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FUNDAMENTAL CANCER RESEARCH

Report of a Committee Appointed by the Surgeon General

In accordance with the National Cancer Institute Act, approved August 5, 1937, the purposes of which are set forth as "to provide for, foster, and aid in coordinating research relating to cancer; to establish the National Cancer Institute; and for other purposes," Surgeon General Parran appointed a committee of leading scientists to formulate, as far as this could be done, the fundamental aspects of the cancer problem and to suggest various lines of work which merit investigation.

This committee is composed of the following members:

Dr. Stanhope Bayne-Jones, professor of bacteriology and dean of the school of medicine, Yale University.

Dr. Ross G. Harrison, chairman of the National Research Council and Sterling professor of biology, Yale University.

Dr. Clarence C. Little, director, Roscoe B. Jackson Memorial Laboratory.

Dr. John Northrop, member, Rockefeller Institute for Medical Research.

Dr. James B. Murphy, member, Rockefeller Institute for Medical Research, chairman.

The text of the report prepared by this committee follows.

During the past 30 years of extensive investigation into the problem of cancer, sufficient information has accumulated to justify an attempt at a formulation and clarification of this material that will serve as a basis for future investigation. Three main lines of attack have contributed definite fundamental facts regarding the nature of malignancy, but the difficulty in the past has been that each of these three lines of investigation has been developed independently, taking little account of the knowledge gained in the other fields.

What appear to be the more fundamental data have been selected here and tentative conclusions have been drawn as a basis for discussion. The three lines which have yielded the data for analysis are:

I. The study of transplantable tumors which has yielded information on the biology of the malignant cell;

II. The conditions governing the experimental induction of malignant tumors;

III. The part played by genetic factors in the development of cancer.

I. BIOLOGY OF THE CANCER CELL

Whatever may be the contributing cause, malignancy once acquired becomes a fixed character of the cell. As shown by the study of transplants in animals and in tissue culture, the continuation of the condition is not dependent on the factors which were responsible for its development. The cells will continue their malignant course uninterruptedly without the continued presence of carcinogenic agents in hosts without endocrine disturbance, liver dysfunction, or action of estrogenic hormones. For instance, transplanted mammary gland cancer grows as well in the male and castrated animal as in the female. Not only does the malignant state become a fixed character, but the type, the tendency to distinct histological arrangement, growth rate, invasiveness, and general behavior are more or less constant for each tumor.

All of this points to the conclusion that malignancy once it is established in a cell becomes an automatic process independent of the presence of a continuously acting agent of outside origin, that the new character of the cell becomes a fixed one which is passed unchanged to the descendants.

Attempts to establish fundamental differences between normal and malignant cells of the same tissue type have failed to bring out any striking variation in chemical make-up, enzyme content, metabolism, or structure. Even transplantation is limited by the same laws as those governing normal tissues, and factors which influence the growth of cancer grafts are equally effective in their influence on the growth of normal tissue grafts. It has not been proved that cancer cells are more sensitive to the action of physical agents (heat, cold, X-ray, or radium) than normal cells of the same type. Even function (secretion) may not be interfered with by the development of malignancy. Possible exceptions to these general statements are: (*a*) Individuality differentials may act in transplantability of normal tissues but not in tumors; (*b*) differences in metabolism (oxidation) between tumors and normal tissues.

Mammalian cancer can be transmitted only by grafts of living tumor cells, and the cells of the host do not become malignant by intimate contact with the cells for which it supplies blood vessels and supporting stroma. The very exhaustive study of mammalian cancer has disclosed a complete lack of evidence of its infectious nature. It has been definitely shown that the animal parasites and bacteria, which may incite malignancy in other organisms, play no part in the continuation of the process. The present evidence tends to indicate that the same may be true for the viruses. As causes of the continuation of the malignant process the many microorganisms which have been described as specific etiological agents may be disregarded.

These findings indicate that malignancy is the result of a fundamental change in cell physiology.

II. PRESENT STATUS OF CARCINOGENIC AGENT STUDY

The isolation and identification of the particular group of compounds present in coal tar which are responsible for its carcinogenic action have led to a concentration of interest in the induction of malignant tumors. The structural relationship of these particular hydrocarbons to certain compounds naturally occurring in the living animal has stimulated extensive investigation of the possibility that these natural substances may act, under certain conditions, as carcinogenic agents or may be transformed by some abnormal condition in the body into active carcinogenic substances. Important as these observations are in evaluating the deductions which may be drawn, proper consideration should be given to the fact that there are many other potent carcinogenic agents entirely unrelated chemically to these hydrocarbons. The simple chemicals, such as arsenic and chloride of zinc, may induce malignant changes. The biological agents, parasites (notably tape worms in their cystic form in the liver, Bilharzia infection of the bladder), germs, tubercle bacilli, particularly in infection of the skin, the syphilis organism, notably in mouth infections, may induce malignancy. Recently it has been demonstrated that under special conditions a virus may act in very much the same way as do the chemical carcinogenic agents. That is, it appears to start a chain of events which tend to go over into malignancy; but there is as yet no evidence that the maintenance of malignancy is dependent on the continued presence of the virus.

The first deduction derived from the experimental study of carcinogenic agents, namely, that the process was one of simple chronic irritation, may probably be definitely discarded. The most active agents are practically devoid of irritative action in the strict sense of the word. There is perhaps sufficient evidence to indicate that they are not cell stimulants, but that they actually tend to inhibit the growth of cells.

The general conclusions which may be drawn from this extensive study are as follows: (1) So-called carcinogenic agents appear to start a process which may lead to malignancy; but once the process is started, the agents apparently play no further role in the picture. Examples: X-ray cancer, cancer of the skin from external application of coal tar; the disappearance of active virus from papilloma before malignancy appears. In fact, the malignancy may occur months or even years after the exposure to the carcinogenic agent. (2) Almost all, if not all, classes of cells may be rendered malignant under the influence of one or more agents. Not only may this be concluded from the fact that among naturally occurring tumors practically all types of cells are represented, but malignancy has been induced experimentally in skin, connective tissue, liver, lungs, stomach, gall bladder, kidney, testicle, muscle, bone, bone marrow, lymphoid cells, the uterus, mammary gland, and other tissues. It may be deduced from this that malignancy is a universal cell potentiality. (3) The expectation that with the method of inducing tumors it would be possible to trace the transformation of normal cells into malignant cells has not yet been realized. In the area of tissue disturbance induced by the agents a new race of cells appears quite suddenly with no apparent gradation.

The general systemic effect of carcinogenic agents has not yet received adequate consideration. It has been shown that the simple application of coal tar to the skin of an animal will produce general changes in the internal organs, particularly the lymphoid system and the liver, changes indistinguishable from those produced by a generalized exposure to the X-ray. The application of either of these agents causes a distinct lowering of resistance to transplants of cancer and may break down a highly developed resistance to the growth of cancer cells. Injections of the chemical agents, even in small amounts, cause a marked increase of a natural tendency in certain strains to develop tumors of certain organs (cancer of lung), or in the rabbit, accompanying the liver and lymphoid damage, there is a tendency to tumor formation in the uterus. The active hydrocarbons of coal tar injected into immature animals cause a permanent stunting of growth.

Recently a virus (the Shope virus) has been discovered in cottontail rabbits which produces papillomas and which is capable of inducing similar growths in domestic rabbits. These growths in domestic rabbits enlarge with great rapidity and frequently become cancerous as seldom happens in the cottontails. Yet from the majority of the papillomas of domestic rabbits the virus cannot be recovered, nor has it ever been gotten from the cancer. The present state of the investigation of this interesting material does not disclose the part which the virus plays in cancer etiology.

While the active virus has not been demonstrated in cancers arising in virus-induced papillomas its presence has been indicated indirectly by serological methods. The virus injected intravenously will readily localize in areas of skin previously treated with coal tar and will cause a malignant change much earlier and more frequently than would be the case with tar alone. When acting in this way it appears to be the precipitating factor in cancer. The virus will also localize in cell proliferations caused by noncarcinogenic agents and no malignant changes result. The question is still unsettled as to whether the cancers induced by the virus in tarred skin may not be entirely due to a stimulating effect upon cells already rendered malignant by the tar.

From the virus point of view the fowl tumors still continue to furnish material of the greatest importance. Here the transmitting agents apparently cause a direct transformation of normal into malignant cells, and the continuation of the process seems to depend on the continued presence of the transmitting agent. So far no agents have been procured by the extensive investigation of mammalian tumors with which cancer can be directly transmitted, but the possibility that such a substance or substances do exist in mammalian tumors is certainly worthy of future consideration. The difficulty in their demonstration may be due to the relative impenetrability of the mammalian cell or the agents may be less stable than those from the fowl tumors. A notable property of the group of agents causing tumors of fowls is that they not only stimulate proliferation of cells, but also cause differentiation of the cells into complex tissue organizations. This property, in addition to some others, has opened the question as to whether this group of agents is of endogenous origin. In any case it seems unjustifiable at the present time to draw any conclusions as to tumors in general from the behavior of this group.

III. HEREDITARY FACTORS IN MALIGNANCY

The first contribution in this field was the observation that families of mice having a markedly higher cancer rate than the average for a mixed population could be segregated. The tendency to develop cancer in a given family is confined, for the most part, to an organ or special tissue, so that we have lung cancer families, mammary cancer families, sarcoma families, and the like. This inherited tendency is not that of a generally unstable cell system, as shown by the fact that a strain showing a high cancer rate for the lung or the breast will often prove more refractory to induced cancer of the skin than strains having a very low cancer rate. The tendency for cancers to form in a definite organ or tissue may be accentuated by environmental conditions. For example, mammary gland tumor rate in mice may be increased by intensive breeding, by mammary duct blockage, or by excessive dosage of theelin, and the lung tumor rate may be increased by the surface application or subcutaneous injection of chemical carcinogenic agents. In the latter instance, whether the agents are absorbed and act directly on lung cells or whether the tumors result from the release of the inherited tendency by a lowering of general resistance is undetermined. The present indications are that the type of inheritance is not the same for different strains of tumor. Cancer of the lung appears to be dependent more directly on genetic influence, whereas the inherited tendency to cancer of the breast is transmitted in greater intensity by the female than by the male, which suggests a supplementary hormonal or extrachromosomal influence.

Regardless of the nature of the inheritance, it would appear that the manifestations are the same. The inherited state is an unstable or poorly balanced cell system confined to an organ or tissue type, in which the specific cells tend to become malignant either from functional strain or from unfavorable environmental conditions. This response to physiologic strain is illustrated by the following facts: The females of a family of mice which, under normal breeding conditions, give a high rate of cancer of the breast will show an increased rate if subjected to forced breeding. If the females of such a strain are prevented from breeding the rate may be very low. For example, one strain which gives a rate of 70 to 80 percent among the normally breeding females, showed only 12 percent in the virgin females. Prepuberty castration reduces the rate to a negligible one. The males of such strains castrated in early life and engrafted with ovaries or injected with estrogenic compounds will develop mammary tumors about as often as do the virgin females. Families not inheriting a natural tendency do not develop cancer from overstimulation with estrogenic hormones or breast blockage.

Another example of the variation in threshold effect is found in the lung cancer families where, in hybrids of varying degree of tendency to spontaneous lung cancer, the particular degree of susceptibility is paralleled by the percentage in which lung tumor may be induced by carcinogenic agents.

Whatever the mode of inheritance of the cancer tendency, the condition inherited seems to be a poorly balanced cell system or cells with a higher potentiality for malignancy. The condition responsible for the initiation of the process likewise may be inherited (endocrine unbalance) or it may be an outside agent (lung tumors).

The following formulations or definitions may be tentatively proposed:

1. Malignancy is a universal cell potentiality, in that any cell has inherent in its make-up the potentiality for unlimited or uncontrolled growth.

2. The degree of the potentiality for malignancy is a variable quantity for each tissue or cell type and this degree is determined largely, if not entirely, by hereditary factors.

3. The malignancy potentiality of a cell may be developed in the more sensitive groups by the strain of normal physiological processes but may be set off even in resistant groups by a variety of inciting agents.

4. The change from a normal to a malignant cell represents an alteration in the cell itself by virtue of which proliferation becomes an automatic process independent of the presence of a continuously acting provocative agent.

5. The new property of the cell appears to develop suddenly, becomes a fixed character, and is transmitted to its descendents. It gives evidence of being a somatic mutation.

RESEARCH OBJECTIVES

For the practical purpose of investigation, the cancer question may be divided into two distinct objectives: First, the *causal genesis* of tumors, including the inciting causes leading up to the development of malignancy, and, second, the *formal genesis*, or the factors responsible for the nature of the cancer cell and its tendency for unlimited multiplication. These objectives may be combined or kept distinct from each other, but they should be considered and properly adjusted in the planning and conduct of any experiment.

For the first problem, the causal genesis, there are sufficient data to indicate that there are multiple and diverse causal conditions and that these probably vary for each type of cancer. This diversity of causes is sufficiently distinct to justify investigating the different types almost as if they represented different diseases. For the second, or formal genesis of cancer, if the present conceptions are correct, the changes in the cell responsible for the state give evidence of being the same regardless of the cause. The different manifestations of the disease appear to be dependent on the degree of malignancy, the type of cell affected, and the environment (location, physical condition of the host, and the amount of resistance). The agents concerned in the causal genesis do not at present appear to play any part in continuation of the malignant state or to influence the final outcome.

CAUSAL GENESIS

For the investigation of the causal genesis the following fields are indicated in the light of present knowledge:

Heredity.—Based on the evidence of the prominent part played by the degree of potentiality for malignancy exhibited by cell groups in different strains of animals, the relative importance of heredity warrants intensive investigation with particular stress on the different cancer types. From the practical side this general field has assumed an importance, for it is probable that preventive measures will come from knowledge gained from this type of investigation. Already such leads have been opened up as the demonstration that the mode of inheritance is not the same for all tumor types; that an inherited tendency for endocrine disturbance may be the determining factor in at least one type of cancer; that liver dysfunction may be prominent in another; that induction of mammary tumors by the estrogenic hormones is definitely correlated with the inherited tendency to breast tumor; that, in general, the ease with which a tumor may be induced experimentally depends on the genetic make-up of the host.

In this connection it has become evident that pure strains of animals of known hereditary tendencies are as important for cancer research as pure chemicals are for the chemist.

It is equally evident that the development of this important line of investigation should include continued study of the particular influence and nature of inheritance for each of the major types available for observation. Since experimenters have come to realize that different factors are operative in different types of tumors, it has become progressively more probable that much light may be thrown on the tumor problem.

A comprehensive study of the incidence of cancer in men with reference to both environmental and hereditary factors should be made. Such a study should be based on clinical data and family histories built upon direct observational methods. In the analysis of the hereditary factors advantage should be taken of the recently developed statistical methods for the detection of linkages.

The carcinogenic agents.—There already exists a formidable list of chemical, physical, and biological agents, including viruses, which are known to initiate conditions tending to go into a malignant state. It is a question as to how useful it will be to extend this list unless the effort is directed more toward finding agents which may have some connection with the naturally occurring human tumors. The greatest need in this field is the development of an investigation directed toward the clarification of the mode of action of these diversified agents rather than a search for more agents. However, the possibility exists that some yet undiscovered agent may supply the key to the understanding of the mode of action.

There are two possible leads which might be the entering wedge in the study of this problem of mode of action: One is the fact that several prominent representatives of the chemical group do not act primarily as stimulators of growth but actually inhibit growth even in high dilutions. If this property is common to the various types of agents known to incite cancer (and this should be determined), it should be possible to establish the particular effect on cell metabolism or other factors responsible for this action. It is important to determine whether prolonged inhibition of cells may not tend to produce mutants with excessive growth capacity.

The second lead is based on the fact that another property of several of the carcinogenic agents, perhaps of all of them, is their general effect on the animal body, even when the applications are made on the unbroken skin. These changes are not fully determined but prominent among them are disintegration of the lymphoid tissue, liver damage, uterine changes, and undoubtedly other effects. Among the agents, tar, X-ray, and the pure carcinogenic hydrocarbons have been found to lower an animal's resistance to the growth of cancer cells. Such observations may open up the question as to whether a carcinogenic agent is effective because of a dual action, inciting a local cell derangement and a disturbance of the body's mechanism for dealing with such a disturbance.

Whether or not the foregoing leads prove of importance, there is no question about the advisability of directing an effort toward learning the mode of action of the carcinogenic agents, and also of the importance of keeping the investigation in the general realm of agents which have a part in the naturally occurring disease.

Somewhere between the inherited or acquired cell tendency and the factor which releases this tendency lies the crux of the cancer problem as far as the inception of the disease is concerned.

FORMAL GENESIS

The fundamental problem in cancer still continues to be the formal genesis of the cancer cell. The evidence that whatever may be the part played by the various factors, and however different these factors may be for different tumor types, the end result is the same—a cell with a capacity for unlimited or uncontrolled growth. Have they become "fast" to the conditions which normally control cell growth in the body or is there a break in the internal control mechanism of the cell, or is there a loss in body control of cell activity? This, the core of the problem, has been almost entirely neglected. As there is an apparent lack of appeal to workers it may be necessary to foster the development of an investigation along this line.

This investigation of characteristics of the cancer cell belongs in the field of cell physiology, and the understanding of the process must be dependent on the advance in the understanding of growth and differentiation of normal cells. But, as in general the knowledge of function (for example, the glands of internal secretion) has been gained largely by the study of dysfunction, should not the cancer cell be for the cell physiologist what the acromegalic or the cretin is for the glandular physiologist?

This field requires definite nurturing and it is felt that an important function would be served if this line of investigation were stressed.

The recognized limitations of cancer therapy justify carefully planned and executed animal experimental work aiming at the discovery of new therapeutic agents. Reference is made in this connection to newly discovered artificially produced radio-active isotopes, bacterial filtrates, tissue extracts, and certain synthetic organic chemicals, which may possess therapeutic properties. In order to avoid the many pitfalls, a conservative and critical attitude is particularly essential in work along this line.

The Committee approves the plans of the National Cancer Institute for developing its clinical contacts and recommends that this development proceed, with a view to detecting and utilizing clinical problems for research in the laboratory, and to testing in the clinic promising methods suggested by laboratory experimentation.

In any program for cancer research, patience and the adoption of a long-time point of view are absolutely essential.

STUDIES ON TRICHINOSIS

XII. THE PREPARATION AND USE OF AN IMPROVED TRICHINA ANTIGEN [1]

By JOHN BOZICEVICH, *Associate Zoologist, Division of Zoology, National Institute of Health, United States Public Health Service*

In connection with a series of studies carried on at this institution on various aspects of trichinosis, an attempt was made to effect improvements in the preparation of trichina antigen in order that more reliable results might be obtained in the diagnosis of trichinosis by the precipitin and intradermal tests.

Extracts of parasites have been used extensively for the diagnosis of various parasitic infestations by means of dermal or serological reactions. Bachman (*1*) in 1928 employed an acid hydrolyzed extract of *Trichinella spiralis* as an antigen for use in intradermal and precipitin tests for trichinosis. In 1932, Augustine and Theiler (*2*), using an antigen extracted with Coca's solution, reported results of skin tests for trichinosis. In 1933, McCoy, Miller, and Friedlander (*3*) used an antigen extracted in buffered saline. Later, Trawinski (*4, 5*) noted that antigens prepared with extracting fluids other than physiological saline gave some false readings, particularly when used in connection with the precipitin test; he stated that reactions ob-

[1] In the following are listed the preceding papers of this series:

I. The incidence of trichinosis as indicated by post-mortem examinations of 300 diaphragms. By Maurice C. Hall and Benjamin J. Collins. Pub. Health Rep., 52: 468–490 (Apr. 16, 1937).

II. Some correlations and implications in connection with the incidence of trichinae found in 300 diaphragms. By Maurice C. Hall and Benjamin J. Collins. Pub. Health Rep., 52: 512–527 (Apr. 23, 1937).

III. The complex clinical picture of trichinosis and the diagnosis of the disease. By Maurice C. Hall. Pub. Health Rep., 52: 539–551 (Apr. 30, 1937).

IV. The role of the garbage-fed hog in the production of human trichinosis. By Maurice C. Hall. Pub. Health Rep., 52: 873–886 (July 2, 1937).

V. The incidence of trichinosis as indicated by post-mortem examinations of 1,000 diaphragms. By M. O. Nolan and John Bozicevich. Pub. Health Rep., 53: 652–673 (Apr. 29, 1938).

VI. Epidemiological aspects of trichinosis in the United States, as indicated by an examination of 1,000 diaphragms for trichinae. By Maurice C. Hall. Pub. Health Rep., 53: 1086–1105 (July 1, 1938).

VII. The past and present status of trichinosis in the United States, and the indicated control measures. By Maurice C. Hall. Pub. Health Rep., 53: 1472–1486 (Aug. 19, 1938).

VIII. The antigenic phase of trichinosis. By John Bozicevich. (In manuscript.)

IX. The part of the veterinary profession in the control of human trichinosis. By Willard H. Wright. (In press: J. Am. Vet. Med. Assoc.)

X. The incidence of light infestations of dead trichinae in man. By Leon Jacobs. J. Wash. Acad. Sci., 28: 452–455 (Oct. 15, 1938).

XI. The epidemiology of *Trichinella spiralis* infestations and measures indicated for the control of trichinosis. By Willard H. Wright. (In press: Am. J. Pub. Health.)

tained with such antigens should be interpreted with considerable care. It is apparent, therefore, that various workers have used different extractive fluids with a view to improving the antigen.

METHOD OF PREPARING THE ANTIGEN

Trichina larvae are obtained in the usual manner by infecting rats with trichinous meat and killing the rats 4 to 6 weeks after infection. The rats are skinned and eviscerated and the remainder of the carcass is ground in the meat grinder. The ground trichinous meat is then ready for digestion.

Hobmaier and Meyer (*6*) have described a modification of the Baermann apparatus for the recovery of trichina larvae. For this purpose, the author prefers a funnel of 3-liter capacity, to the stem of which a centrifuge tube is attached by means of a short piece of rubber tubing. A pinchcock is placed on the tubing to permit closing when the centrifuge tube containing the larvae is removed after digestion of the infested meat. A 6-inch perforated porcelain plate, such as is used in a desiccator, is placed in the funnel, and over this plate are laid 4 or 5 layers of cheesecloth having 40 to 44 mesh apertures to the inch.

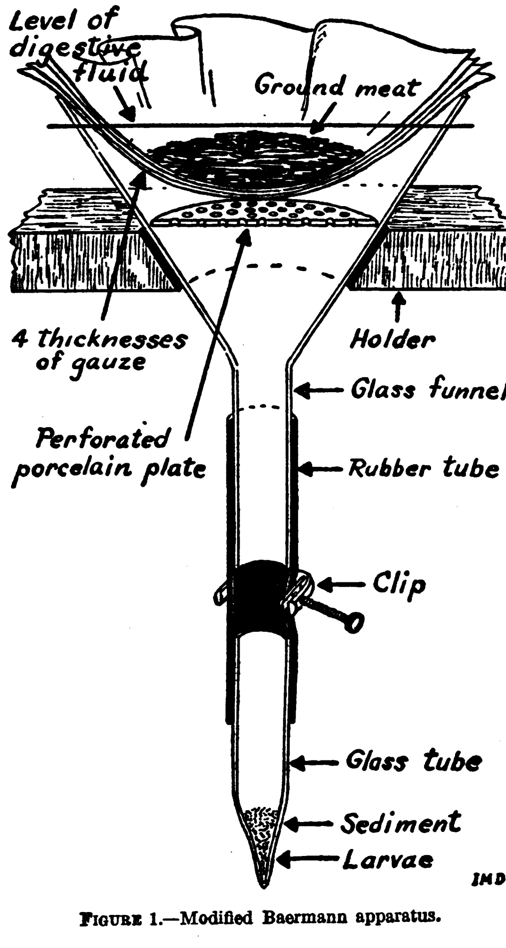

FIGURE 1.—Modified Baermann apparatus.

The digestive fluid is prepared by adding 15 grams of pepsin to 3 liters of warm tap water; this amount is sufficient to digest approximately 70 grams of infested meat. The mixture is stirred until the pepsin dissolves and then 21 cc of HCl (specific gravity 1.10–1.19) are added. The digestive fluid is placed in the modified Baermann apparatus (fig. 1). The meat is added carefully, without agitation, on top of the cheesecloth. The funnel containing these materials is placed in an incubator at 37° C. for a period of 15 to 18 hours. As the larvae are liberated from their cysts by the digestive fluid, they gravitate to the bottom of the centrifuge tube and, by means of their constant agitation, set in motion the fine sediment which settles over them when they become static. As a result, a clean, concentrated collection of larvae may be obtained, thus necessitating only a minimum amount of manipulation in removing rat protein.

At the termination of the incubation period the pinchcock on the rubber tubing is closed and the centrifuge tube removed. The supernatant fluid, along with the layer of sediment, is removed by means of a pipette. In order to neutralize the acid adsorbed from the digestive fluid, a washing solution of a pH 7.5 is used. The larvae are washed three times with this washing solution during a period of 2 hours. After each washing, the larvae are allowed to settle and the supernatant fluid is drawn off. After this, neutral physiological saline is used; at the end of this fourth washing a biuret test is performed on the supernatant fluid and, if the test is negative, the larvae are ready to be dried.

The larvae, with a minimum amount of fluid, are placed in a sausage dialyzing skin 12 inches long and ½ inch in diameter; this is placed in front of an electric fan. Evaporation is completed within 2 to 3 hours, depending upon the amount of fluid in the skin; the larvae are then ground in an agate mortar. Since there is still considerable moisture remaining, the mortar containing the material is placed in a vacuum desiccator containing sulfuric acid or phosphorus pentoxide. After evacuation of the air, drying is continued overnight and, when thoroughly dried, the mass is pulverized and weighed.

The extraction strength is calculated on the basis of the dried powder in a neutral 0.85 percent solution of sodium chloride; a 1:20 dilution is usually the basic dilution used for precipitin work. After an extraction period of 3 to 4 hours at room temperature, the pH of the suspension is adjusted to 7, if necessary, and the extraction is continued in the refrigerator for 15 to 18 hours. Following this, the suspension is centrifuged for one-half hour at the highest available speed. The sediment is discarded and the supernatant fluid is placed in a water bath at 58° C. for 1 hour. It is again centrifuged and the sediment discarded. The pH is rechecked and adjusted, if necessary. At this point a portion of this fraction is set aside for use in making precipitin tests, and the remainder is diluted for skin testing in concentrations of 1:8,000 or 1:10,000, depending on the antigenic strength as determined by the precipitin titer given by a high titered antiserum. A high titered undiluted antiserum will give a good positive reaction with a 1:3,000 dilution of antigen and the solution used for skin testing is diluted to approximately three times the precipitin titer.

The antigen to be used for skin testing is distributed in 2 cc vials in quantities of 0.10 cc, which is more than sufficient for one intradermal test. The vials are hermetically sealed in an oxygen flame. When the vials have cooled to room temperature, they are placed, sealed end down, in a beaker containing water stained with eosin or any suitable dye. The beaker containing the vials is placed in a vacuum desiccator and house vacuum of 500 to 600 mm is applied. In this manner, any deficient seal will be detected readily when air is readmitted to the desiccator, since the stained water will enter any inadequately sealed tubes; such tubes should be discarded.

After checking the seals, the tubes are placed in a water bath at 58° C. for 1 hour. They are then removed and allowed to stand at room temperature for 12 to 15 hours and again are placed in the water bath at 58° C. for a period of 1 hour. Fractional sterilization is continued until sample tubes show no aerobic or anaerobic growth. The sterile tubes are labelled and stored in the refrigerator.

ADVANTAGES OF IMPROVED ANTIGEN

Antigen prepared in the above-described manner has excellent keeping qualities. Samples of this antigen exposed to sunlight and room temperatures for a period of 6 months have shown no loss of potency when tested by the precipitin method. In this connection, Bachman (*7*) reported that the antigen which he prepared lost most of its antigenic properties after 1 month.

McCoy, Miller, and Friedlander (*3*) have reported false positives with trichina antigen when used on subjects infested with other parasites, especially *Trichuris*. Augustine (*8*) has found that the administration of certain drugs, such as arsenicals or quinine, may cause false reactions when serum from individuals treated with such drugs is used for precipitin tests. While the author has not tested such individuals, he has tested numerous subjects harboring infestations with *Trichuris*, *Ascaris*, hookworms, tapeworms (*Hymenolepis nana*), and pinworms, both intradermally and by precipitin, and has obtained no false positives in these cases. The fact that the author's antigen is extracted with a neutral solution without the use of preservatives or added extra salts may explain its marked specificity. Trawinski (*4*, *5*) showed that false positives are frequently obtained in precipitin tests with antigen extracted with Coca's solution. The author has encountered very few reactions of this sort. However, in early stages of experimental trichinosis in rabbits, cloudy serum is often obtained; if an antigen extracted with Coca's solution is used with this serum, false negatives may be encountered at times.

If Bachman's acid hydrolyzed antigen is injected intradermally, control sites may show reactions with extractive fluid alone, provided the pH is not corrected. Bachman (*7*) used an antigen extracted with 0.01N HCl for skin reactions in rabbits, and stated:

It was frequently observed in the control test with the acid, and also with the acidified antigen on normal animals, that a slight necrotic area often developed at the site of the injection which was possibly due to the reaction of the hydrochloric acid on the tissue. Likewise with the control test with Coca's solution slight inflammatory edema developed which was possibly due to the phenol in the Coca's solution. None of the control tests, however, showed the progressive changes observed in the actual antigen tests, and they disappeared in the course of several hours.

If egg albumin or *Ascaris* protein is extracted with Bachman's acid hydrolyzing fluid or with Coca's solution and the resulting products are injected intradermally into rabbits not sensitized to these proteins, with or without sensitization to trichina protein, the sites of injection of either of these products will show larger areas of infiltration than will the sites of injection of the respective extractive fluids alone; moreover, the reactions caused by the protein products will persist for a longer period of time than will the reactions caused by the extractive fluids alone. It is apparent that the use of these two extractive fluids probably results in the formation of certain products which act as tissue irritants. Since trichina protein is probably affected in the same manner, some of the false positives encountered with trichina antigen prepared with these extractive fluids may be attributed to this factor. This is also true, but to a lesser degree, of the buffered saline and phenol antigen as used by McCoy, Miller, and Friedlander (*3*).

In comparing results of intradermal tests on the same animals with the four different types of trichina antigen, namely, those prepared with Bachman's acid hydrolyzing fluid, Coca's solution, the buffered saline of McCoy, Miller, and Friedlander, and saline without preservatives, it is the author's opinion that better and more accurate results can be obtained by the elimination of acid and alkaline solutions, of salts other than saline, and of preservatives. Heat sterilization by the fractional method is superior to filtration through the Berkfeld or Seitz filters since both of these filters adsorb antigen. This may be demonstrated easily by performing precipitin tests on the same serum before and after filtration.

When trichina antigen in a 1:400 dilution is heated for a period of 48 hours at 58° C. no harmful effects or decrease in titer are observed. However, when heated to boiling, a marked decrease in precipitin titer occurs. Furthermore, if the serum is inactivated the precipitin titer is decreased considerably.

METHOD OF PERFORMING THE INTRADERMAL TEST

The following method is recommended for performing the intradermal test and for reading the reaction.

The forearm of the patient, which is the preferred site for making the test, is scrubbed with alcohol and allowed to dry. Using a syringe fitted with a 26-gage, ½ inch needle, 0.01 cc of the 1:10,000 (or 1:8,000) dilution of the antigen is injected intradermally. Since the antigen is prepared with physiological saline solution, a similar solution may be used as a control for the test.

A positive reaction to the intradermal test is of the immediate type and appears usually within 15 to 20 minutes after the injection of the antigen. In rare cases there may be a delayed reaction which does not reach its height before 24 hours. In using the antigen, it is advisable, therefore, to observe the patient at the end of 24 hours, provided the initial reading has been negative. Since no arbitrary standard can be laid down for evaluating the test, judgment must be used in interpreting the reaction. However, it is considered usually that the formation of a wheal, the diameter of which exceeds that of the control wheal by 3 millimeters or more with or without pseudopodia, represents a positive reaction to the test. The wheal is usually surrounded by a zone of erythema, but the amount of erythema is not so important from the standpoint of diagnosis as are the size of the wheal and the presence of pseudopodia.

Instead of using the greatest diameters of the wheal and erythema in determining the extent of the reaction in intradermal tests in animal experiments, a simple method is employed which consists in drawing the picture of the wheal immediately after injection of the antigen and of the control solution. This may be done by placing a piece of cellophane over the site of injection and making an accurate tracing with a fountain pen. The tracing is repeated at intervals and, when the final reading is taken, the areas as outlined on the cellophane can be measured by means of a planimeter. This is a more accurate method than measuring the reaction by means of the greatest diameter and has the advantage of preserving a progressive graphic picture of the reaction.

USE OF TRICHINA ANTIGEN IN AN OUTBREAK OF TRICHINOSIS AT FORT ETHAN ALLEN, VT.

Trichina antigen prepared in the manner described above was used for precipitin and intradermal tests in an outbreak of trichinosis among 44 Civilian Conservation Corps enrollees of the 1136th Company at Camp Charles M. Smith, Vermont, and excellent results were obtained. A complete study of this outbreak was reported by Ferenbaugh, Segal, and Schulze (*9*).

Forty-five patients were hospitalized at Fort Ethan Allen and, of this number, 44 eventually gave positive precipitin and intradermal reactions. One patient gave no reaction to either test and, when questioned, stated that he had not been ill at any time and came to the hospital merely for the purpose of avoiding work.

Blood samples were taken from the patients for the precipitin test, and 2 hours later skin tests were given. Of the 44 patients, 6 failed to react positively to the first precipitin test made 26 days after the date of infestation as established by Ferenbaugh, Segal, and Schulze. At this time, 4 patients did not show an immediate reaction to the intradermal test. However, 2 of the 4 cases gave a delayed type of intradermal reaction which appeared within 24 hours and the other 2 gave positive skin reactions when the intradermal tests were repeated several days later. It is possible, of course, that these 2 patients may have been sensitized by the first injection and that the results of the second test were influenced by the previous one. The 6 patients who failed to react positively to the initial precipitin test gave positive intradermal reactions and the 4 who failed to react positively to the first intradermal test gave positive precipitin reactions. This demonstrates that results of precipitin and intradermal tests must be interpreted judiciously, and that clinical symptoms and other factors must be taken into consideration in attempting to establish a diagnosis. Because the blood serum of patients in early stages of trichinosis contains considerable chyle, or is very cloudy, the precipitin reaction may be concealed and great care must be exercised in reading these reactions. Certain factors, such as the one involving the appearance of precipitins in several cases before a positive intradermal reaction, are difficult to explain. As a rule, intradermal reactions precede precipitin reactions by 2 or 3 days.

Larvae were found on biopsy in 2 of the enrollees who gave delayed skin reactions. Thirty days after the infestation all 44 cases gave positive skin and precipitin reactions.

Twenty enrollees of 136 who remained at camp, some of whom gave a history of trichinosis, were positive to intradermal and precipitin reactions for trichinosis. Nine of the twenty in this group gave reactions to antigen prepared from *Ascaris lumbricoides*. The intradermal tests with *Ascaris* antigen were performed by Dr. H. E. Medivetsky, Medical Department, University of Vermont Medical School, at the same time that the trichinosis intradermal tests were performed. He obtained also 22 positive reactions for *Ascaris* in the hospitalized trichinosis patients. There were 19 negative reactions to *Ascaris* antigen in this group; 4 of the patients received no *Ascaris* tests. Of the 5 enrollees who were positive for trichinae on biopsy, 3 gave positive reactions to *Ascaris* antigen, but all 5 gave positive precipitin and intradermal reactions with trichina antigen.

It is believed by some that there is a group reacting factor in nematode protein and that for this reason *Ascaris* antigen should give reactions in trichina infestations and vice versa. However, the results of the intradermal tests with trichina and *Ascaris* antigens on these patients clearly demonstrate that there is a species specificity. It may be that if the *Ascaris* antigen had been diluted in a manner equivalent to the dilution of the trichina antigen (1:10,000) no reactions with *Ascaris* antigen would have been obtained unless there had been previous contact with *Ascaris* protein.

The intensity of the intradermal and precipitin reactions cannot be taken as an index of the degree of infestation in trichinosis. For example, it was found in comparing the number of larvae found on biopsy that a patient who harbored 50 larvae per gram of muscle gave as great a reaction by the intradermal and precipitin tests as did one who harbored 800 larvae per gram of muscle.

In addition to the tests made on the trichinosis patients, 50 Civilian Conservation Corps enrollees in the 1184th Company at Fort Ethan Allen, Vt., were tested for trichinosis by the intradermal method; these individuals were used as controls, since, so far as known, they had not been exposed to trichinosis. Of this control group, two gave slightly positive intradermal reactions, but negative precipitin reactions. Individuals in this control group were approximately of the same ages and social-economic status as those in the 1136th Company at the Charles M. Smith Camp. When compared with the incidence of trichina infestation disclosed by studies in the National Institute of Health, this finding of 2 positives among 50 persons is low for this particular age group.

SUMMARY AND CONCLUSIONS

Methods are described for the recovery of trichina larvae with a minimum of debris, and for the preparation of trichina antigen by extraction with a neutral 0.85 percent solution of sodium chloride without the use of preservatives. Antigen prepared in this manner and sterilized fractionally by heat undergoes no deterioration and shows no loss of titer even when maintained in sunlight and at room temperature for 6 months. Such antigen may be put up in hermetically sealed vials and stored until needed for use.

The value of the saline-extracted antigen was compared by means of precipitin and intradermal tests with that of antigen prepared by three other methods of extraction, and the saline-extracted antigen was found superior as a diagnostic agent to the other types of antigen.

The saline-extracted antigen was used for precipitin and intradermal tests on 44 cases of trichinosis among Civilian Conservation Corps enrollees hospitalized at Fort Ethan Allen, Vt. All the patients eventually reacted positively to both tests. However, since there is considerable variability in the time of appearance of fixed and circulating antibodies in clinical cases of trichinosis, too much reliance should not be placed on a single intradermal or precipitin test in diagnosing suspected cases of the disease. Evidence indicates that clinical symptoms, the differential blood picture, and other factors should be taken into consideration in establishing a diagnosis.

Forty-one of the 44 trichinosis patients were tested intradermally with an antigen prepared from *Ascaris lumbricoides*. While 22 of the patients reacted positively to the relatively concentrated *Ascaris* antigen, reasons are given to indicate that there was in this case a species specificity and not a group nematode specificity as believed by some writers.

A convenient method for measuring intradermal reactions on experimental animals is described. The progressive development of the reaction is followed by means of cellophane tracings which can afterwards be measured by means of a planimeter. By this method a progressive graphic picture of the reaction may be preserved for further reference.

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DEATHS DURING WEEK ENDED NOVEMBER 12, 1938

[From the Weekly Health Index, issued by the Bureau of the Census, Department of Commerce]

	Week ended Nov. 12, 1938	Corresponding week, 1937
Data from 88 large cities of the United States:		
Total deaths	7,361	[1] 8,122
Average for 3 prior years	[1] 7,859	
Total deaths, first 45 weeks of year	363,942	389,155
Deaths under 1 year of age	429	[1] 514
Average for 3 prior years	[1] 506	
Deaths under 1 year of age, first 45 weeks of year	23,572	25,036
Data from industrial insurance companies:		
Policies in force	68,295,010	69,931,141
Number of death claims	7,752	11,069
Death claims per 1,000 policies in force, annual rate	5.9	8.3
Death claims per 1,000 policies, first 45 weeks of year, annual rate	9.2	9.7

[1] Data for 86 cities.

PREVALENCE OF DISEASE

No health department, State or local, can effectively prevent or control disease without knowledge of when, where, and under what conditions cases are occurring

UNITED STATES

CURRENT WEEKLY STATE REPORTS

These reports are preliminary, and the figures are subject to change when later returns are received by the State health officers.

In these and the following tables, a zero (0) indicates a positive report and has the same significance as any other figure, while leaders (------) represent no report, with the implication that cases or deaths may have occurred but were not reported to the State health officer.

Cases of certain diseases reported by telegraph by State health officers for the week ended Nov. 19, 1938, rates per 100,000 population (annual basis), and comparison with corresponding week of 1937 and 5-year median

Division and State	Diphtheria				Influenza				Measles			
	Nov. 19, 1938, rate	Nov. 19, 1938, cases	Nov. 20, 1937, cases	1933-37 median	Nov. 19, 1938, rate	Nov. 19, 1938, cases	Nov. 20, 1937, cases	1933-37 median	Nov. 19, 1938, rate	Nov. 19, 1938, cases	Nov. 20, 1937, cases	1933-37 median
NEW ENG.												
Maine	37	6	1	2	18	3	1	1	280	46	28	18
New Hampshire	0	0	2	0	------	------	------	------	------	------	42	12
Vermont	0	0	2	1	------	------	------	------	27	2	44	4
Massachusetts	6	5	5	9	------	------	------	------	209	177	82	82
Rhode Island	0	0	0	2	------	------	------	------	------	------	2	2
Connecticut	12	4	8	3	9	3	4	4	186	62	5	22
MID. ATL.												
New York	10	24	25	32	[1] 8	[1] 11	[1] 11	[1] 11	127	315	111	287
New Jersey	16	13	17	20	12	10	7	9	22	18	262	41
Pennsylvania	28	54	33	57	------	------	------	------	34	66	1,032	138
E. NO. CEN.												
Ohio	36	46	46	83	------	------	6	32	12	15	119	63
Indiana	20	13	32	72	5	3	23	23	27	18	16	16
Illinois	30	46	44	55	18	27	10	22	21	32	368	21
Michigan [2]	31	29	36	26	------	------	1	1	58	54	78	46
Wisconsin	4	2	2	5	59	33	33	31	175	98	56	56
W. NO. CEN.												
Minnesota	14	7	13	13	4	2	1	1	307	156	3	45
Iowa	49	24	2	13	6	3	------	------	102	50	5	5
Missouri	38	29	55	64	5	4	41	41	9	7	588	31
North Dakota	66	9	1	5	30	4	------	2	2,873	389	5	11
South Dakota	23	3	2	4	15	2	------	------	324	43	------	4
Nebraska	23	6	1	12	4	1	------	------	4	1	2	6
Kansas	36	13	14	26	22	8	1	1	31	11	19	8

See footnotes at end of table.

Cases of certain diseases reported by telegraph by State health officers for the week ended Nov. 19, 1938, rates per 100,000 population (annual basis), and comparison with corresponding week of 1937 and 5-year median—Continued

Division and State	Diphtheria: Nov. 19, 1938, rate	Diphtheria: Nov. 19, 1938, cases	Diphtheria: Nov. 20, 1937, cases	Diphtheria: 1933–37 median	Influenza: Nov. 19, 1938, rate	Influenza: Nov. 19, 1938, cases	Influenza: Nov. 20, 1937, cases	Influenza: 1933–37 median	Measles: Nov. 19, 1938, rate	Measles: Nov. 19, 1938, cases	Measles: Nov. 20, 1937, cases	Measles: 1933–37 median
SO. ATL.												
Delaware	20	1	0	1	—	—	—	—	40	2	—	—
Maryland [3]	43	14	41	26	22	7	5	5	174	56	3	8
Dist. of Col	91	11	5	11	17	2	—	—	17	2	5	1
Virginia	164	85	81	72	227	118	—	—	71	37	73	28
West Virginia	34	12	31	42	28	10	21	32	48	17	47	23
North Carolina [3]	175	117	80	80	10	7	2	7	290	194	222	94
South Carolina [3]	44	16	12	15	790	284	214	313	11	4	10	10
Georgia [3]	24	14	23	45	52	31	—	—	22	13	—	—
Florida	37	12	15	16	9	3	5	1	100	32	55	4
E. SO. CEN.												
Kentucky	61	34	25	44	37	21	4	4	21	12	62	7
Tennessee [3]	40	22	40	61	68	38	47	47	11	6	95	19
Alabama [3]	56	31	33	45	99	55	116	31	22	12	6	6
Mississippi [2] [3]	36	14	13	19	—	—	—	—	—	—	—	—
W. SO. CEN.												
Arkansas	74	29	21	16	176	69	28	13	38	15	8	1
Louisiana	44	18	27	27	7	3	3	6	122	50	—	5
Oklahoma	63	31	34	25	117	57	15	42	39	19	4	4
Texas [3]	71	84	61	61	186	220	237	127	4	5	44	15
MOUNTAIN												
Montana	10	1	2	2	—	—	—	4	1,093	113	23	22
Idaho	0	0	4	0	—	—	5	3	582	55	31	4
Wyoming	0	0	0	0	—	—	—	—	89	4	—	4
Colorado	78	16	7	7	107	22	—	—	54	11	24	3
New Mexico	74	6	6	6	—	—	2	—	37	3	40	19
Arizona	51	4	13	5	147	116	41	32	25	2	1	2
Utah [3]	30	3	54	0	60	6	—	—	70	7	17	17
PACIFIC												
Washington	25	8	2	1	6	2	—	—	47	15	30	55
Oregon	15	3	8	1	56	11	27	27	41	8	16	18
California	29	34	51	53	28	33	34	37	380	449	47	50
Total	38	953	980	1,309	60	1,229	945	970	111	2,703	3,730	2,229
46 weeks	22	25,448	23,718	31,702	60	56,018	282,053	147,875	691	775,362	260,293	353,300

Division and State	Meningitis, meningococcus: Nov. 19, 1938, rate	Meningitis, meningococcus: Nov. 19, 1938, cases	Meningitis, meningococcus: Nov. 20, 1937, cases	Meningitis, meningococcus: 1933–37 median	Poliomyelitis: Nov. 19, 1938, rate	Poliomyelitis: Nov. 19, 1938, cases	Poliomyelitis: Nov. 20, 1937, cases	Poliomyelitis: 1933–37 median	Scarlet fever: Nov. 19, 1938, rate	Scarlet fever: Nov. 19, 1938, cases	Scarlet fever: Nov. 20, 1937, cases	Scarlet fever: 1933–37 median
NEW ENG.												
Maine	0	0	0	0	0	0	0	1	24	4	30	20
New Hampshire	0	0	0	0	0	0	0	1	72	7	12	10
Vermont	0	0	0	0	14	1	0	0	54	4	15	9
Massachusetts	0	0	3	2	0	0	3	2	85	72	126	126
Rhode Island	8	1	0	0	0	0	1	0	38	5	31	12
Connecticut	0	0	1	1	0	0	0	0	126	42	61	38
MID. ATL.												
New York	1.2	3	3	5	0.8	2	7	7	100	249	348	328
New Jersey	0	0	1	2	2.4	2	2	2	102	85	85	95
Pennsylvania	3	6	2	2	2	4	0	3	160	312	340	395

See footnotes at end of table.

Cases of certain diseases reported by telegraph by State health officers for the week ended Nov. 19, 1938, rates per 100,000 population (annual basis), and comparison with corresponding week of 1937 and 5-year median—Continued

Division and State	Meningitis, meningococcus				Poliomyelitis				Scarlet fever			
	Nov. 19, 1938, rate	Nov. 19, 1938, cases	Nov. 20, 1937, cases	1933-37 median	Nov. 19, 1938, rate	Nov. 19, 1938, cases	Nov. 20, 1937, cases	1933-37 median	Nov. 19, 1938, rate	Nov. 19, 1938, cases	Nov. 20, 1937, cases	1933-37 median
E. NO. CEN.												
Ohio	0	0	4	4	0	0	0	0	193	249	225	441
Indiana	1.5	1	0	1	0	0	1	1	225	150	139	176
Illinois	0.7	1	4	6	0.7	1	4	3	190	287	355	381
Michigan [2]	0	0	2	2	1.1	1	5	5	457	423	417	252
Wisconsin	0	0	0	0	1.8	1	1	2	219	123	181	203
W. NO. CEN.												
Minnesota	0	0	2	1	0	0	2	2	165	84	135	121
Iowa	0	0	2	1	0	0	4	2	143	70	175	84
Missouri	0	0	1	1	2.6	2	4	2	149	114	176	125
North Dakota	0	0	0	0	0	0	0	0	177	24	43	48
South Dakota	0	0	0	0	0	0	1	0	256	34	34	34
Nebraska	0	0	2	0	0	0	3	3	96	25	35	35
Kansas	0	0	0	0	0	0	2	1	378	135	118	118
SO. ATL.												
Delaware	0	0	0	0	20	1	0	0	180	9	12	6
Maryland [2]	0	0	0	1	0	0	1	1	124	40	90	90
Dist. of Col	0	0	3	3	0	0	0	0	83	10	19	17
Virginia	6	3	3	1	4	2	0	1	116	60	45	74
West Virginia	0	0	4	1	0	0	0	0	240	86	102	125
North Carolina [3]	1.5	1	2	2	1.5	1	0	1	108	72	66	94
South Carolina [3]	2.8	1	1	0	6	2	0	0	36	13	12	11
Georgia [3]	1.7	1	1	0	1.7	1	1	1	64	38	32	32
Florida	3	1	1	0	3	1	3	0	69	22	0	2
E. SO. CEN.												
Kentucky	11	6	5	0	1.8	1	0	2	171	96	69	69
Tennessee [3]	4	2	9	3	1.8	1	0	3	164	91	64	92
Alabama [3]	9	5	6	1	0	0	3	1	47	26	20	31
Mississippi [2] [3]	0	0	2	0	2.6	1	7	1	28	11	12	19
W. SO. CEN.												
Arkansas	2.5	1	0	0	2.5	1	3	1	74	29	20	15
Louisiana	0	0	1	1	0	0	1	2	64	26	21	20
Oklahoma	0	0	1	0	2	1	1	1	94	46	59	23
Texas [3]	0	0	1	1	0.8	1	2	2	82	97	113	66
MOUNTAIN												
Montana	10	1	1	0	0	0	0	0	252	26	32	32
Idaho	0	0	0	0	11	1	0	0	137	13	21	21
Wyoming	0	0	0	0	0	0	0	0	111	5	11	15
Colorado	0	0	0	0	0	0	4	4	136	28	32	42
New Mexico	0	0	0	0	0	0	0	0	247	20	30	26
Arizona	0	0	0	0	0	0	0	0	63	5	5	17
Utah [2]	0	0	0	0	0	0	1	0	121	12	65	31
PACIFIC												
Washington	0	0	0	0	0	0	2	2	135	43	39	39
Oregon	5	1	0	0	0	0	4	3	213	42	24	39
California	0.8	1	1	2	0.8	1	12	12	177	209	180	211
Total	1.5	36	69	63	1.2	30	85	91	148	3,673	4,276	4,588
46 weeks	2.3	2,589	4,930	4,930	1.4	1,596	9,187	6,962	144	164,148	195,700	195,700

See footnotes at end of table.

Cases of certain diseases reported by telegraph by State health officers for the week ended Nov. 19, 1938, rates per 100,000 population (annual basis), and comparison with corresponding week of 1937 and 5-year median—Continued

Division and State	Smallpox				Typhoid and paratyphoid fever				Whooping cough		
	Nov. 19, 1938, rate	Nov. 19, 1938, cases	Nov. 20, 1937, cases	1933–37 median	Nov. 19, 1938, rate	Nov. 19, 1938, cases	Nov. 20, 1937, cases	1933–37 median	Nov. 19, 1938, rate	Nov. 19, 1938, cases	Nov. 20, 1937, cases
NEW ENG.											
Maine	0	0	0	0	6	1	1	1	116	19	34
New Hampshire	0	0	0	0	0	0	0	0	0	0	7
Vermont	0	0	0	0	0	0	2	1	0	0	7
Massachusetts	0	0	0	0	1	1	2	2	158	134	130
Rhode Island	0	0	0	0	15	2	1	1	314	41	53
Connecticut	0	0	0	0	12	4	2	1	195	65	60
MID. ATL.											
New York	0	0	0	0	3	8	8	12	265	659	392
New Jersey	0	0	0	0	1	1	4	5	473	394	98
Pennsylvania	0	0	0	0	16	31	19	23	224	438	------
E. NO. CEN.											
Ohio	1	1	3	1	9	12	3	10	118	152	106
Indiana	15	10	21	2	2	1	4	4	21	14	22
Illinois	1	2	6	1	11	16	13	14	332	501	93
Michigan [3]	15	14	4	0	9	8	4	9	318	295	164
Wisconsin	4	2	2	16	2	1	1	1	775	435	170
W. NO. CEN.											
Minnesota	8	4	8	5	0	0	2	1	73	37	43
Iowa	6	3	24	3	6	3	0	3	59	29	30
Missouri	54	41	4	4	3	2	5	6	29	22	63
North Dakota	89	12	32	2	22	3	0	2	59	8	34
South Dakota	0	0	2	2	8	1	1	1	38	5	34
Nebraska	0	0	1	1	4	1	2	0	69	18	12
Kansas	8	3	2	1	3	1	1	5	56	20	70
SO. ATL.											
Delaware	0	0	0	0	0	0	2	2	120	6	6
Maryland [3]	0	0	0	0	16	5	5	12	106	34	100
Dist. of Col.	0	0	0	0	8	1	0	1	75	9	5
Virginia	0	0	0	0	13	7	4	7	85	44	66
West Virginia	0	0	0	0	17	6	11	11	101	36	40
North Carolina [3]	0	0	0	0	15	10	2	4	406	272	152
South Carolina [3]	0	0	0	0	8	3	2	4	97	35	28
Georgia [3]	0	0	0	0	22	13	5	6	25	15	16
Florida	0	0	1	0	19	6	1	3	53	17	8
E. SO. CEN.											
Kentucky	18	10	5	0	21	12	9	14	46	26	93
Tennessee [3]	2	1	6	1	9	5	4	11	41	23	45
Alabama [3]	0	0	0	0	5	3	5	7	79	44	12
Mississippi [2] [3]	0	0	2	0	3	1	4	5	------	------	------
W. SO. CEN.											
Arkansas	3	1	9	0	8	3	22	4	38	15	18
Louisiana	0	0	3	1	44	18	13	11	20	8	6
Oklahoma	8	4	2	1	27	13	10	11	14	7	28
Texas [3]	0	0	2	2	27	32	46	46	65	77	136
MOUNTAIN											
Montana	19	2	17	8	58	6	2	3	348	36	23
Idaho	0	0	13	1	85	8	2	2	21	2	22
Wyoming	0	0	13	2	0	0	0	0	22	1	7
Colorado	5	1	3	7	5	1	0	0	209	43	7
New Mexico	0	0	0	0	62	5	10	10	111	9	74
Arizona	13	1	0	0	76	6	1	1	25	2	------
Utah [3]	10	1	0	0	10	1	0	0	251	25	16

See footnotes at end of table.

Cases of certain diseases reported by telegraph by State health officers for the week ended Nov. 19, 1938, rates per 100,000 population (annual basis), and comparison with corresponding week of 1937 and 5-year median—Continued

Division and State	Smallpox				Typhoid and paratyphoid fever				Whooping cough		
	Nov. 19, 1938, rate	Nov. 19, 1938, cases	Nov. 20, 1937, cases	1933–37 median	Nov. 19, 1938, rate	Nov. 19, 1938, cases	Nov. 20, 1937, cases	1933–37 median	Nov. 19, 1938, rate	Nov. 19, 1938, cases	Nov. 20, 1937, cases
PACIFIC											
Washington	3	1	10	10	16	5	1	3	198	63	81
Oregon	41	8	17	0	15	3	0	1	5	1	32
California	2	2	3	1	8	9	6	9	92	108	245
Total	5	124	215	85	11	279	242	327	174	4,244	2,888
46 weeks	12	13,395	9,316	6,313	12	13,408	14,121	16,271	167	187,136	------

[1] New York City only.
[2] Period ended earlier than Saturday.
[3] Typhus fever, week ended Nov. 19, 1938, 56 cases as follows: North Carolina, 2; South Carolina, 2; Georgia, 28; Tennessee, 1; Alabama, 7; Mississippi, 3; Texas, 13.

SUMMARY OF MONTHLY REPORTS FROM STATES

The following summary of cases reported monthly by States is published weekly and covers only those States from which reports are received during the current week:

State	Meningitis, meningococcus	Diphtheria	Influenza	Malaria	Measles	Pellagra	Poliomyelitis	Scarlet fever	Smallpox	Typhoid and paratyphoid fever
October 1938										
Alabama	10	277	144	1,468	34	35	11	152	3	35
California	2	122	47	70	875	4	6	562	18	44
Florida	3	51	6	70	45	26	2	32	0	12
Georgia	0	310	166	519	23	85	5	124	0	34
Idaho	0	1	11	--------	78	--------	1	47	4	11
Indiana	4	165	62	23	33	--------	6	496	29	27
Iowa	2	74	36	--------	51	--------	6	205	4	17
Kentucky	9	213	77	23	30	2	8	325	0	74
Louisiana	3	99	10	60	78	9	0	52	0	49
Maryland	5	40	22	4	95	--------	4	100	0	59
Michigan	10	84	1	6	179	--------	8	1,169	19	25
Minnesota	0	42	14	--------	301	--------	3	260	16	10
Nevada	0	0	--------	--------	7	--------	0	7	0	0
New Mexico	1	35	14	1	70	2	1	59	0	32
New York	16	66	--------	12	347	--------	15	641	0	71
Ohio	11	236	54	5	59	--------	6	999	0	53
Rhode Island	0	4	--------	--------	2	--------	0	21	0	9
Tennessee	7	248	187	198	22	25	4	288	0	47
Texas	5	278	543	638	--------	121	5	302	--------	173

Summary of monthly reports from States—Continued

October 1938

	Cases
Actinomycosis:	
Michigan	1
Anthrax:	
California	1
Louisiana	1
New York	1
Botulism:	
California	1
Chickenpox:	
Alabama	19
California	790
Florida	24
Georgia	34
Idaho	34
Indiana	82
Iowa	81
Kentucky	187
Louisiana	4
Maryland	100
Michigan	927
Minnesota	203
Nevada	2
New Mexico	12
New York	1,059
Ohio	978
Rhode Island	23
Tennessee	61
Conjunctivitis:	
Georgia	3
Idaho	1
New Mexico	1
Dengue:	
Georgia	7
Diarrhea:	
Maryland	96
New Mexico	5
Ohio (under 2 years; enteritis included)	96
Dysentery:	
Alabama (amoebic)	4
California (amoebic)	16
California (bacillary)	75
Florida (amoebic)	51
Florida (bacillary)	23
Georgia (amoebic)	15
Georgia (bacillary)	8
Iowa (bacillary)	1
Kentucky (bacillary)	14
Louisiana (amoebic)	1
Louisiana (bacillary)	1
Maryland (amoebic)	1
Maryland (bacillary)	67
Michigan (amoebic)	2
Michigan (bacillary)	27
Minnesota (amoebic)	3
New Mexico (amoebic)	2
New Mexico (bacillary)	9
New Mexico (unspecified)	5
New York (amoebic)	10
New York (bacillary)	142
Ohio (bacillary)	9
Tennessee (amoebic)	3
Tennessee (bacillary)	19
Encephalitis, epidemic or lethargic:	
Alabama	1
California	11
Florida	1
Indiana	1
Iowa	5
Kentucky	1
Louisiana	2
Michigan	2
Minnesota	6
New York	7
Tennessee	1
Texas	2
Food poisoning:	
California	58
New Mexico	2
German measles:	
California	93
Idaho	3
German measles—Con.	
Kentucky	1
Maryland	3
Michigan	33
New York	55
Ohio	14
Rhode Island	1
Tennessee	4
Granuloma, coccidioidal:	
California	8
Hookworm disease:	
Florida	157
Georgia	1,902
Louisiana	3
Impetigo contagiosa:	
Maryland	68
Tennessee	24
Jaundice:	
Maryland	6
Michigan	4
Lead poisoning:	
Ohio	6
Leprosy:	
California	1
Louisiana	1
Mumps:	
Alabama	33
California	1,198
Florida	11
Georgia	36
Idaho	9
Indiana	29
Iowa	32
Kentucky	22
Maryland	84
Michigan	149
Nevada	2
New Mexico	7
Ohio	290
Rhode Island	21
Tennessee	28
Ophthalmia neonatorum:	
California	3
Louisiana	1
New York [1]	3
Ohio	81
Tennessee	2
Puerperal septicemia:	
Ohio	3
Tennessee	2
Rabies in animals:	
Alabama	38
California	94
Florida	3
Indiana	29
Iowa	2
Louisiana	16
Minnesota	1
New York [1]	1
Rhode Island	1
Rabies in man:	
Alabama	1
Relapsing fever:	
California	3
Rocky Mountain spotted fever:	
Indiana	1
New York	1
Ohio	1
Septic sore throat:	
California	2
Florida	6
Georgia	34
Idaho	2
Indiana	1
Iowa	3
Kentucky	15
Louisiana	7
Maryland	25
Michigan	5
Minnesota	19
New Mexico	7
New York	88
Septic sore throat—Con.	
Ohio	72
Rhode Island	5
Tennessee	18
Tetanus:	
Alabama	9
California	5
Florida	3
Georgia	1
Kentucky	1
Louisiana	6
Michigan	2
Minnesota	1
New York	6
Trachoma:	
California	64
Florida	6
Kentucky	3
Tennessee	2
Trichinosis:	
California	5
Michigan	5
New York	6
Tularaemia:	
Florida	1
Georgia	3
Iowa	1
Louisiana	2
Michigan	1
Nevada	2
New Mexico	1
Ohio	2
Tennessee	1
Typhus fever:	
Alabama	60
California	2
Florida	8
Georgia	112
Louisiana	2
Michigan	1
New York	2
Tennessee	3
Undulant fever:	
Alabama	4
California	24
Florida	6
Georgia	2
Indiana	3
Iowa	12
Kentucky	2
Louisiana	1
Maryland	8
Michigan	18
Minnesota	11
New York	19
Ohio	8
Tennessee	4
Vincent's infection:	
Florida	7
Idaho	1
Maryland	5
Michigan	12
New York [1]	75
Tennessee	15
Whooping cough:	
Alabama	95
California	576
Florida	83
Georgia	70
Idaho	9
Indiana	107
Iowa	61
Kentucky	116
Louisiana	38
Maryland	94
Michigan	910
Minnesota	147
Nevada	9
New Mexico	49
New York	1,784
Ohio	478
Rhode Island	132
Tennessee	188

[1] Exclusive of New York City.

CASES OF VENEREAL DISEASES REPORTED FOR SEPTEMBER 1938

These reports are published monthly for the information of health officers in order to furnish current data as to the prevalence of the venereal diseases. The figures are taken from reports received from State and city health officers. They are preliminary and are therefore subject to correction. It is hoped that the publication of these reports will stimulate more complete reporting of these diseases.

Reports from States

	Syphilis		Gonorrhea	
	Cases reported during month	Monthly case rates per 10,000 population	Cases reported during month	Monthly case rates per 10,000 population
Alabama	1,824	6.30	247	0.85
Arizona	108	2.62	185	4.49
Arkansas	1,156	5.64	361	1.76
California	1,802	2.93	1,435	2.33
Colorado	127	1.19	84	.78
Connecticut	145	.83	120	.69
Delaware	308	11.80	53	2.03
District of Columbia	522	8.33	407	6.49
Florida [1]	767	4.59	87	.52
Georgia	1,986	6.44	494	1.60
Idaho	19	.39	20	.41
Illinois	2,443	3.10	1,441	1.83
Indiana	352	1.01	116	.33
Iowa	237	.93	201	.79
Kansas	155	.83	85	.46
Kentucky	880	3.01	329	1.13
Louisiana	886	4.16	124	.58
Maine	34	.40	57	.67
Maryland	1,027	6.12	317	1.89
Massachusetts	516	1.17	529	1.20
Michigan	1,183	2.45	661	1.37
Minnesota	266	1.00	209	.79
Mississippi	2,058	10.17	2,535	12.53
Missouri	840	2.11	238	.60
Montana	74	1.37	29	.54
Nebraska	55	.40	72	.53
Nevada	26	2.57	23	2.28
New Hampshire	12	.24	5	.10
New Jersey	974	2.24	304	.70
New Mexico	135	3.20	40	.95
New York	5,283	4.08	1,801	1.39
North Carolina	5,749	16.46	752	2.15
North Dakota	28	.40	81	1.15
Ohio [1]	1,417	2.10	380	.56
Oklahoma [1]	328	1.29	271	1.06
Oregon	65	.63	139	1.35
Pennsylvania	1,252	1.23	176	.17
Rhode Island	118	1.73	56	.82
South Carolina [2]				
South Dakota	15	.22	30	.43
Tennessee	1,024	3.54	416	1.44
Texas	1,301	2.11	380	.62
Utah	23	.44	42	.81
Vermont	17	.44	32	.84
Virginia	1,283	4.74	348	1.29
Washington	177	1.07	287	1.73
West Virginia [1]	411	2.20	130	.70
Wisconsin	65	.22	146	.50
Wyoming	8	.34	3	.13
Total	39,480	3.10	16,278	1.28

See footnotes at end of table.

Reports from cities of 200,000 population or over

	Syphilis		Gonorrhea	
	Cases reported during month	Monthly case rates per 10,000 population	Cases reported during month	Monthly case rates per 10,000 population
Akron, Ohio [3]				
Atlanta, Ga	339	11.29	153	5.10
Baltimore, Md.[3]				
Birmingham, Ala	369	12.54	43	1.46
Boston, Mass	219	2.75	180	2.26
Buffalo, N. Y	114	1.90	35	.58
Chicago, Ill	1,717	4.68	1,103	3.01
Cincinnati, Ohio	266	5.63	115	2.43
Cleveland, Ohio	211	2.23	66	.70
Columbus, Ohio	52	1.66	34	1.08
Dallas, Tex	224	7.37	90	2.96
Dayton, Ohio	126	5.68	8	.36
Denver, Colo.[3]				
Detroit, Mich	651	3.59	315	1.74
Houston, Tex.[3]				
Indianapolis, Ind.[2]				
Jersey City, N. J	28	.86	4	.12
Kansas City, Mo	69	1.60	10	.23
Los Angeles, Calif.[2]				
Louisville, Ky	281	8.29	74	2.18
Memphis, Tenn	292	10.00	73	2.50
Milwaukee, Wis.[3]				
Minneapolis, Minn	55	1.10	48	.96
Newark, N. J	323	7.11	133	2.93
New Orleans, La	72	1.47	59	1.21
New York, N. Y	3,923	5.24	1,262	1.68
Oakland, Calif.[2]				
Omaha, Nebr	19	.85	18	.80
Philadelphia, Pa	538	2.68		
Pittsburgh, Pa	238	3.38	25	.35
Portland, Oreg	27	.84	85	2.65
Providence, R. I	62	2.39	39	1.50
Rochester, N. Y	61	1.78	45	1.32
St. Louis, Mo	404	4.79	119	1.41
St. Paul, Minn	39	1.36	22	.77
San Antonio, Tex	86	3.29	56	2.14
San Francisco, Calif	154	2.23	227	3.29
Seattle, Wash	48	1.24	78	2.01
Syracuse, N. Y	67	2.97	19	.84
Toledo, Ohio [2]				
Washington, D. C	522	8.33	407	6.49

[1] Incomplete.
[2] No report for current month.
[3] Not reporting.

WEEKLY REPORTS FROM CITIES

City reports for week ended Nov. 12, 1938

This table summarizes the reports received weekly from a selected list of 140 cities for the purpose of showing a cross section of the current urban incidence of the communicable diseases listed in the table.

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
Data for 90 cities:											
5-year average	275	132	38	499	536	1,081	7	347	42	931	--------
Current week [1]	153	81	26	562	416	755	3	257	17	1,260	--------
Maine:											
Portland	0	1	0	0	1	0	0	0	0	0	16
New Hampshire:											
Concord	0	------	0	0	0	0	0	0	0	0	11
Manchester	0	------	0	0	0	2	0	0	0	0	10
Nashua	0	------	0	0	0	1	0	0	0	0	7
Vermont:											
Barre	0	------	0	0	0	0	0	0	0	0	2
Burlington	0	------	0	0	0	1	0	0	0	5	9
Rutland	0	------	0	0	1	0	0	0	0	0	9
Massachusetts:											
Boston	0	------	1	5	15	31	0	8	0	32	192
Fall River	2	------	0	0	1	0	0	1	0	0	24
Springfield	0	------	0	8	0	0	0	1	0	1	27
Worcester	0	------	0	0	3	1	0	0	0	13	39
Rhode Island:											
Pawtucket	0	------	0	1	1	0	0	0	0	3	10
Providence	0	------	0	0	2	5	0	3	0	21	59
Connecticut:											
Bridgeport	0	------	0	0	1	3	0	3	0	0	30
Hartford	0	------	0	1	1	2	0	0	0	0	35
New Haven	1	1	0	1	2	2	0	1	0	14	35
New York:											
Buffalo	0	------	0	9	8	17	0	1	0	14	108
New York	12	14	0	23	71	41	0	58	2	143	1,282
Rochester	0	------	0	6	3	1	0	1	0	8	55
Syracuse	0	------	0	1	1	6	0	1	0	9	53
New Jersey:											
Camden	0	------	0	0	1	4	0	0	0	0	29
Newark	0	------	0	2	3	5	0	5	0	33	72
Trenton	0	------	0	0	1	4	0	1	0	0	27
Pennsylvania:											
Philadelphia	2	------	3	6	13	28	0	26	3	82	442
Pittsburgh	2	3	1	0	15	22	0	6	0	16	153
Reading	7	------	0	0	1	1	0	2	0	0	30
Scranton	0	------	--------	0	-------	5	0	-------	0	1	--------
Ohio:											
Cincinnati	14	------	0	0	10	15	0	7	0	11	132
Cleveland	3	10	0	2	12	41	0	11	0	51	157
Columbus	3	------	0	0	3	7	0	0	0	2	72
Toledo	0	------	0	1	1	14	0	1	0	6	50
Indiana:											
Anderson	0	------	0	0	0	2	0	0	0	0	4
Fort Wayne	2	------	0	1	5	4	0	1	1	0	31
Indianapolis	6	------	1	4	14	19	2	5	0	1	107
Muncie	0	------	0	0	1	0	0	0	0	0	12
South Bend	0	------	0	0	1	5	0	0	0	0	23
Terre Haute	1	------	0	0	0	2	0	0	0	1	21
Illinois:											
Alton	1	------	0	0	1	0	0	0	1	0	11
Chicago	15	7	3	8	28	87	0	25	1	386	582
Elgin	0	------	0	0	4	0	0	0	0	0	10
Moline	0	1	1	1	2	1	0	0	0	3	14
Springfield	1	------	0	0	2	2	0	0	0	2	22
Michigan:											
Detroit	18	------	0	8	11	94	0	5	0	124	217
Flint	0	------	0	9	5	36	0	1	0	0	24
Grand Rapids	0	------	0	1	1	17	0	0	0	1	36
Wisconsin:											
Kenosha	0	------	0	0	0	4	0	0	0	13	6
Madison	1	------	0	0	0	2	0	0	0	1	18
Milwaukee	0	------	0	2	7	45	0	2	0	106	93
Racine	0	------	0	2	0	4	0	0	0	8	11
Superior	0	------	0	1	0	2	0	0	0	0	8

[1] Figures for Charleston, W. Va., and Raleigh, N. C., estimated; reports not received.

City reports for week ended Nov. 12, 1938—Continued

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Small pox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
Minnesota:											
Duluth	0	------	0	0	1	3	0	0	0	0	17
Minneapolis	0	------	0	60	10	16	0	0	0	4	81
St. Paul	0	------	0	31	5	10	0	2	0	8	61
Iowa:											
Cedar Rapids	0	------	------	1	------	2	0	------	0	2	------
Davenport	2	------	------	0	------	2	0	------	0	0	------
Des Moines	0	------	0	0	0	9	0	0	0	0	35
Sioux City	0	------	------	33	------	4	0	------	0	5	------
Waterloo	5	------	------	0	------	6	2	------	0	3	------
Missouri:											
Kansas City	3	------	0	0	9	12	1	3	0	0	95
St. Joseph	0	------	0	0	3	2	0	2	0	0	23
St. Louis	3	------	0	1	6	9	0	4	1	6	160
North Dakota:											
Fargo	0	------	0	199	0	4	0	0	0	0	6
Grand Forks	2	------	------	1	------	0	0	------	0	0	------
Minot	0	------	0	0	0	2	0	0	0	0	8
South Dakota:											
Aberdeen	0	------	------	0	------	0	0	------	0	0	------
Nebraska:											
Lincoln	0	------	------	1	------	2	0	------	0	0	------
Omaha	1	------	0	0	3	1	0	0	0	1	30
Kansas:											
Lawrence	0	2	0	0	0	1	0	0	0	0	3
Topeka	2	------	0	0	1	5	0	0	0	3	22
Wichita	1	------	0	1	0	2	0	0	0	0	27
Delaware:											
Wilmington	0	------	0	1	1	3	0	0	0	0	15
Maryland:											
Baltimore	4	3	0	22	13	2	0	5	1	15	208
Cumberland	0	------	0	0	0	0	0	0	0	0	9
Frederick	0	------	0	0	1	0	0	0	0	0	3
Dist. of Col.:											
Washington	7	2	1	2	11	4	0	7	0	13	143
Virginia:											
Lynchburg	0	------	0	0	1	3	0	0	0	0	14
Norfolk	4	------	0	0	0	5	0	0	0	0	23
Richmond	0	------	0	1	0	4	0	5	0	0	55
Roanoke	1	------	0	0	1	0	0	0	0	0	15
West Virginia:											
Huntington	0	------	------	0	------	0	0	------	0	0	------
Wheeling	0	------	0	0	0	0	0	0	0	6	17
North Carolina:											
Gastonia	1	------	------	0	------	0	0	------	0	0	------
Wilmington	1	------	0	1	1	0	0	0	1	2	8
Winston-Salem	0	------	0	18	2	6	0	1	0	0	13
South Carolina:											
Charleston	0	9	0	0	1	0	0	0	1	0	16
Florence	0	------	0	0	0	0	0	0	0	0	8
Greenville	2	------	0	0	4	0	0	0	0	0	25
Georgia:											
Atlanta	2	12	3	0	12	8	0	3	0	0	74
Brunswick	1	------	0	0	1	1	0	0	0	0	3
Savannah	0	6	0	0	4	1	0	2	3	5	29
Florida:											
Miami	0	------	0	1	3	1	0	2	0	0	20
Tampa	0	1	0	0	3	0	0	1	0	3	22
Kentucky:											
Ashland	14	------	0	0	0	1	0	0	0	0	9
Covington	0	------	0	0	1	5	0	0	0	0	19
Lexington	0	------	0	0	2	2	0	2	0	0	23
Louisville	3	1	0	0	4	15	0	3	0	0	46
Tennessee:											
Knoxville	2	------	2	0	3	3	0	2	0	0	23
Memphis	0	------	3	0	8	6	0	1	0	13	89
Nashville	2	------	3	0	0	9	0	2	1	0	63
Alabama:											
Birmingham	1	4	2	0	8	3	0	2	0	1	63
Mobile	0	------	0	0	4	1	0	2	0	0	29
Montgomery	2	------	------	0	------	3	0	------	0	0	------
Arkansas:											
Fort Smith	0	2	------	0	------	1	0	------	0	0	------
Little Rock	1	------	0	0	2	1	0	2	1	0	5

City reports for week ended Nov. 12, 1938—Continued

State and city	Diphtheria cases	Influenza: Cases	Influenza: Deaths	Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
Louisiana:											
Lake Charles	1	------	0	0	0	1	0	0	0	0	4
New Orleans	10	3	0	4	13	3	0	5	0	11	142
Shreveport	0	------	0	0	7	1	0	2	0	2	35
Oklahoma:											
Muskogee	------	------	------	------	------	------	------	------	------	------	------
Oklahoma City	0	------	0	0	1	7	0	2	5	0	35
Tulsa	1	------	0	0	0	4	0	0	0	0	------
Texas:											
Dallas	2	------	0	0	4	8	0	4	0	0	59
Fort Worth	1	------	0	0	1	12	0	0	0	0	49
Galveston	0	------	0	0	3	0	0	1	0	0	13
Houston	3	------	0	0	4	2	0	2	0	0	74
San Antonio	0	2	1	1	6	3	0	5	0	1	48
Montana:											
Billings	0	------	0	0	1	1	0	0	0	0	8
Great Falls	0	------	0	0	0	3	0	0	0	0	4
Helena	0	------	0	4	0	0	0	0	0	0	8
Missoula	0	1	------	0	------	2	0	------	0	0	------
Idaho:											
Boise	0	------	0	0	1	0	0	0	0	0	5
Colorado:											
Denver	8	------	1	0	1	6	0	0	0	26	64
Pueblo	1	------	0	0	2	3	0	0	0	0	13
New Mexico:											
Albuquerque	0	------	0	0	0	1	0	3	0	0	10
Utah:											
Salt Lake City	0	------	0	0	1	4	0	0	0	4	31
Washington:											
Seattle	1	------	1	0	3	6	0	2	1	9	85
Spokane	0	------	0	3	2	0	0	0	0	0	33
Tacoma	0	------	0	0	1	0	0	0	0	7	21
Oregon:											
Portland	0	------	0	1	1	17	0	0	0	0	65
Salem	0	1	------	1	------	4	0	------	0	0	------
California:											
Los Angeles	9	2	0	6	6	34	0	10	0	17	279
Sacramento	0	------	0	0	2	0	0	1	0	1	25
San Francisco	0	------	2	107	5	6	0	6	0	9	163

State and city	Meningitis, meningococcus: Cases	Meningitis, meningococcus: Deaths	Poliomyelitis cases
New York:			
Buffalo	1	0	0
New York	1	2	3
Pennsylvania:			
Philadelphia	0	0	3
Ohio:			
Cleveland	1	0	0
South Carolina:			
Greenville	0	1	0
Georgia:			
Atlanta	0	0	1
Tennessee:			
Nashville	2	2	0
Louisiana:			
Shreveport	0	4	0
Oregon:			
Portland	0	0	1
California:			
Los Angeles	1	0	0

Encephalitis, epidemic or lethargic.—Cases: New York, 1; Camden, 1.
Pellagra.—Cases: Charleston, S. C., 3; Atlanta, 2; Savannah, 2; Mobile, 1; Los Angeles, 2.
Typhus fever.—Cases: Charleston, S. C., 1; Atlanta, 3; Savannah, 1; Mobile, 1; Dallas, 1.

FOREIGN AND INSULAR

CANADA

Provinces—Communicable diseases—2 weeks ended November 5, 1938.—During the 2 weeks ended November 5, 1938, cases of certain communicable diseases were reported by the Department of Pensions and National Health of Canada as follows:

Disease	Prince Edward Island	Nova Scotia [1]	New Brunswick	Quebec	Ontario	Manitoba	Saskatchewan	Alberta	British Columbia	Total
Cerebrospinal meningitis				1	3	1		1		6
Chickenpox		5	1	267	330	72	51	43	93	862
Diphtheria		3	5	186	10	20	8	13		245
Dysentery					7				1	8
Erysipelas				5	7	5	1	2	5	25
Influenza		10			7				33	50
Measles		3	3	211	262	22	8	11	48	568
Mumps			1		21	28	16	6	9	81
Paratyphoid fever					2				1	3
Pneumonia		2			34				9	45
Poliomyelitis				3	3	16		2	1	25
Scarlet fever		16	23	213	230	77	40	37	48	684
Smallpox							3			3
Trachoma									9	9
Tuberculosis	8	59	50	90	115	39	10	2	34	407
Typhoid fever	1		3	37	20	7	5	3	37	113
Undulant fever					4				1	5
Whooping cough				98	408	24	8	12	41	591

[1] For 2 weeks ended Nov. 9, 1938.

FINLAND

Communicable diseases—September 1938.—During the month of September 1938, cases of certain communicable diseases were reported in Finland as follows:

Disease	Cases	Disease	Cases
Diphtheria	241	Paratyphoid fever	114
Dysentery	3	Poliomyelitis	194
Influenza	1,207	Scarlet fever	481
Lethargic encephalitis	1	Typhoid fever	15

CHOLERA, PLAGUE, SMALLPOX, TYPHUS FEVER, AND YELLOW FEVER

NOTE.—A table giving current information of the world prevalence of quarantinable diseases appeared in the PUBLIC HEALTH REPORTS for November 25, 1938, pages 2107-2119. A similar cumulative table will appear in future issues of the PUBLIC HEALTH REPORTS for the last Friday of each month.

Cholera

China.—During the week ended November 12, 1938, cases of cholera were reported in China as follows: Hong Kong, 8; Macao, 14; Shanghai, 10.

India—Negapatam.—For the week ended November 12, 1938, two cases of cholera were reported in Negapatam, India.

Plague

Brazil—Pernambuco State.—During the month of August 1938, four cases of plague with two deaths were reported in Pernambuco State, Brazil.

Hawaii Territory—Island of Hawaii—Hamakua District—Hamakua Mill Sector.—A rat found on November 9, 1938, in Hamakua Mill Sector, Hamakua District, Island of Hawaii, Hawaii Territory, has been proved positive for plague.

Tunisia—Tunis.—During the week ended November 19, 1938, one case of bubonic plague was reported in Tunis, Tunisia.

Smallpox

China—Amoy.—During the week ended November 5, 1938, one case of smallpox was reported in Amoy, China.

Typhus Fever

Syria (Lebanese Republic).—During the week ended October 22, 1938, one case of typhus fever was reported in Syria (Lebanese Republic).

Yellow Fever

Ivory Coast.—Yellow fever has been reported in Ivory Coast as follows: On November 10, 1938, one suspected case on a plantation north of Tiassale, and one case in Abengourou; on November 14, 1938, one suspected case in Agboville, and one suspected case in Katiola.

Sudan (Anglo-Egyptian).—On November 12, 1938, one suspected case of yellow fever was reported in the region of Juba, Anglo-Egyptian Sudan.

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