THE CONTRIBUTORY CAUSES

of

PULMONARY TUBERCULOSIS.

By

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C O N T E N T S .

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Chapter .... 1. Introductory.

In submitting for your consideration some observations on the "Contributory causes of Phthisis Pulmonalis," I am deeply conscious of the vastness of the undertaking, and of my inability to do more than touch the fringe of the subject. At the same time I have hopes that although I may not add anything new to the information contributed by able investigators, it will be some satisfaction to be able to confirm what has been written by those who have not only had great opportunities, but by reason of perseverance and ability, have been able to make use of them.

Of all diseases which attack the human race, phthisis pulmonalis, or consumption of the lungs, is, in our country, the most fatal. No disease, therefore, deserves more consideration from the Medical Profession, and no subject has for many years occupied so many pages in medical literature as this insidious and treacherous disease. For the purpose of obtaining the necessary material to enable me to come to some decision on the contributory causes of phthisis, I have, for the most part

relied on the information gained by a personal enquiry into the deaths from consumption in a Rural District of about 25.000 people in which I have acted as Medical Officer of Health for the past ten years. Little of this information was obtainable until after the death of a patient, but in 200 of the cases careful enquiry was made as to occupation, family history, condition of the patient prior to the supposed onset of the disease, the possibility of direct infection from other persons in the same house suffering from consumption, overcrowding, social conditions, insanitary environment, housing, habits, and in a less satisfactory degree, the food of the deceased. Further, information will be submitted as to the Geological formation of the District. Each case has been carefully tabulated and the results have been most instructive. In a less methodical manner, I have gained valuable information from the study of many of the cases I have seen in private practice. I also profited considerably by a short holiday to Davos Platz, Switzerland, where tuberculosis forms the principle topic of consideration, both amongst the laity and the medical profession.

In a lecture delivered before the Guy's Physical Society on February 28th, 1846, Dr Addison makes use of the following sentences:- "Credultiy may be less unamiable than Scepticism; we may be disposed to regard the former as an infirmity untainted by selfishness, whilst we look upon the latter as too often blended with envy, hatred, or malice; still are they both alike opposed to the advancement of truth." With Addison's

grave warning before me, I have attempted to classify such observations as I have made in an open and honest spirit, at the same time examining them in all their bearings, so that if possible I might not become the victim of either credulity or scepticism.

As grief, pain, and the necessities of man's body and mind called for a cure before there was any reliable knowledge of disease, it may be advantageous to mention some of the views of earlier writers in phthisis, so that we, who have the fuller light, may appreciate the difficulties that have been overcome by those whose work has been incessant in this field of labour. φθίσις (φθίνω = I waste) occurs frequently in the early hippocratic writings, and means pulmonary consumption.

The early Greek physicians distinguished between Empyema as suppuration outside, and phthisis as suppuration inside the lung. The following is a quotation from Celsus:- "The third kind of decline (tabes) and by far the most dangerous is what the Greeks have named φθίσις. It usually arises from a cold in the head, whence it settles in the lungs and then causes ulceration. Then follows a slight feverish movement, which remits and comes again. There is a constant cough, raising of yellow matter, and sometimes of blood."

It was not, however, until the beginning of the 19th century that a clearer knowledge of this disease was to be obtained, and perhaps no man, excepting Koch, has done more to elucidate the pathology of this disease than Laennec. In 1819 he taught that the existence of tubercles is the cause, and constitutes

the true anatomical character of consumption. Laennec's views were not, however, accepted by many of the eminent physicians in the first half of the 19th century.

In a paper "On the Anatomy of the Lungs," read before the Royal Medical and Chirurgical Society. April 23rd. 1841. Addison made the proposition that pneumonia and inflammatory tubercle were identical.

In his paper on the "Pathology of Phthisis" read before the Guy's Physical Society, Jany 4th, 1845. Addison argued that the two disease, pneumonia and phthisis, follow one another and that phthisis is but a secondary affection to the pneumonia. He did not consider the tubercle, per se, to have much to do with the disease, as can be gathered from his words:- "By tubercle-pneumonic phthisis is meant a very common form of the complaint, in which although tubercles are present, the really efficient cause of the phthisical mischief is pulmonic inflammation. In this form of the disorder tubercles sufficiently indicate the strumous or cachetic habit of the individual, and manifestly predispose to the inflammatory change; nevertheless, they do not, beyond this, seem to be either primarily or essentially concerned in the serious changes observed to take place in the pulmonary tissue."

Again in that same paper he states:- "It may be fairly doubted whether in perfect strictness of language, the term tubercular, taken in an exclusive sense, can with propriety be applied to any form of phthisis; for, however strange and paradoxical it may appear, I venture to submit that at the present moment we are not in possession of any conclusive evidence

that either tubercle or its disintegration ever constitues the primary and essential cause of the disorganization which characterises fatal phthisis. We have phthisical disorganization without tubercle, and we have phthisical disorganization with tubercle."

Clearly, then, Addison looked upon phthisis as being in no direct way connected with the manifestation of tubercle, although he admits that the existence of tubercles made the patient susceptible to phthisis. He believed that "inflammation constitutes the great instrument of destruction in every form of phthisis," but he failed to discern tubercle as being the cause of the inflammation. Niemeyer in 1867 taught that in the majority of cases, tubercles, if found in the lungs after death, have been of recent origin, and have complicated the disease when it was already in an advanced stage."

Dr Lathan observed that "The pulmonary consumption is no more than a fragment of a great constitutional malady."

In Watson's "Principles and Practice of Physic", (1857) he states that "tubercles themselves are composed of unorganized matter deposited from the blood, of a yellowish colour, opaque, friable, and of about the firmness and consistence of cheese."

Graves, in his"Clinical Medicine" Vol. 11, p. 114, states as follows:- "It is obvious that phthisis may prove fatal by the rapid and extensive development of tubercles, without any of the peculiar phenomena of pneumonia or bronchitis; yet it most commonly happens that owing to their being produced by the same cause, we have the three different forms of scrofulous

inflammation in the same phthisical patient, although it is by no means rare to meet with them in a separate and distinct state.

Another way in which inflammation acts as a cause of tubercular development, I must not omit stating, namely, by bringing more generally unhealthy blood to the lung, and thus encouraging the formation of morbid deposits; and this leads us to the consideration of another question, why are tubercles so common and so copious in the lungs more than in any other tissue.? I believe there has not been as yet any satisfactory solution of this phenomenon; but it may tend to throw some light on this obscure subject, if we call to mind one of the most striking peculiarities of the lung, namely, that it is the only organ through which the entire mass of the blood circulates.

It is in the lungs also that the change which the blood undergoes takes place exclusively, and its particles experience that mutation which renders them subservient to the purposes of life.

Tubercles are a disease of nutrition, a process which depends intimately on the blood; and it may not seem strange that they should be most frequent and numerous in an organ which has a more intimate connection with the sanguineous circulation, than any other."

It is evident that Laennec had perhaps a clearer idea of phthisis and its pathology, than many of the eminent men who succeeded him. Further, he recognised, what we now recognise

that phthisis has most frequently a predisposing cause, as instanced by his remarks on mental anxiety, depression of spirits, and several diseases which frequently lay the foundation for phthisis.

In speaking of some of the religious orders of France, particularly those to which females are attached, he says that it is to be lamented that they were so unreasonable in their mode of life; for the confinement and want of variation and exercise which attend their mode of living, concurring with their rigid abstinence, produce consumption in a few years. While Laennec knew nothing of the existing cause (tubercle bacillus) he clearly recognised that the anatomical lesion, (the tubercle) was the cause of other changes in the lungs, and that certain habits and conditions of the body were frequently predisposing causes to this disease. Little more than he taught in the beginning of the 19th century was added to our knowledge, until Cohnheim in 1880 demonstrated that tubercle was an infective disease by inoculating the anterior chamber of the eye with tubercular matter. This almost seemed a preparation for the great announcement in March 1882 that Koch had discovered the causal organision. Perhaps there have been few discoveries in medicine which have had such far reaching results, not only in enabling us to decide what is tubercular, and what is not, but holding out hopes that in the spheres of prevention and cure of this fearful malady, a fuller knowledge of this organism and its products may still prove to be a great boon.

Pathologists are now agreed that Koch's bacillus is not

only present and can be demonstrated in all cases of tuberculosis, but that it is the causal organism. They are further agreed that such a bacillus must gain an entrance to its host from without, and the only known sources of infection are man and animals affected with the disease. The continuation of tuberculosis, therefore, depends on the transference of the bacillus from host to host.

From these facts, phthisis pulmonalis may therefore be classified amongst the infectious diseases, since infection is the causa sine qua non, and the disease cannot generate de novo. But, in my opinion, there is one great difference between phthisis pulmonalis and most of the other infectious diseases. "I have heard", says Dr Burton-Fanning, "on the highest authority, that consumption has never been known to attack any of the numerous visitors at Davos, or any of those engaged in occupation there, who were free from tubercular taint on their arrival."

It is a recognised fact that medical attendants and nurses of consumptive hospitals rarely contract pulmonary tuberculosis, and this was the case when the nature of the disease was not well understood, and the precautions then taken were not such as would be considered efficient at the present time.

There has never been any suspicion that the incidence of the disease has been more marked in the neighborhood of any Sanitorium than in adjoining districts, and indeed in the village of Görbersdorf, the incidence of consumption has decreased since Brehmer established his sanitorium there.

In the sweeping of a street of any good sized town, in the

railway station waiting room, in many large, but badly ventilated and dusty halls, where the consumptive, along with the general crowd, may enter, and of necessity must cough and disseminate amongst those around him a plentiful supply of bacilli, there is every opportunity for infection.

In circumstances similar to these, the average man of the town must of necessity drift into weekly, or even daily, and yet without any bad results. Even making allowance for the rapid destruction of the organism by sunlight and fresh air, there must be a very large proportion of humanity who are constantly being infected with the bacillus of tuberculosis, and yet without harm.

With a similar exposure to the infection of scarlet fever, and in the unvaccinated to small pox, there can be little doubt that the results would be very different. If a large number became exposed to infection and only a few acquire the disease, surely the factor of susceptibility to the disease is of greater consequence than even the causal organism. In my opinion this is of still greater consequence in the prevention of pulmonary consumption. For many years prior to the discovery of the bacillus by Koch, the death-rate from the disease had been on the decline. The passing by Parliament of every Public Health Act and Factory Act, has resulted in a further diminution of the death-rate from phthisis by the exterminating of those slums and areas of overcrowding, the carrying out of land drainage, and efficient sewerage, and the improvement of the conditions of labour in the Factory, and the Mines. This surely, indicates the removal of some of the contrubutory

causes.

Although typhus fever is undoubtedly due to some specific organism, the recognition that overcrowding, want of ventilation, filth, debility and privation, were the predisposing causes, has enabled sanitary authorities to make the disease one of the past, and although, now and again in this country some cases make themselves manifest, they are always associated with the predisposing causes mentioned.

It will, therefore, be seen that in typhus fever, although the isolation of the infected persons and the disinfection of the premises are necessary factors in its prevention, the most important, to have any permanent result, must be the removal of the conditions on which typhus is known to thrive. So in phthisis pulmonalis, while it is of very great importance to destroy all tubercular matter, and to teach the patient how to prevent infecting others as well as himself, it will be of still greater importance, so far as possible, to remove these conditions which are favourable to the development of phthisis, whether they be in his environment or in himself.

Let me, therefore, briefly discuss such conditions or contributory causes favourable to the development of pulmonary consumption, and submit for your consideration such evidence as I have been able to accumulate on these points. First, let me consider such contributory causes as affect the environment of the patient; but before doing so, I beg to state that there is no rule without exceptions, and that the good or evil effects in the prevention of phthisis are frequently nullified by other conditions which are unfavourable or favourable.

CHAPTER.....2.

CLIMATE AND ITS RELATION TO PULMONARY TUBERCULOSIS.

Climate.

According to Dr. Theodore Williams, the chief factors of any climate are:-

(1) Latitude.

(2) Altitude.

(3) The relative distribution of land and water, and especially the presence of vast tracts of either desert or ocean, the former accentuating extremes of temperature, and the latter tempering them.

(4) The presence of ocean currents flowing from higher and lower latitudes (as the case may be) and qualifying thereby the climate.

(5) The proximity of mountain ranges, and their influence on the shelter from wind, and on the rainfall.

(6) The soil -- Its permeability and impermeability to moisture.

(7) The rainfall -- Its amount, and annual distribution.

(8) The prevailing winds.

Latitude.

The only effect which latitude can have on the incidence of pulmonary tuberculosis, is temperature, and temperature, per se, is no hindrance to its prevalence. Temperature, however, is not altogether governed by latitude. Quito, the capital of Ecuador, on the equator, has an altitude of 9,500 feet, and possesses the climate of a perpetual spring, having

a mean temperature of 60 °F. for every season. Moosne, a trading station on the north shore of Hudson's Bay, is in the latitude of London, but enjoys a typical arctic climate. There are, therefore, other conditions affecting temperature besides latitude. Pulmonary tuberculosis occurs in all climates, tropical, temperate, and arctic, and although there are no very reliable statistics for the purpose of comparison, there is an undoubted marked prevalence in the temperate zone, especially in the big centres of population.

Many authorities consider that phthisis is much more rapid in its course in the tropical countries. Severe cold does not seem to act in any way as a preventative. Of 162 entries in the death register at Moosne, 68 were due to consumption.

There is distinct evidence, on the other hand, to prove that natives of a tropical climate going to dwell in a temperate climate, and even natives of one tropical climate going to dwell in another tropical climate, are frequently the subjects of pulmonary tuberculosis.

I have it on the authority of a negro medical gentleman from the west coast of Africa, that the majority of natives who come to study medicine in England from that district, die from pulmonary tuberculosis before completing their curriculum.

According to Dr. Andrew in his Lumleian Lectures on the Etiology of Phthisis, there are tropical countries where phthisis is little known. He instances Senegambia, and on the Gold Coast, where at that time, out of 4.000 cases of sickness annually, not one was a case of phthisis. His conclusions on latitude in the etiology of phthisis are as follows:- "On the whole,

the facts, as far as they are known which bear upon the relation between climate and phthisis do not justify any more distinct conclusion than this, viz: that, as a matter of fact, phthisis may, and does exist in any climate, and that the influence of climate, whatever it may be, so far as it depends upon latitude, is but slight, and constantly modified or overridden by other more potent conditions."

ALTITUDE.

Of all climatic conditions affecting pulmonary tuberculosis, altitude perhaps plays the most important part. Several explanations have been given as to the favourable influence of altitude. The purity and aseptic condition of the air in the mountainous district has been advanced as a sufficient reason for the beneficial effects obtained. In the Alps and Rocky Mountains, infusion of meat will keep for a long time exposed to the air in winter, but in the mountain villages where there are large aggregations of human beings or animals, putrefaction soon sets in. Further, although mountain races are specially immune from tuberculosis, this immunity depends also in the avoidance of overcrowding, indoor life and insufficient dietary. Many of the native's huts, which I have seen in Switzerland, must be very deficient in ventilation and lighting in winter, and one cannot be surprised that pulmonary tuberculosis does develop under certain of the conditions that exist.

When we consider that bacteria will not float in the atmosphere in a suspended condition without rafts of organic matter, or dust, or minute droplets of fluid as their carriers; and

when we remember that such mountainous districts are nearly all free from works, or dust producing conditions, it is not to be wondered at that the atmosphere should be so free of disease producing germs.

Perhaps the rarefaction of the atmosphere, and its effects on respiration and circulation is the most important factor in accounting for the comparative absence of pulmonary tuberculosis in most of the high altitudes, and the therapeutic value of the high altitudes in the treatment of pulmonary tuberculosis.

To get a clear view of the effect of rarefied air on the respiration and circulation, it would be advantageous to consider the effects of the compressed air bath as used in the Brompton Hospital. Respiration is slower, deeper and easier than ordinary respiration. Von Vivenot has found respiration to fall to 4, or even 3 in a minute. Inspiration is easy, but expiration undergoes considerable modification, lasting twice, or even three times as long as inspiration. The pulse is slower, smaller in volume, but of increased arterial tension, the capillaries small, and the veins less full of blood.

The therapeutic value in asthma and bronchitis is said to be great, but in pulmonary tuberculosis it is considered by many as of no use, and indeed some authorities consider it to be harmful, as many of the cases develop haemoptysis while in the bath, or immediately afterwards.

When I visited Davos in January 1896, I have a very distinct recollection of my difficulty in walking, and the breathlessness induced by the slightest exertion, and this is the

experience of most people on their arrival at this place. The circulation in the first instance is quickened, and the heart's impulse becomes more powerful. After some weeks residence, the pulse becomes less rapid. Respiration is affected in a similar way, but this also becomes slower, The breathing becomes deeper, the inspiration longer, and the expiration more complete. When the pulse becomes slower, a well marked change almost always takes place in the thorax in various directions, causing an increase in the circumference of from one to three inches at different levels. This chest development is due to rarefaction of the air, as proved by the facts that those who take most exercise have the most marked enlargement in the thorax, and that the chest development is not always permanent, some of the cases returning to the original shape and measurement, on their return to sea-level.

Another beneficial factor common to all altitudes in addition to the rarefaction of the atmosphere, is the quality of diathermacy, or the increased facility by which the sun's rays are transmitted through the attenuated air. The effect of sunlight on tubercle bacillus, and tubercular patients, is now well known. How can we, therefore, doubt that the sun's rays in so effectual a manner must be beneficial in the prevention of tuberculosis.

THE EFFECT OF ALTITUDE ON THE BLOOD.

In Ehrlich's Diseases of the Blood, edited by Stengel, a peculiar phenomenon is described regarding the behaviour of blood to altitude, which

from a therapeutic standpoint, may be of considerable importance. From the investigations of Viault, and others, it appears "that the number of red blood corpuscles in a healthy man with a normal average of 5.000.000 per c.mm. begins to increase immediately after arrival at a high altitude, and continues to increase for ten to fourteen days. This is in excess of the original average in accordance with the difference in height above the sea-level, between the former and the present habitation. Moreover, indivduals born and residing at elevated regions have a physiologic average in excess of that seen at sea-level, and this average is as a rule, somewhat greater than that seen in the acclimatised:- i.e, those who reside only transiently at an elevation.

The following scale shows to what extent increase in height produces a deviation in the number of blood corpuscles from the normal average. (5.000.000.)

Writer	Place	Height above sea-level	Increase about
V. Jaruntowski	Gorbersdorf	561 M	800.000
Wolff & Koppe	Reiboldsgrun	700 M	1.000.000
Egger	Arosa	1800 M	2.000.000
Viault	Cordilleras	4392 M	3.000.000

Exactly the opposite is seen when an individual with this high corpuscular count descends towards the sea-level."

Various observers seem to interpret this phenomenon in various ways, and it seems that there is still a great deal of uncertainty as to what actually takes place. Koppe considered

that the increase is due to the contraction and division of the red blood corpuscles already in existence, and that the change is a physiologic adaptation to the lowered pressure of the air which makes the taking up of oxygen more difficult.

Other observers consider that there is only a relative, and not an absolute increase in the red blood corpuscles, due to an increased concentration of the blood, the result of an increased excretion of water at these heights. There can be little doubt that intense light form a purer air, lowering of temperature, muscular effort and increased activity of respiration are important factors in an altitude which must make some change in the circulation, and the result is beneficial in increasing the vitality, and the resisting power of the individual.

Whatever may be the true significance of these observations, I have several times noticed the change in patients who have spent several months in high altitudes, and who, after returning to this country for a few weeks, and even living under very favourable health conditions, have complained of that want of bouyancy which they enjoyed at high altitude, and have felt an increasing want of power for muscular effort.

On the other hand, I have evidence of permanent good results being gained as a result of a short holiday at high altitude. T.Y. aged 21 years had been delicate since birth, with a very bad family history. When he was 16 years of age, and had finished with his education, his health caused his guardians the greates concern. No evidence of any disease could be found, but he was badly developed, susceptible to colds

and easily fatigued. He was sent out at that time to Davos for the winter. When he returned, he had put on flesh, could walk miles without fatigue, had lost his stooping gait, and had increased in chest measurement. These conditions have continued now for five years. Altitude in this case was capable of removing a condition which I fear in town life would have proved an important contributory cause to pulmonary tuberculosis.

So far as altitude is concerned, the Sunderland Rural District is bounded on the east by the German Ocean, and there is no part of the district which is more than 300 feet above sea-level. I am, therefore, not in a position to give any statistics that will be of any use in this matter.

The relative distribution of land and water, and the presence of ocean currents flowing from higher and lower latitudes can only affect temperature and humidity. There does not seem to be much actual proof that a warm moist air can act in any way favourably to the prevention of pulmonary tuberculosis. Years ago Madeira was considered an ideal place for sending a consumptive, and in many respects the judgement was good. The winters are warm, and the summers cool. Patients are able to live in an equable temperature, the effects of the warm air being soothing and comforting, especially where there is much catarrh, or laryngeal complications.

In spite of this comfort it is very questionable whether such a climate has much power in increasing the resisting power of the patient.

The following case may illustrate my point. E.K. aged 24

had spent the two winters of 1895 and 1896 at Madeira without any appreciable effect. She had consolidation in one apex, and a good deal of pulmonary catarrh. In 1897 she went to Davos, with the result that her appetite and weight increased, her cough ceased, and by the end of six months, excepting for the dulness at left apex, she had no other evidence of any chest disease. These comparative results have much to do with the curative effects of climates, but may we not safely assume that the climate which has, or has not, a beneficial therapeutic affect in pulmonary tuberculosis may also be, or not be, a factor in its prevention.

I am, therefore, of opinion, that a warm moist climate, per se, can have little, if any effect in the incidence of pulmonary consumption.

The proximity of mountain ranges and their influence on the shelter from wind, and on the rainfall.

As through ventilation (or perflation) of a house is a hygienic necessity of the utmost importance, not only for the supply of pure air for respiratory purposes, but for cleaning out micro-organisms which may be suspended in the atmosphere, or deposited on the floor surface or furniture, so wind performs a similar hygienic purpose to the outside atmosphere in dispersing noxious exhalations, whether animal or vegetable, in maintaining the circulation of air which is necessary for its purification, and in drying the earth's surface. Thus far the winds must be considered as unfavourable to the incidence of pulmonary tuberculosis, for we shall see later on that atmospheric impurities probably play a most

important part in the contributory causes of this disease.

When, however, the winds are cold and cutting, (east winds) or when they bring with them large quantities of moisture which for reasons to be explained, deposit the moisture as rain, then such conditions will induce in the body a considerable loss of heat, and produce catarrhs which are so favourable to the development of consumption.

The precipitation of rain is due to one of three causes.

(1) Ascending currents of air, which becomes cooled, and are therefore compelled to deposit their moisture.

(2) Warm air coming against the cold surface of the earth.

(3) The mixture of masses of air of different temperature.

It will, therefore, be seen that mountains may not only act as a protection from wind, but have a most important influence on the rainfall of a district.

Wind coming across an ocean may strike a mountain range, and either by forcing the current upwards, and thereby cooling it, or by chilling the current when it strikes the cold earth, cause the deposit of its suspended aqueous vapour. The district lying to the lee side of such mountain ranges are, therefore, not only protected from the prevailing winds, but have a much smaller rainfall. In this respect the proximity of mountain ranges and their influence on the shelter from wind and on the rainfall, must play a somewhat important part in diseases caused by loss of heat, and, therefore, act indirectly as a contributory cause of phthisis pulmonatis. Much rain renders even moderate degrees of cold unendurable.

The soil - Its permeability and impermeability to moisture.

Perhaps no one has done more in providing absolute proof of the direct relationship which exists between dampness of soil and the incidence of pulmonary tuberculosis than Dr Buchanan. In his report to the Privy Council in 1886, he established the fact that the drying of soil affected by sewage works is generally followed by a great reduction in the mortality of phthisis. In the following year he published another report "On the distrubution of phthisis as affected by dampness of soil.

The results of his enquiry were so striking, that I may be permitted to quote his conclusions:-

(1) Within the counties of Surrey, Kent and Sussex, there is, broadly speaking, less phthisis among populations living on pervious soils, than among populations living on impervious soils.

(2) Within the same counties, there is less phthisis among persons living among high-lying pervious soils, than among populations living on low-lying pervious soils.

(3) Within the same counties, there is less phthisis living on sloping impervious soils, than among populations living on flat impervious soils.

(4) The connection between soils and phthisis has been established in this enquiry; (a) by the existence of general agreement in phthisis mortality between districts that have common geological and topographical features; (b) by the existence of general disagreement between districts that are diff erently circumstanced in regard of such features; (c) by the

discovery of pretty regular concomitancy in the fluctuation of the two conditions from much phthisis with much wetness of soil, to little phthisis with little wetness of soil.

But the connection between wet soil and phthisis came out last year in another way which must be here recalled: (d) by the observations that phthisis had been greatly reduced in towns where the water of the soil had been artificially removed, and that it had not been reduced in other towns, where the soil had not been dried.

(5) The whole of the foregoing conclusions combine into one which may now be affirmed generally, and not only of particular districts, that wetness of soil is a cause of phthisis to the population living upon it.

(6) No other circumstance can be detected after careful consideration of the material accumulated during the year, that coincides on any large scale with the greater or less prevalence of phthisis, except the one condition of soil.

(7) In this years inquiry, and in last year's also, single apparent exceptions to the general law have been detected. They are probably not altogether errors of fact in observation, but are indications of some other law in the back ground, that we are not yet able to announce.

Since 1867, so far as I am aware, these arguments have never been refuted, and although much has been made of the exceptions quoted in paragraph 7, such as a slight increase in the death rate from consumption after the drying of the soil by sewerage works, there no doubt have been other conditions

to mask the general law laid down by Dr Buchanan.

As a matter of interest, and with a view of testing the laws laid down by Dr Buchanan on the dampness of soil and its relation to the mortality from pulmonary tuberculosis, I have compared the average death rate in the various parishes in the Sunderland Rural District for the past ten years.

The geology of the Sunderland Rural District is simple, The surface is capped by boulder clay of varying depths. Below this is a thick mass of permian magnesian limestone, or dolomite. Chemically this rock is a mixture, in equal proportions, of carbonate of lime and carbonate of magnesia, showing by weight 54 $\frac{1}{3}$ per cent of the former, to 45 $\frac{2}{3}$ of the latter. This geological formation is fairly uniform throughout the Sunderland Rural District, excepting for a pocket of sand here and there of so small dimensions as to have little effect on ground formation.

The bed of the River Wear, which runs through the district, is fringed by alluvial deposits, due to the action of the river itself. Add to these the sand of the sea shore, and every formation of the Sunderland Rural District is complete.

The following are the details of each of the parishes.

(a) Ryhope

Area 441 acres.

Estimated population to middle of 1905 = 10655.

Average death rate per million of estimated population during the past ten years from

pulmonary tuberculosis = 1311

Average number of inhabitants per house at last census = 6.3

Formation of land. This parish is bounded on the east by the North Sea, and has rows of houses for the most part running at right angles to the sea border for a distance of three quarters of a mile. There is a steady rise until the western border of the parish is reached which stands 232 feet above sea-level.

(b) Tunstall.

Area 808 acres

Estimated population to middle of 1905 = 5960

Average death rate per million of estimated population from pulmonary tuberculosis = 1541

Average number of inhabitants per house at last census = 5.06

The position of the residential part of this parish is about a mile to the west of the parish of Ryhope, and stands about 100 feet higher, but is in a different water shed.

The township lies in a hollow, about 500 yards to the east, the altitude is 325 feet, falling to 300 feet at the eastern border of the village.

About the same distance to the south the altitude is 356 feet, falling to 314 feet at the southern border of the township. From the west side there is a fall of 15 feet in 200 yards, and from the north west there is a fall from 300 feet

to 200 feet at the north east corner.

It is, therefore, to the north east corner of this township that all surface water from the surrounding area must make its way, converging, in the first instance, slowly and steadily towards the township, and then gradually passing towards the north east corner. It is true that there is some provision for drainage which, no doubt, does much to overcome the harmful effects of the damp soil. But the streets are unpaved, and even surface gulleys will not be effective if a surface impervious to water is not provided. The houses are very much better than in the parish of Ryhope. There is less poverty, overcrowding and intemperance, but from the dampness of the soil, and from that cause alone, there is 17 per cent larger death rate from pulmonary tuberculosis.

It will be noted that the fall in the land all round the township is small but regular. Steeper banks would probably have been less harmful.

(c) **Ford**

Area 1029 Acres

Estimated population to middle of 1905 =	3070
Average death rate per million of estimated population for past ten years from pulmonary tuberculosis. =	981
Average number of inhabitants per house at last census =	6.1

Formation of land. This village is built on the banks of the River Wear. The banks are steep, the highest part of the

village being half a mile from the river 232.8 feet above sea-level with a steady fall to it which is about 32 feet above sea-level. There is a considerable amount of poverty, much overcrowding, and many insanitary conditions. In spite of these defects the average death rate is only 981 per million of the estimated population.

(d) Hylton.

Area 2593 acres

Estimated population to middle of 1905	= 2012
Average death rate per million of estimated population from pulmonary tuberculosis.	= 823
Average number of inhabitants per house at last census	= 5.1

There are two small villages in this large rural parish. They are both built close to the river side on land which has a fall of about 75 feet. The social and sanitary conditions of the inhabitants of these villages are much better than in the previous parish mentioned. Hence there is a smaller death rate from pulmonary tuberculosis in spite of the less favourable geological formation of the land.

(e) Fulwell.

Area 649 acres

Estimated population to middle of 1905	= 3335
Average death rate per million of estimated population for past ten years from pulmonary tuberculosis	= 1544

Formation of land. This parish borders on the sea. The western border stands 150 feet above sea-level, and at this part are a number of houses of the better class. The conditions are favourable for a dry soil.

About four hundred yards to the east of this is the village proper. Its altitude is 99 feet, and for another 400 yards to the east it has a slight rise of 2 feet. In this area large numbers of new cottages have been erected during the past eight years, and the drainage, both of surface water and sewerage, has been a source of difficulty to the authority. The streets, until this year have been unpaved, and as a result after wet weather they have been little else than swamps.

As a proof that special area had had much to do with the increased mortality from pulmonary tuberculosis, the average death rate for the first five years was 1138 per million of the estimated population, and for the past five years the death rate has been 1932. No other reason could possibly be brought forward as a cause for the increased death rate than the dampness of the soil.

(f) Bishopwearmouth Without. This parish is so truly rural, with a small population of 192 scattered over 460 acres, that no deductions for the present purpose would be of any value.

From the above evidence, there is, in my opinion, abundance of proof of Buchanan's third conclusion that there is less phthisis among populations living on sloping impervious soils than among populations living on flat impervious soils, and

had the social, housing and sanitary conditions in the parishes of Ryhope and Ford been more equal to the conditions existing in the parishes of Tunstall and Fulwell, the contrast would have been still more marked.

Of the climatic conditions already discussed, altitude and dampness have, undoubtedly, a marked influence on the incidence of pulmonary tuberculosis. It cannot be said that in altitude there is anything which will render proof its inhabitants against pulmonary tuberculosis, for the uplander frequently develops this disease when he descends to the lowlands, and, on the other hand, the lowlander frequently gets cured when he takes to the high altitude. There is, however, obtained for the man in the high altitude, a constitutional resistance to phthisis, which cannot be considered permanent, but is lasting so long as he lives under such favourable conditions. In soil dampness, on the other hand, there must be produced a condition in the host rendering him more susceptible to the invasion of the parasite; that so far as man's action modifies the influence of soil dampness, there is a corresponding decrease in the results of this contributory cause.

Chapter..... 3.

THE DWELLING AND ITS RELATION TO PULMONARY TUBERCULOSIS .

Damp Dwellings.

Damp dwellings is a subject of probably greater importance than dampness of soil, because a house built under modern bye-laws with provisions for the whole site to be concreted, through ventilation underneath the ground floor, damp proof course and cavity walls will be a prevention in overcoming much of the evil from dampness of soil. I have stated that the topographical conditions of Tunstall were such as to favour a very high death rate from pulmonary tuberculosis. Here very few of the older houses comply with the conditions stated above. to prevent dwellings becoming damp. Since the revision of the building bye-laws in 1900, 91 houses have been erected complying with these bye-laws, and it is noteworthy that from these houses not a single death from pulmonary tuberculosis has been reported. I admit that there has not been the same test of time, as in the other houses. Yet, in the law of averages, if all conditions had been equal some deaths would have occurred in them.

In the inspection of the houses in the Sunderland Rural District in which deaths from pulmonary tuberculosis occurred, I have taken careful note of these points, and the following is the result:-

Dwellings without a damp proof course = 102

Dwellings without provision for ventilation under the floors.	= 131
Other dwellings which had provision for ventilation on the front part of the house, (the kitchen floor being brick, and laid immediately on to the earth.)	= 80
Dwellings in which the walls were actually found to be damp at time of visit	= 29

Doubtless unfavourable weather would have revealed a very much larger number of damp houses.

To prove my argument that damp dwellings act in some measure as a contributory cause to pulmonary tuberculosis, I must further prove that these insanitary conditions exist in a greater proportion in the houses where there have been cases of pulmonary tuberculosis than in other houses in the same area. To this I can only make a general statement. I am well aware of the fact that the great majority of houses in the Sunderland Rural Area cannot be considered ideal, that many have structural faults of a serious nature, but that 10 per cent of the houses in the district would be found to be damp, is an allegation that I would not hesitate to contradict. Indeed, I would be much surprised if the total number would exceed 2 per cent. For since the last Local Government Act of 1894, there are so many agencies at work, that such property has now a poor chance of escaping the notice of the Sanitary Aut-

hority.

But why should damp dwellings be a contributory cause of pulmonary tuberculosis? To enable me to answer this question I am much indebted to a translation of Dr Gemund's paper on "Damp Dwellings" which appeared in Public Health, June 1905.

First the thermal conditions of the dwelling house, and with them the heat regulating economy of the occupant, are very unfavourably affected by damp walls. While 1.000 calories are necessary to raise one cubic metre of water 1 °C., only 500 calories are necessary to raise the same amount of masonry through the same range of temperature. Further, 0.3 calories are necessary to raise one cubic metre of air 1°C. It will therefore, be clear that a dry wall whose pores are filled with air, must use up much less heat than a damp wall, whose pores are filled with moisture. Damp walls will remain cold when the air in the room enclosed by these walls is perfectly warm. Air and water are both bad conductors of heat, but the relative conductivity of water, compared with air, is in the proportion of 27 to 1. Therefore, in rooms which have damp walls the heat is lost much more rapidly than in dry rooms. There is a further loss of heat by the raising of water to a condition of aqueous vapour. According to Rubner, the greatest loss of heat to our bodies is by conduction and radiation. This, therefore, must take place when the bodies around us are cooler than ourselves.

It will, therefore, be seen that when we are in a room where the temperature is sufficient for our comfort, but when the walls are damp, and therefore, colder than the atmosphere in

the room, ther must be a considerable loss of heat by radiation from our bodies. This fact accounts for the feelings of shivering which we feel, when our faces are to a comfortable fire, and our backs are to an outer damp wall. In this case there may be little loss of heat by conduction, but loss of heat by radiation may be so appreciable as to lower the body temperature. As a result there is a weakening of the mucous membranes of the respiratory and gastro-intestinal tract, producing catarrhal symptoms, the relationship of which to pulmonary tuberculosis I will discuss at a later stage.

But a further, and probably an equally pernicious effect of damp dwellings, is the favourable conditions for the growth of lower vegetable life. It is a well known fact that in a well built, dry house permitting of a free circulation of air, the growth of fungi is almost impossible. In a damp house, on the other hand, there is a stuffy smell characteristic of the presence of fungi. This is especially the case in the advanced stages, particularly moulds, when the spores of the fungi die and decay as a consequence of their being invaded in turn by bacteria.

Woolpert has proved that the breathing of tainted air lowers organic metabolism. Hueppe has shewn that attenuated infective bacteria regain their virulence when inoculated with the products of decomposition. It has always been a recognised fact that the lower classes, who live in dark and damp dwellings, become a ready prey to disease in epidemic form. For the above reasons this can be readily understood. Can we won-

der, then that the tubercle bacillus once introduced into such dwellings as are described, will retain all its virulence, and readily infect the occupants of such damp dwellings whose vitality has been materially lowered, not only by loss of heat, but by breathing for a lengthened period the tainted musty air characteristic of these dwellings.

Ventilation.

In attempting to elucidate the relationship between the conditions of the housing of the people, and pulmonary tuberculosis, we have to encounter other factors, which are almost always associated with the crowding together of people. For, speaking generally, the more crowded a community is, the greater the amount of intemperance, want, misery and filth, and these conditions are favourable to the propagation of all infectious diseases. It has been shown, however, (a) that phthisis is most common in large cities. (b) That phthisis is most common in the most densely populated parts of large cities, and (c) that there is a relationship existing between defective ventilation and phthisis mortality, as shewn by the death rate from this disease in the back to back houses of Leeds, and other big centres of population, where such houses are in comparatively large numbers.

For the purpose of excluding the other unfavourable conditions associated with aggregation of population, and proving that defective ventilation, in itself, is a serious contributory cause to pulmonary tuberculosis, I beg to quote the following:-

(a) In the report of the Royal Commission on the sanitary condition of the Army, published in 1858, it was found that as the result of overcrowded and insufficiently ventilated barracks, the phthisis mortality among troops was excessively high. Among the Foot Guards, the phthisis mortality has been highest. During the ten years 1837-46, it was 11.9 per thousand. For the seven years 1864-70, it had been reduced to 2.3 per thousand. The mean of the phthisis mortality in the Household Cavalry, Cavalry of the Line, Foot Guards and Infantry was for the years 1837-46, 7.89 per thousand. In the year 1888 the mortality from phthisis in the British Army at home, was 1.2.

For the most part we can consider the conditions of the soldier in 1888 to be much the same as in 1837, excepting for the conditions of ventilation and overcrowding. He may be somewhat better fed and clothed than in 1837, but for the purposes of this thesis, the discontinuance of overcrowding, and the provision of more efficient ventilation, must be considered as the main causes of the enormous diminution of the mortality from phthisis pulmonalis. Again, "in the defectively ventilated prison of Leopoldstadt, at Vienna, during the years 1834-47, there died of phthisis 51.4 per thousand of the prisoners, while in the well ventilated House of Correction in the same city, during the years 1850-54, only 7.9 per thousand died." Here again, ventilation and overcrowding must be considered as the great cause of the difference in the mortality.

In Dr Ransome's Milroy lecture, it is stated "that in

Canada, a country comparatively free from phthisis, the death rate among English soldiers, was 23 per thousand. After the barracks had been properly drained and ventilated, the mortality had sunk from 23 per thousand in 1865, to 9.5 in 1872, and 6 in 1874."

I will make one further quotation to prove the relationship between ventilation and phthisis. It is from Sir John Simon's Report to the Privy Council in 1860, and is as follows:- "In proportion as the male and female populations are severally attached to indoor branches of industry, in such proportion, other things being equal, their respective death rates by lung disease increased." And Stevenson and Murphy add, "there are medical reasons for assuming the augmented lung disease to be phthisis."

Overcrowding and defective ventilation are not, theoretically speaking, convertible terms. It is possible for one man to occupy a sufficiently large room, yet, from structural defects, or from the closing up of the natural means of ventilation to make the atmosphere in that room harmful. Where the number of persons occupying a room is too great to have the atmosphere in that room kept sufficiently pure without producing draughts, the usual consequences are the closing of all inlets, and the producing of a system of defective ventilation. So that, practically, the results of overcrowding and defective ventilation are the same.

For the present we will leave out of consideration the effects of dust, and confine our attention to the pernicious effects of organic impurities in the atmosphere.

Wherever the conditions are such as to prevent the free circulation of pure air, not only in a dwelling, but around a dwelling, then we get a fouling of the air and its consequences. In this respect, the amount of impurities in the form of decomposing organic matters varies in towns, not only because of an overbuilt and badly arranged area of houses, preventing a free circulation of air, but because of the proximity of old fashioned privy middens and other insanitary conditions, all adding organic effluvia to an atmosphere which is already too tainted for the health of its users. If an atmosphere of this condition surrounds a dwelling, whatever may be its structural efficiency, such tainted air must enter the dwelling, and so affect the health of its inmates. So too, in large tenemented properties, there is an absolute necessity, not only for free circulation of pure air surrounding such buildings, but abundance of provision for cross ventilation within such buildings.

The great work which Miss Florence Nightingale did in pointing out the dangers which existed in the aggregation of large numbers in hospitals, and its relationship to resultant septicaemia so common at that time, is only another example of the pernicious effects resulting from overcrowding and deficient ventilation. For whereas septicaemia is an acute infectious disease, and makes itself manifest soon after its invasion, the bacillus of pulmonary tuberculosis, although slower in its effects, may, under such conditions, be none the less certain in its action.

To prove from experience the serious results arising from a badly constructed and overcrowded site, I have taken such an area in the parish of Ryhope consisting of 55 houses, nearly all tenemented. They are all built in the form of small squares, with little open space about them. The yards, or courts, were mostly unpaved, and the sanitary conditions as a result were bad. During the ten years, 1896-1905, 14 deaths from phthisis occurred in these fifty five houses. While 122 deaths from the same cause occurred, for the same time, in the whole parish of Ryhope. The number of inhabited houses in the whole parish at the last census was 1509. The number of deaths per hundred houses in the whole parish would, therefore, be 8.08. The rate per hundred houses in this small area of 55 houses would be 25.4. It is only fair to state that 3 deaths occurred in one house, and 2 in another. There can be little doubt, therefore, that the enormous death rate from pulmonary tuberculosis in this small area is greatly the result of the want of a free circulation of pure air without, as well as within, the dwellings.

It is interesting to contrast with the above the conditions existing in the Peabody Block Buildings in London. These buildings are number of tenements, built on a very small area, but arranged so that each will receive sufficient air, and fulfil all other sanitary requirements. In 1884, there were 10.144 living rooms inhabited by 18.453 persons. The recorded death rate, corrected for hospitals, was 19.1 per thousand, or 1.2 below the general death rate of London. I have not the

death rate for phthisis pulmonalis, but as a high death rate from phthisis is usually associated with a high death rate from other respiratory diseases, it may be assumed that the Peabody Buildings were also satisfactory in this respect.

It is evident that it is not so much the overcrowding of area, or the aggregation of a large number of people on a small site that is the cause of so much pulmonary tuberculosis; but the absence of efficient means for the supply of a sufficiency of pure air, and the presence of conditions favourable to the decomposition of organic matter.

Before considering the structural defects in the individual dwellings, it might, at this stage, be advantageous to answer the questions, what are the efeects of defective ventilation, and what are the results of the habitual breathing of air rendered impure by overcrowding, or defective ventilation? Carbonic Acid is present in pure mountain air in 0.3 per thousand volumes. In town streets, Angus Smith found it to average 0.36 per 1000 in London, and 0.40 in Manchester. 0.6 per 1000 is held to be the limit compatible with health and yet it may be increased to nearly 15 per 1000 before it commences to produce headache and nausea. It must, therefore, be accepted that Carbonic Acid must play a very small part in the pernicious effects of defective ventilation in a dwelling. One of the sources of Carbonic Acid is from the fermentative, putrifactive and respiratory processes of vegetable and animal life, and it is in this respect that Carbonic Acid is used as an index of impurity of its relation to the organic

matter thrown off during respiration.

With defective ventilation, the products of respiration, (including the tubercle bacillus) the exhalations from the body, the products of imperfect combustion in consequence of defective heating and lighting, must all necessarily accumulate in the atmosphere of the room.

Without a free current of air, the organic compounds from the expired air, and from the surface of the body, accumulate on the floor, the furniture, and the walls, and then decompose. The harmful influence of a close impure atmosphere is well known to most of us. It shows itself both at the time, and afterwards, by headache, yawning, coldness of the extremeties, nausea and even fainting. As I have already stated, this is not due to carbonic acid, nor is it due to a decrease in the amount of oxygen. The real causes are probably organic toxins excreted by the lungs, and given off along with water vapour. When this is condensed and injected into healthy animals, a toxic effect is produced. This toxine is not so diffusible as carbonic acid, hence it acts indirectly in badly ventilated rooms, by weakening the resistance of the tissues.

But while the weakening of the resistance of the tissues will make the host more susceptible to the invasion of the parasite, the conditions of a badly ventilated room must necessarily increase the risk of infection. If the bacilli of tuberculosis, by any means, gain access to such an atmosphere, they will probably be retained, either in a condition of suspension, or deposited on the floors, walls or furniture.

Efficient ventilation, therefore, means something more than the supply of air sufficiently pure for respiration. It means even something more than air containing not more than 0.6 per thousand volumes of carbonic acid. Efficient ventilation means, that a current of air shall, from time to time, sweep through that room, carrying with it not only all atmosperic impurities, but also it must be strong enough to remove from the surface of the floor, walls and furniture all micro-organisms and organic matter that have already been deposited thereon. Hence the necessity, not only of sufficient inlets and outlets in a room, but, further, that the relative position of these inlets and outlets shall be such as to encourage the current of air to sweep through the entire room.

We will now see that many of the houses are constructed so that this perflation, or through ventilation, cannot possibly be carried out.

Houses may be wholly detached, with four exposed sides, or may be semi-detached, with three exposed sides, or may be built in a row with two expoesed sides. Each of these types of houses will permit of a through ventilation, For the purposes of ordinary ventilation the window, or the door, will be the inlet, and the fireplace the outlet. For the purposes of perflation, however, the current must be between the window and the door. Hence, the great advantage there is in having the window and the door in opposite sides of the room.

There is, however, another class of house, known as the back-to-back house. These are constructed by building two

terraces of houses rear to rear, or back-to-back, leaving only one side of the house exposed. Here the only ventilation that is possible between window and door must be thus :-

instead of thus

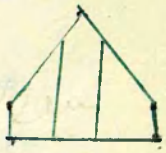
in a house permitting of through ventilation, with the result that the cleansing effect of a current of air under such structural arrangements, will only permit of a very small part of the room being thoroughly flushed. Irrespective of the amount of open space surrounding the exposed side of these houses, such defective buildings have always been associated with a large mortality from pulmonary tuberculosis.

As a result of an inquiry by Dr Gordon Smith, and Dr Barry, into the death rate of the Greengate and Regent Rd registration sub-districts of Salford, we have, to my mind, conclusive proof of the pernicious effects of back-to-back houses as a contributory cause to pulmonary tuberculosis. They divide these two sub-districts into three parts as follows:-

		Population	Death rate per 1000 living from All Causes	Phthisis
Greengates Sub District	Group 1 back-to-back houses	8713	27.5	2.8
	Group 11 average proportion of 25% back-to-back houses.	11749	29.2	3.3

		Population	Death rate per 1000 living from All Causes	Phthisis
	Group 111 average proportion of 56% back-to-back houses.	11405	30.5	3.6
Regent Road	Group 1 no back-to-back houses.	54264	26.1	2.7
Sub District	Group 11 average proportion of 18 % back-to-back houses.	8773	29.1	2.7
	Group 111 average proportion of 50% back-to-back houses.	4380	37.3	4.5

Up till two years ago, in the Sunderland Rural District, there was a row of houses answering completely to the description of back-to-back houses of the very worst type. Originally they had been five roomed cottages, with two good living rooms downstairs, and three small bed rooms upstairs, of a capacity of from 504 cubic feet to 674 cubic feet. There were no fire-places in the bed-rooms, and to make matters worse the bed-rooms were built in the roof, as shown in the section and were badly lighted. In the interest of economy and for the purpose of providing more houses for workmen, the owners closed up the communicating doors between the front living room, and the back living room, and between the front bed-room, and the two back bed-rooms, thus absolutely destroying the through ventilation. As may be expected, the returns



for these houses during the past ten years shews a marked increase in the mortality rate from pulmonary tuberculosis, when compared with the other houses in the same parish. In the 72 houses of the back-to-back type, there were 12 deaths from pulmonary tuberculosis, or 16.6 deaths per hundred houses, while the rate for the houses in the same parish providing for through ventilation, was 7.6 per hundred houses. The conditions of life may be considered to have been fairly equal, as the great majority of the employees in this parish are miners, and have to take any house which might be provided for them.

Evidence of this nature brings home very forcibly the evil results of deficient ventilation as a contributory cause in all infectious diseases, and especially in pulmonary tuberculosis.

In 12 of the houses where deaths occurred from phthisis, from statements made by friends, overcrowding must have existed. I have stated that, practically speaking, overcrowding produces the same results as deficient ventilation.

I have not made any comments on the efficient lighting of premises. It may be stated generally that deficient arrangements for lighting usually existed in the same houses as those where there was deficient ventilation. Especially was this the case in the badly constructed courts to which reference has already been made. The well known effects of the rays of the sun on the tubercle bacillus proves that the lighting of a dwelling is of paramount importance.

Chapter 4

O C C U P A T I O N .

Unfortunately, my experience concerning occupation is limited, and cannot be considered of much value. Probably not less than four fifths of the bread winners in the Sunderland Rural District are employed in, and about, the coal mines.

There are four small factories in the Sunderland Rural District, but some of the employees in factories situated in the Borough of Sunderland reside in the Sunderland Rural District, hence the difficulty of arriving at a correct view of the relation of industry to pulmonary tuberculosis.

Of the 288 deaths registered as due to pulmonary tuberculosis, 108 were bread winners.

The following are details of occupation.:-

Mines	Miners	42
	Screenmen	3
	Banksmen	2
	Total.....	47
Indoor work	Sailmaker.....	1
	Engineers.....	3
	Rivet Makers.....	2
	Fismonger.....	1
	Watchmaker.....	1

	Teacher	2
	Tailor	1
	Draper	1
	Glassmaker	1
	Papermakers	5
	Grocers	2
	Blacksmiths	2
	Joiners	2
	Plumber	1
	Nurse	1
	Hairdresser	1
	Potter	1
	Publican	5
	Stoker	1
	Musician	1
	Domestic Servants	4
	Total ..	39
Out-door	Labourers	8
Workers	Masons	2
	Policeman	1
	Gardener	1
	Hawker	1
	Shipbuilder	1
	Soldier	1
	Mariner	1
	Ticket Collector	1

Plater 1

Platelayer 1

Bricklayer 1

Farmers 2

Total..... 22 Grand total 108

My short experience in the practice of medicine in a colliery village in the West of Scotland, and in Midlothian, seemed to have left an impression on my mind that phthisis was an exceedingly common disease among the miners in these districts, and I had always associated their conditions of labour with the nature of the malady from which they suffered. Since coming to Sunderland, in 1891, I have had a fair share of my work amongst the miners, and comparing the number of patients suffering from phthisis amongst miners, and other artisans, in the same neighbourhood, I have deliberately come to the conclusion that I have seen less phthisis amongst miners, than amongst those artisans who live beside them. Perhaps this may be, in some measure, due to the fact that the struggle for existence is not so keenly felt with the miner, as it is with the shipyard worker. Work is more regular, a living wage is more frequently obtained, and the provision by the employer of a house and coals does much to lessen the anxiety for his existence. The results of the preceeding table does much to confirm this view. For I am absolutely certain that the number of deaths (47) amongst miners, and those working about mines, is a much smaller ratio to the number of men employed, than the number of deaths (62) (males) amongst those

engaged in other occupations.

It is usually held that an atmosphere loaded with the dust of different trades is a potent indirect cause of phthisis, producing a catarrhal, or mechanically injured condition of the mucous membranes of the lungs, and so favours the entrance and activity of the bacillus.

Of the evil effects of hard and angular dust, like steel and stone, there can be no doubt. Coal dust, on the other hand, is a questionable factor. When dust of any kind is inspired, it is usually taken up by the leucocytes, and deposited throughout the lung substance. This is so in the case of the miner, producing the characteristic black lung, seen so commonly in post-mortem examinations on coal workers.

But coal dust is soft, and comparatively free from angles, and cannot produce the traumatic effects produced by the harder and more angular particles of dust in many other trades. Indeed, it is held by some observers, that coal dust, per se, instead of having a harmful effect, affords some protection against phthisis. When the death rate from phthisis amongst miners is found to be large, it will usually be found that the ventilation of the galleries in which they work is not all that it should be.

In Mr Simon's fourth report, he drew special attention to the fact that the colliery miners of Durham and Northumberland, differ from other miners, in not suffering from any important excess of pulmonary disease, and argued that the reason for this is the good ventilation of the mines in which they work. He adds

however, that he was still disposed to think that this operated mainly by removing the coal dust and powder smoke.

Two stonemasons in the Sunderland Rural District died from phthisis. There are very few stonemasons employed in this district, very few stone built houses being erected.

It has always been recognised that dust is not the only industrial contributory cause to phthisis. Exposure to great vicissitudes of temperature, inhaling hot air, either very dry or very moist, and workmen being obliged to carry on their labours in a cramped position, interfering with the respiratory movements, are all aids to the development of pulmonary tuberculosis. From the list of occupations already furnished, I am not in a position to give much proof of this statement. Occupations favouring these conditions are not plentiful in the Sunderland Rural District. It is worthy of note, however, that 39 persons engaged in indoor work died from pulmonary tuberculosis, compared with 22 of the out-door workers. I am satisfied that, excluding miners, there is at least as large a proportion of men who earn their bread out of doors in the Sunderland Rural District, as indoor workers.

This again proves Sir John Simon's words "In proportion as the male and female population are severally attracted to indoor branches of the industry, in such proportion, other things being equal, their respective death rates by lung disease is increased."

Of 6,000 cases admitted into the Brompton Hospital in ten years, two thirds followed indoor occupations, amongst them

milliners, sempstresses and tailors, who work, and possibly live, in close rooms to which they are almost entirely confined, furnish the quota." The Factories and Workshops Act of 1901, will probably do much in time to reduce this number.

Hawkers, and other out-door traders, though much exposed, are shown to be less liable to catarrh than indoor workers.

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Chapter ..... 5.

HEREDITY & FAMILY PREDISPOSITION.

In discussing the relationship of heredity and family predisposition to pulmonary tuberculosis, it will be necessary to enlarge the scope of inquiry to tubercular disease in general, as only by so doing will it be possible to show the relationship between heredity and disease.

The subject of heredity, and its relationship to disease seems to me to be so obscure and speculative, that I have had considerable difficulty in following the writings of Weismann and others, and when it is found that there are so few points on which authorities on this subject are agreed, I may be pardoned if I fail to trace the actual relationship that exists between tuberculosis and heredity, or family predisposition.

The older writers recognised to some extent the theory of the continuity of the germ plasm. Lucretius says "Sometimes, too, the children may spring up like their grandfathers, and often resemble the form of their grandfather's fathers, because the parents often keep concealed in their bodies many first beginnings mixed in many ways, which, first proceeding from the original stock, one father hands down to the next father; and then from these Venus produces forms after a manifold

chance, and repeats not only the features, but the voices and hair of forefathers. And the female sex equally spring from the father's seed; and males go forth equally formed from the mother's body; since these distinctives no more proceed from the fixed seed of one or other parent than our faces and bodies and limbs. The birth is always formed out of two seeds." Lucretius here demonstrated the inheritance of physical features.

Before going further, it might be better to tabulate the results of my inquiry into the family history of 195 persons who died from pulmonary tuberculosis. I admit at once that I am not satisfied with the result of the inquiry. Partly from ignorance of the cause of death of their ancestors, and partly from a desire to hide the fact of a tubercular family history, I believe that my results are understated. Further, I found it impossible to make any satisfactory inquiry into the causes of death in collateral branches of the family, (Uncles, Aunts and Cousins,) and this I regret. I am now satisfied that most valuable information can be gained by inquiry into other branches of the family, as can be shown by the following case.:-

J.S. (aged 30 years) died from pulmonary tuberculosis. Inquiry into the direct line of descent did not give the slightest evidence of a bad family history. Further evidence into the collateral branches of the family showed one of the worst family histories that I have seen, yet in the direct line of descent there was no evidence of a predisposition to tubercu-

losis. The following are the facts:-

J.S. (grandfather) died at 60 years of age from typhoid fever.

M.S. (grandmother) " " 86 " " " from senile decay.

They had ten children, six of whom married and had children.

(1) M.S. (aged 80 years at death) cerebral haemorrhage. 3 children, none of whom were tubercular.

(2) M.S. (aged 60 years at death) unmarried. Tubercular disease of spine.

(3) H.S. (aged 52 years at death) Small-pox, married, 6 children.

(a) Tom M. died aged 24 years from phthisis.

(b) James M. " " 22 " " "

(c) Maggie M." " 26 " " "

(d) Janet M. " " 16 " " "

(e) Eliza M. suffers from scrofulous glands.

(f) John M. apparently in good health.

(4) A.S. married, (two sons) died, aged 71 years from pulmonary tuberculosis.

(a) James, died, aged 17 years, from pulmonary tuberculosis.

(b) William died " 42 " " rhuematic fever.

(5) J.S. died at 19 years of age from scrofula.

(6) E.S. " " 68 " " " " bronchitis.

(7) R.S. died at 64 years of age from cancer of stomach.

(a) J.S. died, aged 30 years from Pulmonary tuberculosis.

Three other members of family in good health.

(8) J.S. Alive and in good health.

(a) W.S. died from diptheria aged 17 years.

(b) J.S. " " bronchitis " 24 "

(c) S.S. " " diptheria " 9 "

Three other members of family in good health.

(9) R.S. (unmarried) 71 years, died from tubercular disease of spleen and tubercular pleurisy.

(10) M.S. married, suffers from tubercular disease of bone. She married her cousin and had nine children.

(a) James G. aged 39 years, suffers from tubercular disease of glands.

(b) Tom G. died at 4 years from tubercular disease of spine.

(c) Marian G. died at 3 years of age from tabes mesenterica.

(d) Eliz. G. died 5 yrs of age from tabes mesenterica

Five other members of the family are apparently in good health.

Nothing is in-common with the above family, or their families but relationship. They all live far apart, and rarely see one another.

Of the 195 cases inquired into, a family history of tuberculosis was obtained in 99 cases.

Synopsis of relations affected in the above cases of family phthisis.

Grandparents (without parents and brother or sister)	5
Both parents	1
Father	9
Mother	12
Parent, and brother or sister	20
One brother or sister	26
Two or more brothers or sisters	12
Children	11
Total	96

From the above table it was seen that:-

(1) No. of family histories taken = 195

(2) No. of cases where there is evidence of bad family history = 96

(3) No. of cases of bad family history but with out risk of infection from other members of the same family = 26

(4) No. of cases of bad family history but with opportunities of infection from other members of the same family = 70

(5) No. of cases of possible risk living in the same house, but having no blood relationship = 15

Amongst the diseases associated with heredity are diabetis, cancer, syphilis, haemophylia, and tuberculosis. Since the discovery of the microbic origin of tuberculosis, opinion concerning the influence of heredity has changed. At one time no factor in the etiology of this disease was of such consequence as family history. It appears to me that now the swing of the pendelum has gone to the other extreme, and leading authorities give little or no place to the influences of heredity. Von Behring in his Cassel lecture, makes the following ing statement:- "I must decline to accept another widespread view, namely that hereditary influences are deciding factors. Theoretically, an intrauterine infection with tuberculosis is certainly possible, and in a few cases such an infection has actually been proven. But neither the parental, not the preparental transmission of tubercle bacilli, nor the hypothetical transmission of a body predisposition to tuberculosis, is of any practical importance." Von Behring considers that nearly all cases of tuberculosis are associated with post-genital conditions, and in face of the positive opinion of an authority so eminent, one hesitates before expressing an opinion which may, in any way fail to harmonise with his. I would here note the distinction between tubercular infection, and tubercular consumption.

Dr Naegeli, of Zurich, was unable to discover in his post-mortem examinations a single case over 30 years of age, which did not show evidence of tubercular infection in some part of

the body. Between the ages of 18 and 30, there were 96 per cent, between 14 and 18, 50 per cent, between 5 and 14, 33 per cent, and between 1 and 5, 17 per cent. In the bodies of infants under one year of age, on the other hand, definite tubercular signs were invariably absent. If we are to accept these figures, and consider them in the same light as cases of tubercular consumption, then we must dismiss the question of heredity and family predisposition, and admit, with the old Griefswald physician, that "We are all a bit tuberculous."

Koch's tuberclin, however, will take us a little further, This water soluble substance derived from the tubercle bacilli, when injected into the body of a patient having a tubercular infection, unites with the soluble anti-body, prepared in the body of the host, to form an insoluble body. The result to the patient is an increase of temperature. It is more than possible that this test does not demonstrate the existence of every tubercular infection; but every reaction to the test, irrespective of physical signs, is a certain demonstration of the existences of tubercular infection. Dr Franz, an Austrian Army Surgeon, by the use of Koch's tuberclin, investigated the condition of two regiments of infantry. In order to prevent much constitutional disturbance he only injected 1 to 3 mgs. of the tuberclin, and with this small dose he demonstrated the existence of tubercular infection in 61 per cent of the first year of service, and 68 per cent in the second year of service. Where he used the dose recommended by Koch, (1 centigram) his percentage for the twenty first year of life

approached Naegeli's results very closely. Turning our attention now to the effect of the tuberclin test on very young children, Dr Nikelaus Berend of Hungary, examined 96 very young children. He injected 1 centigram of tuberclin in each case, and although many of the children were feeble individuals, and some were the children of tubercular parents, he failed to obtain a single positive tubercular reaction.

I have not had the opportunity of examining children by the tuberclin test. My mortality results, however, do not bear out the fact that tubercular infection, and tubercular consumption is such a rare thing in early childhood. I submit the age periods at which death occurred, putting in the first line these cases in which a tubercular family history was obtained, and in the second line those cases in which there was no evidence of tuberculosis in the family.

No. of cases.	Under 1 year	1 year & under 5 years	5 years & under 15 yrs	15 years & under 25 yrs	25 years & under 40 yrs	40 yrs & under 65 yrs	65 yrs & up wards
107	7	3	14	34	31	17	1
90	3	9	8	20	26	20	4
197	10	12	22	54	57	37	5

It has been asserted, in proof of the effects of heredity, that cases with a bad family history die at a younger age than cases with a good family history. This statement, on the whole, is borne out by my table. The proportion of cases under one year of age, seems large, especially in the light of the evidence of Dr Berend, just quoted. I know that in two of the

cases, the diagnosis was verified by a hospital physician. Regarding the other cases, I cannot give any proof of the accuracy of the diagnoses. Taking the statistics as they are, however, it is a noteworthy fact that the number of deaths under one year with a bad family history, was as 2 to 1, when compared with those of good family history. The true value of family history in the etiology of pulmonary tuberculosis is so frequently clouded by the fact that other members of the same family are, or have been, suffering from that same affection, and that there is, therefore, the risk of constant and repeated infection. The previous table which I have given, showing family history, and risk of infection, proves this statement. On the other hand, there are so many cases of bad family history, where there could be no possible risk of infection between one member of a family and another, that it is impossible to doubt the influence of heredity in pulmonary tuberculosis.

W.W. aged 25 years, married, died from pulmonary tuberculosis on 28th. August, 1896. He was a miner, wrought in a healthy mine, lived a healthy and temperate life in a good sanitary house. He had never any financial troubles, and never had any acute illness until he was seized with pulmonary tuberculosis. He was one of three children who were taken into an Irish workhouse in infancy, where his mother died from pulmonary tuberculosis. His sister died at the age of 16 years, in the workhouse, from the same disease. The regulations of the workhouse prevented him seeing much of his sister, and, therefore, direct infection from sister to brother can be

eliminated. The deceased and his brother were taken out of the workhouse when young lads, and came to England, settling down in villages ten miles apart, and rarely ever seeing one another. Both brothers lived to manhood, and both died from pulmonary tuberculosis. Here we have four members of one family, all dying from pulmonary tuberculosis, and, so far as one can see, without the possibility of one infecting the other. With cases like these, it seems to me we are bound to admit the influences of heredity in tuberculosis, and that it still has a claim to an important position in its etiology.

"Out of 3,000 cases at Brompton Hospital, Dr Thompson found that 36 per cent of the males, and 58 per cent of the females, gave a family history of phthisis. In 80 cases of well marked family phthisis, he found a history of paternal inheritance in 24 instances, and maternal inheritance in 14 cases, and atavism in 12 cases. Among these 80 families, 385 children were born, of whom 203 were males, and 182 females. In them phthisis appeared in 98 males, and 96 females, there died in childhood 21 males, amd 16 females, and there remained apparently healthy 84 males, and 70 females. In other words, 194 of these tainted families, or one half, died from phthisis, 37 of the deaths occurring in childhood." In compiling these statistics, Dr Thompson was careful to exclude the possibility of one member of the family infecting another. He shows, too, that where there is a family history of phthisis, the members of that family contract the disease at an earlier age, than do persons with a good family history. Further,he points out

that where the inheritance is both paternal and maternal, the members of that family contract the disease at a still earlier age.

Insurances Offices find, like most commercial undertakings, that the competition of the age demands that no stone be left unturned in order that they may do business. They are advised by many of the most capable physicians and actuaries, and would , doubtless, be prepared, if they believed that there was nothing in family history, to take the risk of insuring persons with a history of family phthisis, providing that the lives were satisfactory in every other respect. Yet we find that in their instructions to medical examiners, they insist on careful information on this point, and they still "load" persons, whose parents, or near relatives, have died from phthisis, and the results of their death returns justify their action.

I here submit tables from the Mutual Life Insurance Company of New York, of deaths from tubercular diseases.

Table 1. Showing the mortality from tuberculous diseases in non-consumptive, and consumptive families respectively.

Ages at death	Deaths in non-consumptive families.			Deaths in consumptive families.		
	From all causes	From tuberculosis	Percentage of deaths from tuberculosis	From all causes	From tuberculosis	Percentage of deaths from tuberculosis
To 29 years	106	38	35.8	60	31	51.6
From 30 to 39 yrs	372	98	26.3	194	77	39.6
" 40 " 49 "	566	100	17.6	284	70	24.6
" 50 " 59 "	724	49	6.7	489	77	15.7
" 60 " 69 "	613	36	5.8	569	47	8.2
" 70 " 79 "	325	12	3.7	398	8	2,2

Table 11. Showing the proportionate mortality from tuberculosis among those with tainted record, arranged according to the degree of taint, and the age of Insurance.

Number of members of family affected.	Ages at Insurance.				
	Total	20 to 29 years	30 to 39 years	40 to 49 years	50 yrs & upwards
	per centage	per centage	per centage	per centage	per centage
Both parents	14.3			50.0	
Father	20.7	43.3	16.1	10.4	9.6
Mother	15.5	36.5	17.1	7.5	4.6
Parent and brother or sister	11.0	18.2	21.4	5.5	
One brother or sister	14.3	34.1	20.6	10.2	4.3
Two or more brothers or sisters	19.4	62.5	42.4	7.5	7.5
Tuberculosis percentage	12.7	28.5	13.3	6.8	4.6

With results such as these, it is not to be wondered at, that Insurance Companies still decline to accept the dictum of Von-Behring, that hereditary influences are not deciding factors on the etiology of tuberculosis, nor are the companies likely to under-estimate their value.

It may be well now to discuss how heredity can possibly determine an increased risk to death from tuberculosis, and for that purpose I will consider it under three heads.

(1) Infection of the germ cell.

(2) Infection of the foetus through the placenta.

(3) The inheritance of a tendency to tubercle, or the existence of a certain vulnerability of tissue which predisposes persons to be more readily affected by tubercle bacillus.

(1) THE INFECTION OF THE GERM CELL. The existence of this possibility is almost always denied by pathologists. They usually take up this attitude for two reasons, (a) because the bacillus of tubercle has never been discovered in the germ cell, and (b) because, if it did infect the germ cell, it would destroy it, and it would be impossible to have a mature child. I think that both these conclusions are arrived at with too little consideration. Too much stress has been laid on the absence of the bacillus, and negation is no proof.

It will be conceded that syphilis is of microbic origin. It will be further admitted that congenital syphilis is not unfrequently seen when the cause of the disease must be the father, and the father only. Yet the very existence of the

child is proof that the micro-organism of syphilis does not always destroy the germ cell. In support of my contention I may be permitted to quote in full Weismann's views on hereditary tuberculosis, whose work on heredity I have gone through, although there is much in it I have failed to grasp. On page 389 he states , "It is, however, also conceivable that both causes - the transmission of abnormal predispositions, and infection of the germ cell - might combine to bring about the transference of a disease from one generation to another. Without desiring to encroach upon the domain of pathology, I am inclined to suppose that this is the case as regards "hereditary" tuberculosis; there is no doubt about the occurrence of the "tuberculous habit"- that is a certain complication of structural peculiarities which is commonly connected with the dissease, such as a narrowness of the chest, for instance. These peculiarities must result from the construction of the germ plasm, in which a definite variation of certain determinants must have taken place, and they are, therefore, certainly transmissible. The disease itself, however, is not due to this habit, but is caused by the presence of specific parasites, the tubercle bacilli, which have a harmful effect upon the various living tissues. They may be introduced artificially into the blood, and then produce the disease even in perfectly normal individuals. They may, however, enter the body spontaneously, e.g. by some natural means, and will there also give rise to the disease. But in the latter case the probability of infection seems to depend upon the susceptibility, or power

of resistance of the individual, and at the present day, pathologists are of opinion that persons exhibiting the tuberculous habit already referred to, have a much slighter power of resistance to the parasites which have passed into the body than strongly built people. The inheritance of the disease, would accordingly, depend on the transmission of a constitution very liable to infection.

Without wishing to deny the existence of such a predisposition to infection, I do not believe that the transmission of tuberculosis is due merely to the inheritance of a greater degree of susceptibility. A large number of facts, seem to me, on the contrary, to support the view, that <u>infection of the germ plays the chief part</u> of the process. It would not be out of place to enter into particulars, and attempt to prove the view here - the question belongs to the province of the pathologists. I merely wished to point out in this connection that a combination of hereditary transmission and infection of the germ is conceivable. The phylectic origin of such constitutional diseases is presumably to be explained as being due to the occurrence of certain individuals possessing constitutions which were abnormally susceptible to a certain kind of microbe. Such persons would be more readily attacked from without by this particular disease. If, however, it once attacked them, and were it of such a kind to cause death only after some time, a further and surer opportunity was offered to the microbes for transferring themselves to other hosts, than was previously the case, when they passed into the body

from without :- they settled in the germ cells of the individual affected, and were thus transferred to the descendants of this individual. Although the presence of parasites in the germ cells has not yet actually been proved in the case of tuberculosis, in my opinion it by no means follows that such infection does not, nevertheless, take place; we do not even know whether such microbes are of the ordinary form and size. In any case, they must possess different vital qualities; for did they multiply in the egg, or sperm cell, in the same manner as in the tissues in which they are known to occur, the germ cells would soon be destroyed. Numerous adaptations to the host may have occurred in this case, as in that of other parasites; and moreover, latent periods of development may have arisen, during which the parasite does not undergo multiplication. It seems impossible that such arrangements should not be met with, and that the parasite should not make use of the favourable opportunity of becoming distributed with the greatest certainty. Latent periods very commonly occur in the germs of animals and plants, whenever they are useful, and hence this arrangement must come about without any great difficulty. Even although our most eminent pathologists, such as Ernest Ziegler, are now of opinion that tuberculosis is not transmitted by infection of the germ, because such a transmission has not been directly proved, and because, on the other hand, infection from without cannot be conclusively disproved in any individual instance, I am inclined to believe that they have been too cautious in their con-

clusions, of which only a negative proof is furnished by either factor. For neither of these in the least proves that infection of the germ does not take place; from a more general, biological point of view, indeed it seems to be far more probable that it does."

It is unfortunate for my purpose that Weismann does not enter into"particulars", and prove his views. Nevertheless, there is in his arguments much that must commend itself to one as being at least possible. Mr James Martin, Veterinary Surgeon, Wellington, Salop, has given me some particualrs of an outbreak of tuberculosis amongst his hens, which, in my opinion, gives some proof of the possibility of the infection of the germ cells. On killing several of his hens for table purposes, he discovered that they were affected with tuberculosis, and resolved to destroy the lot. As the hens were of a very special kind, he gave a setting of eggs to his mother-in-law, who lives several miles from him. She got a brooding hen from elsewhere, and as a result, six chickens were hatched. After killing one of the chickens for table purposes, it was discovered that it had tuberculosis, and Mr Martin thereupon killed the other five chickens, and found them all to be in the same condition. Here again, it may be argued that infection from without has not been disproved; but from all reasonable grounds, it must be inferred that the germ cells were infected, and that it is possible to include direct infection of the germ cells as a cause of tuberculosis. There may be points in the theory of germ cell infection that science has

not yet grasped, but are there not points in the whole subject of heredity, that are equally, if not more obscure than the infection of the germ cell? How can we explain Telegony, for instance. Breeders hold such strong views on "harking back", or infection, that there must be some fact in it.

In the breeding of Bedlington terriers, Oliver points out that it is desirable to obtain dogs with as powerful jaws as possible. A bedlington bitch is, therefore, first covered by a bull terrier dog, and the litter is destroyed. Covered subsequently by a bedlington terrier dog, the litter is practically pure, with the exception that the puppies have stronger jaws than they otherwise would have had, and they also show much of the gameness of the bull terrier. Anatomical structure, and physiological characters are present in the second litter which do not belong to the breed, and could only have come from the bull terrier. We cannot explain this in any way than that there is some absorption from the foetus into the blood of the mother. Whatever the explanation may be, it is easier to imagine the possibility of germ cell infection, than give proof of the cause of telegony.

Charin and Gley, for five years conducted experiments for the purpose of ascertaining the influence on the offspring of parental reception of Virus. Either both male and female parents, or one parent, were inoculated with the bacillus of blue pus, or its toxins. The results varied. Most frequently there ensued sterility, abortion, or birth of progeny that die immediately. In rare instances the offspring survive;

more rarely still are they healthy. Certain rabbits were born with enormous epiphyses, the shafts of the bones being shortened. Of course this experiment proves nothing. The effects on the offspring may have been due entirely to the toxins of the bacillus of blue pus, for, undoubtedly, toxins do exert an enormous effect on the germ cells. Dr George A. Watson conducted some experiments on the effects of Abrin and Ricin on the progeny of inoculated animals. Here, too, he found that the effect of prolonged and increasing doses of the toxin was to diminish the power of producing offspring in both sexes, and that when offspring are produced, they are either born dead, or do not long survive birth. Further, it has been proved "that not only can lead produce abortions and miscarriages in females, but that male workers with lead, suffering with lead poisoning - their wives being free - have devitalised offspring, which either die in utero, or survive only a short time after birth."

I do not think, however, that there is the same evidence of the activities of toxins in tuberculosis as in syphilis, abrin and ricin, or lead. On the contrary, there is much evidence that the toxins are neutralised. For it is a recognised fact that plural, and quickly repeated births, are common in tuberculous families. Nor have I noted in tuberculous families that premature births were at all common, except when the case was complicated by a syphilitic taint.

(2) INFECTION OF THE FOETUS THROUGH THE PLACENTA.

In a review of the works of Professor Francis Harbitz of Christiania, which appeared in the British Medical Journal of August 19th, 1905, two cases of congenital tuberculosis are mentioned, in one of which the child died when only nine days old, and in the other, death occurred at the age of three weeks. Other cases, eight in number, are given of children dying under three months of undoubted tuberculosis It is inferred that the origin of these cases was placental infection. For, however, excellent a filter the placenta may be, it sometimes fails, thereby causing direct infection of the foetus. Harbitz also quotes another case in which a tuberculous mother gave birth to a six months child which survived only about five hours. Harbitz inoculated three guinea pigs, two with material from the placenta, and the third with portions of the liver and spleen of the foetus. Both the former developed tuberculosis, while the third remained free.

Perhaps the number of cases caused by this means is not very large, and serves to demonstrate, as someone has said, "a pathological curiosity." If it be conceded that infection of the germ cell is possible, I am still at a loss to know, in such cases, how they could be diagnosed from germ cell infection.

(3) THE INHERITANCE OF A TENDENCY TO TUBERCLE.

The soil counts for as much as the micro-organisms, and although this is exemplified more markedly in pulmonary tuberculosis, it is by no means confined to it. Experience has taught

us that there are families more susceptible to infectious diseases than others. Few practioners must have failed to notice that some families seem to get every form of zymotic disease that is prevalent, and others, although equally exposed always seem fortunate enough to escape. Neither can this always be ascribed to environment, nor even in many cases to general physicial condition. It has been frequently noted how in a family in the best of condition, living a healthy life under the best hygienic surroundings, have, one after another, succumbed to pulmonary tuberculosis. W. P. Deweis of Philadelphia, cites a noteworthy case as an example. "The predisposition arose on the side of the mother, who died at 43 years of age from phthisis. She had 23 children, all of whom died under 30 years of age from phthisis. The family lived in the country, was very wealthy, and always accustomed to the means generally found successful in destroying the predisposition, or lessening its influence."

In cases like the above, we are bound to believe that there is the accidental incidence of the tubercle bacillus upon tissues that are susceptible through the influence of heredity. It has been proved by experiment that when the blood and tissues of the body are chemically altered, germ resistance is diminished. It has also been proved that if an animal be impregnated with sugar, it becomes a ready prey to the diphteritic poisons.

What this constitution may be which make some families more susceptible to the tubercle bacillus than others, we will now

proceed to discuss. And, first, I would note the fact, that there are families, who, by inheritance, have a peculiar form of chest, or have contracted, and abnormally narrow nasal cavities necessitating almost continous mouth breathing. In the latter case, it is common for such persons to suffer from laryngeal and bronchial troubles, causing a breach of the epithilium of the mucous membrane, the result of mouth breathing. Without any unusual vulnerability of the pulmonary tissues, it is natural to expect that such persons would have a less resisting power to the influence of the tubercle bacillus, because of the frequent catarrhs affecting their respiratory passages.

The liability to pulmonary tuberculosis amongst those inheriting a peculiar form of chest, cannot be explained in this way. While it must be remembered that flattening of one, or both sides of the chest, may be due to pulmonary disease, it nevertheless, frequently happens that persons who are in good health, have always had a narrow contracted chest, especially in the region of the apex.

R. K. aged 72 years, has always been round shouldered, and, as a result, the chest wall at the lung apices is markedly narrow and flat, and the respiratory movements in that region are limited. He has one son, and two daughters, who show the same peculiarity. Three years ago the son had a slight haemoptysis. Examinations revealed very slight dulness in the second right intercostal space. He was seen by a consultant, who considered the case to be one of pulmonary tuberculosis,

and with a winter in Davos Platz, the disease was arrested. I have frequently noticed that the chest in many families who have a bad history, is deeper and longer in proportion to width, than normal, and it is possible that this may likewise indicate a predisposition. There has been much discussion as to the cause of pulmonary tuberculosis manifesting itself first at the apices of the lungs. As I have previously stated, phthisical patients frequently improve rapidly at high altitudes. Respiratory movements are there necessarily vigorous, and one of the first results noted is an increased chest measurement. The first rib, even in women, is very little raised by inspiration, and in the case of a person with a narrow chest, the respiratory movements at the apex are especially weak, thereby perhaps, weakening the naturally elastic movements of the lung, and causing the air to stagnate in that region. If that is so, the virus will thereby, have an opportunity of settling down and developing.

In support of the theory that pulmonary tuberculosis at the apices may be due to diminished respiratory movement, I would note the fact that in Progressive Muscular Atrophy, and in Bulbar Paralysis, death is frequently due to pulmonary tuberculosis, due, no doubt, to diminished muscular movement, the result of the paralysis. In my opinion, it is therefore possible for such persons who have inherited this unnatural chest formation to develop pulmonary tuberculosis, because of the chest formation alone. But with all that has been said about predisposition, there is still a very large number of cases of

family phthisis to account for - cases in which the most careful physical examination will fail to reveal any abnormal condition of the organs of respiration, or of the chest wall. In these cases there must be some defective vital resistance or inability of the respiratory tissues to combat with the tubercle bacillus, and its spores. This vital resistance varies in different people, being stronger amongst those who do not inherit a strong tendency to tuberculosis. This brings us to the question of immunity. When a person has had an attack of scarlet fever, he has, at least for a considerable period, and probably for the reaminder of his life, acquired an immunity against that disease. But there are others who have never had an attack of scarlet fever, and yet, although regularly exposed to infection, and without any special protection, seem to be successful in evading the disease. This we talk of as a natural immunity. To a varying extent, this would seem to be the case, not only with all the diseases we call fevers, but also in recent times we are beginning to see more clearly the application of the same truth to tuberculosis. For, although the whole subject of immunity is still very speculative, certain facts concerning it have been demonstrated which will in future make the whole subject of more practical use to humanity.

When bacteria grow in some of the tissues of the body, they are usually killed in a few days. It has been found that the blood and tissues of the body will afford abundant nutriment for the bacteria. It is, therefore, not the want of

food supply. Nor is it the toxins generated by the bacteria themselves; for although this may take place when bacteria are grown in test tubes, the living body is capable for a long time of removing these toxins from the site of growth, through the circulation. The only other explanation, therefore, must be the adaptation of the blood and tissues of the body to the bacteria. The cells of the body produce what we call anti-toxins, or anti-bodies, which assist in the destruction of the bacteria, and the neutralizing of their toxins. At one time, the leucocytes alone recived the credit of this work; now we believe that the whole organism is actively engaged in the process, the white blood corpuscles acting as the scavengers of the body. The formation of these anti-bodies is evidently one of great complexity. Ehrlich has introduced what is known as his "side chain theory," and Metchnikoff has propounded his theory of"phagocytosis", both of which may theoretically go a long way towards explaining the formation of the antitoxins; but in both theories, some difficulties will be found in explaining everything concerned with their formation. Recently Wright, Douglas and Leishman, have done much to clear up some of the difficulties. If the blood serum be taken from a person who has had a given infectious disease, and be mixed with the leucocytes of a person who has not had that disease, it will be found, on microscopic examination, that these leucocytes are much more active in the taking up of the bacteria of that disease than they otherwise would have been. Something therefore, circulating in the fluids of the body has been elab-

orated by the presence of the bacteria. These have been called by Wright "opsonins." Other protective substances have been found in human blood which we call "agglutinins."

Agglutinins are substances which enter into chemical combination with tubercle bacilli, in such a way as to immobilise and conglomerate them.

Opsonins are substances which modify tubercle bacilli in a manner which renders them an easy prey for the phagocytes.

Antitoxins may pass from the bodies in the various secretions. Much has been written by Von Behring on the fact that it passes by the milk, and, thereby, he recommends the feeding of children with the milk from highly immunised cows. It has been found in the yolk of eggs, and Bullock has found that it is transmitted from the mother to her offspring.

Perhaps the above account of acquired immunity might have been more suitably described while discussing infection, but it leads up to the point I want to bring out - viz. natural immunity. One species of animal may be very susceptible to a micro-organism, while to another species it may be perfectly harmless. So in man, as I have stated, one person may be very susceptible to fever, while another, although equally exposed, may escape. I am, at the present time, attending a man with a severe attack of typhoid fever contracted through eating uncooked mussels, gathered at the sea-shore near a sewer outfall. He had a companion who partook of a large number of mussels gathered from the same place, and he escaped. Of course the dosage of the virus counts for much. It has been proved by

experiment that you can produce immunity in animals by injecting into the blood a gradually increasing dose of the virus, and if too large a dose be given early, the animal will succumb. Now in Wright's observations on opsonins he came to the following conclusions.:-

(1) That the opsonic power is approximately the same in all healthy people.

(2) That the opsonic power of healthy people varies very slightly, or not at all, from day to day.

(3) That an increased opsonic power is to be expected in these cases where there has been an active response on the part of machinery of immunization, to the stimulus of infection.

(4) That a decreased opsonic power is to be expected in individuals in whom there is an inherent deficiency of opsonic power, or in whom the machinery is becoming exhausted.

It would be too soon yet to be very positive on opsonins, and what may be inferred by their presence in increased, or decreased amount. I have been impressed by the last clause of the fourth conclusion. After all may not the machinery of immunization, or the machinery for the production of opsonins be the important part which heredity may play. What that machinery may be, of course, I cannot explain, but I would submit the following points in support of the theory:- (a) In my opinion persons usually run greater risks of infection during the period of infancy than at any other time of life.

Prof. Von Behring has shown that when anthrax bacilli, free

from spores, were introduced with milk into the stomachs of adult guinea pigs, they were rapidly thrown off by the excreta, and had no effect at all. When the same dose of bacilli were introduced into the stomachs of guinea pigs under eight days old, they died just as rapidly from anthrax, as by the usual method of infection, He next tried anthrax bacilli whose virulence had been reduced. These are harmless when injected subeutaneously into guinea pigs. "After feeding new born guinea pigs with weakened bacilli, the blood of the animals contained anthrax bacilli, though the animals did not die." He next turned his attention to the tubercle bacillus. He fed guinea pigs with a single feeding of the tubercle bacilli, carefully weighed out, and in the case of the older guinea pigs, no result happened, while in the case of the young guinea pigs, they developed tuberculosis. This experiment, he states, proves the ready penetrability of the infantile mucous membrance, and a ready acess to the circulation of the new born, may be gained for all bacteria infecting milk. Of these, not the least common is the tubercle bacillus. But milk may not be the only source of infection to the very young.

Kitasato has pointed out that in Japan the death rate amongst children from tuberculosis is very much the same as in Europe; that the native cattle of the present day are not infected with tuberculosis; and that if a mother cannot suckle her child, the child is usually fed by a foster mother. He believes that the child ingests the bacilli with dirt from the floor. With risks such as these, I think I am justified,

therefore, in asserting that the risks of early infancy contracting tuberculosis must be great.

(b) Tubercular glands is one of the commonest affections in family phthisis. I know of a tubercular stock of five families, and in three of these families there are several children suffering from tubercular glands. Dr Urwick, in his paper on opsonins, points out that it is always found that the opsonic power of people suffering from tuberculous glands, is low.

(c) I have already pointed out that where there is a bad family history, statistics prove that death takes place at an earlier age than where the family history is good.

(d) I have also cited Dr Nikelaus Berend's examination of ninety six children by tuberculin and without a single positive reaction.

Even some imperfect machinery will run smoothly for a time. But from the four arguments stated, and from the fact that tubercular lesions are found in increasing numbers as the age limit advances, would it be too rash to assume that the machinery of immunization, or the machinery for the production of opsonins is usually working smoothly in infancy and for some time afterwards, but becomes more readily exhausted, in these with a bad family history. Unless there be some reasons of this kind, it becomes more difficult to understand, how an infant with all its risks, can so frequently escape the ravages of this awful disease. Nor, so far as I know, can we explain the child's escape on the theory of the bacilli remaining lat-

ent.Professor Harbitz suggests that the tubercle bacilli can only remain latent for a few months, or at the most a couple of years.

ATAVISM. In Locksley's Hall sixty years afterwards" Tennyson says:-

"Evolution, ever climbing after some ideal good,
And reversion ever dragging evolution in the mud."

If we always reverted to a foresire whose constitution had been weaker than the parent, Tennyson would have expressed the whole truth. But inheritance sometimes favours us with the physical characteristics of a grandparent, which were better than those of the parent, and so we have to be thankful to reversion. It is no uncommon experience, on the other hand, to come across cases where pulmonary tuberculosis has missed a generation, and for some unexplainable reason, appears in the grandchildren. Possibly environment may have something to do with atavism. The members of the third generation may not have been so fortunate in circumstances as those of the second generation. At any rate, as the granchild may develop the physical characteristics of the grandparent, and not the parent so in pulmonary tuberculosis, we do find the members of the third generation developing the disease, which was present in the first generation, but has not manifested itself in the second.

CONSANGUINEOUS MARRIAGES AND IN BREEDING. The marriage of cousins, which is at all times, a doubtful procedure, becomes specially dangerous to the offspring where there is a tuber-

cular taint in the family. In the few such families that I have attended, I have found the early manifestations of tubercle either in glands, bone, meninges, or lungs, and that a large number of the family become affected by the disease. It is natural to expect that, at least, the effect would be as bad as in these cases where both parents are tubercular, although there is no blood relationship.

With all the possibilities of healthy individuals of a good family history marrying into families who are tainted, it would not take many generations until everyone belonged to a tainted family, if there was no provision for lessening the effects of heredity. We might then consider ourselves a race of weaklings. I think, however, that in reviewing a number of family histories, we are driven to the belief that nature either acts as an eliminator, by cutting off the unfit in the early period of adolescence, or by the intermarriage of the tainted with those of a strong constitution, lessens to a great degree, the evil effects of heredity in the offspring.

In an adress on "Mendelian heredity, and its application to Man." delivered before the Neurological Society of London, on February 1st, 1906. Mr. W. Bateson, M.A., F.R.S quotes the following remarkable example from vegetable life. "Biffen has found in wheat that the condition of greater resistance to the rust disease is a recessive character, I have seen, myself, his wheats growing, and can attest that that is so. He crossed a breed of wheat which has comparative immunity, or a greater resistance to rust, with one which has less resistance, whose

leaves are yellow with rust. The first generation is affected heavily with rust, like the bad parent. In the next generation, one sees that yellowed plants, with green or recessive ones standing among them comparatively immune; and when the seeds of the resisting plants are saved - they are self fertilized before the flower opens - the offspring are comparatively disease resisting." So may we hope, and from experience we are encouraged in that hope, that some similar wise provision exists for overcoming the evil effects of a tubercular taint in the family.

In discussing heredity, and family predisposition in tuberculosis, so many factors must enter into our consideration, and so many points in the doctrine of heredity are so speculative, that it is difficult to give each its true scientific significance. Nevertheless, it cannot be disputed, that heredity does play some important part in a large number of the cases we meet with, and to under-estimate its value, is to fail to understand the disease in all its bearings. In the close intimacy that exists between an infected mother and her child, there are, doubtless, some opportunities given after birth for infection, but the soil counts for as much as the micro-organism. Further, because the death rate from phthisis is diminishing, that is no proof that the disease is not hereditary. That may only be the result of environment increasing the vital resistance, and, in some measure, overcoming the hereditary influence. In closing this section of my thesis, I cannot do better than quote Luys, who sums up the whole ques-

tion of heredity as follows:- "Heredity governs all the phenomena of degeneracy with the same results, and the same energy as it controls moral and physical resemblance in the offspring. The individual who comes into the world is not an isolated being separated from his kindred. He is one link in a long chain which is unrolled by time, and of which the first links are lost in the past. He is bound to those who follow him, and to the atavic influences which he possesses; he serves for their temporary resting place, and he transmits them to his descendants. If he come from a race well endowed, and well formed, he possesses the character of organization which his ancestors have given him. He is ready for the combat of life, and to pursue his way by his own virtues and energies. But invariably if he spring from a stock which is already marked with an hereditary blemish, and in which the development of the nervous system is incomplete, he comes into existence with a badly balanced organization; and his natural defects are ready to be developed when some accidental cause arises to start them into activity."

CHAPTER 6

IS PULMONARY TUBERCULOSIS COMMUNICABLE, AND, IF SO, UNDER WHAT CONDITIONS.?

I have still a clear recollection of one of my old teachers who is now dead, in words of eloquence and, undoubtedly, with settled convictions, answering this question in the negative. At the close of his arguments he used words something like the following:- "I have never yet seen the love and devotion of a wife, or mother, in nursing a husband or child suffering from phthisis, rewarded for all that love and devotion can do by contracting, through infection, the same fearful malady. The thought of it is intolerable, and would seem to be against all natural laws."

Long before Koch disclosed the microbic origin of the disease, although infection from pulmonary tuberculosis was generally denied, there seems to have been some uneasiness as to the possibility of infection, for nearly all writers, while denying its existence, are careful to be at the trouble to discuss the question. For instance, in Watson's Principles and Practice of Physic - page 216, we read:- "Is phthisis contagious? No: I verily believe it is not. A diathesis is not communicable from person to person. Neither can the disease be easily (if at all) generated in a sound constitution. Nor is it ever imparted, in my opinion, even by one scrofulous

individual to another. Yet in Italy a consumptive patient could not be more dreaded if he had the plague. And in this country the suspicion will now and then arise that the disease may be infectious. A girl dying of phthisis is nursed by her sister, who afterwards droops and dies of the same complaint. Here the presence of the peculiar diathesis is strongly presumable. But the parties may be different in blood. A wife watches the death bed of her consumptive husband; and presently sinks herself into consumption; and there may be no traceable or acknowledged example of scrofula in her pedigree. Yet even here the latent diathesis may fairly be presumed to have existed. Very few families are perfectly pure from the strumous intermixture. The predisposition may be slight; it may be dormant for a generation; or, like other inherited peculiarities, it may alight capriciously on some individuals only of the kindred. In both the supposed cases there have been other influences at work, more authentic than the alleged contagious property in calling forth the fatal malady. Watching, the want of rest, confinement to the unwholesome air of a sick chamber, and, above all, protracted mental anxiety, than which no single cause perhaps has more power to foster and forward the inbred tendency to phthisis. The disorder, I am satisfied, does not spread by contagion. Nevertheless, if consulted on the subject, I should, for obvious reasons, dissuade the occupation of the same bed, or even the same sleeping apartment, by two persons, one of which was known to labour under pulmonary consumption."

I may be pardoned for quoting so fully, but with all the denial of the possibility of infection by Watson, do his views differ very much from the authorities of the present day, who are living under the fuller light of the pathology of that disease called phthisis? We shall see.

It was not until Koch in 1882, described the tubercle bacillus as the causal organism of tuberculosis, that the profession generally gave the question of the communicability of pulmonary tuberculosis any serious and systematic examination.

It was at once recognised from Koch's observations, that if there could be no tubercle without the bacillus of tuberculosis, and if the organism was not directly inherited, then it must come from without. If the sputum of a phthisical patient was known to swarm with tubercle bacilli, what more natural method, in the dissemination of pulmonary tuberculosis could there be than by infection from case to case. Accordingly the British Medical Association issued a circular to the profession on January 6th, 1883, to determine the communicability of the disease.

The circular was a direct question as follows:- "Have you observed any case, or cases, in which pulmonary phthisis appeared to be communicated from one person to another?" 1078 replies were received. 673 were simple negatives containing the word - No, without any further remark. The Collective Investigation Committee of the British Medical Association classified the remainder as follows:-

Class 1 Affirmative observers 261

Class 2 Doubtful observers..... 39

Class 3 Negative observers 105

158 of the affirmative returns refer exclusively to cases observed between husband and wife. Communication between husband and wife is mentioned, together with other cases, in 34 of the remaining returns. So that 192 observers report cases of supposed communication of phthisis occurring between husband and wife; in 69 from wife to husband. It is stated in 130 of these cases there existed no family predisposition to phthisis in the partner to which it was supposed to be conveyed.

Statistics lend themselves to various interpretations, and it is not always safe to make deductions from figures. The Collective Investigation Committee of the British Medical Association, composed, as it no doubt would be, of men of eminence, came to the following decision:- "One fact these returns seem to establish beyond all question, and that is, that if phthisis is a communicable disease, it is only so under circumstances and conditions of extremely close personal intimacy, such as persons sharing the same bed, or the same room, or shut up together in close, ill ventilated apartments."

In the number of cases into which I have inquired, I have found nine in all (8 males, and 1 female) in which it was alleged that pulmonary tuberculosis was contracted through infection from their partner in life, and the history of each case bore out the allegation. The total number of married men

who died from pulmonary tuberculosis was 37, and married women 43. The percentage of cases contracted by infection from an affected partner was, therefore, 11.2. When one considers that the wife, in attendance on a sick husband is usually from anxiety, work and confinement indoors, more likely to go down in health, and be more susceptible to the infection than a husband, it seems remarkable that so many of my cases should be males. Furthermore, my figures are not borne out by the statistics of the British Medical Association Inquiry, and the results must, therefore, be considered as accidental.

Another remarkable feature in the infected males was that two of the eight males were brothers. In no other member of the family was I able to find the slightest evidence of tuberculosis. Both brothers had been married for several years, and were in excellent health at the time of marriage. In both cases, there was a clear evidence of the wives being first affected, and although both couples lived in the same village, there was not much intimacy between the two families.

In all the other six males who died, there was no evidence of any family predisposition. The only female who was supposed to have contracted pulmonary tuberculosis from her husband, showed no evidence of disease until twelve months after her husband's illness started, and she died two years before him. The ages at death were interesting. The ages of the males were 28, 30, 32, 34, 34, 52, 59, 63. The age of the female was 25.

Turning now to the other cases for evidence of infection

I would submit the following:-

1 Margt. Jane R. - aged 59 years, had an excellent family history. She never had any illness of any consequence. She went to Bournemouth for a short holiday to see a lady friend (no relation) who was suffering from phthisis , and while there she lived in close intimacy with her, and slept with her. Shortly after her return, she developed pulmonary tuberculosis, and ultimately died from that disease. I am unable to ascertain the sanitary conditions of the room she shared at Bournemouth, but her own home left nothing to be desired.

2. Gilbert P. died at 13 months of age from undoubted pulmonary tuberculosis. There is a good family history. He was almost daily nursed by a neighbour who suffers from chronic phthisis, and who coughs and expectorates a great amount. I cannot help associating the child's death with the neighbour's illness.

3. Three persons occupied different rooms in a small tenemented property in the village of Ford, which did not provide for through ventilation. These persons were in no way related. In one of the cases there was a bad family history. The unhygienic condition of the property generally, lends countenance to the possibility of direct infection from one to the other.

I now submit some cases of several members of the same family suffering from pulmonary tuberculosis, and living in insanitary houses.

1. Joseph R. M. - died at ten months of age, and the death was registered as due to pulmonary tuberculosis. The house was one of the back-to-back group, and, therefore, did not provide for through ventilation. At the time of the death, the mother and two other children were suffering from consumption.

2. Mary E. L. - died at four months of age, and the death was registered as due to pulmonary tuberculosis. The house was a back-to-back one, and the mother, who ultimately died from pulmonary tuberculosis, was in an advanced stage of the disease at the time of the child's birth.

3. Christopher B. - aged 22 years, died from pulmonary tuberculosis, in a back-to-back house, and at the time of his death two sisters, and one brother, were suffering from the same disease.

I have made use specially of the above cases from back-to-back houses, because, as we shall see, the dosage of the virus is an important factor in infection, and as these houses would be infected by millions of bacilli coughed up by affected members of the household, and as there would be little provision for sunlight, and less provision for through ventilation, the virus would live on and accumulate, and the infection, and re-infection be repeated and constant.

But it is not alone limited to the recognised insanitary houses that infection may take place. I submit the following two instances of deaths in families living in sanitary houses. Of course, there must have been family phthisis in both cases,

but the fact that death took place in such regular sequence, proves that there was something more than family phthisis to account for so many deaths in one family.

In the following case I was unable to get any history of phthisis in the family. Grandparents on both sides lived to a remarkably long age. The mother had died from cancer at 60. The father, who was the only member of the family alive, was 63 years of age, and was in good health. They lived in three small rooms, never opened the windows, and it is possible that at one time there was overcrowding. The whole family of five died at the following ages:

Mary C ..... aged 22 years
George C ..... " 22 "
Thomas C ..... " 21 "
Charles C .... " 21 "
Robert C ..... " 21 "

The next case is more remarkable for the clearing out of a whole family of seven, in eight years. I was unable to get the cause of death of any of the grandparents. The house they lived in was perfectly satisfactory from a sanitary point of view. As the premises were licensed, the police are careful to notice any insanitary conditions, and this factor is considered each year by the licensing bench. The cases would illustrate the danger there is, in licensed premises, from the visitors at the bar, coughing and spitting. The following are the dates of the deaths of each member of the family.

Mary P. - mother died on February 16th, 1894.
Robert P.- son " " " 17th, 1895
Nelly P. -daughter " " December 29th, 1898.
Elizabeth P. " " " June 20th, 1901.
Thomas P. -father " " December 6th, 1901.
David P.- son " " January 12th, 1902.
Frederic P. son " " March 14th, 1902

Hillier states that "pulmonary tuberculosis, as we see it amongst the poor, is a strictly infectious disorder, but that it attacks the well-to-do classes more or less speradically." If this is so, and statistics generally prove it, there must be some conditions associated with infection, and this, more markedly in pulmonary tuberculosis than in any other infectious diseases.

Dr. I. Niven states "tuberculosis is a disease not readily or easily acquired, and the onset of phthisis marks either exposure to intense infection, or else some profound weakness." And again he states, "Although tuberculosis is an infectious disease it is generally, more than any other infectious disease, dependant on assisting causes for its propagation."

Dr Wilson Fox states, "There are few writers who have not admitted the possibility of some contagion, but I venture to think that the evidence, as it stands, shows that, even if this possibility has an authentic foundation, the extent and degree to which contagion ordinarily extends is singularly small." From these statements, therefore, it will be inferred that, fortunately for the human race, the propagation of tuber-

culosis by infection is limited by conditions, each of which may play an important part in preventing, or favouring the virus to settle down. From the evidence of many investigators there seems to be little doubt that there is no scarcity of virus.

Sir Hugh Beever has drawn attention to the fact that the sparse populations of country districts die from phthisis in as large proportion as the populations of towns, provided only that the factor of overcrowding is eliminated. This shows how widely diffused the tubercle bacilli must be. He puts it as follows:- "When death returns, and local agencies inducing susceptibility are considered together they confirm the common opinion - avoiding susceptibility one avoids infection."

A remarkable series of cases bearing out the fact that infection is limited in its action, is mentioned by Dr Andrew in his Lumleian lectures on the etiology of phthisis. Miss R. - aged 48, a dressmaker, living in a rather lonely cottage in Bedfordshire, had three apprentices, young girls from 17 to 19 years of age, not related, from three adjoining villages, who took it in turn to remain in the house and sleep with her, each for one week at a time. During their apprenticeship, Miss R. was taken with phthisis, of which she died. In less than two years afterwards, all three apprentices died of phthisis, although in the family history of each, no kind of phthisis existed. No harm came to any of the friends of the unfortunate apprentices."

Here there must have been conditions concerned with the

cottage favourable to the accumulation and vitality of the virus. The same infection had no power to harm any of the apprentices' friends.

WHAT ARE THE SOURCES OR CHANNELS OF INFECTION:?

1. Inoculation. Pathologists and butchers from time to time contract a local tuberculosis on their hands from the handling of tubercular material. The condition is little more than a warty excrescence, and is entirely local. I have never read of any case of pulmonary, or general tuberculosis occurring as the result of such inoculation, although theoretically, I suppose that it is possible.

2. Inspired air. When the consumptive coughs and spits, he expels billions of bacilli, and if these be deposited in such a position where they may dry, and be not exposed to currents of fresh air, or the sun's rays, their vitality may be retained for months after being ejected by the patient. Fischer and Ransome have from observations, proved that their vitality and resistance may be retained from 130 to 184 days. On the other hand, the bacillus of tubercle perishes rapidly if exposed to currents of fresh air, or direct sunlight. It is important to note that the mere drying of the sputum will have no effect on the vitality, or virulence of the bacilli.

Dr Byron Bramwell has reported a case in which there seems reason to suppose that tubercle bacilli, or their spores had retained their vitality in a certain room for four years. Under conditions of cleanliness, plenty of fresh air, and of sunlight there should be little anxiety concerning the vitality of the

tubercle bacillus, or its spores.

Cornet examined 118 samples of dust from hospital wards, and the rooms of consumptives, and found 40 to be infective, and capable of producing tuberculosis.

Into the nostrils of 29 assistants and nurses at the Charite Hospital, Paris, Strauss placed plugs of cotton wool, to collect the dust of the wards, and in 9 cases he was able to produce tuberculosis in animals from the tubercle bacilli collected in the plugs. So it must be plainly seen from the sputum alone, there must be abundance of infective material to infect the inspired air in a room, or even a house in which there is a coughing consumptive. And here I ought to say that it may not always be possible to trace the source of infection, since a consumptive may infect a room, and, thereby, infect another person, without actually being in contact with that person. There have been some well authenticated instances of successive occupants of a dwelling falling victims to consumption, but in my experience, I have never been able to establish the certainty of a single case, although I have been on the outlook for such cases. Such a condition might be possible in furnished apartments, but the ordinary precaution taken at removals, of cleaning and washing a house thoroughly before the furniture is put in, and the opening of the windows and doors for the purpose of drying, is, I think, sufficient to destroy the vitality of the bacilli.

From the infected inspired air, the poison may take one of several courses. First, it may alight on a larynx, bronch-

ial mucous membrance, or even the terminal air cells of the lungs. Or it may alight on the tonsils, and pharynx, and from that site be absorbed and cause tuberculous disease of the cervical glands. Further the bacilli may be swallowed and be absorbed from the gastro-intestinal tract, producing tuberculous disease of the mesenteric glands.

3. FOOD AND DRINK. It is a well known fact that milk, butter and butcher meat not unfrequently contain large numbers of tubercle bacilli. Probably 30 per cent of the cows in the United Kingdom suffer from tuberculosis, and about three per cent of them have tuberculous disease of the udder. Where there is udder disease, large numbers of bacilli make their way into the milk. Even in some samples of butter, tubercle bacilli will be found in large numbers. Although tuberculous meat is probably used to a larger extent than is suspected, the Royal Commission on tuberculosis was of opinion that the danger from this source was probably exaggerated, as the cooking of the meat would, most likely, destroy the virus; but the risks from milk and butter cannot be in this way overlooked, unless we accept Koch's dictum, that bovine tuberculosis is not transmissible to man. Koch's views were founded on four points, and it may be well at this stage to consider these points.

(a) INFREQUENCY OF PRIMARY INTESTINAL TUBERCULOSIS. From the experience of an enormous number of post-mortem examinations, Koch satisfied himself that primary tuberculosis of the intestinal tract was rarely found, and that in the vast majority

of the cases, the primary lesion was to be found in the bronchial tubes, or lungs. The opponents of this view point to the Registrar General's returns, which show that while pulmonary tuberculosis steadily diminsihes year by year, tabes mesenterica shows but little diminution. Koch's followers discredit these returns. They state that many of these deaths, so registered, are not due to tuberculosis, and if so, are not due to primary diseases of the intestinal tract.

In the British Medical Journal of August 12th, 1905, there is a short account of 400 antoposes on children by Bruning of Leipzig. He notes his experience that in children he has found that several organs, or sets of lymphatic glands, are usually invaded, as distinguished from that of adults, when a single infective focus is commonly found. It is not for me to attempt to express any opinion on this intricate subject, but I have been much impressed by Von Behring's views on the production of pulmonary tuberculosis, as stated in his Cassel Lecture, and if his observations be correct, it should do much to clear up matters, touching which the opinions of authorities are so diverse. Von Behring states, "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. 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 tubercul-

osis 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 the 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 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 develope pulmonary consumption."

In another part of the lecture he states "I have long considered it probable that in many cases in which tuberculous disease is developed, the tubercle bacilli, or their spores which happen to lodge on abraded, or unhealthy surfaces, do not produce local disease at the point where they fall, but are either (a) absorbed and conveyed by the lymphatic vessels to the adjacent lymphatic glands where they lodge, and subsequently develope; or, (b) are carried by the blood vessels and lymphatic glands to more distant tissues and organs." If these views of Von Behring are to be accepted, they must necessarily weaken Koch's first argument concerning the infrequency of primary intestinal tuberculosis.

(b) Koch's next argument is, that milk and butter containing bacilli were often consumed with impunity. This brings us back to the question discussed in the first argument, viz:- the registrar General's returns on tabes mesenterica.

(c) Koch gave instances of inoculation of man with bovine tuberculosis, which had not led to the development of tuberculosis in the persons inoculated. But similar evidence can be obtained against human tuberculosis. Pathologists, like butchers, contract by inoculation, a local warty excrescence, which is really a very limited tuberculosis, but I am not aware of a single authentic case of either general, or pulmonary tuberculosis which has ever been produced either in pathologists or butchers.

(d) The results obtained by Koch, by feeding or injecting into cattle and pigs, bacilli obtained from human tuberculosis, and others with bovine tuberculosis. Broadly speaking the results obtained were that the disease was produced by the use of bacilli from bovine tuberculosis, but that the animals infected with bacilli from human tuberculosis remained free from tuberculosis. I think that it is now generally recognised that tubercle bacilli from cattle are more virulent than are human tubercle bacilli, and if that be the case, for that reason alone, Koch might fail to induce tuberculosis in animals with tubercle bacilli from human tuberculosis; while successful with tubercle bacilli from bovine tuberculosis.

Koch has proved that he can confer immunity against perlsucht on goats, donkeys, and cattle, by the injection of human tub-

ercle bacilli. All investigators prove that you cannot obtain immunity from one virus by the use of another, and, therefore, the only important difference between the tubercle bacillus of human tuberculosis, and that of bovine tuberculosis is one of virulence.

Returning again to our arguments on the channels of infection, it will be seen that for all practical purposes, the results of inoculation may be discarded; that infection by food and drink must be via the gastro-intestinal tract; and that infection by inspired air, may be either by the respiratory passages, by absorption to the cervical glands, or by ingestion through the gastro-intestinal tract. Whatever may be the relative frequency of each mode of infection , it must be admitted that each mode of infection is possible, and therefore, ought to be protected against.

In discussing the conditions under which infection may take place, we have to consider the intensity of the poison, on the one hand, and the resisting power of the individual on the other.

Dr Byron Bramwell discusses each of these conditions under three heads.

(1) The intensity of the poison depends upon (a) dose. (b) the source of the infection and the channel through which the poison is introduced into the system. (c) The virulence of the poison.

(2) The resisting power of the individual depends on (a) His inherent, or constitutional peculiarities. (b) His condition

of health at the time when the virus is introduced into the system. (c) The condition of the tissues and organs with which the tubercle bacillus, or its spores, are brought into contact.

I will now consider the various heads under which the intensity of the poison is discussed.

(a) Dose. In an earlier part of my thesis, I have stated that in small, and not oft repeated doses of the tubercle virus, the natural immunity possessed by man was probably sufficient to overcome such an amount of the virus. Further, from the experiments conducted by Von Behring on cattle, and from the knowledge that tubercular infection amongst adults is common, I think we are safe in assuming that there is a further acquired immuntiy against the tubercular virus. But such a degree of immunity must necessarily fail, if the dosage of the virus be large, repeated, or almost continued for a length of time. Here, too, may be the explanation of the fact, as stated by Hillier, that pulmonary tuberculosis as we see it amongst the poor is a strictly infectious disorder, but that it attacks the well to do classes more or less speradically. The conditions of living, the overcrowding, the badly ventilated and the badly lighted rooms, all conduce, with the presence of a spitting consumptive, to the accumulation of the tubercular virus, and, thereby, produce the opportunities for other inmates of the same house receiving an almost continous infection. In support of the argument, that good sanitary conditions in a house rapidly overcame the baneful effects of the tubercle bac-

illus, I mention the case of a girl, who, after eight years illnes from chronic pulmonary tuberculosis in a religious institution, succumbed to that disease. The house was well appointed and clean, and had ample provision for cross ventilation and lighting. There were usually about 40 other inmates, and from inquiries made, no special provision was made to isolate the invalid from the other inmates. She lived with them, slept in the same room with some of them, dined with them, and, in short, when her health permitted, mixed with them, quite the same as any of the other inmates. Now eight years is a long time, and forty is a large number. Yet the closest inquiry failed to reveal the slightest suspicion that the coughing consumptive had infected any other member of the household. The conditions were such as not to prevent infection, but evidently were sufficient to prevent the accumulation of virus so that the dosage might have harmful results.

Although different doses may have different effects on different people, depending on the individual resisting power, still there may be, and probably there is a dose of the tubercular virus to which all may succumb.

(b) The source of the infection, and the channel through which the poison is introduced into the system.

Experimental inoculation in animals has proved that intravenous injection, and intraperiteneal injection are the most certain methods of producing tuberculosis, and a general tuberculosis is more likely to be produced by intravenous injection. The dog possesses a high degree of immunity against

tuberculosis, yet by either intravenous, or intraperotoneal injection, tuberculosis can be produced, In the more susceptible animals, especially the guinea pig, there does not seem to be much difficulty in inducing a general tuberculosis. On the other hand, from recent experiments, one would infer that in the very young, ingestion seems to be as successful a method, if not more so, for inducing general tuberculosis. Returning now to the channel through which the poison is introduced in man, I am of opinion that, to a certain extent, what is true in the experiments on guinea pigs, is true in man. We may take out of our consideration intravenous, and intraperiteneal injection as a natural channel in man. But in considering either the dangers of inhalation, or ingestion, we ought to take into consideration the age of the patient. To the very young, I cannot conceive a more certain channel than by ingestion. As I pointed out in an earlier part of this thesis, the whole epithelium of the gastro-intestinal tract is in a condition to welcome the virus. And if the first line of defence has broken down, it must be harder for the second line to tackle the invaders, more especially as their numbers will probably be increased.

Excepting the childran, however, there cannot be much doubt that infection by inhalation is the more common, and probably the more certain channel. As in the infection of guinea pigs over eight days old, by ingestion, so in adult man, if there is no breach of continuity in the epithelium of his gastro-intestinal tract, the organism will probably pass through with-

out harm.

(c) The virulence of the poison.

It has already been stated that the bacillus of tuberculosis under certain conditions rapidly looses its virulence, and under other conditions more favourable to the organism, it loses its virulence very slowly. But it is doubtful whether there is any condition outside of its host where it can thrive and multiply. Depending on these conditions there are various grades of virulence from the fresh bacillus of bovine tuberculosis to the dead bacillus. For the remarkable fact has been demonstrated that the introduction of tubercle bacilli in the dead condition, can produce tubercle-like nodules, and, further, it has been proved by S. Stockman that such cases will give a tuberculin reaction. The virulence of the organism may depend on the site of its growth. The inoculation of tubercle bacilli into a guinea pig, from a case of pulmonary tuberculosis, will produce tuberculosis more rapidly than bacilli from a tubercular gland.

We will now discuss the resisting power of the individual.

(a) His inherent, or constitutional peculiarities. This has already been gone into in detail in the section of heredity and family predisposition.

(b) His condition of health when the virus is introduced into the system. Any condition produced by most diseases, or by a lowering of the vitality generally, will be favourable to infection from phthisis. Some authorities consider that the rhuematic constitution is not favourable to the growth of tub-

cle, and that when tubercle does develope the rhuematic diathesis hinders its growth. I have been unable to find any evidence which would go to prove this assertion.

There is little doubt, however, that worry, mental over strain, alcoholism, anaemia, and many other diseases, will increase the susceptibility by lowering the vitality of the patient. It has been noted that typhoid fever has frequently been the forerunner of general tuberculosis, and some writers consider the cause to be due to the disturbance caused by the disease in the gastro-intestinal tract, thereby leaving a ready entrance to the virus. I have reasons to doubt the explanation. I have had two well marked and tedious cases of typhoid fever in the Isolation hospital, who, during the stage of convalesence, have developed acute tuberculosis, and died from that disease. During their illness from typhoid fever, they were fed with milk only, and that was obtained from tested cows. Further, there were no patients around them suffering from tuberculosis, and there was no possible source of infection that I could note. Although there was neither evidence nor history of any tuberculosis when they came into hospital, I am inclined to think that these cases were the result of some previous infection, which became lit up as the result of the lowered vitality from the typhoid fever.

(c) <u>The conditions of the tissues and organs with which the bacillus and its spores are brought into contact.</u>

Diseases, like measles and whooping cough, not only shatter the general health, and lower the vitality, but irritate the

lungs, and induce the bacillus to settle there by whatever channel it has gained access. For changes in the tissues as a predisposing cause to tuberculosis is not only confined to the respiratory, or the gastro-intestinal tract. In the great majority of cases of tuberculous disease of the joint , it is customary to get the history of a sprain, or some similar accident, which, though trivial, but frequently neglected, has prepared the soil, especially in the predisposed, for the settling down of the organism. It would seem to be unnecessary to produce any marked structural change in the lungs in order to induce pulmonary tuberculosis. Any cause lowering the vitality of the tissues seems to be sufficient. Colbeck in his book on "Diseases of the heart" in discussing congenital heart disease,uses these words. "If the patient reaches adult life the most common cause of death is tubercular disease of the lungs. In some instances, the fatal termination is brought about by cardiac failure." In my inquiry I made special notes in every case as to the existence of any illness immediately prior to the onset of pulmonary tuberculosis and the frequency of such diseases as measles, pneumonia, influenza, whooping cough, and even quinsy, acting as forerunners to the tubercular mischief impressed one with the relationship that must exist between the previous change, or weakening of the tissues, caused by the primary ailment and the pulmonary tuberculosis; for here there is a preparation of the soil which not only permits the virus to rest, but in which it will find a fitting nidus. If mere lodgment of the tubercle bac-

illus on the mucous membrane were sufficient to produce phthisis in persons, the larynx and intestine ought to become affected in every case of phthisis. But probably no condition plays such an important factor in the preparing of the tissues for the organism as what we know as catarrh, be it in the respiratory, or gastro-intestinal tract.

Of the number of cases inquired into, no fewer than 32 bore evidence of having suffered for some time with repeated colds, or cold lasting for some time before the illness started. I am aware that the onset of pulmonary tuberculosis frequently similates an ordinary catarrh, but many of these cases have been subjects of catarrh due to living in damp houses. As the late Dr J. B. Russel so aptly puts it, "One of the most powerful predisposing causes of phthisis, and one which we have not yet done much to diminish, is the universal catarrh of the lungs, which is provoked by our damp smoke laden atmosphere. Lungs in such a condition are like the field ploughed and pulverized for the seed scattered by the husbandman."

LATENT TUBERCULOSIS. In Dr Burton-Fanning's book on pulmonary tuberculosis he states "that out of one hundred consecutive cases, nearly half of the number narrated that they had in the past suffered from disorders in various parts, which disorders, there is good reason for believing, were tubercular in nature. Thus in sixteen patients there had been definite disease of the glands - one had suffered from corneal ulcer, and two from ischio-rectal abcess. As many as twenty eight of the patients gave a distinct history of pleurisy, which had

occurred long before the manifestation in the lungs themselves. In twelve instances haemoptysis occurred, which the patients had been assured did not arise from pulmonary disease, and which was followed by no other symptoms until the lapse of a considerable time - in one case twenty years." From cases like these Dr Burton-Fanning, and many others are inclined to argue that a large proportion of the population have some active, although perhaps small and isolated focus of tubercular mischief, ready to break out when there is any lowering of the vitality from any cause. This theory is, no doubt, supported by the results of tuberculin tests which have been made, and by the results of post-mortem examinations on persons who have died from diseases other than those tubercular. It must be remembered, however, that, as I have already stated, dead tubercle bacilli will cause a tuberculin reaction.

Further, that evidence of haemoptysis, although tubercular in origin, twenty years prior to the onset of a recognised pulmonary tuberculosis, is no argument that the second condition was only a continuation of the first. An isolated tubercular focus, however small in extent, must necessarily cause some tissue changes, and although free from the active bacillus prior to the more definite tubercular disorder, it will form a fitting nidus for the organism. On the other hand, there is a strong possibility that many of the definite tubercular lesions which we see, may be only secondary to some other ill-defined lesion, which we have failed to recognise.

The public conscience has been recently awakened, and there

is a general desire to do something to mitigate the dreadful effects resulting from tuberculosis, in all its varied forms. Whether the disease will ever be stamped out is very problematical. At any rate, it is only by being able to appreciate the subject in all its bearings, that we will be able to grasp the many aspects of its etiology, and thereby formulate plans whch may have some measure of success.

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