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A SELF-HELP SOLUTION OF STATE PERSONNEL PROBLEMS [1]

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More work—fewer workers. These four words describe the plight of almost every public health agency today. From all sides the cry is raised, "Our organization is being destroyed! We are losing our personnel—they are either going into the armed forces or leaving to take better jobs elsewhere!"

These crys are prompted by varying degrees of urgency. Some of them are hardly justified by actual circumstances and are really prayers that the lightning will not strike. Others are the protest of health officers who are just beginning to feel the pinch of the wartime manpower shortage. Still others sound ominously like the death rattle of agencies about to give up the ghost.

One gets the impression that the catastrophe broke suddenly, without previous warning. Yet this is far from the truth. From the outbreak of the war in Europe it was apparent that the United States would not escape some form of participation. Later events pointed to full participation and complete transformation from a peacetime to a war economy. The present crisis and its effects were foretold again and again by responsible Government spokesmen and analysts of public affairs. Today on every hand we see evidence of how little these warnings were heeded.

The failure of some health departments to adjust themselves to coming events may perhaps be attributed to a disproportionate sense of their own worthiness and essentiality. They believed that those responsible for manpower policies could not disregard their needs. Somehow the war would be fought without disturbing their personnel structure or program content. When the necessity of recruiting and training replacement personnel was pointed out, objections were raised. It was claimed that the requirements of public health work were such that no one could be inducted into the mysteries of the profession without extended training. Merit systems of personnel administration, many of which had not evolved beyond the discussion

[1] From the States Relations Division.

and preliminary paper-work stage, were held up as insurmountable obstacles to emergency adjustments. Many health officers stated the belief that the Federal Government should, could, and would help them through the crisis. They said, "The Government is taking our workers and putting them in military service—why shouldn't it replace them?" or, "The Government is responsible for our headaches. It is putting up big camps and war plants in our midst, and turning the community inside out. Why shouldn't the Government solve the problems it has created?" Indeed, the early steps taken by the United States Public Health Service to relieve the situation in certain critical areas were misinterpreted by some as an indication that the Federal Government intended to shoulder most of the burden.

When the President declared a national emergency the Public Health Service realized that serious local problems would be created for which the Federal Government would have to make special provisions. Accordingly, the Service obtained an appropriation to help meet certain emergency health and sanitation needs in areas where local resources were inadequate. An emergency corps of professional personnel, including physicians, engineers, nurses, and laboratory workers, was recruited. A special effort was made not to select experienced public health workers who were needed in the armed services and in State and local health departments where they were then employed. Consequently, the educational background of most of the recruits was limited to basic training in medicine, engineering, nursing, or allied fields. In order to provide these workers with a modicum of instruction in public health policy and practices, a series of 4-week orientation training courses was instituted. Approximately 700 recruits were trained in this manner. At the conclusion of their orientation training most of them were assigned to State health departments, from whence they were reassigned to duty in some 200 of the more critical war communities.

The purpose of this corps of Federal personnel was to level off some of the more outstanding peaks of need which might obstruct the progress of the war program. The purpose was not to ease the recruitment problems of the States or to assume the responsibilities of State and local health agencies. Therefore, it was hoped that the State health departments would organize recruiting and training programs of their own.

The need for greater self-reliance on the part of the States was emphatically brought out by the Administrator of the Federal Security Agency in his address to the Fortieth Annual Conference of the United States Public Health Service with the State and Territorial Health Officers in March 1942. No doubt the Administrator hoped that his remarks would stimulate some concerted plan of action, or at least an effort by some States to meet the situation individually.

The Surgeon General of the Public Health Service specifically called the attention of the Conference to the need for recruiting additional workers and to the necessity of adjusting State merit systems so that such personnel as were available could be employed for the duration of the emergency.

At long last, one State—North Carolina—has developed an organized plan. On January 4, 1943, the State Board of Health instituted its own orientation training course for public health workers recruited for emergency service within the State.

North Carolina's initiative is the more remarkable in view of the fact that it has not lost so high a percentage of its health personnel as many other States. Only 4 local health officers and 1 medical officer from the State health department staff have been lost to the armed services. One local health officer has resigned to enter private practice. Depletion of sanitarians has been more serious; out of a normal complement of 87 trained sanitarians in local health units, 28 have been lost, and of the normal State sanitation staff of 20, 7 have gone. Of the 332 public health nurses on duty in the State at the beginning of 1942, 71 have left for military service or other employment.

In organizing its recruiting and training program, North Carolina's first step was to make the necessary adjustments in its merit system. The following emergency classifications were established, subject to acceptance by the State merit system council and review by the Federal agencies contributing to budgets through which the personnel will be paid after their assignment to actual duty:

Local Health Officer IV (War Emergency).—Qualifications: (1) Graduation from a class A medical school and 1 year of internship in an approved hospital; (2) possession of a license to practice medicine in North Carolina or eligibility for such a license.

Junior Public Health Nurse (War Emergency).—Qualifications: (1) Education equivalent to 4 years of high school; (2) graduation from a school of nursing; (3) registration with the North Carolina State Board of Nurse Examiners.

Junior Sanitarian (War Emergency).—Qualifications: (1) Graduation from an accredited high school; (2) 5 years within the past 10 of successful full-time paid employment, preferably in a field involving contact with the public.

Junior Follow-up Worker in Venereal Disease Control (War Emergency).—Qualifications: (1) Graduation from an accredited high school; (2) 5 years within the past 10 of successful full-time paid employment, preferably in a field involving contact with the public.

Provision was made for abolition of the new classifications at the expiration of the present emergency or at such time as personnel can be found to meet the qualificational requirements for the regular classifications. This provision was considered necessary in order to protect the merit system against future encroachment.

The State health department then instructed its district nursing consultants and district sanitarians, as well as all local health officers, to seek likely candidates. The State nursing association provided a list

of nurses who might be available. The Vocational Rehabilitation Division of the State Board of Education submitted the names of persons with various types of physical disability which would not interfere seriously with the kind of work to be done. The Women's College at Greensboro suggested several women who had received special instruction in dietetics and home economics, and who were considered suitable candidates for the position of junior sanitarian. WPA service and clerical projects which were undergoing liquidation provided another source of recruits.

A total of 80 applications was received, divided as follows with respect to the various job categories: Physicians, 3; nurses, 29; sanitarians, 34; follow-up workers, 14. Applications were received from virtually every section of the State. Each candidate was interviewed by a district nurse or sanitarian, and applications were submitted to the State health department central office and the State merit system supervisor for review. From the list of applicants the following were selected: Physicians, 3; nurses, 14; sanitarians, 13; follow-up workers, 9.

It is interesting to note the ages and backgrounds of the nursing and subprofessional applicants who were accepted. The nurses ranged in age from 22 to 59, and included seven married, five single, and two widowed women. All but three of them had received their training in schools of nursing approved by the State Board of Nurse Examiners and about half of them had been trained in hospitals with a capacity of more than 100 beds. Actual postgraduate professional experience ranged from 2 months of general staff duty to 22 years of private duty nursing. Three nurses had some public health nursing experience, and one woman's background included 8 years of sanatorium duty, 1 year in the Navy Nurse Corps, 1 year of private duty, and 3 years of public health nursing.

The 21 sanitarian and follow-up worker recruits ranged in age from 23 to 57. Six of them had college training of from 1 to 4 years. Six had physical defects which barred them from military service. All except two were employed at the time they submitted their applications, but a majority were engaged in activities which were suffering wartime curtailment. Among the occupations included were salesmen (candy, electrical appliances, automobiles, refrigerators, insurance), construction contractor, meat market operator, home wax manufacturer, accountant, grocer, textile worker, city fireman, and a college student in mechanical engineering. There were several school teachers and WPA project supervisors. Among the latter was a woman sanitarian recruit with experience in institutional dietetics and school lunch programs. On the whole, the calibre of the recruits was considerably higher than had been expected in view of the known manpower shortage.

On January 4 the recruits reported to the State health department in Raleigh to begin their orientation course. The training program was conducted by the Division of County Health Work, and began with a 2-week period of classroom instruction, including lectures, demonstrations, motion pictures and slide films, group meetings and discussions, quizzes, and a final written examination. Lectures and other instruction were given by members of the State health department staff, officers of the United States Public Health Service, and members of the faculty of the University of North Carolina School of Public Health. The schedule for the 2-week period is given on the following page.

Throughout the 2-week course student participation in the program of instruction was encouraged. Discussion was stimulated through true-and-false quizzes, with the students called upon to defend the answers they gave. A noteworthy feature was the division of the class into two groups, each representing the staff of a local health department and each under the direction of one of the physician students. Each group was presented with a hypothetical community having specified resources and health problems, and then told to go ahead and organize its staff and activities for meeting these problems. The two groups met separately for 30 minutes prior to the daily classes, mapped out their methods of operation, and reported their hypothetical progress. At the end of the 2 weeks each group reported to the entire class and the reports were compared and discussed. This procedure introduced an element of competition and gave the students an opportunity to apply the information they gained from the lectures and classroom demonstrations.

At the end of 2 weeks the recruits were assigned to certain local health departments regularly used by the University of North Carolina School of Public Health as field training centers. Field training for the nurses consisted of demonstrations of bag technique, home visits, observation of various types of clinics and school health service programs, and familiarization with nursing records and reports. The sanitarians observed and assisted in typhus control programs, food and milk control, privy construction, water supply protection, and field inspection and correction of insanitary conditions reported to the local health units. The follow-up workers learned the technique of interviewing venereal disease patients and conducting epidemiological investigations. They also received field instruction in methods of venereal disease education and preparation and use of reports and records. All field training was directly supervised by members of the local health department staffs who reported the progress of each student to the State staff members in charge of training the respective groups.

As this article goes to press the field training course is nearing

North Carolina State Board of Health—Orientation Course for Emergency Personnel, January 4–16, 1943

	9:30–10:15	10:30–11:15	11:30–12:30	1:30–2:15	2:30–3:15	3:30–4:30
Monday, January 4	Registration	Introductory lecture by State health officer.	Lecture — Organization and functions of State board of health.	Film—"The Work of the U. S. Public Health Service."		Instructions regarding courses.
Tuesday, January 5	Lecture — Federal, State, and local relationships.	Lecture—Field relationships of State health department employees.	Lecture—Public health nursing on Federal, State, and local levels.	Lecture — Wartime sanitation activities.	Film—"Stream Pollution."	Group conferences (Nurses, sanitarians, follow-up workers).
Wednesday, January 6	Lecture — County health work.	Lecture—Work of the Division of Epidemiology.	Lecture—Work of the State laboratory.	Lecture—Work of the Division of Sanitary Engineering.	Lecture — Preventive medicine.	Group conferences.
Thursday, January 7	Lecture — Public health nursing in the State.	Lecture—National organization of venereal disease control.	Lecture—State organization of venereal disease control.	Lecture—The venereal diseases.	Lecture — Venereal disease epidemiology and case-finding.	Venereal disease film—"Know For Sure."
Friday, January 8	Lecture—Cafe and hotel sanitation.	Lecture—Food sanitation		Film—"Eating Out"	Lecture—Rural sewage disposal.	Group conferences.
Saturday, January 9	Lecture—Oral hygiene services.	Review quiz				
Monday, January 11	Lecture—Local health departments		Lecture—Special wartime health problems.	Lecture — Wartime nursing.	Lecture — Wartime sanitation.	Lecture — Wartime venereal disease control.
Tuesday, January 12	Lecture—Milk sanitation		Discussion—Milk sanitation.	Lecture—Typhus fever control.	Film—"Rodent Control."	Discussion — Typhus fever control.
Wednesday, January 13	Lecture—Health education.	Lecture and film on nutrition.	Lecture — Tuberculosis control.	Lecture — Industrial hygiene.	Lecture — Public health laws.	Group discussion on laws.
Thursday, January 14	Lecture — Public health nursing.	Lecture—Epidemiology	Lecture—Intestinal parasites.	Group conferences		Individual conferences.
Friday, January 15	Lecture—Malaria control.	T. V. A. Malaria film	Field assignments	Review questions in preparation for final examination.		
Saturday, January 16	Final examination					

completion. Students who complete the training course successfully will be assigned to local health departments. Most of them will be assigned to their home communities. They will be paid with funds already budgeted for salaries covering positions left vacant by those who have gone into military service or other employment. Salary scales lower than those for the regular classifications have been adopted. During the training period the students received a monthly stipend, a per diem allowance, and travel expenses.

While the North Carolina program cannot be evaluated fully until the actual achievements of the new personnel in the field are noted, it is believed that the results will be distinctly worth while. Members of the State staff and representatives of the Public Health Service who helped organize and conduct the training course were most favorably impressed with the quality of the personnel recruited and the way in which they reacted to the instruction. When the program was launched it was expected that a definitely substandard group would be enrolled. This did not prove to be the case, as can be seen by the education and background of the recruits. Indeed, a considerable proportion of them possess the qualities which will enable them, with the benefit of experience and further instruction, to become permanent members of the State and local health organizations.

This raises the possibility that public health organizations may be making a mistake by adhering uncompromisingly to an "all or none" attitude with regard to the educational requirements of personnel. The public health structure as it exists today was built and developed mainly by persons who might be termed "field hands." They grew up and went to school on the job. They had a general concept of what they were striving for, and on the basis of that concept they did their work. The present manpower shortage makes it necessary for health agencies once again to employ field hands and train them on the job. The process should be easier today than in former years because the general level of education has risen considerably.

Some health officers hesitate to fill wartime vacancies because they fear that after the war they will be confronted with returned service men and women seeking jobs in which others have managed to entrench themselves. This attitude is based on the assumption that postwar health programs will be frozen at prewar levels, and that there will be a surplus of qualified manpower. I do not believe that either of these assumptions is warranted. We are fighting this war not only to maintain our present degree of social security but also to guarantee that we shall have the right and the opportunity to extend that security in the future. Such extension will require more attention to public health than ever before, and health departments should expect to expand rather than suffer curtailment. Moreover, it is unfortunate but true that some of those now in military service will

not return, and others will be incapacitated. Still others will not wish to re-enter the field of public health; the war will have given them an entirely different outlook, and they will want to take up other occupations. And a considerable force of trained personnel will undoubtedly be needed abroad to help the liberated nations recover from the terrible ordeal they have been through.

All these considerations give special importance to the pioneer program undertaken in North Carolina. In summary, the chief features of this program are: (1) The State made a careful analysis of its personnel problem; (2) it established emergency classifications in its merit system; (3) it is striving to meet its requirements by recruiting its own citizens, finding employment for them prior to induction, and meeting the costs with funds budgeted and available for salaries; (4) it has organized a systematic training program which includes initial instruction, planned field training, and continuing supervision in local health departments.

The pattern of action thus established by North Carolina may well be studied by other States and emulated where conditions warrant.[1] Similar programs in other States would fortify the health departments for the increased responsibilities and tasks imposed by the war, and would preserve the framework of organization for more complete health services in the postwar era.

THE TOXICITY AND HISTOPATHOLOGY OF SOME AZO COMPOUNDS AS INFLUENCED BY DIETARY PROTEIN[2]

By M. I. Smith, *Chief Pharmacologist*, R. D. Lillie, *Senior Surgeon*, and E. F. Stohlman, *Assistant Pharmacologist, United States Public Health Service*

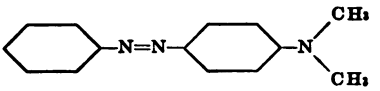
Kinosita's (*1*) discovery of the production of carcinomatosis of the liver of the rat by dimethylaminoazobenzene fed in a rice diet stimulated many investigators to search for an explanation of the phenomenon from the standpoint of chemical constitution in relation to carcinogenesis as well as from the point of view that dietary factors might influence this process. Nakahara and coworkers (*2*) showed that liver supplements had a counteracting effect against dimethylaminoazobenzene while Ando (*3*) ascribed similar results to yeast. Confirmatory reports have subsequently appeared concerning the protective action of liver (*4*), yeast (*5*), and millet (*6*). Miller and coworkers (*7*), using semisynthetic diets, were able to demonstrate a protective action with casein, liver, egg, and yeast proteins while xanthine, l-cystine, i-inositol, and choline had no such effects. The ineffectiveness of cystine has been confirmed by Mori (*8*), though

[1] After this article was written word was received that the Tennessee State Health Department would institute a similar training program beginning March 15.

[2] From the Division of Chemotherapy and Division of Pathology, National Institute of Health.

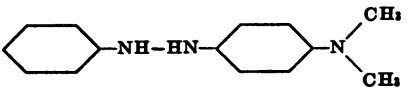
Gyorgy's (*9*) experiments would seem to indicate that cystine and choline together afford some protection. From the experiments of Kensler and associates (*10*) it would appear that at least two dietary factors, riboflavin and casein, are concerned with this antagonistic action. The most recent suggestion implicating biotin comes from Laurence (*11*); it is based on West and Woglom's (*12*) observation of high biotin content of cancerous tissues compared with normal. According to this, biotin deficiency might lead to retardation of the cancerous process, and in line with this DuVigneaud and associates (*13*) present evidence to indicate that biotin might play a precancerous role.

The question of chemical constitution in relation to liver injury produced by p-dimethylaminoazobenzene (I)



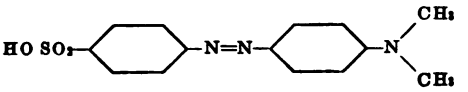
I

has also received considerable attention. Kinosita (*14*) has shown among other things that the hydrazo derivative (II) of



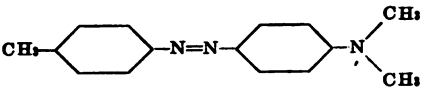
II

p-dimethylaminoazobenzene and the 4-sulfonic acid derivative (III) were devoid of the carcinogenic properties of the



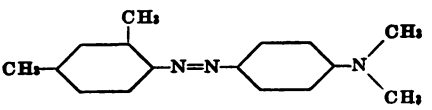
III

parent substance. Nagao (*15*) examined the effects of methylation of one of the benzene rings of p-dimethylaminoazobenzene and found that the 4-methyl derivative (IV) differed

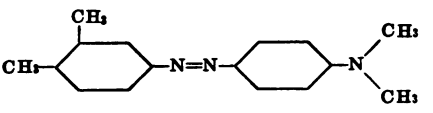


IV

little from the parent substance while the 2-4-dimethyl derivative (V) had definitely less and the

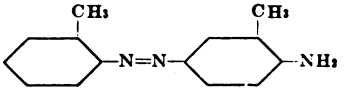


V

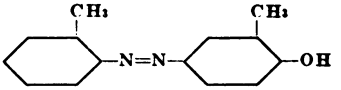


VI

3-4-dimethyl (VI) markedly less carcinogenic action than the parent substance. Law (*16*) reported greater carcinogenic action from the subcutaneous injection in mice of 4′-amino-2,3′-azotoluene (VII)

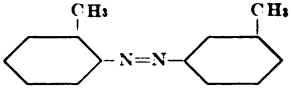


VII



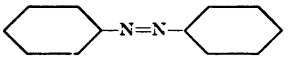
VIII

than from p-dimethylaminoazobenzene (I), 4′-hydroxy-2,3′-azotoluene (VIII) or 2,3′-azotoluene (IX).

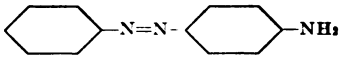


IX

The present report concerns the chronic toxicity and liver pathology of rats fed azobenzene (X), p-aminoazobenzene (XI), and p-dimethylaminoazobenzene (I)



X



XI

in a semisynthetic diet of low or high protein content. The drugs were dissolved in olive oil and incorporated into the food mixture consisting of casein or other protein as specified, McCollum's salt mixture No. 185 4, cod liver oil 2, olive oil 8, dried brewers yeast 5, and sufficient starch to make 100. The low protein diets contained 4 to 5 percent casein supplemented with 0.1 percent cystine in addition to the 3 percent protein derived from the yeast. No cystine was included in the diets with optimal or supraoptimal protein content. Wistar rats of either sex weighing 80 to 120 gm. were used. The animals were weighed at weekly intervals. In many cases hematological studies were made to ascertain the hemoglobin level, the reticulocyte count, and other abnormalities if present. In some of the experiments a liver function test was performed before sacrificing the animals. This was done by injecting intravenously 25 mg. per kg. rose bengal as a 0.5 percent aqueous solution and determining the degree of dye retention in the plasma at exactly one hour. The concentration of the dye in the plasma was measured spectrophotometrically as previously described (*17*). In a series of 20 control rats weighing from 60 to 315 gm. so treated with rose bengal the dye retention at one hour varied from 0.5 to 1.5 mg. percent with an average of 0.97 mg. percent and a standard deviation of 0.37. Retention in excess of 2.08 mg. percent ($0.97+3\times$standard deviation) should, therefore, be regarded as an indication of impaired liver function.

RESULTS

Azobenzene fed in the proportion of 0.1 percent proved so toxic that most animals died within a few days. The concentration of the drug had to be reduced to 0.06 percent to permit sufficiently long survival. The toxicity of the drug did not appear greater in the animals receiving low protein (4 percent casein) than in those receiving high protein (27 percent casein). Even at this low level of drug intake many animals died within a month. The hematological findings in these animals were essentially negative.

These rats developed rather regularly a peculiar hyaline degeneration in the liver. This was present in 21 of 23 rats killed after 25 to 63 days. The liver cells in the central one-third to one-half of the lobule presented changes varying from granular cytoplasmic oxyphilic to hyaline oxyphilic masses continuous directly with the more or less reticulated basophilic cytoplasm. In many areas these hyaline masses were rounded and separated from the rest of the cytoplasm, lying free within vacuoles. This hyaline material tinges only slightly with methyl violet, basic fuchsin, or Sudan IV. It is pale blue green with toluidin blue. It stains pink with eosin-polychrome methylene blue. The lack of metachromasia to methyl violet is against an amyloid nature of the material.

Nine additional rats were fed as above for 47 days and then returned to an 18 percent casein, azobenzene-free diet for 30 days. When killed, their livers contained no hyaline material. The Kupffer cells, however, showed more or less swelling and hemosiderosis in both groups.

The spleens in the azobenzene-fed animals were generally enlarged, their follicles showed no consistent significant alterations, the blood content of the pulp was usually about normal, and there was generally a fairly marked peritrabecular to diffuse infiltration by large basophilic cells, the nuclei of which were more or less pachychromatic. These are myeloid cells, probably of the erythroblastic series, as rather numerous normoblasts were generally present elsewhere in the pulp. Inconstantly, numbers of megakaryocytes were noted. In the rats which were given a recovery period of 30 days on normal diet, this somewhat marked erythropoietic reaction was reduced to a slight reaction. In all rats, including the "recovery" group, there was a moderate to marked hemosiderosis of the pulp reticuloendothelium comparable in grade in the two series.

The renal convoluted tubules almost regularly showed basally striated, fat-free epithelium with moderate to marked accumulation of finely granular brown pigment in the epithelial cells. This pigment usually remained brown with eosin methylene blue and did not tinge with Sudan IV. In a variable proportion of individual tubules (from

one-fourth to practically all) this pigment was deep blue with acidulated potassium ferrocyanide solution, remaining brown in the rest. Either all the pigment in a given tubule, or none of it, appeared to react. Iron-positive pigment was more plentiful in the distal tubules. As previously suggested (*19*) this iron-negative brown pigment probably represents a desiderized hemosiderin. Miller (*20*) has adopted a similar view in regard to the renal pigmentation of lead poisoning, and Edwards and White (*21*) in regard to that of p-dimethylaminoazobenzene poisoning. In the animals in the "recovery" group the renal pigmentation was much decreased in amount, often not detected without the ferrocyanide reaction, and with this it involved perhaps an eighth of the tubules on an average in place of about five-eighths in the azobenzene group.

In most of the azobenzene animals there was seen the nodular papillary hyperkeratosis of the proventriculus of the stomach, which is commonly seen in low protein, yeast-containing diets regardless of presence or absence of toxic substances in the diet. This was not noted in the animals which had been given an adequate diet for the last 30 days after a low protein, azobenzene regime.

Summarizing, azobenzene fed to rats on a low protein diet produces a centrolobular hyaline alteration of liver cells, a more or less marked splenic erythropoietic reaction, and a hemosiderosis of spleen, liver, and kidneys. During recovery, the hepatic lesions disappear without trace, the erythropoietic reaction is greatly decreased, the hemosiderosis decreases most in the liver and kidney, least in the spleen.

Para-aminoazobenzene appeared to be less toxic in rats since it was generally tolerated up to 0.1 percent. This substance readily produced anemia in rats characterized by anisocytosis with numerous macrocytes, polychromatophils, reticulocytes, Howell-Jolly bodies, and a few normoblasts. The hemoglobin level was often reduced to as low as five gm. The anemia was especially pronounced in the low protein group. A series of rats fed this substance at a level of 0.1 percent in both low and high protein diets were sacrificed at from 35 to 41 days for histologic study.

As with azobenzene, the liver cells of the low protein group in the central one-third to one-half of the lobule presented granular cytoplasmic oxyphilia, grading over to hyaline oxyphilic masses and to globules of similar material lying within vacuoles in the cytoplasm. The Kupffer cells often presented swelling and hemosiderin pigmentation.

In the spleen the follicles showed no important changes. The pulp showed fairly marked peritrabecular to diffuse infiltration with large basophilic cells and rather numerous normoblasts around them. At 6 weeks, this erythropoietic reaction was somewhat similar in grade in the high and low protein groups. Hemosiderosis of the pulp reticuloendothelial cells was also marked in all animals.

With p-aminoazobenzene the epithelium of the renal convoluted tubules was usually fat free, basally striated, and more or less heavily pigmented with granular brown pigment. This pigment was iron positive in some tubules and iron negative in others. The renal hemosiderosis was possibly somewhat more marked in the low protein diet group, but was present also in considerable grade in the high protein diet group.

The same papillary keratosis of the forestomach seen in other animals on low protein, yeast-containing diets was present also in the low protein group of p-aminoazobenzene-fed rats.

Summarizing, p-aminoazobenzene fed to rats on a low protein diet produces a centrolobular hyaline alteration of liver cells, a considerable degree of anemia, and a more or less marked splenic erythropoietic reaction with hemosiderosis of the spleen, kidneys, and liver. Both the anemia and the liver injury may be largely overcome by high dietary protein. The relative effects of p-aminoazobenzene on the respective diets are shown in tables 1 and 2. Abnormal rose bengal retention was present in 10 of 11 animals on the low protein diet, and in only 4 of 11 on the 27 percent casein diet. The morphological alterations in the liver, as noted in the tables, were generally consistent with the functional changes.

Para-dimethylaminoazobenzene.—This substance was fed at a level of 0.1 percent. In conformity with previous reports on this subject hepatic cirrhosis was observed within 30 to 50 days with a rapidly rising incidence of tubular or solid or both types of adenomatosis, so that by 80 to 100 days this is usually almost constant when para-dimethylaminoazobenzene is fed in a diet containing 7 to 8 percent protein. However, both the incidence and especially the extent of

TABLE 1.—*Experiments with p-aminoazobenzene fed at a level of 0.1 percent in a diet containing 4 percent casein supplemented with 0.1 percent cystine*

Number	Weight		Days	Rose bengal retention Mg. percent at 60′	Microscopic pathology[1]	
	Initial	Final			Hepatic hyaline degeneration	Splenic myelosis
1	108	80	35	7.45	++	+
2	100	78	35	4.07	++	±
3	110	90	35	5.23	++	++
4	112	98	35	3.29	+	+
5	110	116	35	3.10	+	++
6	118	108	35	4.60	+	+
7	100	78	36	1.00	+	+
8	108	86	36	2.52	++	+
9	92	78	36	4.70	+	+
10	108	96	37	3.01	+	++
11	112	106	37	4.79	++	++

[1] In this and subsequent tables:
− designates none.
± slight or doubtful.
+ definite.
++ marked.

adenomatosis of the liver are low and the survival is decidedly better on the high protein (37 percent casein) diet. Thus, in one series of 9 rats on the low protein diet surviving 120 to 150 days all showed extensive involvement; in a parallel series of 12 rats on 37 percent casein, 8 showed microscopic evidence of adenocarcinoma, 2 showed only slight evidence, and 2, none. This was repeated in another series of experiments, as shown in tables 3 and 4, with the result that 5 of 11 animals on the low protein diet showed extensive carcinomatosis of the liver within 47 to 133 days with abnormal rose bengal retention in all cases. In a parallel series on the high protein diet, only one animal showed a somewhat elevated rose bengal concentration in the plasma though microscopic evidence of carcinoma was present in 4 out of 10. Liver cirrhosis was noted in all animals of both groups.

The favorable influence of casein prompted an investigation of the effects of three other proteins. Yeast was tried because of its high nutritive value and high biotin content. Egg white was used for its avidin content capable of inactivating what biotin there might be in

TABLE 2.—*Experiments with p-aminoazobenzene fed at a level of 0.1 percent in a diet containing 27 percent casein*

Number	Weight		Days	Rose bengal retention Mg. percent at 60′	Microscopic pathology	
	Initial	Final			Hepatic hyaline degeneration	Splenic myelosis
1	86	65	38	3.19	+	+
2	86	102	38	1.34	−	+
3	90	88	40	.57	+	++
4	94	108	40	1.42	±	+
5	92	155	40	1.11	−	++
6	82	126	41	3.19	±	++
7	84	150	41	.87	−	++
8	92	142	41	2.37	±	++
9	82	138	41	2.84	±	++
10	84	124	41	1.69	±	++
11	82	78	41	1.78	−	+

TABLE 3—*p-Dimethylaminoazobenzene fed to rats at a level of 0.1 percent in a diet containing 5 percent casein plus 0.1 percent cystine*

Number	Weight		Days	Rose bengal retention Mg. percent at 60′	Microscopic liver pathology			
	Initial	Final			Hyaline degeneration	Cirrhosis	Duct proliferation	Adenocarcinomatosis
1	100	64	110	10.45	+	+	+	±
2	130	76	110	3.37	−	+	−	−
3	100	78	110	8.63	−	+	+	+
4	100	65	110	14.89	−	+	+	+
5	108	68	47	13.15	−	+	±	−
6	100	78	124	6.99	+	+	+	+
7	124	88	124	13.56	+	+	+	+
8	110	80	63	20.00	−	+	+	−
9	106	92	63	12.77	−	+	−	−
10	130	132	133	7.45	+	+	+	+
11	130	124	133	7.45	+	+	+	−

the basal ration. In a third group of rats gelatin was used as a protein of low biologic value, it being partially or wholly deficient in the essential amino acids—tyrosine, tryptophane, methionine, and cystine. The last two proteins were fed at a level of 32 percent in addition to the 5 percent casein contained in the basal diet, while the yeast was fed at a level of 53 percent, the approximate equivalent of 32 percent protein.

TABLE 4—*p-Dimethylaminoazobenzene fed in a diet containing 37 percent casein*

Number	Weight		Days	Rose bengal Mg. percent at 60′	Microscopic liver pathology				Splenic myelosis
	Initial	Final			Hyaline degeneration	Cirrhosis	Duct proliferation	Adenocarcinomatosis	
1	86	144	133	1.55	±	+	+	±	+
2	86	164	133	0.97	±	+	+	+	+
3	90	170	133	1.25	±	+	−	−	+
4	90	180	133	1.08	+	+	+	±	+
5	82	100	133	2.44	+	+	+	+	+
6	90	188	133	0.71	+	+	+	±	+
7	110	140	80	1.68	+	+	+	−	+
8	104	176	80	0.93	+	+	−	−	±
9	104	176	80	0.62	+	+	+	+	±
10	100	120	80	0.56	±	+	+	+	+

TABLE 5—*p-Dimethylaminoazobenzene fed in a diet containing 5 percent casein plus 53 percent dried brewers yeast*

Number	Weight		Days	Rose bengal Mg. percent at 60′	Microscopic liver pathology				Splenic myelosis
	Initial	Final			Hyaline degeneration	Cirrhosis	Duct proliferation	Adenocarcinomatosis	
1	118	106	87	0.84	−	+	+	±	+
2	108	70	87	3.37	−	+	+	±	+
3	108	76	46	4.05	+	+	−	−	+
4	92	70	46	6.48	−	+	+	±	+
5	110	86	46	3.84	+	+	−	−	+
6	96	70	46	4.27	+	+	+	−	+
7	110	102	47	1.55	+	+	−	−	+

TABLE 6.—*p-Dimethylaminoazobenzene fed in a diet containing 5 percent casein plus 32 percent ovalbumin. All animals were sacrificed after 112 days*

Number	Weight		Rose bengal retention Mg. percent at 60′	Microscopic liver pathology				Splenic myelosis
	Initial	Final		Hyaline degeneration	Cirrhosis	Duct proliferation	Adenocarcinomatosis	
1	98	130	0.93	±	+	−	−	−
2	110	128	.63	++	±	−	−	±
3	98	112	.93	+	+	++	−	−
4	104	106	1.18	±	++	+	±	−
5	98	130	1.26	+	+	−	−	±
6	108	126	1.33	±	−	−	−	±
7	88	95	1.50	+	+	+	±	−
8	100	100	1.80	+	±	−	−	±
9	86	100	.93	+	±	−	−	±
10	110	106	3.85	+	±	−	−	−
11	100	104	3.01	+	±	−	−	−
12	98	124	.93	±	−	−	−	−

TABLE 7.—*p-Dimethylaminoazobenzene fed in a diet containing 5 percent casein plus 32 percent gelatin. Survivors sacrificed after 79 days*

Number	Weight		Rose bengal Mg. per-cent at 60′	Microscopic liver pathology				Splenic myelosis
	Initial	Final		Hyaline degener-ation	Cirrhosis	Duct pro-liferation	Adeno-carcino-matosis	
1	100	60	12.76	±	+	+	±	−
2	118	108	8.01	±	+	+	+	+
3	100	60	6.74	−	+	++	+	−
4	102	100	8.77	−	+	+	−	±
5	104	78	5.0	−	+	+	±	±
6	102	84	8.47	±	++	±	±	+
7	100	54	13.54	±	++	±	±	+

The results of these experiments are shown in tables 5, 6, and 7. The toxicity of p-dimethylaminoazobenzene, curiously enough, was so greatly enhanced in the yeast diet that it was difficult to carry more than a very small percentage of the animals beyond 40 to 50 days. Of a series of 40 rats only 7 survived sufficiently long (47 to 87 days) to permit observation of some of the effects. In this small group all the livers showed cirrhosis, and none showed definite adenocarcinomatosis. Duct proliferation was present in 4 of the animals. Rose bengal retention was abnormally high in all but 2 of the animals.

Table 6 shows the marked protective action of egg albumin. There were no deaths in this group of animals. Rose bengal retention was somewhat above normal in only 2 of the animals. Hepatic cirrhosis was noted in only 5 of the animals, duct proliferation in 3, and in none was adenocarcinoma seen. Only centrolobular hyaline degeneration of the liver such as is produced by the less hepatotoxic agents, azobenzene and p-aminoazobenzene, was noted in the majority—in 8 of 12 animals. Splenic myelosis so general with the azobenzenes was scarcely noticeable in this group of experiments.

The effects of gelatin are shown in table 7. The mortality rate was high in this group of experiments, and the few survivors were sacrificed after 79 days. Abnormal rose bengal retention was present in all, as was hepatic cirrhosis. Duct proliferation was present in 5 and adenocarcinomatosis in 2. Centrolobular hyaline degeneration was not much in evidence and the incidence of splenic myelosis was low.

The results of the experiments with p-dimethylaminoazobenzene are summarized in table 8.

The development of the cirrhosis appears to proceed as follows: The previously described centrolobular hyaline alteration of the liver cells seen in rats, after being fed for 3 to 4 months on high protein diets containing azobenzene and aminoazobenzene and often at shorter intervals on low protein diets, appears to be an initial phase in the development of the cirrhosis. Apparently first in such areas of hyaline

TABLE 8.—*Summary of experiments with p-dimethylaminoazobenzene*

Diet	Rose bengal retention average Mg. percent at 60′	Liver pathology, percent incidence			
		Hyaline degeneration	Cirrhosis	Duct proliferation	Adenocarcinomatosis
	Percent	*Percent*	*Percent*	*Percent*	*Percent*
Basal, 5 percent casein	10. 80	45	100	73	45
37 percent casein	1. 18	60	100	80	40
32 percent yeast protein	3. 48	57	100	57	0
32 percent gelatin	6. 26	0	100	71	29
32 percent egg white protein	1. 52	66	42	25	0

degeneration and sometimes evidently proceeding from the wall of the hepatic venule there appears an interstitial proliferation of fibroblasts. Sometimes atrophy and disappearance of liver cells precedes fibroblast proliferation, so that one sees a capillary net engorged with blood or collapsed, with no intervening liver cells, and usually hemosiderin pigmentation. Such pictures, however, are infrequent. More often one finds interstitial fibroblast proliferation proceeding to form trabeculae by extension to neighboring areas and also to portal areas. Liver cells, with or without cytoplasmic hyaline, may be included in the developing trabeculae, and may be seen in varying phases of shrinkage, cytoplasmic oxyphilia and karyopyknosis, and coagulative and karyorrhectic necrosis. Almost from the start, and throughout this intoxication, in purely cirrhotic areas there are present in the trabeculae more or less numerous phagocytes laden with finely granular dark brown pigment not tinged appreciably by thiazine dyes or basic fuchsin. This finely granular pigment is almost regularly iron positive. In the same cells one often finds among the hemosiderin granules small clear vacuoles and, in frozen sections, small fat droplets. This fat is not acid fast and is not demonstrable with Sudan IV in paraffin sections. These pigment phagocytes are often quite large and may contain multiple, irregularly scattered nuclei.

In contrast with Edwards' findings (*21*), coarse fatty infiltration of parenchyma is often absent. However, when one considers that Edwards and White used the low protein diet of White and Jackson (*22*), the reason for this difference becomes clear, as our animals fed low protein diets also often showed more or less pronounced fatty changes in the parenchyma, while in those given high and adequate protein diets, fatty changes were usually absent.

In many of the fatty livers there was usually found a small amount of acid-fast material This occurred chiefly as rims about large fatty vacuoles and also as smaller solid globules in liver cells. Infrequently, small droplets of similar material occurred free or in phagocytes in the trabeculae. This material was found in many of the animals on the 4 percent casein diets and on the 32 percent gelatin, but occurred in none of those receiving high casein, egg white, or yeast diets.

This acid-fast material, like that seen by Edwards (*21*) in his studies, stains with Sudan IV after paraffin imbedding, is insoluble in fat solvents, is iron-negative, and fails to stain with dilute fuchsin or eosin methylene blue. In the last two particulars it differs from the much more copious, acid-fast ceroid (*23*) of the low protein cirrhosis of rats, which stains blue green with eosin methylene blue and pink with dilute fuchsin.

Since, as Edwards and White (*21*) note, this material occurs in animals receiving deficient diets and no p-dimethylaminoazobenzene, and since animals developing cirrhosis from this chemical on adequate protein diets do not show it, it is concluded that the acid-fast material is not due to the intoxication but to the deficient diet.

In the further development of the cirrhosis, areas of bile duct proliferation appeared. Often these ducts are regular, but in many instances one also sees eccentrically much thickened hyperbasophil epithelium which apparently grades over into solid epithelial cords and thick walled tubules. Accompanying this duct proliferation, there may be more or less marked lymphocyte infiltration and fibrosis. Some of these hyperbasophilic tubules show partial necrosis; some may be filled with intact and fragmenting leucocytes.

One also sees in the segregated nodules of hepatic tissue isolated giant liver cells with hyperchromatic vesicular nuclei, areas of generalized cell enlargement and hyperplasia and gradation to nodules of large coherent polygonal cells, giant tubules lined by very large cuboidal or columnar cells, cystic areas with papillary ingrowths, and blood cysts which may be invaded and lined by similar tumor tissue.

While the classification and genesis of the tumors arising in this cirrhosis is beyond the primary purpose of this paper, it is not believed that either the hepatic cell or the intrahepatic duct cell is the sole source of these tumors. That the adenocarcinomas may arise from parenchyma, as Edwards (*21*) states, seems quite evident but it cannot be agreed that there are no transitions from the non-neoplastic duct proliferations.

In the renal convoluted tubules basal striation is usually retained, manifest fatty changes are absent, and there is almost regularly a deposition of fine to medium brown pigment granules in their epithelium. In perhaps half of the animals a variable proportion of the tubules showed a positive iron reaction for this pigment, the rest a negative reaction.

As with azobenzene, the principal splenic changes are splenic myelosis, chiefly erythropoietic in type, and splenic hemosiderosis. The diet appeared to have no consistent effect on either of these.

Summarizing the liver changes, p-dimethylaminoazobenzene fed to rats on either high or low protein diets produces hepatic cirrhosis, an inconstant preliminary phase of which appeared to be a centrolobular

hyaline degeneration of liver cells like that seen in azobenzene and p-aminoazobenzene poisoning. The fatty changes observed in the liver parenchyma in many animals appear to be associated with low or inadequate protein diets and not with the intoxication. The acid-fast lipoprotein formed in liver cells and occurring sometimes in intratrabecular phagocytes appears to occur exclusively in more or less fatty livers, and hence is probably not due to p-dimethylaminoazobenzene, but is related to the ceroid formed in the protein and choline deficiency cirrhosis of rats (*23*).

The hepatic cirrhosis is thus probably primarily centrolobular in origin, but soon involves portal areas. The trabeculae contain numerous hemosiderin and fat phagocytes and areas of lymphocyte infiltration, fibrosis, and duct proliferation. The last apparently grades into duct carcinoma. There is also a liver cell hyperplasia which becomes nodular, gradually evolving into solid, tubular, and papillary carcinoma which invade veins and cystic blood spaces.

COMMENT

Both azobenzene and p-aminoazobenzene are capable of producing centrolobular hyaline degeneration in rats, a condition which is fully reversible upon withdrawal of the substance from the diet, and which can be prevented by liberal inclusion of casein in the diet. The functional capacity of the liver as judged by retention of intravenously injected rose bengal parallels the morphological changes. p-Aminoazobenzene in addition can also produce anemia, this too being aggravated by low dietary protein and preventable to a large extent by high dietary protein.

p-Dimethylaminoazobenzene usually produces the centrolobular hyaline degeneration, followed by liver cirrhosis, duct proliferation, and carcinomatosis of the liver. These progressive changes are most pronounced when the rat is fed a low protein diet. Pronounced retention of intravenously injected rose bengal is present in all such animals. High dietary casein has modified the picture somewhat, the most marked change being a practically normal rose bengal clearance. This appears to be due to the high rate of liver cell regeneration. High gelatin and high yeast protein have not had a favorable effect except for reducing the incidence of carcinomatosis, but this is probably explained by the shorter survival period. The most pronounced ameliorating effect was noted with egg white protein. If the suggested procarcinogenic effect of biotin (*13*) may be accepted, then the favorable influence of egg white protein is explainable on the basis of its avidin content which can inactivate biotin. It seems more likely, however, that egg white protein can partially detoxify p-dimethylaminoazobenzene in some unknown manner. Similar pro-

tective action of egg white protein against the liver injury and anemia produced by selenium has been reported previously (*18*).

SUMMARY

The toxic effects of azobenzene, p-aminoazobenzene, and p-dimethylaminoazobenzene have been studied in rats, and the influence of dietary protein thereon has been investigated.

Azobenzene and p-aminoazobenzene produce centrolobular hyaline degeneration of the liver with marked impairment of liver function as measured by retention of intravenously injected rose bengal. p-Aminoazobenzene also produces anemia. Both the hyaline degeneration and anemia are preventable by high dietary protein.

The structural and functional liver damage produced by p-dimethylaminoazobenzene is favorably influenced by the inclusion of a high percentage of casein in the diet, but not by yeast protein or gelatin. Egg white protein has greatly reduced the extent and incidence of both hepatic cirrhosis and carcinomatosis within an experimental period of 112 days. The nature of the detoxifying action of egg white protein is not known, though it is possible that it may be due to its avidin content which inactivates biotin.

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AN OUTBREAK OF *MICROSPORON LANOSUM* INFECTION FROM A KITTEN [1]

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Microsporon lanosum infection of the scalp in children is not uncommon. However, the following report of four cases is of interest since it has been possible to trace the infection to the animal which initiated it. Three children and one adult were infected. The infection was limited to the glabrous skin, an unusual finding because in children infected with *M. lanosum* the scalp is usually involved.

CLINICAL CASES

Case 1.—R. H., male, 13 months of age. The infection had existed for about 3 or 4 weeks. A sharply circumscribed scaly patch was seen on the left cheek. Cultures revealed *M. lanosum*. The scalp was free of lesions.

Case 2.—T. W., white male, 8 years of age. The onset of skin eruption occurred 3 to 4 weeks before the patient was first seen. There were numerous oval patches of scaling on the trunk, as well as large lesions on the face and forearms. The patches were about 2 x 1 cm. They showed no surrounding inflammatory areola but in a few

[1] From the Division of Industrial Hygiene, National Institute of Health.

instances showed tiny vesicles at the periphery. Culture of the lesions revealed *M. lanosum*. The scalp was free of any signs of infection.

Case 3.—L. R., white female, 6 years of age. The onset occurred about 2 to 3 weeks before the patient was seen. On the right cheek were oval patches, several centimeters at the longest diameter, covered with fine scales with some tendency to clear in the center. The scalp was free of any lesions even under the Wood's light. Culture revealed *M. lanosum*.

Case 4.—Mrs. H., mother of baby R. H. (case 1). A short time after a lesion appeared on the baby's cheek the mother developed, on the right cheek, an oval area of scaling 1½ cm. at its longest diameter with peripheral vesicles and some tendency to clearing in the center. There was no surrounding zone of erythema.

Close questioning in an attempt to find a possible common source of infection revealed that all of the children were neighbors and had played with a kitten about 6 weeks old. Examination of the kitten showed scaling around the nose and infected hairs around the ears and head. Culture of the infected hairs from the kitten showed *M. lanosum*.

DISCUSSION

Fox and Blaxall (*1*) were apparently the first to record transmission of ringworm from cat to man. The usual organism isolated from the cat was *M. lanosum* (*felineum*). It is interesting to note that only young cats are usually affected, which would parallel the observation that the hair is affected only in children below the age of puberty. Hare and Tate (*2*) state that among school children in London the infection with animal ringworm is negligible. The infection is usually spread from child to child.

Davidson and Gregory (*3*) report that more than half of the cases of ringworm due to microsporon in Winnipeg were due to *M. audouini* while two-thirds of the separate outbreaks were due to *M. lanosum* (*felineum*). There was some evidence in about one-half of their cases that the infection had initiated in a cat or a dog.

Bloch (*4*), Lewis and his coworkers (*5*), Wise and Sulzberger (*6*), and others have repeatedly drawn attention to the fact that the fungi which usually affect the hairs and hairy areas can be divided into two categories: the animal pathogens among which are *M. lanosum* and *Trichophyton gypsium* which commonly affect lower animals and occasionally man, and the human pathogens which commonly affect man and not lower animals (*M. audouini*). The importance of this division is in the recognition that the animal pathogens have a relatively strong tendency to spontaneous healing while the human pathogens have little or no tendency to spontaneous healing. *Microsporon audouini* infection of the scalp, however, heals spontaneously at puberty.

While it has always been recognized that scalp infection in children may be caused by contact with infected animals, the series of cases reported here stresses the importance of seeking animal sources, even in tinea infections of the glabrous skin both in children and adults. These animal sources may be horses, dogs, or cats. The most practical method of terminating an epidemic of this kind is to find and then cure or destroy the animal which initiated it. Not only may the infection spread from the animal to human contacts but it may also spread from animal to animal and thus multiply the foci for new infections. Too often has it been assumed that infections were direct from child to child and the possibility that a pet initiated the original infection was overlooked. Whether the infection can be spread from persons to animals has not been investigated but there is a possibility that this may occur.

Infections of both the hair and the glabrous skin, when due to *M. lanosum*, are more amenable to local treatment than are the cases caused by human pathogenic fungi such as *M. audouini*. This is because the animal fungi are prone to cause the development of a hypersensitivity to the fungi and their products which can be demonstrated by means of a trichophytin test. The trichophytin reaction is usually negative in infections with *M. audouini*. Usually X-ray epilation of the scalp must be resorted to in treating infections with human pathogens; local treatment is successful in a great many cases infected with *M. lanosum*.

In this particular group of cases the infection was transmitted from the kitten to the children and from one of the children to his mother. While the two older children played with each other and with the kitten, they had no direct contact with the baby. The baby, however, played with the kitten. The mother maintained that she had never touched the kitten; the lesion which developed on her cheek was situated at the level where the infected spot on the baby's face rested.

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REPORT ON MARKET-MILK SUPPLIES OF PUBLIC HEALTH SERVICE MILK ORDINANCE COMMUNITIES [1]

Compliance of the Market-Milk Supplies of Certain Public Health Service Milk Ordinance Communities With the Grade A Pasteurized and Grade A Raw Milk Requirements of the Public Health Service Milk Ordinance and Code, as Shown by Compliance (Not Safety) Ratings of 90 Percent or More Reported by the State Milk-Sanitation Authorities During the Period January 1, 1941, to December 31, 1942.

The accompanying list gives the semiannual revision of the list of certain Public Health Service Milk Ordinance communities in which the pasteurized market milk is both produced and pasteurized in accordance with the Grade A pasteurized milk requirements of the Public Health Service Milk Ordinance and Code and in which the raw market milk sold to the final consumer is produced in accordance with the Grade A raw milk requirements of said ordinance and code, as shown by ratings of 90 percent or more reported by State milk-sanitation authorities.

These ratings are not a complete measure of safety, but represent the degree of compliance with the Grade A requirements of the Public Health Service Milk Ordinance and Code. Safety estimates should also take into account the percentage of milk pasteurized, which is given in the following tables.

The milk ordinance recommended by the Public Health Service is now in effect in hundreds of communities ranging in population from 1,000 to 3,500,000 and located in 37 States and 1 Territory.

The primary reason for publishing the rating lists from time to time is to encourage these communities to attain and maintain a high level of excellence in the enforcement of this ordinance. No comparison with communities operating under other milk ordinances is intended or implied.

It is emphasized that the Public Health Service does not intend to imply that only those communities on the list are provided with high-grade milk supplies. Some communities which have high-grade milk supplies are not included because arrangements have not been made for the determination of their ratings by the State milk-sanitation authority. In other cases the ratings which have been determined are now more than 2 years old and have therefore lapsed. In still other communities with high-grade milk supplies there seems, in the opinion of the community, to be no local necessity nor desire for rating or inclusion in the list, nor any reasonable local benefit to be derived therefrom.

The rules under which a community is included in this list are as follows:

[1] From the States Relations Division.

(1) All ratings must have been determined by the State milk-sanitation authority in accordance with the Public Health Service rating method (Pub. Health Rep., **53:** 1386 (1938). Reprint No. 1970), based upon the Grade A pasteurized milk and the Grade A raw milk requirements of the Public Health Service Milk Ordinance and Code.

(2) No community will be included in the list unless both its pasteurized milk and its raw milk ratings are 90 percent or more. Communities in which only raw milk is sold will be included if the raw milk ratings are 90 percent or more. Communities which receive, without local inspection, milk from other sheds will be included in the list only if the locally inspected supply, as well as the shipped-in supply, shows a rating of 90 percent or more.

(3) The rating used will be the latest rating submitted to the Public Health Service, but no rating will be used which is more than 2 years old. In order to promote continuous rigid enforcement rather than occasional "clean-up campaigns" it is suggested that when the rating of a community on the list falls below 90 percent no resurvey be made for at least 6 months, resulting in removal from the next semiannual list.

(4) The Public Health Service will make occasional check surveys of cities for which ratings of 90 percent or more have been reported by the State. If such check rating is less than 90 percent but not less than 85, the city will be removed from the 90-percent list after 6 months unless a resurvey submitted by the State during this probationary interim shows a rating of 90 percent or more. If, however, such check rating is less than 85 percent, the city will be removed from the list immediately. If the check rating is 90 percent or more, the city will be retained on the list for a period of 2 years from the date of the check survey unless a subsequent rating submitted during this period warrants its removal.

Communities are urgently advised to bring their ordinances up to date at least every 5 years, since ratings will be made on the basis of later editions if those adopted locally are more than 5 years old.

Communities which are not now on the list and desire to be rated should request the State milk-sanitation authority to determine their ratings and, if necessary, should improve their status sufficiently to merit inclusion in the list.

Communities which are now on the list should not permit their ratings to lapse, as ratings more than 2 years old cannot be used.

State milk-sanitation authorities who are not now equipped to determine municipal ratings are urged, in fairness to their communities, to equip themselves as soon as possible. The personnel required is small, as in most States one milk specialist is sufficient for the work.

TABLE 1.—*Public Health Service Milk Ordinance communities in which all market milk is pasteurized. In these communities market milk complies with the Grade A pasteurized milk requirements of the Public Health Service Milk Ordinance and Code to the extent shown by pasteurized milk ratings of 90 percent or more* [1]

Community	Percentage of milk pasteurized	Date of ratfng	Community	Percentage of milk pasteurized	Date of rating
ILLINOIS			KENTUCKY		
Brooklyn [2]	100	Oct. 8, 1941	Louisville	100	Jan. 31, 1942
Cahokia [2]	100	Do.			
Canteen [2]	100	Do.	MINNESOTA		
Centerville [2]	100	Do.			
Champaign	100	July 23, 1941	Rochester	100	May 29, 1941
East St. Louis [2]	100	Oct. 8, 1941	Winona	100	Sept. 4, 1942
Elgin	100	Nov. 20, 1942			
Fairmont City [2]	100	Oct. 8, 1941	MISSOURI		
National City [2]	100	Do.	Ladue	100	Jan. 1942
Stites [2]	100	Do.	Richmond Heights	100	Do.
Washington Park [2]	100	Do.	St. Louis	100	June 9, 1942
Waukegan	100	Apr. 29, 1942	Webster Groves	100	Jan. 1942
IOWA			NORTH CAROLINA		
Paullina	100	Jan. 5, 1942	Greenville	100	Apr. 10, 1942

[1] Note particularly the percentages of milk pasteurized in the various communities listed in these tables. This percentage is an important factor to consider in estimating the safety of a city's milk supply.
[2] Part of East Side Health District.

The inclusion of a community in this list means that the pasteurized milk sold in the community, if any, is of such a degree of excellence that the weighted average of the percentages of compliance with the various items of sanitation required for Grade A pasteurized milk is 90 percent or more and that, similarly, the raw milk sold in the community, if any, so nearly meets the requirements that the weighted average of the percentages of compliance with the various items of sanitation required for Grade A raw milk is 90 percent or more. However, high-grade pasteurized milk is safer than high-grade raw milk, because of the added protection of pasteurization. To secure this added protection, those who are dependent on raw milk can pasteurize the milk at home in the following simple manner: Heat the milk over a hot flame to 165° F., stirring constantly; then immediately place the vessel in cold water and continue stirring until cool.

TABLE 2.—*Public Health Service Milk Ordinance communities in which some market milk is pasteurized. In these communities the pasteurized market milk complies with the Grade A pasteurized milk requirements and the raw market milk complies with the Grade A raw milk requirements of the Public Health Service Milk Ordinance and Code to the extent shown by pasteurized and raw milk ratings, respectively, of 90 percent or more* [1]

[NOTE.—All milk should be pasteurized or boiled, either commercially or at home, before it is consumed. See text for home method]

Community	Percentage of milk pasteurized	Date of rating
ALABAMA		
Dothan	89	Apr. 16, 1942.
Montgomery	45	Nov. 28, 1941.
Tuscaloosa	91	June 17, 1942.
ARKANSAS		
El Dorado	45	Sept. 1941.
Little Rock	56	Oct. 1941.
Pine Bluff	43	Nov. 1941.
Texarkana	62	Sept. 1941.
COLORADO		
Pueblo	59	Apr. 1941.
FLORIDA		
Coral Gables	98	May 20, 1942.
Dania	95	May 1, 1942.
Deerfield	95	Do.
Fort Lauderdale	95	Do.
Hallandale	95	Do.
Hollywood	95	Do.
Homestead [2]	94	May 25, 1942.
Jacksonville	78	Apr. 1941.
Marianna	96	Feb. 12, 1942.
Miami	98	May 20, 1942.
Oakland Park	95	May 1, 1942.
Panama City	55	July 1942.
Pompano	95	May 1, 1942.
Tallahassee	50	Sept. 1941.
ILLINOIS		
Chicago	99.8	Apr. 11, 1941.
Glencoe	99.6	Apr. 17, 1942.
Highland Park	99.6	Do.
Kenilworth	99.6	Do.
Lake Bluff	99.6	Do.
Lake Forest	99.6	Do.
Oak Park	99.8	Jan. 17, 1941.
Winnetka	99.6	Apr. 17, 1942.
IOWA		
Davenport	94	Nov. 11, 1942.
Humboldt	87	Jan. 12, 1942.
Mt. Vernon	48	Feb. 2, 1942.
Sheldon	61	May 18, 1942.
Washington	74	Jan. 7, 1941.
Webster City	55	May 13, 1942.
KANSAS		
Kansas City	79	July 30, 1942.
Lawrence	84	May 29, 1942.
Pratt	61	Nov. 1941.
Wichita	72	Dec. 1941.
KENTUCKY		
Bowling Green	71	Sept. 14, 1942.
Glasgow	52	June 1941.
Hazard	40	Dec. 1941.
Henderson	64	Nov. 21, 1942
Lexington	71	Mar. 14, 1942.
LOUISIANA		
Alexandria	81	Apr. 24, 1942.
Monroe	48	July 24. 1942.
Pineville	81	Apr. 24, 1942.
Shreveport	83	Mar. 14, 1942.
MICHIGAN		
Iron River	60	Nov. 15, 1942.
Stambaugh	60	Do.
MINNESOTA		
Moorhead	88	Feb. 14, 1941.
MISSOURI		
Brentwood	98	Jan. 1942.
Clayton	99.8	Do.
Ferguson	79	Do.
Glendale	99.3	Do.
Kirkwood	89	Do.
Maplewood	91	Do.
Overland	92	Do.
Rockhill	88	Do.
University City	99.5	Do.
NEW MEXICO		
Albuquerque	77	Dec. 20, 1941.
Clovis	63	Mar. 18, 1942.
Las Cruces	54	Feb. 2, 1942.
Las Vegas	65	July 18, 1941.
Taos	42	Mar. 6, 1942.
NORTH CAROLINA		
Asheboro	63	Nov. 6, 1941.
Asheville	76	May 1942.
Bethel	17	Apr. 10, 1942.
Biltmore Forest	70	May 1942.
Black Mountain	70	Do.
Concord	57	June 27, 1942.
Enka	70	May 1942.
Farmville	5	Apr. 10, 1942.
Fayetteville	73	May 7, 1942.
Greensboro	96	Sept. 24, 1942
Hendersonville	61	May 30, 1942.
High Point	94	July 17, 1941.
Hope Mills	73	May 7, 1942.
Kannapolis	57	June 27, 1942.
Kinston	30	July 31, 1942.
Mars Hill	15	Jan. 10, 1941.
Mt. Pleasant	57	June 27, 1942.
New Hanover County	86	June 30, 1942.
Roxboro	32	Jan. 16, 1942.
Swannanoa	70	May 1942.
Weaverville	70	May 1942.
NORTH DAKOTA		
Fargo	91	Feb. 16, 1941.
Valley City	33	July 24, 1941.
OHIO		
Athens	83	Nov. 5, 1942.

[1] Note particularly the percentage of milk pasteurized in the various communities listed in these tables. This percentage is an important factor to consider in estimating the safety of a city's milk supply.

[2] Has not adopted the milk ordinance recommended by the Public Health Service.

TABLE 2.—*Public Health Service Milk Ordinance communities in which some market milk is pasteurized. In these communities the pasteurized market milk complies with the Grade A pasteurized milk requirements and the raw market milk complies with the Grade A raw milk requirements of the Public Health Service Milk Ordinance and Code to the extent shown by pasteurized and raw milk ratings, respectively, of 90 percent or more*—Continued

Community	Percentage of milk pasteurized	Date of rating
OKLAHOMA		
Ada	74	Feb. 3, 1942.
Bartlesville	63	Jan. 21, 1942.
Blackwell	38	Nov. 15, 1941.
Okmulgee	64	Apr. 8, 1942.
Shawnee	48	Mar. 26, 1942.
Tulsa	83	May 20, 1942.
Wewoka	68	Dec. 14, 1942.
OREGON		
Astoria	82	June 23, 1942.
Eugene	82	Nov. 6, 1942.
Portland	82	June 16, 1942.
TENNESSEE		
Bristol	80	Dec. 1941.
Memphis	96	Nov. 1942.
TEXAS		
Brownwood	64	May 31, 1941.
Gainesville	65	Mar. 31, 1942.
Lamesa	47	Mar. 26, 1941.
Lubbock	80	Nov. 21, 1941.
Seguin	18	Sept. 10, 1941.
Sherman	53	Mar. 25, 1941.
Texarkana	82	Oct. 21, 1942.
UTAH		
Ogden	93	Sept. 15, 1941.
Salt Lake City	98	Oct. 29, 1942.
VIRGINIA		
Abingdon	38	Mar. 21, 1941.
Bristol	80	Dec. 1941.
Pulaski	99	Dec. 18, 1941.
South Boston	75	May 29, 1941.
Waynesboro	98	Nov. 15, 1941.
Williamsburg	55	May 26, 1941.
WASHINGTON		
Camas	6	June 18, 1941.
Pullman	87	Aug. 26, 1941.
Walla Walla	61	May 28, 1941.
Yakima	72	May 14, 1941.
WEST VIRGINIA		
Huntington	79	July 23, 1942.
WYOMING		
Casper	83	Nov. 1942.
Cheyenne	89	Oct. 1942.

TABLE 3.—*Public Health Service Milk Ordinance communities in which no market milk is pasteurized, but in which the raw market milk complies with the Grade A raw milk requirements of the Public Health Service Milk Ordinance and Code to the extent shown by raw milk ratings of 90 percent or more* [1]

[NOTE.—All milk should be pasteurized or boiled, either commercially or at home, before it is consumed. See text for home method]

Community	Date of rating
ALABAMA	
Albertville	May 1,1942
Boaz	Do.
Bridgeport	May 27, 1941
Fort Payne	Mar. 25, 1942
Guntersville	May 1, 1942
Scottsboro	May 27, 1941
Stevenson	Do.
KANSAS	
Horton	Mar. 30, 1942
KENTUCKY	
Owenton	Nov. 1941
LOUISIANA	
Haynesville	Mar. 10, 1942
Homer	July 23, 1942
NORTH CAROLINA	
Bladenboro	June 4, 1942
Clarkton	Do.
Elizabethtown	Do.
Hemp	Apr. 30, 1942
VIRGINIA	
Blackstone	May 29, 1941
Boydton	Apr. 4, 1941
Lawrenceville	Oct. 23, 1941
WEST VIRGINIA	
Grantsville	May 12, 1941

[1] Note particularly the percentage of milk pasteurized in the various communities listed in these tables. This percentage is an important factor to consider in estimating the safety of a city's milk supply.

PREVALENCE OF COMMUNICABLE DISEASES IN THE UNITED STATES

January 3–30, 1943

The accompanying table summarizes the prevalence of nine important communicable diseases, based on weekly telegraphic reports from State health departments. The reports from each State are published in the Public Health Reports under the section "Prevalence of disease." The table gives the number of cases of these diseases for the 4-week period ended January 30, 1943, the number reported for the corresponding period in 1942, and the median number for the years 1938–42.

DISEASES ABOVE MEDIAN PREVALENCE

Influenza.—The number of cases of influenza rose from approximately 11,000 during the preceding 4-week period to 18,397 for the 4 weeks ended January 30. The incidence was about 10 percent above that for the corresponding period in 1942, which figure (16,925) also represents the 1938–42 median incidence for this period. Excesses were reported from regions along the Atlantic Coast and from the East North Central region, the largest excess being reported from the South Atlantic region, but other regions reported fewer cases than might normally be expected. An increase in this disease normally occurs at this season of the year and as the peak is not usually reached until the middle or latter part of February, further increases may be expected.

Meningococcus meningitis.—For the current period there were 1,267 cases of meningococcus meningitis reported, as compared with 230, 163, and 129 for the corresponding period in 1942, 1941, and 1940. The corresponding median number is 212. The largest numbers of cases were reported from New York, 123; Pennsylvania, 63; Rhode Island, 61; Maine, 53; California, 101; Oregon, 64; Virginia, 87; Maryland, 59; South Carolina, 56; and Missouri, 51. For the country as a whole, the current incidence is the highest on record for this period. Each section of the country contributed to the high incidence, the increases ranging from 3.3 times the median in the West South Central region to 17.3 times the normal incidence in the New England region. Since the peak is not normally reached until the latter part of March or April, a further increase in the number of cases of this disease may also be expected.

Poliomyelitis.—The number of cases of poliomyelitis continued at a relatively high level—136 cases as compared with a median of 109 cases. The New England, West North Central, West South Central, and Pacific regions reported excesses over the expectancy; in the East North Central, East South Central, and Mountain regions the incidence stood at about the normal seasonal level, and in other regions the disease was less prevalent than in preceding years.

DISEASES BELOW MEDIAN PREVALENCE

Diphtheria.—The number of cases of diphtheria reported for the 4 weeks ended January 30 was about 90 percent of the number reported in 1942 and less than 75 percent of the 1938–42 median incidence for the corresponding period. The Mountain and Pacific regions reported excesses over the normal seasonal expectancy, but in all other regions the incidence was relatively low.

Measles.—For the country as a whole the number of cases (36,101) of measles was slightly lower than in 1942, as well as being below the 1938–42 median incidence for this period. A comparison of geographic regions, however, shows that the disease was unusually prevalent in the North Atlantic, Mountain, and Pacific regions, with minor excesses over the seasonal expectancy in the East North Central and East South Central regions. In the South Atlantic region the

Number of reported cases of 9 communicable diseases in the United States during the 4-week period January 3–30, 1943, the number for the corresponding period in 1942, and the median number of cases reported for the corresponding period 1938–42

Division	Current period	1942	5-year median	Current period	1942	5-year median	Current period	1942	5-year median
	Diphtheria			Influenza [1]			Measles [2]		
United States	1,335	1,481	1,829	18,397	16,925	16,925	36,101	36,328	36,655
New England	13	28	50	70	21	77	5,064	2,720	2,583
Middle Atlantic	152	174	230	187	97	155	14,088	7,049	7,049
East North Central	165	233	351	571	480	480	3,786	2,650	3,634
West North Central	117	94	131	404	176	686	2,033	3,187	3,187
South Atlantic	253	314	420	6,163	4,497	5,419	794	6,712	5,098
East South Central	91	147	171	1,244	1,900	2,284	1,059	606	900
West South Central	309	357	357	7,162	7,835	7,835	788	4,912	989
Mountain	77	65	73	1,031	1,181	1,181	3,353	2,161	1,579
Pacific	158	69	112	365	738	738	5,136	6,331	4,426
	Meningococcus meningitis			Poliomyelitis			Scarlet fever		
United States	1,267	230	212	136	109	109	14,150	13,722	16,487
New England	173	23	10	11	7	1	1,968	1,434	1,134
Middle Atlantic	226	49	47	7	21	13	2,732	3,094	4,190
East North Central	104	21	21	14	21	16	4,032	4,134	5,490
West North Central	99	11	11	14	9	9	1,445	1,491	1,891
South Atlantic	258	53	46	12	10	18	1,198	1,275	1,150
East South Central	82	19	22	10	10	10	581	783	666
West South Central	72	24	22	29	11	11	356	391	533
Mountain	54	7	8	7	7	7	929	467	569
Pacific	199	23	16	32	13	13	909	653	981
	Smallpox			Typhoid and paratyphoid fever			Whooping cough [2]		
United States	128	61	320	201	315	329	15,835	17,374	16,857
New England	0	0	0	10	21	17	1,894	2,266	1,551
Middle Atlantic	18	0	0	33	41	54	3,992	4,667	4,481
East North Central	65	13	64	21	34	44	3,827	4,427	3,647
West North Central	7	26	122	14	10	28	674	756	756
South Atlantic	5	1	8	49	84	84	1,672	2,062	2,164
East South Central	9	2	5	9	26	26	536	501	466
West South Central	13	11	47	43	70	74	1,260	482	482
Mountain	5	5	64	12	9	24	538	743	713
Pacific	6	3	20	10	20	20	1,442	1,450	1,450

[1] Mississippi, New York, and Pennsylvania excluded; New York City included.
[2] Mississippi excluded.

incidence was the lowest on record for this period, while decreases of approximately 35 percent and 10 percent were reported from the West North Central and West South Central regions respectively.

Scarlet fever.—The incidence (14,150 cases) of this disease was slightly higher than that reported for the corresponding period in 1942, but it was only about 85 percent of the 1938–42 median incidence. The New England, South Atlantic, and Mountain regions reported excesses over the normal seasonal expectancy, but all other regions reported a comparatively low incidence.

Smallpox.—For the current period there were 128 cases of smallpox reported, as compared with 61 in 1942 and a median of 320 cases for the corresponding period in 1938–42. Of the total cases, Pennsylvania reported 17, Ohio 44, and Indiana 16—more than 60 percent of the cases occurred in those 3 States. Sections of the country in which this disease is usually most prevalent reported very significant decreases from the 1938–42 median figures for this period.

Typhoid and paratyphoid fever.—The incidence of this disease was also relatively low, 201 cases being reported for the current period, as compared with 315 cases in 1942 and a median of 329 cases for the corresponding period in 1938–42. The situation was favorable in all sections of the country.

Whooping cough.—The number of cases (15,835) of whooping cough was low—about 10 percent below the seasonal expectancy. In the West South Central region the number of cases was about two and one-half times the median and the New England, East North Central, and East South Central regions reported minor excesses, but in all other regions the numbers of cases were below the 1938–42 median incidence.

MORTALITY, ALL CAUSES

For the 4 weeks ended January 30 there was a weekly average of 10,251 deaths reported, as compared with a weekly average of 9,731 deaths for the corresponding period in 1940–42. The current figure represents an increase over the 3-year average of about 5.1 percent. Death rates for these cities will be published when current population estimates are available from the Bureau of the Census.

DEATHS DURING WEEK ENDED FEBRUARY 6, 1943

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

	Week ended Feb. 6, 1943	Corresponding week, 1942
Data for 89 large cities of the United States:		
Total deaths	10,016	8,912
Average for 3 prior years	9,764	
Total deaths, first 5 weeks of year	51,216	46,890
Deaths under 1 year of age	681	538
Average for 3 prior years	544	
Deaths under 1 year of age, first 5 weeks of year	3,655	2,835
Data from industrial insurance companies:		
Policies in force	65,334,607	64,898,360
Number of death claims	13,741	13,086
Death claims per 1,000 policies in force, annual rate	11.0	10.5
Death claims per 1,000 policies, first 5 weeks of year, annual rate	11.1	10.4

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 FEBRUARY 13, 1943

Summary

Of the nine common communicable diseases included in the following tables, the incidence of only influenza, meningococcus meningitis, and poliomyelitis for the current week is above the respective 5-year (1938–42) medians, and only for these diseases are increases shown over the preceding week's figures.

The total of 403 cases of meningococcus meningitis reported for the current week (exclusive of 43 delayed reports in Virginia) is more than has been reported for any other week during the past 16 years, the period for which comparable weekly records are available. The accumulated total for the first 6 weeks of the current year is 2,058, more than 3 times the largest number reported for the corresponding period of the past 5 years, and more than 6 times the median. Reports for the current week show increases over the preceding week in all of the nine geographic areas except the North Central group of States. The largest numbers reported by States (preceding week's figures in parentheses) are as follows: Virginia, 61 (18); New York, 43 (39); California, 33 (24); New Jersey, 26 (12); Pennsylvania, 25 (16); South Carolina, 21 (10); Alabama, 20 (4); Rhode Island, 19 (23); Massachusetts, 19 (6); Texas, 16 (13); Washington, 15 (11).

There were 5,371 cases of influenza reported, as compared with 4,327 for the preceding week and a 5-year median of 5,180. Of the total increase for the week, 76 percent is accounted for in the reports from Texas (1,923 cases), Virginia (903), and South Carolina (733). The next largest number of cases was reported in Utah, 314.

A total of 30 cases of poliomyelitis was reported, as compared with 28 last week and with a 5-year (1938–42) median of 18. The current figure is above that reported in the corresponding week of any of the past 10 years, and the accumulated figure for the first 6 weeks of the year (194) was exceeded only once (in 1940) for the corresponding period since 1932.

Of the total of 12,803 cases of measles reported for the week, as compared with 13,444 for the preceding week and a 5-year median of 14,062, the largest numbers were reported in Pennsylvania (2,481) and New York (1,272).

Other reports for the week include anthrax, 2 cases, in Pennsylvania; dysentery, 378 (142 cases, bacillary, in New York City); tularemia, 12; endemic typhus fever, 45.

For the current week, deaths in 89 major cities of the United States totaled 9,697, as compared with 9,970 for the preceding week. The accumulated figure for the first 5 weeks of 1943 is 60,654, as compared with 58,638 for the same period in 1942.

Telegraphic morbidity reports from State health officers for the week ended February 13, 1943, and comparison with corresponding week of 1942 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 1938–42	Week ended		Median 1938–42	Week ended		Median 1938–42	Week ended		Median 1938–42
	Feb. 13, 1943	Feb. 14, 1942		Feb. 13, 1943	Feb. 14, 1942		Feb. 13, 1943	Feb. 14, 1942		Feb. 13, 1943	Feb. 14, 1942	
NEW ENG.												
Maine	0	0	1	------	------	1	13	264	155	4	3	0
New Hampshire	0	0	0	------	------	2	8	15	15	2	1	0
Vermont	0	1	0	1	------	------	280	3	10	0	0	0
Massachusetts	4	3	2	------	------	------	562	430	430	19	0	1
Rhode Island	0	0	0	2	------	------	59	82	13	19	0	0
Connecticut	0	0	0	4	8	8	367	169	169	5	1	0
MID. ATL.												
New York	14	26	26	[1] 19	[1] 14	[1] 36	1,272	404	673	43	3	3
New Jersey	6	4	9	12	22	29	733	167	167	26	2	1
Pennsylvania	9	14	25	3	------	------	2,481	1,083	1,083	25	5	7
E. NO. CEN.												
Ohio	13	13	15	7	16	16	126	222	222	2	1	3
Indiana	4	10	18	15	44	44	244	57	57	3	2	0
Illinois	12	15	30	9	23	134	323	230	230	5	0	0
Michigan [2]	2	3	9	2	1	2	215	173	323	7	1	0
Wisconsin	0	2	2	50	44	65	616	328	585	9	1	1
W. NO. CEN.												
Minnesota	0	1	3	1	------	1	28	570	359	2	0	0
Iowa	0	2	8	------	9	9	114	102	102	0	0	0
Missouri	6	2	10	1	6	42	253	85	74	11	3	1
North Dakota	0	4	2	1	32	32	0	134	15	1	0	0
South Dakota	5	4	1	------	------	4	87	16	16	0	0	0
Nebraska	2	1	1	2	2	2	125	41	22	0	0	0
Kansas	2	6	10	14	7	24	185	291	291	5	0	1
SO. ATL.												
Delaware	0	1	1	------	------	------	16	0	1	0	0	0
Maryland [2]	4	6	7	9	22	103	35	462	61	12	2	2
Dist. of Col	0	4	4	3	2	5	88	19	14	6	0	0
Virginia	5	10	12	903	385	553	173	148	148	[3] 61	0	1
West Virginia	3	5	7	18	43	46	21	641	134	2	0	1
North Carolina	11	15	20	36	16	38	32	1,171	859	7	2	2
South Carolina	1	6	6	733	784	784	46	151	47	21	2	2
Georgia	8	16	11	169	152	152	131	374	202	3	1	1
Florida	3	9	9	3	3	3	29	161	94	4	0	0
E. SO. CEN.												
Kentucky	6	1	10	9	10	51	614	48	108	5	0	3
Tennessee	4	6	10	112	63	168	96	55	64	6	2	2
Alabama	2	6	5	227	659	536	38	198	284	20	1	2
Mississippi [2]	4	3	4	------	------	------	------	------	------	4	0	2
W. SO. CEN.												
Arkansas	13	6	8	166	293	293	146	270	112	3	1	1
Louisiana	8	3	5	7	23	44	92	112	15	11	0	1
Oklahoma	8	4	8	164	100	284	87	321	60	1	2	1
Texas	40	62	51	1,923	1,818	1,818	324	2,025	270	16	1	2
MOUNTAIN												
Montana	2	6	1	51	14	14	160	96	28	0	0	0
Idaho	0	0	1	2	------	------	402	37	37	1	0	0
Wyoming	0	0	1	53	227	4	69	62	14	1	1	0
Colorado	9	9	9	55	29	29	287	128	85	1	0	0
New Mexico	4	1	2	5	1	7	28	55	55	2	0	0
Arizona	0	0	2	155	153	168	18	151	13	3	0	0
Utah [2]	0	0	0	314	------	24	360	44	81	9	0	0
Nevada	0	0	------	1	------	------	18	15	------	0	0	------
PACIFIC												
Washington	2	1	2	1	------	4	754	41	70	15	0	0
Oregon	1	1	2	15	18	54	276	94	94	11	0	0
California	62	13	22	99	137	137	372	2,317	433	33	4	1
Total	279	305	378	5,376	5,180	5,180	12,803	14,062	14,062	[3] 446	42	46
6 weeks	1,959	2,109	2,628	27,124	27,772	27,772	62,348	64,741	64,741	2,058	332	323

See footnotes at end of table.

Telegraphic morbidity reports from State health officers for the week ended February 13, 1943, and comparison with corresponding week of 1942 and 5-year median—Con.

Division and State	Poliomyelitis			Scarlet fever			Smallpox			Typhoid and paratyphoid fever		
	Week ended		Median 1938–42	Week ended		Median 1938–42	Week ended		Median 1938–42	Week ended		Median 1938–42
	Feb. 13, 1943	Feb. 14, 1942		Feb. 13, 1943	Feb. 14, 1942		Feb. 13, 1943	Feb. 14, 1942		Feb. 13, 1943	Feb. 14, 1942	
NEW ENG.												
Maine	0	0	0	1	31	19	0	0	0	0	0	0
New Hampshire	0	0	0	9	13	7	0	0	0	0	0	0
Vermont	0	0	0	18	6	8	0	0	0	0	0	0
Massachusetts	0	0	0	506	374	255	0	0	0	2	0	1
Rhode Island	0	0	0	21	8	10	0	0	0	0	0	0
Connecticut	0	2	0	59	48	90	0	0	0	0	0	0
MID. ATL.												
New York	0	2	2	473	388	647	0	0	0	0	9	5
New Jersey	1	1	0	130	147	172	0	0	0	0	0	0
Pennsylvania	0	0	0	303	367	370	0	0	0	6	5	5
E. NO. CEN.												
Ohio	0	0	0	285	306	306	0	0	5	1	4	1
Indiana	0	0	0	97	160	188	9	0	6	0	2	2
Illinois	1	0	0	186	231	518	1	0	2	1	2	2
Michigan [1]	2	1	1	150	253	261	0	0	6	2	1	1
Wisconsin	1	1	1	256	208	206	0	0	5	0	3	1
W. NO. CEN.												
Minnesota	0	0	0	40	93	112	0	0	5	0	0	0
Iowa	2	0	0	83	49	70	0	1	9	1	0	0
Missouri	0	0	0	97	62	91	0	2	3	0	4	1
North Dakota	0	0	0	15	27	16	0	0	0	0	0	0
South Dakota	1	0	0	19	35	23	0	0	1	1	0	0
Nebraska	1	0	0	31	32	32	1	0	0	0	0	0
Kansas	0	0	0	84	98	98	2	0	1	0	0	0
SO. ATL.												
Delaware	0	0	0	8	65	10	0	0	0	1	0	0
Maryland [2]	0	0	0	81	88	82	0	0	0	0	0	1
Dist. of Col	0	0	0	28	12	15	0	0	0	1	1	1
Virginia	2	0	0	37	46	47	0	0	0	2	1	1
West Virginia	1	0	0	39	37	50	0	0	0	0	2	2
North Carolina	0	0	0	41	42	50	0	0	0	2	1	2
South Carolina	0	0	0	16	6	6	1	0	0	0	2	3
Georgia	1	0	0	31	27	22	1	0	0	3	28	3
Florida	3	0	0	11	17	11	0	0	0	0	5	2
E. SO. CEN.												
Kentucky	1	0	0	65	78	78	0	0	0	1	0	3
Tennessee	1	3	0	48	42	54	0	0	1	0	3	3
Alabama	1	1	1	25	24	22	0	0	0	2	0	3
Mississippi [2]	0	0	1	16	4	5	0	2	1	2	1	0
W. SO. CEN.												
Arkansas	1	0	0	7	6	9	4	1	2	2	2	2
Louisiana	0	1	0	6	4	7	0	0	0	3	4	4
Oklahoma	0	0	0	32	26	31	5	1	1	2	1	1
Texas	4	1	1	62	43	75	4	21	21	2	6	8
MOUNTAIN												
Montana	0	0	0	17	28	28	0	0	0	1	0	0
Idaho	0	0	0	18	2	17	0	0	1	0	0	0
Wyoming	0	0	0	54	20	8	0	0	0	0	0	0
Colorado	0	0	0	23	32	37	0	0	6	0	0	0
New Mexico	0	0	0	5	8	13	0	0	0	1	1	3
Arizona	0	0	0	9	14	13	0	0	1	0	0	0
Utah [2]	0	0	0	75	57	31	0	0	0	2	0	0
Nevada	0	0	--	5	0	-------	0	0	------	0	0	------
PACIFIC												
Washington	0	0	0	33	45	59	0	0	2	0	1	1
Oregon	1	0	0	9	11	22	0	0	0	0	0	1
California	5	3	2	159	92	140	0	1	1	2	4	5
Total	30	16	18	3,823	3,812	4,595	28	29	63	43	93	93
6 weeks	194	154	154	22,010	21,857	25,951	183	113	453	292	493	493

See footnotes at end of table.

Telegraphic morbidity reports from State health officers for the week ended February 13, 1943, and comparison with corresponding week of 1942 and 5-year median—Con.

Division and State	Whooping cough			Week ended February 13, 1943								
	Week ended		Median 1938–42	Anthrax	Dysentery			Encephalitis, infectious	Leprosy	Rocky Mt. spotted fever	Tularemia	Typhus fever
	Feb. 13, 1943	Feb. 14, 1942			Amebic	Bacillary	Unspecified					
NEW ENG.												
Maine	40	37	61	0	0	0	0	0	0	0	0	0
New Hampshire	12	28	4	0	0	0	0	0	0	0	0	0
Vermont	11	53	26	0	0	0	0	0	0	0	0	0
Massachusetts	151	220	220	0	0	6	0	0	0	0	0	0
Rhode Island	21	44	25	0	0	0	0	0	0	0	0	0
Connecticut	52	113	64	0	0	0	0	0	0	0	0	0
MID ATL.												
New York	328	417	394	0	0	146	0	1	0	0	0	0
New Jersey	145	195	158	0	0	0	0	0	0	0	0	0
Pennsylvania	319	214	341	2	1	0	0	0	0	0	0	0
E. NO. CEN.												
Ohio	194	240	216	0	0	0	0	0	0	0	1	0
Indiana	24	65	19	0	0	0	0	1	0	0	1	0
Illinois	174	158	107	0	0	2	0	1	0	0	0	0
Michigan [2]	351	192	189	0	0	1	0	0	0	0	0	0
Wisconsin	182	232	152	0	0	0	0	1	0	0	0	0
W. NO. CEN.												
Minnesota	71	53	53	0	1	6	0	0	0	0	0	0
Iowa	20	24	24	0	0	0	0	0	0	0	0	0
Missouri	20	28	28	0	0	0	0	0	0	0	0	0
North Dakota	3	13	15	0	0	0	0	0	0	0	1	0
South Dakota	3	11	11	0	0	0	0	0	0	0	0	0
Nebraska	10	17	10	0	0	0	0	0	0	0	0	0
Kansas	39	55	55	0	0	0	0	0	0	0	0	0
SO. ATL.												
Delaware	0	1	7	0	0	0	0	0	0	0	0	0
Maryland [2]	60	53	71	0	0	0	1	0	0	0	0	0
Dist. of Col	17	38	15	0	0	0	0	0	0	0	0	0
Virginia	149	70	70	0	0	0	36	0	0	0	0	0
West Virginia	90	46	43	0	0	0	0	0	0	0	0	0
North Carolina	92	148	302	0	1	0	0	0	0	0	0	4
South Carolina	24	45	61	0	0	0	0	0	0	0	0	1
Georgia	24	37	37	0	0	2	2	0	0	0	2	5
Florida	15	16	17	0	0	0	0	0	0	0	0	10
E. SO. CEN.												
Kentucky	38	84	60	0	0	0	0	1	0	0	0	0
Tennessee	83	26	41	0	0	0	0	0	0	0	6	0
Alabama	15	37	21	0	0	0	0	0	0	0	0	2
Mississippi [2]	----	----	----	0	0	0	0	0	0	0	1	0
W. SO. CEN.												
Arkansas	63	11	11	0	0	3	0	0	0	0	0	0
Louisiana	8	3	9	0	2	1	0	0	0	0	0	2
Oklahoma	16	6	6	0	0	0	0	0	0	0	0	0
Texas	382	172	172	0	8	128	0	1	0	0	0	21
MOUNTAIN												
Montana	33	21	13	0	0	0	0	0	0	0	0	0
Idaho	3	11	8	0	0	0	0	0	0	0	0	0
Wyoming	0	3	3	0	0	0	0	0	0	0	0	0
Colorado	15	36	36	0	1	0	0	0	0	0	0	0
New Mexico	22	53	37	0	0	0	0	0	0	0	0	0
Arizona	16	56	26	0	0	0	21	0	0	0	0	0
Utah [3]	33	40	46	0	0	0	0	0	0	0	0	0
Nevada	1	8	----	0	0	0	0	0	0	0	0	0
PACIFIC												
Washington	27	111	111	0	0	0	0	0	0	0	0	0
Oregon	5	31	28	0	0	0	0	0	0	0	0	0
California	269	244	244	0	1	8	0	2	0	0	0	0
Total	3,670	3,816	3,958	2	15	303	60	8	0	0	12	45
6 weeks	23,409	25,517	25,517	----	----	----	----	----	----	----	----	----

[1] New York City only.
[2] Period ended earlier than Saturday.
[3] Inclusive of delayed report of 43 cases in Virginia.

WEEKLY REPORTS FROM CITIES

City reports for week ended January 30, 1943

This table lists the reports from 86 cities of more than 10,000 population distributed throughout the United States, and represents a cross section of the current urban incidence of the diseases included in the table.

	Diphtheria cases	Encephalitis, infectious, cases	Influenza		Measles cases	Meningitis, meningococcus, cases	Pneumonia deaths	Poliomyelitis cases	Scarlet fever cases	Smallpox cases	Typhoid and paratyphoid fever cases	Whooping cough cases
			Cases	Deaths								
Atlanta, Ga.	0	0	25	0	10	0	2	0	6	0	0	1
Baltimore, Md.	8	0	5	3	5	15	17	0	37	0	0	60
Barre, Vt.	0	0	------	0	0	0	0	0	0	0	0	0
Billings, Mont.	0	0	------	0	0	0	1	0	0	0	0	3
Birmingham, Ala.	0	0	4	1	0	0	7	0	1	0	0	1
Boise, Idaho	0	0	------	0	0	0	0	0	0	0	0	0
Boston, Mass.	1	0	------	1	143	5	18	2	111	0	0	37
Bridgeport, Conn.	0	0	------	0	1	2	0	0	9	0	0	0
Brunswick, Ga.	0	0	------	0	0	0	0	0	0	0	0	2
Buffalo, N. Y.	0	0	------	3	80	1	11	0	12	0	0	23
Camden, N. J.	1	0	1	1	1	2	0	0	2	0	0	3
Charleston, S. C.	1	0	86	0	1	2	6	0	3	0	0	0
Charleston, W. Va.	0	0	------	0	0	0	0	0	0	0	0	0
Chicago, Ill.	7	0	4	3	176	4	42	0	58	0	1	71
Cincinnati, Ohio	1	0	------	1	25	1	8	0	45	0	1	4
Cleveland, Ohio	1	0	7	0	6	1	8	0	45	0	0	72
Columbus, Ohio	0	0	------	0	6	0	4	0	14	0	0	3
Concord, N. H.	0	0	------	0	0	0	1	0	2	0	0	0
Cumberland, Md.	0	0	------	0	0	0	0	0	0	0	0	0
Dallas, Tex.	1	0	------	1	1	1	7	0	2	0	0	9
Denver, Colo.	6	0	45	1	106	1	6	0	10	0	0	7
Duluth, Minn.	0	0	------	0	0	0	1	0	3	0	0	2
Fall River, Mass.	2	0	------	0	0	0	1	0	11	0	0	13
Fargo, N. Dak.	0	0	------	0	0	0	0	0	5	0	0	1
Flint, Mich.	0	0	------	0	1	0	3	0	3	0	0	0
Fort Wayne, Ind.	0	0	------	0	0	0	2	0	0	0	0	0
Frederick, Md.	0	0	------	0	0	0	0	0	0	0	0	0
Galveston, Tex.	0	0	------	0	0	0	2	0	1	0	0	3
Grand Rapids, Mich.	0	0	------	0	0	2	3	0	3	0	0	4
Great Falls, Mont.	0	0	------	0	8	0	1	0	1	0	0	15
Hartford, Conn.	0	0	------	0	18	0	7	0	0	0	0	3
Helena, Mont.	0	0	------	0	3	0	0	0	0	0	0	0
Houston, Tex.	6	0	------	1	2	0	13	0	7	0	0	4
Indianapolis, Ind.	2	0	------	1	169	1	12	0	32	0	0	29
Kansas City, Mo.	0	0	------	2	15	0	10	0	39	0	0	3
Kenosha, Wis.	0	0	------	0	1	0	0	0	2	0	0	0
Little Rock, Ark.	0	0	------	0	1	0	2	0	1	0	0	0
Los Angeles, Calif.	3	0	26	2	38	1	6	0	27	0	1	33
Lynchburg, Va.	1	0	------	0	1	0	1	0	1	0	0	3
Memphis, Tenn.	0	0	4	0	0	1	7	0	7	0	0	7
Milwaukee, Wis.	0	0	------	0	104	0	5	0	86	0	0	48
Minneapolis, Minn.	0	0	------	0	9	0	8	0	14	0	0	9
Missoula, Mont.	0	0	------	0	0	0	0	0	0	0	0	0
Mobile, Ala.	0	0	1	1	1	2	1	0	2	0	0	0
Nashville, Tenn.	0	0	------	1	26	0	4	0	5	0	0	0
Newark, N. J.	0	0	9	0	12	1	6	0	16	0	0	5
New Haven, Conn.	0	0	5	0	1	1	3	0	3	0	0	9
New Orleans, La.	0	0	1	0	6	1	12	0	9	0	1	0
New York, N. Y.	11	0	14	3	91	18	99	1	235	0	2	81
Omaha, Nebr.	0	0	------	1	4	0	4	0	6	0	0	3
Philadelphia, Pa.	0	0	2	2	1,241	2	49	0	105	0	0	79
Pittsburgh, Pa.	1	1	7	3	1	3	9	0	6	0	0	23
Portland, Maine.	1	0	------	0	3	6	8	0	0	0	0	33
Providence, R. I.	0	0	------	0	5	8	0	0	3	0	0	7
Pueblo, Colo.	0	0	------	0	2	0	2	0	3	0	0	4
Racine, Wis.	0	0	------	0	22	0	0	0	27	0	0	3
Raleigh, N. C.	0	0	------	0	0	0	1	0	0	0	0	3
Reading, Pa.	0	0	------	1	56	0	1	0	1	0	0	7
Richmond, Va.	0	0	2	1	5	2	9	0	4	0	0	5

City reports for week ended January 30, 1943—Continued

	Diphtheria cases	Encephalitis, infectious, cases	Influenza		Measles cases	Meningitis, meningococcus, cases	Pneumonia deaths	Poliomyelitis cases	Scarlet fever cases	Smallpox cases	Typhoid and paratyphoid fever cases	Whooping cough cases
			Cases	Deaths								
Roanoke, Va	0	0	------	0	0	0	1	0	0	0	0	0
Rochester, N. Y	0	0	------	1	9	2	4	0	8	0	0	18
Sacramento, Calif	3	0	------	0	6	0	3	0	2	0	0	4
St. Joseph, Mo	0	0	------	1	0	0	5	0	0	0	0	0
St. Louis, Mo	2	0	------	0	10	4	15	0	22	0	0	12
St. Paul, Minn	0	0	------	0	0	0	3	0	3	0	0	52
San Antonio, Tex	2	0	3	3	2	0	10	0	1	0	0	1
Savannah, Ga	0	0	43	3	1	1	4	0	1	0	0	1
Seattle, Wash	2	0	------	2	54	1	11	0	5	0	0	0
Shreveport, La	0	0	------	0	0	0	4	0	1	0	1	0
South Bend, Ind	0	0	------	0	1	0	2	0	2	0	0	1
Spokane, Wash	0	0	1	1	135	0	0	0	1	0	1	0
Springfield, Mass	0	0	------	0	12	0	4	0	94	0	0	1
Superior, Wis	0	0	------	0	2	0	1	0	0	0	0	12
Syracuse, N. Y	0	0	------	0	5	1	4	0	16	0	0	11
Tacoma, Wash	3	0	------	0	33	2	3	0	1	0	0	1
Tampa, Fla	0	0	------	0	0	1	1	0	0	0	0	0
Terre Haute, Ind	0	0	------	0	0	1	4	0	3	0	0	1
Topeka, Kans	0	0	------	0	19	0	2	0	0	0	0	0
Trenton, N. J	0	0	------	0	6	0	1	0	8	0	0	0
Washington, D. C	1	0	4	1	51	4	15	0	29	0	1	10
Wheeling, W. Va	0	0	------	0	0	0	2	0	0	0	0	13
Wichita, Kans	1	0	1	0	3	0	7	0	2	0	0	3
Wilmington, Del	0	0	------	0	2	0	5	0	2	0	0	2
Wilmington, N. C	0	0	------	0	10	0	2	0	0	0	0	8
Winston-Salem, N. C	2	0	------	0	0	0	0	0	1	0	0	7
Worcester, Mass	0	0	------	0	65	0	11	0	16	0	0	7
Total	70	1	300	43	2,830	101	554	3	1,243	0	9	900
Corresponding week 1942	79	3	435	40	1,859	24	445	1	1,125	0	16	1,254
Average, 1938–42	112	------	[1] 1,541	[1] 99	[2] 3,539	------	[1] 602	------	1,160	25	18	1,002

[1] 3-year average, 1940–42. [2] 5-year median.

Dysentery, amebic.—Cases: New York, 3.
Dysentery, bacillary.—Cases: Los Angeles, 7; New York, 7; St. Louis, 1.
Leprosy.—Cases: New Orleans, 1; New York, 2.
Typhus fever.—Cases: Atlanta, 2; Brunswick, 1; Charleston, S. C. 1; Galveston, 1; Nashville, 2; Savannah, 1.

TERRITORIES AND POSSESSIONS

Hawaii Territory

Plague (rodent).—During the 2 weeks ended January 23, 1943, 1 rat proved positive for plague was reported in Honokaa, Hamakua District, Island of Hawaii, T. H.

Virgin Islands of the United States

Notifiable diseases—October–December 1942.—During the months of October, November, and December, 1942, cases of certain notifiable diseases were reported in the Virgin Islands as follows:

Disease	October	November	December	Disease	October	November	December
Filariasis	3	4	12	Pneumonia	------	------	1
Gonorrhea	15	6	5	Schistosomiasis	1	------	------
Hookworm disease	2	6	------	Syphilis	22	17	17
Leprosy	1	------	------	Tuberculosis	1	1	3
Malaria	2	5	1	Typhus fever	1	------	------
Mumps	3	1	1	Whooping cough	6	471	140
Pellagra	------	1	------				

FOREIGN REPORTS

CANADA

Provinces—Communicable diseases—Week ended January 16, 1943.—During the week ended January 16, 1943, cases of certain communicable diseases were reported by the Dominion Bureau of Statistics of Canada as follows:

Disease	Prince Edward Island	Nova Scotia	New Brunswick	Quebec	Ontario	Manitoba	Saskatchewan	Alberta	British Columbia	Total
Chickenpox		16		245	535	91	56	53	60	1,056
Diphtheria	1	10	3	26	4	8				52
Dysentery (amebic)					1					1
Dysentery (bacillary)				3						3
Encephalitis, infectious									1	1
German measles				3	12		2	4	8	29
Influenza		27			62	7			17	113
Measles		21	4	79	122	29	30	7	47	339
Meningitis, meningococcus			2	1	4				1	8
Mumps	1	71	1	31	1,119	119	81	110	195	1,728
Poliomyelitis				1		1				2
Scarlet fever		5	3	116	141	16	20	20	32	353
Tuberculosis (all forms)	2	3	3	52	48	13	1	12	25	159
Typhoid and paratyphoid fever		1		8	1					10
Undulant fever				1						1
Whooping cough		9		116	128	38	6	39	15	351

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

NOTE.—Except in cases of unusual prevalence, only those places are included which had not previously reported any of the above-mentioned diseases, except yellow fever, during the current year. All reports of yellow fever are published currently.

A cumulative table showing the reported prevalence of these diseases for the year to date is published in the PUBLIC HEALTH REPORTS for the last Friday in each month.

(Few reports are available from the invaded countries of Europe and other nations in war zones.)

Plague

Morocco—Casablanca.—During the week ended January 9, 1943, 3 cases of plague with 2 deaths were reported in Casablanca, Morocco.

Palestine—Jaffa.—During the week ended January 9, 1943, 2 cases of plague were reported in Jaffa, Palestine.

Typhus Fever

Bulgaria.—For the period January 1–13, 1943, 99 cases of typhus fever were reported in Bulgaria.

China—Shanghai.—For the period October 25 to November 21, 1942, 40 cases of typhus fever were reported in Shanghai, China.

Hungary.—For the week ended January 23, 1943, 20 cases of typhus fever were reported in Hungary.