



# **TB - the Burden that Lasts a Lifetime**

**by Dr. Konrad Werthmann**



Tuberculosis (TB) is an archaic epidemic that occurs all over the world and has existed ever since mammals (warm blooded creatures and humans) came into being. In 1882, Robert Koch discovered that the *Mycobacterium tuberculosis* was the trigger for the epidemic, also known as the „white plague“.

Eva Medina, a biologist at the Society for Biotechnological Research [*Gesellschaft für Biotechnologische Forschung, GBF*] said recently in a press interview that „about a third of the world’s population are carriers of TB pathogens“. Therefore, in the future, too, TB will continue to play a significant role in day-to-day medicine and the way we treat „infections“.

No therapist working according to holistic principles can ignore TB. In order to be able to recognise it in each of its forms, the therapist requires a different perspective from what was formerly expected. At the present time, it is regarded by many doctors and lay people as a disease which has „died out“ nowadays or is subdivided into the diseases of many organs. On the contrary, for a number of years it has been looming larger with a steady increase in the number of multi-resistant tuberculosis pathogens. This is confirmed by both the DZK (German Central Committee for the Control of TB) and the Reference Centre for Mycobacteria. In the years between 1960 and 1980 – i.e. in the period of upswing and of positive attitudes to medical successes – the opinion that TB had been vanquished was a common one and certainly partly justified.

Today, 20-30 years on, it is the opinion of myself and of the experts mentioned above that we need to modify our position; anything else is fallacy.

### **The background to TB**

There are many layers to the background to TB, and everyone has their own opinion according to their medical bias and attitude to treatment. In order to give an exact picture of TB and its progression, I am quite consciously resorting to a book which describes this disease very exactly and nevertheless may still be classed as modern. Trendelenburg and Forschbach (T&F) describe tuberculosis and its affliction of the individual organs in great detail, also giving me the opportunity to utilise and compare facts from the standpoint of 1977. The possibilities for practical treatment and treatment by medication will also not be ignored. T&F define tuberculosis in the following way (1977): „*Tuberculosis plays a leading role among the infectious diseases in human beings which are caused by mycobacteria. In its chronic form, it progresses in a series of attacks and very often also alongside life events. Phases of acute or slow progression, regression and arrest alternate with or proceed alongside one another in one or more organs at the same time. Treatment by medication or surgery improves the course of the disease and has a crucial effect upon its prognosis, enabling recurrent prophylaxis and a clinical cure.*“

Initially, one must be acquainted with the different pathogens and

their frequency. Here, most notably it is an important fact that infections as a result of mycobacteria can have many faces, such as afflicting failed organs or as a stand-alone illness (e.g. as leprosy). However, TB plays a leading role – particularly as tuberculosis of the lungs – among the human infectious diseases known in Europe which are caused by mycobacteria. If people speak of TB today, in both professional and lay people’s language, what they generally mean is TB of the lungs (pulmonary TB). But in principle, any organ (in the extra-pulmonary form) can be affected by TB, including the peritoneum, eyes, ears and even the brain (tuberculoma/ aspergilloma).

The tuberculosis pathogens are immobile forms of mycobacteria which live aerobically and are distinguished by a high lipid level and the resulting acid resistance. They are affected by neither the hydrochloric acid of the stomach nor temperatures of  $-70^{\circ}\text{C}$ . *Mycobacterium tuberculosis* (human type) is regarded as the most common pathogen and *Mycobacterium bovis* (bovine type) as the least common.

The so-called „atypical mycobacteria“ include among others *Mycobacterium avium*. This is cited only in cases of tuberculosis of the bones and joints, where it is thought that infection has entered via the lymph or blood, with possible preliminary damage to the lungs. Atypical mycobacteria show geographical differences. It would currently appear to be the case that *Mycobacterium avium* is known as a cause of TB in the south and



south-west of Germany and *M. kansasii* more so in the western part of Central Germany. Poultry and pigs are regarded as sources of infection with *M. avium* and fishponds, aquaria and milk as sources of infection with *M. kansasii*.

Leprosy is a particular manifestation. It is true that it does not affect Europe directly, but a large number of people suffer from this dermal mycobacteriosis. It is a chronic granulomatous infectious disease caused by *Mycobacterium leprae*, and despite the high doses of antibiotics given in treatment, the danger of infection is still great. Above all, people with latent leprosy do not register as a result of the social consequences and are a source of contagion, particularly when they work in the food trade. The customers of such leprosy patients know this and yet still buy their food from these people.

The frequency of TB is interesting, as it varies from country to country. 200–300 people per 100,000 head of population still contract the disease today in parts of Africa, Asia and Oceania. In Europe, on the other hand, the figure is 20–30 per 100,000. The death rate from TB of foreigners in the population is considerably higher than that of Germans. In terms of epidemiology, T/F regard droplet infection by one person of another as the most important opportunity for the mycobacteria to be passed on. In 1977, it would appear that the digestive tract no longer plays a part as the site of initial infection through infected milk, following the reduction of

bovine infection to 0.99% as a result of the decontamination of beef cattle stocks back in 1965. According to the latest reports, however, TB in Germany has by no means been conquered – a fact which became obvious again only a short time ago with an outbreak of the disease in a herd of cattle in the Gifhorn district.

#### **Factors in the disease**

Opinions at this time about the factors involved in the development of the disease are remarkable and interesting, with the number, virulence, type and resistance to medication on the part of the pathogens playing an important role in the progression of the disease. In the host organism hereditary factors are said to be responsible for the development of tuberculosis, with references made to observations of Africans, Indians and Eskimos. Hormonal factors are blamed for the lack of defence against TB in infants and old people. Thyroid and pancreatic disorders also work in favour of tuberculous diseases. Particular attention is paid to the prescription of steroids, as these activate dormant foci. Social moments and environmental factors are also blamed. But these are all truisms.

In my opinion, the Head of Biology at the Berlin Charité University, **Prof. Dr. Günther Enderlein (d. 1968)** was someone who had a great appreciation of the internal interrelationships in tuberculosis. Around the turn of the century, he made several discoveries in human vital blood under the darkfield microscope, which for his time were unbelievable, even sensational.

Although modern biology already recognises many parts of these paradigms as factors which exist, they are still known all too little. In contrast to Enderlein's fundamental knowledge, the assertions made by Trendelenburg and Forschbach only make the opinions of their time clear. One has to say that the way of thinking of someone like Professor Enderlein or of isopathy can only be identified in minimal approaches, although these insights were already over 60 years old at the time the assertions were made. In the following section, I first want to give some facts about TB as seen by Prof. Enderlein and then talk about my experiences with TB in my own practice.

#### **Enderlein's discoveries about tuberculosis**

Enderlein's discoveries are relatively unknown as there are not even any modern publications. Despite this, Professor Enderlein's recommendations and stipulations for therapy should not be ignored.

The following facts are the most important of his conclusions:

##### **1) The milieu is everything**

It determines which pathogens can grow in the carrier or in the individual organs. Different diseases and how strong they are depend on the person's milieu. Enderlein came to this conclusion on the basis of research by the French scientists Béchamp and Bernard.

This idea can be explained using two examples: paediatric experience shows that varicella is

absolutely contagious. This means that anyone who has not yet had varicella and is in close contact with someone who is suffering from this disease will „infect“ themselves. My older daughter caught chickenpox; my wife, who had never had varicella, and our younger daughter remained free of the disease. The following year, the younger one caught varicella, and shortly afterwards so did my wife. This means that the milieu in the body of my wife and our younger daughter was not suitable for the varicella pathogens at the time my older daughter developed varicella. Another example comes from the laboratory: minor changes to the pH or composition of the culture medium in a Petri dish will allow other bacteria to grow than those desired. Each strain of bacteria requires its own specific conditions.

Of course, the milieu depends on many individual factors, such as the dynamic conditions (see also Schneider, P.: „Hierarchische Multi-Regulation“ [„Hierarchical multi-regulation“], SANUM-Post no. 58, pp. 17-23), the lack or overdose of certain parameters (e.g. pH, lack of minerals, stress, heavy metals, too high a level of protein or carbohydrate in the blood, the remote effects of a focus via meridians or psychological factors). Now, one must know that many functions of archaic microbes or their particles in the body are dependent on their local internal milieu. The more pathological

or abnormal the milieu is, the more pathogenic these microbes become in the way they work or the more pathological their function will turn out. Such conditions can be observed very clearly in the microbes which from time immemorial have been taking over those functions within the human body which are important to life.

The microbe *Mucor racemosus* was described by Enderlein as „the endobiont“ as it is to be found in every cell in the human body – including blood cells. In the course of evolution, this microbe has taken over the function of blood coagulation, and at the same time, is responsible for problems in the blood vessels. In a drop of vital blood under the darkfield microscope the endobiont is seen in line with the conditions described above either as a physiological part of the cyclogeny (apathogenic protite, symprotite or – in the modern way of saying things – nanobacteria) or as pathogenic forms (bacterial and viral stages) and/or as a preliminary stage of the fungus *Mucor racemosus*.

The equally important cyclogeny of *Aspergillus niger* offers a similar relationship. The upward and downward developments in the cyclogeny of *Aspergillus niger* can – like the classification of *Mucor racemosus* – also be observed as physiological particles, as nanobacteria and/or also as a preliminary stage of the fungus *Aspergillus niger*. The

upward development of the *Aspergillus niger* cyclogeny is the **tubercle (Koch's) bacillus**. Indeed, one needs to know that the two microbes *Mucor racemosus* and *Aspergillus niger* are always present in every human being and warm-blooded mammal. *Aspergillus niger* is also responsible for the function of the cell membrane and the transfer of particles from the extracellular space into the intracellular space and *vice versa*.

From my experience, the isopathic remedies which influence the cyclogeny of *Aspergillus niger* are of help – particularly in diseases of the intestinal tract, skin, respiratory tract, lymphatic vessels and nodes, urinogenital tract, bones and joints, CNS and brain. For this reason, it is my opinion that an aspergilloma is also a tuberculin disease.

**2) Basic apathogenic forms can reduce the pathogenicity of so-called „upward developments“, i.e. pathogenic forms**  
This, Enderlein's second discovery, is something totally new and incomprehensible to therapists who deal in the destruction of bacteria. Innumerable therapeutical successes mean that we cannot repudiate this fact of isotherapy. It can, however, also be proved by using smears of vital blood in darkfield microscopy.

Thus, one only needs basic forms of the particular cyclogeny to reduce the pathogenicity and at

the same time modify the milieu conditions.

In the following points, I want to address modern experiences with the facts influencing the milieu as well as Enderlein's findings. First, however, it must be made clear that cyclogeny means the upward and downward movement of the development phases. It is therefore possible for someone to develop a tubercle bacillus from the aspergillus cyclogeny within his body several times a day without falling ill, as the pathogenicity of this bacillus is reduced again and again by apathogenic forms, so long as the milieu is right!

A change in eating habits or the increased frivolous abuse of medications will result in a milieu which is favourable to bacteria (i.e. abnormal), and in the long term, this can lead to „tuberculin or aspergillic weakness“.

### **Tuberculin weakness**

From the point of view of the practitioner, all the systems named above are particularly susceptible to TB. Because of the similarity of its progression to that of TB, this susceptibility is called a „tuberculin or aspergillic weakness“. This means that the disease is not contagious like TB but is similar in the way it develops.

### Chronicity:

The description (by T/F) of the progression of TB also fits exactly with the description of any other chronic illness. A chronic illness imitates the progression of a TB illness but without having its infectious nature – or, expressed

in different terms: as a therapist faced with a chronic illness, one thinks immediately of tuberculin weakness. In conclusion, it can therefore be claimed that the chronicity of diseases is influenced by *Aspergillus niger* and/or that **chronicity represents a tuberculin weakness**. This too, is initially described very well by T/F. In their formulation, it is possible to see the chronic illness clearly. T/F say „In its chronic form, it progresses in a series of attacks and very often also alongside life events. Phases of acute or slow progression, regression and arrest alternate with or proceed alongside one another in one or more organs at the same time.“ Here, it is of no matter whether the chronicity depends on one focus or on CWD (cell wall deficient) micro-organisms, is supported by a deficiency syndrome or is caused by poor nutrition.

In this context, it is necessary to point out that cell wall deficient forms are the original pathogens and initiate a tuberculin reaction and generally severe symptoms of the disease. These dud cells without any immunological markers, in part also so-called partial antigens, move around inside the body and likewise are capable of triggering chronic diseases. These are old phenomena, in which the modern medical/biological view needs to renew its interest.

Leprosy is a good example of this. One feature showed itself even at an early stage in the development of this protracted disease: the frequently repeated occurrence of so-called leprosy reactions. These

are understood to be an acute inflammatory phase in or outside existing granulomatous foci and according to *Klingmüller* are activated by bacterial toxins. This may be true, but we must also consider the other possibility – that these are the consequences of CWD, formed as a result of the high doses of chemotherapy which were tried out very early on in the era of antibiotics. These leprosy reactions are accompanied by fairly severe acute symptoms or activations and consequent worsening. *Klingmüller* („*Innere Medizin*“ [„*Internal medicine*“] in *Praxis und Klinik*, Georg Thieme Verlag, special edition, 2<sup>nd</sup> impression, 1977) calls this a **tuberculoid** reaction which with the emergence of chemotherapy has become very significant. The word „tuberculoid“ implies both a similarity in the progression and a lack of contagiousness. These facts again prove the nature of *Aspergillus niger* and the way in which it is able to bring into line the diseases initiated by itself and/or its neighbouring organs in accordance with its properties.

### **Components which are ostensibly not recognised and yet can support chronicity**

*Mucosa enteralis* and the immunoglobulin A (IgA) which it secretes are always also responsible for the development of disease. For human beings, the intestinal mucous membrane is a central organ because of the many and varied functions of IgA in the immunology. The IgA secretion becomes noticeably less as soon as enteral allergic processes destroy the *mucosa enteralis* partially or



incompletely, particularly in the first nine months of life. Only long-term total elimination of primary antigens (products made from cow's milk and hens' egg) will re-build the *mucosa* and can guarantee normal IgA secretion once again. Otherwise, the result is atrophy of the *mucosa enteralis* with consequent discomfort (cf. Werthmann, K.: „Ratgeber für Allergiker und chronisch Kranke“ [„Advice for people with allergies and chronic illnesses“], available from the Semmelweis Publishing House).

If the IgA is absent, then on the one hand proteins without IgA marking can make their way into the body's interior unchecked and important mineral salts and trace elements can no longer be absorbed. On the other hand, the mast cells can degranulate more easily, consequently the IgA is able to react to the mast cell and within the tissue unchecked. This again means a flaring up of symptoms which are contingent upon degranulation, such as bronchial asthma, neurodermatitis and colitis syndrome.

At the same time, the immunoglobulin A cements together all the surface intercellular windows of the *mucosa enteralis* and marks the intraluminal toxins and bacteria, so that as little as possible of the toxic or bacterial/viral material can penetrate into the interior of the body. Even so-called haptens structures can be repelled in this way. That is important, since antibiotics, cosmetics and immune suppressives are haptens. It is not without interest that in every case,

infestation occurs only as a result of atrophy of the intestinal mucous membrane with consequent deterioration of the bacterial lawn. The bacterial lawn is only a tool that aids the intestinal mucous membrane. One will have little success if one treats the bacterial lawn without taking into account the allergic destruction of the *mucosa enteralis*, for a considerable number of bacteria need the oxygen-rich milieu on the intestinal wall to produce important enzymes.

The intestinal milieu is thus crucial to the general milieu and for pathogenic upward developments in the *Aspergillus niger* cyclogeny. The worse the intestinal milieu, the better it is for tuberculous developments. One consequence is that as a result the tubercle bacillus coming from outside (food) can very easily penetrate the body without the body defending itself against it. Infants who are burdened with artificial milk too early suffer partial or incomplete atrophy of the intestinal mucous membrane, and old people suffer from physiological atrophy of the villi – the so-called „leaky gut syndrome“. That is the reason for greater susceptibility to infection (see T/F for comparison).

### **Cow's milk and TB**

When bovine tuberculosis was still common in Germany, people used to say that „bovine tuberculosis is like tuberculosis in children“. Today, it is worth taking note that whilst it is true that milk is no longer regarded as the major carrier of TB bacteria because of TB-free herds, other new allergens have

arisen and are arising alongside the actual cow's milk allergy as a result of the change in milk structures. Cow's milk also contains CWD forms. Looking at this critically, human beings „conjure up“ the terrain of the new antigen structures. This view is greatly responsible for the increasing tendency towards tuberculin / aspergillitic weakness. Over 40 years (!) ago, I took a tour through the dairy in Innsbruck, where it was pointed out that „nowadays“, one no longer had to reckon with a shortage in milk „production“. So, one could order the different parts of the milk from various federal states and then process them into normal milk. In this way, if no fresh milk were available, one could re-process whey from Lower Austria, milk fat from the Tyrol and the rest of the constituents of the milk into fresh (?) cow's (?) milk. Put as simply as possible: our natural milk is sold as artificially processed „fresh milk“.

Depending on the technical process, one may suppose that the processing is done even better nowadays, and that as a result, the milk being sold in shops today is even more of an artefact. Allergies are increasing, the terrain of inflammations of the intestinal mucous membrane is becoming stronger, and this results in an underlying change in the milieu and promotes the upward development of bacteria.

If we look at the infinite number of possibilities nowadays for cell wall deficient forms to develop, we should be really concerned about the tuberculin complaints of the



future. No antibiotics will be of help any more and they will even produce new forms of these microbes. One cannot even begin to imagine the scenario when a weak organ is affected by a CWD form of the tubercle bacillus: the disease is almost incurable.

We could cause this scenario to turn out rather better if we took note of two facts. First, we have to consider the immunoglobulin A (IgA) which enables antibodies to develop and thus forms a good terrain for health. Then, we need considerably fewer allopathic medications, and the result will be a lower number of cell wall deficient forms with all their consequences.

Many CWD forms must be regarded as partial antigens. As soon as a carrier is offered to the partial antigens, they become full antigens and thus are capable of being excreted by the body. Being immune absorbers, SANUKEHL remedies are able to do this. They empty the pool of partial antigens and thus remove the potential for opportunities for chronic diseases. These immune absorbers are more effective if immune complexes are present, and IgA is required for such immune complexes to form.

### Conclusion

40 years ago, people still believed that we could achieve a world free of TB. However, this hope has not come true. It is true that conventional cases of TB have become fewer in number, but tuberculosis has changed the way it looks (pleomorphism) by changing from the acute form to the chronic one. This chronic progression can take

years, does not show any positive TB reactions, often also shows no increase in the reaction to the reduction in the blood sedimentation rate (BSR), even shows a normal leucocyte count and no signs of bacteria in the usual attempts at culture. This is understandable, for the immune system and traditional bacteriology cannot prove the presence of CWD forms.

This may soon be a major problem, but I am certain that isopathy can very quickly get this phenomenon under control. In my many years of practice, familiar „open“ TB patients have returned again and again, with the rest of the family needing „anti-tuberculosis“ therapy in order to protect the milieu. They were prescribed only the remedies listed below. The patient in hospital followed this course of treatment along with them but „under our roof“. After 10 days at most, the whole family was being given the same prescription. One must assume that the average hospital stay lasts eight weeks.

This formula – always with the same basic features – and tested over more than 30 years – is simple but takes into account Enderlein’s requirements whilst combining them with modern knowledge:

- 1) MILIEU of the small intestine:  
2x ½ to 1 tsp ALKALA N in warm water daily  
of the *Aspergillus*:  
2x 10 drops CITROKEHL daily  
of the *mucosa enteralis*:  
a diet without any primary antigens (products made from cow’s milk or hens’ eggs)

- 2) Pencillium-dependent medications: at the same time as Item 1, begin with FORTAKEHL 5X (drops or tablets) or EXMYKEHL 5X drops: 2x 10-15 drops daily over 1-2 weeks, then change to
- 3) 1x 10 MUCOKEHL 5X drops in the morning and 1x 10 NIGERSAN 5X drops in the evening (or 2x 10 drops SANKOMBI 5X daily) over a number of months, but always in the pattern 5-2-5-2: that is, MUCOKEHL/NIGERSAN from Monday to Friday (5 days) and EXMYKEHL at weekends (2 days), etc.
- 4) At the same time as Item 3, begin with 1x 5 drops UTILIN „S“ 6X rubbed in daily, and at the same time, the same quantity taken orally; in addition 1x 5-10 drops SANUKEHL Myc 6X are to be taken orally or rubbed in daily.

The treatment is carried out over a period of several months. At first glance, this prescription may appear laborious and tedious. However, hospitalisation is reduced from an average of 2-3 months to 10-14 days. Within 10-14 days, chronic illnesses show a significant reduction in the intensity of the symptoms and a corresponding comeback of the functions of the relevant organs.

Please note: Blood, secretions from the joints or ulcerations without proof of the presence of pathogens in laboratory cultures at the same time of the presence



of clinical symptoms are a sure sign of cell wall deficient microorganisms as the cause of the disease.

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**First published in the German language in the SANUM-Post magazine (64/2003)**

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