WORKS TRANSLATED BY

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The Suppression of Tuberculosis.
Together with Observations concerning Phthisiogenesis in Man and Animals, and Suggestions concerning the Hygiene of Cow Stables and the Production of Milk for Infant Feeding, with Special Reference to Tuberculosis. By Professor E. VON BEHRING, University of Marburg. Authorized Translation by CHARLES BOLDUAN, M.D. 12mo, vi + 85 pages. Cloth, $1.00.

THE SUPPRESSION OF TUBERCULOSIS

TOGETHER WITH

OBSERVATIONS CONCERNING PHTHISIOGENESIS IN MAN AND ANIMALS

AND

SUGGESTIONS CONCERNING THE HYGIENE OF COW STABLES AND THE PRODUCTION OF MILK FOR INFANT FEEDING, WITH SPECIAL REFERENCE TO TUBERCULOSIS

BY

PROFESSOR E. VON BEHRING
University of Marburg

AUTHORIZED TRANSLATION

BY

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TRANSLATOR'S PREFACE.

THE suppression of tuberculosis is of universal interest. A disease which is so wide-spread and which attacks the human race from so many points can be successfully combated only by the cooperation of many different agencies. It is therefore of great importance that the origin of tuberculosis be well understood not only by physicians, but by many outside the medical profession, such as dairy- and cattle-men, officers of health boards, sanitary engineers, etc. In order to present to the English reader in a concise and authentic form von Behring's views on the origin and suppression of tuberculosis, the translator has made selections of von Berhing's articles from several different sources.

The principal article is translated from a lecture delivered in Cassel, September 25, 1903. In reply to certain criticisms of a statement in this lecture, von Behring published an explanation in the *Deutsche medizinische Wochenschrift*, No. 6, 1904. This is reproduced, in part, in the Appendix. The two articles, "Observations on Phthisiogenesis"

and "Suggestions concerning the Hygiene of Cow Stables" are taken from *Beiträge zur experimentellen Therapie*, No. 8, 1904.

Each article, with the exception of the one just mentioned, is reproduced in full and without changes. In a subject of such controversial nature it is essential to reproduce faithfully the exact meaning of the original, and this has been done even though at times it may have led to awkwardness of expression. In a few cases where an exact equivalent for the German could not be found the translator has used the nearest English equivalent, followed by the German word in brackets.

CHARLES BOLDUAN.

BROOKLYN, July, 1904.

CONTENTS.

THE SUPPRESSION OF TUBERCULOSIS.

THE CASSEL LECTURE.[1]

ALTHOUGH in this lecture I have planned to discuss mainly the suppression of human tuberculosis, my experiences with bovine tuberculosis cannot be omitted; for not only have I strong reasons for believing that bovine tuberculosis plays an important rôle in the etiology of human tuberculosis, but I am also firmly convinced that by means of the knowledge gained from the study of bovine tuberculosis, we shall be able to prevent to a great extent human tubercular consumption, and favorably influence the course of already existing tubercular diseases.

Both human pulmonary consumption and *perlsucht* of cattle (which often leads to consumption) are caused by minute vegetable organisms, which it is impossible to differentiate either microscopically or culturally,—the tubercle bacilli of Koch. It is impossible to have either tubercular pulmonary

[1] Address delivered at the seventy-fifth meeting of Naturalists and Physicians in Cassel, September 25, 1903.

consumption or *perlsucht* without the presence of these tubercle bacilli; and no matter how susceptible to tuberculosis an individual may be, a tubercular infection cannot take place if these bacilli be absent.

In order for the disease to develop, three factors must always be present: first, the susceptible organism; second, the vegetable parasite; and third, the necessary opportunity to infect. Under the last I understand all the conditions necessary to bring the virus to such a part of the living organism as will enable it to enter either into the circulation or into other body juices.

Every human individual is by nature susceptible to the action of tubercle bacilli which have entered his body juices. This is true not only of human but also of bovine tubercle bacilli. I emphasize this now but shall give my proofs later. But not every tubercular infection of the tissues leads to consumption. Frequently the tubercular infection is followed by such slight disturbances of health that it remains latent throughout an entire lifetime and is only discovered at autopsy.

Whether the entrance of tubercle bacilli into the tissue juices of an individual shall end disastrously or not, depends on a large number of factors. *Congenital* differences in the degree of susceptibility undoubtedly play a subordinate rôle. A more important factor is the degree of virulence of the inoculated virus, and this may fluctuate within

wide limits. Of great importance also is the amount of the virus and whether the inoculation is single, repeated, or almost continuous for a longer time. Of transcendent importance, however, are the age of the individual, his physiological or pathological condition at the time of infection, other previous diseases, mode of feeding, occupation, hygienic conditions of his surroundings, etc. In short, all that we understand by the term *acquired susceptibility*, or *acquired predisposition*. Finally, I must mention the location and character of the point of entrance of the virus.

We see at once, therefore, that, in the analysis of a tubercular infection, it is not sufficient to have determined the presence of the three necessary factors, susceptibility, the tubercular virus, and the opportunity for infection. If we wish to understand the manifold variety of the reaction phenomena exhibited by an individual infected with tubercular virus, we must thoroughly study all the conditions above mentioned as well as a great variety of accidental conditions.

Nowadays the term tuberculosis is applied to everything which constitutes a change in the organism excited by the entrance into it of tubercle bacilli. In statistics, galloping consumption is thus classed with tubercular local infection of a joint, gland, abdominal organ, of a little nodule in the skin of the face, etc. Calcified as well as other remains of previous lesions are classed as tubercular.

We have every reason to praise as a great scientific advance the discovery that in manifestations so varied in their importance to life and health the etiological factor is the same. One must not, however, make the mistake of classing as one and the same thing tubercular infection and consumption and prognosticate an inevitable and painful end on making the diagnosis of tubercular infection. How little the facts justify such a course is seen in the statistics of bovine tuberculosis and will be seen also when I come to speak of the statistics of human tuberculosis.

In studying the statistics of bovine tuberculosis of this province, I have had the hearty support of the government officials as well as of many county veterinary surgeons. After my colleague, Dr. Römer, had examined several thousand head of cattle we ceased our labors because we were confronted by the depressing fact that larger herds of cattle were rarely ever free from tuberculosis. As a test to discover the presence of tuberculosis we made use of small injections of Koch's tuberculin. We are aware that there are tuberculous animals which do not react to this test. *On the other hand, if the method of making the test be faultless, a positive reaction always indicates that the animal in question is infected with tuberculosis.* Our figures for these cases are therefore without doubt too low. Despite this, and despite the fact that young cattle, even though infected with tuberculosis,

often do not react to the usual dose of tuberculin, we found frightfully high figures for herds of more than twenty animals. We had to admit that in this respect we were no better off than other countries. I will cite one of the most experienced of the investigators of tuberculosis, Prof. Bang of Copenhagen, who says that in Denmark, in stables of more than fifty head of cattle, an average of 60% of the animals are tuberculous. Bang says further: "If one is dealing with a large herd in which tuberculosis has existed for many years, one may as well omit testing the full-grown animals. *Most of them will react, even though they appear perfectly healthy.*" To this, as a result of many years' observation, I should like to add, *and though they may apparently remain perfectly healthy for their entire life.*

As a rule a herd of cattle on a large farm, though infected with tuberculosis, presents a fine appearance. Nor does their state of nutrition, milk production, or calf-raising leave anything to be desired. Now and then a cow coughs, or, despite plentiful food, loses flesh. This animal is put aside and the herd, as such, resumes its magnificent appearance. If in a herd reacting to tuberculin things are allowed to go on in this way for a number of years, especially if the cattle be barn-fed, then, to be sure, the number of coughing animals increases, the offspring deteriorate, the milk and meat production decrease, until finally the scourge of tuberculosis is plainly marked.

Carefully conducted epizootic researches in our province have led me to the discovery that a single, native, mountain breed of cattle, that of Vogelsberg, was probably free from tuberculosis as late as twelve years ago. Through imported cattle the tubercular infection was then introduced. But even now the percentage of animals responding to the tuberculin test is about one-quarter that of other breeds. What is curious, however, in these cattle is, that whereas, according to common experience in all other breeds, the percentage of reacting animals increases with their age, in these Vogelsberg cattle the percentage from the fifth year on even decreases. This may depend on the smaller number of animals infected five years ago. Some of the exceptions, however, I must account for by the definite healing of lesions and a consequent disappearance of the reaction. The favorable termination of the tuberculosis in these Vogelsberg cattle I ascribe to the smaller accumulation of the tubercular virus in the still relatively little-infected stables, or, what amounts to the same thing, *to a smaller dose of the virus infecting the suckling calves. These, of course, take up the infecting agent not only from the mother cow, but also from the other cattle.* One can plainly see how the percentage of reacting cattle in this Vogelsberg breed increases from year to year, and I feel certain that ten years hence these cattle will be as badly off in this respect as other breeds, provided measures are not taken to check the ad-

vancing infection. Such measures we have had at our command for some time, and their usefulness has been thoroughly proven. I know of a large number of herds which, by means of Bang's rules, have been made free from tuberculosis and kept so. It is, however, an expensive procedure and requires the constant supervision of an expert, and even then is usually possible only on large estates where any reacting animal may immediately be separated from the unaffected herd. When, therefore, I published a method of protective inoculation against tuberculosis, one which I had proved in my own stables, I was pleased with the spirit of hearty cooperation shown by the owners of large dairies who had already experimented largely with Bang's procedures.

Through the courtesy of Count Zedlitz I was enabled to make numerous preventative inoculations on cattle in three counties of Hesse-Nassau; from there the experiments were extended to the neighboring grand duchy of Hesse, and to the large dairy of Bolle in Köpenik near Berlin. As a result of this work I was able in the beginning of this year to establish the harmlessness of the procedure. Since then I have had abundant opportunity to inoculate calves under three months on the immense Hungarian possessions of Prince Ludwig of Bavaria; also on those of Archduke Frederick of Austria. Under my direction scientific researches regarding the protection afforded by the inoculations have

been undertaken by Prof. Eber in the veterinary school of the University of Leipzig and by Prof. Schlegel in that of the University of Freiburg; further also by Ober-med. Rath Lorenz in Darmstadt and by Prof. Hutyra in Budapest. In Marburg I myself have made studies on highly immunized cattle regarding the production of antibodies, have reared calves on cows immune to tuberculosis, and have investigated the subject of immune milk.

All the details have been carefully recorded. Some of these protocols have already been printed, and at the close of this year I hope to commence the publication and working out of the rest of this enormous mass of material in my "*Beiträge zur experimentellen Therapie.*"

In the mean time, however, the practical application of my immunizing procedure continues steadily to gain in extent. Austrian as well as Swedish investigators, sent by their governments to study the inoculation technique at Marburg, are to make extensive inoculations in their native countries. The government of the grand duchy of Hesse has ordered that the counties use part of their police fund to defray the expense of inoculating all calves free of charge, provided that the owners bind themselves to allow the county veterinarian to supervise the inoculated animals. From all sides I receive requests for this preventative virus.

Until recently I prepared this virus myself and distributed it free of charge with the help of private funds. I was able to do the entire scientific, technical, and administrative work with only a few assistants. But this is no longer possible, for the expenses have outgrown not only my financial means, but also, even with great zeal for the work, my bodily strength. I should gladly have postponed any change in the manufacture and distribution of the virus until the Prussian government was ready to take charge of the entire matter. But the daily increasing labor has made it necessary for me to rid myself of this burden now, and I have therefore provisionally arranged that the firm of Drs. Seibert & Ziegenbein of Marburg undertake the manufacture.

That this free distribution of the protective virus for tuberculosis of cattle has been completely justified is seen by the report of Koch's Institute, entitled, "*Ueber Immunisierung gegen Tuberculose.*" To be sure, this report contains nothing positive that, as a result of a large number of experiments, I had not already maintained for some years. And it lacks, what is especially important in the practical immunization of cattle, namely, proof in the form of protocols. Possibly these were omitted because they would fail to substantiate Neufeld's assertion that Koch, previous to my publication regarding protective inoculations of cattle with living tubercle bacilli, had already made use of the same procedure.

I cannot refrain from observing that Neufeld treats this question of priority in rather unusual fashion, for he now claims for Koch an immunizing procedure which I had published as long ago as 1901. It would surely have been more reasonable to have made these claims sooner, before the success of the method became so generally recognized in agricultural circles. Finally, I must criticise the author for making similar uncalled-for claims for the English investigator Macfadyan. As the one most concerned, such claims might have been made by Macfadyan himself, yet, so far as I know, that investigator has not even suggested such a thing. In the case of my diphtheria-serum discovery I can entirely overlook similar baseless and unjust claims for Roux and for Ehrlich. Such claims have often been made by medical authors, but neither Roux nor Ehrlich has ever authorized such a perversion of facts.

However, apart from these inaccuracies, the report of Neufeld regarding Koch's tubercular immunizing experiments shows an agreement with my results which extends into the smallest details. This, then, must demonstrate to the world the absolute reliability of my immunizing principle. The entire suppression of bovine tuberculosis is now only a question of conscientious and properly conducted protective inoculations, and, of course, also a matter of time.

Just now I am studying the important practical

point which for a long time occupied the attention of students of vaccination. We now know that protection against variola is only then secured when the vaccination is followed by a typical inflammatory reaction. Are the requirements the same for bovine tuberculosis? This I cannot yet definitely answer; and even under the most favorable conditions I shall be able to answer it only after a lapse of two years. By that time the calves that were injected only once and that did not, so far as we could ascertain, react to the inoculation, will have grown to maturity.

Another important problem, one regarding hereditary immunity, is already nearer solution. I believe I am warranted in saying that, as a rule, the immunity against tuberculosis is not transmitted by the cow to her calf during intra-uterine life. One can readily be deceived by cases of apparent hereditary immunity. For example, in examining the fourteen-day-old calf of a highly immunized cow, I found the calf to possess a comparatively high degree of immunity against *perlsucht* bacilli. Without my previous experience with infantile immunity to guide me, I might easily have been led into the error of assuming this to be a case of hereditary immunity. Comparative tests, however, have made it very probable that the immunity, undoubtedly possessed by this calf, was derived from the milk of the immune cow with which the calf was nourished.

It will still be some time before I can present any experimental evidence to show whether a cow, less highly immunized than the one just mentioned, also produces a milk which possesses protective immunity, and if so, how far it is possible to increase this property; whether such a milk can be used to combat human tuberculosis; and finally, whether it can be transported long distances without losing this property. I have, to be sure, every reason to hope that we are on the right track when we believe that immune milk constitutes a remedy for tuberculosis with which no other remedy can even remotely compare. You will, I am sure, believe that I shall leave nothing undone which will lead to a realization of these hopes.

Right here, perhaps, it is well to say that, for the purpose of making very careful therapeutic experiments, I am delivering immune milk to only a few of my medical friends. I cannot, therefore, meet the many demands for this remedy coming from medical and other sources. They are so numerous that I cannot even answer them all. When, as a result of these clinical experiments, the conditions and restrictions are determined under which immune milk exerts its therapeutic action, I shall make public all the facts in regard to this remedy, and thus make it accessible to every one.

In the mean time I believe it will serve a very useful purpose if I publish in readily comprehensible form the scientific facts on which my plans for

the suppression of tuberculosis rest. With this, of course, will come a discussion of controversial points in the field of tuberculosis investigation which have attracted the attention not only of the medical profession, but also of the laity.

I shall commence with the question as to what relations exist between human and bovine tubercle bacilli. Two years ago, in London, Koch said that the excitants of bovine *perlsucht* have nothing to do with human consumption. If this be true, then I must at once admit that my hopes regarding the utility of cattle immunization for human therapeutic purposes have very little foundation. It is well known that the protection afforded by immune bodies excited by the injection of any virus extends only to the same species of virus, not to that of different species. Nor, in our entire investigations in immunity, do we possess a single example showing the possibility of protecting animals or man by means of one infectious agent against the action of another of a different species. All this is now so well known that I need not discuss it. When, therefore, the researches at the Koch Institute show that it is possible to protect goats, donkeys, and cattle against *perlsucht* by inoculating them with human tubercle bacilli, does it not show that in these diseases there is no difference of species?

According to the recently expressed opinion of his co-workers, Schütz and Neufeld, Koch in his London address did not maintain that there is a

difference in species. He merely maintained that the virus of human consumption is not identical with the virus of bovine *perlsucht*, and that, likewise, bovine tuberculosis is not identical with human tuberculosis. That, to be sure, would be something quite different from what the rest of the world understood. Human pulmonary consumption and bovine *perlsucht* are anatomically so dissimilar that it required extraordinarily clever researches and keen reasoning to lead to the discovery of a common etiological factor in these diseases. But one can easily go further than this. One can maintain without exaggeration that tubercular lupus, tubercular scrofula, and other human tuberculosis localized in joints, serous surfaces, etc., are just as little identical with pulmonary consumption as are the cases of bovine *perlsucht*. The term *identical* does not even fit all cases of consumption. Strictly speaking, a process or thing is identical only with itself. Certainly not all tubercle bacilli are identical, nor yet all kinds of tubercle bacilli. Koch's assertion that there is an essential difference between human and bovine tubercle bacilli, and that these differences are not bridged by any connecting links, provoked the strongest opposition. This assertion has since called forth observations from all over the world which positively demonstrate the existence of intermediary stages in the virulence of tubercle bacilli derived from mammals. Generally, tubercle

bacilli derived from cattle are more virulent for all animal species, thus far examined, than are human tubercle bacilli. And the opinion is constantly gaining ground that bovine tubercle bacilli are also more virulent for man.

In the scientific controversy as to whether the virus of consumption and that of *perlsucht* belong to the same species or not, various misunderstandings have prevented an agreement. Very often the terms "similar species" and "different species" have been used in a different sense. It is, of course, wholly a matter of individual judgment where to draw the line limiting the membership of what, in our minds, constitutes a species. Even if we apply Darwin's definition and include under the term "species" only "organisms of similar origin," the term is still very elastic. However, in the light of our present biological knowledge, based on Darwin's theory of evolution, we are under no circumstances permitted to take as a criterion for like and unlike species such an easily influenced factor as the degree of virulence, i.e., the capacity for giving rise to pathological changes in another organism. *Nowadays all botanists and zoologists are agreed on this, that membership in a species is determined by the origin of the organism.* An anthrax culture, which has been made entirely avirulent by means of Pasteur's procedure for reducing virulence, surely belongs to the same species as does the virulent variety from which it was derived. And in this

case the difference in the degree of virulence is far greater than it ever is between human and bovine tubercle bacilli. If we keep in mind all the functional differences that have experimentally been shown to exist between tubercle bacilli which have long lived in a human body and those which have long lived in a bovine body, we shall surely not go wrong when we assume that with a little patience and expert knowledge we shall be able to make these two varieties absolutely similar again, even in respect to their virulence.

Passing now to the discussion of the real subject of my lecture I shall try to give you an idea of the distribution of tuberculosis in the human species. Although the number of persons dying of consumption is frightfully large, the number of tubercular, and tuberculariy infected, persons is much larger. Not until recent years have we secured tolerably reliable data to enable us to judge of the distribution of tuberculosis in civilized countries.

If we classify as tuberculous every case in which there are pathological changes due to tubercle bacilli, then surely the oft-quoted saying of the noted Greifswald physician is true, "We are all a bit tuberculous." It has not been easy to demonstrate this fact scientifically, and it would have been impossible of demonstration without the two epoch-making discoveries of Robert Koch, that of tubercle bacilli, and of tuberculin.

Not even a man of the scientific attainments of

Virchow was able to judge what constitutes tuberculosis, although during fifty years he had endeavored to formulate data for the differential diagnosis of post-mortem findings. Since we have learned that Koch's bacillus is the bond of union between all the various tubercular diseases, we see that many inflammatory processes which Virchow described as entirely distinct and different are nothing else than special stages in the tubercular infection. The anatomical unit of the tubercular process is the tiny, translucent gray nodule of millet-seed size described by Laennec and Virchow, the miliary tubercle. But what may develop from this may take the form of most widely divergent pathological lesions, and it was not granted to Virchow to puzzle out the endless variety of things etiologically the same. Yet it is *etiologically* that the vital processes must be analyzed if we wish to have a firm basis for our struggle against preventable diseases. This has more and more become the opinion of modern physicians. The progress that has been made, first in surgery and then in internal medicine, progress that only fifty years ago was declared by the most eminent representatives of medicine in those days to be impossible, has been possible only through the etiological investigations developed by Pasteur and by Koch.

The first principle in the etiological investigation of all vital processes, and therefore of those concerned in infectious diseases caused by micro-

organisms, is thus formulated by Darwin: "That which is of the same origin belongs to the same species." Two infectious agents may resemble each other ever so closely, but if they have not the same genealogy, i.e., if they are phylogenetically widely separated, then, biologically, they belong to different species. And conversely, size, form, and other properties of certain micro-organisms may be ever so different. If, however, the organisms are of the same origin, then, biologically, they belong to the same species. The virus of anthrax occurs in two forms: as bacilli, and as oval spores. The quotidian malaria parasite has an extraordinarily complicated cycle of development. Nevertheless we have no hesitancy in speaking of either an anthrax virus or of a malaria parasite.[1]

The virus of human tuberculosis, the tubercle bacillus of Koch, possesses narrowly limited morphological characteristics. It is readily recognized since Koch published his very accurate description of it in 1882, and especially since Ehrlich, shortly afterward, published a specific staining procedure. The tubercle bacilli are familiar to us as rods of varying length, but of fairly constant thickness, which occasionally show granular degeneration. I believe it is now everywhere accepted

[1] On the other hand, certain stages in the development of various malaria parasites resemble each other very closely; but we distinguish the parasites definitely because they are of different origin.

that the presence of these bacilli in a lesion in the human body indicates the tubercular character of that lesion. Leprous lesions with somewhat similar bacilli must, of course, be taken into account, but these, as a rule, can already be differentiated macroscopically.

Recognizing this significance of the presence of Koch's bacilli and applying all other known methods for the identification of tuberculosis, Dr. Naegeli of Zürich, working under the direction of Prof. Ribbert, was unable to discover at autopsy a single body over thirty years old in which there were not some signs of the occurrence of a tubercular infection. Between the ages of 18 and 30 there were 96%; between 14 and 18, 50%; between 5 and 14, 33%; and between 1 and 5 years, 17%, which showed the presence of tubercular lesions. In the bodies of infants under one year, on the other hand, definite tubercular signs were invariably absent.

The astonishing results of these careful anatomical investigations have been verified by reports from pathological anatomists in other cities; yet at first sight, they seem to contradict all medical and other experience, for according to these results all of us assembled in this hall are tuberculous! But the perfected diagnosis of tuberculosis in the living person leaves no doubt that Naegeli's figures, at least for thickly populated centers, possess general applicability. To be sure, if we count only the patients who come to the physician

because of tubercular or supposedly tubercular symptoms, then our figures do not agree with Naegeli's; nor will the statistics of living persons in whom we can discover tubercle bacilli agree with the autopsy statistics. The agreement, however, becomes very close if we make use of a diagnostic method furnished us by the second of Koch's above-mentioned discoveries, the tuberculin injection.

Koch's tuberculin is a water-soluble tubercular toxin, given off from the bodies of the tubercle bacilli to the culture medium, and concentrated together with glycerine. Injected either subcutaneously or intravenously it causes no reaction in persons free from tubercular infection. On the other hand, it is one of the strongest poisons for those who are under the influence of such an infection. Even before the infection has led to clearly recognizable lesions, and long before there are any symptoms of tubercular disease, and even if the most careful physical examination fails to discover a suspicion of tuberculosis during the entire lifetime of the individual, his peculiar susceptibility to this tuberculin injection shows that somewhere in his tissues or body fluids tubercle baccilli are producing their peculiar changes.

The nature of these changes is becoming somewhat clear to us since there have been discovered in the extra-vascular blood of tubercularly infected men and animals coagulation and agglutination phenomena which are entirely absent in the blood

of non-infected individuals. It appears that the activities of the tubercle bacilli in the body of the host excite the production of a soluble anti-body. When this anti-body comes into contact with the water-soluble substances derived from the tubercle bacilli, Koch's tuberculin, it is transformed into an insoluble body. According to my own researches I believe it probable that this anti-body is formed by the smallest arterioles in the neighborhood of the infected area. The extent of the agglutination phenomena varies according to the amount of anti-body and of the tuberculin with which it comes into contact. This manifests itself clinically, by the degree of fever, and anatomically, by intravascular coagulations. The latter, in some cases may lead to exudations or to the escape of blood from the pathologically altered vessels. As a result of the tubercular poisoning, we would then have, at autopsy, the typical picture of a tuberculin reaction.

Tuberculin, in its action as a blood poison for an individual infected with tuberculosis, behaves like many other infectious poisons. Very small fractions of the amount sufficient to threaten life cause a distinct reaction. This is manifested by a rise of temperature preceded by a sharp fall. I know, through personal experience, of a case of human tuberculosis in which more than a hundred times the usual diagnostic dose of tuberculin was administered. But aside from several days of high fever

and a considerable feeling of illness, it had no damaging influence on the patient's general condition. Koch, the discoverer of tuberculin, once took a strong dose of tubercular poison in the form of dead tubercle bacilli and became very ill. In his case probably a hundredth of the amount would have sufficed to cause transient temperature changes and thus have demonstrated that he also had once been infected with tubercle bacilli. Ten years ago I myself reacted to a dose of 4 mg. with fever and a pronounced feeling of illness which confined me to my bed for several days in San Remo. Therefore I have no doubt about the tubercular infection of my body.

The most instructive evidence to confirm the general truth of Naegeli's figures is furnished by the results of investigations made by the Austrian army surgeon, Dr. Franz, on soldiers of two regiments of infantry. In order to avoid injuring the health of the individuals tested, Franz used only very small doses of tuberculin, 1 to 3 mg., which, in case the injection was repeated, was increased to 5 mg. In spite of this, and in spite of the fact that the soldiers represented the healthiest individuals of the population, he found in one regiment in the first year of service (1901) 61%, and in the second year of service 68% of tubercularly infected cases. Franz adds to his report, which at present I have only in manuscript, that when he employed the dose originally recommended by Koch, namely one

centigram, his percentage for the twenty-first year of life approached Naegeli's very closely, 96%!

On the other hand the Hungarian investigator Dr. Nikolaus Berend has not obtained a single positive tuberculin reaction in ninety-six very young children, in spite of the fact that among these were some very feeble individuals, and children of parents manifestly tubercular; and further, despite doses as high as 1 cg. We see then that herein also the statistics coincide with Naegeli's post-mortem statistics.

Another proof that human tuberculosis is much more widely disseminated than was heretofore believed is furnished us by a diagnostic method devised by the French clinician André Jousset, namely "inoscopy." By means of inoscopy we can examine microscopically, for tubercle bacilli, coagulable inflammatory exudates and the blood of suspected cases of tuberculosis, even though only very few bacilli are distributed in large amounts of fluid. And we examine these fluids directly, not indirectly by means of cultures. As a result of the coagulation the bacilli are fixed by the fibrin, from which they are freed by dissolving the fibrin with an appropriate digesting fluid.[1] The bacilli

[1] Formula for the digesting fluid:

Pepsin	1–2 gm.
Glycerin	10 c.c.
HCl 22° Baumé	10 c.c.
Sodium fluorid	3 gm.
Distilled water	1000 c.c.

are then separated by centrifuge and can be examined directly in microscopical preparations. With the aid of this very valuable diagnostic method we are enabled to demonstrate the tubercular origin of almost all serous pleurisies, of many exudative peritonites, of accumulations of fluid in the peritoneal cavity of alcoholic individuals with liver cirrhosis, of joint inflammations, of exudative meningites, of many cases of heart-disease and of other classes of symptomatic affections, where formerly most physicians did not think of the possibility of these affections being tubercular.

I cannot fail to express my conviction of the general diffusion of tuberculosis in densely crowded populations, and of the consequent futility of all attempts to suppress the disease by means of isolation and segregation. What would be the outcome if we were to adopt the suggestion seriously put forth, to send all the tubercularly infected soldiers to hospitals, later discharging them as unfit for service? We should not have more than 5% left for active service, and even this small number would probably after the lapse of a few years be declared tuberculous. At the most I can assent only to the separation of the coughing consumptive from the apparently healthy individual; and these should be sent, not to sanitaria [Heilstätten], but to homestead colonies [Heimstätten] such as we formerly maintained for lepers.

We need not, however, idly fold our hands and

become fatalists who see the inevitable destruction of the human race by tuberculosis. *Tubercular infection does not by any means signify tubercular consumption.* It is just this enormous diffusion of tubercular infection which demonstrates, better than anything else, the curability of many tubercular diseases, their *liability to spontaneous cure;* for I must admit that I have little faith in the curative action of any of the numerous methods of treating tuberculosis. Here also the maxim formerly applied to diphtheria holds true, "mild cases go on to recovery, severe infections are fatal"; and here as in diphtheria I was confronted by the question whether there are ways and means to prevent severe infections with bad prognoses, or to convert severe infections into mild ones with favorable prognoses. You will be able to answer this question yourself if I now give you the results of my experimental investigations on the occurrence and prevention of tubercular consumption. I shall begin by discussing the requirements necessary for the development of tubercular consumption in man.

But first of all I must say that according to my ideas there has not yet been a single well-authenticated case in which pulmonary consumption has originated in adult persons as the result of a tubercular infection developing epidemiologically, i.e., under essential conditions for infection occurring in nature. Even counting those cases in which mortuary assistants, butchers, and labora-

tory workers have been severely infected through the subcutaneous tissues, I still fail to find any proof that a traumatic infection has caused pulmonary consumption in an individual not yet infected with tubercle bacilli.

I am well acquainted with the argument by which it is sought to prove that pulmonary consumption may develop as a result of inspiration of particles of dust or moisture containing tubercle bacilli. The facts on which this argument is based are the greater occurrence of tuberculosis and a higher mortality rate from that disease among nurses, occupants of houses in which there are pronounced cases of phthisis, among the inmates of prisons, etc. But, considering the figures previously given, showing the enormous diffusion of tuberculosis, the objection is surely justified that the persons thus dying of consumption already had a tubercular focus in the lungs and that this pulmonary disease, under a mode of life favorable to tuberculosis, was converted into florid phthisis.

In order not to be misunderstood, I wish to emphasize here that I do not at all deny that infection can be caused in adults by inoculation with tubercular virus. In fact, I assume that few of us in advanced life escape such infection. But that this infection leads to cavity formation in the lungs, is, I believe, fully as unproved as the assertion that bovine tubercular virus has caused human pulmonary consumption in even a single instance.

Koch has very properly pointed out the entire absence of proof for this last statement. I can go still further in my concessions to the prevalent view that consumption results from the inhalation of particles of dust or moisture laden with tubercle bacilli. I concede not only the possibility but the actual occurrence of pulmonary tuberculosis going on to consumption, as a result of infection of an adult person. I concede this in the sense that on the basis of an infantile infection a pulmonary tuberculosis has developed which becomes manifest only through the agency of the additional infection. However, the opportunity for infection with tubercle bacilli cannot by itself be a deciding factor in the development of pulmonary consumption. I can here cite the experience of Dr. Moritz Schmidt of Frankfurt a. M., who has examined a great number of cases of tubercular laryngitis. In his experience of over forty years he has certainly been exposed, more than others, to tubercular infection. But neither he nor any of his numerous assistants has ever become consumptive.

My experiments on animals have shown me that the lesions characteristic of human pulmonary consumption are developed only after there have been extensive and long-continued disturbances of the vital functions of the organism. Our ancestors introduced the term "dyscrasia" and "diseased constitution" to express this idea. I have succeeded, especially in goats, but also in other

animals, in producing a clinical picture exactly similar to that of human pulmonary consumption. In these animals I first produced a moderate degree of immunity against tuberculosis by a lengthy course of treatment, and then I injected a strong tubercular virus into the circulation. I regard the lesions in pulmonary consumption as being produced in similar fashion. They are the expression of an infection in an individual who, owing to a very early previous infection with tubercle bacilli, is less susceptible to the new infection. These late infections may in isolated cases be referable to the inhalation of tubercle bacilli. They may, however, be due to already existing tubercular lesions, and so be regarded as auto-infections or metastases. *Were we to inject into the tissue juices of a person not yet partially immunized against tuberculosis an amount of tubercle bacilli equal to that usually found in the lungs of consumptives, the person would die of an acute miliary tuberculosis, but he would never develop pulmonary consumption.*

There is another argument against the common assumption that primary infection by way of the respiratory organs is the cause of consumption, and this is furnished by an analysis of the anatomical findings. If we allow an individual, entirely free from tuberculosis, to breathe tubercle bacilli, the opportunity for an intestinal[1] infection is surely

[1] The intestine reckoned from the pharynx down through the stomach to the rectum. See appendix, p. 81.—TRANSLATOR.

presented; on the other hand, that any bacilli whatever reach the lungs directly cannot be positively affirmed. Infection of the organs of the pharynx and larynx in these cases always corresponds to disease of the lymph-vessels and glands in the neck, and gives the individual the scrofulous habitus. Now let us recall the neck of consumptives. It appears almost as if, at the time when an individual may be designated as being a candidate for consumption, the organs of the neck were already quite immune against a vulgar tubercular infection.

I could multiply the arguments in favor of my assertion that, in order to have pulmonary consumption follow infection with inspired tubercle bacilli, it is necessary to have constitutional changes in the organism due to an early previous infection. I hope, however, to have sufficiently established my reasons for rejecting the current theory of the origin of consumption, a theory due mainly to the valuable and detailed researches of Cornet.

I must decline to accept another wide-spread view, namely, that hereditary influences are deciding factors. Theoretically an intra-uterine infection with tuberculosis is certainly possible, and in a few cases such an infection has actually been proven. But neither the parental nor the preparental transmission of tubercle bacilli, nor the hypothetical transmission of a body predisposition to tuberculosis, is of any practical importance. Nevertheless, according to my researches, the view preva-

lent among the laity regarding the important influence of parents, grandparents, and other near relatives, in the etiology of consumption, is entirely justified and proper. I, too, am of the opinion that one can properly speak of the bad prognosis in cases of *family tuberculosis*. If, in taking the history of a patient, I should elicit the fact that several near relatives had died of consumption, and if, then, by means of the tuberculin reaction or of inoscopy, I made the clinical diagnosis of a tubercular infection, I should be very pessimistic in my prognosis, even though the infection were not otherwise manifest.

I may very properly be asked how I can deny hereditary transmission and yet place so high a rating on the influence of the ancestors, cognates, and house-fellows, in the production of pulmonary consumption. A little explanation regarding the meaning of certain terms will make my ideas perfectly clear

The expression *hereditary transmission of tuberculosis*, or rather, *of tubercle bacilli*, may be construed in several different ways. It may mean the hereditary transmission from father or mother, or from grandfather or grandmother, or from ancestors still further back. If we designate the parental transmission as *congenital heredity*, transmission from further back as *pregenital heredity*, then in my view of the origin of pulmonary consumption, generally neither congenital nor pre-

genital heredity comes into play. And, looking at the matter as I do, if one is still desirous of speaking of the hereditary influences of relatives, one ought to use the term *postgenital.* It is now almost everywhere conceded that human tuberculosis as a rule is actually of postgenital origin.

Experience has taught me that if, in scientific investigation, one wishes to discover something new, one should study the exceptions to the rule. In actual practice, on the other hand, it is well to keep to the rule. We may therefore safely ignore the cases of congenital tuberculosis, but must all the more thoroughly study the circumstances which in extra-uterine life govern the tubercular infections which lead to consumption. And here I believe I have discovered a new principle which may be expressed thus:

"*The milk fed to infants is the chief cause of consumption.*"

This assertion will at first sight be surprising, for it has long been maintained that the suckling infant receives milk free or almost free from germs. Mother's or nurse's milk is taken by the child in this condition, and the cow's milk for artificially nourished children is usually first boiled or scalded. In later life, to be sure, much less attention is paid to securing a milk as sterile as possible. How, then, is this to be reconciled with the above statement, that it is especially the *milk fed to infants* which constitutes the chief danger in causing tuberculosis?

And yet this statement is true, not because the milk fed to infants is at all worse than other milk, but because the human infant, like the young of all other mammals, is destitute of the protective agencies in his alimentary system which at a later period of life prevent the entrance of disease germs into his tissues. It has taken many years of experimental work to demonstrate this fact conclusively. At present, however, the chain of evidence is so strong that I have not the least hesitancy in building on it my entire plan for the suppression of tuberculosis.

In this lecture I can do no more than summarize the main proofs for my assertion regarding the ready penetrability of the infantile alimentary tract for all disease germs, but especially for tubercle bacilli.

I began with a very interesting fact discovered by my fellow-worker, Dr. Römer. He showed that true albumins penetrate unchanged the intestinal mucous membrane of new-born foals, calves, and smaller laboratory animals, and that they produce the same action on the organism as when they are injected directly into the circulation. In adult animals of all species, on the contrary, the true albumins must first be digested into peptones before they can pass through the mucous membrane. The anti-diphtheria serum and the anti-tetanus serum contain curative substances in the form of true albumins. If such a serum be introduced into the stomach of a healthy, full-grown

animal or man, not a trace of these bodies passes into the blood. On introducing the serum, however, into the stomach of the new-born, the unchanged antitoxic albumin can almost entirely be demonstrated in the blood. This discovery indicates that the mucous membrane of adults, acting as a dialyzing membrane, does not allow the large molecules of true albumins to pass through unchanged, whereas the mucous membrane of sucklings behaves more like a very porous filter.

It was but a step from this discovery to the assumption that the mucous membrane of infants might behave similarly toward bacteria. For my first experiments I selected anthrax bacilli, which, when free from spores and given in milk per stomach, do not affect adult guinea pigs at all. They are quite rapidly thrown off with the excreta, remaining, however, a little longer in the cæcum. When the same dose of bacilli was administered in this way to guinea-pigs less than eight days old, they died just as rapidly of anthrax as by the customary method of infection. Next I tried anthrax bacilli whose virulence had been reduced. These are harmless when injected subcutaneously into guinea-pigs. *After feeding these weakened bacilli to new-born guinea-pigs, the blood of the animals contained anthrax bacilli, though the animals did not die. Incidentally, a fact of considerable theoretical importance was discovered: that the anthrax bacilli*

possess a very intimate affinity for the endothelium of the heart and blood vessels.

Having thus studied the fate of anthrax bacilli introduced into the stomach of new-born and adult guinea-pigs, I now turned to a similar study of tubercle bacilli. Together with Dr. Römer I studied the behavior of guinea-pigs toward a definitely weighed quantity of tubercle bacilli given in *one single feeding.* And here also, as in the case of anthrax bacilli, it was found that when the tubercle bacilli could be demonstrated microscopically nowhere else in the alimentary tract, they were often present in the cæcum. After a single feeding of a small quantity of tubercle bacilli, only the new-born or the few-days-old guinea-pigs became tuberculous. When larger doses were administered it happened that older animals also became tuberculous. At the post-mortem examination of the new-born, a few days later, there were found sub-miliary thickenings, with tubercle bacilli in the great and the lesser omentum. There were also little nodules at a point on the root of the mesentery, not far from the cæcum. Of especial interest is the further development of this alimentary tuberculosis in the guinea-pigs which survived. In these animals, even while their general health remains good, it is always possible to demonstrate a tuberculosis of the glands of the neck, a type of disease which may be said to correspond to scrofula in man. Not infrequently there is later on

developed that type of guinea-pig tuberculosis which has heretofore been regarded as the expression of an inhalation tuberculosis.

In the results of all these investigations I see experimental support for the view I have for some time maintained, namely, that the origin of the epidemiological pulmonary tuberculosis in man and that of the epizootic pulmonary tuberculosis in cattle is a primary intestinal infection occurring in very early infancy. In this I leave undecided whether the bacilli gain access to the body through feeding or through inspiration.

Upon reflection it will at once be seen that from these experimental data showing the ready penetrability of the infantile mucous membrane, another logical deduction follows: If even non-virulent anthrax bacilli introduced per stomach gain ready access to the circulation of the new-born, then it must be possible for all the bacteria of milk to do the same, and we may therefore expect that the accidental presence of pathogenic bacteria in the milk fed to infants will exercise a damaging influence on the infant organism. In considering their pathogenic possibilities the amount of the infectious germs which enter the system must, of course, be a matter of some importance. Under certain conditions, however, even a few germs may be sufficient to excite disease, for in the intestines, especially in the cæcum, they find an excellent place of incubation where

they can multiply. A milk very poor in disease germs may thus lead to a virulent infection. In breast-fed infants the danger of introducing disease germs, excepting tubercle bacilli, of which I shall speak later, is not very great, for it is very unusual for living germs derived from the interior of the body to appear in the milk. The germs which can be found, even in perfectly fresh milk, are derived from the surface of the body, or from the mouths of the lacteals, or possibly even from the glandular epithelium, as researches conducted by myself, assisted by Mr. Rösler, have shown.

But in *artificially nourished infants* the matter is altogether different. It would be a miracle if, after all the manipulations to which the milk supplied to our large cities is subjected, it did not occasionally contain disease germs derived from the milker or from other persons who have handled the milk.

If, by the time the milk reaches the city, the proliferation of these pathogenic micro-organisms has already gone on to a considerable extent, then usually the milk will contain a number of poisonous substances in addition to the micro-organisms. Some of these germs are killed by the scalding to which the milk is usually subjected before feeding, and the virulence of the rest is much diminished, so that in boiled milk practically no danger is to be apprehended from the micro-organisms. We are not at all sure, however,

that we have made the toxins innocuous by this boiling, and probably a great many cases of intestinal catarrh in artificially nourished children are due, not to a parasitic, but to a toxic infection.

I said it would be a miracle if artificially nourished infants did not frequently suffer from milk infections, and I can add that this miracle does not, in fact, occur. One need only glance at the mortality statistics of artificially nourished infants in order to realize that my experimental results absolutely agree with the facts. The following figures are taken from the excellent report, "Gesundheitswesen des preussischen Staates im Jahre 1901," which has recently been published by the Prussian government.

In the city of Stettin, the mortality for the first year of life was 473.52 for every 1000 living children of the same age; whereas in the period of 10 to 15 years the mortality was 2.94 to each 1000 living children of that age. In other words, *during the same length of time* 161 *times as many infants up to one year died as did children over ten years.*

Berlin, with a mortality of 286.29‰ for the first year of life, stands about midway in the list of Prussian cities having over 100,000 population. Cassel, with 183.54‰, shows about the lowest figure, and even this is inordinately high, for it is not in the nature of things that this is so. We are not facing a necessity of nature to which we must submit like fatalists. This can be readily seen by

observing that there are towns and whole regions in which the mortality figures for the first year are kept within moderate limits. In Ireland and Scotland, as well as in Norway and Sweden, the mortality for this period scarcely exceeds $10^0/_{00}$, about one-fiftieth the mortality in Stettin. In Stockholm I visited a foundling asylum with an organization bound to excite admiration and wonder, in which, as I recollect, the mortality was still less.

Nowadays the assertion that the character of the milk fed to infants is responsible for the great differences in the mortality statistics is nowhere seriously questioned. There are, however, wide differences of opinion as to what the determining factors are, and how, in places where the mortality figures are so outrageously high, we can remedy the evil.

According to my researches into this subject this problem will not be successfully solved by the efforts now being made to secure the use of sterilized milk. I am, in fact, in doubt whether milk sterilization as at present practised can much longer pass as a hygienic measure. For the present, to be sure, we have nothing better. But the discussion of this question does not fall within the scope of this lecture. I have quoted the statistics of the high infant mortality in our large cities merely in order to advance a further epidemiological argument for my assertion that the infant alimentary tract is defenseless against infectious agents whether these

are living or not. Even the infectious toxins pass unchanged through the intestinal mucous membrane of very young individuals, though not through that of healthy older ones. A real advance in milk hygiene can, however, be begun even now if the milk be pasteurized at the dairies and not at the large receiving-stations in the cities. In the raising of calves, this procedure has proven of great value.

I have made exhaustive studies to discover why the intestinal mucous membrane of the young should offer so little resistance to the passage of corpuscular infectious substances. I shall content myself here with the statement that the mucous membrane of new-born individuals possesses no continuous epithelial covering and that the gland-tubes of the ferment-producing glands are little, if at all, developed at this time.

By having thus presented to you the results of my experiments and explained my epidemiological views, I have not really deviated from the subject of the suppression of tuberculosis. We have seen that the tubercle bacilli which gain access to the system through the alimentary tract in infancy constitute the important etiological factor in the production of the tubercular infection which leads to consumption, and I believe that the realization of this great fact will supply us with a rational plan for combating tuberculosis. It will be necessary to strive more than ever to secure a suitable milk diet for new-born and very young children,

one based on sound experimental investigations. The as yet unsolved problem, that of a rational milk hygiene in the suppression of tuberculosis, coincides with the problem of milk-feeding of infants in general. The mode of infection is everywhere the same, but the infectious agents are of great variety. Most of them excite acute diseases which end either fatally or in entire recovery; in the latter case with a simultaneous development of immunity. The virus of tuberculosis, however, behaves quite differently, creeping in most insidiously, all unnoticed, and being in this respect analogous only to the virus of leprosy, of syphilis, or possibly of malaria in tropical countries. It may be months, years, or decades before the infection leads to manifest disease. This depends on the virulence of the virus, which is generally much greater in the virus of bovine tuberculosis than in that of human tuberculosis. It also depends on the number of bacilli introduced per stomach, and whether such introduction is single or oft repeated. In the human being months and years may elapse before the infection is followed by any sensitiveness to tuberculin injections in the usual dose. If, then, at the time of puberty, or after an exhausting puerperium, after too great a demand on the milk secretion (especially with insufficient food), after so-called colds and other unfavorable meteorological conditions, after muscular overexertion, under conditions unfavorable to life, such

as improper nourishment, confinement in insufficiently or badly ventilated rooms, etc., if, after any of these, pulmonary disease develops whose tubercular nature we cannot doubt, then we are dealing with the *beginning of consumption;* the *beginning of tubercular lesions* is much further back; and the first introduction of the disease germs, in other words, the *beginning of the infection*, is far back in earliest infancy. This must be so, for we see many individuals, though subject to the most unfavorable conditions, for example, those confined in unsanitary prisons, wholly escape tuberculosis.

The results of all these scientific investigations lead us back to the old folk belief in family tuberculosis and hereditary consumption, with this difference, however, that now we believe the germs of consumption to be transmitted *postgenitally* from parents, relatives, or house-companions. If a coughing consumptive lives together with a newborn child, especially if cleanliness leaves much to be desired, it is impossible for this child to avoid infection with tubercle bacilli. They are present in the particles of sputum which are scattered about everywhere, and they thus gain access to the mouth and nose of the infant. From there they reach the intestinal mucous membrane, which they penetrate, and so they invade the body juices. Not alone infants, but older persons as well, are endangered in the home of the consumptive. In

these, however, there must previously have been pathological changes in the alimentary tract, or an overwhelming dose of the infectious agent, in order to effect an intestinal infection. Pathological changes, accompanied by the shedding of epithelium, occur especially in the exanthemata, and particularly after measles. The laity has long noticed this close relation between measles and tuberculosis. In the infant, the disposition to intestinal tubercular infection is entirely physiological and normal. The healthiest and strongest infant is exposed fully as much as the weak, sickly one, and perhaps more so, for in the latter other parasites are contending for the cells on which they feed.

I have now given you a general idea of the origin and spread of the tubercular infections ending disastrously. This brings us at once to certain practical points in dietetic hygiene which, though never entirely neglected, are now brought into prominence.

It is unnecessary for me to further emphasize the necessity, in infant feeding, to insist under all circumstances on milk absolutely free from tubercle bacilli; nor, what is equally obvious, that it is absolutely necessary to keep coughing consumptives away from infants. But I should like to observe that not only infants but also older persons should be protected against the possibility of infection, if we have any reason to fear that the

alimentary tract is anywhere deficient in its protective epithelial covering. I have already pointed out the importance of the exanthemata in this respect, and I need only remind you of the many other disturbances associated with the shedding of epithelium, disturbances due to catching cold, to indigestion, or the after-effects of certain diseases associated with intestinal ulceration, etc. There is one other condition which I must not fail to mention, namely, the temporary exacerbations of a tubercular process, in which one cannot be too careful in ordering the diet. For that large class of individuals threatened with consumption, I believe we have a valuable healing agent in the dietetic therapy made prominent during the past decade, especially by von Leyden and his pupils. In the same sense we must regard the temporary residence of tuberculous individuals in sanitaria as most valuable, for even if the lesions do not heal there, the progressive downward course of the disease is checked and the patients learn for the rest of their lives to appreciate what will benefit and what will harm them. In many cases, therefore, these sanitaria will prove themselves homes for the prevention of consumption, even if they are not homes for the cure of tuberculosis.

My own efforts in the field of tubercular therapeutics do not, to be sure, concern themselves with sanitarium treatment. *Their last aim is to make all homes for the prevention of consumption, all sani-*

taria, etc., unnecessary by means of a protective agent similar to that by which Jenner made smallpox pest-houses unnecessary. Institutions for persons bodily wrecked are like those for persons morally wrecked; they are products of our civilization, but not desirable products. At best they are necessary evils.

OBSERVATIONS CONCERNING THE STUDY OF PHTHISIOGENESIS IN MAN AND IN ANIMALS.

1. It is possible in experiments on guinea-pigs to produce pulmonary phthisis by infecting these animals from the oral cavity in such a manner that every direct infection of the lungs (alveolar infection through the trachea = aerogenous infection of the lung) is excluded.

(*a*) Infection through the parenchyma of the tongue.

(*b*) Feeding of tubercle bacilli with milk.

2. Ascribing the pulmonary phthisis which I succeeded in producing experimentally to a lymphogenous or hæmatogenous infection of the lung following previous scrofulous disease. Definition of the term "scrofula": multiple, caseating areas of disease in lymph-glands and in other organs, caused by Tb. infection. Concerning the etymology of the word "scrofula" (Greek = choeraden), see Virchow's "Geschwülste," Vol. II, p. 558.

3. Other varieties of experimental phthisiogenesis:

(*a*) v. Baumgarten's experimental method of causing pulmonary phthisis by means of primary infection of the urogenital apparatus.

(*b*) The experiments of Troje and Tangl with tubercle bacilli artificially weakened in virulence.

4. Critical analysis of the so-called "Inhalation Tuberculosis" of guinea-pigs and rabbits.

(*a*) My own experiments, in which the typical picture heretofore regarded as that of an inhalation tuberculosis, in the sense of an aerogenous alveolar infection, was produced by lymphogenous or hæmatogenous introduction of tubercle bacilli, with the complete exclusion of a primary alveolar or bronchial infection.

(*b*) The experiments of Weleminsky (Hüppe).

(*c*) Signs distinguishing tubercular pulmonary consumption from so-called inhalation tuberculosis.

5. Improbability, so far as importance as a phthisiogenetic factor is concerned, of a primary bronchial, or even primary alveolar Tb. infection following aerogenous introduction of Tb. into the mouth and nose, through the inhalation of dust or droplets containing tubercle bacilli.

6. Proof for my assumptions,

(*a*) That inhaled tubercle bacilli under circumstances occurring in nature are taken up by the lymphatic receptive apparatus without exciting, at the point of entry into the lymph-channels, any tubercular disease,

(*b*) That inhaled tubercle bacilli, after they have entered the lymph-channels of the throat, take the following courses: some find lodgment in the submental glands and glands of the neck; some are transported to the mediastinal (bronchial?) glands; some gain the circulation and thus cause hæmatogenous infections, especially at the peripheral (sub-pleural) endings of the pulmonary artery, from which then the lung parenchyma can be infected; finally, some are carried through the stomach to the lowest portions of the intestines, from where they can reach the mesenteric lymph-glands, the portal vein, and the peritoneum.

(*c*) That the introduction of Tb. into the lymph-channels is accomplished primarily by the leucocytic wandering cell, which takes up the tubercle bacillus.

7. In the herbivora the Tb. are most commonly carried from the cæcum to the mesenteric lymph-glands. In man, the agminated lymph-follicles of the small, and the solitary lymph-follicles of the large intestine also serve as points of entry (cf. Carl Hof, "Ueber primäre Darmtuberculose," Kieler Dissert., 1903. Compare also v. Hansemann, "Ueber Fütterungs Tuberculose," *Berl. klin. Wochenschr.*, 1903, No. 7).

8. Reference to the peculiar features of the stomach of ruminants and remarks on primary tuberculosis of the stomach (Schottelius).

9. The primary development of localized foci in the lesser omentum after infection through the stomach in early nursing period.

10. Peculiarities of the infection of nurslings through the alimentary tract, experimentally and statistically determined.

(*a*) Feeding experiments with anthrax bacilli and other bacteria.

(*b*) Feeding of spores.

(*c*) Tubercular virus behaves in many ways more like the resistant form [spore form] of anthrax virus, especially when the Tb. virus is present in cheesy pus.

(*d*) The observations of Adalbert Czerny and Paul Moser on the occurrence of bacteria in the blood of living human nurslings (1894).

(*e*) Carl Weigert's statements concerning the penetrability, for Tb. virus, of the intestinal apparatus of very young children. (From 1883, cited in *Deutsche medizin. Wochenschr.*, 1903, No. 41.)

(*f*) Raw's communication (*British Med. Journ.*, 1903) concerning 300 cases of *tabes mesenterica*, not one of which was found to have developed in a child nursed exclusively at the breast, but rather in those who had been nourished for a considerable time on cow's milk.

11. It is probable that in thickly populated countries practically every person is at some time or other infected with tuberculosis. Aside from

the quantity and quality of the Tb. virus, the outcome of the tubercular infection is dependent to a high degree on the physiological state of the infected individual and on accidental conditions of infection. (Intercurrent pathological factors; endogenous and exogenous conditions of infection.)

12. Not a single unexceptionable case has been brought forward to show that under the conditions of life usually present in civilized lands, an adult person has ever contracted pulmonary, bronchial, tracheal, or laryngeal tuberculosis without having previously been infected and thus rendered oversensitive to the tubercular poison.

13. Against the action of tubercle bacilli entering the intestinal apparatus, healthy, full-grown persons apparently possess sufficient protection in the character of the mucous surfaces and the anti-bacterial action of the digestive juices. It has, too, still to be proved that healthy, full-grown persons become ill with tuberculosis as a result of eating food (milk, butter, meat) derived from tuberculous cattle.

14. Very probably adult persons frequently acquire intestinal tuberculosis through food containing tubercle bacilli, if the epithelial covering of the intestinal mucosa is defective, or if perhaps there exist ulcers which extend down to the parenchyma of the wall of the alimentary tract. (Exanthematic diseases, typhoid, dysentery, carcinoma, etc.)

15. Whether adult persons in whom the condi-

tions are favorable for an intestinal infection with Tb. will develop primary tubercular lesions in the intestinal wall, or in the mesenteric glands and the peritoneum, will depend mainly on the circumstance whether or not, owing to a previous infection, they have become oversensitive to tuberculin. *Individuals oversensitive to tuberculin are inclined to develop lesions at the point of entry of the tubercular virus*, if opportunity is given for the introduction of the virus by means of leucocytic wandering cells. This opportunity is lacking in the virile infecting period at such places where the lymphatic receptive apparatus is destroyed [verödet]. (Mucous surfaces of the faucial ring of consumptives?)

16. In order to explain the mode of origin of cheesy pneumonias and tubercular broncho-pneumonias it is necessary at autopsy to regard most carefully the possible direct extension of the infection from cheesy mediastinal and bronchial glands to the bronchi and their branches, before thinking of aerogenous or hæmatogenous pathogenesis. (Compare [Ribbert] Sievers, "Marburg Dissertation," Aug. 14, 1902.)

17. Critical analysis of several statistical statements which seek to show that alveolar pulmonary tuberculosis is referable directly to inhaled tubercle bacilli; especially the statement of Knopf (New York) cited by Mitulescu (*Zeitschrift für Hygiene*, 1903), that in Lansing, Mich., twenty employés of a library became consumptive through handling

Tb. laden books.[1] Probability of the correctness of my assumption that Knopf was misled by unscientific communications from Lansing. Proof that Mitulescu again misunderstood Knopf.

18. It has not yet been proved that persons cutaneously infected with human or bovine Tb. have as a result of this developed phthisis. (My own observations on cases of infection on the hand in persons working with tubercle bacilli of various origins.)

19. Justification of the statement by Virchow in his "Phymatie, Tuberculose und Granulie," that "the history of phthisis is concerned much more with cheesy hepatization than with tubercles" (Virchow's "Tuberkel," Begriff).

20. The analysis of the origin of pulmonary consumption must begin with the primary attack (primary regionally as well as chronologically) of the Tb. introduced into the organism.

21. *As a rule we can regard as points of primary infection polynuclear leucocytes in the blood and lymphatic receptive apparatus; next in order are the muscular elements in the walls of the smallest blood-vessels.* Endothelium and epithelium may become carriers of Tb. through the action of polynuclear leucocytes which have wandered into these tissues.

22. In studying the results of an infection with tubercular virus, aside from the degree of virulence,

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[1] See appendix, page 84.

from the dosage, the single or repeated Tb. inoculation, and the primary point of attack (regional and cellular), we have particularly to consider the age at which the primary infection occurs. I distinguish four periods:

(*a*) Infantile period of infection;
(*b*) Puerile " " "
(*c*) Virile " " "
(*d*) Senile " " "

23. It seems to me that in the epidemiological origin of pulmonary consumption the infantile Tb. infection, followed by latent or manifest scrofula in the puerile period, is of great significance, so that we can formulate the following doctrine: "*An infantile tubercular infection predisposes to tubercular pulmonary consumption.*" Under "scrofula" I here include the alteration in the muscle of the blood-vessels, caused by the Tb. infection, which finds its expression in the increased sensitiveness to tuberculin, and which in general is equivalent to "scrofulous diathesis" of the older authors.

24. The primary infection from the mouth or nose with tubercular virus derived from food or even from inhalation, in the small quantities that under ordinary conditions of life are concerned, is followed after the bacilli have entered the circulation, by alterations in the walls of the smallest vessels. These manifest themselves as follows:

(*a*) Microscopically, by a loosening of the vessel wall, between whose elements, shortly after the

infection, tubercle bacilli can be found. These bacilli, brought here by the wandering cells, are set free on the destruction of the cells.

(*b*) In primary functional disturbances which can be recognized by the temperature curve and the heart action.

(*c*) In secondary tuberculin hypersensitiveness.

25. Following mild infections the alterations in the vessel walls may retrogress with a suppression [Beseitigung] of the tubercle bacilli. Without exception, however, the hypersensitiveness to tuberculin remains, varying in degree and length of time according to the virulence of the infecting Tb. and to their more or less locally limited action on the vascular system.

26. After a moderately severe infection there is a formation of transparent, submiliary eruptions (nowadays our "gray miliary tubercle"), especially about the smallest vessels of serous membranes. *These eruptions are capable of becoming organized. In fact, when they have healed they form a tissue entirely identical with the tissue in which they originated.* (Bichat, Lebert, Empis, and many other older investigators.) Aufrecht, *Deutsch. Arch. f. klin. Med.*, Vol. LXXV.

27. The introduction of a tubercular virus so strong as to cause the smallest vessels to become occluded, especially Cohnheim's terminal arteries of the spleen, lungs, and kidneys, results in the exudation of a coagulable fluid and in necrobiosis

of the extravascular region supplied by those vessels. (Aufrecht, *Arch. f. klin. Med.*, Vol. LXXV.) Into this dead area tubercle bacilli are dragged by the wandering cells. These bacilli multiply and cause chemical changes which manifest themselves first in a fatty (steatomatous) and then in a cheesy metamorphosis. (Cf. Koch, Volume II of the *Mittheilungen aus dem Kaiserlich. Gesundheitsamt*," p. 21, and Plate IX, Figs. 45 and 46); my own observations; Aufrecht, l. c.; compare also the critical reference by Virchow to the works of older authors, such as Vetter, Gendrin, Lobstein, Cruveilhier, Bayle, Baillie, Laënnec, Rilliet and Barthez, Vulpian, Craigie, etc., in "Phymatie, Tuberculose und Granulie," and in Vol. II of "Die Krankhaften Geschwülste."

28. We may enumerate as the result of primary tubercular focal disease

(*a*) Anatomically demonstrable residues.

(*b*) Functional alterations.

29. Functional alterations may remain without any anatomically demonstrable residues of the primary infectious processes. I divide functional alterations into

(*a*) Alterations which leave behind them an immunity against living Tb. virus, probably to be sought for in the peculiar condition of the vessel musculature and, in the beginning at least, always associated with hypersensitiveness to the soluble tubercular poisons.

(*b*) Scrofulous diathesis, consisting in such changes in the vascular system and the lymphatic apparatus that a renewed Tb. infection very readily causes cheesy tubercular lesions.

30. As scrofulous infectious processes, I regard

(*a*) Lupus, which I interpret as a cutaneous *additional* effect of a tubercular infection.

(*b*) Gland scrofula,[1] inclusive of tuberculosis of the mediastinal, bronchial, and mesenteric glands.

(*c*) Bone scrofula and joint scrofula.

(*d*) Scrofula in the domain of the external body covering, of the mucous membranes, and of the lymph-channels.

(*e*) Cheesy metamorphosis in internal organs, inclusive of the organs of sense, and the vessel intima.

31. The acute miliary tuberculosis in man, which can be clinically diagnosticated, is to be regarded as a provocative secondary infection, resulting from scrofula of the blood-vessels' intima when, on the disintegration of cheesy intima tubercles, a great many tubercle bacilli are thrown into the circulation at once. (Weigert-Ponfick.)

32. Disease of the lung apices occurring in the

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[1] It will be well to retain the name "Scrofula" for those tubercular processes *outside* of the lung which go on with cheesy degeneration. Only in that way can the connection between the histogenetically so important results of earlier investigations be maintained.

virile period of infection and, because of its important bearing on the origin of consumption, considered separately, is preceded by the consequences of an infantile infection. Foremost among these is the secondary hypoplasia of the smooth muscle tissue (of vessels, bronchi, and intestinal wall); next in order come wasting [Veröding] of the lymphatic apparatus (quantitative and qualitative reduction of the follicular receptive apparatus of the *tubus alimentarius*); destruction of lymph-glands; and the secondary hypoplasia of other primary points of attack for Tb. action (in the spleen, bone-marrow cavities, on serous surfaces of the large body cavities, and of the joints). The predilection of the thoracic dome for immobilizing changes can probably be ascribed to its exposure to the Tb. infection in connection with precedent mediastinal-gland scrofula; while the predilection of the lung apices for caseating lesions can again be brought into causal relation with secondary ossifying processes, of scrofulous origin, in the joint structures of the thoracic dome (cf. Aufrecht, l. c.).

33. In my experiments on tubercular cattle I succeeded in producing eruptions of gray, non-caseating tubercles, running an acute course, by means of injections of tuberculin. At the same time it was noticed that not infrequently after the intercurrent exacerbation had subsided, the old infectious process had been favorably influenced.

34. Even when caseating lesions are present, the

simultaneously developing submiliary transparent eruptions (granulie, of the older authors) are to be regarded as capable of being organized and spontaneously cured. They are much more numerous in man than has heretofore been assumed. This is shown, for example, by the fact that upon opening the abdominal cavity of such young individuals as showed no clinically diagnosticated symptoms of a miliary tuberculosis, these eruptions were by chance demonstrated.

It is of the highest interest to determine the fate of such individuals with a healed miliary tuberculosis of the peritoneum, that is, whether in virile period of life the disposition in them to pulmonary consumption is increased or diminished.

35. The clinical picture of scrofula in the puerile period of infection is etiologically complicated by other infectious processes, especially in the domain of the outer body covering. The functional alterations in the vascular system, due to an infantile Tb. infection, find their expression not only in the organism's increased sensitiveness to tuberculin, but also in a marked instability [labilität] of the dynamic equilibrium of the circulation (lymphatic constitution). In consequence of this, eczematous eruptions due to parasitic and toxic agents result much more readily in these individuals than in those who have not suffered such an infantile Tb. infection (one incompletely healed).

36. The symptoms of the so-called "inclination

to consumption" are the expression of a defective overcoming of the infantile and puerile infectious periods. The hindered development and weakness of the organs made up of smooth muscle fibres (muscles of the blood-vessels, intestinal wall, and bronchi) are comprehensible when we remember the great share that the smooth muscle tissue has in the reaction against the Tb. circulating in the blood. The question whether the weakened conditions and hindered development in the domain of the striped muscles are due directly or indirectly to the Tb. infection cannot be answered without further investigations. Similarly, we do not yet know the mechanism of the origin of the chest anomalies seen in candidates for consumption. A partial impoverishment of the intestinal lymphatic receptive apparatus is presumably accountable for the fact that even a plentiful supply of food is unable to increase the accumulation of fat.

37. The possibility must also be considered that in the course of the puerile period of infection the development of a caseating tubercle may proceed in the joint tissues between costal cartilage and breast-bone, and that such a scrofulous thoracic affection is clinically much less readily diagnosed than a similar affection in bones of the extremities. Further, that such a lesion in the lower extremity is much more readily recognized than one in the upper extremity, since even slight alterations in the bony tissues of the lower extremity, because of

functional disturbances in walking and running, make themselves manifest.

38. For a detailed analysis of the origin of pulmonary consumption, beside the results of infantile and puerile infections, there must still be considered

(*a*) Additional virile Tb. infections (cf. Romberg, *Deutsch. Arch. f. klin. Med.*, 1903).

(*b*) Combined action of a complicating infection.

(*c*) The co-action of general hygienic and dietetic injuries.

39. For my plans for suppression of tuberculosis, therefore, the following points are essential:

(*a*) Prevention of the introduction of Tb. with food, especially with milk, during infantile life.

(*b*) Introducing Tb. anti-bodies with the milk in earliest infancy in order to render innocuous any inhaled tubercle bacilli.

40. I have made separate communications regarding feeding with Tb.-free milk. The production of such a milk will be very easy if my method of cattle immunization in practice fulfils the hopes which I have for it; and these hopes I am convinced are fully justified.

41. In order to produce immunity by means of anti-bodies it is probable that anti-bodies derived elsewhere will have to be added to the milk given to infants.

SUGGESTIONS CONCERNING THE HYGIENE OF COW-STABLES AND THE PRODUCTION OF MILK FOR INFANT-FEEDING, WITH SPECIAL REFERENCE TO TUBERCULOSIS.

The Bonn Rules.

1. Until it has been definitely decided whether or not my protective cattle inoculation protects cattle against tuberculosis when these are kept in stalls or herds strongly infected with Tb. virus, it should be sought to reduce the danger of infection as much as possible by following Ostertag's recommendations and segregating all the cattle with an open tuberculosis.

2. The milk of cows which react positively to tuberculin must not be used for feeding calves, nor, of course, for infant-feeding.

3. Whenever the circumstances permit, the separate housing of the protectively inoculated animals in a stable free from tuberculosis is to be recommended. In constructing new stables it is strongly urged that in place of *one large* stable, several small

ones be erected; besides this, attention should be paid to the placing of the animals. They should be placed with the heads toward the lateral walls, and not head to head, facing a common feeding-trough along the middle of the stable.[1]

4. Particular attention should be paid to the cleanliness of the feeding-troughs. Every fourteen days they are to be filled with hot water and freed from any adherent dirt by mechanical means. The feeding-buckets are to be similarly cleansed from time to time.

5. The drinking-water supplied to cattle should meet essentially the same requirements as are demanded of water supplied to dwellings.

6. In maintaining the health of the young cattle an important factor is the pasturage. For larger dairies an arrangement used at our Marburg experimental station, consisting of a lightly constructed shelter within a large enclosed pasture, is to be recommended.

7. The disinfecting of infected stables is to be done in accordance with the government regulations.[2] [This refers to the German regulations of

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[1] Rules 1, 2, and 3 have been *purposely* neglected in certain dairies in Savár, Teschen, and Mecklenburg, as can be seen by the reports of Messrs. Strelinger, Rösler, and Ebeling (*Beiträge zur experim. Therapie*, No. 8, 1904), without, thus far, injuring the inoculated animals.

[2] § 2 includes the following disinfecting agents:

1. Water, steam, hot water, boiling for one hour.

2. Soap-suds.

June 23, 1880, and May 1, 1894.] Here it is to be remarked that disinfection of the stables by means of formaldehyde generation is not even then accomplished when one far exceeds the requirements formulated by Flügge for the Breslau method. Utilizing the opportunity presented by a number of courses given at Marburg in the method of making protective inoculations, etc., we have found that even in the stable of the pest laboratory here, which is very easily closed off, the above-mentioned formaldehyde fumigation is ineffectual; for by treating the disinfected objects with ammonia water it was found that neither anthrax virus nor cocci, in the dry state, were certainly destroyed by the fumigation.

8. More important than the disinfection by means of chemicals is the prevention of the accumulation of infectious materials on the stable utensils, in the food, on the body surface of the

3. Soda-lye solution (at least 2 kilos soda in 100 liters water).

4. Freshly slaked lime, powder, and milk of lime, 1:2 aq. and 1:20 aq.

5. Sol. chloride of lime, 1:3 aq. and 1:20 aq.

6. Sol. carbolic acid, 5%.

7. Sol. crude cresol, 5% (Liquor cresoli saponatus of the German pharmacopœia).

8. Coal-tar and wood-tar.

9. Fire.

The most reliable and cheapest agent for rendering harmless Tb. virus in stables is hot 2% soda solution, whenever a surface disinfectant is desired. Disinfection of the stable air is as yet to be classed only as a pious wish.

cattle, on the body and clothing of the stablemen. Training the stablemen to the use of warm or hot water and soap; the use of towels and dishcloths which have been rendered germ-free by means of hot water or dry heat before throwing them among the soiled clothes; special regard to a rational care of the milker's hands, including the removal of the dirt under the nails; all of these are the most important measures for the prevention and suppression of stable infection.

9. If it is desired to secure a milk as free from dirt and germs as possible (not over 400 germs per c.c.), the following additional measures must be taken:

I. Presupposing that the milk is to be used for infant-feeding, the same is to be filled into bottles holding 250 to 500 c.c. All parts of the bottle, including the air-tight and germ-tight closing contrivance, must be constructed so as to be readily cleansed, and further, so that they can, without injury, be sterilized by heat.

II. In order to meet all the requirements for cleansing and sterilizing milk-bottles, the following rules are recommended. The bottles returned by the consumers are in a soiled condition and contain fluid remnants of milk:

(*a*) The cleansing of the bottles should be undertaken in a well-ventilated room which can readily be kept clean and which may also serve as the bottling room. This room is to be so

separated from the stables that the stable odors cannot gain access.

(*b*) The following manipulations are required to clean the bottles:

(α) Energetic rinsing with 10% warm soda solution.

(β) Rinsing with quartz gravel by means of a stream of hot water.

(γ) Cold rinsing.

(δ) Sterilization in the dry chamber. These cleansing manipulations occupy about forty-five minutes.

III. The stable itself should be so built that it can readily and thoroughly be cleansed, with special regard to the following points:

(*a*) Draining of the fluid excreta, etc., by means of drains with sufficient pitch.

(*b*) Ready removal of solid refuse without raising dust.

(*c*) A plentiful supply, by means of pipes, of pure, wholesome water. Also a hot-water supply for cleansing purposes.

(*d*) Good ventilation for the high lying stalls.

(*e*) Broad alleys separating the rows of stalls.

(*f*) Each stall to be constructed in such manner that the cow is obliged after feeding to step back, thus compelling her to empty her excreta into an open drain connected with the main drain. This is effected by means of the so-called "drop-railing contrivance."

(*g*) Water-tight flooring.

IV. The spreading of peat instead of straw to keep the stalls dry.

10. Another deciding factor in the production of a pure milk for infant-feeding is the cleanliness of the stable and dairy attendants, the milkers, the cows, and the stable and dairy utensils.

I. Aside from general body cleanliness, particular attention is to be given to a healthy condition of the milking person's hands. Before proceeding to milk, these persons are to don a clean gown made of white linen.

II. The cows are to be kept scrupulously clean. The udders and tails are to be so clean that they can be touched with white-kid gloves without causing appreciable amounts of dirt or of odorous substances to soil them. In order to keep the udder and teats clean the following is recommended:

Just before milking, the udder, which should always be kept dry, is to be rubbed off with a clean flannel cloth which has been greased with a little lard. By this means dirt and odorous substances are most readily removed. Following this, the parts are rubbed dry with the aid of a little bran.

III. The milk vessels should be of tinned sheet iron.

IV. The milk should be strained through aluminium-nickel gauze or brass gauze, which is then to be cleaned and disinfected.

V. All the milk of one milking period is pumped

high into the bottling-room and conducted into a collecting reservoir. This passage to the reservoir, during which it is cooled and aired, occupies only a few minutes and the milk reaches the reservoir at a temperature of 4°–5° C.

VI. From this reservoir the milk is bottled without delay by means of a bottling-machine, the milk having a temperature of about 5°–7° C. on bottling. *A temperature of less than 2° C. has an injurious influence on the anti-bacterial substances in the milk, and should therefore be avoided.*

11. If the milk hygienic rules above mentioned are carefully followed, and if attention is paid to the experiences regarding a rational method of feeding milch cows, it is possible, without any further method of preserving the milk, to have the same capable of being transported and kept for sixteen hours without losing its adaptability as infant food. In Berlin I saw such a milk reach the consumer with a germ-content of not over 1000 germs per cubic centimeter. On the other hand, I have seen milk derived from dirty cows and collected without particular hygienic precautions, after being transported six hours, contain a hundred to a thousand times as many germs. In such cases, soon after milking, the milk often contains from 30,000 to 100,000 germs per cubic centimeter.

In the strictly fresh milk from my own cows the number of germs per cubic centimeter varies from 10 to 50 to 200. Such milk kept at room

temperature does not coagulate until after three to five days.

At present I regard 1000 germs per cubic centimeter at the time of feeding to be a safe limit for milk fed to infants.

12. If a milk collected in accordance with the preceding regulations is to be made capable of being transported and kept for three days (in which case coagulation factors must be reckoned with) without losing its qualifications as an infant food, the addition of formaldehyde in the proportion of 1 : 40,000 (B. f. milk) is to be recommended.

The following manipulations are then to be carried out:

I. Preparing a formaldehyde stock solution containing 0.5% formaldehyde. (One part of commercial formaldehyde and 79 parts of boiled water.) This solution will keep for two or three days.

II. Pouring 50 c.c. of this 0.5% stock solution (=.25 gramme formaldehyde) into an empty tin pail in which a mark has been made at the 10-liter level.

III. Milking into this pail up to the mark.

IV. Straining, airing, cooling, etc., according to paragraph 10, IV, V, and VI.

13. In order not to conflict with the government sanitary regulations the milk producers at present delivering this B. f. milk have agreed to supply it only to certain private institutions designated by me.

14. Should the authorities later decide to allow the sale of this B. f. milk, it is recommended that

licenses be issued, out of the proceeds of which veterinary physicians be appointed to see that this B. f. milk is produced in accordance with these regulations.

15. Containers for B. f. milk should have trade-marked labels indicating that the milk has been produced in conformity with the requirements. Beside this the label should bear in clear figures the time and date of milking.

16. Whenever for any reason it is impossible to raise a calf directly on the udder, B. f. milk should be preferred to milk sterilized by heat. In the production of B. f. milk tubercular and suspected tubercular cows are to be excluded on principle.

17. Where it is desired to suppress an infectious process in the digestive system of cattle, the temporary employment of a stronger formaldehyde milk (1 : 25,000 to 1 : 16,000) under the direction of a veterinary physician is recommended.

18. In the suppression of "*Kälbersterbe*," beside the increased requirements regarding the cleanliness of attendants' clothing and body, of stable and stable utensils, the proper care of the navel with the aid of a $\frac{2}{5}\%$ aqueous solution of formaldehyde is recommended.

19. The radical suppression of bovine tuberculosis by means of my protective inoculation is to be sought for.

20. The following article shows the method of making these inoculations at present in use at Marburg.

SYNOPSIS FOR PROTECTIVE INOCULATION OF CATTLE IN AGRICULTURAL PRACTICE.

GENERAL CONSIDERATIONS.

For the protective inoculation carried out by us in practice we make use of "Mxt. Tb." in the form of our "Culture No. 1," which has been studied by us for years and which we have minutely described in the *Beiträge*, Nos. 5 and 6. The accumulation of orders for this virus on the one hand and the multitudinous experimental labors in the institute here on the other, compelled us to rid ourselves of the technical labor of manufacture, weighing off and shipping of the virus, which accordingly has been undertaken by the local firm of Drs. Siebert & Ziegenbein. The virus, however, continues to be tested as to purity, virulence, etc., in the scientific department of the institute at Marburg.

The virus is accompanied by directions for use which have already appeared in the *Zeitschrift für Thier-medicin.* These are as follows:

I. Selection of the Animals to be Inoculated.—As a rule only animals without external evidences of disease, from three weeks to three months (for the first inoculation) should be inoculated. In healthy animals of this age a previous tuberculin test is unnecessary, even if the animal comes from a notoriously tubercular herd.

II. Numbering the Inoculated Animals.—Every inoculated animal must be marked with a running number. The marking must be of such a character as to be distinct, not liable to be mistaken for some other, and to last the lifetime of the animal. (Ear-marking, tattooing, etc.) If necessary, the marking is to be repeated should the first mark become indistinct.

III. Keeping of Records.—See under "Conditions governing the distribution of the virus, etc.," p. 75.

IV. Technique of Taking the Temperature.—The body temperature is determined by means of a self-registering thermometer completely inserted into the rectum. Before introducing it, the rectum is to be cleared of any hard fecal masses. A tape about a foot long, having a clamp at its end, is tied to the neck of the instrument. The thermometer is left in the rectum for four minutes, the clamp meanwhile being fastened to the hairs at the root of the tail. In order to save time, thermometers are introduced into a number of animals (about six) consecutively. When the last thermometer

has been inserted it will usually be time to extract and read the first. In this way one can take the temperature of fifty head of cattle in 1–1½ hours.

V. The Virus.—The inoculating virus consists of living tubercle bacilli whose action has been accurately tested in the Marburg Institute for Experimental Therapy. The tubercle bacilli have been dried without losing their vital powers in any way. These dry tubercle bacilli (Trocken Tb.), kept in sealed glass tubes, will retain their action on cattle unchanged for a period of thirty days. If, therefore, a tube of Tb. bears the date VII–1–02, the contents can be used for cattle immunization until VIII–1–02. After thirty days, although the immunizing power is not entirely lost, it is so far decreased as to render it ineffective in the dosage recommended.

VI. Dosage of the Virus.—For the first inoculation, one immunizing unit, 1 I. E. [=1 Immun Einheit] is used for each calf; for the second inoculation, which is not to be undertaken until at least twelve weeks after the first, five units (5 I. E.) are used for each calf. As a rule the dose of 1 I. E. is 0.004 gramme dry Tb., that of 5 I. E. for the second inoculation is therefore 0.02 dry Tb.

If the tube contains the quantity requisite for the first inoculation of twenty cattle, it will bear the label 20 I. E. In order at all times to control the manner of production of the dry Tb. each tube

also bears in Roman numerals the consecutive laboratory number thus:

Op. No. IV.
20 I. E.
VII–17–02

This label is interpreted as follows:

Dry Tb., which in the records of the Marburg Institute bears the laboratory number IV, contain on the day of delivery, i.e., on VII–17–02, twenty immunizing units, and retain this strength until viii–17–02.

For the inoculation the virus is uniformly mixed with 1% salt solution which has previously been boiled and cooled. The procedure is as follows:

The entire contents of the tube are placed in a small mortar and crushed with the pestle. Then 2 to 3 c.c. salt solution are added and the whole rubbed into a uniform mixture or emulsion, after which it is poured into a graduated cylinder holding 50 c.c. A little more salt solution is then added to the mortar, and thus the remaining particles of virus are added to the previous mixture. The fluid in the graduate is then made up to 30 c.c. with salt solution, and then poured into a sterile wide-mouthed bottle holding 100 c.c. Any remaining emulsion is then washed out of the graduate with an additional 10 c.c. salt solution and added to the 30 c.c. in the bottle. The bottle thus contains virus ready to inoculate, and each 2 c.c. will be the dose for the first inoculation of a calf, provided that the tube originally contained 20 I. E.

The dose for a second inoculation would then be contained in 10 c.c. of this fluid.

VII. INSTRUMENT CASE.—In order to carry out the inoculations in agricultural practice, the instrument case constructed by W. Holzhauer, Marburg, is to be recommended. It contains the following:

(*a*) Two bottles for the virus, each 100 c.c., made of colored glass.

(*b*) One bottle lysol.

(*c*) One bottle alcohol.

(*d*) One Erlenmeyer flask (for 1% salt solution).

(*e*) One mortar and pestle.

(*f*) One graduated cylinder 50 c.c.

(*g*) One graduated cylinder of 10 c.c.

(*h*) One basin to hold disinfectant solutions.

(*i*) Six thermometers with tapes and clamps. (Price 10.50 marks or 1.75 marks each.)

(*k*) Two virus syringes with two strong canulas. (Price 11 marks.)

(*l*) One small hand-scale and weights.

(*m*) One alcohol stove and cover.

(*n*) Sterile cotton.

(*o*) One wire basket.

The entire box is supplied with a canvas cover and a leather handle. The lid, which bears four movable legs, can, when the same is removed from the box, be set up like a saucepan and used as a sterilizer. For this purpose the vessels and instruments are placed into the wire basket, the sterilizer half-filled with 2% lysol solution, and the whole

heated just to boiling by means of the alcohol stove.

The price of the complete outfit is 85 marks. By omitting the thermometers and syringes the price is reduced to 63.50 marks.

VIII. Method of Making the Intravenous Injection.—The injection is best made by means of a 5-c.c. glass syringe with an asbestos plunger (paragraph VII, *k*), which is cleaned by means of the lysol solution and rinsed in sterile salt solution. The canula attached to the syringe is cleaned in the same manner. One immunizing unit (1 I. E.) is then drawn up into the syringe and any air-bubbles are expelled, care being taken that any drops of fluid expelled in this manner do not fall on the floor but are caught up in the basin. The filled syringe, together with the canula, which is now detached, is then placed on the previously mentioned basin. The virus is injected into the left jugular vein. When possible the cattle should be inoculated, each in its own stall, so as to avoid exciting them. After washing the left side of the neck with 2% lysol solution, the operator, by pressing his thumb on the jugular vein, stops the circulation, causing a distinct, sausage-shaped, fluctuating swelling to appear. With his right hand he now takes the canula from the basin and thrusts it, just above the compressing thumb, upward into the vein at an angle of 45°. One recognizes that the vein has been entered, by the blood that at once flows through the canula. If no blood flows, the vein has not been entered.

In that case the canula is withdrawn slightly but not completely, and again thrust into the fluctuating swelling. As soon as blood flows, the left hand ceases making pressure and grasps the canula, whilst the right hand fits the syringe thereto. Then the virus is slowly and uniformly injected into the vein. When the syringe is emptied, the skin at the site of injection is pressed together and the syringe and canula withdrawn. As a rule, bleeding ceases almost at once. The region is then rubbed over with 2% lysol solution and the inoculation is completed.[1]

CONDITIONS GOVERNING THE DISTRIBUTION OF THE VIRUS PROTECTIVE AGAINST TUBERCULOSIS.

1. The virus is supplied by the firm of Drş. Siebert & Ziegenbein in Marburg a. d. Lahn, Germany.

2. This firm supplies the virus in packages containing 5 I. E. and 20 I. E. The price for the former at present is 40 pf. per I. E. and for the latter 25 pf. per I. E. In ordering, the style of package is to be specified.

3. The recipients bind themselves to fill out and return the following records:

A. Temperature charts: The temperature is to be recorded as a curve.

B. Other data as per blank herewith enclosed (see next page).

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[1] See appendix, page 84.

A	B	C	D				E			F				G		H	I
Consecutive Marburg Number.	Consecutive Inoculation Number.	Breed, Herd and Designating Number.	Nationality.				Manner of Raising.			Protective Inoculation.				Tuberculin Test.		Weights.	Special Remarks.
			I.	II.		III.	IV.	I.	II.		I.	II.	III.	IV.				
				Location.					Artificial.									
				A	B				a	b								
			Owner.	At Birth.	At Time of Inoculation.	Breed.	Sex.	Natural (Suckling Calf).	With Non-sterilized Milk.	With Sterilized Milk.	Laboratory Number of the Virus.	Dose in Grammes or in c.c.	Date and Manner of Inoculation.	Degree of Reaction.	Date.	Degree of Reaction.		

DIRECTIONS FOR FILLING THIS BLANK.—Column A is to be left blank by the attending physician; column B contains the running number of the animal inoculated; C is to be recorded only in fitting cases; D, on the other hand, is never to be omitted; under E the fitting column is to be marked thus: "!"; in column F, IV, the degree of reaction following the inoculation is to be noted by signs, thus: O= Failure of any reaction whatsoever (R= O); I= short febrile reaction; II= 2–4 days of fever; III= 5–8 days of reaction associated with other disturbances of health (cough, loss of weight, diminution of appetite, diarrhœa, etc.).

In the same manner the degree of the reaction to a later tuberculin test is to be recorded (column G). In column H the weight is always to be accompanied by the date.

The returns are to be made as soon as the data for about 100 *cattle have been secured.*

C. General report: This includes a report on the manner of keeping the inoculated cattle, the hygienic conditions of the stables, previous occurrence of *perlsucht* in the cattle of the herd, previous use of other means of suppression of tuberculosis and their result, etc., etc.

4. The owners of the cattle must see to it that the inoculations are properly made and they themselves must bear all consequences resulting from defective inoculation. Drs. Siebert & Ziegenbein can in no way accept responsibility for this.

The preceding conditions are still adhered to in many large dairy farms. To a number of smaller dairies we have allowed easier conditions. An example is afforded in the following order of the Ministry of the Interior of the Grandduchy of Hesse.

MINISTRY OF THE INTERIOR OF THE GRANDDUCHY OF HESSE,

DEPARTMENT OF PUBLIC HEALTH.

To the Grandducal County Veterinarians:

Whereas, following the proclamation of the Ministry of the Interior dated Aug. 19, 1902, a large number of replies has been received from the farmers, it is ordered that the inoculations be hereafter made in accordance with the following principles:

1. The protective inoculation consists in the intravenous injection of a *single* immunizing unit of tubercle germs (derived from man), rubbed up with sterile salt solution; and in a second intravenous injection, three months later, of *five* immunizing units of the same germs.

2. The germs in question can be obtained in glass tubes containing 5 or 20 units.

3. As a rule only cattle without external manifestations of disease, aged from three weeks to four months (at the first inoculation) should be inoculated. With healthy animals of this age it is unnecessary to test them previously with tuberculin, even if they belong to a notoriously tubercular herd.

4. In exceptional cases older animals (from four months to two years) may be inoculated with the virus, but only when they are entirely free from disease, *and when a tuberculin test made on them results without any reaction whatsoever.*

5. The inoculated animals are to be kept in their stalls two days before and two weeks after the inoculation.

6. In all cases in which the owners can manage the rectal temperatures, the same are to be taken two days before the inoculation (morning and evening) and again on the morning of the inoculation. After this they are taken once more in the evening and then once daily for the next five days. If, after this, the animals still have temperatures above

39.2° C., the temperatures are to be taken daily until the same reach 39.2° C.

7. In animals not over four months old at the time of the first inoculation, the temperature may be omitted if there be any difficulties in taking it. In those, however, which are over that age at the time of the first inoculation, the temperature must invariably be recorded according to paragraph 6. Even when inoculating animals in infected herds the above-mentioned measurements should be taken whenever practicable.

8. If possible, the inoculated animals should be weighed every fourteen days on a cattle scale.

9. Owners of cattle are to keep record of all the temperatures and weights. You are to collect the records concerning temperatures as soon as they have been completed; the records of the weights, however, only after three months. The results noted in the temperature returns are to be entered by you on the charts herewith provided, and a temperature curve plotted. These charts are to be made in duplicate, one copy to be sent to our representative and the other to the Experimental Division of the Hygienic Institute, Marburg.

10. The inoculated animals are all to be permanently marked. Those on which temperature and weight observations have been made should also be numbered.

The method of thus marking and numbering the animals is left to your judgment.

11. Should you not be informed in regard to the previous notifications respecting the inoculations, kindly place yourself in communication with the grand-ducal "Kreis Amt." Following this, as soon as you have informed yourself regarding the age and health of the animals to be inoculated, notify our representative as to the number of these animals suitable for inoculation. The reports for each village are to be made out separately.

12. The necessary virus and utensils, as well as explicit directions for use, will be furnished by us.

13. We shall see that your first inoculation is attended by our representative.

(Signed) ————

DARMSTADT, Nov. 16, 1903.

APPENDIX.

NOTE TO PAGE 28.

. . . Nowadays, however, there are few authorities who do not admit that tubercle bacilli which have gained lodgment in the nasopharynx or in the oral cavity may cause *primary lesions in the lung* in an entirely different way [different from direct infection by inhalation], namely, through *lymphogenous* or *hæmatogenous channels* after an *intestinal infection* (the intestine reckoned from the pharynx down through the stomach to the rectum).

Whether this inhalation of tubercle bacilli from the air into the nasopharynx is followed by a pulmonary tuberculosis, and whether, if it be demonstrated that this actually occurs, the disease is to be ascribed to *intestinal* or *pulmonary* infection, is the thema probandum. . . . How little I deny the possibility of an inhalation of tubercle bacilli and the consequent danger of infection to many persons can be seen from two quotations, one from my Vienna lecture, March 12, 1903, and the other from the lecture held at Cassel, Sept. 27, 1903.

"In my plans for the suppression of tuberculosis

I give the central place to the feeding of infants with milk both harmless and capable of healing. But as already stated in my previous communications, it is absolutely important to keep coughing consumptives away from the nursing infant. The tubercular virus scattered throughout the dwelling of the consumptive surely finds its way in one way or another into the mouth of the nursling, and then, just like the tubercle bacilli in the milk, it reaches the lymph-vessels and the circulation. It will, of course, also get into the infant's milk, and when, therefore, in my lecture in Cassel, I stated that 'the milk fed to infants is the chief cause of consumption,' I presumed that my auditors and readers were familiar with the following paragraph of my Vienna lecture: 'If I may apply the experiences gained in studying the conditions under which tubercular and other cattle are housed, etc., I must say that model hygienic housing can do little to prevent the spread of tuberculosis, if at the same time the scattering of tubercle bacilli by the cattle with open tuberculosis is not prevented or at least lessened. It is absolutely impossible to determine all the numerous ways in which tubercle bacilli coughed up, or otherwise scattered about by the tuberculous animals, will finally reach the organism of the other cattle in the stable; and I regard it as almost impossible, by means of mechanical or other measures, to prevent the spread of virus from a case of open tuberculosis to the other inmates of the

same room. For adults who, in healthy circumstances, possess a strong protection against the action of the tubercle bacilli, in the form of the epidermis, the epithelial covering of the mucous membranes, and the anti-bacterial ferments, I regard the danger of infection as not particularly great, provided, of course, the dose introduced is not as large as in our laboratory experiments. On the other hand, the new-born, whose mucous membrane is so very permeable and lacks the protective ferments, are so very much exposed to the danger that I cannot conceive such individuals escaping infection if they be in the same room with a coughing consumptive. It has been amply proved that the air can carry not only dry but also moist tubercle bacilli. If, then, this virus be present in the air (in a measure 'volatile'), its introduction into the mouth and so into the intestinal tract cannot be avoided.

"These facts apply equally well to infants and to new-born calves."

NOTE TO PAGE 51.

Mar. 3, 1904.

Excellence E. von Behring, Marburg, Germany.

My dear Professor Behring:

I have seen in recent discussion, reported in the medical journals, a reference to Dr. Knopf in regard to the infection of record-books, which he attributed to an occurrence in the Health Bureau of Lansing, Michigan, and seems to have reported in one of the French journals.

Dr. Henry B. Baker, who is the Secretary of the State Board of Health of Michigan, and who is a personal friend of mine, has been applied to for more particular information, and it appears that the matter refers to a newspaper (published in Detroit Oct. 14, 1899) and *undoubtedly refers to a claim made by some one in Russia, where such an infection is said to have taken place.*

I send you herewith a copy of the notice that has been sent out by the Lansing Health Bureau, and may say that Dr. Baker writes me that nothing of the sort has occurred in the office at Michigan.

I would be greatly pleased if you could send me a copy of Dr. Knopf's publication in the *Press Medicale* to which you have referred. .

Thanking you in advance, I remain

Sincerely yours,

KARL VON RUCK.

NOTE TO PAGE 75.

. . . One unpleasant possibility [in the making of inoculation] is to be mentioned, one which I did not originally think of and which was called to my attention by Ober-med. Rath Lorenz. It consists in the general collapse of the inoculated calf, of very threatening aspect, when the inoculating fluid has been injected *too cold*. This happened on a Hessian farm on a cold winter day, and resulted in the collapse of *all* the inoculated calves. (It would, by the way, have happened if only the cold fluid without the virus had been injected.) As it was, all the inoculated animals had recovered by the following day. This teaches us to warm the inoculating fluid to about body temperature before injecting it. This is readily done by placing the bottle containing the fluid into a little lukewarm water.

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