Medical Research Archives

European Society of Medicine

OPEN ACCESS

Published: February 29, 2024

Citation: Maes, R., 2024. Tuberculosis is a looming next pandemy. Medical Research Archives, [online] 12(2). https://doi.org/10.18103/mra.v12i2.5036

DOI: https://doi.org/10.18103/mra.v12i2.5036

ISSN: 2375-1924

REVIEW ARTICLE

TUBERCULOSIS IS A LOOMING NEXT PANDEMY

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ABSTRACT

This review describes the mode of action of the vaccine Bacille Calmette=Guérin (BCG) and demonstrates its inefficacity. It analyses the skin test that is commonly used to verify the vaccination with the BCG and concludes that this skin test results from an awakening of a tuberculous infection. The review analyses thereafter the usefulness of a blood test that evaluates the presence of antibodies against the pathogen. This test was banned by the WHO claiming its inaccuracy, because a large load of the pathogen as demonstrated by bacilloscopy was associated with an absence or low level of antibodies, which runs counter the received belief that it must be the opposite. The WHO ignored the immunosuppressive capacities of the pathogen, which reduced to nil the level of antibodies produced by large loads of the pathogen. Tuberculosis is an immunological problem that could find a solution in an immunological treatment. Such a treatment was advocated by Stanford who recommended the injection of Mycobacterium vaccae. The failure of this treatment was traced to an absence of understanding of its mode of action. The use of a chemical that boosts the production of nitric oxide is indicated to help the tuberculous patient to recover. In the meantime, new drugs are developed (e.g. delamanid, clofazimine, bedaquiline) that prove as dangerous to use as the previous ones and need close supervision during their administration, to avoid fatal issues.

Key words: BCG (bacille Calmette=Guérin), M. vaccae, Tuberculosis, mycobacteria, immune supression.

Introduction

Today tuberculosis kills about 4000 people every day, worldwide. TB kills more people each year than HIV and malaria put together. The victims are largely invisible because they are confined in sanatoria and do not encumber regular hospitals.

This bacterium is currently neglected but may expand again.

I will first describe the vaccine BCG and the tuberculin skin-test. It is a complex and long story, and I will devote some time on it because it was a gigantic fraud and delusion at the root of the TB debacle. This will be followed by the blood test, which was rejected by the WHO, the immuno-therapy also scorned by TB-experts, and the search for new drugs, which proved disappointing.

The BCG Vaccine.

1. THE PROTECTIVE VALUE OF THE BCG

"Mr. Fougerousse is an ignorant who knows nothing about Tuberculosis. The (French) experts are honest, competent, and knowledgeable; they are there to tell the truth without dogmatism or occultation. They found that the number of (tuberculosis) cases among the French vaccinees was less than 2 per million whereas it amounted to 15 per 100.000 among not vaccinated. And the vaccine is particularly *efficacious in infants, against tuberculous meningitis".*

I read this on February 2, 1978, in the local newspaper "the Last News of the Alsace" [Les Dernières Nouvelles d'Alsace" (DNA)]. Fougerousse was a junior deputy of the mayor of Ostwald, a neighborhood of Strasbourg (France), who held in December 1977 a modest conference to his constituency, in which he dared express doubt about the value of the BCG vaccine against tuberculosis. BCG stands for Bacille Calmette Guérin, the names of the two investigators who developed the vaccine at the Pasteur Institute of Lille. That same year, J. Frimodt-Moller (1) published that the BCG was not safe, i.e. was iatrogenic.

France had imposed the BCG as sole vaccine against TB in 1950, with the active support and participation of the WHO:

Fig. 1: Poster edited by the French Ministry of Health in 1964. The caption reads: to vanquish TB is only a play, thanks to the BCG.

Fig.1. Posters and post-stamp extolling the merits of the BCG vaccine.

Contrary to what is claimed, to vanquish TB is far from being a play but is a war, and we are losing it despite the BCG, which does not protect.

Fig.2. The tubercle bacillus.

Tuberculosis and leprosy are mycobacteria. There exist at least 64 different species of mycobacteria, infecting mammals, birds, reptiles, and fishes. With billions of doses of BCG vaccine distributed over the past sixty years, with 122 countries presently covered, with 85% of the world population vaccinated, the epidemiological proof of the failure of the BCG vaccine is given.

How could this vaccine have been used for so long despite the evidence of its malfunctioning, given in 1927? Because TB regressed in the West after World War II. This improvement was traced to the policy of vaccination with the BCG and stamping the bug out with 4 drugs: isoniazid in 1952, pyrazinamide in 1954, ethambutol in 1962 and rifampicin in 1963, (plus streptomycin in 1944, for the resistant cases) given during 6 to 9 months, which was claimed necessary and sufficient to eliminate TB. All the Public Health agents in charge of TB in France and by the WHO thought it would soon be eradicated. Throughout the decennia, a flood of dithyrambic praise of the BCG muffled any question of its efficacy. No contradictory opinion was tolerated. The reservations expressed by Fougerousse were sacrilegious.

Fougerousse based his depreciative argumentation on a book published that year 1977 by Prof. Marcel Ferru, entitled: *The failure of the*

BCG, testimonies of yesterday and today (La faillite du BCG. Témoignages d'hier et d'aujourd'hui, eds: Ferru). Ferru found no editor to publish his book and resolved to publish it himself (2).

The pediatrician Ferru, who had vaccinated his own family with the BCG, went through the whole of the BCG problem, starting in 1926 with the vaccination of children by Calmette and the opposition to it by Lignières in 1927, and wrote:

Chapter 2: The rude controversy Calmette-Lignières
Chapter 5: The arrogant rejection of the critics
Chapter 6: The silent contestation
Chapter 7: The international pseudo-congress of Paris
Chapter 8: The inordinate practice of the vaccination

Chapter 9: Massive and monopolized propaganda
Chapter 10: Denatured information
Chapter 11: Oppressive dogmatism
Chapter 12: The masquerade of retaliation
Chapter 13: Back to facts

This is a violent denunciation. He warned that TB will return with a vengeance.

The regression of the tuberculous endemy grinded to a halt in France ten years later, in 1987. Pr. Grosset mentioned 10,000 new cases in 1994 (3) in France and concluded that the fight against TB was a total failure. Janet Cornwall replied tartly in 1997 that the reason of the failure was the greed and ineptness of the TB-actors, who refused to adopt the available, effective means to combat it (4).

2. THE WHO, PRINCIPAL TB-ACTOR

Tuberculosis and leprosy are age-old diseases. In the West, the industrial revolution, the creation of a derelict impoverished urban proletariat associated with an increase in population leading to promiscuity, favored the surge of TB in the 18th century. The Western nations created Work Medicine institutions whose primary vocation was to fight the disease. The WHO was founded in 1948 with a mandate to coordinate international health policy, and its prime concern in 1948 was TB.

The Constitution of the WHO stipulates that it *"shall enjoy immunity from every form of legal process"*, with the perceived need to take crucial public health tasks out of the clutches of party politics. The lofty goal of the WHO founders was adulterated forthwith by the experts they put in charge of the TB extirpation. The problem was handled by pediatrician Debré, Chairman of the UNICEF (United Nations International Children's Emergency Fund) subcommittee on medical projects and brother of the Prime Minister of France. Guided by the will of the president of the Republic Charles de Gaulle to "Make France Great Again" after the debacle of World War II, they focused their attention on the financial needs of the Pasteur Institute.

To this end, they secured the backing and huge financial support of the WHO to promote the BCG. This institution discovered by itself that the BCG vaccine generated excess TB cases among the vaccinees in Finland and Denmark (5). The French Ministry of Health, the Pasteur Institute and the WHO occulted these adverse results, voluntarily ignored the observed dysfunction, and organized a lucrative and prosecution-free gigantic fraud.

3. THE BCG FRAUD

A. Background

The incidence of tuberculosis peaked in the West between the 18th and 19th century. In Germany alone, hundreds of thousands of people died of it every year. The intense chauvinistic mentality of the western nations (Prussia had badly beaten France in 1870) led to fierce competition for the development of a diagnostic and of a treatment.

Koch and his colleagues in Berlin used the Ziehl-Neelsen stain in

1882 to observe under the microscope the presence of the tubercle bacillus

in sputum (this is referred to as "bacilloscopy" and commonly said "smear-positive" or "smear -negative"). Bacilloscopy became a standard diagnostic and remained so until today. Koch announced an additional standard diagnostic and a treatment, based

on a TB extract named "Old Tuberculin". The second standard diagnostic, -the skin test (which is still abundantly used today)-, was the intradermal inoculation of a small quantity of tuberculin. Three days later, a swelling appears at the site of inoculation if the subject had been previously infected with tuberculosis. Using Old Tuberculin as a cure, Koch made a fortune before conceding that the Old Tuberculin awakened dormant tuberculosis. The competition for a vaccine remained open.

B. Vaccines

An American killed TB vaccine applied in Jamaica in 1939 and 1944 was abandoned for reasons I ignore, just as was a British vaccine based on a vole mycobacterium (*M. microti*). Friedmann, an Austrian Jew, developed a vaccine based on *M. chelonae,* which was satisfactory. It was recognized by Germany in 1919 and Italy in 1922 but repudiated by Hitler and Mussolini after 1933. Calmette in France promoted in 1926 a life vaccine based on *M. bovis*. The Health Committee of the League of Nations (predecessor to the World Health Organization) adopted the BCG vaccine in 1928 but it was rapidly abandoned because it had proved defective by the veterinarian Lignières (6). After World War II, the French State stamped out the Friedmann vaccine that gave satisfaction and imposed the BCG.

C. The Bacille Calmette Guerin (BCG)

1. Early evidence of inefficacy and infectiousness of BCG.

According to Ferru, Calmette observed that children having developed a mild extrapulmonary tuberculous infection after drinking milk contaminated with *M. bovis,* appeared protected against more serious forms of pulmonary TB.

This observation prompted him to attenuate a strain of *M. bovis* during 13 years by repeat subculture *in vitro* [meaning growth of the bacterium in a liquid substrate on glass instead of in animals (*in vivo*)]. The attenuated live bacillus so obtained limited, in guinea pigs, the dissemination of inhaled TB bacilli towards the liver as well as their secondary dissemination towards the lung. However, this live vaccine infected various organs for several months and provoked a general lymphatic disease that Calmette and his collaborator Guerin claimed to heal spontaneously within 3 weeks. The vaccine lent no protection at all to monkeys at risk: all vaccinated monkeys contracted TB upon exposure (5).

Calmette refused to take this crucial observation into account although this was an indication that BCG could favor TB infections. The apparently positive results obtained with guinea pigs prompted Calmette to vaccinate newborns in 1926. The proof of efficacy of the vaccine in newborns was, according to Calmette, the skin-allergy, which he assumed the BCG elicited, and he held that a successful vaccination generated an allergy betrayed by a positive tuberculin skin test.

Lignières showed in 1927 and 1928 that the BCG was neither completely attenuated nor efficacious. In England, the statistician Greenwood observed in 1928 that the results obtained by Calmette to prove the efficacy and harmlessness of this vaccine were flawed because the vaccinated children were kept in a protected environment under tight medical supervision by nurses who fed them well and cared for them whereas the unvaccinated

children used as controls were children living in the slums of France (2). Greenwood was right: the general infantile mortality, in the protected environment where the vaccinated children were confined, was 17% in 1922 and had dropped to 5.1% in 1926, at the moment they were vaccinated, indicating that the reduction in mortality in the protected environment started well before the vaccination took place.

2. Proof of protection based on tuberculin allergy.

Calmette observed in 1928 that only 28 % of the vaccinated children held in a protected environment reacted on an intradermal tuberculin injection. This was half the frequency of reactors observed at the same time among vaccinated children kept in an open environment, which amounted to 60 %.

How explain these results, when the expectation was that the BCG would induce a positive skin test at 100%?

Calmette did not comment on the poor reactivity of 28% where he had expected 100% and attributed the twofold increase in reactivity among the exposed vaccinees at risk (60%) to an unapparent tuberculosis infection. He was right in this, but he did not consider the possibility that BCG does not or only rarely provoke a Delayed Type Hypersensitivity reaction betrayed by a skin test, -as he should have concluded from these results- and did not explain the scarce 28% reactors observed among vaccinees held in confinement.

Were these 28% reactions due to the vaccine -as he assumed- or were they also indicative of asymptomatic TB infections? Could it be that the vaccine was iatrogenic and induced *per se* TB infections that were, in this case, unapparent?

The conclusion should have been that the positive skin test observed after a BCG vaccination was due to a TB infection induced by the BCG, similar to the reactivation of dormant TB observed by Koch after vaccination with Old Tuberculin. Such a conclusion implied concordance with the German, which the hyper-chauvinist Calmette could not conceive, and the ubiquitous presence of the tuberculosis pathogen, even in secluded areas, without any evidential proof, and this conclusion was not envisioned.

The first Congress of BCG held the 18th of June 1948 at the Pasteur Institute deviously claimed unanimously (with one abstention by Ferru) that BCG provokes within a short time a neat and lasting allergy induced by tuberculin with a skin test. The observation of Calmette, i.e. only 28% of the vaccinees showed a positive skin test, was ignored. This allergy is a Delayed Type Hypersensitivity (DTH) because the allergy becomes apparent with a delay of 3 days after the challenge and was taken as proof that the vaccine had been efficacious and protective. A second vaccination was recommended if the first one had not elicited a positive tuberculin reaction. This was a sure way to induce a TB infection, unapparent (17) or symptomatic.

3. Vaccination campaigns

France imposed in 1950 by law the BCG vaccination of all infants and young tuberculin-negative adults of the country and throughout the French Empire. Recalcitrant parents faced 2 years of prison.

The vaccination campaign started in Djibouti and Madagascar. G. Comstock, a US Health

Service expert who followed TB clinical trials, reported (7) in 1995 that nothing was done to verify the efficacy of the vaccine, which was immediately reported nefarious by the local Public Health officers, who observed excess TB cases among the vaccinees (5). The WHO organized the vaccination in Libya. The Netherlands and the USA refused the BCG on a routine basis, a measure that in no way favored the endemy above that observed in covered countries.

The consequence of the mass vaccination (13.874.000 subjects) with a deficient vaccine was a rampant spread of tuberculosis and leprosy throughout the world.

a) Leprosy.

The promotion of leprosy by BCG was published in 1960 (8) and again twice in the nineties (9,10) In addition, A. Bagshawe and co-workers observed in 1989 in New Guinea, a TB-free zone, a 9-fold excess of leprosy cases which affected only vaccinees less than 5 years old, during the first five years following the BCG vaccination (11,12). These repeat warnings were all ignored, and "The Hindu" of January 29, 2017, announced an alarming recrudescence of leprosy in India*: "Why India needs to step up its fight: In 2015, the country accounted for 60% of new cases of leprosy globally'".*

b) Pulmonary Tuberculosis.

India.

Notwithstanding the bad results observed in the use of the BCG, the WHO and UNICEF provided support for a BCG vaccine Production Center at Guindy, Madras/Chennay, in 1948. Vaccination was extended to schools in almost all states of India in 1949. A year later, P. V. Benjamin reported that tuberculosis infection was so widespread that no part of the country was free from it. J. Frimodt-Moller vaccinated Indian villagers also in 1950 and published in 1978 an excess of 90% TB cases among the vaccinees (1).

To salvage the BCG, a committee appointed jointly by the Indian Council Medical Research (ICMR) and the WHO acknowledged that the BCG is powerless against lung TB but that it provides substantial protection against childhood forms of TB such as tubercular meningitis; it recommended to give the vaccine before the end of the first year after birth.

The WHO and the US Health Service organized the vaccination of 260,000 Indians in 1970 (the Chingleput trial), controlled by Comstock.

S.P. Tripathy reported in 1986 that this vaccination resulted after a year in a 100 % excess of symptomatic TB among the vaccinees. Four years after vaccination, the excess in symptomatic cases was 150 %. (13)

Brazil.

Ph. Lagrange and H. David, and the American B. Bloom, member of the US National Academy of Sciences, published with colleagues in 1997 in the Proceedings of the National Academy of Sciences, US (PNAS USA) that the epidemic of TB occurring after the vaccination of the Yanomamo Amazonian tribe with BCG in 1994 was due to its immunological naivety. (14)

The "immunological naivety" explanation is a myth. It propped up in Sweden, the Netherlands, and the United Kingdom during the coronavirus pandemy of 2020 and is a narrative that was initially created to not deal

with genocide. R. Dunba-Ortiz exposes in *An Indigenous peoples' history of the United Sates* (ISBN: 978-080705783-4; 2015) that the Native Americans would not have been so vulnerable to diseases if white settlers had not strived to wipe them out. Their food sources were taken from them, their trade routes were cut off and many were enslaved. It is this unrelenting physical and psychological assault that depressed native peoples' immune systems and rendered them vulnerable, not immunological naivety.

Were the Yanomami immunologically naïve?

The PNAS publication reports that the first case of TB among the Yanomami was observed 29 years previously, in 1965, with more cases appearing in the 1970's. The population was vaccinated in 1994 because tuberculosis cases were uncovered among its members. The population was exposed to TB before the vaccination took place and the TB patients present in its midst did not cause an epidemic. The result of the vaccination was that 82% of the vaccinated population contracted TB. A presumed immunological naivety had nothing to do with this epidemic. The epidemiological proof that the origin of the epidemic was the vaccine is indisputable. The PNAS publishes without referees controlling the soundness of the conclusions drawn, and B. Bloom could publish whatever he wanted. The authors indulged in deception.

c) Tuberculous meningitis

As I said, the newspaper DNA had proclaimed in 1978 the beneficial activity of BCG against tuberculous meningitis. It reaffirmed it on July 14, 1993.

The proof of infectiousness of BCG for meninges had been given by Lignières in 1928. It was confirmed in Iran in 1999 by N. Bagghaie, M. Masjedi and A. Velayati (15). In this Iranian study of 100 children suffering from meningitis, only 10% responded to a tuberculin test (90% were thus immune-depressed) and only 22% were meninges-smear positive.

Of the 30 children with a history of vaccination, 9/30 died (30%) versus 20/70 (28.6%) among the non-vaccinated children. These percentages of mortality are very close. The authors noted that the symptoms among the vaccinated surviving children were milder. This is in line with the affirmation published in the German *Algemeine Zeitung* of 29 September 1982 that the BCG is not efficacious against meningitis and, at most, attenuates the symptoms. [I remind here the affirmation in the Journal DNA February 2, 1978: "The (French) experts are honest, competent, and knowledgeable; they are there to tell the truth without dogmatism or occultation. And the vaccine is *particularly* efficacious in infants, against tuberculous meningitis"].

Nevertheless, the claim that the BCG protects against meningitis continues to be asserted repeatedly with effrontery, and the Pasteur Institute continued to advise BCG-vaccination in France in 2018, even if no more obligatory since 2007.

4. The tuberculin proof of the iatrogenic activity of BCG.

The skin allergy induced by a BCG inoculation was found in 1999 in Saudi Arabia to be of the order of 7.8 %, five years after vaccination. An Italian study conducted in Turin by M. Bugiani

and co-workers confirmed this, also in 1999, and attributed the skin induration not to BCG but to a tuberculous infection (16). These observations came in support of G. Comstock, who had followed the Chingleput trial. He remarked in 1994 that vaccinees responded either not at all or else with an induration similar to the one obtained with TB patients. He also had observed that the positive response was obtained mainly among vaccinees who had been former TB patients. This observation reminds of the Old Tuberculin, whose use as a vaccine by Koch lead to a reactivation of dormant TB.

These proofs that the reactivity to tuberculin following a BCG vaccination was not due to the vaccine but to TB were spectacularly corroborated by a Swedish study analyzing the sensitivity to tuberculin of vaccinated and unvaccinated children, published in 1992 (17).

Sweden had wisely discontinued the BCG vaccination as early as 1975. This allowed A. Lind and co-workers to conduct a comparative study of the sensitivity to tuberculin of vaccinated and non-vaccinated children. Three percent of the non-vaccinated controls were tuberculin reactive, which was proof of a latent TB infection, and 49 % of the vaccinated children were positive. Lind attributed this huge number not to a latent TB infection induced by the vaccine, as he should have, but to a sensitization directly due to the BCG, as claimed by the Pasteur Institute and the WHO. The children in Sweden spend the long dark winter months at home, caring for pet animals (birds and fishes). The Swedish investigators controlled the skin reactivity of their subjects not only with tuberculin but also with *avianin* and *scrofulascein*, which would indicate infections with *M. avium* and *M. scrofulasceum*, mycobacteria that infect birds and fishes. They found that 58% of the vaccinated children reacted to *scrofulacein* and 67% to *avianin*, while the frequencies found with these two sensitins in non-vaccinated children were only 25% for *scrofulacein* and 32% for *avianin*.

Lind concluded correctly that BCG favored infections by atypical mycobacteria but refused to extend this obvious conclusion to tuberculosis because it challenged the paradigm under which he worked, namely that BCG induces a skin reactivity.

5. Side effects

Linières observed in 1928 that one of two BCG-vaccinated sisters died from a BCG infection. This was of no significance for the French experts who all affirmed loud and repeatedly whenever a suspicion arose, that the

BCG was innocuous.

V. Romanus and co-workers published in 1993 that the adverse reactions in healthy and immunocompromised children under six years of age vaccinated with the Danish BCG vaccine, were ten times more frequent than was previously reported. (18)

4. Conclusion

The failure of the BCG is, for most of the mycobacteriologists, an enigma. Because its failure is now patent, one looks in all kinds of directions for a recent change as an explanation, i.e. the genetics of BCG changed recently, the genetics of TB changed recently, and the genetics of the population changed recently, as if the English laundry women of Victorian times were genetically different from the women of leisure they served, and similar

in their genetic set-up then to the set-up of contemporary Russian prison inmates, now.

Dilemmas and paradoxes.

The existence since 1948 of a vaccine and of a simple chemotherapy based on four drugs, plus one applied to resistant cases, induced the triumphant TB-actors to ignore the dilemmas and paradoxes posed by the vaccination and the therapeutic strategy they applied. Despite vaccination, at least a quarter of humanity is infected but only 5% to 10% of these subclinical cases develop into a clinical illness (19). The regression of the endemy was similar or better in a country that never applied the BCG vaccination, versus those where 98% of the population was covered. A drug shown active *in vitro* may be non-effective *in vivo.* At the benign end of the spectrum, in pleural tuberculosis, healing may occur without treatment.

To explain these enigmas and paradoxes, new tools were needed.

1. SEROLOGICAL BLOOD TEST

A. Development of a blood test

It is generally assumed that antigen-detecting diagnostic tests show if you currently have the pathogen, different from antibody-detecting serological tests, which show if you have previously had it. This is a simplified elementary erroneous view: antibodies last long after the elimination of the pathogen that provoked the formation of antibodies but are also produced during the infection. The detection of antibodies can thus diagnose an active infection.

During the last 70 years, the WHO relied on diagnostic antigen-tests that showed the presence of TB cells essentially in the sputum of the patient. A negative result leads to inaction. A positive result leads to "stamp the bug out with chemistry".

This obsession of pathogen detection imperatively needed to start a treatment was prompted by insecurity: one initiates a treatment only when one is sure the TB bug is there, because the treatment is harsh. It led to the development of ever more sophisticated antigen-tests of which the Xpert MTB/RIF test (detection of tuberculosis nucleic acid) is a good example. Today, the problem of TB is handled with the concepts that were in force during the 19th century. New sophisticated methods are nothing more than gimmicks that consolidate the 19th century concept: molecular tools build a technical scaffold in front of a facade that stands alone, without the building that it is supposed to clad. The technical skills involved in these methods help hiding the poverty of the results obtained. These huge health care expenditures reflect potential huge corporate profits rather than human needs and the gap between these expenses and promises and performance make manifest our vulnerability to propaganda.

In the hope to substitute an antibody test to the tedious and cumbersome bacilloscopy, multiple attempts to develop a blood test designed to monitor antibodies against TB led to the conclusion that blood tests were unsuitable as a diagnostic because many patients whose sputum was loaded with TB cells showed no presence of antibodies against TB (these were said to be "false negatives") while many asymptomatic controls were with antibodies, and one speaks of "false positives".

These deviant results were attributed to a default of a test that clearly does not perform correctly. However, if the test performs well, the deviant results observed must find another explanation.

I developed a blood test in 1989 (20)

B. Applications of the blood test to diverse situations.

1. Latent tuberculosis infections.

One enigma that the antibody blood-test solved immediately was the ubiquitous presence of the bug throughout a population, even where the incidence is very low. About 50% of blood donors in Nice (France) had IgM antibodies against antigen 60, indicating that the pathogen is ubiquitous and generates transient, fleeting asymptomatic infections betrayed by a surge of IgM antibodies that are sufficiently efficient to quietly eliminate the pathogen. Only about 4% of the blood donors were with IgG and/or IgA antibodies. They were healthy, asymptomatic.

F. Mandler evaluated in 1991 the presence of IgG antibodies against Antigen 60 in 940 healthy Italians and showed that about 4% of the general population in this low TB incidence country has antibodies (21). These 4% were restricted to two groups, HIV-sero-positives and drug addicts.

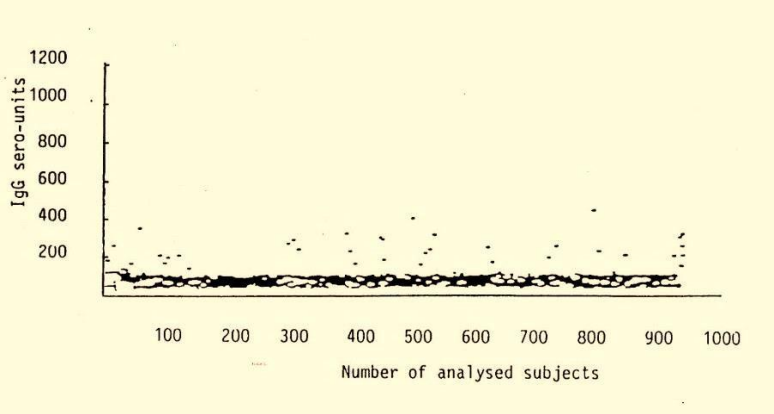

Fig. 3. Sero-analysis of IgG antibodies against Antigen 60, in 940 healthy adult Italians. The results are assembled in a solid line, with only 4% of the values above this baseline.

C. Wirrmann confirmed in 1990 the IgG-sero-positivity of the employees of a supermarket in Strasbourg (France). He found that some cashiers, and only these, not the employees shielded from contact with customers, were positive (22).

R. Patel and co-workers mentioned in 1994 infections due to nontuberculous mycobacteria in kidney, heart, and liver transplant recipients (23). J. Kaustova showed in 1996 that mycobacterial infections affected only some types of cancers (24). J. Graham and co-workers showed in 1998 nontuberculous mycobacterial infections in children with cancer (25).

2. Efficacy of the BCG vaccine.

a.) C. Delacourt and co-workers at the Hospital Necker-Enfants

Malades, Paris (France), published in 1993 (26) that non-BCG vaccinated infants less than 2 years old were without IgG antibodies against

BCG antigen 60, as expected. Antibodies of the IgG class were also absent in most vaccinated children that were 2 and 5 years old, a result that was not expected.

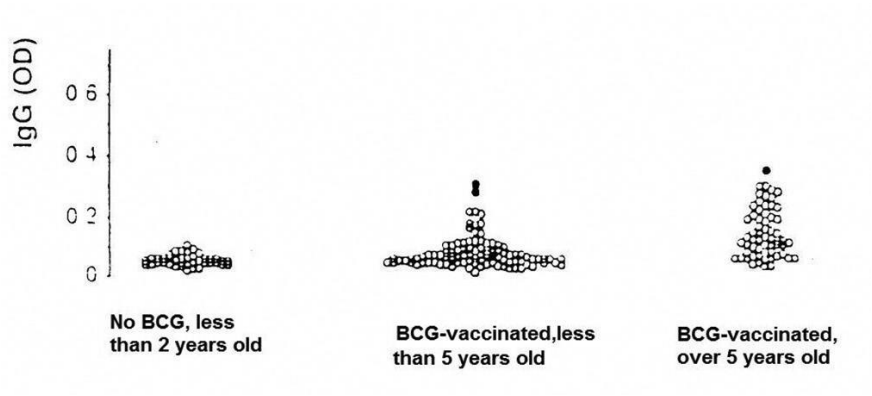

Fig.4. IgG antibodies' absence in vaccinated infants and toddlers.

The very few borderline-positive cases (3 in total, black dots) observed may be traced to unapparent TB infections due to increased exposure of the children to contacts outside the home (i.e. nursery, kindergarten and relatives). In view of the high frequency of fleeting unapparent tuberculous infections taking place among blood donors, as betrayed by an IgM response, such contaminations are expected.

*b.)**S. Rota,* U. Beyazova, T. Karsligil and C. Cevheroğlu followed in a hospital of Ankara (Turkey) for three years the humoral immune response against antigen 60 of BCG, of infants vaccinated at birth as recommended by the Pasteur Institute and the WHO.

This superb study published in 1994 (27) that, at birth, about 50% of the children had IgG antibodies against antigen 60, received from their mothers. At birth, about half of the babies were thus passively protected by maternal IgG antibodies, which disappeared from the circulation two to four months after birth, despite the BCG vaccination. These IgG antibodies remained low ever thereafter. IgM antibodies against A60, which do not cross the placental barrier, were absent at birth but did not rise 2 and 4 months after vaccination. Fifteen months after vaccination, about 50% of the children produced IgM antibodies against A60 but no IgG antibodies. This IgM frequency increased from 50% to 100% when PPD (purified protein derivative, which is purified tuberculin) was used as capture antigen. PPD contains A60 antigen but also an array of other molecules, among which a mycobacterial factor of virulence, lipoarabinomannan.

c.) ***The conclusion*** is that the BCG does not induce the synthesis of IgM and IgG antibodies. The study made on blood donors in Nice has shown that fleeting TB infections betrayed by IgM antibodies are common, but IgG antibodies were not frequent. The same observation was made in Ankara. The great number of mothers with IgG antibodies observed in Ankara is probably because we are dealing with an exposed group that consults at the hospital.

3. The blood test as a diagnostic.

The desire to replace the cumbersome bacilloscopy, which requires to obtain sputum

(not saliva) from the patient, smear the sputum on a microscopy slide, stain the bacteria with Ziehl-Neelsen stain, mount the slide with a glass-cover and count TB bacilli observed in 100 fields of the slide, with an easily performed blood test, prompted a number of clinical laboratories to test this possibility, with responses ranging from vehement rejection to enthusiastic endorsement, depending on various factors not all necessarily of a scientific and medical nature.

A disquietingly large number of students and scholars systematically and easily follow pundits without exercising their own capacity of analysis. The constantly repeated erroneous interpretation volunteered by a pundit who may be greedy and corrupt, backed by a subsequent blind and respectful consensus resting on a stubborn refusal of individual expenditure of mental energy, may persist during decennia without correction. The rejection of the blood test followed the consensus taught by university professors during decennia, and asserted by the WHO in 2011, that patients whose sputum was loaded with TB cells showed no presence of antibodies against TB while many asymptomatic controls were with antibodies (28).

H. Schaaf and A. Zumla edited the textbook "Tuberculosis. A comprehensive clinical reference, published in 2009. Dick Menzies, Kevin Schwartzman and Madhukar Pai wrote its chapter 19: "*Immune-based tests for tuberculosis*". They state on page 189 that:
"The sensitivity of serological assays which target mycobacterial antigens putatively associated with active TB should be highest among persons with smear-positive pulmonary disease – because of the high bacillary load and ensuing stimulation of antibody production".

These authors pretended to ignore the immune depression induced by the bacillus, which reduces the antibody output against any antigen sometimes to zero during active TB. Contrary to what they state, the higher the bacillary load, the lower the antibodies' level, which was abundantly proven well before the textbook was published.

Children with TB are frequently misdiagnosed. TB can mimic many common childhood diseases, including pneumonia, generalized bacterial and viral infections, malnutrition, and HIV infection. However, the main impediment to the accurate diagnosis of active TB is the paucibacillary nature of the disease in children. Younger children produce smaller amounts of sputum, which is usually swallowed rather than expectorated. Consequently, bacteriological confirmation is the exception rather than the rule.

Khalilzadeh and co-workers analyzed in 2001 (29) the value of IgG serology for the diagnosis of children in Tehran (Iran). They reported: *"the sensitivity of serological diagnosis of A60 antigen is 77.3%, higher than culture (51.2%) and smear test (43.1%). Therefore, use of ELISA tests for the detection of anti-A60 antigen is a simple and rapid diagnostic method, which can greatly facilitate the diagnosis of TB in children."*

In a document addressed to the Indian Ministry of Health, the Association of Diagnostic Manufacturers of India wrote:

The tests are regularly used by clinicians for two major clinical scenarios:

*****Early diagnosis in smear-negative pulmonary tuberculosis: Wherein the tests***

are used to confirm clinical suspicion of tuberculosis prior to eventual sputum smear positivity, thereby saving precious treatment time

*****Early diagnosis in extra-pulmonary/occult/pediatric tuberculosis cases: Wherein, the inability to access site-specific specimens for conventional smear, culture or PCR leaves the only available option of a serum test to possibly indicate the illness.*

What is important in these two statements of usefulness are the words

"early diagnosis", meaning a moment in time when the bug had not been given the possibility to exercise in full its immune-depressive capacities.

4. The blood test as a prognosic.

Patients who consult for TB are generally poor and consult only when the symptoms are of such intensity that they feel compelled to do so.

The follow-up of patients at entry with the monitoring of IgM, IgG and IgA production divulged that the patients were immune depressed at entry, for all three classes of antibodies and that a surge of antibodies occurred upon successful treatment. It also showed that this surge could be stopped during the treatment with immunosuppressive drugs, which lead to relapse.

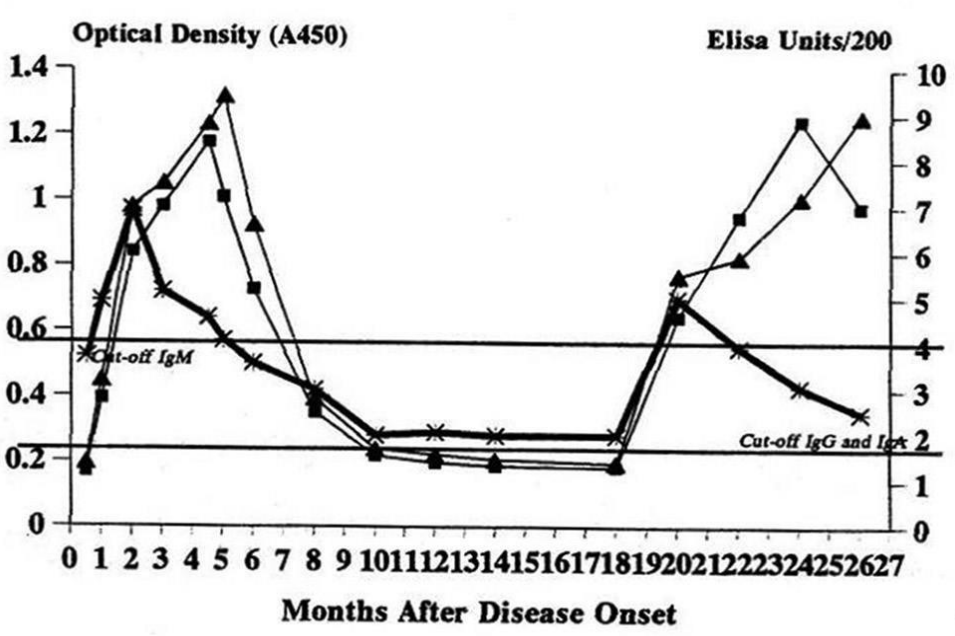

Fig.5. Follow-up with three classes of antibodies in the first treatment and treatment after relapse.

One observes that IgG, IgA and IgM productions are very low at entry. The treatment is beneficial, and a surge of antibodies is observed. IgM declines two months later, which is expected, followed by an abrupt decline of IgG and IgA after 5 months of treatment. This is not normal: they should have continued to be synthesized. However, the drugs eliminated the pathogen, and the patient was released but the drugs

had killed the memory cells able to synthetize IgG and IgA antibodies in case of a new challenge. The patient relapsed 18 months later with a surge of antibodies like the one observed previously: the patient was devoid of memory cells.

The consequence of this observation is twofold. First, there is a possibility to monitor the efficacy of the chemo-treatment: if the level of IgG antibodies is high when the patient seeks specific treatment, the chemo treatment will be an adjunct. If the level of IgG antibodies is low, indicative of a severe immune-depression induced by the pathogen, help is imperatively needed, and the success of the chemo-treatment will be revealed by a rise in specific antibody levels. If the chemotherapy evacuates the pathogen without restoring the immune functions of the patient, this patient is bound to relapse because his immune defenses have been destroyed.

C. Rejection of the blood test.

More than three hundred patients in the care of the Clinique du Sport (36, bd Saint Michel, 75005, Paris) suffered from spondylodiscitis, meaning that their vertebrae were infected, with terrible, unendurable sufferings. The possibility of a mycobacterial infection had been rejected on the ground that no mycobacterium was observed: no pathogen detected, hence no absolute proof and consequently no treatment.

On 21 November 2008, I gave to Dr Sagnet, director of the Clinic, all information needed for him to apply the serological test to his patients. He directed me to the Committee of Experts in Nosocomial Diseases ("noso-comial" means accidentally gotten disease as a collateral injury in a hospital). This was obviously a dead end. I then offered a serological kit to Dr Desplaces (Hôpital de la Croix St Simon, Paris) in charge of the analyses, but she returned the kit to me, unused. The reader must know that physicians have no obligation of results, otherwise many of them would land in prison, but have an imperious obligation of means: they may neglect none to establish a diagnosis or apply a treatment.

J. Grosset, the civil servant in charge of TB in France, Veronique Vincent in charge of the mycobacteria Unit at the Pasteur Institute, the Minister of Health and all other French TB-experts who all knew the existence of the serological test and its merits, preferred to leave the patients of the Clinique Du Sport unattended for three years. Grosset trampled his Hippocratic Oath yet did so with the full approval and active assistance of the French medical establishment. The etiological agent was *M. xenopi*, which had survived the standard sterilization regularly applied to surgical instruments. French justice was however administered: three physicians from the Clinique du Sport were indicted for inoculating infectious products although they bore no responsibility for this infection.

2. The WHO

Policies rest on conclusions drawn from a careful analysis of data. The WHO commissioned a systematic review of published research on TB blood tests. (30)

The meta-analysis (31) was data-rich and intelligence-poor. The authors of this meta-analysis, the WHO experts and the assembly of approving WHO scientists who endorsed it (31), analyzed with statistics the accuracy of TB

serological data, relying on the P value (P stands for Probability). As early as 1999, S. Goodman warned against *"The P Value Fallacy"* (32). M. Pai teaches Biostatistics at Mc Gill University, and the authors of the meta-analysis could not have ignored this warning, nor could the WHO experts, nor could the WHO scientists. All three bodies disregarded this warning and endorsed at unanimity a policy relying on glaringly false conclusions.

The meta-analysis was performed with a disarming mathematical zeal meant to command adhesion but relied on a statistical method (*the P value*) unsuited for the elaboration of a policy.

The whole process was a scam.

3. India

India applied the ban on the 7th of June 2012. This was the RNTCP (Revised National Tuberculosis Control Programme).

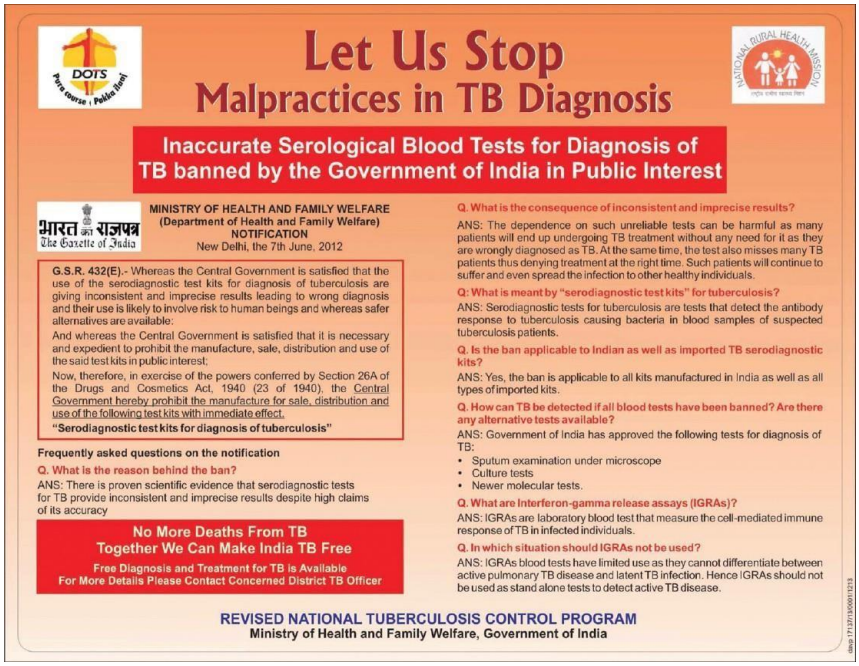

Fig.6. The Indian RNTCP ban of serology, 7th June 2012.

Losing no time, prof. Virander Chauman wrote in the newspaper "*The Hindu*" of September 3, 2012: *"serological tests for TB are not only of no value but also add to the gravity of the problem by resulting in multi-drug resistant (MDR)*

TB. …. Moreover, highly efficient tests based on detecting genetic material from the TB bug are now available and in use….These tests are highly reliable". This efficient highly reliable test was the Xpert/rif assay (33).

The application of the Revised National Tuberculosis Control Program was initiated in June 2012 (fig.6). It resulted in a disaster.

On February 16, 2013, The Wall Street Journal wrote that India's emergency strategy to defeat the disease encourages its spread: *"Global TB fight hits a wall. India's new*

strategy actually makes disease more drug resistant, doctors say".

M. Pai wrote (J Lab Physicians 2013; 5:1-4): "Promoting affordable and quality tuberculosis testing in India": "The Government of India, acting on the 2011 WHO policy against serological tests, banned the use, import, sale, and manufacture of antibody-based blood tests for TB. This historic ban has had a big impact in reducing the use of inaccurate serological tests in India. Diagnosis in the private sector is characterized by overuse of unreliable blood tests. Blood-based antibody tests are not accurate and discouraged by the World Health Organization (WHO). Thus, for the money patients were paying for inaccurate tests, they can now get WHO-endorsed, high-quality tests. The Revised National Tuberculosis Control Programme (RNTCP) has made good progress by providing basic TB diagnosis and treatment free of cost to all patients in the public sector".

In line with the WHO's **Guidance on ethics of tuberculosis prevention, care and control** published in November 2010, the Indian Journal of Medical Ethics urged on June 2013 the Indian Ministry to phase-in the nation-wide deployment of Xpert/RIF test at all points of care of the country as soon as possible.

On January 9, 2014, Dr. S. Panchavati posted on the LinkedIn group "Healthcare India": ***Ethics take a back seat, as opportunism goes into overdrive & leads from the front):*** *"there is a huge possibility of medication or diagnostics being advised disproportionate to the need, and people at large feel that is what is happening*".

Kalikesh Deo, a sitting Member of Parliament from Bolangir in Odisha, wrote on February 6, 2017:

"The scenario for the ordinary Indian is bleak with regard to TB diagnosis and treatment. Affected Indians are dependent on an ineffective Revised National Tuberculosis Control Programme (RNTCP), which is riddled with delays, mismanagement and inefficiencies. This, coupled with a lack of political will and reducing health budgets, has made India's TB problem only worse.

An unsympathetic and ill-equipped public sector makes for a hostile environment and forces many to seek treatment in the private sector. Today, the largest section of the TB affected seek care – not with the RNTCP – but in the private sector. Here, the techniques used for diagnosis or treatments prescribed are often incorrect. Yet, the private sector is the first responder to TB patients when a government programme fails. Yet, nothing is possible until the public and private players work together. The effort for this will have to come from the government. Till then, India will continue to lose the battle against TB".

K. Deo states that the private sector uses incorrect diagnostic techniques. He obviously ignores the plea the Association of Diagnostic Manufacturers of India addressed to the Indian Ministry of Health, not to ban blood tests, and the reason why it should not ban them, namely because they are essential for a correct early detection.

V. Chauman, in the editorial of "*The Hindu*" of September 3, 2012, mentioned above, said that the opinion of the private clinicians in the

field, who claimed on the contrary that blood tests helped fight drug resistant TB, was of no value: the Ministry of health had widely consulted in the public sphere, was very serious and knew what was best for them all. The private sphere was ignored.

Prof Harinath published an editorial in 2017: *"Then, may I ask, why the hurry to ban serological tests and give the impression that serology itself is not useful for TB detection?… Based on meta-analysis by Pai and associates, WHO banned serodiagnostic kits…. The announcement of blanket ban was done without seminar or discussion in different scientific forums."*

The Indian public sector went out of its way to alienate the private sector. The Indian private sector used the blood test appropriately with intelligence for early diagnosis and excellent results.

The very same year 2017, the "Guide to Tuberculosis Diagnostic Tools" edited by the TAG (Treatment Action Group, whose mission is to ensure that all people with TB receive lifesaving treatment, care, and information), fully corroborated this appallingly erroneous appreciation of blood tests. It stated (page 20): *All currently available blood-based, or serological (also called serodiagnostic), tests for the detection of pulmonary TB and ExtraPulmonary TB are not WHO-recommended for use. In fact, the WHO issued a negative recommendation (meaning "do not use this test" for serological TB tests). The reason leading to the lack of endorsement of the blood-based tests for active TB is that such tests have low sensitivity (high false-negative results) and low specificity (high false -positive results). Activists advised the banning of such tests for use in India, particularly in the private sector, and such efforts successfully led to India banning the tests.*

This successful ban was a dishonor for the Indian Ministry of Health, a sin against science and medicine, and a felony for the Indian TB patients and their physicians abruptly deprived at the urging of the WHO counseled by Pai, of an exceptionally useful diagnostic and prognostic tool.

4. corruption

K. Deo also ignores corruption that led to the imposition of an ineffective, complex, expensive antigen detection test (the Expert /Rif test) to replace the useful blood test.

In many African and Asian countries, except for some privileged locations, the laboratory personnel cannot perform sophisticated interferon assays and Xpert/rif assays: no electricity, no pipettes, no incubators, no running water, no training, no brooms, and soap and no what have you. They even have also difficulty using Elisa tests. To impose on them sophisticated assays as the Xpert MTB/ RIF test and interferon tests is a non-sense.

Keertan Dheda of South Africa and Sarman Singh of India both warned in 2014 (36) and later (37,38) that *"the Xpert MTB/RIF results may be a disastrous step for TB control programs, as this test gives alarmingly high false-negative results"*. Nevertheless, South Africa and India meekly followed the recommendation of the WHO: they continue to use the Xpert MTB /RIF test extensively and pay a high price for their subservience and credulity.

BC. Harinath wrote in 2017 an editorial (Int J Mycobacteriol; 2017; 6: 323-325) that speaks of itself and exposes the problem:

"I always wondered how an august body like the WHO has decided to take the unprecedented move to recommend a ban of serological tests for TB, despite the fact that it once advocated the exploration of serology to develop affordable tests for the detection of TB in resource-limited developing countries. …… Why the hurry to ban serological tests and give the impression that serology itself is not useful for TB detection? This drastic step is one of the reasons that have led to the stagnancy in TB diagnostic research…….. There appears to be some plan, by intent or accident by commercial interests to promote the molecular assay "Xpert MTB/RIF" and remove the opposition from the use of affordable serodiagnostics in rural areas with a blanket ban of all tests, without discrimination…… Now let us come to Xpert MTB/RIF test, which was aggressively marketed, demolishing the serology. It is sad to observe that it is not advantageous cost-wise, access-wise, and simplicity-wise to use in primary health centers and rural hospitals in developing countries…… The time has come for the WHO, in the interest of successful control of TB, to review the ban on TB serological tests (in the process serology itself) and the usefulness of the costly Gene Xpert, to take corrective steps in developing countries, in particular in rural areas". This vibrant plea has remained without effect.

Immunotherapy.

Immuno-therapeutic agents are useless for those patients whose chemo-treatment is sufficient to kill the pathogen and heal the patients. As I exposed *supra*, most patients who consult do so only when compelled and have low levels of specific IgG antibodies due to pathogen-induced immuno-depression. These antibodies surge during a successful treatment. The essential contribution of immuno-therapeutic agents would be to assist chemicals powerless in the suppression of an established immunosuppressive infection.

The figure 5 *above* shows the absence of antibodies at entry, the rise of antibodies during successful treatment and the immunosuppression of IgG and IgA antibodies occurring on the 5th month of the treatment, probably inflicted by the harsh treatment. In this case, a boosting of the multiplication of lymphocytes is indicated.

A food supplement able to stimulate the synthesis of nitric oxide, a powerful universal antibiotic generated by macrophages and other cells, exists, is available (39) and was shown to be very effective (40,41, 42,43).

A third use of immunotherapy relies on a mycobacterium that would be non-pathogenic yet able to add to the immune capacities of the patient. The reasoning is to mobilize his defenses against a non-pathogenic mycobacterial helper and hasten his recovery, shortening his treatment from 9 months down to 6. This was the approach elected by Stanford and coworkers, with *M. vaccae*.

1. M. vaccae

A.) success and failure of trials.

J. Stanford and colleagues answered the TB challenge with *M. vaccae* immunotherapy in 1994. The proposed strategy elicited high expectations as well as violent rebuttals. Promising results were published in 1995 (but the study performed at Durban in 1999 (44) ruined the project. This study's purpose was to shorten anti-tuberculosis chemotherapy (about 9 months) by the inoculation, at start,

of killed *M. vaccae,* which would decrease the time needed to achieve a negative sputum culture. The clinicians followed the Helsinki protocol that demands that the control group (placebo) receives the best available treatment, against which the test product will be evaluated.

It did not work.

This outcome was evident. A single inoculation of killed *M. vaccae* at the beginning of the successful chemo-treatment of patients of whom all the controls not receiving the immune-therapy survived, had no effect, and could not have one. As a result, *M. vaccae* immuno-therapy was claimed, mistakenly, useless.

What should have been done is to reserve the immunotherapy to chronic patients. The same erroneous approach was taken in the following two trials made in 2000 and 2002. Yet, proof of efficacy had been given in 1999 and was successful because the immuno-depression of the patients was considered. When the agent was used repeatedly, i.e. injected intramuscular monthly for 6 months to chronic patients resistant to isoniazid, streptomycin and rifampin, 9 of 24 cases were cured after 18 months follow up, versus 1 of 24 cases in the control group (45).

B.) Conclusion.

The potential of the *M. vaccae* immunotherapeutic agent, essentially the formation of antibodies interacting with surface and with cytoplasmic antigens of TB, is masked in those patients whose chemo-treatment is amply sufficient to heal the patients and is manifest in patients where the drugs are either not available on a regular basis or unable to fight the progress of the disease. The very weak cellular immune reactivity of *M. vaccae* is overcome by repeat inoculations of the immunotherapeutic, which proved save (43).

General Conclusion

The need for an immunotherapeutic approach, in view of the current failure of the WHO-drug-policy, is evident. Stanford proposed a specific immunotherapy based on *M. vaccae*. I proposed an unspecific booster of the synthesis of Nitric Oxide *via* uleine (Pau aspido) and of the multiplication of lymphocytes (RNO) (39).

Both approaches are valid provided they are applied in accordance with the immune status of the individual patient under treatment. But this immune status is willfully ignored, and both approaches are up till today strenuously opposed and derided although their use would benefit those patients suffering from an immuno-depression despite chemotherapy, be this depression due to the absence of effect of the drugs used (drug resistance) or else due to immune-depressive drugs themselves.

2. Directly observed Therapy Short term (DOTS)

Reduction of the TB problem to detection and compliance led to the concept of Directly Observed Therapy (DOT). DOT, as a means to control the progression of the disease, is based on good sense. Untreated TB is lethal and, if the drugs are not taken on a regular basis, healing is jeopardized. Good sense also teaches that the enforcement of DOT is, in most parts of the world, impossible. The method was successfully applied in New York and other American cities because the number of TB patients was relatively low,

because these patients were largely co-operative and because the strong political will to eradicate the disease was backed by almost unlimited funds.

The exorbitant cost of such a strategy, the intrinsic immunosuppressive activity of some of the drugs used, the short time of administration and a restriction to only four chemicals, were potential obstacles that left the organizers of this strategy undaunted. It worked in developed countries, but the predicted failure concretized rapidly in developing countries where the stigma of TB and the impossible control of the patients, added to the unsupportable harshness of the side effects, inevitably led to massive escape.

To palliate this, an effort was made to shorten the chemo-treatment and apply a DOT Short term. Stanford's immune therapy addressed this problem, with the failure I mentioned *above*.

3. Multi-drug Resistant TB

The Indian RNTCP policy resulted in the rise of Drug Resistant Strains. Be the patient old, young, starved, or immune-depressed was ignored by those who applied the treatment, with sometimes lethal consequences. I witnessed at the TB–Care station of Zabol (East-Iran), an elderly patient die on the night that followed his initiation of the DOTS therapy. The local health agents attended the funeral but not the experts who imposed the DOT treatment.

The only way to handle these refractory patients seems to be a specific immunotherapy and/or the boosting of their immune system, yet only additional drugs are considered.

4. New drugs in addition to SOP.

The UK All Party Parliamentary Group on Global Tuberculosis evaluated on 24 March 2015 the number of deaths due to Drug-Resistant TB at 75 million people by 2050. The Stop TB partnership, counseled by J. Grosset, advocated Bedaquiline, Clofazimine and Delamanid to meet the surge of MDR-TB, in the immediate term. These are given in addition to the regular regimen, whose side effects are expressed in the following picture:

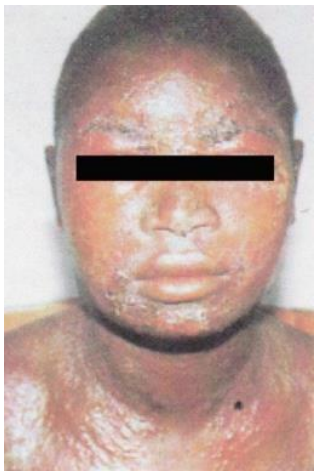

Fig.8. Cutaneous effect of streptomycin, isoniazid, and thiacetazone on a HIV-positive tuberculous woman observed 3 weeks after the beginning of the treatment (Clinical tuberculosis. Ed Davies, Chapman and Hall Medical 1994)

A.)**Bedaquiline.** The drug is known to elicit mycobacterial drug resistance and associates with a disquieting number of dangerous side effects, among which liver toxicity and a potentially serious disturbance in the heart's electrical rhythm. Bedaquiline's long half-life means that these and other side-effects pose risk to patients even after discontinuation of therapy.

B.)**Clofazimine** is said to reduce the time of treatment of MDR-TB cases from about two years down to 9 months. The drug generates crippling side effects. It was discovered in 2014 and 2017 that some MDR-TB patients without prior Clofazimine or Bedaquiline exposure demonstrated preexisting resistance to these drugs, presumably associated with prior TB treatment.

C.) **Delamanid.** The Stop TB Partnership announced on 16 July 2015 that WHO recommends the use of Delamanid for XDR-TB treatment. An examination of the data given in this communication reveals that 17 patients were treated with Delamanid for 6 months in addition to the regular regimen. The failure of the treatment at 24 months amounted to 35%. This is not good.

These drugs are merely an extension of former approaches, which have proven disappointing. New drugs similar to those in use (44) are mirages that arouse hopes which as quickly as they had come would be replaced by others. Tuberculosis patients lurch from one treatment to the next in hopes that something changes the fate of a fight racing toward doom.

5. Conclusion.

The Treatment Action Group (TAG) stated on March 15, 2018, that a single treatment for MDR-TB in the USA costs $294,000. This treatment is unworkable in rural areas due to unbearable costs, to crippling side-effects (vomiting, liver damage, eye damage, and sometimes death), demands so many expensive monthly controls during follow-ups, presents drug cross-resistance and finally encounters a difficulty in tracing patients who escape the harsh treatment as soon as recovery is noticed, that it is not feasible nor realistic to apply it in those countries that need it most. Specific immunotherapy and/or the boosting of the immune system in addition to chemotherapy, accompanied by a monitoring of the humoral immune capacities are novel and constructive means to meet the MDRTB rising peril.

How did the WHO lose the war against TB?

At least 85,416 peer-reviewed publications have covered the Tuberculosis problem, but the problem went from bad to worse. Knowledge is not lacking but the will to use it appropriately.

What has been done, -and not done-, to achieve this result? Who recommended the policy that generated this disaster? How many WHO-TB leaders, for how many decades, have seen and known what is coming but have decided that it is more expedient to keep it behind closed doors? The powerlessness of WHO in the face of the TB contemporary rise is traced to a policy of blindness and deliberate implementation of ignorance, from the beginning on, and a deliberate preference not to act on the patients' interest.

An adversarial relationship with the truth about tuberculosis materialized as early as the 1950's with the inexcusable, appalling

promotion by the French Ministry of Health, the Pasteur Institute and the WHO of the BCG vaccine known by them to be iatrogenic and ended up in an unmitigated disaster with the Revised National Tuberculosis Control Program (RNTCP) initiated by India in 2011 under the advice of the WHO. So dreary a chapter of errors, omissions and crimes has been played out that we badly need policymakers who can face the risky reality they have created.

It should not be contentious that an organization set up to give you advice should have to be told that the advice has to be good for you. A standard utilitarian logic presupposes that health policy should be judged in terms of outcomes for human welfare. The WHO is sovereign, and the patients respond trustfully to the recommendations given them. Behind the spectacle, public health policies are shaped by interaction of a few governing elites that represent insatiable business interests.

National and international institutions appointed experts who rigged the system for their own benefit. The system helps the wealthy at the detriment of the poor. It provides to inept investigators and unworthy corporations huge benefits that they do not deserve. These deeply abhorrent actions injure the many thousands of patients of reduced means of subsistence who thrive in the hope of a relief these very experts deny them.

WHO-TB is struggling with a crisis of both legitimacy and efficiency. Efficiency sputters, sizzles, and ceases to exist, legitimacy creaks and dissatisfaction rises. The TV channel ARTE denounced on April 4th, 2017 (“*The WHO in the claws of lobbyists?*”) the corruption of the TB section of the WHO.

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