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A Perspective on Q-Cycles

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Abstract

An examination is made of both the Q-cycle and *b*-cycle formulations of electron transfer and energy conservation in the cytochrome bc_1 complex. A working hypothesis for the complex is presented, based upon the Q-cycle notion of vectorial reaction sites, but incorporating the *b*-cycle feature of semiquinone movement between these sites.

Key Words: Q-cycle, *b*-cycle, *bc* complexes; *bf* complexes; semiquinone; quinol oxidation; quinone reduction.

Introduction

Before introduction of the Q-cycle hypothesis (Mitchell, 1976), major problems existed in finding suitable proposals in chemiosmotic terms to explain both the observed proton/electron stoichiometry of the bc_1 complex and the unusual kinetic behavior of the redox components involved. The hypothesis has since led to a great deal of useful experimental endeavor. During this time, many modifications to the basic concept have been suggested and several of these have withstood the test of time and have produced "modified Q-cycle" schemes. The basic Q-cycle mechanism in its original and present forms is represented in Fig. 1. The essential features are two vectorial reaction sites for reaction with quinones and a transmembrane electron transfer role for the cytochromes *b*.

In addition to these developments have been the concurrent suggestions of a "*b*-cycle" mechanism of operation to explain the protonmotive and electron transfer properties of the complex (Wikstrom and Krab, 1980; Wikstrom *et al.*, 1981). At least in its original form, one essential feature of such a model was the involvement of the cytochromes *b* in a proton-pumping

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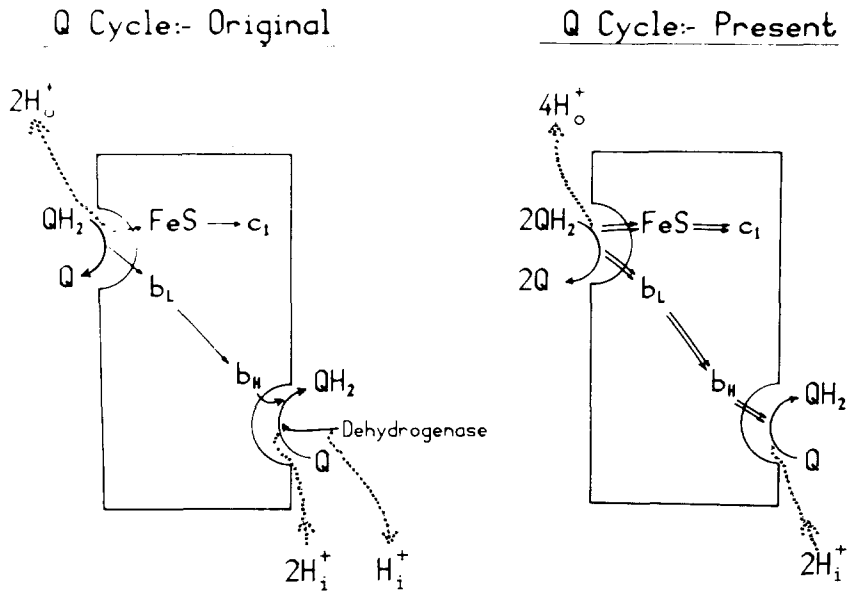

Fig. 1. Original and present forms of a Q-cycle. In all diagrams the top is the positive side of the membrane. The "o" and "i" sites are on the left and right of each representation. b_H and b_L, the high- and low-potential *b* hemes; FeS, the Rieske center. c_1, cytochrome c_1 or *f*. Subscripts o and i refer to the phases with which the species are associated (o = positive phase, i = negative phase). Types of transfer are: solid lines, electron transfer; dotted lines, proton transfer; dashed lines, semiquinone transfer. Possible protonations of the *b* hemes on reduction have not been included.

role of some sort, a postulate which was necessary since all reactions of the quinol/quinone system were postulated to occur in contact with the positive side of the membrane. A second essential feature was the ability of the semiquinone produced by quinol oxidation to act as an oxidant for cytochrome b_H. The latest version of the *b*-cycle scheme (Wikstrom and Saraste, 1984) has adopted a transmembrane location for reaction sites, hence removing the requirement for a "proton pump," while retaining the (now electrogenic) movement of semiquinone between these sites, as shown in Fig. 2.

It may be noted that the two types of model have tended to converge. In our own case new experimental data have caused us to adopt a working hypothesis which contains features implicit in original forms of the Q-cycle and *b*-cycle. A dominating feature of the model is still a transmembrane (at least in functional, if not in spatial, terms) pair of reaction sites, one of which oxidizes quinol and the other of which reduces quinone/semiquinone. It is therefore a derivative of the original "Q-cycle" proposal of protonmotive electron flow through the *bc* complexes. It allows, however, for the possible

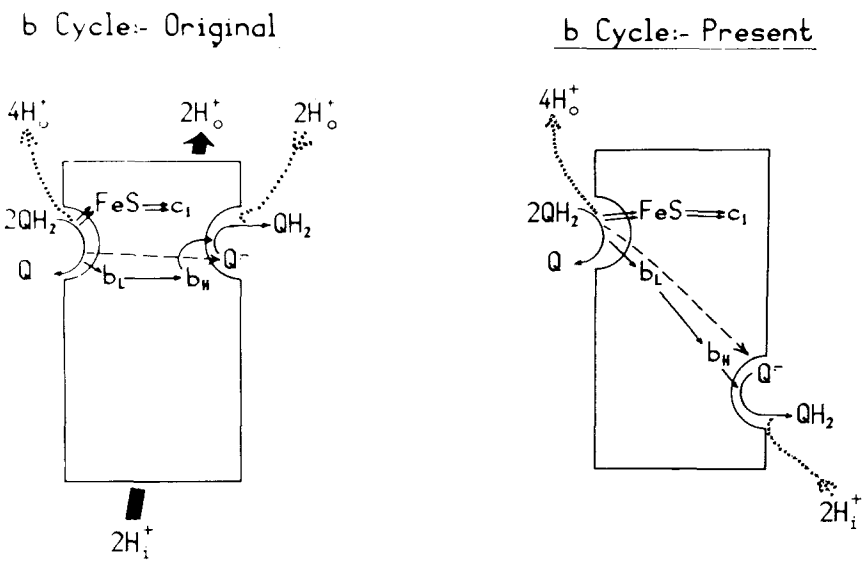

Fig. 2. Original and present forms of a *b*-cycle. See the legend to Fig. 1. For the original form, a proton pump is represented by a large arrow through the complex.

(electrogenic) movement of semiquinone between these sites and hence incorporates a central feature of the *b*-cycle schemes.

I intend to deal in this short report with several selected aspects only, chosen to emphasize how the above working hypothesis has been derived from recent experimental data, while attempting to avoid any substantial overlap with the other contributions in this issue.

Terminology

Central to this discussion is the occurrence and location of two reaction sites in the *bc* complexes by which quinol/quinone may interact with the redox components. The sites were termed the "o" site and the "i" site by Mitchell (1976)—the sites facing *outwards* to the positive side and *inwards* to the negative sides of the mitochondrial inner membrane respectively. This terminology has created some confusion especially in the chloroplast area where the positive side of the membrane is inside.

For consistency with previous literature, I intend to continue the use of these labels, but without their previous abbreviated meaning. The "o" site is defined as that at which quinol oxidation occurs, and the "i" site as that at which quinone/semiquinone reduction occurs.

The nomenclature of various redox and protonation states of quinones is well established and most easily described in the "scheme of squares" as depicted, for example, in Fig. 1 of Rich (1985).

Established Developments

Over the last ten years a number of key observations were made which have become well established and have contributed greatly to the ways in which both the original Q-cycle and *b*-cycle ideas have developed. These might be summarised as follows.

Identification of Two Sites for Redox Interaction of Quinone Species with the Complex

A major prediction of the Q-cycle model was that two distinct sites were available for the reaction with quinone redox couples. These are the centers "o" and "i" as described above. Probably the most convincing evidence for two such sites came with the identification of inhibitors which reacted independently and additively at these two sites. The mode of action and classification of these inhibitors have been described in detail recently (Von Jagow and Link, 1984). Their use has shown conclusively that two routes of cytochrome *b* reaction with quinone are possible and the extreme specificity of many of them has allowed detailed investigation of each route independently of the other.

Whether these two sites are vectorially arranged, and what the distance between them might be, is rather less clear. The data which are available, however, do tend to favor a transmembrane arrangement of sites:

(1) Extrapolations from primary sequence to tertiary structure for the cytochrome *b* polypeptide place the *b* hemes perpendicular to the membrane plane and transmembrane, with a center-to-center separation of around 20 Å (Saraste, 1984; Widger *et al.*, 1984). Since center "o" and center "i" inhibitors affect cytochrome b_L or b_H respectively, one might expect these centers to be similarly transmembrane;

(2) The ATP-induced midpoint potential shift of cytochrome *b* also supports transmembrane positions for the two *b* hemes (Mitchell, 1976);

(3) EPR studies of spin quenching of cytochrome *b* EPR signals originally placed both *b* hemes toward the outer face of the mitochondrion (Case and Leigh, 1976). However, more recent refinements of the technique have now produced data more in accord with their transmembrane locations (Ohnishi, personal communication);

(4) Electron transfer from Q_0 to cytochrome b_L in bacterial chromatophores is not electrogenic (Crofts, 1985). This contrasts with a contribution of around 40 and 60% of the electrogenic reaction from the $b \rightarrow b$ and $b \rightarrow Q_i$ steps respectively in both the chloroplast (Jones and Whitmarsh, 1985) and the chromatophore systems (Glaser and Crofts, 1984; Crofts, 1985);

(5) The positions of the redox centers of the iron–sulfur center and cytochrome c_1 (f) are in the positive phase (Hauska *et al.*, 1983). Since the iron–sulfur center forms at least part of the Q_0H_2 binding site (Trumpower, 1981), it is likely that the "o" site is also in the positive phase. In contrast, it would appear that cytochrome b_H is accessible (via the "i" site) only from the matrix (negative) surface of the mitochondrial membrane (Kunz and Konstantinov, 1984).

Overall, then, the presence of two transmembrane sites for reaction with quinones is very much favored experimentally. These sites are inherent in the Q-cycle formulation and are also included in the most recent b-cycle scheme. To the present author it is this feature, combined with a movement of charge in some way between the sites, which is the key feature of the energy-transducing model. With these, the required proton/electron stoichiometry of $2H_0^+/e$ (1 q/e) is automatically satisfied whatever the details of electron transfer mechanism at the two sites. Because of this, one might classify the Q-cycle and the recent b-cycle formulations as the same general type of model. Their previous distinction on the basis of energy transducing mechanism has vanished, and instead a distinction on the basis of detailed electron transfer mechanism (see below) might be made.

Oxidant-Induced Reduction of Cytochromes b and the Reaction at Center "o"

A key experimental observation which had been reported before the postulation of cyclic schemes was that of "oxidant-induced reduction" of the cytochromes b. In this effect the cytochromes b are rapidly reduced when a pulse of oxidant is added to oxidize via the cytochrome c_1, provided that a quinol donor for the complex is also present. An elegant explanation was originally offered by Wikstrom and Berden (1972) in terms of the two $n = 1$ redox couples of ubiquinone, one of which ($QH\cdot/Q$) operated at high and the other ($QH_2/QH\cdot$) at low potentials.

It was later realized that, given the extreme instability of the semiquinone species in the membrane or in the "o" site, this ordering should be reversed (Mitchell, 1976). By including the probability of a low pK of the semiquinone and the likelihood of protonation/deprotonation reactions being rapid at the "o" site, we arrive at the generally held view of reaction sequence of

$$QH_2 + FeS \longrightarrow Q\cdot^- + FeS^- + 2H_0^+ \quad (1)$$

$$Q\cdot^- + b_L \longrightarrow Q + b_L^- \quad (2)$$

This sequence is in accord with the midpoint potential value calculated for ubiquinone in aqueous solution of around +380 and −240 mV for the couples $QH_2/Q\cdot^-$ and $Q\cdot^-/Q$ respectively at pH 7 (using data from Rich and Bendall, 1980). Such values are close to those which should be operative at the "o" site, if the stabilization of the semiquinone is small and if the site is fully accessible to water. Any stabilization of semiquinone will bring these potentials closer by 60 mV for each decade of stability increase, as previously discussed (Mitchell, 1976). The observation that the semiquinone species can be observed kinetically but not in a redox poised system supports the notion of an unstable semiquinone at the site (DeVries *et al.*, 1981).

Such a reaction mechanism at this site has recently been questioned by a number of groups. Experimentally, rapid turnover experiments have shown that cytochrome *b* reduction can often be faster both in initial rate and in time constant than cytochrome c_1 (re)reduction (Selak and Whitmarsh, 1982; Peters *et al.*, 1984), a result which is not expected from the above scheme. More quantitative considerations, however, reveal several possible sources of experimental anomaly: cytochrome *b* reduction may occur at center "i" (although the anomaly can still be observed when center "i" is inhibited); cytochrome c_1 (*f*) reduction involves concomitant reduction of other components (FeS, *c*, plastocyanin, P700) so that the flux of reducing equivalents to the FeS (and hence the rate of $Q\cdot^-$ generation) is much higher than that calculated from cytochrome c_1 (*f*) reduction; less than one electron may appear on cytochrome *b* for each quinol being oxidized by FeS (possibly suggesting simultaneous reduction and oxidation processes for cytochrome *b*). It has been shown that these factors are the likely cause of the anomaly in bacterial photosynthetic systems (Crofts *et al.*, 1983), and recent quantitative simulations with the chloroplast system have revealed that these factors will cause such anomaly in this system also (Rich *et al.*, 1985).

A more fundamental objection to the mechanism has recently been raised by Hendler *et al.* (1985) who have suggested that the notion of formation of a low-potential semiquinone reductant of cytochrome *b* is not justified. Their arguments are based on the idea that equilibration of the semiquinone at center "o" with the quinone pool must occur rapidly and so will have an E_m (*sic*, but presumably intended to be an E_h) which is too high to significantly reduce the cytochromes *b*. There are, however, several rather questionable points in these deliberations which concern the interplay between kinetic and thermodynamic factors.

(1) Although binding of Q and QH_2 to center "o" is expected to be rapid, binding rate constants of semiquinone are expected and predicted to be slow. Occupation of the site with semiquinone will prevent rapid Q or QH_2 exchange. This does not invalidate the notion that semiquinone is only weakly stabilized—the semiquinone stability is kinetic rather than thermodynamic in origin.

(2) The exchange reactions (pp. 58 and 59) suggested to be a dismutation are not so—a disequilibrium amount of semiquinone will remain after any number of these exchanges. Only via a true dismutation can the expected equilibrium ratios of components be attained.

(3) The electron transfer process from quinol to the FeS/c_1 region must occur in two one-electron steps. The kinetic question therefore is not why an $n = 1$ reaction is preferred over an $n = 2$ reaction involving simultaneous reduction of the FeS and c_1. It is rather a question of why the reactive semiquinone preferentially reduces the b_L rather than reducing the FeS a second time. We have previously even suggested that a switching between these two possible routes may form the basis of an explanation of variable proton/electron stoichiometry in the chloroplast cytochrome *bf* complex, although there are no data to support such a model at present (Rich, 1984).

Because of the above arguments, there appear to be no compelling reasons at the present time to abandon the conventional "o" site reaction mechanism represented in Eqs. (1) and (2).

One point which has received little attention concerns the mechanism by which such a reaction is made to occur rapidly in the "o" site. I have previously reviewed our data which suggests that catalysis may involve a positive charge which promotes the electrochemically active anionic form of the quinol (Rich, 1984). There have been no further data on this problem and so the arguments will not be reiterated here.

Oxidation to Cytochromes b—the Reaction at Center "i"

In my previous review article, I suggested that cytochrome *b* reoxidation was the least clear step in the catalytic cycle (Rich, 1984). I still consider this to be the present situation. In the original Q-cycle schemes, the reoxidation of cytochrome *b* occurred as one of the two one-electron steps of reduction of quinone to quinol; the other one-electron reduction was provided by a dehydrogenase. It rapidly became clear, however, that such a scheme was inconsistent with many observations, for example, that the isolated bc_1 complex did not require any dehydrogenase component for full activity. A scheme in which the cytochrome b_H provided electrons for two successive one-electron reductions of quinone to fully reduced quinol was widely settled

upon (see Fig. 1). The quinone involved in this reaction was thought to be provided by the quinone pool:

$$b_H^- + Q_{pool} \longrightarrow b_H \cdot\dot{\cdot} \cdot Q$$

$$b_H \cdot\dot{\cdot} \cdot Q + b_L^- \longrightarrow b_H \cdot^{2-}\cdot\dot{\cdot} \cdot Q$$

$$b_H \cdot^{2-}\cdot\dot{\cdot} \cdot Q + 2H_i^+ \longrightarrow b_H + QH_2$$

Having two successive electron transfers from cytochrome b_H to different quinone couples has been an unattractive feature to some. It may be noted, however, that stabilization of the semiquinone by binding at the "i" site prevents either step being energetically unfavorable and so removes any thermodynamic objections which might be raised. Such stabilization at this site has been confirmed experimentally (Ohnishi and Trumpower, 1980). Indeed, a precedent for such a mechanism is provided by the secondary acceptor site, Q_B, of photosynthetic systems where a similar stabilization of semiquinone occurs (Wraight, 1979). In fact, there appears to be some similarity of inhibitor sensitivities between the "i" site and the Q_B site, which may further indicate a similarity of reaction mechanism.

The predictions of this Q-cycle model are that both quinone and semiquinone are rapid oxidants of the cytochrome b_H at center "i". This is in contrast to a *b*-cycle mechanism, where the only oxidant for cytochrome b_H is envisaged to be the semiquinone which is produced at the "o" site (Figs. 1 and 2). Some scrutiny of the experimental data on this point is warranted.

A number of experiments have certainly indicated that pool quinone can act as a rapid oxidant and pool quinol as a rapid reductant of cytochrome *b*.

(1) The rapid oxidation or reduction of the mitochondrial quinone pool by addition of fumarate or succinate, when myxothiazol has been added to prevent "o" site reactions, causes a rapid oxidation or reduction of the cytochrome b_H. These are sensitive to antimycin A and so are occurring via the "i" site (Von Jagow and Link, 1984).

(2) Recent experiments with bacterial chromatophores have suggested that the rates of reoxidation of cytochrome *b* after flash reduction are directly related to the amount of oxidized quinone in the pool, suggesting a second-order reaction for quinone oxidation of cytochrome b_H (Crofts *et al.*, 1983).

(3) In bacterial chromatophores under appropriate conditions, a rapid reduction of cytochrome b_H by flash-generated quinol can be observed to occur through the "i" site (Dutton *et al.*, 1984; Crofts, 1985).

Although the above support the Q-cycle formulation for reaction at this site in which quinone or quinol from the pool can react rapidly with cytochrome b_H, a number of further observations indicate that an oxidant produced by the "o" site may act as a preferential oxidant of the cytochrome b_H.

(1) If the operation of the bc_1 complex requires oxidized pool quinone, then one might expect an inhibition of its turnover when the quinone pool becomes highly reduced. This does not appear to be the case, and there have equally been no indications that the quinol–cytochrome *c* oxidoreductase activity of the complex is autocatalytic after addition of a pulse of quinol to start the reaction.

(2) Triphasic reduction kinetics of cytochrome *b* are observed on addition of a pulse of reductant to mitochondria or to isolated cytochrome bc_1 complex (Jin *et al.*, 1981; Rich, 1983). It has been noted that the second phase of cytochrome *b* reoxidation is greater than that expected by the redox state of the quinone pool—only during the third and slow reduction phase does the cytochrome *b* E_h approach that of the quinone pool. Additionally, equilibration with quinol through center "i" is faster if center "o" is made to be inoperative. These effects have been interpreted to indicate that the oxidant of cytochrome *b* in phase 2 is produced by center "o" but does not rapidly equilibrate with the quinone pool and in this way a transient oxidation of cytochrome *b* beyond the pool E_h is possible. This was envisaged as the production of a quinone at center "o" which had a favored (compared to pool Q) access to the "i" site, although from more recent developments the possibility that the species is a semiquinone should also be considered.

It would appear that the detailed mechanism of cytochrome *b* reoxidation at center "i" has yet to be resolved in detail, especially since it is this step which contributes most to the electrogenic reaction.

The Components of the Electrogenic Reaction

In a Q-cycle mechanism, the electrogenic reaction was originally suggested to be caused entirely by electron transfer from b_L to b_H. In the original forms of the *b*-cycle, the electrogenic event was presumably an electrogenic proton movement associated with the cyclic conformational changes of a "proton pump." Evidence that the dominant energy conservation occurred at the $b \rightarrow b$ step came from studies of protonmotive force-induced changes in relative redox poise of the cytochromes *b*, from structural predictions of the positions of the cytochrome *b* hemes, and from the fact that antimycin A abolished the electrogenic reaction associated with the bacterial cytochrome bc_1 complex (see above).

A number of developments have altered this view. In particular, a closer analysis of the effects of inhibitors of the slow electrochromic shift in chloroplasts and in chromatophores has indicated that a block in cytochrome *b* reoxidation abolished around 60% of this electrochromic shift. This has indicated that the majority of the electrogenic reaction is associated with cytochrome b_H reoxidation, rather than with its reduction by b_L. Further

studies have indicated that the reduction of b_L by Q_0 is nonelectrogenic, indicating that *b* to *b* electron transfer contributes the remaining 40% of the overall charge separation across the membrane (Jones and Whitmarsh, 1985; Crofts, 1985). The basic schemes have had to be altered only in detail to encompass these observations.

Perhaps a more serious question has been raised with experiments which have demonstrated an antimycin-sensitive cytochrome *b* oxidation under conditions where the cytochromes *b* are fully reduced at the outset of the experiment (Wikstrom and Saraste, 1984; Rich and Wikstrom, 1986). In a Q-cycle formulation, one might expect full prereduction of the cytochromes *b* to prevent formation of quinone at center "o" and so prevent formation of an oxidant which can act at center "i". This has led to the suggestion that the semiquinone produced at center "o" (even if the cytochromes *b* are fully reduced) might itself move to the "i" site to effect antimycin-sensitive cytochrome *b* reoxidation. Such a notion fits well with the idea that an oxidant produced by center "o" is the preferred oxidant at center "i" (see above).

In the present form of the *b*-cycle, such a reaction would constitute 50% of the electrogenic reactions—the first semiquinone reduces cytochrome b_H via b_L, as in a Q-cycle, but the second semiquinone produced by the "o" site moves electrogenically as $Q\cdot^-$ across the membrane to reoxidize the cytochrome b_H.

Accommodation of electrogenic $Q\cdot^-$ movement into the Q-cycle produces a more flexible scheme since it could allow for any percentage of the electrogenic charge movement occurring by this route, as recently noted by Mitchell (personal communication). Any balance of charge movement through the cytochromes *b* versus via a semiquinone species would still produce the required proton/electron stoichiometry. It might be pointed out that a model in which a semiquinone at center "o" shares its electron transiently with cytochrome b_L, but then moves electrogenically to center "i" and shares its electron with cytochrome b_H, is not at present experimentally distinguishable from the conventional route of cytochrome b_H reduction via b_L. In both cases the final result is the same, but in the former it is binding energy changes which are conserved as protonmotive energy whereas in the latter it is redox energy which is conserved.

A particular attraction of allowing the possibility of electrogenic semiquinone movement is that it provides in part an explanation for the observations of cytochromes *b* oxidation from the fully reduced state by oxidant pulses (see above) and for the observations that the redox state of cytochromes *b* in chloroplasts does not affect the kinetics of the slow electrochromic shift (Girvin and Cramer, 1984; Joliot and Joliot, 1985; Moss and Bendall, 1985). One further prediction, however, would be that the amplitude

of the slow phase in chloroplasts at low potentials might be up to twice that at higher potentials since in the former case both electrogenic semiquinone movement *and* electrogenic cytochrome *b* oxidation are expected. Joliot and Joliot (1985) have presented some evidence for a larger slow phase at low potentials, although this was not found by Girvin and Cramer (1984).

Even despite such difficulties, it remains to be established whether such a mechanism is ever operative outside the rather severe experimental conditions which are imposed in such experiments. This doubt is particularly pertinent since it is quite well established that electron transfer between the cytochromes *b* is indeed rapid in the *bc* complexes (Meinhardt and Crofts, 1983), although whether this is also the case for the chloroplast cytochrome *bf* complex has recently been questioned (Crofts, 1985). The demonstration of an electrogenic route via cytochromes *b* casts doubts on whether a second electrogenic route is necessary or possible. Nevertheless, the type of Q-cycle scheme outlined here represents a useful working hypothesis at the present for future experimental endeavor.

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