ACQUIRED IATROGENIC DEATH SYNDROME (AIDS)
Pneumonias & Lung Diseases

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PCP accounts for the majority of Western AIDS diagnoses - yet the diagnosis of AIDS can be given on the basis of a presumptive diagnosis of PCP alone. A positive "HIV"antibody test is not even necessary. Is this definition alone creating a hothouse of fear and, with its assumptions, distracting patients and doctors from recognising the real processes of risk? In particular, the revelation that PCP is not an opportunistic infection challenges itself the wisdom of drug prophylaxis.

In 1996, the respected British medical journal The Lancet began publishing a series of special articles about HIV and AIDS, addressing various aspects from vaccines to the nervous system. All the articles rashly assumed that an isolated retrovirus called HIV causes AIDS.

In the following article, Dr. Heinrich Kremer responds to one such article, by Dr. Miller, on "HIV-associated respiratory diseases". In a knowledgeable and inquisitive manner he brings into unrelenting focus the deeply disturbing way in which damaging medication has helped create rather than cure the problems of PCP and other conditions.

Pneumonia is a frightening prospect for anyone, treating physicians included. Undoubtedly prevention is better than cure. But does this involves a fresh commitment to look behind the plague-mongering to the sensitivity of our biological systems, and the pressures of present and cumulative chemo-toxicity?

> "Progress comes from individual creation and imagination, not from the narrow dogmatism of a burgeoning AIDS establishment."
>
> -- The Lancet editorial, July 6,1996

It was one of the early pioneers of modern medicine, the German physician Rudolf Virchow (1821-1902) who, at the height of his career, said he wanted to become an MP in order to see to the completion of Berlin's antiquated sewage system, otherwise he could not successfully fight tuberculosis. How right he was! Only 100 years ago one worker in three died of tuberculosis. But until about 1950 tuberculosis had become rare in Western industrial countries, practically without recourse to drugs, which only became available towards the end of the 1940s. Above all, improvements in hygiene, living conditions and nutrition were instrumental in curbing tuberculosis of the lung.

Nowadays, however, there are modern successors to Virchow's causes of tuberculosis to surprise us, namely,

> "the association between HIV infection and tuberculosis is well described ... Tuberculosis in an HIV-infected individual is an AIDS-defining illness ..."

This claim is one of the many assertions by Dr. Miller in his article "HIV-associated respiratory diseases," which he divides into eight infectious and four non-infectious diseases: Infectious: 1) Upper respiratory tract infections, 2) Acute bronchitis, 3) Acute sinusitis, 4) Bacterial pneumonia, 5) Pneumocystis carinii pneumonia, 6) Mycobacterium tuberculosis, 7) Mycobacterium avium intracellulare, 8) Fungal pneumonia;
Non-infectious: 1) Kaposi's sarcoma, 2) Lymphoma, 3) Non-specific interstitial

pneumonitis, 4) Lymphoid interstitial pneumonitis.

Now, it turns out to be something of an advantage for me as a medical doctor to have kept away from conferences and not to have much beyond schoolboy English, because I had to check up on the exact meaning of 'association' and 'associated' in the Oxford Dictionary, to find that they mean 'connection in the mind' - in other words, we are dealing with a suggested mental, rather than causal, connection between HIV and respiratory disease, as well as between HIV and tuberculosis. Does Dr. Miller want to lecture us on ideology rather than biology? Not at all, Dr. Miller quickly explains:

> "tuberculosis is a potent stimulator of cell-mediated immunity, activating HIV production in lymphocytes and monocytes/macrophages latently infected with HIV, which brings out the spread of HIV infection to other cells."

It dawns on me why tuberculosis -1.7 billion infected worldwide, 600 million cases annually, 2 million deaths, of which 95% are in developing countries; in Western countries less than 0.05% of the population is affected by the disease, of which more than 95% are homeless, alcoholics, IV-drug users, asylum seekers - "is an AIDS-defining illness". Up till now AIDS-defining illnesses were supposed to be the consequence of alleged causative HIV infection. It now seems that diseases which first have to rouse "dormant HIV" from CD4 lymphocytes and macrophages are also part of the AIDS collection.

This means every disease process which has in any way reacted with thymus-matured immune cells (T-cells) can henceforth be renamed an "AIDS-defining disease", if it can simultaneously be "associated with HIV." There are no limits, therefore, to what can be done in the virtual, toytown world of AIDS, since in practically all serious diseases there is some contribution of cellmediated immunity. Dr. Miller grabs this opportunity by the throat and lists all "11 IV-associated respiratory diseases," and observes that "the clinical features of upper respiratory tract infections, acute bronchitis and acute sinusitis are the same in HIV-infected individuals as in those without HIV, but their frequency is increased."

So saying, Dr. Miller has deftly dealt with three of his eight "infectious HIV-associated respiratory diseases."

Bacterial Pneumonias

But it gets more serious when Dr. Miller classifies as the fourth HIV-associated disease group "bacterial pneumonia".

> "The spectrum of bacterial pathogens is similar to that of community-acquired pneumonia in the non-HIV-infected population."

In this disease group, too, Dr. Miller offers an intellectual prop to the "HIV-association":

> "bacterial pneumonia occurs more frequently in HIV-infected individuals than in the general population and is especially common in HIV-infected intravenous drug users."

Indeed, in the general population in Germany, for example, less than 1% of the population is affected by bacterial pneumonias. IV-drug users suffered more frequently from bacterial pneumonias long before AIDS came along for reasons well known to the venerable Virchow - unsatisfactory hygiene, malnutrition, bad housing etc., etc. (see above). The really important point, however, Dr. Miller conceals from us: IV-drug users classified as "HIV-infected" suffered from bacterial pneumonias, as a comprehensive study in Berlin has shown, whereas non-bacterial pneumocystis carinii pneumonia (PCP) the most frequent "HIV associated respiratory disease" in Miller's list (and the most frequent "HIV associated disease" or "AIDS-defining illness" altogether in the West) does not to all intents and purposes feature at all in IV-drug users who are not homosexual.

We can now see more clearly why bacterial pneumonias that are on the official list of AIDS-indicator diseases in adults only if they occur more than twice a year, have to be

wangled in under the guise of 'HIV-associated.' Without the creation of "HIV" no-one would ever have dreamt of the need to diagnose IV-drug users as "AIDS," because PCP and Kaposi's sarcoma, the most common "AIDS indicator diseases", do not occur in non-homosexual IV-drug users in Western countries despite a laboratory finding of 'HIV-positive.'

Why does Dr. Miller conceal these facts in his Lancet article? Dr. Miller lumps together everything which in patients labeled "HIV positive" could be called respiratory tract diseases. Because the introduction of a new cause for these long-known diseases from the same long-known causes, in the very same long-known categories of persons, would seem rather untrustworthy, he makes these 'old-timers' among the common respiratory tract diseases the driving force of the newly invented "HIV" infection and lumps them together as "HIV-associated disease" or "AIDS related processes" as he likes to call them. That these patients are thereby exposed to an increasing number of pharmaceutical drugs does not seem to worry Dr. Miller unduly, as his treatment of PCP, the fifth of the eight "HIV-associated" diseases in his list shows.

Pneumocystis Carinii Pneumonia

Strangely, pneumocystis carinii pneumonia turns up here as a "genuine" AIDS-indicator disease under the conditional heading of "HIV-associated respiratory disease." Has Dr. Miller lost faith in the orthodox belief in "AIDS"? Not at all. he quickly goes on to explain:

> "P. carinii remains a common respiratory pathogen in individuals with AIDS."

Miller again uses the idiosyncratic phrase "in individuals with AIDS." A doctor's diagnosis of PCP alone is entirely sufficient according to the most official AIDS definition to say "this patient is an AIDS case" or "this patient has AIDS." And this attribution of "AIDS" due to the diagnosis of PCP alone, establishes 40% of all clinical "AIDS cases" and about 80% of all "AIDS deaths" in Western countries.

The official definition of the authoritative American CDC has since 1987 remained unchanged - an AIDS diagnosis is justified even if only a presumptive diagnosis of PCP exists, and in the absence of any laboratory suggestion of "HIV-positivity," and without any noticeable decline of immune cell values in blood serum.

Thus, PCP was from the begining synonymous with AIDS, even without the 'S' (for Syndrome, of another 28 diseases) and without the 'ID' (for immune-deficiency, interpreted from decrease in CD4 lymphocytes in the bloodstream), and without the 'A' (for Acquired "HIV infection"). Naked PCP therefore is the seed of "AIDS". Everything else Dr. Miller and his colleagues associated, as "connections in the mind".

In plain language, if there are other good reasons for the occurrence of PCP in homosexual patients, the "HIV-associated diseases" just melt away as yesterday's snow.

In this respect Dr. Miller comes up with a genuine surprise. Miller explains in considerable detail that according to the latest research findings, the pathogen responsible for PCP is an airborne fungus and not, as thought up till now by AIDS doctors, a unicellular animal parasite. This implies an enormous difference from the diagnostic and therapeutic point of view: and in the real case of an individual patient, this reclassification can mean the difference between forecasting life and death. Until now epidemiologists assumed PCP was a case of zoonosis, ie. a ubiquitous animal, prevented by cell-mediated immunity from breaking through the body's immune barrier and causing devastating pneumonias. This assumption was seemingly based, according to Dr. Miller, on the finding that 90% of children and adults in Western countries had antibodies to P. carinii without contracting that form of pneumonia. But now it turns out the essential assumption - that the presence of antibodies indicates the presence of the pathogen - was fundamentally mistaken:

> "P. carinii cannot be detected with DNA amplification or monoclonal antibodies in bronchoalveolar ravage fluid or necropsy lung tissue of

> immunocompetent individuals and low levels of P. carinii are detected in the lungs of only 20% of immunosuppressed HIV-positive patients with respiratory episodes and diagnoses other than P. carinii pneumonia."

The conclusion is, therefore, that "HIV" was invented in order to explain the apparent fact that CD4 lymphocytes in ostensibly 'hitherto healthy' individuals could suddenly no longer hold in check the pneumocystis protozoa which had been there all along. The simple explanation which the now shattered HIV/AIDS theory led to, was: "HIV" is transmitted in semen, blood and blood products to the recipient, "HIV" destroys the thymus matured CD4 lymphocytes, the pneumocystis protozoa escaped their dead guards and kill their up till then healthy host. "HIV" was invented in order to explain the apparent fact that CD4 lymphocytes in ostensibly 'hitherto healthy' individuals could suddenly no longer hold in check the pneumocystis protozoa which had been there all along. According to this nightmare scenario anyone with "HIV" in his CD4 cells dies.

But suddenly now, everything turns out to be completely different:

Pneumocystis protozoa cannot escape from the CD4 immune cells, because pneumocystis protozoa are not there. Instead, since P. carinii is a fungus, is not transmitted in semen or blood, and is passed on through the air. This fungus, as Miller informs us, can be disposed of easily in 80% of "immune-suppressed HIV positive patients with respiratory episodes and diagnoses other than P. carinii pneumonia," without leaving a trace, and leaving in the rest of these "immune-suppressed HIV-positive patients" just "low levels" of P. carinii (whatever that may mean).

So, what has the laboratory finding of "HIV-positive" got to do with P. carinii pneumonia? What conclusions does Miller draw from his newly discovered findings? Answer: none. Miller simply reports the fact and carries on treating his patients as before:

> "The regimen of first choice for primary and secondary prophylaxis of P. carinii pneumonia is co-trimoxazole, 960 mg once a day or three times a week.... For treatment, first choice is high dose co-trimoxazole (100 mg/kg per day of sulphamethoxazole and 20 mg/kg per day of trimethoprim) in two or four divided doses, orally or intravenously, for 21 days."

The important point arises - how does the metabolism of a unicellular animal (protozoon) which normally just vegetates as a harmless opportunist in the undamaged environment of a lung differ from the metabolism of a unicellular fungus - an external "recycling specialist" - which, even in "immune-suppressed patients" apparently thrives only when suitable growth conditions are present in the lung? Miller, unsurprisingly, is silent on that question.

Another question is who or what is responsible for the substrate, the suitable growth conditions, for P. carinii in the lung? "HIV"? The patient? Or his treating doctors?

The imaginary retrovirus "HIV" or a shortage of CD4 cells (allegedly massacred by "HIV") cannot be decisive for creating the special environment in the lung which enables P. carinii to multiply freely. Miller himself observes that:

> "many immunosuppressed HIV-positive patients do not have in the lung any P. carinii or show only traces of it."

The question arises, therefore, whether cell-mediated immune deficiency, the laboratory finding of "HIV-positive", and the production of the substrate for P. carinii could not all be traced back to a systemic change in the body's metabolism. Miller provides an important clue by mentioning that administration of corticosteroids to rats can provoke PCP. Experiments of this kind date back to the 1950s which Miller does not mention, after what was later called PCP was first recognised in the 1930s in premature babies. Similar symptoms of atypical non-bacterial pneumonia (as opposed to typical bacterial pneumonia) were diagnosed in the 1940s in children and adults in famine conditions. So, what do the steroid-treated rats, the premature babies and the starving children after the Second World War have in common? (Note: PCP was at the time practically unknown in the United States).

The premature babies hardly stood a chance before modern treatments came along. They suffered from their immature lung cells a highly acute oxidative stress which in turn led to massive hypercortisolism. They mostly died from bacterial infections. These could be controlled more successfully after the introduction of the first broad-spectrum sulphonamide, prontosil, at the end of the 1930s. But then they died instead from PCP. Although the sulphonamide, which is a folic acid antagonist, [i.e. prevents the building of folic acid] successfully halted the production of bacterial proteins and hence bacterial reproduction itself, at the same time they raised the catabolic stress [see footnote]. Because the necessary maturation of CD4 lymphocytes (T-cells) in the thymus gland is very susceptible to hypercortisolism and systemic oxidative stress, the task of T-lymphocytes to dispose of the extremely increased turnover of cells became practically impossible: and the resulting decomposition products of the catabolic metabolism especially in the lungs which are particularly susceptible to oxidative stress, built the special conditions for the ubiquitous airborne spores of P. carinii to thrive in.

These rather complex pa/ho-physiological processes (unknown about, of course, in the 1930s) would also explain the PCP seen in rats which had been treated with corticosteroids while under antibiotic treatment.

Hypercortisolism induces a characteristic 'famine metabolism' which leads to complicated systemic changes in growth and decay of the body at the molecular level, and provides the substrate for the highly specialised Pneumocystis fungi to grow on.

If this vital emergency condition, under constant stress factors becomes a fixed lasting condition, as in the starving children of post-war Europe or in parts of Africa today, thymus-dependent cells (T-cells) decrease. In the normal course of events these T-cells have to get rid of 1012 spent body cells a day: by halting the maturation of T-cells the table becomes richly set for P. carinii and other microbes to thrive. These unwelcome scroungers can only be chased away from this paradise of theirs, by abolishing the fixed emergency conditions that created it. This explanation is confirmed by the animal experiments quoted by Dr. Miller - 75% of P. carinii were found to have been disposed of within one year of ending the artificially induced hypercortisolism.

So, what could Dr. Miller have learned from this brief glance into the history of PCP to benefit his patients, ostensibly stricken with "HIV-associated respiratory diseases"? First, that PCP and the fungal pneumonias could thrive very happily before AIDS came along - and long before any hypothesised retrovirus (which is not supposed to have existed before 1978) could have been involved - under the systemic environmental changes in the lung due to excessive situations of oxidative stress under a persistent catabolic level of metabolism.

Secondly, Dr. Miller could have learned that the common factor between the phenomena called "CD4 cell immunodeficiency", P. carinii growth conditions, and the laboratory finding of "HIV positive" can be found in the fact of excessive forced oxidative stress.

The construction rules of the "anti-HIV antibody test" lead also to this conclusion. Dr. Gallo and his colleagues brewed their test soup from already overstimulated CD4 lymphocytes obtained mainly from the serum of PCP patients as well as from cells of a particularly division-prone leukaemia cell line, spiced this with powerful oxidising agents, called mitogens, added a generous dash of hydrocortisone, and incubated it thoroughly. They then fished out of this brew a mixture of proteins which they ascribed to a hypothetical retrovirus, HIV. It follows that these proteins (antigens), released under the oxidative stress in the test-tube, will necessarily bind to their complementary proteins (antibodies) from the serum of patients who had themselves, due to pathophysiological processes, formed proteins analogous to the test antigens from Gallo's brew. Antibodies found in HIV-positives are therefore to be seen as nothing other than increased levels of auto-antibodies against endogenous proteins which have been produced as a result of highly increased cell-turnover under chronic oxidative stress.

Thirdly, Dr. Miller could have learned from all these findings that these laboratory artefacts known as "HIV-positives", represent anything but the presence of a transmissible

mass epidemic due to semen and blood.

The annual incidence of the false diagnosis "HIV-associated diseases" for the whole population of Germany is 0.002%. This result contradicts eloquently the absurd apocalyptic predictions of AIDS doctors. The official annual rate of new infections in the general population in Germany of the clinical misdiagnosis "HIV associated P. carinii pneumonia" is practically 0.00%, and among gays amounts to just about 0.05%. This true rate, differing starkly from the predicted rate, is well within the range of other epidemiological burdens of other population groups, ea. the annual incidence of lung cancer in all smokers is 0.1%. On the other hand, the annual rate of miscarriages due to folic acid shortage in mothers is also around 0.1% (and is strikingly close to the incidence of folic acid inhibition following medication with co-trimoxazole, of which more below.)

Medical Treatment

Perhaps the most important lesson for survival of those affected is the question: what is the effect of the medical treatment on the development and course of "HIV-associated" P. carinii pneumonia.

Dr. Miller is quite revealing in two respects of this without seemingly being aware of the consequences this entails. First, he is perplexed:

> "despite the widespread introduction of effective primary and secondary prophylaxis, P. carinii pneumonia remains a common respiratory pathogen in individuals with AIDS and continues to account for almost half of all respiratory episodes."

Without in any way justifying the alleged efficacy of his primary and secondary prophylaxis, he defines his preferred mixture of co-trimoxazole with seemingly precise milligram amounts as a multipurpose-weapon prophylaxis against prokaryotic and eukaryotic unicellular life forms in three domains of life at the same time: against fungi (including P. carinii pneumonia), protozoa (including toxoplasmosis gondii) and "bacterial infections."

> "The regimen of first choice for primary and secondary prophylaxis of P. carinii pneumonia is co-trimoxazole 960 mg once a day or three times a week; this may also afford some protection against bacterial infections and against reactivation of cerebral toxoplasmosis. "

The curious reader also gets to learn from Dr. Miller, 15 years after being first reported by the CDC (June 1981) about the failure of treating homosexuals with co-trimoxazole who had PCP (two out of five died), but nothing of the mechanism of this chemotherapeutic agent (often wrongly prescribed as an 'antibiotic').

Co-trimoxazole (better known under its trade names Bactrim and Septrin) contains a combination of sulphamethoxazole, a sulphonamide, and trimethoprim, a cytostatic agent which is also used to treat leukaemia in the same form, ie. to destroy white blood cells! Sulphamethoxazole inhibits the synthesis of folic acid which is essential to life, by substituting the pare-amino-benzene (PABA) moiety, so that the enzyme responsible for folic acid synthesis is consequently blocked.

Trimethoprim inhibits conversion of folic acid into the biologically active form of tetrahydrofolate by blocking the enzyme dihydrofolate reductase. Without tetrahydrofolate, essential precursors for new DNA cannot be synthesised. For example, the nucleoside uridine has to be methylated by methylentetrahydrofolate to form the essential DNA building block, thymidine triphosphate (TTP). This is the same component that is displaced with the notorious cell poison, azido-thymidine, better known as AZT, Zidovudine or Retrovir. Co-trimaxazole, therefore, works in a different way, but with a similar result to AZT, as a DNA blocker!

The consequences of inhibiting essential metabolic pathways for growth, cell differentiation and division are fatal. The synthesis of essential nucleic acids, proteins and

enzymes develops faultily, or ends completely.

Cell Damage

This treatment with combined trimethoprim/sulphamethoxazole (=co-trimoxazole) is especially serious for the functioning and fine structure of mitochondria in nucleated (eukaryotic) unicellular and multicellular species (protozoa, fungi, plants, animals, humans). Mitochondria - so-called organelles - are the major suppliers of energy in human cells (except in red blood cells). They are endosymbionts (former bacteria with a double membrane). They contain remnants of their ancestral genome. This mitochondrial DNA (mtDNA) is irreplaceable in the synthesis of protein sub-components of the respiratory chain. For respiration, activated electrons in the respiratory chain from nutrients using oxygen are built into the universal energy source for the entire cell, adenosine triphosphate (ATP).

If the synthesis of precursors of DNA is harmed through chronic or high dose treatment with co-trimoxazole the mitochondrial DNA is damaged and altered which in consequence impairs mitochondrial proteins, as well as the proteins of the respiratory chain, and ATP production therefore decreases. This leads to increased oxidative stress and to an increase in toxic oxygen free radicals. A vicious circle is set up once the ATP levels reach a critical low, and if the special molecules which normally remove harmful oxygen intermediaries are all used up, then further DNA damage arises. The cell initiates programmed cell death, because the ion pumps which regulate the balance of the flow of manifold molecules of building supplies and working materials into and out of the cell necessary to maintain cell function, fail for lack of fuel in the form of ATP.

Incidentally, DNA blockers such as co-trimoxazole and AZT, fundamentally damage predominantly the mitochondrial DNA, because the mitochondria cannot repair any mismatches or breaks in their DNA unlike the much longer and specially protected double-stranded DNA in the cell nucleus which can. DNA in the nucleus is passed on by sexual reproduction and is recombined, whereas mitochondrial DNAis propagated asexually through the maternal egg cell, which means that mutation errors are not corrected but conserved. The mutation rate of mitochondrial DNA is 5-10 times higher than for nuclear DNA. The nuclear DNA, being surrounded by its own membrane is physically better shielded, as well as benefiting from protective proteins and enzymes from any damaging effects of the metabolism than is mitochondrial DNA, which in bacteria is scattered loose throughout the cell plasma, and in several copies.

The above basic facts of cell biology apply, of course, with greatest force in rapidly maturing cells with short half-lives, especially the thymus-matured lymphocytes (T-cells), whose job is not only to recognise and, with the help of other immune cells eliminate, foreign proteins, but also to remove altered selfproteins without causing inflammation. If this cannot be done adequately because of infectious, toxic, nutritional, psychological or other overload, the body enters a state of emergency: the B-cell system is stimulated to produce antibodies and autoantibodies as well as macrophages and many inflammatory mediators and the entire metabolism is transformed. Over the short term, the body can deal with such a state of emergency. If this state persists, however, a chronic maturation deficit of T-lymphocytes (T-helper cell deficiency) arises, and the now permanently changed environment becomes the feeding ground (substrate) for the recycling activity of fungal parasites (in Greek, parasite means unwelcome scrounger) and as a consequence of B-cell activation, specific autoantibody profiles make the "anti-HIV antibody test" turn positive, just as in some autoimmune diseases such as rheumatoid arthritis and lupus erythematosus.

Under these conditions of highly acute state of emergency such as is found in "immune-suppressed patients," it does not require the wisdom of Solomon to see that the treatment methods of Dr. Miller and his colleagues will induce precisely that which they seek to avoid, namely, an Acquired Immune Deficiency Syndrome (AIDS) induced through wrong medical practice.

Why does Miller apparently not know anything of the special vulnerability of mitochondria to co-trimaxazole? Does he not know that in wanting to stop fungi, protozoa and bacteria prophylactically, he simultaneously attacks the driving force of all body cells, the

mitochondria. as well. since they are former bacteria themselves?

Dr. Miller comments truefully:

> "as many as 25% of patients receiving prophylactic co-trimoxazole develop adverse drug reactions ... 50% of patients receiving treatment doses likewise develop adverse reactions."

But Dr. Miller describes only the massive "side-effects" of short term therapy, though he appears to be genuinely surprised by their intensity in "HIV-associated diseases."

> "There is no clear explanation for such a pronounced increase in adverse reactions to co-trimoxazole which is about 20% greater than seen in the general population."

It is a pity that he does not give some thought to the long-term use in prophylaxis of folic acid inhibitors like co-trimoxazole (let alone in combination with AZT (zidovudine), ddC (zalcitabine) ddI (didanosine), D4T (stavudine), 3TC (2'-deoxy-3'-thiacytidine), the newer protease inhibitors (saquinavir, ritonavir, indinavir, neltinavir) or the newest non-nucleoside reverse transcriptase inhibitors (delavidine, nevirapine and others.)

Are not the long-term damaging effects of using combined folic acid inhibitors really the major cause and not the consequence of what Dr. Miller and colleagues perceive to be "HIV associated disease?"

Drug History

Co-trimoxazole was brought into clinical use in the early 1970s, ie. more than 20 years ago. Individually, sulphamethoxazole and trimethoprim inhibited the growth of pathogens only, whereas both together as co-trimoxazole killed off a wide range of microbes.

This was of great significance in the treatment of multiple infections of a minority of homosexuals in the large Western metropols. The purpose of treatment in most cases was simply to suppress as quickly as possible the wide spectrum of microbial growth encountered. Co-trimoxazole quickly became the wonder drug with specialist doctors and their homosexual patients in Western metropols; this double-action folic acid inhibitor was used not only to treat but to forestall (incredibly, often by selfadministration), unthinkingly, in exactly the same way as nowadays Dr. Miller and his colleagues do, too - moreover, in far too high doses and for far too long a time - especially against the often refractory urinary tract and intestinal infections, and atypical pneumonias in this group of patients.

A literature search since 1970 does not come up with a single publication about the "side-effects" of co-trimoxazole on the functioning and fine structure of mitochondria, nor on the connection between T-cell deficiency itself and co-trimoxazole; yet there has always been ample evidence in the literature for the damage caused to all white blood cells (including lymphocytes) in various groups of patients, even during short-term use of co-trimoxazole! The only investigation of up to 45 days after beginning treatment with co-trimoxazole was conducted in Britain by the General Practice Research Group in 1988-93. Since folic acid reserves in man can last up to 4-5 months, significant damage to patients with "HIV-associated infectious diseases" often manifests itself only after long-term prophylactic use exceeding eight weeks, which is then interpreted as AIDS symptoms.

Surprisingly, until the appearance of AIDS in 1981 there were no reports in the medical or pharmaceutical press about the damaging effects of co-trimoxazole use amongst homosexuals, although the "side effects" in this group should have stood out like a sore thumb, because of the high dosages and duration of treatment. The matter was obviously declared a taboo subject until P. carinii fungi as a recycling agent began to run amok in the lungs of these patients, sometimes after they could not be controlled even with high doses of co-trimoxazole (as was first reported by the CDC as long ago as June 1981).

Instead of at least by then asking themselves what the damaging consequences of

chemotherapy in this group might be, all those concerned indulged in a fit of collective mental repression regarding the interpretation of the symptoms they were witnessing, which later on became rationalised as a "new lethal syndrome due to a new pathogen."

Egged on by a prurient media relishing plague fantasies, the medical establishment transformed a set of new and old symptoms into an apparently uniform disease process using the codes "AIDS-related processes" and "HIV-associated diseases" which supposedly resulted as a wide chain-reaction of the primary effect, a virus invasion, which anyone could catch through sex and blood.

This interpretation had the tremendous advantage over more mundane explanations that neither the doctor nor the patient had to question their own role in the dynamic of the case history; the new syndrome started, so to speak, a-historically. The pharmaceutical industry could exploit the ensuing fear of death with impunity, instead of initiating a fundamental reappraisal of unphysiological chemotherapy and treatment, involving the testing and use of ever more powerful combinations of highly toxic mixtures on a global scale, financed by all of us, in a seemingly heroic battle against a "plague threatening humankind."

The world's largest manufacturer of co-trimoxazole has meanwhile confirmed in writing that there have never been any investigations into the effect of folic acid antagonists on mitochondrial integrity. Strangely enough though, the indications (recommended circumstances under which to prescribe a drug) at least in Britain and America for co-trimoxazole, have been severely reduced, because of frequent side effects during short term use (cf. BNF, FDA guidelines). Excepted from this change: special indications of prophylactic and therapeutic high doses and longterm use and in frequent intermittent therapy (itself a long-term use because of cumulative damage) were expressly permitted for "HIV-infected" patients and "PWAs".

In this light, the concerned statement of Dr. Miller:

> "despite the widespread introduction of effective primary and secondary prophylaxis, P. carinii pneumonia remains a common respiratory pathogen in individuals with AIDS"

becomes a self-fulfilling prophecy.

In the early 1980s the use of co-trimoxazole had reached the very high annual incidence of 5% of the population, about equal to that of alcoholism, while use of co-trimoxazole by homosexuals in Western countries (the taboo subject), in particular in metropols and in the neighbourhoods of specialised practices and clinics must have likely been, and continue to be, considerably higher. It was recommended to restrict the general indication of the drug, because of the high level of damage to blood cells, (including lymphocytes) while, gruesome to relate, the prophylactic and therapeutic indication for already immune-suppressed patients, stigmatised as "HIV infected" and "PWAs", were relaxed.

If the number of CD4 cells necessarily declines because of DNA blockage and mitochondrial damage through co-trimoxazole (because of increased cell death and inhibition of maturation) then "AIDS" will be diagnosed, and the range of DNA blockers and mitochondria killers constantly enlarged. The blind zeal of virus hunters leads them to use ever more frantic chemistry without, as Dr. Miller shows, ever stopping to think about the vital basic conditions of the intertwined biospheres of our bodies. The patient will go through all the stages of AIDS described in textbooks, and at the end of it, all the participants will feel bitter at having lost the battle, at great sacrifice against a fickle enemy, despite calling on all means available; the patient will have dutifully suffered a ritual death for the sake of a plague-hungry society; the frustration of the doctors and their companions in death will have been transformed into aggression against those who have insisted all along on a genuine re-evaluation of their actions.

Pre-existing Immune Deficiency

If the premise of an inexplicable immune deficiency affecting hitherto completely healthy individuals had turned out to be true, then the virus-AIDS theory could have been a

reasonable working hypothesis. But because AIDS (according to the official CDC explanation) is supposed to be a serious disease of acquired immune deficiency without pre-existing or induced immune deficiency, it has to be stated quite unequivocally that such AIDS cases have never been found up till now, except in the form of a medical mantra of plague propaganda, because in all verifiable cases, demonstrable immune-suppressive disease and/or treatment have always preceded them.

There has never been a need, therefore, to explain an inexplicable immune deficiency, because the causes were there for all to see. And a new virus was entirely redundant for an understanding of the disease process, irrespective of whether the suggested retrovirus HIV existed or not. For example, the annual incidence in Germany of AIDS in the population as a whole is just 0.002%, and amongst homosexuals 0.1%. Intriguingly, more than 60% of all "AIDS-cases" in a population of 80 million occur in the immediate vicinity of six large university clinics in six towns, which rather supports the view that AIDS should be called an "Acquired Iatrogenic Death Syndrome."

Furthermore, according to Dr. Jager (in a live interview), one of the leading German AIDS-authorities, (President of the Curatorium for Immunodeficiency, Munich) in the period from the ostensible beginning in 1981 to 1996, there has not been a single case of male or female HIV infection in the age group 14-20 (not even in homosexuals!), although every school kid has had the exact opposite drummed into him, which is proof that the advocates of HIV/AIDS are by now unwilling to separate fact from fiction, even for the sake of the patients entrusted to their care.

Plague Mania

Dr. Miller really should have another look at Virchow's writings in order to understand why TB had practically lost all its horrors in Western Europe without use of chemotherapy, and why now, in his own words:

> "multiple drug resistant (MDR) tuberculosis has emerged as an important clinical problem in HIV-infected patients in the USA."

Dr. Miller fails to mention, however, that chemotherapy for mycobacterium tuberculosis and mycobacterium avium intracellulare (No's 6 & 7 in his list of "HIV-associated infectious diseases") also require high levels of folic acid, thereby inducing a relative shortage of folic acid, ideal conditions for DNA mutation to occur. In Africa by the way, in a different mycobacterial disease, leprosy, the anti-HIV antibody test reacts positive as well.

Because African AIDS (apparently 90% of all AIDS cases world-wide) is as a rule nothing other than old well-known clinical conditions such as TB, malaria, hepatitis, diseases caused by worms and 'slim', (all the result of poverty, hunger and inadequate hygiene, in the sense that Virchow meant), there the "HIV associated respiratory tuberculosis" in Dr. Miller's terminology, turns out to be just plague-mongering. As Dr. Miller rightly says:

> "TB is a potent stimulator of cell-mediated immunity"

but he doesn't say that if the store of immunity is constantly being used up, and, for all the reasons explained above not properly replenished, then a deficit does indeed arise, resulting in an "Acquired Immune Deficiency Syndrome". However, even in such cases, immune-suppressive disease and/or treatment have preceded the appearance of the syndrome; it is not a case of independent AIDS in the CDC sense, which requires an explanation based on "HIV association". The HIV association is, as far as this elementary disease process is concerned, exactly the reverse a life-threatening "connection in the mind" which has led to a collective plague mania, which has completely lost sight of the true causes of disease.

Anniversary

It might be appropriate to mention that the German medical profession these days is

commemorating the 50th anniversary of the Nuremberg Doctors' Trials, during which doctors stood accused of crimes committed under the Nazis. Amongst other things one can see in the transcripts that experiments were performed on concentration camp inmates including homosexuals using sulphonamides to treat infectious diseases that had been induced specially for this purpose. Sulphonamides were used knowingly to cause death in innocent victims. Nowadays the chemical cudgel of sulphonamide + trimethoprim (=co-trimoxazole) + AZT, etc. etc., is hurled about (of course, only with best intentions and not to be compared with the crimes of doctors in Nazi times).

The lethal effect of AZT on mitochondria has by now been amply proved; an analogous effect of long-term use of co-trimoxazole is equally plausible. Does it not occur to anyone, bearing the Nuremberg Trials in mind, that it is high time to discuss the ethical consequences of the "virtual medicine" currently practiced, which under the presence of an imagined globe] epidemic, force-feeds highly toxic drug cocktails to terrorised patients, on the basis of a laboratory artefact.

What Dr. Miller and colleagues clearly find hardest to do, is to question their own doing. Anyone who uses co-trimoxazole, AZT etc. etc., which we know very well induce immune deficiency in the long run, to combat immune deficiency, is put in the same position as someone in the 19th century prescribing blood-letting to treat anaemia, knowing full well that millions perished as a result of that treatment. Just 10 days' use of co-trimoxazole can, as has been observed in some instances, result in anaemia. Nonetheless, Dr. Miller and colleagues continue to advocate co-trimoxazole as the "prophylactic treatment" of first choice - until the patient dies.

Since the life-threatening consequences of this long term iatrogenic intoxication with co-trimoxazole, in complete analogy to the no-longer-to-be-denied consequences of long-term AZT use, are not ascribed anymore to the Co-trimoxazole-treatment itself (alone or in combination with AZT) but are misinterpreted as the predicted "HIV-associated infectious diseases" and "AIDS-related processes", Dr. Miller arrives at the unbelievable [suggest: breath" taking] conclusion:

> "in patients with adverse reactions, desensitisation to co-trimoxazole is successful in as many as 80% of cases."

What Dr. Miller does not say is that mortality due to PCP, as a fraction of the total number of AIDS deaths in the Western world is despite (or because of?) medication with co-trimoxazole also 80%!

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