Alzheimer’s Disease and Age-related Macular Degeneration could simultaneously be treated or prevented with a single therapeutic intervention

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ABSTRACT

Alzheimer’s diseases becomes the most common type of dementia in the world. There is more of 5.4 million AD patients in America, more than 6 million patients in China, and 35.6 million patients worldwide. AD and other dementias worldwide will increase to 67.5 million by 2030 and 115.4 million by 2050. In the aging population, two of the most common neurodegenerative diseases, AD and AMD, could simultaneously treated or prevented with single therapeutic intervention.

Keywords: AD, AMD, Melanin, hydrogen, energy, glucose.
Background:
Age related macular degeneration (AMD) is the leading cause of visual impairment in elderly persons in industrialized countries [1]. Alzheimer’s disease and AMD have long been hypothesized to share a common pathogenesis based on several lines of evidence. Both conditions have similar histopathological changes [2], as drusen accumulations, containing β-amyloid, lipid, and other neurodegeneration waste products. Supposedly these deposits may lead to subsequent retinal pigment epithelial changes, photoreceptor malfunctions, neovascularization, and eventually atrophy.

In Alzheimer’s disease, an accumulation of extracellular β-amyloid, axonal and dendritic waste products from dystrophic neurons has been documented. These deposits form senile plaques and neurofibrillary tangles in the cortex and hippocampus of the brain that lead to neuronal malfunction and cell death.

AMD and AD share similar vascular risk factors [3], such as hypertension, and cigarette smoking. Both AMD and AD have been linked to an increased risk of stroke [4].

The similarities shown in the various studies have led to the belief that in the aging population, two of the most common neurodegenerative diseases, AD and AMD, could simultaneously be treated or prevented with a single intervention [5].

Introduction:
In biology and thereby medicine, there are two widely and deep-rooted dogma: First) Glucose has a double function as universal precursor of any organic matter in the body, and second) melanin is a simple sunscreen that tends to disappear with civilization. It is alleged that people in high latitudes in Europe and east Asia independently evolved lighter skin to produce vitamin D more efficiently with less sunlight [6]. However, the results of our study shattered both dogmas.

In 1990, we began a descriptive, observational study about the three main causes of blindness (AMD, Diabetic retinopathy, and glaucoma) and its possible correlation with anatomical variations of the minute vessels that pass through the optic nerve. Due to the optic nerve in humans is so small, 1200 microns, it was necessary to use significant enlargement of images obtained from retina’s patient to get enough definition of anatomic details of these blood vessels; magnification was achieved through optical and digital means. Photographic files of 6000 patients were included along 12 years of the study.

A few weeks after the study started, we noticed the constant presence of melanin in the vicinity of the optical disc (figure 1), so we decided to include it as a variable in study, after the blood vessels of the optic nerve, which until then was the main variable in study.

Figure 1) Image of the optic nerve of the left eye, the anatomical details of the blood vessels of the optic nerve are appreciated, and in turn, a dark edge can be detected in the meridian of 2 to 5 that corresponds to the melanin (arrows).
Our study leads us to find three axioms:

**Melanin is present nearby optic nerve of all human beings.** It is an ancient observation, since Helmholtz Ophthalmoscope invention (1851), however, the analysis of retina photographs raises in our team the awareness of the ever presence of melanin around the optic nerve in humans. The traditional explanation about these significant phenomena is roughly as embryonic remnants, or sometimes are sequels of local or systemic inflammatory processes in the past, and so on. However, nature just insist in important things, thereby we tried to find some other explanation.

As our research progressed, we began to detect that the blood vessels seemed to respond to the presence of melanin, and as the presence of melanin was constant in all the patients we examined, also the substantive antiangiogenic effect of melanin was remarkably persistent. Finally, we built a second axiom:

**With a greater amount of melanin there will be a lower number and caliber of blood vessels and vice versa.**

Now, we had to explain the mechanism. By one side, melanin is an amorphous substance, therefore it has not something that could be considered as factory of peptide factors; and neither has something that could resemble as a receptor of some type. Thereby, the cross-talk between endothelial cells of blood vessels and melanin cannot be through peptide factors or something similar that could be produced by endothelial cells, because melanin does not have some type of receptor biologically accepted or at least described.

Our partial conclusion was that blood vessels obey melanin presence through some type of unknown communication which is remarkably efficient. Our efforts to find the answer led us to detect a third axiom:

**A higher amount of melanin means a higher level of oxygen in the tissues and vice versa.**

This finding represented the long-sought answer because the best anti-angiogenic agent is known are the elevated levels of oxygen in the tissues.

The doubt was resolved, but what was next was trying to find where so much oxygen came from, because in our working conditions the difference reached 34%. We analyzed the metabolic processes described in the literature concerning the tissue in question, but we could not find any process or molecule that could donate such a quantity of oxygen so consistently.

So, we began to examine the molecule with greater presence in the eye and that is the water, since more than 95% of the eyeball is constituted by it, at least in number of molecules. It seemed feasible that oxygen would come from the water of the Vitreous body, but the water does not release the oxygen easily, it is necessary some mechanism to remove it from there when it is dissolved in enough amount, or the next possibility was that melanin could dissociate the molecule of the water.

And the conditions were given. The eye has 40% more melanin than the skin, its water content exceeds 90%, and the amount of light is adequate, and even concentrated by the natural lenses that are the crystalline and the cornea. A few experiments in the laboratory were enough to demonstrate the hitherto unknown intrinsic property of melanin to dissociate the water molecule [7].

Once we could take the biological event to the test tube, the results left us astonished because the melanin is not only able to dissociate the molecule of the water, but it is also capable of reforming it (Figure 2).

\[
2\text{H}_2\text{O} \rightarrow 2\text{H}_2 + \text{O}_2 \rightarrow 2\text{H}_2\text{O} + 4e^-
\]

Figure 2) Melanin absorbs visible and invisible light and dissipate the absorbed energy dissociating the water molecule. However, melanin can support the opposite reaction: the back-bonding of Hydrogen and oxygen; and for each two molecules of re-formed water 4 high energy electrons \((4e^-)\) are generated.

The conclusions of that study were surprising, melanin has an unsuspected bio-energetic biological role. Glucose is the universal
precursor of any organic matter but cannot provide the energy that its own metabolism requires \[^8\].

So, the dogma that our body gets energy by combining glucose with oxygen gradually (graduated combustion?) was difficult to accept because oxygen is toxic to any concentration, and on the other hand the sacrosanct role of glucose as a source of energy is broken into a thousand pieces.

**The role of energy in the functioning of the human body**

It is widely accepted that energy metabolism plays a role in health and aging as well as disease. Energy must be available for the work of synthesizing new cellular material, maintaining membranes and organelles, and to fuel movement and active transport.

In the 19\textsuperscript{th} century researchers first released that metabolism can be viewed as a highly complex network of connected biochemical reactions. Enzymes catalyze these reactions, this is, they enable them and modulate the rate at which they take place. However, an enzyme does not make possible an impossible chemical reaction.

Since the first scientific article on a metabolic process in the 17\textsuperscript{th} century nearly six million articles on metabolism have been published. And every day articles that seem to add one more piece of the puzzle about the mechanisms of metabolic processes in humans and many other organisms continue to be published. To understand the metabolism, we need to know how the reactions and the enzymes catalyzing them fit together and act as a whole.

The idea that energy and mass come from the same source (food); it is very old, and that glucose specifically is the main molecule that provides at the same time to the organism with carbon chains and energy began to emerge in the 17\textsuperscript{th} century. However, of the about 70000 chemical reactions described in the literature that have been published trying to build a logical sequence that explains the apparent double function of glucose, just in 199 there is consensus and, in the rest, (=6801) is controversial \[^9\].

The importance of metabolism (continuous change) is such that several of the most prevalent diseases in modern society, including diabetes, cardiovascular diseases and obesity involve disruptions of metabolic processes. Even in cancer, metabolism also plays an important role, as a tumor requires energy to grow \[^10\].

It is to attract the attention that no one uses in the clinical practice the ATP to raise the levels of energy of the patients. And the reason is that in experimental animals as well as in some very small clinical trials the results have been disastrous \[^11\]. What was to be expected because if glucose was a source of energy the diabetics patients would fly.

**Energy in the clinical practice**

Roughly speaking, 40\% of the energy the body gets from glucose is used to boost the necessary chemical reactions, and 60\% dissipates and maintains the proper body temperature. Therefore, the measurement of body temperature gives us an approximate idea of the energy state of the organism, but not for the above reasons. Temperature measurement is a coarse, inaccurate method.

IF ATP were the universal Energy exchange currency then the determination of the concentration of the energy carrier molecule, adenosine-5´-triphosphate (ATP) supposedly may assess the energy state in cells but given the complexity of the procedure is not of practical use in the clinic, besides that the levels of energy change constantly. It is argued that cellular ATP is an important element of cell death by apoptosis or necrosis \[^12\]. Thereby, it is thought that a decreased cellular ATP level is characteristic for cell death, but there is no systematic investigation whether the decrease is the cause or consequence of cell death, or simply a detectable part of an extraordinarily complex biological system.

Because in everyday clinical practice we only have a limited number of paraclinical studies, for
practical purposes mitochondria and ATP are supposed to be the basis for the exchange of energy in the eukaryotic cell, and if blood glucose levels are within adequate limits, it is assumed that mitochondria and ATP will also be. And the clinician automatically focuses on detectable alterations by physical examination and para-clinical test.

However, the biology of eukaryotic cell and our organism can be divided in mass (everything that has a place in the space) and energy (everything that produce a change). The current medicine is mainly based on the examination of the mass, since the energy is difficult to evaluate and it is generally accepted that if the glucose is well, then the levels of energy will certainly be the appropriate ones.

But neither mitochondria nor ATP are energy sources, because the molecular hydrogen (H\textsubscript{2}) derived from the dissociation of the water molecule and the high energy electrons (e\textsuperscript{-}) that are generated when water is re-formed, can cover the full energy needs of the eukaryotic cell. The generation and distribution of energy from melanin are surprisingly accurate and fast processes. They happen the same way since the beginning of time. And the changes we observed in the biomass reflect the accuracy of the energy generated by the melanin, as the continuous changes or metabolism that organic molecules have continuously in the human body, also occur with an astonishing accuracy.

**The impairment of generation and distribution of energy**

The biological processes of the body are very sensitive to changes in the available energy. and sooner or later they are reflected in the functioning of the organism. When the accuracy in the generation and distribution of energy is perturbed, for instance, by cold; then our body goes in imbalance and clinical manifestations should ensue.

It is relatively unpredictable the type of signs or symptoms that could be presented when the balance between mass and energy occur in human body.

In the case of the macula of the eye, the delicate anatomy of this highly specialized zone is deformed by the presence of blood, inflammation, neovascularization, atrophy, etc. But finally, the patient notices the distortion of images. which is progressive according to how the anatomy of retina is more and more altered. We call it age-related macular degeneration (AMD) because it is more frequent from the 60s. It is the leading cause of visual impairment in elderly persons in industrialized countries. The term degeneration can be defined as “deterioration of a tissue or an organ in which its function is diminished, or its structure is impaired”, in other words: “A retrogressive pathologic change in cells or tissues in consequence of which their functions are often impaired or destroyed; sometimes reversible; necrosis results in the early stages”.

In accordance with different sources of information, i.e. National Eye Institute of NIH, currently, no treatment exists for early AMD. The benefit of nutritional supplements will not restore vision already lost from intermediate AMD and perhaps help slow vision loss in people who already have late AMD. In the case of neovascular AMD, typically results in severe vision loss. And several treatments are discussed: intraocular injections, photodynamic therapy, and laser surgery. However, the panorama is bleak, as AMD remains as the leading cause of blindness in industrialized countries as it has been for the past 60 years or more. This indicates that the treatments currently available for AMD are not working, because the incidence and prevalence of this disease has not varied significantly.

**The good news**

As we understood and deepened the fundamental role of melanin in molecular biology, we could understand that the usual manifestations of AMD are basically an imbalance between mass and energy in the organism and that can manifest in the macula. That is why despite the exhaustive studies at both the macro and microscopic level, both
anatomical and histochemical; the pathophysiology of AMD has not been understood, and the adage is fulfilled that we cannot cure what we do not understand. There are some studies about energy disturbances in diseases such as heart failure and Alzheimer’s disease, and even treatments have been attempted in human patients, but they have failed because researchers were considering energy as an important etiological factor, but they insisted. Insist on including glucose and ATP in the theoretical schemes of energy metabolism.

So, we developed some pharmacological strategies to try to restore the lost balance between mass and energy according our research, and the results exceeded our own expectations.

**Case studies:**

Patient 1) Female patient, M.G., in the eighth decade of Life, who presented a gradual loss of vision in the left eye. She was attended by other physicians, but the loss of vision advanced inexorably. When months later she presented the same symptoms in the contralateral eye and realizing the null usefulness of the available treatments; the patient decided to go with us.

The ophthalmological examination showed data compatible with AMD of neovascular type (Figure 3). It was explained to the patient and relatives the type of treatment we started to offer and given the little or no effectiveness of the various treatments she had tried in the left eye, she decided to use them. Because it is not a propaganda article, only of information that we consider important to be known, we only mention the therapeutic results, which by the way are extraordinary (Figure 4).

**Case study 2)**

Female patient in the seventh of life, who has distortions in the vision of the left eye for a few months, which have been aggravated despite several treatments by ophthalmologists. When they proposed the application by intravitreal injection of anti-VEGF antibodies, she decided to assist to our office.

The ophthalmological examination performed at the first consultation showed compatible findings with subretinal neovascularization or neovascular AMD (Figure 5). The anatomic and visual improvement were substantive (Figure 6)

![Image](image-url)

**Figure 3) Case study 1.** The photograph shows the typical appearance of choroidal neovascularization that grows disorderly and abruptly, deforming the anatomy and physiology of the over-lying retinal tissues.
Figure 4) Case study 1. The patient was reviewed within 21 days of commencement of treatment. Since she walked into the office without help, we realized things were going well. And so it was, the vision had improved 80%, and the ophthalmological examination showed an astonishing improvement in the anatomy of the macula.

Figure 5) Case study 2: The appearance of the retina suggests that the current lesion (yellow arrow) had been preceded by previous imbalance events (blue arrows), one of the scarred, which is denoted by melanin presence in the area. The photo corresponds to the first test before the beginning of the treatment.

Figure 6) Case study 2: The patient went to review three months after being continuously using our treatment. The improvement is manifested by the notable decrease in exudates (yellow arrow) and by the decrease of the retinal folds (blue arrows) caused by the edema of the tissues.
Case study 3:
Female patient in the seventh decade of life, who consulted us via email from France, because a family member who lives in our city told him from our research. The patient had been diagnosed as AMD in intermediate stages by an ophthalmologist. Her retinal studies of the right eye (Figure 7) showed changes compatible with the diagnosis of AMD, so we advise you to use our treatment.
Periodically, the patient wrote to us and told us that it began to improve, to the degree that the Ophthalmologist told her that it had been a miracle. Figure 8 clearly shows the disappearance of the cystic spaces inside the retina, characteristic of AMD. The difference between the two studies is one year. (Figure 8)

Figure 7) Case study 3: Typical appearance of abnormal accumulation of fluorescein in the Macular area (arrows) which is normally dark. The study was performed prior to the commencement of treatment.

Figure 8) Case study 3: IN the upper part is observed the study of optical coherence tomography practiced in October 2009, where we appreciate a cystic lesion (yellow arrow) that is characteristic of AMD. In the lower part, we observe the study of optical coherence tomography dated September 22, 2010, which shows that the cystic lesion disappeared, and the contour of the macular region is now within normal limits.
Case study 4)
Female Patient, in the sixth decade of life, with long-evolving axial myopia, who begins with metamorphopsias that gradually were taking the form of a dense central scotoma. At the ophthalmological examination, a hemorrhage below macular region is seen (Figure 9). At 8 weeks of treatment, sub-macular hemorrhage has decreased significantly. (Figure 10). At twelve weeks of initiation the treatment the blood disappeared in its entirety with scarce scar tissue. (Figure 11).

Figure 9) Case study 4. IN The first examination, a remarkable blood extravasation is seen (arrows), covering the entire macular region.

Figure 10) Case study 4: At 8 weeks after the treatment started, the blood has disappeared for the most part, with little or no residual reaction.
Figure 11) Case study 4: After twelve weeks of treatment, the result is very encouraging, because the blood already disappeared in its entirety or null fibrosis reaction.

Case study 5)
A 45-year-old female Patient, who develops a hemorrhage in the macular region consequent a bronchopneumonia. (Figure 12, Upper, left) After one week of treatment, the blood has reabsorbed largely (Figure 12, up, right). At the third week of treatment, the blood disappeared completely, without apparent tissue damage, except for a depigmentation zone. (Figure 12, down, right and left)

Figure 12) Case study 5: In the upper part, on the left, there is a hemorrhage that covers the fovea centralis. At the top, to the right; Blood that has been reabsorbed remarkably with treatment. At the bottom, it is appreciated that, at three weeks after the initiation of treatment, the blood has disappeared completely without retinal alterations, except for a thin line of hypopigmentation.
Case study 6)
Female Patient in the eighth decade of Life, with intermediate myopia from the youth, who presents fluctuations of vision in right eye of three weeks of evolution, which have been worsening.

A major hemorrhage in sub-macular region is observed in ophthalmological examination. (Figure 13). At the sixth month of treatment, the hemorrhage has completely disappeared, and the vision has recovered substantially, as the patient refers that he could no longer cook, and now he can do it, as well as wander without help. (Figure 14)

Figure 13) Photograph of the right eye retina taken in the first exam. A major sub-macular hemorrhage is seen.

Figure 14) Case study 6: The Blood has completely reabsorbed, the whitish striations observed, have been seen before. The Foveola centralis, or is observed in good condition with the blue filter (Up, right)
The Six cases of macular degeneration that we present here are demonstrative, because they are not the only ones or the best, in fact are the cases of macular degeneration that we can show with a couple of photographs.

AMD, the Alzheimer’s disease of the eye?

The cross-sectional association between low cognitive function and early AMD has been carefully documented \[13\]. It can be sustained, beyond reasonable doubt, that AMD and cognitive impairment may share similar complex pathogenesis and risk factors.

Neurodegenerative diseases, the leading cause of morbidity and disability, are gaining increased attention as they impose a considerable socioeconomic impact, due in part to the ageing community. Neuronal damage is the pathological hallmark of Alzheimer’s disease (AD) and Age-related Macular Degeneration (AMD) and various neurodegenerative illnesses such as Parkinson’s diseases, amyotrophic lateral sclerosis, Huntington’s disease, spinocerebellar ataxia and multiple sclerosis. A common feature of neurodegenerative diseases is chronic activation of innate immune cells within the CNS, and in other diseases such as multiple sclerosis (MS), New concepts about immune environment of CNS are emerging from the study of the influx of peripheral immune cells across the blood–brain barrier (BBB), identification of lymphatic drainage, as well as by detailed studies on immune cell trafficking in the choroid plexus (CP). \[14\]

There is indirect evidence that free radicals and excited-state species play a key role in both normal biological function and in the pathogenesis of human diseases. Generation of activated species by inflammatory cells mediate important components of the inflammatory response. The evidence for a role for electronically activated species in human diseases has long been prevalent. However, free radicals and other activated species are so difficult to measure under biological conditions, so the evidence for their role for electronically in biological process is normally indirect and circumstantial. \[15\]

Thereby AD and AMD present with increased production of activated species or with increased levels of radical- generating substances. The pathogenic pathways leading to neurodegeneration in AD include accumulation of aberrant or misfolded proteins, ubiquitin-proteasome system dysfunction, excitotoxic reactions, oxidative and nitrosative stress; mitochondrial injury, synaptic failure, altered metal homeostasis dysfunction of axonal and dendritic transport, and misoperation of chaperones \[16\]. Such a widespread failure is only possible when the problem is energy.

So far, the major obstacle in managing the disease and thereby the designing of rational therapeutic targets is the incomplete understanding of the pathogenesis of the disease. Several hypotheses have been proposed to explain the pathogenesis of AD, including theories involving amyloid deposition, tau phosphorylation; oxidative stress; metal ion dysregulation, and chronic inflammation. Unfortunately, no single one of these theories is enough to explain the spectrum of abnormalities found in the disease. However, the generation and distribution of energy that comes from melanin through dissociation and re-formation of water in form of molecular hydrogen (H2) and high energy electrons (e-) seems to be a good candidate as fundamental initiator of the pathological cascade.

The cases of Alzheimer's which we present and have been treated with the Therapeutic strategy based on Neuromelanin as a source of energy are a significant test of the importance of the light, visible and invisible, as a main energy source of the CNS.

Case study 7:

Female Patient in the eighth decade of life, who begins with forgetfulness, fears, sudden changes in mood, and progressive incapacitate of taking care of herself. During the previous 4
years she had received treatments of various types without results.

**Case study 8)**

Female Patient in the seventh decade of life, who a few years after having retired from a social security institution in Mexico, began with increasing memory problems, which were added intense depression, aggression, excessive fears, and growing disorientation. Despite different treatments used in the previous 3 years, the general state of the patient continued to deteriorate, so that her relatives decided to go to our Study center.

![Photograph taken during the first exam](image1.png)

**Figure 15)** Case study 7: Photograph taken during the first exam, the expression is typical of Alzheimer's disease (anger, deep sadness, fear, aggression).

![Close-up](image2.png)

**Figure 16)** Case study 7: The close-up shows in greater detail the usual characteristics of facial expression of Alzheimer's patients. The photograph was taken at the time of the first exam.
Figure 17) Case study 7: The photograph was taken two years after we started treatment with drugs developed by us. The improvement is substantive.

Figure 18) Case study 8: The patient’s facial expression reveals fear, sadness, fear, anxiety. Note the position of the family’s hands (left). The photograph was taken during the first exam.

Figure 19) Case study 8: The position of the hands of the relative (left) now looks more relaxed, after three months of treatment, because the changes she has noticed in the sick are egregious. The photograph was taken three months after we started our treatment.
Case study 9)
Female Patient at the beginning of the eighth decade of life, who presented mild problems of behavior and memory that have gotten worse. Relatives have tried various treatments without much result, so they come to us for an evaluation.

Case study 10)
Female Patient in the eighth decade of life, with progressive dementia of several years of evolution, which has been worsening despite the care of relatives. When the patient attended the first consultation, no longer communicating in any way, was in a wheelchair and required the use of diapers. She suspended the medication she was using so far and started to use our treatments.

Figure 29) Case study 9: In the photograph taken during the first examination, the facies that characterize the dementia type Alzheimer, the most common one, are seen. Note the aggressiveness, the fear, the deep depression.

Figure 30) Case study 9: After three months of treatment initiation, changes in facial expression are substantive.
Case study 10)
Figure 31) Case study 10: The picture shows the appearance of the patient who could be seen in the first exam. The physical and mental condition were deplorable.

Figure 32) Case study 10: After six months of treatment, noticeable changes are seen in the expression of the sick, who already ate better, socialized more, was able to ask for water or food.

Case study 11)
Female Patient in the ninth decade of life, with DX of AD since 2007, was examined for the first time in our study Center in 2010.

Case study 12)
Female Patient in the seventh of life, who presents alterations of behavior that have been get worse, because he does not want to leave his room, leaves the stove on, does not want to bathe, his behavior is aggressive.
Figure 33) Case study 11: The patient's depression was so intense that she did not raise her head during the entire consultation. Photograph taken in the first exam.

Figure 34) Case study 11: This photograph was taken six months after our treatment started. The improvement is surprising.

Figure 35) Case study 11: The patient's gaze at six months of initiation treatment is very significant.
Figure 36) Case study 12: Photo taken during the first exam, notice the lack of personal care.

Figure 37) Case study 12: Three months after the initiation of treatment, the patient shows a noticeable improvement. Facial expression is different, and her personal care has improved ostensibly.

Case study 13)
Male Patient in the seventh life, who starts with behavior problems that have gradually worsened, to the extent of getting lost in the street, can no longer work.

Case study 14)
Female Patient in the ninth decade of Life, who presents invalidity of several years, requires wheelchair, marked depression, loss of memory moderate to marked at times, and uses 14 medicines of different types. Denies Diabetes mellitus.
Figure 38) Case study 13: The appearance of the sick in the first exam. There is apathy, loss of interest; depression.

Figure 38) Case study 13: 4 months later, and despite not having followed the treatment properly, there was improvement, which can be glimpsed in this photograph, because there is a significant improvement in his mood.

Figure 39) Case study 14: Photograph at first examination.
Case study 15)
Female Patient in the eighth of life who began with forgetfulness without apparent importance but have been increasing to constitute an obstacle to their daily life, four years of evolution.

Case study 16:
Female Patient in the eighth decade of Life, which has changed behavior for 5 years, which have gradually worsened.

Figure 41) case study 15: The appearance of the patient in the first examination, reflects fatigue, depression.

Figure 41) case study 15: After three months of treatment, the facial expression has changed, Since the 15 days of treatment, the relatives refer that noticed positive changes in the patient, which have been increasing.
Figure 42) Case study 16: Depression is a constant finding in patients with progressive cognitive problems, as reflected in the clinical photograph of the patient at the time of the first exam.

Figure 43) case study 17: At two months of treatment the changes are already perceptible in facial expression.

Case study 17:
Female Patient in the sixth decade of life, who has mild cognitive alterations that have progressed and begin to obstruct daily life. Five years of evolution.
Conclusion:
The cases of study presented in this article and with a growing number each day, are a proof not only significant even substantive because they demonstrate beyond the reasonable doubt the role of hydrogen as a carrier of energy in the human body, possibility that had already been proposed by Prof Szent Györgyi, in 1961 [17]; Even he said that if there is a constant hydrogen source it would be easy to explain biology; but not knowing at that time the unsuspected bioenergetic role of melanin; the professor Szent-Györgyi argued that glucose was the packaging by which nature transported the hydrogen from the outside into the human body[18].

The energy metabolism of the glucose-based organism resulted in false news that appeared and lasted in the textbooks.

The cases we present are not the only or the best, but the ones we can publish succinctly and that show beyond reasonable doubt, that we must discard the mistaken idea of an energy metabolism based on the oxidation of glucose.

Glucose as source of energy It Is a collective mistake that already has too many decades and every day that passes causes more and more damage, as well as unnecessary suffering.

The purpose of this article is, above all; make a call to collective consciousness so that each of us does what is necessary for the new biology, which implies a new era in medical practice; get to the general public as soon as possible and can be used massively for the benefit of the sick and society.

The challenge is formidable, for it is ideally that the new knowledge about that the human body can directly use sunlight, like plants as well; It should appear in the shortest possible time in textbooks from elementary degrees of education to the most advanced, washout glucose as source of energy.

Likewise, all the actors in the different social structures will have to do their part to favor the
profound change that is required to modify the course of the society towards a preventive and corrective medicine very different to the current one but that is more human and efficient. We talk about politicians, educational authorities, health authorities, tax authorities; Universities, research centers, professionals; associations, academies; Pharmaceutical laboratories of all sizes, in short; of each and every one of the elements that make up society to do their part to propitiate the rapid establishment of a new era in biology and medicine that will be, without a doubt; a huge collective benefit. It is better to recognize as soon as possible that universities and research centers were wrong to continue to perpetuate the long-lasting mistakes even more.

References:

   Handbook of free radicals and Antioxidants, Vol I.
16. Parihar, Mordhwaj S. Brewer, Gregory J.
   Mitoenergetic failure in Alzheimer’s Disease. Am
   J Physiol Cell Physiol 292: C8- C23, 2007- doi:
   10.1152/ajpcell.00232.2006
   On the energy transfer in biological systems.
   Institute for muscle Research at the Marine,
   Woods Hole, Massachusetts. Communicated
   Sept 15 1951.
   Nov. 1956, Vol. 124, No. 3227,pp873-875