

Received: 2009.02.23
Accepted: 2009.08.31
Published: 2010.02.01

The “continuum” of a unified theory of diseases

George Vithoulkas, Stefano Carlino

International Academy of Classical Homeopathy, Alonissos, Greece

Source of support: Self financing

SR

Summary

This essay’s theme was inspired by a question asked by a child: ‘Why do I get ill?’ The question is very interesting, but has no easy answer. This paper discusses a few possible answers to this difficult question. Through the life of a person, from birth to death, there is a “continuum” in the pathological conditions a person may experience. The body, as a whole, suffers deeply any time there is an acute or a chronic condition that is either maltreated or neglected. Chronic and acute diseases in the medical history of a person constitute a rigidly related chain of immune responses in the form of a real “continuum” that at every point in time indicates the end result of this continuum. The idea promoted here is that suppression of diseases, through excess of chemical drugs or other means, many times overwhelms the body’s natural defenses and forces the immune system to compromise and start a deeper line of defense, which then constitute the beginning of a new chronic condition. Thus, the original inflammation of an acute condition may continue as a sub-acute inflammatory process on a deeper level.

Acute inflammatory conditions must therefore be treated very carefully from their beginnings in childhood in order not to force the immune system to compromise. It is also suggested here that all chronic degenerative conditions have a sub-acute inflammatory character, and that “inflammation” constitutes the main common parameter of all diseases.

key words: continuum of unified theory of diseases • homeopathy • fever • acute and chronic disease • subacute inflammation • unified theory

Full-text PDF: <http://www.medscimonit.com/fulltxt.php?ICID=878341>

Word count: 5836

Tables: –

Figures: –

References: 129

Author’s address: George Vithoulkas, International Academy of Classical Homeopathy, Alonissos, Northern Sporades 37005, Greece, e-mail: george@vithoulkas.com

BACKGROUND

Through the life of a person, from birth to death, there is a “continuum” in the sequence of natural diseases, acute and chronic. When acute diseases are not properly treated and the patient has a weakened immune system, the overall health of the individual is permanently compromised.

The acute diseases of childhood (not the epidemic, which manifest primarily in the systems that are more exposed to the external environment, namely the respiratory, digestive and epidermic systems), have to be carefully treated and not suppressed with excessive medication, otherwise the acute diseases will continue in modified form as a kind of sub-acute inflammatory process, triggering the expression of the genetic predispositions of the body, and thus manifesting the chronic degenerative diseases. All chronic conditions also have an inflammatory character, and that “inflammation” constitutes the main parameter that characterizes all diseases.

If the body, while running a high temperature, is repeatedly stressed in an aggressive way through strong or excessive chemical drugs, the immune system, being already in a weakened state, may eventually be compromised to such a degree that it may no longer be able to react by producing a high fever, even if exposed to virulent microbes [1]. One of the best examples of this is chronic fatigue syndrome, also called “post viral syndrome”. In such a condition it is known that after a viral infection a chronic condition can develop [2], characterized by sometimes persistent debilitating fatigue, with muscular weakness, mild fever, tender lymph nodes, headache and depression. Another example is acute viral hepatitis, which may continue as a hepatic derangement and finally cirrhosis [3], as well as the acute rheumatic fever which ends in chronic heart condition [4–12]. It is also known that elderly persons have a lower ability to raise a high fever after an exposure to an infectious agent [1,13]. In all such cases, when high fever due to a viral infection is strongly arrested by suppressive means, the general level of health is severely compromised. At the same time a new chronic degenerative condition begins, for which the body is genetically predisposed.

The questions that here can arise is: “What is the relation between acute inflammatory processes and chronic diseases characterized by acute exacerbations?”, and also, “Is it possible that they form one and the same line of basic disturbance?”

In acute conditions, following aggressive interference through strong chemical substances, the body will descend in its overall health condition, by giving up its defense on a peripheral level and proceeding to defend the body on a deeper level. If this new line of defense is again attacked it will go on to an even deeper level. The lowering of the defense will follow a hierarchical plan which seems to be an archetype in all humans – peripheral infections going deeper to a more central level.

Therefore, the hypothesis we can draw is that the immune system, whose main purpose is to maintain life at any cost, is structured in such a way as to respond and react on different levels in different ways. The immune system has several

levels of defense. The first line of defense will be to raise a high fever. If this is not possible anymore, due to a deficiency of the immune system, then the second line of defense will be a sub-acute inflammatory process, which is more diffused and therefore more debilitating, by involving vital organs or systems [14,15].

The infinite complexity of the human being

If we consider the human being as a whole, with intelligence, feelings, language, cognitive and creative capacities, **no other organism on this planet is as complex and multi-dimensional as are humans**. There is therefore no way that any kind of biochemical examination can provide us with a really definite answer as to the degree of health of an individual at a certain moment. Laboratory tests can give us only a very gross idea of what is going on at a biochemical level at a certain fixed time, but cannot tell us the true overall state of health. In this essay we will try to give some parameters that will help the medical practitioner have a better idea of the patient's overall state of health.

Role of the environment in creating and shaping diseases

Usually diseases begin to manifest from the very early days of life, when the environment becomes hostile to a newborn. We live in an environment in which there are disease-bearing organisms or substances which force the body to defend itself. The relation between the ability of the individual to adapt and to defend itself and the ability of the hostile organism to challenge the health of the individual will determine whether a disease process will be set in motion. For an person to start becoming ill, it is obvious that a stressor is needed, but in addition the immune system must be in a weakened state and have a sensitized predisposition towards the hostile organism. This is quite true at a first level, e.g. there may be a microbe and an organism that is susceptible to this microbe which may start a microbial infection, or there may be a chemical substance to which a sensitized organism is exposed, and this can cause the onset of an illness [16,17].

Role of the lifestyle in forming diseases

Another important reason why we become ill is that illness is often the result of our own way of life, habits, eating habits, thoughts, something that breaks a law of nature. If we exceed these limits laid down by nature, we inevitably lose the balance of homeostasis. For example, if we exert ourselves beyond a certain level of endurance, at some point the body will react by developing a disease. When we nurture negative feelings, we may provoke inherent predispositions to chronic conditions.

“Predisposition to diseases”

Environment and lifestyle are not sufficient to cause illness; there is a disease-bearing factor and the **human body may be sensitized to this factor** and therefore may develop an acute illness. It is a well known fact that two or more men could have contact with a woman infected with gonorrhea and only one of them gets ill. An organism develops a disease when it has a predisposition, a weakness, towards a particular disease-bearing factor (not everyone will develop

tuberculosis when exposed to *Mycobacterium tuberculosis*). In other words, when the relation between the strength of the stressor and strength of one of the defense mechanisms is in favor of the stressor, the body falls ill, or, if this supremacy of the stressor is excessive, it may even kill the patient. For the most part predispositions are congenital [18–21], but the use of certain drugs, like the antibiotics [22–31], or exposure to free radicals or to other chemical substances present in the environment [32,33], may cause mutation in the DNA that may lead to the development of “acquired” predispositions, due to the weakening of certain organs or tissues. Normally, about 10^4 DNA bases are damaged per cell per day and each cell continuously repairs this damage to maintain genomic integrity. Fortunately, this very complex mechanism is very efficient, but its malfunction can play a role in the development of new predispositions [34].

The suppression of acute diseases as a cause of the onset of chronic diseases

Let us take this idea a little further; we have diseases that we call acute, and diseases which are chronic and degenerative. It is important to distinguish between why someone falls ill with an acute disease versus with a chronic disturbance. We want to discover what actually happens in a person who is born and has health problems and how those problems develop over the life span.

Almost everyone has health issues. There is not a single child born who does not potentially have health problems, at one time or another, whether acute or chronic.

The general picture of disease in humans is characterized by two main groups, from the point of view of febrile expression: one with high fever and the other with low or no fever. To the first group belong the acute, to the second the chronic diseases. The main purpose of this essay is to show the relation between such diseases in the same individual; in other words, to show that there is a continuum within a specific organism that determines the reaction of the individual's immune system.

A very interesting observation is that many chronic diseases have exacerbations and remissions [35–44]. Let us take for example a person who is suffering from epilepsy. In the crises stage he has an epileptic seizure, but when he does not have the crises, what changes occur in his body and what changes occur in order for an epileptic seizure to take place? The same question is valid with multiple sclerosis, bronchial asthma, hay fever and other chronic degenerative conditions.

The next legitimate question therefore is: “Are the exacerbation waves that occur during a chronic condition comparable to acute diseases, as the body having a relapse?” **If we can understand how the body functions, perhaps we can arrive at a theory of disease that is completely different from what is taught to students in a conventional medical school.**

In medical schools students learn how to discern chronic and acute diseases, about the various syndromes, how all the acute states are characterized and how each one must be dealt with, separately, with a particular treatment. Is this knowledge sufficient for a doctor to cure a patient who comes

to him with an asthmatic crisis? Usually the doctor knows what he must do in an asthmatic crisis - he prescribes bronchodilators, or if the crisis is very strong he provides corticosteroids and the patient will come through the crisis safely. A little later the crisis will return and the condition will be worse. The crises will multiply over the years, becoming more stubborn, and we will finally be faced with patients such as the one I treated recently who could not breathe and spoke to me bent double. According to Scientific American magazine, June 2000, page 30, “Asthma Worldwide”: “*Asthma was rare in 1900, but now it has grown into an epidemic: more than 15 million are affected in the U.S. and up to 10 times that many around the world. Every year it kills 5,000 Americans, mostly older adults, and 180,000 annually worldwide, according to the World Health Organization. Why asthma rates have risen is not entirely understood, but clues come from studies showing that its prevalence tends to be highest in Western countries, particularly the English-speaking ones; it is virtually absent in parts of rural Africa.*” Also the American Academy Allergy Asthma & Immunology shows very worrying statistical data concerning the increased incidence of asthma [45]. It is evident that the ‘Western countries’ way of treating diseases is not exactly what ‘curing’ people should be. By suppressing the symptoms, you can get the initial impression that overall the disease is ameliorating, but then the disease becomes worse over and over. It is as if you had a pressure cooker on the stove (cause of the disease) and you see steam coming out through the safety valve (symptom) and, instead of turning off the burner (removing the cause), you close the security valve (suppression of the symptom), causing a very dangerous increase of the pressure inside the cooker. In routine daily practice a correlation has been demonstrated between tonsillectomy and risk of inflammatory bowel disease [46,47], because the surgical removal of tonsils, which seem to be the cause of the troubles in the patient, can cause deeper and more serious diseases. Since the 17th–18th centuries the suppression of hemorrhoids, menstrual evacuation and skin eruptions has driven the disturbances inward in the body, causing the occurrence of asthma and dyspnea [48]. If we look at the official statistics of the U.S. government from the beginning of the last century concerning death rates, we see a decrease in mortality due to infectious diseases, but increased mortality due to cancer [49]. In other words, we clearly assisted to a shift of the disturbances to a deeper level. The decrease in mortality due to infectious diseases was not due to the introduction of antibiotics or vaccinations, because these were introduced at a time when the declining trend of these pathologies was almost finished [50]. Analogously, we can see a worrying increase in the incidence of autism in American children from 1992–1993 to 1999–2000 of about 2,500% as average [51]. We, as therapists, have the responsibility to carefully consider this phenomenon.

The totality of symptoms as a reaction of the body attempting to restore balance

Going back to the asthmatic crisis, the thing that interests medicine today is whether we have the ability to either reduce the severity of the crises or to cure the patient.

What are the parameters that tell us if a patient can be cured? These parameters are of great interest to the medical practitioner. The child presumably had repeated illnesses one after the other and asked himself “Why do I get sick? My

friend at school does not get sick, or not so often". Perhaps the answer lies primarily in the complexity of the individual and their specific heredity. Within this hereditary predisposition his defense mechanism tries to adapt to the environment in order to survive, maintaining homeostasis, without needing to manifest a series of pathological symptoms. With the manifestation of pathological symptoms the body actually tries to restore its lost balance. For instance, in a very hot environment the reaction of the body is to perspire in order to cool down. But if the cooling down is abrupt, the system will react with a common "cold" that, in order to bring back the balance, will develop a fever, bringing the reaction to a pathological degree. It is pure intellectual invention to claim that symptoms are a negative manifestation that must be eliminated or suppressed. We feel pain in a joint when the affected part needs to be immobilized in order to minimize the local disturbance and allow maximum and fast recovery. Pain suppression – which on one hand gives freedom of movement - may sometimes lead to severe organic damage; thus, **the development of a symptom is a useful mechanism**. Biology considers it a developmental mechanism and a mechanism for adaptation. We should, therefore, deduce that epidemics of childhood diseases, for instance, are necessary in order to "train" the immune system and make it stronger in order to survive later in life.

"The meaning of symptoms"

We may hypothesize that acute illnesses are often the body's learning processes which must be understood by the doctor as such in order to avoid or guard against a suppression and a transformation into chronic illness. When an organism first experiences a new environment, it must learn in which direction it must develop and strengthen its immune system. The body expresses its discomfort through symptoms, and in symptoms there is so much information, very useful for doctors, to guide them in treating patients and then being able to answer questions such as: "Can my disease be cured?", or "Can I be helped, and to what degree?". In chronic cases, the conventional doctor can very rarely say that he will be able to cure the patient. What he should say is that he will provide the patient with medication which will make him feel more comfortable, and that his pain and all other symptoms will not bother him so much. He cannot, however, affirm that this particular patient will be cured. In homeopathy, things are a little different. That is to say, the doctor, using information from the "totality of symptoms", can assert in many instances that this patient can or cannot be cured.

The defense mechanism and the hierarchy in the human body

Every living organism has its own defense system, either in the animal or in the plant kingdom [52–58] and the doctor must, if possible, find the key which corresponds to the way each organism reacts to diseases. This is a basic rule in homeopathy. It is not accidental that in a newborn baby diseases appear mainly in the most external parts of the body. Pediatricians agree that it is the respiratory, digestive and cutaneous systems that are primarily affected in infancy. These three systems are related to the contact we have with the environment. These systems are subject to the greatest percentage of assaults by different microbes and chemical

substances that cause diseases that we call "acute", characterized by high fever. The urinary system with the kidneys, the vascular system with the heart, and the neurological system with the brain are less vulnerable and much more protected, and they are much less affected in early childhood with acute infections. This happens because the child's immune system is usually in quite good condition and in general is at a better level of health than in an adult. It is obvious that an inflammation of the skin or the guts or the bronchi is less dangerous to the patient's life than an inflammation of the kidneys, the heart or the brain. "*The blood-brain barrier (BBB) provides both anatomical and physiological protection for the central nervous system (CNS), strictly regulating the entry of many substances and bloodborne cells into the nervous tissue*" [59] and this indicates how the body is designed to actively protect the systems that are most vital to survival. We have to accept that the body maintains a hierarchy in its organs and systems as far as the protection of these systems and organs are concerned. It will therefore try to keep the disturbance on a rather peripheral level and as far as possible from more important organs and systems. The first infections we observe in children are in the upper respiratory track; they are primarily tonsillitis, rhinopharyngitis, common colds, and etc.60, and not encephalitis, meningoen- cephalitis and central nervous system infections in general. The tonsils are one of the gates that keep the infection away from the lungs, which are situated at a rather central level, so that a more serious infection which would put the whole body into danger is prevented. The body has a rule, an intelligence, which is not expressed in a logical way, but in a way that can be noticed by us, taking into consideration the facts we mentioned above. For instance, a child develops tonsillitis and takes antibiotics and soon after there is another infection and the next year the child has repeated infection, again treated with antibiotics. **The next year he may not have tonsillitis any more, but rather an established tracheo-bronchitis infection**, and again he receives antibiotics. After a few years this, the body, already weakened, will have infections starting from the lungs, and pneumonia. The lungs (with pneumonia), which are the most important organs of the respiratory system, are now affected. Very often allergic rhinitis is a condition that precedes the asthmatic pathology, especially when the rhinitis is suppressed with pharmacological treatment [61–67]. In other words, the disorder leaves the peripheral area of the respiratory system and proceeds ever deeper into the system's organs, which are absolutely basic for the individual's survival. If this hierarchical order is an archetypical principle in all humans, the choice made by the defense mechanism to keep the inflammation on a peripheral level (tonsils) is the best possible choice and should be respected by the doctor who understands and accepts these principles. In such a case the chosen therapy must not suppress the inflammatory process, pushing it to deeper levels, but only support the body, helping it to overcome the problem.

Let us consider another system, the urinary one. We have a patient with repeated urinary tract infections and eventually he develops interstitial cystitis, painful bladder syndrome [68], or permanent kidney damage [69]. When you investigate the case history of each of these patients, you will see a similar process. The body initially tries to keep the infection at a peripheral and more superficial level, and therefore less dangerous to the whole body. By interfering with

strong chemical drugs, we do not allow the disturbance to stay on this peripheral level, causing a compromised immune system [70–74] and forcing the infection to proceed to a deeper level, until it reaches and infects the deepest part of this system, which in this example are the kidneys.

Let us consider another example – there are patients that easily get diarrheas and because of some type of infection; these diarrheas are treated with chemical drugs and sometimes antibiotics, and eventually pseudo-membranous colitis develops [75–77] that is dealt with by other drugs. After a while ulcerative colitis and finally cancer of the colon can appear [78–84]. The antibiotic treatment causes the modification of the intestinal flora, favoring certain anaerobic strains that, being normally present in a few colonies, remain innocuous, while in this case they begin to overgrow. As a result of their overgrowth the whole body can be seriously damaged. Recently, the hypothesis that the quality of the micro-flora can play an important role in the pathogenesis of autism is becoming more accepted [85–87].

All these instances are similar in a particular respect: at the beginning, the body tries in all cases to keep the disturbance at a level which is not deep, is peripheral and therefore easily managed. The question is whether we understand this principle and try to keep the disturbance on a peripheral level, or we ignore this vital principle and go ahead and suppress the manifestation of an acute condition. Most chronic disturbances start when babies, affected by skin eruptions, are treated with corticosteroids – the skin eruptions are suppressed, not allowing the body to express its deeper disturbance on the skin. Antibiotics are frequently prescribed to prevent urinary infections, not because of any manifestation of symptoms, but simply because urine cultures are positive for certain types of bacteria. There is no inquiry by the part of the doctor as to why the body “needs” these colonies of bacteria, but as a rule a strong antibiotic is prescribed. This is certainly not an optimum way of treating diseases, and many provocative thoughts and ideas presented in this paper may deserve consideration. In the United States, for example, the latest statistics mention 328,000 patients with kidney failure undergoing blood hemodialysis, which is a frightening number. Why there were no such numbers of patients with kidney failure in the past? When we examine the case histories of these patients, we find repeated infections of the lower urinary tract, which were treated with antibiotics. Eventually the infections are manifested deeper in the bladder, then in the pelvis (pyelonephritis) and finally in the kidneys (glomerulonephritis), that greatly disturb the function of these important organs. The question is to what extent are much antibiotics responsible this course of events?

Diseases as the result of a chain of events

What we are interested in is seeing whether final illnesses that appear in humans and in each individual in particular are linked through a chain of pathological incidents that originated as acute inflammatory events and finally resulted in a chronic degenerative disease. For instance, someone gets rheumatoid arthritis or any other chronic degenerative disease and according to conventional medical thinking this is an accidental event, but the question is “Is this an accidental event or is this the result of a precise, almost mathematical, course of pathology events that led this patient to

this final condition?” It is very important to find out whether this condition that appeared at the age of 35, 40, 50 or 55 is linked to the person’s whole medical history. My experience in taking the medical history of thousands of patients from birth to childhood, or to the time they came to consult me with chronic problems, shows me that their immune system invariably had tried to keep the disturbance on a peripheral level by inflaming a non-vital organ, such as the tonsils, and by reacting to the intruders with high fever, but such a reaction was almost always suppressed fast by strong chemical drugs. Because of the heavy treatments administered at the time of acute inflammation, the body was not allowed to perform all the necessary biochemical processes that could restore the lost homeostasis, and the body adopted a second line of defense, which means that the chronic condition started with a sub-acute inflammatory process. We may assume that the heavy chemical interference did not give the body enough time and space to rearrange himself in order to be able to defend against the next assault by the microbes.

It is well known that the immune system learns how to defend itself through the experience of epidemics. When we do not allow this process to take place, then we will not have a stronger, but a weakened immune system. It is also true, of course, that if we allow all acute diseases to run their natural course some patients will die. Sometimes the “pneumonia” will be stronger than the defenses and the patient will succumb. This is the course of all acute diseases – they have a prodromal period, a climax, and an ending that results to either cure (lysis-dissolution) or death. This is where we have homeopathy’s and other alternative method’s contributions. It is possible, instead of suppressing an infection, to help the body to overcome the acute phase in a natural way by strengthening the defenses with a remedy that produces similar symptoms to the ones of the disease. In this way we help the body to regain its equilibrium. I previously spoke of a theory which I call “The continuum of a unified theory for diseases, acute and chronic.” According to this theory all chronic diseases, and all naturally occurring degenerative chronic illnesses, are “inflammatory processes” [88]. The main difference between an inflammatory process in a chronic condition and an acute inflammation is the manifestation of high fever during the acute phase [89–98].

The body, when it has the possibility of producing a high fever, is in a relatively good state of health. When it no longer has this ability, it means that we did not allow this inflammatory process to remain on the peripheral level, and now the inflammatory process has gone to a deeper level, to a vital organ or even to the whole system (e.g. systemic lupus erythematosus). The new situation is that now the body is eaten up with a sub-acute inflammatory process, with low fever or without apparent fever, but also without the possibility of a final dissolution (lysis). On the contrary, what takes place now is that the sub-acute inflammatory process is constantly destroying new areas and there is a continuous worsening of the chronic condition [99–108].

We have a similar inflammation like the peripheral acute but at a deeper level, and the body can no longer overcome it by producing a high fever, in spite of sporadic efforts. For example, people who suffer from migraines almost routinely say that they have periodic attacks [109–114], e.g. two or

SR

three times a week. In these cases, if we inquire, we will find that there were several acute diseases before the initiation of migraines, with high fever, such as acute tonsillitis, cystitis or bronchitis, which were mistreated and suppressed before the migraines appeared. It is the same inflammation that was not allowed to express itself when it was tonsillitis, and now the body regularly regains strength and makes an effort to reproduce the original acute state. The patient feels this effort as the syndrome we call migraine or "cluster headache". Some years ago, when I was talking about the continuum of diseases, I did not know the biochemical mechanism that would create these sub-acute inflammations. Later on, I read about the idea of inflammatory proteins [115–121]. One of my students brought me the results of a study conducted in the U.S., showing that schizophrenia is most probably an inflammatory process [122]. The immediate reaction of the medical researchers was to find an anti-inflammatory drug for curing schizophrenia. So, all these years we have not learned that this way of thinking is wrong, and we continue even in these last stages to insist on this way of thinking – eliminating the stressor, instead of strengthening the immune system. The conventional medical thinking is "Let us find the disease producing agent, find a chemical drug that can eliminate it and cure the patient." They fail to realize that these are inflammatory proteins and cannot be exterminated with a chemical, but **can only be treated by boosting the immune system**. If medicine does not part company with this way of thinking, humanity will continue to experience ever more complex illnesses, which will be increasingly difficult to cure.

Homeopathy is capable of bringing the immune system back to its original strength

The body has an ability to react against stressors from the environment, which must be augmented rather than suppressed so as to overcome the inflammation in a natural way, without pushing it to deeper levels. This whole process of reaction of the body that produces an acute inflammation with high fever is "the result of millions of biochemical reactions," having the purpose of re-establishing the lost balance – the homeostasis. If this process is interrupted and is forced to disappear by brutal chemical force, does not reach the climax of its healing mission which would bring about a homeostasis, the body is obliged to re-arrange its defenses and the immune system decides to take a deeper line of defense, internalizing the inflammatory process. This is the starting point of a chronic disease. It may be a collagen disease, lupus erythematosus, psoriasis, multiple sclerosis, neuromuscular disease, psychiatric disease, autism, etc., all have behind them a similar process. The important difference is that this time the immune system cannot anymore muster the strength to raise again a high fever in order to accomplish a cure. If there was a better system of treating the acute diseases with milder means, then the immune system would not need to compromise and accept the disturbance on a deeper level. Suppression of fever in pregnant mothers or in young child can cause the onset of autism [123]. Autistic children rarely have fevers – they had fevers before they became autistic, e.g. many of them had repeated otitis media with high fever that were suppressed by antibiotics or strong antipyretics. It is interesting to remark that if they develop fever their autistic condition ameliorates [124]. With the correct treatment the fevers will return, while autistic

behavior improves dramatically. Diseases of our modern societies are more and more affecting the peripheral and the central nervous systems. Given the fact that the most important organ is the brain, if we continue pushing disease to the centre of the body, we will soon be witnessing a tremendous increase in psychotic diseases.

Japanese researchers have found that they can cure cancer by producing fever [125] through different antigens in order to cause the rise of temperature, and they have had some really remarkable results. They introduced fever-inducing agents into the body, the body reacted and they saw an improvement in the cancer [126]. The same has been observed in children affected by autism [127].

We hope we gave the child an answer to his question – "Why do I get sick?" – why he had begun with tonsillitis and now has got to the point of having asthma.

DISCUSSION

Every step forward that man has taken in his scientific endeavors has always been met by resistance. Human history is full of such social and scientific revolutions that have shaken the existing foundations and beliefs of humans. Invariably such discoveries have required a period of many years for elaboration and acceptance. Each one of these important steps has, however, opened ever wider horizons that have allowed humans to evolve. Homeopathy represents one of these great revolutions, and since it acts on still unknown and little explored levels of the human world, is requiring more than two hundred years (since the time of its discovery by Samuel Hahnemann) before the scientific world open its mind to a serious evaluation of the tens of thousands of proofs, today in our hand, on its wonderful effect on humans and animals [128–129].

We are all aware of the fact that every living being cannot escape the laws of thermodynamics. According to the second of these laws, the entire universe spontaneously tends towards the maximum possible disorder. Only the supply of free energy in a system can counteract this increase of chaos. Nobody can deny the presence of a dualism in every living being, in which the universal tendency toward disorder (something that spontaneously occurs after the death of the being itself) is continuously counteracted by an intrinsic tendency toward the order and harmony in equilibrium of forces that maintain the being "alive".

The discovery of the complex and wonderful biochemical mechanisms of life induced humans to experience the possibility to affect them, by the introduction of other molecules into the body's systems, attempting to modify the pathways and control the "causes" of the malfunctioning. Actually, in an energetic system that has self-managed itself for more than four billion years, the biochemical mechanisms that we observe, when the system is unbalanced (for example during a high fever), are nothing other than the last effects of the best possible solution that the wonderful and intelligent defense mechanism found for trying to restore order in the system which has been upset by a stressor. Hence, these metabolic effects should not be interfered with by use of other chemical agents because they do nothing more than impede the defense mechanism in its intelligent expression

of recovery. On the contrary, it is necessary to promote the capacities of the defense mechanism by removing the “energetic” causes of its weakening.

CONCLUSIONS

Every human being is affected by diseases, acute and chronic, which are inter-connected throughout life in a “continuum of a unified substratum of diseases”, which leads up to the final disease condition that marks the end of life.

The question is whether medicine can discover ways of treating acute diseases – which constitute the beginning of the imbalance – with milder means, that promote and enhance the natural reaction of the immune system rather than suppressing it with strong chemical drugs and perhaps damaging the immune system irreparably. The defense mechanism as a whole appears to have a “higher” intelligence that is able to maintain optimum balance under any stress. But if, under certain conditions, the body cannot overtake and neutralize the stressor while the problem is on a peripheral level, this compromises its overall wellbeing and transfers the defense to a deeper level by mobilizing the defenses in a deeper and therefore more important organ or system, in this way marking the “beginning” of a chronic degenerative disease.

The model we present here emerges from almost fifty years of direct observations of tens of thousands of patients.

REFERENCES:

- Ahkee S, Srinath L, Ramirez J: Community-acquired pneumonia in the elderly: association of mortality with lack of fever and leukocytosis. *South Med J*, 1997; 90(3): 296–98
- Mowbray JF, Yousef GE: Immunology of postviral fatigue syndrome. *Br Med Bull*, 1991; 47(4): 886–94
- Lee WM: Hepatitis B virus infection. *N Engl J Med*, 1997; 337(24): 1733–45
- Guilherme L, Faé K, Oshiro SE, Kalil J: Molecular pathogenesis of rheumatic fever and rheumatic heart disease. *Expert Rev Mol Med*, 2005; 7(28): 1–15
- Guilherme L, Ramasawmy R, Kalil J: Rheumatic fever and rheumatic heart disease: genetics and pathogenesis. *Scand J Immunol*, 2007; 66(2–3): 199–207
- Guilherme L, Faé KC, Oshiro SE et al: Rheumatic fever: how S. pyogenes-primed peripheral T cells trigger heart valve lesions. *Ann NY Acad Sci*, 2005; 1051: 132–40
- Guilherme L, Ramasawmy R, Kalil J: T cell response in rheumatic fever: crossreactivity between streptococcal M protein peptides and heart tissue proteins. *Curr Protein Pept Sci*, 2007; 8(1): 39–44
- Guilherme L, Kalil J: Rheumatic fever: from sore throat to autoimmune heart lesions. *Int Arch Allergy Immunol*, 2004; 134(1): 56–64
- Guilherme L, Kalil J: Rheumatic fever: the T cell response leading to autoimmune aggression in the heart. *Autoimmun Rev*, 2002; 1(5): 261–66
- Chopra P, Gulwani H: Pathology and pathogenesis of rheumatic heart disease. *Indian J Pathol Microbiol*, 2007; 50(4): 685–97
- Gölbasi Z, Uçar O, Keles T et al: Increased levels of high sensitive C-reactive protein in patients with chronic rheumatic valve disease: evidence of ongoing inflammation. *Eur J Heart Fail*, 2002; 4(5): 593–95
- Otto CM: Aortic stenosis – listen to the patient, look at the valve. *N Engl J Med*, 2000; 343(9): 652–54
- Norman DC: Fever in the elderly. *Clin Infect Dis*, 2000; 31(1): 148–51
- Vithoulkas G: The three levels of the human being. In: Vithoulkas G (ed.), *The Science of Homeopathy*. Grove Press, 1st ed. NY, 1980; 23–44
- Vithoulkas G: The energy complex of the human body. In: Vithoulkas G (ed.), *A new model for health and disease*. North Atlantic Books, 1st ed. Berkeley, 1991; 42–61
- Kim YJ, Choi JY, Paek D, Chung HW: Association of the NQO1, MPO, and XRCC1 polymorphisms and chromosome damage among workers at a petroleum refinery. *J Toxicol Environ Health A*, 2008; 71(5): 333–41
- Nebert DW, Roe AL, Vandale SE et al: NAD(P)H: quinone oxidoreductase (NQO1) polymorphism, exposure to benzene, and predisposition to disease: a HuGE review. *Genet Med*, 2002; 4(2): 62–70
- Miki Y, Swensen J, Shattuck-Eidens D et al: A strong candidate for the breast and ovarian cancer susceptibility gene BRCA1. *Science*, 1994; 266(5182): 66–71
- Hugot JP, Chamaillard M, Zouali H et al: Association of NOD2 leucine-rich repeat variants with susceptibility to Crohn's disease. *Nature*, 2001; 411(6837): 599–603
- Tanaka S, Kobayashi T, Nakanishi K et al: Association of HLA-DQ genotype in autoantibody-negative and rapid-onset type 1 diabetes. *Diabetes Care*, 2002; 25(12): 2302–7
- Sumnik Z, Cinek O, Bratanic N et al: Risk of celiac disease in children with type 1 diabetes is modified by positivity for HLA-DQB1*02-DQA1*05 and TNF-308A. *Diabetes Care*, 2006; 29(4): 858–63
- Li PY, Chang YC, Tzang BS et al: Antibiotic amoxicillin induces DNA lesions in mammalian cells possibly via the reactive oxygen species. *Mutat Res*, 2007; 629(2): 133–39
- Arabski M, Kazmierczak P, Wisniewska-Jarosinska M et al: Interaction of amoxicillin with DNA in human lymphocytes and H. pylori-infected and non-infected gastric mucosa cells. *Chem Biol Interact*, 2005; 152(1): 13–24
- González C, Nájera O, Cortés E et al: Susceptibility to DNA damage induced by antibiotics in lymphocytes from malnourished children. *Teratog Carcinog Mutagen*, 2002; 22(2): 147–58
- Friedman GD, Oestreicher N, Chan J et al: Antibiotics and risk of breast cancer: up to 9 years of follow-up of 2.1 million women. *Cancer Epidemiol Biomarkers Prev*, 2006; 15(11): 2102–6
- Sesti F, Abbott GW, Wei J et al: A common polymorphism associated with antibiotic-induced cardiac arrhythmia. *Proc Natl Acad Sci USA*, 2000; 97(19): 10613–18
- Velicer CM, Heckbert SR, Lampe JW et al: Antibiotic use in relation to the risk of breast cancer. *JAMA*, 2004; 291(7): 827–35
- Knekt P, Adlercreutz H, Rissanen H et al: Does antibacterial treatment for urinary tract infection contribute to the risk of breast cancer? *Br J Cancer*, 2000; 82(5): 1107–10
- Didham RC, Reith DM, McConnell DW, Harrison KS: Antibiotic exposure and breast cancer in New Zealand. *Breast Cancer Res Treat*, 2005; 92(2): 163–67
- Voiculescu C, Stanciu L, Voiculescu M et al: Experimental study of antibiotic-induced immunosuppression in mice. I. Humoral and cell-mediated immune responsiveness related to *in vivo* antibiotic treatment. *Comp Immunol Microbiol Infect Dis*, 1983; 6(4): 291–99
- Voiculescu C, Stanciu L, Voiculescu M et al: Experimental study of antibiotic-induced immunosuppression in mice. II. Th, Ts and NC cell involvement. *Comp Immunol Microbiol Infect Dis*, 1983; 6(4): 301–12
- Kim YJ, Choi JY, Paek D, Chung HW: Association of the NQO1, MPO, and XRCC1 polymorphisms and chromosome damage among workers at a petroleum refinery. *J Toxicol Environ Health A*, 2008; 71(5): 333–41
- Nebert DW, Roe AL, Vandale SE et al: NAD(P)H: quinone oxidoreductase (NQO1) polymorphism, exposure to benzene, and predisposition to disease: a HuGE review. *Genet Med*, 2002; 4(2): 62–70
- Fortini P, Pascucci B, Parlanti E et al: The base excision repair: mechanisms and its relevance for cancer susceptibility. *Biochimie*, 2003; 85(11): 1053–71
- Tomioka R, Tani K, Sato K et al: Observations on the occurrence of exacerbations in clinical course of systemic lupus erythematosus. *J Med Invest*, 2008; 55(1–2): 112–19
- Panitch HS: Interferons in multiple sclerosis. A review of the evidence. *Drugs*, 1992; 44(6): 946–62
- Rancé F, Boguniewicz M, Lau S: New visions for atopic eczema: an iPAC summary and future trends. *Pediatr Allergy Immunol*, 2008; 19(Suppl.19): 17–25
- Brand HS, van Beusichem FF, van Nieuw Amerongen A: Behçet's disease. *Ned Tijdschr Tandheelkd*, 2008; 115(6): 340–45
- Abularraja CJ, Slidell MB, Sidawy AN et al: Quality of life of patients with Takayasu's arteritis. *J Vasc Surg*, 2008; 47(1): 131–36
- Susac A, Babić S, Lipozencić J: An overview on atopic dermatitis in children. *Acta Dermatovenol Croat*, 2007; 15(3): 158–66

41. Van Rossum MM, van der Avoort IA, de Hoop D et al: Lichen sclerosis. *Ned Tijdschr Geneesk*, 2007; 151(22): 1225–31
42. Dunand M, Lalive PH, Vokatch N, Kuntzer T: Myasthenia gravis: treatments and remissions. *Rev Med Suisse*, 2007; 3(110): 1185–86, 1188–90
43. Shields LB, Hunsaker DM, Hunsaker JC III: Schizophrenia and suicide: a 10-year review of Kentucky medical examiner cases. *J Forensic Sci*, 2007; 52(4): 930–37
44. El-Shanti HI, Ferguson PJ: Chronic recurrent multifocal osteomyelitis: a concise review and genetic update. *Clin Orthop Relat Res*, 2007; 462: 11–19
45. American Academy of Allergy Asthma & Immunology. Asthma statistics. http://www.aaaai.org/media/resources/media_kit/asthma_statistics.stm
46. Koutroubakis IE, Vlachonikolis IG, Kapsorita et al: Appendectomy, tonsillectomy, and risk of inflammatory bowel disease: case-controlled study in Crete. *Dis Colon Rectum*, 1999; 42(2): 225–30
47. Maté-Jimenez J, Correa-Estañ JA, Perez-Miranda M et al: Tonsillectomy and inflammatory bowel disease location. *Eur J Gastroenterol Hepatol*, 1996; 8(12): 1185–88
48. Timio M, Capodicasa E, Ippolito Albertini and Michael Albertus: disparate old and innovative theories on dropsy and edema. *Am J Nephrol*, 2002; 22(2–3): 220–24
49. USA deaths statistics. http://www.whale.to/a/usa_deaths.html
50. Statistics graphs. <http://www.whale.to/a/graphs.html>
51. Autism increase. http://www.whale.to/a/autism_increase.html
52. Bruce TJ, Pickett JA: Plant defence signalling induced by biotic attacks. *Curr Opin Plant Biol*, 2007; 10(4): 387–92
53. Dangl JL, Jones JD: Plant pathogens and integrated defence responses to infection. *Nature*, 2001; 411(6839): 826–33
54. McPhee JB, Scott MG, Hancock RE: Design of host defence peptides for antimicrobial and immunity enhancing activities. *Comb Chem High Throughput Screen*, 2005; 8(3): 257–72
55. Kraus D, Peschel A: *Staphylococcus aureus* evasion of innate antimicrobial defense. *Future Microbiol*, 2008; 3: 437–51
56. Lee HY, Andalibi A, Webster P et al: Antimicrobial activity of innate immune molecules against *Streptococcus pneumoniae*, *Moraxella catarrhalis* and nontypeable *Haemophilus influenzae*. *BMC Infect Dis*, 2004; 4: 12
57. Blanco JL, Garcia ME: Immune response to fungal infections. *Vet Immunol Immunopathol*, 2008; 125(1–2): 47–70
58. Lanier LL: Evolutionary struggles between NK cells and viruses. *Nat Rev Immunol*, 2008; 8(4): 259–68
59. Pachter JS, de Vries HE, Fabry Z: The blood-brain barrier and its role in immune privilege in the central nervous system. *J Neuropathol Exp Neurol*, 2003; 62(6): 593–604
60. GreenFacts. Scientific facts on respiratory diseases in children. <http://www.greenfacts.org/en/respiratory-diseases/t-3/01-effects-children.htm#0p0>
61. Polosa R, Al-Delaimy WK, Russo C et al: Greater risk of incident asthma cases in adults with allergic rhinitis and effect of allergen immunotherapy: a retrospective cohort study. *Respir Res*, 2005; 6: 153
62. Guerra S, Sherrill DL, Martinez FD, Barbee RA: Rhinitis as an independent risk factor for adult-onset asthma. *J Allergy Clin Immunol*, 2002; 109(3): 419–25
63. Leynaert B, Bousquet J, Neukirch C et al: Perennial rhinitis: An independent risk factor for asthma in nonatopic subjects: results from the European Community Respiratory Health Survey. *J Allergy Clin Immunol*, 1999; 104: 301–4
64. Leynaert B, Neukirch C, Kony S et al: Association between asthma and rhinitis according to atopic sensitization in a population-based study. *J Allergy Clin Immunol*, 2004; 113(1): 86–93
65. Shaaban R, Zureik M, Soussan D et al: Allergic rhinitis and onset of bronchial hyperresponsiveness: a population-based study. *Am J Respir Crit Care Med*, 2007; 176(7): 659–66
66. Mete N, Sin A, Gulbahar O et al: The determinants of bronchial hyperresponsiveness in patients with allergic rhinitis. *Ann Allergy Asthma Immunol*, 2004; 93(2): 193–99
67. Valdesoiro L, Bosque M, Marco MT et al: Allergic rhinitis and bronchial hyperreactivity. *Allergol Immunopathol (Madr)*, 2004; 32(6): 340–43
68. Warren JW, Brown V, Jacobs S et al: Urinary tract infection and inflammation at onset of interstitial cystitis/painful bladder syndrome. *Urology*, 2008; 71(6): 1085–90
69. Orellana P, Cavagnaro F, Baquedano P et al: Risk factors for permanent kidney damage in children with urinary tract infection. *Rev Med Chil*, 2002; 130(10): 1147–53
70. Heinle S, Stünkel K, Zähler H et al: Immunosuppressive effects of the macrolide antibiotic bafilomycin towards lymphocytes and lymphoid cell lines. *Arzneimittelforschung*, 1988; 38(8): 1130–33
71. Tanouchi Y, Shichi H: Immunosuppressive and anti-proliferative effects of a macrolide antibiotic, tetracycline. *Immunology*, 1988; 63(3): 471–75
72. De Simone C, Pugnali L, Cilli A et al: Pharmacokinetic assessment of immunosuppressive activity of antibiotics in human plasma by a modification of the mixed lymphocyte reaction. *Crit Care Med*, 1984; 12(6): 483–85
73. Horáková L, Nouza K, Pospíšil M et al: Immunosuppressive properties of the antibiotics cytosipin and vermiculine. *Folia Biol (Praha)*, 1980; 26(5): 312–26
74. Siefert G, May DJ, Günther B: Immunosuppressive effect of common antibiotics. *Arzneimittelforschung*, 1971; 21(12): 2109–12
75. Bartlett JG, Chang TW, Gurwith M et al: Antibiotic-associated pseudomembranous colitis due to toxin-producing clostridia. *N Engl J Med*, 1978; 298(10): 531–34
76. Bartlett JG, Moon N, Chang TW et al: Role of *Clostridium difficile* in antibiotic-associated pseudomembranous colitis. *Gastroenterology*, 1978; 75(5): 778–82
77. Bartlett JG: Antibiotic-associated pseudomembranous colitis. *Rev Infect Dis*, 1979; 1(3): 530–39
78. Rufo PA, Bousvaros A: Current therapy of inflammatory bowel disease in children. *Paediatr Drugs*, 2006; 8(5): 279–302
79. Botoman VA, Bonner GF, Botoman DA: Management of inflammatory bowel disease. *Am Fam Physician*, 1998; 57(1): 57–68, 71–72
80. D'Haens GR, Lashner BA, Hanauer SB: Pericholangitis and sclerosing cholangitis are risk factors for dysplasia and cancer in ulcerative colitis. *Am J Gastroenterol*, 1993; 88(8): 1174–78
81. Lewis JD, Gelfand JM, Troxel AB et al: Immunosuppressant medications and mortality in inflammatory bowel disease. *Am J Gastroenterol*, 2008; 103(6): 1428–35
82. Hordijk ML, Shivananda S: Risk of cancer in inflammatory bowel disease: why are the results in the reviewed literature so varied? *Scand J Gastroenterol Suppl*, 1989; 170: 70–74
83. Mir-Madjlessi SH, Forouzandeh B, Ghadimi R: Ulcerative colitis in Iran: a review of 112 cases. *Am J Gastroenterol*, 1985; 80(11): 862–66
84. Pongprasobchai S, Manatsathit S, Leelakulsong S et al: Ulcerative colitis in Thailand: a clinical study and long term follow-up. *J Med Assoc Thai*, 2001; 84(9): 1281–88
85. Finegold SM, Molitoris D, Song Y et al: Gastrointestinal microflora studies in late-onset autism. *Clin Infect Dis*, 2002; 35(Suppl.1): S6–S16
86. Parracho HM, Bingham MO, Gibson GR, McCartney AL: Differences between the gut microflora of children with autistic spectrum disorders and that of healthy children. *J Med Microbiol*, 2005; 54: 987–91
87. Song Y, Liu C, Finegold SM: Real-time PCR quantitation of clostridia in feces of autistic children. *Appl Environ Microbiol*, 2004; 70(11): 6459–65
88. University of California, Berkeley Wellness Letter The newsletter of nutrition, fitness, and self-care. 2008;24: 4 From the School of Public Health – WellnessLetter.com 10/2/2009
89. Naudin J, Mège JL, Azorin JM, Dassa D: Elevated circulating levels of IL-6 in schizophrenia. *Schizophr Res*, 1996; 20(3): 269–73
90. Kluger MJ, Kozak W, Conn CA et al: Role of fever in disease. *Ann NY Acad Sci*, 1998; 856: 224–33
91. Kluger MJ: Fever. *Pediatrics*, 1980; 66(5): 720–24
92. Cunha BA: Symposium on infections in the compromised host. Significance of fever in the compromised host. *Nurs Clin North Am*, 1985; 20(1): 163–69
93. Bernheim HA, Block LH, Atkins E: Fever: pathogenesis, pathophysiology, and purpose. *Ann Intern Med*, 1979; 91(2): 261–70
94. Soszynski D: The pathogenesis and the adaptive value of fever. *Postepy Hig Med Dosw*, 2003; 57(5): 531–54
95. Kluger MJ: Phylogeny of fever. *Fed Proc*, 1979; 38(1): 30–34
96. Kluger MJ, Kozak W, Conn CA et al: The adaptive value of fever. *Infect Dis Clin North Am*, 1996; 10(1): 1–20
97. Hasday JD, Fairchild KD, Shanholtz C: The role of fever in the infected host. *Microbes Infect*, 2000; 2(15): 1891–904
98. Hasday JD, Garrison A: Antipyretic therapy in patients with sepsis. *Clin Infect Dis*, 2000; 31(Suppl.5): S234–41

99. Romanovsky AA, Székely M: Fever and hypothermia: two adaptive thermoregulatory responses to systemic inflammation. *Med Hypotheses*, 1998; 50(3): 219–26
100. Meira LB, Bugni JM, Green SL et al: DNA damage induced by chronic inflammation contributes to colon carcinogenesis in mice. *J Clin Invest*, 2008; 118(7): 2516–25
101. Ernst P: Review article: the role of inflammation in the pathogenesis of gastric cancer. *Aliment Pharmacol Ther*, 1999; 13(Suppl.1): 13–18
102. Chavarria A, Alcocer-Varela J: Is damage in central nervous system due to inflammation? *Autoimmun Rev*, 2004; 3(4): 251–60
103. Butterfield TA, Best TM, Merrick MA: The dual roles of neutrophils and macrophages in inflammation: a critical balance between tissue damage and repair. *J Athl Train*, 2006; 41(4): 457–65
104. Kundu JK, Surh YJ: Inflammation: Gearing the journey to cancer *Mutat Res*, 2008; 659(1–2): 15–30
105. Fujita N, Sugimoto R, Ma N et al: Comparison of hepatic oxidative DNA damage in patients with chronic hepatitis B and C. *J Viral Hepat*, 2008; 15(7): 498–507
106. McKay CJ, Glen P, McMillan DC: Chronic inflammation and pancreatic cancer. *Best Pract Res Clin Gastroenterol*, 2008; 22(1): 65–73
107. Farrow B, Sugiyama Y, Chen A et al: Inflammatory mechanisms contributing to pancreatic cancer development. *Ann Surg*, 2004; 239(6): 763–69
108. Farinati F, Cardin R, Bortolami M et al: Hepatitis C virus: from oxygen free radicals to hepatocellular carcinoma. *J Viral Hepat*, 2007; 14(12): 821–29
109. Meinel E, Krumbholz M, Derfuss T et al: Compartmentalization of inflammation in the CNS: A major mechanism driving progressive multiple sclerosis. *J Neurol Sci*, 2008; 274: 42–44
110. Leroux E, Ducros A: Cluster headache. *Orphanet J Rare Dis*, 2008; 3: 20
111. Greenfield DP, Hariharan S: Diagnosis and clinical management of headaches. *CNS Spectr*, 1999; 4(9): 32–47
112. Alstadhaug KB, Bekkelund S, Salvesen R: Circannual periodicity of migraine? *Eur J Neurol*, 2007; 14(9): 983–88
113. Alstadhaug K, Salvesen R, Bekkelund S: Insomnia and circadian variation of attacks in episodic migraine. *Headache*, 2007; 47(8): 1184–88
114. Alstadhaug KB, Salvesen R, Bekkelund SI: Seasonal variation in migraine. *Cephalalgia*, 2005; 25(10): 811–16
115. Alstadhaug KB, Salvesen R, Bekkelund S: Weekend migraine. *Cephalalgia*, 2007; 27(4): 343–46
116. Zeremski M, Petrovic LM, Talal AH: The role of chemokines as inflammatory mediators in chronic hepatitis C virus infection. *J Viral Hepat*, 2007; 14(10): 675–87
117. Driscoll KE: Macrophage inflammatory proteins: biology and role in pulmonary inflammation. *Exp Lung Res*, 1994; 20(6): 473–90
118. Hachicha M, Naccache PH, McColl SR: Inflammatory microcrystals differentially regulate the secretion of macrophage inflammatory protein 1 and interleukin 8 by human neutrophils: a possible mechanism of neutrophil recruitment to sites of inflammation in synovitis. *J Exp Med*, 1995; 182(6): 2019–25
119. Smith RE, Strieter RM, Zhang K et al: A role for C-C chemokines in fibrotic lung disease. *J Leukoc Biol*, 1995; 57(5): 782–87
120. Zoccali C, Mallamaci F, Tripepi G: Inflammatory proteins as predictors of cardiovascular disease in patients with end-stage renal disease. *Nephrol Dial Transplant*, 2004; 19(Suppl.5): V67–72
121. Chait A, Han CY, Oram JF, Heinecke JW: Thematic review series: The immune system and atherogenesis. Lipoprotein-associated inflammatory proteins: markers or mediators of cardiovascular disease? *J Lipid Res*, 2005; 46(3): 389–403
122. O'Brien KD, Chait A: Serum amyloid A: the “other” inflammatory protein. *Curr Atheroscler Rep*, 2006; 8(1): 62–68
123. Naudin J, Mège JL, Azorin JM, Dassa D: Elevated circulating levels of IL-6 in schizophrenia. *Schizophr Res*, 1996; 20(3): 269–73
124. Torres AR: Is fever suppression involved in the etiology of autism and neurodevelopmental disorders? *BMC Pediatr*, 2003; 3: 9
125. Hobohm U: Fever therapy revisited. *Br J Cancer*, 2005; 92(3): 421–25
126. Hobohm U: Fever and cancer in perspective. *Cancer Immunol Immunother*, 2001; 50(8): 391–96
127. Curran LK, Newschaffer CJ, Lee LC et al: Behaviors associated with fever in children with autism spectrum disorders. *Pediatrics*, 2007; 120(6): e1386–92
128. Mastrangelo D, Loré C: The growth of a lie and the end of “conventional” medicine. *Med Sci Monit*, 2005; 11(12): SR27–31
129. Mastrangelo D: Hormesis, epitaxy, the structure of liquid water, and the science of homeopathy. *Med Sci Monit*, 2007; 13(1): SR1–8