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INTRODUCTION

Industrial communities that consume large quantities of bituminous coal suffer more or less from what has come to be called the "smoke nuisance," and in many of them steps have been taken by private or public agencies to abate it. In the fall of 1911 Professor Robert Kennedy Duncan, Director of the Department of Industrial Research of the University of Pittsburgh was provided by a Pittsburgh business man with a fund for making an investigation into this subject with particular reference to Pittsburgh. Prominent citizens had frequently expressed themselves to the effect that the smoke problem is the greatest single civic problem confronting Pittsburgh. The donor of the fund was actuated by the belief that a thorough investigation would reveal not only the nature, extent, and precise causes of the smoke nuisance, but also the remedies that would make its abolition possible and practicable.

At the present time the investigation is being carried on by a staff of twenty-five specialists, six of whom are giving their entire attention to this work, while the remaining nineteen have been entrusted with the preparation of special reports concerning particular phases of the subject.

The investigation as a whole may be divided roughly into two parts,—the analytical or diagnostic, and the constructive or remedial part. Under the first head may be grouped the Meteorological, the Botanical, the Chemical, the Physical, the Architectural, the Hygienic and Medical, and the Economic researches, as well as certain sections of the Engineering and of the Legal and Administrative investigations. Under the second caption may be grouped the Experimental and Educational activities of the staff, as well as the more important phases of the Engineering investigation and of the Legal Administrative investigation.

Apart from these two main divisions of the work, provision has also been made for a brief History of the Smoke Nuisance in Europe and in the United States, and for an exhaustive Bibliography of the subject surpassing in scope and thoroughness anything hitherto attempted in this line.

SMOKE AND THE WEATHER

Dr. H. H. Kimball, of the federal Weather Bureau at Washington, D. C., has been entrusted with the task of determining the effects of smoke and soot upon fogs, winds, temperature, sunlight and weather conditions generally, with particular reference to soft-coal communities like Pittsburgh. Inasmuch as similar investigations, both official and unofficial, have been made in a number of European countries, this investigation will, for purposes of comparison, be based upon similar methods wherever these methods are found to be scientifically satisfactory. The main topics covered by this section of the investigation are:—atmospheric gases; suspended particles in the atmosphere; dust layers of the atmosphere; the surface dust layer; quantity of soot in the air of cities; limits of visibility; meteorological effects of the smoke cloud; effects of smoke upon condensation; city and country fogs; effect of smoke upon fog dissipation; duration of sunshine in cities; intensity of sunshine in cities; quantity of daylight in cities; effect of smoke clouds upon city temperatures; effect of smoke clouds upon maximum and minmum temperatures; effect of smoke clouds upon the range of temperature.

Data have been obtained from the records of the Government Weather Bureaus at Pittsburgh, Harrisburg, Philadelphia and Williamsport, regarding the reduced atmospheric transparency caused by soot. Observations are being made at several stations in Pittsburgh and vicinity, and will be continued for a year, to determine the intensity of daylight, more especially, on cloudy days when the sunshine recorder furnishes no information on this subject. These observations, made by chemical and photometrical methods, in the clear country air, as well as in the smoky atmosphere of the city itself, will not only be of scientific significance in themselves, but will provide part of the basis necessary for calculating the increased cost of artificial illumination in Pittsburgh due to smoke in the atmosphere, and will be of value to the physicians of the staff in their study of eye-strain in smoky cities.

HOW IS VEGETATION AFFECTED BY SMOKE AND SOOT?

The answer to this important question involves first of all a botanical survey of the "Pittsburgh District,"

with a view of determining by observation what plants thrive in Pittsburgh and what plants can be grown here under the present difficulties. It includes, furthermore, a study of the physiologic response of plants as a whole and a determination of the reasons why plants that ought to grow here cannot be cultivated successfully.

This work, which is in charge of J. F. Clevenger, botanist of the Pennsylvania State College, will also comprise a series of laboratory experiments so arranged as to determine the effect of definite quantities of soot, of varying composition, upon the seedlings of plants which seem to be most influenced by soot, and upon the seedlings of the hardier plants.

Investigations made elsewhere have indicated that the factors in air pollution which prejudicially affect vegetation are:—the smoke clouds limiting the available sunlight; the tarry matter coating over the leaves and choking the stomata; the presence of free acids in the air, tending generally to lower the vitality of the plant; the effect of the free acids falling upon the soil and limiting the activity of the soil organisms, principally those of nitrification. The measurement of these forms of effective damage to vegetation by smoke, with particular reference to Pittsburgh, will prove of convincing interest to those who are interested in the defoliation and ruin of gardens, trees, flower-beds, and public and private pleasure-grounds.

THE CHEMISTRY OF SMOKE AND SOOT

What is the nature of soot? In view of the agitation against smoke and soot, begun in England as early as 1819, when Parliament appointed the first Special Commission on Smoke Prevention, it would seem that we ought now to know a great deal about the precise composition of soot. This, however, is not altogether true, for comparatively few scientific investigations have been made into this phase of the subject. Moreover, the analyses of soot which have been made from time to time by different experimenters show great variations in composition. When one considers the very different conditions under which coal is burnt it is obvious that the character of the soot must vary. For soot is a product of incomplete combustion, and is formed partly by the mechanical re-

moval of ash by the chimney draught, and partly by the decomposition of the volatile portion of the fuel which is formed by the process of destructive distillation. Hence there are noteworthy differences in the relative amounts of ash and tar contained in soot. Again, the character of the soot varies with the distance, from the grate, at which it has been deposited as well as the temperature of the furnace, amount of air, method of firing, etc.

For these reasons, the different varieties of soot produced in Pittsburgh are being subjected to careful chemical analysis, under the direction of Dr. Raymond C. Benner.

THE PHYSICAL PROBLEMS OF SMOKE

It is important to know not only how much carbon, tar, oil, water, etc., exists in collection of smoke, but also the shape and size of the smoke particles; how the properties of the different particles vary; what constituents are in the solid, the liquid, or the occluded gaseous conditions; whether the smoke particles are electrically charged; what effect temperatures have upon the nature of the smoke particles; and how these particles form nuclei for the condensation of fog and rain.

The combustion of coal consists of the union of the combustible portions of the coal with the oxygen of the air. The smoke particles are born, so to speak, wherever incomplete combustion takes place. These smoke particles are usually electrified and subjected to intense forces that result in their aggregation into larger particles. There are three main periods in the life of the products of combustion. The formative and adolescent period of the smoke particles is followed by a comparatively quiescent period of maturity, during which they float in the air or unite with mist particles or rain drops. Finally, in their next stage, they are deposited on our clothes, on buildings, on plants and animals, or on the ground.

The nature of these processes is being investigated by Dr. W. W. Strong, who will determine, by the use of the ultra-microscope, and by the photometric and electrical methods, the physical properties of the smoke in furnaces and flues, in the air, and on the ground. The facts thus ascertained will be of value to those in charge of other phases of the investigation. They will, more-

over, furnish a basis for certain preventive devices to which reference will be made later on.

DETERIORATION OF BUILDINGS AND BUILDING MATERIALS.

The acids contained in soot attack mortar, masonry, wood-work, metal-work and building materials generally. In many European cities it has been specifically charged that soot causes the rapid disintegration of statuary and public monuments. The damage done to inside decorations appears to be no less important than the effect upon the exterior of buildings.

The numerous and varied aspects of this branch of the inquiry are being studied by a group of five architects and experts in building materials,—Messrs. E. B. Lee, Richard Hooker, C. T. Ingham, Richard Kiehnel and Carlton Strong. With particular reference to Pittsburgh, these gentlemen are engaged in the preparation of reports on the following topics: The effect of smoke and soot on sandstone, limestone, marble, terra cotta and other building materials; the effect on outside and inside painting, on wall paper and on interior decorations; the effect on sheet metals, copper, galvanized iron, etc.; modifications in electrical work and in the distribution and maintenance of light because of smoky and sooty atmospheric conditions; the effect on cleaning and on the costs of maintenance of buildings; skylights, their use and arrangement as affected by smoke and soot; the influence of the above factors on architectural design in exterior and interior work.

This part of the inquiry involves numerous experimental features. Various kinds of stone and building materials have been exposed to smoky atmosphere, after being examined and photographed. After a year's exposure, they will be again examined and photographed to discover the amount of soot and dirt taken up by the different materials, and the effect of this soot and dirt upon them. The samples will then be cleaned by various recognized methods, for the purpose of revealing the cost, difficulties, and effects of different cleaning methods.

An expert in paints, Mr. Karl K. Stevens, is engaged in gathering comparative data regarding the effect of smoke and soot upon different colors and compositions of

paints, and regarding the durability of paints in Pittsburgh and certain other cities having a less smoky atmosphere.

A number of different metals, including copper, galvanized iron, plain iron, aluminum, brass, lead and tin plate, have been exposed to the weather in different parts of Pittsburgh in order to determine the corrosion due to the acid gases occluded in the soot.

SMOKE AND DISEASE

The effect of smoke and soot on human morbidity and mortality has perhaps received more attention than any other phase of the subject. To determine as accurately as may be possible, and with especial reference to Pittsburgh, the influences that these kinds of air-pollution actually have upon certain forms of disease and upon the death-rate of the population, the Department has secured the co-operation of the following Pittsburgh physicians: Oskar Klotz, W. L. Holman, S. R. Haythorn, E. W. Day, W. W. Blair, B. A. Cohoe, W. C. White and R. T. Miller.

No matter how much one may be convinced of the deleterious effects on health produced by breathing a smoke-laden atmosphere, ordinary medical statistics afford little definite information. Inasmuch, however, as many cities have for years collected and published statistics of mortality, a careful analysis of these figures will be made, in the hope that they may throw some light upon the relationship, first, between the death-rate and air-pollution, and, secondly, between air-pollution and smoke. Although the testimony of specialists is somewhat divergent on this subject, it is noteworthy that an over-whelming majority of investigators both in this country and abroad have reached the conclusion that smoke and soot are injurious to the respiratory organs, injurious to the eyes, and responsible for a lowering of human vitality and an increase in the death-rate.

The available information will be interpreted from two points of view, Dr. C. W. White giving his attention to the effect of smoky and sooty air on tuberculosis and its treatment, and Dr. W. A. Cohoe devoting himself particularly to the effect on acute respiratory diseases, including pneumonia.

Attention has been called in this connection to the practice of house-keepers in having the windows shut for fear of the soot that floats in when they are open; and it has been asserted that this also contributes "to the mentally and physically depressing effect of the pall that shuts out the life-giving and germ-destroying air and sunshine." Indeed, English official investigators have declared it "more than proable that living in a foul atmosphere which diminishes vitality increases the desire for stimulants, induces drunkenness and its concomitants of brutality, immorality and crime."

Whether such statements are scientifically warranted as regards Pittsburgh, and, if so, to what extent these consequences of smoke and soot are noticeable here,—are questions which physicians of the staff have undertaken to answer.

Particular attention is being given to diseases of the respiratory organs. Post-mortem examinations have been made and will continue to be made by Dr. Klotz for the purpose of determining the amount and distribution of soot deposited in the organs of Pittsburgh residents engaged in different occupations.

Dr. Holman and Dr. Klotz are making a bacteriological survey of the air of Pittsburgh. Dr. Haythorn is engaged in the work of determining the manner in which carbon is deposited in the lungs and the nature of the resultant lesions.

The effect of a smoky atmosphere upon eye-strain, diseases of the eye, and allied physical disorders, is being investigated by Dr. W. W. Blair. Data regarding the influence of fogs, poor light, and artificial light, upon the work and conduct of school children, are being collected through the courtesy of the Superintendent of Pittsburgh Schools, Mr. S. L. Heeter.

Are residents of smoky cities predisposed to diseases of the nose and throat, or may smoke and soot be regarded as causing those ailments? What effect has smoke and soot on the time and difficulties involved in effecting a cure? These are questions to which a scientific answer is being sought by Dr. E. W. Day.

It is recognized that successful surgery depends to a great extent upon operative technique and cleanliness. How either of these is affected by a smoky atmosphere,—if at all affected,—will be investigated by Dr. R. T. Miller.

By means of experimentation upon animals a study will be made of the extent to which living in a smoky atmosphere predisposes to infection by consumption and pneumonia. It is also planned to determine experimentally the effect to which persons living in a smoky air are predisposed to conjunctivitis.

A psychiatrist will investigate the relations between smoke-induced fogs and certain forms of mental depression.

WHAT THE SMOKE NUISANCE COSTS

Whether it is more profitable for a manufacturing establishment to produce smoke or to so modify its installation as to cease producing smoke, may, in a few instances at least, be a debatable question. There can be no doubt, however, that the widespread practice of filling the air with soft-coal smoke results in increasing the cost of operating certain business enterprises and in damaging certain kinds of merchandise. Nor is it improbable that certain branches of manufacturing and commerce are carried on in Pittsburgh with greater difficulties than would be the case if the city were smoke-free.

In the case of certain manufacturing establishments, the necessity for clean air and abundant light involve extra expenditure for purifying the air, for ventilating devices, and for artificial illumination. In the case of retail and wholesale stores, the cost of outside and inside cleaning, the losses through soiled merchandise, the frequent need of re-painting and re-decorating, the labor and precautions required to keep stores and goods clean,—all these are noticeably increased by the soot-laden atmosphere of smoky cities. Certain trades such as textile manufactures, millinery, stationery and art goods, vegetable gardening,—can be carried on in such cities at greater cost and inconvenience than elsewhere.

Already considerable evidence has been accumulated to show that the cost of operating office buildings, hospitals, libraries, schools and public buildings generally in Pittsburgh, is increased in noteworthy and fairly measurable proportions by the smoky and sooty atmosphere of the city. It will be more difficult, however, to determine the economic damage done to the clothing of the people and to their homes and home furnishings. But these

features of the problem affect every man, woman and child in the community, in the guise of larger laundry bills and bills for dry-cleaning clothes, in the form of greater wear and tear on clothing, curtains, carpets, wall-paper, rugs, and other household goods, and in the additional labor required to maintain even a moderate standard of domestic cleanliness.

To determine, as nearly as may be possible, the economic significance of all of the above items, expressed in dollars and cents, and of elements that make the smoke nuisance an economic burden to Pittsburgh, is the task that has been assigned to Dr. C. W. A. Veditz and Mr. J. J. O'Conner, Jr.

They will also seek to interpret in economic terms the loss of fuel and of power involved in the present methods of power-production used in Pittsburgh, as revealed by the inquiries made by the engineers of the staff into the mechanical conditions of smoke-production. The economic inquiry will necessarily be comparative throughout its entire scope. For it will be necessary to obtain data not only from Pittsburgh but also from other cities that have less or no smoke, in order that a more correct appraisal may be made of the several items under consideration.

It has been estimated that the smoke nuisance costs Cleveland $6,000,000, Cincinnati $8,000,000, and Chicago $50,000,000 per annum. Herbert W. Wilson, of the United States Geological Survey is authority for the statement that the country as a whole suffers a loss of over $500,000,000 each year, in damage done to merchandise, defacement of buildings, tarnishing of metals, injury to human life and to plant life, the greatly increased labor and cost of housekeeping, and the losses of manufacturers due to imperfect combustion of coal. The economists of the Pittsburgh investigating staff are endeavoring, by means of more accurate methods than have been employed elsewhere, to determine the total loss suffered by Pittsburgh on this score.

WHO MAKES THE SMOKE?

The relation of mechanical engineering to the problem of smoke abatement is in some respects the most important branch of the entire inquiry, for the question

whether smoke is industrially necessary or not is after all a question of mechanical engineering. Therefore, the plans for this branch of the investigation are somewhat elaborate, both as regards the study of causes and the study of remedies. The plans for this part of the work now being carried out by Mr. A. B. Bellows and Mr. O. R. McBride, include the following main lines of study: A geographical and topographical survey of Pittsburgh and the "Pittsburgh District"; an account of its principal industries,—their nature and relative importance; a classification of the fuels employed, together with a statement of the amounts used and the cost of each; an inventory of the industrial establishments producing smoke; a study of the nature and extent of locomotive and steamboat smoke; the determination in detail of the causes of smoke production by the principal offenders; a careful inquiry into the installation, operation, fuel economy and power production of plants that have ceased to produce smoke or that have succeeded in the noteworthy abatement of smoke-production; and a brief treatise on the subject of combustion and a descriptive discussion of smokeless fuels and of various fuel-burning devices now in use, with particular reference to smokeless combustion in boiler, metallurgical, and other furnaces using bituminous coal.

In ascertaining the sources of smoke, the engineering inquiry distinguishes (a) the business section of the city, (b) manufacturing plants, (c) railroads, (d) river steamboats, (e) residences, and (f) miscellaneous plants,—such as contractors' hoisting engines and steam rollers, which make smoke in the streets and discharge it at low levels near doors and windows.

The plants investigated will be graded as follows:—(a) Those producing no smoke; (b) those which produce smoke in quantities within the limits permitted by the present city smoke ordinance, or which can be brought within the city ordinance by proper operation; and (c) those producing smoke, but which cannot be brought within the city ordinance without alterations or new installations. In the case of the smoke-producing plants, a study is being made of the precise causes in each case, of the changes in equipment or operation that will remedy the evil, and of the cost of these changes.

In connection with the engineering investigation (and of such other phases of the whole inquiry as admit of it) photographs and charts are being made to give definiteness and convincing character to the observations made and to the conclusions reached.

LAWS AND ORDINANCES CONCERNING SMOKE

The law took cognizance of the smoke nuisance in England as early as 1273, when the use of coal was prohibited in London as prejudicial to the public health. Since that time numberless proclamations, laws and ordinances have given attention to the smoke nuisance in one form or another. Most of the larger cities of all industrial nations now have ordinances dealing with the subject. These ordinances, however, vary greatly in purpose, scope, character, and stringency. They are, moreover, enforced with degrees of vigor that range all the way from zero to comparative efficiency.

The legal specialist of the staff, Joseph A. Beck, Esq., is making a collection of the laws of the several states of the United States, of Great Britain, Germany and France, relating to this subject, and of the ordinances of cities of the United States. These laws and ordinances will be analyzed, classified, and criticized, and an investigation made of the interpretation placed upon them by the courts, and of the constitutional questions involved. A study also will be made of the rights of individuals under the common law (irrespective of the statute law) to secure the abatement of a smoke nuisance or to recover damages for the injury suffered. A systematic inquiry into means and methods of enforcing the laws and ordinances concerning smoke, into the rights, duties and activities of smoke inspectors, and into the nature, frequency and effects of the penalties imposed for violation of the smoke ordinances, is already well under way.

GENERAL EXPERIMENTAL WORK

It has already been pointed out that a number of Pittsburgh establishments burning soft coal are operated almost smokelessly, and that a considerable number of the others could be operated without producing more smoke than the law permits, if proper care were always

taken by trained stokers. To aid in bringing this about, experiments have been made for the purpose of devising a cheap automatic apparatus,—a "smoke monitor and recorder,"—that will give smoke signal, such as ringing a bell, whenever the furnace is making more smoke than the laws allows, and that will keep a record of the frequency and duration of the transgression.

With the aid of such a device,—several of which have already been constructed and found satisfactory,—it will be possible for the superintendent of a plant to know which of his furnaces and which of his employees is responsible for illegal smoke-production, and which of his men have exercised sufficient care and intelligence to produce no objectionable smoke. Such an appliance, moreover, might conceivably be made a valuable and trustworthy ally of the city smoke inspectors, who can cover only a limited area and who can make hardly any "smoke observation" at night or under adverse weather conditions.

Experimental work is also being done for the purpose of constructing a simple device for recording the density of smoke,—a "smoke meter" that will substitute definiteness for the present inaccurate terms used to distinguish various grades or degrees of smoke. Furthermore, the experimental staff, consisting of Dr. R. C. Benner, Dr. W. W. Strong and Mr. P. F. Shuey, is working upon the problem of precipitating smoke electrically or otherwise before it leaves the chimney or flues.

SMOKE MEANS WASTE AND INEFFICIENCY

In the scientific literature on combustion there is a surprising unanimity of opinion to the effect that smoke is unnecessary and positively wasteful in an overwhelming majority of establishments burning soft coal. It is a consequent and visible proof of imperfect combusiton of fuel, and therefore evidence of a waste of part of the fuel. This waste, however, is not nearly so important to the owner of the establishment as the losses of heat and of power that are its inevitable concomitants.

Few manufacturers who have taken the trouble carefully to investigate the subject, and certainly no expert mechanical engineers, would deny this. But it is quite a different matter to persuade the owners of smoke-producing plants to adopt an effective system of burning fuel

without smoke, unless it offers a substantial monetary saving more than sufficient to cover the initial outlay. Nevertheless, reputable mechanical engineers, both in this country and abroad, now contend that in at least a large number of instances the introduction of smoke-consuming devices and more scientific methods of stoking would result in a saving of fuel and a more economical production of heat and power, sufficient to equal in a relatively short time the entire original cost of these changes. Abundant and convincing practical demonstration of this contention is furnished by the achievements of the Hamburg Manufacturers' Smoke Abatement Society and the London Coal Smoke Abatement Society.

The abolition of the smoke nuisance, therefore,—unlike many other social nuisances against which an outcry has been made,—would result in direct and immediate gain both to the public at large and to those chiefly responsible for the nuisance. For this reason, it may be said that the mechanical engineers of the staff are engaged in a most important constructive task. This task will involve the careful and detailed study of Pittsburgh plants to which reference has already been made. It will also involve a study of fuel economy and fuel-efficiency in these plants, as a basis for an appeal to business instinct as well as to civic pride.

Equipped with a thorough knowledge of the numerous devices for smoke-prevention that are now on the market, and with an understanding of the needs and problems of each plant, the engineers of the staff should be able to do the work of veritable "smoke doctors," pointing out the precise remedy suited to each individual case of smoke-production.

THE EDUCATION OF THE PUBLIC

Experience has shown that even manufacturers, though presumably on the alert for every improvement, however slight, in the economical operation of their plants, are creatures of habit and tradition. If their plants have already produced smoke in the past, it is in some instances necessary to bring pressure to bear before the change is apt to result in more economical as well as smokeless operation.

In these cases an active and intelligent public opinion, that subjects the offending parties to public opprobrium, may help to accomplish the desired end. At all events, the formation of an enlightened public opinion upon the smoke problem is one of the principal objects of the whole investigation. Should it be found, for example, that certain diseases are less frequent and more easily cured in a smoky atmosphere than in a clear atmosphere, that fact will be made public property. Again, should it be discovered after careful inquiry that no way has yet been devised to prevent the emission of black smoke from certain classes of metallurgical furnaces, save at an expenditure that would greatly increase the costs of production, this fact will likewise be made known. But if, on the other hand, both of these supposed conclusions are unfounded, it will be none the less necessary to publish the facts. Too often a public demand for certain reforms has been based upon imperfect knowledge or upon a mistaken notion of the factors involved.

Should the work and conclusions of the staff be known to the investigators themselves, they will prove of little practical avail. It has therefore been decided (a) to make a systematic effort to enlighten civic and business organizations on this subject, and to arouse them to combined action; (b)* to organize a staff of lecturers prepared to address these organizations in Pittsburgh and elsewhere; (c) to secure the co-operation of the public press; and (d) to publish in book form the results of the entire inquiry.

THE QUESTION OF LEGAL REGULATION

Should the intelligent self-interest of manufacturers, combined with an insistent public demand for smoke abatement, fail to result in an adequate improvement in conditions, then the public authorities have not only the right but a positive duty to interfere and to enforce more drastic measures. But the legal appeal should be the last appeal. If, however, such an appeal is made it should be made effectively. The mere enactment of a smoke ordinance is not sufficient. Such an ordinance must be both enforceable and enforced. Based upon the information obtained from other cities and countries, the legal expert of the

*A leaflet concerning these lectures will be sent on request.

staff will draw up a model ordinance. Under such an ordinance, provision should be made for an adequate corps of qualified inspectors. It should provide, moreover, for a system of co-operation between private and public agencies of enforcement, with proper regard for the industrial interests and the industrial development of the city of Pittsburgh.

Mellon Institute of Industrial Research and School of Specific Industries

Smoke Investigation Bulletin No. 9

Papers on the Influence of Smoke on Health

Edited by
Oskar Klotz and Wm. Charles White

University of Pittsburgh
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Introduction

The present bulletin contains the papers representing the work done by the physicians and laboratory investigators on the Staff of the Smoke Investigation.

Much difficulty was experienced by the physicians in arriving at definite conclusions, because the present state of our knowledge does not admit of satisfying and positive pronouncements of the relation of smoke to diseases of the eye, ear, nose and throat and other diseases requiring surgical intervention. The psycological aspects of the problem have been discussed by J. E. W. Wallin, Ph. D. in Bulletin No. 3 of this series.

Doctor Cohoe has prepared his resume of the literature especially for this bulletin. The papers of Doctors Klotz, Holman, Haythorn and White have all been published or read elsewhere in scientific circles.

Doctor Cohoe's paper gives a review of the history of the thoughts and work of men leading up to our present knowledge and attempts no decision of the vexed problems.

The work undertaken by Doctor Klotz deals with the gross pathological changes which arise from the excessive inhalation of a carbon-laden air. Whereas the presence of small quantities of smoke has no harmful effect upon the tissues, it is shown that the continuous accumulation of carbon may act quite differently. This feature alone in our problem deserves further attention.

The work of Doctors Haythorn and Holman are both very valuable contributions to this subject, containing the results of original research that aid greatly our search after the truth of the relation of smoke to health. Doctor Haythorn's paper brings out some suggestive points in indicating the histological changes induced by smoke and their relation to pneumonia.

It will be seen in reading all of these papers that practically all investigators whose opinions are based on

grounds other than theory, are agreed that smoke has a tremendous influence in increasing the incident severity and mortality of acute diseases of the air passages. It would appear that this increased susceptibility is, in part, the result of the lowering of our natural body resistance. In simple terms, the smokier the atmosphere, the more the colds and bronchitis, and the more money paid to doctors.

On the other hand, the relation of smoke to tuberculosis is one of greatly divided opinion and the burden of proof is that if smoke has any influence at all, it is not a harmful one. The work of Doctor Klotz and of Doctor Haythorn offers some explanation of this probability. If this be the truth and tuberculosis is not influenced by smoky atmosphere, it is time to stop the utterance of this popular fallacy which can do naught other than harm in sacrificing the confidence of the public in those who should guide them.

We feel very sure that a careful perusal of this bulletin will fully repay every reader of it.

WM. CHARLES WHITE.

The Relation of Atmospheric Smoke and Health

B. A. Cohoe, AB., MB.
Pittsburgh, Pa.

INTRODUCTION.

In the present paper an attempt has been made to cull from scientific literature the evidence, pro and con, which has been adduced by various scientists whose opinions would seem to merit consideration, concerning the role played by a smoke-laden atmosphere upon the bodily health of the community.

Thanks to the triumphs of the modern science of sanitation, a successful assault has been waged against such water-borne diseases as typhoid fever and cholera by a close surveillance of the water supply and sewage disposal within our cities, while, more recently, the efforts of the Public Health Bureau have rendered it possible for our citizens to obtain pure food stuffs, free from adulteration. Meanwhile, strangely enough, the problem of pure air has received but scanty consideration.

This omission appears the more astounding when regarded in the light of a fact stated by Cohen that every man and woman breathes about two thousand gallons of air in twenty-four hours, or about thirty-four pounds in weight, as contrasted with a daily intake of five and one-half pounds of liquid and solid foods. Or, in other words, the weight of the air inhaled daily is more than six times the weight of the daily consumption of food.

Fortunately, within recent years, largely owing to the organization of Smoke Abatement Leagues, an agitation has been begun advocating an examination of the air in a manner comparable to the careful scrutiny now required for our water and food supplies and of the sewage disposal.

In a survey of the various factors which tend to contaminate the atmosphere of cities, smoke is conceded by all to be the greatest source of pollution. The Smoke Abatement Leagues, in their efforts to purge the air of this menace, have encountered numerous obstacles, notably an apathetic public and a certain antagonism on the part of manufacturers against any attempt to curtail the amount of industrial smoke. The lethargy of the public in regard to the smoke evil doubtless has arisen from the circumstance that, until recently, the opinion has prevailed in the minds of the laity, and indeed of physicians as well, that a smoky atmosphere is not only not injurious but at times even beneficial to the public health. This supposition gained favor from an observation, largely erroneous, that coal miners are not prone to contract tuberculosis.

A belief in the antiseptic qualities of smoke has been entertained by many physicians for several centuries. It is a matter of historical interest that during the Plague Year in London fires were kept burning in the hope of suppressing the epidemic for "it was alleged that the sulphurous and nitrous particles that are often found to be in the coal, with the bituminous substance which burns, are all assisting to clear and purge the air and render it wholesome and safe to breathe in."

Quite apart from any mooted question as to the ultimate effect on health, the citizens of Pittsburgh have, for many decades, been keenly alive to the bodily discomfort resulting from the omnipresent smoke pall in the community. As early as 1804, General Neville, the then Burgess of Pittsburgh, is quoted (O'Connor) as having stated with reference to the smoke nuisance "that not only the comfort, health, and in some measure, the consequence of the place, the peace and harmony of the inhabitants, depend upon speedy measures being adopted to remedy the nuisance." Nor, indeed, were visitors, then as now, slow to observe the baneful influence of the smoke upon the inhabitants for, in 1818, Eswick Evans wrote that "'owing to the exclusive use of coal here, both by the man-

ufacturers and by private families, the whole town presents a smoky appearance. Even the complexion of the people is affected by this cause." Major Forman noted in 1797 that "the coal smoke is such as to affect the skin of the inhabitants." Another writer of note who corroborated these observations was Henry Bradshaw Fearon, a London surgeon, who, in 1817, remarked of Pittsburgh that "the smoke is extreme, giving to the town and its inhabitants a very sombre aspect."

Such casual observations, extending over a period of more than a century, only serve to emphasize a deplorable degree of apathy on the part of the citizens in tolerating a nuisance whose injurious effects have been so long and so clearly proclaimed. Yet Pittsburgh fares no more ill than many of the large industrial centers of the world with respect to the smoke evil.

In the present resume of the literature, the aim has been to present an analysis of the vital statistics, special investigations, and experimentations bearing upon the problem of the relation of smoke and health, and no attempt has been made to deduce other than a few generalizations concerning local conditions in Pittsburgh. A special study of the local conditions and vital statistics from a viewpoint of the status of smoke as a possible factor in increasing the local death rate has been reserved for another bulletin of the present series.

I.

EVIDENCE FAVORABLE TOWARDS SMOKE.

In a general survey of the literature concerning the role of smoke in the causation of disease, it is not surprising that an expression of doubt concerning the harmfulness of smoke in the community should be encountered in certain quarters, nor, indeed, that certain scientists should entertain a sentiment favorable towards smoke. While the smoky atmosphere of an industrial center continues to be the index of the prosperity of the town, the community is likely to overlook the discomfort and danger of the same and foster any attempt to defend its antiseptic qualities. In an impartial weighing of the evidence, pro and con, regarding smoke such favorable opinions, coming from observers whose authority cannot be overlooked, merit consideration.

The popular idea that smoke does not act injuriously on health, but only uncomfortably, has served to retard in no small measure the work of smoke abatement. Our standard works on hygiene take little or no cognizance of the harmful effects of smoke. Many authors, who do not regard smoke as injurious, attempt to establish their claim by a citation of the low death rates of certain industrial towns. At the Manchester meeting of the Smoke Abatement League of Great Britain, 1911, a delegate made the assertion that his town (Coatbridge, a smoky industrial town) was one of the healthiest of towns, admitting, however, that the smoke was at times very annoying. The reason for his defense was obvious in his statement that "furnaces did make smoke and to prohibit the making of smoke would prohibit the making of puddled iron." A writer in the Revue Industrielle (November, 1899) voices the same sentiment in stating: "In spite of the abundance of smoke, people are no worse off in Leeds than elsewhere.

Facades of houses are soiled but the inhabitants do not suffer. Are we going to learn one of these days that smoke, thanks to its antiseptic properties, contributes to making the atmosphere healthful?"

The medical profession, indeed, has at times given utterances to similar opinions. At a conference held in the Franklin Institute, Philadelphia, a few years ago a physician is quoted as saying: "I do not mean to intimate that the smoke nuisance injures health in any ordinary sense; on the contrary, I am inclined to think that the essence of this, which is always a minute amount of free carbon in the air, is rather healthful than otherwise." Similarly, following a discussion of the smoke evil at a meeting of the Philadelphia County Medical Society, 1906, it was recorded, "that the nose, throat and eyes are directly injured by smoke was admitted beyond a doubt, but whether the presence of soot in the human lungs is an indifferent matter, or an injury, was left undecided. There is little evidence to show that the presence of smoke in the air increases the morbidity or the mortality of the community."

In a study of anthracosis as a factor in the causation of lung diseases, Trotter has commented upon the rarity of pulmonary tuberculosis among miners, and from his observations he was lead to regard coal dust as productive of little, if any, harm to the organism. His conclusions would seem to show that coal dust renders miners more or less immune to tuberculosis.

1. Coal dust may remain imbedded under the skin for years without producing any irritation whatever.

2. The death returns from phthisis in a colliery district show a greater proportion of deaths among females than males.

3. Colliery surgeons almost invariably state that phthisis is not so common in mining as in other districts.

4. In the majority of phthisis cases which do occur, there is a strong family history on one or both sides.

So popular has been this fancy that coal dust acts beneficially in pulmonary tuberculosis that it has been for years a common custom for persons affected with this disease to resort to coal mines, or to build fires and inhale the smoke, hoping thereby to affect a cure, on the quasi-scientific supposition that the inhaled carbon promoted healing. As will be shown later, more careful recent investigation has demonstrated the fallacy of this supposition and has placed a revised interpretation on the apparent low death rate of miners from tuberculosis.

II.

EVIDENCE OF A DOUBTFUL NATURE.

In a second grouping can be assembled the opinions of a number of scientists who, while they would be constrained to regard smoke as a cause of ill health, yet in a spirit of scientific conservatism consider the case as not proven. The study of the problem of the effect of smoke upon health presents many complicating factors, which render judgment difficult.

Among these, Mehl states that "no one has yet shown that smoke and fog are injurious to health," but the same writer proceeds to demonstrate by means of statistics that the increased death rate from respiratory diseases, in large industrial centers, must be referred to the influence of smoke and dust. Cohen, one of the most active workers in the crusade against industrial smoke, while realizing that it is quite true that pulmonary disease is much higher in towns than in the country, concedes that "we cannot single out smoke as the cause, because the problem is complicated by so many other factors."

Another writer, Glinzer, while regarding smoke as truly injurious to health in a degree not as yet determined, appreciates its twofold qualities. Smoke, he believes, possesses excellent germicidal and disinfecting properties. "Obnoxious vapors and other harmful products carried by the air are absorbed and retained by the material elements of smoke, and are finally carried away harmlessly by the rain, so that smoke, on the contrary, can be stamped as a purifier of the atmosphere and a benefactor of the human race. In my opinion, this is doubtless true; it is only a question of which influence is the stronger. I believe we can safely depend on our instinct, and this tells us decidedly that smoke does not agree with us."

Another careful investigator who regards the proof as incomplete is Rubner. He believes that while there can be scarcely any doubt of the possibility, perhaps even the probability, that the smoke content of the air is an important element of injury to health; a matter of the coincidence of bad air and disease is another matter. "Such a proof can scarcely be attained even by the standard of very exact etiological investigations. If there is an entire lack of knowledge as to how the pollution of the air by smoke is distributed in the country in general, and as to whether the regions with impure air coincide to some extent with those of more frequent lung diseases, there is still further lack of proof as to whether the condition of the air is alone responsible for the injury, or whether the remaining within doors does not also contribute to it. The injuries resulting from a pollution of the air might also be traced to climatic influences."

III.

EVIDENCE OF A POSITIVE NATURE.

Passing over the foregoing evidence, which is more or less equivocal in kind, we come to the evidence of a more positive nature, which has been gleaned from the many monographs appearing in the literature dealing with the smoke problem, in which the authors have attempted to establish proof concerning the pernicious influence of smoke and its constituents upon the individual and public health. In this connection, the significant fact is revealed that the more exhaustive the investigations undertaken by the various scientists, from the viewpoint of sanitation, vital statistics, and animal experimentation, by so much the more do the conclusions evolved attest the deleterious effect of smoke on health. Such observations have been made from many angles—the effect of smoke as a whole, and the effect of its various ingredients, as well as what may be regarded as the direct primary effect together with the more remote secondary effects of a smoke-laden atmosphere. Dealing first, therefore, with the opinions promulgated as to the general effect of smoke, or the influence of smoke as a composite entity upon the health of the community, we shall pass to the evidence obtained as to the effect of the various constituents of smoke, such as carbon, soot, and gaseous elements upon the human organism, and finally, to the indirect effects of smoke in impairing the health of a community.

(A) The Effects of Smoke as a Whole.

The general effects of a smoky atmosphere upon the health of inhabitants have been broadly summarized by Wainwright, who states that smoke interferes with the welfare of a community—

1. By conducing to the formation of fog and rain.

2. By shutting out sunlight and depriving us of certain qualities of light of great importance in regard to changes in organic matter.
3. By depositing soot and rendering houses or their contents dirty.

Moreover, with respect to the individual he believes the acid content of smoke and the irritating particles suspended in it are harmful to the tissues of the nose, throat and eyes, and especially the lungs and air passages whether these be in a healthy or other condition. He further indicts smoke as a menace indirectly to the sick of a community, averring that "it aggravates to the discomfort of those suffering from all forms of heart trouble; increases the distress of those who have nervous complaints; lowers the tone of general health; is a peril to the aged; diminishes buoyancy of spirit as well as reducing still further an already lowered resistance to disease."

Valuable evidence has been presented by a number of Medical Health Officers of smoky industrial cities concerning prevailing health conditions. This is especially true of the city of Manchester which may well be selected as a type of smoky towns. Concerning local conditions in the city, Dr. Niven, M.H.O., has written: "Manchester is still conspicuous for its high mortality, and it is especially conspicuous for its excessive death rate from phthisis, pneumonia, bronchitis, and heart disease. All forms of septic disease are unduly prevalent. The windows of houses and factories are closed on account of the dirt which enters by the open window. Less attention is paid to cleanliness than is needful for health. Fresh air is needed not only in the treatment of consumption, but is equally necessary for the raising of healthy children. * * * There has been a great advance in the physical well-being of the population, but the death rate is still among the highest prevailing in great towns. Manchester, like other towns, exhibits an immense improvement as regards some diseases, more especially smallpox, enteric fever, and scarlet fever. * * * The mouth breather is at a much greater disadvantage in Manchester

and the large towns than he is in the country and it is important that adenoids should be attended to."

The report of Dr. Tatham, M.H.O., in 1890, speaks even yet more strongly of the local conditions in Manchester. This writer states that the working life of the people in central Manchester is curtailed by ten years. To quote from his report: "Our people lose 30% of their lives. * * * The acids of smoke and carbon particles operate upon the lungs for years before they finally destroy them."

If the criterion of physical fitness for army service may be taken as an index of the health of the citizens of a smoky city, the statistics for Manchester, according to Horsfall, reveal an appalling condition. This author writes: "In Manchester in 1899 of 11,000 men who wanted to enlist only 9% were found to be physically fit for the regiments of the line, and at the present moment the Navy could only accept 14% of the boys belonging to all grades of society who wanted to join. There are no figures among races of the world comparable with these in their revelation of physical deterioration."

Among the numerous investigations which have been undertaken to determine the direct effect of smoke as a source of ill health, it is not surprising to find that the burden of proof has been sought in the relation of smoke to diseases of the respiratory organs, since the lung tissue is necessarily first invaded by the smoke content of the air inhaled. Accordingly, the majority of observers have based their studies upon the incidence of lung disease in smoky industrial cities. While vital statistics may not furnish, perhaps, the ideal criterion of the health of a community, they afford without doubt the most readily available standard of comparison.

In reference to the irritating effect upon the lung tissue of the constant breathing of a smoky atmosphere, Reed writes: "The slight morning cough with the equally slight expectoration of black mucus is an experience familiar to denizens of a smoky town, but an experience which, to the medical mind, suggests a persistent although

slight irritation of the upper air passages that are thus made hospital avenues for tuberculous infection."

Supfle, another observer, corroborates the opinion of the former writer in saying: "Climate has much to do with the effects of smoke on health. Southern cities, with warm climate, do not suffer so much as those in the north. Cities lying in the valleys suffer most. The acids given off in fumes provoke a hypersecretion of the mucous glands of the trachea and bronchi. In time, one accommodates oneself to the new conditions and one apparently feels well. And herein lies the great danger, as one ceases to cough and outward symptoms no longer appear, the injurious effects remain."

According to statistics cited by Wainwright, the death rate from diseases of the respiratory organs in Manchester was in 1874, 7.7, and in 1893, 23.2 per 1,000 of deaths from all causes; and in Westmoreland, England, the death rate from the same diseases was but 2.29 in 1888, and 13.7 in 1893. These increased death rates he charges to an increased consumption of coal and consequent smoke.

Russell has utilized the following comparative table to show the extreme frequency of death due to lung diseases occurring in and about the smoky atmosphere of Glasgow in the year 1880:

	Contagious Diseases	Lung Diseases	Other Diseases	Total
Rural Districts	289	354	996	1,639
City of Glasgow	773	1,024	1,232	3,029
Thinly populated part of Glasgow (36 persons per acre)	450	600	870	1,920
Densely populated part of Glasgow (512 persons per acre)	1,020	1,860	1,600	4,480

Similarly, a table compiled for Manchester reveals a strikingly high incidence of deaths from respiratory diseases. The death rate in different parts of Manchester

per 100,000 for the year ending the third quarter of 1891 was as follows:

	Contagious Diseases	Lung Diseases	Other Diseases	Total
Thinly populated part	241	534	954	1,729
Densely populated part	510	1,544	1,798	3,752

Concerning southeast Lancashire, a smoky industrial center, Dr. Brown states that the death rate in that district from respiratory diseases is very considerably higher than in the southern and western parts of the country.

Far and away the most comprehensive study of the direct effect of smoke upon the respiratory organs has been made by Dr. Louis Ascher of Konigsberg. He has published several monographs dealing with the subject both from the standpoint of vital statistics and animal experimentation. His conclusions, while not universally accepted, have been drawn from the most careful scientific investigation of the problem of smoke and health yet presented. His work merits more than a passing survey.

In pursuing his observations, Ascher utilized the statistics of the Imperial Health Bureau of the German Empire. Respiratory diseases he separated into two main groups, the tuberculous (designated T) and the non-tuberculous (NT). In some instances the German statistics were compared with those of other countries, as England and America. A study of these statistics, dating back to 1875, lead him to conclude that since 1875 there has been an increase in acute inflammatory diseases of the respiratory organs. The possible bearing of a diminished power of resistance, or of climatic or infectious causes, as contributing to this increase was considered by the author, but each of these in turn were found not to be a factor in the increase of acute lung diseases. Was this increase confined to Prussia during this period? An examination of the mortality tables gave a negative answer and revealed a similar increase in the whole of Germany, as well as in England and America. In the latter countries he found

an increase in the death rate from pneumonia between 1890-1900 of 186.9 to 192 of every 100,000 of the population. He made a study of conditions in agricultural districts where he found a lowering of the death rate from tuberculosis and a rise in acute lung diseases. His conclusion from his earlier statistical studies was as follows: "The increase in the mortality of acute lung diseases must be the result of some harmful factor which, it is true, is found in agricultural communities, but with a much higher increase in industrial centers. This factor is not limited to the places of industrial work but is also found in the homes, as proven by the mortality tables for infants and old people. The cause of this increase can only be the smoke of the coal fires."

Ascher was fully cognizant of the opposition that such conclusions might engender on the part of the critics. The lethargy of the public in regard to the smoke problem has been due, he believes, to the fact that smoke has not been considered harmful. The reasons why smoke has not been regarded as a menace he summarizes as follows: (1) Coal smoke does not act as an irritant, causing discomfort or inflammation on the body tissue with which it comes in contact, as does ordinary dust. And since there is relatively little coal dust in the air, it is inconceivable that it should have much pathological effect. (2) The researches of Arnold tended to show very slight harmful effects from coal dust in comparison with other forms of dust. In answer to these contentions, Ascher asserts that the action of smoke cannot be represented as an inflammatory one due to mere physical ingredients. Since smoke consists in the greater part of chemical ingredients, the effect upon the human lungs is rather an indirect than a direct one, due to the fact that the normal constituents of the air are replaced by abnormal ones. In order to obtain relevant statistics concerning the effect of smoke upon health, it is necessary to assume that smoke belongs to the same category of harmful agents which have an indirect predisposing effect, such as alcohol, unsanitary dwellings, etc.

Another reason which has been urged against the harmfulness of smoke has been the fact that coal miners show an especially favorable death rate from tuberculosis. According to statistics, very few miners die from tuberculosis. Ascher claims that an explanation of this can be found in the fact that they die of acute lung diseases. The death rate among miners from acute lung diseases is much higher than among other inhabitants of the same age in Prussia. Again, he states, every miner becomes within forty years an invalid and is then no longer considered in the statistics. From this apparently low death rate from tuberculosis amongst coal miners has arisen the belief that coal dust, smoke and soot were not only not harmful but even beneficial to health. Ascher has compiled the following table to show the fallacy of this view:

TABLE I.

FIFTY-FIFTH ANNUAL REPORT OF THE REGISTRAR GENERAL OF THE UNITED KINGDOM.

Death Rate per 10,000.

	T.	NT.
Laborers in Agricultural Districts	18.8	18.6
Coal Miners	14.10	32.6
Chimney Sweeps and Soot Merchants	37.1	43.1
Coal Heavers	29.7	65.6

Here it is apparent that, while the death rate from tuberculosis appears low among coal miners, it is not low among other laborers working in coal dust and smoke. Coal carriers, chimney sweeps, and soot handlers show a high mortality both from tuberculosis and non-tuberculous disease. Ascher maintains that the low mortality from tuberculosis among coal miners is due to the choice of picked workmen, selected for hard work, and who earn a higher scale of wages and live under good social conditions. Hence, the better physical and economic conditions of coal miners serves to explain the low death rate from tuberculosis. These same conditions ought to reveal a

lower mortality from non-tuberculous lung diseases, but as a matter of fact the death rate from such diseases among miners is far above the average.

From German statistics, Ascher found a similar state of matters among coal miners.

TABLE II.

Death Rate per 10,000.

	T.	NT.
Workmen in Prussia (15-60 yrs. of age)	28.8	16.5
Coal Miners in the Ruhr District	13.1	39.2

Here, again, Ascher explains the low death rate from tuberculosis among miners as due to the fact that they are very robust and earn high wages. The high death rate from non-tuberculous lung disease is again very striking.

Statistics of the mortality from tuberculous and non-tuberculous lung diseases for the whole population of Prussia are given in the following table:

TABLE III.

Death Rate per 10,000 of Population.

	T.	NT.
1875-1879	31	16
1880-1884	31	20
1885-1889	29	22
1890-1894	25	22
1895-1899	21	26
1900-1904	19	27

This table shows for Prussia a striking decrease in the death rate from tuberculosis and an increase in other acute lung diseases.

He further compared the death rate of infants in agricultural and industrial districts of Prussia:

TABLE IV.

	East Prussia (agricultural)			Silesia (industrial)			Rhine Province (industrial)		
	NT	S&M	D	NT	S&M	D	NT	S&M	D
1876	4.0	3.1	13.7	3.4	10.9	4.4	3.7	2.1	2.8
1880-81	1.6	4.7	34.9	5.0	8.7	5.5	6.4	3.4	3.6
1885-86	3.4	18.2	30.3	7.3	7.8	8.6	12.2	4.1	2.6
1890-91	4.2	9.9	25.0	10.7	4.1	7.9	15.3	2.3	2.1
1895-99	5.9	7.4	13.9	15.2	3.9	3.2	16.7	2.7	2.2
1900-01	6.9	7.1	10.5	19.4	6.4	2.7	21.2	2.8	1.6

S. (Scarlet Fever.) M. (Measles.) D. (Diphtheria.)

An analysis of the death rate of infants under one year of age for the period 1876-1901 in certain rural districts and industrial districts of Prussia showed the following:

Death Rate per Hundred Under 1 Year of Age.

	NT.	Scarlet Fever, Measles, Diphtheria, Croup.
In six rural districts of East Prussia	4.3	29.8
In six industrial districts of Rhineland	12.6	5.2

He points out here that the contrast between acute lung diseases and contagious diseases among those who are most subject to them, viz., children under one year of age, is very great. This table proves that the higher death rate from acute lung diseases in the Rhenish districts is not due to worse economical conditions nor a more unfavorable climate, for the six districts of East Prussia have a considerably poorer population than the six industrial districts of Rhineland. The former also have a

longer and harder winter and a climate subject to great variations of temperature, whilst the six Rhenish districts have a mild and even temperature. Here, he states, we see a rapid rise of NT diseases, both in the Silesia and the Rhine industrial districts, an increase of from 500% to 600% during a period of 25 years. That such increase is not merely an expression of increased opportunity for infection is clearly proven by the varying behavior of the death rate from the infectious diseases, scarlet fever, measles and diphtheria, the death rate from the latter being highest in the agricultural regions of East Prussia and much lower in the industrial regions.

The death rate of infants in rural and urban districts is again compared in the following table:

TABLE V.

	——NT.——		Acute Infectious Diseases	
	Male	Female	Male	Female
Country	6.24	5.19	25.37	23.81
Small Town	12.61	10.52	18.10	17.75
Medium Sized Town	17.33	15.34	11.36	11.48
Large City	26.30	23.34	10.52	10.52

This table shows significantly an increase in acute lung diseases and a decrease in infectious diseases.

The mortality of infants from acute lung diseases for a period of years in the whole of Prussia is shown in the following:

TABLE VII.

	Death Rate per 1,000.	
	Male	Female
1876-1879	8.3	6.9
1881-1884	11.6	9.6
1885-1889	13.1	10.9
1890-1894	17.3	14.5
1895-1899	20.2	16.2
1900-1909	22.10	17.7

This constant increase in the death rate among infants Ascher believes can be attributed to the increase in smoke. Smoke and soot in small quantities decrease the resistance, especially in weakened individuals, *i. e.*, infants and the tuberculous.

For the purpose of comparing the rates of death for tuberculous and other acute lung diseases, Ascher has taken some statistics from England:

TABLE VIII.

Deaths in England for Every Million of Population.

Years	1850-4	1855-9	1860-4	1865-9	1870-4	1875-9
Tuberculosis	3655.0	3448.0	3367.6	3326.0	3013.4	2903.0
Acute Lung Diseases	2769.0	3155.2	3409.2	3415.4	3607.2	3981.0
T : NT.	1.34	1.09	0.99	0.97	0.84	0.75

Another table for a later period shows:

TABLE IX.

	1881-85	1886-90	1891-95
Tuberculosis	18.30	16.24	14.63
Bronchitis, Pneumonia, Pleurisy	32.09	33.22	33.63
T : NT.	0.57	0.49	0.43

Both tables show a progressive decrease in the ratio, a lowering of the death rate from tuberculosis, and an increase in acute lung diseases.

Ascher's next step was to undertake a comparison of the death rates in smoky and textile districts. He cites the work of Finkelburg, who showed that the mortality from affections of the bronchial tubes is more than half as great again in towns as in the country and, moreover, that it rises to an unusual height not in textile towns, but in places where coal is burned. Ascher's own investigations corroborated the findings of Finkelburg. He con-

trasts the statistics obtained for Krefeld, a typical textile town, and Essen, a smoky town.

TABLE X.

	Krefeld (Textile)			Essen (Smoke)		
	T	NT	T:NT	T	NT	T:NT
1876	5.92	0.61	9.7	4.70	1.18	4.0
1880-81	4.87	1.48	3.2	4.21	3.37	1.2
1885-86	5.37	1.49	3.6	3.66	4.12	0.9
1890-91	3.83	2.81	1.4	2.98	4.48	0.7
1895-96	3.10	2.49	1.2	2.12	3.79	0.6
1900-01	2.37	2.50	0.9	1.91	5.05	0.4

He concludes from this that we always find tuberculosis in textile districts and acute lung diseases in smoky districts.

He has employed the following table, taken from statistics of the Royal Prussian Offices, to show that the mortality from acute lung diseases is 30% higher in smoky than in textile districts:

TABLE XI.

Mortality per 1,000 of Population.

	T	NT	T+NT	T:NT
Textile Districts	1.83	2.23	4.06	0.82
Smoky Districts	1.77	2.93	4.70	0.62

A selection of a group of non-industrial and industrial districts has offered a striking contrast of the frequency of non-tuberculous and tuberculous mortality in the two classes. The statistics are taken for the period 1898-1907.

TABLE XII.

Death Rate per 10,000.

Non-Industrial Area			Industrial Area		
	NT	T		NT	T
Amsberg	28.19	23.59	Dortmond	36.88	14.99
Meschede	26.99	27.35	Bochum	35.89	13.78
Brillon	24.50	29.36	Gelsenkirchen	37.35	15.20

In analyzing this table, Ascher points out that the three industrial districts are wealthier and, for that reason, presuppose a lower death rate from tuberculosis, but they also have a more smoky atmosphere and therefore a higher death rate from acute lung diseases.

For the purpose of establishing the fact that this pernicious influence of smoke is active at all stages of life in increasing the death rate from acute lung diseases, he has compared the statistics from two equally large towns, situated in close proximity, in the industrial district of Westphalia. These towns are built in the same style and differ only in the amount of coal smoke in the air. The town of Hamm, which lies easterly, receives coal smoke only from the west, while Gelsenkirchen, situated centrally, has an atmosphere constantly charged with smoke.

TABLE XIII.

Death Rate per 10,000 during years 1900-1902.

	Hamm Pop. 32,435 30.6 NT	Glesenkirchen Pop. 37,834 57.4 NT
According to age		
0-1 years	228.9	258.9
1-5 "	57.1	131.1
5-10 "	6.5	17.3
10-15 "	1.8	2.6
15-60 "	10.7	34.7
60+ "	140.7	210.2

The influence of smoke here in raising the death rate from acute lung diseases is quite apparent. A similar cause, he believes, serves to explain the increased death rate found in the industrial regions of Westphalia and Upper Silesia, as noted in the following table:

TABLE XIV.

Death Rate per 10,000. Years 1905-1909.	
Death rate from acute lung diseases in all German towns with 15,000 of a population or more	24.0
In equally large towns of Rhenish Westphalia (industrial area)	34.0
In the industrial districts of Silesia	36.0

A study of urban and industrial districts in England yielded a similar result. The average death rate from acute lung diseases for the industrial urban districts was found to be 26.5 per 10,000, and for the rural counties only 17.5.

The later studies of Ascher have disclosed a significant fact in regard to the relation of smoke to tuberculosis. He has demonstrated that tuberculous patients, both male and female, die at an earlier age than formerly. For the year 1876, of every 100 persons dying of tuberculosis in Prussia, 36.64 male and 32.64 females were over fifty years of age. Later, in 1901, only 28.20 tuberculous males and 23.54 females survived the age of fifty. Rahts has similarly shown that of every 1,000 persons of all ages dying of tuberculosis, in the city 112, and in the country 206, have passed the age of sixty. These figures would seem to justify the conclusion that with increasing industrial activity and a thickening of the population, there arises a greater deterioration of public health, not necessarily an increase in the mortality but an earlier age of death. Since the mortality from tuberculosis and other lung diseases is dependent inversely upon the resistance of the individual, the earlier age of death in industrial districts is not to be attributed to an earlier opportunity for infection, but to a lowering of the power of resistance. The factor which causes a lowering of the resistance, Ascher believes, lies in the presence of smoke from coal fires.

Again, Ascher maintains that the tuberculous lesion *per se* does not cause the death of the individual, since the tendency of such a lesion is always towards healing, a

fact readily demonstrated by pathological investigation. The fatal results are due to the secondary infection of the lungs with other micro-organisms, such as the streptococcus, pneumococcus, or influenza bacillus. Therefore, he states, "we can easily imagine that a harmful factor which increases the disposition towards acute lung diseases causes a quicker course in tuberculosis." A proof of this was seen in the influenzal epidemic in Germany in which the tuberculous patients died very quickly. From these facts, Ascher concludes that smokes causes a predisposition towards acute lung disease and hastens the course of tuberculosis.

Ascher hoped to confirm his statistical studies by means of pathological examinations of human subjects. He was unable, however, to obtain sufficient material and was obliged to forego this line of investigation. His animal experimentations were more successful. The points he sought to elucidate were:

1. Does smoke cause a disposition to acute lung disease in rabbits, *i. e.*, are rabbits which have breathed smoke for some time seized by acute lung disease, and are animals not so exposed less liable to contract such disease?

2. Does smoke cause a more rapid course in pulmonary tuberculosis, *i. e.*, do rabbits which have been inoculated with tuberculosis die more quickly if they have inhaled smoke than if they have not?

In order to investigate these questions, ten rabbits were innoculated with tubercle bacilli and these, together with ten control normal rabbits were exposed from 90 to 120 days, 10 hours daily, to a smoky atmosphere. The innoculated animals died on the average of 53.9 days, while the tuberculous control animals lived almost double the number of days, or an average of 90.3 days. In addition to tuberculosis of the lungs, other conditions which developed were bronchitis, purulent pneumonia, and peritonitis. Nor was the smoke, indeed, quite devoid of effect on the entire organism. Some of the rabbits exposed to smoke showed eczema, loss of hair, and scaling. The amount of coal dust in the lungs was small, as a rule,

except in unaffected parts where, at times, it was found in abundance. He also found that moisture combined with smoke intensified its effects. Similar, but less distinct results were obtained by causing animals to breathe soot. The same predisposition to acute lung disease and a quicker course of tuberculosis was demonstrated.

From these statistical and experimental studies, as yet the most comprehensive found in the literature dealing with the smoke evil, Ascher has drawn the following generalized conclusions:

1. The mortality of acute lung diseases is certainly increasing, especially among children and old people. The cause of this increase is due to the impurification of the air by smoke because, in the first place, the increase is greatest in industrial centers and not in agricultural districts. Since 1875, the mortality from such diseases among nursing infants has increased as much as 600%.

2. Within industrial districts a difference in mortality can also be noted, the death rate from acute lung diseases, in districts with a strong smoke development, being higher than in other industrial centers, *e. g.*, textile districts.

3. The mortality among coal miners from acute lung diseases is a much higher one (135%) than among the other male population of the same age. Here also differences can be observed in that in districts with a larger native population the mortality from acute lung diseases is a higher one than in districts where the miners have lately moved from agricultural districts.

4. The conclusion already noted, that the impurification of the air by smoke causes a predisposition to acute lung diseases and hastens the course of tuberculosis.

As was to be expected, Ascher's conclusions have elicited more or less criticism in certain quarters. Indeed, Ascher himself admits that not all doubts as to the assumption of smoke *per se* being the one factor in causing an increase in the mortality from acute lung diseases, and in hastening the course of tuberculosis, have been eradicated. It is conceivable that there may be other contrib-

uting causes which future scientific studies of the problem will be able to demonstrate.

Liefmann is an investigator who has reviewed Ascher's work and corroborated much of it. He further points out that the small industries are more culpable than the larger ones where special attention is apt to be paid to smoke consumption. He regards the supposition that smoke breathed is a menace to health as difficult to prove, and concerning the statistics of Ascher writes:

"Even though the statistical results would scarcely be in a position to give infallible proof the harmfulness of smoke, when considered along with other evidence, they have considerable weight. The high mortality from acute lung diseases among miners should cause serious reflection."

Renk is another investigator of note who would seem to regard the effects of smoke upon health as general rather than specific.

Bartel and Neumann obtained results comparable to those of Ascher in a series of animal experimentations. In experimental inhalation of tubercle bacilli by guinea pigs, these authors found that guinea pigs which had inhaled a moderate amount of smoke on account of their being kept in a large city, died from pulmonary tuberculosis in less time than those which showed smoke-free lungs.

The Sixty-sixth Annual Report of the Registrar General of England discloses the fact that there was an increase in the mortality from bronchitis and pneumonia in England during the quinquennium 1891-1895, and a considerable decrease since then. In connection with this fact Chubb has observed that eighty cities, London, Manchester, Liverpool, *et al.*, have reported a decrease in smoke since that time.

Very few observers have attempted to explain the manner in which air polluted by smoke and dust acts injuriously upon the body tissues. Bachman is one of the few who has offered an explanation. He believes that the blood of people living in cities with vitiated air becomes

impoverished, resulting in an anaemia (or better, dysaemia). This, he states, is apparent in the skin of the city dweller. The effect is not due to a local action upon the skin, but to an admixture of such air in the blood of the lung capillaries. The presence of carbon dioxide, sulphuric acid, etc., in the air inspired acts as a protoplasmic poison interfering with cellular activity and causing an inhibitory action on the ciliated cells of the respiratory tract. When the function of these ciliated cells is interfered with, only coughing and hawking can clear the bronchial tubes so as not to plug up the bronchioles. The organism has, he states, a further protection in the lymphatics and leucocytes, but eventually these auxiliary aids are insufficient to cleanse the air and the whole lymph system becomes flooded with the taking up of poisons. The lymphatic glands constitute the third protection against the dust particles. In this condition of chronic impurity of the blood, the organism suffers and is rendered predisposed to infection with pathogenic germs. The author believes that arteriosclerosis may result from this chronic poisoning. "By breathing impure air we render the body fluids impure, and this leaves the body more prone to disease."

(B) THE EFFECT OF THE INDIVIDUAL CONSTITUENTS OF SMOKE UPON HEALTH.

Smoke is composed of solid carbon particles, or soot, and certain volatile gases such as carbon dioxide, sulphur dioxide, sulphuric acid, and other compounds. We shall consider seriatim the more important of these.

Soot.

In the literature, this term is employed with more or less latitude, at times more broadly as synonymous with coal dust, and at other times as referring simply to carbon particles. Concerning the nature of soot, Cohen defines it as consisting mainly of tar and mineral matter ash, to-

gether with small quantities of sulphur and nitrogen compounds, and frequently possessing an acid character. Various analyses show considerable variation in its composition. Soot is a product of incomplete combustion, and domestic soot, as compared with boiler soot, is said to be richer in carbon and the volatile products such as tar, ammonium chloride, and sulphate, and poor in ash. Russell, of London, first observed that soot contains sulphuric acid, the quantity, as estimated by Cohen, varying from 0.28-1.62%. According to Ascher, soot contains about 31% of mineral particles, principally silicates and iron.

The question of the fate of the inhaled carbon particle has been one that has evoked much discussion. It has been generally believed that it may enter the lung either by inhalation or by absorption from the intestinal tract and the lymphatic system. Since Villaret in 1862 suggested the probability of an intestinal origin of pulmonary anthracosis, many investigations to establish or disprove his claim have been made. His assertion that soot gets into the blood stream through the intestines has not been corroborated. As a result of a series of experiments, Arnold admits the presence of carbon particles in the interior of the intestinal tract, but never in the intestinal wall or in the lymph vessels and mesenteric glands, except in isolated instances, where barely a few pigmented cells could be seen in the intestinal tissue and in the mesenteric glands. Aschoff, Schulze, Cohen and Beitzke, in experimenting with China ink introduced into the abdominal cavity, found that this was absorbed by the lymph ducts of the mesenteric glands and of the diaphragm, the thoracic duct, the vena mammaria interna, and the blood. Thence they found it entered the bone marrow, the spleen and the liver, but not the lungs. The evidence of these authors would appear to prove that anthracosis of the lungs is caused directly by inhalation. Calmette, Guerin, von Behring and others, from experimental data, believe that particles absorbed in the intestinal tract may be carried by the lymphatics to the lungs. Oliver inclines to the latter view as a possibility "but that on this account the

intestinal canal should be regarded as the mode by which the insoluble dust more frequently reaches the lungs, rather than by direct inhalation, I am not prepared to admit. The engagement of the lymphatics in the deeper structure of the bronchi and around the blood vessels suggests the possibility of an intestinal source of infection, but the changes observed in the alveoli of the lungs in the early stages of anthracosis points to the irritation of the epithelial lining by direct contact with dust."

Oliver further points out the danger from continual exposure to dust as a factor in producing structural changes in the lungs, such as the replacement of the normal spongy tissue by fibro-connective tissue. He regards pulmonary fibrosis in the early stages as due solely to the irritation by dust. Von Behring believes that pulmonary tuberculosis in such fibrosed lungs frequently develops from the lighting up of disease long latent in the lymphatic glands as a result of a probable intestinal infection contracted during childhood. Oliver cautions persons who are employed in dusty occupations and to whom, as a result of exposure to dust, the ciliated epithelial cells of the trachea are lost, carefully to rinse their nostrils with warm water before leaving the factory.

The manner in which the carbon particles penetrate into the lung tissue after being inhaled is another mooted question. The earlier observers believed that the individual coal particles, because of their inherent hardness and angularity, bored into the tissue, while a later view is that the dust particles are drawn into the tissue by the migrating dust cells. Knauff believes that the dust penetrates the tissue both free and attached to cells. Ruppert maintains that the greater part of the dust enters the tissues without being attached to cells, since he found no evidence of inflammation around pigmented areas. He does not regard it as probable that coal, in the form of soot particles, has the physical strength for boring into the tissues, although this might be a possibility for other forms of dust.

Ruppert conducted a number of animal experiments in the Polytechnic Institute, Heidelberg, upon dogs and rabbits for the purpose of elucidating the following points:

1. What changes are caused by the inhalation of the dust in the epithelium of the air passages in the deeper tissues of the organ of respiration?

2. How does the dust penetrate into the tissue, in a free state or enclosed in cells?

3. What passage does it use in entering?

4. What forces impel it forward in the process?

From the results of his experiments he writes: "So little injurious was the soot to the respiratory organs of the animal that even weak animals can be exposed to the smoke for weeks without suffering particularly." In order to force a sufficient amount of soot into the respiratory passages, he was obliged to tracheotomize the animals. He believes that when dry coal dust is in the form of soot it is less irritating than other forms of dust, *e. g.*, stone dust. When, however, it is suspended in a fluid, as in the experiments of Slavjansky, it has an important irritating effect. The latter found severe acute pneumonia in many cases, but such changes rarely occur except when large quantities of dust have been inhaled. From Ruppert's experiments the conclusion may be drawn that soot is less harmful in its effects on the respiratory organs than the various forms of mineral dust.

Other writers express a similar opinion, that soot, *per se*, is not especially harmful, and the fact would seem to be fully well substantiated that non-carbonaceous dust (mineral and other) is more serious in its effects upon health than is carbonaceous. Sir James Crichton Brown states "that of all mineral dust carbonaceous dust seems to be the least injurious to the human organism. * * * Besides being in some degree antiseptic, carbon dust is less irritating and scarifying than many other industrial dusts, and it is really by their irritating and scarifying power that the lethal effects of dusts are to be measured." Dr. Evans of Chicago writes: "Smoke carbon is probably as little harmful as any solid which can be taken into

the human body. It is quite inert chemically. Physically, it irritates but little. The harm that it does is that it transports bacteria and secures entrance for them where alone they would be repulsed."

The question as to whether soot and coal dust possess active antiseptic properties has long been a subject for dispute. Formerly, soot and coal dust were believed to have an inhibitive action on the growth of the tubercle bacillus and tuberculous lesions. An evidence of this belief is found in the fact, cited by Jacobi, that the metal grinders of Sheffield had, until twenty-five years ago, the habit of going into places filled with coal dust after having been in the metal dust all day. The majority of such artisans contracted "Grinder's Asthma," which at present is regarded as tuberculous. This belief in the protective power of soot is now known to be largely erroneous, and Mendelssohn, as early as 1885, stated that he had met many persons dying from tuberculosis whose symptoms never showed themselves until they worked in coal dust and smoke.

Oliver thinks that soot acts in a manner different from coal dust. "Soot," he states, "increases the action of incipient tuberculosis, whereas coal dust has an unfavorable effect on the tubercle bacilli. Soot has only a mild action in preventing infection by tuberculosis, whereas coal dust is active in its immunizing qualities. The acid elements of the soot are not only an irritant, but an aid to tuberculous development. * * * It is a common experience that the course of pulmonary tuberculosis is hastened by living in a smoky atmosphere. Also that smoke predisposes to acute lung diseases. Soot differs from coal dust in being a spongy material capable of absorbing sulphuric acid and hydrochloric acid up to 10%, besides retaining other free acid gases and certain oxidation products of a tar-like nature."

According to Lehman (cited by Oliver), the sulphur-dioxide contained in soot is absorbed by the nasal mucous membrane and the particles of carbon are carried further into the respiratory passages, and are finally deposited in

the lung tissue, having meanwhile in their descent given up to the bronchial mucous membrane and the lining membrane of the lungs some of the acids which they retained.

Cornet, by means of animal experimentation, demonstrated that soot did not contain any qualities which would stop or inhibit the tuberculous process.

A number of pathological conditions in the respiratory organs have been attributed to the deposit of soot and coal dust in the lungs by different observers. True it is, that the burden of opinion, until recent years, has been that anthracosis was a condition productive of no specific phenomena of disease, and that lung lesions were independent of the inhalation of dust, and further, that coal miners were almost always immune to tuberculosis. Of late, these older views have been questioned. One condition that has been ascribed by Seltmann and others to a deposit of coal dust in the lung is that of dyspnoea. As quoted by Schlockow: "Seltmann came to the conclusion that a deposit of coal in the lungs, as soon as it reaches a certain degree, diminishes the gaseous exchange by decreasing the breathing surface, checks the formation of blood, and so causes anaemia and dyspnoea." Croque similarly attributes the dyspnoea of miners to anaemia which he regards as due to the faulty aeration of the blood in the lung capillaries around which the deposits of coal press upon the vessels so as to interfere with the flow of blood. He offers no explanation of the fact that many cases of anthracosis are not accompanied by dyspnoea.

Chronic bronchitis and emphysema are two further associated conditions towards which the inhalation of coal dust and soot is believed to exert a predisposing influence. The frequency of chronic bronchitis among coal miners is a well-known fact. Hirst believes that the inhaled particles of coal dust clog the bronchial secretions. Tobold states that the carbon itself of smoke is deposited in the nose, throat and bronchi in a fine form which acts as an irritant causing morning cough. The ciliated cells are no longer able to cope with the deposit of carbon and a chronic inflammatory condition of the mucous membrane

results. It is commonly observed clinically that emphysema follows in the train of chronic bronchitis. Merkel explains this as due to the fact that during spasms of coughing the glottis becomes compressed and the air is forced through the lower bronchi, causing distention of the alveoli and ultimately of the thorax as a whole.

Schlockow regards the dust in the air of mines as especially well adapted to produce emphysema, since certain parts of the lung tissue, due to the deposit of coal dust, are thrown out of function either temporarily or permanently. This entails increased function in the remainder of the lung tissue and, as a result, an abnormal distension of the pulmonary alveoli. Ultimately, as this phenomenon is repeated, the pulmonary vesicles lose their elasticity and become permanently distended. Racine asserts that coal dust may cause emphysema directly through this excessive inspiratory distension without the initial factor of chronic bronchitis. Every inspiration of dust laden air entering the aveoli tends to obstruct them. Each following inhalation introduces more air which, on account of these partly occluded alveoli, must find other air space. Gradually, the open air cells have an increased function thrust upon them and permanent distension results. As a proof of his view, he cites the case of two healthy young miners in whom dyspnoea developed, due to their using lamps producing an unusual amount of soot. After leaving the mine, the dyspnoea gradually disappeared but within a year in each of the miners emphysema developed without the presence of any previous bronchitis.

Concerning the incidence of chronic bronchitis and emphysema among the miners in his district, Racine has compiled the following tables:

TABLE XV.

Among a total of 870 miners there were the following reports of illness for the years 1880-1882:

	1880	1881	1882
From all causes	221, or 27.8%	242, or 29.8%	273, or 27.1%
Chronic Bronchitis	16, or 7.2%	15, or 6.2%	18, or 6.5%
Emphysema	18, or 8.1%	19, or 7.9%	23, or 8.5%

The average for these years was

For Chronic Bronchitis	6.6%
For Emphysema	8.1%

The deposit of coal dust in the lung tissue has been regarded as the exciting factor in the causation of pleurisy. The occurrence of a dry fibrinous pleurisy is frequent among coal miners. Racine, along with Merkel, believes that this may be due to a rather large deposit of coal dust in the neighborhood of the lung periphery, acting as an irritant.

A more remote injury to lung tissue dependent upon the presence of coal dust in the lungs, as observed by Seltmann and Eulenberg, is the production of small cavities, following a localized pneumonia or a limited gangrenous process within the lung tissue. They found that such areas presented the appearance of an inky fluid and at times contained pus. Racine believes that the limited necrosis is due to the pressure exerted by the deposit of large amounts of coal dust, exciting inflammatory changes which result in the formation of abscesses and cavities.

Regarding the disputed question of the inhibitive action of soot against a tuberculous process in the lungs, Racine's views are somewhat at variance with those of Ascher. He states: "My own observation leads me to ascribe to anthracosis of the lungs a protective influence against tuberculosis." He believes that soot has consid-

erable disinfecting power, and that its presence is deterrent against the growth of bacteria.

Coullard has made an exhaustive study of the effect of smoke upon the health of firemen, and his observations are of interest in their bearing upon the problem of industrial smoke and health. He describes the following symptoms arising from the inhalation of soot:

1. Effect upon the eyes. Redness and congestion of the conjunctiva, accompanied by copious tears and a sensation of prickling, as of a foreign body in the eye. In cases of severe irritation, the ciliary glands, and the lachrymal glands also, may be inflamed; or still more serious ailments (including a hypersecretion).

2. Effect upon the nasal passages. The carbon particles or heavy vapors, inhaled through the nose, cause a rapid inflammation of the nasal mucous membrane. This congestion is accompanied by hypersecretion and is frequently complicated with the frontal sinus. This inflammation is the frequent cause of frontal headaches.

3. Effect upon the pharynx. Very frequently in firemen, enlargement of the tonsils is found, which Dr. Henning of Leipzig does not hesitate to attribute to the irritating effect of smoke. Redness and congestion of the pharynx are very frequent.

4. Effect upon the respiratory organs. Irritating smoke causes inflammation of the larynx which may even develop into oedema, or swelling of the glottis, the irritation being most often betrayed by a dry, spasmodic, and very painful cough. The vapors and particles of smoke penetrate into the trachea and the bronchial passages, and in individuals subject to emphysema, or cough, bring on asthmatic symptoms. In extreme cases, bronchial pneumonia and lobar pneumonia may result, but most often pneumonia, in the case of firemen,

is attributable to the cold caused by the great quantities of water poured upon the fire.

This author further employed animal experimentation in pursuing his study concerning the effect of smoke. From the results of his experiments he concludes that the carbon particles play a large role in the production of serious symptoms. He believes that a certain number of slight disorders, frequently observed in firemen, are due to the action of carbon particles upon the mucous membranes, but he states "although it is true that animals poisoned by filtered smoke (*e. g.*, smoke from which soot has been withdrawn by filtering) return to life more easily than those which have been subjected to common smoke," he yet maintains that the poisonous gases of the smoke are responsible for the grave symptoms produced by the inhalation of smoke among firemen, rather than the soot.

Apart from its more obvious effect upon the respiratory organs, soot has for many years been more or less fancifully believed to create a predisposition towards the production of cancerous growths among workmen who are brought into contact with it. Only recently Sir Thomas Oliver has again called attention to this possible relationship. While the handling of coal itself is not apparently attended by any risk from cancer, there is some evidence for the belief that working with soot does seem to predispose towards it. To quote from Oliver: "In Great Britain we are familiar with chimney sweeps' cancer. Something, therefore, is present in soot in a chemically active form which irritates the skin and leads to cancer. That the scrotal cancer of chimney sweeps is the result of irritation caused by soot is confirmed by the youthful age and occupation of its victims. Years ago, Earl published notes of three cases of cancer occurring on the hands of gardeners, who had been distributing soot among plants. All the men were under the age of thirty. Earl's father described a case of cancer of the scrotum in a boy eight years of age who was a chimney sweep, and Sir James Paget observed the disease in the ears of workmen who had carried sacks of soot on their shoulders. Mortality

figures show that in England and Wales cancer among chimney sweeps is twice as frequent as in occupied males generally. The comparative mortality figures for cancer among chimney sweeps between the ages of twenty-six and sixty-five was, for the three years ending 1903, 133, as compared with 63 for occupied males at the same ages. We must therefore admit that the chimney sweeps' occupation is a cause of cancer." It is scarcely conceivable that the amount of soot in the air of industrial towns is sufficient in amount to be an exciting cause of cancer, as it may possibly be in the case of the chimney sweep.

EFFECTS OF THE GASEOUS CONSTITUENTS OF SMOKE UPON HEALTH.

The gaseous components of smoke, which are believed to exert a baneful influence upon health, include carbon monoxide, carbon dioxide, certain sulphur and arsenic compounds, and nitrous and chloric vapors.

Carbon Monoxide.

Carbon monoxide is a product of combustion, which is known physiologically to act as a poison on the human organism if inhaled in sufficiently large amounts. According to Gruber, an atmosphere becomes dangerous when it contains 0.05% of carbon monoxide, while Haldane believes that symptoms may be caused by as small an amount as 0.02%. Kinnicut and Sanford state that air containing 0.3% of the gas causes death, 0.2% very dangerous symptoms, and that mice will quickly show the effects of the gas when the air contains only 0.005%. The smoke from iron furnaces, it is stated, may contain as much as 25%-35% of carbon monoxide gas. When present in lethal proportions the principal symptoms produced are severe headache, vertigo, a vague feeling of illness, marked muscular weakness, and frequently nausea and vomiting. If the amount of gas be greater, drowsiness and loss of consciousness and death may result.

Gautier made an analysis of air taken from the open street and found that only 1 part in 500,000 of carbon monoxide, a proportion presumably inert in regard to health. He believed, however, that the air near factories would contain a sufficient amount of this gas to prove a menace to health. Very few estimations of the amount of carbon monoxide in the air of industrial towns have been made.

Fodor, from the results of animal experimentation, concludes that human beings are much more sensitive to the effects of this gas than are the lower animals. "If present," he states, "in greater quantities than 0.15% it is dangerous to health, especially if breathed continually, and when present in quantities of 0.05%, or even perhaps as low as 0.023%, it produces a bad effect." One of the results of long continued breathing of carbon monoxide is the production of a severe anaemia. So violent, he writes, is the action of carbon monoxide that even when an animal has recovered consciousness, after poisoning by carbon monoxide, the danger is not passed; the animal, and likewise the human being, may pay the penalty with his life, although the inhalation of carbon monoxide has ceased. The affinity of carbon monoxide for the blood, or conversely, of blood for carbon monoxide, is such that carbon monoxide is taken into the organism and into the blood circulation when the atmosphere contains no more than .004% or 1/25,000 part." For the reason that such minute amounts are harmful, Fodor does not believe that any minimal quantity is permissible in the air, and he maintains that to be perfectly healthy an atmosphere should contain no trace of carbon monoxide.

Tobold believes that, while the amount of carbon monoxide in our dwellings is not appreciable ordinarily, small quantities of it may cause headache and indisposition. Sambere regards the determining factor of the effect upon the human organism as the proportion of carbon monoxide to the oxygen breathed, rather than the absolute amount of carbon monoxide.

Coullard states that the effects of carbon monoxide and carbon dioxide inhalation by firemen are not, as a rule, very serious. The symptoms produced in firemen are mainly headache, weakness, nausea, and vomiting, fainting, dyspnoea, and a rapid pulse, coldness of the extremities, profuse perspiration with pallor of the face, diarrhoea, neurasthenia, and the development of tuberculous lesions. Practical trials prove that a robust man, entering a smoky atmosphere which contains 1%-1.5% of carbon monoxide, is affected by asphyxial disorders only very slightly serious if he does not remain in this atmosphere more than ten or fifteen minutes. For carbon monoxide to produce very rapid effects, Coullard estimates the gaseous mixture inhaled must contain at least 15% of it—ten times as much as is contained in ordinary smoke.

Carbon Dioxide.

According to Renk, normal air contains about .03% and city air about .03%-.05% of carbon dioxide. The air of factories, during the daytime, has about an average of 10.1 volumes per 10,000 of the gas, while at night, when the gas is burning, it has been estimated that there are about 17.6 volumes per 10,000. In an atmosphere in which oxygen has been reduced to 1.5% or 3%, a proportion of 12%-15% of carbon dioxide produces fatal results.

Schaffer states that in London 100,000 tons of carbon dioxide are poured into the air each day as smoke. Every ton of completely burned coal gives rise to about three tons of carbon dioxide and monoxide. According to Tobold, carbon dioxide in a proportion of 1:10 acts as a poison, causing headache and shortness of breath, while if present in a proportion of 30%, death may result. Evans believes that we can stand a much higher percentage of carbon dioxide than is ever found in the outside air, and that while carbon monoxide is directly toxic, carbon dioxide is only depressant and remotely toxic and is never fatal in "one whiff in any concentration." But, as he wisely adds, "neither does a child get a complete education in five minutes in a grammar school."

Coullard studied the poisonous effects of carbon dioxide in observing the effect of smoke on firemen. He states that a smoky atmosphere is sometimes extremely poisonous, due to the presence of quite large quantities of carbon dioxide rather than to a deficiency of oxygen. The fact that Pettenkofer was able to pass several hours in an atmosphere containing 1% of carbon dioxide without being made ill by it lead Coullard to regard this gas as not especially poisonous. Another observer breathed without difficulty for ten minutes an atmosphere in which there was 4% of carbon dioxide.

The effect of carbon dioxide inhalation may be explained as follows, according to Bert (cited by Coullard): "From the accumulation of carbon dioxide in the blood there results a progressive abatement of the oxidation within the organs, and from this as a consequence, a considerable lowering of the temperature of the body. The central nervous system in the general action upon the organism first manifests that it is affected by the loss of reflex transmission, first to the limbs, then to the eyes, then finally to the respiratory center, from which death results without any agitation or convulsive movement."

While the air of smoky towns does not contain carbon dioxide in a proportion sufficient to produce the foregoing results, nor, indeed, to prove instantly a menace to health, yet it is altogether probable, as has been suggested, that "small amounts of excess carbon dioxide, continued for long periods of time would, within limits, tend to have the effects somewhat similar to the effects of large amounts breathed for a short period of time."

Sulphur.

The sulphur compounds found present in smoke occur mainly in the form of sulphur dioxide and sulphuric acid. The amount of sulphur, in this form in a smoky atmosphere, is at times so great that it cannot be disregarded as a probable exciting cause of ill health. It has been estimated that in London 981,792 pounds of sulphur are

poured into the air each day, or over 500,000 tons in the course of the year. In Glasgow and Manchester, it is stated, twenty tons escape each day in the smoke. According to Rideal, the quantity of sulphur found present in the air of London, from different analyses, is from 0.015-0.77 grams per 100 cubic feet. At Kew, as much as 2% of sulphur was found in an analysis of dust from an exposed surface. According to Nicholson, one-half cwt. of sulphuric acid is deposited over every square mile of Manchester, and in Chelsea very much greater deposits.

There is much unanimity of opinion regarding the deleterious influence of the sulphurous gases upon health. Concerning the action of sulphur dioxide on the human organism, Cushny states that 5 parts in 10,000 acts as an irritant causing sneezing, coughing, and lachrymation, and that, in somewhat greater concentration, it becomes entirely irrespirable; still smaller quantities in the air cause bronchial irritation and catarrh when inhaled for some time. Lehman believes that sulphur dioxide in amounts as small as .001% (0.1% being fatal) causes discomfort, and that 3 parts in 100,000 renders some persons decidedly ill after a few minutes. It is the opinion of Harrington that sulphur dioxide affects the digestive tract rather than the respiratory organs.

According to a theory expressed by Markel, sulphur is possessed of a somewhat ferment-like action in the air in its tendency to automatic renewal. This author states that sulphurous acid coming into contact with iron is immediately oxidized into sulphuric acid which, in turn, reacts upon iron forming ferrous sulphate and iron oxide. The latter drops off as rust and begins a new sulphur cycle. It is also commonly known that sulphur dioxide possesses certain disinfectant properties, being germicidal in the proportion of not less than 92 grams of sulphur per cubic metre. The presence of moisture enhances this disinfecting action and at the same time, it is believed, increases the injurious effect of sulphur dioxide upon the human organism.

It is probable that sulphurous fumes are the most deadly of all the gaseous constituents of smoke. In this connection Evans states: "Sulphur compounds are very objectionable and probably more harmful than carbon compounds. Probably before long our dense smoke ordinances will be changed so as to add to the carbon control other provisions which will control sulphur compounds. Possibly, also, the combustion experiments will likewise be directed more to the solution of the sulphur problem."

Coullard's studies among firemen did not lead him to regard the inhalation of sulphurous acid fumes as productive of any very serious disorders. Opposed to this, is the opinion of Schaefer, who has made a special study of the effects of sulphur gases on health, and who attributes lasting and serious results due to the inhalation of these gases. Sulphur fumes, he believes, play a large role in the etiology of asthma. To quote him: "The importance of sulphur dioxide as an impurity of the air of our cities and its injurious effects upon the organs of respiration is a subject that has not reached the attention, in works on hygiene, that it demands. The writer has been studying the noxious effects of sulphur dioxide as an impurity of the atmosphere for the past ten years, and he has arrived at the conclusion that it is one of the most potent causal factors of asthma."

Ascher has cited experimental work done by Kimball on rabbits which demonstrated the fact that, by causing rabbits to breathe small quantities of sulphuric acid fumes, tuberculous infections were increased. The same author states that other experimental work has shown that sulphur inhalation causes a decrease in the bactericidal action in tuberculous lungs and a lowering of the power of resistance.

Arsenic.

It has been found that most varieties of coal contain small quantities of arsenic, probably in the form of arsenical pyrites. Cohen and Ruston believe that the

arsenic which is found present in the air and water comes from the smuts of coal smoke. Traces of arsenic have been found in household dust (0.010%—0.004%) and an analysis of rain water in London showed 0.003 parts per million of arsenic.

According to Delepine, "the large amount of arsenic in soot causes a marked arsenical contamination of the air in Manchester and may account for the bad effects of air on vegetation." In regard to the effect upon health, he writes: "I have a suspicion that soot from towns where arsenical coal is used is far more irritating to the lungs than pure coal dust. The reason why I say so is that I have noticed there is generally more fibrous tissue produced in the lung in town anthracosis than when the coal is inhaled as dust, for instance in the case of coal miners. Lungs of coal miners may be as black as soot without very distinct evidence of inflammatory reaction whilst, on the contrary, in towns, where the amount of carbon collected in the lungs is smaller, there are frequently capsules of fibrous tissue in the lungs around small masses of carbon which have accumulated, indicating some irritating action on the part of soot. All town dust, as well as town air, is never free from arsenic."

In connection with the influence of arsenic inhalation upon health, Cushny's statement concerning the miners of Reichenstein, who are constantly exposed to arsenic owing to its being contained in large quantities in the ore, is of interest. Concerning these, he writes: "These people are described by Geyer as shortlived, very subject in childhood to severe rickets and in adult life to dropsies and respiratory diseases; they offer little resistance to microbial infection and frequently present the skin and nervous symptoms of arsenic poisoning."

Nitrogen and Chlorine Gases.

These gases occur mainly in the smoke of industrial centers, especially in the fumes from factories using nitrate of mercury and from chemical works where sul-

phuric acid is manufactured. They probably play a minor role in the effect of smoke upon health. Coullard has called attention to the manner in which they appear to act injuriously. The nitrous vapors act, he states, (1) by powerfully irritating the bronchi and the small pulmonary vessels to the point of producing centers of apoplexy, and (2) by producing a special impairing of the blood." The chlorine fumes, he believes, while slightly irritating, do not cause serious disorders except through prolonged inhalation, in which case it is not uncommon for a tuberculous process to become lighted up.

As a general resume of the effects of the various constituents of smoke upon health, the following note of Mehrsten's is clarifying. This author writes: "It is not even the dust only that is injurious, but it is the invisible products of combustion escaping from the chimney in the form of gases, of which we need to take into consideration only carbonic acid, nitrogen, carbonic oxide, and sulphurous acid. The first two, which are heavy gases, can probably be regarded as injurious only when they are driven directly into dwellings and this cannot happen unless the chimneys are not high enough. The carbonic oxide, which is extremely poisonous, is usually present in such slight quantities that it becomes greatly diffused as soon as it escapes from the chimney. On account of its lightness it also arises rapidly and is lost in the higher strata of air; but, on the other hand, the gaseous sulphuric acid (sulphur dioxide) generated from the sulphur contained in the coal, which is considerably heavier than the air, is such an injurious gas that first of all our efforts must be directed toward preventing it from doing harm, all the more as it is not only the human organism that is injured but also, in a positively destructive manner, the vegetation, soil, and buildings. These facts show that any smokeless furnace with a direct combustion has little value for the public as far as the purification of the air is concerned unless the dust and the sulphurous acid are retained at the same time."

INDIRECT EFFECTS OF A SMOKY ATMOSPHERE UPON THE HEALTH OF THE COMMUNITY.

Fogs.

It is a fact long well known that a smoky atmosphere predisposes towards fogs. When an atmosphere laden with suspended particles becomes charged with moisture a fog is precipitated. According to Nicholson, over 25% of the fogs are caused by smoke.

Not infrequently, following a fog period, there is a sharp rise in the death rate of a community, the rise being due mainly to the great increase in deaths from diseases of the respiratory organs. Scientific observers of vital statistics have here once again placed varying interpretations upon this increase, a few authorities regarding the increased mortality as dependent upon the alterations in the temperature and other factors, while others maintain that the fog, *per se,* is the exciting cause of the increase. Among the former class of conservative writers is W. J. Russell of London, who, in commenting upon the effects of London fogs, writes: "By far the greater number of fogs occur when there is a great fall in temperature, and clearly this is followed closely after a few days by a great increase in the death rate; but how much of this is to be attributed to the fog and how much to the fall in temperature may be difficult to determine, but we have evidence that when fogs occur without fall of temperature they do not appear to be followed by any remarkable increase of the death rate, for on December 15, 1889, there was a dense fog and the temperature was even above the average; under the conditions, the death rate remained far above the average. On December 13 and 14 of the same year there was a dense fog, an average temperature, and only an average death rate; and the same thing happened on February 4, 1890, when, notwithstanding a dense fog, the death rate remained low, and last winter on November 13 and 14 there was a dense fog, a high temperature, and an

average death rate. With these four exceptions, depression of temperature goes with fog. There is no case of depression of temperature not followed by increase of death rate. That many people suffer, both physically and mentally, from the effects of fog there can be no doubt, but so far as I can interpret these returns of the Registrar General, they do not confirm the popular impression that fog is a deadly scourge; at the same time, it is beyond doubt that an atmosphere charged with soot dust and empyreumatic products is an unwholesome atmosphere to breathe; but I think that the principal cause of the great increase of deaths when fogs occur is attributable rather to the sudden fall of temperature than to the fog itself. As to bacteria—the experiments of Dr. Percy Frankland show that fogs do not tend to concentrate or nurture them, for he found that there were remarkably few bacteria in London air during a time of fog."

In the opinion of Rollo Russell, while cold is doubtless a contributing factor in raising the mortality during a fog period, the most potent cause lies in the smoke constituents of the foggy atmosphere. He writes: "Great cold combined with fog is not productive of much illness in the country. In smoky towns, the case is far different. Thus, in London, the death rate was raised in a single fortnight, from January 24 to February 7, 1880, from 27.1 to 48.1 per thousand. The fatality and prevalence of respiratory diseases were enormously increased. The excess of deaths over the average in the three weeks ending February 14 was 2,994, and in the week ending February 7 the deaths from whooping cough were unprecedently numerous, 248, and those from bronchitis numbered 1,223. At least 30,000 persons must have been ill from the combined effects of smoky fog and cold. * * * The large excess of carbon dioxide, sulphurous acid, and of microorganisms and effete organic products was partly concerned in these ill effects, but the factor of greatest importance was the finely divided and thickly distributed carbon, or carbonaceous matter, which irritated the breathing passages and lungs. The results corresponded rather

closely with the gradual ill effects of the dusty trades. * * * After a fortnight of dense fogs, the deaths in London for one week ending January 2, 1892, exceeded by 1,484 the average number, being at the rate of 42 per thousand. Increases took place in the following diseases: Measles, 114%; whooping cough, 173%; phthisis, 42%; old age, 36%; apoplexy, 58%; diseases of the circulatory system, 106%; bronchitis, 170%; pneumonia, 111%; other respiratory diseases, 135%; accidents, 103%. These results are in the main attributable to the concentration of the ordinary London air with moisture and intense cold to help their deadly work. The majority of the fatal cases were weakened constitutions, though many were among the robust. The experience of large towns is that the power of recovery after illness is less within their confines than in the country. In fog, the evil influences of town air are many times multiplied." The same writer, in another place, contrasts the influence on health of a country and a London fog by quoting statistics of the death rates of Croyden and London during the great fogs of 1880. In Croyden, the number of deaths rose from 35-36 in three weeks, while in London the number rose 2,994 above the average during that time.

Ascher attributes the baneful influence of fog to the fact that the fog concentrates great quantities of smoke which is inhaled into the lungs in much larger quantities from the damp air of foggy weather than from dry air. This, he claims, has been proven experimentally. Moreover, he studied the mortality tables of Manchester in relation to fog, and drew the following conclusions: "We see that the mortality from respiratory diseases and phthisis increases during a period of fog, while the incidence of contagious diseases is not affected by it, a fact that has been known in England a long time." He was of the opinion, however, that it would not be justifiable to draw an unqualified conclusion regarding the influence of fog since other attendant conditions might also increase the mortality from lung diseases, e. g., the sudden fall in temperature, which usually precedes a fog.

Des Voeux studied the effects of two fog periods in Glasgow upon the subsequent death rate. In the autumn of 1909 there occurred two periods of smoke fog in that city, each of several days' duration but separated by an interval of a few weeks. The death rate rose suddenly during the first period from 18 to 25 per thousand, and during the second week to 33 per thousand, while the death rate in the rural environments of the city was increased only very slightly. It was estimated that 1,063 deaths could be attributed to the foggy weather.

Niven has made a similar study concerning the influence of fog periods upon the death rate in Manchester. He found that when fog periods were charted out and the number of deaths from phthisis and other forms of respiratory diseases tabulated, an unmistakable rise in death rate could be demonstrated occurring within a few days from the onset of the fog. He believes that an increase of micro-organisms occurs during fogs and that thereby a diffusion of bronchitis and other diseases finding entrance to the lungs is facilitated by fogs. The following tables showing the bearing of fogs upon the death rate are taken from his report.

Table I. Showing deaths from phthisis in each six weeks preceding, and in each of six weeks containing or following a fog, for the twenty years 1891-1910, added, the fogs not being of longer duration than six days.

Weeks preceding fogs:

6th	5th	4th	3d	2d	1st
2,192	2,040	2,049	2,135	2,161	2,224

Weeks of fog and following:

1st	2d	3d	4th	5th	6th
2,377	2,468	2,360	2,339	2,334	2,399

"It thus appears that there is an increase in deaths, greatest in the first three weeks but continuing into subsequent weeks. The greatest number of deaths is in the week after a fog."

Table II. Showing deaths from Pneumonia during weeks similarly disposed to fogs, in the same manner for the years 1897-1910, added.

Weeks preceding fogs:

6th	5th	4th	3d	2d	1st
1,351	1,389	1,345	1,442	1,442	1,494

Weeks of fog and following:

1st	2d	3d	4th	5th	6th
1,572	1,638	1,657	1,710	1,631	1,589

"There is here a clearly marked influence on mortality ascribed to pneumonia, greatest in the fourth week following the fog and next greatest in the third week. The effect on pneumonia is clearer than that on phthisis and ites maximum intensity is differently disposed, as if time were required for the development of the pneumococcus and the course of the disease."

Table III. Showing deaths from bronchitis during weeks similarly disposed for the years 1897-1910.

Weeks preceding fogs:

6th	5th	4th	3d	2d	1st
1,317	1,301	1,330	1,526	1,479	1,627

Weeks of fog and following:

1st	2d	3d	4th	5th	6th
1,808	1,864	1,848	1,699	1,729	1,749

"Here the maximum effect is manifested in the first three weeks following, the greatest effects being produced in the second week as in phthisis. The increment observed in the different forms of respiratory diseases before the occurrence of fog is due to the fact that the different fogs interfere from the proximity to each other. Thus,

while the effect of the individual fog is diminished, the total effect in producing an increase in mortality becomes more conspicuous. The increase in mortality from bronchitis, like the increase from phthisis, follows more closely on fog than does that from pneumonia. The processes are probably different. In the case of phthisis and bronchitis, fogs cause congestion; in the case of pneumonia, they probably light up pneumonia. When the fogs are of longer duration than six days, the numbers are comparatively small."

Table IV. Respiratory diseases, other than Phthisis, for the years 1891-1905.

Weeks preceding fogs:

6th	5th	4th	3d	2d	1st
304	312	289	355	363	389

Weeks of fog and following:

1st	2d	3d	4th	5th	6th
394	484	417	398	375	332

"No effect is observable under phthisis. The effect of fogs in producing mortality from respiratory diseases is unmistakable."

Diminution of Sunlight.

That a smoky town means diminished sunlight in the community is a fact universally conceded, even by such as may be skeptical concerning any direct influence of smoke on health. Indeed, some authorities are inclined to attribute the evil effects of smoke to this decrease in sunlight rather than to its irritating action on the organs of respiration. In this connection, Kister writes: "The development of smoke and soot has without doubt a bad effect upon the daylight and sunshine of a place, and, be-

cause of the hygienic significance of sunshine, is inimical to our physical, or at least our psychical, welfare. But it is not easy to prove such a direct injurious effect of smoke upon our respiratory organs."

The precise manner in which a diminution of sunlight acts injuriously on health is a subject more or less under dispute. It is well known that sunlight is germicidal. Tubercle bacilli quickly succumb to the action of direct sunlight, a few hours' exposure sufficing to destroy their vitality, although they may live indefinitely in darkened surroundings. Quite apart from this bactericidal action there are apparently other factors, in decreased sunlight, which militate against health. Liefman has grouped these harmful influences under three headings. "If," he states, "we group all these experiences in the significance of light for our life and health, and concentrate them upon the problem which here interests us, we shall be led, in my opinion, to the conclusion that a darkening of the atmosphere of our great cities is injurious to health in three ways: (1) An exciting impulse which influences our disposition is weakened and the energy of metabolism, especially as it concerns respiration, is diminished. (2) The illumination and warming of the earth, the water, and the air within the precincts of our great cities is diminished and in this way a series of hygienically important processes is influenced or depressed. (3) The chemical and bactericidal effect of the sun's rays is decreased and thus bacteria, especially the pathogenic ones, are permitted to thrive.

Sir William Ramsay regards smoke as harmful by virtue of its power directly to absorb light, and by its effect in the formation of clouds and fogs which, he states, are peculiarly fitted to absorb the blue, the violet, and ultra violet rays, these being the rays that are especially germicidal. In this manner a diminution of sunshine causes an increase of bacteria, both pathogenic and non-pathogenic, in the atmosphere. The same writer believes that sunlight has a direct influence on the human skin as well as upon mental states.

In order to ascertain the possible bearing of diminished sunshine on the mortality returns from respiratory diseases, Niven has compiled the following statistics for the industrial cities of Manchester, Birmingham, Sheffield and London:

RELATIVE MORTALITY FROM RESPIRATORY DISEASES AND RECORD OF SUNSHINE

	PHTHISIS				BRONCHITIS				PNEUMONIA				SUNSHINE (Hours)			
Year	Man-chester	Birm-ingham	Sheffield	London	Man-chester	Birm-ingham	Sheffield	London	Man-chester	Birm-ingham	Sheffield	London	Man-chester	Birm-ingham	Sheffield	London
1901	2.09	1.73	1.41	1.71	2.22	2.07	1.51	1.62	1.96	1.73	1.45	1.35	1192	1144		1567
1902	2.08	1.65	1.18	1.65	2.44	1.91	1.52	1.71	1.98	1.62	1.45	1.47	928	1048		1228
1903	1.85	1.45	1.36	1.62	1.87	1.73	1.71	1.15	1.87	1.48	1.58	1.28	1119	972	1216	1445
1904	1.98	1.54	1.27	1.70	1.97	2.06	1.51	1.40	2.18	1.72	1.39	1.45	1031	1239	1325	1459
1905	1.56	1.45	1.15	1.50	1.85	1.68	1.56	1.33	1.62	1.55	1.44	1.53	1005	1149	1432	1420
1906	1.71	1.29	1.05	1.53	1.74	1.68	1.46	1.18	1.59	1.47	1.32	1.45	1069	1143	1438	1735
1907	1.70	1.29	1.20	1.51	2.06	1.76	1.76	1.32	2.02	1.66	1.70	1.66	894	952	1428	1417
1908	1.65	1.39	1.28	1.44	1.96	1.73	1.65	1.15	1.75	1.35	1.59	1.46	992	981	1281	1634
1909	1.70	1.44	1.17	1.44	2.00	1.77	1.32	1.35	1.72	1.46	1.54	1.68	999	1129	1332	1641
1910	1.49	1.25	1.01	1.28	1.59	1.51	1.26	1.12	1.40	1.33	1.52	1.49	982	1011		1380

It is difficult to draw any exact conclusions from this table, the evidence presented being, on the whole, somewhat equivocal. A more or less general decrease in respiratory diseases (with the exception of a slight rise in pneumonia in London and Sheffield) may be noted but it cannot be demonstrated that this general decrease is concurrent with an increase in the number of hours of sunshine in the four cities. London and Sheffield show a slight increase in the number of hours of sunshine; Birmingham and Manchester present a continuously decreasing number of sunlight hours.

Some statistics of Kister for the city of Hamburg, one of the smokiest of German towns, are comparable with those of the English towns. The following table is taken from a report of this writer on the Investigation on Smoke and Soot in Hamburg:

MORTALITY FROM NON-TUBERCULOUS DISEASE OF THE RESPIRATORY ORGANS.

For the City of Hamburg.

(Diphtheria and Whooping Cough are not included.)

Year	Under 1 yr.	1-15 yrs.	15-30 yrs.	30-60 yrs.	60-70 yrs.	Over 70	Total	% of Dead	.01% of Living
1894	412	305	17	143	258	173	1308	12.08	21.636
1895	559	400	22	153	289	280	1703	14.50	27.513
1896	509	377	14	152	261	324	1537	14.03	24.204
1897	417	279	20	149	260	246	1371	12.37	20.996
1898	527	309	18	139	245	255	1578	13.51	23.589
1899	405	363	27	158	339	348	1630	13.74	23.807
1900	527	414	20	218	417	1318	2914	16.50	28.935
1901	548	405	19	156	328	324	1780	14.49	24.845
1902	444	293	21	169	369	342	1748	14.55	23.854
1903	390	333	33	177	364	322	1619	12.99	21.674
1904	441	310	22	155	373	313	1614	13.37	21.021
1905	422	315	51	325	265	384	1762	14.09	22.262
1906	535	358	44	296	223	322	1778	14.24	21.768
1907	466	262	62	419	280	429	1918	15.37	22.709

The comments of the author upon this table are, in part: "If we collect the figures for Hamburg, the following (the table) shows a decrease or perhaps an increase

in recent years from other diseases (non-tuberculous) of the respiratory organs. * * * Even though this fact cannot be referred, without further investigation, to the development of smoke, it is one of the phenomena that must be taken into account in the question of the hygienic effect of smoke."

OTHER REMOTE EFFECTS OF SMOKE UPON HEALTH.

The psychological aspect of the smoke nuisance has been dealt with at length in another Bulletin of this series. The present day recognition of the importance of the mental influence of mind upon bodily health is a hopeful sign of the times. It is doubtful whether the maximum of mental acumen and bodily efficiency may for long be preserved in a smoke laden atmosphere. True it is that, at times, certain minds can rise above their environments, but whether the sum total of the mental efficiency of the community can be equal to that of a community living within cleanliness, aerial and environmental, seems scarcely conceivable. Not infrequently one hears the complaint of the casual visitor to a smoky city with respect to a feeling of mental depression provoked by the smoky atmosphere. Certain physicians have pronounced themselves upon this aspect of the problem. Dr. Evans of Chicago writes: "It (smoke) serves to lower the general tone of a community. A spotless town is more apt to be moral than a dirty town. It is useless to try to get a spotless town and leave the smoke. If the air is dirty it is very hard to get the streets, the yards, the clothes, the people clean." C. A. L. Reed speaks in a similar strain: "Physical dirt is closely allied to moral dirt and both lead to degeneracy. It is too much to expect the best results in the public schools that exist beneath the sombre shade of smoke. It is difficult to imbue the young with a sense of the beautiful when the beauty itself is bedaubed with soot." Jacobi believes "that a clean community has the better chance to avoid degeneracy," and

Des Voeux asserts that "dirt and darkness are the twin children of smoke, and to them are closely related poverty, drunkenness and crime."

A further quite obvious factor which renders a smoky town unhealthy is the necessary curtailment of free house and factory ventilation. To open a window carries with it the certain penalty of soiling a curtain, a rug, or a counterpane. The zealous housewife accordingly is prone to guard the cleanliness of her house at the expense of fresh air. Out of door sleeping porches are, for a similar reason, conspicuous by their absence in smoky towns, in contrast with their increasing use in more cleanly modern cities. The fine shower of soot detracts from the comfort of sleeping in the open air and speedly soils the bed linen. Such a more or less universal avoidance of fresh air militates strongly against the health of the community and predisposes its inhabitants to tuberculous and other diseases.

Bibliography

Ascher, L. Die Einwirkung von Rauch und Russ auf die menschliche Gesundheit. Deutsche Med. Wchnschr. 1909. xxv., 585; 633.

——— Coal Smoke Abatement in England. Journ. Roy. San. Inst., London., 1907-8. xxviii., 88-93.

——— Die Rauchbekämpfung in England and Deutschland: ein Reisebericht Deutsche Vrtljschr. f. öff. Gsndhtspflg. 1907. xxxix., 291-303.

——— Tuberclose und nicht tuberculose Erkrankungen der Athmungsorgane in Preussin. Seit 1875. Berlin Klm. Wchnschr. 1903. xl., 1012-1016.

——— Zur Beurteilung der Rauch- und Russfrag. Ztsch. f. Hyg. u. Infeck. Krank. Leipz. 1911. lxviii., 551-568.

——— Die Rauchbekämpfung in der englischen Abteilung der Internationalen Hygiene-Ausstellung. Rauch u. Staub. No. 2. Nov., 1911. p. 41.

——— Luft untersuchungen. Rauch u. Staub. No. 3. 1910. p. 78.

——— Die Rauchbekämpfung in Königsberg. Rauch u. Staub. No. 7. April, 1911. p. 218.

——— Die nächsten Aufgaben der Rauchbekämpfung. Rauch u. Staub. No. 1. Oct., 1910.

——— Die Einwirkung von Rauch u. Russ auf die menschliche Gesundheit. Rev. Rauch u. Staub. No. 1. Oct., 1910. p. 27.

——— Weiteres Material zur Rauch frage. Rev. Rauch u. Staub. No. 8. May, 1911. p. 268.

Bachmann, Dr. Verunreinigte Luft als Krankheitsursache. Rauch u. Staub. No. 4. Jan., 1911. p. 129.

Cohen and Ruston. Smoke: A study of Town Air. London. Edwin Arnold.

Coullard, H. L'intoxication par les fumees chez les sapeurs-pompiers. Ann. de' hyg. Par. 1909, 4. s. xii., 490-541.

Des Voeux, H. A. (et al.) Discussion on Smoke Abatement. Brit. Med. Jr. Lond. 1908, ii., 566.

Evans, W. A. An address before the Illinois Fuel Conference at the University of Illinois. Chicago, Ill. 1909.

Fodor, J. V. Carbon Monoxide in its relation to health. Budapest. 1880.

Gautier, A. Les fumées de Paris; influence exercée par les produits de combustion sur l'atmosphere de la ville. Rev. d'hyg. Par. 1901. xxiii., 97.

Glinzer, C. Address on the Smoke Nuisance and its Abatement before the Industrial Society of Hamburg. 1890.

Jacobi, A. Smoke in relation to health. Journ. Am. Med. Ass. Chicago. 1907. xlix., 813-5.

Kister. Bericht über die in Hamburg ausgefuhrten Rauch- und Russuntersuchungen. Gesundheits-Ingenieur. Munchen. 1909. xxxii., 841. 1910. xxxiii., 30.

Liefmann, H. Ueber die Rauch- und Russfrage, insbesondere von gesundheitlichen Standpunkte u. eine Methode des Russnachweises in der Luft. Deutsch Vrtljschr. f. off. Gsndhtspl. Brnschwg. 1908. xl., 282-344.

Lungenentzündung durch Raucheinatmung. Zeitschr. f. Gewerbe-Hygiene. 1912. No. 6. S. 138. Rev. Rauch u. Staub. No. 9. June, 1912. p. 261.

Mehl, W. Rauch u. Russ in hygienscher Bedeutung. Gesundheit. Leipz. 1905. xxx., 747.

Mehrstens, John H. Solution of the Problem of the Smoke Nuisance. Glasers Annalen für Gewerbe u. Bauwesen. vol. 60. 1907.

Nicholson, W. Smoke Abatement. J. San. Inst. Lond. 1903-4. xxiv., 749.

Niven, J. The Smoke Nuisance. Public Health. Lond. 1890-91. iii., 236.

O'Connor, John J., Jr. History of the smoke nuisance and of smoke abatement in Pittsburgh. Industrial World. v. 92. 1913. p. 352-354.

Oliver, Sir Thomas. Soot and Cancer. Lancet. Lond.

——— Dust and Fume, Foes of Industrial Life. The Lancet. Lond. Sept. 28. 1912.

——— On diseases of the lung caused by dust. J. Preventive Med. Lond. 1905. xiii., 317-333.

Racine, Dr. On the relation of Empysema and Tuberculosis to Anthracosis in Miners. Vrtljhrschr. f. gericht. Med. n. s. vol. 40-41.

Ramsay, W. Les dangers de l' obscurussiment de l'air par les fumées. Rev. d' hyg. Par. 1896. xviii., 857-867.

Reed, C. A. L. The smoke question viewed from a national standpoint. Am. Med. Phila. 1905. ix., 703-05.

Renk, F. The Air. 1886.

Rubner, Max. Ueber trübe Winterlage nebst untersuchungen zur sog. Rauchplage der Gross-stadte. Archiv. für Hygiene. 1906. lviii., 323-328.

——— Die Rauchplage in der Gross-städten. Arch. f. Volkswohlf. Berl. 1907-8. i., 153-166.

Ruppert, H. Experimental Investigation on the Inhalation of Coal Dust. Virchow's Archiv. vol. 72. 1878.

Russell, W. J. On town fogs and their effects. Trans. Seventh Internat. Cong. Hyg. and Demog. Lond. 1891-2. v., 11-22.

Russell, F. A. Rollo. The atmosphere in relation to human life and health. Smithsonian Report. 1895.

——— London fog and smoke.

Schaefer, T. W. Contamination of the air of our cities with Sulphur Dioxide. Boston Medical and Surgical Journ. vol. 157. 1907.

Smoke Abatement. Papers of the Royal Sanitary Institute Conference at London. Dec., 1905.

Smoke nuisance with special reference to the conditions prevailing in Philadelphia. J. Frankl. Inst. Phila. 1899. cxlvii., 244-247.

Soot fall in London. The Times. Lond. Jan. 5, 1912.

Süpfle, K. Die Rauchplage. Klin. therap. Wchnschr. Berl. 1911. xvii., 279-285.

Tobold. Die Rauchbelastigung. Bl. f. Volksgsndhts. pflg. 1903. iii., 65-71.

Trotter, R. S. The so-called anthracosis and phthisis in coal miners. Br. Med. Journ. Lond. 1903. i., 1197.

Wainwright, J. W. Bituminous coal smoke: its chemistry and effects on the health of man and plants, works of art and commerce. Med. Rec. N. Y. 1908. lxxxiv., 791-793.

Pulmonary Anthracosis—A Community Disease

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Definite observations upon the presence and nature of pigment within the lung substance and its associated lymphatic structures are of relatively recent date. Nevertheless, as early as 1717, Ramazzini discussed the presence of carbonaceous material within the lung and indicated an association with definite pulmonary diseases. His observations were made upon various laborers who, through the inhalation of angular stone particles, became predisposed to asthma and tuberculosis. His observations, however, did not suggest that any of the foreign material contained within the lung consisted of a carbon deposit.

Not until Pearson in 1813 studied the problem and applied the term anthracosis or coal miner's lung, followed by a report by Laennec in 1819, was a more acute attention attracted to the subject. Pearson indicated that individual coal particles when inhaled became deposited in the lung tissue and upon the accumulation of larger quantities of this pigment, the lung gave macroscopic evidence of its presence.

This contention was further supported by Gregory, who in 1831, described the pigment in the lungs of a coal miner with definite tissue changes within the organ. Other English authors (Thompson, Simpson and Stratton) made similar observations and indicated the importance of anthracotic deposits as a type of occupational disease.

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Although Pearson's views were accepted in England, they were strongly combatted in Germany, particularly by Koschlakoff, as well as by Virchow and Henele, who regarded the coloring matter in the lung substance and in the lymph nodes of haematogenous origin. Virchow did not believe that the lung substance could be penetrated by inert foreign particles. As late as 1855, Barthelmess discussed "pulmonary melanosis" as a progressive pigmentation of the lung resulting from repeated haemorrhages and inflammation. Subsequently, however, Pearson's observations were confirmed in Traube's clinic (1860) where some carbon pigment, presumably having its origin from charcoal, was demonstrated in the lung substance. Since then the deposition of carbon from smoke has been amply confirmed by studies upon human lungs as well as by animal experimentation. The first confirmatory animal experiments were carried out by Knauff (1867) and repeated by Konradi (1869).

In the earlier discussion upon the inhalation of dust, including the carbon particles of smoke, it was indicated that as every one inhaled more or less of it during his lifetime, little pathological change, other than the storage of pigment, occurred in the lung tissue. It was, however, recognized that the inhalation of different foreign substances had a varying effect upon the respiratory organs. It was deemed that carbon pigment had the least harmful effect and, hence, the deposits which were found in the adult lungs, could be disregarded as having any association with respiratory diseases or clinical symptoms during life.

With the admission that the black pigmentation of the lung was the result of the inhalation of carbon particles contained in the air, a considerable controversy began concerning the manner in which the foreign material made its entrance into the tissues. It was not uncommonly observed that the sputum of individuals working in smoky atmospheres would, for considerable periods of time, contain black pigment particles. Some of this foreign material was free, while some was contained with-

in cells. Knauff believed that these cells were desquamated epithelial structures of the bronchi, which had lost their cilia. It was thought that with the active desquamation of the epithelial lining that a more ready access for the pigment to the deeper structures was possible. Sikorsky and Klein both believed that foreign particles were able to pass between the uninjured epithelial cells and directly enter the lymphatics.

On the other hand, Arnold and Schottelius opposed the view of the direct migration of the pigment into the tissues and claimed that the transport was accomplished only through the agency of cellular activity. This phagocytosis they believed could be accomplished by the bronchial epithelium, lymphoid wandering cells and by the alveolar epithelium. Traube thought that the acicular nature of the carbon particles would account for their tendency to pierce the delicate alveolar walls and then to migrate to other parts by the lymphatic channels. Rindfleisch accepted this latter view, laid stress upon the gritty hardness of the particles and believed that through the impact of the air current the particles may be driven through the superficial tissues. He furthermore pointed out that when these foreign particles had entered the lymph spaces they not uncommonly became incorporated within cells having phagocytic properties.

The theory of the phagocytic transport of the foreign particles continued to gain ground but there was no agreement concerning the nature of the active cell. Slavjansky and Ins believed that the leucocytes were most active, while Ruppert, Schottelius, and others believed that the alveolar epithelium picked up the carbon from the air sacs. Arnold was divided in his views, considering that both types of cells were capable of carrying out this function, while, furthermore, he believed that under certain conditions migration of pigment might occur in the absence of phagocytic cells. Even to the present time, observers are not in unison concerning the taking up and storage of the pigment which reaches the lung. The exact nature of the phagocytic cells, and these seem to be the

active participants in the accumulation of carbon pigment in the pulmonary structures, is still in debate. In a recent study, however, it appears to us that Haythorn has conclusively shown that although pigment may appear in a variety of cells, the important cell acting as a carrier from the air sac to the interstitial tissue and the lymphatics of the lung, is an endothelial cell.

Although, in the early days of cellular pathology (1858) the finding of extensive pulmonary anthracosis was unusual, the situation has changed much in the present day. Then, as now, the most intense examples of pigmentation of the lung were found among the coal miners, and it was particularly to these that Pearson, just a century ago referred, when he first applied the name anthracosis to the pulmonary condition. Apparently, early in the last century, in the course of ordinary life the accumulation of pigmented dust bodies within the lung was hardly sufficient to attract the attention. At that time, the use of coal was less general among the housekeepers and the combustion of wood was relatively complete, with but slight pollution of the air. Then, too, coal was not in as general use in industries nor had the use of the steam engine found a definite place in manufacture.

To-day the use of coal forms the main source of energy for the remarkable industries, which began in the middle of the nineteenth century. We need not indicate by figures or statistics the extent to which our progress is determined by the use of coal. Nor is it within our province to indicate the enormous losses entailed in the incomplete combustion of coal. The main fact stands before us to-day that in every city where householders use coal or in which manufactures of any capacity are located, the air shows a greater or less pollution by carbon particles. To-day it is almost possible to gauge the extent of the manufactures within a city by estimating the quantity of carbon in the atmosphere. In other words, what, not so many years ago was a rather unusual aerial condition, to-day forms a constant finding and has added a nuisance which affects the well-being of the community. No longer may we re-

gard the presence of carbon in the air of large cities as a harmless factor. And furthermore, the gradual accumulation of this foreign material within the respiratory tract has a definite effect upon the tissues in reducing their functional activity and in possibly leading to secondary disturbances affecting our general bodily health.

The following observations have been made upon a series of autopsies, in which the deposit of carbon pigment within the lungs was particularly noted. These observations were made upon civilians not engaged in coal mining. The majority of them had been residents of the Pittsburgh district for the greater part of their lives. We were unable to account for the great variation, which occurred in the intensity of the pigmentation among the different individuals, particularly when their respective occupations appear to have had no relation to the amount of the pigment deposit in the lungs. Thus one of the most markedly pigmented lungs was obtained from a peddler, in whose history we could find no particular association with a sooty atmosphere or industry. It is possible that the tissues of different individuals store the foreign particles from the air with different degrees of activity.

Admitting that but few individuals to-day can escape the accumulation of carbon particles in the respiratory system, it may be suggested that the condition should be looked upon as a normal process. This attitude has been the dominant one in the discussion of pulmonary anthracosis. As, however, we must to-day freely admit that individuals living under different circumstances and in different communities suffer unequally from the quantity of inhaled dust, it is impossible for us to designate all of these as normal conditions. That a small amount of anthracosis of the lungs is not incompatible with good health is obvious to all who have observed the condition in many autopsies. That, however, certain communities are subject to greater pollution of the air by smoke than others, and that the individuals in these communities suffer in an equally greater degree from the inhalation of soot and smoke is also obvious to those who have had an oppor-

tunity of comparing the lungs from different localities. The pathologist has no difficulty in recognizing lung specimens from large manufacturing cities.

Thus we are able to observe variations from the almost non-pigmented lung tissue obtained from those living far distant from cities, to the more intensely pigmented lungs, the coal miner's lung illustrating the extreme degree of carbon deposit. There is, however, some difference between the deeply pigmented coal miner's lung and that obtained from the city dweller. The carbon dust as inhaled in the coal mines is considerably coarser than the fine particles of soot found in the city air. Moreover, the dust in coal mines is made up of fine angular and rough particles, while soot is a mixture of a very fine amorphous carbon and ash.

ANATOMICAL CONSIDERATIONS.

The deposition of carbon pigment in the lungs from the dust-laden air is dependent upon the respiratory function and the activity of the lymphatics. The inhaled air with its carbon particles is carried to varying depths into the lung tissue. The major amount of the foreign material adheres to the moist walls of the respiratory passages and never reaches the lung tissue proper. It is possible, and microscopical analysis seems to confirm this, that the foreign material that adheres to the mucous membranes of the nose, pharynx, trachea, and larger bronchi is but rarely carried into the tissues of these tracts, but lying in the secreted mucous, is carried upwards and is eventually expelled. The relatively lesser quantity of dust and carbon which reaches the lung alveoli also becomes adherent to the moist surfaces of the alveolar sacs and then by the activity of certain cells which have been studied and described by Haythorn, these particles are eventually carried into the lymphatics of the alveolar wall where they are disposed of by the lymphatic system of the part. The subsequent distribution is to a great extent determined by the site of absorbtion within the lung. Thus

the carbon particles which have found their way into the air sacs near the surface of the lung, gradually accumulate within the lymphatics of the visceral pleura, while the carbon which is collected from the more centrally placed alveoli, accumulates about the lymphatic channels which drain that particular area. The tendency of this absorbed carbon is to pass from the finer lymphatic channels of the alveolar walls to the larger passages, eventually reaching a lymph node where the onward progress of the particles is impeded by the filtering action of this structure. In the main, the lymphatic drainage of the entire lung converges at the hilus and passes into the peri-bronchial glands located in this part.

The lymphatics of the visceral pleura form an intricate network of channels which surround each lobule. The lymphatics upon the pleural surface of these lobules can, at times, be recognized by the naked eye. Many anastomoses occur and the larger channels drain towards the hilus. Communications between the surface lymphatics and those within the organ have also been demonstrated.

A somewhat similar system of lymphatic channels have been demonstrated about those lobules which lie within the lung. Not only is there a system of lymphatic channels about the lobules, but small passages extend into the individual alveolar walls. Sikorsky, as well as Wittich, claimed to have demonstrated small patent communications between the lymphatic channels in the alveolar walls and the air sacs. By this means, it was suggested, that foreign materials within the air sacs could find a ready passage into the interstitial lymphatic system. In 1878 Rindfleisch suggested similar passages for the entrance of coal pigment into the lung tissues. He believed that the fine dust particles could pass directly from the air sac into the interstitial lymphatic channels without the intervention of phagocytic cells. He did appreciate the role of phagocytosis in the subsequent transportation and storage of the foreign material. It would appear, however, with the most recent studies that the migration of

the dust particles from the air sacs occurs only through the agency of certain wandering cells.

To thoroughly appreciate the progressive pigmentation of the lung substance by the inhalation of carbon particles, the general mechanism of respiration as well as the efficient lymphatic drainage of all the air sacs must be understood. The important conclusions of Beitzke and Most, that the lymphatics of the lung and visceral pleura have no direct communication with those of the head, neck, or abdomen, and the fact that carbon particles are rarely found in the circulating blood, indicate that pulmonary anthracosis is developed through the activity of the respiratory functions alone. It is, therefore, quite out of place, here to discuss the claims of Calmette, and his associates, for the origin of pulmonary anthracosis in the alimentary tract. A more extensive review and study of the relation of intestinal absorption to anthracosis is given by Montgomery. This author from his own experiments concluded that the respiratory route alone was the important one leading to pulmonary anthracosis.

DISTRIBUTION OF PIGMENT BENEATH THE VISCERAL PLEURA.

At first sight, when the lung is examined externally, the distribution of the deposit of carbon pigment seems to be irregular and without any association with the anatomical structure of the lung. The pigment is deposited in small granular masses, which, in their beginning, occupy areas less than of pin-head size. Sometimes it would appear that the deposit is in the nature of lines which, however, on slight magnification are found to be the coalescence of numerous small granular points.

As the deposit becomes more extensive, the pigment is found to follow a definite arrangement and the anatomical structures which, in the non-pigmented lung are not visible, are mapped out by the deposit. Thus the subpleural pigmentation is found to pick out the septa dividing the lobules of the lung. This geographical marking is

more particularly evident in the early stages of the pigmentation prior to the diffuse deposition of the pigment with the consequent obliteration of the early linear markings. Whereas in the early stages of the deposit, the septa of the lobules show fine linear deposits of pigment, the increasing accumulation of the carbon leads to an irregular thickness of these lines and the conversion of them into small chains of nodules or to the development of flat or shot-like masses in the sub-pleural tissues. Gradually the deposit extends from the septa into the tissues of the lobules until blotches of pigment become prominent. All gradations, from the finest hair-like lines in the septa, to diffuse pigmented areas in which the normal color of lung substances can not be recognized, are not uncommonly seen in the same lung.

The macroscopic appearance of the sub-pleural tissues is a very good gauge as to the actual amount of carbon pigment contained in the lung. That is, with the deposit of such an obvious pigment no difficulty is experienced in distinguishing its presence or in gauging the amount present in each portion of the tissue examined. It is well, however, to recognize that the amount of carbon pigment on the surface does not necessarily indicate the extent or distribution of the pigment in any part of the lung tissue. There are factors arising with each lung and within each lobe which tend to modify the amount of pigment within the tissue.

Although the pigment follows the septa of the lung lobules, the distribution upon the surface is by no means uniform. It has been repeatedly observed that the amount of pigment in the different lobes as well as in the different portions of the same lobe varies very considerably. The distribution of the pigment in the sub-pleural tissues is dependent upon the course of the lymphatic stream. But, as has been indicated by S. R. Haythorn, the presence of carbon pigment within the lung may have a marked effect upon the subsequent condition of these lymphatics. Thus, as we shall discuss later, the deposition of carbon pigment resulting through the activity of certain phagocytic cells

has a tendency to stimulate tissue changes which modify the architecture of the organ. It is most probable that by this means the deposit occurring in the lung tissue does not always appear in the same characters, but according to the particular tissue reaction (fibrosis) a modification of the lymphatic system leads to an altered physiological process in which the amount of deposit may be increased or decreased.

In the examination of a series of lungs it soon becomes evident that there are certain areas in the normal organ, which become involved earlier than others, and which usually show the most intense pigmentation in the later stages of the process. Thus in young adults, who show no evidence of other disease processes in the lung, the pigment is more prominent in the apex of the upper lobe, the anterior border of the upper lobe, and the posterior border of the upper and lower lobes. Even in these three areas the distribution of the pigment is by no means uniform, for in different individuals the grades of intensity of the deposit differ somewhat within these locations.

It is the usual observation that the least pigmented portions of the pleural surface of the lung are the diaphragmatic and the interlobar surfaces. It is not uncommon, however, to observe a sharp line of pigmentation separating the outer surfaces of the pleura from the interlobar areas. At the border of each lobe as it lies in apposition to its fellow, there is a marked pigmented zone, more intense than the deposit upon the free surface, and serving as a boundary between the pigmented pleura and the non-pigmented interlobar surfaces.

The distribution of pigment in the apex of the lung varies according to the shape of the part and to the character of the dome of the chest cavity. As has been pointed out by Schmorl it is not uncommon to have an unusual prominence of the upper ribs and irregular folds of the parietal pleura forming bands which divide the otherwise round dome of the chest cavity into several smaller compartments. These abnormal ridges are very common, but are not constant in their disposition. Not uncommonly

they pass from behind inwards and forwards, crossing the dome in an arched and rather spiral direction, the anterior extremity passing towards the hilus of the lung. At other times, folds of parietal pleura pass from behind upwards and forwards, crossing the highest points of the pleural sac. When these folds are marked a definite depression is left upon the lung surface, particularly if the lung is unduly distended by emphysema or pneumonia. This depression is observed in the nature of a groove looking not unlike the vertical grooves seen over the right lobe of the liver (Liebermeister's groove). These grooves become more marked and more permanent with the age of the individual. Not only do they present depressions in the soft and spongy tissues of the organ, which, in the early years of life, can easily be obliterated, but in the course of years they remain as definite areas of retraction where the lung substance does not expand nor develop equally with the rest of the tissue. Thus in the apical grooves the lung tissue is inhibited in its growth and its functional activity is hindered by obstructing bands. Moreover, the lung tissue opposite the depths of the grooves is prone to become fibrosed or to develop adhesions to the parietal pleura.

These apical grooves are also to be recognized in the variation of the deposit of the pigment. The grooves when well marked attract the attention by showing a lessened amount of pigment than the surrounding tissue. When several well marked grooves occupy the apex of the lung, then this part of the lobe appears to contain decidedly less pigment than other parts. Yet on closer examination, although the apical pleura may appear to contain less pigment than the other pleural areas, this is due to the absence of pigment in the grooves themselves, and not to the variation of pigment in the parenchyma of the lobe.

The ridges bounding these grooves usually show an unusual pigmentation. The extent, however, of the deposit on the borders of the grooves, is not uniform in that it is not uncommon to observe one border deeply pig-

mented while its fellow on the opposite side contains but little carbon.

The intensity of the pigmentation along the posterior border of the upper and lower lobes is commonly the most marked in the entire organ. From the early beginning of a tortoise shell marking indicating the division of the lobules, the condition progresses until the pigmentation produces one diffuse coloration of the pleural tissues.

Furthermore, there are two important considerations respecting the localization of anthracotic pigment in the pleura. The first of these is the relation of the pigment deposit to the position of the intercostal spaces and the ribs. The second is the relation of the pigment deposit to the opposed pleural surfaces between the neighboring lobes.

In respect to the relation of the pigment to the ribs and intercostal spaces there have been a number of views expressed. As above stated, our attention has been particularly attracted to this question through the observations of Schmorl upon the apical grooves of the lung. Similar rib impressions are found in adults upon the surfaces of both the upper and lower lobes. Schmorl in 1901 indicated that the uppermost ribs produced individual impressions upon the lung substance which were easily recognized at autopsy. Schmorl found these depressions in children, but noted that they tended to disappear with advancing age. He believed that the depressions were the result of the undeveloped chest pressing upon the lung substance and that with the development of the thorax, in the normal individual, the pressure upon the lung was much relieved. He noted, however, that in those individuals, whom we are prone to look upon as possessing the anatomical character of a tuberculous subject, the flat chest, these grooves or rib depressions upon the lung remained permanently. Thus he believed that the anatomical characteristics of the chest altered the relationship of the lung to the pleural cavity which in the undeveloped condition was prone to bring about those anatomical changes of the lung, inviting tuberculosis.

The grooves in the lung tissue had the effect of compressing both the lymphatic and blood vessels. Likewise a certain interference might be produced in compression of the bronchial tree. These pathological conditions tended towards a stasis of the circulation of the part, permitting a more ready development of the tuberculous process.

It has, furthermore, been shown that not only do the ribs in the uppermost portion of the thorax leave their impression on the lung tissue, but that such marks may be distinguished for the entire series of ribs down to the eight or ninth. These rib markings or impressions are more readily followed by observing the deposit of pigment than by the actual depressions produced upon the lung substance.

As one will readily appreciate, the intensity of the impressions of the ribs upon the lung varies in different individuals. Not uncommonly, the thorax is of such dimensions or its capacity bears such a relation to the lung, that little or no effect of rib pressure is to be noted. Under those conditions in which the volume and consistence of the lung is increased, as in lobar pneumonia, the rib depressions are temporarily more decidedly marked.

Peiser has studied a series of cases and finds that the rib grooves are not well marked in the infant. In this he differs from Schmorl. He believes that the rib grooves increases in their depth as the individual assumes the upright position and the thoracic wall sinks. As the thorax, with increasing age, gradually assumes its new level, the upper ribs become more prominent on the inner wall of the thorax. These then produce depressions upon the lung surface. Not only does the sinking of the thoracic wall lead to the prominence of the rib margins, but the respiratory movements are altered, there being a diminished respiratory activity established. This in its turn has the effect of producing a pulmonary stasis and a lessened elasticity of the lung. Peiser believes that with the altered condition of the respiration, the character of the lung sub-

stance changes so that the rib grooves are more readily produced.

Further observations have recently been made by Orsos. He studied the mechanics of respiration as regards the relationship of the expanding thoracic wall to the spongy lung substance within. He indicates that the thorax, constituting a closed cavity, has its walls made up of parts which are of different composition. In part, the wall consists of solid structures, the ribs, while in other places soft portions, make up a part of the active walls. He points out that the effect of these two types of tissue upon the lung substance is different. The solid ribs, he believes, are more active in producing a suction by the expanding chest and a compression by the contracting chest wall. This greater activity in relation to a part of the chest wall has its effect upon the lung substance in that the tissue immediately opposite the firm ribs is functionally more active during the respiratory movements. The inactivity of the intercostal spaces is not only to be observed in the smaller alveolar spaces, but also in the more sluggish lymphatic drainage leading to the greater deposition of the insoluble carbon particles. Thus in the adult the intercostal spaces become more richly marked by the deposit of anthracotic materials.

In discussing the views expressed by Orsos, an opposite stand was taken by Marchand, Aschoff and Beitzke, in that each of them expressed his belief that the greater deposit of pigment occurred in the areas mapped out by the ribs.

In our own observations, we must, in the main, agree with the findings of Orsos. Some difficulty is experienced in determining which portion of the lung lay opposite the ribs, particularly when there have been no marked depressions, while the deposit of pigment is quite decided. There can, however, be no doubt, as to the depressions opposite the first, second, or third rib, and in these situations, the grooves which are very decided contain less pigment than the high points of the ridges. At this point, however, it is necessary to introduce a word of explana-

tion in discussion of the pigment deposit in and about the costal grooves. It is best to study those lungs which are moderately advanced in the anthracotic process, and which are not altered by the presence of adhesions. Inflammation introduces a factor which modifies the normal distribution of pigment so that we can no longer ascribe our findings to the influence of the costal grooves alone. We shall discuss the effect of inflammation upon the deposit of coal pigment at another place.

It is, furthermore, to be indicated, that the deposit of pigment along the intercostal areas is not uniform. Although the margins of the grooves as well as the intercostal spaces contain the greater amount of the pigment while the depth of the groove is almost always free, it is impossible to make a common statement as to the exact outline of the deposit for each groove. No doubt, the intensity of the pigmentation is determined to a certain extent, by the individual characters, such as the prominence of the ribs, the corresponding depth of the groove, and the local pressure upon the lymph and blood vessels.

In support of the views of Orsos that the cavity of the grooves exhibit less pigmentation than the surrounding portions, is the fact that the natural depression as well as the opposed pleural surfaces between the lobes have the same characters as the rib grooves in being less pigmented than other parts. It is the common observation to find a pale non-pigmented pleura on the interlobar surfaces while the external visceral pleura is mottled by a pigment deposit. The same is true of the diaphragmatic surface. Here, too, a less amount of pigment accumulates. This variation in the distribution of the pigment upon the pleural surfaces is not dependent upon the difference of the respiratory function of the lung alveoli beneath these parts, nor is it due to a difference in the character of the distribution of the lymphatic channels which surround the lung alveoli, but it is dependent upon outer influences of pressure which modify the capacity both of the alveoli and lymphatics. In the normal lung these influences of pressure are to be observed mainly in the rib grooves, the

interlobar and diaphragmatic surfaces. It is possible that the presence of points of pressure upon the lung tissue has the quality of massaging the parts during respiratory activity and thus driving the particles of pigment more rapidly to other parts. We are inclined to believe that this quality of massaging the part by intermittent friction, plays the important role of preventing the accumulation of carbon pigment in the given regions of the lung. As we shall point out later, the lack of flow in the lymphatic system does not prevent the accumulation of foreign particles. Stasis of the lymphatic system, although preventing the fluid within the channels from flowing with normal rapidity has little effect upon the migration of the cellular elements, which are the main means by which the foreign material is transported. Thus, although stasis prevents the proper flow of the serum through the lymph channels it permits the wandering of phagocytes into the obstructed region where these may accumulate in undue proportion. These wandering cells with their pigment burden are the chief causes for the pigmentation of the given areas of lung tissue.

INTERSTITIAL PULMONARY ANTHRACOSIS.

The nature of the distribution of carbon pigment in relation to the pulmonary alveoli within the lung is very similar to that observed upon the pleural surface. We do not, however, have an opportunity of viewing the pigment in the same manner. Thus in a cross section of the lung we do not have the opportunity of observing the surface of the lobules, but see only cross sections of the partitions. Thus for the most part our attention is attracted to the deposition of pigment at the points where the partitions meet. In these situations we observe small nodular deposits not uncommonly the size of pin heads. At first sight, it would appear that the amount of pigment within the lung is relatively less than that observed on the surface. Nevertheless, it can be observed that the total amount of pigment within the lung tissue bears a rela-

tion to the quantity observed on the surface. In the normal lung, however, the distribution within the tissue is more uniform than the distribution of carbon in the pleura and there is not the macroscopic variation in different parts of the lobes, save at the hilus where the parenchyma is more pigmented on account of the greater accumulation in small lymphatic channels and nodes.

Furthermore, the unequal distribution of the pigment as it is observed upon the pleural surface has no direct relation to the deposition within the organ. The lack of pigment upon the interlobar surfaces and in the rib grooves is only a superficial condition and does not affect the deeper underlying lobules. The earliest deposits of coal pigment are to be looked for mainly in the perivascular lymphatics of the smaller branches of the pulmonary artery, subsequently, pigment appears in the regions of the small bronchi and venules. In all of these situations its presence becomes more marked with the increasing quantities of soot that are constantly inhaled.

As the accumulations of pigment gradually increase, they not only form lines along the septa of the lobules and the vascular channels, but nodular collections appear at the points of junction of the various lymph channels, where small receptacula are formed. These nodules becomes so prominent that they are readily felt by the finger and at times the course of the lymph channel can be detected by the feel.

Tissue changes may or may not accompany these larger depositions of pigment. In the majority of instances, however, a process of fibrosis, not accompanied by any inflammatory exudate, makes its appearance and surrounds each pigmentary nodule. These can be detected by the naked eye, while the larger ones, which are shot-like and gritty, are commonly spoken of as "anthracotic nodules."

In none of our specimens were we able to observe any uniform variation in the deposit of the anthracotic pigment within the lung of normal individuals. The greater quantity of pigment along the various channels has been

indicated above, but no unequal distribution of pigment has been observed which would in any way correspond to the unequal distribution beneath the pleura. True it is, however, that certain pathological processes in the lung tissue may modify the distribution of the pigment to a very great degee. We have, however, failed to find any evidence of excessive deposit in the deep tissues at the apex of the upper lobe. In fact, in our experience more pigment was found toward the hilus than at the periphery, regardless of the pleural distribution. Furthermore, the more marked areas of pigment deposit in the pleura are confined to this superficial layer and do not involve the underlying parts.

In several specimens of lungs from elderly individuals, who showed a moderate amount of emphysema in portions of the lobes near the surface, it was observed that an unequal distribution of the anthracotic pigment was present. Those lobules showing emphysema contained less pigment than elsewhere. This condition was not only apparent by the greater area occupied by the emphysematous tissues, but was real, in indicating less carbon in the affected tissues. When such emphysematous areas occupy the surface alveoli and when these lie upon the ridges of the costal markings, it is then found that the borders of the grooves contain less pigment than the surrounding areas. Thus the contention of Marchand and others that the ridges between the intercostal grooves accumulate less pigment may have its explanation in the presence of these emphysematous alveoli.

THE MODIFICATION OF ANTHRACOTIC DEPOSITS BY OTHER FACTORS.

It is evident from what we have said, that every individual has a greater or less quantity of carbon accumulate in the lungs, and that this accumulation varies in the normal lung according to the amount of carbon in the inspired air. With advancing age, the quantity of pigment continues to increase until a relative standard for

the community in which he resides is reached. This pigment in the normal lung becomes deposited, through the agency of phagocytic cells, within the lymphatics and its particular location beneath the pleura of the lung, is dependent upon the distribution of the lymph channels and the relationship of the opposed pleural surfaces which varies to some extent in all individuals. The distribution within the normal lung substance appears to be entirely determined by the circulation within the lymphatics.

Whereas under normal conditions we may look for certain common features in the anthracotic deposits in the lungs, there are also pathological processes which bring about a modification of the deposit. Thus we find that certain disturbances within the lung tissue have an effect of inducing greater deposits of pigment within localized areas. And it is probable that this new condition of excessive pigment deposit brings about further changes instituting a vicious circle.

A. LOCAL PLEURAL INFLAMMATION.

It is not an infrequent observation to find a greater quantity of pigment in the immediate vicinity of a band of pleural adhesions. By some it has been suggested that these adhesions are the result of the unusual deposit which leads to an excessive irritation in the surrounding tissues. When, however, we study the development of pleural adhesions we find that the fibrous bands in children show little or no difference in the deposit of pigment from other parts of the lung. With advancing age, however, the accumulation of carbon at the point of attachment of the adhesion to the lung becomes greater. A difference is noted, too, in the character of the adhesions, for those which have only a superficial attachment and do not induce a fibrosis of the neighboring lung tissue, show less deposit. It is obvious that we must differentiate those pigmentary processes associated with primary pleural adhesions from those that we associated with primary lung

disturbances (tuberculosis), in which adhesions may also be present. Of this latter type, we shall speak again.

The best example of pleural adhesions for study are those developing between two surfaces which are in constant frictional contact, as well as the bands of adhesions which sometimes follow fibrinous pleurisy in early life. Of the former type we meet with adhesions at the apex arising from a rib groove which, under ordinary circumstances, is non-pigmented. Here a firm band of adhesion binds a portion of the lung to the chest wall. The fibrous band not alone attaches itself to the surface of the visceral pleura but bands of tissue enter to a greater or lesser extent the fibrous layer of the lung covering and the interstitial septa, and alveolar walls. The fibrosis spreads diffusely through the tissue surrounding the blood vessels and encroaches upon the loose tissue of the lymphatics. Some of the lymph channels become completely obliterated, others are altered in their course.

It is probable that some of these bands of adhesions develop without the presence of an acute process and like the presence of milk spots of the heart, induce a progressive fibrosis which alters the relationship of the surrounding tissues. The pleura with its vascular tissues is altered to a sclerosed structure in which the lymph channels are reduced to mere clefts. In this condition not only is there a stasis of the fluid within these channels, but there is also a filtering out of the phagocytic cells which are constantly wandering from the alveoli towards the larger lymphatic system at the hilus. Gradually the accumulation of cells is sufficient to show the increased quantity of pigment within the part. It would appear according to Haythorn, that these migrating cells may live for a considerable period with the pigment within their protoplasm. Other phagocytes probably liberate their contents which become deposited in the interstices of the fibrous areas. It is probable that the liberated carbon remains in the clefts between the cells and does not enter fixed tissue cells.

If the opportunity for the absorption of carbon pigment from the alveoli be great, then the accumulation of this foreign material in the vicinity of adhesions becomes very marked. Nodules are formed which are hard and encroach upon the lung tissue. The lung alveoli are surrounded by a progressive fibrosis containing much carbon. It is more than probable, that when such excessive quantities of pigment are deposited that these again act as irritants, inducing greater adhesions. We do not believe that the inhalation of carbon in the normal lung will induce pleural adhesions unless some other factor within or upon the lung acts as a primary exciting cause. We have upon repeated occasions observed the lungs of mill workers and coal miners in whom the lung tissue had become intensely black through carbon deposit without there being any evidence of pleural adhesions.

We have never observed acute pleurisy to alter the deposit of pigment in the pleura. It has been observed that in acute inflammatory processes where the lymphatic channels of the pleura are filled with migrating and phagocytic cells that a considerable amount of pigment may be removed from the pleura to other parts. The exact bearing which this inflammatory migration might have upon the total pigment content could not be determined. The changes, however, were insufficient to produce any difference in the amount of pigment to be noted by the naked eye.

On the other hand, the chronic processes of the pleura not uncommonly had an effect similar to that observed associated with individual tags of adhesions. In cases where there were universal fibrous adhesions, the effect was not observed in the quantity of pigment deposit except where denser bands had developed. The diffuse and veil-like adhesions were without change in the vicinity of their attachment to the lung. A study of these indicated that the fibrous tissue of these adhesions had only a superficial attachment and did not involve the deep layer of the pleura. Where, however, the intensity of the chronic adhesive pleurisy was not uniform and where irregular bands

were attached to the lung substance at various points, here a more marked pigment deposit was prone to form.

In all instances where the more intense deposit of carbon pigment was associated with bands of adhesions, the process remained fairly superficial and localized. There was no invasion of the deeper parts by the continuous accumulation of pigment.

B. INFLAMMATION OF THE LUNG SUBSTANCE.

Much has been indicated to associate pneumokoniosis with acute and chronic respiratory diseases. As early as 1717, Ramazzini drew attention to certain air borne occupational diseases, and since then the greatest attention has been paid to diseases associated with inhalation of dust. Naturally, much interest has centered about the effect of inhaled dust of various kinds upon the lung and more particularly the relation, if any, that existed between these changes brought about by the deposits and inflammatory processes induced by bacteria. The manner in which this relationship was established has not been entirely clear. By many (Ascher and others), however, tuberculosis has been looked upon as a process secondary to the deposition of the dust.

On the other hand, the relative infrequency of pulmonary tuberculosis amongst those who are engaged in work associated with much coal dust, has been pointed out by a number of authors (Ogle, Sommerfeld, Hirt). In the statistics, tuberculosis appears rather rare among coal miners. It would, therefore, appear that the inhalation of coal dust does not predispose to tuberculosis. The explanation for this appears to depend upon the morphological characters of the dust particles. On the other hand, Kuborn, Villaret, Versois, and others believe that the continued contact with coal dust leads to a true immunity against tuberculosis. Racine believed that coal contains substances which are antiseptic and disinfectant and that this quality inhibits the growth of the tubercle bacilli, and Holman has shown similar disinfectant qualities in

soot. Another (Idel) believed that the porous coal dust absorbed the tubercle bacilli and rendered them inert, while Wainwright and Nichols thought the partially soluble calcium salts contained within the coal gave the animal body protection against these organisms. The indication that the presence of coal dust within lungs had a favorable effect upon respiratory diseases, led Guillot to use the inhalation of coal dust for therapeutic purposes. As early as 1793 Beddoes established a sanitarium near Bristol where he treated chronic diseases, as asthma and consumption by the inhalation of charcoal. The patients were placed in a dusting box where by mechanical means the charcoal was distributed into the air. However, it was later shown by Papasotiriu that coal dust had no influence upon the growth of the tubercle bacilli upon glycerine agar cultures, while Cornet was unable to protect animals against air borne tuberculosis by means of the inhalation of carbon dust. It has been indicated by Bartel and Neuman that anthracosis increases the virulence of tuberculosis in experimental animal infection.

It is more than probable that the infrequent presence of tuberculosis amongst those developing extensive anthracosis has its explanation in certain anatomical changes in the respiratory system and it is possible, as is indicated by Fraenkel and admitted by Racine, Wainwright and Nichols, that the infrequence of progressive tuberculosis among the coal miners is due to tissue obstruction of the lymphatic channels brought about by the anthracosis.

Ascher's observations that the extensive inhalation of smoke as well as soft coal increases the mortality in tuberculosis, is not in agreement with other general findings. It has, however, been shown by Hart that there is a difference in the composition of smoke particles and coal dust, and that the former contains some of the products of coal distillation. Again it has been shown by others that laborers engaged in atmospheres containing much coal dust, such as stokers, coal heavers, and chimney sweeps, are just as immune as coal miners to tuberculosis (Markel, Versois). Lewin found that 92.3 per cent. of chimney sweeps

who had followed this occupation for more than 10 years were free from respiratory diseases.

Our own observations have concerned themselves in determining the influence of the pigment upon the lung tissue as well as its relation to the tissue changes in acute and chronic processes within the lung. We can offer no statistics which show the relation which the pigment deposit has to the occurrence of infections of the lung. This study also deals with the effect of certain respiratory diseases upon the subsequent deposition of carbon pigment.

As it has been shown that the anthracotic material owes its presence to the activity of certain phagocytic cells it is evident that an interesting problem confronts us in determining what role similar cells stirred to activity by a bacterial irritant will have upon the foreign materials, as carbon pigment, which are already present in the interstitial tissues.

In the study of lung tissues showing acute pneumonia one is confronted with the picture of a lesser pigmentation in the areas involved in the pneumonia. The appearance is quite decided and a fairly sharp line of demarcation separates the pneumonic area from the more healthy tissues. Within the consolidated portion of the lung the carbon pigment is seen only in the more prominent nodular deposits while the pigment observed along the interlobular septa in the normal lung can no longer be traced. The diffuse pigmentary deposit in the alveolar walls is also overshadowed by the color of the exudate, be this grey or red. However, when viewing the lung from its pleural surface no change in the amount of pigment deposit is observed in the superficial portions.

Although a decided diminution of pigment within the consolidated area is apparent, the fact that pigment is actually removed from the tissue involved in the inflammatory process can not be demonstrated in the lung after its recovery from pneumonia. We have not been able to define the areas of consolidation after recovery from the disease, by the amount of pigment in the tissues.

It does seem, however, that some of the pigment in the lung tissue becomes dislodged during the active migration of cells. During the late stages of pneumonia, the lymphatic channels contain a greater number of pigment bearing cells than are observed in the uninvolved lung. It may be that, due to the stagnation of the lymphatic system in anthracosis, these pigment bearing cells do not have an opportunity of migrating from the pulmonary structures, but remain stagnant in the dilated lymph channels. The macroscopic appearances of a diminution of carbon pigment during the acute stages of the pneumonic process is more apparent than real and is due to the overshadowing of the normal lung structures by the cellular exudate of the inflammation.

On the other hand, we have repeatedly observed that in isolated areas of fibrosis of the lung where no evidence of tuberculosis was found that the amount of anthracotic pigment was much increased over that present elsewhere in the same lung. We can, however, hardly offer this as an indication that the sequel to an acute inflammatory process, ending in fibrosis is associated with an excessive pigmentary deposit. In a single case of well advanced organized pneumonia we observed some increase in the amount of macroscopic pigmentation within the fibrosed area as well as microscopic evidence of such increased deposit. Naturally, it is difficult to estimate the exact variations from the normal deposit in different parts of the same lung.

In no instance has the examination of pneumonic lungs shown that the presence of the anthracotic deposit has in any way modified the distribution of the acute process. It can not be shown that the more intensely pigmented tissues are more readily subject to pneumonia than the other less involved areas. It has, however, been suggested by Haythorn that aside from the local pigmentation in the vicinity of the individual air sacs, the anthracotic process of the lung has a definite effect upon the lymphatic system and particularly the lymphatic channels. These channels, which become narrowed and partly obstructed,

are less efficient for carrying off the debris which accumulates in the acute inflammatory process. This stagnation impairs the process of resolution with the result that proper repair of the lung following pneumonia does not take place. Conditions of unresolved pneumonia and gangrene of lung are more common in lungs with marked anthracosis than in the less affected organs.

We have in many examples made observations upon the anatomical relationship between the anthracotic deposit and tuberculosis. It is immediately apparent that in the discussion of such a relationship we must clearly define the type of tuberculosis. Naturally, the effect of the distribution of the tuberculous process upon the anthracotic deposit will be different in acute miliary tuberculosis than in chronic localized tuberculous lesions, and similarly the reverse relationship, if such exists, will also differ with the various forms in which one meets the tuberculous process. Individually both processes are dependent, for their local distribution, upon similar factors, the phagocytic activity of cells and the distribution of the lymphatics.

We have not been able to observe any direct bearing of the anthracotic process upon acute miliary tuberculosis, nor have we observed a greater tendency for the development of tuberculous lesions in the anthracotic areas than in other parts of the lung. In fact, lungs showing moderate anthracosis will have more acute miliary tubercles in the uninvolved portions of the lung than in the anthracotic nodules. Nevertheless, we have observed that in the later stages of the process when the miliary tubercles had advanced to larger and more definite caseating areas that the localized areas of anthracosis not infrequently had gray tuberculous centers. It is probable, therefore, that the absence of tubercles within anthracotic nodules during the acute stage of the infection is, in part, due to the intense pigmentation obliterating the early tuberculous focus. As the anthracotic deposit is associated directly with the course of the lymphatic streams and particularly with those surrounding the blood vessels, it is to be ex-

pected from what we know of the distribution of tuberculosis that many tubercles will develop along these systems, in spite of the presence of anthracosis. As the individual miliary tuberculous foci increase in size they gradually obliterate the anthracotic areas with the change from an intensely pigmented tissue to one showing numerous gray nodules of various sizes. With the increase in number, the tissue gradually loses the intensity of its pigmented appearance.

A still more marked loss of anthracotic pigment from the lung is seen in caseous pneumonia. Here, instead of having many small gray nodules gradually obliterating the pigment within the lung, we observe a diffuse gray caseous tissue whose light color is in strong contrast to the pigment in other portions of the lung. Only a moderate amount of pigment deposit is seen in the caseous area, and this pigment lies in the areas of former intense deposit. The gray color of the caseous areas not only represents the necrotic exudate within the alveoli, but also indicates tissue changes, first proliferative, later degenerative of the alveolar walls, and their contents. It is during the process of proliferation in the alveolar walls and lung trabeculae that the former pigmented cells are stimulated to proliferation and probably migration, which leads to a removal of the pigment in the particular area. What eventually becomes of the disturbed pigment in the lung tissue during the tuberculous process is difficult to say. In part, it finds its way towards the lymphatics at the hilus of the lung. In part, it may become removed by the destruction of the tissue and subsequent expectoration.

In the above processes, acute miliary tuberculosis and caseous pneumonia, it is evident that the anthracotic process has no influence in localizing the infection. We have, however, observed that miliary tuberculosis is more prone to develop into a chronic caseous miliary form in lungs presenting much pigmentation than in those not affected.

Quite a different outcome is observed in localized chronic caseous tuberculous foci. The early stages of the

tuberculous process simulates the lesions which we have just described. As the lesion enters the chronic stage one observes that instead of there being a diminution of pigment in the involved area that gradually and in direct proportion to the amount of fibrosis the pigment deposit increases. Thus the periphery of the lesion in which area the healing of the tuberculous mass is taking place, larger amounts of pigment are continuously laid down. We have never observed the macroscopic increase of pigment before the development of fibrosis in the tuberculous lesion. Eventually the fibrosed mass becomes intensely black and hard. These areas vary in size from a pea to a mass the size of a golf ball. When fully developed the tissue with its pigment deposit resembles in consistance and color a hard rubber ball.

We have observed all stages of these pigmented masses surrounding tuberculous foci and it is evident that the pigment deposit develops upon the tuberculous lesion. The extent of the pigmented area is entirely dependent upon the reaction in the tissue of the tuberculous focus, and this reaction is always of the development of fibrosis. Where a tuberculous process by progressive caseation has lead to cavity formation there is no excessive pigmentation in the vicinity of the cavity until repair by fibrosis has begun in its walls.

Microscopically, it has been shown that the same cells, which form the tubercle and which in themselves are phagocytic for tubercle bacilli, are also the cells most phagocytic for carbon pigment. Thus these cells, constituting the tubercle, are adapted for the localization of foreign dust particles, and being in excess of the number present in the normal parts of the lung, may bring about an anthracotic deposit, with the tubercle. However, by the time the pigment has accumulated in sufficiently large quantities to be recognized macroscopically, there has developed a secondary fibrosis inducing a vicious circle by obstructing new lymphatics and accumulating greater numbers of pigment laden cells.

TISSUE CHANGES INDUCED BY CARBON PIGMENT WITHIN THE LUNG.

To-day we have come to recognize that the term anthracosis does not refer alone to the presence of coal pigment within the lung, but also includes the tissue changes which accompany this deposit. As we have previously indicated we have come to recognize that the deposition of the carbon in the lung is brought about through the agency of phagocytic cells. It is not probable that inert carbon can enter the lung tissues by mechanical means alone. The contention of Klein, Sikorsky, Merkel and others that the physical characters of the foreign material is such that it may migrate between the cells in the alveolar walls without the assistance of wandering cells can no longer be supported. Hence it is evident that the very process of accumulating and carrying the pigment is a vital one and has to do with the cells arising from the pulmonary tissues. It has been shown that the number of cells acting as phagocytes found within the alveoli is proportionate to the quantity of pigment in the air sac and thus, too, the activity of the wandering cells is dependent upon the inhaled carbon. As the engulfed pigment is prone to remain fixed for considerable periods of time, it even being claimed by some that the phagocyted pigment remains permanently within the wandering cells, there is a progressive accumulation of these cells in the lymph spaces of the alveolar walls. Their direction is mainly towards the larger lymphatic system at the hilus of the lung, but it is also probable that these cells may not only lie inactive for varying periods of time, within the interstitial lymph spaces, but are still capable of returning to the air sacs to encumber themselves with still more foreign material.

How long these cells of an endothelial type are able to remain dormant but still living, is very difficult to say, yet it has been demonstrated in tissues that pigmented cells having every appearance of fixed connective tissue

when thoroughly analyzed and segregated from their surroundings were found to be endothelial cells.

It is the common observation to find a progressive accumulation of pigment bearing cells within the alveolar walls with advancing ages. As the cells increase in number within the lymph spaces the wall becomes thicker and the tissue has a more or less hyaline appearance between the aggregations of pigment granules. To a certain extent the increase in tissue is the result of a direct increase in the number of wandering cells. On the other hand, we have also been able to show that there is a definite increase in the connective tissues about the lymph channels with the laying down of heavy collagen strands.

With this fibrosis there is no increase in the elastic tissue, in fact, the areas of extensive change are poorer in elastic fibers than normal.

As we have previously indicated the distribution of the inhaled dust in the lung is quite uniform, save for its distribution in the lymphatics of the pleura. Some (Arnold and also Boer) maintain that the deposition of soot is considerably greater in the upper lobe. This has not been our finding, though at times a difference has been observed between the two lungs. The accumulation of dust to that extent which induces secondary fibrosis will thus give rise to a fairly uniform tissue change in all lobes of the lung. This is a common finding in as far as the lung tissue proper is concerned. It is probable that the fibrosis thus produced assists further with preventing a proper lymphatic circulation (Haythorn) and leads to the greater number of phagocytic cells becoming localized in the alveolar walls.

It is probable that the very nature of the phagocytic cells, being large and sluggish in activity, leads to their more ready localization in the lymph clefts than the more active leucocytes which deal with acute disturbances. If the normal functions of the endothelial phagocytic cells would be continuously carried out, it is improbable that as great a quantity of carbon would localize in the parenchymatous tissue of the lung, more of it finding its way

to the large lymphatics and lymph glands at the hilus. The very condition which is brought about by the obstruction of the lymph clefts and small channels as well as the blocking of the lymph sinuses in the nodes about the bronchi tends to increase the localization of the large phagocytes close to the alveoli from which they obtain their pigment. Thus the nature of the pigment phagocytosis and the localization within the lymphatic spaces tends to bring about a vicious circle which, when a certain degree of anthracosis has developed, permits of a still more rapid deposit of pigment in the alveolar walls. It is about in this stage of the condition that the developing fibrosis leads to structural changes which impair the function of the lung tissue.

Other than inducing a diffuse fibrosis within the lung, there are also the nodular fibrotic masses surrounding accumulations of pigment and pigment bearing cells at the junction of the lymphatic channels. The more common of these are the size of wheat grains. The fibrosis assumes a concentric arrangement enclosing pigment which to a great extent lies free but much of which is contained in the original phagocytic elements. Such nodules, however, may become much larger, forming isolated masses, three or four cms. in diameter. It is probable, however, that these larger masses arising in the lung tissue have had other factors superadded, leading to their unusual development. The consistence of these is that of hard black rubber. Where calcareous masses are found in the center of such nodules, the previous existence of tuberculosis is strongly suggested. This association of anthracosis with chronic tuberculosis we have discussed above.

ANTHRACOSIS AND EMPHYSEMA.

With extensive and diffuse development of pulmonary anthracosis in which tissue changes to a greater or less degree are developing, the activity of certain parts of the lung is impaired to such a degree that compensatory changes occur in other and more active parts. These com-

pensatory changes are mainly evidenced in the development of emphysema. It would be difficult to indicate the sequence of events in laborers or coal miners. Here, from the very nature of their work emphysema would readily occur. We may, however, observe emphysema in individuals with diffuse anthracosis whose work or whose thoracic condition would offer no explanation, for the compensatory expansion of certain lung areas. This we have on several occasions observed and we were unable to find an explanation save in the diminished functional activity in those portions of the lung with marked anthracosis and fibrosis. The development of the emphysema observed in the positions is seen under other conditions.

The apex and the anterior border of the upper lobe are usually most involved. A rather remarkable feature associated with this emphysema is the disappearance of the anthracotic pigment from the emphysematous area. Where the alveoli become usually distended the pigment gradually disappears until the tissues look quite white (pulmonary albinis). This has been commented upon by Beitzke and others.

From our observations it would appear that this loss of pigment from the lung is the result of the greater local activity during the process of development of the emphysematous areas. The condition would simulate the lack of pigment observed in the interlobar pleura where the massage of these areas by constant friction seems to drive the pigment bearing cells into the larger lymphatics. This is probably also the case during the development of the emphysema where the lung alveoli are acted upon by the greater air pressure having the effect of repeated compression and relaxation. Thus the air contained within the alveoli has the effect of massaging the alveolar walls and likewise of driving onward the cells containing the pigment. A similar effect would also be had upon the free pigment within the lymphatic spaces of the alveolar wall. In these emphaysematous areas the removal of the pigment is not associated with an inflammatory process assisted by leucocytic phagocytes.

QUANTITATIVE ESTIMATION OF CARBON IN LUNG.

As we have indicated, a fair estimate for comparison can be made of the carbon deposit by the naked eye appearance. The pleural deposit of carbon, although not directly related to the presence of pigment in the inner portions of the lung, is, nevertheless, a good guide to the quantity of foreign material in the organ. The pale gray or grayish-pink color of the lung of the rural inhabitant is readily distinguished from the mottle, streaked or slaty black tissues of the city dweller. Moreover, as we have indicated, the progressive increase of the carbon deposit, in the lungs of every citizen in manufacturing communities, can be recognized and grouped into the age periods by decades, when the individual has lived fairly constantly in the same district. Individuals of similar occupation are exposed to relatively equal amounts of atmospheric carbon, and their respiratory tissues receive similar quantities of carbon by inhalation. On the other hand, in communities where within short ranges of distance the atmospheric conditions differ, and with this the carbon content of the air is very unequal, the peoples living or working but short distances apart are subjected to diverse conditions, the one inhaling much larger quantities of soot than the other.

There are so many factors associated with the deposit of soot in the lungs of human individuals that it is impossible to make any general statement indicating the amounts for each. In truth, it is plain that those in smoky atmospheres have larger deposits, but we are often misled in our reference as to occupational influence. The millworker employed within the sheds in the manufacture of steel is often less exposed than his wife living within a quarter-mile range enveloped by the smoke clouds from the multitudinous stacks. The lungs of a peddler selling his wares to the foreign population of our smoke-laden valleys

were found to contain more carbon than those of the mill-hand (see table below).

As we feel convinced from our observations, that the intestinal route has little or no practical significance for the deposit of carbon in the lungs, it does not appear that the degree of cleanliness—particularly of the mouth—bears any relation to pulmonary anthracosis. Carbon particles once lodging upon the moist surfaces of the nose, mouth, pharynx and trachea, never assist in increasing the carbon of the lung. It is probable, as was shown by Haythorn's experiments, that only those carbon particles lying within the alveolar sacs can reach a permanent interstitial abode and that little if any carbon is phagocyted and carried into the tissues from the bronchi or bronchioles. Furthermore, it would appear, both from experimental and other observations, that the carbon reaching the lung alveoli is only a very small portion of the carbon content of the air as inspired, and this portion has reached the lung because it escaped contact with the moist mucous surfaces of the respiratory tract. Under the most trying circumstances of a smoky atmosphere we are amply protected by the sticky surfaces of tortuous tubes.

Difficult as it seems for carbon to reach the lungs, it appears equally difficult to dislodge the pigment when once it has been incorporated by the tissues. In fact, we may well believe that, save under very abnormal circumstances, carbon once within lung tissue remains for life, and hence each year we add that amount to our store as we may have been exposed to city smokes. To gain some accurate information of the quantitative deposit of carbon in the lungs an analysis was made of the tissues. Previous analyses have been made determining the quantity of iron, silicate, copper and other metalic deposits in the lungs of laborers.

Saito in a series of experiments estimated the quantity of dust inhaled from the air. Using measured quantities of dust (white lead) he determined the quantity taken up the animal when exposed to the dust-laden air. He observed that only 4 to 24 per cent. of dust entering the

nose was deposited in the respiratory organs, while the remaining quantity found its way to the intestine.

More recently Boer has made a relative quantitative estimation of the soot content of small portions of lung tissue. By his method, using only 3 ccm. of lung tissue, errors of calculation may possibly be great. He points out the error which would be obtained in comparing lung tissue of unequal density or consistence, as for example that of emphysema or oedma, and confined his examination to normal lung tissue. Here, too, much variation may be encountered, whether or not much pigmented pleura is included in the portion under examination. Care in selection of the tissue can not wholly rule out errors of serious import in the results. Furthermore, as the amount of carbon in these small portions of tissue was too small to weigh, he has used a colorimetric method suggested by Liefmann. The amount of carbon isolated from the lung examples was suspended in a mixture of oil and ether, and compared with a set of standard suspensions, prepared by suspending weighed quantities of naphthalin soot in the same vehicle. Such a colorimetric method cannot be relied upon, owing to the difference in the nature of the carbon in the lungs and naphthalin soot. Fresh soot has physical and chemical properties widely different from the carbon isolated from the lung by treatment with antiformin and alcohol. Isolated carbon from lungs has lost its flakiness and is quite granular, devoid of its phenols and acids. Its bulk is much less than the original soot from which it was derived, and in suspensions taken, weight for weight, it does not compare with the apparent mass of soot. It is furthermore, to be noted that in isolating the lung carbon, care must be taken to free the final product of its fat and foreign calcareous matter which tends to remain incorporated in the residue.

In our determinations we took an entire lung, dissected away the glands, large bronchi and adventitious tissue at the hilus, and minced the entire organ in a meat machine. The pulp was then divided among four half-liter flasks and to each was added enough of a seventy-

five per cent. solution of antiformin to well fill the flask. The flasks were placed in the incubator and repeatedly shaken for four to six days. Two hundred cubic centimeters of alcohol were then added to each flask and the mixture centrifugalized, the residue being collected and returned to clean flasks. These materials were again subject to fresh digestion with antiformin for a period of four days, recollected, washed and for a third time acted upon by antiformin. After again collecting the residue and washing it, it was treated with ten per cent. hydrochloric acid, repeatedly agitated and allowed to remain in contact for forty-eight hours. The residue now collected by the centrifuge and washed, was in turn treated with acid-alcohol and ether until the supernatent fluid showed no evidence of fat. The ether suspension was then allowed to evaporate to dryness, and the collected residue repeatedly washed with distilled water to rid it of any contained salts. The final product consisted of a pure black, fine powder, denser than the light, fluffy soot masses found in the air. Under the microscopic, angular carbon particles were alone present.

Case	Age	Occupation	Residence	Side	Quantity of Carbon
218	22	Laborer	Pittsburgh	Right	3.2
73	28	Peddler	"	Left	5.3
154	37	Laborer	"(6 yrs.)	Right	1.7
163	37	Housekeeper	"	Right	2.1
158	39	Clerk	"	Left	1.2
164	44	Housekeeper	"	Right	2.6
A-Q-8	47	Storekeeper	Ann Arbor	Right	0.145
A-Q-12	68	Laborer	"	Right	0.405
239	69	Carpenter	Pittsburgh	Right	2.81

(NOTE.—I am indebted to Prof. A. S. Warthin for the material from Ann Arbor.)

In our examination it is shown that the lungs of adult individuals resident in the Pittsburgh district have materially more carbon deposit than the lungs of the two individuals resident in a lesser manufacturing community.

Our number for comparison is very small, but is, nevertheless, suggestive of community characteristics. On account of the slow and rather tedious process in isolating the carbon in a pure form, only one lung was examined in each case, so that the total pulmonary content is about double of that indicated in the table. Furthermore, it is to be noted that the isolation of the carbon did not include that present in the peri-bronchial glands, where dense deposits are commonly found.

As was previously indicated the lungs showing marked anthracosis are decidedly heavier than normal organs, but it must not be inferred that the extra weight is due to the foreign dust in the lungs. From our analysis of the carbon pigment in the lung it is evident that no material increase in weight is obtained directly from this source. On the other hand, it is well shown that a relatively small quantity of carbon in the lung can induce massive fibroid changes which alter the architecture and increase the bulk.

SUMMARY.

Pulmonary anthracosis (not in coal miners) is distinctly an urban disease, and is proportionate to the smoke content of the air.

The soot is inspired and lodges in the pulmonary alveoli, from which it is carried by phagocytes into the lung tissue to become lodged in some portion of the pulmonary lymphatic system.

Although small quantities of carbon deposit in the lung may remain without harm, yet the quantity accumulating in the dweller of the larger cities has an accompanying greater or less fibrosis impairing the elasticity as well as altering the functional capacity of the organ.

The distribution of carbon is fairly uniform in the parenchyma of the different lobes, but there is a considerable variation in the distribution of the pleural deposit. The interlobar and diaphragmatic pleural surfaces show the least pigment. Moreover, less pigment is found in the grooves produced by the ribs or abnormal bands.

Carbon tends to accumulate at the nodal points of junction of the lymphatic channels. The cellular migration of carbon may lead to unusual accumulations in certain areas particularly well demonstrated in the deposit about chronic tuberculous lesions.

Carbon deposits by inducing fibrosis tend to encapsulate chronic tuberculous foci.

Pulmonary anthracosis by itself does not appear to stimulate the production of pleural adhesions.

The actual amount of carbon present in the lungs of different individuals varies considerably and is dependent, in part at least, to the age, occupation, residence and condition of the lungs (emphysema, collapse, tuberculosis).

Bibliography

Ascher. Der Einfluss des Rauches auf die Atmungsorgane. Stuttgart. 1905.

Aschoff. Sitzungs der. d. Ges. z. Beforder d. ges. Naturwissench zu Marburg. 1906.

Verhand. d. d. Path. Gesell. 1912. xv., p. 148.
Path. Anatomie. 1909. Jena 11. p. 271.

Arnold. Untersuchungen uber Staubinhalation und Staubmetastase. Leipsig. 1885.

Barthelmess. Inaug. Disert. Erlangen. 1855.

Beck. Uber Veraenderungen der Lunge. Wurzburg. 1895.

Beitzke. Anthracosis. Virchows Archiv. 1907. 187. p. 183.

Verhand. d.d. Path. Gesell. 1912. xv., p. 148.

Bartel. Weiner Klin. Wochensch. 1895. x., p. 10.

Bennecke. Beitrage zur Klinik der Tuberculose. 1906. vol. vi., p. 139.

Boer. Arch. of Hygiene. 1911. vol. 74. p. 73.

Calmette and Guerin. Annales de l'Institut Pasteur. 1905. xix., p. 601.

Cornet. Die Tuberculose. Nathnagels Handbuch. 1899. p. 240.

Fraenkel. Deutsch. med. Wochensch. 1895. x., p. 10.

Gregory. Edinburg Med. & Surg. Journ. 1831. xxxvi., p. 389.

Guillet. Archiv. gener. de Medicin. 1845. vol. vii., p. 1.

Haythorn. Journ. Med. Research. 1913. vol. xxix., p. 259.

Henele. ref. Beck. Inaug. Dissert. Wurzburg. 1895.

Hirt. Die Krankheiten der Arbeiter. 1871. Breslau.

Holman. Amer. Journ. Public Health. 1913. iii., p. 1210.

Hart. Lubarsch Ostertag. 1910. Abt. 1, p. 405.

Ins. Archiv. f. expl. Path. 1876.

Idel. Inaug. Diss. Wurtzburg. 1889.

Koschlakoff. Virchows Archiv. 1866. Bd. 35.

Knauff. Virchows Archiv. 1878. Bd. 73.

Konradi. Untersuchungen uber den Schwarzen Farbstoff in Lunge. Dorpat. 1869.

Kuborn. Presse. Medic. 1862. No. 27.

Lewin. Die Inhalationstherapie. Berlin. 1865.

Montgomery. Jour. Med. Research. 1910. xxxiii., p. 3.

Marchand. Verhand. d. d. Path. Gesell. 1912. xv., p. 148.

Merkel. Deutsch. Arch. f. Klin. Med. 1871. viii., p. 210.

Neuman. Wiener Klin. Wochensch. 1906.

Nichols and Wainwright. Amer. Jour. Med. Sc. 1905. Sept.

Orsos. Verhand. d. d. Path. Gesell. 1912. xv., p. 148.

Ogle. Internat. Kongr. f. Hygiene & Demographie. London. 1891.

Pearson. Philosoph. Trans. London, 1813. 11, p. 120.

Peiser. Cent. f. Alleg. Path. 1908. xix., p. 817.

Papasotiriu. Munchn. Med. Wochenschr. 1901. No. 13.

Ramazzini. De morbis. artificum diatribe. 1717.

Rindfleisch. Lehrbuch. der. Path. Gewebelehre. 1878. p. 392.

Racine. Vierteljahr. Sch. f. Gerichl. Med. 1884. vol. 40, p. 300.

Saito. Arch. f. Hygiene. 1912. lxxv., p. 134.

Stratton. Edinburgh Med. & Surg. Jour. 1838. vol. 49, p. 490.

Schottelius. Virchows Archiv. 1878. vol. 73.

Sikorsky. Cent. f. Med. Wissench. 1870. N. 52.

Slavjansky. Virchows Archiv. 1869. vol. 48, p. 826.

Schmorl. Munchn. Med. Wochenschr. 1901. vol. 48, p. 1995.

Sommerfeld. Die Schwindsucht der Arbeiter, ihre Ursache. Berlin. 1891.

Schmitt. Lubarsch Ostertag. 1894. Abt. 11. p. 16.

Thompson. Med. Chir. Trans. London. 1837. xx., p. 230.

Traube. Deutsch Klinik. Berlin. 1860. xii., 475.

Virchow. Virchows Archiv. 1847. B. I. p. 40.
Virchows Archiv. 1866. B. 35.

Versois. Schmidts Jahrb. B. 100.

Villeret. Cas rare d'Anthracosis. Paris. 1862.

Zenker. Deutsch. Archiv. f. Klin Med. B. I. p. 116.

The Bacteriology of Soot*

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The study of soot from a bacteriological standpoint has received but scant attention from the numerous investigators of the problems of the smoke nuisance.

Needless to say the subject has not been left out of the discussions; in fact, it is rather frequently mentioned in a more or less general way. As far as I have been able to ascertain, however, there has been but little definite work on the Bacteriology of Soot as discussed in this paper.

In an article in the *Revue Industrielle* (1), is found the question, "Are we going to learn one of these days that smoke, thanks to its antiseptic properties, contributes to making the atmosphere healthy?" Glinzer (2) refers to the fact that the particles contained in soot possess excellent germicidal and disinfecting qualities. Racine (3) in a discussion on the "Relation of Emphysema and Tuberculosis to coal-lungs in miners," believes from his own observations that anthracosis of the lungs acts as a protective influence against tuberculosis and that the only correct view is that coal has a great disinfecting power and that the conserving action of the coal dust is, perhaps, to be explained "from its action as a hinderance to the growth of microorganisms such as the bacilli of tuberculosis." We were unable to find in the literature any original work upon which these conclusions could be founded. Percy Frankland (4) shows that fogs do not tend to con-

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centrate or nurture bacteria, for he found there were remarkably few bacteria in London air during a fog. On the other side of the question, Russel (5) includes the increased number of bacteria in a list of contributory causes of the high death rates during fogs. Sir William Ramsay (6) first brought forward the theory that smoke by directly absorbing light, through the action of clouds and fogs, which are particularly fitted to absorb the blue, the violet and ultra-violet rays, which are the germicidal rays in light, contributes to the development and increase of bacteria, pathogenic as well as others, in the atmosphere. Liefmann (7) also believes that under the above conditions "bacteria, especially the pathogenic ones, are permitted to thrive." In passing it may be mentioned that there is no evidence that bacteria ever grow in the air. They are carried into it from a variety of sources, are continually falling out of it by gravitation, being carried out by rains, or destroyed in a number of ways. The effect of the gases in the air upon bacteria is another problem, although it is closely related to the one under discussion. The contamination of the air of our cities with sulphur dioxide is well known (8); also that surphur dioxide in the air acts as a disinfectant, the necessary proportion being not less than 92 grams of sulphur per cubic meter (9). Cohen and Ruston in "Smoke—A study of town air," have devoted some space to the consideration of the effects of acid rains upon the bacteria of the soil. They point out that the greatest reduction takes place in the numbers and the activity of the nitrifying or nitrate producing bacteria, there being also a marked inhibitory effect on the nitrogen fixing organisms.

In our investigations we examined the air of Pittsburgh to determine the numbers and distribution of bacteria in different parts of the city and to ascertain, if possible, if any relation exist between the bacterial count and the smoke conditions.

For this purpose twenty glass evaporating dishes seven inches in diameter filled with agar-agar were ex-

MAP OF PITTSBURGH.

Showing Wards and Location of Agar Plates Exposed.

posed for from five to fifteen minutes in various districts. Notes were taken of the presence or absence of smoke, the wind direction, the condition of the earth, whether moist or dry, and time of exposure. The number of colonies developing in forty-eight hours at 37° C. varied within very wide limits and it was found impossible to discover any influence of the smoke content of the air on the bacterial count. The difficulty of attempting to draw any definite conclusions from these experiments will be patent to everyone, the factors which influence the bacterial content of the air being so numerous and so variable as—rain, moist or dry soil, winds, sunlight, and other conditions. The map of the city shows the points where plates were exposed and Table I gives our findings in a condensed form.

One quite striking observation, however, was made and that was the comparative infrequency of the development of colonies around the soot particles. Rough microscopic counts were made of the total number of visible soot particles and of those which were infected, as indicated by a black speck in the center of the colony. Out of about 500 counted, 100 showed growth. Many of these particles which we counted as soot, were, in all probability, specks of dust from other sources, so that our proportion of infected soot particles is too high. In several of the plates the soot particles were too numerous to count and around the majority of these no growth was to be noted. This was particularly true in the case of Plates 9 and 10, where the atmosphere was decidedly smoky. Considering the sources from which bacteria enter the air this is not to be wondered at. The smoke as it leaves the chimney is free from bacteria and apparently is not a favorable nidus for their collection from the air into which it is poured.

The common types of aerobic Gram positive spore-bearing bacteria and chromogenic organisms were isolated on our plates. B. Coli Communis and Communior, B. Alkaligenes Fecalis, B. Proteus Vulgaris, B. Pyocyaneus,

Plate Number See Map.	Time of Exposure in Minutes.	Weather.	Wind.	Smoke.	Count.	Notes.
1	15	Cloudy, rain 3 hrs. before.	N. W	None	75	B. Coli Communis isolated.
2	15	Cloudy, rain 3 hrs. before.	N. W.	"	53	
3	5	Cool, cloudy; soil damp.	W.	"	15	
4	10	Cool, cloudy; soil damp.	W.	"	68	
5	15	Clear, warm; soil damp.	W.	Blowing towards plates	26	B. Coli Communis. B. Coli Communior.
6	10	Bright sunshine; soil damp.	W.	but high.	43	B. Alkaligenes. Fecalis.
7	10	Cloudy, sultry; soil dry.	S.	Blowing towards plates.	51	5 soot particles, no growth.
8	15	Cloudy, sultry; soil dry.	S.		144	15 soot particles, 3 infected.
9	10	Partly cloudy.	S.	Smoky.	60	Soot particles very numerous, 1 infected.
10	15	Warm, dry.	S.	"	108	Soot particles very numerous, 3 infected.
11	5	Clear, warm, sultry; soil dry.	S.	Hanging low.	18	Several soot particles, no growth.
12	15	Clear, warm, sultry; soil dry.	S.	Same.	46	Few soot particles; 1 infected. B. Proteus Vulgaris.
13	10	Bright, hot, dry, sultry.	S.	Valley smoke not dense	59	No soot particles. Nocardia.
14	15	Threatening rain.	S.	near plates.	—	Fly contaminated plate.
15	5	Threatening rain; rain 1 hr. before.	E.	Blowing towards plates.	116	B. Pyocyaneus. Soot particles 130; 14 infected.
16	8	Threatening rain; rain began.	E.	Same.	124	Soot particles 174; 17 infected.
17	10	Threatening rain; 2 hrs. before.	E.	Air smoky.	19	No soot particles.
18	10	Hot, clear, dry, dusty.	S. W.	Dirty, dusty, smoky.	196	Soot particles 11; 4 infected.
19	10	Hot, clear, dry, dusty.	S. W.	Valley smoky, clear near plates.	160	Soot particles 19; 11 infected; air nocardia.
20	5	Hot, clear, dry, dusty.	S. W.	Blowing towards plates.	131	Soot particles 72; 30 infected.
21	10	Hot, clear, dry, dusty.	S. W.	Blowing towards plates.	128	Soot particles 56; 14 infected.

and others, presumably from manure dust, and a few examples of air nocardia were also found.

Our next problem was to determine the action of soot on the growth of bacteria. For this purpose a quantity of soot was obtained from the special chimney in the Department of Industrial Research.

The partial analysis was as follows:

Tar,	3.84%
Ash	1.19%
Fixed Carbon	94.97%

This specimen was used throughout the experiments.

The analyses of specimens of soot collected from the air in different parts of the city showed some wide variations.

Woods Run.	
Tar	0.82%
Ash	62.6%
Fixed Carbon	36.58%
State Hall.	
Tar	0.36%
Ash	66.68%
Fixed Carbon	32.96%

This soot, even after standing around the laboratory in an ordinary cardboard box frequently open to the air, was shown by experiment to be almost free from bacteria. Five agar plates dusted with small quantities of this unsterilized soot failed to show growth with the exception of one colony on one plate. The soot was now added in varying amounts to test tubes of ordinary broth. No growth occurred. However, when larger quantities of this same soot were added to 150 cc. of broth in flasks, growth of bacteria did appear. The organisms developing being of the B. Subtilis group.

Experiments were next undertaken to determine the bactericidal action, suggested by the above observations.

Efforts were made to determine any difference in growth in flasks of broth containing soot from other control flasks without soot. Dilution and plating methods were employed but the mechanical interference of the soot particles in the one case ruled out the method as one without even approximate exactness. The counts, however, were uniformly lower in the soot broth than in the control. A series of agar plates, one having the surface sifted with soot, the other free, were next exposed in the laboratory and counts made of the colonies from the aerial contamination. No marked difference in the number of colonies was found.

The following series of experiments have, however, given us quite definite results indicating a very marked bactericidal action of soot. Five grams of soot were thoroughly mixed with 100 cc. of broth 0.6 acid to phenolphthalein. After autoclaving, the mixture was allowed to stand several days. The soot particles were then filtered out and the filtrate after sterilization was seeded with B. Typhosus. A control flask was inoculated at the same time. No growth occurred in the soot-treated broth while a good growth developed in the control.

A second experiment was carried out. The broth after treatment with soot as before was tubed and inoculated with a series of organisms. Controls of plain broth from the same batch were used for comparison. Fresh twenty-four hour cultures were employed. The results are seen in Table II.

The soot broth tubes which showed no growth after seeding and incubation, did not show any growth on further transfers with the exception of B. Subtilis. The spores in this case had most probably withstood the action of the bactericidal substance. B. Coli Communior and B. Indicus showed much less growth in the soot-treated broth than in the control. B. Pyocyaneus was apparently unaffected while B. Paratyphosus (Achard) and B. Proteus Vulgaris showed definite agglutination in the soot-treated broth.

TABLE II.

Culture.	Soot Broth.	Control.
Staph. Albus	—	+ + +
Staph. Pyog. Aureus	—	+ + +
B. Coli Communis	+ +	+ + +
B. Coli Communior	+ +	+ + +
B. Mucosus Capsulatus	+	+ + +
B. Acidi Lactici	—	+ + +
B. Typhosus (3)	—	+ + +
B. Typhosus (90)	+	+ + +
B. Paratyphosus B.	—	+ + +
B. Paratyphosus Achard	+ +	+ + +
B. Dysenteriæ (Flexner)	—	+ + +
B. Pseudodysenteriæ	—	+ + +
B. Iliacus	—	+ + +
B. Proteus Vulgaris	+ + +	+ + +
B. Alkaligenes Fecalis	—	+ + +
V. Cholera	—	+ + +
B. Pyocyaneus	+ + +	+ + +
B. Indicus	+	+ + +
B. Subtilis	—	+ + +
B. Mesentericus	—	+ + +
B. Xerosis	—	+ + +
B. Diphtheriæ (230)	—	+ + +

(The intensity of the growth is indicated by the number of + signs.)

We believed that this inhibitory and bactericidal action was due to the phenols contained in the soot. The soot-treated broth gave a marked reaction for phenols by Millon's reagent while the control broth gave a negative reaction. After testing the acidity, phenolphthalein being used as the indicator, it was found that the soot treated broth gave a difference of 1.7 per cent. acid over the untreated broth, the acidity of which was 0.6 per cent. To determine whether this acidity was the potent inhibitory factor we carried out the following tests.

A new lot of broth was prepared and the reaction made 0.6 per cent. acid. To 500 cc. of this broth 25 grams of soot were added. The mixture was thoroughly shaken, autoclaved and allowed to stand for two days, with repeated shaking and finally filtered. The reaction titrated against N/20 sodium hydrate expressed in terms of hydrochloric acid was found to be 2.2 per cent. acid. One-

half of the broth was used at this acidity, 2.2 per cent. the other half being reduced to 0.6 per cent. by addition of sodium hydrate. Half of the untreated control broth had its acidity raised to 2.2 per cent. acid with hydrochloric acid. The four lots were then tubed, sterilized and inoculated with a number of organisms from twenty-four hour agar slants. After an incubation of seventy-two hours, the results obtained were as shown in Table III.

From this it will be seen that we must consider at least two factors in the antiseptic action of the soluble content of soot in broth. The effect of the increased acidity which is seen most markedly in the cultures of the cholera vibrio where practically no growth took place in either of the acid broths is of great importance. The cholera organism is shown to be particularly sensitive to the presence of acid and for its cultivation an alkaline or neutral medium must be employed. Schroeder (10) has shown that the Vibrio choleræ is killed after an exposure of five hours to peat dust. It is especially effective, he says, if the peat be of an acid reaction.

The growth of B. Anthracis and B. Subtilis is also inhibited in the broths of high acidity, while B. Alkaligenes Fecalis and B. Iliacus, are definitely affected. It is interesting to note the effect of the acidity in interfering with the production of the coloring matter in the cultures of B. Pyocyaneus. Russel, Cohen and Ruston, and others point out the variable acid content of soot. On the other hand we notice, that, independently of the acidity, the soot-treated broth exercises a marked interference with growth. This is to be seen with B. Indicus, one strain of B. Coli Communis, B. Typhosus, and in B. Paratyphosus to a marked degree. The two latter organisms are not generally affected by small amounts of acid, and are, therefore, valuable in testing out this second bactericidal effect of the soluble parts of the soot.

Soot probably has the power of absorbing many gases from the air especially those associated with the combustion of coal.

The next problem was to study the effect, if any, of soot on the destruction of bacteria in the process of dessication.

TABLE III.

Culture.	Control Broth. 2.2 Acid.	Soot Broth. 2.2 Acid.	Control Broth. 0.6 Acid.	Soot Broth. 0.6 Acid.
B. Indicus	Heavy cloud	Trace of cloud.	Heavy cloud, heavy precipitate.	Slight cloud.
B. Pyocyaneus..	Heavy cloud, powdery scum, trace of green color.	Same, no green color.	Same, bright green on top.	Heavy cloud, thick compact scum, green color throughout.
B. Anthracis.....	Clear, no precipitate.	Clear, no precipitate.	Clear, abundant precipitate.	Clear, slight precipitate.
B. Subtilis	No growth.	No growth.	Heavy ring, heavy precipitate, granular cloud.	Ring on glass, precipitate.
B. Coli Communis (52) and (14).....	Marked cloud.	Slight cloud.	Marked cloud.	Slight cloud.
B. Coli Communis from urine (780) ..	Fair cloud.	Same.	Same.	Same.
B. Typhosus	Cloud.	Slight cloud.	Heaviest cloud.	Faint cloud.
B. Paratyphosus B.	Marked cloud.	Faint cloud.	Marked cloud.	Faint cloud.
B. Proteus Vulgaris	Heavy cloud, no scum.	Heavy cloud, scum.	Very heavy cloud, no scum.	Very heavy cloud, scum.
B. Iliacus.....	Slight cloud.	Fair cloud.	Heaviest cloud.	Heavy cloud and ring.
B. Alkaligenes Fecalis	Faint cloud.	Clear.	Marked cloud, scum.	Slight cloud.
V. Cholera.....	Clear.	Trace of cloud.	Cloud.	Cloud.

Dessication is one of the most valuable natural means of disinfection and many organisms succumb to its effects very rapidly as the gonococcus, the B. Influenza, the meningococcus and others. The micro-organisms of the air, on our streets, and in our houses are continually being destroyed by this process and we have been able, we believe, to show that soot increases very decidedly, this bactericidal action.

TABLE IV.

Exposure.	Influence of Soot on B. Indicus.		
	Control. Cover Glass. Dipped in Broth and Dried.	Cover Glass I. Dipped in Soot while Moist, then Dried.	Cover Glass II. dried as in Control. Buried in Soot.
1 day.....	+	+	+
2 "	+	+	+
3 "	+	+	+
4 "	+	—	+
5 "	+	—	+
7 "	+	—	—
8 "	+	—	+

(The + sign indicates that growth was obtained in the test cultures.)

TABLE V.

Exposure.	Influence of Soot on B. Typhosus and Streptococcus Fecalis.			
	Culture.	Control Dipped in Broth and Dried.	Cover Glass I. Dipped in Soot while Moist, then Dried.	Cover Glass II. Dried as in Control. Buried in Soot
2 days.....	B. Typhosus	+	—	
	S. Fecalis	+	+	
3 "	B. Typhosus	+	—	+
	S. Fecalis	—	—	—
	B. Typhosus	+	—	+
	S. Fecalis	+	—	+
4 "	B. Typhosus	+	—	+
	S. Fecalis	—	—	+
5 "	B. Typhosus	+	—	+
	S. Fecalis	+	—	—
6 "	B. Typhosus	+	—	+
	S. Fecalis	—	—	—
9 "	B. Typhosus	+	—	+
	S. Fecalis	—	—	—
10 "	B. Typhosus	+	—	—
	S. Fecalis	+	—	—

The technique of our experiments was as follows: Fresh twenty-four hour broth cultures of the organisms to be tested were prepared. Small sterile cover glasses were thoroughly moistened by dipping them into the cultures. These were dried under sterile precautions for sixteen to twenty-four hours over caustic soda. They were then transferred to sterile petri dishes and used as controls.

Cover glasses indicated under the heading I in the table, were dipped into the broth cultures and then into sterile soot and finally dried as in the control. Cover glasses under heading II in the table were prepared in the same way and at the same time as the control, and when dry were packed in sterile soot. From time to time these cover glasses were dropped into tubes of dextrose broth to test the viability of the organisms.

It will be noted that the failure of growth is shown very clearly in the case where the moist cover glass was dipped in soot and then dried. In the majority of cases those cover glasses which were first dried and then packed in soot failed to show growth much later than the above, but usually before the control. This is well shown in Table V.

The long period that the Streptococcus Fecalis, 174 days, and Staphylococcus Pyogenes Aureus, 225 days, remained viable under these abnormal conditions is quite remarkable. (See Table VI.)

In Table VII is shown the peculiar behavior of yeast. It survived longest, 41 days, on the cover glasses dipped in soot while still moist, the conditions under which all the other organisms tested rapidly died out. Marshall (11) quotes Hansen as stating that compressed beer yeast mixed and dried with charcoal kept as long as ten years. The marked resistance of the yeast organism to the action of acids is also of importance in this connection.

The great natural disinfectant of the atmosphere and our surroundings is the bactericidal action of the sun's rays. Direct sunlight is most destructive and its activity

TABLE VI.

Time of Exposure	Incubation Period	Influence of Soot on B. Coli, B. Typhi, Streptococcus Fecalis and Staphylococcus Aureus.			
		Culture	Control. Dipped in Broth and Dried	Cover Glass I. Dipped in Soot while Moist, then Dried	Cover Glass II. Dried as in Control. Buried in Soot
2 days	24 hours	B. Coli	+	+	
		B. Typhosus	+	+	
		Strep. Fecalis	+	+	
		Staph. Pyog. Aureus	+	+	
5 "	24 "	B. Coli	+	—	+
		B. Typhosus	+	+	+
		Strep. Fecalis	+	—	+
		Staph. Pyog. Aureus	+	—	+
	48 "	B. Coli	+	+	+
		B. Typhosus	+	+	+
		Strep. Fecalis	+	—	+
		Staph. Pyog. Aureus	+	—	+
6 "	24 "	Strep. Fecalis	+	+	+
		Staph. Pyog. Aureus	+	—	+
7 "	24 "	B. Typhosus	+	—	+
		Strep. Fecalis	+	+	+
19 "	24 "	B. Coli.	+	—	+
		B. Typhosus	+	—	—
		Strep. Fecalis	+	—	+
		Staph. Pyog. Aureus	+	—	+
	48 "	B. Coli	+	—	+
		B. Typhosus	+	—	—
		Strep. Fecalis	+	+	+
		Staph. Pyog. Aureus	+	—	+
34 "	24 "	B. Coli	+	—	—
		B. Typhosus	+	—	—
		Strep. Fecalis	+	—	+
		Staph. Pyog. Aureus	+	—	+
	48 "	B. Coli	+	—	—
		B. Typhosus	+	—	+
		Strep. Fecalis	+	—	+
		Staph. Pyog. Aureus	+	—	+

TABLE VI (*Continued*).

Time of Exposure	Incubation Period	Influence of Soot on B. Coli, B. Typhi, Streptococcus Fecalis and Staphylococcus Aureus.			
		Culture	Control. Dipped in Broth and Dried	Cover Glass I. Dipped in Soot while Moist, then Dried	Cover Glass II. Dried as in Control. Buried in Soot
62 "	24 "	B. Coli	+	—	—
		B. Typhosus	—	—	—
		Strep. Fecalis	—	—	—
		Staph. Pyog. Aureus	—	—	—
	48 "	B. Coli	+	—	—
		B. Typhosus	—	—	—
		Strep. Fecalis	+	—	+
		Staph. Pyog. Aureus	+	—	+
174 "	24 "	Strep. Fecalis		—	+
		Staph. Pyog. Aureus	—	—	+
225 "	24 "	Staph. Pyog. Aureus	+	—	—
247 "	24 "	Staph. Pyog. Aureus	—	—	—

upon bacterial life depends directly on the amount of moisture and dust in the air.

Smoke, in contributing very great numbers of minute particles to the air adds to the conditions favoring fogs and clouds. Smoke, fogs, and clouds, all absorb more or less, the blue, the violet, and ultra-violet rays of the sunlight. This is well seen in the familiar red sun of a smoky atmosphere. These particular rays, which are absorbed, give the important bactericidal action to the sunlight. Most of the above facts have been amply proved by other experiments and it was not considered advisable to repeat them. We have, however, made a few observations on the protective action of soot for bacteria.

The technique of our experiments was briefly as follows: Agar-agar, well seeded with the test organism, was poured into petri dishes. Soot was sifted over one half

of the cover of the petri dish while the other half remained free. These plates were then exposed to the sunlight for definite periods, then incubated and the results read.

A number of plates, seeded with Staphylococcus Aureus, were exposed to sunlight for varying periods every day for two weeks. The results obtained are shown in Table IX. Notwithstanding the irregularity of these results the protection, afforded by the soot in the air and clouds against the action of the sun's rays, is, I believe, clearly shown.

There are a number of very interesting questions which arise as a result of these experiments. It has been shown that soot in contact with bacteria has very decided bactericidal properties. These properties are also demonstrated in the soluble content of the soot. The soot particles from the air as they fell on our plates were generally sterile. This may be due to the solution of the bactericidal substances of the soot by the moisture on our plates, with the consequent destruction of any bacteria adherent to the soot. Or the moisture condensing around the soot as it does in the formation of fogs and clouds, may have acted in the same way before it was collected on our plates. The third possibility is that the majority of the soot particles never came in contact with bacteria after leaving the chimney, at which time they were, of course, bacteria free.

That this disinfectant substance requires moisture in order to have its most powerful effect is well shown in the drying experiments where the organisms treated with soot, while still moist, succumbed very much sooner than the others. There is also the possibility that the soot absorbs moisture from the bacteria and hastens thereby its death by thorough drying. This was suggested in the experiment in which dried organisms were buried in soot and frequently killed off earlier than in the control.

TABLE VII.

Time of Exposure	Incubation Period	Influence of Soot on B. Diphtheriæ and Yeast (Saccharomyces). Culture	Control. Dipped in Broth, then Dried	Cover Glass I. Dipped in Soot while Moist. Dried	Cover Glass II. Dried as in Control. Buried in Soot
3 days	48 hours	B. Diphtheriæ	+	—	
		Yeast	+	+	
	72 "	B. Diphtheriæ	+	+	
		Yeast	+	+	
7 "	24 "	B. Diphtheriæ	+	—	+
		Yeast	—	—	—
	48 "	B. Diphtheriæ	+	—	+
		Yeast	+	+	+
	72 "	B. Diphtheriæ	+	+	+
		Yeast	+	+	+
16 "	48 "	B. Diphtheriæ	+	—	+
		Yeast	—	+	+
	72 "	B. Diphtheriæ	+	+	+
		Yeast	—	+	+
27 "	72 "	B. Diphtheriæ	+	—	+
		Yeast	—	+	—
38 "	48 "	B. Diphtheriæ	+	—	+
	5 days	B. Diphtheriæ	+	+	+
41 "	48 hours	B. Diphtheriæ	+	—	+
		Yeast	—	+	—
77 "	72 "	B. Diphtheriæ	—	—	—
		Yeast	—	—	—

That the conditions, similar to those of our experiments, are to be found in every smoky city, will be evident to any one who has noticed the black, smeary deposit of soot on damp days where the concentration of soluble substances from the soot in crevices and corners must be very high. We have found, as others have demonstrated, that the effect of soot in fogs and clouds in diminishing the action of the run's rays on bacteria, is quite definite. The relative importance of the protective qualities of soot against sunlight to the bactericidal effect of the constituents of soot remains an open question.

There are, therefore, two divergent results brought about by the presence of soot in our atmosphere and upon the earth's surface. One of these is beneficial and the other harmful to the life of vegetable micro-organisms, and from the bacteriological and hygienic point of view may serve as a direct aid in propagating or preventing the spread of infectious disease.

TABLE VIII.

Organism Tested.	Inhibition of Solar Bactericidal Activity.			
	Time of Exposure	Incubation	Soot Covered Half.	Unprotected Half.
Strep. Fecalis	5 mins.	24 hrs.	Few colonies	None
	5 "	48 "	Many "	Many colonies
	15 "	48 "	Few "	No growth
	30 "	48 "	Few "	Many colonies
	45 "	48 "	Few "	Many "
	45 "	48 "	Many "	Few "
	120 "	48 "	Many "	Few "
B. Indicus	10 "	48 "	Many "	Many "
Staph. Aureus	10 "	48 "	Many "	Many "
	30 "	48 "	Many "	Many "
	45 "	48 "	Many "	Few "
	75 "	48 "	No "	No "
B. Mesentericus	15 "	48 "	Few "	Few "
	30 "	48 "	Few "	Few "
	45 "	48 "	Few "	Few "
	60 "	48 "	Few "	Few "
	75 "	48 "	Few "	Few "
	180 "	48 "	Few "	Few "

General Conclusions.

1. Soot has a definite bactericidal action on bacteria, due either to the absorption of moisture from the organisms or more probably, to the action of its contained germicidal acids and phenols.

2. Soot as it exists in the air does not form a favorable nidus for the collection and distribution of bacteria.

3. Broth and other fluids treated with soot have conferred upon them a decided germicidal power.

TABLE IX.

(Inhibition of Solar Bactericidal Activity by Smoke. Test Organism Staphylococcus Pyogenes Aureus.)

	Sun.	Clouds.	Smoke.	Result.	Note.
1	Bright		Clouds low.	Killed 1 hr.	
2	Dull	Present.	Clouds passing over sun.	Slight difference in 1 hr.	
3	Bright	Passing across sun.	None.	Killed 1 hr.	24 hr. incub.
4	Bright	Same.	"	Lessened in 15 and 30 mins. in proportion.	
5	Hidden, dull	Snowing in morning. Present.	"	Killed in 1½ hrs.	
6	Bright	Thin and white.	Around horizon.	Not killed in 1 hr., lessened.	
7	Clear	—	Haze around horizon.	Same.	
8	Bright and clear	Very few.	—	35 mins., much lessened; 1 hr., almost all killed.	
9	Bright and variable ..	Cloudy.	—	Not killed in 1 hr.	
10	Bright	None.	Much, low down.	Not killed in 1 hr.	Around edge.
11	Dull, foggy	Foggy.	—	Little change in 1½ hrs.	
12	Dull	Many, raining.	Much.	Almost all killed in 1½ hrs.	
13	Variable, free, dull	Variable.	—	Little change 1¼ hrs.	
14	Bright	None.	Haze.	Killed in 1 hr.	

4. This germicidal action is due not only to the acids contained in the soot but also to some other agent, probably some of the phenols.

5. Soot, as it occurs in smoke, clouds, fogs, and as a non-transparent covering for our streets and houses, protect micro-organisms from the destructive action of the sunlight.

I wish to express my thanks to Mr. C. H. Marcy for his valuable assistance in the early part of the work, more

especially in the study of the bacteriology of the air of Pittsburgh. I wish also to thank Dr. O. Klotz for many suggestions and Mr. Benner for the samples of soot and the chemical analysis of the same.

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Bibliography

1. Revue Industrielle, November, 1890.
2. C. Glinzer, "Address on the Smoke Nuisance and its Abatement," before the Industrial Society of Hamburg, 1890.
3. Racine, Vierteljahrsschrift für Gerichtliche Medicin. n. s. xl-xli.
4. Quoted by W. J. Russel, "Town Fogs and Their Effects." Nature. 1891. xiv., p. 10.
5. F. A. Rollo Russel, "The Atmosphere in Relation to Human Life and Health." Smithsonian Report. 1895.
6. Quoted by Liefmann.
7. H. Liefmann, "The Smoke and Soot Question Especially from the Hygienic Point of View." Halle. 1908.
8. Theo. W. Shaefer, "The Contamination of the Air of our Cities with Sulphur Dioxide, the Cause of Respiratory Disease." Boston Medical and Surgical Journal. clvii. 1907.
9. "How much Carbolic Acid or how much Sulphurous Acid is Necessary for the Destruction of the Smallest Form of Life?" Vierteljahrsschrift für Gesundheitspflege. vol. xii., 1879.
10. Schröder, J. Society Chem. Ind. xiii., 538.
11. Marshall, Microbiology. Philadelphia. 1911.

Some Histological Evidences of the Disease Importance of Pulmonary Anthracosis*

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The histological evidences as to the disease importance of anthracosis of the lungs, as set forth in this paper, are the results of work done by the author in an attempt to determine whether or not extensive deposits of dust and coal pigment within the body tissues have or have not any "real disease" significance. The problem as originally planned included a study of all of the microscopic effects of smoke and soot upon the body as a whole. Such a piece of work has been impossible in the time at our disposal so that in the present paper we shall deal only with the microscopic effects of smoke and soot, as observed upon the air passages and lungs, which are the chief portals of entry for these substances into the body. We have also included a consideration of the association of the resultant pulmonary lesions with those of tuberculosis and pneumonia.

The paper naturally divides itself into three parts, and for the sake of clearness these will be discussed under the headings of: I. The anthracotic process; II. The association of anthracosis and tuberculosis; and III. The association of anthracosis and pneumonia.

In the use of the term anthracosis in this report it must be understood that it refers only to the fairly well

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advanced stages of the process, as will subsequently be described.

The tissues studied were obtained in part from the surgical and autopsy materials of the Department of Pathology of the University of Pittsburgh, and from the Mercy Hospital, and in part from experimentally produced lesions in animals.

I. The anthracotic process.—Anthracosis is a term applied to a condition in which carbon particles of extraneous origin are deposited in the tissues or organs. It has been described as occurring in the lungs, spleen, liver, intestinal tract, and certain sets of lymph nodes, and is always accompanied by more or less fibrosis on the part of the tissues in which it is found. The most common site for anthracosis is in the lungs, and in individuals who work or live in smoky, sooty atmospheres; it often reaches a marked degree of development.

The fine particles seem to gain entrance into the lung substance by way of the air passages, and it has been stated by some that the mucous membranes of the nose, mouth, pharynx, larynx, trachea, bronchi, and bronchioles contribute largely to the taking in of the pigment, but our observations tend to show that in this community (Pittsburgh), at least, such is not the case. In the nasal cavities, the vibrissæ, and turbinates collect a great deal of carbon, which is subsequently removed by the secretions, for in the examination of nasal polypi and strips of turbinate mucosa removed at operations we have never seen a single example of free pigment or pigment bearing cells beneath the epithelial layer. Maclachlan[1] in his paper on "Tonsilitis" in which he reported a histological study of three hundred and fifty pairs of tonsils found only one example of a carbon pigment bearing cell in that structure, and about as many more tonsils and adenoids have since been examined without finding another instance. In the air passages below the larynx, pigment bearing cells have frequently been observed, but these were either free in the lumina or were surrounded by bits of mucus and other

cells and were not found penetrating the epithelial layers. In abscesses of bronchi, both the abscess contents and remaining parts of the mucosa have been seen to contain pigment bearing cells, though one could not say that any of these were definitely passing into the tissues. Moreover, our contention is supported by the following two experiments:

Experiment I.—For twenty minutes daily a young guinea-pig was placed in a box through which a dense black smoke was made to pass. The smoke was generated by burning xylol and entered the box through a funnel. After each exposure the animal showed a profuse nasal secretion and sneezed repeatedly. His furry coat was completely blackened. At the end of ten days he was killed with chloroform and autopsied. Macroscopic description: The gross findings of soot deposit were entirely limited to the blackened nasal mucosa, which was especially marked over the anteriar portions of the turbinates. Microscopic description: Sections showed soot particles adherent over the epithelium of the nasal mucosa. There was no evidence of pigment phagocytosis. (**Note.**—Nothwithstanding the fact that three guinea-pigs with early spontaneous anthracosis have come under our notice we failed in our attempts to reproduce the condition experimentally, apparently on account of the efficient way in which the intricately arranged turbinates protected the air passages beyond them.)

Experiment II. A white rabbit was given sixty inhalations in eighty-seven days of finely powdered lamp black in the following way: The nose piece of a powder insufflator was placed in the animal's mouth and the lamp black was forced in under pressure. At first the inhalations were given every two or three days, but later the treatment was carried out daily. The animal became very much emaciated and died. The autopsy was performed while the tissues were still warm. Macroscopic description: All of the mucous membranes of the mouth, pharynx, esophagus, stomach, larynx, trachea, and bronchi were coated or crusted with black pigment. Many of the lesser bronchi were completely plugged with masses of lamp black so that the foci of lung beyond them were devoid of air and in a stage of collapse. A few fine black granules were seen in the lung tissue. Microscopic description: In no part of the upper air passages was the phagocytosis of pigment observed. No pigment was seen in or beneath the epithelial layers of the bronchi, though some of them contained sufficiently large masses of pigment in their lumina to cause a flattening of the epithelial lining cells. In the alveoli many pigment phagocytes were seen (Fig. 5), and these cells were especially numerous in the atelectatic areas; some of them had already penetrated into the spaces of the alveolar walls, but as yet none had reached the perivascular, peribronchial, or subpleural lymphatics. The peribronchial lymphoid tissue was also free from pigment. The carbon pigment cells were large and round and were so completely filled with granules that their identity was obscured.

It will be seen by these two experiments that although we were able to bring an abundance of carbon pig-

ment in direct contact with the epithelial linings of the oral cavity and upper air passages we were unable to obtain any evidences of phagocytosis of such pigment in any of these structures. Coupling these results with our negatice findings in the surgical and autopsy materials cited above, we feel justified in concluding that the amount of pigment phagocytosis in the air passages above the lung alveoli must be a negligible quantity.

As our studies on the anthracotic process have to do largely with the lung alveolus and with the lymphatics of the lung a hasty review of our knowledge of these structures seems advisable at this point.

The alveoli, or air sacs, are minute cuboidal or spheroidal cavities which open directly into the small bronchioles through the infundibuli. The cavities are lined by a single layer of very flat pavement cells, which rest on a very thin basement membrane. Small openings called stomata sometimes connect one alveolus with its neighbors. The walls contain both smooth muscle and elastic tissue and are exceedingly vascular. The vessels are capillaries with definite endothelial walls, and they form a network completely surrounding the alveoli.

The lymphatics have their beginnings as small intercellular spaces in the alveolar walls. These connect with larger lymph spaces in the interlobular septa, which then empty into definite channels also having endothelial linings. These latter channels are found in three sets which are called, according to their location, perivascular, peribronchial, and subpleural. The larger channels have valves and foci of lymphoid tissue distributed along their courses. They all terminate finally in the peribronchial nodes.[2]

Most text books on general pathology discuss the process of anthracosis, but they do not enter into a description of the steps by which the condition develops. We have based our conception on the study of a great many lungs in the various stages of the anthracotic condition, and a description of the appearances of lungs in these var-

ious stages will serve to explain how we reach our conclusions.

The finding of free carbon particles in the alveolar spaces was unusual, though in some of the very advanced cases black pigment masses filled some of the alveoli in such a way as to resemble a complete cast. In the early stages of the anthracotic process, the condition was manifested only by the presence within the air sacs of large round mononuclear phagocytes filled with carbon pigment, while in the slightly more advanced stages these cells were not only present in the alveoli but were also found in the interalveolar lymph spaces, in lymphatics of the interlobular septa, in those about the vessels, and beneath the pleura, and in those of the lower layers of the bronchial mucosæ. A little later these cells were found gathered in nests of considerable size, and many of them were seen to be compressed into spindles so that only the elongated nests of granules were visible. The very advanced conditions consisted only of a quantitative increase in all of the features mentioned above and if one examines a lung in a late stage of anthracosis, such as we have commonly observed in Pittsburgh, the following points will be noted: The deep layers of the bronchial mucosa may or may not show pigment bearing cells, and when they are present they are generally grouped about the small mucosal vessels. The alveoli contain pigment casts, pigment bearing phagocytes and rarely free pigment particles. The phagocytes are found in the lymph spaces between the alveoli, about the vessels, in the septa, and beneath the pleura, and those about the vessels are so numerous as to form rosette-like nodules consisting of alternate layers of pigment cells and of connective tissue trabeculæ (Figs. 1 and 7). The amount of connective tissue may be out of proportion to the number of pigment cells present. The striking point in the whole picture is the extensive plugging and obliteration of the small and medium sized lymphatics and the compression of the large

ones. In the pleura and in the peribronchial lymph nodes the picture is a similar one, and in the latter the lymphoid tissue is often completely replaced by a scar tissue, the meshes of which are full of pigment spindles. By following the findings from stage to stage, the process appears to consist in the taking up of the carbon pigment by cells in the lung alveoli and its transportation through the lymph spaces and channels to the peribronchial lymph nodes. During the process of transportation many of the pigment cells appear to be caught in the lymph spaces causing a backing up of similar cells in the spaces behind them. Here the cells seem to act as irritants and cause the proliferation of connective tissue, which holds them firmly. The nodules appear to be formed by the alternate blocking of small perivascular lymph spaces by pigment bearing cells and the formation of new spaces about the outside of the occluded ones. The new spaces then seem to become obstructed by the pigment cells in a similar way and so on until the concentric nodular anthracotic rosette is formed. The cells nearest the center are always more flattened and spindle shaped than those in the periphery. The continuation of the process over a long period of time leads gradually to a very severe embarrassment of the lymphatic drainage of the lung. It seems well to observe here that the separation of anthracosis by some authors into diffuse and nodular forms is one of degree only, as the condition which is diffuse in the early stages becomes nodular in the later forms.

Four very interesting questions arise in connection with the anthracotic process which we have up to the present purposely omitted from the discussion, because they seemed to be sufficiently important to deserve consideration under separate headings.

1. The question of the part played by the alveolar epithelium in the phagocytosis of pigment. Several authors state that the epithelial cells of the lung take up anthracotic pigment and that the pigment bearing cell is merely a desquamated alveolar lining cell. The author

devised a combined staining method based on Heidenhain's hematoxylin as a nuclear stain, in combination with Mallory's aniline blue connective tissue method, which brought out the epithelial lining cells of the alveoli as clearly defined cells with red protoplasm and dark brown nuclei, resting upon a pale blue basement membrane. This stain was applied to sections from two hundred anthracotic lungs, and no pigment granules were found in the attached epithelial cells (Figs. 3, 4). After they had become desquamated it was difficult to identify the epithelium from other mononuclear cells present, except in certain instances where the desquamation occurred in strips of several cells, attached end to end. In none of these instances were epithelial cells seen to contain pigment.

While not wishing to state definitely that epithelial cells cannot and do not take up pigment under some conditions we have no hesitation in saying that it is not the usual procedure and that we must look to other sources for the identity of the common pigment phagocyte.

2. Concerning the question of the identity of the pigment phagocyte. Beitzke[3] says that the pigment phagocyte is sometimes derived from the alveolar epithelium and at others is a wandering mononuclear white cell from the blood. Other authors have described them as endothelial leucocytes, those cells formerly called transitional and large mononuclear leucocytes which have been shown to come from the endothelial lining of the blood and lymph spaces.[4] We were able to get no specific stain to definitely settle this point. However, the pigment phagocyte observed in the alveolus and the endothelial leucocyte have many points in common. They are indentical in size and appearance, they are both phagocytic, for blood pigment, as well as coal pigment, they both take up all kinds of cells and cellular debris and lastly we will show—later that the pigment phagocyte and the early cell of the tubercle, which we believe to be an endothelial cell, is one and the same cell.

By injecting pigment, in the form of Higgin's India ink, diluted one in five with normal saline we have attempted to determine the indentity of the cells which acted as phagocytes.

Experiment III.—Guinea-pig 2. Two cubic centimeters of dilute India ink was injected beneath the scapula at 5 P. M. The animal was found dead at 8:30 A. M. on the following morning, and presented the appearance of having died some hours before. Macroscopic description: The subscapular region was found much blackened and the neighboring lymphatics were deeply injected with black pigment. Microscopic description: In the sections many polymorphonuclear leucocytes were present in the region and a few of them contained a scant number of granules of pigment. A relatively few endothelial leucocytes were present, but all of them contained abundant granules.

Experiment IV. A similar dilute ink injection was made into the interstitial tissues of the abdominal walls of a guinea-pig. The animal was autopsied at the end of twenty-four hours. Macroscopic description: There was present in the tissues of the abdominal wall a localized nodule of swollen, edematous, and blackened tissue from which both smears and sections were made. Microscopic description: Both smears and sections showed a considerable number of endothelial leucocytes, and all of them contained a great number of pigment granules. There was a very marked polymorphonuclear leucocytic exudate present, but only occasional leucocytes were found containing pigment granules (Fig. 6).

Experiment V.—This experiment was similar to the preceding, save that the injection was repeated at the end of twenty-four hours. The animal was examined at the end of forty-eight hours. Macroscopic description: The gross appearance of the abdominal wall was similar to that in the preceding experiment. Microscopic description: A severe local endothelial leucocytosis was shown in the sections and most of these cells contained pigment. Many polymorphonuclear leucocytes were also present, but the phagocytosis of the pigment by these cells was insignificant.

It appears from these experments that the polynuclear leucocytes may take up pigment, but that their action is transient and unimportant, while the observations point to the endothelial leucocyte as the chief pigment phagocyte of the body.

3. The length of time during which the enclosed pigment remains intracellular is very important. As we have described above, the pigment bearing cells often become caught in the lymph spaces and when they have been surrounded by scar tissue appear simply as pigment spindles and look like nests of free granules, or as if they were incorporated in spindle shaped connective tissue cells

(Fig. 7). In acute inflammation of the lung, and more particularly in edema, the spindles again assume a more or less round or oval form (Fig. 8), suggesting that the spindle shape is due entirely to the pressure of the scar tissue. In two experiments we were able to reproduce the condition.

Experiment VI.—A small amount of dilute India ink was injected interstitially into the ear of a rabbit. At the end of twenty-six days sections were made. Almost all of the pigment had been taken up and was found in the form of spindles firmly fixed between strands of connective tissue (Fig. 9).

Experiment VII.—The ear of another rabbit was treated in a similar way. After forty days the ear was dipped into water at 60° C. for three minutes. At the end of fifteen hours a very extensive edema of the ear had developed and the animal was brought to autopsy. Sections showed a very extensive interstitial serous-exudate, without any cellular elements, to separate the strands of connective tissue very widely. Almost all of the pigment was found to be present in cells which were of the large round type (Fig. 10).

In some processes within the lung, such as abscess, gangrene, the various granulomata, and tumors, which are accompanied by necrosis, the anthracotic pigment is found extracellular and is seen widely distributed as free granules. These granules sometimes remain free in caseous areas, or in scar tissue, or they may again be engulfed by large mononuclear phagocytes. From our own observations we feel justified in saying that carbon pigment once taken up by the cells remains intracellular indefinitely, unless freed by some process producing general necrosis of the tissues.

4. How long do phagocytic cells remain free in the alveoli? There is at present no accurate means of determining the length of time during which a cell may remain free in the alveolus, though there are several points which indicate that this is longer than is generally supposed. One may observe many cells in the lung alveoli which are only partially filled with pigment, but to find such a one in the intra-alveolar lymph spaces is quite unusual. This suggests that the phagocytic cells are generally pretty well loaded when they leave the alveoli.

In pneumonia and edema of the lung where individuals have been confined to the hospital for some time and presumably not inhaling very much pigment, the alveoli often contain numerous pigment bearing phagocytes. The almost constant presence in the alveolar spaces of "Herzfehler Zellen" in brown induration of the lung is hardly a terminal condition, and establishes further evidence that phagocytic cells may remain free in the alveoli for a considerable length of time. In Experiment II. we found that although the soot inhalations had been carried on for eighty-seven days, pigment bearing cells were found only in the alveoli and inter-alveolar spaces and there was no evidence that any had migrated as far as the large lymphatics. All of these points seem to indicate that the phagocytic cells are not transient scavengers, but in a more leisurely manner gather their pigment-load and transport it to the tissue spaces.

Summarizing the foregoing work we conclude:

1. The quantity of pigment taken up by the upper air passages is a negligle one.

2. The lining cells of the lung alveoli take little or no active part in the phagocytosis of carbon pigment.

3. The process consists in the phagocytosis of the pigment within the lung alveolus by a cell, which is probably an endothelial leucocyte. This cell then passes into the pulmonary lymphatics where it sometimes lodges and becomes surrounded by connective tissue. The pigment remains intracellular until acted upon by some process producing a local necrosis of the tissues.

4. The importance of the sequence of the process lies in the fact that the lymphatics of the lung become obliterated either mechanically or by fibrosis.

II. The association of anthracosis and tuberculosis.—Pulmonary tuberculosis in adult individuals in Pittsburgh is constantly associated with more or less anthracosis, so that ample opportunity was afforded for the study of the effect of the one upon the other. The important lesion of tuberculosis is the tuberculous granuloma

or "tubercle," and the following discussion is confined to a consideration of the related lesions of this characteristic tuberculous lesion, and anthracosis.

The present day knowledge of the histogenesis of the tubercle is the result of the accumulated work of more than a generation of pathologists. Point by point has been added year by year, and credit cannot be given to any single individual or group of individuals, and more especially is this true for the reason that our facts are still deficient in certain details. The names of Baumgarten[5] working in Germany and of Borrell[6] in France, however, stand out prominently among the founders of the experimental studies, to which we owe so much. The formation of the tubercle as we understand it at present, seems, in general, to occur in the following way:

When tubercle bacilli enter the smaller vessels or lymph spaces they are shortly incorporated by the cells[c] lining these structures. In both blood vessels and lymph spaces the process appears the same so that the description of the former will suffice. The cells which have taken up the tubercle bacilli undergo rapid proliferation and develop a nest of proliferated endothelial cells which tends to fill the vessel, obliterate it, cut off the nutrient supply to the nest, and leave it an extra-vascular structure. The endothelial cells have by this time changed their morphology and somewhat resemble epithelial cells, for which reason they have long been known as "epithelioid" cells. About this time a few polymorphonuclear leucocytes may frequently be seen in the margins of the structure, but, in the later stages, lymphocytes alone are present. The next change which is usually observed is the appearance, near the center of the nest, of a multinucleated or giant cell, which was described by Langhans in his early studies on tuberculosis and to which his name has been given. That the Langhans' giant cell is the product of the endothelial cell seems now to be unquestionable, but a considerable dispute still exists as to whether it arises by a fusion of the endothelial cells or by a division of the nucleus

without a multiplication of the cell. Caseation necrosis is next seen near the center of the nest and commonly first manifests itself in the center of the giant cell. With the appearance of caseation there is not uncommonly a proliferation of connective tissue about the margin which is an attempt on the part of the tissues to encapsulate the lesion. This completes the structure of the miliary tubercle (Fig. 15). Complete encapsulation and healing may follow with more or less absorption of the necrotic matter by the tissues,[p] or the deposit of calcium salts within the caseated areas. On the other hand a local or general spread of the tuberculous process may follow. A certain amount of local invasion from the original tubercle may take place by direct extension leading to the development of numerous closely aggregated and fused tubercles. The lesion thus produced is known as a conglomerate tubercle. A more extensive local spread, even to the involvement of an entire organ, often takes place by way of the anastomosing lymph spaces.

Recently, considerable evidence has been advanced to show that the so-called epithelioid cells are at all times endothelial cells, and if this is true the term "epithelioid," which is somewhat confusing, should be discontinued. Mallory has called attention to the constant involvement of endothelial cells in the earliest recognizable lesions of tubrculosis, and has shown that no fibrils are demonstrable by stains in the tubercles until the stage of encapsulation is reached, at which time fibrils appear in conjunction with the proliferating connective tissue cells in the outer border. Since endothelial cells are not known to produce fibrils, the evidence is in favor of the essential cell of the tubercle being endothelial in character. Bowman, Winternitz, and Evans have shown experimentally that tubercles may be developed in the liver from the Kupffer stellate cells, which are also considered as endothelial cells. By first inducing a vital staining of these cells in animals by injecting trypan blue into the circulation, then inoculating the same animal with tubercle bacilli, they

were able to follow the formation of tubercles by the stained cells. I have also induced the development of tubercles experimentally, the essential cells of which were phagacytic both for pigment granules and the tubercle bacillus (Experiments VIII., and IX., and X., and Figs. 17, 18, and 19), and if we were right in concluding that the endothelial cell is the general pigment phagocyte of the body, we have another link in the chain which indentifies the "epithelioid" as an endothelial cell.

As to the nature of the Langhans' giant cell we have, in several instances, observed multinucleated cells containing both pigment granules and tubercle bacilli. This would indicate that the cells exhibiting the phagocytic properties to foreign particles and tubercle bacilli were of the same origin and that the giant cells were but a morphological modification of these. As to the way in which the phenomenon of multinucleation occurs we have no conclusive additional evidence. An experiment was planned by which it was hoped to discover a constant ratio between the amount of pigment taken up and the number of nuclei present in the giant cell. The experiment failed, firstly, because time was not allowed for complete phagocytosis of the pigment before the tubercle bacilli were introduced and, secondly, because the amount of pigment used was so great as to completely obscure many of the nuclei in the cells. The experiment though unsuccessful seems worthy of repetition.

Lesions illustrating the association of the various stages of tubercle formation with all degrees of anthracosis were found in the materials secured from the autopsies and the following interesting observations were made: (1) Examples of early tubercles were found which consisted of simple nests of endothelial cells, some of which contained black pigment granules though they differed apparently in no other way (Fig. 16). (2) Pigment granules were found in many of the giant cells, and when caseation necrosis was also present in these cells the nuclei were found arranged in the periphery with the

pigment granules grouped in circles about them (Fig. 20). (3) In tubercles where caseation was advanced much of the pigment was found to have been liberated and occurred either as free granules diffusely distributed throughout the necrotic foci or, as was more often the case, gathered in the margins of the caseous areas where it was found undergoing a second phagocytosis by endothelial cells (Fig. 21). (4) Many partially and completely encapsulated tubercles were seen with great numbers of pigment spindles caught in the meshes of the capsules, and in these instances the connective tissue was unusually abundant. Obliteration of the neighboring perivascular lymph spaces by anthracotic fibrosis was a common additional finding in some of these lesions.

The number of cases examined was too small to allow the drawing of a general conclusion, but it seemed that there was less local spread and more extensive fibrosis in this latter type with anthracosis than is usual in ordinary pulmonary tuberculosis. It further seemed that the presence of pigment within the endothelial cells did not interfere with their development of typical tubercles (Experiments VIII., IX., and X.).

Experiment VIII.—Two cubic centimeters of a very dilute suspension of India ink was shaken up in normal salt solution with two loops of bacillus tuberculosis bovinus (kindly furnished by Dr. W. L. Holman) and was injected into the deep muscles of the thigh. After twenty-four days the animal was killed with chloroform and autopsied. Macroscopic description: Indefinite grayish-black tubercle-like nodules were found among the muscle fibers, and some of the intermuscular lymph spaces appeared as blackened lines. There was no evidence of involvement of the inguinal nodes, nor of the other organs or tissues of the body. Microscopic description: Many early tubercles were seen in the process of formation and the cells composing them often contained black pigment granules (Fig. 17). Tubercle bacilli and these pigment granules were repeatedly found in the same cells. Relatively few multinucleated cells were seen, but in some of them bacilli and pigment granules were found together. Practically no fibrous proliferation was present about the lesions and the cells did not show any kind of fibrils when special stains were applied.

Experiment IX.—One cubic centimeter of a I in 5 suspension of India ink in normal saline was shaken up with two loops of bovine tubercle bacilli and injected underneath the scapula of a rabbit. Ten days later inhalations of soot similar to those used in Experiment II were begun. These were carried out about every five days

and about fifteen inhalations were given. After seventy-four days the animal died from general tuberculosis. Macroscopic description: A large caseous mass about 4 centimeters in diameter,[p] near the center of which was a small black nodule was found beneath the scapula. The lungs, liver, spleen, and kidneys showed very advanced caseous tuberculous lesions. A few rather indefinite black granules appeared about the margins of the lung tubercles. Microscopic description: The lungs alone are of interest in this experiment. They contained all stages of tuberculous lesions, and a few fine pigment granules were observed in some of the cells forming the tubercle. Two vascular lesions were of interest because they showed direct extensions of early tubercles into the lumina of vessels without causing rupture of the walls. The subscapular lesion was in a very advanced stage of caseation necrosis and was valueless for study.

Experiment X.—Injections as carried out in Experiment VIII. were made into the interstitial tissue of a rabbit's ear. The animal was brought to autopsy at the end of twenty-six days. Macroscopic description: The ear showed one large diffuse black area involving approximately one-half of the ear. The tissues were somewhat thickened and contained three large and several small grayish-black nodules. At the base of the ear was found an enlarged and blackened lymph node. Microscopic description: Sections of the ear and lymph node both demonstrated the presence of early tubercles, with black pigment granules in the cells of the tubercle, and in both instances tubercle bacilli and pigment granules were found in the same cells (Fig. 19).

In all three of these experiments we were able to induce miliary tubercles,[p] formed by cells which were filled with pigment granules. This indicated that the presence of pigment in the specific cells did not exert any inhibitory influence to the development of tubercles. By demonstrating pigment granules and bacilli in the same cells of the tubercle, we were able to show that a single cell may actively phagocyte both materials. In other words, the essential cell of the tubercle and the pigment phagocyte were found to be identical. Still more interesting was the finding in Experiment X. in which both the pigment phagocytes and tubercle forming cells were found in a neighboring lymph node, where tubercles were being formed by cells which contained pigment.

Summing up our observations on the associated lesions of anthracosis and tuberculosis we came to four conclusions:

1. The cell which takes the early active part in the formation of a tubercle is phagocytic for tubercle bacilli

and for pigment granules and is probably an endothelial cell.

2. The presence of pigment granules within these cells does not interfere with the cells taking part in the formation of a tubercle.

3. The presence of pigment-bearing cells in the connective tissue about tuberculous lesions acts as an additional stimulus to fibrosis and encapsulation.

4. The obliteration of the pulmonary lymph spaces in the anthracotic process is unfavorable for the local spread of tuberculosis, and aids in the localization of the condition.

III. The association of pneumonia and anthracosis.—With pneumonia the relation of anthracosis is a very different one. The process is one of acute inflammation wherein the alveoli, bronchioles, and often the smaller bronchi become filled with a fibrino-purulent exudate. Ordinarily this exudate remains in the alveoli for a relatively short time, usually from five to nine days, and then undergoes resolution. The process of resolution consists, firstly, in a softening and liquefaction of the exudate, which takes place through the autolytic action of the different elements, and is known as puriform softening, and secondly, in getting rid of the resulting debris. Much of the resolved exudate is coughed up, but the greater part of it passes back into the circulation by way of the lymph spaces and through the lymph nodes.[7] During resolution these spaces are found to be dilated and to contain fluid, fragments of fibrin and many types of exudative cells. Among these cells are numerous phagocytes containing other cells, cell debris, fibrin, and fat globules (Fig. 13). The contents of the sinuses of the lymph nodes is exactly the same (Fig. 12). It is evident that for the removal of this debris as well as for active phagocytosis comparatively large channels are necessary, and it is obvious that anything which may impede the passage through the lymphatics must interfere with the healing process. The mechanical blocking of the spaces forces the debris to find

other channels of exit, and the imperfect drainage leads to the accumulation of waste products which, in turn, may retard the activity of the proteolytic enzymes.

When for any reason resolution of pneumonia fails to take place the microscopic picture presents certain characteristics. The cellular elements of the exudate are not numerous and are often lymphocytic; the fibrin becomes contracted into firm hyaline, spindle, or fan-shaped masses which are bathed in fluid, and the alveolar wall appears thickened and edematous. At somewhat later periods these fibrinous masses occasionally undergo organization in a way comparable to the repair of a wound. Abscesses are also very common findings in pneumonic lungs where failure of resolution has occurred, and gangrene is also seen in some instances. Not only are such lungs unusually susceptible to secondary infection, but the actual presence of the undigested fibrin is claimed to be extremely toxic.

Our evidence of the effects of anthracotic conditions on pneumonia was obtained by a microscopic study of sixty-two cases of the disease. Of these, thirty-eight were lobar, twenty were of the bronchial type, four were hypostatic, and two were the end result of general septicemia.

Of the thirty-eight lobar pneumonias, nine, or nearly twenty-five per cent., were diagnosed "unresolved" at autopsy and two others presented microscopical evidences of the same condition. Two of the bronchial type contained much hyaline fibrin and abscesses, and one of the hypostatic variety showed advanced organization.

This unusually large percentage of failures in resolution suggested a careful study of the pulmonary lymph spaces in these cases. This was carried out as far as we were able in the sections. Our attention was mainly directed toward the large channels because the small lymph spaces were obviously involved in all of the cases, and also because a severe involvement of the larger lymphatics indicated an extensive participation of the smaller contributory vessels over a considerable area. We found the

large perivascular and subpleural lymph spaces closed or compressed in four cases by anthracotic fibrosis. Two cases presented a similar picture save that the amount of scar tissue seemed to be out of all proportion to the number of pigment cells. One instance was found where the lymph spaces were closed by scar tissue alone, and in two others there was no local evidence which could be construed as having anything to do with failure of resolution. The two remaining cases were pneumonias in which there were coexistent septicemias, and were omitted from consideration because the origin of the lung abscesses might have occurred as a part of the general process. Both of these lungs were examples of late stages of anthracosis. Our results, then, showed that in six of the nine cases of unresolved pneumonia the larger lymph channels were severely embarrassed by the anthracotic process.

The failure of resolution in pneumonia is commonly attributed to the low state of the general bodily condition, although Pratt[8] called attention to the frequency of delayed resolution in individuals who had been previously attacked, and attributed it to the fibrosis of the lymph spaces following the lymphangitis which occurs during resolution.

The results of the microscopic examination of the lymphatics in the total number of lobar pneumonias were as follows: Eighteen cases presented dilated lymph spaces filled with serous or sero-purulent exudate; five were closed by acute fibrino-purulent exudate, nine were practically obliterated by anthracosis; two were obliterated by scar tissue, and four were in the stage of resolution with large numbers of phagocytes in the lymph sinuses.

An analysis of these findings seems to indicate that during an attack of pneumonia in an otherwise normal lung the lymph vessels are generally engaged in an active exchange of fluids. Even in those instances where these channels are filled with a fibrino-purulent exudate this

exudate has a better chance of becoming liquefied and returning to the circulation than a similar exudate in the alveoli. So that even under these circumstances they would again be available for drainage.

The presence of the phagocytes in the channels and in the sinuses of the lymph nodes shows fairly conclusively that the lymphatics are important drains during the stages of resolution of pneumonia, and if these are closed the waste products of autolysis of the exudate must accumulate, and such an accumulation undoubtedly interferes with proper enzymatic action. In several of the above mentioned cases where the lymphatics were found to be closed, death occurred before the stage of resolution was reached. It is, of course, impossible to say that the presence of the anthracotic condition had any definite connection with the fatal result. Yet it is likely that the lymphatics in inflamed areas are just as important for the entrance of fresh fluids as for the exit of waste materials. It has been variously shown that the cultures of pneumococci obtained from pneumonic lungs after crises are less virulent than those made in the early stages, and this suggests some immune reaction against the organisms. If we are correct in the supposition that immune substances are the products of the entire body as well as of local origin, we must recognize the need of a constant influx of fresh fluid containing such immune products. It seems, then, that the closed lymph spaces may have had some effect even in this latter class of cases.

Our series although small seems to point toward a certain relation between "unresolved pneumonia" and anthracosis with chronic obliterating lymphangitis.

An interesting collateral finding, brought out by the newly devised staining method used in demonstrating alveolar epithelium, showed that desquamation of alveolar epithelium in pneumonia is inconstant and occurs only in some alveoli. Instances were observed in which the pneumonia had reached a stage of resolution and yet practically all of the alveolar epithelium was intact.

1. Any process which interferes with the free lymphatic drainage during pneumonia will delay resolution, both by actual mechanical obstruction to the migration of cells, and by aiding in the accumulation of waste products probably embarrassing enzymatic digestion of the exudate.

2. Anthracosis is an important process in the lung interferring with lymphatic drainage, and is a factor in causing delayed resolution.

Summarizing the various points indicating the disease importance of anthracosis which we have gathered from histological evidence we draw the following conclusions:

1. Moderate anthracosis in an otherwise normal lung is not in itself detrimental to health.

2. In tuberculosis and granulomatous conditions in which the reactions are chiefly centered in focal lesions of the tissues, the anthracotic condition is either entirely passive, or is active in assisting healing, in that it is an additional stimulus to fibrosis and encapsulation and in that it aids in the localization of the process through the obliteration of the lymph spaces.

3. In acute inflammatory conditions where the lymphatics are important for proper resolution, anthracosis becomes seriously detrimental, because of the obliteration of these spaces.

[I wish to express my thanks to Dr. Oskar Klotz for his very able advice during the preparation of this paper and to Mr. William Coburn for his efficient aid in carrying out the experimental work.]

Bibliography

1. Tonsillitis. W. W. G. Maclachlan, Publication from the University of Pittsburgh, September, 1912, 4.

2. Aschoff. Pathologische Anatomie. 1912, ii., 280.

3. Beitzke. Ibid., ii., 316.

4. Mallory. Cirrhosis of liver. Johns Hopkins Bulletin, March, 1911. 72.

5. Baumgarten. Uber Tuberkel und Tuberkelose, Berlin, 1885. Lehrbuch der pathol Mykologie. 1890.

6. Borrel. Tub. experiment du vein. Annales de l'Inst. Pasteur. 1894.
Tub. pulmon. experiment. Ibidem. 1893.

7. Aschoff. Pathologische Anatomie. 1912. ii., 290.

8. Pratt. Jour. Boston Soc. of Med. Sci. 1900. Journ. of Med. Research, iv., 183.

Description of Plates XIV-XVII

DESCRIPTION OF PLATES XIV-XVIII.

Plate XIV., Fig. 1.—Perivascular arrangement of nodular anthracosis.

Fig. 2.—Subpleural type of anthracosis.

Fig. 3.—Alveolus containing many pigment phagocytes and a pneumonic exudate. Note the alveolar epithelium swollen and free from pigment.

Fig. 4.—Alveolus containing pigment phagocytes with alveolar epithelium intact. Note one cell containing both pigment and a red blood corpuscle.

Plate XV., Fig. 5.—Experimental anthracosis. Pigment phagocytes in alveoli and inter-alveolar lymph spaces.

Fig. 6.—Injection of carbon pigment in abdominal wall. Pigment present in large round cells and in inter-cellular spaces. Polynuclears contain no pigment.

Fig. 7.—Spindle-like arrangement of pigment in perivascular anthracotic nodules.

Fig. 8.—Effect of edema on spindle-like pigment occlusions in perivascular anthracosis.

Plate XVI., Fig. 9.—Experimental fibrosis produced by the injection of India ink into a rabbit's ear.

Fig. 10.—Effect of artificially produced edema on rabbit's ear which had been treated in a similar way to that seen in the preceding figure.

Fig. 11.—Alveoli containing pneumonic exudate in stage of resolution. Note epithelial cells intact on alveolar walls.

Fig. 12.—Lymph spaces of peribronchial lymph node containing phagocytes from preceding figure.

Plate XVII., Fig. 13.—Perivascular lymph space filled with broken up fibrin and phagocytes from same case as preceding figure.

Fig. 14.—Perivascular lymph spaces completely obliterated by carbon pigment and fibrosis. From severe case of unresolved pneumonia.

Fig. 15.—Formation of early lung tubercle.

Fig. 16.—Early tubercles containing pigment bearing cells (human).

Fig. 17.—Experimental tubercle in rabbit's thigh. Pigment bearing cells actively forming the tubercle.

Plate XVIII., Fig. 18.—Oil immersion view of same tubercle. Cells contain both pigment granules and tubercle bacilli.

Fig. 19.—Tubercles containing pigment bearing cells in lymph node.

Fig. 20.—Arrangement of pigment in giant cells in late stage of tuberculosis.

Fig. 21.—Distribution of carbon pigment in the periphery of caseous tubercle.

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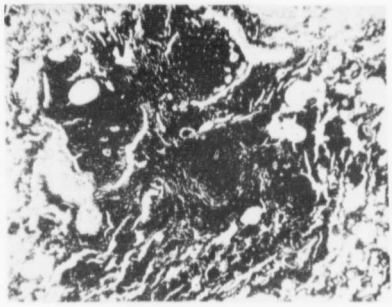

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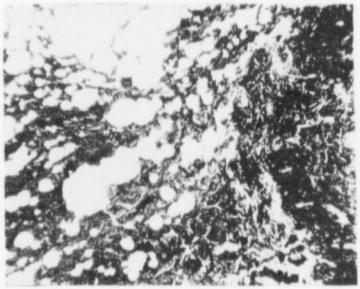

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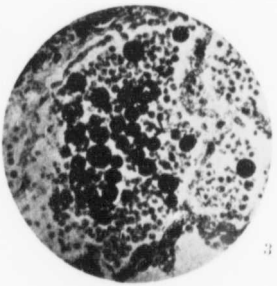

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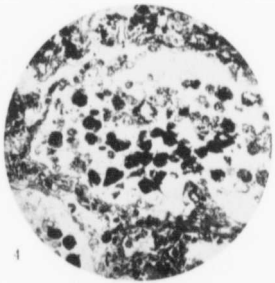

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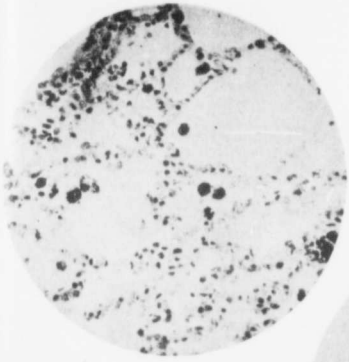

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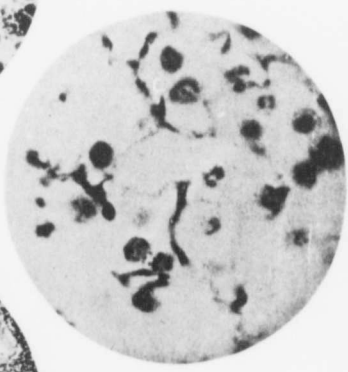

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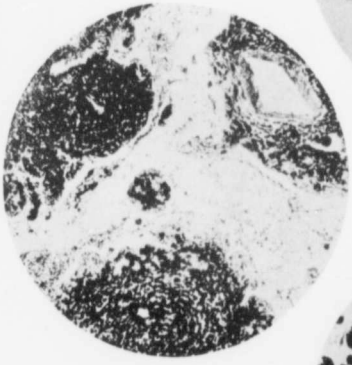

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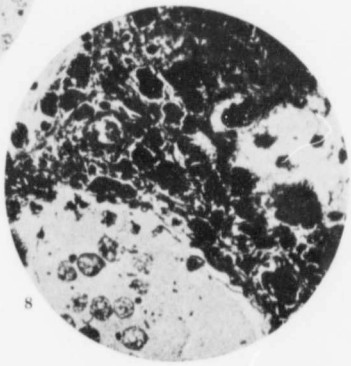

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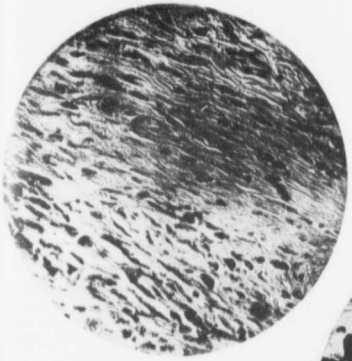

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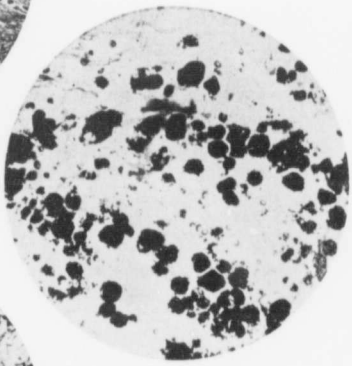

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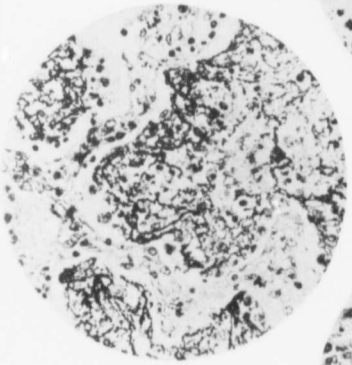

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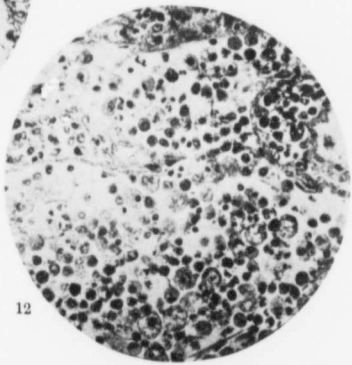

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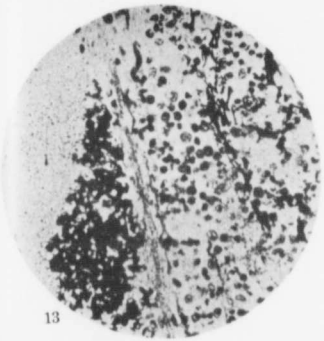

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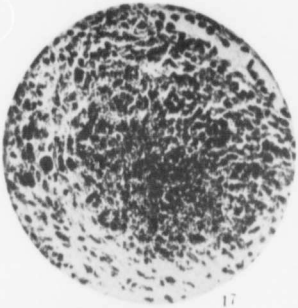

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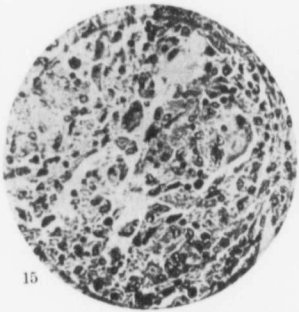

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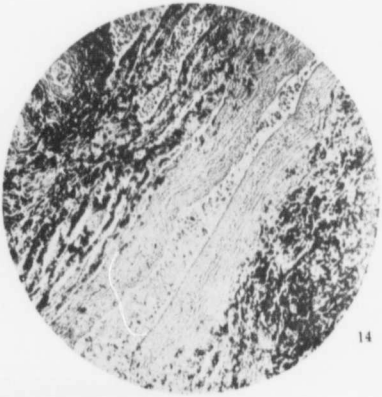

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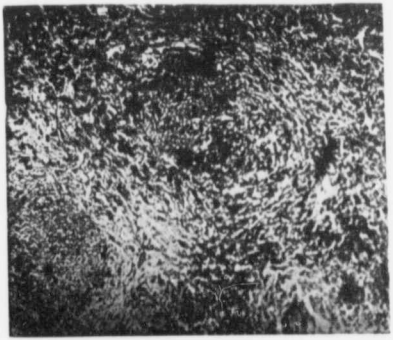

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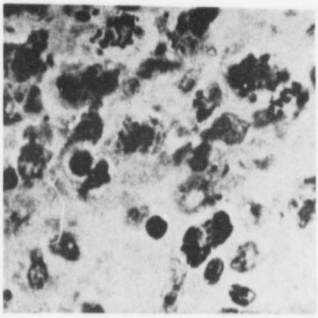

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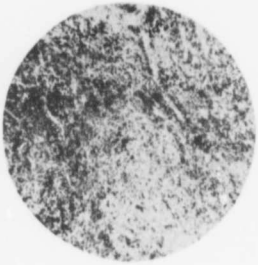

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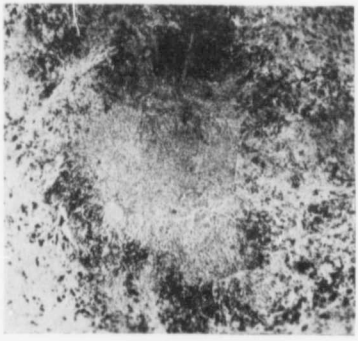

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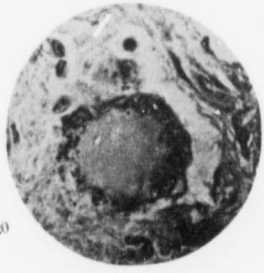

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A Study of the Influence of Varying Densities of City Smoke on the Mortality from Pneumonia and Tuberculosis*

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Associate Professor of Medicine, University of Pittsburgh.
AND
C. H. MARCY.

As part of the investigation into the smoke problem, which is being carried on at the University of Pittsburgh, through the generosity of Mr. R. B. Mellon, we have made

Pittsburgh, Pa.
Showing Smoke Areas
by Wards

Chart I

*Reprinted from the Transactions of the Fifteenth International Congress on Hygiene and Demography, held at Washington, D. C., September 23-28, 1912.

COMPARISON OF TUBERCULOSIS DEATH RATE

BY WARDS AND SMOKE CONTENT OF AIR BY WARDS

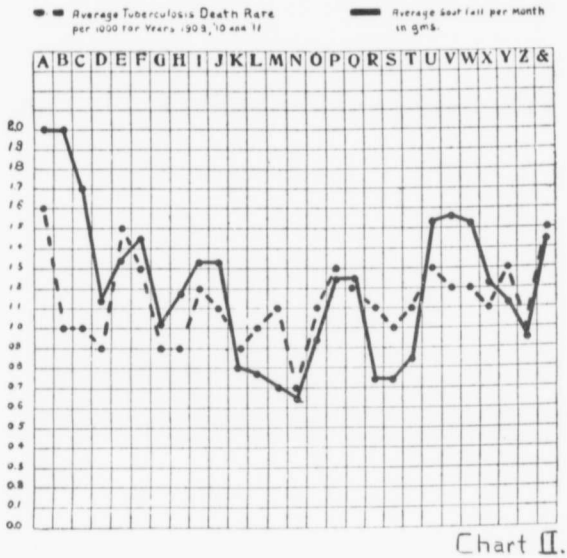


Chart II.

a study of the influence which varying densities of smoke have on the mortality from tuberculosis and pneumonia as typifying acute and chronic pulmonary infections. Perhaps in none of the smoky cities of the world is it possible to go into the influence of smoke in the same way as in Pittsburgh. The peculiarity of the relation of high hills and valleys, in close proximity, within the city limits, makes a well-defined variation in the smoke content of the air. It will be noticed on the map (chart 1) that, along the river frontage, where for the most part the land is low-lying, the air content of smoke is very dense; skirting this dense smoke area, the hills rise more or less abruptly to

varying heights. On the lower hills, as exemplified by C, D, E, J, Y, Z, and &, there is an area of moderate smoke density. Where the hills, however, rise directly from the river to the height of 400 or 500 feet, as exemplified by R, S, T, G, K, L, N, and M, there is comparatively little smoke at any time of the year. During prosperous times, the valley, or dense-smoke area near the rivers, is always heavily laden with smoke. This is true even when there is a brisk breeze blowing.

It is possible, from the division of the city into wards, to rule out a number of other factors, such as poverty, race congestion, and so forth, as an influencing factor in the point of relation which we have brought out by these studies.

It is unfortunate that our figures only cover a period of two years, but this is due to the fact that no records taking account of the wards of the city have been kept prior to this time and also that the number of wards was changed from 56 to 27 three years ago. This renders useless any previous comparison on the ward basis.

The figures which we have obtained are, from one aspect, very convincing, although from another aspect very confusing. It will be noticed for instance, in Chart 4, that in Chicago, where there is a comparatively low smoke content, there is still a very high pneumonia death rate; while in Pittsburgh, with the highest smoke density, there is no higher pneumonia mortality.

The question of smoke density for the various cities has been based upon the reports of the United States Government Weather Bureau. In the various cities which are included in this report the method of determining this density is based on the distance of vision, considering certain fixed objects in the city from an observation center. In Charts 2 and 3, of Pittsburgh, the basis has been certain carefully devised studies of precipitation of air content of carbon dust. These scientific observations have been made in connection with the present smoke study by Messrs. R. C. Benner and C. H. Marcy. In Chart 4, for the sake

of comparison, we have adhered to the Government Weather Bureau report.

The mortality tables, on which Charts 2 and 3 are based, were derived from the health table of statistics of the city of Pittsburgh, and include the years 1910 and 1911.

COMPARISON OF PNEUMONIA DEATH RATE BY WARDS AND SMOKE CONTENT OF AIR BY WARDS.

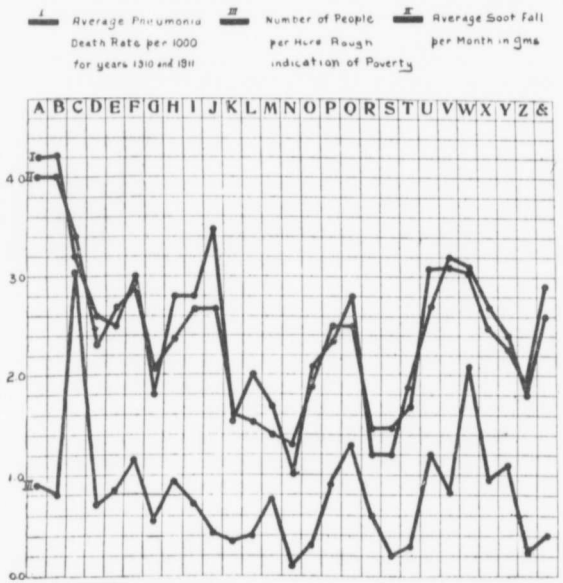


Chart III.

The mortality tables for pneumonia, on which Chart 4 is based, were secured from the Bureau of Census statistics.

We desire to acknowledge here the courtesy and kindness of Director Durand, of the Government Department of Commerce and Labor, for the prompt return of the smoke studies and statistical tables from the various cities, which we scarcely could have obtained without his aid.

Since 1905, Ascher, of Konigsberg, has published tables and studies on the relation of smoke to pneumonia and tuberculosis. Ascher's main conclusions are as follows:

1. The deaths from acute lung diseases are most frequent in children and old people. There is, from year to year, a steady increase in the number of these deaths. This is explained, in part, by the increased contamination of the air by smoke, because the increase in the number of deaths is greatest in districts of an industrial character and not in farming districts. Since 1875 the deaths of infants from pneumonia have increased 600 per cent.

2. There is a noticeable difference in the deaths of acute lung diseases, in those industrial districts where smoke contamination is greater than in those industrial districts where the smoke contamination is less. The number of deaths in coal workers from acute lung diseases is 130 per cent higher than the number of deaths in other workmen from the same cause and of the same age. Hand in hand with the increase of acute lung diseases there is a decrease in the age at death from tuberculosis. This means that the fatal course of tuberculosis in smoky districts is shorter.

3. Animal experiments show that the inhaling of smoke increases the susceptibility of animals to infection by aspergillus. Pneumonia develops in animals which have inhaled smoke more easily than in the control animals.

Ascher's experimental work can scarcely be used as a comparison with the influence of the smoky atmosphere of cities, because he used the smoke or soot of burning petroleum, which forms an insignificant amount of the smoke content of cities and differs very materially from the main content of the smoke of city air.

So far as Ascher's statistical tables are concerned, there can be very little doubt that acute lung diseases are taking a different course to tuberculosis, and one of the most appalling things of modern civilization has been the sharp increase of the mortality rate from acute diseases of the respiratory tract coincident with industrial activity and the consequent irritation by smoke of the

respiratory tract, this being in sharp contrast to the diminution of the chronic diseases of the respiratory tract, as exemplified by tuberculosis.

The pneumonia death toll for 1900 for the United States was 105,971; for 1909, 122,400; and for 1910, 136,000; an increase of 10 per cent in one year.

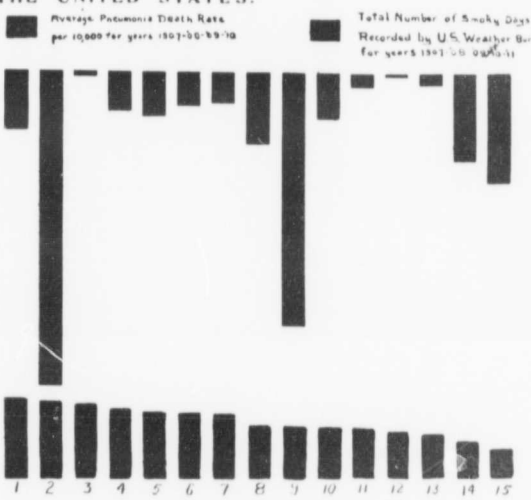
The average death rate from tuberculosis per 100,000 for the 10-year period 1900-1909 was 183; in 1909 it was 160.8; and in 1910, 160.3. This shows a steady decrease.

These figures are examples of the merit which this subject has for our most earnest study.

The chief fact which has stimulated our interest in the pneumonia problem in the city of Pittsburgh has been the terribly acute and fatal type of pneumonia which fills the wards of our hospitals. It can hardly be that this severity of infection has to do alone with the virulence of the germ; nor does it seem likely that it has to do with the generally low type of resistance which Pittsburghers have to this type of infection; rather, it seems more likely that there must be some factor present in Pittsburgh which does not operate in other cities. As we approached this subject we felt sure that we would be able to prove that this factor was the smoke of the iron and steel mills. How signally we have failed in this will be very evident from chart 4, in which is seen the striking contrast between Boston, with almost no smoke, and Pittsburgh, swamped with smoke, yet with approximately the same pneumonia death rate. The same sort of thing is apparent between Mobile, Ala., with almost no smoke, and St. Louis, Mo., with a very high smoke content in its air.

When we attempt to analyze the city of Pittsburgh on the basis of air content of smoke and pneumonia death rate, so striking is the correspondence between the pneumonia death rate and the smoke content of the air of the ward that we are convinced that smoke is a very important factor in the severity of the disease and that some other factor must operate in those cities where the smoke content of the air is not the determining factor in the

COMPARISON OF PNEUMONIA DEATH RATE AND SMOKE CONTENT OF FIFTEEN CITIES OF THE UNITED STATES.



1-Chicago
2-Pittsburgh
3-Boston
4-New Orleans
5-Richmond Va
6-New York
7-Charlestown S.C.
8-Cincinnati, O
9-St Louis
10-Philadelphia
11-San Francisco.
12-Mobile Ala
13-Cleveland
14-St Paul
15-Portland Ore

Chart IV

pneumonia death rate. This correspondence is more striking even when we put in a line showing that there is no definite bearing of such other factors as poverty, race, and congestion. When, however, one turns to tuberculosis and analyzes the death rate from this disease by wards and charts it in comparison with the smoke content of the air, one finds that there is no association whatever. This corresponds with our clinical observations on between four and five thousand cases of tuberculosis in the last six years. As the result of this clinical study we have come to the conclusion that the general death rate from tuberculosis in Pittsburgh is low—that there is nothing in the smoke content of the air which in any way stimulates the onset of the tubercular process or militates against the rapidity of recovery from tuberculosis when once this disease has been contracted.

In other words, after having made an analytical study of the relation of smoke in the City of Pittsburgh, where it is possible, by virtue of its contour, to separate the atmosphere into "densely laden," "moderately laden," and "comparatively no smoke" areas, and in such a way as to rule out such influences as poverty race, and congestion, we are forced to the conclusion that the smoke content of the air has an apparently important bearing on the pneumonia death rate and comparatively little bearing on the tuberculosis death rate.

From the careful analytical studies of the character of the smoke in the city of Pittsburgh, carried out by Dr. Klotz and Dr. Holman in connection with this same work, there is added a purely chemical and physical reason for this same conclusion, for they have found that the percentage of phenol around the carbon, which pollutes the air, is sufficient to destroy most or many of the organisms with which they have studied when suspensions of these are mixed with suspensions of air smoke.

With this fact in mind it is probably legitimate for one to turn to the pathological studies of these two infections. Pneumonia is a catarrhal condition, and a pre-

disposition for it may be prepared by the irritation of the mucous membrances with foreign substances; but the second (tuberculosis), being granulomatous in type, in which the microorganisms are sequestered and surrounded by cells, the cure of which is accomplished by fibrosis, may naturally be supposed to be aided in the direction of cure by any deposit which stimulates granulation and fibrosis. Some strength is given to this theoretical view by the evidence which we have from anatomical studies, in which we find depositions of carbon particles around the healed tuberculous focus.

One can not help wondering why, when the facts concerning these two diseases are known, so little has been done on the question of pneumonia prevention, when so much has been accomplished on the tuberculosis side of pulmonary infection; and in addition to my remarks upon the relation of smoke to this disease, I should like to again call attention to certain suggestions which I offered a year ago in an address before the Ontario Medical Association for the control of a certain portion of the evils arising from pneumonia. These are as follows:

First. The proper segregation of pneumonia patients and their utensils in hospitals; cleaning, by sprays and washes, the noses and throats of all who nurse and come in contact with these patients; careful hand washing of nurses and attendants after handling; careful destruction of sputum and other discharges; sterilization of linen of patients; fumigation of rooms after occupancy; and the use of gauze, which can be burned, instead of handkerchiefs. These will be the center of the educational crusade.

Second. To have attached to our dispensaries certain nurses who have received special instruction on nursing and preventing the spread of pneumonia, these to be sent to all pneumonia cases in home-nursing work.

Third. The reporting of all such cases to the health department governing the district where the disease exists and the fumigation of the quarters in which the disease has occurred by the department after the death or recovery of the patient.

Fourth. The instruction of the public by pamphlets and school lectures on the necessity for keeping the noses and throats cleansed, especially during winter months; the necessity for controlling the dust of streets by better sprinkling and night sweeping; the evils of bad ventilation in house, public building, and school; of alcohol; of badly cooked poor food; of lack of rest; of worry; of the handkerchiefs; of the bearing on pneumonia of spitting, as well as on other diseases; of the increased resistance generated by open-air sleeping; and similar knowledge. This I am sure, can best be engrafted on the child's mind rather than on that of the adult.

The Influence of Smoke on Acute and Chronic Lung Infections*

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AND

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A year ago we started an investigation on the influence of smoke on the various problems of public health. We chose for our study the respiratory tract, as that in which smoke would have its greatest influence by direct contact.

The investigation was carried on under a grant given by Mr. R. B. Mellon to the University of Pittsburgh for the consideration of this problem from all its aspects.

At the International Congress of Hygiene and Demography we made a preliminary report of what had been accomplished up to that time, and while the work reported in the former paper was very incomplete, there seemed to be a rough direct ratio between the number of smoky days in any given city and the number of deaths occurring from pneumonia. On the other hand, there seemed to be an inverse ratio between the number of smoky days and the number of deaths from tuberculosis.

In choosing these two diseases, we were guided by the fact that the one (pneumonia) is an acute inflammatory process of short duration, likely to be influenced by acute irritation, such as would come from foreign particles, and that it represents the most striking malady from the standpoint of increased mortality in the greater number of our cities.

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The other (tuberculosis) is a chronic infection, characterized by infiltration and healing by fibrosis, extending over a long period of months or years, and more likely to be stimulated to healing by irritation of inert foreign particles, and yet presenting a constantly decreasing mortality.

Following the presentation of the paper before the Congress of Hygiene and Demography, Dr. John S. Fulton, Secretary of the Congress, called our attention by letter to certain factors, which he felt were of more importance than the simple ratio of smoke to pneumonia mortality, and we have included these in the substance of our present study. The main points in Dr. Fulton's arguments against the conclusions which we tentatively drew from our study were as follow:—

"I would expect Boston to have a greater mortality from pneumonia than Pittsburgh, on the sole basis of fact that Boston has relatively more people in the pneumonia ages. I would expect Chicago to have a higher pneumonia mortality than Pittsburgh, because there is a pneumonia obsession in the minds of the medical profession of Chicago.

"Chicago has relatively more people in the pneumonia ages than Pittsburgh has, and relatively fewer in the pneumonia ages than Boston. I do not think that the pneumonia figures admit of sound reasoning as to magnitude, unless, in the first place, distinctions are made as to the age-distribution of the populations which are to be compared; and, in the second place, unless the pneumonia mortality is divided sharply into two groups, those under and those above the age of three years, and the comparisons made with reference to these distinctions.

"A comparison of pneumonia and tuberculosis magnitudes, as among the cities which you mention, does not prove that the prevalence of tuberculosis in Pittsburgh is low, or that the prevalence of pneumonia is high.

"By mere inspection of those pneumonia charts, without any key to the names of the cities concerned, I would

say that 1, 2, 3, 4, 5, 6 and 7 (Chicago, Pittsburgh, Boston, New Orleans, Richmond, New York and Charleston) are cities fifty years old or older. The last eight (Cincinnati, St. Louis, Philadelphia, San Francisco, Mobile, Cleveland, St. Paul and Portland) are cities less than fifty years of age, and probably situated west of the Alleghenies. I would be right with respect to the first group, and, with respect to the last group, my two errors would be Philadelphia and Mobile."

In choosing the cities for study, we have taken the larger cities scattered widely over the United States, and have tried to get as widely varying conditions from the standpoint of age of settlement, density of population, years of incorporation, flatness of contour as it was possible to obtain.

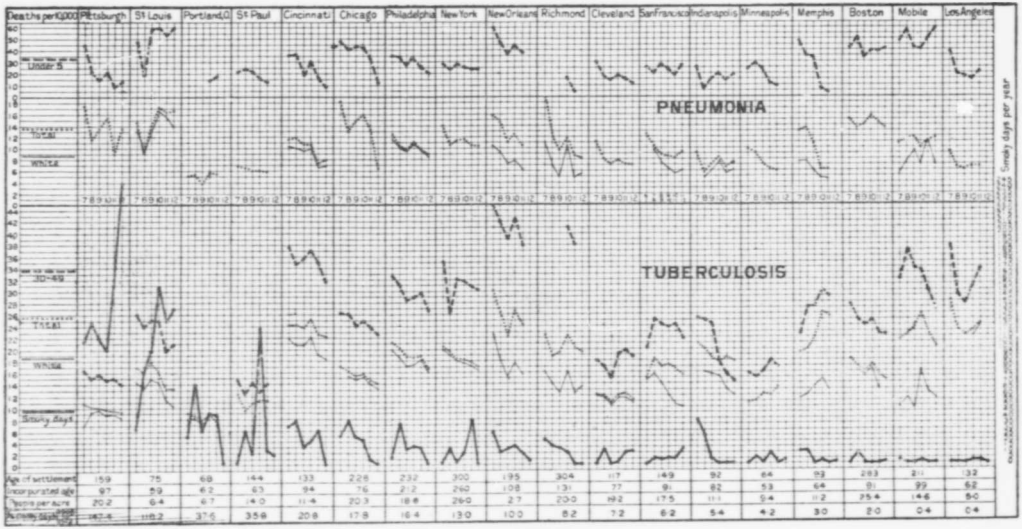
We have also analyzed more carefully the data on which we have completed our present study, and have ruled out as much as possible, in drawing our conclusions, the years and material which were unsafe to use by virtue of their lack of thoroughness.

In the charts accompanying the present study, in the smoke curve we have plotted the number of smoky days per year. In arranging the cities on the charts we have put the smokiest cities first, and so graded on down to the cities in which the smoke was the least. We have considered in arranging them in this way only the 1908 to 1912 periods, preferring to neglect 1907 on account of the unreliability of the mortality data of that year. It will thus be seen that Pittsburgh, St. Louis, Portland, St. Paul, Cincinnati, Chicago, Philadelphia, New Orleans and New York may be grouped as the smoky cities. In this group, however, Pittsburgh, St. Louis, Portland and St. Paul may be classed as very smoky cities.

Beneath these charts the figures represent:—

First (the top line), the number of years since the settlement of the city;

The second line, the number of years since the city was incorporated;


Deaths per 1000
Pittsburgh
St Louis
Portland,O
St Paul
Cincinnati
Chicago
Philadelphia
New York
New Orleans
Richmond
Cleveland
San Francisco
Indianapolis
Minneapolis
Memphis
Boston
Mobile
Los Angeles
Under 5
PNEUMONIA
Total
White
1890
TUBERCULOSIS
30-49
Total
White
Smoky days
Smoky days per year
Age of settlement
Incorporated age
Years per acre
Persons per acre
1890
1900

The third line, the density of population on the basis of the number of people per acre; and the

Fourth line, the average number of smoky days per year for the five-year period, 1908-1912.

In studying the death ratio of the different cities, we plotted the total death-rate and the death-rate of the white population, as well as the following groups:

Total population under 5 years.
" " from 5 to 9 years.
" " " 10 " 19 "
" " " 20 " 29 "
" " " 30 " 49 "
" " " 50 " 69 "
" " " 70 years upwards.

While we plotted all these in the original chart from which this study was made, we have only included a few of them outside the total death-rate in the accompanying charts. In these curves, it is important to bear in mind the history of the registration area of the United States and the great variation in the thoroughness with which vital statistics are kept in the various cities. It was only in 1907 that the registration area approximated 50 per cent. of the population; and only in 1910 was any special attempt made at a uniform method of classification and registration, and this uniform method is not yet in operation in all of the municipalities which we have used. For instance, this classification is not in use in Philadelphia, nor in St. Lous.

In the various curves it will be observed that the year 1907 does not conform to the ratio which has been found to hold in years following this. This may safely be explained by the fact that 1908 was really the beginning of better attempts to place vital statistics upon a more uniform basis; and only in 1910-1911-1912 did we arrive at any real fair basis for comparison.

It must be borne in mind that in comparing with other cities, cities having a large coloured component of population, the mortality by age must be compared on a basis of the ratio of the mortality of the white population to the entire population, because the negro death-rates are much higher than the white.

It will be obvious immediately to anyone who attempts to study this field that the number of smoky days, as furnished by the United States Weather Bureau, is apt to be a very variable quantity, both from the personnel of the recorder and the method by which these readings are made.

The present method of determining the number of smoky days is by fixing the clearness with which certain established objects can be observed from the fixed point of the Observation Bureau. Such factors as the height of the Bureau from the ground, the acuteness of vision of the observer, probably the habits of the observer, the interest which the observer has in the problem, and similar circumstances make it almost impossible to lay down any fixed curve which will be comprehensive for all cities and it would seem a reasonable conclusion that if there is even a rough relation between the conditions which we are studying, it would be safer to say that with more careful figures a more intimate relation could be determined. For instance it is inconceivable that there should be no smoky days for 1910-1911-1912 in a city like Boston, which is largely a manufacturing centre. In the report of the District forecaster for Boston, there have only been two smoky periods in five years. These two periods were five days following September 1, 1908, and five days following October 15 in the same year, and were due to forest fires. While it seems almost incredible that in a manufacturing city, subject to fogs, there should not be more smoky days than is indicated by the forecaster's report, yet it will be noticed that in the Boston curve during this smoky period the pneumonia death- rate- decreased. It is a feature probably not of very serious import that during

that year the pneumonia death-rate fell in all pneumonia groups save that of 5 years of age. This evidence of the reverse influence of the number of smoky days per year on the mortality curve from pneumonia is apparent in several other cities, as for example, Cincinnati.

Between St. Paul and Minneapolis, lying very near together, there is also a marked discrepancy—St. Paul having in 1910 nearly 120 smoky days, and yet the reply from the Forecaster was as follows: "You are informed that said days with record of smoke had reference only to and not to local smoke due to factory chimneys, &c." It seems impossible that St. Paul should be affected with forest fires and Minneapolic not, with a few miles difference in position. This peculiarity of the smoke curve for St. Paul would, of course, put it out of place in this chart, and would explain the difficulty in tracing the same relation which is present in the other cities.

The same thing applies to Portland, from which we have not been able to receive an explanation of the large number of smoky days reported from that city. Portland is probably out of its place in this scheme, as its statistics are not complete.

In summing up these charts, which have been done as impartially as possible, the only constant factor which seems to have any relation is the smoke; in other words, where age of settlement, number of people per acre, and age of incorporation have any apparent influence, this influence must be coupled with the number of smoky days before any satisfactory conclusion can be drawn. It will be seen, then, that if we except Portland and St. Paul, there is a general tendency of the tuberculosis death-rate to rise as the number of smoky days in the city decreases. On the other hand, it will be seen that there is a general tendency for the number of deaths from pneumonia to fall as the number of smoky days in the city decreases. In this instance also, Portland, St. Paul and Boston must be excepted. There seems to be no definite relation, however, between the number of smoky days and the death-rate

under 5 years of age in the pneumonia group. This might readily be expected if we consider as the explanation of the influence of smoke on pneumonia the irritative changes which go on in the mucous membrane of the upper air passages as the underlying factor in this relation, and that these changes would probably take years in their production, or, as Dr. Haythorn has shown, the pneumonia difficulty may be largely one of absorption of exudate, which anthracosis by plugging the lymph spaces largely impedes.

In general, the tuberculosis age-groups are rather uniform in their relation to each other when one comes to the study of individual influences; probably nothing is more striking than the difference between the curve for the total death-rate of the white population as opposed to the coloured. This is most strikingly seen in such southern cities as Memphis, Mobile, New Orleans, and Richmond. There is a striking difference, also, in San Francisco and Los Angeles in the total death-rate from tuberculosis, due, likely, to importation from the middle-west and northern parts of the country.

When one studies individual cities, one finds, as in Pittsburgh, St. Louis, Cincinnati, Chicago, New Orleans, Richmond, &c., a noticeable similarity between the total pneumonia death-rate and the total number of smoky days. This is almost entirely absent in comparing the tuberculosis yearly death-rate, which has persistently dropped in most of the individual cities, save the southern ones, in which there have been curious rises. It is not our intention to enter into explanation of this feature in this paper.

We are at a loss to explain the high mortality rate from tuberculosis in Cincinnati, which seems to be out of its place in the general contour of this chart.

In Boston, in addition to the fact that we believe it out of place from the number of smoky days from a manufacturing standpoint, Dr. Fulton had suggested in his criticism of our former paper that the high pneumonia death-rate in Boston was probably due to the large num-

ber of people in the pneumonia ages (extremes of life). This our age grouping has not demonstrated, as the pneumonia death-rate in all ages is high in Boston. We believe that the factor which is absent in the compilation of this city is the number of smoky days in the year.

Chicago, on the other hand, where Dr. Fulton believes there is a pneumonia obsession in the minds of the physicians, follows very closely what one would expect from the readings of the smoky days. As nearly as we can find, Chicago has been very careful, and since 1910 has forwarded its certificates to Washington, where they have been classified by the Vital Statistics Division of the Census Bureau in order to obviate the reflection of local bias.

We believe that if it were possible to establish a reading of smoky days on the basis which Dr. Benner has established in Pittsburgh, *i. e.*, the precipitation of soot, and have this uniform in the various cities, that we would be able to establish a much more intimate relation between the number of smoky days and the number of pneumonia deaths in any city.

One of the conspicuous things to us in Pittsburgh has been the virulency of the pneumonia infection, which, of course, varies from year to year, but seems to carry its toll off more quickly in Pittsburgh than in any other of the four cities in which I have lived and worked in this field.

It may be well here to again call your attention to the fact that pneumonia is in the main an increasing death-rate in many cities and in the country as a whole; that it takes its victims from the extremes of life; and also takes off many of our most useful middle-aged business men, *i. e.*, many on whom most has been spent in education, at a time when they are most useful to the community; and if it were possible by municipal ordinance to control in some way the production of useless smoke in the cities, much might be done to conserve that on which the community has expended the most, and from which it

may reasonably expect returns in place of death by a rapid illness, such as the one with which we are dealing.

Source of Data.

The mortality statistics are based on reports received through the courtesy of Dr. C. L. Wilbur, Chief of the Division of Vital Statistics of the United States Census Bureau and also through the courtesy of the various Boards of Health of the different cities.

The population statistics and age-distribution for 1910 were obtained through the courtesy of Director E. Dana Durand, of the United States Census Bureau.

The smoke data we obtained through the courtesy of the Chief of the United States Weather Bureau.

Since the Census Bureau report for the year 1900 was as of the population on June 1 while the Census Bureau report for 1910 was as of population on April 15, we utilized the method used by the Census Bureau in estimating the population figures for intercensual years, and after determining the rate of increase, we reduced the estimates of populations to a uniform mid-year basis, *i. e.*, we have them to relate to July 1, with the exception of San Francisco, in which our mortality figures were for the fiscal year. For this city we took the population as of January 1.*

It was necessary to plot population curves to provide a comparison between the population statistics which are furnished on the basis of ten-year periods, starting with five as its unit digit after 35. The mortality statistics, on the other hand, are furnished on the basis of ten-year periods with zero as the unit digit above 30 years of age.

After the population statistics were plotted on this basis, computation was made from these curves for the age periods corresponding to the mortality statistics. The mortality rate per 10,000 was then computed and used in the building up of the curves of the other charts.

* Bulletin 108, p. 9, and Bulletin 109, p. 9, United States Bureau of Census, Department of Commerce.