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SKIN HAZARDS IN AIRPLANE MANUFACTURE

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This report is based on studies made in nine airplane factories located in various parts of the United States, employing over 100,000 workers, and making various types of aircraft—training planes, commercial planes, and all types of fighting planes and bombers. Four of these plants had reported 117 cases of dermatitis to the Compensation Commission during the preceding 15 months. Records were not available from the other five plants. Thirty-six active cases of dermatitis were seen during the course of our inspection, 24 of which were definitely of occupational origin.

The three principal parts of an airplane, (1) the motor, (2) fuselage, wings, and controls, and (3) the flying instruments, are made in different factories but are all assembled into the complete machine at the factory where the fuselage, wings, and controls are built, and where our studies were made.

PROCESS

The airplane consists essentially of a chassis or framework of stainless steel metal tubing, the parts welded together somewhat as the framework of a bicycle. Around this framework is built the fuselage, and to the fuselage are attached the wings, the controls, and the motor.

The fuselage is made of sheets of duraluminum alloy called "dural". The sheets of metal are molded into proper shape by presses or by hand and the parts are riveted or welded together. In most of the larger planes the wings, tail, and controls are also made of aluminum alloy, the composition of which may vary somewhat according to the part of the plane into which it goes. In most small planes, and some larger ones, the wings and controls consist of a framework of metal over which is drawn a fabric covering made impervious by the application of "dope." "Dope" consists essentially of cellulose acetate, or nitrate, dissolved in a solvent such as acetone, amyl acetate, etc.

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Other alloys of aluminum, such as Alclad and Dow Metal, are used in various parts of the plane.

The rivets, screws, nuts, and bolts which are used where welding cannot be done, are made of iron or steel, made rustproof by cadmium plating or by anodizing.

The various parts of the plane, when completed, are assembled on the assembling line in a manner similar to that of an automobile, but the line moves very much more slowly.

Dural, an alloy consisting of aluminum, copper, magnesium, manganese, and iron, Alclad, containing the same materials but in different proportions, and Dow Metal, an alloy of magnesium, manganese, silica, and aluminum, come into the factory in large sheets, several sheets being crated together in wooden crates for shipment. Some of the sheets are heavily coated with an oil called "fish oil", the composition of which is said to consist of 96 percent highly refined Mid-Continent Neutral Oils to which is added a 4 percent winter pressed fish oil.[1] In one large factory, many workers exposed to this oil developed a hypersensitivity to it as shown by patch tests, and dermatitis of the forearms, face, and other exposed parts ensued.[2] None of the workers uncrating the metal from the wooden crates were affected, perhaps because they all wore gloves and coveralls, primarily as protection against cuts while lifting the thin, sharp-edged sheets, but which evidently also served to prevent contact of the skin with the oil. Workers who handled cut sheets before the oil was washed off did not wear such protective clothing and they were more or less constantly in contact with the oil. It was in this group of workers that dermatitis caused by "fish oil" was observed (fig. 1).

We have had reports of dermatitis occurring among workers in a motor manufacturing plant exposed to an oil sprayed on metal parts to prevent corrosion. This oil also has a fishy odor, and was thought to be "fish oil," but inquiry from the manufacturer revealed that it consisted of fatty oils derived from rapeseed, lard, and rice bran, plus butyl alcohol, and about 3 percent of a tertiary phenolic amine, a condensation product of dimethylamine with phenol in the presence of formaldehyde. This tertiary phenolic amine is said to neutralize free acid, thus preventing rancidity of the fatty oils and also preventing corroding of the metal. The tertiary amine has the fishy odor characteristic of the methylamines, and this may lead one to think that there is fish oil in the compound. The tertiary phenolic amine is also an irritant and sensitizer if allowed to remain on the skin for a considerable length of time. This, or another inhibitor or noncorro-

[1] An oil of animal or vegetable origin is added to the petroleum oil in order to make it adhere to the metal.

[2] Lounsberry, C. Ray: Occupational dermatoses in the aircraft industry. California and West. Med., 51:309-313 (1939).

sive with similar action that may be in the winter pressed fish oil, is more likely to be the actual cause of the dermatitis occurring among the workers exposed to the so-called fish oil on the dural sheets than any animal, vegetable, or fish oil which it may actually contain, as such oils are usually harmless.[3]

In some factories, the dural sheets when received were covered with a coat of varnish which was applied to them by the makers in order to protect them from scratches and cuts in shipping and handling. Some workers were found who had dermatitis caused by sensitivity to this varnish. Sheets of uncoated dural were also received in these factories and in some places the sheets, after being uncrated, were dipped into a tank containing a preparation called "line oil" in order to protect the surface from scratches in the process of manufacture. "Line oil" consists of soybean oil and spent varnish containing various resins dissolved in Stoddart solvent (a petroleum distillate). The sheets, after being dipped into the tank of "line oil", are lifted out, allowed to drip and dry, leaving a dry surface coating consisting of the oil and resin. The workers at this job had their arms, hands, and clothes soiled with the "line oil" and a number of cases of dermatitis have resulted. This dermatitis may be due to the defatting of the skin by the Stoddard solvent or it may be caused by the development of allergy to the resins or the soybean oil. There were no cases of dermatitis found in any of the factories that could be attributed to the uncoated aluminum or magnesium alloys. Therefore, it is believed that the so-called "dural poisoning" in airplane factories is not caused by the alloy itself, but by the oils, varnishes, and paints which are applied to its surface.

Dermatitis among this group of workers can be prevented by having all sheets of oiled or varnished alloy which come into the factory washed clean of coating before permitting them to be handled by the workers. The men employed at cleaning the coating from the sheets should wear protective clothing such as rubber boots and gloves, aprons, and sleeves made of impervious material such as the synthetic resins, pliofilm, koroseal, vinylite, etc. Men exposed to "line oil" should be similarly protected.

Since there is no particular starting point in airplane manufacture, the various processes in which skin hazards occur will be described in alphabetical order.

ANODIZING DEPARTMENT

Anodizing imparts to the metal a dull gray finish which is rustproof and tarnishproof. Before anodizing, all dirt, grease, and scale must be removed from the surface of the metal. This is done by immersing

[3] The makers deny the presence of an inhibitor in this oil.

in acids and solvents and washing in a hot alkali solution, then in water, then again in a solution of soap (Kelite), again rinsing in water, and then immersing in the anodizing tank. The anodizing tank contains a solution of chromic acid and dichromates. Although most of the dipping of the metal parts into the various solutions is done by mechanical means and the anodizing tank is vented and usually kept closed when not in use, splashes from the tanks containing the cleaning solutions and fumes of chromic acid and dichromates from the anodizing tank may affect the worker, causing dermatitis on the intact skin, and ulcers if the irritant liquid enters abrasions.

In some factories this department also contains tanks of nitric acid and hydrofluoric acid into which the metal is dipped. Dermatitis may occur from splashes and fumes of both these powerful acids.

Various coal tar and petroleum distillates as well as trichlorethylene are used in this department for degreasing metals and for removing paint from them before they are anodized. Dermatitis has occurred from exposure to these degreasers which are fat solvents and sensitizers. When the wet metal is lifted out of the anodizing tank and hung up to dry, workers handling it may develop ulcers and dermatitis from the chromic acid solution remaining on the metal as well as from the fine gray dust which coats the parts after they are dry. In addition to proper local exhaust ventilation over these tanks,[4 5 6] the workers in this department should be furnished with long rubber gauntlets over which they should wear sleeves of impervious material buttoned at the wrist. Aprons of a similar material would prevent the soiling of the clothes and dermatitis of the covered parts. Workers exposed to the fumes issuing from chromic acid and hydrofluoric acid tanks should insert vaseline into the nostrils several times a day in order to protect the nasal mucosa from the corrosive effect of these chemicals.

Because the various solvents used in this department act to defat the skin and thus cause drying, chapping, and chronic eczema, it is recommended that no strong soaps, bleaches, or solvents be used by the workers for cleaning the skin after work. A neutral sulfonated castor oil containing 2 percent of a wetting agent (such as Duponol, Aerosol, Santomerse, Naconol, or Igepon) should be provided for the workers, instead of soap, for cleansing the hands after work. Such a mixture, because of its vegetable oil content, will not defat the skin and yet will clean it. Workers with dry, chapped skins should also rub into the skin before and after work a mixture of an-

[4] Bloomfield, J. J., and Blum, W.: Health hazards in chromium plating. Pub. Health Rep., 43: 2330 (1928). Reprint No. 1245.

[5] Riley, E. C., and Goldman, F. H.: Control of chromic acid mists from plating tanks. Pub. Health Rep., 52: 172 (1937). Reprint No. 1801.

[6] Bloomfield, J. J.: Poisoning by chromium compounds. Safety Eng., 61: 222 (1931).

hydrous lanolin and olive oil. This will act to buffer the action of the fat solvents on the skin and will tend to replace whatever fats such solvents may remove from the skin.

DEGREASING

Degreasing tanks are usually located in the anodizing and cadmium plating departments. Some of them are large rectangular tanks with the surface of the solvent, usually trichlorethylene, about three feet below the top of the tank. A few inches above the level of the liquid, and running around the inside of the tank, there are cooling coils for the purpose of preventing the evaporation and escape of fumes. Some degreasing tanks are simple covered containers with no other safety appliances. Workers dipping metal parts into the tanks are exposed to the fumes and vapors which may escape, and to splashes when the metal parts enter the liquid, and drippings of the solvent as the metals are taken out. Trichlorethylene is a fat solvent and can cause a chronic dry, cracked, fissured eczema of the hands and arms. It is also a sensitizer and can cause a more or less generalized acute eczematoid type of dermatitis which begins as an erythema, becomes papular, then vesicular, and is followed by oozing, crusting, and desquamation.

All degreasing tanks should be so constructed that fumes cannot escape.[7] The workers should be protected against splashes and dripping by protective clothing. Rubber and the ordinary impervious films are attacked by trichlorethylene and carbon tetrachloride, the chemicals usually used for degreasing, but the polyvinyl alcohols are not. Aprons, sleeves, and gloves made of the polyvinyl alcohols can be obtained and should be used by workers on degreasing operations where the chlorinated hydrocarbons are used as degreasing solutions.[8]

To counteract the defatting action on the skin of these solvents, a skin cleanser and protective ointment should be used similar to that described under anodizing.

"DOPE" ROOM

Here the fabric parts of the plane (wings and controls) are fitted over the metal framework and made impervious by the application of the so-called "dopes". The "dopes" are applied by hand brushes. They consist essentially of a solution of cellulose nitrate or acetate in a volatile solvent such as acetone, amyl acetate, etc., which, after evaporating, leaves the fabric impregnated and coated with the cellulose compound, making it impervious. On entering this room, the

[7] Witheridge, W. N., and Walworth, H. T.: Ventilation of a trichlorethylene degreaser. J. Ind. Hyg. and Toxicol., 22: 175-187 (May 1940).

[8] "Resistoflex" is the trade name of a polyvinyl alcohol which will resist the action of the chlorinated hydrocarbons, but it will not resist the action of water and steam.

strong odor of solvents first irritates the nose, throat, and eyes, causing coughing and lacrimation, but after a few minutes these symptoms cease, probably because of the anesthetic action of these esters on the mucous membranes. Most of the "dope" rooms have exhaust ventilation of some kind, but in spite of this the "dope" permeates the air.

The "dopes" have a defatting action on the skin and can cause dry, chapped hands, and chronic, fissured eczemas of the hands and arms. A small percentage of workers become sensitized and develop acute eczematoid types of dermatitis of the hands, arms, face, and whatever other parts may be exposed to the solvents or their fumes (fig. 2).

Workers exposed to "dopes" should wear fabric-lined rubber gloves over which sleeves made of impervious fabrics should fasten at the wrist. Long aprons of the same materials will protect the clothes from being soiled. Workers who are hypersensitive should apply to the face, neck, and other exposed parts the protective ointment of lanolin and olive oil described above. Those workers with dry and defatted skin should use the mixture of sulfonated castor oil and wetting agent described above for a hand cleanser instead of the usual soaps or volatile solvents.

It was observed that some of the girls employed at sewing the fabric coverings of the wings and controls of airplanes developed blisters and irritation of the fingers from the long, sharp needles which they used. Many of them wore leather finger shields for protection. Such shields should be furnished to all engaged in this occupation.

DROP HAMMER DEPARTMENT

Many of the metal sheets are shaped to the desired form by placing them on molds and allowing hammers, the faces of which fit into the molds, to fall on them. The hammers are raised by hand ropes, and dermatitis of the hands has occurred from the mechanical action of friction in handling the ropes as well as from the oil and resin with which some of the ropes are treated in order to make them strong and serviceable. Petroleum oils and grease are used on the dies and metal plates in order to protect the metal, and the clothes often become soiled with oil and grease, resulting in folliculitis of the thighs and forearms, especially among workers who do not frequently change to clean clothes or who neglect cleaning their skin after work. To prevent these conditions, workers at the drop hammers should wear leather or canvas gloves and impervious sleeves and aprons.

The same hazards are present in the hydraulic press department where larger pieces of metal are molded in similar manner by hydraulic presses.

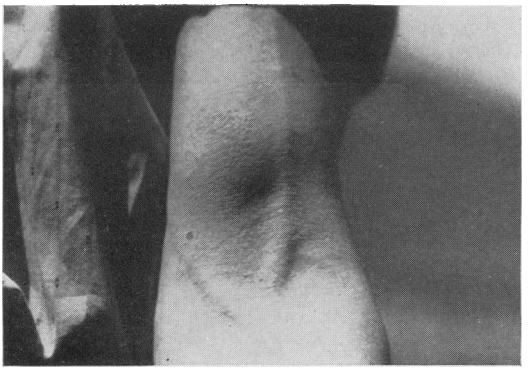

FIGURE 1.—Allergic dermatitis from "fish oil."

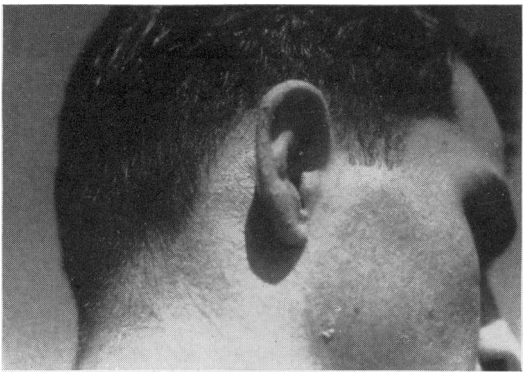

FIGURE 2.—Dermatitis caused by volatile solvents used in "dopes."

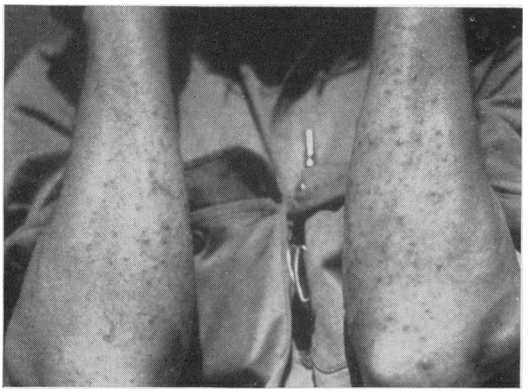

FIGURE 3.—Oil acne caused by cutting oils.

GAS TANKS

Gas tanks are made of molded dural plates welded together. The complete tanks are dipped in a vat containing a solution of potassium dichromate and nitric acid resulting in a grayish-green color on the surface of the tank which prevents rusting. The vat containing the potassium dichromate and nitric acid is an open one and workers should be protected against splashes and fumes by rubber gloves, impervious sleeves, and aprons, and by placing vaseline in the nostrils as described under anodizing.

A sealing compound is applied to the surface of the gas tank. This compound consists of zinc chromate, asbestos, mica, a synthetic resin, and a drying oil in a thinner such as ethyl acetate. Dermatitis may result among workers applying this sealing compound to the gas tanks. The solvent may cause dermatitis either by its fat solvent action or by its sensitizing action, and skin sensitivity to zinc chromate and synthetic resins has also been observed among these workers. Workers engaged in applying this paint to the gas tanks should wear the protective clothing described above under the discussion of the hazards in the "dope" room.

Gas tanks are enclosed in a covering of a rubber-like compound which is said to plug up bullet holes. The composition of this substance was not learned, but it is soft and doughy, resembling unvulcanized synthetic rubber. No cases of dermatitis were noted from handling this substance. A leather casing encloses the gas tank and its covering.

HEAT TREATMENT

Heat treatment increases the strength of the metal parts. Some of the parts are dipped in a tank containing molten sodium nitrite to which a small percentage of sodium dichromate has been added. The tank is covered with a lid, but when the lid is lifted, fumes of nitrous and chrome compounds are given off and may irritate the nose of workers standing over the tank. Therefore, in addition to exhaust vents over the lid, workers around this tank should insert vaseline into the nostrils to prevent nasal mucitis. Gloves are indispensable because of handling hot metal, but impervious sleeves and aprons should also be worn because the fumes may irritate the skin, causing dermatitis and ulcers.

Some of the metal is treated by immersion in molten sodium cyanide. The vessels containing the molten sodium cyanide usually have over them exhaust vents to prevent the fumes of hydrocyanic acid from coming in contact with the workers. Nevertheless, vaseline in the nostrils insures the protection of the nasal septum from whatever cyanide fumes faulty vents may allow to escape. Gloves and imper-

vious sleeves and aprons should be worn to protect the skin from the corrosive action of cyanide.

HYDROFLUORIC ACID

Tanks containing hydrofluoric acid are located in some of the departments. Into them are dipped metal parts for etching and also for coating with fluoride so that they can be "spot welded." Burns of the skin and ulceration of the nasal septum are hazards to workers dipping metal into these tanks.[9] Tanks of hydrofluoric acid should be kept covered and vented so that the workers are not exposed to fumes and splashes of this corrosive liquid. The workers should insert vaseline into the nostrils several times a day to prevent nasal mucitis, and should wear sleeves, aprons, and gloves of impervious material.

MACHINE SHOP

The metal parts are cut and drilled in the machine shop. The lathes and other machines are lubricated by oils both soluble and insoluble. The soluble oils consist chiefly of sulfonated mineral oils which are miscible with water, forming a milky emulsion, and are used mainly to cool the cutting tools, although they also facilitate the actual cutting operation. The chief function of the insoluble oils is to make cutting easier and to save the cutting edges of the lathes, drills, etc., but the insoluble oils also act as cooling agents. The insoluble oils are composed of mixtures of mineral oils to which are added animal and vegetable oils, as for instance, the so-called lard oil which consists of a mixture of mineral oil and lard oil. When animal and vegetable oils are contained in cutting oils, the manufacturers of the oils usually add a small amount of preservative in order to prevent the oil from becoming rancid. Such preservative must be antacid and noncorrosive. The oils circulate in the machines, being filtered in the course of circulation in order to screen out metal chips and dirt. In some factories the oil in the machines is changed once a week, the used oil being thrown away. In other factories the change is made at irregular intervals whenever the worker thinks it should be, and the used oil may be reclaimed or thrown away. In still other factories, the oil is rarely changed, additional antiseptic being added to it from time to time as it becomes rancid. In the latter case, oil has at times been found to contain as high as 10 percent of the phenolic compound usually used as an antiseptic.

When used oil is reclaimed, it is usually filtered and centrifuged in order to remove metal chips and dirt; it is then heat-sterilized and reused.

[9] The burns resulting from hydrofluoric acid are not felt until several hours after contact. The resulting ulcers are painful, and show but little healing tendencies. Curetting the base, followed by aseptic dressings, is required to heal them.

In most airplane factories, the workers are provided with as many clean wiping cloths as they may require. The factory has these cloths washed and the metal chips removed; they are then used again by the workers.

Dermatitis is of frequent occurrence in the machine shop. It may be caused by metal slivers cutting the skin and the subsequent development of infections resulting in boils. Since some workers spit into the oil reservoir of the machines, and since even *Bacillus coli* has been isolated from cutting oils, it cannot be said that cutting oils do not contain bacteria, but they do not contain more bacteria than is usually found on the skin. Infection is more likely to occur from bacteria present on the skin than from bacteria in the oil, because pure mineral oils are unsuitable culture mediums for bacteria, and those cutting oils that contain animal or vegetable oils also contain antiseptics.

Folliculitis on the extensor surfaces of the forearms and the thighs is a frequent form of dermatitis from cutting oils. It is usually caused by long contact of the skin with oil-soaked sleeves and trousers. The oil plugs up the follicles of the skin causing comedones and acne, and secondary infection may follow, causing either folliculitis or boils (fig. 3).

Occasionally a worker becomes allergic to something in the cutting oils and develops an eczematoid type of dermatitis. Antiseptics and preservatives in cutting oils are sensitizers and may be the actual causes of the eczematoid type of cutting oil dermatitis. Some workers exposed to cutting oils and greases develop small, flat, brown, slightly elevated papillomata on the dorsum of the hands and forearms.

Those cutting oils which are composed principally of mineral oil also have a defatting action on the skin, tending to make it dry and chapped.

To prevent dermatitis from cutting oils, the oils should be frequently changed and no additional antiseptics or preservatives should be added to them. If they are reclaimed they should be filtered, neutralized, and heat sterilized. The workers should have daily changes of clean working clothes and should wear sleeves, aprons, and coveralls of impervious material to prevent soiling of the clothes. Showers should be provided for cleaning up after work. The workers should be supplied with clean wiping cloths, free from metal chips. Those who have chapped, dry skins should rub into the hands before and after work an ointment consisting of lanolin and olive oil. This ointment acts to fill the pores and prevent the entrance of the irritating cutting oil. If a wetting agent is incorporated in the lanolin-olive oil mixture, it will aid in emulsifying it and removing it from the skin after work. The use of strong soaps, solvents, and bleaches for skin cleansing pur-

poses should be prohibited. The sulfonated castor oil-wetting agent mixture described above is recommended as a substitute.

The tooling department, in which jigs and dies are made, also uses cutting oils and the workers are subject to the same hazards as in the machine shop. In addition to this, the workers in the jig department also come in contact with painted metal parts both dry and wet and dermatitis from zinc chromate paint was found among them.

The routers cut flat metal sheets into the desired shape by guiding electric cutting tools, fixed to a movable arm, along the edges of a pattern fastened on top of the sheets of the metal. The routers are sprayed with the cutting oil thrown from the cutting tool. Their clothes become saturated with this oil and cutting oil dermatitis has been found among them. Routers should also observe the precautions given above against cutting oil dermatitis.

MAGNETIC INSPECTION

Bolts and screws are inspected for cracks and other flaws by dipping them in kerosene containing filings of iron oxide. They are then placed in a machine called a magnescope. When the current is turned on, the particles of iron are deposited in whatever cracks there may be in the metal piece that is being tested, thus revealing the flaws. The parts that have been inspected and found to be flawless are then washed in Stoddart solvent and, after drying, are dropped in a dye solution consisting of methyl violet in wood alcohol which dyes them and shows that they have been inspected and found to be perfect.

Dermatitis may occur in the magnetic inspection department from the defatting action on the skin of the kerosene and the Stoddart solvent. Allergic dermatitis may also be caused by methyl violet [10] although no cases were found in this study. There is also a hazard of poisoning from the fumes of wood alcohol given off by the uncovered tank of dye solution. Workers engaged in magnetic inspection should wear impervious gloves, sleeves, and aprons. Ordinary rubber gloves are easily affected by the petroleum distillates. Therefore, it is recommended that the gloves be made of synthetic rubber, which is less easily affected, or of polyvinyl alcohol (Resistoflex) which is not affected at all by petroleum solvents. Sleeves and aprons of impervious material should also be worn.

PAINT SHOP

Painting is usually done by the spray method in booths, and the back wall of the booth has an air exhaust appliance to pull the fumes of the thinner and solvents away from the worker. In some of the paint shops there is a continuous sheet of water flowing along the

[10] Schwartz, Louis: Skin Hazards in American Industry. Part I, Pub. Health Bulletin No. 215, U. S. Government Printing Office, 1937. Page 23.

entire back wall, back of which the exhaust fan is located so that the fumes are pulled through the sheet of water which collects some of the ingredients of the paint. In some of the factories, these are recovered from the water. In spite of the exhaust ventilation, there is a strong odor of the thinners and solvents in the paint shop. Toluol, turpentine, and petroleum distillates are the principal thinners used in the paints. The pigment is usually zinc chromate. The paint also contains a drying oil and a resin. Dermatitis may result from the defatting and sensitizing actions of the thinner on the skin and from hypersensitivity to the resin and the zinc chromate. The workers in the paint department should wear gloves, sleeves, and aprons made of an impervious material, such as described under magnetic inspection, which is not affected by the thinner and solvents. They should also be furnished with an ointment consisting of anhydrous lanolin and olive oil to rub into the skin before and after work. Those workers whose skins already show the drying, cracking effect of these solvents should be provided with the sulfonated castor oil-wetting agent mixture, described under anodizing, for washing the hands instead of the ordinary soaps and cleansers.

PASSIVATING

The tanks containing nitric acid solution in which this operation is performed should be closed and vented and the workers around it should wear impervious sleeves, aprons, and gloves to protect them from the fumes of nitric acid.

PLANISHING

Planishing consists in smoothing dents from small metal parts by means of electric hammers. Oil and grease are used on the metal parts and the workers become splashed with the oil. They should wear protective clothing, sleeves, gloves, and aprons, to protect them from oil dermatitis.

PLASTER SHOP

Here the men are engaged in making plaster casts in which the molds are made. A compound consisting of stearic acid in coal oil is put on the plaster to prevent it from sticking to the molds. Dermatitis develops among some of the men from the defatting action of the coal oil. It would be advisable to substitute a vegetable oil such as castor oil or linseed oil for the coal oil in order to prevent dermatitis.

PLATING

Only cadmium plating is done. The parts to be cadmium plated are first sandblasted and then degreased by washing in strong alkali

soap. They are then dried and immersed in a plating solution which contains about 4 percent of sodium cyanide. The plating tanks are usually well vented, but accidental splashes and drippings from the metal parts may fall on the workers. Therefore, they should wear protective clothing in the form of impervious gloves, sleeves, and aprons, and it would also be advisable to have them insert vaseline into the nostrils to prevent possible nasal mucitis from cyanide fumes that may escape from the tanks.

TUBING DEPARTMENT

In the tubing department, workers cleaning and painting tubes should wear aprons, sleeves, and rubber gloves because dermatitis has occurred among workers from the cleaners and paints. Some cases have also occurred from copper tubing, probably caused by the lacquer which is applied to some of the tubing.

Oil is run through the tubing, which constitutes the chassis of the fuselage of the plane, in order to act as a protective against rust. The workers engaged in this operation are splashed with the oil and should wear gloves, boots, aprons, and sleeves of an oilproof material.

WELDING

Metal parts are welded together by electric welding, by oxy-acetylene welding, or by the so-called spot welding. Parts made of dural or of stainless steel are welded together by the use of fluxes containing fluorides. The workers lean over the flame of the welding torch and their faces are exposed to the fluoride fumes given off during the operation. Nasal mucitis, as evidenced by nose bleed and ulceration of the nasal septum, occurs among these workers. They should insert vaseline into the nostrils several times a day to prevent this condition.

In spot welding, a mixture of hydrofluoric acid and tragacanth is brushed on the metal to act as a flux. The men brushing it on and washing it off should wear rubber gloves, impervious sleeves, and aprons to prevent hydrofluoric acid burns, and they should also insert vaseline into the nostrils. These same precautions should be observed wherever a flux containing a fluoride, zinc chloride, or chromic acid is used.

WOOD SHOP

Here the various parts requiring wood are made. In some of the wood shops, dermatitis has occurred from a wood known as Honduras mahogany or to the workers as "Tabasco" mahogany because of its irritant properties. The dust of this wood is said to be irritating to a considerable percentage of the workers. Only such workers as are known to be nonsusceptible should be permitted to work with this wood.

X-RAY

Many of the metal parts of the plane are X-rayed for defects. The machines used are powerful and totally enclosed, and those handling them are well protected against X-ray exposure. No dermatitis or burns of the skin were found in this department.

ZINC CHROMATE

Zinc chromate is the pigment that is used to the greatest extent on the metal parts of airplanes. It is applied both for purposes of a filler and a paint. It is sprayed on or applied by hand brush. Several cases of dermatitis were found among persons showing by patch test a hypersensitivity to the zinc chromate itself. The prevention of dermatitis from zinc chromate has been mentioned previously under the discussion of hazards occurring in the paint shop.

SUMMARY

An inspection of airplane plants employing over 100,000 men shows that there are many skin hazards in airplane manufacture.

The principal ones are those from cutting oils, thinners, and solvents used in paints and "dopes," plating and rustproofing of metals, fluxes used in welding, and solvents used for cleaning and degreasing.

Preventive measures consisting of wearing of impervious clothing, the use of protective ointments, and the use of nonirritating skin cleansers, in addition to proper general and local ventilation, are described.

A PROTECTION TEST IN MICE FOR IDENTIFICATION OF LEPTOSPIROSIS ICTEROHAEMORRHAGICA (WEIL'S DISEASE) [1]

By CARL L. LARSON, *Assistant Surgeon, United States Public Health Service*

Demonstration of the uniform susceptibility of young white mice (*Mus musculus*) to *Leptospira icterohaemorrhagiae* (*1*) made it possible to develop a mouse protection test which is specific for leptospirosis icterohaemorrhagica. The data presented in this paper show the value of the test in differentiating Weil's disease from other diseases which are apt to be confused with it.

Konar, Roy, and De (*2*) include influenza, infectious jaundice, secondary syphilis, septicemia, malaria, blackwater fever, dengue, typhus fever, and yellow fever in the differential diagnosis of Weil's disease. Soper (*3*) points out that the latter disease must be distinguished from yellow fever and this is especially emphasized in view

[1] From the Division of Infectious Diseases, National Institute of Health.

of the confusion which led Noguchi (*4*) erroneously to report *L. icteroides* as the etiologic agent of yellow fever. Fairley (*5*), Davidson et al. (*6*), Schüffner (*7*), and Walch-Sorgdrager (*8*) have pointed out that as many as 60 percent of patients infected with *L. icterohaemorrhagiae* may be anicteric and Weil's disease may be overlooked because this symptom is lacking.

In most cases of Weil's disease laboratory methods must be resorted to in order to establish a diagnosis firmly. The organism can be isolated from blood or urine before death or from post-mortem material by cultivation on Verwoort's or other suitable medium, or by transmission to susceptible animals. Davidson et al. (*6*) isolated leptospirae from the blood in 9 of 22 patients, and from the urine in 5 of 64 attempts.

Agglutinins, lysins, and complement-fixing antibodies are developed during the second week of illness and tests for their presence and increase in titer are made to confirm the clinical diagnosis in the majority of instances. The adhesion test adapted by Brown and Davis (*9*) and the agglutination-lysis test of Schüffner and Mochtar (*10*) are the most widely used serological aids. Schüffner (*7*) states that agglutinins may persist in the blood of recovered patients for at least 8 years, and Fairley (*11*) demonstrated their presence 12 years after onset of the disease. Davidson (*12*) used the agglutination test to diagnose subclinical and asymptomatic cases in retrospect.

Pfeiffer's phenomenon and immune reactions among guinea pigs have been demonstrated by Inada and his coworkers (*13*) but these results have not been applied to any great extent.

The methods mentioned above for laboratory studies of Weil's disease present difficulties of technique and interpretation which make it desirable to describe the findings concerning a specific mouse protection test against *L. icterohaemorrhagiae*.

MATERIALS AND METHODS

L. icterohaemorrhagiae, strain 1653, was used as the infectious agent throughout most of the work. It was originally isolated by Dr. A. Packchanian from a wild rat trapped in Washington, D. C. It is highly pathogenic for 3-week-old white mice and has been carried through a number of generations in mice since June 20, 1940. Strain 11, used occasionally, was isolated directly in white mice from the kidney of a wild rat trapped in Arlington County, Virginia. It has been transferred from mice to mice since July 12, 1940, and is also highly pathogenic for them.

It is necessary that young white mice, 3 to 4 weeks old, be used, in order to obtain maximum mortality in experimental leptospirosis, and mice of this age, bred at the National Institute of Health, have been employed routinely as test animals.

The test is carried out in the following manner. The kidneys and liver of a mouse dead or dying of leptospirosis are removed, weighed, and transferred to a mortar. They are finely ground with the aid of an abrasive, and sufficient normal saline solution is added to make a 10 percent suspension. The presence of leptospirae in the suspension should be checked by dark-field examination. Further 10-fold dilutions of this suspension, which have been allowed to settle by gravity, are made so that final dilutions from 10^{-1} to 10^{-5} are obtained. The serums to be tested are diluted to 10^{-1} with saline and passed through a Berkefeld N filter in order to insure sterility. Equal quantities of serum diluted to 10^{-1} and tissue suspensions diluted to 10^{-2} are mixed and allowed to stand at room temperature for 1 to 2 hours. Ten mice are inoculated intraperitoneally with 0.3 cc. of each dilution of tissue suspension and a similar number with 0.6 cc. of each serum-tissue mixture to be tested. In this way equal quantities of the infectious agent contained in a 10^{-2} dilution of infected tissue is given to each mouse to which this dilution of material is administered. The infectious agent has been titrated in the experiments to be reported.

The mice are observed for 2 weeks after inoculation before the experiment is terminated and the animals discarded. Mice dying before the fourth day following injection are not included in the final results of the test (*14*) as they die of secondary infection, trauma, or other conditions not directly due to the agent in question. All mice surviving this period are carefully observed for the development of jaundice, and all deaths are recorded. Observations are made daily at 8:30 a. m. in order to decrease the error in the noted survival time of individual animals. Protection is stated in terms of mice surviving 14 days compared to those still alive 4 days after inoculation.

The serums used in these studies were obtained from cases of Weil's disease and from cases of infectious jaundice, syphilis, typhus fever, "Q" fever, tularemia, malaria, poliomyelitis, rat-bite fever, and influenza, in order to determine the specificity of the test. Samples of serum from animals immunized against Rocky Mountain spotted fever virus, influenza virus, *L. canicola*, and *L. icterohaemorrhagiae* were likewise examined. Material was also obtained from animals suffering from, or recovered from, rat-bite fever, relapsing fever, and syphilis, as well as from normal rats and humans.

One sample of blood from a dog with leptospirosis was obtained from Dr. J. C. Lange, Greensboro, N. C. It agglutinated *L. canicola* to a titer of 1:1000 and *L. icterohaemorrhagiae* to a titer of 1:100.

The various rat serums (*R. norvegicus*) studied are listed in table 1, together with data relating to the diagnosis of leptospirosis in these animals. The serums of 15 wild rats were examined. Thirteen of these rats gave positive agglutination tests for the organism, having titers varying from 1:100 to 1:100,000. *L. icterohaemorrhagiae*

was noted by dark-field examination of the kidneys of 10 of them, isolated by animal transmission in 3 instances and cultured on Verwoort's medium from 7 others.

TABLE 1.—*Data concerning leptospirosis in wild rats (R. norvegicus), the serums of which were tested for the presence of protective antibodies against L. icterohaemorrhagiae*

Rat No.	Origin	Date examined	Titer of agglutination vs. *L. icterohaemorrhagiae*	*L. icterohaemorrhagiae* isolated by culture	*L. icterohaemorrhagiae* isolated by animal inoculation	*L. icterohaemorrhagiae* seen microscopically (dark-field kidney)
A8	Arlington, Va.	July 12, 1940	1:100	+	+	−
A9	do	do	1:100	−	−	+
A11	do	do	1:100	+	+	+
A12	do	do	1:1,000	+	−	+
A17	do	July 13, 1940	1:10,000	−	−	−
A19	do	July 15, 1940	1:1,000	−	−	+
A20	do	do	1:1,000	+	−	+
A24	do	July 16, 1940	Negative	−	−	−
A25	do	July 29, 1940	1:1,000	+	+	+
A30	do	July 30, 1940	Negative	−	−	−
A48	Washington, D. C.	Aug. 21, 1940	1:10,000	−	−	+
A50	do	Aug. 22, 1940	1:10,000	+	−	+
A52	do	Aug. 23, 1940	1:100,000	−	−	−
A55	do	do	1:1,000	+	−	+
A57	do	Aug. 27, 1940	1:10,000	−	−	+

In table 2 is listed information concerning the 25 human cases of leptospirosis whose serums were tested for the occurrence of protective antibodies. The data available show that from a clinical viewpoint these cases were typical of Weil's disease, since jaundice, fever, and leucocytosis characterized the infection. This view is further enhanced by the results of laboratory investigation of the agglutinin content of the blood. Serum from case 1 was examined on three occasions during the course of illness. Sample A, taken on December 6, 1940, the sixth day of the disease, failed to agglutinate *L. icterohaemorrhagiae*. Four days later sample B showed a titer of 1:10,000, and in about 3 weeks the titer of sample C had increased to 1:100,000. Case 4 had Weil's disease in 1935. At the time of illness the patient's serum agglutinated *L. icterohaemorrhagiae* to a titer of 1:80,000. In 1937 when an agglutination test was performed against this organism in Uhlenhuth's laboratory the titer had fallen to 1:6,000 and by 1940 the titer had decreased to 1:250. Cases 2 and 3 were ill in 1938 and serum was obtained in July 1940, about 2 years later. The serums of Cases 7 and 21 were tested 18 and 16 months, respectively, after onset of Weil's disease. The fact that we were able to study serums taken during the time antibodies were developing, when antibodies were at their height, and as late as 5 years after onset of symptoms enables us to measure the protective value of serum through all phases of the disease.

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TABLE 2.—*Summary of human cases of Weil's disease whose serum was tested for presence of protective antibodies against L. icterohaemorrhagiae*

Case No.	Location	Name	Age	Sex	Color	Date of onset	Occupation	Presence of:			*L. icterohaemorrhagiae* isolated	Result of agglutination test vs. *L. icterohaemorrhagiae*
								Jaundice	Fever	Leucocytosis		
1	Richmond, Va	GM	25	♂	C	Dec. 1, 1940	Poultry picker	+	+	+		(*a*) Negative. (*b*) 1:10,000. (*c*) 1:100,000.
2	Jefferson Co., Ala	RC		♂		1938	Coal miner	+	+			1:100.
3	Cincinnati, Ohio	WD	48	♂	C	Mar. 3, 1940	Ditch digger	+	−	+	+	1:10,000.
4	Worcester, Mass	ED				1935						1:250.
5	Atlanta, Ga	TH	23	♂	W	July 25, 1940		+	+	+		1:100,000.
6	New Britain, Conn	CL	47	♂	W	Aug. 1, 1940	Salesman	+	+	+		1:100,000.
7	Milwaukee, Wis	CB	51	♂	W	May 27, 1939	Sewer worker	+	+			1:1,000.
8	Philadelphia, Pa	JW	18	♂	W		Unemployed	+	+	+		1:1,250.
9	Chelsea, Mass	SL	40	♂	W	Sept. 16, 1940	Fish cutter	+	+			1:10,000.
10	Baltimore, Md	SE		♂		Dec. 10, 1940	Poultry picker	+	+	+	+	1:100,000.
11	do	PE	50	♀	C	Aug. 19, 1940		+	+	+	+	1:100,000.
12	Philadelphia, Pa	GK	18	♂	W	Aug. 18, 1940	Unemployed (Picked up a rat)	+	+			1:100,000.
13	Jefferson Co., Ala	RS	24	♂	W	Oct. 31, 1938	Miner	+	+			1:1,000.
14	Puerto Rico	ED	39	♂	W	Nov. 5, 1940	Farmer	+	+	+		1:100,000.
15	Milwaukee, Wis	CS	27	♂	W	January, 1941	Slaughter-house worker	+	+	+	+	1:100,000.
16	Chelsea, Mass	RT	48	♂	W	Jan. 4, 1941	Fish cutter	+	+	+		1:10,000.
17	Cincinnati, Ohio	GL		♂		July, 1940		+	+	+		1:10,000.
18	Jefferson Co., Ala	CF	39	♂	W	June 19, 1940	Miner	+	+	+		1:3,000.
19	do	RQ		♂	C	Aug. 9, 1940	do	+	+			1:1,000.
20	do	BM	27	♂	W	Mar. 27, 1939	do	+	+	+		1:1,000.
21	Cincinnati, Ohio	HA	21	♂	W	Sept. 28, 1939	Laborer	+	+	+		1:100.
22	Detroit, Mich	JE					No data					1:100,000.
23	Hartford, Conn	?3073					No data					1:10,000.
24	Milwaukee, Wis	?	48	♂	W		Sewer worker	+	+	+		1:1,000.
25	Jefferson Co., Ala	LR		♂	C	May 25, 1940	Miner					1:100,000.

EXPERIMENTAL

Test of protection afforded mice infected with L. icterohaemorrhagiae by serums from cases of infectious jaundice and Weil's disease.—Serums from cases of infectious jaundice were obtained from five persons who contracted the disease in Jenkins County, Georgia, in the fall of 1940 and these, together with serums from nine cases of Weil's disease, were tested at the same time. The test was made in the manner previously described and the results are given in table 3.

TABLE 3.—*Results of testing for presence of protective antibodies against L. icterohaemorrhagiae in serums from cases of infectious jaundice and Weil's disease*

Serum	Type of serum	Source of serum	Agglutination titer vs. *L. icterohaemorrhagiae*	Date of inoculation [1]	Number of deaths, by days														Survivors at end of 4 days	Survivors at end of 14 days	Ratio of protection
					1	2	3	4	5	6	7	8	9	10	11	12	13	14			
CL 44	Infectious jaundice	Human	Negative	Dec. 17, 1940						1			5	1	1		1		10	1	1/10
CL 57	do	do	do	do						1			8				1		10	0	0/10
CL 58	do	do	do	do							4	4	1		1				10	0	0/10
CL 59	do	do	do	do								6	1		2		1		10	0	0/10
CL 60	do	do	do	do								7	3						10	0	0/10
1 (a)	Weil's disease	do	do	do								1	3	1	1		3		10	1	1/10
1 (b)	do	do	1:10,000	do											2				10	8	8/10
2	do	do	1:100	do		1				1							2		9	6	6/9
3	do	do	1:10,000	do		1	2												7	7	7/7
4	do	do	1:250	do		1													9	9	9/9
5	do	do	1:100,000	do															10	10	10/10
6	do	do	1:100,000	do															10	10	10/10
7	do	do	1:1,000	do															10	10	10/10
8	do	do	1:1,250	do															10	10	10/10
9	do	do	1:10,000	do															10	10	10/10

TITRATION OF INFECTIVE AGENT FOR MINIMAL FATAL DOSE (NO SERUM)

Dilution of infective tissue emulsions (dose, 0.3 cc.)	Date of inoculation	Mice dying, by days														Survivors at end of 4 days	Survivors at end of 14 days	Ratio of survival
		1	2	3	4	5	6	7	8	9	10	11	12	13	14			
10^{-1}	Dec. 17, 1940						4		6							10	0	0/10
10^{-2} [2]	do								10							10	0	0/10
10^{-3}	do								8	2						10	0	0/10
10^{-4}	do									1	2	1		1		10	5	5/10
10^{-5}	do															10	10	10/10

[1] Test dose of serum-leptospira mixture, 0.3 cc. 10^{-1} dilution of serum +.3 cc. 10^{-5} suspension of leptospira infected tissue.
[2] Dilution of leptospira infected tissue employed in serum mixture.

It is apparent that none of the 5 serums from cases of infectious jaundice (CL 44, 57, 58, 59, and 60) protected mice against infection with a tissue suspension containing *L. icterohaemorrhagiae*. Only 1 of 50 mice so treated survived. Among the 10 samples of serum originating from cases of Weil's disease, 7 protected all of the mice surviving until the fourth day after inoculation. Included in these are the serums from Case 4 which were taken 5 years after onset of Weil's disease, and from Case 7 which were examined 18 months after the original illness. Serum from Case 2 protected only 6 of 9 mice. This sample had an agglutination titer of 1:100 and was drawn 2 years after the patient had been ill. The first sample of serum from Case 1, taken on the sixth day of illness, contained no antibodies capable of shielding mice against leptospirosis, but the second lot, drawn on the tenth day, protected 8 of 10 mice injected with it.

Test of protective ability of syphilitic serum and of serum from cases of Weil's disease, and from rabbits immunized against L. icterohaemorrhagiae.—A test was set up to determine whether or not serum taken from syphilitic patients having positive Wassermann reactions was capable of protecting young white mice against leptospirosis. Six serums obtained from the Naval Medical School, Washington, D. C., gave strongly positive Wassermann reactions. They were tested together with 5 serums from patients with Weil's disease and 3 from rabbits (R–11, R–S, R–1653) immunized against *L. icterohaemorrhagiae*.

TABLE 4.—*Results of testing for presence of protective antibodies against L. icterohaemorrhagiae in serums from cases of syphilis and Weil's disease, and from rabbits immunized with this organism*

Serum	Type of serum	Source of serum	Agglutination titer vs. *L. icterohaemorrhagiae*	Date of inoculation	Number of deaths, by days														Survivors at end of 4 days	Survivors at end of 14 days	Ratio of protection
					1	2	3	4	5	6	7	8	9	10	11	12	13	14			
A	Syphilis	Human	Negative	Jan. 6, 1941						1	8	1							10	0	0/10
B	do	do	Negative	do						1	2	7							10	0	0/10
C	do	do	Negative	do	1					2	6	1							9	0	0/9
D	do	do	Negative	do		1				1	2		3	1					9	2	2/9
E	do	do	Negative	do						3	4	3							10	0	0/10
F	do	do	Negative	do						2	6	2							10	0	0/10
10	Weil's disease	do	1:100,000	do								1							10	9	9/10
11	do	do	1:100,000	do															10	10	10/10
1 (c)	do	do	1:100,000	do										2					10	8	8/10
12	do	do	1:100,000	do															10	10	10/10
13	do	do	1:1,000	do	1														9	9	9/9
R16[illegible]	Anti-icterohaemorrhagiae	Rabbit	1:1,000,000	do															10	10	10/10
R8	do	do	1:1,000,000	do															10	10	10/10
R11	do	do	1:1,000,000	do															10	10	10/10

TITRATION OF INFECTIVE AGENT FOR MINIMAL FATAL DOSE (NO SERUM)

Dilution of infective tissue emulsions (dose, 0.3 cc.)	Date of inoculation	Mice dying, by days														Survivors at end of 4 days	Survivors at end of 14 days
		1	2	3	4	5	6	7	8	9	10	11	12	13	14		
10^{-1}	Jan. 6, 1941						6	3		1						10	0
10^{-2}	do					1	2	3	3	1						10	0
10^{-3}	do								3	2	3	1				10	1
10^{-4}	do															10	10
10^{-5}	do															10	10

The results obtained show conclusively that no antibodies against *L. icterohaemorrhagiae* were present among the small group of syphilis serums examined (table 4).

A serum (Case 13) obtained 2 years after Weil's disease appeared afforded ample protection to mice to which it was administered, as did serum from Case 1 (C) examined about 3 weeks after onset of illness. Serums from the 3 rabbits immunized against *L. icterohaemorrhagiae* fully protected mice against leptospirosis.

Test of protection afforded by yellow fever immune serums and by serums from cases of Weil's disease, and from rabbits immunized against L. canicola.—Yellow fever antiserum was drawn from individuals who had been immunized with attenuated yellow fever virus at the Rocky Mountain Laboratory, Hamilton, Mont. None of the subjects had ever had jaundice prior to immunization. The immunization procedure induced the production of antibodies against yellow fever virus as shown by mouse protection tests. No agglutinins against either *L. canicola* or *L. icterohaemorrhagiae* could be demonstrated (table 5). When tested for the presence of protective antibodies against this latter organism none of these serums protected mice, while 4 specimens of serum from cases of Weil's disease containing agglutinins against *L. icterohaemorrhagiae* protected at least 9 of the 10 mice into which they were inoculated. Serum from rabbits (P–b, P–c) immunized against *L. canicola* either failed entirely to protect mice or gave equivocal results. In 1 case, only 1 of 10 mice with murine leptospirosis survived after having been given a mixture of anti-canicola serum and infective tissue, and in another case 4 of 10 mice died after being subjected to the same procedure.

TABLE 5.—*Results of testing for presence of protective antibodies against L. icterohaemorrhagiae in serums from cases of yellow fever, Weil's disease, and from rabbits immunized with L. canicola*

Serum	Type of serum	Source of serum	Agglutination titer vs. *L. icterohaemorrhagiae*	Date of inoculation	Number of deaths, by days													Survivors at end of 4 days	Survivors at end of 14 days	Ratio of protection
					1	2	3	4	5	6	7	8	9	10	11	12	13	14			
82	Yellow fever	Human	Negative	Jan. 28 1941						1	2	4	1	1					10	1	1/10
83	do	do	do	do						2	4	2	1						10	1	1/10
84	do	do	do	do						1	1		2		1	1	2		10	2	2/10
85	do	do	do	do							2	1	1			1	1	2	10	2	2/10
86	do	do	do	do							3	2	3			1			10	1	1/10
87	do	do	do	do							5	4	1						10	0	0/10
P-b	Anticanicola	Rabbit	10^{-3}	do							4	3			1		1		10	0	0/10
P-e	do	do	10^{-3}	do							1		2	1					10	6	6/10
14	Weil's disease	Human	10^{-4}	do															10	10	10/10
15	do	do	10^{-4}	do								1							10	9	9/10
16	do	do	10^{-4}	do											1				10	9	9/10
10	do	do	10^{-4}	do															10	10	10/10

TITRATION OF INFECTIVE AGENT FOR MINIMAL FATAL DOSE (NO SERUM)

Dilution of infective tissue emulsions (dose, 0.3 cc.)	Date of inoculation	Mice dying, by days														Survivors at end of 4 days	Survivors at end of 14 days
		1	2	3	4	5	6	7	8	9	10	11	12	13	14		
10^{-1}	Jan. 28 1941					1	1	3	2	2			2			10	0
10^{-2}	do							2	4	4						10	0
10^{-3}	do														2	10	8
10^{-4}	do															10	10
10^{-5}	do															10	10

Further tests of the ability of various types of antisera to protect mice infected with L. icterohaemorrhagiae.—Tests were carried out on 25 other samples of serum which originated from various sources other than Weil's disease. Poliomyelitis antiserum from 2 cases occurring near Charleston, S. C., were found to possess protective antibodies against poliomyelitis virus. The samples from those persons with "Q" fever and typhus fever also had antibodies against the respective etiologic agents. Specimens from 2 cases of malaria were studied; one of these was taken during the height of fever, while the other was taken during an afebrile period. An influenza serum originated from a case in San Diego, Calif., and was shown to have antibodies capable of protecting mice against infection with this virus. Material from 3 cases of tularemia possessed agglutinins to a high titer for *B. tularense*, and that from a single case of rat-bite fever of the Haverhill type agglutinated *Streptobacillus moniliformis*. All of the cases from which serum was obtained were characteristic of the disease involved, and in no instance were agglutinins for *L. canicola* or *L. icterohaemorrhagiae* demonstrated.

The serums of 4 guinea pigs having *Spirillum minus* in their blood were tested for protective antibodies against leptospirae. A rabbit infected with *Treponema pallidum* presented an active local lesion at the time blood was obtained for experimental use. A specimen of serum from a rabbit which had been hyperimmunized against Rocky Mountain spotted fever virus contained protective antibodies against that virus. Five monkeys exposed to relapsing fever were also studied; one specimen of serum (364) was taken when the monkey was ill and the others 6 months after the last exposure of the animals to *Spirochaeta recurrentis*. The organism had been noted in all of them at some stage of the disease. No agglutinins for leptospirae were found in any of the samples of serum discussed. Various members of the Public Health Service supplied most of the material examined.

Table 6 shows the results of agglutination tests against *L. icterohaemorrhagiae* and of protection tests against murine leptospirosis performed with these specimens. It will be noticed that they contained neither agglutinins nor protective antibodies against the leptospirae. While the number of samples studied from each disease is necessarily small, the results obtained appear to show that the test is specific. At the time the above serums were being examined a group of 22 samples taken from persons and rats with leptospirosis were also studied and served as controls for the former group. The results of the agglutination and protection tests are shown in table 7.

TABLE 6.—*Results of testing for the presence of protective antibodies against L. icterohaemorrhagiae in serums derived from miscellaneous sources*

Serum	Type of serum	Source of serum	Agglutination titer vs. *L. icterohaemorrhagiae*	Number of deaths, by days														Survivors at end of 4 days	Survivors at end of 14 days	Ratio of protection
				1	2	3	4	5	6	7	8	9	10	11	12	13	14			
232-3	Malaria	Human	Negative					2		1	5	2						10	0	0/10
246-A	do	do	do							5	3	2						10	0	0/10
ML	Poliomyelitis	do	do							3	5	2						10	0	0/10
AB	do	do	do							6	1		3					10	0	0/10
450	Syphilis	Rabbit	do				2	1	1	5	1							8	0	0/8
JY	Rat-bite fever (Haverhill)	Human	do							3	2		2					10	3	3/10
223	Rat-bite fever (S. minus)	Guinea pig	do	1							7	2						9	0	0/9
232	do	do	do		1		1			2	1	5						8	0	0/8
253	do	do	do								5	5						10	0	0/10
262	do	do	do							1	6	1	1					10	1	1/10
918	Relapsing fever	Monkey	do						5		4	1						10	0	0/10
364	do	do	do						4		4	1						10	1	1/10
67	do	do	do						4		5	1						10	0	0/10
75	do	do	do		1	1					6	2						8	0	0/8
357	do	do	do						4		4						1	10	1	1/10
12844	Tularemia	Human	do			1		1	1		5	2						9	0	0/9
19252	do	do	do						6		4							10	0	0/10
13010	do	do	do			1			1		6	1		1				9	0	0/9
FSU	Influenza	do	do							5	3	1	1					10	0	0/10
451	do	Rabbit	do						1	2	2	2	2					10	1	1/10
452	Rocky Mountain spotted fever	do	do						1	3	2	3	1					10	0	0/10
RN	"Q" fever	Human	do							3	1	3	1	1				10	1	1/10
EM	do	do	do							4	3	3						10	0	0/10
MT	Typhus fever	do	do						2	1	5	2						10	0	0/10
IB	do	do	do						2	2	2		2		1			10	1	1/10

TABLE 7.—*Results of testing for the presence of protective antibodies against L. icterohaemorrhagiae in serums from cases of murine, canine, and human leptospirosis*

Serum	Type of serum	Source of serum	Agglutination titer vs. *L. icterohaemorrhagiae*	Number of deaths, by days														Survivors at end of 4 days	Survivors at end of 14 days	Ratio of protection
				1	2	3	4	5	6	7	8	9	10	11	12	13	14			
A8	Leptospirosis	Rat	1:100			1												9	9	9/9
A11	do	do	1:100											1				10	9	9/10
A20	do	do	1:1,000					1		1								10	8	8/10
A50	do	do	1:10,000			1	1											8	8	8/8
A52	do	do	1:100,000			1												9	9	9/9
A55	do	do	1:1,000															10	10	10/10
A57	do	do	1:10,000					1										10	9	9/10
A12	do	do	1:1,000															10	10	10/10
A25	do	do	1:1,000				1											9	9	9/9
A48	do	do	1:10,000															10	10	10/10
CL56	do	Dog	1:1,000												1			10	9	9/10
14	do	Human	1:100,000			1												9	9	9/9
17	do	do	1:10,000															10	10	10/10
18	do	do	1:3,000															10	10	10/10
19	do	do	1:1,000															10	10	10/10
20	do	do	1:1,000					1										10	9	9/10
21	do	do	1:100		1	1												8	8	8/8
22	do	do	1:100,000															10	10	10/10
23	do	do	1:10,000															10	10	10/10
8	do	do	1:1,250															10	10	10/10
10	do	do	1:100,000															10	10	10/10
15	do	do	1:100,000													2		10	8	8/10

It is readily seen that serum developed in the presence of leptospirosis acts specifically to preserve mice against infection due to *L. icterohaemorrhagiae*. While only 10 of 241 mice (4.1 percent) treated with nonspecific serum and which survived the fourth day following inoculation recovered from the infection, 204 of 212 mice (96.2 percent) treated with specific serum failed to develop leptospirosis and remained well during the period of observation. In the main, those diseases which may be confused, clinically, with leptospiral jaundice are easily differentiated from it by the mouse protection test.

Titration of protective ability of leptospiral antiserums.—An attempt was made to determine whether the protective ability of serum possessing agglutinins against *L. icterohaemorrhagiae* varies in relation to the agglutinin content. Table 8 shows the results obtained using 2 negative serums, 2 with agglutinins to a titer of 1:100, one with a titer of 1:1,250, 3 having titers of 1:1,000, 3 with titers of 1:10,000, and 2 with agglutinins to a titer of 1:100,000 against this organism. There is a wide range (1.78×10^{-3}) of protection afforded by these serums, as measured by the 50 percent end point method. While the ability of serum to protect mice generally increases as the agglutinin content increases, this does not necessarily hold in all cases. Thus, the greatest protection was afforded mice by injection of a serum (8) containing agglutinins to a titer of only 1:1,250, while one serum (25) with a titer of 1:100,000 had a 50 percent end point of only 2.37×10^{-2}. One serum (13) drawn 2 years after injection had a 50 percent end point of 2.68×10^{-2}. The relation between protecting ability and agglutinin content does not remain constant. A low concentration of protective antibodies may be encountered in the presence of abundant agglutinins and the reverse may also hold.

TABLE 8.—*Results of titration of protective antibodies against L. icterohaemorrhagiae contained in serums from rats and humans with leptospirosis*

Serum No.	Source of serum	Agglutination titer vs. *L. icterohaemorrhagiae*	Number of mice used	Serum dilution					50 percent end point
				10^{-1}	10^{-2}	10^{-3}	10^{-4}	10^{-5}	
5	Human	1:100,000	5	[1] 5/5	5/5	2/5	1/5	0/5	1×10^{-3}.
25	do	1:100,000	5	4/5	4/5	1/5	0/5	0/5	2.37×10^{-3}.
A17	Rat	1:10,000	5	5/5	5/5	0/5	0/5	0/5	3.16×10^{-3}.
3	Human	1:10,000	5	5/5	3/5	0/5	0/5	0/5	4.68×10^{-3}.
A27	Rat	1:10,000	5	5/5	3/5	0/5	0/5	0/5	4.68×10^{-3}.
8	Human	1:1,250	5	4/5	5/5	4/5	0/5	0/5	1.78×10^{-3}.
A19	Rat	1:1,000	5	5/5	5/5	1/5	0/5	0/5	4.22×10^{-3}.
24	Human	1:1,000	6	6/6	5/6	1/6	0/6	0/6	3.16×10^{-3}.
13	do	1:1,000	5	5/5	4/5	0/5	0/5	0/5	2.68×10^{-2}.
2	do	1:100	5	4/5	0/5	0/5	0/5	0/5	2.37×10^{-1}.
A9	Rat	1:100	6	6/6	0/6	0/6	0/6	0/6	3.20×10^{-1}.
A24	do	Negative	5	0/5	0/5	0/5	0/5	0/5	0.
A30	do	Negative	5	0/5	0/5	0/5	0/5	0/5	0.

[1] Numerator=number of mice surviving; denominator=number of mice inoculated.

DISCUSSION

The data presented show that serum obtained from individuals or from animals which have been infected with, or immunized against, *L. icterohaemorrhagiae* possesses antibodies capable of preventing young white mice from becoming infected with this organism when serum and tissue suspension containing leptospirae are mixed *in vitro* before injection into the animals. Such serums also contain specific agglutinins for the organism. The fact that such antibodies could be demonstrated in this material suggests the use of a mouse protection test for diagnostic and laboratory procedures in studies of Weil's disease.

Serums derived from cases of disease other than leptospirosis were examined in order to ascertain whether the test was specific. In none of them were either agglutinins or protective antibodies demonstrated. With the exception of dengue fever and blackwater fever, material from all diseases usually considered in the differential diagnosis of leptospirosis was examined, with negative results. The protective antibodies produced in animals following contact with *L. icterohaemorrhagiae* possess a specific affinity for these organisms and are not produced in response to infections of the other diseases studied.

Protective antibodies appear in the blood stream in about 2 weeks following the onset of illness at about the same time as agglutinins become evident. They remain in the serum of individuals who have recovered from Weil's disease for a period of at least 5 years. In cases where the clinical diagnosis of Weil's disease has not been made or where the agglutination titer of the serum is so low as to be of a controversial nature, the protection test serves as a specific and sensitive diagnostic procedure.

Although the protective antibody content of serum from Weil's disease patients and from rats infected with *L. icterohaemorrhagiae* has a certain general relation to the agglutination titer, this has been shown to hold only to a limited degree. The mouse test would probably not be of significance in attempting to follow the course of illness by measuring the titer of antibodies developed as the disease progresses.

In the few tests which have been made using serums from rabbits hyperimmunized against *L. canicola* to protect mice against infections with *L. icterohaemorrhagiae*, it appears that there is a marked inability of such serums to act against heterologous leptospirae. This is of especial interest when the marked protection afforded by the single serum obtained from a dog suffering from leptospirosis is noted. Although it agglutinated *L. canicola* to a titer of 1:1000 and *L. icterohaemorrhagiae* to a titer of only 1:100, it protected 9 of 10 mice infected with the latter organism.

CONCLUSIONS

1. Serums from 25 humans, 13 wild rats, and 1 dog all suffering from leptospirosis were tested for the presence of specific protective antibodies against *L. icterohaemorrhagiae*. Material from three rabbits hyperimmunized against *L. icterohaemorrhagiae* was also tested.

2. Protective antibodies were detected in all of the above serums.

3. No protective antibodies against leptospirae were produced following influenza, malaria, poliomyelitis, Rocky Mountain spotted fever, typhus fever, "Q" fever, tularemia, rat-bite fever, relapsing fever, infectious jaundice, syphilis, and yellow fever.

4. Indefinite results were obtained with serums derived from rabbits hyperimmunized against *L. canicola*.

5. Protective antibodies develop during the second week of the disease and persist for at least 5 years.

6. The protective antibody titer roughly follows the agglutinin titer.

7. The mouse protection test devised can be used for the diagnosis of leptospirosis and offers a specific and easily interpreted test for this purpose.

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A STUDY OF THE RELATIVE TOXICITY OF THE MOLECULAR COMPONENTS OF LEAD ARSENATE [1]

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The extensive use of lead arsenate in agricultural spray material is of interest to public health because of the amount of spray residue present in the marketed product. This residue consists principally of unchanged dilead orthoarsenate ($PbHAsO_4$), although as a result of weathering a portion of the material on the surface of the fruit may be converted to a more basic form having the composition $Pb_4(PbOH)(AsO_4)_3.H_2O$, according to McDonnell and Graham (*1*).

The toxic properties of lead arsenate have been variously ascribed to different portions of the molecule. The assumption has been made, for instance, that the toxicity is due to the arsenate radical, i. e., that any arsenate would serve as a poison and that lead is chosen as a vehicle merely because of the physical properties of the compound, lead arsenate. It has also been considered that lead arsenate itself is a unique poison—that the combined toxic action of lead and arsenic specifically makes lead arsenate an especially effective insecticide. Furthermore, it has been suggested that the lead and arsenic are synergistic in action, each increasing the toxicity of the other when they are administered as lead arsenate. Whether the lead itself, or the arsenate group only, or whether molecular lead arsenate is the important factor in its toxic action on animals has not so far received the experimental attention that it deserves.

It has been shown by Newcomer (*2*) that, as an insecticide at least, calcium arsenate is just as effective as lead arsenate in the control of the codling moth. More recently Fleming, Baker, and Koblitsky (*3*) have found that as insecticides various lead salts, such as the acetate, borate, carbonate, chloride, fluoride, and acid phosphate were not toxic at the concentration used, whereas the arsenates of other metals, such as aluminum, iron, and magnesium, were invariably toxic. They conclude that the insecticidal action is to be attributed entirely to the arsenate radical. While calcium arsenate is apparently effective as an insecticide some of the commercial preparations of this salt are prone to cause leaf burn due to free arsenic acid formed by hydrolysis (*4*). The use of lead arsenate has also been favored because it has a

[1] From the Division of Industrial Hygiene, National Institute of Health. This is the fifth of a series of investigations concerning lead arsenate. The preceding publications are as follows:

Fairhall, L. T., and Neal, P. A.: Absorption and excretion of lead arsenate in man. Pub. Health Rep., 53: 1231-1245 (1938). Reprint No. 1960.

Fairhall, L. T.: The solubility of lead arsenate in body fluids. Pub. Health Rep., 54: 1636-1642 (1939). Reprint No. 2097.

Fairhall, L. T., and Sayers, R. R.: The significance of the excretion of lead in the urine. Pub. Health Rep., 54: 2016-2019 (1939). Reprint No. 2113.

Fairhall, L. T., Sayers, R. R., and Miller, J. W.: The relative toxicity of lead and some of its common compounds. Pub. Health Bull. No. 253. Government Printing Office, 1940.

greater spreading or covering power, so that the protective coat is more uniform.

Nonetheless, calcium arsenate is used commercially to a large extent even as compared with lead arsenate. According to Roark (*5*), in 1936 the estimated annual consumption of calcium arsenate in the United States was 45,000,000 pounds, while that of lead arsenate was 40,000,000 pounds. A large part of the calcium arsenate so used has been employed for dusting.

While the primary interest in the toxicity of lead arsenate has in the past largely related to its insecticidal properties, an increasing interest has been shown in its toxic action on animals (*6*). The present investigation was not concerned with the degree of toxic action of lead arsenate itself, as much as with finding which portion of the molecule, i. e., the lead or the arsenic, is the principal factor as the toxic agent.

Since this question exists as to the cause of the toxic action of lead arsenate, experiments were undertaken with animals to determine whether the toxicity is due to the lead radical, to the arsenic radical, or to both.

EXPERIMENTAL PROCEDURE

White rats were fed an experimental diet over a period of 2 years. The diet consisted of wheat flour, corn meal, oat meal, dextrin, powdered milk, dried yeast, and liver powder, with the addition of an adequate mineral ration and supplemented at intervals by greenstuff. The diet was uniform except that lead arsenate was added to the diet of one group, an equivalent amount of lead as lead carbonate was added to the diet of the second, and an amount of arsenate equivalent to that of the lead-arsenate group was added as calcium arsenate to the diet of the third group. The amounts of salts were weighed, suspended in water, and thoroughly mixed with the dried food materials and the resulting dough was rolled out into thin biscuit form and lightly baked. This insured a fairly uniform distribution of intake. Calcium arsenate and lead carbonate were chosen only because of their low solubility (comparable to that of lead arsenate) and not because calcium arsenate is also used as an insecticide. A control group of rats of similar age and weight was placed on the same diet free from the substances under test. The amount of lead or arsenic in the food was so arranged that on the basis of a 10-gram ration each rat would ingest daily approximately 10 milligrams of lead arsenate, or its lead or arsenic equivalent in lead carbonate or calcium arsenate.

The young white rats used weighed from 70 to 90 grams at the beginning of the experiment and were divided according to diet as follows: Calcium arsenate 99, lead arsenate 49, lead carbonate 55,

and controls 24. A larger number of rats was used for the calcium arsenate diet because those originally set aside for this purpose were females, while all the other animals were males. For this reason a second group of male rats was added for this diet. No marked difference was found, however, in the effect of calcium arsenate on the sexes. At the beginning it was not anticipated that the experiment would extend over a period of 2 years; it was assumed that the experiment would terminate within a few months because of the large dosage of 10 milligrams a day of lead arsenate or its equivalent. No attempt was made to estimate the degree of absorption of lead or arsenic by determining the fecal and urinary output of arsenic and lead, and comparing these figures with the intake.

The indices of toxicity included mortality figures and such outward signs of morbidity as loss of weight, refusal of food, diarrhea, poor posture, and gait. Throughout the experiment, however, the indices of morbidity revealed nothing of a striking nature. Finally, the distribution of lead and arsenic in the tissues was determined and the various tissues and organs were studied microscopically for pathological changes.

A certain number of deaths occurred in the various groups in the early stage of the experiment but these were due to middle ear infection and occurred in the control group as well as the others (table 1, fig. 1). It is possible, however, that the addition of either lead arsenate, calcium arsenate, or lead carbonate to the diet of the respective groups increased the mortality from disease at this point.

TABLE 1.—*Cumulative mortality rates of rats on experimental diets*

Duration of experiment (months)	Control, 24 animals		Lead carbonate, 55 animals		Lead arsenate, 49 animals		Calcium arsenate, 99 animals	
	Total deaths	Percent deaths	Total deaths	Percent deaths	Total deaths	Percent deaths	Total deaths	Percent deaths
1	3	13	9	16	4	8	14	14
2					5	10	20	20
3	4	17	10	18			23	23
4							25	25
5					6	12	29	29
6			11	20	7	14	35	35
7							37	37
8					9	18		
9			12	22	10	20	40	40
10					13	27	41	41
11			13	24	19	39	46	46
12			14	26	22	45	48	48
13								
14							53	54
15								
16			15	27	23	47	54	55
17	5	21					55	56
18			18	33	25	51	56	57
19			19	35	27	55	58	59
20							59	60
21			20	36				
22	8	33	21	38	28	57		
23			22	40	29	59	64	65
24	10	42	23	42	30	61	66	67

At the end of the first year, approximately half of the surviving rats of each group were killed. Portions of the kidneys, liver, and spleen were set aside for pathological investigation and the remainder analyzed for lead and arsenic. The remaining animals were retained at the same level of lead or arsenic intake until the end of the second year, when they were killed and their tissues analyzed. At the end of the experiment, each rat in its respective group had received a total of approximately 7.2 grams of lead arsenate, 5.5 grams of lead carbonate, or 4.8 grams of calcium arsenate.

EXPERIMENTAL RESULTS

The mortality figures over a period of 2 years, exclusive of the animals killed, indicate a difference between the arsenate groups and

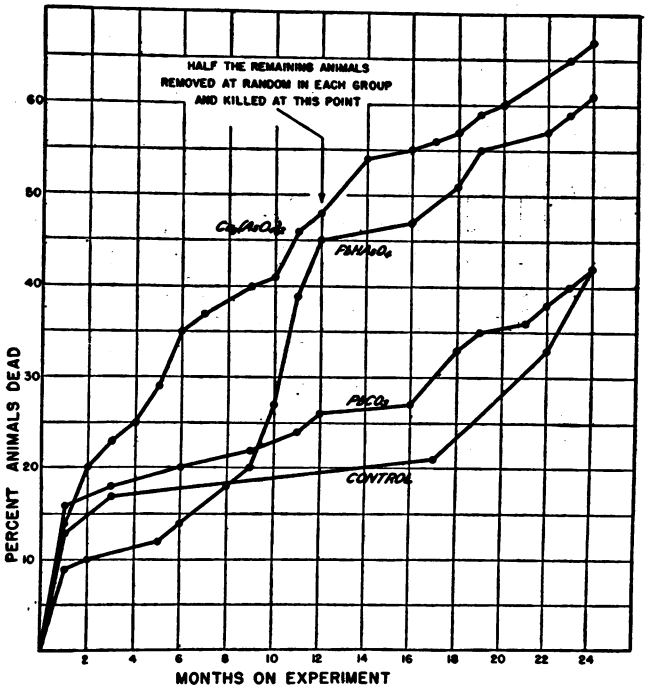

FIGURE 1.—Mortality (excluding animals sacrificed at end of first year) of white rats over a period of 2 years when fed 10 mg. of lead arsenate per day or its lead or arsenate equivalent.

the carbonate group. Rats on both the lead-arsenate and the calcium-arsenate diets showed a consistently greater mortality. From the second month until the end of the 2-year period, the rate of death was highest in the calcium-arsenate group. The mortality of the animals in the several groups is shown in figure 1.

The amount of lead in the tissues was determined by ashing and analysing by the chromate method (7), or by the colorimetric diphenyl-

thiocarbazone procedure (*8*), while the arsenic content was determined by the Gutzeit method (*9*).

The distribution of lead and arsenic in the tissues, liver, kidney, and bone, expressed in terms of 10 grams of wet tissue (with the exception of a few cases where specimens were unavoidably lost) is given in table 2.

A study of these figures shows several interesting relationships. As in previous investigations (*10*) the concentration of lead is greater in the kidney than in the liver, both in the case of lead arsenate and of lead carbonate. This is true both for the 1-year group and for the 2-year group. In the 1-year lead-arsenate group the average content of lead in the liver was 0.029 mg./10 grams as compared with 0.187 mg./10 grams of kidney, while for the 2-year group the values were 0.025 mg. and 0.124 mg., respectively. In the case of lead carbonate these amounts were, respectively, 0.039 mg. and 0.499 mg. for the 1-year group and 0.019 mg. and 0.297 mg. for the 2-year group.

With respect to arsenic, the liver and kidney storage was greater than with lead in the case of lead arsenate, namely 0.177 mg. and 0.197 mg. for the 1-year group and 0.166 mg. and 0.266 mg. for the 2-year group. In the case of calcium arsenate, the amounts in the liver and kidney were 0.129 mg. and 0.733 mg., respectively, for the 1-year group, and 0.223 mg. and 0.701 mg. for the 2-year group.

Our experience has shown that the lead and the arsenic content of liver and kidney may be diminished rapidly if the animals are placed on a normal diet even a few days before death. No attempt was made to determine the decrease in arsenic content of the liver and kidney with time as determined by Blumenfeldt (*11*) for acute arsenical poisoning, but a similar relationship between the liver and kidneys as temporary reservoirs of arsenic was noted.

TABLE 2a.—*Distribution of arsenic in tissues. Calcium arsenate*

Days on test	Weight at death, grams	Grams $Ca_3(AsO_4)_2$ received (estimate)	Mg. As/10 gm. tissue		
			Liver	Kidney	Bone
	1 year				
381	163	2.5	0.202	0.580	0.023
381	168	2.5	.158	.469	.047
388	160	2.5	.104	.443	.043
388	165	2.5	.088	.300	.073
388	176	2.5	.169	.902	.018
391	158	2.5	.081	.840	.037
391	126	2.5	.170	.545	.137
391	178	2.5	.147	.721	.035
391	205	2.5	.071	.950	.072
393	166	2.6	.157	.835	.058
393	164	2.6	.150	1.418	.060
393	162	2.6	.128	.473	.064
394	172	2.6	.150	.781	.094
394	186	2.6	.073	.712	.030
394	180	2.6	.086	1.025	.071
Average			.129	.733	.057
	2 years				
739	210	4.8	.106	.372	.004
739	220	4.8	.131	.460	.068
740	250	4.8	.067	.311	.000
740	215	4.8	.111	.636	.001
740	200	4.8	.077	.866	.013
742	225	4.8	.110	.366	.009
742	200	4.8	.133	.536	.007
742	200	4.8	.100	.292	.007
743	220	4.8	.072	.720	.017
743	225	4.8	.160		.009
743	220	4.8	.191	1.672	.011
740	356	4.8	.219	2.790	.000
740	350	4.8	.281	.346	.000
740	203	4.8	1.250	.244	.000
740	320	4.8	.283	.208	.000
740	405	4.8	.077	1.515	.000
740	390	4.8	.470	.207	.000
740	285	4.8	.232	.350	.000
Average			.223	.701	.008

TABLE 2b.—*Distribution of lead and arsenic in tissues. Lead arsenate*

Days on test	Weight at death, grams	Grams $PbHAsO_4$ received (estimate)	Mg. Pb/10 gm. tissue			Mg. As/10 gm. tissue		
			Liver	Kidney	Bone	Liver	Kidney	Bone
	1 year							
338	118	3.4	0.036	0.120	3.01	0.212	.067	0.048
341	142	3.4	.025	.151	3.63	.185	.081	
354	224	3.5	.033	.086	1.91	.109	.100	.051
378	178	3.8	.020	.236	2.77	.202	.079	.015
378	185	3.8	.031	.214	1.83	.077	.271	.018
379	204	3.8	.016	.238	1.48	.136	.143	
379	165	3.8	.038	.280		.132	.250	
379	191	3.8	.042	.367	2.98	.205	.633	.103
379	230	3.8	.032	.174	3.11	.267	.110	.026
379	284	3.8	.027	.099	1.98	.200	.187	.089
351	213	3.5	.024	.086	2.21	.224	.249	.034
Average			.029	.187	2.49	.177	.197	.048
	2 years							
737	230	7.4	.010	.057	1.46	.116	.223	.004
737	240	7.4	.021	.037	1.37	.249	.113	.028
737	270	7.4	.021	.150	3.44	.361	.490	.003
730	265	7.3	.013	.110		.236	.511	.004
737	200	7.4	.037	.159	2.81	.181	.206	.009
738	240	7.4	.034	.214	2.39	.107	.321	.004
693	230	6.9	.037	.149	2.43	.130		.006
738	240	7.4	.035	.150	3.50	.092	.128	.009
738	260	7.4	.020	.045	1.13	.055	.130	.006
739	260	7.4		.091	2.52	.099	.142	.002
739	250	7.4	.021	.150	1.65	.201	.400	.008
Average			.025	.124	2.27	.166	.266	.008

TABLE 2c.—*Distribution of lead and arsenic in tissues. Lead carbonate*

Days on test	Weight at death, grams	Grams $PbCO_3$ received (estimate)	Mg. Pb/10 gm. tissue		
			Liver	Kidney	Bone
	1 year				
343	138	2.6	0.028	0.387	3.44
367	155	2.8	.025	.417	4.83
367	192	2.8	.058	.613	3.99
367	202	2.8	.028	.444	3.37
367	116	2.8	.121	.372	4.86
352	258	2.6	.036	.351	4.00
368	229	2.8	.032	.528	3.43
368	215	2.8	.038	.643	3.99
368	225	2.8	.035	.696	2.90
371	245	2.8	.027	.642	2.84
371	211	2.8	.041	.455	5.61
371	281	2.8	.027	.417	3.28
378	250	2.8	.030	.425	4.90
378	255	2.8	.030	.594	4.45
Average			.039	.499	3.99
	2 years				
731	200	5.5	.023	.290	5.26
731	225	5.5	.027	.323	4.84
732	330	5.5	.018	.372	4.39
726	300	5.5	.016	.210	4.59
717	270	5.4	.021	.300	4.99
732	220	5.5	.024	.332	5.69
702	230	5.3	.031	.308	3.85
733	270	5.5	.024	.286	5.08
718	285	5.4	.011	.216	3.87
729	270	5.5	.009	.297	4.99
729	270	5.5	.012	.294	4.09
Average			.019	.297	4.69

TABLE 2d.—*Distribution of lead and arsenic in tissues. Controls*

Days on test	Weight at death, grams	Mg. Pb/10 gm. tissue			Mg. As/10 gm. tissue		
		Liver	Kidney	Bone	Liver	Kidney	Bone
	1 year						
395	161	0.013	0.139	0.000	0.007	0.087	0.008
395	134	.056	.050	.000	.007	.000	.000
395	213	.027	.046	.000	.017	.046	.009
395	180	.020	.050	.098	.006	.070	.016
395	164	.010	.033	.000	.010	.079	.012
Average		.025	.063	(.019)	.009	.056	.009
	2 years						
663	250	.023	.016		.020	.072	
743	250	.008	.033		.049	.060	
743	325	.003	.035		.008	.014	
743	160	.008	.041		.000	.000	
743	240	.005	.085		.037	.048	
Average		.009	.042		.023	.039	

A striking difference is apparent in the amount of lead deposited in the bone in the case of lead arsenate as compared with lead carbonate. For animals given lead arsenate, in the 1-year group the average amount of lead in bone tissue was 2.49 mg./10 grams, as compared with 3.99 mg./10 grams for animals given lead carbonate. In the

2-year group the respective figures were 2.27 mg./10 grams and 4.69 mg./10 grams. In other words, twice as much lead was deposited in the bones of those animals receiving lead carbonate as in those receiving lead arsenate, the actual amount of ingested lead being identical in each case. It would therefore appear that in the presence of the arsenate radical the amount of lead absorption from the gastrointestinal tract is either decreased, or that the excretion of lead is

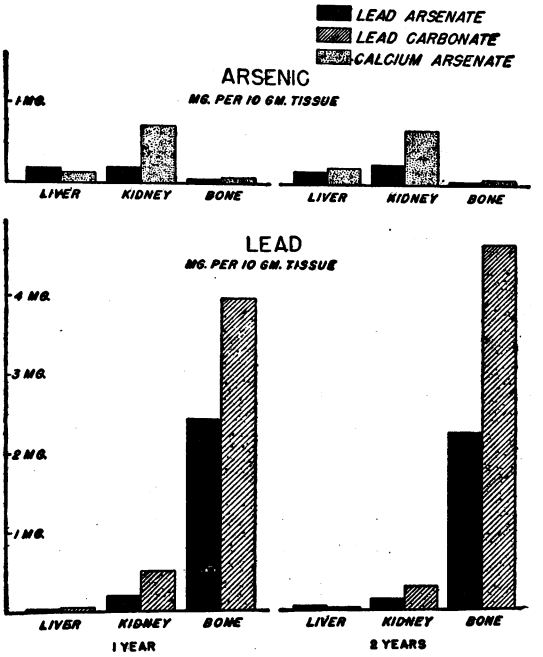

FIGURE 2.—Distribution of lead and arsenic in the tissues following the ingestion of lead arsenate or its lead or arsenate equivalent.

increased. This variation in distribution is shown graphically in figure 2.

There was no comparable relationship between the lead and arsenic content of the tissues, indicating that storage of lead and arsenic as lead arsenate itself probably does not occur in these tissues.

PATHOLOGY

Tissue from the animals killed both after 1 year and after 2 years was submitted to pathological examination. Paraffin sections were made from the liver, spleen, kidneys, heart, pancreas, stomach, duodenum, jejunum, ileum, and large intestine. Sections were routinely stained by Lillie's (*12*) eosin-polychrome methylene blue method. Sections from all of the spleens and a representative number of kidneys were stained by ferrocyanide to demonstrate the presence or absence of iron-bearing pigment. A number of sections, when

indicated, were stained by Lillie's [2] current modification of Gallego's elastic and connective tissue stain. Special microchemical methods described later were employed in studying the changes in the kidneys. An estimate of the degree of involvement of each organ was recorded for each rat in terms of a numerical code in which 0 designated no change from normalcy; minus plus, very slight; plus minus, slight or few; one plus, moderate; two plus, numerous; three plus, marked or many; and four plus, very marked or very many. These findings have been summarized in table 3 in which the average or typical reaction is entered. In every group of rats, of course, there were individual differences in reaction. These differences in response were considered in drawing conclusions from the data entered in the table.

TABLE 3.—*Summary of the principal pathologic findings in rats fed calcium arsenate, lead arsenate, and lead carbonate for periods of 1 and 2 years, respectively* [1]

	Calcium arsenate		Lead arsenate		Lead carbonate		Controls	
	1 year	2 years	1 year	2 years	1 year	2 years	1 year	2 years
Spleen:								
Hemosiderin	++	+++	++	++	±	±	±	0
Myelosis	++	++	+	++	∓	±	++	+++
Follicular phagocytosis	∓	±	∓	±	±	0	0	0
Kidney:								
Swollen cells in convoluted tubules	++	+	++	+	+++	+++	0	0
Oxyphil intranuclear inclusions	0	0	∓	+	+++	++	0	0
Brown pigment in convoluted tubule cells	+++	+	++	+	+++	+++	∓	++
Brown pigment in proximal tubule cells	+++	+	++	+	+++	+++	0	++
Brown pigment in distal tubule cells	++	0	0	∓	++	+	0	0
Casts	±	++	±	+	±	+	0	0

[1] 0, no deviation from normal; ∓ very slight, very few; ±, slight, few; +, moderate; ++, moderately marked, numerous; +++, marked, many; ++++, very marked, very many.

A total of 1,190 histological sections from a representative group of 87 rats was studied.

CALCIUM ARSENATE

Spleen.—The findings of the 1-year experiments were similar in all respects to the findings of the 2-year experiments but were less conspicuous. The splenic corpuscles were small, well defined, and surrounded by fairly large zones of paler staining cells, probably areas of perifollicular anemia. A moderate to a marked splenic myelosis (myeloid hyperplasia) with accompanying megakaryocytes was present in all of the animals. The degree of myelosis paralleled to some extent the amount of hemosiderin present. The cavernous veins were filled with blood, the amount of which varied inversely with the degree of myelosis and perifollicular anemia. Diffuse iron reaction (hemosiderosis) of a few to a considerable number of cells was

[2] Personal communication from Dr. R. D. Lillie.

found in all but 2 of the 14 rats in the 1-year series and in all of the 2-year group (fig. 3). In addition, a few round golden brown granules, some of which reacted for iron, but most of which did not, were present in the pulp. They showed considerable variation in size and were morphologically similar to granules found in the kidneys. These granules were not numerous and occurred more frequently and in greater numbers in the 2-year series. Lymphocytic infiltration of the trabeculae, usually slight to moderately marked in degree, was present in all of the rats. Nuclear fragments were found in the follicles of only 4 animals of the 1-year series but occurred in 9 of the 11 rats fed calcium arsenate for 2 years.

Kidneys.—Pathologic changes, with the exception of casts in the straight collecting tubules, were more conspicuous in the 1-year series than in the 2-year series. The cells in isolated tubules or in groups of convoluted tubules were swollen and contained large, vesicular nuclei. Many contained fairly large basophilic nucleoli but the oxyphil nuclear inclusions, so frequent in the lead carbonate series, were absent. The cytoplasm of the swollen cells was granular, not radially striated, and the cells sometimes occluded the lumina. Round, golden brown particles, variable in size, were found in a large proportion of the cells. They were most numerous in the swollen, convoluted tubule cells but also were present in the apparently normal cells. These particles were found both in the cells and in the lumina of the convoluted tubules. No iron was demonstrated in a random selection of sections. Some cells appeared to be breaking down and discharging the brown granules.

Both the brown pigment and hypertrophied cells were found only in the convoluted tubules and were more frequent and numerous in the proximal convoluted tubules. They were also more marked in the 1-year series, and were often absent in the distal convoluted tubules in the 2-year experiments. Discussion of these particles will be presented later. Congestion of interstitial capillaries and glomeruli was insignificant in both the 1- and 2-year experiments. Hyaline casts, sometimes in great numbers, especially in the 2-year series, were present in the straight collecting tubules and in the ducts of Bellini. Serum was present in an occasional glomerular space in a few of the animals, especially those showing very large numbers of casts.

Liver.—The cytoplasm of the cells was finely granular and generally fairly dense. An occasional and infrequent large nucleus was noted but no oxyphil inclusion bodies in the nuclei were present. Periportal lymphocytic infiltration was usually absent; when present it was slight in degree. No differences were noted in the livers of the animals from the 1-year and the 2-year experiments and in the controls.

No changes of note occurred in the stomach, duodenum, jejunum, ileum, large intestine, pancreas, and heart.

LEAD ARSENATE

The pathologic changes observed in this series of animals resembled the changes resulting from ingestion of calcium arsenate but differed in degree of involvement.

The *spleens* of the 1-year group of animals showed findings similar to those observed in the calcium-arsenate series. The degree of splenic myelosis was slightly less. Hemosiderosis was the same but the other changes were less prominent. In the 2-year series the amount of hemosiderin was less than in the 1-year animals and in the calcium-arsenate-fed rats.

Iron-bearing pigment was present to a large extent in the pulp but was also noted in the perifollicular zone of anemia and in a few of the follicles.

The *kidneys* showed the same changes noted with calcium arsenate but a few intranuclear oxyphil inclusions not present in the calcium-arsenate group were found. These inclusions were more numerous in the 2-year animals. The swollen cells of the convoluted tubules were more numerous and the amount of brown pigment was greater in the 1-year series than in the 2-year series. The position of the brown granules in the cells of the convoluted tubules was the same and they were noted more often in the cells of the distal tubules than in the same region in the calcium-arsenate series. The number of casts was less than observed with calcium arsenate in the 2-year series. Congestion was occasionally noted in the 1- and 2-year groups but appeared to be of no importance.

The other organs showed nothing of note.

LEAD CARBONATE

The pathologic changes in the lead-carbonate group of animals were in general similar to those observed in the animals fed calcium arsenate and lead arsenate.

Hemosiderosis of the *spleen* was much less marked than with calcium arsenate and lead arsenate and was occasionally absent. Follicular phagocytosis was also diminished. Splenic myelosis was less marked than in the controls. Lead carbonate was the only one of the three compounds tested which caused a significant reduction in splenic myelosis.

The *kidneys* showed a marked increase in the number of swollen cells with large vesicular nuclei. Large oxyphil intranuclear inclusions were numerous and conspicuous and occurred in a large number of the swollen cells. The brown granules noted in the other two groups were present. They were more numerous in the proximal convoluted tubules where they were more prominent than in the distal tubules. These particles were noted in the lumina and in the cyto-

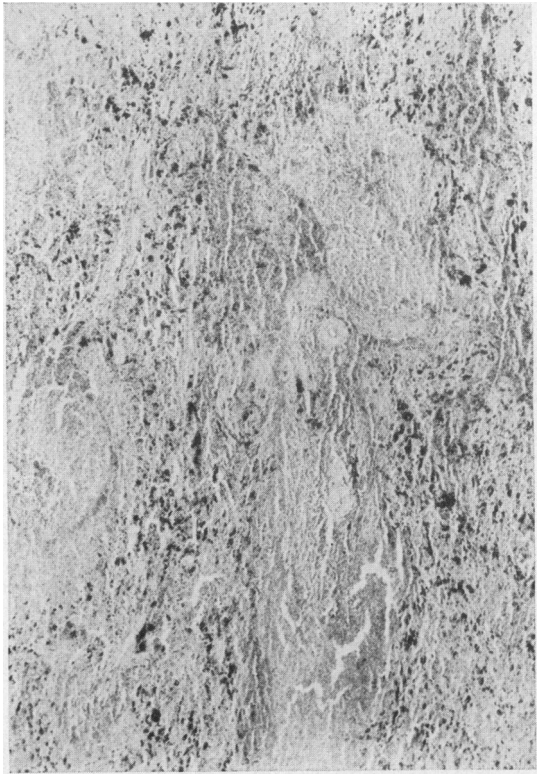

FIGURE 3.—Spleen of rat fed calcium arsenate for 2 years; treated with ferrocyanide to show amount of distribution of hemosiderin. (87 ×)

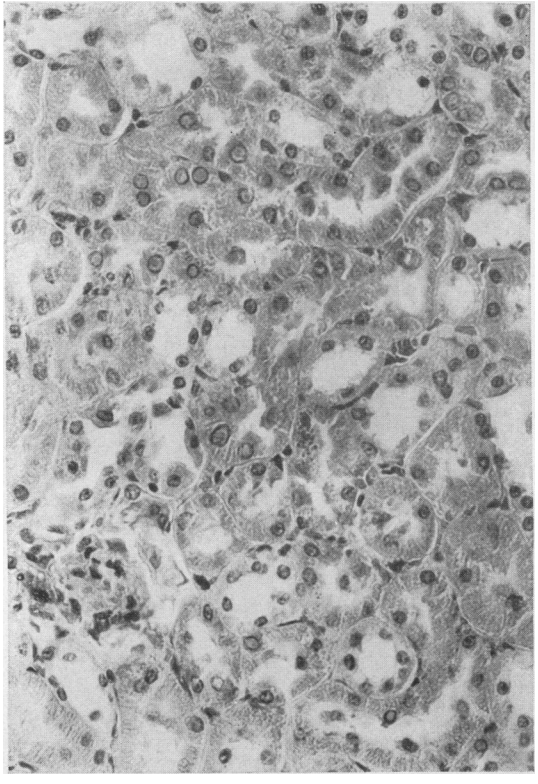

FIGURE 4.—Kidney of rat fed lead carbonate for 2 years. Large cell and nuclei with oxyphil intranuclear inclusion body adjacent to cross section of convoluted tubule with brown cytoplasmic granules. Other large nuclei and pigment particles are seen in the section. (495 ×)

plasm of large and normal-sized convoluted tubule cells. The number of casts was less than in the animals fed the other two compounds, but was definitely increased in the 2-year series.

The other organs showed essentially no change.

DISCUSSION OF PATHOLOGY

In the spleen, the most significant findings were the presence of hemosiderin and the behavior of the myeloid (hemopoietic) tissue. Hemosiderosis has been generally accepted by most investigators as signifying blood destruction. Myeloblasts, as Naegeli (*13*) believes, following the idea of the dual origin of the blood cells, give rise to granulocytes, monocytes, and erythrocytes. The presence of myeloid cells, usually in large numbers, has been observed in the spleens of untreated rats in this laboratory. Jaffe (*14*) states that great numbers of a "neutrophilic element," which from his description are evidently myelocytes, are found in the spleens of rats but rarely occur in rabbits and guinea pigs. Hence, it appears that myeloid cells are a usual histologic finding in the spleen of the rat and are associated with blood formation. The term splenic myelosis seems preferable to hyperplasia or metaplasia in describing this erythroleucopoietic activity in rats and is so used throughout this discussion.

Hemosiderosis occurred in the following order of decreasing magnitude in the four groups of animals: Calcium arsenate, lead arsenate, lead carbonate, and the controls. In the control group, only traces were found in a few of the animals. The value of splenic hemosiderosis as an indication of destruction of red blood cells and its use as a pathologic index of the relative toxicity of lead and its compounds has been previously mentioned (*10*). This seems to be borne out further by these experiments. Splenic myelosis was present in the animals fed calcium arsenate, less in those fed lead arsenate, and most in the control animals, but was definitely reduced in the lead-carbonate group. Thus, lead carbonate appears to have less destructive action on the blood than calcium arsenate and lead arsenate as indicated by hemosiderosis in this particular instance, but interferes with blood regeneration as shown by decreased splenic myelosis. This would seem to indicate that lead anemia to some extent may be caused by interference with blood formation.

Nuclear fragments in the splenic follicles, indicative of recent cellular destruction, were not a significant finding in any group of animals.

All three compounds caused well-defined pathologic changes in the kidney. The outstanding finding was the occurrence of large swollen cells in the proximal and to a lesser extent in the distal convoluted tubules. Tubules lined entirely by these large cells occurred singly and in clusters. Sometimes the cells were so large as to occlude the lumina. Their cytoplasm was granular and radial striations were

absent. The nuclei were large, vesicular, and contained a relatively small amount of chromatin. In the lead-carbonate series, a strongly oxyphil body, usually very large, was present in many of the large nuclei and occurred in all of the anmals. In the lead-arsenate series, most of the animals showed only a few of the intranuclear inclusions and they were absent in the calcium-arsenate group and in the controls in both 1- and 2-year series. Blackman (*15*) describes these acidophilic bodies in detail and attributes them to lead poisoning.

Peculiar brown granules of widely varying size were also seen in the cells of the convoluted tubules. They were round and occurred in both swollen and normal-sized cells and were found free in the lumina. They were more frequent in the proximal than in the distal convoluted tubules, and were most numerous in both the 1- and 2-year lead-carbonate series and the 1-year calcium-arsenate groups and less numerous in the 1- and 2-year lead-arsenate-fed animals. A few were observed in the controls, mostly in the 2-year group. The brown particles failed to show the presence of iron with ferrocyanide in all but one of about 20 animals so tested. They were insoluble in concentrated sulfuric acid after 72 hours. They were insoluble in strong ammonia (6 hours). Hydrogen peroxide (2 percent) failed to bleach the particles in 1 hour and hydrogen sulfide produced no color change. The particles showed no birefringence with polarized light. Potassium bichromate caused no change in color in 9 days and showed no reaction for lead. Acid para-amidoazobenzaldehyde, a test for bile salts, gave no reaction. On differentiation with 95 percent alcohol, the particles did not retain basic fuchsin. Thoroughly deparaffined formol-fixed sections on quartz slides failed to fluoresce when viewed with the ultraviolet microscope at 3,650Å, isolated by a monochromator, but this does not wholly eliminate the porphyrins, as insufficient information exists as to the value of lack of fluorescence in fixed tissues. It is entirely possible, according to Lillie (*16*), that the particles are hemosiderin from which the iron has been dissolved.

Hyaline casts were present in the straight collecting tubules and ducts of Bellini in a number of the test animals but were absent in the controls. They were particularly numerous in the 2-year calcium-arsenate group. Congestion of the interstitial capillaries and glomeruli was insignificant and often absent. Interstitial lymphocytic infiltration was demonstrated in only four of the entire number of rats examined.

The livers of all of the animals showed no histologic changes of note. In a few animals, scattered throughout the three series, the presence of a relatively large basophilic nucleolus was noted in some of the nuclei of the liver cells. The hepatic acidophilic inclusions mentioned by Blackman (*15*) were not encountered.

Sections from the heart, pancreas, stomach, duodenum, jejunum, ileum, and large intestine showed nothing of note.

CONCLUSIONS

An investigation of the effect of ingestion of lead arsenate, extending over 2 years, was made on rats in order to determine whether the lead or the arsenic component of the molecule, or whether these components in combination, were chiefly responsible for the toxicity of the substance.

Lead arsenate was compared with calcium arsenate on the one hand and lead carbonate on the other.

Based upon mortality rates over the 2-year period, the order of toxicity of the three substances at equivalent levels of intake was as follows: Calcium arsenate was most toxic, lead arsenate less, and lead carbonate least toxic.

Pathologic studies showed significant changes in the kidney and spleen.

The large hyperregenerative cells with large vesicular nuclei and cytoplasmic brown pigment granules in the renal convoluted tubules were most frequent in rats fed lead carbonate, less with lead arsenate, and least with calcium arsenate. The large oxyphil intranuclear inclusions appeared in the same order in the animals fed lead carbonate and lead arsenate but were absent in the calcium-arsenate group. This seems to indicate that lead is the causative agent for these reactions.

Splenic hemosiderosis, considered indicative of blood destruction, occurred in greater amounts in the rats fed calcium arsenate and lead arsenate than in those fed lead carbonate. Splenic myelosis (erythroleucopoietic activity) was distinctly reduced in the lead-carbonate series but not appreciably diminished in the calcium-arsenate and lead-arsenate series or in the control groups. If splenic hemosiderosis is accepted as a sign of blood destruction and splenic myelosis is accepted as signifying blood formation, it appears that the action of lead carbonate on the spleen in rats may be both hypoplastic and hemolytic while that of the arsenate radical is primarily hemolytic. The degree of both splenic myelosis and hemosiderosis found in the animals fed the three compounds decreased in the following order: Calcium arsenate, lead arsenate, and lead carbonate, which parallels the order of toxicity as determined by the mortality rates.

The distribution of lead and arsenic in the tissues of the 1- and 2-year groups indicates less storage of lead than of arsenic in the soft tissues of animals fed lead arsenate.

The kidney content of arsenic in the calcium-arsenate group was distinctly greater than that of the lead-arsenate group, both in the 1-year and in the 2-year animals.

The concentration of arsenic in the liver of the 1-year calcium-arsenate group was somewhat less than that of the 1-year lead-arsenate group but greater in the 2-year animals.

With reference to the two lead compounds studied, there is a greater degree of deposition of lead in the tissues of the rats given lead carbonate than in those given lead arsenate, both in the 2-year and 1-year groups. This is strikingly apparent in the case of bone-deposited lead where practically twice as much lead was deposited in the bones of the lead-carbonate group as in the lead-arsenate group.

Since bone-deposited lead is a somewhat safer index of absorption than the degree of lead deposition in the softer tissues, it would appear that the arsenate radical either decreases the absorption or increases the excretion of lead.

ACKNOWLEDGMENT

The authors wish to thank Surgeon P. A. Neal and Passed Assistant Surgeon W. C. Dreessen for numerous suggestions, also Junior Scientific Aide F. L. Weaver, Jr., and Assistant Scientific Aide S. W. Jones for analytical and technical assistance, Medical Technician E. C. Thompson and Junior Medical Technician A. S. Gerber for technical assistance in preparing histological sections, and Bacteriological Technician R. D. Reed for the photomicrography.

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DEATHS DURING WEEK ENDED JULY 26, 1941

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

	Week ended July 26, 1941	Corresponding week, 1940
Data from 88 large cities of the United States:		
Total deaths	7,601	8,855
Average for 3 prior years	7,696	
Total deaths, first 30 weeks of year	261,597	262,759
Deaths per 1,000 population, first 30 weeks of year, annual rate	12.2	12.2
Deaths under 1 year of age	555	546
Average for 3 prior years	510	
Deaths under 1 year of age, first 30 weeks of year	15,762	15,128
Data from industrial insurance companies:		
Policies in force	64,389,697	65,055,294
Number of death claims	9,975	11,718
Death claims per 1,000 policies in force, annual rate	8.1	9.4
Death claims per 1,000 policies, first 30 weeks of year, annual rate	9.9	10.0

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 AUGUST 2, 1941

Summary

Although another increase was recorded in the number of cases of poliomyelitis, the rise was less sharp than in the two preceding weeks. For the current week 326 cases were reported (8 percent increase) as compared with 302 for the preceding week (an increase of 23 percent). The 5-year (1936–40) median expectancy is 197. The largest number of cases for the corresponding week of the past 5 years (414) was reported in 1937, in which year 2,485 cases had been reported to August 7, as compared with 1,851 cases to date this year.

The highest incidence continues in the South Atlantic and East South Central States, which reported 200 cases, or 61 percent of the current total, as compared with 70 percent of the total for the preceding week. Larger increases were recorded for the current period in some of the Northern States than were reported last week. The States currently reporting more than 10 cases are as follows, with last week's figures in parentheses: Georgia, 71 (79); Alabama, 49 (58) Florida, 27 (16); Maryland, 14 (3); Pennsylvania, 15 (8); Ohio, 16 (11); Illinois, 13 (4); Tennessee, 13 (24); New York, 12 (11).

As compared with the preceding week slight increases were also reported for diphtheria, meningococcus meningitis, typhoid fever, and whooping cough. Only 6 cases of smallpox were reported—the same as for last week. Five of the current cases occurred in the North Central States.

North Dakota reported 54 cases of encephalitis, South Dakota 19, Minnesota 35, and Colorado 3. Connecticut reported 7 cases of undulant fever, Maryland 2, and South Dakota and North Carolina 1 case each. Of 70 cases of endemic typhus fever, Texas reported 25 cases, Georgia 19, and Alabama 13.

Marmots from San Miguel County, Colorado, have been found plague infected. This is believed to be the first instance of plague infection reported in that State.

The death rate for the current week in 88 large cities is 11.9 per 1,000 population, as compared with 10.6 for the preceding week and with a 3-year (1938–40) average of 10.8.

Telegraphic morbidity reports from State health officers for the week ended August 2, 1941, and comparison with corresponding week of 1940 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 1936–40	Week ended		Median 1936–40	Week ended		Median 1936–40	Week ended		Median 1936–40
	Aug. 2, 1941	Aug. 3, 1940		Aug. 2, 1941	Aug. 3, 1940		Aug. 2, 1941	Aug. 3, 1940		Aug. 2, 1941	Aug. 3, 1940	
NEW ENG.												
Maine	0	0	0	------	------	------	53	44	25	0	0	0
New Hampshire	0	0	0	1	------	------	3	0	3	0	0	0
Vermont	0	0	0	------	------	------	48	10	10	0	0	0
Massachusetts	2	2	2	------	------	------	125	315	118	3	0	1
Rhode Island	1	0	0	------	------	------	10	29	7	0	0	0
Connecticut	1	0	1	------	1	------	51	9	14	0	2	0
MID. ATL.												
New York [1]	15	7	19	[2] 1	[2] 4	[2] 2	202	351	261	6	2	7
New Jersey	0	4	3	2	1	1	117	200	73	0	0	0
Pennsylvania	6	5	17	------	------	------	264	151	117	3	3	3
E. NO. CEN.												
Ohio [1]	3	4	6	3	6	3	123	22	77	0	0	0
Indiana [3]	4	5	6	4	2	------	32	10	2	0	1	1
Illinois	16	12	15	------	6	3	50	93	25	2	0	2
Michigan [4]	0	5	7	------	4	------	122	193	68	1	0	1
Wisconsin	1	0	1	5	8	10	188	285	32	0	1	1
W. NO. CEN.												
Minnesota [5]	1	0	2	------	2	1	5	18	18	2	1	0
Iowa	1	1	1	------	------	------	25	36	21	1	3	0
Missouri	5	0	6	2	------	18	23	3	3	2	0	0
North Dakota [5]	1	1	2	2	------	2	14	1	1	0	0	0
South Dakota [5]	4	4	1	------	------	------	0	3	------	0	0	0
Nebraska	0	0	1	------	------	------	2	6	2	0	0	0
Kansas [1]	2	5	2	1	1	1	21	15	7	1	1	1
SO. ATL.												
Delaware [3]	0	0	0	------	------	------	2	1	------	0	0	0
Maryland [3 4]	0	1	4	------	1	1	101	6	13	2	0	0
Dist. of Col	0	3	3	------	------	------	11	2	5	0	0	0
Virginia	5	21	17	61	15	------	102	43	35	2	0	1
West Virginia [4]	2	3	3	6	2	6	24	6	6	1	1	0
North Carolina [1 3]	7	9	9	------	------	------	63	26	26	0	1	2
South Carolina [1]	1	3	8	61	116	63	63	19	9	0	1	1
Georgia [1]	9	1	16	10	3	------	44	11	------	0	1	2
Florida [1]	2	4	4	23	------	1	25	6	4	0	0	0
E. SO. CEN.												
Kentucky	1	5	4	------	1	1	21	41	5	1	2	2
Tennessee	2	1	4	9	6	6	33	9	7	2	0	1
Alabama [1]	8	9	11	2	5	5	12	30	3	1	0	1
Mississippi [4]	4	2	9	------	------	------	------	------	------	0	2	0
W. SO. CEN.												
Arkansas	2	2	5	2	15	5	32	2	2	0	0	0
Louisiana	0	3	8	1	2	9	2	2	2	0	0	0
Oklahoma	1	2	3	7	9	5	16	1	3	0	0	0
Texas [1]	27	14	21	345	137	53	101	70	34	1	1	2
MOUNTAIN												
Montana	0	6	1	5	------	------	0	17	12	0	1	0
Idaho	0	1	0	------	------	1	0	4	4	0	0	0
Wyoming	5	1	1	3	------	------	2	3	3	0	0	0
Colorado [5]	8	7	7	22	------	------	31	5	9	0	0	0
New Mexico	0	0	2	2	------	------	26	14	6	0	0	0
Arizona	1	1	1	22	14	10	29	13	9	0	0	0
Utah [4]	0	0	0	------	------	------	8	19	12	0	0	0
Nevada	0	------	------	------	------	------	0	------	------	0	------	------
PACIFIC												
Washington	1	1	1	------	------	------	5	7	16	0	0	C
Oregon	0	1	1	4	------	3	7	13	13	0	0	0
California [1]	15	8	16	38	8	8	111	82	91	0	0	1
Total	164	164	272	644	369	263	2,349	2,246	1,153	31	24	33
31 weeks	7,287	8,535	13,093	598,539	168,338	151,020	827,376	225,925	268,586	1,359	1,118	2,072

See footnotes at end of table.

Telegraphic morbidity reports from State health officers for the week ended August 2, 1941, and comparison with corresponding week of 1940 and 5-year median—Con.

Division and State	Poliomyelitis			Scarlet fever			Smallpox			Typhoid and paratyphoid fever		
	Week ended		Median 1936–40	Week ended		Median 1936–40	Week ended		Median 1936–40	Week ended		Median 1936–40
	Aug. 2, 1941	Aug. 3, 1940		Aug. 2, 1941	Aug. 3, 1940		Aug. 2, 1941	Aug. 3, 1940		Aug. 2, 1941	Aug. 3, 1940	
NEW ENG.												
Maine	4	1	1	0	2	10	0	0	0	2	7	2
New Hampshire	0	0	0	1	0	0	0	0	0	1	0	0
Vermont	0	0	0	2	1	1	0	0	0	0	1	1
Massachusetts	5	1	1	64	26	31	0	0	0	4	3	2
Rhode Island	1	0	0	2	0	2	0	0	0	0	0	0
Connecticut	6	0	0	4	6	7	0	0	0	2	6	3
MID. ATL.												
New York [1]	12	4	9	80	73	71	0	0	0	16	11	18
New Jersey	5	0	2	37	27	20	0	0	0	4	5	7
Pennsylvania	15	0	1	40	48	72	0	0	0	13	14	21
E. NO. CEN.												
Ohio [1]	16	13	7	49	72	72	0	0	1	17	8	21
Indiana [3]	5	15	1	9	8	19	1	0	0	4	0	6
Illinois	13	7	7	46	59	82	0	8	7	21	20	19
Michigan [4]	8	8	8	44	57	60	2	1	1	4	4	4
Wisconsin	3	0	0	37	28	48	0	5	2	1	0	3
W. NO. CEN.												
Minnesota [5]	3	2	4	10	11	25	0	1	1	0	1	1
Iowa	1	9	3	9	21	21	1	4	4	1	5	5
Missouri	1	9	2	24	5	15	0	1	1	8	10	14
North Dakota [5]	0	0	0	1	8	5	0	0	0	0	0	0
South Dakota [5]	5	3	1	3	4	5	1	3	3	0	0	1
Nebraska	0	1	3	1	3	4	0	0	2	0	4	0
Kansas [1]	0	23	4	6	15	23	0	0	1	6	7	7
SO. ATL.												
Delaware [3]	0	0	0	1	1	0	0	0	0	0	0	0
Maryland [3 4]	14	0	1	23	8	8	0	0	0	8	4	11
Dist. of Col	0	0	0	2	1	1	0	0	0	0	0	0
Virginia	4	1	3	5	18	9	0	0	0	4	10	19
West Virginia [4]	1	13	1	12	13	13	0	0	0	1	9	10
North Carolina [1 3]	0	1	2	16	19	17	0	0	0	16	10	18
South Carolina [1]	5	0	0	0	2	2	0	0	0	3	19	13
Georgia [1]	71	0	5	10	8	8	0	0	0	13	29	36
Florida [1]	[6] 27	1	1	2	2	3	0	0	0	4	4	4
E. SO. CEN.												
Kentucky	7	6	3	16	17	17	0	0	0	14	8	39
Tennessee	13	1	3	16	8	9	0	4	1	13	12	32
Alabama [1]	49	1	1	14	13	11	0	0	0	7	11	19
Mississippi [4]	9	0	4	5	6	6	0	0	0	16	13	13
W. SO. CEN.												
Arkansas	1	1	2	1	3	3	1	0	0	20	31	31
Louisiana	5	11	0	3	5	5	0	0	0	9	16	16
Oklahoma	0	6	0	4	9	7	0	5	0	1	24	19
Texas [1]	4	7	7	14	8	21	0	0	0	42	37	57
MOUNTAIN												
Montana	0	8	1	3	2	4	0	0	0	1	0	1
Idaho	0	4	1	0	3	3	0	0	1	0	0	1
Wyoming	0	0	0	0	1	3	0	0	0	1	0	1
Colorado [5]	2	0	1	8	3	9	0	1	1	1	0	2
New Mexico	0	1	0	0	3	4	0	0	0	6	5	5
Arizona	0	0	0	1	3	1	0	0	0	1	0	1
Utah [4]	1	1	0	3	2	6	0	0	0	2	2	1
Nevada	0	------	------	0	------	------	0	------	------	0	------	------
PACIFIC												
Washington	1	15	1	8	24	18	0	0	0	4	4	2
Oregon	1	3	1	7	6	6	0	1	1	3	2	2
California [1]	8	20	20	40	43	49	0	0	7	7	23	16
Total	[6] 326	197	197	677	705	927	6	34	52	301	379	497
31 weeks	1,851	1,403	1,403	90,786	117,703	135,655	1,173	1,927	7,847	3,813	4,208	6,096

See footnotes at end of table.

Telegraphic morbidity reports from State health officers for the week ended August 2, 1941, and comparison with corresponding week of 1940—Continued

Division and State	Whooping cough	
	Week ended	
	Aug. 2, 1941	Aug. 3, 1940
NEW ENG.		
Maine	19	84
New Hampshire	16	0
Vermont	11	35
Massachusetts	195	108
Rhode Island	22	4
Connecticut	49	46
MID. ATL.		
New York [1]	235	311
New Jersey	99	76
Pennsylvania	229	437
E. NO. CEN.		
Ohio [1]	343	467
Indiana [2]	13	17
Illinois	164	181
Michigan [4]	317	272
Wisconsin	225	99
W. NO. CEN.		
Minnesota [5]	53	30
Iowa	53	36
Missouri	72	36
North Dakota [5]	30	3
South Dakota [5]	7	6
Nebraska	7	2
Kansas [1]	79	53
SO. ATL.		
Delaware [3]	2	6
Maryland [3 4]	84	141
District of Columbia	20	6
Virginia	50	56
West Virginia [4]	13	27
North Carolina [1 3]	244	111
South Carolina [1]	104	35
Georgia [1]	34	13
Florida [1]	36	2
E. SO. CEN.		
Kentucky	61	71
Tennessee	44	71
Alabama [1]	22	10
Mississippi [4]	----------	----------
W. SO. CEN.		
Arkansas	7	11
Louisiana	17	7
Oklahoma	39	25
Texas [1]	232	209
MOUNTAIN		
Montana	29	1
Idaho	11	11
Wyoming	6	5
Colorado [6]	123	30
New Mexico	17	34
Arizona	16	17
Utah [4]	29	76
Nevada	0	----------
PACIFIC		
Washington	110	59
Oregon	29	19
California [1]	335	317
Total	3,952	3,673
31 weeks	139,624	100,575

[1] Typhus fever, week ended Aug. 2, 1941, 70 cases as follows: New York, 2; Ohio, 1; Kansas, 1; North Carolina, 1; South Carolina, 3; Georgia, 19; Florida, 4; Alabama, 13; Texas, 25; California, 1.
[2] New York City only.
[3] Rocky Mountain spotted fever, week ended Aug. 2, 1941, 5 cases as follows: Indiana, 1; Delaware, 1; Maryland, 2; North Carolina, 1.
[4] Period ended earlier than Saturday.
[5] Encephalitis, week ended Aug. 2, 1941, 111 cases as follows: Minnesota, 35; North Dakota, 54; South Dakota, 19; Colorado, 3.
[6] Delayed report of 6 cases included.

PLAGUE INFECTION IN CALIFORNIA AND MONTANA

IN FLEAS FROM GROUND SQUIRRELS IN KERN AND SISKIYOU COUNTIES, CALIF.

Under dates of July 24 and 26, 1941, Dr. Bertram P. Brown, State Director of Public Health of California, reported plague infection proved, by animal inoculation and cultures, in a pool of 201 fleas from 10 ground squirrels, *C. beecheyi*, submitted to the laboratory on July 10 from a location 1 mile south and 1 mile east of Keene, Kern County, Calif.; and in 2 pools of fleas from ground squirrels, *C. douglasii*, from

a ranch 8 miles east and 3 miles south of Montague, Siskiyou County, Calif., one a pool of 387 fleas from 8 ground squirrels submitted to the laboratory on July 9, and the other a pool of 290 fleas from 8 ground squirrels submitted on July 11.

IN FLEAS FROM GROUND SQUIRRELS IN BEAVERHEAD COUNTY, MONT.

Under date of July 23, Dr. N. E. Wayson, Medical Officer in Charge, Plague Suppressive Measures, San Francisco, Calif., reported that a pool of 49 fleas from 120 ground squirrels, *C. columbianus*, shot July 8 on a ranch 12 miles west of Wisdom, Beaverhead County, Mont., had been found positive for plague.

WEEKLY REPORTS FROM CITIES

City reports for week ended July 19, 1941

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

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
Data for 90 cities:											
5-year average	78	25	11	964	281	334	5	345	51	1,373	----
Current week [1]	34	23	13	909	200	255	0	316	25	1,448	----
Maine:											
Portland	0	----	0	0	2	0	0	0	0	3	22
New Hampshire:											
Concord	0	----	0	0	1	1	0	0	0	0	6
Manchester	0	----	0	0	0	1	0	0	0	0	11
Nashua	0	----	0	0	0	0	0	0	0	3	----
Vermont:											
Barre	0	----	0	0	0	0	0	0	0	0	1
Burlington	0	----	0	0	0	0	0	0	0	0	8
Rutland	0	----	0	0	0	0	0	0	0	0	10
Massachusetts:											
Boston	0	----	0	47	12	18	0	14	1	35	213
Fall River	0	----	0	0	1	1	0	1	0	0	20
Springfield	0	----	0	24	0	2	0	0	0	3	25
Worcester	0	----	0	4	4	9	0	0	0	9	45
Rhode Island:											
Pawtucket	0	----	0	0	0	0	0	0	0	0	10
Providence	1	----	0	13	0	5	0	1	0	28	50
Connecticut:											
Bridgeport	0	----	0	11	2	0	0	0	0	3	35
Hartford	0	----	0	1	1	3	0	0	0	2	31
New Haven	0	----	0	3	0	3	0	1	0	3	33
New York:											
Buffalo	0	----	1	8	4	7	0	4	0	15	112
New York	10	1	1	89	28	28	0	69	6	142	1,142
Rochester	0	----	0	22	1	1	0	0	0	9	49
Syracuse	0	----	0	17	0	2	0	0	0	20	36
New Jersey:											
Camden	0	----	0	2	2	0	0	0	0	3	23
Newark	0	1	1	20	4	6	0	14	0	19	105
Trenton	0	----	0	4	0	3	0	1	1	0	29
Pennsylvania:											
Philadelphia	0	----	0	14	11	13	0	25	2	54	379
Pittsburgh	1	2	3	30	5	2	0	5	1	43	122
Reading	0	----	0	2	0	0	0	2	0	1	21
Scranton	0	----	----	17	----	0	0	----	0	2	----
Ohio:											
Cincinnati	0	----	0	0	2	2	0	5	0	11	148
Cleveland	0	----	0	6	1	12	0	5	0	57	166
Columbus	0	----	0	9	0	3	0	3	0	12	81
Toledo	0	----	0	125	2	2	0	4	0	60	77

[1] Figures for South Bend estimated; report not received.

City reports for week ended July 19, 1941—Continued

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
Indiana:											
Anderson	0	------	0	3	0	0	0	0	0	0	2
Fort Wayne	0	------	0	1	1	0	0	1	0	0	24
Indianapolis	1	------	0	8	5	1	0	5	0	4	86
Muncie	0	------	0	0	1	1	0	1	0	0	7
Terre Haute	0	------	0	1	1	0	0	0	0	0	16
Illinois:											
Alton	0	------	0	1	2	0	0	0	0	0	12
Chicago	4	------	1	16	14	30	0	26	0	95	597
Elgin	0	------	0	0	0	0	0	0	0	6	3
Moline	0	------	0	0	0	0	0	0	0	3	10
Springfield	0	------	0	26	0	1	0	0	0	1	20
Michigan:											
Detroit	1	------	0	58	8	34	0	9	1	103	225
Flint	0	------	0	1	4	2	0	0	0	10	23
Grand Rapids	0	------	0	20	1	2	0	0	2	1	32
Wisconsin:											
Kenosha	0	------	0	2	0	0	0	0	0	0	5
Milwaukee	0	------	0	101	1	6	0	2	0	65	74
Racine	0	------	0	33	0	0	0	0	0	3	--------
Superior	0	------	0	2	0	1	0	0	0	23	9
Minnesota:											
Duluth	0	------	0	0	0	0	0	0	0	22	19
Minneapolis	0	------	0	4	1	5	0	0	0	19	84
St. Paul	0	------	0	0	5	1	0	3	0	16	50
Iowa:											
Cedar Rapids	0	------	--------	0	-------	0	0	-------	0	1	--------
Davenport	0	------	--------	3	-------	1	0	-------	0	0	--------
Des Moines	0	------	--------	0	-------	0	0	-------	1	9	28
Sioux City	0	------	--------	1	-------	0	0	-------	0	5	--------
Waterloo	0	------	--------	5	-------	2	0	-------	0	0	--------
Missouri:											
Kansas City	0	------	0	21	1	4	0	2	0	6	86
St. Joseph	0	------	0	0	3	0	0	0	0	1	13
St. Louis	0	------	0	11	4	3	0	5	1	43	168
North Dakota:											
Fargo	0	------	0	0	0	0	0	0	0	6	9
Grand Forks	0	------	--------	0	-------	0	0	-------	0	0	--------
Minot	1	------	0	2	0	0	0	0	0	0	5
South Dakota:											
Aberdeen	0	------	--------	0	-------	0	0	-------	0	0	--------
Sioux Falls	0	------	--------	0	-------	0	0	-------	0	0	11
Nebraska:											
Lincoln	1	------	--------	5	-------	0	0	-------	0	7	--------
Omaha	0	------	0	2	1	1	0	0	0	2	53
Kansas:											
Lawrence	0	------	0	0	0	0	0	0	0	5	1
Topeka	0	------	0	0	1	4	0	0	0	32	10
Wichita	0	------	0	0	1	0	0	0	0	2	27
Delaware:											
Wilmington	0	------	0	0	2	0	0	0	0	0	19
Maryland:											
Baltimore	0	1	1	128	3	8	0	16	0	82	179
Cumberland	0	------	0	1	0	0	0	0	1	0	16
Frederick	0	------	0	0	0	0	0	0	0	0	1
District of Columbia:											
Washington	0	------	0	30	4	1	0	17	0	9	130
Virginia:											
Lynchburg	0	------	0	19	0	0	0	1	0	2	10
Norfolk	0	------	0	11	2	0	0	1	0	0	27
Richmond	0	------	0	2	0	0	0	2	0	0	49
Roanoke	0	------	0	2	0	0	0	0	0	5	17
West Virginia:											
Charleston	0	------	0	0	2	0	0	0	0	1	18
Huntington	0	------	--------	0	-------	0	0	-------	0	2	--------
Wheeling	0	------	0	4	0	2	0	0	1	10	18
North Carolina:											
Gastonia	0	------	--------	0	-------	0	0	-------	0	4	--------
Raleigh	0	------	0	0	1	0	0	0	0	23	7
Wilmington	0	------	0	3	1	1	0	0	0	15	8
Winston-Salem	1	------	0	4	1	0	0	1	0	2	13
South Carolina:											
Charleston	0	------	1	2	2	0	0	1	1	3	22
Florence	0	------	0	0	0	0	0	0	0	1	16
Greenville	0	------	0	0	0	0	0	0	0	2	8

City reports for week ended July 19, 1941—Continued

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
Georgia:											
Atlanta	0	3	1	0	4	1	0	6	0	0	79
Brunswick	0	------	0	0	1	0	0	0	0	1	6
Savannah	0	------	0	6	1	1	0	1	0	1	34
Florida:											
Miami	0	------	0	1	2	0	0	2	1	3	32
St. Petersburg	0	------	0	0	0	0	0	0	0	0	16
Tampa	0	------	0	0	2	0	0	1	0	2	27
Kentucky:											
Ashland	1	------	0	3	0	1	0	0	0	2	5
Covington	0	1	0	0	1	1	0	1	0	0	18
Lexington	1	------	0	0	0	0	0	3	0	3	12
Louisville	0	------	0	14	2	3	0	1	1	19	74
Tennessee:											
Knoxville	0	------	0	2	0	1	0	1	1	1	31
Memphis	0	------	0	10	1	0	0	3	0	27	73
Nashville	0	------	0	1	3	1	0	5	0	5	--------
Alabama:											
Birmingham	0	------	0	2	0	0	0	5	1	2	77
Mobile	0	------	0	1	2	1	0	0	0	0	24
Montgomery	0	------	--------	0	-------	1	0	-------	0	2	--------
Arkansas:											
Fort Smith	0	------	--------	0	-------	0	0	-------	0	0	--------
Little Rock	0	------	0	1	4	1	0	5	0	0	31
Louisiana:											
Lake Charles	0	------	0	0	0	0	0	0	0	0	2
New Orleans	3	1	1	1	4	1	0	15	2	43	140
Shreveport	0	------	0	0	2	0	0	1	0	0	28
Oklahoma:											
Oklahoma City	0	------	0	2	2	0	0	3	0	1	31
Tulsa	0	------	0	3	0	0	0	2	0	0	24
Texas:											
Dallas	1	1	1	12	2	0	0	0	1	1	55
Fort Worth	0	------	0	0	1	0	0	0	0	2	38
Galveston	0	------	0	0	3	0	0	2	0	0	19
Houston	0	------	0	3	1	1	0	7	2	1	81
San Antonio	0	------	0	0	1	0	0	3	0	1	63
Montana:											
Billings	0	------	0	0	1	0	0	0	0	0	13
Great Falls	0	------	0	1	1	0	0	0	0	5	5
Helena	0	------	0	2	0	1	0	0	0	1	7
Missoula	2	------	0	0	0	0	0	0	0	0	6
Idaho:											
Boise	0	------	0	0	0	0	0	1	0	1	4
Colorado:											
Colorado Springs	0	------	0	2	0	0	0	1	0	6	7
Denver	6	3	0	8	1	0	0	0	0	112	62
Pueblo	0	------	0	3	0	0	0	0	0	3	10
New Mexico:											
Albuquerque	0	------	0	3	2	1	0	0	0	2	21
Arizona:											
Phoenix	0	10	0	7	0	0	0	0	0	9	--------
Utah:											
Salt Lake City	0	------	0	2	2	0	0	0	0	23	34
Washington:											
Seattle	0	------	0	1	3	1	0	1	0	21	97
Spokane	0	------	0	1	2	3	0	0	0	9	37
Tacoma	0	------	0	0	0	1	0	0	0	13	23
Oregon:											
Portland	0	------	0	2	2	1	0	1	0	0	78
Salem	0	1	--------	0	-------	0	0	-------	0	0	--------
California:											
Los Angeles	1	9	0	16	3	7	0	9	0	58	306
Sacramento	2	------	0	1	2	2	0	1	0	15	31
San Francisco	0	1	1	4	5	2	0	9	0	24	158

City reports for week ended July 19, 1941—Continued

State and city	Meningitis, meningococcus		Poliomyelitis cases
	Cases	Deaths	
New York:			
Buffalo	1	0	0
New York	1	1	4
Ohio:			
Cleveland	0	0	4
Illinois:			
Chicago	1	0	1
Michigan:			
Detroit	0	0	4
Wisconsin:			
Madison	0	0	1
Superior	0	0	1
Maryland:			
Baltimore	3	0	3
District of Columbia:			
Washington	0	0	1
South Carolina:			
Charleston	0	0	1
Georgia:			
Atlanta	0	0	19
Savannah	0	0	1
Florida:			
Tampa	0	0	1
Kentucky:			
Louisville	0	0	2
Tennessee:			
Knoxville	0	0	1
Alabama:			
Birmingham	0	0	15
Montgomery	0	0	1
California:			
San Francisco	1	1	1

Encephalitis, epidemic or lethargic.—Cases: New York, 2; Toledo, 2; Fargo, 12; Minot, 6; Baltimore, 1; Birmingham, 1. Deaths: Fargo, 1; Baltimore, 1; Birmingham, 1.

Pellagra.—Cases: Boston, 1; Baltimore, 1; Savannah, 3; Montgomery, 1.

Typhus fever.—Cases: Miami, 1; Birmingham, 1; Mobile, 3; New Orleans, 1; Dallas, 2; Houston, 1. Deaths: Miami, 1.

FOREIGN REPORTS

CANADA

Provinces—Communicable diseases—Week ended June 28, 1941.—During the week ended June 28, 1941, cases of certain communicable diseases were reported by the Department of Pensions and National Health of Canada as follows:

Disease	Prince Edward Island	Nova Scotia	New Brunswick	Quebec	Ontario	Manitoba	Saskatchewan	Alberta	British Columbia	Total
Cerebrospinal meningitis	1	2	2	2	11	2			3	23
Chickenpox		13		72	184	52	34	84	36	475
Diphtheria	1	12		43	6	4	1			67
Dysentery				9						9
Influenza		2			4	1			1	8
Measles	1	4	2	97	729	28	27	21	106	1,015
Mumps	2			99	133	14	11	5	2	266
Pneumonia	5	7			2				5	19
Poliomyelitis				1		2				3
Scarlet fever	5	10	4	44	153	12	8	7	15	258
Trachoma									1	1
Tuberculosis	2	11	22	90	56	2		1		184
Typhoid and paratyphoid fever				12	5			1		18
Whooping cough				85	124	1		2	20	232

CANADA

Manitoba—Poliomyelitis.—Information received under date of August 1, 1941, states that 90 new cases of poliomyelitis were reported in the Province of Manitoba for the week ended July 31, making a total of 191 cases, with 8 deaths, during the month of July.

The disease was stated to have spread from the original focus in Winnipeg, where most of the cases have occurred, to rural districts within a radius of 60 miles of the city. A few cases have been reported from remote localities in the Province. In only two instances has more than one member of a family been attacked.

The records so far indicate that 40 percent of the cases have shown muscular weakness or paralysis in some degree, not all of which, however, were severe.

Therapeutic serum is being administered. Donors are not limited to persons who have recently had the disease, but include those who have had the disease at any time, and preferably those who were afflicted with paralysis. The average amount of serum used was

stated to be about 20 cc. per patient, administered within 3 or 4 days from initial onset of symptoms, provided no paralysis is apparent.

The report states that unusually high temperatures are prevailing in Manitoba.

JAMAICA

Communicable diseases—4 weeks ended July 5, 1941.—During the 4 weeks ended July 5, 1941, cases of certain communicable diseases were reported in Kingston, Jamaica, and in the island outside of Kingston, as follows:

Disease	Kingston	Other localities	Disease	Kingston	Other localities
Chickenpox	5	7	Puerperal fever	----	2
Dysentery	----	3	Tuberculosis	19	92
Erysipelas	----	1	Typhoid fever	4	31
Leprosy	----	3			

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