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Dr. med. Heinrich Kremer: A silent revolution in cancer and AIDS therapy

New research results concerning the factual causes of disease and death, confirm the effectiveness of a therapy based on biological compensation.

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Excerpt from chapter XI: pp. 416-443

“The lifesaving knowledge on healing“

On the diagnosis, prophylaxis and applied therapy for AIDS and other systemic diseases – balance instead of elimination

[excerpt begins on page 416]

Disposition factors can be epidemiologically justified. They also explain why pathogenic distribution patterns manifest in a Gaussian curve, as equally for excessive exposures as in collective populations and differing risk groups.

While the majority of the individuals so exposed will maintain a variable redox balance with flexible cytokine patterns, a minor percentage will develop a distinct Type-1 overreaction leading to a distinct Type-2 counterregulation. To presume a highly contagious and inevitably fatal epidemic was an *a priori* medical construction far beyond any biological-evolutionary reality.

In the case of so-called HIV-induced AIDS, it was particularly questionable to presume that the humoral (antibody-supported) immunity was operating successfully (Mildvan 1982), while the cellular immunity of the T4 helper cells against intracellular germs failed such that opportunistic infections could develop. Only when patients were treated with AZT chemotherapy did massive bacterial infections occur, as a consequence of the inhibited maturation process of bone marrow cells (Rosenthal 1994), (Marco 1998, Cox 1998).

Because the evolutionary-biologically programmed co-action of exposure and disposition factors have not been sufficiently understood, HIV/AIDS medicine has provoked the appearance of acquired immunodeficiency on both the cellular and humoral levels (severe combined immunodeficiency, or SCID), by prescribing chemotherapeutics on the basis of the objectively incorrect theory that “HIV causes AIDS.” The clinical and epidemiological results demonstrate with abundant clarity, the deficits of modern medicine that result from the underestimation of toxic and pharmatoxic stressors, and from combining sophisticated 20th century biotechnology with a narrow fixation on outdated 19th-century germ theories.

The massive projection of a supposed pandemic by the HIV/AIDS establishment reveals that with the situation of doubtlessly increased collective load of immune stressors (endemic multiple infections, contaminated drinking water, malnutrition, adverse general life conditions, [industrial toxins,] etc.), special disposition factors must also play a role in triggering acquired immunodeficiency.

Prof. Duesberg, retrovirus-cancer scientist and molecular biologist at UC Berkeley, California, is one of the most severe critics of the "HIV causes AIDS" theory. He considers the presumed HI-Virus to be a harmless "passenger virus", regarding toxic reasons like excessive consumption of street drugs, nitrate inhalation as a sexual doping agent, medical chemotherapy, and the intake of highly concentrated coagulated protein [unpurified Factor VIII] as the true causes of AIDS. At the occasion of the July 2000 AIDS Specialist's conference in Pretoria, South Africa, he stated:

"In the light of this hypothesis, the new epidemic of HIV-antibodies would simply reflect a new epidemic of HIV-antibody testing—introduced and inspired by new American biotechnology. This technology was developed during the last 20 years for basic research to detect the equivalents of biological needles in a haystack, but not to "detect" the massive invasions of viruses that are necessary to cause ALL conventional viral diseases (Duesberg, 1992; Duesberg & Schwartz, 1992; Duesberg, 1996; Mullis, 1996; Duesberg & Rasnick, 1998; Mullis, 1998). But this technology is now faithfully yet inappropriately used by thousands of AIDS-virus researchers and activists, to detect latent (ie. biochemically and biologically inactive) HIV or even just antibodies against it (Duesberg & Bialy, 1996)! The same technology also provides job security for other virologists and doctors searching for latent, and thus biologically inactive, viruses as their preferred causes of Kaposi's sarcoma, cervical cancer, leukaemia, liver cancer, and rare neurological diseases - without ever producing any public health benefits... (Duesberg & Schwartz, 1992). To all of us who have been subjected to the American AIDS rhetoric, and indeed the rhetoric of our first meeting in Pretoria last May, about the "catastrophic dimensions" of African AIDS (Washington Post, April 30, 2000), the healthy African growth rates come as a big surprise. Take as an example of this rhetoric President Clinton's recent designation of AIDS as a "threat to US national security ... spurred by US intelligence reports that looked at the pandemic's broadest consequences, ... particularly Africa ... [and] projected that a quarter of southern Africa's population is likely to die of AIDS..." (Washington Post, April 30, 2000). The alarming tone of WHO's joint United Nations Programme on HIV/AIDS, "AIDS epidemic update: December 1999" (UNAIDS December 1999), announcing that Africa had gained 23 million "living with HIV/AIDS", because they are "estimated" carriers of antibodies against HIV, since the "early 80s" (WHO, Weekly Epidemiological Record 73, 373-380, 1998) is equally surprising in view of information available to the agency. Neither the WHO nor the United Nations point out that Africa had gained 147 million people during the same time in which the continent was said to suffer from a new AIDS epidemic.

Likewise, South Africa has grown from 17 million to 37 million in 1990 (United Nations Environment Programme, June 15, 2000), and to 44 million now ("HIV/AIDS in the Developing World", U.S. Agency for International Development & U.S. Census Bureau, May 1999). In the last decade South Africa has also gained 4 million HIV-positive people (A. Kinghorn & M. Steinberg, South African Department of Health, undated document probably from 1998, provided at the Pretoria meeting). Thus South Africa has gained 4 million HIV-positives during the same decade in which it grew by 7 million people. Moreover, although the 23 million "estimated" HIV-antibody positives are said to be "living with HIV/AIDS" by the WHO, the agency does not offer any evidence for morbidity or mortality exceeding the modest numbers, i.e. about 75,000 cases annually, reported by the it's Weekly Epidemiological Records (0,012% of Africa's whole population) (WHO Weekly Epidemiological Records 73,373-380, 1998). The agency's estimates of HIV-positives are indeed just "estimates", because according to the 1985 Bangui definition of African AIDS, as well as the current "Anonymous AIDS Notification" forms of the South African Department of Health, no HIV tests are required at all for an AIDS diagnosis (Widy-Wirski et al., 1988; Fiala, 1998). In addition, the WHO promotes the impression of an infectious AIDS epidemic, by reporting African AIDS cases cumulatively rather than annually (WHO's Weekly Epidemiological Records since the beginning of the epidemic). This practice creates the deceptive impression of a nearly exponential, ever-growing epidemic, even if the annual incidence has declined (Fiala, 1998). It follows that the estimated increase in African "HIV antibody"(!)-positives should correlate with a decrease in population, yet this is not this case.

On the contrary, the increase in African "HIV antibody"-positives correlates simultaneously with unprecedented increases in the continental population (increase of 617 million inhabitants of sub-Saharan Africa) - hardly the "catastrophe" (after the assertion of President Clinton) imagined by the Washington Post and propagated by the WHO and the American AIDS establishment. For those who are not aware of the facts, this deceptive propaganda prevents a truly scientific analysis of African AIDS." (Duesberg 2000)

In other words, the actual data recorded in the WHO epidemiological reports total morbidity and death rates in African states to be hardly higher than in western nations. Namely, 0.012% of Africa's total population fall ill and die of AIDS per year (WHO Weekly Epidemiological reports since 1991), compared to 0.001 to 0.002% of total populations in western countries (CDC 1999, Robert Koch Institute 1999). The absurd propagandistic claims insisting on an "African pandemic" distributed to the international media by the WHO, are based on arbitrary extrapolation of small random samples received by abusing "American biotechnology", using the so-called "anti-HIV antibody" test (Duesberg 2000). Based on the unreliable acquisition of pathogen data, compared to western countries, and because of the small pool of medical research results in developing countries, it is much less obvious to draw conclusions on the interaction of exposition and disposition for morbidity and mortality, in causal connection with the systemic diseases of Type-2 cellular dysbiosis. However the population explosion in African countries is comparable to demographic processes in western countries 150 years ago: a decline in infectious disease and a rise in toxic stressors, with the gradual improvement of general life conditions and of medical and social standards. Increased collective exposure to a variety of non-sexual immunostressors and the "surprisingly" (Duesberg 2000) low AIDS incidence (WHO Weekly Epidemiological Records since 1991) as compared to Western countries, in parallel with the African population explosion, gives reason to suppose that disposition factors must also play a role. Consequently, the recommendation for developing countries is the same as for Western nations: protection from the "abuse of American biotechnology" (Duesberg 2000) and the "blessings" of Western chemotherapy and chemoantibiotics, while promoting knowledge of evolution-biologically programmed redox protection.

Disposition factors affect the system that controls peroxidation (creation of hydrogen and lipid peroxides) and nitrosylation (the bonding of NO and its derivatives with the hydrogen sulphide groups of proteins containing cysteine, RSNO) of transcription proteins. In the event of extreme glutathione expenditure, this regulatory control system acts to increase the activity of the antioxidative gene, and thus the rate of H₂O₂ and lipid peroxidation and RSNO biotransformation (Hausladen 1996). After the depletion of glutathione and other antioxidant enzymes (catalase, superoxide-dismutase, selenium-dependent glutathione peroxidase, glutathione transferases, NADH dependent glutathione reductase), a hypoxic/pseudo-hypoxic emergency routine will be switched on. In the context of biological evolution, the early and sustained switch of the cytokine balance to the humoral antibody-supported immune response was advantageous, as an efficient mechanism against the bacterial threat predominant in the course of evolution. Bacteria proliferate faster than opportunistic pathogens. They can be efficiently inhibited and destroyed through the defense mechanisms of the non-cell-mediated humoral immunity: complement formation, opsonisation (the coating of bacterial membranes by special target molecules for antibodies) and of the antibodies themselves, which are formed by B-lymphocytes matured in the bone marrow. The majority of fungal pathogens and parasites, equipped with mitochondria, and mycobacteria provided with a special cell membrane, are stopped most efficiently by the co-action of the specific and non-specific immune cell network. If not inhibited by the NO gas attack in a timely manner, many parasites can inhibit the NO gas synthesis by special surface molecules (glycoinositol phospholipids). Extra- and multi-cellular parasites can emit specialized tissue-destructive proteins (proteases), which trigger a Type-2 cytokine response; the Th2 immune response is a suitable reaction in this case, since combating, for example worms, [via Th1 defense] would need such a large quantity of NO gas that the host's own tissue cells would be damaged.

Metastatic cancer cells also utilize proteases to deactivate the production of NO gas in neighbouring cells; in this manner they can pervasively invade tissue. Characteristic to cancer cells is a degradation of gas production (Ignarro 2000) and extreme irritability to high levels of intracellular NO gas (Xie 1996, Chinje 1997). Consequently, a one-sided Th2 (Type-2 cytokine) immune response creates an unfavorable disposition for blocking both intracellular pathogens (fungi, parasites, mycobacteria, some virus species) and metastases (Zvibel 1993, Liew 1994, 1995a, 1995b, Mosmann 1996, Abbas 1996, Lucey 1996, Xie 1996). Thus, everything depends on an appropriate and flexible combination of the defense and regulatory strategy.

The redox-sensitive disposition for lasting Type-2 cytokine immune response was once an evolutionary advantage, which has changed in relevance to the toxic and pharmatoxic effects of civilization; by the net result of civic progress and the advancement of modern medicine, especially through the introduction of vaccination programs and antibiotics over the past 50 years, the impact of these toxic stressors has grown in importance.

Persons with a particularly high redox-sensitive disposition are at a disadvantage because they respond to toxic influences faster and with an exaggerated Type-2 counterregulation, in the manner normally suited for inhibiting extracellular bacteria or multicellular parasites. Thus, the immune system activates the biological program that is appropriate in an evolutionary context, yet is the “wrong” strategy for our modern times; the system is misled by toxic stressors that did not exist as part of natural history.

This development is reflected by the steady rise of cancer and other systemic diseases over the past 100 years in the developed countries. The quintessential source of toxic exposure which favours the development of cancer and other systemic diseases in the industrialized countries, are presently: toxin residues in food and beverages, in the environment and at working places, and the use of tobacco (Loeppky 1994, Walker 1998, Waite 1998, North 1998), as well as pharmatoxic medication and their toxic metabolites.

An individual's disposition for the efficiency of redox-dependant detoxification capacity is a critical disease factor for the actual incidence of Type-2 cell dysbiosis via toxic and pharmatoxic nitrosylation and peroxidation.

On occasion of the Nobel prize laureate conference held in Lindau on Lake Constance in 1967, Warburg made his historic declaration: “there is no disease whose prime cause is better known than cancer” (Warburg 1967). Since then, an expanding field of research has been established, that deals with an individual's disposition for metabolic processes involving the detoxification of medical drugs in the human organism.

This field of research has very quickly expanded on the direct and indirect effects of toxic substances on cancer genesis (overview: Kalow 1993, Daly 1994). These studies concentrate, according to the focus on molecular genetics in mainstream cancer research, on the variability of genetic expression for biosynthesis of detoxification enzyme-proteins (genetic enzyme polymorphism): The paradigm of carcinogenic chemical products' mechanisms of application is well established in a cell culture model and in animal systems. [???

Studies on human beings support the possibility that most cancer forms can be triggered via contact with chemicals and nutrition, and that they progress through various precancerous stages of tumorigenic tissue damage, consisting of partially transformed cells to fully developed metastases (Vogelstein 1993).

The progression stadium can be increased in rat models through treatment with tumour promoting substances that are not necessarily carcinogenic (Hennings 1993). Affixing the mutation in the genome, those chemicals are to be visualized as intermediaries for cell

proliferation. Another class of chemicals called non-genotoxic carcinogens was described in rat model systems (Jackson 1993, Barret 1995, Costa 1995). These substances are not activated as genotoxic metabolites, but presumably alter control of the cellular division cycle. Many non-genotoxic carcinogenics are also tumour promoters, but their mechanisms of action are as of yet unknown.

The concept persists that humans differ in their sensitivity to cancer. Certain individuals can be more sensitive while others are more cancer resistant. This may be due to a number of factors that include state of health, nutritional condition, and gender. Considering all the known facts regarding the mechanism of carcinogens, the general presumption is that genetic background might play an important role. Complying with such a theory, obvious candidates are the genes that code for enzymes (Gonzales 1995, Nebert 1996). The expression of such enzymes in variable quantities, can result from an increase or decrease in carcinogenic activation. It is indeed well established that genetic differences appear during the expression of detoxification enzymes (Hirvonen 1999). Re-foetalized tumour cells can appear as a matter of time (via Type-2 counterregulation of cellular dysbiosis).

In certain kinds of cells, working with prooxidative chemotherapeutics allows for the selective forcing of the desired apoptosis/necrosis (Type-1 cell symbiosis overregulation); on the other hand, this can also accelerate other kinds of cells into fully transformed metastases. Primarily in tumour tissue, and also secondarily in other sorts of tissue, all phases of the yet-to-be compensated cell dysbiosis, can switch over unpredictably into a decompensated cell dysbiosis stadia.

It is characteristic for the chemotherapeutical treatment principle that patients with a particularly increased redox sensitivity disposition, who fell ill because of this genetically and supra-genetically disposed redox sensitivity, will not only have exact cell dysbioses in manifest tumour tissue but also in other tissue types and that tumour cells will respond to the chemotherapeutic target attack in manifest decompensated tissue in different manners in different stadia. [??]

Therefore a homogenous response of tumour cells to chemotherapy is a conditional assumption, and the results of such a therapy on an individual cannot be sufficiently calculated. Such is the distinctive genetic polymorphism of carcinogen-activating detoxification enzymes, made manifest in the course of a systemic disease.

Redox-sensitive variability in detoxification enzymes applies mainly to cytochrome P450-dependant and flavin-containing monooxidase, epoxide hydrolase, glutathione transferase, N-acetyl transferase, NAD(P)H-ubiquinone oxidoreductase, myeloperoxidase and others (overview: Wilkinson 1997, Hirvonen 1999). Such patients depend most urgently on an immediate balancing of the thiol pool and redox state. Chemotherapeutical treatment and the consequent extreme prooxidative stress inevitably results in a counterproductive effect, because such a therapy is usually done without compensating for the depletion of the thiol pool. Chemotherapy triggers not only the desired cytotoxic effects, but also dysbiotic cellular counterregulations that cannot be predicted on an individual basis; among other things, this leads to systemic wasting syndrome (cachexia).

Though resulting in questionable conclusions, genetic tests have been developed to determine the individually-disposed variability of detoxification enzyme isoforms. In the USA, for example, prophylactic mastectomies are being performed in order to avoid breast cancer. Such prognostication by means of genetic tests is for different reasons to be assessed most critically. If they have any significance at all, they could only give reason to provide balance and regulation therapy via nutrition, as a targeted influence on an individual's exposition-disposition interaction. In order to determine which metabolic genome types indicate a possible risk, a rapid progress in methodology must take place. This progress includes minimally invasive methods for extracting test probes (for example cells from oral mucosa or

cell probes from urine), automatic DNA extraction in combination with processing probes by robots, and genetic test methods based on specialized analysis of oligonucleotides.

At present, many research laboratories are researching the association between genetics and cancer. Some contradictory reports have appeared in the technical literature, which may be potentially false interpretations; initially, it is the smaller studies that show a positive correlation. This results in significant problems when gauging their importance, during the planning of follow-up studies. Many public reports of a high correlation between gene aberrances and cancer incidence have eventually turned out to be false, and therefore are equally problematic. Furthermore, recent discussions have assessed only one side of the issue, resulting in the selective publication of only positive associations.

If the above mentioned, potentially false interpretations can be meticulously controlled, genetic triage studies can become more helpful in the near future for identifying susceptible persons and population subgroups exposed to environmental toxins. Genetic biotechnology corporations now offer genetic tests to both companies and individuals. Until these methods are scientifically and ethically beyond any shadow of doubt, the only ones that will benefit from these tests are the manufacturing firms that sell them. Regarding the social consequences of public health care, there exists a need to raise important ethical questions (Hirvonen 1999).

The development of these genetic tests demonstrates the predominant tendency to overemphasize genetic structural aberrations, rather than considering the bioenergetic conditions for expression of enzymatic proteins, studying exposition risks, and managing individual dispositions by gentle methods of prevention.

The entirety of all available experimental, clinical and epidemiological data make it necessary to consider in clinical practice more acting-directing[?] principles of diagnosis, prophylaxis and therapy of systemic diseases. Because of the relative straightforwardness of the cause-and-effect ratio between exposition and disposition factors, Pre-AIDS and AIDS present a good model to describe the overregulation and counterregulation of the symbiotic cellular interactions in immune and non-immune cells and their consequential systemic processes.

Should a patient find himself stigmatized as “HIV positive” by the result of the so-called “HIV test”, there is no cause for panic. Any prognosis of death is merely an expression of limited medical knowledge, and not justified by biological facts. The alleged incubation period between so-called HIV seroconversion and manifest symptoms averages 12 – 15 years. In the USA, where patients are treated aggressively and early with prooxidative chemotherapeutics (“hit hard, hit early”), about 5% of patients stigmatized as HIV-positive become ill. Consequently, it would take 20 years for most so-called HIV-positives to actually fall ill. However, illness incidence in fact depends on the persistence of primary exposition risks, on secondary exposition risk through aggressive therapy schemes, and the absence of systematic compensatory and regulatory therapy, if even necessary at all. Rather than merely concluding the patient belongs to a risk group, first and foremost a meticulous anamnesis (case history) should be called for.

A predisposition to allergies, atopic skin diseases, asthma etc. can be important indicators for a patient’s disposition for Type-2 cytokine reactions and increased antibody production. Among other indicators, the childhood absence of typical bacterial diseases can also be a sign for a Type-2 disposition.

More than 70 symptomatic conditions can result in a positive “HIV test” result. Hence even HIV/AIDS medical treatment categorizes 5% of all so-called positive HIV tests as *a priori* insignificant diagnostic findings. Therefore, medical care cannot and must not be guided by

the positive result of any so-called HIV test, unless an actual immune deficiency virus “HIV” has been [definitively] proven.

Taken by itself, the number of differentiated cells measured within the immune cell network and the immunoglobulin classes, cannot be considered a reliable indicator for the actual existence of an immune cell deficiency in symptom-free patients. Within any healthy population, there will always be 5% that will report bloodstream CD4 cell values below 500/ μ L.

For patients who have tested HIV positive, the CD4 count is currently interpreted by HIV/AIDS dogma as a reason for a chemotherapy and antibiotic intervention. In healthy individuals, the CD4 can drop even below 200/ μ L without any serious loss of immune function. Without seriously limiting their functionality, the number of helper T immune cells in the blood stream depends on multiple influences. [??]
Valid information can be obtained through a DTH antigen recall test (delayed-type hypersensitivity skin reaction).

As a functional assay for Type-1 cytokines (which activate a cytostatic NO-gas defense against the intracellular pathogens following antigenic stimulation), a strong DTH test reaction is considered to be a reliable indicator (Christou 1986, 1995, Mosmann 1989, Hässig 1998b). The actual danger of intracellular opportunistic infections is not made clear simply from a positive result of a so-called HIV test.

A weak or anergic (ineffective) DTH skin test reaction, indicates the probability of a prevalent shift to the Type-2 cytokine state and an increased risk for opportunistic infections. Equally essential is the quantification of reduced glutathione in the plasma; both in the lung mucosa and inside peripheral CD4 lymphocytes (for the laboratory method: Buhl 1989, Herzenberg 1997, Nutrall 1998).

Also important is the measurement of plasma cysteine levels. Major deviations from the standard value of non-protein thiol must be treated, even in symptom-free patients. As a rule, the organism's need for thiol tends to be grossly underestimated. After the predominant scenarios in the “thiol-ester-iron world” one of the essential conditions for the origin of life in the prebiotic world (before the creation of cellular organisms), was the capacity of sulfur to generate bonds and exchanges between protons of the S-H groups via “weak interactions” (De Duve 1991).

Seawater is naturally high in sulfur, but terrestrial life forms face a consistent danger of latent deficiency of non-protein thiols and sulfates. Both are indispensable, being responsible for the regulation of the redox environment, the functioning of the cell symbiosis in immune and non-immune cells, and innumerable biosyntheses and biochemical reactions (Wrong 1993, Hässig 1999). The pathognomic symptom of cellular immune deficiency (AIDS) and other systemic diseases is lack of cysteine and glutathione. (Herzenberg 1997, Dröge 1997 b, Peterson 1998, Hässig 1998 d, Kremer 1999).

In both symptomatic and asymptomatic patients, lack of thiol must be strictly compensated in individually adjusted doses. For an intact cell symbiosis, the “semiconductor barriers” of redox-sensitive gene expression must be modulated in a sustainable and enduring manner by the redox potential, which for retuning enzyme activities depends on the glutathione system. Since glutathione synthesis depends on enzymes which are created under normal redox conditions, for 2 to 4 weeks at the beginning of the biological-compensatory therapy at least 5 grams of glutathione and 10 to 30 grams of N-acetyl-cysteine must be orally administered per day simultaneously.

As a barrier against opportunistic pathogens, the glutathione concentration of the mucous membranes tends to be much higher than in the blood plasma (e.g., the lung membrane fluid

contains about 150 to 200 micromoles of glutathione, while the blood plasma less than 5 micromoles). An important cofactor for cellular immune deficiency against the *Pneumocystis carinii* fungi (causing the most common AIDS-indicating disease, PCP) is a lack of glutathione in the secretory layer of the lung. In the special case of nutritional malabsorption caused by infectious and non-infectious alterations of the intestinal mucosa, doses of glutathione and N-acetyl-cysteine, correspondingly reduced, can be administered intravenously.

After the compensation of the intracellular and the plasma levels in the thiol pool and the glutathione concentration in the lung and the intestinal mucous membrane liquids, cysteine treatment should be continued for another six months with a daily dosage of 5 to 10 grams of N-acetyl-cysteine. Cysteine and methionine are both converted in the liver to cysteine. They can be taken in addition to daily nutrition. It is found in low-fat cottage cheese and other traditional dairy products (Bounous 1993). Due to the deficit of convertible protons, thiol deficiency causes a glutamine decrease with exaggerated protein reduction in the skeletal muscles (loss of body weight, wasting syndrome). The oral intake of high-dosage glutamine, up to 40 grams/day, can retune the synergistic effect of glutamine and cysteine levels on the maturation of T-helper cells (Shabert 1999).

Simultaneously, this effect improves the regeneration of intestinal and lung mucosa, energetic metabolism of the cellular symbioses, and systemic pH balance. Glutamine facilitates liver detoxification via the glutathione system; it slows down urea production due to decrease in the arginine splitting in urea and ornithine. If in case of a clear arginine deficit and the resulting lack of NO gas production (as part of pre-AIDS and AIDS), the thiol and glutamine balance is complimented by doses of up to 30 grams of arginine per day and up to 2% of the caloric intake respectively. By these means, the functionality of the cellular immune network (T4 helper cells, natural killer cells, neutrophilic granulocytes) can be significantly improved (Barbul 1990, Bower 1990).

In cases of massive immune cell deficiency, forced [an]aerobic glycolysis, malignant cell transformation and cell degeneration, as well as a pronounced wasting syndrome, adjustment of dysregulation of the amino acids cysteine, glutamine and arginine, can be achieved by using small intestinal probes [suppositories?] or, if necessary, by parenteral infusion. In critical cases of disease, glutathione can be administered intravenously [brand-name Tationil]

High-dosage compensation for thiol deficiency and amino acid dysregulation must be seen as the foundational therapy of the redox environment and of detoxification ability. They will provide the organism with natural and much-needed survival resources, to facilitate its self-regulation. Therapeutic success must be continuously monitored by laboratory testing adapted to individual requirements, as the N-acetyl-cysteine lifts glutamine and arginine levels in the plasma (Dröge 1997 a).

In the case of pre-AIDS or AIDS, a rigorously administered compensatory therapy during a well-monitored treatment phase produces better and more cost effective results, when compared to the counterproductive prescription of chemotherapeutic agents (AZT, HAART, cocktail therapy, etc) and a lifetime prophylaxis with antibiotics (Septra, etc). Chemotherapeutic agents may bring short-term results, but also will aggravate the system. Should a chemo-antibiotic (Seprin etc.) be administered for a short time because of acute opportunistic infections, it is just as critical to administer a consequently metered compensation of the thiol deficiency.

This important compensatory therapy can be effectively supported by a set of specific regulatory means during the symptom-free phase of the acquired immune deficiency, as well as in the phase of systemic secondary diseases. Hepatitis is often evident in members of pre-AIDS and AIDS risk groups, i.e. promiscuous homosexuals, I.V. drug users and recipients of highly contaminated stored blood (Hässig 1996 b, 1998 e). This calls for

additional liver protection to bring relief to the glutathione system and phase-II detox enzymes (Wilkinson 1997).

In contrast to the phase-I enzymes, which generate reactive electrophiles (electron generating substances) and activate carcinogens, phase-II enzymes inhibit electrophilic bonds and turn them into water-soluble excretable substances. 'Oltipraz', is a synthetic agent that has proven highly effective in practice. It was originally designed as an antihelminthic against schistosomes, which trigger Type-2 cytokine dominance (Lucey 1996) analogous to the early stadia of AIDS. 'Oltipraz' is a sulphur-containing dithiolthione. In general, it activates the enzyme family of glutathione-S-transferases. It exerts a protective function in the liver and in many other cell systems, especially the intestinal mucous membrane. Besides the protective effect against opportunistic germs and endoparasites, this drug has demonstrated antiviral and anticarcinogenic effects (overview in: Wilkinson 1997). These findings are significant, especially after prior prooxidative damage to the mitochondrial cell symbiosis caused by AZT and Septra. Oltipraz is equally efficient in the activation of the detoxification enzymes in the T-helper cells (Gupta 1995). Because Oltipraz causes sustainable triggering of the phase-II detoxication enzymes with low side effects, an adequate dosage for this is 125 to 250 milligrams/m². [??]

Among the natural substances, sulfurous isothione-cyanates provide an effective protection by triggering a variety of phase-II detox enzymes (overview: Hecht 1995). These thiocyanates are inherent in vegetables like garlic, onions, broccoli and other cabbage species. The other important family of natural liver-protecting agents are polyphenols. Animal organisms are not able to synthesize aromatic benzene rings, and like vitamins must therefore obtain them via ingestion plants and algae (Hässig 1997 c).

The redox-cycling between the glutathione system and the polyphenolic substances is crucial for the balancing of the redox environment and the detoxification performance by polyphenols, as well as the triggering of the phase-II detoxification enzymes and the inhibition of the phase-I enzymes respectively.

Essentially, polyphenols assist enzymes by cooperating with reduced and oxidized glutathione, glutathione-peroxidase, glutathione-reductase, glutathione-S-transferases, katalase, NAD(P)H-quinone-oxidase, and they inhibit the enzymes of the cytochrome P450 family (overview: Wilkinson 1997). Polyphenolic antioxidative protection of the cellular symbioses in the liver, and other cell systems including the immune cells, is of particular importance in the highly acute AIDS state, if, due to the failure of the cytotoxic NO-gas blocking of the Th1 helper cells, intracellular opportunists can proliferate without inhibition.

In this precarious situation, Type-2 cytokine production is amplified on one hand, but on the other hand the non-specific immune reaction of the phagocytes (macrophages) and the microglial cells in the brain are hyperactivated by the modulation of pro-inflammatory cytokines (Interleukin-12, Interleukin-1, Tumour Necrosis Factor-alpha and others, inflammatory mediators, and nitrosative and oxidative radicals).

The elevated quantity of neopterin (as a product of folic acid metabolism) and the Beta-2 protein in the circulating blood are indirect markers for an hyperactivation of the pro-inflammatory cytokine activity of the nonspecific immune cells with a simultaneous suppression of the cytotoxic NO-gas production of the specific immune cells (full-blown AIDS clinical pattern) (Mauri 1990, Odeh 1990, Fuchs 1990, Harison 1990, Matsuyama 1991, Krwon 1991, Hässig 1993, Valdez 1997). Consequently, well-balanced cell protecting backlash fails and the simultaneous cytotoxic overregulations (prevalence of Interleukin-12 compared to the Type-2 cytokine Interleukin-10) incapacitate the feedback functions. In the case of a thiol deficiency and a too high consumption of other antioxidants, the redox-compensation collapses in the competing cytokine chaos (Cossarizza 1995).

The clinical studies concerning polyphenols during recent years have mainly been concentrating on ellagic acid [pomegranates?]. Polyphenols are contained in green and white tea, curcuma (turmeric), silymarin (milk thistle) and others (overview: Stonder 1995, Conney 1997, Wilkinson 1997, Zhao 1999, Plummer 1999). Another possibility is the galenic combination of glutathione with polyphenolic anthocyanins (Reconstat, Ohlenschläger 1994) and with the ginkgo biloba polyphenol (S-acetyl-glutathione, SAG) respectively. In the case of chronic hepatitis B [and C], liver protection by the phytotherapeutic polyphenolic complex 'Padma 28', produced in Switzerland from a traditional recipe of Tibetan medicine (PADMA AG, Schwerzenbach), containing 20 herbal flavonoids and tannins, has proven reliable (Brzosko 1992, Liang 1992, Hässig 1997).

By providing a supply of glucuronic acid, liver cell symbiosis can additionally be reinforced; it plays an equally important role as a phase-II regulator of prooxidative and carcinogen-activating foreign agents in the liver, and acts to transform toxins into [urine] excretable substances. Kombucha, the organic product of tea fermentation from ancient China containing a symbiotic yeast/bacterial hybrid, is a natural source of glucuronic acid; beside the high concentration of glucuronic acid, it also contains B-vitamins and antibiotic substances. Kombucha can be made at home from black tea and sugar, once a culture is obtained (Frank 1992).

The progression of the prostaglandin synthesis under the influence of Type-2 cytokine dominance is in the case of pre-AIDS or AIDS, a characteristic part of the Type-2 counterregulation. This progression can be countermodulated therapeutically as well and preventatively. Like the Type-2 cytokines, elevated quantities of PGE₂ inhibit the synthesis of the cytotoxic NO gas and thus enhance opportunistic infections. The prostaglandins are products of the arachidonic acid, an [omega-6] essential fatty acid. Arachidonic acid is enzymatically metabolized into prostaglandin within the cell plasma by the enzyme cyclooxygenase (COX).

In the case of AIDS, cancer and other systemic diseases, the COX-2 isoform appears to be elevated. COX-2 increases the PGE-2 production. It also raises the production of the Type-2 cytokine Interleukin-6, which can trigger wasting syndrome (Hack 1996). A typical symptom for all systemic diseases like AIDS and cancer, wasting syndrome can be influenced by the selective inhibition of the COX-2 (O'Hara 1998).

PGE-2 is enzymatically generated by COX-2. In the same sense as growth factor TGB-Beta, it activates the generating of polyamides from the arginine derivative, ornithine. Therefore, the blockade of COX-2 by medication also inhibits tumour growth, reduces wasting syndrome, and improves the Th1-Th2 balance of the cellular immunity (Subbaramiaiah 1997, Huang 1998, Jones 1999, Lipsky 1999a, 1999b, Sawaoka 1999, Golden 1999, Masferrer 2000, Kune 2000, Prescott 2000, Reddy 2000, Hiashi 2000, Stolina 2000).

In the case of symptom-free patients with a weak or anergic population of Th1 immune cells, prostaglandin modulation via essential fatty acids is a better option for treatment.

In animal testing, Th1 helper cells population reactivity in the DTH skin reaction test was inhibited when 15% of the intake of calories consisted of linolenic acid [omega-6 vegetable oils], but not with the same quantity of fish oil with its high content of omega-3 fatty acids (Alexander 1990).

As cold-water fish can supply their needs of essential fatty acids by eating sea microalgae, patients can cover their requirement of essential fatty acids for prostaglandin modulation by the nutritional intake of contamination-free microalgae in powder or in tablet form (e.g. *Chlorella vulgaris*; manufactured by Yaeyama and Jarrow).

It is indeed necessary to ingest a couple of grams per day for several weeks in order to stimulate immune cell reaction and inhibiting tumour creation. The effect of the mitochondrial cell symbiosis protection is improved by the simultaneous substitution with cysteine,

glutamine, arginine and RNA (Bower 1990, Cossarizza 1995, Chuntraskaul 1998, Gianotti 1999).

The low or high fluidity of the cell symbiosis' micro-Gaian environment, just like the fluidity of the cell membrane, reflects the type and composition of the polyunsaturated fatty acids (Bower 1990, Fernandes 1998, Simonopoulos 1999, Zelenuich-Jaquotte 2000).

The interaction between the synthesis of NO and other derivatives, and the prostaglandin PGE-2, which is synthesized from the [Omega-6] arachidonic essential fatty acid, is to a minor degree antagonistic (overview: Lindoln 1997, Minghetti 1998).

This interaction is of vital importance for the prevention and therapy of Type-2-counter-regulations of cell symbioses (systemic diseases) including the Type-1 to Type-2 cytokine shift (cellular Th1 immune deficiency, Pre-AIDS) combined with proinflammatory macrophage hyperactivation (opportunistic infections, AIDS full-blown clinical pattern). It is possible to counter-regulate massive regressions of the cell symbioses by type omega-3 multi-unsaturated fatty acids and its derivatives (Velerod 1997, Imoberdorfr 1997, Gogos 1998, Albert 1998, Ogilvie 1998, De Langeril 1998, Tashiro 1998, Rose 1999, Bougnoux 1999, Burns 1999, Bartsch 1999, Biasco 1999).

Using micronutrients (vitamins, minerals and oligo-elements) must be considered in a differentiated way when used as part of a biological-compensatory and regulation therapy for prevention of Pre-AIDS and AIDS, as well as other systemic diseases.

Currently, vitamin E in combination with vitamin C is the standard antioxidant treatment worldwide. In "The antioxidant supplement myth", Herbert critically analyzes this standard (Herbert 1994). He coherently demonstrates that its effects are disadvantageous; pharmacological dosages of a single polyphenol, for example vitamin E, in combination with vitamin C and beta-carotene, can have some potential benefit but may in many cases cause ill effects, depending on the recipient's iron state.

As redox compounds, they simultaneously demonstrate both prooxidative and antioxidative effects. Therefore the summary of their use: "[A micronutrient] supplement can help some consumers, harm others, but for most, they don't show any effect whatsoever."

It has been demonstrated that vitamin C (ascorbic acid), in the presence of redox-active transition metals like iron (Fe) or copper (Cu), can act as a prooxidant, and so by the so-called Fenton reaction aids in the development of highly reactive hydroxide radicals (Fenton 1894, Halliwell 1993, Cottier 1995).

The synthesis of hydrogen peroxide (H_2O_2) occurs through the slow, pH-dependent dismutation of superoxide radicals.

Note: As chelators of free metals, tannins can contribute useful services in this situation. Herbert's critical statement has been extensively affirmed by the studies of Kim et al., who could not find any life-extending effect by the use of isolated and unbalanced vitamins and mineral nutrition supplements. They identify the annual expenditure of \$3.3 billion for nutrition supplements, as a virtually useless burden on health care costs (Kim 1993). "In conclusion, we would like to state: a sufficient nutritive supply of a natural mixture of tannins and flavonoids is indispensable for a reliable and side-effect free antioxidative effect" (Hässig 1997c).

Vitamins E and C generate radical chain reactions as intermediate states, which must be compensated for by the glutathione system (Ohlenschläger 1994); therefore a case of thiol deficiency can be aggravated by high dosage supplementation of these vitamins. Micronutrient requirements, in the case of Pre-AIDS and AIDS, should be evaluated in the context of the precise regulation of a rigorous biological-compensatory and regulation therapy, because deficits of several micronutrients depend on the redox state, the mitochondrial activity, the cytokine balance, the presence of wasting syndrome, any given resorption dysfunction, severe diarrhoea, toxic and infectious stress-factors, alloantigen

overcharge, chemotherapeutic agents, chemoantibiotics, antiparasitics, antifungals, antivirals, excessive alcohol, recreational drug use, cigarette smoking and many other factors. Unmonitored self-medication does not make much sense, and can in some cases even be dangerous.

An overview study with ambulatory Pre-AIDS and AIDS patients in relatively good health, without clinically-defined wasting syndrome or heavy diarrhoea, measured levels of the following: Vitamin A and complete carotene, vitamins C, E, B₆, B₁₂, folic acid, thiamine, niacin, biotin, riboflavin, pantothenic acid, free and total choline, carnitine, biopterin, inositol, copper, zinc, selenium, magnesium and glutathione.

The results of the study confirm a reduction in the circulating concentrations of glutathione, and quite often lower serum concentration for magnesium, complete carotene and complete choline, along with increased niacin levels. The remaining values were in normal range and lower for a minority of the tested persons respectively, sometimes with self-medication by vitamin and mineral supplements (Skurnich 1996).

A general survey of state-of-the-art clinical research on HIV/AIDS medicine concerning several micronutrients as influencing factors for states of acquired immune deficiency (Pre-AIDS and AIDS), display also an individual dependency on deficiencies in a wholistic context of dysfunctional cell symbiosis.

“Singular micronutrient deficiencies are known for their disadvantageous influence on the immune system by lowering cellular and humoral immunity and damaging the phagocytosis” (Beisel 1982, Klurfeld 1993). PWAs can react particularly sensitively to nutrition based deficiency states, which damage the already suppressed immune functions. In an earlier study with people infected by HIV we found that carotene and ascorbic acid (vitamin C) have been lowered in 27% and vitamins E and A in 12% of the patients” (Bogden 1990).

“The serum levels of micronutrients in HIV positive patients have been associated with immune function- and disease stage-markers (Fordyce-Baum 1990, Baum 1991, 1992, Semba 1993). The studies demonstrate that an abnormal nutritional status at once, goes along with and precedes the progress of the HIV disease (Semba 1993, Coodley 1993, Tang 1993, Abrams 1993). These studies evaluated the nutrition intake or the serum concentration of one or of a few micronutrients in selected patient-cohorts” (Skurnick 1996). The relevance of the primary influence of micronutrients on the prevention and therapy of cancer has been qualified in relative terms compared to the relevance of the redox status, NO- and prostaglandin syntheses, the cytokine balance and cell symbiosis activity (World Cancer Research Fund 1997).

Additionally, measuring of the serum ferritin level must be considered essential; in the case of pre-AIDS or AIDS, as in all proinflammatory stages of macrophage overactivation, ferritin elevation is evident (Gupta 1986) and plays an important role in all Type-2 counterreactions (Gherardi 1991, Weinberg 1992, Herbert 1992, Gelman 1992, Lacroix 1992, Kiefer 1993). Apart from the compensatory therapy of the redox state (Pippard 1989, Hässig 1993), the reinforcement of basic tissue has an important function, in view of the regulation of balancing iron.

The extracellular base-tissue, in which all tissues and organs are embedded, functions as filter for the cell symbioses' entire bioenergetic, substantial, hormonal and sensory input and output.

Among other, base-tissue is composed of a complex network of high-sulfate protein molecules (glycosamine glycanes, proteoglycanes), which make up the negative redox potential. Re-fetalization of the extracellular matrix of the base-tissue into sulphate free hyaline acid, as present in early embryonic tissue, is characteristic for many carcinomas (Heine 1997).

For prevention and therapy, the extracellular matrix can be reinforced with a regular supply of polyanions: chondroitin sulfate in the form of [bovine] cartilage preparation, shark cartilage,

micro-algae (agar) or macro-algae (seaweed). (Hässig 1992). The redox potential equalization of the base-tissue is synergistically supporting the glutathione system and discharges cell symbioses in prooxidative and systemic stress stages (Hässig 1992, 1997a, 1998b) [??]. Direct activation of the mitochondrial cell symbioses can be stimulated by Coenzyme Q10 (Folkers 1986) and L-carnitine (Bremer 1990).

Coenzyme Q10 plays an important role in the electron transfer in the mitochondrial respiration chain. In symptom free so-called HIV positives, a CoQ-10 deficit is already traceable and will be progressively so in aggravating pre-AIDS and AIDS.

Toxic stress factors and pro-oxidative medication (AZT, Septrin, etc.) are decisive factors in leading to mitochondrial respiration chain dysfunction and secondary defects in the mitochondrial DNA. CoQ-10 improves the cell symbioses performance in immune cells and non-immune cells, and can be administered in a daily dosage of 200 milligrams for a several months without any noticeable side-effects (overview: Folkers 1988).

L-carnitine is involved in the transport of long-chain fatty acids (triglycerides) inside the mitochondria for oxidation. L-carnitine deficiency increases the glucose metabolism and facilitates a switch to [an]aerobic glycolysis (Warburg phenomenon). The disturbance in the triglyceride transport causes lipid accumulation, as often seen under the treatment with HAART and protease inhibitors (Brinkmann 1999). Pre-AIDS and AIDS can be viewed as systemic dysfunctions in lipid metabolism and of the lipid composition of the T-lymphocytes, in context with an L-carnitine deficiency (De Simone 1991). Administration of high dosage L-carnitine (6 grams per day for two weeks) has improved T-helper cell proliferation, lowered the triglyceride serum level, and decreased the circulating Beta2-microglobulin and the Tumour Necrosis Factor-alpha serum values as indicators for hyperactive macrophages in patients with AIDS, and patients tested HIV positive. It also seems that L-carnitine stabilizes the cytokine balance by ameliorating the mitochondrial performance (overview: De Simone 1993).

Decreased mitochondrial performance as a result of long-term chemotherapy, i.e. damaged mitochondrial DNA after the intake of AZT etc. and Septrin etc., can additionally be compensated for by a daily dose of 600mg lipoic acid (alpha-lipoic acid) plus 300mg thiamine (vitamin B₁) for a month or longer.

Systematic [systemic?] mitochondrial activation is of particular significance to so-called "HIV positive" patients, but equally as well for cancer patients threatened with multiple organ failure (myocardial infarction, sepsis, cerebral infarction, hepatic coma, myopathy). The cytokine balance and resulting the equilibrium between cell-mediated and antibody supported immunity interacts, like all organ systems, with the sensory and hormonally controlled stress system.

The retroactive hormonal stress axis between the hypothalamus, the pituitary, and the adrenal cortex modulates the cytokine profiles via equilibrium between cortisol and dehydroepiandrosterone (DHEA), both produced by the adrenal cortex. The final cortisol synthesis takes place in the mitochondrial cell-symbionts of the adrenal cortex cells (Tyler 1992). Consequently, disturbance and damage to these cells can favour grave psychosomatic stress diseases and systemic diseases like AIDS, cancer and many other symptoms. During states of high stress, the synthesis and the release of cortisol increases relative to the DHEA level. This causes an inhibition of cytokine synthesis via the interaction of cortisol with transcription factors (Brattsand 1996).

Persistent cortisol elevation facilitates the antibody-supported immune response, and weakens the cellular immune reaction. A reluctance in the Type-1 cytokine pattern, under strong stress stimulation of the macrophages through antigens and toxins, the release of nitrogen- and oxygen-radicals, and of the inflammation mediators Interleukin-1 and Tumour Necrosis Factor-alpha can be increased within said macrophages. As immediate scope for

quantification of macrophage-activation through inflammation, neopterin and ferritin and indirect measurements (for example the C-reactive protein) can serve well as indicators of acute phase reactions, i.e. C-reactive proteins (Hennebold 1994, Hässig 1997d, 1998b). [??]

If a cortisol/DHEA ratio can shift the cytokine pattern from Type-1 (Th1) to Type-2 (Th2) dominance, it also suggests the reverse: a moderation of stress-induced hypercortisolism will amplify the effect of DHEA towards Type-1 cytokine synthesis. Therefore, augmenting the cortisol/DHEA ratio in favour of DHEA can improve cellular immunity by activating the Type-2 cytokine Interleukin-2. There is indeed a direct correlation between the CD4 cell count, the cortisol level, and the level of DHEA sulfate (DHEA-S being predominantly synthesized form). The occurrence of acquired cellular immune failure syndrome is associated with an increasing deficit of DHEA-S (Biglierei 1988, Raffi 1991, Mulder 1992, Christeff 1996, Ferrando 1999). In people with AIDS, the 24-hour cortisol levels have been shown to be elevated (Vilette 1990). These findings resulted in the hypothesis that DHEA-S substitution (as a cortisol antagonist) could enhance cellular immunity for the prevention and therapy of opportunistic infections in the case of pronounced pre-AIDS and AIDS (Frissen 1990, Wisniewski 1993).

The DHEA-S level as counterbalance to the ACTH cortisol system is of vital importance, not only for cytokine controlled functions of the immune cells cell symbioses, but also for other cell systems (Parker 1985, Ebeling 1994, Lavallee 1996). DHEA as a precursor molecule for sex hormones, and DHEA-S dysregulation are both co-determining factors in the case of tumours in hormone dependent organs such as the mammary gland or the prostate as much as of tumours in other organs (Vermeulen 1986, Heinonen 1987, Barrett-Connor 1990, Stahl 1992, Le Bail 1998, Lissonie 1998, Svec 1998, Eaton 1999).

In many cases, moderation of hypercortisolism and direct Type-2 [?] cytokine stimulation, via DHEA-S, can be supported by nutritive measures. Among other things, the increase of the extracellular content of glycosaminoglycans (heparin, heparinoids) is useful. They reduce the influx of calcium ions into the inner cell, and inhibit the binding of cortisol on the intracellular receptors. This can be achieved by ingestion of cartilage extracts (chondroitin sulfate) or agar from sea algae (Hässig 1993, 1998b).

Simultaneously, the proinflammatory hyperactivation of macrophages in Type-1 to Type-2 cytokine shifts can be repressed by binding surplus NO and O₂ radicals, through intercepting excessive free iron and an increase in catabolic proteases generation by using phytotherapeutic complexes such as Padma 28, a Tibetan combination preparation made up of polyphenolic flavonoids and tannins (Liang 1992, Hässig 1993, Gebbers 1995). Cortisol moderation and the reactivation of DHEA-S in interaction with “proinflammatory macrophage stimulation inhibition” is important, because in addition macrophages through their phagocytosis performance capacity represent a preferred reservoir for intracellular opportunistic pathogens (Rubin 1988, Meltzer 1992).

Sooner or later, the counterregulation against strong and/or long-lasting nitrosative, pro-oxidative and systemic stress—with the consequence of an elevated cortisol/DHEA-S ratio, failure of cellular immunity and of the cytotoxic NO gas defence through a Type-2 cytokine shift and the pro-inflammatory mobilization of opportunistic residents in the macrophages (fungi like pneumocystes, candida, histoplasma, cryptococci, parasites and toxoplasma; bacteria like mycobacteria, listeria, legionella and chlamydia; and many actually existing viruses, in contrast to the so-called HIVs)—must lead to clinical full-blown AIDS. If the primary stress factors cannot be minimized, the supply and demand of available protons remains imbalanced, and the dysregulation of the cell symbiosis will be further aggravated by the use of chemotactical weapons.

In full-blown AIDS, this results in a crucial antagonism inside the nonspecific immune response of the CD4 cells: the cortisol brake for the biosynthesis of Tumor Necrosis Factor

is disregarded[?] in the macrophages by the activation of Interferon-gamma under strong or/and long-lasting stress stimulation (Luedke 1990) and the cortisol/DHEA-S ratio thus skews in favor of DHEA-S, due to the anti-inflammatory influence of cortisol. After receiving signals from the antigen-presenting dendritic cells, the T4 helper cells, now poor in glutathione, predominantly synthesize Type-2 cytokines (Peterson 1998).

The CD4 cells then inhibit the cytotoxic NO gas synthesis, contrary to the macrophages (loss of helper Th1 cell functions) and stimulate instead the antibody production (overview: Mosmann 1996, Lucey 1996, Abbas 1996, Hässig 1996d, Lincoln 1997). This results in the increase of the cortisol/DHEA-S ratio on the cost of DHEA-S (Wisniewski 1993, Christeff 1996, Ferrando 1999).

From this antagonism of unspecific inflammatory events combined with the mobilization of opportunistic pathogens on one hand and the loss of the specific Th1 gas production against intracellular opportunists on the other hand results the contradictory clinical symptoms of manifest AIDS.

The prooxidative, glutathione consuming and mitochondria-toxic chemotherapy with AZT etc. and the continuous prophylaxis with Septrin etc. are not able to control the chaos of competing cytokines resulting from nonspecific immune hyperactivation within the macrophages (Type-1 overregulation: among others, the Type-2 cytokine Interleukin-10 antagonistic against Interleukin-12; in the case of wasting syndrome: Type-2 cytokine increased; in the case of tumour cells: TGF-beta increased: NO and O₂ production inhibited).

The most efficient option is to compensate for the thiol deficiency, empowering cysteine to slow down the toxic cell effects of the tumour necrosis factor within the hyperactivated macrophages, and improving glutathione biosynthesis (Cossarizza 1995).

The preventive and therapeutic aim must be to balance the redox environment, to restore fluidity to the micro-Gaian milieu, to reconstruct the cytokine balance, and to reduce the concurrence between the Type-1 overregulation of the nonspecific immune reaction and simultaneously moderate the Type-2 counterregulation of specific immunity via a synergistic therapy of compensation and regulation.

Last but not least, "it is essential to decidedly confront the popular and official doctrine that every HIV-infected person must sooner or later enter full-blown AIDS and thus inevitably die (Hässig 1992b). To the contrary, we should give "HIV-infected" persons hope that they may be spared from succumbing to AIDS, if they learn to adapt their lifestyle to the possibilities and limits given by nature. For this, dealing with nutritional problems seems to be most adequate. In our overview work, "Umdenken bei AIDS [*Rethinking AIDS*]", published about one year ago, we ask if this could lead to a paradigm shift in medicine (Hässig 1992b).

Nowadays, we tend to assume, such a shift will happen. The use of AZT and analogous antiviral medications as recommended by the responsible authorities, is based on the antibiotics paradigm, which aims for the toxicological extinction of microbial inflammation germs. By the same token, man lives in ongoing symbioses with a whole slew of microorganisms, hence the question is justifiable if it wouldn't be more sensible to support the organism's probiotic, physiological mechanisms of self-healing.

The variety of the effective and non-toxic intervention options demonstrates a possible change within medical practice "from antibiosis to symbiosis". Therefore, it is the primary duty of medical doctors to reduce the paralyzing and destructive fear of death, and instead encourage people afflicted with systemic cell dysbioses, reinforcing their natural will to survive via a clarification of the actual standard of knowledge.

The most efficient protection against the abuse of a "violent medicine" (Albonico 1997) as a modern instrument of terror and fear, is the rational knowledge that any kind of risk for, and

any targeted attack on the cell symbioses of immune cells and non-immune cells will find a constitutional evolutionary biological response.

The imagined “HIV retrovirus” would not be any exception, even if it really existed. The clinical symptoms of pre-AIDS and AIDS could, if a biologically active “HIV causative agent” actually was the cause of the disease, also ultimately result from the disturbance of the redox balance, damage to cellular symbiosis, and the [subsequent] shifting within the micro-Gaian environment. The preventive and therapeutic treatment for the inactivation of such an “HIV retrovirus” (not proved in biological reality) would be principally the same as for all other prooxidative exposure factors.

These fundamental therapy concepts are universally valid, irrespective of their exact nature, be they toxic, pharmatoxic, traumatic, inflammatory, infectious, nutritive, radioactive, alloantigenic, psychic, or any assortment of such. People with a strong redox-sensitive disposition must be treated in every case with the same manner to avoid exposition risks and orient their nutrition according to their blood group as a cipher for genetically disposed polymorphism of the enzyme systems (D’Amato 2000).

The vital importance of a synergy between a conscious compensatory and regulatory therapy is deduced from the logic of natural laws pertaining to the co-evolution between microbes and mankind, the processing of toxins and other bioactive stress factors, including the consequences of an underfeeding or malnutrition. A profound change of the scientific attitude towards healing, points from antibiosis (greek: *anti* for against, *bios* for life) to symbiosis (greek: *sym* for with, together). The inevitable end of the lethal hunt for the virus, and of the one-sidedly aggressive [slash and burn] cancer expunging, at the same time for all concerned—for medical therapists as well as the general population—a self-critical act of relief from the staging of collective terrorism and the exploitation of fear.

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