The Cell originated through Successive Outbreaks of Networking and Homing into Associations for the Mutual and Reciprocal Sharing of Advantages and of Disadvantages, between the Partners, with a Benefit only for their Wholeness. [1]

(Les Associations à Avantages et Inconvénients Réciproques et Partagés. L'origine endosyncénotique de la cellule : avantages et inconvénients partagés entre partenaires indissociables et bénéfice global pour le nouveau tout émergeant)

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Abstract : A living system is integrated into an ecoexotope.[2] The interactions between its ecoexotope and its endophysiotope shape its morphogenesis. In order "to survive" an organism has "to transform disadvantages into advantages" and "to avoid that advantages become disadvantages". Alone survived Associations for the Mutual and Reciprocal Sharing of Advantages and of Disadvantages[3], in which each parcener is both a guest and an hostage. They originated from the juxtaposition and the encasement of previous systems. Each guest must maintain its identity into the new wholeness, through the fitness between "the capacity of to be hosted" of its endophysiotope and "the capacity of hosting" of its ecoexotope. This is allowed only through the simultaneous losses by each protagonist of the capacity to destroy the other one(s). Their whole's outcome results from their simultaneous metamorphoses.

The comparison of the cell's organelles with the moneres and the study of the apoptosis, necrosis and tumourisation of the cell, make possible to resume the paradigm of the cell's phylogeny. The amalgamation, without membranes coalescing, of a population of side by side aggregated moneres, had made their endophysiotopes to become continuous, and was at the origin, of both the nuclear envelope and the endoplasmic reticulum. These new internal spaces were delimiting thus a continuous intermediary lumen, an ecophysiotope, which has played the role of a buffer between the ancient endophysiotope and the previous ecoexotope. It allowed a new network, the Golgi apparatus, to merge.

The first need, in order to survive, is "to eat and not to be eaten". Each organelle has its specific phages and descends from previous free-living moneres. The mitochondria and the plastids, which are delimited by two membranes, originated from the merging of Associations, for the Mutual and Reciprocal Sharing of Advantages and of Disadvantages, between preys and predators. The nude centrosome, which has the structure of a viral capsid, came from the melting of organisms with the same common membrane. The lysosomes and peroxisomes, with a single membrane, originated from the fusion of moneres having the same membrane interfaces.

Résumé : Tout organisme est indissociable de son écoexotope dans lequel il est accueilli. Sa survie et son ontogénèse résultent des interactions entre son écoexotope et son endophysiotope.[4] "Survivre c'est transformer des inconvénients en avantages et éviter que des avantages deviennent des inconvénients." Quel que soit le mode d'intégration d'un système vivant, seules survivent les Associations à Avantages et Inconvénients Réciproques et Partagés.[5] Leur mise en place émerge d'une adéquation entre la capacité d'être accueilli de l'endophysiotope et la capacité d'accueil de l'écoexotope. Elle est réalisée par la perte simultanée par chaque protagoniste de la capacité de détruire l'autre.

La mise en place de l'organisation spatio-temporelle de la cellule, sa cancérisation, son apoptose ou sa nécrose, son ontogénèse par comparaison avec celle des monères, permettent de retrouver sa phylogénèse. L'accolement, puis la fusion hyaloplasmique, mais sans fusion membranaire, de monères apparentées dont l'endophysotiope devient ainsi

[1] For the **Complementary Data (Tables and Figures)** and the **Supplementary References, quoted *** into the text, and for a more **detailed plan**, please do look at: **http://minilien.com/?MKOkk2v5Nv**

[2] BRICAGE P. (2002a) *The Evolutionary "Shuttle" of the Living Systems.*, 6 p. 5th ECSS (Heraklion, Creta, Greece). **http://www.afscet.asso.fr/resSystemica/Crete02/Bricage.pdf**

[3] BRICAGE P. (2002b) Only sustainable development can ensure both care of the environment and intra-generational equity., 1p. *Global Ethics for a Humane World*, UNO & IISD globus conference (Utrecht, Netherlands). **http://spitswww.uvt.nl/globus/morales/conference/second/contributions/bricage1.htm#bricage1**

[4] BRICAGE P. (2001) La nature de la décision dans la nature ? Systèmes biologiques: production, consommation, croissance et survie. Quelles règles ? Quels degrés d'exigence ? Quels bilans ?, 16 p. *La décision*. Coll. Afscet (Andé, France). **http://www.afscet.asso.fr/Decision.pdf**

[5] BRICAGE P. (2002c) Héritage génétique, héritage épigénétique et héritage environnemental: de la bactérie à l'homme, le transformisme, une systémique du vivant., 28 p. *Évolution du vivant & du social* Coll. Afscet (Andé, France). **http://www.afscet.asso.fr/heritage.pdf**

continu, est à l'origine, à la fois, de l'enveloppe nucléaire et du réticulum endoplasmique.[6] Leurs espaces internes, en continuité, délimitent un espace intermédiaire nouveau, qui joue le rôle d'un tampon entre le nouvel endophysiotope et le nouvel écoexotope: l'écophysiotope. La fusion résulte de leurs métamorphoses simultanées. L'appareil de Golgi, structure émergente unique, en constitue le réseau de coordination.

Survivre c'est "manger et ne pas être mangé" pour se survivre.[7] Tout organite descend de monères à vie libre avec ses phages spécifiques. Délimités par une double membrane, mitochondries et plastes ont pour origine la mise en place d'associations à avantages et inconvénients réciproques et partagés entre proies et prédateurs, par pertes simultanées par chacun de la capacité de détruire l'autre. Non délimité par une membrane, le centrosome, dont la structure est celle d'une capside virale, provient de la fusion d'organismes délimités par la même membrane. Organites à une seule membrane, lysosomes et peroxysomes proviennent de la fusion de monères délimitées par la même interface membranaire.[8]

1. ProKarya, the initial old Compartments[9]: Archaea and EuBacteria, the Moneres.

A MON is made of a single CPT limited by 1 MB. MONs are able to resist to difficult EXOs, if agglomerated. An hyperosmotic stress induces the acidification of their ENDO and their sporulation.

1a. The membrane, interface between endophysiotope and ecoexotope, and the wall.

The MB isolates from the EXO. Controlling the exchanges, it allows to avoid its DISs, while keeping its ADVs.* The W* doubles the MB and determines the form. Protoplasts, without W*, are very little resistant to osmotic shocks (what is a DIS), but their nude L deforms itself easily (what is an ADV). Aggregation or fusion between protoplasts* is triggered by a high pH, an osmotic shock or a VIR.* Gram- BACTs have one more MB containing POR.* Gram+ BACTs have only a 1X MB.* ARKs* do not sporulate. Only 1 PROT can control both the MB structure and the transfers of genes.*

1b. To survive it is "To eat and not to be eaten".

Ancient MONs survived from early organic compounds. Their first metamorphosis resulted from the depletion of nutriments and accumulation of waste products. BACT are eaten by phage VIRs.

1c. Emergence and level of organisation: the EoMonere, biodiversity and exaptation.*

Each reaction is reversible.* The same structure can have several functions.* The phenotype is changing with the EXO changes. With the time, the common becomes particular and the rare common.

Genes controlling metabolism and division of ARKs, BACTs and EUKs have a common ancestor (Figure 3).* ARKs* and MITs have cytochromes. ARKs and BACTs* have ferredoxines. ARKs, MITs and cBACTs have a common ancestor. The EoMON was anaerobic and survived in a hot reducing EXO. Its progeny survived by acquiring the capacity to use O2. BACTs merged in a milder EXO.* The lack of W, a DIS in an hypoosmotic EXO, allows also the ingestion of preys that is an ADV.* The entry of H2O, by increasing the volume, is a simple means to escape the ingestion by a predator.*

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[6] BRICAGE P. (2003) *Organisation, intégration et espace-temps des systèmes vivants*., 31 p. Coll. Afscet (Andé, France). **http://www.afscet.asso.fr/pbAnde03.pdf**

[7] BRICAGE P. (2000) *La Survie des Organismes Vivants*. Afscet Systémique & Biologie., 44 p. Fac. Médecine St Pères (Paris, France). **http://www.afscet.asso.fr/SURVIVRE.pdf**

[8] **Mots clés:** "capacité d'accueil", "capacité d'être accueilli", cellule, écoexotope, émergence, endophysiotope, métamorphose, mode d'intégration, monère, niveau d'organisation, ontogénèse et phylogénèse, virus, acteur "clé-de-voûte":
BRICAGE P. (2004) La gouvernance du vivant: les acteurs et les systèmes., 26 p. *La gouvernance*. Coll. Afscet (Andé, France). **http://www.afscet.asso.fr/pbAnde04GV.pdf**

[9] Terms in use: **abbreviations, definitions**, & **word roots** according to
CAILLEUX A. & KOMORN J. (1981) *Dictionnaire des Racines Scientifiques*., 263 p. CDU-SEDES (Paris, France).
Archaea (ARK) from the Greek: **archo** first, **archeo** primitive, **Bacterium (BACT)** from the Greek: **bacter** baton,
ecoexotope (EXO) from the Greek: **exo** external, **tope** space, **eco** of habitat, of welcome (BRICAGE P. 2002a),
endophysiotope (ENDO) from the Greek: **endo** internal, **tope** space, **physio** of that grows and reproduces (Figure 1.)
ecophysiotope (ECO): the space of interactions between the ecoEXOtope and the endoPHYSIOtope,
(eo)monere (MON) from the Greek: **mono** unit, **moner** the simplest (HAECKEL 1866), **eo** of the early beginning,
hyaloplasm & hyaloplasmic (HYA) from the Greek: **plasm** what is fashioned,
integration from the Greek: **integer** whole, that makes only one with its ecoexotope, that is inseparable from it,
Prokarya, prokaryotes (PROK), Eukarya, eukaryotes (EUK) from the Greek: **pro** before, **karyo** nucleus, **eu** true,
symbiotes: the joined partners in a symbiotic association **(symbiosis SYM), symbionte**: the whole, the association.
advantage: ADV, disadvantage: DIS, compartment: CPT

Without W, the hyperosmoticity of the EXO avoids the bursting and becomes an ADV. The acquisition of photosynthesis, metamorphosis that has allowed the surviving in an organic matter deprived EXO, is at the origin of cBACTs.[10] This huge ADV loads a huge DIS, the release of O2. Its accumulation has forced the MONs to escape in anoxic EXOs or to new metamorphoses for the utilisation of this toxic waste. The acetic fermentation[11] and oxidations* have transformed 2 DISs, alcohol and toxic O2, into 1 ADV.*

To survive (to grow) it is "to transform disadvantages into advantages" and "to avoid that advantages become disadvantages", in order to " survive itself" (to reproduce).*

2. Simultaneous merging of the nucleus and the reticulum: the cell, a network of moneres.

The CEL* is built with modules, limited by MBs, the organelles.* It merged from the fusion, without membranes coalescing, of a population of side by side aggregated moneres. The HYA, a reducing CPT, is their EXO. The today L contains a redox system, consumer of O2, which acidifies the HYA.*

2a. Organisation and integration: the cell is an endosyncoenosis.

The HYA is specialised in the metabolism of sugars without O2.* The PER is an ubiquitous and pluripotent MON (see 4).* The MIT (see 3b) is a specialised MON that converts lipids and sugars in ATP.* The cPLST (see 3c) is a MON that is specialised in sugars synthesis.* As MIT, it owns its nucleotides, proteins and lipids metabolisms. The NY is an emergent CPT (see 2c) that is specialised in nucleic acids synthesis and storage.* The ER and the GA (see 2d) are specialised in PROT metabolism.*

2b. Metabolism flows and membranes: the merging properties.

The inhabitant organelles are protected form VIR* by the HYA, their inhabited MON of welcome* in which the abundance of H2O favours the hydrolysis, not the dehydrating syntheses. The metabolic pathways are juxtaposed and encased in the same manner that CPTs that contain them are.[12]

2b1. The basic dynamics of predator/prey interactions.

The phagocytosis is an emergent process associate to the SQ, lacking in PROK.* The integration of the MIT into the CEL is posterior to SQ acquisition. Regularly, protagonists reply to an attack by a defence and to a stronger defence by an again stronger attack. The no-resolution of the conflict, "cause and consequence of the natural selection"*, starts an escalation of ripostes with a cost always greater.* How to end to a cheap stable steady state which is imperative to optimise the survival?

2b2. The control of proteins flows between the hyaloplasm and the organelles.

A PROT, which is aimed at an organelle, contains a signal of transportation and entry. A MB translocator controls its passage. A protease removes the signal, which is a source of AAs that allows the organelle to "eat" even in hyperosmoticity.* VIR PROTs are the same way sent to NY and MITs.

2b3. The internal membranes originated from moneres plasma membranes.*

MIT OMB and ER MB have the same lipid contents.* NY MB and ER MB have a common ancestor, GA MB and ER MB also, MIT IMB and L of Gram- BACTs have another one, LYS MB and EUK L have a common ancestor.* NY IMB and EUK L* have the same properties of endocytosis, MIT IMB and VAC MB have the same ATPases VIR receptors* EUK L, ER MB, and NY OMB carry the same ATP dependent proteases, absent of cPLST and MIT OMBs (Tables* A1 & B1)

2c. The nucleus: the actors and the system.*

[10] Other abbreviations of terms in use:
cellular level: apoptosis/tic **APO**, cell/cellular **CEL**, centrosome **CENT**, Cyanobacteria **cBACTs**, cytoskeleton **SQ**, endosome **END**, envelope **ENV**, Golgi apparatus **GA**, glyoxysome **GLY**, lysosome **LYS** , stroma **MAT**, membrane **MB**, outer MB **OMB**, inner MB **IMB**, single membrane **1X MB**, double membrane **2X MB**, membrane of sequestration **SQMB**, mitochondria **MITs**, nucleus/ar **NY**, nucleolus/ar **NU**, skeletal wall **W**, peroxysome **PER**, plasma membrane **L**, vacuolar membrane **T**, microsome **MICR**, microtubule **TUB**, phagosome **PHAG**, plastid **PLST**, amyloplaste **aPLST**, chloroplast **cPLST**, perforine **PERF**, porine **POR**, aquaporine **aPOR**, endoplasmic reticulum **ER** (smooth **LER** or granular **RER**), thylakoïd **THYL**, vacuole/ar **VAC**, virus/al **VIR**
molecular level: amino acid **AA**, calcium **Ca++**, carbon dioxide gas **CO2**, electron **e-**, enzyme/atic **ENZ** (**CAT** catalase, **OXY** oxidase, **PEROX** peroxidase, SuperOxydeDismutase **SOD**), proton(s) **H+**, water **H2O**, hydrogen peroxide **H2O2**, dioxygen gas **O2**, protein/aceous **PROT**.

[11] TORTORA G.J. & al. (2004) *Microbiology*., 924 p. Pearson (San Francisco, USA).

[12] De DUVE C. (1987) *Une visite guidée de la cellule vivante*., 437 p. De Boeck Université (Bruxelles, Belgique).*

Containing at least 1 NU, that can reach the size of a MON, the NY is delimited with a 2X MB pierced with pores. Each pore is a place of junction between OMB and IMB and a place of exchanges between NY ENDO and HYA ENDO. With its symmetry and dimensions it looks like a VIR capsid.

2c1. The membrane interface between endophysiotope and ecoexotope.

The NY OMB is in continuity with the ER MBs.* The NY MBs and the L are homologous.

The periNY and intraER spaces are equivalent.* They built an interactive space in continuity between the EXO and the ENDO, the ECO, which is both more and less than the ENDO and EXO.

The common origin of NY IMB and L, and of NY OMB, ER MBs and L is proved by their reassembly from the ER[13] after mitosis.* They carry the same VIR domains (Table B2*). The NY OMB and L behave the same manner during the HIV induced fusion.* The NY pores are interMB specialised opened[14] junctions, through which actin[15] filaments extend. The NY and HYA contain the same SQ. TUBs are attached to the L and to the NY OMB. Nevertheless, the interface is mobile. It is a slipping surface that allows the NY, coated with its IMB alone, to turn on itself. The pores can be broken and rebuilt. The intraER space is a slipping surface also (Figure 2a). The simultaneous emergence of the central NY and the outlying ER results from a position effect within an aggregate of preARKs.

2c2. Like the cell, the nucleus is also a polymonere endosyncoenosis.

The NY of the Echinides' coelomocytes* contains inclusions of MON type: - organising centres with a 2X MB, a PROT canvas, crystalline like a PER, containing RNA, that migrate between NY and HYA, - globules analogous to the proMITs, forming in the NY and migrating to the HYA, by dilations of the 2X MB NY ENV. The NU with its RNA nucleoid and its plasmatic heterogeneity, has also a PROK organisation, but without MB.* It happens as if the NU was an eomonere with an RNA genome and the NY a polyeomonere endosyncoenosis, with its own phages*, initially of the RNA sort.[16]

2d. Endoplasmic Reticulum, Golgi Apparatus and Centrosome are indivisible.

The ER represents 50 % of the cell's MB surface. Like the MIT, it insures the sequestration of Ca++ and detoxication processes through OXY. It surrounds, and isolates from the ENDO, the senescent organelles, in digestive VACs. The GA and ER*, specialised parts* of the ECO, have the same role of convergence and distribution[17] like the interMB space of Gram-. The GA grows by extension and fission of a previous GA. During the fusion of 2 CELs, the GAs fuse in only 1 structure, as CENTs do.[18] The NY OMB, ER and GA have the same dynamics.* The GA, PER and CENT may be formed de novo, while being self-dividing. Their "enclosed dangers"* reveal the origin of the organelles.* Each one has its own phages. VIRs built and bud on NY IMB, ER MB and GA. A unique PROT allows the MB fusion of VIRs and the fusion of CELs into a syncytium with only 1 L*, this indicating the common origin of these MBs. There are so much potential hosts for a VIR that CPTs are (Table C).* The integrity of GA, CENT and NY is depending on the SQ.[19] Their movements and the maintenance of the shape and movement of the CEL is the duty of actin or tubulin filaments. The TUBs are formed of PROT fibres arranged in a repeated motive delimiting an internal canal. This topology is that of a VIR, like the tobacco mosaic VIR. The CENT resembles a VIR with 2 nude empty capsides.*

3. The Actors and the System: About the Origin of the Mitochondria and Plastids.

MITs and PLSTs possess a genome. Their OMB and IMB contain the same PROTs, but more concentrated in the "simplified" OMBs, specialised in the control of H2O and solutes flows.

3a. The membrane, interface of confrontation and reciprocal constrained exchanges.

In anaerobiosis, the ADV of the absorption of sugars by the BACT* L or CEL* L, is associated with a DIS, an entering flow of deleterious H+. the excretion of which is ATP consuming.

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[13] BURKE B. & ELLENBERG J. (2002) *Remodelling the walls of the nucleus.*, p. 487-97. Nature Rev. Mol. Cell Biol. n° 3.
[14] HOELZ A. & BLOBEL G. (2004) *Cell biology: popping out of the nucleus.*, p. 815-6. Nature n° 432.
[15] LÉVY N. & CAU P. (2003) *Anomalies du noyau et maladies génétiques.*, p. 50-6. Pour La Science n° 313.
[16] Do look at: **http://minilien.com/?MKOkk2v5Nv** and at **http://www.minilien.com/?LUeZbdsNCH**
[17] SMALLRIDGE R. (2002) *Endocytosis. A new recycling route.*, p 315. Nature Rev. Mol. Cell Biol. n° 3.
[18] PELLETIER L. & al. (2002) *Golgi biogenesis in Toxoplasma gondii.*, p. 548-52. Nature n° 418.
[19] CAMERA P. & al. (2003) *Citron-N is a neuronal Rho-associated protein involved in Golgi organization through actin cytoskeleton regulation.*, p. 1071-8. Nature Cell Biol. n° 5.

3a1. The mitochondrion is an hostage trapped into the hyaloplasm.

The LYS, END or MIT pH regulative ATPases/synthases are always turned inwards the ENDO. They have the same reversible functioning, whatever is the MB.* Alone factors of regulation or modulation differ. But, if the Ca++ATPase of the L ejects outside in the EXO the toxic Ca++, that of the MIT is forced to pump it in its own ENDO.* That is a DIS. But, in the presence of O2, the excretion of H+ is linked with the IMB chain of e- transport and allows the synthesis of ATP. 2 DISs become 1 ADV. To survive in oxygenated and acidic EXO, alone the IMB is necessary. To what serves the OMB ? The MIT ATP is exchanged against carbohydrates, lipids and PROT from the HYA. The prePROTs entry, through sites of contact between OMB and IMB equalling to NY pores, and their activation is ATP consuming, but this DIS is compensated by the ADV of the digestion of PROTs cleaved part.

PROTs analysis indicates a kinship between IMB and Gram-[20] L, or OMB and EUK L.

3a2. The origin of the Outer Membrane: an attack/defence balance system.

During the sequestration process, the future host produces PERFs against the L of the future inhabitant and the future inhabitant produces PERFs against the L of the future inhabited. The SQMB becomes the containing BACT PORs OMB of a future organelle.* The IMB contains PORs but not BACT ones.* PORs allow the transfer of DNA* of the lodged host to the lodging one. In the NY genome are coded ORFs* of EUK origin (of the ancient system of attack/defence against a PROK) for the MIT IMB, and ORFs of PROK origin (of the ancient system of attack/defence against an EUK) for the MIT OMB. The resulting PROTs are joined in a unique hybrid one* that contains the signal for the IMB of the lodged, and that for the OMB of the inhabited.* The LER MB and MIT OMB are identical .The MIT IMB and BACT L react the same to osmotic shocks.[21] The lipids compositions of BACT OMB and MIT OMB are different. The MIT OM is a SQMB with an original periMON exchange system.* When there is a 2X MB, the OMB is a SQMB and the IMB is equivalent to L.

3a3. Porines* and Perforines: the Attack and Defence Molecules.

The hyperosmoticity accelerates H+ excretion. The acidity favours the absorption.* A poor watered hyperacid ECO allows the exit of H2O, the entry of sugars and the synthesis of ATP. It is like that the MIT is constrained to work by the HYA!* The universal presence of PORs* and of PROT domains intervening both in the adressage and protection of PROTs and CELs division*, proves that all MBs have a common MB ancestor and that the pleiotropy is the motor of exaptation mechanisms. POR genes are expressed preferentially in strong or weak osmolarity EXOs.* Always excreted, PORs are present in the OMBs of Gram- BACTs, MITs and cPLSTs, but also in EUK L and ER MB. The OMBs of GLYs, PERs and MITs have the same PORs.* The MIT OMB, whose alteration turns the APO on, contains the same PORs that the L. APO PROTs (of the HIV and the VHB*) or antiAPO ones (of the myxoma VIR) reacts with this type of PORs. Besides their role in the hydric status in Gram- BACTs, PORs* intervene in adherence and invasion mechanisms.* Their synthesis increases in anaerobiosis, The OmpF POR intervenes both in the structure of pores and the hydrolysis of RNA and DNA.* The lymphocytes kill BACTs, or VIR infected CELs, by releasing PERFs* that form open pores in BACT or CEL MBs.* The APO is triggered by PERFs.[22]* Reciprocally, VIRs*, especially these that induce syncytia*, or BACTs*, produce PERFs that kill their targets by using PORs or/and by playing the role of PORs.* The Bcl APO factors are PERFs.

3a4. The origin of the membranes*: modularity, pleiotropy* and exaptation.*

The ER and L carry the same ATPases. The MIT OMB and L carry the same channels of Ca++ exchange. The aPORs*, H2O channels located in Gram- BACT OMB and MIT OMB, intervene in the intraCEL adherence and the APO. Structures show the common origin of L and MIT IMB, and that MIT OMB is a true SQMB. But the L has been modified as the MB at the origin of the Gram- BACT OMB has been. The early endosymbiote did not resemble a current PROK and no more an EUK.* The modularity creates the variety with a light genetic or phenotypic load. A PROT is an assemblage of juxtaposed and encased domains, that can be present in different PROTs.* Mistaken replication, transcription and translation, decrease the output in functional PROTs, it is a DIS. But that allows the appearance of new PROTs, with an added or removed domain*, what can be an ADV. All CEL is a mosaic of juxtaposed and encased organelles, with limited life durations, and that are continuously recycled and renewed. In the HYA,

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[20] SANCHEZ-PULIDO L. & al. (2003) *POTRA: a conserved domain in the FtsQ family and a class of β-barrel outer membrane proteins.*, p. 523-6. TRENDS Biochem. Sci. n° 28.

[21] MÜELLER A. (2002) *VDAC and the bacterial porin PorB of Neisseria gonorrhoeae share mitochondrial import pathways.*, p. 1916-29. EMBO J. n° 21.

[22] KELLY J.M. & al. (2004) *Granzyme M Mediates a Novel Form of Perforin-dependent Cell Death.*, p. 22236-42. J. Biol. Chem. n° 279.

there is a competition between RNAs for nucleotides, a competition between PROTs for AAs, a competition between the DNA and PROTs for nucleotides, a competition between RNAs for the integration* into the DNA, a competition between RNAs and DNA... The local competition between the parts is eliminated through the global assistance to the survival of their whole.

3b. The mitochondria.*

A MIT is a MON with a 2X MB. Mobile, it changes of form, grows and divides in the HYA.

3b1. Endophysiotope and ecoexotope: proteome and genome, membranes.

The MIT IMB and BACT L have the same ATPase/synthase complex. The permeable OMB allows with solutes from the HYA, the recreation into the interMB space of the liquid EXO of the BACT ancestor. The selective permeability of the IMB, its PROT hyperconcentration, its crests, are totality explicable as the consequence of a plasmolysis (Table F*).

This dehydration is at the origin of the production of both water and ATP* (to control the tender flows of metabolites against the osmotic gradient). The MAT[23] is the halophile hyperoxic ENDO of a plasmolysed sequestered BACT protoplast that survives, in an acidic EXO, with the H2O producing respiratory chain to struggle against hyperosmolarity, and with ATP for its metabolic surviving trades with HYA. It happens as if foods combined with O2, but in fact they combine with H2O: it is a cold combustion or a hydrolysis without H2O! The hyperoxic MIT ENDO allows the survival of the HYA that is maintained free of toxic O2. The integration of the cPLST has consolidated the role of the MIT, in the same manner that the integration of the MIT had given a new role to the PER. The functional structure of the IMB is the cause and the consequence of pH changes.* The e- flows are similar to those of anoxygenic photosynthetic BACT.* The interMB space is the same as the periplasm interMB space of Gram- BACTs.* The MIT originates from an aerobic photosynthetic BACT (or a parasite* BACT) which was a sweet water MON trapped in a hyperosmotic EXO.*

Besides the aPORs of the OMB and the ATPases of the IMB, a glutathion PEROX as in the cPLST, a PROT of the PER MB, at least a clathrin of the L, all elements of the genome of retroVIRs, and transposons are present. Imported PROTs for the IMB or the MAT* indicate an hypotonic acidic ancestral ENDO.* The PROTs addressed to OMB or L have the same hydrophobic signal.[24] PROTs without signal are excreted in the space of sequestration. Transfers of genes take place between MITs and NY and between cPLSTs and MITs.* During fusions between PLSTs and MITs* the MIT morphology is changing, MITs become turgid, without crest. The MIT genome[25] contains genes, introns*, plasmides, coming from VIRs, NY or other organelles (Table E*).

3b2. An Association for the Mutual Sharing of Advantages and DisAdvantages.

The MITs eat the waste products of the HYA and provide back useful products to the cPLSTs (H2O, CO2) and to the PER (urea, ammonia). MIT stocks Ca++, whose content has to be maintained very low in the HYA. The haem synthesis for building the haemoPROTs of MITs, HYA and PERs, begins in the MIT continues in the HYA and ends in the MIT.* "The survival of one, or of the whole, has a prerequisite that all the other ones survive, and reciprocally." The MIT IMB hyperactivity[26] provokes the CEL APO. HYA proteases destroy the NY and MITs, foreigner endosymbiotes hosted by the HYA. It is the reject of the no-self. "In order that the wholeness (the symbionte) survives, it is necessary first that each parcener (the symbiotes) survives." The OMB is the SQMB through which the CEL has manipulated the MIT ancestor and has been manipulated by it.[27] In symbiosis, all what is an advantage to one parcener is a disadvantage for the other ones and reciprocally.* The MIT looks like a thermoacidophile ARK that survives to low temperatures in the CEL.*

The MB of enveloped VIRs is like an IMB.[28] A VIR PROT inserts into the OMB and induces, as a PERF does, the MIT lysis.[29] A VIR PROT recognises[30] the IMB. The MIT is the EXO for the VIR DNA

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[23] For a review look at : **http://www.els.net/ doi:10.1038/npg.els.0001381 ***

[24] REHLING P. & al. (2003) *Protein insertion into the mitochondrial inner membrane by a twin-pore translocase*., p. 1747-51. Science n° 299.

[25] CLIFTON S.W. & al. (2004) *Sequence and Comparative Analysis of the Maize NB Mitochondrial Genome*., p. 3486-503, Plant Physiol. n° 136.

[26] SANCHEZ-ALCAZAR J.A. & al. (2000) *Increased mitochondrial cytochrome c levels and mitochondrial hyper-polarization precede campothecin-induced apoptosis in Jurkat cells*., p. 1090-100. Cell death & Differentiation n° 7.

[27] BOUCROT E. & al. (2005) *The Intracellular Fate of Salmonella Depends on the Recruitment of Kinesin*., p. 1174-8. Science n° 5725.

[28] COCKBURN J.J.B. & al. (2004) *Membrane structure and interactions with protein and DNA in bacteriophage PRD1*., p. 122-5. Nature n° 432.

replication.[31] The MIT has its own phages (Table G*). The SQMB protects it from VIRs (Table D*). The genome of the TomboVIRs codes for an adsorption PROT* that inserts into the OMB with an anchorage domain into the IMB. This PROT is a module of a complex of survival of VIRs* and MITs[32], that prevent the APO. The MIT is the specific host of VIRs, independently of the host of the MIT, so a plant VIR can infect an animal.* The animal CytomegaloVIRs and plant PotyVIRs have now 2 hosts: 1 old host of adsorption (the MIT) and 1 new host of integration of the genome (the NY). The NodaVIRs, MIT phages with RNA genome, possess a transMB PROT of adsorption on the MIT ENV. The synthesis of their RNA takes place in the MAT and they bud in the interMB space.[33]* VIRs with a RNA1+ genome* recognise both ER MB, L, T, PER MB and MIT OMB, the proof of the common origin of MBs and of the antiquity of VIRs.* The ENDO of eoMONs was a RNA's world.

The APO by a VIR[34] results of a positive feedback[35] between ER and MIT IMB. The HYA releases cofilin, a toxic for MITs. In return MITs release cytochrome c that is toxic for the HYA.[36] The SQMB rupture triggers the partition of the association.* PROTs preventing the MBs lysis must continuously maintained the survival of MITs. VIRs, which divert the HYA PROT synthesis and suppress this maintenance*, must restored it in order that the CEL may survive to allow the VIRs to survive itself.* During APO the defence system of the lodging host turns on and acts against the lodged host. The ER[37] induces the MIT lysis, whose death induces that of the CEL.* The one can not survive in destroying the other one.* The APO has to be continuously prevented.[38] In order that the one survives, it is necessary first that the other ones survive: it is a synallagmatic agreement*, with mutualism and subsidy.*

3b3. The Origins of Mitochondria and Gram- Bacteria? * (Figures 2b, 2c & 3)*

The MIT OMBs fuse themselves and are in continuity with the L as if it was a reverse endocytosis.* During the struggle before a CEL organelle merges, the association could have evolved backward, with the disappearance of the CEL HYA ending to a MON with a 2X MB: a Gram- BACT.

3c. The Plastids.

A cPLST may have the size of a CEL. It looks like a cBACT of the genera Synechocystis. Like the MIT it has been acquired by endocytosis.* To the opposite of the MIT, it is rich in glycolipides.

3c1. The three types of membranes of the chloroplast.

Like in the MIT a permeable OMB and a selectively permeable IMB delimit an interMB space. The IMB neither contains an e- transportation chain-, or an ATPase. They are fixed on saccules surrounded by the IMB, the THYLs. Into the MAT, they form a network equalling to ER and GA. THYLs are isolated MIT crests, but inverted after a lysis. The intraTHYL space and the MIT interMB space are identical. The OMB fuse with the ER MB, the NY OMB, or other cPLST or MIT OMBs.

3c2. Mitochondria and chloroplasts are working in constrained reverse ways.

As for the MIT respiratory chain, the THYL photosystems result from the juxtaposition and encasement of PROK OXPHOS units* into a supramolecular structure allowing "constrained tender flows".

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[29] CHANTURIYA A.N. (2004) *PB1-F2, an Influenza A Virus-Encoded Proapoptotic Mitochondrial Protein, Creates Variably Sized Pores in Planar Lipid Membranes*., p. 6304-12. J. Virol. n° 78.

[30] GIBBS J.S. & al. (2003) *The Influenza A Virus PB1-F2 Protein Targets the Inner Mitochondrial Membrane via a Predicted Basic Amphipathic Helix That Disrupts Mitochondrial Function*., p. 7214-24. J. Virol. n° 77.

[31] RIEDINGER H.J. & al. (2005) *Replication of simian virus 40 (SV40) DNA in virus-infected CV1 cells selectively permeabilized for small molecules by Staphylococcus aureus a-toxin: Involvement of mitochondria in the fast 02-dependent regulation of SV40 DNA replication*., p. 557-66. Biochem. J. n° 386.

[32] EVERETT H. & al. (2002) *The Myxoma Poxvirus Protein, M11L, Prevents Apoptosis by Direct Interaction with the Mitochondrial Permeability transition Pore*., p. 1127-40. J. Experiment. Medicine n° 196.

[33] GUO Y.X. & al. (2004) *Membrane Association of greasy Grouper Nervous Necrosis Virus Protein A and Characterization of Its Mitochondrial Localization targeting Signal*., p. 6498-508., J. Virol. n° 78.

[34] CATTEAU A. & al. (2003) *Expression of dengue ApoptoM sequence results in disruption of mitochondrial potential and caspase activation*., p. 789-93. Biochimie n° 85.

[35] BOEHNING D. & al. (2003) *Cytochrome c binds to inositol (1,4,5) trisphophate receptors, amplifying calcium-dependent apoptosis*., p. 1051-61. Nature Cell Biol. n° 5.

[36] CHUA B.T. & al. (2003) *Mitochondrial translocation of cofilin is an early step in apoptosis induction*., p. 1083-9. Nature Cell Biol. n° 5.

[37] GERMAIN M. & al. (2005) *Endoplasmic reticulum BIK initiates DRP1-regulated remodelling of mitochondrial cristae during apoptosis*., p. 1546-6. EMBO J. n° 24.

[38] KONG M. & al. (2004) *The PP2A-Associated Protein α4 Is an Essential Inhibitor of Apoptosis*., p. 695-8. Science n° 5696.

But, metabolic flows are in opposite: the MITs produce H2O and consume NADH, the cPLSTs consume H2O and produce NADPH. The osmotic properties are in opposite. During the cPLSTs extraction*, an osmotic shock breaks the 2X MB but not the THYLs. Only a very hypoosmotic shock does break THYLs. The plasmolysed MIT is specialised in H2O export.* In an hydrophobe* EXO the turgid cPLST is specialised in H2O import. It is an hydrolytic CPT, but it works in condensation.

3c3. Does the chloroplast merge from an EndoSynCoenosis ?*

OMB and IMB have similar translocators.* OMB, L and T have similar aPORs.* MIT IMB, cPLST IMB and GLY MB resemble by their carriers and ENZs of the lipid metabolism. The proteome indicates a kinship between MAT and THYL. PROTs resemble those of Rhodobacter capsulatus.* The cBACTs, contrarily to the cPLSTs, do not have site of adhesion between OMB and IMB. Their interMB space is more greater.* The Mg++ dependent ATPase of the IMB is insensitive to oligomycine and controlled by calmodulin and Ca++ (as that of the ER), it is also K+ insensitive (contrarily to that of L) but that of THYLs is different.* The IMB resembles THYLs, MIT IMB and the ER., but differs from an OMB. The OMB is not a BACT OMB, it really is a SQMB. And the IMB delimited CPT is born from a colony of eaten MONs that reorganised in a new whole intermediary between MONs and EUKs.

3c4. A Balance of Interactions between What Sort of Partners? (Figure 3)

cBACTs and cPLSTs have the same chlorophylls. The sequencage of their genomes has proved their kinship.* But, the OMB and IMB contain channels of nonphotosynthetic pathogen BACTs and receptors and channels of nonPROK origin. The OMB, SQMB of EUK origin, contains a POR which is a BACT cytolysis turning APO on, an EUK ATP POR, and a POR of intermediate origin (Figure 2d). The genes of the rRNAs have the same location in the genomes of the cPLST, Escherichia coli (a Gram- BACT) and Anacystis nidulans (a cBACT).* The tRNA genes of the cPLST contain coding introns as those of MITs and ARKs. The bicatenar circular DNA of Chlamydomonas[39] contains VIR inverted complementary sequences, whose transcription is controlled by antisens RNA.* In cPLST and Rhizobium the metabolism of sugars is linked to the adaptation to osmotic shocks and the nitrogenous metabolism to adaptation to thermal shocks.* The THYLs and L have the same behaviour to cold.* The interMB space is not a Gram- BACT periplasm, but an interface with a new EXO, the ECO. Granar vesicles may be born from side reversal aggregation of lysed cBACT IMBs, after a thermal shock, in a thermosensible mutant of a thermophile BACT (as Anacystis), or following a VIR lysis?

3c5. In what a sort of Whole are Costs and Profits mutually and reciprocally shared?

PROTs of RNA VIRs are formed only with the use of the cPLST genetic code.* The TymoVIRs have a 2X or a 1X MB, formed from the cPLST MBs.* The fact that their VIR EXO is both the NY and the cPLST results from the transfer of the cPLST genome into the NY and is due to the fact that the early VIR EXO was the cPLST ENDO. Their replicase belongs to an adsorption complex onto the PLST.[40] The photosynthesis* is indispensable to their development. They do exist as complete VIR only in the common EXO of NY and cPLSTs, the HYA. As the NodaVIRs they merged from a common ancestor of cPLSTs and MITs. Vesicles containing the replicative form of ClosteroVIRs appear by PLSTs lysis.* The cPLST is the prey of specific phages.* Soon or late, cPLSTs are lysed like Rhizobium bacteroids are, by a HYA induced autolysis. The HYA is a predator of cPLSTs and MITs.[41]

The cPLST is the CPT for the reduction of nitrites and sulphates, for the synthesis of AAs. The nitrites come from the HYA reduction of absorbed nitrates, because it is maintained at a reducing state thanks to PERs and MITs detoxication of O2. Along the daily light/darkness cycle, cPLSTs and MITs, through their opposite variations of ATP production, maintain a constant HYA "energy content".* Each parcener will survive only if the other ones and their wholeness first survive.

4. The Single-Membraned Organelles: Polymorphism, Pleiotropy, and their Origin's Unity.

A 1X MB limits the PER.* That MB resembles L, ER and MIT IMB, as predicted by the model of the origin of the EUK CEL (see 2). The PER volume* is adjusted to the O2 content of its EXO.

4a. The diverse phenotypes of Peroxisome: pleiotropy and metamorphoses.*

[39] MAUL J.E. & al. (2002) *The Chlamydomonas reinhardtii Plastid Chromosome*., p. 2659-79. Plant Cell n° 14.
[40] PROD'HOMME & al. (2003) T*argeting of the Turnip Yellow Mosaic Virus 66K Replication Protein to the Chloroplast Envelope Is Mediated by the 140K Protein*., p. 9124-35. J. Virol. n° 77.
[41] The CEL endosyncoenosis is identical to the lichen one; the lodging host soon or late eats the lodged hosts.

Depending on EXO changes, the PER ENDO contains ENZs of AAs and purines metabolism like a MON, ENZs of ß-oxidation of lipids like a MIT, ENZs of cholesterol synthesis as in LER and it works in neoglucogenesis, as the HYA. But it is the alone CPT of H2O2 detoxication. By reverse functioning it becomes an CPT of defence and attack (producing H2O2) and even of regulation (producing NO*). It allows both, the resistance to O2, the utilisation of fermentation's wastes* and the resistance to high temperatures. The PER is a keystone actor of the CEL.* As with the MIT, its flows are constrained by hyperosmolarity*, it produces H2O* but it can not use it, except under its LYS avatar.* PER, cPLST and MIT have 1 common ancestor.* The PER merged from a hyperhalophile HaloARK whose genome coding for detoxication ENZs is today in the NY genome.* As its ancestral ARK, it can eat sugars.*

The various MICRs are avatars of the polymorph[42] PER. The ontogenesis of PERs, ENDs and LYSs is the same.* Depending on the HYA EXO changes*, the PER (that allows the CEL not to be eaten) develops or metamorphoses into GLY, glycosome[43], nitrogenosome, uricosome, magnetosome, liposome, or LYS (that allows the HYA to eat). The PER merged from a pluripotent* and ubiquitous MON, whose pleiotropy[44] permitted both the PER integration and the CEL exaptation.

The PER is a lodged but not sequestered MON, at the cross of evolutionary paths.

4b. The peroxisome genesis: sequestration, half-autonomy and the origin of Gram-.

The PER does not own a nucleoid, or a transcription-translation system. With at least 224 ORF genes[45], its genome is fully integrated in the NY and is highly preserved from an organism to an other.

One of its PROTs* stimulates its growth, an other one inhibits it.* More than 20 ones control* its cycle of development.[46] Previous PERs by division[47] give new PERs. It is an half-autonomous organelle (Table I). Its adressage signal is present in PROTs of MONs and EUK VIRs.[48] The PPARs, that control PERs proliferation, act in the regulation of the hepatitis B VIR cycle.* All TomboVIRs recognise[49] the PER MB where they bud. [50] The lipid metabolism of PERs is diverted by RNA VIRs for their ontogenesis.* The PER has its specific phages, dangers that are contained by the endoSYM association. During the transfer of the PER genome to the NY one, these constrained dangers may have been free. The association has been able to evolve back to the disappearance of the HYA partner, giving birth to a new free PROK with a 2X MB, the ancestor of Gram- BACTs (Figure 3).

cPLSTs* show such a regression of the CPT of welcome to the benefit of the welcomed CPT.

4c. The survival through the reciprocal sharing of advantages and disadvantages.

In plant CELs the narrow contacts between PERs, cPLSTs and MITs allowed the photorespiratory glyoxylate cycle to merge. This defence against the O2 excess has a cost in sugars. It is a DIS. But the transformation of these sugars in AAs is a huge ADV. cPLSTs provide the raw material, which PERs transform into glyoxylate then into glycine, that MITs turns into serine. The HYA controls the reorientation of wastes that become foods with the use of the ATP provided by the glycolysis. Simultaneously the PER can furnish back the cPLST in glycerate. If the EXO turns favourable, photosynthesis starts again. The association works in tender flows, because the wastes of ones are the foods of others and reciprocally. The PER fatty acids oxidation is cyanide insensitive, in the case of MIT dysfunctioning it is a relief way. Soon or late, the lodger always eats the lodged hosts. During the senescence, lipids are recycled through the cPLSTs

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[42] De DUVE C. (1983) *Les microcorpuscules de la cellule vivante*., p. 80-91. Pour la Science n° 69.

[43] GUERRA-GIRALDEZ C. & al. (2002) *Compartmentation of enzymes in a microbody, the glycosome, is essential in Trypanosoma brucei*., p. 2651-8. J. Cell Sci. n° 115.

[44] DONALDSON R.P. & al. (2000) *Plant Peroxisomes and Glyoxysomes.* Nature Encyclopedia of Life Sciences. **doi:10.1038/npg.els.0001677**

[45] SMITH J.J. & al. (2002*) Transcriptome profiling to identify genes involved in peroxisome assembly and function*., p. 259-71. J. Cell Biol. n° 158.

[46] HONSHO M. & al. (2002) *The Membrane Biogenesis Peroxin Pex16p. Topogenesis and Functional Roles in Peroxisomal Membrane Assembly*., p. 44513-24. J. Biol. Chem. n° 277.

[47] KOCH A. & al. (2003) *Dynamin-like Protein 1 Is Involved in Peroxisomal Fission.*, p. 8597-605. J. Biol. Chem. n° 278.

[48] MOHAN K.V.K. & al. (2002) *Identification of a Type 1 Peroxisomal Targeting Signal in a Viral Protein and Demonstration of Its Targeting to the Organelle*., p. 2543-7. J. Virol. n° 76.

[49] NAVARRO B. & al. (2004) *Expression of the Cymbidium Ringspot Virus 33-Kilodalton Protein in Saccharomyces cerevisiae and Molecular Dissection of the Peroxisomal Targeting Signal*., p. 4744-52. J. Virol. n° 78.

[50] RUSSO M. & al. (2002) *Molecular Characterization of a Tombusvirus Isolated from Diseased Pear Trees in Southern Italy*., p. 161-6. J. Plant Pathol. n° 84.

degradation, then the PROTs are, with the lysis by the HYA first of MITs then of PERs, this in the backward order of the integration events.

4d. An early Constrained Danger, when discharged, becomes a KeyStone Actor.*

The PER controls the CELs fate.* Subordinated to the EXO status, the surexpression of NY PPAR* is carcinogenic[51], or turns on the APO of cancerous CELs.[52] MITs, cPLSTs and the HYA are able to produce H2O2 in response to invaders. But alone the PER owns the defence mechanism against H2O2 shock. Alone its ancestor could not only "not to be eaten" but also "to invade and eat". It is both because it has not made that (or has not been able to make it) and because the CEL itself has no more destroyed it (or has not been able to make it) that their inseparable union merged through a qualitative evolutionary jump. The PER autophagy[53] takes place by sequestration. At least 1 gene of the lodging host allows the sequestration of the lodged host and the digestion of the selected prey.* At least 1 gene prevents this sequestration. During a plant infection, the necrotic[54] defence starts with the PER/GLY.

4e. The origins of membranes: "capacity of welcome" and "capacity to be welcomed".

When a predator enters in a CEL, it is sequestrated, separated from its host by a 2X MB. If that lodged host is extracted, the 2X MB ENV is preserved!* Additional ER MBs surround the organelles during their autophagy. When a predator invades by break-in, there is no SQMB. A Gram+ BACT derived organelle will have only 1 MB, it is the case of the PER. If derived from a VIR, it will have no MB, it is the case of the CENT (Table J, Figure 3).

5. The centrosome is a half-autonomous organelle coming from a virus.

The CENT is an evolutionary invariant that is exchangeable between species.*

Like the PER, it is both completely recreated while being able of division.*

Formed of 2 perpendicular interconnected centrioles, without any MB, it is composed of more than 80 PROTs.[55] As the Centre of polymerisation of the SQ and TUBs, the CENT[56] is indispensable to the CEL division.[57] Organising place of the assemblage of VIRs*, it has the topology of a capsid of a VIR[58] whose ancient RNA genome should nowadays be integrated in the DNA genome of the NY.

Like PERs it controls the CEL fate.*

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[51] FAJAS L. & al. (2001) *Peroxisome proliferator-activated receptor–γ : from adipogenesis to carcinogenesis*., p. 1-9. J. Mol. Endocrinol. n° 27.

[52] BEGUM N.M. & al. (2002) *Expression of peroxisome proliferator-activated receptor γ and the growth inhibitory effect of its synthetic ligands in human salivary gland cancer cell lines*., p. 599-605. Internat. J. Oncol. n° 20.

[53] STRÖMHAUG P.E. & al. (2001) GSA11 *Encodes a Unique 208-kDa Protein required for Pexophagy and Autophagy in Pichia pastoris.*, p. 42422-35. J. Biol. Chem. n° 276.

[54] COTS J. & al. (2002) *Pathogenic attack and carbon reallocation in soybean leaves (Glycine max. L.): reinitiation of the glyoxylate cycle as a defence reaction*., p. 91-6. J. Plant Physiol. n° 159.

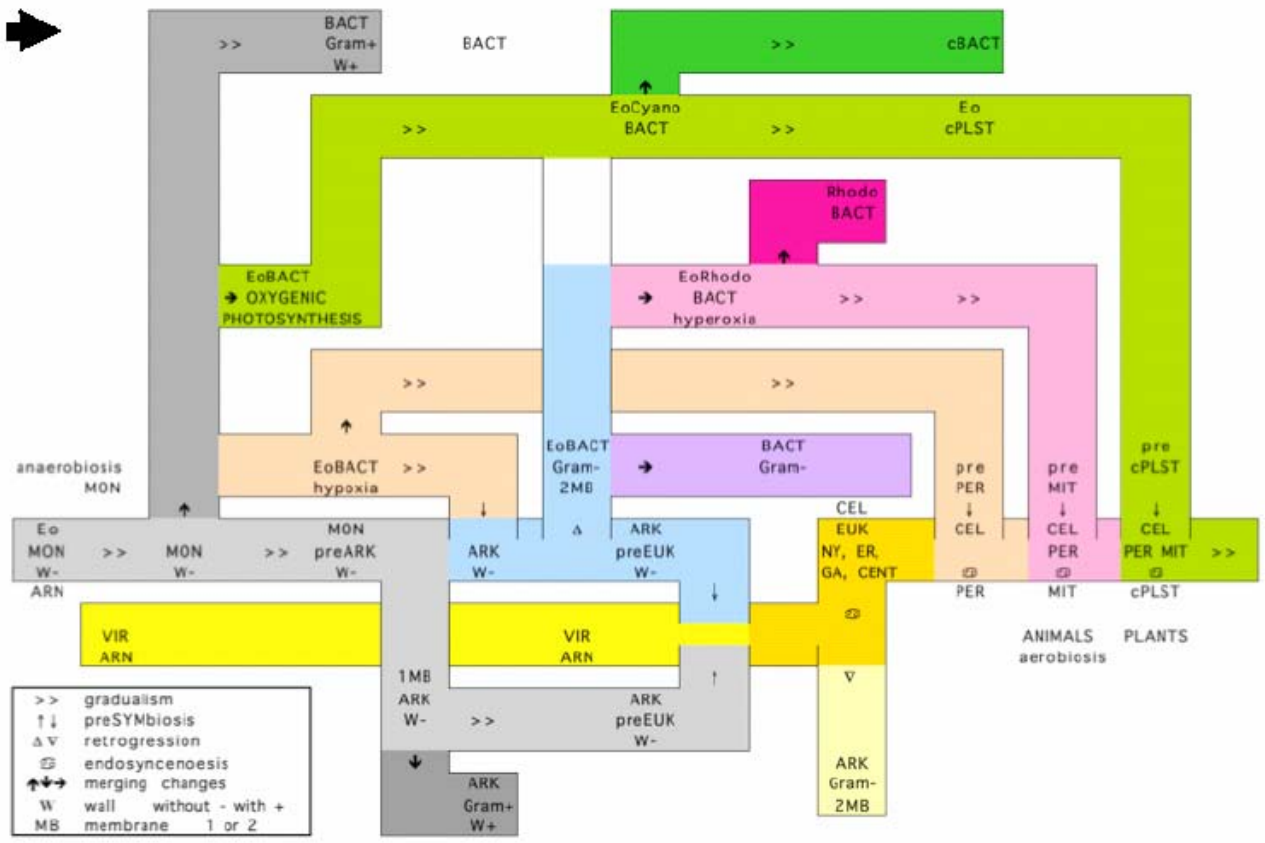
[55] STEARNS T. (2003) *The centrosome yields its secrets*., p. 14. Nature Cell Biol. n° 6.

[56] GLOVER D. & al. (1993) *Un architecte des cellules : le centrosome*., p. 50-8. Pour La Science n° 190.

[57] BORNENS M. & PIEL M. (2001) *Un agent de division*., p. 25. Pour La Science n° 283.

[58] MARTELLI G.P. & CANDRESSE T. (2003) *Closteroviridae*. Nature Encyclopedia of Life Sciences. doi:10.1038/npg.els.0000747

Figure 3. The Moneres, the PreOrganelles and the cells' phylogeny.[59]



Eo down, **Pre** just before, **MON** monere, **ARK** Archaea, without – or with + wall **W**, with **1** or **2** membranes **MB**, **BACT** Bacteria, Gram+ or Gram-, **EUK** Eukarya, **CEL** cell, **NY** nucleus, **ER** endoplasmic reticulum, **GA** Golgi apparatus, **PER** peroxisome, **MIT** mitochondrion, **cPLST** chloroplast (the time's arrow runs from left to right).

The progenote EoMON was a preARK, without W, surviving in anaerobiosis by fermentation. Its RNA genome, with disjoined genes, in repeated copies, coded for small peptides, actually juxtaposed in PROTs. The VIR induced endosyncoenotic fusion of aggregated MONs initiated the emergence of the EUK structure with both NY, ER. and CENT. As each organelle, it could be a prey or a predator. MONs of similar volumes cannot eat each other, but after the plasmolysis of some ones the other ones can. It is in that manner that the MIT ancestor had been trapped.[59]

A bacterium is not a cell. A cell is a polyMON endosyncoenosis that is stabilised by containing dangers[60], a wholeness in which the interdependence allows to transform disadvantages into advantages, and the pleiotropy avoids that advantages become disadvantages.

[59] Do look at **http://minilien.com/?MKOkk2v5Nv** for complementary Tables and Figures.

[60] BRICAGE P. (2005) *The Metamorphoses of the Living Systems: The Associations for the Reciprocal and Mutual Sharing of Advantages and of Disadvantages.* 10 p. 6th SSEC, ENSAM (Paris, France).

Do look at **http://www.minilien.com/?LUeZbdsNCH** for complementary informations about MON, CEL & VIR.