is much stronger than their interaction with the membrane, forcing the membrane to follow their conformation in space. Therefore, the LH1 dimers can be considered the backbone structure of the vesicles and the soft membrane is mainly necessary to separate the two sides of the photosynthetic proteins from each other, sealing the vesicle. From this, one expects the bc_1s to be separated from the array of the LHCs.

The proton flux outside of the vesicle

Seen from the outside, the vesicle takes up protons via the RCs and the bc_1s and ejects them by the ATPase. To satisfy the overall balance, every proton entering the vesicle has to leave again. Thus, the overall proton turnover can be set to the maximal throughput of the one ATPase of the vesicle of some 400 protons/s and vesicle at maximum (for details, see (9)). Due to the bc_1's Q-cycle, half of these 400 protons are taken up by the RCs (and packed onto the QH_2s), while the other half is pumped into the vesicle by the bc_1s. Because the RCs—due to the size of their surrounding LH1s—are spread out over the surface of the vesicle, to a first approximation the whole surface of the vesicle is covered sparsely by them. Consequently, the proton uptake rate per vesicle area through the RCs is rather small. The other half of the protons are taken up via the few bc_1s, which means that each of them has to capture approximately four-times as many protons per second as one RC while occupying $<$1/10th of the membrane surface.

Moreover, the proton efflux through the single ATPase is very localized: all 400 protons per second leave the vesicle at a small spot between the ATPase's *a*- and *c*-subunits. This creates a high local proton density right at this spot from which the protons diffuse away into all directions. Assuming isotropic diffusion, approximately half of them will bump into the vesicle membrane again close to the ATPase.

Consequently, it would be most efficient to place the bc_1s close to the ATPase, where they benefit from the locally increased proton density, increasing their proton capture probability. Half of the protons leaving the ATPase would thus end up being recaptured immediately by the bc_1s. Note that under steady-state conditions this argument is independent of the diffusion coefficient of the protons and only requires the spatially isotropic diffusion of the emitted protons. The diffusion coefficient itself determines how fast a proton will hit the vesicle membrane but not the probability for this collision to occur. As a side effect, this recycling of the protons by the bc_1s placed close to the ATPase efficiently reduces the acidic load that the cell has to cope with.

PROPOSING A MODEL VESICLE

Piecing together all information collected in the previous sections and in the accompanying article (9) we come up with a model of the chromatophore vesicles as depicted in Fig. 3: the main defining structure, i.e., the backbone of the vesicle, is a linear array of parallel-assembled LH1/RC dimers. These bent LH1/RC dimers define the curvature of the vesicle membrane and therefore its diameter. Attached to this belt of LH1s around the vesicle's waist, one finds the smaller LH2 rings. Due to their smaller size, the spherical caps of the vesicle can be efficiently filled with them, making best use of the available membrane area. As the LH1s and the LH2s are built from the same α–β dimers, one may expect that the LH2s are mechanically attached to each other and to the LH1s with a comparable strength as the LH1 dimers to each other. The coupling between the LH1s and the LH2s has to be strong to efficiently transmit the excitons from the captured photons. As proposed above, the ATPase and bc_1s then sit on one of the endcaps of the vesicle close to each other, separated from the array of the light-harvesting complexes (LHCs).

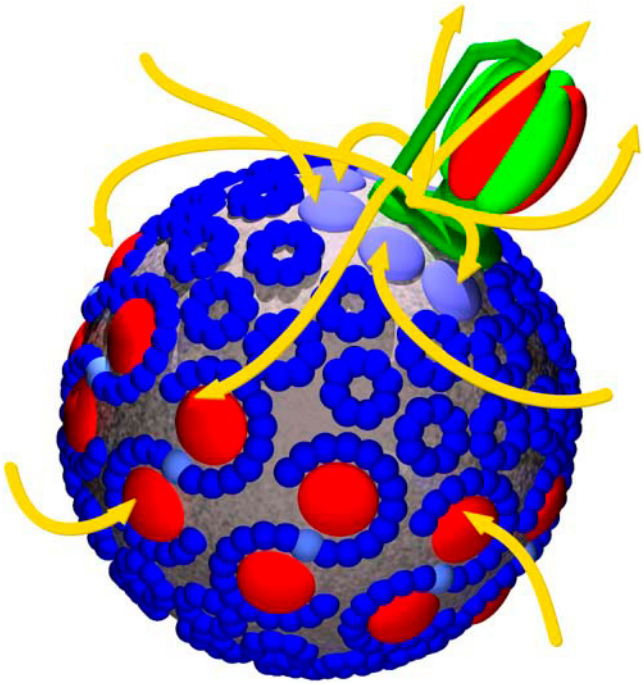

FIGURE 3 Proposed setup of a chromatophore vesicle from the data compiled in this study (compare this to the textbook-style rendering in the accompanying article, from Fig. 1 of Geyer and Helms (9), and to Fig. 1 of this article): the Z-shaped LH1/RC dimers (*blue/red*) form a linear array around the equator of the vesicle, determining the vesicle's diameter by their intrinsic curvature. Located next to this array are the smaller LH2 rings (*blue*), permitting efficient exciton transfer onto the LH1s. The ATPase (*green/red*) and the bc_1 complexes (*light blue*) are consequently found at the vesicle's poles. We suggest a setup of ATPase and bc_1 complexes close together at the same end of the vesicle being favored due to the increased proton density close to the ATPase. The diffusion of the protons out of the vesicle via the ATPase and to the RCs and bc_1s is exemplified by the yellow arrows. For a more detailed explanation, see text.

This picture nicely fits with the observed AFM images: the mechanically weak spots of the vesicle are the calotte-shaped endcaps. Here, the vesicle will start to break apart when subjected to the preparation process before the AFM imaging, preferentially on that side which holds the huge protruding ATPase. The belt of LH1s, the central structure of the vesicle, survives the highest stress and remains mostly intact, while the LH2s sticking to this structure in hierarchies have to reorder together with the remains of the lipid membrane. The ATPase and the bc_1s, on the other hand, which need not be coupled electronically (and mechanically) to the LHCs, are only associated with this LHC agglomerate via the comparatively weak lipid membrane. Accordingly, neither ATPase nor bc_1 complexes are found any more on the AFM images of the flattened membranes, while the fragments of the LH1 belt are the dominating structures in the recent AFM images (3). Consequently, a strategy to image the bc_1s would be to look for membrane patches still attached to the comparatively huge ATPase. These, then, should contain some bc_1 dimers.

The accompanying article (9) presented the two scenarios for the number of the bc_1s that may either be associated with the RCs or with the ATPase. This reasoning was purely based on stoichiometries and kinetic information, only considering the respective inputs and outputs of the proteins, so that the electron and proton cycles would be closed most tightly. However, one may also consider as an evolutionary argument which of the two cycles came first, the RC-bc_1-electron cycle or the bc_1-ATPase-proton cycle? In the first case, one could speculate that the bc_1s should be spatially connected to the RCs, while in the second case the bc_1s may be preferentially placed next to the ATPase. Actually, the bc_1-ATPase-proton cycle is not unique to photosynthesis but also part of the respiratory chain—which is older from an evolutionary point of view. This supports our suggestion to relate the bc_1s to the ATPase—both kinetically and spatially—and consequently to place them together as a unit onto one of the endcaps of the vesicle.

SUMMARY AND CONCLUSIONS

This study was motivated by a recent AFM study of chromatophore vesicles from purple bacteria where the light-harvesting complexes and reaction centers are clearly visible, but the other two components, the Cytochrome bc_1 complex and the F_0F_1–ATPase, could not be identified on the images. With the aim to assemble a complete vesicle we collected the available experimental information about all the parts of the photosynthetic apparatus of *Rb. sphaeroides*. Hopefully, this effort would help us in identifying the positions of the bc_1 complexes, too.

Our arguments for the placement of the bc_1s come from the observation that the LH1/RC dimers assemble into rather

rigid arrays, which then determine the size of the vesicles, and from the spatial properties of the proton flux into and out of the vesicle: the beltlike LH1/RC array spanning around the equator of the vesicle is proposed to push the bc_1s and also the large ATPase to the poles of the vesicle. As the protons leave the vesicle at a single spot at the ATPase, but are taken up over the rest of the surface, the bc_1s may be favorably placed into the high local proton concentration next to the ATPase, i.e., right around the ATPase. This arrangement would explain why, during the preparation process, both the ATPase and the bc_1s are lost together and consequently do not show up on the AFM images.

This article about the spatial setup of the chromatophore vesicles of *Rb. sphaeroides* is closely related to the accompanying article (9), where we discuss the kinetic aspects of the photosynthetic apparatus. Both articles together present a comprehensive minimal model of these vesicles. As these vesicles are small, naturally closed systems with only a few different proteins and a manageable total number of moving molecules, we now have at hand a well-suited model system for molecular simulations. With it, one can not only learn the technical aspects of how to conduct simulations in such a coarse-grained description, but also use it to complete our understanding of photosynthesis.

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