people were. 'All they do is carve variously-sized wooden bears. Who in their right mind would buy one?' 'Well, Mike, we did', we replied. He got a little quieter (just a little). When Mary and I got home, Mike had had his bypass operation and our wooden bear arrived. We looked at each other and said 'Mike!' We sent it to him with a note saying that he had admired it so much he should have it. No reply from Mike for months – then a short note. 'Well, I've had it on my mantel three months. We don't seem to have lost any friends because of it. Some people even admired the thing. I guess we're going to have to learn to live with each other.' When our daughter was born in 1981, her first present was a stuffed bear, which promptly was named 'Gill-bear.'

We miss you, Gill-bear.

References

1 Gill, D. M., Pappenheimer, A. M., Brown, R. and Kurnick, J. T. (1969) *J. Exp. Med.* 129, 1–21
2 Gill, D. M. (1975) *Proceedings of the 10th Joint Conference on Cholera* (Kyoto, October, 1974). The US–Japan Cooperative Medical Science Program, pp. 119–128
3 Gill, D. M. and King, C. A. (1975) *J. Biol. Chem.* 250, 6424–6432
4 Gill, D. M. (1975) *Proc. Natl Acad. Sci. USA* 72, 2064–2068
5 Enomoto, K. and Gill, D. M. (1980) *J. Biol. Chem.* 255, 1252–1258
6 Gill, D. M. and Meren, R. (1983) *J. Biol. Chem.* 258, 11908–11914
7 Rubin, E. J., Gill, D. M., Boquet, P. and Popoff, M. D. (1988) *Mol. Cell. Biol.* 8, 418–426
8 Chardin, P., Boquet, P., Madaule, P., Popoff, M. D., Rubin, E. J. and Gill, D. M. (1989) *EMBO J.* 8, 1087–1092
9 Benjamin, R. C. and Gill, D. M. (1980) *J. Biol. Chem.* 225, 10493–10508
10 Ikejima, M. and Gill, D. M. (1987) *J. Biol. Chem.* 262, 17641–17650

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TALKING POINT

Restricted diffusion in photosynthetic membranes

Jérôme Lavergne and Pierre Joliot

The structural organization of membrane proteins and their linkage by diffusion are topics of much debate. Functional studies in photosynthetic membranes, where rapid equilibration of electron transport between redox centers appears restricted to isolated domains, shed new light on the subject.

ELECTRON TRANSFER CHAINS in bioenergetic organelles combine two sets of processes. Most reactions occur through electron tunneling between redox centers that are strictly positioned within integral membrane proteins. These large blocks can be considered immobile on the time-scale of electron transfer, and redox transfer between them is achieved through shuttling of smaller diffusible carriers[1]. In chloroplasts, two such carriers are involved[2], the lipid-soluble plastoquinone and the water-soluble plastocyanin, which is confined in the lumenal space within the thylakoid sacs. Homologous carriers in the bacterial photosynthetic electron transport chain[3] are ubiquinone and cytochrome c_2, which is confined to the periplasmic space. We will show, however, that in several instances these molecules do not diffuse freely within the membrane or along its surface, at least on a rapid time-scale. Evidence for such restrictions to diffusion gives useful clues as to the lateral organization of membrane proteins. Although the scope of this article concerns only photosynthetic membranes, similar considerations apply to respiratory electron transfer.

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Such information is especially valuable since it provides a bridge between two levels on which most structural investigations have focussed. Our knowledge of structure on the molecular scale is rapidly increasing thanks to biophysical and biochemical techniques and to the X-ray crystallographic solution of the three-dimensional structure of bacterial reaction centers[4,5]. On the other hand, electron microscopy and associated techniques give pictures of the large-scale organization of the membrane[6]; in particular they have established the lateral heterogeneity of the chloroplast membrane between the appressed regions of grana stacks and unappressed regions.

Our approach is based on the idea that a restricted diffusion of electron carriers, confined within isolated 'domains', will prevent global equilibration of the photochemically-generated redox equivalents. This will appear as discrepancies between the quasi-equilibria resulting from illumination and thermodynamic data derived from equilibrium titrations in the presence of redox mediators. Experimental data has revealed many instances of such discrepancies, which, combined with appropriate kinetic studies, suggest roles for novel structural features[7–10].

Equilibrium within isolated domains

Before giving specific examples, we would like to outline the general ideas that are involved in this approach (see Ref. 7 for more details). We consider a section of a photosynthetic electron transfer chain, say, on the donor side of a reaction center (although the same principles will apply to an acceptor chain). This donor chain includes redox

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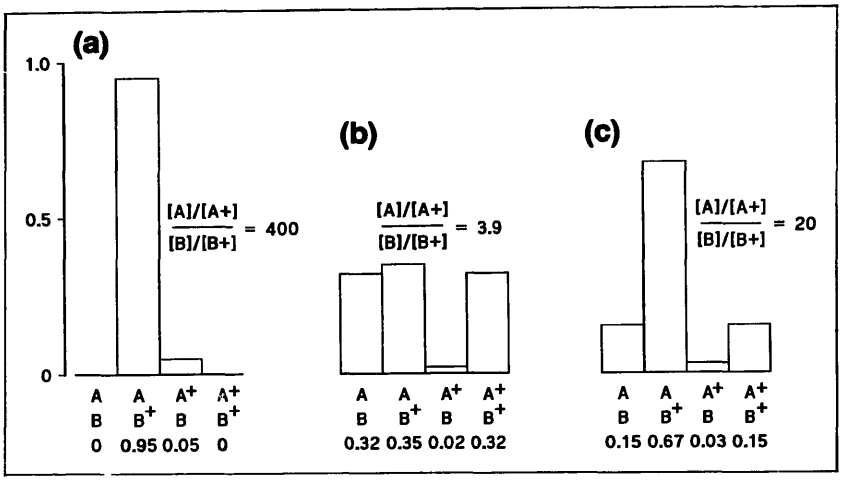

Figure 1

Basic features in the behaviour of isolated domains can be illustrated by considering a two-partner donor complex with a primary donor, A, and a secondary one, B, with lower potential. The mid-point potentials of the two carriers are assumed to be independent of the redox state of the partner (e.g. no electrostatic interaction between the redox centers) and their difference is taken around 75 mV, corresponding to an equilibrium constant K_{eq}=20. A photochemical reaction extracts an electron from A and rapid relaxation then occurs within the complex until $[AB^+]/[A^+B]$=20, according to the scheme: $AB \xrightarrow{h\nu} A^+B \leftrightarrow AB^+$. The diagrams show the distribution of the various states of the complex after this intra-complex equilibration has taken place, where one positive charge is present in the complex. **(a)** One single turnover flash has been given, injecting a single positive charge in all the complexes (so that states AB and A^+B^+ are not populated). **(b)** Following a period of weak continuous illumination, the complex has a (near) poissonian probability of having undergone 0 (state AB), 1 (states $A^+B \leftrightarrow AB^+$) or more than 1 (state A^+B^+) photochemical turnovers. (a) and (b) relax towards **(c)**, the global inter-complex equilibrium that may be reached through complex–complex collisions or through an exogenous redox mediator at low concentration. Only in this state of global equilibrium is the ratio $([A]/[A^+])/([B]/[B^+])$ equal to K_{eq}. By contrast, the one-flash distribution (a) corresponds to an under-oxidation of A and the continuous illumination (b) to an over-oxidation of A. In the latter case, when K_{eq} is large (say, >50), the distribution becomes totally independent of K_{eq} (Ref. 8), with $([A]/[A^+])/([B]/[B^+])$=4.6.

centers bound to membrane proteins and, possibly, mobile carriers that may, within a certain diffusional domain, connect the complexes. By shining light on the system, electrons can be removed from the chain through photochemical turnovers of the reaction center. An additional experimental condition is to prevent any re-reduction, for example, by use of an appropriate inhibitor, thereby creating a cul-de-sac. Following a period of illumination that leaves the system in a partially oxidized state, a relaxation will take place within any domains connected by the diffusing carriers. If all the redox chains can interact with each other (i.e. if the domains are large or exogenous redox mediator is added) a state of global equilibrium will be reached. The respective oxidation states of two redox centers within a chain is controlled by an equilibrium constant that can be deduced from the difference in the mid-point potentials. If, however, the diffusion of the mobile carriers is restricted to small domains, the resulting state of the various redox centers may vary quite significantly from the global equilibrium, depending on the history of the system, particularly the illumination procedure used. Feeding charges into the system through an irreversible photochemical process is not equivalent to balancing the system at some redox potential and will not, in general, result in the particular distribution of redox states that would fit global equilibrium.

An illustration is given in Fig. 1, where a simple two-partner complex (primary donor, A, and secondary donor, B) is considered. Illumination by a single turnover flash forces the same degree of oxidation in all domains, leaving two of the four possible states of the system (AB and A^+B^+) unpopulated. Although equilibrium is achieved within each complex, the overall result is an under-oxidation (compared with global equilibrium) of the high potential (primary) donors (A) with respect to the lower potential (terminal) ones (B). Conversely, continuous illumination will cause over-oxidation of the primary donor (A^+) by totally removing electrons in a fraction of the domains. This is because of the poissonian distribution of photons during continuous illumination: while some domains receive less photons than average, some receive more and become completely photo-oxidized earlier. The effects illustrated in Fig. 1 are significant only when the size of the domains (the number of redox centers per domain) is small. However, similar deviations from global equilibrium may occur in larger domains if their individual stoichiometry is not fixed, but varies around an average value, as is discussed later about the acceptor chain of photosystem II.

Super-complexes in bacterial chains

Our first example is the donor chain in the photosynthetic bacterium *Rhodobacter sphaeroides*[8]. The relevant redox centers are P, the primary donor (the bacteriochlorophyll special pair in the reaction center), cytochrome c_2 as a secondary donor, then cytochrome c_1 and the Fe–S center that both belong to the cytochrome *b–c* complex (analogous to cytochrome b_6–*f* in chloroplasts). Experimentally, this section of the bacterial electron-transfer cycle can be easily isolated kinetically by using myxothiazol as an inhibitor of quinol reoxidation by cytochrome *b–c*. Cytochromes c_1 and c_2 have similar mid-point potential values, and their equilibrium constant with P (K_{eq}= $[P][Cyt^+]/[P^+][Cyt]$) is about 70. Starting from a reduced donor chain, continuous light can be shone on the system, the oxidation kinetics of P and the cytochromes can be monitored, then (P_{red}) against (Cyt_{red}) can be plotted, as shown in Fig. 2. The data are very different to the equilibrium curve (K_{eq}=70), drawn as a solid line. They would actually suggest, when starting from a totally reduced state, an equilibrium constant of about six, meaning a considerable over-oxidation of P in comparison with the expected equilibrium. Distortion caused by a kinetic limitation is unlikely, since the intensity of the light corresponded to a photochemical turnover rate much slower than that of electron transfer from cytochrome c_2 to P. Indeed, when the light intensity was lowered by an order of magnitude, the same curve was obtained. A quasi-equilibrium of some sort has thus been achieved, although it differs grossly from the global equilibration. However,

data in agreement with the global equilibrium is obtained when the relaxation time is extended to several minutes. This was seen by modulating the redox balance of the cell in the dark through the respiratory rate. The above data provide a strong indication that the carriers are organized in small domains, such as 'super-complexes'. Rapid equilibrium occurs within one super-complex, while equilibration between super-complexes is much slower, unless accelerated by exogenous redox mediators. During the photooxidation process, some super-complexes will have received more photons than others and become totally oxidized earlier, which accounts for the observed over-oxidation of P.

The precise stoichiometric and kinetic features of the super-complexes were deduced from additional experiments. Firstly, the total amount of photo-oxidizable cytochrome (c_1+c_2) is only 87% of the amount of P. Since there are roughly equal amounts of c_1 and c_2, this suggests, as a first approximation, a basic stoichiometry $[(P)_2(c_2)(b–c)]_n$. Following illumination with a weak flash, which randomly samples a small fraction of the centers, total rereduction of P^+ is completed in less than 1 ms; this excludes the possibility of reaction centers disconnected from any secondary donor. On the other hand, when a saturating flash is used, about 12% of P remains oxidized in the 100 ms time-range. This suggests that in a fraction of the super-complexes there are less secondary donors (c_2+c_1+Fe–S) available than there are primary donors. A simple explanation is that there is not enough cytochrome *b–c* available to complete every super-complex in the form $[(P)_2(c_2)(b–c)]$, so that a fraction (slightly more than 2 × 12% because of the photochemical misses) of the super-complexes remain as $[(P)_2(c_2)]$. In these, following a saturating flash, the state $P^+P^+c_2$ relaxes towards $P^+Pc_2^+$, whereas in the complete super-complex enough secondary donors are available to achieve rereduction of both primary donors.

This model can be tested in a rather precise way, since the photo-oxidation process can be simulated using these stoichiometric parameters (and the equilibrium constants between the redox centers) without resorting to the use of a variable parameter. As may be seen in Fig. 2, where the dashed curves show the simulated results, the fit is quite good (and remains so when simulating similar experiments with different initial redox poise, or flash-driven photo-oxidation). This comparison with simulated curves rules out larger structures, such as $[(P)_2(c_2)(b–c)]_2$.

Finer mechanistic details were obtained from flash-induced kinetics in the sub-millisecond time-range. A biphasic time-course of P^+ reduction is observed with a fast (3 μs) phase involving less than half of the centers, and a slow phase in the 100 μs range that is further slowed when a saturating flash is used. In the case of a weak flash (hitting 5% of the centers), the probability of hitting both centers in a single super-complex is very low, thus most P^+ formed will occur as $P\ P^+c_2(b–c)$. The fact that less than half of these react with a 3 μs time constant indicates that only in this fraction of the super-complexes c_2 is properly attached to the P that was photo-oxidized. This agrees with the idea (see the inset in Fig. 2) that within a super-complex, the c_2 molecule has three {or two in the $[(P)_2(c_2)]$ fraction} binding sites, one on each of the Ps, and one on the *b–c* complex. The second phase thus corresponds with the switching time from one of the inactive configurations (c_2 bound on the *b–c* complex or on the reduced P) to the active one (c_2 bound on the oxidized P). When a saturating flash is used, c_2 needs to be rereduced by cytochrome c_1 (when available) before reacting with the second P^+. This mechanism accounts for the slower (130 μs instead of 65 μs) second phase observed in this case.

A dimeric association of the reaction centers has also been found in another

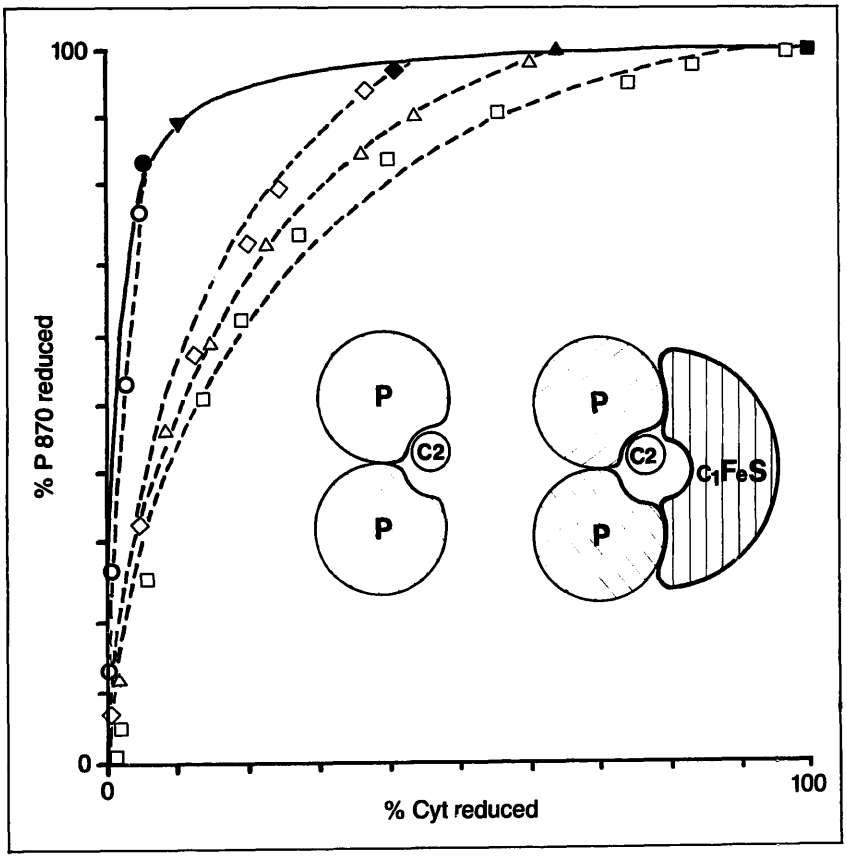

Figure 2

A plot of the amount of reduced primary donor (P-870) versus the amount of reduced cytochrome (c_2+c_1) in myxothiazol-poisoned cells of *R. sphaeroides* (redrawn from Ref. 8). The experimental data points were plotted with various symbols corresponding to different initial redox balances in the dark. These initial states are shown as closed symbols; the corresponding open symbols display the states reached during subsequent photo-oxidation under weak continuous illumination (the common end-point for total photo-oxidation is at the axis origin). The solid line is the equilibrium curve for K_{eq}=70, which closely fits the initial redox distribution (closed symbols). The dashed curves were computed according to the treatment developed in Ref. 7 using the parameters described in the text. The inset is a schematic representation of the two types of super-complexes, and is not intended to give a realistic structural picture.

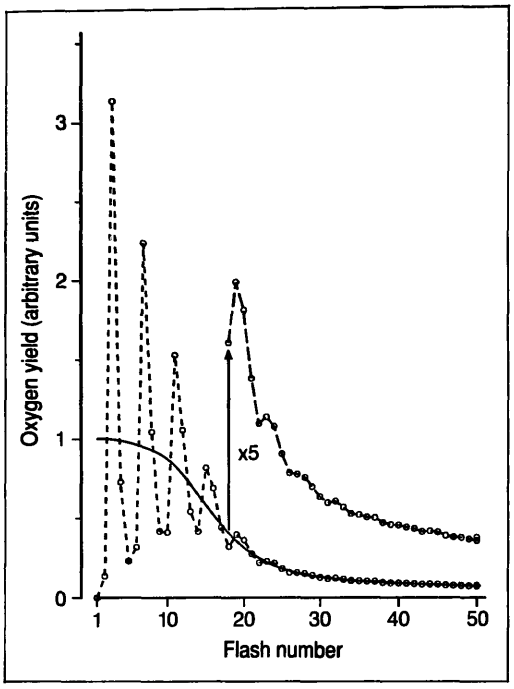

Figure 3

Oxygen evolution during a sequence of short saturating flashes in spinach chloroplasts (anaerobic conditions, no PSI acceptor). When the final part was replotted on an expanded scale (X5), the oscillatory pattern was still discernible (with no phase-shift) at the seventh period. The solid curve was obtained by averaging the oxygen yield on successive groups of four flashes, showing the progressive closing of PSII centers accompanying the reduction of the PQ pool. If global equilibrium (with K_{eq}>50) were achieved during the reductive process, this curve would consist of a plateau followed by a step decrease (i.e. no significant Q_A reduction until most PQ is reduced).

bacterium, *Rhodospirillum rubrum*[9]. There is no association to cytochrome *b–c* in this case, nor tight binding of cytochrome c_2, although there is still a binding region in the dimer that can only accommodate a single c_2. In both cases, the evidence for associations in super-complexes was inferred from functional experiments and still awaits direct confirmation from structural methods.

It has been suggested that the mitochondrial cytochrome-*c* oxidase also exists as dimers *in vivo*. It was shown that covalent binding of one cytochrome *c* per dimer was sufficient to cause inhibition of both oxidases[11], which may suggest a similar arrangement to the $[(P)_2(c)_2]$ super-complex.

The photosystem II acceptor chain

Three integral proteins are involved in the photosynthetic non-cyclic electron transfer chain: photosystem II (PSII), the cytochrome b_6–*f* complex and photosystem I (PSI). Electrons from PSII are carried to the b_6–*f* complex by plastoquinones, while plastocyanin acts as carrier between the b_6–*f* complex and PSI. The overall transfer of electrons from water to NADP is quite rapid and efficient: it thus came as a surprise that PSIi and PSI turned out to be located in different places within the photosynthetic membranes (thylakoid). The vast majority of PSII is segregated in the appressed regions (granal stacks), while PSI is only present in non-appressed regions (stromal lamellae)[1,2,6]. The distance between them is in the order of 0.1 μm. Therefore, long-range rapid diffusion must be achieved by either plastoquinone or plastocyanin, or both. Structural studies do not help in deciding between the two candidates since cytochrome b_6–*f* is found in both regions[12]. In the absence of definitive proof (for review see Ref. 2), many authors have argued in favour of plastoquinone. This does not seem to demand an unacceptably large diffusion coefficient for this molecule in the lipid phase of the membrane[13,14]. Furthermore, plastoquinone has been found approximately evenly distributed in both membrane regions[15]. A more positive indication is that several PSII centers feed electrons into a common pool of plastoquinones, as evidenced by the slower photoreduction of the pool when inhibiting a fraction of the PSII centers[16,17]. It was recently shown by Mitchell and co-workers[14] that the overall kinetics of electron transfer from PSII to PSI could be satisfactorily simulated by a model based on plastoquinone diffusion. An interesting feature of this work is the use of a Monte Carlo approach that takes into account the complicated geometry of the thylakoid structure and the crowding of the membrane by protein complexes (although, in our opinion, this aspect was still under-estimated by these authors). We will describe recent evidence[10] that led us to adopt the unorthodox view that plastoquinone cannot be involved in long-range electron transfer.

The 'primary' acceptor of PSII, a reaction-center-bound plastoquinone named Q_A (the existence of more primary acceptors, chlorophyll and pheophytin is of no relevance here) feeds electrons into the pool of plastoquinones (PQ) diffusing in the lipid phase of the membrane[1]. There are about eight PQ per PSII, each accepting two electrons, so that the average capacity of the pool is about 16 electrons per PSII. Redox titrations indicate a midpoint potential of PQ more than 100 mV above that of Q_A, so that the equilibrium constant is greater than 50 in the direction of PQ reduction. Nevertheless, when the pool is photoreduced under weak continuous light or a train of low frequency saturating flashes, an apparent equilibrium constant of 3–5 is observed between Q_A and PQ (see Ref. 10 and references therein), meaning that the reduction of Q_A with respect to PQ is much larger than expected from equilibrium considerations. Again, however, kinetic limitations are unlikely since the photochemical rate (in the range 10–500 ms) is much slower than the known transfer rate from Q_A^- to PQ (sub-millisecond range). Relaxation towards the global equilibrium occurs in the 10 s time range. The situation is so far similar to that described for the donor chain of *R. sphaeroides*, and might suggest super-complexes of the form $[(PSII)(PQ)_8]_n$. However, the electron capacity of such a structure, even taking n=1, is too large to account for the small apparent equilibrium constant[7]. Furthermore, as alluded above, it has been established that several centers share a common pool (thus, n>1).

A crucial piece of information came from analysing the oxygen evolution pattern under flashing light. Oxygen is evolved after the donor side of PSII has accumulated four oxidizing equivalents, resulting from four successive photoevents on an individual center. In the dark-adapted state, the system is initially reduced so that a characteristic evolution pattern is obtained, oscillating with a periodicity of four flashes[18]. The oscillation is damped due to an intrinsic probability of photochemical misses or double-hits occurring randomly on each center. When reoxidation of the pool is inhibited, a progressive decrease of the yield is observed (Fig. 3), reflecting the closing of reaction centers due to Q_A reduction with a

small apparent equilibrium constant. However, no additional damping is observed under these conditions. If the closing of the centers were controlled by a global equilibrium with reduced PQ, there would be an increased probability of photochemical misses and dramatic damping and phase shift in the sequence would occur; it would be expected that the sequence would be completely smoothed over after a few periods. The absence of damping suggests an all-or-none behaviour: each center is experiencing a large equilibrium constant with a locally accessible pool and the centers that stop emitting oxygen do so only when this pool is completely filled up with electrons. We must resort again to the concept of isolated domains, but now allowing variability in their stoichiometric composition[10] (PQ/PSII ratio). The domains with a smaller PQ pool are the first to achieve total photoreduction, and their PSII close earlier. The actual distribution of pool sizes can be approximated by assuming that the fraction of centers that are closed after flash n+1 in a train of saturating flashes gives the fraction of domains with $n/2$ PQ per center. A satisfactory simulation of the experimental oxygen evolution sequence is then obtained by using this distribution for weighting theoretical sequences corresponding to the various pool sizes (where a large equilibrium constant is assumed).

Knowledge of the distribution of the PQ/Q_A ratio among domains leaves open the problem of the absolute stoichiometries: how many PSII centers are there, on average, per domain? Relevant information can be drawn from experiments where a fraction of the centers is inhibited. As mentioned above, previous experiments[16,17] of this kind had revealed, as a primary effect, the slowing down of the pool reduction, indicating that several centers were connected by a common pool. We reinvestigated this problem, focussing on the total amount of PQ being reduced under such conditions. For a given fraction of inhibited centers, the probability of having all centers inhibited in a domain (thus no local PQ reduction) depends on this stoichiometric ratio. We found a decrease of the rapidly photoreducible PQ when partially inhibiting PSII that leads to an estimate of 4–8 PSII centers per domain.

What physical structure can be envisaged to account for these findings? Our present view is a (non-)percolation model in which the integral membrane proteins delimit closed regions of the lipid phase[19] (Fig. 4). Fluctuations in the number of reaction centers per domain area and, to a lesser degree, fluctuations of the PQ concentration could account for the wide variation in stoichiometry. This view may not be unrealistic since cryofracture micrographs indicate that about half, and possibly more, of the bilayer midplane is obstructed by integral proteins. This level of crowding represents the percolation threshold in two dimensions: randomly crowding the surface above this value will prevent any long-range diffusion of small objects. Non-random associations between integral proteins may alter this threshold in either direction.

When the photoreduction process is interrupted and resumed after several seconds, the $[Q_A^-]$ versus $[PQ_{red}]$ curve is shifted towards the global equilibrium curve. This indicates redistribution of PQ among domains in this time-range. Since the domains contain a small number of PSII centers (less than eight), their size must be much smaller than granal stacks, and the diffusion of PQ from the appressed to non-appressed regions must involve the crossing of many domain boundaries. Such a slow process rules out the long-range diffusion of PQ being responsible for the transfer between both photosystems which occurs over a few milliseconds. However, a good case can be made for the other candidate for this job, plastocyanin. Millisecond oxidation of all cytochrome *f* by PSI through plastocyanin has been reported[2]. Since this includes the large fraction of the b_6–*f* complex located in PSII regions, one must conclude that plastocyanin, which is contained with the thylakoid lumen, manages to worm its way efficiently through the whole lumen, regardless of its complex geometry and of the proteins protruding from the membrane

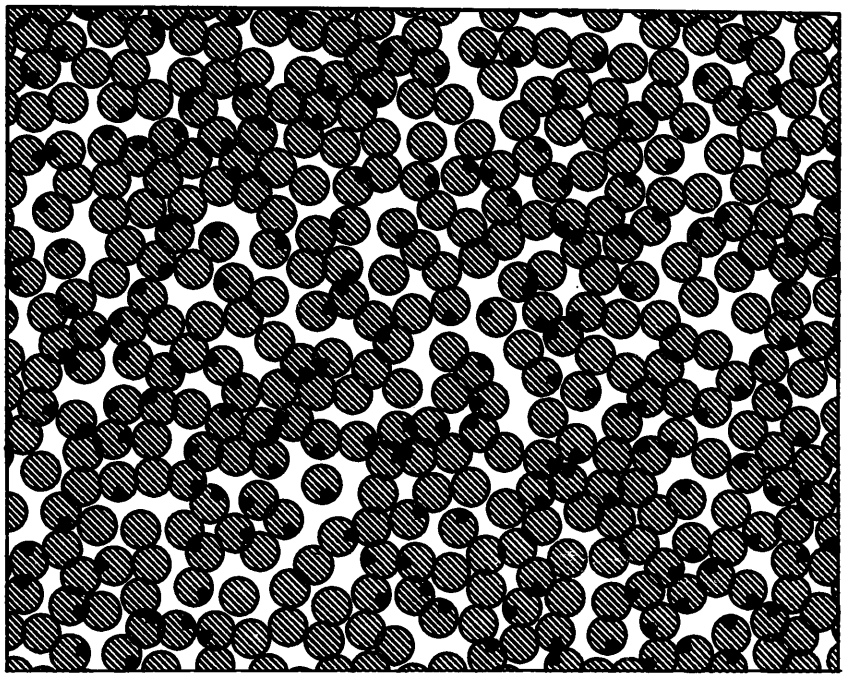

Figure 4

A computer-generated image illustrating our model for restriction of PQ diffusion. The circles stand for membrane proteins, half of them representing PSII centers with the black sector as the binding site for PQ. The white space is the area available for PQ diffusion, consisting of closed domains largely heterogeneous in size. We propose that the fluctuations in the number of PSII active sites per domain area are the origin of the broad distribution found for the PSII/PQ stoichiometry. The program first generated a random pattern of non-overlapping circles; the radius of each circle was then increased by 25% to create the slight overlap. This overlap is a convenient way to visualize the effective barrier for PQ diffusion, taking into account the minimum width of this molecule (7Å). This model is very crude and does not allow for a number of known or expected features such as heterogeneity in particle sizes, repulsive or attractive interactions between proteins and other specific associations. A possibly interesting feature in this simulation is the significant fraction of centers that have no access to PQ: this might be the origin for the experimental finding of a fraction of 'inactive' centers[20–22].

surface. A consequence of this view is that the b_6–f complex present in the non-appressed regions cannot be involved in linear electron transfer from PSII, and must be dedicated to cyclic transfer around PSI.

In comparing the PSII acceptor chain with the investigation of a bacterial donor chain, it is noteworthy that we were led to quite different structural models although the basic 'thermodynamic' information was similar. In both instances, the discrepancy between the quasi-equilibrium states reached during illumination and the global equilibrium suggests that mobile carriers (c_2 or PQ) are confined within isolated domains, on a rapid time-scale. With the bacterial donor chain, the domains are small, associating a few (two or three) membrane proteins. These structures have a precise stoichiometry and probably correspond to reproducible oligomeric entities. The area of the domain of diffusion for cytochrome c_2 is probably less than 100 nm^2. An electrostatic confinement rather than a protein 'pocket' is more likely since the latter structure is not supported by the three-dimensional structure of the reaction center[5]. Much larger domains are involved for the PSII acceptor chain with an area of the order of 10 000 nm^2. These structures, resulting from the more or less random spatial distribution of membrane proteins, are expected to be very inhomogeneous, thus accounting for the broad spread of the stoichiometric ratio PQ to PSII.

References

1 Ort, D. R. (1986) in *Photosynthesis III, Encyclopedia of Plant Physiology (Vol. 19)* (Staehlin, L. A. and Arntzen, C. J., eds), pp. 143–189, Springer-Verlag
2 Whitmarsh, J. (1986) in *Photosynthesis III, Encyclopedia of Plant Physiology (Vol. 19)* (Staehlin, L. A. and Arntzen, C. J., eds), pp. 508–525, Springer-Verlag
3 Dutton, P. L. (1986) in *Photosynthesis III, Encyclopedia of Plant Physiology (Vol. 19)* (Staehlin, L. A. and Arntzen, C. J., eds), pp. 197–232, Springer-Verlag
4 Deisenhofer, J. and Michel, H. (1989) *Science* 245, 1463–1473
5 Rees, D. C., Komiya, H., Yeates, T. O., Allen, J. P. and Feher, G. (1989) *Annu. Rev. Biochem.* 58, 607–633
6 Staehlin, L. A. (1986) in *Photosynthesis III, Encyclopedia of Plant Physiology (Vol. 19)* (Staehlin, L. A. and Arntzen, C. J., eds), pp. 1–72, Springer-Verlag
7 Lavergne, J., Joliot, P. and Verméglio, A. (1989) *Biochim. Biophys. Acta* 975, 346–354
8 Joliot, P., Verméglio, A. and Joliot, A. (1989) *Biochim. Biophys. Acta* 975, 336–345
9 Joliot, P., Verméglio, A. and Joliot, A. (1990) *Biochemistry* 29, 4355–4361
10 Joliot, P., Lavergne, J. and Béal, D. (1990) in *Current Research in Photosynthesis (Vol. 2)* (Baltscheffsky, M., ed.), pp. 879–882, Kluwer Academic Publishers
11 Bisson, R., Jacobs, B. and Capaldi, R. A. (1980) *Biochemistry* 19, 4173–4178
12 Olive, J., Vallon, O., Wollman, F. A., Recouvreur, M. and Bennoun, P. (1986) *Biochim. Biophys. Acta* 851, 239–248
13 Millner, P. A. and Barber, J. (1984) *FEBS Lett.* 169, 1–6
14 Mitchell, R., Spillmann, A. and Haehnel, W. (1990) *Biophys. J.* 58, 1011–1024
15 Chapman, D. J. and Barber, J. (1986) *Biochim. Biophys. Acta* 850, 170–172
16 Joliot, A. (1968) *Physiol. Veg.* 6, 235–254
17 Siggel, U., Renger, G., Stiehl, H. and Rumberg, B. (1972) *Biochim. Biophys. Acta* 256, 328–335
18 Joliot, P. and Kok, B. (1975) in *Bioenergetics of Photosynthesis* (Govindjee, ed.), pp. 387–412, Academic Press
19 Saxton, M. J. (1989) *Biophys. J.* 56, 615–622
20 Chylla, R., Garab, G. and Whitmarsh, J. (1987) in *Progress in Photosynthesis Research (Vol. II)* (Biggins, J., ed.), pp. 237–240, Martinus Nijhoff
21 Graan, T. and Ort, D. (1987) in *Progress in Photosynthesis Research (Vol. II)* (Biggins, J., ed.), pp. 241–244, Martinus Nijhoff
22 Lavergne, J. (1987) *Biochim. Biophys. Acta* 894, 91–107