EXTRACTS FROM

THE MILROY LECTURES

ON THE

ETIOLOGY AND PREVENTION OF PHTHISIS.

Delivered before the Royal College of Physicians of London.

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Lecture II.—The Topography and Distribution of Phthisis.

Ubiquity of Phthisis.—Influence of Climate.—Variations in Countries; in Counties.—Male and Female Phthisis Rates.—Areas of Immunity: a. *In Sparsely Populated Regions;* b. *Elevated Sites;* c. *Sub-Arctic Regions.—Influence of Subsoil.—Malnutrition.—Hardship.—Exposure to the Elements.—Occupation.—Dusts.—Stooping and Constrained Postures.—Variations of Temperature.—Ill-ventilated Workshops.—Rebreathed Air.—Tubercular Infective Areas.—Infected Houses.*

It is not unreasonable to expect that a study of the geographical distribution of phthisis would lead us to the discovery of at least some of the fostering causes of the disease. Our chief sources of information as to the comparative prevalence of phthisis are contained in two great general works, Hirsch's *Geographical and Historical Pathology* and Lombard's *Climatologie Médicale;* but we are able to obtain some data from the *British Army and Navy Medical Reports*, from official statistics of disease in different countries, and from various works by individual medical men.

Lombard's comparative figures are not very satisfactory, as he adopts the fallacious method of comparing the number of deaths from phthisis with each 1,000 of the total deaths—a method that gives a lower ratio from consumption, the higher the general death-rate—and thus a place with double the rate of mortality from phthisis, as compared with another of the same population, may appear in the same grade with it, if only its total death-rate is also twice as great as that of the latter.

Many of the other statistics are also open to objection, but in a general survey, such as we propose to make, it may perhaps be permissible to make use of somewhat imperfect materials.

Hirsch's tables are the most trustworthy, as he gives the mortality from phthisis per 1,000 living, and the years to which the returns apply, together with his sources of information, which are always the best that can be obtained. I have thrown his principal figures into the form of a diagram, which exhibits graphically the variations in the complaint better even than the figures themselves.

The disease appears upon the death-rate of almost every nation. As both Hirsch and Lombard remark, "phthisis is a ubiquitous malady." If we look at the map of the world (page 524), in which various shades are made to denote the presence of the malady in greater or less abundance, it would appear that they are quite justified in their assertions.

With very few exceptions, the degrees of shading denoting variation of prevalence serve also to denote variations in the relative density of the population in different parts of the globe. In other words, in proportion to the numbers of the people aggregated together upon a given space of ground, so also is the rate of mortality from phthisis.

In Dr. Lombard's maps the only portions of the earth's surface from which colour is entirely absent are the arctic and sub-arctic regions, deserts, and high ranges of mountains, and it is precisely in those parts that human beings are fewest and most sparsely scattered over the ground.

In all the capitals of countries, and in the chief cities of Europe, Asia, Africa, and America, there is but little difference in the phthisis-rate, and what differences there are are not to be accounted for by difference in climate. (Diagram.) We are impelled to similar conclusions by a survey of the army and navy returns from all parts of the world.

In the light of our present knowledge of the essential cause of phthisis there is nothing surprising in our thus finding the disease wherever human beings are gathered together, for, so far as we know, the chief source of the organism that provokes the malady is the human race. If, however, we break up the figures presented to us into smaller groups—in other words, if we try to analyse the returns of the disease—we shall find out that it has its predilections, and by observing more narrowly its selection of favourite habitats and breeding grounds we shall obtain important indications that will guide us to a discovery of the conditions that determine its choice.

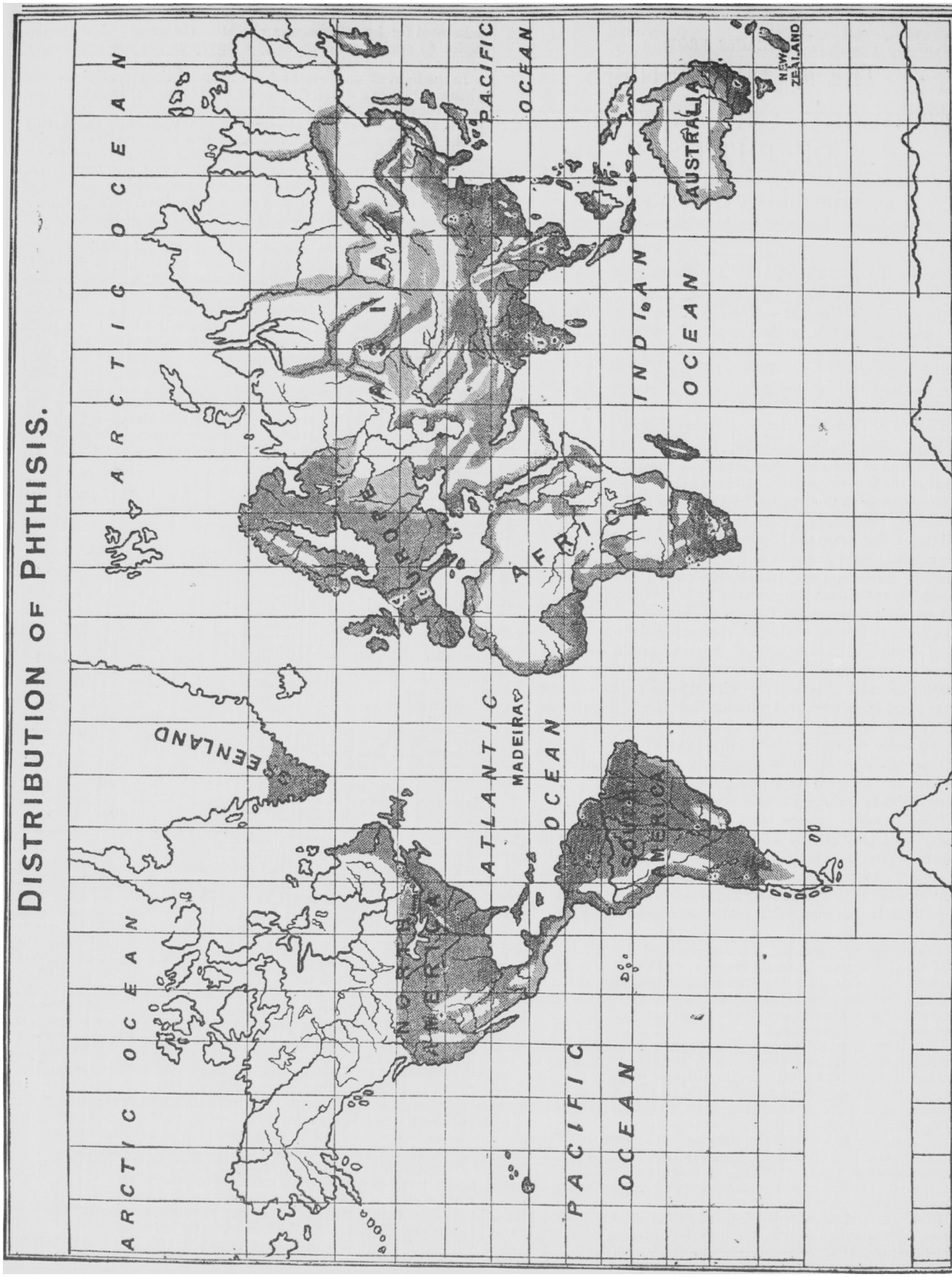
We have already seen reason in the almost universal presence of the malady to conclude that the ancient doctrine of its origin in a damp, changeable climate is erroneous. If we take the map of almost any country in the world, and shade its several provinces so as to denote the varying prevalence of the disease, we shall discover not only that a great variety of tint has to be used, but also that the dark parts alternate with the light, without any reference to geographical position, north or south, east or west. We may take the variations in different parts of England, avoiding the places whose returns might be vitiated from the presence in them of large hospitals. There are only two exceptions to this rule, namely, Hastings and Liverpool; the difference between the highest and lowest returns is more than 50 per cent., and in some the highest reading is more than three times that of the lowest.

Variations in the Consumption Rate at Ages 15 to 55 per 100,000 living at those Ages (Males) (1861-70).

Counties.	Towns.	Highest.	Lowest.
Surrey	Guildford	526	...
	Farnham	—	242
Sussex	Hastings	(H) 643	—
	Battle	—	180
Oxford	Headington	566	—
	Banbury	—	211
Cambridge	Whittlesea	469	—
	Wisbech	—	230
Norfolk	Walsingham	439	—
	Fligg	—	257
Wilts	Salisbury	438	—
	Mere	—	201
Cornwall	Redruth	461	—
	Launceston	—	223
Lincoln	Spilsby	361	—
	Caistor	—	205
Lancashire	Liverpool	(H) 602	—
	Wigan	—	249
York	Reeth	589	—
	Settle	—	253

The same variation is to be found in other parts of the United Kingdom. Thus, in Scotland consumption is almost unknown in the Western Hebrides, but in towns on the west of the mainland, with a very similar climate and a similar race of people, it is very common. In Edinburgh the rate per 1,000 deaths is 102; in Glasgow, 371. In Ireland, again, the rate of mortality from the disease per 100,000 of the population in the ten years, 1865 to 1874, was in the eastern division, 259.62; in the western, 95.64. Similar variations are to be observed in other parts of the world. Lombard gives the number of persons dying of consumption per 1,000 deaths in different places as follows: In Canada the numbers vary from 241 in New Scotland to 138 in Quebec. In Russia, from 190 in Archangel to 30 in Riga. In Holland, from 141 in La Drenthe to 64 in Zealand. In Belgium, from 240 in Limbourg to 122 in Luxembourg. In Germany, Dresden 147; Weimar 74. In Italy, Milan 132; Turin 83. In Portugal, Lisbon 115; Malaga 54. In Egypt the general rate is 25, in Cairo 101. These variations cannot be due to climate, and probably arise from differences in the density of the population and in their mode of living.

But perhaps the strongest proof of the existence of causes much more powerful than climate in producing a tendency to consumption is to be found in the great differences between the male and female rates of mortality from the disease in the same towns and districts. In the following table I have selected from "Lowe's Tables" the male and female rates (1) in places where



the male rate exceeds the female rate and (2) in places where the female rate is much the highest.

Annual Average Proportion of Deaths from Consumption between the Ages of 15 and 55 per 100,000 Persons living at those Ages between the Years 1861 to 1870.

Towns.	Males.	Females.	Differences.
Cambridge	570	395	− 175
Whitechapel	560	430	− 130
Bath	540	255	− 185
Greenwich	525	375	− 150
Brighton	520	345	− 175
Southampton	500	385	− 115
Birmingham	475	345	− 130
Newcastle	470	395	− 75
Salisbury	440	305	− 135
Sedbergh	365	615	+ 250
Congleton	360	595	+ 225
Bootle	225	555	+ 330
Leek	355	525	+ 170
Belper	275	455	+ 180
Buckingham	275	455	+ 180
Sevenoaks	290	455	+ 165
Alston	285	425	+ 140
Camelford	230	295	+ 65
Battle	180	365	+ 185
Pickering	160	315	+ 155
Billesdon	120	265	+ 145

The differences between these figures, concerning two groups, male and female, of the population of the same places, constitute the main point of my argument at present, but I shall presently have occasion to return to them, and attempt to draw from them the other lessons that they teach.

In Lombard's maps we see that there are certain broad spaces untinted, showing regions still untainted by the disease. These spaces may be grouped broadly under three heads:—

1. Deserts, or sparsely populated places.
2. Mountains or high plateaux.
3. Arctic or sub-arctic regions.

It is not merely the scarcity of possible victims that makes the disease uncommon in deserts, for the proportion of cases to the number of inhabitants in these districts is also very low, so far as we can judge from the imperfect data at our command. Thus we are told by both Lombard and Hirsch that there is almost complete immunity from the disease in Nubia and Upper Egypt, that it is very rare in Abyssinia and Central Africa, in the capital town of Oran, with 25,000 inhabitants, it is practically non-existent, and Livingstone states that it is quite unknown in South Africa and in the interior of Cape Colony. It is rare in Asia Minor, Mesopotamia, Syria, Arabia, and Persia. In the steppes of Tartary, the interior of Australia, and in Tasmania, it is almost unknown.

There are, however, certain considerations, apart from the wide separation of these districts from one another, that prevent us from ascribing this freedom from disease altogether to the climate. These spaces are often bordered by regions where the disease is much more frequent. Thus in Asia Minor it is often met with with on the coast or in the principal towns. The Bedouins on the coast of the Red Sea, "who exchange their tents for stone-built houses," suffer from consumption.[1] In Syria it is met with at Aleppo, and in the Soudan at Khartoum. In Zanzibar it is said to be especially common among Arabian women of the higher class, probably on account of their seclusion. Whilst it is rare amongst the native Persians, who live an almost open-air life, it is more common amongst foreigners. In Algeria, whilst the nomad Arabs are free, "amongst the captives many die from the disease;" and in Egypt it is noted that whilst Syrians, Turks, Armenians, and Europeans seldom contract the complaint, Jews often become scrofulous, and die frequently of consumption. At Alexandria the mortality is nearly double that in England. In Australia it is almost as common in the large towns as it is in Europe. All these facts are significant of the influence of indoor life.

Again, we have to note the fact that in many parts of the world formerly almost free from the disease, it is now rapidly increasing in prevalence. Information to this effect comes to us not only from Australia, but from Greece, Brazil, New Zealand, and the United States of America. Some observers state (Hirsch) that consumption is unknown in the western prairies of Illinois, Missouri, Iowa, Wisconsin, and Utah, and also in the Rocky Mountains, but according to others it has of late begun to show itself much more frequently, especially amongst the female immigrants. The climate has not changed, but the conditions under which life is carried on, especially the crowding together of the inhabitants, have altered, hence the increase of the disease.

In all parts of the world, and in all climates, an elevated site seems to be to some extent a safeguard against consumption. The degree of elevation conferring this immunity was supposed by Gastaldi (who was one of the first to notice it) to be over 600 mètres—about 2,000 feet—the disease being as common just under that height as on the plains. But in Western Texas, at a height of 2,100 feet, consumption is not uncommon, and the same observation has been made respecting the elevated plains of Castille and Leon, and of the Styrian highlands.[2]

The population of these elevated regions is much less dense than that of the lower ground, and the air much less polluted with organic matter. That the immunity is not simply due to the rarity of the air is completely proved by Dr. Emil Müller, who has shown that there is no complete immunity from the disease in Alpine Switzerland. A certain proportion of the inhabitants of these high places die of the disease, the rate depending not upon the elevation of their dwellings above the sea, but upon the nature of their occupations. Industrial indoor pursuits give a rate varying from 6.5 to 10.2 per cent., and one of the highest of these rates (9.8) is at an elevation of 3,400 to 4,400 feet; at 4,400 to 5,000 feet, in mixed labour, the rate from the disease was 7.7 per cent. Here, then, we are obliged again to fall back upon the conditions of social life as a much more powerful factor than the influence of climate or locality.

It has been shown by Dr. Bowditch in America, and by Dr. Buchanan in England, that places situated upon a damp impenetrable subsoil are much more subject to consumption than those placed upon a porous soil; good drainage, also, has been found to diminish the prevalence of the disorder by as much as 50 per cent. These results have since been confirmed by Dr. Haviland and by the Registrar-General of Scotland.

And yet there is no necessary connection between dampness of soil and consumption. Dr. Buchanan's experience with respect to the influence of drainage of towns has not always been confirmed by other observers; thus Dr. Kelly, Medical Officer of Health for East Sussex, has shown that in 1861-70 the order of the several districts named by Dr. Buchanan had changed from that of 1851-60 without any difference of drainage. At Ashby-de-la-Zouche the mortality from phthisis rose 19 per cent. after the ground was drained. In Brunswick, according to Reek, "the mortality from consumption has not been greater in the wet parts of the town than in the quarters on a dry soil." "In Dantzig, where a system of main drainage was carried out fully in 1871, the death-rate from phthisis, which had been (according to Licoin) 2.12 per 1,000 in the eight years preceding (1861-70) rose in the nine years following (1871-79) to 2.48 per 1,000." "At Berlin, in like manner, no notable effect in the prevalence of phthisis can be traced to drainage of the ground,"[3] and Hirsch says: "It seems to me to be a more probable explanation that other etiological factors beside the influence of soil come into the account under the given circumstances—factors that have a modifying effect upon the amount of the sickness, and serve to neutralise the benefits even of the most favourable conditions of soil."[4]

Moreover, there are in different parts of the world large tracts of country that are excessively damp, many of them, indeed, wholly given over to malaria and to intermittent fevers, and yet these districts are so free from the prevalence of consumption that some Continental writers have supported the view that ague is antagonistic to phthisis.

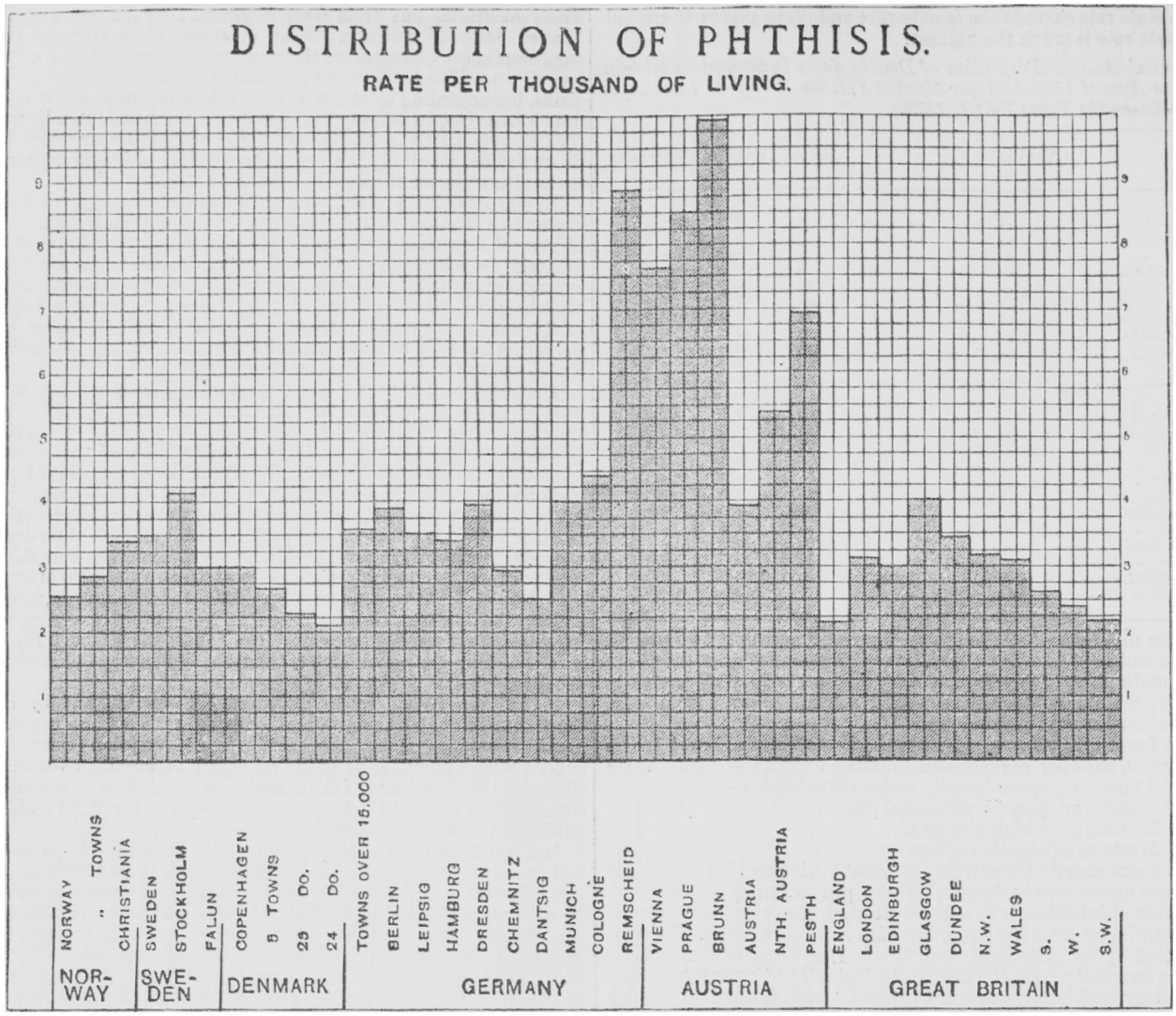
The last group of districts to a great extent exempt from consumption consists of the arctic and sub-arctic countries of Green-

[1] Hirsch, vol. ii, p. 65.

[2] Dr. Marcet has recently carried on a series of observations upon the influence of altitude upon human respiration which may throw some light upon the advantage of high levels in the treatment of phthisis (*Proc. R. S.*, vols. xxvii, xxviii, xxix, xxxi, and xlvi). He proves that the volumes of air required to yield the oxygen necessary for the production of a given weight of carbonic acid are smaller on mountains under diminished pressures than the plains under high pressures (the mean difference was 12 per cent.); and he concludes that the respiratory function is, therefore, carried on more perfectly at these heights than in the plains.

[3] Hirsch, pp. 203-4.

[4] Hirsch, p. 204.



land, Labrador, Iceland, Spitzbergen, Nova Zembla, Finland, Siberia, Canada, and the northern parts of the North American Continent.

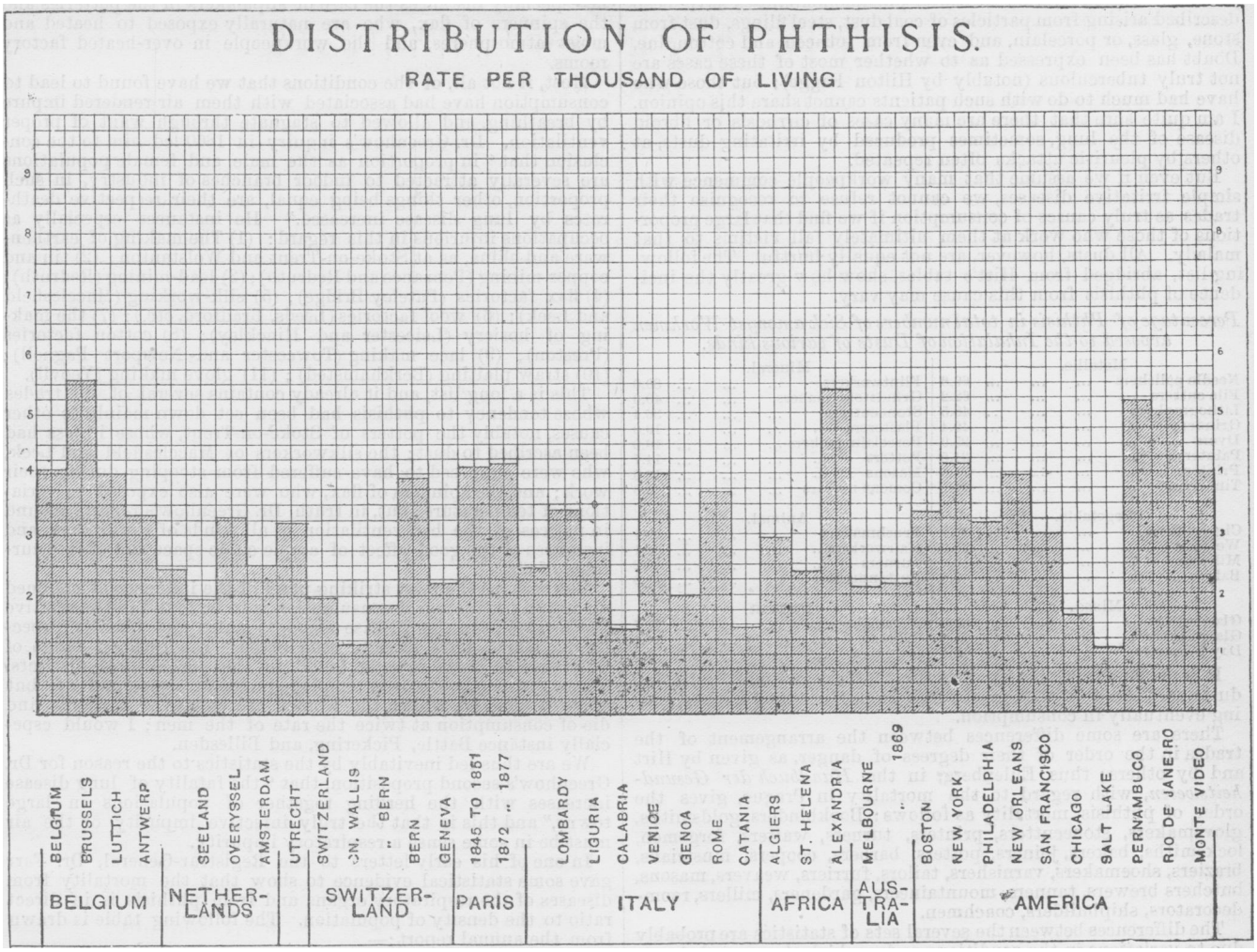
It is now certain that not only is there comparatively little consumption in cold climates, but that many people predisposed to the disease or actually consumptive derive great benefit from a sojourn in the keen, dry, cold air of Canada, or from a winter spent amidst snow and ice at Davos Platz or the Engadine. But it is not yet clear in what way a cold climate exerts this favourable influence; we may, indeed, surmise that the lower amount of humidity in the atmosphere of these regions may have something to do with the result; cold air has a much smaller capacity for aqueous vapours than warm air. It may thus be less capable of sustaining the life of the microscopic organisms that are the exciting cause of consumption; or, again, it is possible that there are smaller quantities of organically charged vapours arising from the ground, frozen as it is for so large a portion of the year. Moreover, in a frosty air the condensed moisture may entangle the organisms in its meshes, and may carry them down out of harm's way.

But all these suggestions are mere guesswork, and there are as yet no certain proofs as to the mode in which the tendency to consumption is lessened in these regions.

Here again, also, we must note the circumstance that all these countries are very sparsely populated, and that there is, therefore, comparatively little respiratory impurity constantly floating in the air; moreover, just as it was found possible for men to override the beneficial influences of an elevated site, so in cold climates also they may live under conditions such as will lead to consumption as certainly as in less favoured countries. The Esquimaux are not seldom affected by consumption, and the fact is hardly strange when we consider the mode in which they exclude all the air they can from their crowded huts. In Greenland phthisis is one of the commonest causes of death, and similar reports come from New Archangel and Alaska. In Canada also the mortality from this disease amongst the troops in the years 1830 to 1837 was no less than 23 per 1,000 of strength, while in healthy districts in England and Wales, at ages corresponding to those of the soldiers, the rate was only 7.7. In all England the rate was 9.2; and in Manchester, with all its unfavourable surroundings, it was 12.4, little more than half the military rate in Canada. The cause of this heavy mortality was discovered to be bad drainage and want of proper ventilation of the barracks, and after these defects had been remedied from 1863 to 1872 the rate was 9.49, and in 1874 it was only 6.0.

We may next inquire statistically into the influence of malnutrition. It is not surprising that at one time tubercular disease should have been ascribed to some form or other of bad nutrition, and especially to a deficiency of fatty food. Thus it was found by M. Marc d'Espine that, at Geneva, in every 1,000 deaths amongst the poor, 233 were from consumption, whilst of the rich only 68 were from this disease.

The terrible mortality from phthisis that prevailed at one time amongst the finest soldiers of the British army was certainly not brought on by starvation or misery. It occurred for the most part when they were not on active service, but in the time of peace, when they were well fed and well cared for, so far as their bodily comfort was concerned, far better in fact than the half starved workpeople and labourers, who only died of the disease at one-third the rate that they did.



Similar remarks, so far as the army and navy are concerned, would apply to the next series of supposed causes of consumption, namely, hardship, exposure to the weather, and cold.

The poor fishermen of Iceland, the hunters and trappers of North America, the nomad tribes of Asia and Africa, the wretched natives of Australia, all these people escape the disease almost entirely, whilst one-third of the deaths of the well protected, well clothed adult inhabitants of towns are from this cause. The Highlanders who inhabit well-built houses on the mainland of Scotland are subject to the same rate as the other inhabitants, whilst the ill-fed, ill-clothed fishermen of St. Kilda and the Hebrides, who are of the same race, hardly ever contract the disease.

It is very doubtful whether exposure to the elements, taken alone, is a common cause of these inflammatory attacks of the respiratory passages. We do not find that men who are much in the open air, and exposed to all vicissitudes of weather, are especially prone to catarrhal affections of the lungs.

A close inquiry into the figures laid before us gives not only negative but positive information as to the secondary causes of consumption, and we may safely draw the following conclusions from them:

1. That the occupations of a people have an important influence over the disease.

2. That its fatality increases with the herding together of populations in large towns.

The mere fact that, in most countries where manufacturing processes are carried on, the worst places are seen to be the manufacturing centres, would in itself be sufficient to rouse suspicion on this head.

In our own chart the two highest columns of disease are those of Brunn, in Austria, and Remscheid, in Germany, and both of these are actively engaged in manufacture, the former of woollen goods, and the latter of iron and steel. Nor are we left without direct testimony on this point: Drs. Headlam Greenhow and Arlidge in England; Finkelburg and Schweig in Germany; Meynne in Belgium; Boudin and Chatin in France—all these observers concur in attributing a malefic influence to certain kinds of work. In his report to the Privy Council in 1858, Dr. Headlam Greenhow pointed out the influence of occupation as a cause of pulmonary disease, and in 1860 and 1861 he followed it up by two admirable special reports upon the subject.

Without going into the minutiæ of these reports, it may be sufficient to summarise his conclusions as to the conditions that render workpeople peculiarly liable to these diseases by arranging them under four principal heads:

1. The inhalation of dusts of various kinds.
2. Stooping postures at work.
3. Exposure to changes of temperature.
4. Bad ventilation.

In considering the etiology of phthisis we are bound to follow the example set by Dr. Greenhow, and to decline to accept the figures placed before us as due to true tubercular disease. It is needful to attempt to distinguish between the direct effects of the occupation itself and those of the ordinary modes of life of the worker.

With these reservations, we may turn to the conditions mentioned as causes of pulmonary disease. It is certain that the kind of disorder at first produced by inhalation of dust is not true

phthisis. Various forms of these pneumonokonioses have been described arising from particles of coal dust, steel filings, dust from stone, glass, or porcelain, and even from tobacco and cotton flue. Doubt has been expressed as to whether most of these cases are not truly tuberculous (notably by Hilton Fagge); but those who have had much to do with such patients cannot share this opinion. I am quite sure that there are many cases of cirrhosis or fibroid disease of the lung, sometimes produced by irritating dusts, at others by pleuritic attacks often repeated.

But even if we assume that many workpeople commence with simple irritative diseases, we cannot refuse to recognise their trades as truly causes of consumption if we find that large proportions of those who work at them ultimately fall victims to that malady. All dusts, however, are not equally hurtful. The following list, abridged from Hirt's tables, show how greatly the incidence of phthisis from this cause may vary.

Percentage of Phthisis in total numbers of Sick amongst Workmen exposed to the Inhalation of Dusts of various kinds.

Metallic.		**Mineral.**	
Needle polishers	69.6	Flintworkers	80.0
File cutters	62.2	Grindstone makers	40.0
Lithographers	48.5	Stonecutters	36.4
Grinders	40.4	Plasterers	19.0
Dyers	25.0	Porcelain makers	16.0
Painters	24.5	Potters	14.7
Printers	21.6	Masons	12.9
Tinkers	14.1	Cement makers	8.0
Vegetable.		**Animal.**	
Cigarmakers	36.9	Brushmakers	49.1
Weavers	25.0	Hairworkers	32.1
Millers	10.9	Turners	16.2
Bakers	7.0	Buttonmakers	15.0
Mixed.		**No Dust.**	
Glasscutters	35.0	Shoemakers	18.7
Glassmakers	17.8	Glovers	10.0
Day labourers	15.1	Butchers	7.9

It is evident from this list that the sharpest and most irritating dusts are those that tend most surely to produce lung disease, ending eventually in consumption.

There are some differences between the arrangement of the trades in the order of their degrees of danger, as given by Hirt and by others; thus Eulenberg, in the *Handbuch der Gesundheitswesen*, with regard to the mortality in Prague, gives the order of phthisis mortality as follows: Bookbinders, goldsmiths, glovemakers, stonecutters, printers, turners, waiters, forgemen, locksmiths, bakers, joiners, potters, barbers, coopers, musicians, braziers, shoemakers, varnishers, tailors, furriers, weavers, masons, butchers brewers, tanners, mountaineers, gardeners, millers, room-decorators, shipbuilders, coachmen.

The differences between the several sets of statistics are probably due to variations in the conditions under which the several classes of artisans did their work, but they add to our reluctance to receive the figures as evidence of the influence of the occupations themselves, and make it probable that the conditions in question had at least as much to do with the result as the occupations themselves.

The next great exciting cause of phthisis mentioned by Dr. Greenhow is the interference with the respiratory apparatus by posture, and he adduces as evidence the heavy mortality of the women and girls employed in the lace and hosiery finishing and winding rooms at Nottingham, the watchmakers of Coventry, the handloom weavers of Macclesfield and Leek:—

Death-rate per 1,000 living from Pulmonary Affections.

	Males.	Females.
Nottingham	8.13	7.03
Coventry	6.61	5.73
Macclesfield	7.43	8.51
Leek	7.80	8.51
Six Northern Standard Districts	2.97	3.04

On the other hand, it is only right to notice the observation of Rokitansky, Wagner, and others, which has been generally confirmed by experience, that hunchbacks are but seldom affected by tubercular disease of the lungs.

Exposure to variations of temperature, moisture, etc., includes the third class of conditions that, according to Dr. Greenhow, render workpeople peculiarly liable to pulmonary disease, and he especially instances the case of slipmakers in the potteries and the spinners of flax, who are naturally exposed to heated and moist atmospheres, and the workpeople in over-heated factory rooms.

Most, if not all, of the conditions that we have found to lead to consumption have had associated with them air rendered impure by breathing, and allowed to stagnate through want of proper ventilation. Dr. Greenhow's inquiry in 1860 led him to the conclusion that "in proportion as the male and female populations are severally attracted to indoor branches of industry, in such proportion, other things being equal, are their respective death-rates by lung disease increased." He instances especially as occupations injurious in this regard: (1) The making of earthenware and china, as at Stoke-on-Trent and Wolstanton; (2) tin and copper mining (Penzance and Redruth); (3) lead-mining (Redruth); (4) flax factories (Pateley Bridge); (5) silk-working (Macclesfield and Leek); (6) wool factories (Leeds, Bradford, etc.); (7) the making of hosiery (Leicester and Hinckley); (8) cotton factories (Preston); (9) lace making (Towcester and Newport Pagnell); (10) straw plaiting (Berkhamsted); (11) glove making (Yeovil).

This is a long list, and it already contains several of the trades whose tendency to phthisis had been set down mainly to other causes, notably the potters of Stoke-on-Trent, whose illness had been ascribed to dust; the silkworkers of Macclesfield and Leek, who were supposed to have suffered from stooping during their work; and the spinners of flax, who were also exposed to variations of temperature; but, in truth, Dr. Greenhow's reports abound in notices of the bad ventilation of all kinds of workrooms, and instances of the good effect of ample cubic space and of free currents of air.

But perhaps the most striking proof of the influence of confined air in the production of consumption is to be found in the relative death-rates from this disease amongst males and females respectively, which have already been given. The greatest range of difference in these rates is found in the agricultural districts, where the men are mostly employed out of doors, and are but slightly affected, whilst the women are employed at home, and die of consumption at twice the rate of the men; I would especially instance Battle, Pickering, and Billesden.

We are thus led inevitably by the statistics to the reason for Dr. Greenhow's second proposition, that "the fatality of lung disease increases with the herding together of populations in large towns," and this is that the truly infective impurity of the air must be in some sense a respiratory impurity.

In one of his early letters to the Registrar-General, Dr. Farr gave some statistical evidence to show that the mortality from diseases of the respiratory organs and from phthisis is in direct ratio to the density of population. The following table is drawn from the annual report:—

Mean Mortality in Three Groups of the Thirty-two Metropolitan Districts, 1839.

Districts.	Square Yards to one Person.	Annual Rate per 100,000.		
		Totals.	Respiratory Organs.	Phthisis.
1 to 10	57	3,321	822	478
11 to 20	78	2,839	768	451
21 to 30	217	2,169	583	354

But the truth was hardly fully accepted until Dr. McCormac took up the subject, and with iteration insisted that "wherever there is foul air, unrenewed air, impure air.....there we meet consumption; there we meet scrofula and an untimely death."

The ravages committed in the ranks of the army by pulmonary disease are to be traced in a great degree to the vitiated atmosphere generated by overcrowding and deficient ventilation, and the absence of proper sewerage of barracks. In reference to this, Dr. Farr said: "The prevalence of phthisis in the armies of Europe is probably due in part to the inhalation of expectorated tubercular matter, dried, broken up into dust, and floating in the air of close barracks," and Dr. Parkes remarks that this prevalence "can scarcely be accounted for in any other way than by supposing the vitiated air of the barrack room to be chiefly at fault."

But we have still not quite reached the limit of the aid to our inquiry that may be obtained from statistics of disease. We may push our investigations into still closer quarters, and, abandoning total death-rates of institutions and towns, we may examine into the mortality of certain districts and even of certain houses. In a paper on "Tubercular Infective Areas," read two years ago before the Epidemiological Society, I gave the details of an inquiry made into the incidence of phthisis in some of the worst districts of Manchester and Salford, an inquiry that was greatly facilitated by the medical officers of health of those boroughs and by the excellence of the mortality tables which they placed at my disposal. Its results showed that the portions of these districts most affected by the disease were the close courts and alleys, the shut-in or blocked-up lanes, and, above all, the houses built back to back, with no through ventilation. I especially noted the cases in which, in the space of five or six years included in the inquiry, double or treble occurrences of the disease had taken place in the same houses, and I found them very numerous. These results have been confirmed by other observers; thus, Dr. Niven, of Oldham, writes to me respecting 3,001 deaths from tuberculosis which occurred in that town during eleven years (1877 to 1887), and states that they took place in the worst class of houses, and in 302 cases there were two or more in a house. He has calculated mathematically the chance of any one house being twice affected (not infectively), and he finds that only 68 on this hypothesis would have thus suffered, whereas 274 were so attacked: and whilst the chance of thrice suffering was only 7.6, 24 were so affected.

Again, Dr. Flick, of Philadelphia, has recently carried out an elaborate topographic study of phthisis in that city, extending over a period of twenty-five years, and he draws the following conclusions:—

1. "That a house which has had one case of consumption will probably have another within a few years, and may have a very large number of cases in close succession."
2. "That when a case of consumption occurs in a house, approximate houses are considerably exposed to contagion."
3. "That houses in localities where endemic after endemic has existed have nevertheless escaped the disease."
4. "That tuberculosis of different kinds occurs in the same localities and often in the same lots as consumption."
5. "That during the twenty-five years scarcely 20 per cent. of the houses of the ward were so affected."

He ascribes these results to contagion in the houses themselves.

These facts must be placed in apposition with Dr. Cornet's researches upon the bacillus-holding properties of the walls of houses in which consumptive patients have resided. They probably afford an explanation of many of the facts detailed in the course of this lecture.

REMARKS
ON
SOME FACTS ILLUSTRATING THE EARLY STAGES OF LEPROSY.

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(*Continued from page 343.*)

The cases which I gave in my last paper were published in order to illustrate, amongst other points, the mode in which leprosy first reveals itself. There is no doubt that there has arisen quite recently a tendency to revert to the old and utterly exploded idea that leprosy is a disease which spreads by contagion. This has been due to several circumstances: first, the discovery of a bacillus which at once placed the disease by the side of tuberculosis and other infectious maladies; secondly, the reported success of inoculation in the case of the Sandwich Island convict, Keanu; thirdly, the seemingly inexplicable character of the recent outbreak in the Sandwich Islands; and lastly, the publication of two or three reputed instances of observed contagion, notably the Dublin case. It becomes, therefore, in view of the revival of this opinion, to ask whether there is anything to be noted in the early stages of the disease which favours it, or in any way explains the mode of supposed transference. If leprosy can be transferred from one individual to another, is such transference affected through a lesion in the skin or mucous membranes, or is it by lung-inhalation? Is the disease contagious in the proper sense of the word, or is it infectious? Or lastly, do the clinical facts oppose the belief in either.

My first proposition shall be: *That there is no evidence whatever in support of the belief that leprosy ever has a primary lesion, or that its transference is effected by means of a sore on the external parts.* There is never anything which corresponds to the chancre in syphilis or the ulcer in malignant pustule. Not even the most zealous advocates of contagion have ever described any such occurrence. It is, indeed, remarkable how uniformly the discussion, or even the mention, of such a possibility is avoided. The conclusions to be derived from a perusal of the cases which I have published are, I may here say, in precise conformity on this point with those of all previous observers who have attended closely to the matter.

There remain open to debate as to the early manifestations of leprosy only a few, and those comparatively unimportant, points. All agree that by far the most common revelation of the malady consists in the appearance of one or more dusky patches of erythema in the skin. These patches multiply, and after a longer or shorter time are developed symmetrically, and over all parts of the limbs, face, and trunk. All such patches assume after a time the characteristic features of anæsthesia in their centres. The first patches are often so insignificant that they are overlooked by the patient; indeed, this is the usual event. Leprosy is seldom suspected until it has been in active existence for several years—a fact of no little importance in connection with the present demand for the systematic segregation of lepers in order to prevent contagion.

The brown patch above described as the first manifestation may in the first instance be a single one; indeed, if discovered soon enough, it probably is so usually. It may remain single for long, that is, for some months—possibly, in rare instances, for a year or more. In such cases the suspicion may arise that this patch is itself the primary lesion, and that the poison is breeding in it prior to its general diffusion. Not improbably the latter half of this proposition is true. We may with much probability suspect that the bacilli of leprosy do multiply in its patches rather than in the blood, and that, gaining access to the substance of wandering cells,[1] they are by them carried to other parts. Thus each patch may be a focus for increased contamination.

In this feature leprosy probably has analogies with infective tuberculosis, with multiple lupus, and a host of other diseases. It does not follow, however, from this admission that the first patch of erythema which appears is the primary lesion of the disease or that it has itself begun from local poisoning. Such a view of the facts is negatived by the following considerations: First, there is never any history of a local sore or injury having preceded the patch, the epidermis of the patch is always unbroken, and the changes always begin in the deeper layers of the skin. Secondly, it is common for these primary patches to appear on several parts of the body simultaneously; they may be quite symmetrical. Thirdly, the patches which are to follow will all be usually similar in all features to the original one. It seems, therefore, far more probable that the early erythema patches of leprosy are due to implanation of the bacilli from the blood than from external contagion. I have at the present time under observation a case which gives point to the above considerations. A boy of 12, born of English parents, in a leprosy district, has a single patch on one forearm of unmistakable character. It is not bigger than a shilling, has dusky, slightly scaly edges, and a whitish anæsthetic centre. On the most careful inspection of the whole surface no other patch can be found anywhere. If there is any exception to this statement it is that the skin near to the patch is at one point a little discoloured, in a manner suggestive of a commencing satellite. Would it be good practice to treat this patch—which I may add has been recognised for a year, and is spreading very slowly—as a primary lesion, and destroy it by cautery or excision? That it is likely to prove infective I do not doubt, but that it is primary in the sense of being of local origin I cannot believe. Probably the lepra bacillus already exists in the boy's blood and tissues at other parts, and is undergoing its slow development, and will in due time make its appearance, irrespective of the removal or otherwise of this. In spite of this latter consideration I must admit,

[1] The bacillus is not believed to possess any independent power of movement from place to place.