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DEDICATION OF THE NEW BOSTON MARINE HOSPITAL

The dedication of the new marine hospital at Boston, Mass., on June 6, 1940, was a significant milestone in the history of the Public Health Service. It was significant for many reasons. The medical and hospital care of American merchant seamen was the first (and long the only) function of the Service; the first treatment furnished seamen under the authority of the act of 1798 was given in Boston in 1799; the first permanent marine hospital built by the Federal Government was that erected in Boston (Charlestown); and, of especial interest today, the marine hospital service may well be considered the first compulsory hospital insurance plan adopted in America. It is of interest to note that the Boston Marine Hospital is also the oldest hospital in Massachusetts, and believed to be the fourth oldest in the United States.

The first marine hospital erected in Boston was built in 1803, on 5 acres of land in Charlestown, and was occupied early in 1804. This hospital cared for the sick and wounded officers and members of the crew of the frigate *Constitution* and prisoners from the *Guerrière* after their engagement in what now may modestly be termed the slight unpleasantness of 1812. Before this hospital building was made available, medical and hospital care was provided seamen in a temporary building, the barracks at Fort Independence, Castle Island.

With the increase in shipping activities at Massachusetts ports, and the resultant increase in the number of seamen employed, increased bed capacity and improved facilities became necessary from time to time, which were provided by successively larger buildings occupied in 1827 and 1860. The present new building was occupied on June 1, 1940.

The principal address at the dedication ceremony was made by Federal Security Administrator Paul V. McNutt, whose remarks are printed in full elsewhere in this issue. Mr. McNutt pointed out that

the act of 1798, providing medical and hospital care for sick and disabled merchant seamen, a group in especial need of such services, was one of the first acts of our young Federal Government which recognized its responsibility for the welfare of the people; and that in this work the present Federal security program actually had its roots in the very beginning of our Government. He referred to the hospital building as the visible embodiment of democracy, and stated that it represents the solicitude of the Government for the sick and is a tangible "reassurance that the individual citizen is still the first concern of the Nation."

Surgeon General Parran reviewed the history and progress of the marine hospitals, summarized his expectations regarding new hospitals, and spoke of the new program for providing hospitals in needy rural areas, the bill for which is now before Congress.

Assistant Surgeon General S. L. Christian, Chief of the Division of Marine Hospitals and Relief, commended the work of the staff, attendants, and others concerned with the task of preparation for the opening of and the transfer to the new building, expressed appreciation of Congressional action in making funds available for the much needed new facilities, and thanked the architects and construction experts who designed and built the hospital.

The principal speaker at the luncheon held in connection with the dedicatory exercises was Dr. Roger I. Lee, professor of public health at Harvard University and a member of the National Advisory Health Council. In speaking of the need for continued progress, Doctor Lee said that a hospital is never actually completed, either physically or spirtually, even though "you have moved in." Not only must the physical walls and equipment be changed, but the mental walls must be changed as well. "The succession of five marine hospitals at Boston,[1] " he said, "was evidence that the marine hospital system of the Public Health Service has been keeping abreast of progress."

A personal connection with the past was provided at the dedication in the presence of Col. David Townsend, the great-grandson of the third commanding officer of the first marine hospital constructed at Boston, Dr. David Townsend. Dr. Townsend succeeded Dr. Benjamin Waterhouse in 1809 and served as medical officer in charge until his death in 1829.

The new hospital is located on a site of approximately 13 acres on Warren Street (No. 77), near Commonwealth Avenue. It is of modified Georgian style of architecture, of red brick and brown limestone construction, and has a bed capacity of 360. On the site are also ample quarters for medical officers, nurses, and attendants,

[1] Counting the barracks at Castle Island which were used for the first or temporary hospital before the first permanent marine hospital was built. Counting all buildings used in Boston for the marine hospital, the present building is the sixth, as a rented building in Charlestown was used from 1825 to 1827.

The four U. S. Marine hospitals built by the Federal Government at Boston. Above: Left, first hospital, 1804–25; right, second hospital, 1827–58. Center: Third hospital, 1858–1940. Below: New hospital, occupied on June 1, 1940.

RULES AND ORDERS

OF THE

United States Marine Hospital,

ESTABLISHED AT CHARLESTOWN

ARTICLE I.

THE OVERSEER or STEWARD is to go through all the wards in the morning before the Physician visits them, to see that the men have washed their hands and faces, and that nothing offensive be left in the rooms; and he is to go again through the wards before bed time to see that all the patients be in the house; and that none remain in it that do not belong to it.

ARTICLE II.

Every patient is to retire to rest on or before nine o'clock in the winter months; and by ten in the summer; and no lights are to be allowed, or fire kept up, after that period, unless some special case should require it.

ARTICLE III.

Every patient in the house is enjoined to pay strict obedience to the orders of the Steward or Overseer; and should any patient think himself at any time aggrieved, he has the liberty of appealing to the Physician.

ARTICLE IV.

Every patient is to be shaved every Sunday and Wednesday; and shirted every Sunday, and oftener, if convenient; and he is to wash his face and hands and comb his head every day, if his case and circumstances will admit of it.

ARTICLE V.

Every patient is forbidden to spit on the floor or hearth, or write on the walls, or mark the wood work, or drive nails in either.

ARTICLE VI.

If any man pertinaciously disobeys the orders of the Physicians or overseers, or gets drunk, or commits riot, or is found guilty of theft, he forfeits the privilege of the hospital, and shall be dismissed.

ARTICLE VII.

No patient is allowed to go to Boston, or to any distance from the hospital without permission from the Physician, or Overseer, or house-pupil.

ARTICLE VIII.

If any patient be found to throw away his medicine, or feign complaints; or who wilfully does any thing to impede his cure, he shall, upon conviction thereof be dismissed

ARTICLE IX

No person is allowed to play cards, or any other game of hazard for money, drink, or any other article.

All games of amusement, accompanied with noise are forbidden, as they disturb the sick.

ARTICLE X.

Whatever patient be out of the house all night without permission from the Physician or Overseer, is from that time dismissed.

ARTICLE. XI.

All the patients shall be in their own wards, and places when the Physician is ready to visit them, of which they will be notified by the ringing of the bell.

ARTICLE I.

NURSES—Are to see that the patients be neat and clean as the nature of their cases will admit. They are to see particularly as to the cleanliness of the bed and bedcloaths. They are to see that the wards be kept extremely clean; and that they be aired, by keeping the windows and doors open in fair weather, a longer or shorter time, as the weather may admit. And they are to see that no nastiness of any kind be thrown out of the windows or doors.

ARTICLE II.

The nurses, male or female, are, upon no pretence, to alter the diet ordered by the Physician; nor to suffer the patients to use any other diet, than what is allowed by the hospital; nor are they to permit spiritous liquors of any kind to be brought into the wards, except what is directed by the Physician. They are to attend to the particular disgusts and cravings of the sick, and report them to the Physician.

ARTICLE III.

The effects of men, who die in the hospital, are to be locked up, and reported as soon as may be to the Physician; and if any nurse, attendant, or any other person, should take away or conceal any article belonging to such as die in the hospital, their crime shall be reported to the *Superintendent.*

ARTICLE IV.

The hours for admission of patients are between 10 and 12; but should any that are lame or weak present themselves before or after that time, the house-pupil, or the overseer, or the head-nurse, may receive them; and give them such articles of food only as are allowed in the *low diet*, until the Physician see them.——No person can be admitted into the hospital with the itch, or any other infectious disorder, the venereal excepted.

ARTICLE V.

No Seaman can be admitted into the hospital without a written certificate from the Custom-House, that he has paid hospital money.

APRIL, 1808.

Benj^n Waterhouse a Physician to M. S. Marine Hosp.

Facsimile of the "Rules and Orders" of the Boston Marine Hospital drafted by Dr. Benjamin Waterhouse in 1808.

laundry, storage facilities, powerhouse, garage, and machine shop. The hospital building has an auditorium with a seating capacity of 300, modern in acoustic construction, and has a stage and dressing room facilities. The auditorium will be available for medical meetings, including those of the local medical society, and for the recreation and entertainment of ambulant patients.

The equipment is the best and most modern available, not elaborate or ostentatious, but of suitable quality for the best care of both bed and ambulant patients.

The staff and personnel of the old hospital were transferred in a body, and will be augmented as required in the future.

AN ADDRESS BY THE HONORABLE PAUL V. McNUTT, FEDERAL SECURITY ADMINISTRATOR, AT THE DEDICATION OF THE U. S. MARINE HOSPITAL, BOSTON, MASS., JUNE 6, 1940

It is reassuring to be in Boston again. In these troubled times Boston, the cradle of American liberty, gains an added significance and a heightened place in our affections.

Every American city has its special appeal. Each one has the characteristics of its region and the peculiar qualities of the people who made it and now live in it. But all over our country, in every city and hamlet in the land, Boston holds a place of its own, unique and unchallenged. It means something to all of us because something of our roots are buried here. It is good to remember that the "tea party" did take place; that Paul Revere took his famous ride; that embattled farmers fought at Lexington.

When I walked across the Common this morning, I felt a certain security of spirit that comes from the knowledge of a great fighting tradition, of a great culture, of achievement in all the arts, in science, in medicine—a sense of communion with great men of the past and a realization of its great citizens of today in every walk of life.

Boston is American in the true sense. It is the melting pot quite as much as other cities in the East, and in the Far West and the Middle West. It has demonstrated the truth of what men are fighting for in Europe: That people of many origins can live together in peace, trusting each other in the fundamentals.

Yes, it is good to be back. And I am happy that the occasion which brings me is the dedication of this Public Health Service marine hospital, the first to be established in the Federal Government's first program for human welfare and the last of the 26 marine hospitals to be rebuilt on modern lines.

When the President created the Federal Security Agency last July, pursuant to the Reorganization Act, he sought, in the modern trend of government administration, to group together agencies having related functions. Why the Public Health Service had been in the Treasury was a question hard to answer since the reason had long been forgotten.

When the Service was first established, it was an agency to furnish medical service and hospital care to sick and injured seamen from American vessels. The collectors of customs were the business agents of the hospitals. They handled the money and paid the bills. As the collectors of customs came under the Treasury Department, so did the marine hospitals. But over a half century ago the collectors of customs ceased to be the business agents of the hospitals. Since that time, Congress has expanded the hospital service to include many duties much more closely related to the work of the Federal Security Agency than to that of the Treasury Department.

The history of the Public Health Service makes us realize that the great Federal security program, administered by the Federal Security Agency, had roots in the beginnings of our Government. The main function of the Public Health Service for so many years, namely, the medical care of sick and injured seamen, was one of the first acts of our young Federal Government in recognition of its responsibility for the welfare of our people.

This program has been extended both in concept and function until today the citizens of our democracy have charged their Government with responsibility for many functions in the service of mankind of which the founding fathers never dreamed in our frontier years. The Public Health Service has evolved from a hospital service for one small group into our first-line defense for the promotion and preservation of the health of all the people.

Each of the other units of the Federal Security Agency also touches millions of Americans. The Social Security Board reaches with its two big insurance features, unemployment compensation and old-age insurance, 50 millions of people, and its public-assistance programs serve many additional millions among the needy aged, dependent children, and the blind.

The National Youth Administration and the Civilian Conservation Corps have as their primary responsibility the conservation of the health and morale of our young people, with emphasis upon training them for self-supporting, intelligent citizenship. The Office of Education is our third instrument to help American youth. Through its advisory services to the schools and its vocational education and rehabilitation program it reaches almost every community in the Union.

The present grouping of these agencies under one administrative unit, the Federal Security Agency, is a visible sign of our realization that all the services designed to "promote the general welfare" are interdependent, one upon the other, and that only with close cooperation can these services be integrated into an effective program. The problems of human welfare which our democracy seeks to solve through these services have common causes, common means of solution, and a common objective, namely, to prove that a democracy can and will function for the protection and the promotion of its human resources.

The oldest member of the Federal Security Agency family is the Public Health Service. The story of its origin, its growth, and achievement is well known to this audience. To me it is of much interest and significance, for if one desires to serve in furthering the objectives of an undertaking, one must know its past and the reason for its beginning.

The active life of the Service began in the first Boston Marine Hospital. That story has been admirably told for all of us here today by Doctor Trask in the little booklet which commemorates this occasion. To him we pay tribute not only for his achievements as physician and hospital administrator, but also for his interest and ability as an historian. His chronicle of the past meets the true test of history. It creates a desire to visit the Boston Marine Society and talk with the ships' captains who are today carrying on the work of an organization nearly two centuries old, to visit Fort Independence, site of the first Boston Marine Hospital, and the grave in King's Chapel churchyard of Doctor Welsh, and many other points of interest which illuminate the past.

These things enrich our understanding of the tradition of service which even today inspires the work of the Public Health Service. As we survey the years of life of the Service, we are forcibly impressed with its role as the searcher, the demonstrator, and the leader in the forward advance of public health. Its work has been and will continue to be the search for new and better ways of dealing with problems affecting the people's health, a demonstration of their most effective use and an example of that use.

That role has been maintained continuously since the very day when this old marine hospital first opened its doors. It was the first hospital in Boston, the fourth in the Nation. Thomas Welsh, first physician in charge, had little to guide him in his task of organizing and creating a general hospital. Within a few years, with the aid of Benjamin Waterhouse, also an outstanding figure in the history of American medicine, this institution became a demonstration of what constitutes a good hospital and good hospital practice. In its early days, too, the hospital took its place in the evolution of American medical education.

It became the "teaching hospital" of the Harvard Medical School and remained so for a number of years.

From those beginnings there has descended in the Public Health Service a long line of important "demonstrations" in the wider fields of scientific research, quarantine procedure, epidemiology, and public health control and administration. It is the desire of all of us that in this role of demonstration and leadership the Public Health Service may continue to make significant contributions, not only in research and public health practice, but in medical practice as well. And our hopes for this new hospital in Boston beget the thought that here, in the years ahead, we may so effectively put to work the newer knowledge in medicine as to present within these walls an inspiring example of modern science at its peak of swift and efficient relief of human suffering.

This hope goes beyond the desire to ease immediate pain. We like to think that the day will come when this hospital is not simply a place to which its beneficiaries resort in emergencies, but a port of call for the prevention of future illness, a harbor from which the patient goes refreshed both in body and in spirit, better able to meet his world and the people and conditions which form it.

This hope is strengthened by consideration of some of the miracles of modern medical science. For example, in the recent use of chemotherapy for infectious diseases, we are witnessing one of the most remarkable developments in the history of medicine. We remember also that man suffers not from infectious disease alone. He is an organism both ruled and tyrannized by his emotions. The emotional factors in disease are also being recognized and studied as of great importance, especially in the treatment of the more chronic and debilitating conditions.

Man is not to be considered a physical body with a separate mind, but rather as a complete and indivisible being. When he is ill, the whole organism is ill. Consideration of the patient's feelings, anxieties, and fears, of the causes of these emotions, and their alleviation in the treatment of his illness are also among the important advances in medicine. So we find that, in the modern hospital, increasing emphasis is being placed upon the personal relationships between the patient and those who care for him, upon the kindliness of his reception when the patient enters its doors, upon allaying unnecessary fear and apprehension, and upon obtaining from him the invaluable cooperation in recovery that comes from his confidence in physician and nurse and in their sympathetic and intelligent understanding of his individual case.

We could find no better place for the fulfillment of this future aim than Boston, one of the great medical centers of our country. In this setting are some of the oldest and most renowned medical schools in

America, leaders in medical research and medical education, distinguished practitioners in medicine and public health—men to whom we owe much of the progress made in the treatment of the sick, in our knowledge of the causes of disease, and in public health administration, men who have been leaders in everything connected with American medicine.

The relationship which exists between the marine hospitals of the Public Health Service and the medical profession of the communities in which they are established is, and always has been, intimate and cordial. Through the continued maintenance of consultant staffs, the hospitals have profited immeasurably by direct contact with practicing physicians and clinicians.

It is our hope that this new Boston Marine Hospital will take its place and make its contribution in the medical world of Boston. Their interests are identical; their objectives the same. Their programs should be mutually helpful and complementary.

As we meet here today near the old Boston harbor where once the *Constitution*, and the *Congress*, and the *President*—frigates of our young Navy—discharged their wounded to the marine hospital at Charlestown, I feel very strongly the importance of such a relationship. In the difficult days ahead for this Nation, as we rise to gird democracy for its defense, let us not for a moment forget that we must have both military defense and a prepared people, physically and spiritually able to perform their tasks.

Without the leadership and guidance of our medical institutions and our men of medicine, without the cooperation between Government and medicine, such as that demonstrated in the Public Health Service hospitals, we cannot raise this bulwark of human defense. We cannot be totally prepared.

In a very real sense, this hospital is the visible embodiment of democracy. It represents the solicitude of the Government for the sick. It is tangible reassurance that the individual citizen is still the first concern of the Nation. It somehow reminds us that whatever happens elsewhere in the world, America is one land where the ideals of humanity and Christian love still prevail as the dominant influence in our national life.

OCCUPATIONAL LEUKODERMA

By Louis Schwartz, *Medical Director, United States Public Health Service*, Edward A. Oliver, M. D., *Chicago*, and Leon H. Warren, *Acting Assistant Surgeon, United States Public Health Service*

About September 1938, the Negro workers of a large tannery began to complain to the management that white spots (fig. 1) were appearing on the areas of skin of their forearms and hands which were covered by the rubber gloves furnished for their use by the factory.

The men working in the beam house and tan house and the color and fat liquor departments were said to be the only ones affected. There was itching, and in some cases a mild dermatitis preceding the appearance of the depigmentation, but there was no great discomfort or any disability. However, the psychological reaction on some of the men was bad, and by the early part of 1939 several of the workers had instituted law suits for damages.

All of the affected workmen were said to have been wearing a heavy gauntlet type of rubber glove having curved fingers, known as a heavy acid rubber glove. Up to April 1937 they had been wearing the same type of glove but with straight fingers. After that time the management furnished them with the curved-fingered gloves.

The condition continued to progress so that in some instances most of the skin covered by the gloves became depigmented (fig. 2), while in other instances the skin covered by the gauntlet was only spotted with areas of leukoderma (fig. 3). A few of the workers also developed leukoderma on parts of the body not covered by the gauntlets, but these parts were all on areas of the body which could have been touched by the gloves (fig. 4). The hairs in the leukodermic areas were not affected. As a result of these complaints, the management discontinued the use of the curved-fingered glove early in May 1939 and consulted the Department of Leather Research of the University of Cincinnati concerning this matter.

The Leather Research Department referred the matter to the Surgeon General of the United States Public Health Service on May 16, 1939. About the same time the representatives of the rubber company who manufactured the gloves came in person to consult the Office of Dermatoses Investigations in regard to the same matter. The Office of Dermatoses Investigations then began the investigation by first trying to ascertain by correspondence whether similar cases had occurred in other tanneries.

In June 1939, one of us (E. A. O.) was called in by the insurance company which carried the insurance of the tannery to see 18 cases of a peculiar depigmentation occurring among a group of Negroes and Mexicans working in the tannery.

A few days later a letter was received by the Office of Dermatoses Investigations from another tannery stating that some of their colored and white workers had depigmentation of the skin of the hands and forearms involving the areas covered by their rubber gauntlets, and asking whether this office had any reports of a similar condition. Since these first two reports, we have received letters reporting the occurrence of similar cases in other industries located in various parts of the United States where rubber gloves of the same manufacture were worn.

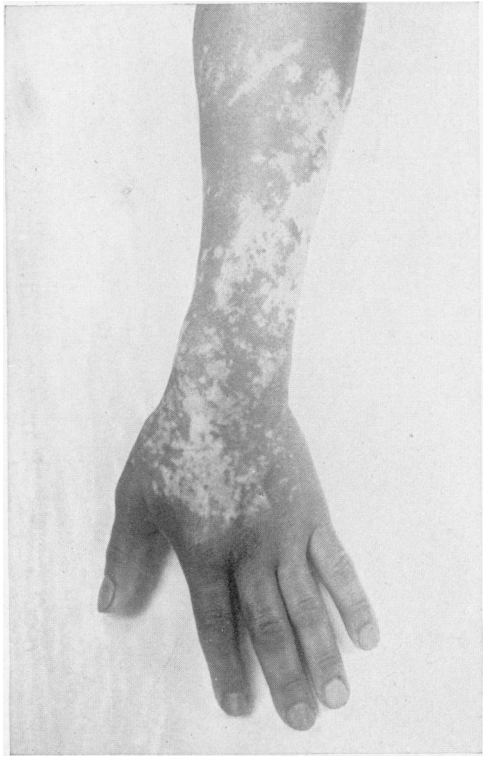


FIGURE 1.—Patchy depigmentation.

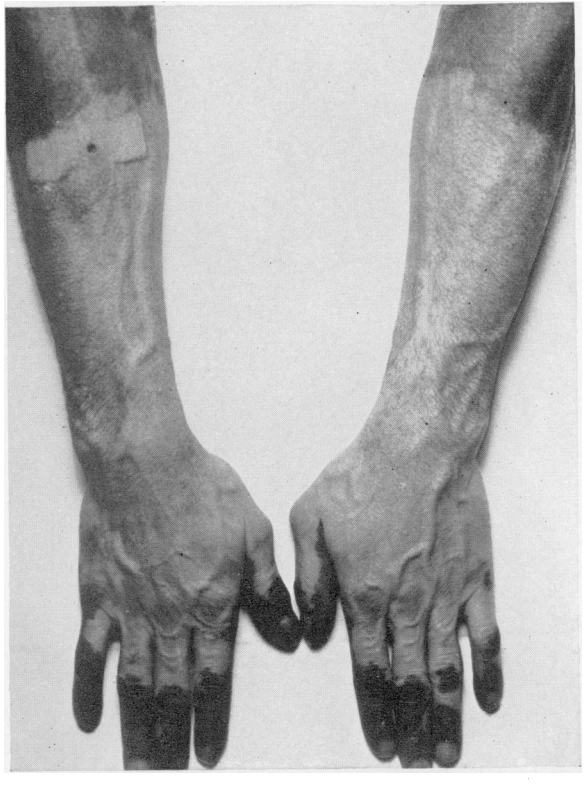


FIGURE 2.—Uniform depigmentation.

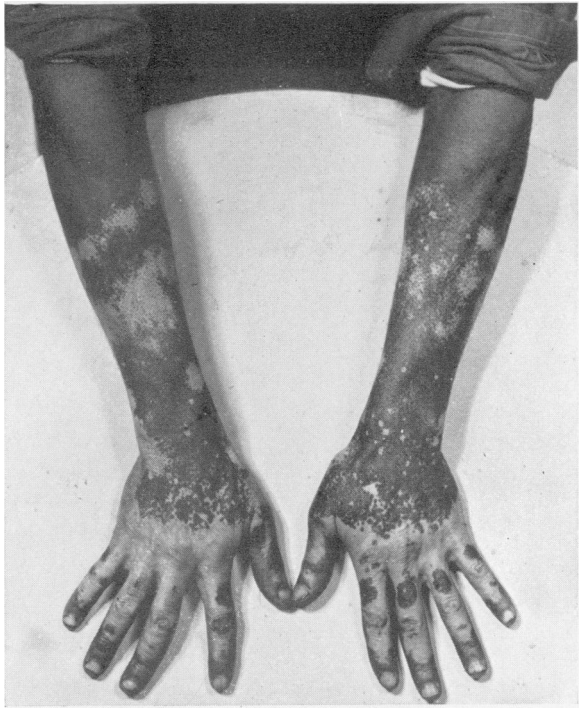


FIGURE 3.—Patchy depigmentation.

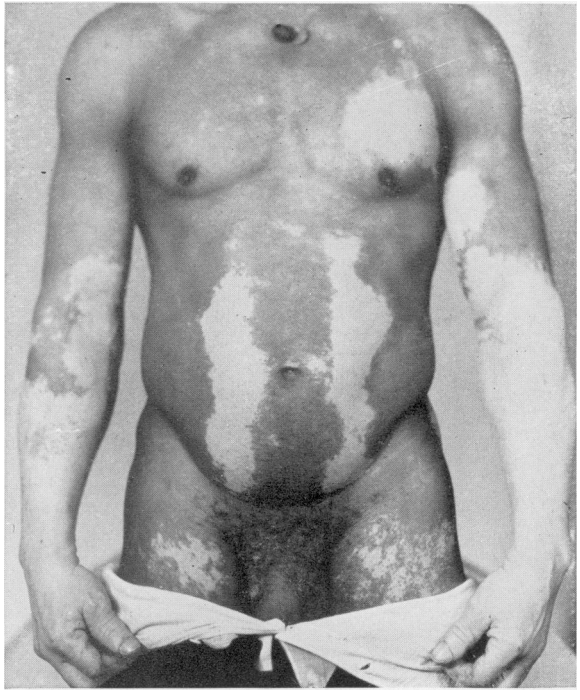


FIGURE 4.—Widespread involvement resembling vitiligo.

The cities in which the two tanneries are located were visited and an active investigation was begun.

In the first tannery all the workers were examined, and it was found that out of about 500 employees only 48 wore the suspected rubber gloves. These 48 were all employed in 4 departments of the tannery. The total number of workers in these 4 departments was 88. Of the 48 who wore the gloves, 25, or 52 percent, had developed leukoderma. Of the 11 workers in the tan house, 10 wore rubber gloves and 6 of these developed leukoderma, an incidence of 60 percent. Of the 4 workers in the hand finishing department, 2 wore rubber gloves and both of these developed leukoderma. Of the 16 workers in the color and fat liquor department, 13 wore the gloves and 7 of these developed leukoderma, an incidence of 54 percent. Of the 57 workers in the beam house, 22 wore gloves and 9 developed leukoderma, an incidence of 41 percent. The janitor who wore the rubber gloves while cleaning also developed leukoderma. Sixty percent of the workers affected were Negroes, 20 percent were Mexicans, and 20 percent were white. On the basis of complexion, 31 percent of the Mexicans wearing the gloves, 29 percent of the Negroes wearing the gloves, and 21 percent of the white Americans wearing the gloves developed leukoderma.

The areas of leukoderma showed a flat white in contrast with the dark skin of the Negroes; the contrast was not so marked against the lighter skin of the Mexicans; and in the case of the white Americans the leukoderma was noticed only in the summer when the skin covered by the gloves failed to tan.

At the time of our examination there were no signs of any acute or chronic dermatitis present on any of the leukodermic areas. In some instances the hairs were lost over the leukodermic areas, in others the hairs were short and stubby as if they had been depilated and grown back, and in other cases the hairs were unaffected. In no case, however, were the hairs depigmented. The loss of hair may not necessarily have been due to anything in the glove, since many of the chemicals used in tanning leather are depilatories and loss of hair on the arms of workers with these chemicals is not uncommon.

In studying the process of tanning used in this factory, we learned that dimethylamine was used as an unhairing agent. Since we had not previously seen this chemical used as an unhairing agent in tanneries, we at first suspected that the leukoderma might have been directly or indirectly caused by its action on the skin. This, however, we disproved later in our investigation.

The company officials in the second tannery stated that their workers had never used the curved-fingered gauntlets but only the straight-fingered ones; yet some of their workers in the beam house, the tan yard, and the fat liquor departments were affected with leuko-

derma on areas of skin touched by the straight-fingered gloves. The officials also stated that there was no dimethylamine used in their tannery, the unhairing being done with the usual mixture of lime and sodium sulfide. In this tannery 16 workers with leukoderma on parts touched by the rubber gloves were seen. Five of them were white workers who stated that they noticed the leukoderma only when the warm weather began and the areas touched by the gloves failed to tan. The first cases of depigmentation were called to the attention of the management in December 1938. This tannery purchased its gloves from the same rubber company as the first tannery.

Since the workers in this tannery did not at any time use the curved-fingered glove, and since they did not use dimethylamine and yet were affected by leukoderma, it now became apparent that dimethylamine was not the cause of the leukoderma, and that the curved-fingered gloves were not the only ones that caused it.

In order to see whether workers in other tanneries in which rubber gloves manufactured by this company were used had the same condition, we visited a third tannery, the name of which was given to us by the jobbers who sold the gloves. In this tannery it was found that both the curved- and straight-fingered gloves were used, that dimethylamine was not used, and that workers who wore either kind of gloves made by this company for any length of time had leukoderma.

Never before in the history of any of these tanneries had such a condition occurred on the skin of the workers. In the knowledge of the authors no similar outbreak has been reported in the literature before our preliminary report in the Journal of the American Medical Association on September 2, 1939.

In order to ascertain whether the leukoderma due to wearing these gloves occurred only in tanneries or whether it occurred in other industries where the rubber gloves were worn, the names of various manufacturing companies where these gloves were used were obtained from the jobbers, and these factories were visited. They included decalcomania manufacturers, electrical apparatus manufacturers, meat packing plants, and electroplating works. In all of them some of the workers who wore this particular brand of gloves were found to have leukoderma on areas covered by the rubber gloves. As in the tanneries, the white workers noticed the condition only with the coming of summer when the skin touched by the rubber gloves failed to tan.

Samples of the gloves causing the leukoderma were obtained and taken to the factory of the rubber company which manufactured them. They identified the gloves as being what they called their "acid-cured" gloves. The officials of this company stated that they had been making these gloves for many years, but that in September 1937 they had made a change in the formula, consisting in the addi-

tion of 0.2 percent of an antioxidant,[1] which they said was monobenzyl ether of hydroquinone, containing less than 1 percent of unchanged hydroquinone as an impurity. Based on the amount of smoke sheet rubber in the rubber compound from which the gloves were made, the amount of the antioxidant added to the formula was 0.5 percent. The formula of the rubber glove which caused the leukoderma was as follows:

Smoke sheet rubber	100 parts.
Chrome yellow	5 parts.
Whiting	125 parts.
Cumar	3 parts.
Stearex beads	10 parts.
Antioxidant (monobenzyl ether of hydroquinone)	0.5 parts.[2]

A small amount of soapstone.

The process of manufacturing the glove is as follows: The ingredients are mixed in a mix mill and rolled into thin sheets. The sheets are cut up into small pieces and placed in metal drums containing petroleum naphtha. There are 7 parts of naphtha to 1 part of compounded rubber. The drums are revolved for about 48 hours, after which time the rubber is completely dissolved. The solution is then pumped into a storage tank from which it is allowed to run into the dipping vats as required. Porcelain forms are dipped into the solution in the vats and allowed to remain for a short time. The rubber deposited on the forms is then allowed to dry. The forms are repeatedly redipped until the desired thickness of rubber is obtained. They are allowed to dry and are then cured or vulcanized by immersing for about 1 minute in a 4 percent solution of sulfur monochloride in benzol. They are again allowed to dry, taken off the forms, turned inside out, again put on the forms and redipped in the sulfur monochloride in benzol, so that both sides of the glove may be cured. They are then allowed to dry, taken off the forms, and dusted with soapstone.

The heavy acid gauntlets have a chocolate-colored layer incorporated between the inner and outer layers. The chocolate-colored layer contains brown oxide of iron instead of chrome yellow. This is done because originally the users of the gloves were accustomed to wearing a glove having such a chocolate-colored middle layer. All the different styles and weights of acid-cured gloves made by this rubber company were made from rubber compound stored in one tank and had the same composition.

All the rubber compound for making acid-cured gloves mixed during the period from September 1937 to October 1938 contained the anti-

[1] Antioxidants are used in rubber to retard the ageing or oxidation of the rubber, in the course of which the action of heat, light, and oxidation causes the rubber to lose its elasticity, to crack, and to discolor.

[2] This equals 0.2 percent of antioxidant based on the weight of the glove.

oxidant monobenzyl ether of hydroquinone. However, as the large storage tank containing the solution of the rubber compound in naphtha, to which the new formula was added, was not emptied before storage of the new stock began, a considerable time elapsed before the rubber compound in the storage tank contained the full strength of 0.2 percent of monobenzyl ether of hydroquinone, and it was not until some time after October 1938 that the antioxidant had entirely disappeared from the rubber compound in the tanks.

Since it takes a considerable time after the gloves are manufactured for them to be in the hands of the users, it was probably not until October or November 1937 that any gloves containing the antioxidant were actually being worn.

In October 1938 the company received a complaint from a factory manufacturing gaskets and brake-lining materials, stating that 10 of 15 girls wearing the acid-cured gloves were affected with dermatitis. As a result of this the antioxidant was withdrawn from the formula. Because the new rubber compound was not emptied from the tanks, gloves containing some of the antioxidant were probably still being made as late as the early months of 1939 and were being sold to workers at an even later date. Sample gloves obtained from the jobber late in May 1939 failed to cause leukoderma or patch-test reactions on the involved workers. These same workers, however, showed patch-test reactions to rubber gloves manufactured in 1938.

Samples of all the ingredients going into the manufacture of the gloves were obtained from the rubber company. Workers having leukoderma were patch tested with these chemicals. The tannery first reporting the cases was selected as the place for doing the major portion of the patch tests because it had the largest number of cases and because the management and the workers showed a willingness to cooperate.

Ten workers who had leukoderma were patch tested with the following substances which went into the manufacture of the rubber gloves:

1. Chrome yellow.
2. Stearex.
3. Antioxidant.
4. Whiting.
5. Soapstone.
6. Cumar.
7. The same antioxidant.
8. Pieces of a new acid-cured rubber glove made by the rubber company.[3]

The patches were allowed to remain on for 7 days.

[3] It was later found that this rubber glove was manufactured about April 1939 and therefore could not have contained more than a trace of the antioxidant.

TABLE 1.—*Results of patch tests allowed to remain on the patients' backs [1] from July 10 to July 17*

	Patch No. 1, chrome yellow		Patch No. 2, stearex		Patch No. 3, antioxidant		Patch No. 4, whiting		Patch No. 5, soapstone		Patch No. 6, cumar		Patch No. 7, antioxidant		Patch No. 8, rubber	
	July 24	Jan. 25	July 24	Jan. 25	July 24	Jan. 25	July 24	Jan. 25	July 24	Jan. 25	July 24	Jan. 25	July 24	Jan. 25	July 24	Jan. 25
C.	-----	-----	-----	-----	+	-----	-----	-----	-----	-----	-----	-----	+	-----	-----	-----
J. T.	-----	-----	-----	-----	(+)	(++)	-----	-----	-----	-----	-----	-----	++	(++)	-----	-----
H. E.	-----	-----	-----	-----	+	(+)	-----	-----	-----	-----	-----	-----	+	(+)	-----	-----
L. E.	-----	-----	-----	-----	+	(+)	-----	-----	-----	-----	-----	-----	+	(+)	-----	-----
S. V.	-----	-----	-----	-----	(+) ++	(−)	-----	-----	-----	-----	++	(−)	(+) +	(−)	-----	-----
M. H.	-----	-----	-----	-----	+	-----	-----	-----	-----	-----	-----	-----	+	-----	-----	-----
J. J.	-----	-----	-----	-----	(+) ++++	(++)	-----	-----	-----	-----	-----	-----	(+) ++++	(++)	-----	-----
D. K.	-----	-----	-----	-----	+	(−)	-----	-----	-----	-----	-----	-----	(+) +++	(−)	-----	-----
L. P.	-----	-----	-----	-----	++	(+)	-----	-----	-----	-----	-----	-----	[2]	(−)	-----	-----
M. S.	-----	-----	-----	-----	(+) ++	(+)	-----	-----	-----	-----	-----	-----	(+) +	(+)	+	(−)

[1] All blank spaces in this table represent tests in which there were no skin reactions and no leukoderma.
[2] Lost patch.
+ and − = skin reactions.
(+) = leukoderma.
(++) = spread of leukoderma.
(−) = no leukoderma.

It will be noted from table 1 that all of these workers reacted to patch No. 3, the antioxidant, and to patch No. 7, also the antioxidant, except one (L. P.) who lost the chemical from under patch No. 7. There was only one reaction to patch No. 8, rubber glove containing only traces of the antioxidant, and one reaction to patch No. 6, cumar. There were no reactions under any of the other patches. It is possible that the reaction to patch No. 6 was due to the fact that this worker was sensitive to cumar, but since leukoderma later developed at this site it is more likely that the patch was accidentally contaminated with the antioxidant from patch No. 7 at the time of its application. On July 24, 1939, or 2 weeks after the patches were applied, there were signs of leukoderma on the antioxidant patch test sites of 5 of the workers (fig. 5). On the next inspection, made on January 25, 1940, 8 of the 10 workers were seen and 3 of them who had shown no leukoderma on July 24 now showed leukoderma at the site of the patch test with the antioxidant (fig. 6). Areas of leukoderma produced by the patch test that were noted on 2 workers at the time of the first inspection had by this time become repigmented. There seemed to be no definite correlation between the intensity of the skin reaction to the antioxidant patch tests and the subsequent development of leukoderma. The leukoderma began to develop about 1 week after the removal of the patches in the majority of the cases. It reached its peak some time between July 24, 1939, and January 25, 1940, so that by January 25, 1940, 2 of the leukodermic patch test sites had become entirely repigmented and the others showed areas of repigmentation.

Repigmentation of the skin on the arms first occurred on small sites around the hair follicles scattered over the leukodermic area (fig. 7). The skin first appeared a light brown color, the color later increasing in intensity. The areas of repigmentation spread and coalesced (fig. 8).

According to the manufacturers the antioxidant is monobenzyl ether of hydroquinone, containing a fraction of 1 percent of unchanged hydroquinone as an impurity. It is a light tan-colored powder with an aromatic odor, melting at 115° to 120° C., and having a specific gravity of 1.26. It is very slightly soluble in water, practically insoluble in petroleum hydrocarbons, but soluble in benzol and in rubber up to 2 percent. It is said to be nontoxic in ordinary handling and not to "bloom" when used up to 1 percent on the rubber. It is nondiscoloring in diffuse daylight and gives a minimum discoloration in direct sunlight. For this reason it is recommended by the manufacturers for use in white and light-colored rubber goods.

The workers continued at their occupations while wearing the patches and we thought that perhaps some of the chemicals with which they worked may have had an influence on the patch tests. In order to determine further whether the tanning liquors containing these chemicals had some effect on the ingredients in the rubber and formed new compounds which may have caused the leukoderma, pieces of the rubber glove used for patch test No. 8 were dipped into the various tanning liquors and applied as a patch test on 6 other workers affected with leukoderma.

Patch No. 1 consisted of the rubber dipped in a 1 percent solution of dimethylamine. (These patch tests were done in the tannery in which the dimethylamine was used in conjunction with lime as an unhairing agent.)

Patch No. 2 consisted of the rubber dipped in the actual unhairing solution consisting of a mixture of dimethylamine and lime. Since this unhairing solution is strongly alkaline and therefore caustic and would cause dermatitis on the normal skin if applied as a patch test, the alkalinity was reduced to pH 11 by the addition of a few drops of sulfuric acid. (It has been found that the normal skin will withstand for 24 hours a patch test with a solution having a pH 11.)

Patch No. 3 consisted of a piece of the rubber dipped into the tanning solution. The tanning solution is a green liquid consisting of about 1.5 percent of chromium sulfate and sufficient sulfuric acid to give it a pH of 3.3.

Patch No. 4 consisted of a piece of the rubber dipped in the liquid expressed from the leather taken out of the fat liquor drums. This liquor consists of remnants of the various dyes and oils used in the fat liquoring process, and is approximately neutral in reaction.

These patches were taken off each day and inspected, remoistened with the liquors, and then reapplied for a total period of 7 days.

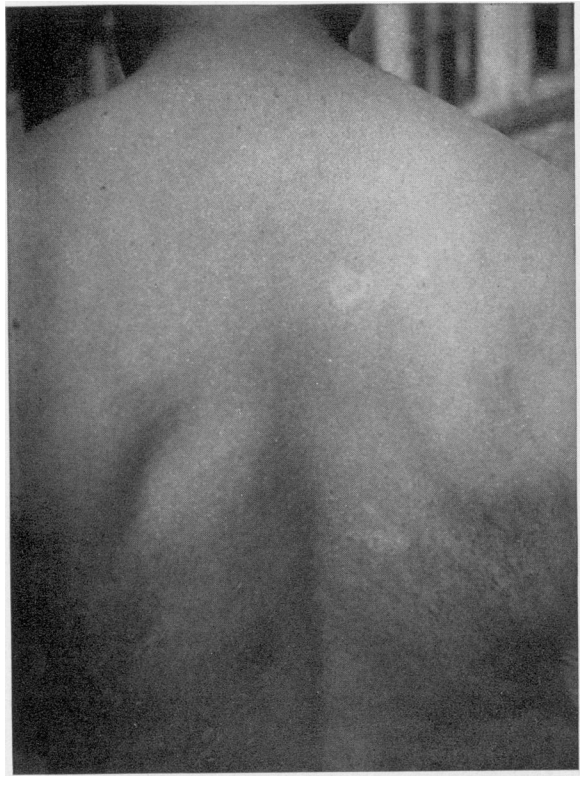


FIGURE 5.—Leukoderma at sites of patch tests with antioxidant powder.

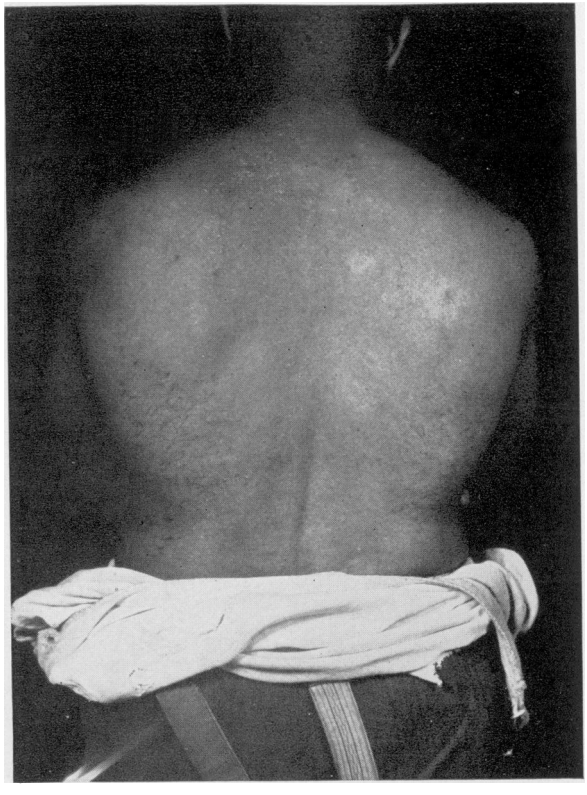


FIGURE 6.—Leukoderma at sites of patch tests with antioxidant.

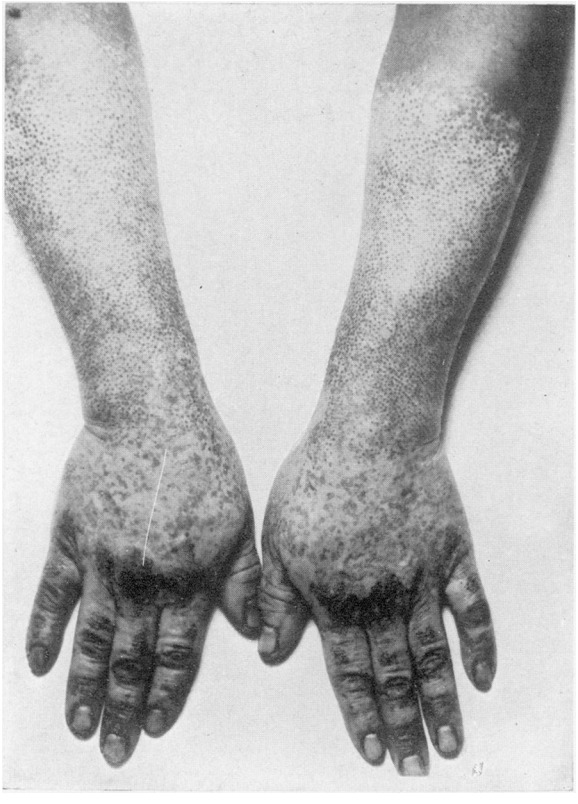


FIGURE 7.—Perifollicular repigmentation.

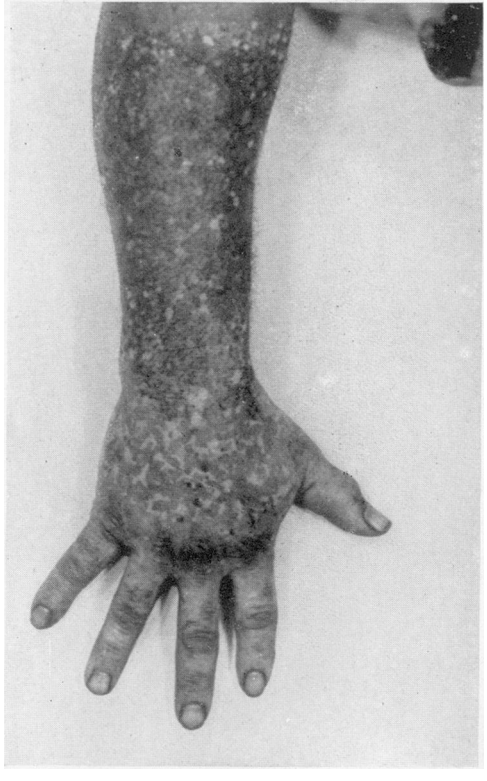


FIGURE 8.—Coalescent repigmentation.

TABLE 2.—*Results of patch tests allowed to remain on the patients' backs from July 11 to 17*

	Patch No. 1	Patch No. 2	Patch No. 3	Patch No. 4
	July 24	July 24	July 24	July 24
O. M.	−	−	−	−
J. P.	−	−	−	−
P. B.	−	−	−	−
C. Mc.	−	−	−	−
J. T.	−	−	−	−
T. D.[1]	+	+	+	+

[1] Delayed reactions appearing one week after patches were removed.

It will be noted that there was no reaction under any of these patches at the end of the week. However, when the patch test sites were again seen 7 days after removal, one of the workers had a mild reaction under each of the 4 patches. This worker was the janitor and was not occupationally exposed to any of the tanning solutions, although he did have leukoderma caused by wearing the gloves while he was engaged in washing and scrubbing. This worker was either sensitive to something in the rubber or to all of the 4 tanning liquors. No leukoderma developed at the site of these reactions, because, as stated before, this rubber glove contained only a trace of the antioxidant. This experiment shows that the tanning liquors did not act on the other chemicals in the rubber gloves in such a way as to change their chemical composition and cause leukoderma.

In order to determine whether acid curing affected the action of the antioxidant in causing leukoderma, the rubber company manufacturing the gloves compounded a set of samples of the rubber glove in the various stages of manufacture.

Sample No. 1 consisted of the antioxidant, 1 part, and smoke sheet rubber, 100 parts, milled together and rolled into a sheet.

Sample No. 2 consisted of the antioxidant, 0.5 parts, smoke sheet rubber, 100 parts, and cumar, 3 parts, milled together and rolled into a sheet.

Sample No. 3 consisted of sample No. 2, plus chrome yellow, 5 parts, milled and rolled into a sheet.

Sample No. 4 consisted of sample No. 3, plus whiting, 125 parts, milled and rolled into a sheet.

Sample No. 5 consisted of sample No. 4, plus stearex, 10 parts, milled and rolled into a sheet.

Sample No. 6 consisted of sample No. 5, with the exception that chrome yellow was omitted and for it was substituted 1.25 parts of powdered oxide of iron, milled and rolled into a sheet.

Sample No. 7 consisted of sample No. 1 dissolved in petroleum naphtha in the proportion of 1 part of rubber to 7 parts of naphtha, deposited on a form and acid cured as described in the process of acid cured glove manufacture.

Sample No. 8 consisted of sample No. 2 treated in the same way as sample No. 7.

Sample No. 9 consisted of sample No. 3 treated in the same way as sample No. 7.

Sample No. 10 consisted of sample No. 4 treated in the same way as sample No. 7.

Sample No. 11 consisted of sample No. 5 treated in the same way as sample No. 7.

Sample No. 12 consisted of sample No. 6 treated in the same way as sample No. 7.

We at first planned to patch test all the workers who had leukoderma with these 12 samples of vulcanized and unvulcanized rubber, but by the time that these samples were finished and received it had already been established that the antioxidant was the sole cause of the leukoderma. Hence it only remained to find whether vulcanization hastened or retarded its action, and also to find whether the small amount of hydroquinone present as an impurity played any part in causing the leukoderma.

Heinz Oettel (Archive für Experimentelle Pathologie und Pharmakologie, **183**:319 (1936)) performed experiments with hydroquinone in connection with its contemplated use as a food preservative. During the course of these experiments Oettel fed cats daily doses of hydroquinone and observed among other signs of chronic hydroquinone poisoning that there was a depigmentation of hair. Thus, as a result of the oral ingestion of hydroquinone, the fur of black cats was temporarily changed to gray.

An ointment consisting of 20 percent of monobenzyl ether of hydroquinone in benzoinated lard was applied daily to the backs of 4 workers at the first tannery. On 3 of the men this ointment was rubbed over an area of the skin on which in the previous experiment the antioxidant powder had been applied for 1 week and no leukoderma had developed. The fourth man had not previously been patch tested with the antioxidant.

The ointment containing the antioxidant, and the 20 percent solution of the antioxidant in ether were applied 5 days a week for a period of 6 weeks to the skin of the four Negroes. Depigmentation on these sites was first observed 39 days after the beginning of these applications and upon final observation 5 months afterward all of the men showed large areas of depigmentation on the back where the ointment and the solution were applied.

To another small area on the back of these men a 20 percent solution of the antioxidant in ether was applied freshly each day.

Twenty-five workers were patch tested with vulcanized rubber containing nothing but 1 percent of the antioxidant. At the end of 1 week the rubber patches were removed, and 22 of these 25 men were examined. Four of these showed positive reactions at the site of the rubber patches, and 1 of the 4 showed a desquamation of the skin, underneath which there was a definite leukoderma. One of the 3 who did not show leukoderma at the time the patches were removed

developed leukoderma at this patch test site 2 weeks afterward. At this time (2 weeks afterward) 2 others showed ill-defined leukodermic spots at the site of the patch and 2 showed a brown scaling at the patch test sites.

Upon final examination on January 25, 1940, 12 out of 19 of the 25 workers patch tested with the vulcanized rubber containing nothing but 1 percent of the antioxidant showed leukoderma at the site of this patch test (fig. 9).

In the second tannery 4 workers were patch tested with the unvulcanized rubber containing nothing but 1 percent of the antioxidant. An ointment of 20 percent of hydroquinone in benzoinated lard was rubbed into an area of the back. A saturated solution of hydroquinone in ether was applied to a similar area. The rubber was allowed to remain on for 72 hours, at the end of which period 2 of the workers showed an inflammatory reaction consisting of erythema and vesicles. No leukoderma was present at this time.

The ointment was applied daily for 4 days, as was the saturated solution of hydroquinone in ether. There were no reactions noted at the end of this period.

These workers were again examined 16 days later, at which time there was a reaction at the site of the unvulcanized rubber patch on another one of them.

They were next observed 5 months afterward, and at this time 2 of them showed areas of depigmentation 1 inch square at the site of the unvulcanized rubber containing the antioxidant patch.

These experiments show that the antioxidant, alone or contained in rubber, either unvulcanized or vulcanized, can cause skin reactions and leukoderma even when applied to the skin for only 72 hours, and that the hydroquinone when applied in strong concentration for 96 hours produced no leukoderma on the same subjects. It therefore seems that the small percentage of hydroquinone contained as an impurity in the antioxidant played no role in the production of leukoderma.

As stated before, only 52 percent of the workers in tannery No. 1 who wore the gloves developed leukoderma. In some of the other factories investigated only about 10 percent of the workers who wore the gloves developed leukoderma. These facts indicate that perhaps some of the workers wore the gloves for longer periods of time than others and thus received larger doses of the antioxidant, or that some of the workers were more susceptible to its action than were others.

In order to find out whether leukoderma could be produced on the normal skin if a sufficient amount of the antioxidant were applied for a sufficient length of time, one of us (E. A. O.) applied the antioxidant in the form of an ointment and in the form of a solution in ether to the

skin of a colored patient in the hospital. Applications were made daily to a circumscribed area of the skin for 2 weeks, at the end of which time leukoderma developed. The leukoderma increased in intensity, and 4 months afterward there was still a definite leukoderma present at the site of the applications (fig. 10).

At the same time, one of us (L. S.) applied a specially prepared piece of vulcanized rubber containing nothing but 5 percent of the antioxidant on a freckled area of his own skin over the right shoulder. The rubber patch was allowed to remain on for 7 weeks. During this period a reaction consisting of erythema and vesiculation was observed at the site of the patch, but despite this reaction the patch was allowed to remain on. When the patch was removed the skin site showed a dermatitis, but no leukoderma or disappearance of the freckles. Since the leukoderma on the white workers was evidenced only after exposure to sunlight, the patch test site was exposed to 2 erythema doses of a mercury vapor lamp, divided into 2 sittings 1 day apart. There was a marked reaction in the form of an erythema at the site of the patch test and a much milder reaction of the surrounding skin, but still no visible leukoderma. The reaction went on to slight scaling and the area of skin surrounding it became tanned, but the site of the patch was erythematous and untanned. The site was observed from day to day, until 28 days later definite leukodermic spots were noticed over the area of the patch test site, and some of the freckles had disappeared.

These experiments show that if the skin is exposed for a sufficient length of time to a strong concentration of monobenzyl ether of hydroquinone it will in the course of 3 or 4 months become definitely depigmented.

After the investigation began, and after gloves containing more than traces of the antioxidant were no longer sold, no new cases of leukoderma developed on any of the workers observed.

At the end of our experiments, which occupied a period of 7 months, it was noted that all of the workers affected showed definite signs of returning pigment over the leukodermic areas (figs. 11 and 12). This indicates that the pigment-forming mechanism of the skin was interfered with but not destroyed by the antioxidant.

Biopsies were performed and the sections studied both by regular staining methods and by the dopa technique. The following is the report of examination of these sections by Dr. Frederick D. Weidman, of the University of Pennsylvania:

Patient J. T., slide No. 2802, a section showing repigmentation stained with the dopa reagent.

There is very slight hyperkeratosis in one portion of the section. Acanthosis is negligible. Pigmentation is spotty in distribution, and extreme in degree along a certain short extent of the epidermis. There is a rather singular occurrence,

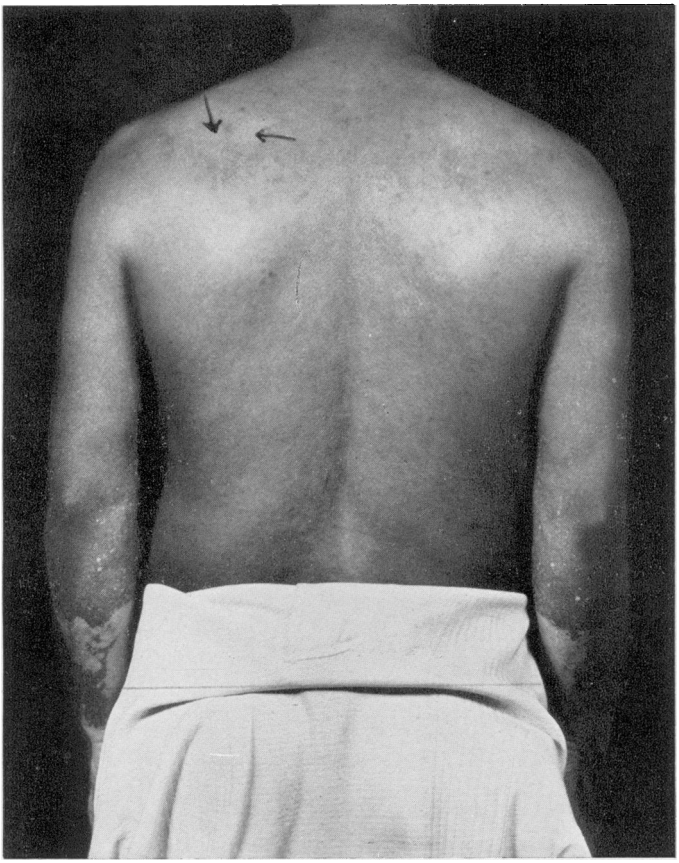


FIGURE 9.—Leukoderma at site of patch test with 1 percent antioxidant in vulcanized rubber.

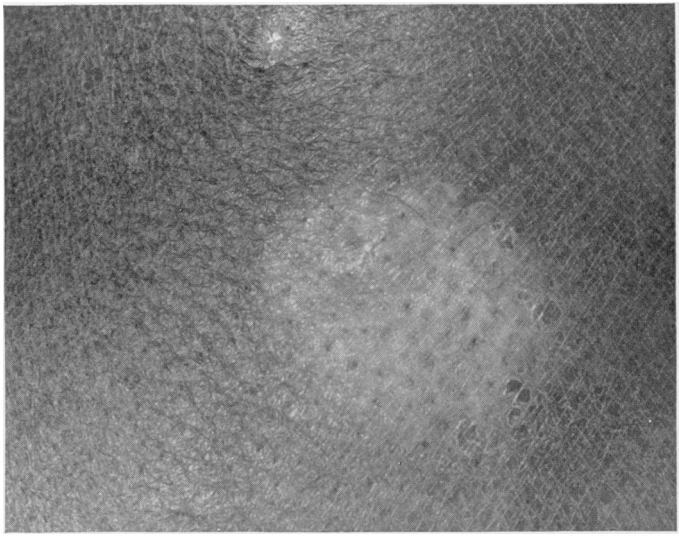


FIGURE 10.—Leukoderma produced by local applications of antioxidant.

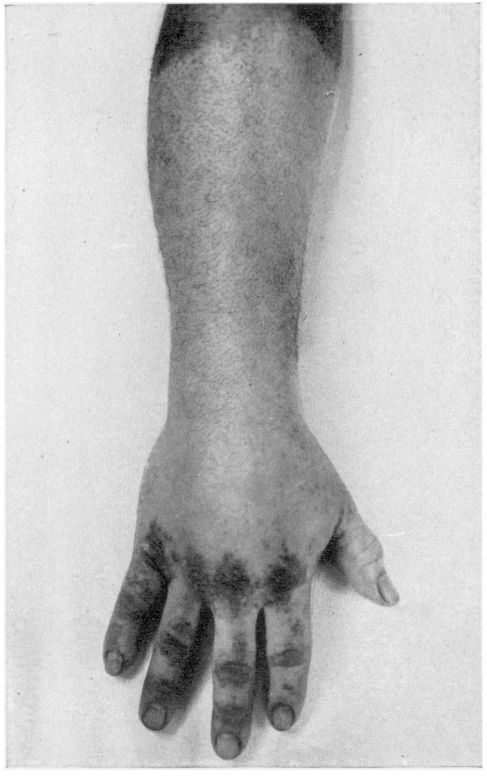


FIGURE 11.—Before regeneration of pigment began.

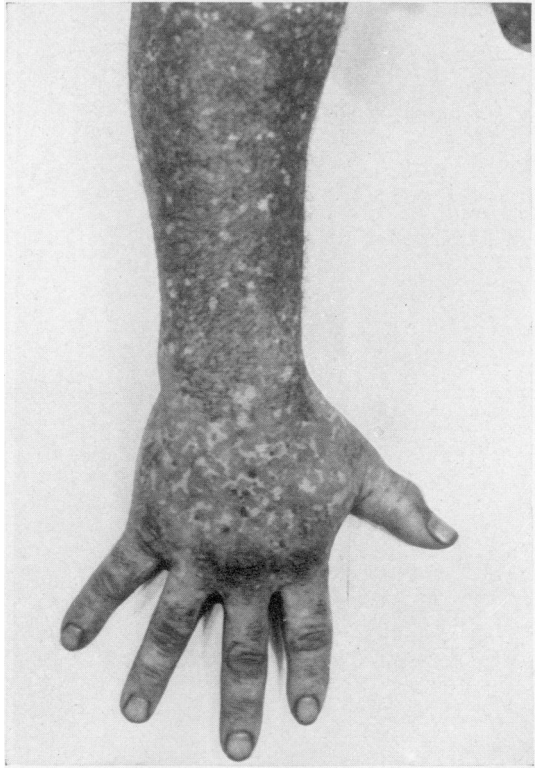


FIGURE 12.—Regeneration of pigment. Same patient as in figure 11, but 5 months later.

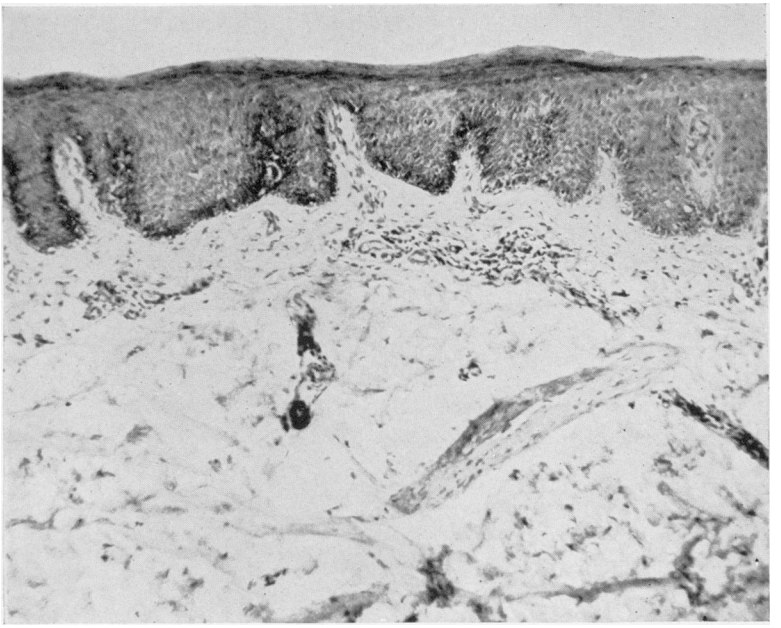


FIGURE 13.—Section from junction of normal and depigmented skin (dopa stain).

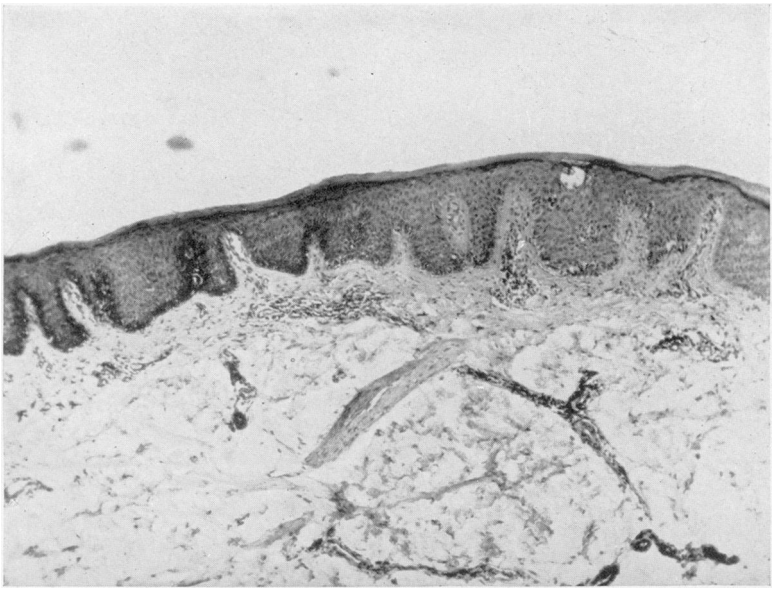


FIGURE 14.—Section from junction of normal and depigmented skin (hematoxylin and eosin stain).

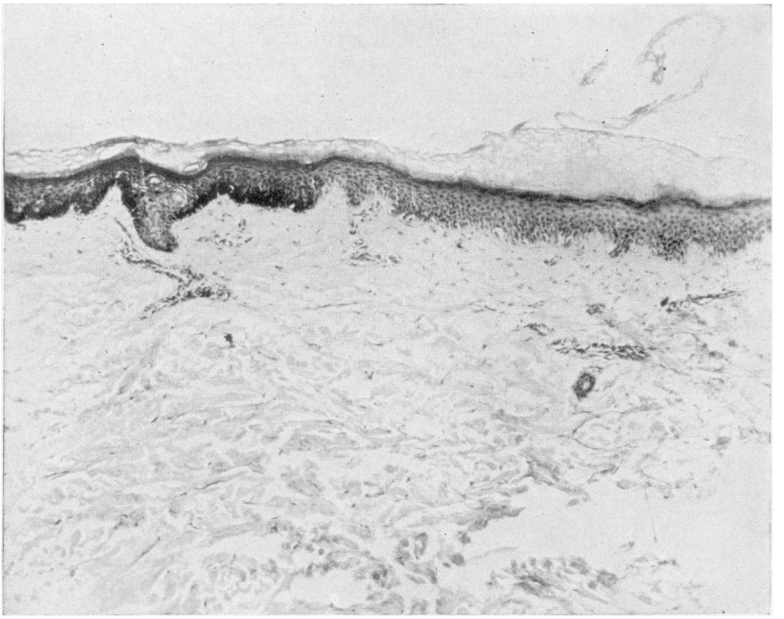


FIGURE 15.—Section from junction of normal and depigmented skin (dopa stain).

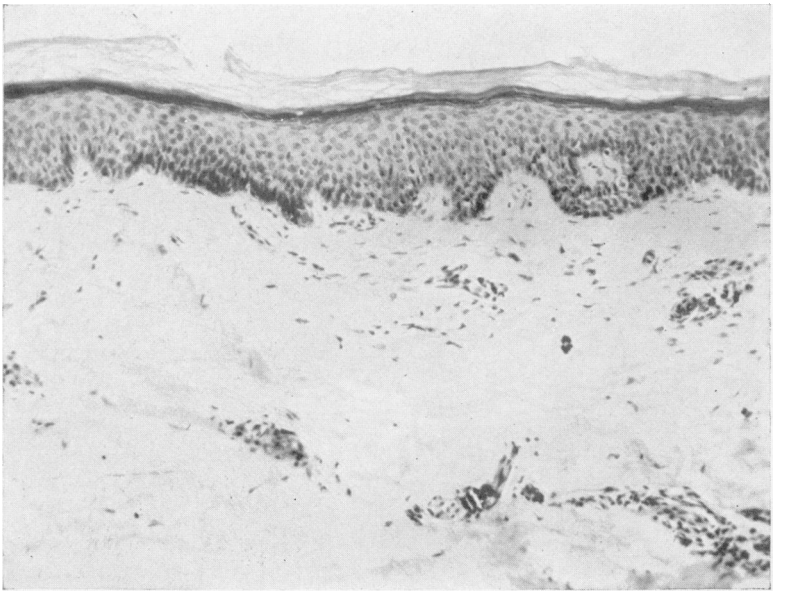


FIGURE 16.—Section exhibiting patchy depigmentation (dopa stain).

namely, that in the stratum corneum and the stratum granulosum the pigment has tended to accumulate in a way that does not appear consistent with the amount of pigment in the basal layers—a very distinctive appearance. In the deeply pigmented foci, pigmentation is greatest in the basal cell layer, with marked hyperpigmentation in the chromatophores in the tips of the dermal papillae. In the less pigmented areas there is also hyperpigmentation in these chromatophores. In general, there is no marked hyperpigmentation in the basal cell layer. The localization or distribution of the pigment is utterly irregular, but even where the pigmentation is not heavy there are fine pigment granules in the intercellular spaces. The dendritic cells are sharply outlined by powdery pigment grains in their stellate processes which extend far up into the epidermis. In all cases the pigment is extremely fine and powdery.

Interpretation of the pigmentation.—The primary activity is in the chromatophores which are present even around the sweat glands, but are most abundant in the tips of the papillae and the blood vessels of the subpapillary layer. The dendritic cells in the epidermis have engulfed this pigment, thus accounting for its transportation upward into the epidermis. A rather unusual phenomenon is this accumulation of pigment in the stratum granulosum and to a somewhat lesser extent in the stratum corneum. The basal cells themselves are not the site of this pigment production. In those places where the basal layer is pigmented it is due more to intercellular location of the pigment. However, there are still small quantities of pigment in the cytoplasm. The corium has a diffuse tingeing of the collagen bundles in addition to the pigment granules. In the sweat glands the cytoplasm of the secretory cells in particular, and to a less extent of the collecting ducts, have excessively finely granular, powdery pigment. The nuclei are not compromised except where the pigment is particularly dense—in which case the nucleus is somewhat pyknotic. There are no hair follicles or sebaceous glands in this section. Even the involuntary muscle around the sweat tubule is stained a diffuse brown. The arrectores pilorum muscles and also the involuntary fibers in the vessel walls have escaped involvement. In the lumina of the sweat glands is a rather shreddy, diffusely brown tinted material with some granules. One peculiar feature is the "backing up" or retention in the stratum granulosum of the above mentioned pigment.

Slide No. 2801, J. T., a section of leukoderma and adjacent normal skin stained with hematoxylin and eosin.

This section is the same as the one above, but the area of intense hyperpigmentation is more extensive. There is a lymphocytic proliferation around one blood vessel. There is a very marked number of chromatophores in the tips of the derma papillae. The pigment is intercellular in the basal layer and evidently located in the dendritic processes. The sweat gland cells are dopa positive (as noted in section 2802 above), but without pigment grains in these cells.

Summary of biopsy findings.—The melanin pigment is present in chromatophores which occupy the dermal papillae and in the intercellular spaces of the basal layer of the epidermis. There are also small quantities of pigment within the cytoplasm of the basal cells. The dopa reaction brings out (in addition to the findings under hematoxylin and eosin staining) that the cells of the granular layer of the epidermis are dopa positive. There are dopa positive granules in the stratum corneum and in the secretory cells of the sweat glands.

The following are reports from different sections made by Dr. J. W. Miller, of the United States Public Health Service:

2801, J. T. (fig. 13). Normal skin and leukoderma. Sections treated with dopa show a rather sharp demarcation between the area of leukoderma and normal

skin. There is a gradual decrease in the number of pigment particles in the cells of the basal layer as the leukodermic area is approached. Dopa positive particles occur in cells of the stratum granulosum, cells of the sweat glands and as intra and extracellular particles in the dermis and as isolated particles in the stratum corneum.

A similar picture is noted in sections stained with hematoxylin and eosin (fig. 14). Fine melanin particles also occur in the stratum germinativum and are apparently extracellular. Such particles are also noted in the dermis in connective tissue cells and in small stellate cells under both normal and leukodermic areas.

2802. Repigmented. Dopa treated sections showed areas containing many dense staining dopa particles shading into areas with no particles in the basal cells. In corresponding hematoxylin and eosin sections much less pigment was noted in the basal cells. Pigment particles also occurred in the stratum granulosum. This section is very similar to 2804. Pigment particles occur in the stratum corneum overlying the pigmented areas, but not over the nonpigmented areas.

2803, O. M. Mc. Normal and leukoderma (fig. 15). Dopa positive particles are found in the pigmented basal cell layer of the normal skin portion of the section. At the junction of the area of leukoderma and normal skin there is a preponderance of dopa positive particles in the last four or five cells before a sharp line of demarcation between the normal and leukoderma area. No dopa positive particles occur in the basal layer of the area of leukoderma.

Dopa positive particles are noted in the dermis of the nonpigmented area, both isolated and some apparently in cells.

The same tissue stained with hematoxylin and eosin shows a similar sharp line of demarcation between the pigmented and nonpigmented cells of the basal layer, but the last few cells of the pigmented portions contain fewer pigment particles than the adjacent cells in the normal pigmented portion. Melanin particles appear throughout the dermis under both portions of the section. Other than absence of pigment, no pathological changes are noted.

2804. Repigmented (fig. 16). Areas of dopa positive particles in the basal cell layer are interspersed by areas of pigment-free cells. There is variation in size of these areas. No dopa positive particles are found in the depigmented regions. There is a gradual shading in the pigmentation from either zone. Sections stained with hematoxylin and eosin show a similar picture. The amount of melanin in the basal cell layer of the repigmented portion is less than that noted in the normal skin in the other section from the same case. No inflammatory reaction was present in the sections studied.

Discussion

THEORIES OF ANTIOXIDANT ACTION IN RUBBER

Although the action of antioxidants in rubber is not well understood there are several theories in this regard. One theory is that the antioxidant, having a stronger affinity for oxygen than the rubber, becomes oxidized before the rubber. The fact that antioxidants in rubber lose their action after a time seems to substantiate this theory. Another theory is that, although the antioxidant takes up the oxygen to which the rubber is exposed, it rapidly gives it up in an inactive form. If this were entirely true, the action of the antioxidant would last indefinitely. A third theory is that the presence of the antioxidant in the rubber prevents oxidation of the rubber taking place, although

the antioxidant itself is not acted upon. In other words, it acts like a negative catalyst. If this were true, the presence of the antioxidant would again prevent the rubber from deteriorating at all. However, antioxidants in rubber lose their action after a time, as is evidenced by the rubber becoming sticky, resinous, and losing its tensile strength and elasticity, despite the presence of even large amounts of antioxidant. The fact that the antioxidant is used up in the rubber is proved by experiments which show that more antioxidant can be recovered from newly compounded rubber than from old rubber. Experiments have shown that about 90 percent of the antioxidant freshly placed in rubber can be recovered, whereas very much less than this can be recovered from old rubber. The oxidation products of the usual antioxidants are dark colored compounds. The oxidation product of monobenzyl ether of hydroquinone is much lighter in color than are the oxidation products of other antioxidants. Sunlight plays an active part in causing the oxidation of antioxidants. This is proven by the fact that in the absence of sunlight, antioxidants do not darken as much as when sunlight is present. The amount of antioxidant placed in rubber depends upon the required service of the rubber, the solubility of the antioxidant in the rubber, the effect of the antioxidant on the rubber matrix, and the cost. The more antioxidant up to a certain amount that there is placed in the rubber, the longer is oxidation of the rubber delayed. But there is always a certain amount of oxidation of the rubber itself taking place in spite of the presence of the antioxidant. In other words, when oxygen is present, most of it will combine with the antioxidant, but some of it will also combine with the rubber.

Heat cure does not affect the action of antioxidants on rubber, nor does it seem to affect their chemical composition. The acid cure, however, causes rapid color changes with most antioxidants, because most antioxidants are attacked by active chlorine compounds. In other words, sulfur chloride oxidizes the ordinary antioxidants. Acid cure, however, does not affect monobenzyl ether of hydroquinone as much as it does other antioxidants, and does not cause it to discolor. This is the reason why this antioxidant is used in light-colored, acid-cured rubber goods.

The action of monobenzyl ether of hydroquinone on the skin in producing leukoderma may very well be the same as it is in preventing the oxidation of rubber. The melanin of the skin is theoretically supposed to be formed by the oxidation of the propigment with a special oxidase. Monobenzyl ether of hydroquinone, being absorbed into the skin because of its solubility, prevents this oxidation from taking place. That monobenzyl ether of hydroquinone itself is used up in this process is shown by the fact that after the workers were no

longer exposed to monobenzyl ether of hydroquinone, the pigment returned.

The biopsies do not show any injury to the cells theoretically involved in the melanin production. This despite the fact that some of the workers stated that they had a dermatitis before the leukoderma developed, and one of the authors (L. S.) developed a marked sensitivity to monobenzyl ether of hydroquinone after having applied it to his skin and had a marked dermatitis over the area before the development of leukoderma. There was no scarring following the sensitization dermatitis in any of the cases.

Antioxidants have been used in rubber for many years, but never before has leukoderma been reported from wearing rubber goods. Therefore, it seems either that monobenzyl ether of hydroquinone has a physiologic action on the skin different from that of other antioxidants, or that other antioxidants do not act on the skin. The action of monobenzyl ether of hydroquinone in rubber differs from that of other antioxidants in that it allows less discoloration of the rubber. This is because the oxidation compound of monobenzyl ether of hydroquinone does not darken as much as do the oxidation compounds of other antioxidants. The reason why monobenzyl ether of hydroquinone causes leukoderma, and leukoderma has not been reported to have been caused by other antioxidants, which like monobenzyl ether of hydroquinone discolor only slightly upon oxidation, may be because monobenzyl ether of hydroquinone is more soluble in water and hence more readily absorbed. It is also possible that these other antioxidants have not been used in rubber goods which are worn next to the skin for long periods of time. Monobenzyl ether of hydroquinone is quite freely soluble in alkalies, and the fact that most of the workers who developed leukoderma from wearing the rubber gloves worked with alkalies may have been a factor in dissolving the antioxidant from the rubber and allowing it to come in contact with the skin in an absorbable state.

It was first thought that perhaps there was an excess of monobenzyl ether of hydroquinone in the rubber which caused it to bloom out and come in contact with the skin and thus be absorbed, but an examination by competent rubber chemists of the rubber gloves containing it did not show any bloom. It was then thought that perhaps the antioxidant was dissolved out of the rubber by the perspiration, and to test this a piece of the rubber glove containing monobenzyl ether of hydroquinone was soaked in water containing sufficient acetic acid so that the solution had a pH of 4, and another piece was soaked in water containing a sufficient amount of alkali to give it a pH of 8, these being about the limits of the range of the pH of the perspiration. To 100-cc. of each solution in a 200-cc. flask were added 5 grams of the rubber gloves cut into thin strips. Since the

maximum concentration of antioxidant in the rubber compound was 0.2 percent, this amount of rubber contained 10 milligrams of monobenzyl ether of hydroquinone. At the end of 14 days each of these two solutions extracted 1 milligram of the antioxidant from the rubber, or about 10 percent of all the antioxidant contained in the rubber was taken out by these solutions. This experiment showed that perspiration, regardless of its pH, could dissolve monobenzyl ether of hydroquinone out of the rubber. It is possible that if the rubber had been left in the solution for a longer period of time, the water would have taken more of the antioxidant out of the rubber, perhaps even the entire amount, because 100 cc. of water is capable of dissolving 100 milligrams of monobenzyl ether of hydroquinone, or about 10 times the amount that was contained in the 5 grams of rubber. This experiment proved that the perspiration of the workers could dissolve this antioxidant out of the rubber gloves which they wore.

The workers exposed to the antioxidant in the rubber gloves wore the gloves for many months before leukoderma developed. In this period of time it was possible that the perspiration dissolved a considerable amount of monobenzyl ether of hydroquinone out of the rubber, and that some of this was absorbed into the skin. This hypothesis is made even more plausible because the workers all worked in alkalies in which the monobenzyl ether of hydroquinone is very much more soluble than it is in water.

In the experiments in which we moistened the sample of the glove with the tanning liquids and performed patch tests, and obtained no leukoderma, it was found that the glove with which we performed the patch test was manufactured many months after the monobenzyl ether of hydroquinone had been taken out of the rubber stock. This accounts for the fact that we failed to obtain positive patch tests or leukoderma from the experiment.

The action of monobenzyl ether of hydroquinone in the skin may very well be the same as it is in the rubber, that is to prevent the formation of color compounds, i. e., the formation of melanin in the skin. This may be due to the monobenzyl ether of hydroquinone uniting with dopa oxidase to form a colorless compound, and thus preventing the dopa oxidase from combining with propigment to form melanin. Or the monobenzyl ether of hydroquinone may so injure the melanoblasts that they cannot produce oxidase. Or the monobenzyl ether of hydroquinone may prevent the formation of propigment or even destroy it. That the inhibition of propigment formation, or its destruction, is not the cause of leukoderma in these cases is shown by the fact that the dopa reaction is negative. The dopa reagent theoretically takes the place of propigment, and if free oxidase were present in the cell, the dopa reaction would have been positive. There are no direct reactions known at present which may be used

to determine whether propigment is or is not present. If it were possible to supply the dopa oxidase to frozen sections as it is possible to supply dopa to them, we might be able to ascertain whether or not propigment is present. Since the dopa reaction was negative in the biopsied sections of leukoderma, the propigment cannot be the missing factor in the leukodermic skin.

Therefore, it must be either that the oxidase producing power of the cell is injured or that the monobenzyl ether of hydroquinone prevents the oxidase from forming a color compound with the propigment. The fact that the cells were not injured is shown by the biopsies. The cells appear normal except for their lack of pigment. Therefore, there only remains the theory that the antioxidant prevents the combination of oxidase and propigment. Whether the monobenzyl ether of hydroquinone acts as a negative catalyst in preventing the combination of oxidase with propigment or whether it actually combines with the oxidase itself can only be theoretically surmised. It is also possible that the antioxidant may combine with the oxidase and then give it up in an inactive form, the antioxidant then being capable of combining with more oxidase, etc. Whether it acts in this manner or as a negative catalyst, once in the cell its action would continue indefinitely and repigmentation would not take place even though exposure to more of the antioxidant was prevented, unless metabolism removed the antioxidant from the cell. The fact that repigmentation took place after the antioxidant was removed from the rubber glove shows either that the monobenzyl ether of hydroquinone had been completely oxidized by oxidase and became inactive, or that it was removed by metabolism. In rubber the antioxidant combines with oxygen and is used up in that manner; hence it seems reasonable to suppose that this same action takes place in the skin.

Monobenzyl ether of hydroquinone is not a pigment bleach. It does not decolorize melanin, but simply acts to prevent the formation of melanin. The depigmentation does not appear until the melanin already present in the skin has been absorbed or destroyed by the metabolism. That this takes a considerable length of time is shown by the fact that the leukoderma did not appear in most of the cases until weeks or months after the application of monobenzyl ether of hydroquinone. If it takes such a long period for the melanin to disappear from the skin, it is conceivable that it takes an equal period for a deposit of monobenzyl ether of hydroquinone to disappear from the skin. This accounts for the many months elapsing after cessation of contact with it, before the leukodermic areas showed repigmentation.

It may be possible that other antioxidants would produce leukoderma if they could be absorbed by the skin. Perhaps if they were dissolved in a vegetable or animal oil and applied to the skin they

could produce leukoderma. But the depigmentation would not be as apparent as that produced by monobenzyl ether of hydroquinone, because the oxidation products of other antioxidants are not colorless compounds, and just as they cause discoloration of rubber they would cause discoloration of the skin and prevent the observation of the disappearance of the melanin.

It was noted in our experiments and in the cases of leukoderma studied that the hairs in the leukodermic areas were not decolorized. This points to a separate pigment-forming mechanism for the skin and hairs. There is, however, a possibility that the antioxidant did not penetrate to the deeply placed hair papillae. But the fact that senile graying of the hair occurs without concomitant loss of pigment in the skin tends to corroborate the theory of separate pigment-forming mechanisms for skin and hair.

As to the use of monobenyzl ether of hydroquinone for bleaching the skin or decolorizing hyperpigmented areas, it must be borne in mind that it takes a long time for depigmentation to be produced by this antioxidant. Moreover, there is a possibility of its action spreading to the surrounding normal skin and depigmenting a larger area than was intended. The fact that sensitization to it was produced in some individuals (produced on L. S.) must also be borne in mind.

Summary

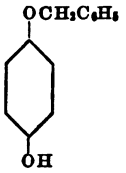
An outbreak of leukoderma among the workers in a tannery was investigated.

It was found that about 50 percent of those wearing a heavy acid-cured rubber glove were affected.

Investigations showed that a considerable percentage of workers wearing the same make of glove in other tanneries and in other industries were similarly affected.

The makers of the glove were traced and their cooperation was obtained in an effort to determine what chemical in the glove caused the leukoderma.

By patch testing with the various chemicals in the glove, it was found that the antioxidant, said by the makers to be monobenzyl ether of hydroquinone,



containing less than 1 percent impurity of unchanged hydroquinone, was the cause of the leukoderma.

It was proved that the impurity of hydroquinone was not a factor in causing the leukoderma.

Repigmentation of the leukodermic areas followed the withdrawal of the antioxidant from the rubber glove.

The fact that the hairs in the leukodermic areas were not depigmented suggests that there may be a separate pigment-forming mechanism for the skin and the hairs.

Leukoderma was experimentally produced on normal skin by long-continued application of this compound.

In some instances allergic reactions were elicited by long-continued applications of the compound.

The theories of the action of antioxidants in rubber are discussed.

It seems likely that the action of the antioxidants in the skin is similar to their action in rubber.

The possibility of using antioxidants for the depigmentation of hyperpigmented areas of the skin is suggested.

The general health of the workers was not affected.

TABLE 3.—*Laboratory studies on workers having leukoderma made at beginning of investigation*

Patient No.	pH of surface of skin	Wassermann test	Kahn test	Red blood cells, in millions	Hemoglobin percentage, based on 15.3 gm.	Hemoglobin, gm. per 100 cc.	Color index	Reticulocytes per 100 red cells
1201		−	−	4.56	81.9	12.6	0.89	0.06
1202		(1)	−	4.86	90.9	14.1	0.9	0.04
1203		−	−	4.69	84.5	13.9	0.9	0.24
1204	5.0+ to 5.7	(1)	−	4.74	92.8	14.8	0.9	0.13
1205	5.0+ to 5.7	−	−	4.75	92.8	14.3	0.9	0.14
1206		−	−	4.86	85.8	13.2	0.89	0.1
1207	5.0+ to 5.7	−	−	5.30	102.8	15.8	0.9	0.14
1208	5.9	(1)	−	5.02	79.4	12.2	0.79	0.08
1209		−	−	5.11	101.8	15.6	0.9	0.39
1210		−	−	4.86	90.3	13.9	0.9	0.1
1211		(1)	−	5.05	78.1	12	0.78	0.2
1212	5.0+ to 5.7	−	−	4.78	90.9	14.1	0.9	0.14
1213		−	−	5.09	90.3	13.9	0.9	0.09
1214		−	−	4.89	95.2	14.6	0.9	0.17
1215		−	−	4.96	80	12.3	0.8	0.24
1216		(2)	(2)	4.56	87.1	13.4	0.9	0.1
1217		−	−	4.86	87.1	13.4	0.9	0.18
1218	5.0+ to 5.7	(1)	(1)	4.86	83.9	12.9	0.8	0.08

Patient No.	White blood cells	Neutrophiles	Eosinophiles	Lymphocytes	Monocytes	Immature (staff)
1201	5,800	52	1	42	3	2
1202	4,600	60	3	36	1	0
1203	8,600	76	2	22	0	0
1204	7,900	65	2	32	1	0
1205	7,200	62	6	29	2	1
1206	7,100	43	0	55	1	1
1207	6,800	42	1	51	6	0
1208	11,700	67	0	23	7	3
1209	5,800	67	4	26	3	0
1210	7,100	53	1	43	0	3
1211	4,300	56	3	37	4	0
1212	8,000	78	1	17	4	0
1213	7,400	61	1	32	6	0
1214	5,100	65	1	33	1	0
1215	9,600	57	3	36	1	0
1216	7,100	57	3	30	5	0
1217	5,100	53	4	40	3	0
1218	5,600	77	1	19	3	0

[1] Anticomplementary. [2] Positive. [3] Strongly positive.

NOTE.—In all the above cases the red blood cells were normal in size and shape, with no stippling or polychromatophilia.

PROVISIONAL BIRTH AND INFANT MORTALITY STATISTICS FOR 1939

BIRTHS

According to provisional tabulations issued by the Bureau of the Census,[1] there were 2,262,726 births registered in the United States in 1939, a decrease of 24,236 as compared with 1938. This decrease in the number of births gives a corresponding decrease in the birth rate from 17.6 to 17.4. The provisional rate for 1939, however, is approximately 5 percent higher than the lowest birth rate, 16.5 in 1933, recorded for the birth registration area since that area was established in 1915, which is probably also the lowest rate for the country as a whole. The Bureau of the Census points out that the slight increases in the last 2 years cannot be taken as an assurance that the general decline in the birth rate in the United States during the past 20 years has been checked.

On the basis of provisional figures, 16 States and the District of Columbia show an increase in 1939 over 1938, 27 States show a decrease, and in 5 States there was no change. The largest increases are shown for the District of Columbia, Delaware, Florida, and South Carolina, while the largest decreases are recorded for Mississippi, Arkansas, and Illinois.

The highest birth rates for 1939 are those for New Mexico (33.7), Arizona (26.0), Mississippi (25.6), and Utah (25.1), while the lowest rates are shown for New Jersey (13.0), Connecticut (13.5), Massachusetts (13.6), and New York (14.4).[2]

The final birth rates, by States, will be published in the Public Health Reports as soon as they are made available by the Bureau of the Census.

The following table gives the birth rates for the expanding birth registration area in continental United States from 1915 to 1939. Since, and including, 1933, the birth registration area includes all of the States.

Birth rate (number of live births per 1,000 population) for the birth registration area, 1915–39

Year	Rate	Year	Rate	Year	Rate	Year	Rate	Year	Rate
1939 [1]	17.4	1934	17.1	1929	18.9	1924	22.4	1919	22.3
1938	17.6	1933	16.5	1928	19.8	1923	22.2	1918	24.6
1937	17.0	1932	17.4	1927	20.6	1922	22.3	1917	24.7
1936	16.7	1931	18.0	1926	20.7	1921	24.2	1916	25.0
1935	16.9	1930	18.9	1925	21.5	1920	23.7	1915	25.1

[1] Provisional.

[1] Vital Statistics—Special Reports, vol. 9, No. 46 (May 22, 1940), pp. 537–542.

[2] The rates for the States are based on the estimated State populations for 1937; the rates for the United States are based on the estimated population for 1938. The birth figures for Massachusetts are partial estimates, as the 1939 data are incomplete.

INFANT MORTALITY

Provisional figures furnished by the Bureau of the Census [3] indicate that the infant mortality rate for the United States in 1939 is the lowest in the history of the birth registration area and no doubt the lowest for the country as a whole. According to provisional tabulations, there were 108,532 infant deaths (under 1 year of age) in the United States in 1939, as compared with 116,702 in 1938. The corresponding infant mortality rates (number of deaths under 1 year of age per 1,000 live births) are 48.0 and 51.0—a reduction of 6 percent in 1939 as compared with the preceding year.

In terms of the infant mortality rate, 42 States and the District of Columbia recorded decreases in 1939 as compared with 1938, while 6 States showed increases. A comparison of the rates for cities with a population of 100,000 or more shows decreases in 65 cities, increases in 27, and the same rate for the 2 years in 1 city.

Fourteen States have rates below 40.0, the lowest rate, 35.4, being reported for Minnesota. The highest rates are those for New Mexico, 109.3, and Arizona, 95.5.

As the figures for 1939 are preliminary, and in some instances probably incomplete, the numbers of infant deaths and the infant mortality rates by States will not be published in the Public Health Reports until final tabulations are available. It is unlikely that delayed certificates and the final count will change the provisional rate for the country as a whole.

The general trend of the infant mortality rate in the United States since 1915 is shown in the following table:

Infant mortality rate (number per 1,000 live births) for the birth registration area, by years, 1915–39

Year	Rate	Year	Rate	Year	Rate	Year	Rate	Year	Rate
1939 [1]	48.0	1934	60.1	1929	67.6	1924	70.8	1919	86.6
1938	51.0	1933	58.1	1928	68.7	1923	77.1	1918	100.9
1937	54.4	1932	57.6	1927	64.6	1922	76.2	1917	93.8
1936	57.1	1931	61.6	1926	73.3	1921	75.6	1916	101.0
1935	55.7	1930	64.6	1925	71.7	1920	85.8	1915	99.9

[1] Provisional.

COURT DECISION ON PUBLIC HEALTH

Statute requiring health certificate of person working in food or drink establishment construed.—(Texas Court of Criminal Appeals; *Sekaly* v. *State*, 136 S.W.2d 854; decided February 14, 1940.) A Texas law provided in part as follows:

[3] Vital Statistics—Special Reports, vol. 9, No. 47 (May 25, 1940), pp. 543-547.

No person * * * operating * * * any * * * place * * * where food or drink * * * is * * * served, sold, or * * * handled * * * shall work, employ, or keep in their employ, in * * * said place * * * any person infected with any transmissible condition of any infectious or contagious disease, or work, or employ any person to work in * * * said place, * * * who, at the time of his * * * employment, failed to deliver to the employer * * * a certificate signed by a * * * physician * * * attesting the fact that the bearer had been * * * examined by such physician within a week prior to the time of such employment, and that such examination disclosed the fact that such person to be employed was free from any transmissible condition of any infectious or contagious disease.

The act also required that a new certificate be secured every 6 months.

In a prosecution under this law it was charged that, on or about a certain date, an operator of an establishment, where food and drink were served, unlawfully worked in said establishment without having in his possession a physician's certificate showing that he had been examined by such physician within 1 week prior to the time that he so worked in said establishment and that such examination disclosed that he was free from any infectious and contagious disease. There was a conviction and an appeal followed.

In considering the case on appeal the court of criminal appeals said that it occurred to it that the statute required that a certificate be secured not more than 1 week prior to the time of employment and be renewed every 6 months, but that it was not necessary that a certificate show that it was secured 1 week prior to any particular time that the operator or employee might have worked. "The law," said the court, "does not seem to require the operator or employee of the establishment to have a certificate attesting the fact that he or she has been examined by a physician within a week prior to any particular time that he might have worked in such establishment." The court pointed out that it was merely alleged that the appellant worked in the establishment on a particular date without having secured a certificate for a week prior to this date.

Regarding the allegation that the appellant did not have a certificate in his possession, the court said that the statute did not require that an employee or operator have actual possession of a certificate but that it only required that the certificate be displayed on the business premises.

The judgment of the lower court was reversed and the prosecution ordered dismissed.

DEATHS DURING WEEK ENDED JUNE 8, 1940

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

	Week ended June 8, 1940	Corresponding week, 1939
Data from 88 large cities of the United States:		
Total deaths	8,579	7,950
Average for 3 prior years	7,773	
Total deaths, first 23 weeks of year	208,252	206,928
Deaths under 1 year of age	521	490
Average for 3 prior years	486	
Deaths under 1 year of age, first 23 weeks of year	11,691	12,124
Data from industrial insurance companies:		
Policies in force	65,353,394	67,253,770
Number of death claims	12,771	12,798
Death claims per 1,000 policies in force, annual rate	10.2	9.9
Death claims per 1,000 policies, first 23 weeks of year, annual rate	10.4	11.4

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

REPORTS FROM STATES FOR WEEK ENDED JUNE 15, 1940

Summary

The incidence of smallpox, typhoid fever, and whooping cough increased slightly during the week ended June 15, as compared with the preceding week, while that of the other 6 important communicable diseases included in the weekly telegraphic reports decreased. The numbers of cases for each of these 9 diseases, however, with the exception of measles, were below the 5-year (1935–39) median expectancy for the current week, and, with the exception of measles, scarlet fever, and whooping cough, were below the figures for the corresponding week last year.

The number of cases of smallpox increased from 62 to 78, of which 71 cases, or 91 percent, were reported from the two North Central groups of States (18 in North Dakota, 14 each in Illinois and Wisconsin, and 10 each in Indiana and Iowa).

The 154 cases of typhoid fever reported currently (preceding week, 130; 5-year median, 292) were more widely scattered, with the 4 West South Central States reporting 46 cases, Georgia 13, Missouri 12, and the 3 Pacific States 14 (California 10).

The number of reported cases of poliomyelitis dropped from 58 to 42, and of meningococcus meningitis from 29 to 24. Of the 42 cases of poliomyelitis, 28 cases, or 67 percent, occurred in two Pacific States, Washington (17, of which 14 were in Pierce County, 2 in Tacoma) and California (11, of which 6 were in Los Angeles).

Of 12 cases of Rocky Mountain spotted fever, 8 cases were reported in the western States and 4 cases in the East. Ten cases of Colorado tick fever were reported in Colorado; 2 cases of undulant fever each in Rhode Island and Arizona, and 3 cases in Maryland; and 2 cases of tularaemia in Utah. Of 20 cases of endemic typhus fever, 10 cases were reported in Texas.

For the current week the Bureau of the Census reports 7,956 deaths in 88 major cities of the United States, as compared with 8,579 for the preceding week and with a 3-year (1937–39) average of 7,623 for the corresponding week.

Telegraphic morbidity reports from State health officers for the week ended June 15, 1940, and comparison with corresponding week of 1939 and 5-year median

In these tables a zero indicates a definite report, while leaders imply that, although none were reported, cases may have occurred.

Division and State	Diphtheria			Influenza			Measles			Meningitis, meningococcus		
	Week ended		Median, 1935–39	Week ended		Median, 1935–39	Week ended		Median, 1935–39	Week ended		Median, 1935–39
	June 15, 1940	June 17, 1939		June 15, 1940	June 17, 1939		June 15, 1940	June 17, 1939		June 15, 1940	June 17, 1939	
NEW ENG.												
Maine	1	3	1	2	1	-------	305	63	81	0	0	0
New Hampshire	0	1	0	-------	-------	-------	6	33	33	0	0	0
Vermont	0	0	0	-------	-------	-------	8	201	47	0	0	0
Massachusetts	4	0	2	-------	-------	-------	1,455	1,015	460	0	1	1
Rhode Island	0	0	0	-------	-------	-------	201	26	26	0	0	1
Connecticut	1	0	2	1	-------	-------	31	468	213	1	1	1
MID. ATL.												
New York	16	14	34	[1]1	[1]6	[1]3	994	1,511	2,546	3	4	13
New Jersey [2]	1	15	10	3	4	4	1,267	32	547	0	1	3
Pennsylvania	15	14	18	-------	-------	-------	496	124	1,408	3	7	7
E. NO. CEN.												
Ohio	9	10	13	12	28	28	53	96	898	1	1	1
Indiana	4	4	4	5	1	3	16	6	97	2	0	1
Illinois	19	19	37	5	9	19	223	33	427	2	0	3
Michigan [3]	2	5	6	1	2	1	793	301	189	0	1	2
Wisconsin	1	0	1	15	31	11	1,111	457	457	0	0	1
W. NO. CEN.												
Minnesota	1	1	1	2	2	2	86	138	190	0	0	0
Iowa	5	3	2	-------	1	1	87	100	100	0	0	1
Missouri	2	5	7	1	-------	22	21	3	50	0	0	0
North Dakota	3	1	1	-------	10	-------	3	5	5	0	0	0
South Dakota	0	0	0	1	3	-------	2	57	3	0	0	0
Nebraska	0	1	1	-------	4	-------	7	110	89	0	1	1
Kansas	1	3	3	1	2	1	242	59	59	1	0	0
SO. ATL.												
Delaware [3]	0	0	1	-------	-------	-------	3	12	9	0	1	0
Maryland [2 3]	0	2	3	-------	2	1	9	120	120	0	0	1
Dist. of Col	0	0	3	-------	-------	-------	6	144	93	0	0	3
Virginia [2]	5	10	7	34	32	-------	156	446	183	0	1	4
West Virginia [3]	2	2	4	7	9	11	10	14	95	0	1	1
North Carolina [4]	6	9	7	-------	-------	1	112	288	196	0	1	4
South Carolina [4]	3	3	3	95	188	56	16	14	30	0	2	2
Georgia [4]	1	7	3	9	17	-------	43	60	0	1	0	0
Florida [4]	2	6	6	-------	4	1	16	47	11	0	0	0
E. SO. CEN.												
Kentucky	2	2	4	2	-------	5	95	20	65	1	0	1
Tennessee	2	3	5	21	22	8	85	101	49	0	3	3
Alabama [4]	1	1	5	10	22	8	31	81	68	1	1	1
Mississippi [3]	0	3	5	-------	-------	-------	-------	-------	-------	0	0	0
W. SO. CEN.												
Arkansas	3	1	3	28	13	10	28	31	31	0	1	1
Louisiana [4]	0	5	10	9	9	15	1	14	14	1	0	3
Oklahoma	5	2	2	13	17	17	22	78	48	1	0	1
Texas [4]	14	30	26	100	91	91	669	309	125	2	1	1
MOUNTAIN												
Montana	2	0	0	-------	2	2	50	84	56	0	0	0
Idaho	0	0	0	-------	-------	-------	31	22	19	0	0	0
Wyoming [3]	1	0	0	-------	-------	-------	34	34	5	0	0	0
Colorado [5]	10	6	4	-------	20	-------	26	56	56	0	0	0
New Mexico	0	2	2	-------	1	1	67	17	46	0	0	0
Arizona	2	1	2	50	33	14	71	8	8	1	0	0
Utah [2 3]	0	0	0	-------	-------	-------	222	92	77	0	0	0
PACIFIC												
Washington [3]	2	2	0	-------	-------	-------	187	769	199	0	0	1
Oregon [3]	5	0	1	5	19	7	111	56	56	0	0	0
California [2 4]	20	21	30	23	36	110	260	1,455	1,097	3	2	3
Total	173	217	330	456	641	540	9,768	9,210	9,239	24	31	64
24 weeks	7,427	9,773	11,648	165,861	148,631	138,052	193,411	322,064	322,064	963	1,108	3,403

See footnotes at end of table.

Telegraphic morbidity reports from State health officers for the week ended June 15, 1940, and comparison with corresponding week of 1939 and 5-year median—Continued

Division and State	Poliomyelitis			Scarlet fever			Smallpox			Typhoid and paratyphoid fever		
	Week ended		Median, 1935-39	Week ended		Median, 1935-39	Week ended		Median, 1935-39	Week ended		Median, 1935-39
	June 15, 1940	June 17, 1939		June 15, 1940	June 17, 1939		June 15, 1940	June 17, 1939		June 15, 1940	June 17, 1939	
NEW ENG.												
Maine	0	0	0	0	11	7	0	0	0	1	0	1
New Hampshire	0	0	0	1	1	3	0	0	0	0	1	0
Vermont	1	0	0	1	7	5	0	0	0	0	0	0
Massachusetts	0	1	1	95	109	188	0	0	0	1	6	1
Rhode Island	0	0	0	4	3	11	0	0	0	1	2	0
Connecticut	0	0	0	48	26	77	0	0	0	1	3	1
MID. ATL.												
New York	1	2	2	389	267	447	0	8	0	7	10	9
New Jersey [2]	1	0	1	147	101	101	0	0	0	2	0	4
Pennsylvania	1	1	0	195	150	261	0	1	0	9	6	12
E. NO. CEN.												
Ohio	0	0	1	142	172	172	0	14	4	4	8	8
Indiana	1	1	0	37	51	53	10	4	4	3	8	3
Illinois	1	1	1	444	173	319	14	9	12	3	13	6
Michigan [3]	0	1	0	211	257	257	0	3	1	1	3	4
Wisconsin	0	0	0	79	50	185	14	0	3	0	1	1
W. NO. CEN.												
Minnesota	0	0	0	44	29	89	0	2	7	0	0	1
Iowa	0	0	0	28	40	54	10	12	19	1	3	3
Missouri	0	0	1	46	38	38	2	8	10	12	4	7
North Dakota	0	0	0	2	0	11	18	3	8	0	0	0
South Dakota	1	0	0	3	7	8	1	7	7	1	0	0
Nebraska	0	0	0	2	6	9	1	6	6	0	0	0
Kansas	2	0	0	21	25	45	1	7	8	3	2	2
SO. ATL.												
Delaware [2]	0	0	0	2	7	3	0	0	0	0	0	0
Maryland [2 3]	0	0	0	20	7	43	0	0	0	2	2	4
Dist. of Col	0	0	0	12	4	7	0	0	0	1	3	0
Virginia [2]	0	0	0	25	16	18	0	0	0	3	13	13
West Virginia [3]	0	0	0	23	22	22	1	0	0	3	9	6
North Carolina [4]	0	2	2	11	18	18	0	4	1	0	11	11
South Carolina [4]	0	28	0	0	0	1	1	0	0	1	21	12
Georgia [4]	0	5	0	6	8	5	0	0	0	13	12	23
Florida [4]	1	1	0	2	5	5	0	0	0	4	2	4
E. SO. CEN.												
Kentucky	1	0	0	21	12	13	0	2	0	2	12	9
Tennessee	1	0	0	26	21	10	0	14	0	2	10	13
Alabama [4]	1	0	2	6	11	5	1	0	0	3	5	10
Mississippi [3]	0	0	1	4	2	6	0	0	0	2	1	7
W. SO. CEN.												
Arkansas	0	0	0	4	3	4	0	2	0	7	7	8
Louisiana [4]	0	0	1	10	9	6	0	0	0	11	11	13
Oklahoma	1	0	0	16	5	11	0	12	1	10	15	11
Texas [4]	0	2	2	18	23	28	2	0	7	18	16	19
MOUNTAIN												
Montana	0	0	0	5	9	8	0	0	7	0	0	1
Idaho	0	0	0	2	1	7	0	0	0	2	0	1
Wyoming [3]	0	0	0	5	0	6	0	4	3	0	0	0
Colorado [5]	0	1	0	13	20	29	1	2	2	1	4	1
New Mexico	0	1	0	5	15	15	0	0	0	4	9	1
Arizona	0	4	0	3	1	7	0	0	0	0	1	2
Utah [2 3]	0	0	0	5	12	15	0	0	0	1	0	0
PACIFIC												
Washington [2]	17	0	0	31	20	25	0	1	3	3	52	2
Oregon [2]	0	1	0	6	18	16	1	1	5	1	1	2
California [2 4]	11	13	6	105	98	149	0	70	14	10	5	10
Total	42	65	65	2,325	1,890	3,033	78	196	196	154	292	292
24 weeks	644	630	575	109,589	107,943	152,197	1,685	8,072	7,078	2,242	3,233	3,233

See footnotes at end of table.

Telegraphic morbidity reports from State health officers for the week ended June 15, 1940, and comparison with corresponding week of 1939 and 5-year median—Continued

Division and State	Whooping cough	
	Week ended	
	June 15, 1940	June 17, 1939
NEW ENG.		
Maine	18	44
New Hampshire	2	0
Vermont	15	46
Massachusetts	156	121
Rhode Island	10	13
Connecticut	44	68
MID. ATL.		
New York	259	427
New Jersey [3]	88	281
Pennsylvania	257	303
E. NO. CEN.		
Ohio	300	320
Indiana	27	42
Illinois	96	179
Michigan [3]	237	168
Wisconsin	100	143
W. NO. CEN.		
Minnesota	21	28
Iowa	23	28
Missouri	55	18
North Dakota	15	1
South Dakota	0	2
Nebraska	8	34
Kansas	43	14
SO. ATL.		
Delaware	7	17
Maryland [2 3]	152	46
Dist. of Col	5	30
Virginia [3]	56	123
West Virginia [3]	31	16
North Carolina [4]	122	203
South Carolina [4]	17	73
SO. ATL.—continued		
Georgia [4]	54	18
Florida [4]	5	28
E. SO. CEN.		
Kentucky	107	12
Tennessee	59	50
Alabama [4]	19	62
Mississippi [3]	----------	----------
W. SO. CEN.		
Arkansas	17	33
Louisiana [4]	76	3
Oklahoma	27	4
Texas [4]	261	146
MOUNTAIN		
Montana	0	6
Idaho	11	5
Wyoming [3]	3	0
Colorado [5]	5	49
New Mexico	45	17
Arizona	48	34
Utah [2 3]	179	55
PACIFIC		
Washington [3]	56	28
Oregon [3]	35	16
California [3 4]	471	181
Total	3,642	3,535
24 weeks	76,890	94,166

[1] New York City only.
[2] Rocky Mountain spotted fever, week ended June 15, 1940, 12 cases, as follows: New Jersey, 1; Delaware, 1; Maryland, 1; Virginia, 1; Wyoming, 3; Utah, 2; Washington, 1; Oregon, 1; California, 1.
[3] Period ended earlier than Saturday.
[4] Typhus fever, week ended June 15, 1940, 20 cases, as follows: North Carolina, 1; South Carolina, 1; Georgia, 3; Florida, 2; Alabama, 1; Louisiana, 1; Texas, 10; California, 1.
[5] Colorado tick fever, week ended June 15, 1940, Colorado, 10 cases.

PLAGUE INFECTION IN RODENTS AND FLEAS IN OREGON AND WASHINGTON

IN A MARMOT IN LAKE COUNTY, OREG.

Under date of May 31, Surgeon L. B. Byington reported plague infection proved in a marmot (*Marmota flaviventris*) shot on May 17, 1940, 7 miles north of Adel, Lake County, Oreg.

IN FLEAS FROM A COTTONTAIL RABBIT IN SPOKANE COUNTY, WASH.

Under date of May 31, Surgeon L. B. Byington reported plague infection proved in a pool of 36 fleas from a cottontail rabbit shot on May 20, 1940, 9 miles west of Spangle, Spokane County, Wash.

WEEKLY REPORTS FROM CITIES

City reports for week ended June 1, 1940

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	131	55	26	5,062	464	1,663	18	385	31	1,209	-----
Current week [1]	56	31	21	3,389	291	1,594	1	316	19	773	-----
Maine:											
Portland	0	----	0	83	2	0	0	0	0	0	25
New Hampshire:											
Concord	0	----	0	0	1	0	0	0	0	0	8
Manchester	0	----	0	0	5	0	0	0	0	0	15
Vermont:											
Barre	0	----	0	0	0	1	0	2	0	0	3
Burlington	0	----	0	0	0	0	0	0	0	0	9
Rutland	0	----	0	0	0	0	0	0	0	0	7
Massachusetts:											
Boston	0	------	0	209	19	30	0	11	1	47	218
Fall River	0	------	0	151	0	1	0	1	0	3	32
Springfield	0	------	0	3	1	6	0	1	0	5	42
Worcester	0	------	--------	236	2	11	0	0	1	4	37
Rhode Island:											
Pawtucket	0	----	0	0	0	0	0	0	0	0	16
Providence	0	----	0	113	2	4	0	1	0	0	55
Connecticut:											
Bridgeport	0	----	0	2	3	5	0	2	0	0	28
Hartford	0	----	0	1	4	6	0	0	0	6	44
New Haven	0	----	2	0	0	7	0	0	0	6	43
New York:											
Buffalo	2	----	0	3	7	20	0	2	0	5	135
New York	12	6	2	395	60	427	0	80	4	68	1,443
Rochester	0	----	1	4	1	9	0	1	0	5	73
Syracuse	0	----	0	0	1	5	0	0	0	3	47
New Jersey:											
Camden	0	----	0	3	1	12	0	0	0	0	32
Newark	0	1	0	460	1	29	0	1	0	10	98
Trenton	0	----	0	0	2	7	0	3	0	2	28
Pennsylvania:											
Philadelphia	0	----	2	161	14	88	0	17	0	14	376
Pittsburgh	1	----	1	2	4	35	0	7	0	13	146
Reading	0	----	0	2	0	0	0	1	0	7	25
Scranton	0	----	-----	0	-----	0	0	-----	0	0	-----
Ohio:											
Cincinnati	1	2	1	0	0	8	0	8	0	17	112
Cleveland	2	10	1	7	11	50	0	9	0	43	174
Columbus	1	----	0	0	1	8	0	1	0	2	70
Toledo	0	----	0	2	1	64	0	3	0	3	51
Indiana:											
Anderson	0	----	0	1	1	2	0	0	0	0	10
Fort Wayne	0	----	1	6	1	2	0	0	0	1	22
Indianapolis	0	----	0	4	5	24	0	6	0	6	99
Muncie	0	----	0	0	0	0	0	0	0	1	9
South Bend	0	----	0	0	0	0	0	0	0	0	12
Terre Haute	0	----	1	0	2	1	0	0	0	2	27
Illinois:											
Alton	0	----	0	0	1	1	0	1	0	0	11
Chicago	11	3	3	104	21	497	0	36	1	26	630
Elgin	0	----	0	0	2	1	0	1	0	0	11
Moline	0	----	0	3	0	0	0	0	0	0	10
Springfield	0	----	0	1	0	1	0	0	0	0	17
Michigan:											
Detroit	1	----	0	261	8	88	0	17	1	70	221
Flint	0	----	0	11	2	8	0	0	0	2	10
Grand Rapids	0	----	0	3	3	21	0	0	0	17	50
Wisconsin:											
Kenosha	0	----	0	69	0	0	0	0	0	0	6
Madison	0	----	0	66	1	1	0	0	0	5	4
Milwaukee	0	----	0	0	2	24	0	7	0	6	88
Racine	0	----	0	3	0	3	0	0	0	1	11
Superior	0	----	0	84	0	2	0	0	0	0	7
Minnesota:											
Duluth	0	------	0	5	0	0	0	0	0	0	15
Minneapolis	0	------	1	1	3	14	0	1	0	12	98
St. Paul	0	1	1	0	2	7	0	3	0	8	61

[1] Figures for Boise estimated; report not received.

City reports for week ended June 1, 1940—Continued

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								
Iowa:											
Cedar Rapids	0	------	--------	38	-------	0	0	-------	0	1	--------
Davenport	0	------	--------	3	-------	6	0	-------	0	0	--------
Des Moines	0	------	0	19	0	3	1	0	0	0	38
Sioux City	0	------	--------	1	-------	1	0	-------	0	0	--------
Waterloo	1	------	--------	5	-------	1	0	-------	0	1	--------
Missouri:											
Kansas City	0	------	0	7	4	5	0	0	1	0	104
St. Joseph	0	------	0	0	0	2	0	1	0	0	24
St. Louis	0	------	0	2	9	12	0	2	0	12	184
North Dakota:											
Fargo	0	------	0	0	0	0	0	0	0	0	6
Grand Forks	0	------	--------	0	-------	0	0	-------	0	2	--------
Minot	1	------	0	1	0	0	0	0	0	0	8
South Dakota:											
Aberdeen	0	------	--------	0	-------	0	0	-------	0	0	--------
Sioux Falls	1	------	0	0	0	1	0	0	0	0	9
Nebraska:											
Lincoln	0	------	--------	1	-------	2	0	-------	0	1	--------
Omaha	6	------	0	9	1	5	0	2	0	1	52
Kansas:											
Lawrence	0	------	0	0	0	0	0	0	0	1	4
Topeka	0	------	0	30	0	3	0	0	0	0	14
Wichita	0	------	0	3	4	1	0	0	0	0	32
Delaware:											
Wilmington	0	------	0	0	1	2	0	0	0	4	25
Maryland:											
Baltimore	1	------	0	2	5	10	0	13	0	66	200
Cumberland	0	------	0	0	0	1	0	0	0	0	8
Frederick	0	------	0	0	1	0	0	0	0	0	3
Dist. of Col.											
Washington	3	------	0	2	7	20	0	10	1	8	126
Virginia:											
Lynchburg	1	------	0	0	1	0	0	0	0	11	17
Norfolk	0	------	0	104	1	1	0	1	0	3	32
Richmond	0	------	0	3	1	3	0	2	0	0	45
Roanoke	0	------	0	27	0	1	0	0	0	0	15
West Virginia:											
Charleston	0	------	0	1	1	1	0	0	0	1	13
Huntington	0	------	--------	0	-------	1	0	-------	0	0	--------
Wheeling	0	------	0	0	2	0	0	2	2	5	13
North Carolina:											
Gastonia	0	------	--------	0	-------	1	0	-------	0	0	--------
Raleigh	0	------	0	1	0	0	0	1	0	1	9
Wilmington	0	------	0	1	1	0	0	0	0	0	9
Winston-Salem	1	------	0	0	0	1	0	2	0	4	21
South Carolina:											
Charleston	0	------	0	0	1	0	0	1	0	0	23
Florence	0	------	0	0	0	0	0	2	0	0	12
Greenville	0	------	0	1	3	0	0	1	0	0	21
Georgia:											
Atlanta	0	4	0	4	3	1	0	3	1	5	70
Brunswick	0	------	0	0	0	0	0	0	0	0	1
Savannah	0	------	0	0	1	2	0	2	0	0	25
Florida:											
Miami	0	1	0	2	2	0	0	1	0	0	35
Tampa	0	------	0	39	2	0	0	0	0	1	21
Kentucky:											
Ashland	0	------	0	0	0	0	0	0	0	0	5
Covington	0	------	0	15	0	1	0	0	0	5	13
Lexington	0	------	0	18	0	1	0	0	0	4	15
Louisville	0	------	0	17	4	20	0	3	0	47	57
Tennessee:											
Knoxville	0	2	2	1	2	2	0	0	1	0	30
Memphis	0	------	0	41	5	8	1	3	0	10	67
Nashville	0	------	0	7	3	2	0	2	1	4	41
Alabama:											
Birmingham	0	------	0	14	2	2	0	4	0	3	61
Mobile	0	2	0	21	2	0	0	1	0	0	15
Montgomery	0	------	--------	1	-------	0	0	-------	0	3	--------
Arkansas:											
Fort Smith	0	------	--------	0	-------	0	0	-------	1	0	--------
Little Rock	0	------	1	0	5	1	0	1	0	5	--------
Louisiana:											
Lake Charles	0	------	0	0	0	0	0	0	1	0	5
New Orleans	2	1	1	0	6	1	0	11	1	42	147
Shreveport	0	------	0	1	4	0	0	2	1	2	38

City reports for week ended June 1, 1940—Continued

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								
Oklahoma:											
Oklahoma City	0	4	0	0	0	4	0	1	0	0	31
Tulsa	0	------	0	0	2	1	0	1	0	8	22
Texas:											
Dallas	0	------	0	218	4	0	0	2	1	19	56
Fort Worth	0	------	0	16	0	0	0	2	0	26	30
Galveston	0	------	0	0	1	0	0	0	0	0	9
Houston	1	------	0	13	3	1	0	2	0	1	53
San Antonio	1	------	0	3	7	1	0	3	0	7	85
Montana:											
Billings	0	------	0	0	1	0	0	0	0	0	10
Great Falls	0	------	0	21	0	0	0	0	0	0	3
Helena	0	------	0	0	0	1	0	0	0	0	6
Missoula	0	------	0	0	0	1	0	0	1	0	6
Idaho:											
Boise	------	------	------	------	------	------	------	------	------	------	------
Colorado:											
Colorado Springs	0	------	0	1	0	1	0	1	0	0	12
Denver	6	------	0	24	3	2	0	2	0	1	81
Pueblo	0	------	0	5	0	1	0	0	0	1	12
New Mexico:											
Albuquerque	1	------	0	0	1	0	0	2	0	1	17
Utah:											
Salt Lake City	0	------	1	340	1	1	0	0	0	69	34
Washington:											
Seattle	0	------	0	130	4	5	0	3	0	10	86
Spokane	0	------	0	1	0	2	0	0	0	2	20
Tacoma	0	------	0	0	1	5	0	0	0	1	27
Oregon:											
Portland	4	------	0	39	1	0	0	1	0	11	79
Salem	0	------	------	3	------	0	0	------	0	0	------
California:											
Los Angeles	2	1	1	15	2	24	0	8	1	36	238
Sacramento	1	------	0	9	1	1	0	2	0	16	24
San Francisco	0	------	0	7	5	4	0	11	0	14	170

State and city	Meningitis, meningococcus		Poliomyelitis cases	State and city	Meningitis, meningococcus		Poliomyelitis cases
	Cases	Deaths			Cases	Deaths	
New York:				Alabama:			
New York	0	1	0	Birmingham	1	0	0
Rochester	1	0	0	Louisiana:			
Michigan:				New Orleans	1	0	1
Detroit	1	0	0	Shreveport	0	1	0
Flint	1	0	0	Washington:			
District of Columbia:				Tacoma	0	0	14
Washington	1	0	0	California:			
West Virginia:				Los Angeles	0	0	5
Huntington	1	0	0				
Wheeling	0	0	1				
South Carolina:							
Charleston	0	0	1				
Florence	0	1	0				

NOTE.—Information has been received that 1 case reported as meningococcus meningitis in Wilmington, N. C., for the week ended May 11, and published in Public Health Reports of May 31, p. 997, was influenzal meningitis.

Encephalitis, epidemic or lethargic.—Cases: New York, 1; Wichita, 1.

Pellagra.—Cases: Charleston, S. C., 1; Miami, 2; Louisville, 1.

Typhus fever.—Cases: New York, 1; Savannah, 1.

FOREIGN REPORTS

CANADA

Provinces—Communicable diseases—Week ended May 18, 1940.—During the week ended May 18, 1940, 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	New Brunswick	Quebec	Ontario	Manitoba	Saskatchewan	Alberta	British Columbia	Total
Cerebrospinal meningitis				1	4		1			6
Chickenpox		19	14	163	332	32	2	1	64	627
Diphtheria		3	1	23		12	4	1		44
Dysentery				3						3
Influenza		4			21		3		2	30
Lethargic encephalitis					1					1
Measles		11	3	293	299	397	289	13	128	1,433
Mumps		6		9	344	11	15		16	401
Pneumonia		14			29	3	1	3	1	51
Poliomyelitis				2						2
Scarlet fever		5	12	97	91	22	2	20	4	253
Tuberculosis	2	10	8	54	59	1	2			136
Typhoid and paratyphoid fever		2		20	1		31	2		56
Whooping cough		46	13	176	138	39	35	11	10	468

REPORTS OF CHOLERA, PLAGUE, SMALLPOX, TYPHUS FEVER, AND YELLOW FEVER RECEIVED DURING THE CURRENT WEEK

NOTE.—A cumulative table giving current information regarding the world prevalence of quarantinable diseases appeared in the PUBLIC HEALTH REPORTS of May 31, 1940, pages 1000-1002. A similar table will appear in future issues of the PUBLIC HEALTH REPORTS for the last Friday of each month.

Cholera

India—Moulmein.—During the week ended June 1, 1940, 10 cases of cholera were reported in Moulmein, India.

Plague

Hawaii Territory—Island of Hawaii—Hamakua District—Paauilo.—A rat found on May 9, 1940, in Paauilo, Hamakua District, Island of Hawaii, T. H., has been proved positive for plague.

United States.—A report of plague infection in Lake County, Oregon, and in Spokane County, Washington, appears on page 1138 of this issue of PUBLIC HEALTH REPORTS.

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