Heinrich Kremer MD: The Silent Revolution in Cancer and AIDS Medicine

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:

The lifesaving knowledge on healing

Disposition factors can be epidemiologically justified. They also explain why the pathogenetic distribution patterns manifest in a Gaussian distribution curve, equally for excessive expositions as in collective populations and differing risk groups. While the majority of the exposed individuals will maintain a variable redox balance with flexible cytokine patterns, a minor percentage will develop a distinct Type-I overreaction or a distinct Type-II counterreaction. To presume a fatal mass-infection transmissible to everybody, ending in inevitable death was *a priori* a medical construction beyond a biological-evolutionary reality. In the case of the "HIV-induced" AIDS, it seemed 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, so opportunistic infections could develop. Once patients were treated with AZT chemotherapy massive bacterial infections happened as a consequence of maturation inhibition of bone marrow cells (Rosenthal 1994), (Marco 1998, Cox 1998). Because the evolutionary and biologically programmed coaction of exposition and disposition factors has not been sufficiently understood, HIV/AIDS medicine provoked the appearance of combined acquired cellular and humoral immune deficiency SCID (severe combined immunodeficiency) by prescribing chemotherapeutics on the basis of the objectively incorrect theory that „HIV causes AIDS". The clinical and epidemiologic results demonstrate the deficits of modern medicine with abundant clarity, resulting from underestimating toxic and pharmacotoxic stressors and of the one-sided fixation on outdated 19th century infection theories with sophisticated 20th century biotechnological methods.

The HIV and AIDS establishment's massive projection of a supposed pandemia (mortal infection spreading on whole populations) reveals that even in the case of doubtless increased collective charging with immune stressors (endemic multi-infectiosity, contaminated drinking water, undernourishment, adverse living conditions et al.), special disposition factors must supervene when triggering acquired immunodeficiency.

Professor Duesberg, retrovirus cancer scientist and molecular biologist at Berkeley, California, one of the most severe critics of the "HIV causes AIDS" theory, considers the "HI virus" to be a "passenger virus". He regards toxic reasons like excessive consumption of illegal drugs, nitrite inhalation as sexual doping agent, medical chemotherapy and also the injection of highly contaminated clotting protein factors against hemophilia as the real causes of AIDS in western countries (Duesberg 1996). On the occasion of the Pretoria specialists' conference, summoned by President Mbeki before the XIII World AIDS Conference in July 2000 (see chapter XII), 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 a, Duesberg 1992 b, 1996, 1998, Mullis, 1996, 1998). But this technology is now faithfully but inappropriately used by thousands of AIDS virus researchers and activists to detect latent, i.e. biochemically and biologically inactive HIV or even just antibodies against it (Duesberg 1996 a)! 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, leukemia, liver cancer, and rare neurological diseases - without ever producing any public health benefits (Duesberg 1992 a)… 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 1980s" (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 and 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 to the current "Anonymous AIDS Notification" forms of the South African Department of Health - no HIV tests are required for an AIDS diagnosis (Widy-Wirski et al., 1988; Fiala, 1998). In addition the WHO promotes the impression of a microbial 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 an ever growing, almost exponential epidemic, even if the annual incidence declines (Fiala, 1998). It would follow that the estimated increases in African HIV antibody (!)-positives do not correlate with decreases in any African population. On the contrary, they correlate with unprecedented simultaneous increases in the country's popu-lations - hardly the "catastrophe" imagined by the Washington Post and propagated by the WHO and the American AIDS establishment. But this deceptive AIDS propaganda biases a scientific analysis of African AIDS by all those who are not aware of the facts" (Duesberg 2000).

In other words, the actual figures recorded in the WHO epidemiological reports on total morbidity and death rates in African states are hardly higher than in western countries. 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 a "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 "anti HIV antibody test" (Duesberg 2000). Based on the not very reliable acquisition of pathogen data, compared to western countries and because of the small fund of medical research results in developing countries, it is much less obvious to draw conclusions on the exposition and disposition interaction for morbidity and mortality in causal connection with systemic diseases of Type-II cell dyssymbiosis. However the population explosion data in African countries shows comparability with demographic processes in western countries 150 years ago. With gradual improvement in living conditions and medical and social standards, infectious disease rates will decline and toxic stress will increase. Increased collective charge, irrespective of gender, with variegated immunostressors with simultaneously "surprising" (Duesberg 2000) low AIDS incidence (WHO Weekly Epidemiological Records since 1991) when compared to western countries and the parallel population explosion in Africa give reason to presume that disposition factors must play a role. The consequences for developing countries as for western countries are the same: protection from the abuse of "American biotechnology" (Duesberg 2000) and the "blessings" of western chemotherapy and chemoantibiotics while promoting knowledge on evolutionary biologically programmed redox protection.

Disposition factors act via the peroxidation control system (creation of hydrogen peroxides, H_2O_2 and lipid oxides) and nitrosylation of transcription proteins (bonding NO and NO derivatives with hydrogen sulphide groups of proteins containing cysteine, RSNO). In the case of too high glutathione consumption this control system initially raises, as a sensor, the activity of antioxidative genes and the metabolization of H_2O_2, lipidperoxidation and RSNO (Hausladen 1996). After depletion of the neosynthesis of glutathione and other antioxidative enzymes (catalase, superoxide dismutase, selenium dependent glutathione peroxidase, glutathione transferases, NADPH dependent glutathione reductase), the hypoxic/pseudohypoxic emergency routine is switched on. From an evolutionary biological point of view, the early and sustained switch of the cytokine balance to humoral and antibody-supported immune response was advantageous, because the bacterial threat predominant in the course of the evolution could be efficiently averted. Bacteria proliferate faster than opportunistic pathogens. They can be inhibited and destroyed efficiently through the defense mechanisms of non cell-related humoral immunity, complement formation, opsonization (coating of bacteria membranes by special target molecules for antibodies) and by the antibodies themselves, which are formed by B-lymphocytes maturated in the bone marrow. The larger fungal and parasitic pathogens, equipped with mitochondria, and mycobacteria with a special cell membrane are stopped most efficiently by interaction between the non-specific and specific immune cell networks. If not inhibited in time by the gas attack, many parasites can invalidate the NO gas synthesis via special surface molecules (glyco-inositol phospholipids).

Multicellular (extracellular) parasites can emit specialized enzymes, which attack tissues (proteinases), and trigger a type-2 cytokine response (Th2 immune response) as a suitable

form of reaction, since combating worms, for example, would require too large quantities of NO gas which would in turn damage the body's own tissue cells. Metastatic cancer cells also utilize the biochemical supply of proteinases for pervading tissue and thereby deactivating the NO gas production in neighboring cells. Cancer cells are characterized by low NO gas synthesis (Ignarro 2000) and greatly affected by high NO gas levels (Xie 1996, Chinje 1997). All in all, a one-sided Th2 (cytokine 2) immune response is a disadvantageous disposition for blocking intracellular pathogens (fungi, parasites, mycobacteria, some virus species) and the inhibition of metastatic cancer cells (Zvibel 1993, Liew 1994, 1995 a, 1995b, Mosmann 1996, Abbas 1996, Lucey 1996, Xie 1996). Crucially, all depends on an appropriate and flexible combination of defense and regulation strategies.

On balance, the importance of the evolutionary advantage of a redox-sensitive sustained type-2 cytokine immune response has changed through advances in civilization and the developments in modern medicine, especially on introduction of vaccination programs and antibiotics over the last 50 years, while toxic and pharmacotoxic effects of the impact of civilization have gained in importance as a threat to cellular symbiosis.

People with a particularly redox-sensitive disposition are now at a disadvantage because they respond to toxic influences faster and with a more sustained Type-II counterregulation that would be better suited to the inhibition of extracellular bacteria or multicellular parasites.

Thus, the immune system chooses the evolutionary biologically programmed, but "wrong" strategy, because it is mislead by toxic stressors, which did not exist as part of natural evolution. This development is reflected in the steady rise of cancer and other systemic diseases over the last 100 years in the industrialized countries. Today, the main sources of toxic exposition which favor the development of cancer and other systemic diseases in the industrialized countries are: toxin residues in nutrition, in the environment and at working places, and the use of tobacco (Loeppky 1994, Walker 1998, Waite 1998, North 1998) as well as pharmacotoxic medication and toxic pharmaceutical decomposition products (Kalow 1993). The individually predisposed efficiency of redox-dependent detoxification capacity is the critical disease factor for the actual incidence of Type-II cell dyssymbiosis through toxic and pharmacotoxic nitrosation and peroxidation.

Indicative of an individual disposition for redox-efficiency and the detoxification capacity of the cellular symbiosis, patients with manifest systemic illness depend most urgently on replenishment of the cysteine/glutathione systems

Since Warburg made his historic declaration at the conference of Nobel laureates held in Lindau on Lake Constance in 1967 that there is no disease whose prime cause is better known than cancer (Warburg 1967) and an expanding field of research has been established dealing with the individual disposition for metabolic processes on the detoxification of medicaments in the human organism. This field of research was very quickly expanded to include the direct and indirect effects of toxic substances on cancer genesis (overview with Kalow 1993, Daly 1994). These studies concentrate, corresponding to the molecular genetic mainstream of cancer research, on the variability of genetic expression for biosynthesis of xenobiotic-metabolizing enzymes (genetic enzyme polymorphism):

"The paradigm for mechanism of action of chemical carcinogens has been well established in model cell culture and animal systems, and studies in humans appear to support the possibility

that most cancers are initiated by chemical/dietary exposures and proceed through various stages of preneoplastic lesions consisting of partially transformed cells to full metastatic cancers (Vogelstein 1993). In rodent models, the progression stage can be enhanced by treatment with tumor promoters, which themselves do not necessarily exhibit the properties of carcinogens (Hennings 1993). These chemicals are thought to mediate cell proliferations that fix the mutation in the genome. Another class of chemicals called nongenotoxic carcinogens has been described in rodent model systems (Jackson 1993, Barret 1995, Costa 1995). These agents are not metabolically activated to genotoxic derivatives but presumably alter cell-cycle control. Many nongenotoxic carcinogens are also tumor promoters. However, their mechanisms of action are not presently known.
It is widely held that humans differ in their susceptibilities to cancer. Certain individuals may be more susceptible, whereas others are more resistant to cancer. This may be due to a number of factors including health, nutritional status, and gender. From what is known about the mechanism of action of carcinogens, it is thought that genetic background could play a significant role. The responsible genes are probably those encoding the xenobiotic-metabolizing enzymes (XMEs) that activate or inactivate carcinogens (Gonzalez 1995, Nerbert 1996). Variable levels of expression of these enzymes could result in increased or decreased carcinogen activation. In fact, it is well established that genetic differences occur in expression of XMEs" (Hirvonen 1999).

In other words, genes are effectors in complex, self-organized networks, which under the influence of redox-dependent sensors encourage or discourage the biosynthesis of enzymes. If the enzyme systems are individually more strongly predisposed to activating carcinogens rather than deactivating them, then there are more demands on the antioxidative capacity. The thiol pool and other activities could become exhausted more rapidly from protecting the respiratory chain, the macromolecules and lipids. The result is that in the long-term the redox milieu changes and the dominant cytokine profiles switch, earlier and in a more sustained manner, to type-2 cytokines. The production of nitrogen and oxygen oxides are choked and the highly fluid micro- Gaia milieu transforms permanently to a less fluid milieu.

Dependent on time, re-fetalized tumor cells could form (Type-II counterregulation of cell dyssymbiosis). If in this situation prooxidative chemotherapeutics are in use the desired apoptosis/necrosis can be selectively forced in a part of the cells (Type-I overregulation of cell symbiosis), but by the same token this can also accelerate a fully developed transformation into metastatic cancer cells inside other cells. Basically, all phases of still to be compensated cellular dyssymbiosis, primarily in tumor tissues but also secondarily in differentiated tissues, can switch over unpredictably to a decompensated cellular dyssymbiosis phase. It is a characteristic of the principle of chemotherapeutic treatment that patients with a particularly redox-sensitive disposition, who become ill because of this genetic and supragenetic predisposed redox sensitivity, will not only have cellular dyssymbiosis in manifest tumor tissues but also in other tissue types and that tumor cells will respond to the targeted attack of chemotherapeutics in manifestly decompensated tissues in a diversity of ways in the various phases. Therefore, there can only be a conditional homogenous responsiveness of tumor cells to chemotherapeutics, and the results of therapy schemata cannot be sufficiently calculated individually. Consequently, for patients with a systemic disease, the distinctive genetic polymorphism of carcinogen-activating detoxification enzymes become manifest in the course of the disease. Redox-sensitive variability of the xenobiotic-metabolizing enzymes applies mainly to cytochrome P450-dependent and flavin-containing monoxygenases, epoxide hydrolases, glutathione transferases, N-acetyl transferases, NAD(P)H-ubiquinone oxidoreductases, myeloperoxidases etc. (overview with Wilkinson

1997, Hirvonen 1999). Such patients most urgently require a balancing of the thiol pool and redox state. Chemotherapeutical treatment and the consequent extreme prooxidative stress must inevitably have had a counterproductive effect on patients with systemic diseases because such therapy is normally effected without compensating the depletion of the thiol pool, the dysregulation of amino acids, the cysteine balance and without moderating the Type-II counterregulations. Chemotherapy triggers the desired destructive cell effects as well as individually non-calculable cell dyssymbiotic counterregulations, causing among other things systemic wasting syndrome.

Genetic tests of questionable value were developed to determine the individually predisposed variability of the isoforms of detoxification enzymes. In the USA, for example, prophylactic mastectomies were performed to avoid breast-cancer on the basis of such genetic tests. Such deterministic prognostics by means of genetic tests are to be viewed most critically for a number of reasons. If they have any significance at all, then at the most as an inducement for purposefully influencing the individual balance and regulation therapies in the interplay between exposition and disposition through nutritional measures.

The deterministic forecast of an individual's disposition based on genetic testing of detoxification-enzyme synthesis is to be criticized for many reasons; in reality, the expression of every single gene is redox-dependent, resting primarily on the relative condition of the bioenergetic redox system

"It is anticipated that rapid advances will be made in methodology to determine potential metabolic at-risk genotypes. These advances may include less invasive collection methods for test samples (e.g., buccal cell and urinary cell samples), automated DNA extraction combined with robotic sample handling, and high-density oligonucleotide array-based genetic test methods. At present, many research laboratories are conducting association studies and contradictory reports are emerging in specialist literature. Several sources of potential bias exist that partly account for these divergent findings, usually an initial small study showing a positive association. This raises the important issue of power calculations in planning subsequent studies. High profile reporting to the public of results of studies that may ultimately turn out to be erroneous is also problematic in this context. Also, there recently has been debate about publication bias-selective publishing of only positive associations. If the potential biases mentioned above are carefully controlled, genetic screening studies may in the near future help us identify susceptible individuals and subgroups in environmentally exposed populations. Companies offer gene tests to individuals and employers. As long as this testing is not scientifically and ethically above reproach, it can benefit only companies selling the tests. There is an urgency to address several important ethical questions with regard to societal and public health" (Hirvonen 1999).

This gene-technological development of tests demonstrates the predominant tendency to overemphasize structural gene aberrations instead of considering the bioenergetic conditions for genetic expression for the biosyntheses of enzyme proteins and studying exposure risks and compensating individual dispositions by non-aggressive prevention.

The guiding criteria for diagnosis amongst those testing "HIV positive"

All available experimental, clinical and epidemiological data result in the main principles of diagnostics, prophylaxis and therapy of systemic diseases in clinical practices. Pre-AIDS and

AIDS, because of the relative straightforwardness of the cause and effect relationship between exposure and predisposing factors, present a good model of the overregulation and counterregulation of the cellular symbiotic interactions in immune and non-immune cells and the consequential systemic processes.

There is no reason for panic should a patient find himself stigmatized as “HIV positive” as a result of the “HIV test”.

Death prognoses are an expression of limited medical knowledge rather than justified in biological fact. The period of incubation from the “HIV seroconversion” to manifest symptoms averages 12 – 15 years. In the USA, where patients are treated aggressively and early with prooxidative chemotherapeutics and chemoantibiotics about 5% of patients stigmatized as HIV positive become ill. Consequently, under these conditions, it would take 20 years for all “HIV positives” to actually become manifestly ill. However the actual incidence depends on the persistence of primary exposition risks, on the secondary exposure risk through the aggressive therapy schedule and on the omission of targeted compensatory and regulatory therapies, if they are necessary in the first place.

A careful anamnesis of the patient is necessary; it is not enough just to state that he belongs to a risk group. Allergy predisposition or atopic skin diseases, asthma etc. can be important indicators for a patient’s disposition to type-2 cytokine reactions and increased antibody production. The absence of typical bacterial children’s diseases can, with other indicators, also be a sign of a type-2-disposition. As more than 70 symptomatic conditions can result in a positive HIV test reaction and even the HIV/AIDS researchers categorize *a priori* 5% of all confirmed positive “HIV tests” as insignificant diagnostic findings, medical actions cannot and must not be guided by the positive result of the “HIV test”, irrespective of the non-isolation of an actual immune weakening “HI virus”.

The determination of the immune cell status and antibody status are obligatory. The number of differentiated cells measured within the immune cell network and the immune globulin classes, cannot alone be considered reliable indicators for the actual existence of an immune cell deficiency in symptom-free patients as within any healthy population roughly 5% have T4 cell values below 500 per microliter in their blood stream. For HIV/AIDS medicine this T4 cell count is already interpreted as a reason for chemotherapeutic and chemoantibiotic intervention in patients testing HIV positive. In healthy people the T4 cell counts can even drop below 200 per microliter without a serious loss of cellular immunity functions. Without seriously limiting their functionality, the number of T-helper immune cells in the blood stream depends on multiple influences. Valid information can be obtained through the DTH recall antigen test (antigen recall test of the skin, delayed type hypersensitivity. A strong DTH test reaction is considered a reliable indicator of the actual functionality of type-1 cytokines, activating a cytotoxic NO defense gas against the intracellular pathogens after antigen stimulation (Christou 1986, 1995, Mosmann 1989, Hässig 1998 b).

(Note: Four weeks after publication of this book (in November 2001), the DTH test was withdrawn from circulation worldwide by its producer, the pharmaceutical industrial group Aventis-Mérieux (which includes the subsidiary Mérieux-Pasteur). As Aventis-Mérieux (now merged with French pharmaceutical group Sanofi) holds the patented monopoly of the DTH test, there is no alternative diagnostic instrument. Biosyn, the German DTH test vendor, pointed out in a written statement, that Aventis-Mérieux had for no apparent reason abruptly stopped delivering the DTH test (including the last charge, contrary to contract). They, Biosyn, had been trying to get a license for the production of the DTH test, but Aventis-

Mérieux prevented their attempt. This behavior can only be understood by considering the background. Aventis-Mérieux had at that time (November 2001), been involved in studies using so-called naked DNA as an “anti HIV vaccine” on human experimental subjects in Uganda and Thailand. Such a vaccination was considered to promise billions of dollars in sales. An easy-to-handle DTH test could be detrimental to this purpose. In African countries, for example, the “anti-HIV-antibody test” frequently reacts positive for people previously infected with tuberculosis or malaria pathogens, which have endemic proportions there. In addition, conventional scientists deny any connections of this reaction to an “HIV infection”. In such cases, the use of DTH skin reaction tests would show a sufficient Th1 immune cell reaction, if subjects of the test didn’t manifest chronically present TB or malaria exposure of clinical relevance. A routine use of a DTH test before a mass-vaccination with naked DNA would consequently have been able to unmask the global “HIV infection” as a scientific campaign of disinformation. In the meantime, experiments with naked DNA against “HIV infection” have been suspended. Only after leukaemia appeared in infants, treated with genetically upgraded stem cells, to fight a serious immune deficiency, has a global moratorium on human trials on gene therapy been declared.)

Therefore, the acute danger of intracellular opportunistic infections is not only due to the positive result of an “HIV test”. A weak or anergic (ineffective) DTH skin test reaction indicates the probability of a prevalent shift to type-2 cytokine status and the danger of opportunistic infections. (Note: Nowadays the cellular immunity can be measured using the lymphocyte transformation test (LTT). This test uses comparable antigens to the no longer available DTH skin test but the costs are higher. Alternatively it can be measured by cytokine 1 – cytokine 2 profiles for determination of a possible Th1-Th2 switch).

Measurement of the values of reduced glutathione in the plasma, in the lung mucosa and intracellularly in the T4 lymph cells of the blood stream is essential (on the laboratory process: Buhl 1989, Herzenberg 1997, Nuttall 1998).
At the same time, the cyst(e)ine level in the plasma must be determined. Major deviations from the non-protein thiol norms must be treated, even in symptom-free patients.

Preventing and treating systemic illnesses with glutathione/cysteine compensation

The organism’s need for thiol is often underestimated or neglected. After the predominant scenarios in the “thioester-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 sulfhydryl groups through “weak interactions”, (De Duve 1991). Saltwater contains naturally an elevated sulfur concentration, but for terrestrial life forms there is a consistent danger of latent deficiencies of non-protein thiols and sulfates. Both are indispensable because they are responsible for the regulation of the redox milieu, the functioning of 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 symptom-free and symptomatic patients lack of thiol must be permanently compensated with individually adjusted doses. The “semiconductor thresholds” of redox-sensitive gene expression must be modulated in a sustainable and enduring manner

by the negative redox potential, which depends on the glutathione system, in order to retune the enzyme activities necessary for intact cellular symbiosis.

Since the stimulation of the neosynthesis of glutathione, due to the redox-dependent enzyme syntheses, is not guaranteed, at least 2 grams of glutathione and simultaneously 5 to 10 grams of N-acetyl cysteine must be orally administered per day for 2 to 4 weeks at the beginning of compensation therapy. As protection against opportunistic pathogens the glutathione concentrations, especially in the mucous membranes, are considerably higher than in the blood plasma (for example, pulmonary mucosal fluids contain about 150 to 200 micromoles of glutathione, blood plasma less than 5 micromoles). Lack of glutathione in the lung secretion layer is an important conditioning factor for cellular immune deficiency against the pneumocystis carinii fungi, the pathogens of the most common AIDS indicator disease, PCP of the lungs.

In cases of distinctive resorption dysfunction caused by infectious and non-infectious changes in the intestinal mucosa, corresponding doses of reduced glutathione and N-acetyl cysteine can be administered intraveneously. After balancing the intracellular and the plasma levels of the thiol pool and the glutathione concentrations in the lung and the intestinal mucosal liquids, cysteine treatment should be continued for another six months with a daily dosage of 5 to 10 grams of N-acetyl cysteine. In addition, cysteine and methionin, the latter is converted in the liver to cysteine, can be taken as part of the daily nutritional regime and can be found in low fat curds and other native organic dairy products (Bounous 1993).

Additional glutamine and arginine compensation to prevent or treat wasting syndrome (cachexia)

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 up to 40 grams per day of high dosage glutamine can be used to retune the synergic effect between glutamine and cysteine levels for T-helper cell maturation (Shabert 1999).

Simultaneously this effect improves the regeneration of intestinal and lung mucous membranes, energy metabolism of cell symbiosis and acid-base balancing. The glutamine facilitates detoxification activity in the liver via the glutathione system and slows down the urea production by reducing the arginine splitting to urea and ornithine.
If there is a clear-cut arginine deficit and the linked lack of NO gas production the cellular immune performance (Th4 cells, natural killer cells, neutrophile granulocytes) can be significantly improved by supplementing the thiol and glutamine balance in pre-AIDS and AIDS with doses of up to 30 grams of arginine per day and up to 2% of the caloric intake respectively (Barbul 1990, Bower 1990). Synergistic adjustment of the dysregulation of amino acids cysteine, glutamine and arginine can be achieved in the case of massive immune cell deficiency, forced aerobic glycolysis, malignant cell transformation and cell degeneration as well as a pronounced wasting syndrome, by using small intestinal probes or, if necessary, by parenteral infusion solutions. In critical cases of disease, glutathione can be administered intravenously.

Highly dosed compensation for thiol deficiency and amino acid dysregulation must be seen as the basic therapy of the redox environment and of a detoxification performance. These are

providing the organism with much needed and natural survival resources to facilitate its self-regulation. The success of therapy must be continuously supervised by laboratory checks adapted to individual requirements, as the intake of N-acetyl cysteine simultaneously raises the glutamine and arginine levels in plasma (Dröge 1997 a).

Rigorously administered compensatory therapy, in cases of pre-AIDS or AIDS, during a well-monitored treatment phase produces better and more cost effective results than the counterproductive prescription of chemotherapeutic agents (AZT etc., “cocktail therapy”, HAART) and permanent prophylaxis with chemoantibiotics (Bactrim etc.) which may bring short-term results, but have been proven to aggravate symptoms. If chemoantibiotics like Bactrim etc. have to be prescribed for a short time because of acute opportunistic infections, then it is vital to administer a strictly metered compensation of the thiol deficiency.

Obligatory compensation therapy can be effectively supported by a set of specific regulatory measures during the symptom-free phase of acquired immune deficiency as well as in the phase of systemic secondary diseases.

Additional liver protection, in particular with acute/chronic hepatitis B or autoimmune hepatitis (Oltipraz, nuitritive isothiocyanate, polyphenols, glucuronic acid)

Hepatitis is often evident in members of pre-AIDS and AIDS risk groups - promiscuous homosexuals, intravenous drug users and recipients of highly contaminated blood products (Hässig 1996 b, 1998 e) - and calls for additional liver protection to bring relief to the glutathione system and phase-II detoxification enzymes (Wilkinson 1997). In contrast to the phase-I enzymes, which generate reactive electrophiles (electron consuming substances) and activate carcinogens, phase-II enzymes inhibit electrophile bonds and turn them into water-soluble excretable substances. ‘Oltipraz’ is a synthetic agent and has proved highly effective. It was originally designed as an anthelmintic against schistosoma, which trigger type-2 cytokine dominance (Lucey 1996) analogous to the earlier stages of acquired immune deficiency. ‘Oltipraz’ is a sulphur-containing dithiolthione and primarily 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. Beside the protective effects against opportunistic germs and endoparasites the agent has been shown to have antiviral and anticarcinogenic effects (overview with Wilkinson 1997). These findings are significant especially after prior prooxidative damage of mitochondrial cell symbiosis caused by AZT etc. and permanent prophylaxis with ‘Bactrim’. ‘Oltipraz’ is equally efficient in the activation of the detoxification enzymes in the T-helper cells (Gupta 1995). A prescription of 125 to 250 milligrams/m2 twice a week for 12 weeks is an adequate dosage, as ‘Oltipraz’ causes a sustainable triggering of the phase-II detoxification enzymes and has very few side-effects. (Note: Dithiothione, trade name Oltipraz, up until now is only available for clinical studies. There has been no approval by the FDA, only publications about cancer treatment with Oltipraz in clinical research).

Among the natural substances, sulfur-containing isothiocyanates provide effective protection by triggering the variegated phase-II detoxification enzymes (overview with Hecht 1995). These thiocyanates are inherent in vegetables like garlic, onions, as well as in broccoli and several other cabbage species. The other important family of natural liver protecting agents are polyphenols. Animal and human organisms are not able to synthesize aromatic bonds with

benzene rings from preliminary stages. Polyphenols must be ingested with nutrition like algae or other plants and are thus similar to vitamins (Hässig 1997 c). The redox-cycling between the glutathione system and the polyphenolic substances is crucial for the balancing of the redox milieu and the detoxification performance by the polyphenols, as well as for the triggering of the phase-II detoxification enzymes or as the case may be, the inhibition of the phase-I enzymes. Above all, polyphenols assist enzymes 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 with Wilkinson 1997).

Antioxidative protection of cell symbioses of liver cells and other cell systems including the immune cells, by polyphenols is of particular importance in the highly acute AIDS state, if intracellular opportunists can proliferate without inhibition, due to the failure of the cytotoxic NO-gas producing Th1 helper cells. 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 microglia cells in the brain are hyperactivated by the modulation of pro-inflammatory cytokines (interleukin-12, interleukin-1, tumor necrosis factor alpha and others, inflammation mediators and nitrogenic and oxidative radicals).

The elevated quantity of neopterine (as a folic acid metabolism product) and the beta-2 protein in the circulating blood are indirect markers for an hyperactivation of the proinflammatory cytokine activity of the unspecific immune cells with a simultaneous suppression of the cytotoxic NO-gas production of the specific immune cells (full-blown AIDS) (Mauri 1990, Odeh 1990, Fuchs 1990, Harrison 1990, Matsuyama 1991, Krown 1991, Hässig 1993, Valdez 1997).

Consequently, well-balanced cell protecting counterregulations fail and the simultaneous cytotoxic overregulations (prevalence of interleukin-12 compared to type-2 cytokine interleukin-10) incapacitate the feedback functions. In cases of a thiol deficiency and an overconsumption of other antioxidants, the redox balance collapses in the cytokine chaos (Cossarizza 1995).

The clinical studies concerning polyphenols over the last years have mainly concentrated on ellagic acid, the polyphenols in green tea, curcumin, silymarin etc. (overview with Stoner 1995, Conney 1997, Wilkinson 1997, Zhao 1999, Plummer 1999).

Another possibility is the Galenic combination of glutathione with polyphenolic anthocyans (Recancostat, Ohlenschläger 1994) or with the ginkgo biloba polyphenol (S-acetyle glutathione, SAG). The polyphenolic complex phytotherapeutic ‘Padma 28’, produced in Switzerland from a traditional recipe of Tibetan medicine, containing 20 herbal flavonoids and tannins, has proved its worth in protecting the liver in chronic cases of hepatitis B (Brzosko 1992, Liang 1992, Hässig 1997 c).

Additionally, reinforcing the supply of glucuronic acid can relieve liver cell symbiosis. Glucuronic acid plays an equally important role as a phase-II regulator of prooxidative and carcinogen-activating foreign agents in the liver by transforming toxins into secretable substances. Kombucha, the organic product from China, containing a symbiosis of fungi and specific bacteria is a natural source of glucuronic acid that contains, besides high concentrations of glucuronic acid, vitamin B compounds and antibiotic substances. Kombucha can be made at home (Frank 1992).

Prostaglandin (PGE2) modulation (essential fatty acids: omega-3/omega-6; COX2 inhibitors)

The characteristic progression of prostaglandin synthesis, especially PGE 2, under the influence of type-2 cytokine dominance in pre-AIDS or AIDS, as part of the Type-II counterregulation, can also be countermodulated therapeutically or preventively. Elevated quantities of PGE2 inhibit, like the type-2 cytokines, the synthesis of the cytotoxic NO gas and thus enhance opportunistic infections. The prostaglandins are products of the arachidonic acid, an essential fatty acid. Arachidonic acid is enzymatically metabolized into prostaglandin within the cell's plasma membrane by the enzyme cyclooxygenase (COX). In AIDS, cancer and other systemic diseases, the COX-2 isoform appears elevated. COX-2 increases PGE2 production and also raises the type-2 cytokines interleukin-6 production, which can trigger wasting syndrome (Hack 1996). Symptomatic for all systemic diseases like AIDS and cancer, wasting syndrome can be influenced by the selective inhibition of the COX-2 (O'Hara 1998). PGE2 is enzymatically generated by COX-2. In the same sense as growth factor TGF-ß, it activates the generation of polyamines from the arginine product ornithine. Therefore, the blockade of COX-2 through medication also inhibits tumor growth, reduces wasting syndrome and improves the Th1-Th2 balance of cellular immunity (Subbaramaiah 1997, Huang 1998, Jones 1999, Lipsky 1999 a, 1999 b, Sawaoka 1999, Golden 1999, Masferrer 2000, Kune 2000, Prescott 2000, Reddy 2000, Higashi 2000, Stolina 2000).

However, in symptom-free patients with a weak or an anergic Th1 immune cell population, prostaglandin modulation through essential fatty acids is a better option for treatment. In animal testing the Th1 immune cell population's stimulability in the DTH skin reaction test was inhibited, when 15% of the intake of calories consisted of linolenic acid, but not with the same quantity of fish oil with its high content of omega-3 fatty acid (Alexander 1990). Just 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. Admittedly it is necessary to ingest a couple of grams per day for several weeks in order to stimulate immune cell reaction and inhibit tumor formation. The effect of the mitochondrial cell symbiosis protection is improved by simultaneous substitution with cysteine, glutamine, arginine and RNA (Bower 1990, Cossarizza 1995, Chuntrasakul 1998, Gianotti 1999).

The low or high fluidity of the micro-Gaia milieu of cell symbiosis and the fluidity of the cell membranes reflect the type and the composition of the multi-unsaturated fatty acids (Bower 1990, Fernandes 1998, Simopoulos 1999, Zelenuich-Jaquotte 2000). The interaction between the synthesis of NO and its derivates and the prostaglandin PGE2, which is synthesized from the arachidon essential fatty acid, is equidirectional in small amounts but antagonistic in larger amounts (overview with Lincoln 1997, Minghetti 1998). This interaction is of vital importance for the prevention and therapy of Type-II counterregulations of cell dyssymbiosis (systemic diseases) including the type-1 to type-2 cytokine switch (cellular Th1 immune deficiency, pre-AIDS) combined with pro-inflammatory macrophage hyperactivation (opportunistic infections, full-blown AIDS). It is possible to effectively counterregulate massive regressions of the cell symbiosis with omega-3 multi-unsaturated fatty acid and its derivates (Veierod 1997, Imoberdorf 1997, Gogos 1998, Albert 1998, Ogilvie 1998, De Lorgeril 1998, Tashiro 1998, Rose 1999, Bougnoux 1999, Burns 1999, Bartsch 1999, Biasco 1999).

Individually customized micronutrient compensation (vitamins, minerals, trace elements)

The use of micronutrients (vitamins, minerals and trace elements) must be considered in a differentiated way regarding compensation and regulation therapies for the prevention of pre-AIDS and AIDS as well as for other systemic diseases.

"Nowadays the intake of vitamin E, in combination with vitamin C and ß-carotene, is worldwide the standard antioxidative treatment. In "The antioxidant supplement myth", Herbert critically analyzes this process (Herbert 1994). He conclusively demonstrates that this process is afflicted with serious disadvantages as pharmacological doses of a single polyphenol, like for example vitamin E in combination with vitamin C and ß-carotene, have some positive, but often damaging effects, depending on the receptor's iron balance. As redox compounds have both prooxidative and antioxidative effects the treatment can be summed up by the sentence: supplementation (of micronutrients) can help some consumers, harm others, but for most people they have no effect whatsoever. Thus, it was demonstrated that vitamin C (ascorbic acid), in the presence of redox-active transition metal ions, like iron (Fe^{3+}) or copper (Cu^{2+}), can act as a prooxidants and indirectly, by the so-called Fenton reaction, help to develop highly reactive hydroxyl radicals (HO) (Fenton 1894, Halliwell 1993, Cottier 1995). The synthesis of hydrogen peroxide (H_2O_2) occurs through a slow pH-dependent dismutation of superoxide radicals: $2O_2^{-} \bullet + 2H^{+} \longrightarrow O_2 + H_2O_2$. Incidentally, as chelators of free metals, tannins can contribute useful services in this situation. Kim et al., who in studies on 14,407 Americans could find no life prolonging effects in the use of isolated and unbalanced vitamins and mineral nutrition supplements, have extensively affirmed Herbert's critical statement. They identified the annual expenditure of $3.3 billion for nutrition supplements as a virtually useless augmentation of the health care costs (Kim 1993). In conclusion, we would like to state that a sufficient nutritive supply of a natural mixture of tannins and flavonoids is indispensable for a reliable and side-effect free antioxidative effect" (Hässig 1997 c).

Vitamin E and vitamin C generate radical chain reactions as intermediate states, which must be compensated for by the glutathione system (Ohlenschläger 1994), therefore a given thiol deficiency can be further aggravated by the intake of high dosages of these vitamins. Micronutrient requirements, in pre-AIDS and AIDS, should be evaluated in the context of a fine tuning of strict compensation and regulation therapy because deficits of individual micronutrients depend on the redox status, the mitochondrial activity, the cytokine balance, the existence of wasting syndrome, any given resorption dysfunction, severe diarrhea, toxic and infectious stressors, alloantigen overcharge, chemotherapeutic agents, chemoantibiotics, antiparasitics, fungistatics, virustatics etc, excessive alcohol, drug or cigarettes usage and many other factors. Uncontrolled self-medication does not make much sense and can in some cases even be dangerous.

A profiling study of ambulatory pre-AIDS and AIDS patients in relatively good health without a clinically definable wasting syndrome or heavy diarrhea quantified:

- Vitamin A and total carotenes, vitamins C, E, B6, B12, folate, thiamin, niacin, biotin, riboflavin, pantothenic acid, free and total choline and carnitine, biopterin, inositol, copper, zinc, selenium, magnesium and glutathione.

The results of the study confirm a reduction in the circulating concentrations of glutathione and relatively common lower serum concentrations of magnesium, total carotene and total choline plus increased niacin levels. The remaining values were within normal ranges or lower in a minority of the test subjects partly through self-medication with vitamins and minerals (Skurnick 1996).

Today's HIV/AIDS clinical research concerning micronutrients as influencing factors for acquired immune deficiency states (pre-AIDS and AIDS) confirmed the individual dependency on deficiencies in a holistic context of dysfunctional cell symbiosis.

"Deficiencies of single micronutrients are known to adversely affect the immune system by depression of cellular and humoral immunity and the impairment of phagocytosis (Beisel 1982, Klurfeld 1993). Individuals infected with the human immunodeficiency virus type 1 (HIV-1) may be particularly vulnerable to nutritional deficiencies that impair already compromised immune function. In a previous study of HIV-1 infected patients, w found that carotenes and ascorbate were below normal in 27% of the subjects, and vitamins E and A were low in 12% (Bogden 1990). Serum levels of micronutrients in HIV-1 patients have been associated with markers of immune function and stage of disease (Fordyce-Baum 1990, Baum 1991, 1992, Semba 1993). Studies have shown that abnormalities in nutriture both accompany and predict HIV disease progression (Semba 1993, Coodley 1993, Tang 1993, Abrams 1993). These investigations assessed dietary intake or serum concentrations of one or a few micronutrients in selected cohorts" (Skurnick 1996).

The primary influence of micronutrients on the prevention and therapy of cancer was also put into perspective in comparison to the importance of the redox status, NO- and prostaglandin syntheses, the cytokine balance and cell symbiosis activity (World Cancer Research Fund 1997).

An additional measuring of the serum ferritine level must be considered essential, which in pre-AIDS or AIDS patients as in all pro-inflammatory stages of macrophage hyperactivation is evidently elevated (Gupta 1986) and plays an important role in all Type-II counterregulations (Gherardi 1991, Weinberg 1992, Herbert 1992, Gelman 1992, Lacroix 1992, Kiefer 1993). In addition to compensation therapy of the redox status, the reinforcement of the matrix has an important function in regard to the regulation of the iron balance (Pippard 1989, Hässig 1993).

Stabilization of the extracellular matrix (polyanions)

The basic extracellular matrix, which embeds all tissues and organs, functions as filter for all the bioenergetic, substantial, hormonal and sensory inputs and outputs of cellular symbiosis. Amongst other things, the matrix is composed of a complex network of sulfate-rich protein molecules (glycosaminoglycans, proteoglycans), which are necessary for the negative redox potential. Re-fetalization of the extracellular matrix into sulfate-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 by a regular supply of polyanions, chondroitin sulfates in the form of cartilage preparations or shark cartilages, of

macroalgae, of agar-agars or by eating macroalgae (Hässig 1992). The balance of the redox potential of the matrix synergistically supports the glutathione system and relieves cell symbioses in states of prooxidative and systemic stress (Hässig 1992, 1997 a, 1998 b).

Direct activation of the mitochondrial respiration chain (coenzyme Q10; L-carnitine; possibly lipoic acid and thiamine)

Direct activation of the mitochondrial cell symbiosis 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 respiratory chain. In symptom-free “HIV positives”, a Q10 deficit is already apparent and progressively increases in pre-AIDS and AIDS. Toxic stressors and prooxidative medication (AZT etc., Bactrim etc.) are decisive factors that lead to dysfunction in the mitochondrial respiratory chain and secondary defects in the mitochondrial DNA. Q10 improves the cell symbiosis performance in immune cells and non-immune cells and can be prescribed in a daily dosage of 200 milligrams for a several months without detectable side effects (overview with Folkers 1988).

L-carnitine supports the participation of long-chained fatty acids (triglycerides) for the oxidation inside mitochondria. L-carnitine deficits increase glucose metabolism and facilitate a switch to aerobic glycolysis (Warburg Phenomenon). The disturbance in triglyceride transport causes lipid accumulation, which is often observed in treatment with HAART and protease inhibitors (Brinkmann 1999). Pre-AIDS and AIDS have been shown, in the context of a L-carnitine deficit, to be systemic dysfunctions of lipometabolism and of the lipid composition of the T-lymphocytes (De Simone 1991). Administrating high doses of L-carnitine, 6 grams per day for two weeks, has improved T-helper cell proliferation, lowered the triglyceride serum levels and decreased the serum values of circulating beta-2 microglobulins and alpha tumor necrosis factors as indicators for hyperactivation of macrophages in HIV positives and AIDS patients. L-carnitine also appears to stabilize the cytokine balance by ameliorating mitochondrial performance (overview with De Simone 1993).

Reduced mitochondrial performance as consequence of chemotherapeutics, caused by damage to mitochondrial DNA after the intake of AZT etc. and Bactrim etc., can additionally be compensated by the daily dose of 600 mg lipoic acid (alpha-lipoic acid) plus 300 mg thiamine (vitamin B1) for a month or longer.

Targeted activation of mitochondria is especially significant for “HIV positives” but also for cancer patients, who years after enforced chemotherapy are still under the threat of multiple organ failure (myocardial infarction, sepsis, cerebral infarction, hepatic coma, myopathies etc.) as a result of potentiating mitochondrial DNA defects.

The moderation of hypercortsolism (DHEA-S; glucosaminoglycans; heparin; heparinoids; phytotherapeutic complexes: flavonoids + tannins)

The cytokine balance and the related equilibrium between cell-mediated and antibody-supported immunity are, as all organ systems, closely related to the sensory and hormonally controlled stress systems. The retroactive hormonal stress axis between the hypothalamus, the hypophysis and the adrenal cortex modulates the cytokine profiles via equilibrium between cortisol and DHEA-S (dehydroepiandrosterone sulfate), both generated in the adrenal cortex. The final cortisol synthesis takes place in the mitochondrial cell symbionts of the adrenal cortex cells (Tyler 1992), so that disturbance and damage of these cells can favor 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 when compared to the DHEA levels. This causes the inhibition of cytokine synthesis through the interaction of cortisol with transcription factors (Brattsand 1996). Persistent cortisol increase facilitates the antibody-supported immune response and weakens cellular immune reaction. On inhibition of the type-1 cytokine profile, however, under the 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 tumor necrosis factor-alpha can be increased within the macrophages. The neopterin and ferritin levels serve as a direct quantification of the extent of the inflammatory macrophage-activation and as an indirect measurement serve such markers that display the scale of the acute phase reaction, like for example the C-reactive protein (Hennebold 1994, Hässig 1997 d, 1998 b).

Inversely, a shift from a type-1 (Th1) cytokine profile to a type-2 (Th2) cytokine dominance through an increased cortisol/DHEA-S ratio means that a moderation in stress-related hypercortisolism amplifies the effect of DHEA-S on type-1 cytokine synthesis. This means an improvement to the cortisol/DHEA-S ratio in favor of the latter can expand cellular immunity by activating the type-1 cytokine interleukin-2.

There is indeed a direct correlation between the balance of T4 helper immune cells and the increased cortisol level (Th1-Th2 switch) or as the case may be the level of DHEA-S, the predominantly synthesized form. The development of acquired cellular immune weakness syndrome is associated with an increasing DHEA-S deficit (Biglieri 1988, Hilton 1988, Raffi 1991, Mulder 1992, Christeff 1996, Ferrando 1999). However, the 24-hour cortisol level seems to be elevated in AIDS patients, (Vilette 1990).

These findings resulted in the hypothesis that DHEA-S substitution (DHEA-S as an anti-cortisol hormone) could enhance cellular immunity for the prevention and therapy of opportunistic infections in cases 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 the cytokine controlled functions of cell symbiosis of the immune cells, but also for other cell systems (Parker 1985, Ebeling 1994, Lavallee 1996). DHEA is a precursor molecule for androgenous sexual hormones and the DHEA-S dysregulation is a co-determining factor in tumors in hormone-dependent organs such as the mammary or prostate glands as well as in tumors in other organs (Vermeulen 1986, Heinonen 1987, Barrett-Connor 1990, Stahl 1992, Le Bail 1998, Lissoni 1998, Svec 1998, Eaton 1999).

The moderation of hypercortisolism and the indirect type-1 cytokine stimulation via DHEA-S

can in many cases be supported by nutritive measures, including an increase in the extracellular content of glycosaminoglycans (heparin, heparinoids). They reduce the influx of calcium ions into the inner cell and inhibit cortisol bonding on the intracellular receptors. This can be achieved through an intake of cartilage extracts (chondroitin sulfate) or of agar-agar from sea algae (Hässig 1993, 1998 b). Simultaneously the proinflammatory hyperactivation of macrophages (as counterregulation) in a cortisol-related type-1 to type-2 cytokine shift can be repressed by binding surplus NO and O_2 radicals by intercepting excessive free iron and the increasingly formed catabolic proteases by using complex phytotherapeutics like Padma 28, a Tibetan preparation made of polyphenolic flavonoids and tannins (Liang 1992, Hässig 1993, Gebbers 1995).

The regulation of cytokine chaos in acute full-blown AIDS requires high-dose cysteine/glutathione compensation + DHEA-S + gammaglobulins, in order to curb the antagonism between simultaneous macrophage hyperactivation and the type-1 cytokine inhibition of T-helper immune cells

The moderation of cortisol and the reactivation of DHEA-S in interaction with the inhibition of pro-inflammatory macrophage stimulation are important additionally, as macrophages because of their phagocytosis performing capacities represent a preferred reservoir for intracellular opportunistic pathogens (Rubin 1988, Meltzer 1992). The counteraction to a strong and long-lasting nitrosative, prooxidative and systemic stress effect results in an elevated cortisol/DHEA-S ratio, a weakening of cellular immunity and an inhibition of cytotoxic NO gas defense through a type-2 cytokine switch and simultaneously in a pro-inflammatory mobilization of opportunistic residents in the macrophages (fungi like pneumocystis, candida, histoplasms, cryptococci, parasites and toxoplasms, bacteria like mycobacteria, listeria, legionella and chlamydia and many actually existing viruses, in contrast to the “HI viruses”). These counterregulations must, sooner or later, lead to clinical full-blown AIDS, if the primary stress factors can not be minimized, the proton demand deficiencies are not balanced and the dysregulation of the cell symbiosis is aggravated additionally by the use of ‘chemo-tactical’ weapons. In full-blown AIDS there is a crucial antagonism between the behavior of the non-specific immune response of the macrophages and the specific immune response of T4 helper immune cells: The cortisol brake for the biosynthesis of tumor necrosis factor in the macrophages is suppressed by the activation of interferon-γ under strong or/and long-term stress stimulation (Luedke 1990) and the ratio of cortisol/DHEA-S in the macrophages is regulated in favor of the latter by their inflammatory cytokines (Hennebold 1994). In contrast, the T4 helper cells remain inhibited under the influence of cortisol and synthesize after receiving signals from the glutathione-depleted antigen presenting dendritic cells, predominantly type-2 cytokines (Peterson 1998). They inhibit cytotoxic NO gas synthesis, contrary to the macrophages (loss of helper Th1 cell functions) and stimulate instead antibody production (overview with Mosmann 1996, Lucey 1996, Abbas 1996, Hässig 1996 d, Lincoln 1997). The net result in the T4-immune cells is a change in the ratio of the cortisol to DHEA-S in favor of the former (Wisniewski 1993, Christeff 1996, Ferrando 1999).

The contradictory clinical symptoms of manifest AIDS result from this antagonism of unspecific inflammatory events combined with the mobilization of opportunistic pathogens on the one hand and the loss of the specific Th1 gas production against intracellular opportunists on the other hand. The prooxidative, glutathione-consuming and mitochondriotoxic

chemotherapy with AZT etc. and sustained prophylaxis with Bactrim etc. are not able to control the competing cytokine chaos resulting from unspecific immune hyperactivation within the macrophages (Type-I overregulation of among others things: type-1 cytokine interleukin-12 antagonistic towards type-2 cytokine interleukin-10; tumor necrosis factor alpha increased, type-1 cytokine interferon-γ increased; NO-and oxygen radicals including toxic hydroxyl groups increased) and the deactivation of the specific Th1 immune response (Type-II counterregulation of amongst others things: type-2 cytokine interleukin-10 antagonistic towards interleukin-12; in wasting syndrome type-2 cytokine interleukin 6 increased; in tumor cells TGF-ß increased; NO and O_{2-} production inhibited). The most effective option is compensating the thiol deficiency, whereby cysteine slows down the cytotoxic effects of tumor necrosis factor within the hyperactivated macrophages and improves glutathione neosynthesis (Cossarizza 1995).

The preventive and therapeutic aim must be to balance the redox milieu, to improve the fluidity of the micro-Gaia milieu, to reconstruct the cytokine balance and simultaneously to moderate the competition between Type-I overregulation of unspecific immunity and Type-II counterregulation of specific immunity. This can only be achieved by a synergistic compensation and regulation therapy.

Overcoming fear and anxiety, and the profound shift in knowledge of healing from chemoantibiosis to nontoxic cellular symbiosis therapy

“Last but not least it is essential to resolutely confront the still widely propagated and officially held belief that every HIV-infected person must sooner or later progress to AIDS and inevitably die (Hässig 1992 b). On the contrary, we should give HIV positives the hope that by adapting their lifestyles to the opportunities given by nature and they may be spared from the limitations of AIDS for a long time, maybe forever. To achieve this they have to come to terms with their nutritional problems. In our overview work, “The Rethinking of AIDS”, which was published about one year ago, we asked whether this could lead to a general paradigm shift in medicine (Hässig 1992 b). Nowadays, we tend to assume that such a shift will happen. The use of AZT and analogous virucidal medication as recommended by the responsible authorities, is based on the antibiotic paradigm, which means the toxicological extinction of microbial inflammation germs. Man lives, however, in an ongoing symbiosis with a whole range of microorganisms, hence the question is justifiable if it would not be more sensible to support the probiotic, physiological mechanisms of self-healing to support organisms”(Hässig 1993).

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 overriding task of physicians to reduce the paralyzing and destructive fear of death and instead encourage people affected by systemic cell dyssymbiosis by reinforcing their natural will to survive by clarification of the actual state of knowledge. The most effective protection against the abuse of “violent medicine” (Albonico 1997) as a modern instrument of terror and fear is the rational knowledge, that every kind of risk for and any targeted attack on, the cell symbiosis of immune cells and non-immune cells is answered according to the laws of evolutionary biology.

An imagined “HIV retrovirus”, if it were to exist, would be no exception. The actual clinically monitored symptoms in pre-AIDS and AIDS would, if a biologically active “HIV agent” actually were to be the cause of the disease, be conditioned by the disturbance of the redox balance, cell symbiosis damage and by the shifting within the micro-Gaia milieu. The preventive and therapeutic consequences of the deactivation of such a biologically undetected “HIV retrovirus” would be principally the same as for all other prooxidative load factors. Irrespective of the type of exposure, these basic consequences are universally valid whether they are of a toxic, pharmacotoxic, traumatic, inflammatory, infectious, nutritive, radiative, alloantigenic, psychic or any other nature. People with an especially redox-sensitive disposition must all be advised to avoid risks of exposure and orientate their nutrition according to their blood group as a code for the genetically predisposed polymorphism of the enzyme systems (D’Amato 2000).

The lifesaving synergy of a conscientious compensation and regulation therapy is derived from the logic of the natural laws of the co-evolution between microbes and man, the processing of toxins and other bioactive stress factors, including the consequences of undernutrition and malnutrition.

The profound change in ‘natural’ scientific knowledge of the sciences progresses from antibiosis (from the Greek: *anti* = against + *bios* = life) to symbiosis (from the Greek: *sym* = with, together). The foreseeable end of lethal virus hunting and of one-sided aggressive cancer expunging represents, both for those concerned and for medical therapists as well as for general population, a self-critical liberation from the staging of a collective and exploitive terrorism of fear.