

## Metabolic Failure as the Cause of Fibromyalgia Syndrome: Exploring the John C. Lowe Thesis

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**ABSTRACT. Introduction and Aim.** The aim of this one year project was to investigate a thesis by John C. Lowe: that fibromyalgia syndrome (FMS) is a hypometabolic condition caused by a reduced effect of thyroid hormones at the cellular level, due either to (1) a deficiency of adequate thyroid hormone (hypothyroidism), or (2) a reduced cellular effect of thyroid hormone despite its production of reference range quantities (partial peripheral thyroid hormone resistance).

**Material and Methods.** 56 female patients with a clinical picture of “classical FMS.” The diagnosis of FMS was verified by specialists for 27 of the patients. Although 29 patients had the classical signs and symptoms of FMS, they were waiting for their diagnosis to be verified by specialists. These 29 patients were excluded from the investigation until a later date when a qualified specialist will verify beyond doubt their correct diagnostic label.

To test Lowe’s thesis, a questionnaire was made. The basis of the questionnaire was the already-existing statistics of symptoms and signs of hypothyroid patients who had not yet achieved metabolic restoration by the use of thyroid hormone medication. The questionnaire was filled out by all 56 patients. The answers from all patients were collected and put into a database. The answers of patients with verified FMS were extracted and compared to the statistical material upon which the questionnaire was made.

In addition, all 27 patients with verified FMS were tested for their thyroid hormone status at the local hospital. Thirteen of the patients were tested with a method developed by Professor Louis-Claude Van Vincent,<sup>[45]</sup> “Van Vincent’s Bio-Electrical-Terrain-Analysis” (Van Vincent BE-T-A). BE-T-A is a system that evaluates the metabolic status of mesenchymal (structurally supportive) and parenchyma (essentially producing) tissues.<sup>[16]</sup>

**Results.** Comparing the questionnaire answers of the 27 FMS patients with the statistics from hypothyroid patients, the frequencies of symptoms fit a pattern of untreated hypothyroidism. The Van Vincent BE-T-A test showed an acidosis of mesenchymal tissues, such as connective tissues and lymphatic and blood vessels. The acidosis can be explained by a lack of thyroid hormone effect at the cellular level, leading to an increase in parenchymal anaerobic metabolism, in which biological processes proceed in the absence of oxygen.

**Conclusion.** The results of questionnaire analysis, use of the Van Vincent BE-T-A testing, and the author’s clinical experience are consistent with the Lowe thesis that metabolic failure underlies FMS. The cause of the failure is plausibly explained by deficiencies of thyroid hormones or reduced cellular effects of the hormones. There are two natural solutions to this problem: (1) increase the amount of thyroid hormones available to the tissues, and (2) normalize the cellular effects of thyroid hormones by removing whatever hinders their normal effects.

**KEYWORDS:** Fibromyalgia syndrome, hypothyroidism, thyroid hormone resistance, Van Vincent BE-T-A test, Bio-Electrical-Terrain-Analysis, energy medicine, John C. Lowe Thesis

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### INTRODUCTION AND AIM

*“The problem is not lack of knowledge, but knowing what we already know”* • Norwegian author Wilhelm Schjeldrup MD

#### PART I: A MEDICAL PROBLEM, BUT NO SOLUTION

**Medical Science in Need of New Concepts.** Medical science has a long standing history for sacrificing complexity to simplicity by using the tradition of “diagnosing”—giving one single noun to disease-entities that often covers a wide range of symptoms and signs;

often going to extreme dichotomy by neglecting the fact that a body is one whole: body, mind, and soul. As observed by O’Connor: “Medical diagnoses are nominalizations. They are substantives, but every disease is a process, and changes during the process of therapy.”<sup>[1]</sup> Such simplemindedness, which omits a host of information, can be justified for simple cases like a broken leg, a torn tendon, or a cut in the skin; where just a single noun is sufficient to transmit inherent meaning; but dealing with complex diseases, single nouns pointing to simple entities fail their purpose dramatically.

Fibromyalgia, the topic of this paper, is not a “simple, fixed entity.” It cannot be clearly identified by a set

of criteria; experience with in depth study of patients given the diagnosis shows a broad spectrum of symptoms, signs, and objective findings.<sup>[2]</sup> The spectrum is indeed so broad that the noun “syndrome” is proper, denoting a variety of symptoms, signs, and objective findings that can vary in number and intensity over a spectrum of possibilities.<sup>[3]</sup>

Recording the finer details leads to even deeper realizations that fibromyalgia syndrome, due to its flexible nature, ought not be regarded as a simple entity. Instead, it should be considered a condition more on the order of “functional diseases.” Such diseases is a new concept formed in the latter part of the 20<sup>th</sup> century when cybernetics and the idea of function entered medicine from the science of physics and biology. Using system thinking and cybernetics, FMS is best seen as a system failure to achieve maximum functionality and energy production, resulting in a host of energy-depletion signs, symptoms, and findings.<sup>[4]</sup>

However, in the history of medicine, doctors have a long tradition of lagging behind current advances in thinking and technology. They have, despite “modern times,” kept searching for simpler “one-noun, one-entity concept” that adheres to the unspoken diagnostic tradition. This thereby pleasing their peers who, for some reason, have built empires upon simple and manageable doctrines that in due time have become the credo of the profession as to what or what not to believe.<sup>[5]</sup>

However, from time-to-time comes great minds to shake up the public, and the recent works of a number of American researchers have brought to light the fact that what is considered disease entities are but fleeting changes in the human framework, as energy is reduced in a number of steps to save life from harmful influences: destruction by environmental toxins,<sup>[6]</sup> lack of proper nutrients,<sup>[7]</sup> psychological and social stress,<sup>[8]</sup> electromagnetic pollution,<sup>[9]</sup> and the misuse of potent modern drugs that have serious side effects meant to be used only for a short time span.<sup>[10]</sup> Due to such influences, we see a decline in the human adaptation system and immunological defense, which in turn lays the body open to all sorts of microbes (natural or man made).<sup>[11]</sup>

**John C. Lowe Discovery: FMS—Just Like Hypothyroidism.** One of these giants among minds is current researcher and science writer John C. Lowe who has delivered a major blow to the concept of one name, one diagnosis, one pathology. His book *The Metabolic Treatment of Fibromyalgia*<sup>[12]</sup> will stand as one of the major intellectual efforts of this century to speak out for a functional interpretation of human suffering. According to the book, FMS and also chronic fatigue syndrome (CFS) are not fixed entities easily contained in the

meaning of the one-noun tradition. Instead, they are complex phenomena resulting from lack of energy produced per-unit body mass to sustain proper functions and life under the ruling conditions of life.<sup>[12,pp.295-341]</sup> Thereby, he has taken away FMS and CFS from the backwaters of nominalization to where real life is, and described the process to which the diagnostic noun points.

Looking to the underlying processes and the resulting clinical scenarios, rather than to the semantics, Lowe has discovered that FMS and CFS patients have exact, if not identical, manifestations of symptoms as people deficient in thyroid hormone, whom we say have “hypothyroidism.”<sup>[12,pp295-341]</sup>

The exception is that in the two former “diseases,” FMS and CFS, traditional testing does not show a deficiency of thyroid hormones. Therefore, patients appear to be “euthyreots.” That is, they appear to have “have sufficient thyroid function compared to a sample of normally functioning human beings.” But facts speak for themselves: these patients have most if not all the symptoms and signs of people diagnosed as being hypothyroid.<sup>[12,pp1107-1127]</sup>

In addition, Lowe has recently found in two well-controlled studies, using indirect calorimetry, that FMS patients—some of whom were euthyreots—had significantly lower resting metabolic rates and basal body temperatures than matched healthy controls.<sup>[86][87]</sup>

According to Lowe, it is therefore intellectually proper to term these two groups of patients, and especially fibromyalgia patients (about which most of the book is written), as hypometabolic, despite being “euthyreot.”<sup>[12,pp243-288]</sup> Logically, the patients’ cells must be somehow resistant to the influence of hormones from the thyroid gland.<sup>[17]</sup> This concept is new to most doctors, but there is no way around the situation: If your laboratory tests spell “hormones within their ranges,” but the clinical picture tells you that the effect of the hormones are lacking, what other choice is there but to call the patient’s condition “hormonal resistance” or “reduced effects of thyroid hormones”?<sup>[18]</sup>

## MATERIALS AND METHODS

*“To find gold you either need a good map or Grace of the gods.”* ● Norse saying

### PART II: MAPPING ENERGY: DEPLETED TERRAIN

**The Van Vincent Test: Mapping Hypometabolic Terrain.** My own experience with Van Vincent testing dates some years back. As in this study, I have perform-

ed Van Vincent testing, taking measurements with equipment customarily used with our cancer patients, from the German company MedTronik.

Through this testing, I discovered that many patients with “unclear clinical pictures” and who were doomed to suffer from “inexplicable diseases,” or described as “psychosomatic cases,” were instead suffering from too little energy.

At that time, within the ordinary health care system in Norway, there was little-to-no lab service available to substantiate a diagnose of too little energy. So I had to rely on clinical evaluations and act accordingly, often with very good results.<sup>[15]</sup> But realizing that in the long run, the situation was unbearable, we finally invested in BE-T-A equipment. “BE-T-A” is an abbreviation for Bio-Electronic-Terrain-Analyzer.

The BE-T-A methodology makes it possible to quantify cellular metabolism by making a statistical analysis of bio-physical parameters of venous blood, sputum, and urine.<sup>[16]</sup>

I will explain the methodology: The BE-T-A method is a typical “Old world” bio-medical device rooted in the marriage of medicine, technology, and holistic thinking that emerged in Europe in the fruitful years from 1920-to-1960. The technique and methodology was single-handedly developed in Strasbourg in 1946 by French hydrologist Professor Louis-Claude Van Vincent. He originally designed the instrument to evaluate the quality of drinking water.<sup>[17]</sup>

Van Vincent could give a good picture of the quality of water by using three well-known physical measurements: pH (acidity),  $rH_2$  (electron activity), and R (resistance).

During 1952 and 1953, while working in Lebanon, he began using his technique on human fluids: saliva, urine, and venous blood. As Elmau wrote, he “was able to show on a large patient base that the physical or bio-electronic measurement values pH,  $rH_2$ , and R—depending on how they relate to each other—are sufficient to indicate whether the organism is sick or healthy.”<sup>[17,p.iii]</sup>

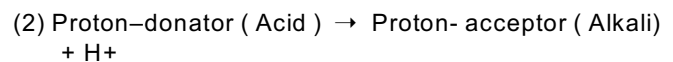
As a result of his pioneering work, Van Vincent was appointed Professor at Ecole d'Anthropologie in Paris. There he continued to build his database and refine his technical equipment. In the 1960ies, his method was introduced to the German-speaking world by Dr. Med. Franz Morell.

Later on, Van Vincent's method spread to all parts of the world. The first world congress on his method was held in Königstein in 1976. The proceedings of the International Society of Bioelectronic Van Vincent have since been applied to veterinary and human medicine, oncology, the evaluation of drinking water, agriculture, hygiene, and the dairy industry.<sup>[17,p.iv]</sup>

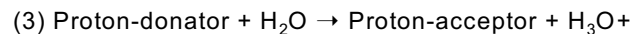
To understand the value of the Van Vincent meth-

odology, one must understand the three basic components, pH,  $rH_2$ , and R, as indicators of cellular energy production. I explain each of these below.

**pH: The Magnetic Factor.** pH is basically a measurement for proton activity. In chemistry, substances that can give away protons are acids, and those that steal protons are alkalies. This leads to the standard textbook wisdom we learnt “back in School,” give which goes back to the Danish chemist Bønsted, who in 1923 defined the terms:



When this reactions takes place in water it may look somewhat like:



pH is equal to the negative decadic logarithm of the concentration of protons in a given fluid. This turns out to be a measurement of proton activity in that fluid. The formula holds good for the range 4.0 to 9.2. For purified water at 18° Celsius, pH = 7.105. At 24° Celsius, pH = 7.0, and at 37° Celsius, pH = 6.78. This latter pH, 6.78, is optimum for kidney activity.

The difference in pH = 1 equals a factor of 10 in proton activity. At pH = 6.8, there is 160 nanomols of dissociated protons per liter of fluid. At pH = 7.4, there is only 40 nanomols of dissociated protons per liter. At theoretical pH = 0, there is a maximum proton activity (dissociated protons), and at pH = 14 there is no proton activity at all.

The technical side to the problem is taken care of with a glass-electron chain using two separate metal electrodes separated by a proper material. The electrode must be calibrated once a week and cleaned properly. If properly taken care in this way, it will have an accuracy of 0.02-to-0.04.

Measuring urine pH using dry chemistry, as with “Lab-stix,” is often cherished in clinical practice. However, an interesting discovery is that, in comparison with dry chemistry, using electrodes to measure urine pH is highly accurate. (The dry chemistry, as with “Lab-stix,” is often cherished in clinical practice. However, an interesting discovery is that using electrodes to measure urine pH is highly accurate.) By comparison, the dry chemistry method is highly inaccurate, giving low readings.<sup>[18]</sup>

Protons work like micro-magnets. Because of this, in a very proton-rich milieu (that is, a low pH or acidic milieu), there will be a stronger magnetic field than in an

alkaline milieu.<sup>[25]</sup> pH thus is also an indirect measurement of a fluid's magnetic properties.

Even these considerations may be too simple for one to get the "whole picture." For example, according to Davis and Day, pure water is a mixture of 18 different molecular forms and 15 kinds of ions. These give rise to 36 different types of substances which may be in a mixture that, for simple reasons, we agree to call "water."<sup>[16,pp67-70]</sup>

**rH<sub>2</sub>: The Electron Activity Factor.** The other entity in BE-T-A technology is rH<sub>2</sub>. This is a measurement of the amount of negative electricity that is uniquely bound to the electron (e-). According to the Clark equation, rH<sub>2</sub> is defined as:<sup>[27]</sup>

(4) Definition:  $rH_2 = -\log(PH_2)$

P stands for partial pressure exhibited by any gas. PH<sub>2</sub> stands for the partial pressure of hydrogen gas. Hydrogen gas arises due to the fact that water H<sub>2</sub>O gives rise to both H<sup>+</sup> ions and OH<sup>-</sup> ions. If electrons are supplied to the water they will bind to protons: H<sup>+</sup> and from atomic hydrogen, H. The atom of hydrogen will bind to another hydrogen atom formed in the same manner, creating hydrogen gas. The amount of gas can be measured by a proper probe immersed in the fluid. With a calibrated meter, we can then read rH<sub>2</sub> on a scale.

In water, there is a balance between H<sub>2</sub> and O<sub>2</sub>. The balance occurs because the surplus of OH<sup>-</sup> freed when H<sup>+</sup> goes to hydrogen gas forms oxygen (O<sub>2</sub>). In water rH<sub>2</sub> will vary between 0 and go to +42. The neutral point of rH<sub>2</sub> is 28, where PH<sub>2</sub> = PO<sub>2</sub>.

Below the neutral point of rH<sub>2</sub> (< 28) there is a surplus of electrons. Water becomes electronegative, and the partial pressure of oxygen is less than that of hydrogen. Above the neutral point of rH<sub>2</sub> (> 28), the situation is reversed.

For venous blood, the neutral point is defined differently, as the mean for healthy persons is rH<sub>2</sub> = 22. Compared to water, then, venous blood is in a slightly reducing state.<sup>[20,pp.25-30]</sup>

rH<sub>2</sub> is thus a measurements of the amount of negative electricity (electrons), while the more wide known pH is a measurement for positive electricity (protons). Both processes are of equal importance because they are dependent on each other; this applies to rH<sub>2</sub> as much as to pH. Generally this is not taken into consideration because most doctors do not have access to the "hidden" factor rH<sub>2</sub>. But one should never forget: when there is an exchange of protons (pH), there is a simultaneous exchange of electrons (RH<sub>2</sub>).

Similar to the acid-alkali concept of pH, rH<sub>2</sub> leads to

the concept of reduction-oxidation:

(5) Electron-donator → Electron-acceptor + e-

According to Nygren-Wigren in 1964:<sup>[20,pp.25-30]</sup>

(6) Oxidation: the oxidating substance donates an electron

(7) Reduction: The reducing substance accepts an electron

In shorthand, this is often written:

(8) Red → Ox + N → e-

(N represents the number of electrons transferred.)  
When the reaction goes to the right Y, we name it "Oxidation"; when it goes to left Z, we call it "Reduction."

The equation says that in any liquid where reduction and oxidation takes place, there is a "Red-Ox" pair. But to what use is this? As will later be shown, energy in the human body is of an electro-chemical nature; shifting around electrons in a number of Red-Ox processes generates free energy; that energy can be stored in energy-rich molecules, such as ATP and NADH. *The shift of electrons and their transport on ions is what in physics is called an "electrical current (I)."*

We are now entering useful territory: If you have pH, you can go to your textbooks and deduct useful information about the production and transport of acids. This is useful if you remember that anaerobic metabolism creates lactic acid, acidifying the internal milieu. Furthermore, if you know your Red-Ox potential, you know at least two things: the flow of electrical currents in body fluids and, if you know rH<sub>2</sub>, you will have a fair estimate of free radicals, since free radicals are electron-grabbers and thus lower rH<sub>2</sub>.

pH and rH<sub>2</sub> are interrelated, as will be seen form the so-called "Nernst Red-Ox potential equation":<sup>[20,p.94-96]</sup>

(9) Definition:  $E = 30 \rightarrow (rH_2 - 2 \rightarrow pH)$

"E" *has to be calculated* on the basis of measurements of parameters rH<sub>2</sub> and pH. The unit used to measure E is volt (V). In biological samples as blood, urine, or sputum, E will be of the order mV (1 mV = 0.001 V). In biological systems, a number of feedback systems tend to keep E within narrow limits, making pH and rH<sub>2</sub> inter-related and mutually dependent. This can be seen if we rewrite equation 9 to read:

(10)  $rH_2 = 0.033 E + 2pH$

If a biological system wants to keep E within certain limits, this implies that when rH<sub>2</sub> increases, pH will follow and visa versa. This is verified by BE-T-A measurements. One may view this in a teleological manner: by keeping E within certain limits and adjusting pH to rH<sub>2</sub>, the body achieves the following—in a milieu with oxidative stress, the various fluids increase their amount of protons (pH goes up), thereby creating more protons to neutralize excessive electrons. Also, the excessive protons create a more intense magnetic field that can be regarded as an effort to promote self healing.<sup>[20,p.94-96]</sup>

**R: The Dielectric Factor:**

The third parameter, resistance R, is to calculate the flow of electrons. Knowing the Red-Ox potential E and R, one can calculate the flow of electrons I: the electrical current:

$$(11) I (\text{calculated}) = E (\text{calculated}) / R(\text{measured})$$

R is given in the unite Ohm (Ω) and was formerly named ρ (rho) in France. It is internationally known as the “di-electric factor.” It expresses the degree of ionization of a fluid under the influence of an electrical force.

R is dependent upon the presence of electrolytes in a fluid. In blood, the major players are natrium (sodium), which provides 92% of all positive electrolytes; chlorine provides 68% of all electrolytes. In practical situations, measurement of R is done with electrodes that are calibrated in a 0.1 mol KCl, corrected according to temperature.<sup>[32]</sup> Having these four entities we have a knowledge of free radicals, the acid-alkaline status, and electrolyte state. By doing enough tests on a number of healthy reference persons, using young sportswomen

measurements to compare healthy people with diseased people. His data for healthy, young individuals are given in Table 1 below<sup>[21]</sup>

**Table 1.** Ideal measured values according to Van Vincent.

Fluid	pH	rH <sub>2</sub>	R
Blood	7.34 (7.10)	22.0	Fluid pH rH <sub>2</sub> 210
Sputum	6.50	22.0	180 (140)
Urine	6.80	24.0	30

By the early 1950s, he learned that the two numbers in parenthesis typical of healthy young individuals. At that time, nutrition was more natural and drinking water less polluted.

It is thus interesting to see that venous blood in 2000 is more alkaline than 50 years back This indicates a decay in human capacity to excrete acids through the kidneys.<sup>[21]</sup> Sputum, which is an indication of the condition of intestinal secretion, tends these days to have a higher R factor. This is interpreted as meaning less ionized minerals in the intestinal secretion due to an overall lack of minerals in our present nutrition.

**The Acidic Mesenchyme (the Structurally Supporting Tissues of Organs).** The use of Van Vincent BE-T-A technology opens an interesting option for clinical investigation into metabolic states of the human body. Going into detail about this issue would demand a number of extra pages, so we focus on what is of interest to the Lowe-thesis: the reduction of energy production in parenchyma (the productive tissues of organs), and the subsequent degradation of the mesenchyma (like connective tissues and lymphatic and blood vessels) that will lead to an accumulation of acids in it. Our findings and the interpretation of these findings for 13 FMS patients is given in Table 2.

The pH of venous blood and sputum taken from FMS

**Table 2.** Reduce energy and accumulation of acids in mesenchyme of 13 FMS patients.

N=13	pH	rH <sub>2</sub>	R	Redox potential mV	Energy μW/cm <sub>3</sub>
Blood	7.44	25	199	303.6	463.1
Urine	5.64	20.77	143	285	566.8

and sportsmen from France, Van Vincent, over a period of several decades, collected a database for the “ideal human being.” He simultaneously compared these reference people to men and women of all ages with all sorts of diseases. Finally, he came up with revealing facts.

**Measurements on Healthy People:** Having established the technology, Van Vincent set out to do meas-

urements to compare healthy people with diseased individuals.

From the statistical works of Van Vincent and others, we know that this is typical for advancing age, stomach dysfunction, nephritis, liver failure, cancers, intestinal symbiosis, and, relevant to the Lowe-thesis, chronic metabolic disorders.<sup>[35][12]</sup> Since none of our patients had diagnosed cancers, old age, or other such conditions, the

test results are an indication of a chronic metabolic disorder. But what kind of metabolic disorder?

During the release of energy in chemical bonds, intracellular glucose is transformed to pyruvate. Pyruvate then enters the mitochondria. There it is converted to acetyl-Co-A, which enters the Krebs cycle and then the respiratory chain to produce ATP.

This process is strongly controlled by the thyroid hormone T<sub>3</sub>, (liothyronine), along with necessary cofactors.<sup>[23]</sup> If the process of cellular respiration slows, pyruvate is transformed to excessive lactate, and the milieu becomes acidic. Surplus acids must then be buffered and transported from tissues to the blood.<sup>[24]</sup>

Direct measurements of pH using DMO and radioactive C have shown the following pH levels in healthy individuals: erythrocytes, 7.2; muscle tissues, 6.9; and the mitochondrial interior, 6.6.<sup>[25]</sup> During failing metabolic activity, these pH values fall to lower levels.

According to Elmau: “. . . so far it is legitimate to consider the blood as a mirror of the metabolic processes, as a reflection of what is happening in the mesenchyme (connective tissues) as a connecting link between the blood (itself a mesenchymal tissue) and the parenchyma.”<sup>[25]</sup> However in degenerative processes, this relationship is not absolute, since the amount of acids produced in the connective tissues is no longer freely transported to the blood to be eliminated by the kidneys. Rather, it is stored in other mesenchymal tissues, such as connective tissues.<sup>[26]</sup>

According to Elmau, what happens is this: During excessive production of acids in the parenchyma, acids are stored and buffered in the mesenchyme, binding protons to potassium, sodium, and mucopolysaccharides. This condition is based on actual tests of mesenchymal (structural connective tissue) measurements in patients.<sup>[27]</sup>

Acidosis is partially compensated for in the mesenchyme, with some acid clearance. At the same time, however, the outward pumping of acids is reduced due to lack of ATP to support active mechanisms of acid transport. As a result, we do not get expected proportions of acidic venous blood; instead, *venous blood becomes more alkaline*, as seen among our FMS patients. More alkaline venous blood is thus a sign of long-standing mesenchymal acidosis. This is not to be confused with acidity of the arterial blood under similar conditions.

**Long Standing Energy Failure.** Complications develop when the buffering capacity of the mesenchyme is exhausted? The body then starts to take cations from the skeletal structures by increasing the activity of parathyroid hormone.<sup>[28]</sup> This again leads to a suppressed production of thyroid hormones as a way to save energy,

thus reducing possible extra acidic load in the parenchyma. *The idea from nature's side seems to be to force the individual into a more idle life while trying to correct the misery of hypometabolism.*

However, the modern lifestyle fails its purpose. Instead of resting, pressured modern man and woman presses on, creating more mesenchymal acidosis.

This leads to a vicious circle: excessive acids are produced and stored in the mesenchyme, giving rise to a number of connective tissue problems such as pain, stiffness, and reduced muscle function. Over time, the skeletal system begins losing cations. On “the path to disaster,” mesenchyme takes in Na<sup>+</sup> and H<sup>+</sup>, thus worsening cellular acidosis. According to the degree of mesenchymal acidosis, the venous blood becomes more alkaline and the urine more acidic. In the extremes, we may find pH values of more than 7.5 for the venous blood and less than 4.5 for urine.<sup>[17]</sup>

This leads over time to K<sup>+</sup> deficiency in parenchyma. BE-T-A testing is actually more revealing of the K<sup>+</sup> deficiency than is measurement of K<sup>+</sup> in the blood.

Of clinical interest is that K<sup>+</sup> is shifted from the cell to the extracellular fluids to be used as a buffer for acids. When this happens, intracellular Mg<sup>+</sup> is also depleted. We therefore end up with a mesenchymal acidosis plus K<sup>+</sup> and Mg<sup>+</sup> cellular deficiency. This situation is often worsened by a deficient alimentary canal that misuses ACTH, cortisone, aldosterone, and diuretics.<sup>[29]</sup>

The presence of mesenchymal acidosis also leads to other shifts in BE-T-A readings. Typical is an increase in rH<sub>2</sub>. This is to be understood as a compensatory mechanism to keep redox potential stable. As mentioned before, the Nernst redox potential is given by equation (9)  $E = 30 (rH_2 - 2pH)$ . So when pH is rising, rH<sub>2</sub> increases to keep E somewhat constant. This leads to an increase in electron activity in the blood, causing more degeneration.

The urine readings indicate that FMS patients are excreting more acids. Even though acids are stored in the mesenchyme, some surplus is transferred to the blood, buffered, and then excreted in the urine. A measured pH = 5.64 clearly indicates an excretion of excessive protons. This reading indicates a twofold situation: excretion of excessive acids from the mesenchyme, plus a decay in kidney function, since healthy kidneys should always produce a pH= 6.8.<sup>[17]</sup>

Going beyond these basic considerations is of little clinical value in an introductory text such as this. My point is this: BE-T-A testing supports the thesis that FMS is indeed a loss of energy production in the parenchyma and a degeneration of the mesenchyme, as proposed by Lowe.<sup>[12]</sup> We therefore conclude, as did Dr. Lowe, that FMS, CFS, and hypothyroidism share the

same underlying biochemical characteristic: too little energy is produced and distributed, and accordingly, cells work most of the time in an inefficient anaerobic mode.<sup>[17]</sup> In this mode, energy production (mostly ATP) is shifted from the high ATP-yielding mitochondrial respiration process to the less effective anaerobic glycolysis that results in accumulation of lactic acid.<sup>[23]</sup>

Forced to work under harsh conditions, the person's expenditure of energy is drastically reduced in a number of body compartments. This results in typical energy-depletion symptoms, such as tiredness, lethargy, slow pulse, falling blood pressure, cramping of peripheral vessels, lowered body temperature, loss of enthusiasm for life, loss of muscle tonus, and cramping of muscles.<sup>[17]</sup> Later, compensatory adjustments occur. These include the accumulation of fat tissue and mucous in the loose connective tissues for insulation against heat loss, reduced sugar metabolism, slowing of adrenal function, and loss of immune competence.<sup>[47]</sup>

**Mitochondria: The Core of It All?** It is common knowledge today among researchers and doctors that the core of this situation rests with the functioning of the cellular energy-producing units—the mitochondria.<sup>[30]</sup>

The mitochondria have been underrated in the history of medicine, ever since the discovery of their important function as cellular energy producers.<sup>[31]</sup> Few researchers have taken advantage of their dysfunction to explain clinical problems.

Three exceptional researchers (Warburg, Seeger, and Eck) should be mentioned. First is German Nobel Prize winner Otto Warburg, who in 1936 discovered an underlying common trait of all cancers.<sup>[32]</sup> He found that tumor cells had lost their ability to produce energy by respiration; instead, they went into a state of fermentation.

Later, German scientist Paul Gerhard Seeger discovered that the cause of this state of fermentation was impaired—even lost—mitochondrial respiration. The reduced respiration forced cells to extract ATP from glucose by the highly inefficient process of glycolysis.

Seeger also impressed the German-speaking world by conducting the largest number of lab tests on cancerous animals done until this date. He examined more than 100,000 lab animals over a period of 50 years. From his work Seeger concluded: not only is Warburg's discovery correct; *all* cancers produce little energy. Cancer can also be completely healed by sufficiently activating energy production. The nucleus of it all is the energy-producing activity of mitochondria.<sup>[33]</sup>

Seeger was never awarded the Nobel Prize for his cancer cures. This was due to one simple fact: he came from the wrong place—East Germany, a country not supported by the medical elite in the West.<sup>[34]</sup> He also

did not have access to the high-tech biochemical products in the West.

He achieved most of his healings by using natural substances known as “antioxidants.” Such substances stem from fresh fruits, berries, vegetables, trees, pollen flowers, and certain unicellular organisms. In Germany, these are known as “hefe.” All hefe have common qualities: they are non-toxic and neutralize free radicals, which are toxic to cells. Hefe have a high content of enzymes, minerals, and vitamins known to be crucial co-factors for enzymes of the mitochondrial respiratory chain.<sup>[35]</sup>

According to Seeger, cancer is a problem of local cell energy production. Healing is but restoring respiration and the local electromagnetic milieu.<sup>[36]</sup> Later, several other researchers came to the same conclusion regarding other diseases.

Toward the end of the 20<sup>th</sup> century, masses of data accumulated that confirmed a related conclusion: without proper energy production and control, the functional capacity of cells starts to diminish. As capacity lessens, life as we like to live it also diminishes, and people eventually come to suffer from a number of depletion syndromes.<sup>[37]</sup>

**Paul Eck: The Bio-holography of Metabolic Control.** Today, a major contribution to the realization expressed in the section above is to be found in the life work of American lab-scientist Paul Eck. Over a 40-year-period, he tested several hundred thousand patients in his lab in Phoenix Arizona. From these tests, he came to realize that diseases in general are equivalent to reduced energy production and control. His conclusion lifted the eye from the mitochondria itself to the *hormonal control* of mitochondrial energy production.<sup>[37]</sup>

The work of Eck, his contemporary Hans Seley<sup>[38]</sup> (“father” of the moniker we call “stress”), and George Watson<sup>[39]</sup> have firmly established a fact: mitochondrial activity is the basis of life. These researchers also established that supplying mitochondria with proper “food,” oxygen, and water, and regulating their supply according to the demand imposed on them and their capacity lies in the neuro-hormonal control system of the human body.

This system was termed the “holographic”<sup>[49]</sup> system by Eck. By this term, he meant that each part in the energy-producing system consists of a number of equally important players, each acting under one common law: produce enough energy at all times to sustain the maximal output of the person; or, if not, shut down production to protect misuse.<sup>[41]</sup>

The process of providing maximum energy output is the stage of stress. In response to excess stress, a number of well-defined stages shut down energy production. The shutdown leads to many processes that doctors give

names called “diagnoses.”

This brings us to a core issue of this paper: the history of the mitochondria has been utterly neglected, and the control of mitochondrial energy production has accordingly been given little thought. Thus, when a researcher like John Lowe rediscovers this fact and relates it to his favorite diagnosis, fibromyalgia, the medical elite are dumbfounded and at a loss for how to meet the challenge.<sup>[42]</sup>

To put the Lowe discovery (or, should one say “revival of a noble tradition”) to the test, I used what knowledge I derived from his splendid book<sup>[12]</sup> and combined it with my roots in the European French-German tradition of men like Enderlein, Warburg,<sup>[43]</sup> Seeger,<sup>[34]</sup> Van Vincent,<sup>[45]</sup> and the American Broda Barnes.<sup>[46]</sup>

*Keep asking the patient and you will get many answers. Keep asking the doctor and you will only get one word: the diagnosis. • Russian proverb*

### PART III: THE CLINICAL PICTURE

**Metabolism: Collecting Relevant Data.** The first step in my testing of the Lowe discovery was to find a suitable clinical definition of hypometabolism. Such definitions exist in the form of statistics from researchers like Means, de Groot, Refetoff, and others.<sup>[47]</sup>

These researchers’ statistics were based on a careful questioning<sup>[47]</sup> and observation of patients who hypo-

**Table 3:** 27 women with FMS versus 177 cases of hypothyroidism from two studies

	FMS patients	Hypo patients		FMS patients	Hypo patients
<b>Heart &amp; circulation</b>			C4: Coarse skin	44.4%	84%
H1: Low BBT	Too few answers given	100%	C5: Stiff / brittle hair	37.0%	75%
H2: Feeling cold	74.1%	92%	C6: Loss of hair	37.0%	49%
H3: Cold skin	66.2%	82%	C7: Brittle nails	44.4%	41%
H4: Dyspnoea	40.7%	64%	C8: Joint pains	81.5%	29%
H5: Paresthesia	66.7%	56%	<b>Muscles</b>		
H6: Peripheral edema	51.9%	56%	M1: Easily fatigue on exercise		
H7: Heart palpitation	63.0%	27%	M2: Muscle weakness	96.3%	61%
<b>Emotional &amp; mental functions</b>			M3: Muscle-pain	96.3%	36%
E1: Failing memory	74.1%	65%			
E2: Depressed	77.8%	60%	<b>Vitality signs</b>		
E3: Restless/nervous	74.1%	43%	V1: General weakness	88.9%	99%
E4: Loss of appetite	14.8%	43%	V2: Lethargy	77.8%	88%
E5: Restless sleep/insomnia	81.57%	25%	V3: Weight gain	59.2%	68%
<b>Connective Tissue</b>			V4: Loss of sex drive	Too few answers given	50%
C1: Dry skin	63.0%	88%			
C2: Swollen eyelids	44.4%	88%			
C3: Swollen face	51.9%	87%			

metabolic patients in their blood samples. The patients therefore got the diagnosis “hypothyroidism,” or deficiency of thyroid hormones.

However, the patients could also have lacked adequate production or effect of other hormones, such as insulin and parathyroid hormone. But due to the thinking of the time, only thyroid hormones levels were measured.<sup>[48]</sup>

Presumably lacking enough thyroid hormones, these hypometabolic patients developed a number of characteristic complaints. In Table 4, these symptoms and their frequency amongst the sufferers are noted. In Table 3, the incidence of symptoms among my FMS patients are compared to those of 177 hypothyroid patients.

These numbers clearly speak for themselves and give a good picture of what it means to be hypometabolic and have “too little energy.”<sup>[49]</sup>

Table 3 is adopted from the statistics of de Groot, Larsen, Refetoff, Stanbury,<sup>[69]</sup> and Broda Barnes.<sup>[12, p.255-256]</sup> Mean numbers arise by adding the various statistics and averaging the numbers. Arrangement of the symptoms and signs under the various headings is the author’s own and can be debated, but these have proven useful for practical purposes. The “basal body temperature” test popularized by Broda Barnes consists of the patient putting a thermometer into the axilla before getting out of bed.<sup>[12, p.255-256]</sup> This test is debatable



since many conditions may lead to reduced temperature under the arm. However, nearly 100% of all cases of hypothyroidism show low temperature before starting proper thyroid hormone therapy.

*“Science is built on the premise that Nature answers intelligent questions intelligently. So, if no answers exists, there must be something wrong with the question.”*

● Nobel Laureate, 1972: Albert Szent-Györgyi

## RESULTS

### PART IV : DATA: ASKING THE INTELLIGENT QUESTION

#### Putting Patient Data into the Hypothyreot Table 3.

Table 3 gave me a form by which I could identify hypothyroid symptoms among FMS patients. With this form, I started to collect data on FMS patients regarding the following “Leit-criteria”: widespread muscle pain and tenderness, muscle fatigue and weakness, lethargy and asthenia, and signs of hypometabolism.

I asked every patient to fill in the hypothyroid questionnaire. Over a period of 2 years, more than 100 patients filled in the form. Some filled in the questionnaire by themselves in their homes after a first consultation where every question was explained. Others filled in the questionnaire at their initial meetings in our office.

The semantics of each question is explained in Appendix II to make clear to every colleague what meaning we gave each phrase.

In November 2005, we collected all the handwritten questionnaires. We found 56 questionnaires with symptoms of hypothyroid symptoms, plus complaints of muscle pain, tenderness, and muscle fatigue.

Of these 56 patients, 27 had diagnoses of FMS made by specialist before coming to our office. Of the remaining 29 patients, 12 had either myogenic encephalopathy/chronic fatigue syndrome, and the other 17 patients had myalgic symptoms plus general health problems that could easily fit a FMS diagnosis. However, this 29 patients had not been diagnosed by a qualified specialist; so to avoid accusations of being “too eager to see the light,” these 17 patients were not included because they were waiting to have their diagnosis verified by a rheumatologist. (The wait was long in that there are only 2 among a population of 120,000 inhabitants in my country, a southern/damp/foggy region of the coastline already overloaded with rheumatic cases).

Of the 27 clear FMS cases, there were additional interesting diagnoses described later in this article (see Table 5). Of the 27 clear FMS cases, 7 patients had a history of thyroid gland disease and were currently on Le-

vaxine (T<sub>4</sub>) and/or liothyronine (T<sub>3</sub>) medication. These patients were obviously under medicated. This was clear in that they all had symptoms or signs of hypothyroidism according to the percentages in Table 4.

Without being “too eager” to prove the point, it is of interest for all to notice that of the 27 female FMS patients, 7 suffered from under-medicated hypothyroidism. It is astonishing, however, that according to the laboratory (their blood samples of TSH and FT<sub>4</sub>) and by their respective doctors’ judgement, they were “adequately treated.” Some 99% of doctors in my region believe the lab tests to be “sufficient testing for evaluating the problem.”

#### Twenty-seven Patients with Classical FMS.

Every questionnaire was now transferred to an Excel data program, and each entry matched with the journals of each patient to ensure 100% correctness—a procedure which lasted for one full month to ensure every entry to be 100% correct. All 56 patients are thus electronically stored. From this databank we extracted the 27 patients (the whole database was sent to the publisher for statistical analysis since there are more relationships to extract for future articles).

When asked about their symptoms, patients were not only asked to state if they had a specific symptom, but also to what degree it bothered them. The use of a subjective scale is not one of absolute unbiased measurement, but rather how the mind ranks a certain physical problem in a given situation where various sensations are weighed against each other. How this weighing is done is far from clear. It seems to me, however, that at least the importance of a certain body function is judged according to its necessity to perform. For example, as will be seen below, in the mind’s experience of symptoms, those of muscle, joint, and cognitive dysfunction always come out high.

Each symptom (read experience of dysfunction) was ranked according to a simple 4-point scale (see “APPENDIX II. QUESTIONNAIRE SEMANTICS” below).

In the questionnaire, patients wrote the severity they found appropriate, between 1 and 3. The entries were added and averaged: Intensity =  $\sum .I(j)/27$  where  $j = 1, 2, \dots, 27$ . (see Table 4). This formula gives a fair number for how intensely a certain symptoms was experienced by the group as a whole. The best picture, of course, would be to present a histogram for each entry, which will be done in a later article dealing with how CFS, FMS, FMS with hypometabolism and hypothyroidism with diffuse muscle pain compare.

**Table 4.** 27 Women with verified FMS from specialist.

Symptom	Frequency	Symptom Intensity	Symptom	Frequency	Symptom intensity
Heart & circulation			C4: Coarse skin	44.4%	0.70
H1: Low basal body temperature	Too few answers given	-----	C5: Stiff / brittle hair	37.0%	0.59
H2: Feeling cold	74.1%	1.48	C6: Loss of hair	37.0%	0.56
H3: Cold skin	66.2%	1.29	C7: Brittle nails	44.4%	0.67
H4: Dyspnoea	40.7%	0.74	C8: Joint pains	81.5%	1.59
H5: Paresthesia	66.7%	1.15	<b>Muscles</b>		
H6: Peripheral edema	51.9%	1.15	M1: Easily fatigued on exercise	Implicated in M2-see appendix	
H7: Heart palpitation	63.0%	0.93	M2: Muscle weakness	96.3%	2.26
<b>Emotional &amp; mental functions</b>			M3: Muscle-pain	96.3%	2.48
E1: Failing memory	74.1%	1.52			
E2: Depressed	77.8%	1.37	<b>Vitality signs</b>		
E3: Restless/nervous	74.1%	1.40	V1: General weakness	88.9%	2.07
E4: Loss of appetite	14.8%	0.11	V2: Lethargy	77.8%	1.81
E5: Restless sleep/insomnia	81.57%	1.63	V3: Weight gain	59.2%	1.14
<b>Connective Tissue</b>			V4: Loss of sex drive	Too few answers	
C1: Dry skin	63.01%	1.15			
C2: Swollen eyelids	44.4%	0.78			
C3: Swollen face	51.9%	0.96			

As seen in Table 4, the average FMS patient had a symptom pattern the same as a hypothyroid patient untreated with thyroid hormone supplements.<sup>[50]</sup> Any endocrinologist who would read the statistics in Table 4—if not knowing the diagnosis his colleague the rheumatologist would give—would probably diagnose all these patients as hypothyroid. As will later be revealed, 7 of these patients were indeed found to suffer from thyroid disease. But, again, most of these of these patients got an FMS diagnosis before it was discovered that they also had a thyroid disease.<sup>[51]</sup> And even more interesting: all of these patients got symptomatic improvement from their FMS symptoms when they received adequate amounts of thyroid hormone (levaxine and liothyronine). And even more interesting, 2 of the hypothyroid patients had to take supraphysiological doses of thyroid hormones as judged by current laboratory standards.<sup>[52]</sup>

Visa versa: the remaining FMS patients who were not diagnosed as having hypothyroidism, all improved from their FMS when they received therapy that improved their metabolism. The 2 patients who received traditional psychoactive drugs showed very little benefit from such medications as to the majority of neuromuscular symptoms.

The reason that muscle pain frequency amongst the 27 FMS sufferers was not 100%, was due to one partic-

ular patient: a 36 year old women who was cured from her muscle pain before entering the study.

Of all the data, one special relationships stands out as being of most interest: In true hypothyroidism due to thyroid gland failure, loss of vitality is not reflected by an abnormal rise in insomnia/sleeping pattern abnormality. But among FMS patients, complaints of insomnia rank of the same order as loss of vitality. This could indicate that loss of vitality in a FMS patient could be due to restless sleep due to muscle pain.

**Additional medical diagnoses at time of first contact.** Except for the patients who received their FMS diagnosis from a qualified specialist in rheumatology, the diagnosis for each patient is a question of medical attitude. Most of the patients, including those with clear-cut FMS diagnoses, had a “host of problems.” Most of the patients’ doctors underrated the other medical problems and focused only on the patients’ major symptoms. See Table 5 for other diseases identified.

**Currently Available Laboratory Testing.** We used the lab tests available in Norway at the time,<sup>[53]</sup> but we screened all patients for their metabolic status. For those receiving thyroid hormones from their doctors, thyroid lab tests were all “within the normal range.”

However, two patients suffered from untreated overt overt hypothyroidism at the time of contact. Two others

**Table 5: Medical status at time of first consultation**

Medical information	Numbers
Average age	49.5 years
Med-requiring cardiovascular disease	3
Central nervous system dis., except ME	1
Med-requiring psychiatric disorders	2
FMS previously confirmed by specialist	27
Subclass (a) simple case of FMS	15
Subclass (b) FMS + hypothyroidism	7
Subclass (c) FMS+ ME or CFS	3
Subclass d) FMS + diabetes	2
Immunity/allergies (i.e., colitis or asthma)	6
Patients on thyroid medication	7
Patients on cortisone	2

previously undiscovered subclinical hypothyroidism were also discovered. Because she was “forgotten by the system,” one of the hypothyroid women was left untreated by her doctor for 20 years!

The remaining patients all had perfectly normal, “within the reference range,” levels of FT<sub>4</sub>, FT<sub>3</sub>, TSH, and rT<sub>3</sub>.<sup>[54]</sup>

**Microbiological Analysis.** In trying to find causes for patients’ FMS and their obvious hypometabolic states, we also tested all for antibodies against five microbes: Epstein Barr Virus, Cytomegalovirus, Chlamydia Trachomatis, Mycoplasma, and Borrelia Burgdorferi.

This testing revealed three cases of chronic, undiagnosed Mycoplasma infections.<sup>[55]</sup> All patients were treated with Doxycillin 200 mg per day, plus antioxidants and probiotic bacteria cultures.<sup>[56]</sup> All three patients are currently under therapy for the 5<sup>th</sup> month and improving.

**Metabolic Status by Use of the Van Vincent Method.** Recapturing the data in Table 2, we note the BE-T-A measurements of 13 FMS patients. If one analyzes venous blood and compares it with the “ideal,” the patients had slight oxidative stress. (Remember that although the breaking point for water is rH<sub>2</sub> = 28, for blood it is rH<sub>2</sub> = 22.) pH is alkaline when R is slightly elevated. The ideal R for women is 190 and for men is 220. (The latter is often given as the ideal in many textbooks on BE-T-A).<sup>[57]</sup>

The data in Table 2 indicates that the FMS patients had mesenchymal acidosis. This is part of the tissue terrain that Van Vincent often called “the terrain for degenerative diseases” or “diseases of modern civilization.” In this part of the terrain, one also finds increased risks for cancers, viral infections, thromboses, and cardiovascular diseases.<sup>[58]</sup>

Urine is highly acidic due to the excretion of excessive acid from the mesenchyme. Acidic urine occurs because there is no blocking of acid transport from mesenchyme to blood, and rH<sub>2</sub> is a very low indication of holding back electrons from the process of proton excretion.

The FMS patients’ urine R is remarkably high: 143 versus an ideal of 30. This high R indicates excretion of mineral ions in the process of pH-rH<sub>2</sub> regulation. Mineral deficiency is to be expected in the long run.<sup>[58]</sup>

Finally, the acidity of the patients’ sputum was low. Sputum is an indication of the function of the digestive system, with low acidity indicating less effective digestion. The low rH<sub>2</sub> indicates a reduced milieu with a possible excess of free radicals (electron-grabbers). In addition, the high R indicates mineral deficiency.<sup>[58]</sup>

*“Our bodies are energy-producing machines. Every aspect of health depends on adequate energy generation. • Lawrence D Wilson, MD*

## CONCLUSION

### PART III: SCIENTIFIC HYPOTHESIS IN SUPPORT OF DR. LOWE’S THESIS

**Reduced Effects of Hormones: the Culprit?** Collecting our own data and those from a number of reliable sources we can conclude:

(1) All FMS patients, when thoroughly questioned and examined, have a clinical picture with a high degree of similarity to those of hypometabolism, as caused by a deficiency of thyroid hormones. Actually, in speaking of this clinical picture, it is necessary to speak in the plural, since we refer to clinical pictures—not just one fixed picture.

But despite having degrees of differences between patients’ clinical pictures, the patients still belong to what in the mathematical theory of patterns is called “of the same class.”<sup>[59]</sup>

(2) When investigated, our 27 FMS patients fell into two distinct categories: first, those who did indeed have thyroid hormone deficiencies, as judged by current laboratory standards; and second, those who were clearly euthyroid (their TSH and thyroid hormone levels “within their reference ranges”) but still had the same clinical picture of untreated hypothyroid patients and were clearly hypometabolic.

Approximately 34% of FMS patients fell into this latter category, by J.C. Lowe’s calculations from the studies available in 2000.<sup>[85]</sup> (Garrison and Breeding credited Lowe’s research, but argued that the percentage of euthyroid FMS patients who recover only with thyroid hormone therapy is substantially higher.<sup>[91]</sup>) Lowe also calculated that 55% of FMS patients had either primary or central hypothyroid. From the evidence of Sweden’s Bo Wikland, the remaining 11% of FMS patients—or perhaps a much higher percentage—could have hypothyroidism not biochemically diagnosable because of their “normal” TSH, FT<sub>3</sub>, FT<sub>4</sub>, and anti-

thyroid antibody levels. Among these patients, an accurate diagnosis of autoimmunity can be made only with fine-needle aspiration cytology.<sup>[88]</sup> Wikland found that 40% of 219 chronic fatigue patients with autoimmune thyroiditis had reference range TSH, FT<sub>3</sub>, FT<sub>4</sub> levels. Only half of these patients had high antibodies.<sup>[89][90]</sup>

(3) With the majority of FMS patients, there was no sign of reduced production of thyroid hormones (except for those who had the combination of FMS plus hypothyroidism). Also, there was no indication of impaired transport of hormones.<sup>[64]</sup>

Adding up these premises, among the euthyret FMS patients, the most plausible conclusion is that there must be a reduced effect of thyroid hormones at the cellular level. This reduced effect could be due to any of several factors. For example, transport might be impaired across the barrier between the blood and the intercellular fluid between cells, where hormones are transported by colloid mineral-complexes to the cellular membranes. From there, the hormones are shuttled across membranes into cells by pinocytosis, where they are carried to the nuclear membrane and attached to enzymes that rearrange T<sub>4</sub> into 4 possible isomers of T<sub>3</sub>. Only 3 of these isomers are effective. The fourth, known as reverse-T<sub>3</sub>, is an inactive variety that seems to hinder the effect of the other three effective isomers.<sup>[60]</sup>

The three active isomers all belong to a family of hormones known as cell-membrane structural rearrangement hormones. These hormones attach to receptors on the DNA. The attachment causes certain genes to start transcription for production of the real powerhouse of the cell, the small energy factories called “mitochondria,” and the host of enzymes that catalyze reactions within the mitochondria.<sup>[61]</sup>

Each step in this process is vulnerable to changes in the local milieu and in the availability of certain cofactors, such as minerals, vitamins, and free fatty acids. If some of these cofactors are deficient, even sufficient thyroid hormone will not produce energy in the form of active ATP.<sup>[61]</sup>

**Mitochondrial Energy Production.** Once it is realized that mitochondrial energy is the major source of biochemical energy—and a seemingly infinite supply of it—the clinical picture gets simpler. It is not possible to understand these matters unless the medical society makes an important shift in its mindset: from the gross (clinical) to the subtle atomic world.

According to late Nobel Prize winner Szent György, future medicine will work on the atomic level, and everything will be explained from there. We are now in the age he spoke of some 60 years ago upon receiving

his Nobel Prize for the co-discovery of one of the major atomic steps in medicine: the ability of ascorbic acid (vitamin C)—which can be properly understood *only* on the atomic level—to deliver free electrons to the energy producing machinery of the cell, the mitochondria.<sup>[62]</sup>

The mitochondria are the engines of the cell. Small bacterial-like structures, they are believed to be the primitive ancestors from a species of bacteria known as *E. Coli*.<sup>[8]</sup> They can reproduce within themselves from their own limited genome, but their genomic source is part of the DNA structure in the nucleus of the cell.<sup>[64]</sup> The thyroid hormone T<sub>3</sub>, amongst other factors, activates the genes that transcript instruction to make more of these “energy producing animals,” increasing their numbers.<sup>[65]</sup>

Inside the mitochondria’s double-layer lipid membrane, acetyl-CoA produces active ATP through a series of steps known as cellular respiration. The source for this raw material is either fatty acids or pyruvic acid from the decomposition of glucose in the cytosol of the cell. Outside of the mitochondria, these breakdown products yield only small amounts of active ATP.<sup>[65]</sup> Once inside a mitochondria, however, acetyl-CoA from free fatty acids or from converted pyruvic acid enters a transformative process known as the “citric acid” or “Krebs cycle.” This cyclic transformation of citric acid (from acetyl-Co A) produces a highly energy-rich molecule known as NADH<sub>2</sub>. What has happened is that enormous electrical energy has been ripped off from the various molecules in the citric acid cycle and has been stored in the chemical bounds of the hydrogen atoms attached to ionize NAD<sup>+</sup>, forming NADH<sub>2</sub>.<sup>[67]</sup>

The newly-produced NADH<sub>2</sub> is then sent to a number of small molecules on the inner wall of the mitochondrion, known as “cytochromes.” There, the energy-rich electrons in the NADH<sub>2</sub> are taken away and transported to a chain reaction that produces active ATP from its precursor, ADP.<sup>[52]</sup> The phosphate bonds in ATP now hold all the electrical energy once stored in NADH<sub>2</sub>, and ATP travels out of the mitochondrion to supply cellular chemical activity with life-giving electrical energy.<sup>[12,pp.165-166]</sup> The protons released when the electrons are transported away eventually combine with oxygen and form water. The need for oxygen as a cofactor to the protons gives name to the process: “cellular respiration.”

I cannot emphasize it strongly enough: mitochondrial respiration is all about taking electrical energy from nutrients (sugar and fatty acids) and putting it into smaller molecules called ATP. ATP can then move freely and support all other life processes throughout the body’s cells. Basically energy is about electromagnetic energy: the energy is either (1) trapped in chemical bounds, (2) freely moving or rotating ions or electrons,

or (3) pure electromagnetic energy waves. Beyond that, there is no other source of energy in the human body.<sup>[68]</sup>

**Mitochondrial Nourishment.** Like any being, unicellular or multicellular, mitochondrial life is sustained the “traditional way”: nourishment enters, energy is released, and waste products in the form of protein-bound toxins are excreted in the process.<sup>[69]</sup> There is no mystery to this, only intense interest of the curious mind. However, not even the most authoritative textbooks from the medical establishment contain a single word about the necessary cofactors in the mitochondrial respiration process, nor anything otherwise about the mitochondrion’s nourishment and toxin production and elimination.

This omission is so glaring that it blinds the eye, which this author personally believes is an intended effect. The intention is to mislead the medical society into believing that energy is produced just from sugars, proteins, and fatty acids. Some textbook authors, however, believe fatty acids to be “dangerous,” and, to be politically correct, they therefore do not even mention the fatty acids.<sup>[70]</sup>

However, there are a number of laboratories and experts on nutritional medicine that do indeed test for and include the nutrients necessary to make sufficient energy available to cells. A good and recommended source of information is a U.S.-based company, US Biotek. Through this company, one can get a listing of life sustaining nutrients known to be necessary for enough energy to be available to cells. Briefly, these nutrients are:

Krebs Cycle:

1. Vitamine B complex
2. Coenzyme Q10
3. Alpha lipoic acid
4. Aspartic acid
5. Alpha keto glutarate
6. Several amino acids (L-phenylalanine, L-arginine, L-isoleucine, L-valine, L-gutamine, L-cystein, L-tyrosine, L-hydroxytyptophan , and N-acetyl-cystein)
7. Minerals (copper, ion, magnesium, manganese)
8. Necessary antioxidants (i.o.w: electron-donors) vitamin C, E, and alpha lipoic acids

(For a complete list of mitochondrial nutrition and metabolic testing: contact US-BioTek, 13500 Linden Avenue North, Seattle, WA 98133 also [www.usbiotek.com](http://www.usbiotek.com).)

These substances cover only what is needed inside the mitochondria. The work of Berlin’s Paul G. Seeger as a research scientists for over 50 active years proved beyond doubt: if essential nutrients are missing, or if electron donors are lacking, mitochondrial respiration suffers, and the production of ATP “sinks like stone.” This results in hypometabolism and eventually cancer.<sup>[70]</sup>

Seeger also showed beyond doubt that the presence of toxins (free radicals) either damaged the cell membranes or disrupted the chemical processes inside the cell. In either case, this also leads to same results: hypometabolism and finally cancer.<sup>[70]</sup>

## PUTTING IT ALTOGETHER

It is now due time to put together the data I have included here. The point is to try to make a synthesis that leads to some basic understanding. I have done so in a series of short statements.

**1. The clinical pictures of FMS and hypothyroid patients are similar:** Comparing the clinical picture (symptoms + signs + objective findings) of FMS and hypothyroidism, one find them to belong to the same class of phenomena. This means that if a doctor is given the clinical picture of a FMS patient with no diagnosis attached, he will in most cases label the patient “hypothyroid.”<sup>[71]</sup>

**2. FMS patients have mesenchymal acidosis:** The Van Vincent BE-T-A test shows that FMS patients belong to a special class of patients; those who have mesenchymal acidosis. This means they have an accumulation of acids in their connective tissues. It also means that unless the patient is actually poisoned by an external acid, his or her parenchymal metabolic rate is reduced, and that, as a result, lactic acid and free radicals are accumulating in his or her connective tissues.<sup>[72]</sup>

**3. Blood sample: results show two etiological subgroups:** When investigating blood samples, we discovered at least two major subgroups of FMS patients: (1) a euthyreot group with no signs of failing thyroid production or transport; (2) another group suffering from a variety of thyroid disease states, such as thyroid hormone deficiency. In general, both groups of patients get simultaneous relief from FMS and any thyroid problem when they receive “enough “thyroid hormone” (meaning, enough to alleviate their symptoms and normalize their metabolism). The former groups of patients, however, may need additional help, such as supplemental cofactors, to restore thyroid hormone effects.<sup>[19]</sup>

**4. Both subgroups deserve the same label: hypo-**

**metabolic states:** A reduced level of thyroid hormones or hindered cellular effects of the hormones both produce the same impact on patients: clinical hypometabolism and mesenchymal acidosis that is typical of mitochondrial failure. We discovered no major difference between the two groups.

Mitochondrial failure explains the acidosis. When glucose is not broken down to acetyl-CoA and transformed to ATP in the Krebs-cytochrome respiration chain, what is produced instead is lactic acid. Mitochondria failure also produces free radicals, and both of these processes explain paracymal acidosis and subsequent mesenchymal acidosis.<sup>[16]</sup>

**5. Loss of mesenchymal electrical properties:** To buffer acids, potassium and magnesium are lost from cells to the mesenchyme. Sodium, on the other hand, enters cells and weakens the electrical potential across the cells' membranes. This leads to further mitochondrial failure. In the later stages, calcium is extracted from the skeletal system to buffer acids. This is in accord with the clinical findings of J.C. Lowe who reported an increased need for calcium and magnesium in FMS patients.<sup>[12]</sup> Potassium is not mentioned, probably because this mineral is abundant in the daily food, while calcium and magnesium are not.<sup>[103]</sup>

**6. Need for more thyroid hormones:** To correct patients' symptoms and signs, more thyroid hormones are clearly needed, together with minerals, vitamins and other cofactors to balance the electrolytes (see patient 5) and nourish the mitochondria.<sup>[76]</sup> At the same time, one needs to find the cause(s) for cellular resistance (such as microbes, toxins, and heavy metals) and try to remove these.

What is the "chicken" and the "egg" may in the end be a matter of philosophy. Clinically, both ends should be dealt with: the lack of hormones and/or any causes of the failed cellular mission of the hormones.

*How it all should be dealt with is according to the understanding of the doctor and the unique condition of each patient, not to forget the possibility of making corrections within the framework of the medical establishment.*

#### **POLITICAL POSTSCRIPT: A DISCOVERY WORTH PROMOTING IN NORWAY**

As this article is being written, the author is currently working to get the Lowe thesis known amongst

colleges. This has proved to be somewhat problematic.

The reason is that in Norway, doctors are compelled to adhere to a doctrine: metabolism is best understood in terms of the "normal range" of TSH, T<sub>3</sub>, and T<sub>4</sub>. As long as these measurements are within their "normal ranges," metabolism is OK, and patient complaints should be ascribed to psychosomatic factors. In one particular case the author was even "reported" to the authorities for giving a different opinion, that of "euthyretot hypometabolism."

On the brighter side is the fact that some doctors now seem to be catching up with the idea of euthyretot hypometabolic syndromes and functional thyroid hormone resistance. Fortunately, Aker University Hospital in Oslo, known for its work on hormones, has met the idea with a positive response.<sup>[77]</sup>

Personally I have found these ideas and discoveries so useful that I have decided to use the rest of my career to go deeper into the relationship between energy production, hypometabolism, and degenerative diseases. Therefore, I express a heartfelt thanks to the pioneer John C. Lowe and to the journal *Thyroid Science* for publishing my findings.

#### **APPENDIX: THE ENERGY VIEWPOINT**

*"All medicine is energy medicine."* • James Oschmann

*"You cannot solve a problem unless you have an outcome."*  
• Joseph O'Connor<sup>[1]</sup>

The Lowe discovery that FMS and CFS are indeed hypometabolic syndromes may well be one of the major breakthroughs in modern clinical medicine. There are others, of course, who independently have made the discovery that most diseases may be viewed as a failure to produce and distribute enough energy. Examples are Paul Eck, George Watson, and Szent György. But the work of John C. Lowe is the first major work to nail a specific disease entity outside the ordinary endocrinological diseases to metabolic failure.

The morphological orientation, or what could be called the 19<sup>th</sup> century medical paradigm, is that a "disease" is something static and unchangeably recognized by the presence of a "list of symptoms, signs, and findings."<sup>[78]</sup> But once we enter the realm of metabolism, we are in for a change of paradigm; we move from a medical science of morphology to a science of energy. The core of metabolism is the production of energy and its distribution and use.

Once entering the realm of energy, medicine enters

the realm of physics where scientists have for 200 years recognized the fact that phenomena are changing. The search is thus not for something static but for underlying laws of nature that make things happen. Physical science is thus occupied with principles and expects natural events to be in movements all the time.<sup>[107]</sup>

The search for natural laws has been the source of success in the physical sciences. Technology in general is far more advanced today in many respects than in medical science, which is somehow, for better or worse, stuck in static 19<sup>th</sup>-century thinking. The gulf between the two worlds can be pictured like this: It is 39 years since U.S. scientists and technocrats put a man on the moon for a fraction of the cost of what is spent on cancer research and treatment per year in the U.S. Despite this fact, there are still few medical cures for cancer and no sensible explanations for this failure of established medicine.<sup>[80]</sup>

This situation is not due to a lack of brilliant medical scientists, lack of enthusiasm, intelligence, good intentions, or brave efforts. It is due simply to this: GPs, by which the community of good men navigate, continue to point to a path of struggle and toil through a barren landscape.<sup>[81]</sup>

Only by change of path will more fruitful landscape be found; and this is what John C. Lowe has done. By asking the right questions, he has single-handedly loaded the scientific GPs with a path that has entered the realm of the physical sciences. This realm is based on energy thinking and the search for underlying laws of nature. Instead of looking at snapshots, one is watching a film and asking for the ways the film's director is "thinking." This orientation is called "functional medicine" or "energy-based medicine." It by no means excludes chemistry or morphology, but rather encompasses these as *part* of the curriculum, rather than the end of all thinking.<sup>[82]</sup>

By doing this, John C. Lowe has followed the path of great American scientists such as the "father of stress," Hans Selye, nutritionalist George Watson, mineral researcher Paul Eck, biophysicist and surgeon Robert O. Becker,<sup>[83]</sup> and luminaries of the Old World such as Nobel Laureate Otto Warburg, cancer-researcher Paul Gerhard Seeger, Hungarian biophysicist and Nobel Laureate Szent Györgye, and many more who wanted to merge the powerful dynamic thinking of physics with the wealth of morphological knowledge of medicine.<sup>[84]</sup>

It is my sincere opinion that understanding FMS and CFS as conditions of metabolic failure is like hand-

ing the GP with a new map to navigate turbid waters, and thereby to help patients and doctors alike to tread a better path of understanding, leading above all to a better outcome.

## APPENDIX II. QUESTIONNAIRE SEMANTICS

0 = I do not sense this symptom/problem.

1 = I feel this is a small problem (of dysfunction). It is weak and gives me little trouble in my personal life.

2 = I feel this to be a medium problem. It causes me trouble, but I can manage to go along with certain compromises.

3 = It bothers me very much. I feel that it hinders me in my daily life, and I am bothered all the time by it. It is amongst the problems I need to reduce to be able to improve.

Answers depend on the questions asked. We tried to select a proper linguistic structure based on NLP-principles to reach clear answers. For readers interested in our linguistic structure, please email me at for a copy of the questions in the questionnaire: bjorn@dr-overbye.

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metabolism in the past and in the present time is their focus on thyroid hormone alone. A number of thyroid books completely overlook the simple fact that the thyroid is but one of many hormones working to regulate cellular production of ATP. Two major steps forward are the work of John C. Lowe and Paul Eck's at Ananalytic Research Laboratoium. In a forthcoming PhD thesis by our colleague Ragnar Wattøe on the nature of complex regional pain syndromes, the mitochondrial aspect is the central focus. The thesis is obtainable from the author: Dr. Øverbye, Boks 348 BN4803 Arendal, Norway.

49. This table is adopted from the book of de Groot, et al.<sup>[14]</sup> In the original work there are two statistics termed A and B (see full exposé in Lowe, J.C.: *The Metabolic Treatment of Fibromyalgia*, Boulder, McDowell Publishing Co., 2000, p.837). My numbers were achieved by taking the mean of the two:  $(A+B)/2$  and rounding off to the nearest whole number, which is mathematically acceptable as long as the basis for the numbers are mentioned.
50. See reference 3. Much can be said about the work of Yunus. The basic problem with his work, however, is that he looked for a muscular disease, not for a metabolic syndrome. His diagnostic criteria therefore fit only a certain percentage of all hypometabolic patients suffering from muscle pain similar to FMS.
51. Simple logics tells us that if lack of thyroid hormones leads to a certain clinical profile characterized by symptoms S1,S2YsN, these symptoms together are what is called a mathematical Group, which we may call "A." If another disease entity has the symptoms S1,S2YsN, the symptoms can form a Group called "B." According to mathematical logic, if  $s_j = S_j$  for  $j=1,2,Y,N$ , then  $A = B$ . So, if hypothyreosis is classed as Group A, and FMS is classed as Group B, by simple logic one would end up saying FMS = hypothyroidism. However, most FMS patients do not have abnormal blood levels of thyroid hormones, warranting classification A. Then the only logical explanation will be found in the fact that symptoms are not an expression of blood thyroid hormone levels. Instead, the symptoms arise from biological effects of the hormones at the cellular level. In other words, we deal with metabolism = the biological effect of hormones and their cofactors. For those who want to go deeper into the subject I recommend Max Black: *Critical Thinking*, 2nd edition, Englewood, Prentice Hall, 1962. John C. Lowe is the only scientist I know of who has so far ventured into the use of logic in the diagnosis of FMS.
52. Many of my hypothyroid patients indeed got the diagnose FMS before getting the diagnosis of hypothyroidism, which is just another support for reference 33.
53. Lowe is one of the advocates of suprphysiological doses of thyroid hormones for FMS, when appropriate, to override cellular resistance to the hormone. Using suprphysiological dose of hormones is nothing new. For example, a number of conditions require this, such as hormonal replacement therapy in postmenopausal women, the p-Pill for fertile women, the clinical use of cortisone (sic) which in Norway is routinely given to even infants to relieve ordinary asthma.
54. The best biochemical metabolic testing available for Norwegian doctors at the present is the Aker University Hospital of Oslo. They can supply the following tests: TSH, FT<sub>4</sub>, T<sub>4</sub>, FT<sub>3</sub>, T<sub>3</sub>, rT<sub>3</sub>, Tg, anti-Tg, anti-TPO, TRAS, PTH, Protein bound Iodid, DHEA, DHEA sulphate, cortisol, ACTH, aldosterone, complete sex -hormone screening, and ADH.
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### POSTSCRIPT

It is a sad fact that FMS and a host of other diseases could be successfully understood, quantified, and treated with quite simple means. This could be accomplished were it not for the rise of a fragmentary and ever more specialized occupation known as current progress in medical science. In the end, this current “progress” will lead to nowhere, except each doctor living on a small island in an ocean of unintelligible data. The energy viewpoint can solve the diseases from which humans suffer gracefully and with beauty.

The author is presently conducting a longitudinal study of the hypometabolic states of FMS patients with BE-T-A testing. The focus of the study is the paradox of alkaline venous blood despite mesenchymal acidosis. This study will be finished by end of 2008.