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The Role of Modified Mediterranean Diet and Quantum Therapy in Oncological Primary Prevention

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Abstract: The Authors provide an overview of useful treatments such as 'Modified Mediterranean Diet', CoQ10, Melatonin and 'Quantum Therapy', testing their effects in Oncological primary prevention. This is done through 'Quantum Biophysical Semeiotics' biological evaluation, clinically monitoring the results and efficiency of ongoing therapies aimed at improving mitochondrial and endothelial function, when it is impaired in any biological systems. This clinical method allows physicians to bedside assess tissue acidosis, before and during different preventive therapies, testing their respective efficacious and utility.

All the investigated treatments have initially ameliorated and then normalized tissue microcirculatory pattern, showing a physiological functioning. Furthermore the tested 'Quantum therapy' generates virtuous genetic feedbacks.

'Quantum Biophysical Semeiotics' theory is an extension of medical semeiotics. It is grounded on a multidisciplinary approach that involves chemistry and biology, genetics and neuroscience, chaos theory and quantum physics. It is based on the method of 'Auscultatory Percussion', through which by means of the common stethoscope, it is possible to listen to the signs that the body gives us when appropriately stimulated. The stimuli are used to induce consistent behaviour in precise and well defined biological systems of the human body, thus giving local qualitative information on the state of health or disease, whether potential, being developed but not yet evident by usual clinical trial, effective or even in chronic phase. The 'Quantum Biophysical Semeiotics' theory provides very detailed case studies based on the latency time, duration, and intensity of the reflexes, which play a central role in such a diagnostic method.

Keywords: Oncology, CoQ10, primary prevention, mediterranean diet, melatonin, quantum therapy, clinical diagnosis, biophysical semeiotics.

INTRODUCTION

In a recent paper [1] we have shown the crucial importance of 'Modified Mediterranean Diet' and the important role of Coenzyme Q10 in the primary prevention of several diseases. CoQ10 has got a central bio-energetic role in mitochondrial redox metabolism and phosphorylation of ADP. Furthermore, we highlight the Melatonin Action Mechanisms in metabolic processes. This is done through Quantum Biophysical Semeiotics bed-side evaluation, which allows to bedside assess CoQ10 deficiency.

Quantum Biophysical Semeiotics – QBS - theory, extension of the medical semiotics, is based on the Congenital Acidosic Enzyme-Metabolic Histangiopathy, CAEMH [2], a unique mitochondrial cytopathy, present at birth and subject to medical therapy. According to QBS, physicians can bedside evaluate, simply using the stethoscope [3], the mitochondrial functionality of their patients in all biological systems.

In the present paper we focus our attention to Oncological clinical and pre-clinical diagnosis, bed-side, in the process of Oncogenesis.

Since birth, it is possible to make a diagnosis in order to assess the presence of Inherited Real Risk of cancer [4], linked to QBS Oncological Terrain (or Oncological Constitution) so that an intelligent prevention in subjects with Real Risk can be implemented. On the basis of QBS constitutions [5], i.e., Osteoporotic Constitution, Diabetic Constitution, the onset of more serious diseases such as osteoporosis, diabetes, cancer, ischemic heart diseases, including myocardial infarction, can be prevented.

The new approach introduced by QBS allows the diagnosis of cancer, even silent or in the very beginning clinical

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stages. The existence of pre-metabolic syndrome[1], pre-clinical stage of still potential diseases (evolution to pathology, pre- morbid state or gray area), can also be assessed, so allowing an effective prevention.

According to QBS theory, genoma's information are transmitted simultaneously both to parenchyma and related microvessels, so that mutations in parenchymal cell n-DNA and mit-DNA are the *conditio sine qua non* of the most common human disorders, like diabetes and cancer. In fact, all these diseases are based on a particular congenital, functional, mitochondrial cytopathy, mostly transmitted through mother, the CAEMH [6]. In addition, parenchymal gene mutations cause a local microcirculatory remodeling, gathering indirect information on inherited modifications of the relative parenchymal cell, since biological system functional modifications parallel gene mutation, according to Angiobiopathy theory [7]. The presence of intense CAEMH – termed CAEMH-α - in a well-defined biological area, involved by gene mutations in both n-DNA and mit-DNA, is the ground for one or more QBS constitutions[2] which could bring about, i.e., the congenital Real Risk – RR[3] - of cancer characterized by microcirculatory remodeling, intense under environmental risk factors.

With the aid of QBS, medical doctors are able to do the clinical evaluation of microvascular dynamics [8-10]. The microvessels carry on a motor activity, autochthonous and chaotic deterministic, which represents one of the most remarkable manifestations of microcirculatory hemodynamics, characterized by a *flow-motion* and rhythmically fluctuating hematocrit due to the particular nonlinear behaviour [11, 12] of both *vasomotility* and *vasomotion*[4].

Furthermore, the 'Inherited Real Risk' (IRR) of cancer, characterized by newborn-pathological Endoarteriolar Blocking Devices, is associated to endothelial dysfunction[5], which doctor can bed-side assess in an easy and reliable way, at rest as well as under stress tests. As a consequence of the above, briefly referred remarks, according with QBS theory, physicians can observe the presence of typical pathological EBDs[6] in microvessels [13, 14], which play a central role in cancer 'Inherited Real Risk' (IRR).

QBS method allows physicians to monitor tissue acidosis revealed by the latency time (Lt) of Gastric Aspecific Reflex (G.A.R.) before and during different preventive therapies, comparing them and testing the respective efficacious and utility.

1. Oncological Terrain and IRR of Cancer: Clinical and Pre-clinical Diagnosis

The objective QBS examination allows physician to bed-side recognize and quantify, in a few minutes, the presence of the 'Inherited Real Risk' (IRR) of cancer or overt cancer, even initial, through the evaluation of several semeiotics signs, i.e., assessing vasomotility, vasomotion and typical pathological EBDs. In following, we briefly resume the easier way for the diagnosis of this pathology or of the Oncological Inherited Real Risk: the Gastric Aspecific Reflex (G.A.R.) through the Auscultatory Percussion of the Stomach [15].

In a supine healthy subject, psycho-physically relaxed, with open eyes, aiming to lower significantly melatonin secretion, a digital pressure of "mean" intensity, applied upon the skin projection's area of cancer's trigger points brings about G.A.R., whose latency time (Lt), duration (D), intensity and Microcirculatory Functional Reserve (MFR) inform on tissue oxygenation at rest, as well under stress situations [16]. In Table **1** is resumed the study case about G.A.R.

According to clinical and experimental evidences [17], tissue pH is related to the reduction of latency time (Lt) and to the extension of the duration of the G.A.R., which expresses the local Microcirculatory Functional Reserve (MFR), calculated as simply as the disappearing time of G.A.R. before the appearance of the next one [18].

In addition, Lt of both caecal and aspecific oncological reflexes (i.e., caecal and gastric dilation) increases significantly, raising to 16 seconds (negative semeiotic sign, absence of cancer and of Oncological Terrain) when digital pressure becomes "intense", hence inducing local metabolic regulation of Tissue Microvascular Unit (T.M.U.), i.e., activating the MFR [6, 7].

[1]Metabolic syndrome is a combination of medical disorders that increase the risk of developing cardiovascular disease and diabetes. It is also known as metabolic syndrome X, syndrome X, insulin resistance syndrome, Reaven's syndrome. The pre-metabolic syndrome, as defined by Stagnaro, is the syndrome that precedes the metabolic one, and is linked with congenital real risks and their associated biophysical semiotics constitutions.

[2]QBS constitutions, detectable since birth, are the inherited congenital ground or terrain of well defined potential diseases clinically hidden, which can last several years before appearing, in the slow transformation process from potential (pre-metabolic syndrome, pre-clinical stages) to effective pathology (metabolic syndrome)

[3]Real Risk – RR - means any mutation, limited at level of cells belonging to a well-defined biological system - for example, beta cells of islets of Langerhans, for diabetes - which occurs in one or more cells when ATP decreases strongly for any reason.

[4]In all tissues, a part from their local different architecture, microvessel diameter oscillates rhythmically during time. The term *vasomotility* refers to small arteries and arterioles sphygmicity, according to Hammersen, and *vasomotion* is the subsequent oscillation of capillaries and post-capillaries venules diameter.

[5]There are mitochondria also in endothels, although in small amount. In the lining of the arteries (endothelial cells) and the smooth muscle cells in the walls of the arteries. The endothelial dysfunction is likely to be multifactorial in these patients and it is conceivable that risk factors such as hypertension, hypercholesterolemia, diabetes mellitus and smoking can contribute to its development.

[6]The Endoarteriolar Blocking Devices (EBDs) are a kind of dam which by opening and closing regulates blood flow in microvessels directed to the parenchyma. If these EBDs are tough, rigid, inelastic, there is a Real Risk of disease. There are EBDs Type I - located in small arteries, according to Hammersen -, and Type II – they can be found in the arterioles that are between small arteries and capillaries -: only type II is ubiquitous, in the sense that it is observed everywhere, in all arteries. Even these physiological types get sick or old. However, the other types, pathological-new-formed, are expressions of the Real Risk of potential disease, they are more occlusive, but through therapy they can be transformed from subtype a) pathological, to subtype b) aspecific, and then to "physiological" type, decreasing gradually their amount. EBDs play a primary role in the regulation of local microcirculatory *flow-motion*: when this is abnormal, there is congenital microvascular remodeling and EBDs bring about impairment of the Microcirculatory Functional Reserve (MFR), which contribute to affect the 'Real Risk' of disorders, like Osteoporosis, whose onset shall possibly occur after years or decades.

Table 1. Comparison of Different Preventive Therapies. Legend: Lt (Latency time); ↑ = Lt Increases from Baseline Pathological Level Liver Gastric Aspecific Reflex - Mean Intensity Digital Pressure on Liver Trigger Points (Liver Skin's Projection)

Latency Time (Lt) in Seconds	Latency Time After Precondi-tioning (Pause of 5 sec.)	Diagnosis	Latency Time During a Combined Treatment of Mediterranean Diet, CoQ10 and Melatonin	Latency Time During (and after) Quantum Therapy	Latency Time During (and after) Sulphurous Thermal Water Therapy
Lt = 8	Lt = 16	Health	==========	==========	==========
Lt = 8	Lt < 16	Inherited Real Risk of liver cancer	Lt = 12	Lt = 16 (12)	Lt = 20 (12)
7<Lt <8	Lt < 16	Inherited Real Risk of liver cancer in evolution	Lt = 12	Lt = 16 (12)	Lt = 20 (12)
Lt≤7	Lt < 14	Liver cancer	Lt = ↑	Lt = ↑	Lt = ↑

On the contrary, *Oncological Terrain sign* is positive in case of "intense" digital pressure when the reflex appears simultaneously, (Lt = 0), revealing the Oncological Constitution. In this last case, the G.A.R. is "simultaneous" [4].

In health – in supine position – digital pressure of mean intensity, applied, i.e., on liver trigger points (skin projection of the liver), brings about liver G.A.R. after a latency time (Lt) of 8 seconds (Table **1**, first column). Liver G.A.R. lasts less than 4 sec., soon thereafter disappearing for 3-4 seconds. Afterwards, a second reflex occurs. The duration of liver G.A.R. unfolds the MFR activity of related microvessels, thus correlated with the function and anatomy of the microcirculatory bed, the T.M.U. At this point of investigation, the physician quickly interrupts the digital pressure for exactly 5 seconds. Then, Lt of G.A.R. is evaluated again: Lt raises to 16 seconds, liver G.A.R. lasts less than 4 seconds, disappearing after roughly 4 seconds: these values evidence a *physiological preconditioning* (Table **1**, second column) [5].

In summary, when digital pressure is of mean intensity, physiological Lt of liver G.A.R. is 8 seconds at the first evaluation (*basal-line value*), but increases clearly doubling in the second as well as in the third one, due to the physiological activation of MFR.

In individuals at risk, i.e., of liver cancer, *base-line* Lt is physiological during the first evaluation (8 seconds). However, liver G.A.R. lasts 4 seconds or more and disappears for less than 3 seconds. Moreover, preconditioning results "pathological", as Lt is less than 16 seconds: these values give evidence of a *pathological preconditioning.* Interestingly, in patients with liver cancer, even clinically silent, the *basal value* of latency time of liver G.A.R. appears to be less than 7 seconds at first evaluation and becomes lower in the second one, in relation to the seriousness of underlying disorder.

In healthy subjects the *preconditioning* brings about, as natural consequence, an optimal tissue supply of material-information-energy, by increasing the local *flow-motion as well as flux-motion*.

On the contrary, if the 'Inherited Real Risk' of liver cancer is present, *preconditioning* data are almost the same as the basal ones, but Lt is a little shorter than physiological one. Finally, in overt disease, *preconditioning* shows an altered and shorter Lt of reflex in relation to the seriousness of the underlying disorders.

At this point, we come back to the former example: in the initial phase of cancer, which evolves very slowly toward successive phases, QBS "basal" data can seem "apparently" normal. However, under careful observation, the duration of liver G.A.R. is equal or more than 4 seconds (the normal value, NN, is less than 4 seconds), indicating a local microcirculatory disorder.

In these cases, *preconditioning* allows in a simple and reliable manner to recognize the pathological modifications, mentioned above, which indicate the altered physiological adaptability, even initial or slight, of the biological system to changed conditions as well as to increased tissue demands. The various QBS parameters related to a defined biological system, parallel and are consistent with the data of *preconditioning*.

2. Oncological Primary Prevention and Therapy with Modified Mediterranean Diet

'Modified Mediterranean Diet' (a greater amount of fish, i.e., protein and Omega-3 fatty acids, than the normal Mediterranean Diet), together with CoQ10 and Conjugated-Melatonin, were successfully tested in Oncological primary prevention and therapy[7]. The QBS method and signs were used to monitor tissue acidosis (revealed by the latency time (Lt) of G.A.R.) before and during these preventive therapies.

The combination of these treatments contributes to diminish as far as normalize tissue acidosis and re-equilibrate acid based balance as proved by a longer Lt (Table **1**, fourth column). We explain the properties of these treatments in short as follows.

a) 'Modified Mediterranean Diet Central Role' in Oncological Prevention

Many studies suggest that Mediterranean diet may be beneficial to health [19], and variants of this diet have improved the prognosis of patients with coronary heart disease

[7]We enrolled in our research thirty three women and two men, aged from 53 to 67.

Table 2. An Example of Modified Mediterranean Diet

1st Day	2nd Day	3rd Day	4th Day	5th Day	6th Day	7th Day
200 gr. Mixed Fresh Salad with Olive Oil	150 gr. Pasta with Olive Oil	200 gr. Mixed Fresh Salad with Olive Oil	200 gr. Mixed Fresh Salad with Olive Oil	150 gr. Pasta with Olive Oil	200 gr. Mixed Fresh Salad with Olive Oil	200 gr. Mixed Fresh Salad with Olive Oil
100 gr Fish	100 gr. Meat	100 gr Fish	100 gr Fish	100 gr. Meat	100 gr Fish	100 gr Fish
100 gr Legumes	200 gr. Mixed Fresh Salad with Olive Oil	100 gr Legumes	100 gr Legumes	200 gr. Mixed Fresh Salad with Olive Oil	100 gr Legumes	100 gr Legumes
150 gr. Fresh Fruit	150 gr. Fresh Fruit	150 gr. Fresh Fruit	150 gr. Fresh Fruit	150 gr. Fresh Fruit	150 gr. Fresh Fruit	150 gr. Fresh Fruit
150 gr. Yogurt	150 gr. Yogurt	150 gr. Yogurt	150 gr. Yogurt	150 gr. Yogurt	150 gr. Yogurt	150 gr. Yogurt
100 gr Cereals	100 gr Cereals	100 gr Cereals	100 gr Cereals	100 gr Cereals	100 gr Cereals	100 gr Cereals

[20]. The Mediterranean diet, in general, was associated with increased survival among older people, especially when modified adding to it unsaturated acids and omega-3, and suggesting physical exercise, walking about 40 min. day [17].

The 'Modified Mediterranean Diet', we suggest (Table **2**), is characterised by a high intake of vegetables, legumes, fruits, and cereals; a moderate to high intake of fish; a low intake of saturated lipids but high intake of unsaturated lipids, particularly olive oil; a low to moderate intake of dairy products, mostly cheese and yogurt; a low intake of meat; and a modest intake of ethanol, mostly as red wine [19-22]. Furthermore, there are novel actions of vitamin D (i.e., it reduces tissue acidosis and it presents anti-inflammatory properties) and it is useful for the prevention or treatment of degenerative disorders such as cancer [20-21]. The authors intend the term "diet" in etymological sense, including, i.e., daily physical exercise, whose paramount importance is highlighted as follows, since it works ameliorating endothelial function, as Conjugated-Melatonin does [2-7, 23].

Aadherence to a Mediterranean diet proved to be efficacious in preventing most common and serious disorders, particularly if personalized, and modified, after therapeutic monitoring [24-26]. However, we have to care a "unique" individual, a "single patient" with particular 'QBS Constitutions', 'Single Patient Based Medicine' is based on. In fact, we must consider accurately in the "single" patient his (her) whole 'QBS Constitutions' [2-5]. Mediterranean diet may prevent cancer because it contributes to diminish as far as normalize tissue acidosis and re-equilibrate acid based balance [6-8]: firstly it ameliorates and then it normalized the mitochondrial activity, always impaired in case of pathology or congenital risk of disease.

In fact, as evidenced in the first chapter, according to QBS theory, tissue pH is related to the reduction of latency time and to the extension of the duration of the Oncological Gastric Aspecific Reflex. By mean of the above mentioned diet, the latency time of the G.A.R. rises, and the duration of the reflex slows down, both tending to physiological levels.

b) Coenzyme Q10 in Oncological Therapy

The present literature underlines the clinical benefits of Coenzyme Q10 (CoQ10) in different disorders, as in cancer therapy [27-34]. Since all common and serious human disorders are based on CAEMH, as mentioned above, ubidecarenone utilization in cancer primary prevention is justified on the ground of its central action mechanism.

The present understanding of the central bio-energetic role of CoQ10 in mitochondrial redox metabolism and phosphorylation of ADP was well demonstrated [27, 30, 35, 36].

Analogously to Conjugated-Melatonin[8] multiple action mechanisms, Coenzyme Q10[9] ameliorate mitochondrial function, impaired in some biological systems in individuals positive for CAEMH. As a consequence, the use of both drugs has shown to be really efficacious in a lot of disorders [37-44], including cancer, especially when administered in earliest stage, i.e., in individuals apparently healthy, but positive for cancer 'Inherited Real Risk' [6, 7, 45].

Anti-aging effect of the antioxidant containing foods and various anti-oxidants, such as coenzyme Q10, was studied just in animals [46]. A clinical study aimed at evaluating the therapeutic efficacy of C0Q10 for primary prevention of osteoporosis in humans was done by one of the author. In spite of the small number of subjects treated (only 5) the results obtained are evidence of the efficacy of this agent which had never before been used in the therapy of osteoporosis. The possible mechanisms of action CoQ10 are discussed in the light of an original interpretation of the etiopathogenesis of this very complex bone disease [36].

c) Melatonin Action Mechanisms in Cell Metabolism

In a previous monograph, new action mechanisms of melatonin were described by one of the authors [47, 48]. In several researches melatonin proved to be really useful in ameliorating metabolism and tissue oxygenation, reducing

[8]We use melatonin conjugated with adenosine as prepared by Dr. Ferrari and Dr. Di Bella.

[9]We use UBIMAIOR 50, twice per day; no important side effects observed. The dose were controlled by bedside evaluation of Co Q10 Deficiency Syndrome [23 – 26]

tissue acidosis and normalizing the microcirculatory flow-motion in humans. The results obtained by other Authors indicate that melatonin treatment improve metabolism and it is useful for the treatment of Oncological disorders [49-66].

According to Stagnaro's [47], such as action mechanism of melatonin in ameliorating metabolism is more complex than generally admitted, including also both the positive effect on adiponectin synthesis and than its efficaciousness on lever, and parietal wall. As a matter of fact, adiponectin have showed a protective effect on metabolism in patients with type 2 diabetes mellitus [67, 68], corroborating the results in individuals with predisposition to degenerative pathologies and under different conditions [2-8, 64-65, 69].

3. A New Way of Therapy: The Quantum Biophysical Approach

Recent clinical experiments about quantum therapy in EHF (Extremely High Frequency) and BRR (Body Resonance Recording) regime showed to be useful in patients with cancer [70-72].

QBS tools are not only useful for diagnostic purposes, but also for therapeutic advices, because they are able to measure the microcirculatory activity before and after each preventive therapy's treatment, in order to understand the effectiveness of remedies.

Quantum Biophysical Semeiotics allows an accurate and direct study of condition and functioning of microvessels and only indirectly of the related parenchyma[10]. If the way of being and functioning of the microcirculation improves, it means that also the way of being and functioning of its parenchyma has improved.

Treatment and prevention, according to QBS, must be geared to improve and normalize metabolism, tissue oxygenation and mitochondrial respiratory chain function, expression of the normal operation of mitochondrial oxidative phosphorylation. Indeed, the mitochondrial functional cytopathy (CAEMH) is the *conditio sine qua non* of the more frequent and severe human diseases, like CAD, type I and II DM, and Cancer.

QBS has recently tested some treatments not yet experimented for preventive purposes as the quantum treatment mentioned above and the water thermal therapy [73]. We consider, among the several diagnostic parameters provided from QBS, the Latency time (Lt) of G.A.R., as illustrated in Chapter 1. In this case the physiological Lt is 8 seconds (NN = 8). If the basal value is less than 8 seconds, then there is Oncological Terrain and Inherited Real Risk of cancer (Table **1**, first column).

Under a continuative preventive therapy based on the combination of Modified Mediterranean Diet, CoQ10 and Conjugated-Melatonin the Lt rises to 12 seconds, so that the Inherited Risk of cancer becomes residual (Table **1**, fourth column) [1, 6, 7, 75]. By this way tissue oxygenation and mitochondrial activity are improved, mitochondria are running well, but the genetic alteration of mit-DNA still remains (CAEMH and Oncological Terrain are still positive). The news is given by the quantum therapy just mentioned, as follows.

We capture, i.e., the liver trigger points' radiations for one minute by means of a quantum device working in BRR mode[11], then we apply the device's crystals with the customized frequencies, on the same trigger points for 10 minutes. At this point the experimental and clinical evidences provided by QBS diagnosis and monitoring on more than 30 subjects at Risk of liver cancer confirm that the Oncological Terrain disappeared [75]. From this moment we observe a very high Microcirculatory Activity never seen before, denoted by a Lt of 16 seconds, which lasts for 7 days.

After a re-structuring period of time (7 days) the Lt slows down to 12 seconds, more than physiological one (Table **1**, fifth column). All QBS parameters from the beginning of the single unique application, till the time-out of genetic re-structuring time, and all QBS monthly diagnosis monitoring confirm the negativity of Oncological Constitution. After 9 months from the day of the unique device's application, the Microcirculatory Activation stops: this is the time-out of the normalization period. From this moment in time there are not anymore biological evidences of quantum treatment in progress, and the Oncological Constitution continue to be negative [74, 75].

Furthermore, we discover that hot springs have great therapeutic properties: by the same way of the quantum treatment above mentioned, after drinking sulfuric thermal water the Oncological Constitution disappears, and the QBS parametrical values are even better than those induced by the quantum treatment: Lt during the genetic re-structuring length of time rises to 20 seconds, before normalizing to 12 seconds for 9 months (Scheme **1**, sixth column) [73, 74].

Recent experiments [74, 75], undertaken with the same group above mentioned, have shown that quantum therapy in

[10]The micro-circulatory remodeling is directed by the way of living and working on the parenchyma: if the subject is healthy, the related parenchyma on the microcirculation is healthy (see angiobiopathy theory, dealing with diseases of blood and lymph vessels in accordance with QBS). Certainly a loss, rheumatism, immune, infectious, can act both directly and indirectly. See [http://www.semeioticabiofisica.it/microangiologia/common.htm]. It may be that in the long run re-organization becomes difficult or impossible because the flow decreases more, and then are built up of feedback mechanisms for which are to activate dormant cancer cells. Aging with free radicals that accumulate contributes to further damage both micro vascular and parenchymal: even endothelium (cell layers lining the inner surface of blood vessels and heart chambers) and smooth muscle cells possess mitochondria. Remodeling micro circulatory type cancer is an expression of mutations of genes within cells in that forum: any change in gene expression - cell finds its expression in the parallel alteration of its microcirculation (tissue microvascular units): the tissue here is around the vessels, interstitial, not the parenchyma! If these processes are blocked, the entire organization stops. Very important is that if there are congenital abnormalities, genetically transmitted through the mother (see CAEMH, mitochondrial cytopathy or mitochondrial functional pathology in the site www.semeioticabiofisica.it) amending the unfolding vital physiological processes occur the most serious human diseases, and not, now real epidemics. Autopoietic networks must therefore regenerate themselves continuously in normal and physiological way, to maintain its organization.

[11]'Quantum therapy' in this paper stands for capturing radiations from the body trigger points for 60 seconds, modulated at 10 Hz, and re-transmitting the same modulated frequencies for 10 minutes in the same place. BRR mode works with a semi-conductor of microwaves (millimeter waves) whose frequencies are in the range 35 – 70 GigaHz.

BRR mode and sulfuric thermal water are able to act and feed back to higher levels, directly on the causes of the diseases, such as healing the alteration of maternal mit-DNA and 'QBS Constitutions', in accordance with the Principle of Recursive Genome Function - PRGF by Pellionisz [76, 77] who argues the chance of a direct bi-directional communication's feedback between DNA and proteins. QBS clinical and experimental evidences have been analyzed and related to PRGF, in order to understand if the genetic alterations of mit-DNA could be reversed, due to the recursive energy, information and communication feedback between DNA, RNA and downstream structures such as tissues, cells, mitochondria and proteins. These evidences [78] are consistent with and fully confirm the above mentioned Principle.

4. CURRENT & FUTURE DEVELOPMENTS

Mediterranean Diet, CoQ10 and Conjugated-Melatonin were successfully tested in Oncological primary prevention and therapy, in accordance with QBS theory. They are able to reduce tissue acidosis improving tissue oxygenation and mitochondrial activity, but the genetic alteration of mit-DNA still remains (CAEMH and 'Oncological Constitution' continue to be positive). Recent positive clinical and experimental evidences provided by a quantum therapy able to capture and re-transmit customized frequencies from Oncological trigger points suggested us to test the preventive effectiveness of this treatment through the assessment of QBS parameters. This quantum therapy allows to improve mitochondrial and endothelial function, and furthermore to heal the 'Oncological Terrain'. The water therapy by means of sulfuric thermal water provides similar results as well as those offered by the quantum treatment.

According with QBS remarks, a new efficient Primary Prevention of cancer can be performed, on very large scale in individuals, involved by 'Oncological QBS Constitution' "and" 'Inherited Real Risk' of cancer, which have to undergo the above-mentioned treatment, rationally prescribed, and bed-side monitored.

CONFLICT OF INTEREST

The author(s) confirm that this article content has no conflicts of interest.

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REFERENCES

[1] Stagnaro S, Caramel S. The role of mediterranean diet, coq10 and conjugated-melatonin in osteoporosis primary prevention and therapy. Curr Nutr Food Sci 2012; 8: 55-2.

[2] Stagnaro S. Istangiopatia congenita acidosica enzimo-metabolica. Una patologia mitocondriale ignorata. Gazz Med It Arch Sci Med 1985; 144, 423.

[3] Stagnaro S. Rivalutazione e nuovi sviluppi di un fondamentale metodo diagnostico: la percussione ascoltata. Atti Accademia Ligure di Scienze e Lettere. Vol. XXXIV, 1978.

[4] Stagnaro S. Reale Rischio Semeiotico Biofisico. I Dispositivi Endoarteriolari di Blocco neoformati, patologici, tipo I, sottotipo a) oncologico, e b) aspecifico. Ed. Travel Factory, Roma, 2009.

[5] Stagnaro S, Stagnaro-Neri M. Le Costituzioni Semeiotico-Biofisiche. Strumento clinico fondamentale per la prevenzione primaria e la definizione della Single Patient Based Medicine. Ed. Travel Factory, Roma, 2004.

[6] Caramel S, Stagnaro S. The role of mitochondria and mit-DNA in oncogenesis. Quantum Biosystems 2010; 2: 250-81.

[7] Stagnaro S, Stagnaro-Neri M. Introduzione alla semeiotica biofisica. il terreno oncologico. Ed. Travel Factory, Roma, 2004.

[8] Curri SB. Le Microangiopatie. Inverni della Beffa, Milano, 1986.

[9] Bucciante L. Anastomosi arterovenose e dispositivi regolatori del flusso sanguigno. Mon zool it 1949 ; 57 : 3-10,

[10] Pratesi F. Microcircolazione e microangiologia. fisiopatologia, clinica e terapia. Ediz. Minerva Medica, Torino, 1990.

[11] Goldberger AL, West BJ. Applications of non-linear dynamics to clinical cardiology, ANN. N.Y. Acad Sci 1987; 504: 195.

[12] Stagnaro S, Stagnaro-Neri M. Deterministic chaotic biological system: the microcirculatory bed, Gazz Med It-Arch Sci Med 1994; 153: 99.

[13] Stagnaro S. Role of coronary endoarteriolar blocking devices in myocardial preconditioning - c007i. Lecture, V Virtual International Congress of Cardiology. 2007; Available at: http://www.fac.org.ar/qcvc/llave/c007i/stagnaros.php

[14] Hammersen F. Zur ultrastruktur der arterio-veno¨sen anastomosen. In: Hammersen F, Gross D (eds). Die Arterio-venoesen Anastomosen Anatomie, Physiologie, Pathologie, Klinik. Verlag Hans Hubert: Bern und Stuttgart. pp 24–37, 1968.

[15] Stagnaro-Neri M, Stagnaro S. Auscultatory percussion evaluation of arterio-venous anastomoses dysfunction in early arteriosclerosis. Acta Med Medit 1989; 5: 141-4.

[16] Stagnaro-Neri M, Stagnaro S. Diagnosi clinica precoce dell'osteoporosi con la percussione ascoltata. Clin Ter 1991; 137: 21-7.

[17] Stagnaro S. Bedside diagnosis of osteoporotic constitution, real risk of inheriting osteoporosis, and finally osteoporosis. Theor Biol Med Model, 2007; 4: 23

[18] Caramel S. Coronary Artery Disease and Inherited Real Risk of CAD, JOQBS, 2010. Available at: http://www.sisbq.org/uploads/5/6/8/7/5687930/cad_caramel.pdf

[19] Huijbregts P, Feskens E, Rasanen L, Fidanza F, Nissinen A, Menotti A. Dietary pattern and 20 year mortality in elderly men in Finland, Italy, and the Netherlands: longitudinal cohort study. BMJ 1997; 315:13-7.

[20] World Cancer Research Fund and American Institute for Cancer Research. Food, nutrition and the prevention of cancer: a global perspective. Washington DC: WCRF and AICR, 1997.

[21] Trichopoulou A. Modified Mediterranean diet and survival: EPIC-elderly prospective cohort study. BMJ 2005; 330: 991

[22] Knoops KT, de Groot LC, Kromhout D, Perrin AE, Moreiras-Varela O, Menotti A. Mediterranean diet, lifestyle factors, and 10-year mortality in elderly European men and women: the HALE project. JAMA 2004; 292: 1433-9.

[23] Werbach MR. Melatonin for the treatment of gastroesophageal reflux disease. Altern Ther Health Med 2008; 14: 54-8.

[24] Bonaccio M, Iacoviello L, de Gaetano G. On behalf of the molisani investigators. The Mediterranean diet: The reasons for a success. Thromb Res 2012; 129: 401-4.

[25] Demarin V, Lisak M, Morović S. Mediterranean diet in healthy lifestyle and prevention of stroke. Acta Clin Croat 2011; 50: 67-77.

[26] Salas-Salvadó J, Martinez-González MÁ, Bulló M, Ros E. The role of diet in the prevention of type 2 diabetes. Nutr Metab Cardiovasc Dis 2011; 21: B32-48.

[27] Society of obstetricians and gynaecologists of Canada. The Canadian consensus conference on menopause and osteoporosis, 2002 update [SOGC guideline no 108]. Ottawa: The Society; 2002. Available at: http://www.sogc.org/guidelines/public/108E-CONS1-Sep-Dec2001.pdf

[28] Stagnaro-Neri M, Stagnaro S. Acidi grassi Ω-3, scavengers dei radicali liberi e attivatori del ciclo Q della sintesi del CoQ10. Gazz Med It-Arch Sci Med 1992; 151, 341-5.

[29] Lenaz G, Parenti Castelli G. Multiple roles of ubiquinone in mammalians cells. Drugs Exptl Clin Res 1984; 10: 481-5.

[30] Lubich T, Bargossi AM, Fiorella PL. Metabolic effects of coenzyme Q treatment in high level athletes. VI Simposio Internazionale su: Biomedical and clinical aspects of coenzyme Q. Chairmen:

Folkers K, Littarru GP, Yamagamy Y. 22-24 gennaio 1990, Roma. Abstracts, 70.

[31] Stagnaro S, Stagnaro-Neri M. Carenza di CoQ10 secondaria a terapia ipolipidemizzante diagnosticata con la percussione ascoltata. Settimana Italiana di Dietologia. Merano 9-13 aprile 1990. Com.. Abstract. 105 e Epatol 1991; 37: 17-22.

[32] Stagnaro-Neri M, Stagnaro S. Auscultatory Percussion Coenzyme Q deficiency Syndrome. VI Simposio Internazionale su: Biomedical and clinical aspects of coenzyme Q. Chairmen: Folkers K., Littarru G.P., Yamagamy Y., 22-24 gennaio 1990, Roma. Abstracts, 104.

[33] Stagnaro S. Ipercolesterolemia e Coenzima Q10. The Pract Ed It 1990; 133: 5-6.

[34] Stagnaro-Neri M, Stagnaro S**.** Sindrome clinica percusso-ascoltatoria da carenza di Co Q10. Medic Geriatr 1993; XXIV: 239-51.

[35] Mitchel P. The vital protonmotive role of Coenzyme Q. VI Simposio Internazionale su: Biomedical and clinical aspects of coenzyme Q. Chairmen: Folkers K., Littarru G.P., Yamagamy Y., gennaio 1990, Roma. Abstracts, 77.

[36] Neri Stagnaro M, Stagnaro S. CoQ10 in the prevention and treatment of primary osteoporosis. Preliminary data. Clin Ter 1995; 146: 215-9.

[37] Baker SK, Tarnopolsky MA. Statin myopathies: pathophysiologic and clinical perspectives. Clin Invest Med 2001; 24: 2258-72.

[38] Leung NM, Ooi TC. Use of statins and fibrates in hyperlipidemic patients with neuromuscular disorders. Ann Intern Med 2000; 132: 418-9.

[39] Gutierrez-Mariscal FM, Perez-Martinez P, Delgado-Lista J, *et al*. Mediterranean diet supplemented with coenzyme Q10 induces postprandial changes in p53 in response to oxidative DNA damage in elderly subjects. Age (Dordr) 2012; 34: 389-403.

[40] Ochoa JJ, Pamplona R, Ramirez-Tortosa MC, *et al*. Age-related changes in brain mitochondrial DNA deletion and oxidative stress are differentially modulated by dietary fat type and coenzyme Q□□. Free Radic Biol Med 2011; 50: 1053-64.

[41] Quinzii CM, Hirano M. Coenzyme Q and mitochondrial disease. Dev Disabil Res Rev 2010; 16:183-8.

[42] López LC, Quinzii CM, Area E, *et al*. Treatment of CoQ(10) deficient fibroblasts with ubiquinone, CoQ analogs, and vitamin C: time- and compound-dependent effects. PLoS One 2010; 5: e11897.

[43] Cordero MD, Alcocer-Gómez E, de Miguel M, *et al*. Coenzyme Q(10): A novel therapeutic approach for Fibromyalgia? case series with 5 patients. Mitochondrion 2011; 11: 623-5.

[44] Sourris KC, Harcourt BE, Tang PH, Morley AL, Huynh K, Penfold SA, Coughlan MT, Cooper ME, Nguyen TV, Ritchie RH, Forbes JM. Ubiquinone (coenzyme Q10) prevents renal mitochondrial dysfunction in an experimental model of type 2 diabetes. Free Radic Biol Med 2012; 52: 716-23.

[45] Stagnaro S, Stagnaro-Neri M. Single Patient Based Medicine. La Medicina Basata sul Singolo Paziente: Nuove Indicazioni della Melatonina. Ed. Travel Factory, Roma, 2005.

[46] Takahashi R. Anti-aging studies on the senescence accelerated mouse (SAM) strains. Yakugaku Zasshi 2010; 130: 11-8.

[47] Stagnaro S, Stagnaro-Neri M. La Melatonina nella Terapia del Terreno Oncologico e del "Reale Rischio" Oncologico. Ed. Travel Factory, Roma, 2004.

[48] Stagnaro S. Endothelial cell function can ameliorate under safer drugs, such as Melatonin-Adenosine. BMC Cardiovascular disorders. 2004; 4: 4

[49] Suzuki N, Somei M, Seki A, Reiter RJ, Hattori A. Novel bromomelatonin derivatives as potentially effective drugs to treat bone diseases. J Pineal Res 2008; 45: 229-34.

[50] Maldonado MD, Pérez-San-Gregorio MA, Reiter RJ. The role of melatonin in the immuno-neuro-psychology of mental disorders. Recent Pat CNS Drug Discov 2009; 4: 61-9.

[51] Paternoster L, Radogna F, Accorsi A, Cristina Albertini M, Gualandi G, Ghibelli L. Melatonin as a modulator of apoptosis in B-lymphoma cells. Ann N Y Acad Sci 2009; 1171: 345-9.

[52] Cutando A, Aneiros-Fernández J, Aneiros-Cachaza J, Arias-Santiago S. Melatonin and cancer: current knowledge and its application to oral cavity tumours. J Oral Pathol Med 2011; 40: 593-7.

[53] Lemoine P, Wade AG, Katz A, Nir T, Zisapel N. Efficacy and safety of prolonged-release melatonin for insomnia in middle-aged and elderly patients with hypertension: a combined analysis of controlled clinical trials. Integr Blood Press Control 2012; 5: 9-17.

[54] Carpentieri A, Díaz de Barboza G, Areco V, Peralta L.M, Tolosa de Talamoni N. New perspectives in melatonin uses. Pharmacol Res. 2012; 65: 437-44.

[55] Sanchez-Barcelo EJ, Mediavilla MD, Alonso-Gonzalez C, Rueda N. Breast cancer therapy based on melatonin. Recent Pat Endocr Metab Immune Drug Discov 2012; 6: 108-16.

[56] Kesemenli C, Necmioğlu S. The role of melatonin as a link between head injury and enhanced osteogenesis. Med Hypotheses 2003; 65: 605-6.

[57] Steinhilber D, Brungs M, Werz O, *et al*. The nuclear receptor for melatonin represses 5-Lipoxygenase gene expression in human B Lymphocytes. J Biol Chem 1995; 270:7037-40.

[58] Susko I, Mornjakovic Z, Alicelebic S, Cosovic E, Beganovic A. Retinal and pineal melatonin-from a circadian signal to therapeutic use. Med Arh 2004; 58: 61-4.

[59] Pablos MI, Guerrero JM, Ortiz GG, Agapito MT, Reiter RJ. Both melatonin and a putative nuclear melatonin receptor agonist CGP 52608 stimulate glutathione peroxidase and glutathione reductase activities in mouse brain *in vivo*. 1998; 18: 49-58.

[60] Reiter RJ. Neuroendocrinology of melatonin. In: Melatonin-Clinical perspectives. Edited by Miles *et al*. Oxford University Press, 1988; 1-24.

[61] Kopin IJ, Pare CMB, Axelrod J, *et al*. The fate of melatonin in animals. J Biol Chem 1961; 236: 3072-5.

[62] Eileen ML. Melatonin and cancer treatment. Le Magazine, January 2004.

[63] World cancer research fund and American institute of cancer research. Diet, nutrition and the prevention of the cancer. Banta Book; 1997.

[64] Rodriguez-Reynoso S, Leal C, Portilla-de Buen E, Castillo JC, Ramos-Solano F. Melatonin ameliorates renal ischemia/reperfusion injury. J Surg Res 2004; 116: 242-7.

[65] Leone AM, Francis PL, Silman RE. The isolation, purification, and characterization of the principal urinary metabolites of melatonin. J Pineal Res 1987; 4: 253-66.

[66] Sanchez-Hidalgo M, Guerrero JM, Villegas I, Packham G, De La Lastra CA. Melatonin: a natural programmed cell death inducer in cancer. Curr Med Chem 2012; 19: 3805-21.

[67] Stagnaro S. Il test Semeiotico-Biofisico della Osteocalcina nella prevenzione primaria del diabete mellito, 2008. Available at: http://www.fcenews.it/index.php?option=com_content&task=view&id=909&Itemid=47

[68] Stagnaro S, West PJ, Hu FB, Manson JE, Willett WC. Diet and risk of type 2 Diabetes. N Engl J Med 2002; 346: 297-8.

[69] Davis PF, Barbee KA. Hemodynamics and Atherogenesis. Endothelial surface dynamics in flow signal transuction. recent advances in atherosclerosis research. Annals of the New York acadèmy of science. 1995; 748: 86-103.

[70] Teppone MV, Avakyan RS. EHF-therapy in Oncology. Millimeter waves in biology and medicine. PAPH Moscow Regional Oncology Dispensary 2003. N(1): 3-19.

[71] Kabisov RK. Millimeter waves in Oncology: reality, problems, perspectives. The P.A. Hertsen Moscow SR Oncologic Institute. Millimeter waves in biology and medicine. 1992; N(1): 55-61.

[72] Kozhemyakin A. Report on clinical evaluation apparatus for EHF-IR therapies device portable with changeable oscillators "Cem Tech". Spinor O'LTD, 2008. Available at: http://www.schungit-mineralien.de/CEM-TECH/docs/Ergebnisse-klinik-eng.pdf

[73] Caramel S. Meccanismi d'azione sconosciuti dell'acqua termale sulfurea. JOQBS, 2012. Available at: http://www.sisbq.org/uploads/5/6/8/7/5687930/h2s.pdf

[74] Stagnaro S, Caramel S. A new way of therapy based on water memory information: the quantum biophysical approach, JOQBS, 2011. Available at: http://www.sisbq.org/uploads/5/6/8/7/5687930/qbtherapy.pdf

[75] Stagnaro S, Caramel S. The genetic reversibility in Oncology. JOQBS, 2011. Available at: http://www.sisbq.org/uploads/5/6/8/7/5687930/reverse_oncology.pdf

[76] Pellionsz AJ. The principle of recursive genome function. Cerebellum 2008; 7: 348-59.

[77] Pellionisz AJ, Graham R, Pellionisz PA, Perez JC. Recursive genome function of the cerebellum: geometric unification of neuroscience and genomics. Chapter in Handbook: "The Cerebellum and cerebellar disorders" Ed. Dr. Mario Manto, Springer Verlag, 2011.

[78] Stagnaro S, Caramel S. The principle of recursive genome function: QBS clinical and experimental evidences. JOQBS, 2011. Available at: http://www.sisbq.org/uploads/5/6/8/7/5687930/prgf_qbsevidences.pdf

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