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PHARMACOLOGICAL AND CHEMICAL STUDIES OF THE CAUSE OF SO-CALLED GINGER PARALYSIS

A PRELIMINARY REPORT

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A peculiar form of paralysis, perhaps unlike anything ever known before, has recently afflicted a relatively large proportion of the population throughout some of the midwestern and southwestern Definite figures on the extent of the disease are not available, but it is certain that the numbers run into the thousands. vestigation of this condition in some of the stricken areas in Ohio and Tennessee seemed to confirm the widespread rumor that the disease is closely associated with the drinking of an adulterated fluid extract of ginger. That it could not be due to the ginger as such became clearly evident from the fact that many of the victims, when questioned, freely admitted having used similar preparations for beverage purposes for from one to five years with no other effects than those derived from the alcohol. It soon became evident, therefore, that the condition must have resulted from some unknown poison or from some known poison whose pharmacologic action was so altered through the ginger or the alcohol, or both, as to render it unrecognizable, which poison in some way got into a manufactured lot of so-called U. S. P. fluid extract of ginger at a relatively recent date.

EPIDEMIOLOGY

It must be borne in mind that the so called U. S. P. fluid extract of ginger has been sold extensively for many years for beverage purposes, mostly and perhaps exclusively throughout the States where the paralysis is prevalent at present. This seems to have followed the ruling of the Prohibition Bureau to the effect that the

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official fluid extract of ginger is a nonpotable beverage, thus removing all restrictions from the sale thereof. Following this, it appears, there soon came upon the market what seems to have been in effect a tincture of ginger adulterated with substances not easily detectable by the chemist and resembling oleoresin of ginger to make it conform with the official fluid extract. Such a preparation has been used very widely as a beverage in the States alluded to.

The close relationship of the disease to the consumption of an adulterated fluid extract of ginger is clearly brought out by the epidemiological studies at the Cincinnati General Hospital by Dr. T. J. LeBlanc, to whom we are greatly indebted for this information. Briefly, Professor LeBlanc's studies indicate that the cases of paralysis. of which they had over 200, first began to show up the latter part of February, that the epidemic reached the peak by the middle of March, and that by April it was well on the decline. Almost exactly the same data were obtained in an epidemiological study by the Tennessee State health department, at Nashville, of 119 cases that occurred for the most part in eastern Tennessee. Both epidemiological studies agree in that the great majority of the patients were males, that it affected all ages between 20 and 80 years, usually about 40, that no cases occurred among children and very infrequently among young adults, and that practically in all cases a history of drinking ginger could be obtained. Another significant fact disclosed by these investigations was that an interval usually of from 10 days to 3 weeks elapsed between the drinking of the suspected ginger and the onset of paralysis. These findings were confirmed by one of us (M. I. S.) by a personal examination early in May of six cases in Ripley, Ohio, and eight cases, including one lawyer and one retired merchant, in Johnson City, Tenn.

Bearing in mind that the "epidemic" first occurred the latter part of February and that there was on an average an "incubation period" of two weeks, it appeared clear that the poisoned ginger must have been shipped for distribution from its source of manufacture (which no one knows) early in February or possibly during the month of January. Furthermore, since the ginger was sold in and about Cincinnati under at least eight different brands and in Johnson City under at least four different brands, and since in no case could paralysis be definitely associated with or definitely dissociated from any one brand, it appears probable that the poisoned "fluid extract" of ginger must have come from some one source at a fairly definite time. The evidence which will follow bears out these early assumptions.

SYMPTOMATOLOGY AND CLINICAL FINDINGS

There is a remarkable uniformity in the history and the symptoms and physical signs. In every case investigated there is a clear history of drinking "U. S. P. fluid extract" of ginger purchased from a local dealer (not from a pharmacist) who distributed the ginger in 1½ or 2 ounce bottles. The brands were different according to the locality sold; Anchor, Peer, Royal, K. D., K. K., Q. C., B. & L., Fulton, Tommac, Deco, Uanaca, and Land were some of the brands drunk by many of the victims questioned. A noteworthy point is the fact that apparently the quantity consumed was not a deciding factor in the outcome, except perhaps in degree. Apparently reliable histories have been obtained indicating that one drink of as little as 1 ounce of the ginger produced essentially the same result, except perhaps for some differences in degree, that followed the drinking of 10, 15, or more 2-ounce bottles over a period of some days. The immediate effects in many cases were none other than those of alcoholic intoxication. In many of the cases there were gastrointestinal disturbances characterized by nausea, vomiting, abdominal pain, and diarrhea. These lasted a day or two. The gastrointestinal disturbances seem to have had no relationship to the brand consumed nor to the ultimate outcome, for fairly definite histories were obtained where two or more in a party drank of the same purchase lot with the early symptoms in some and no immediate effects whatever in others, but with ultimate paralysis in all.

With the exception of the early and transient gastrointestinal disturbances in some of the cases, no effects were noted that could have been definitely attributed to the consumption of the beverage for an interval of from 5 days to 3 weeks, usually about 10 days. The first symptoms were soreness in the muscles of the legs, and only occasionally some numbness in the fingers or toes. The soreness of the leg muscles was usually complained of for several days before it was realized that the toes could not be moved. This was soon followed by bilateral foot drop. In all the cases questioned the onset of the weakness in the fingers and the wrist drop followed the foot drop by an interval of about a week or 10 days, and, upon examination, the disability in the hands and forearms was never so marked as in the feet and legs.

Clinically the victims presented bilateral wrist drop and foot drop of varying degrees of severity. The milder cases could get about with the aid of canes or crutches; the severer cases were bedridden and in many instances were unable to feed themselves. The paralysis in the upper extremities has not been seen to extend beyond the elbows, and in the lower extremities the thigh muscles were seen to be involved in the more advanced cases. There are no sensory

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disturbances, no impairment of tactile, pain, or temperature sensations; the superficial reflexes are normal; the sphincters are normal; there are no visual disturbances; and there is no evidence of involvement of the cranial ganglia or nerves. In brief, the clinical picture is uniformly that of a flaccid paralysis for the most part of the distal muscles of the lower and upper extremities, clearly pointing to involvement of the lower motor neuron remarkably localized to the lower lumbar and lower cervical regions of the cord. Indeed, the only physical sign that in our experience presented any degree of inconstancy is the knee jerk, which has been found diminished or absent in the severer cases, as one would expect, normal or nearly normal in the milder cases, and markedly exaggerated in some of the milder ambulatory cases.¹

THE PROBABLE NATURE OF THE POISON

From the description of the clinical manifestations of the disease considered in the light of the probable etiologic factor it must be evident at once that this form of motor paralysis does not fit in with the known types of multiple peripheral neuritis. It will be readily conceded that it is not pure alcoholic multiple neuritis. Its superficial resemblance to arsenical neuritis has led some observers to suggest arsenic in the ginger as the etiologic factor of this disease. The fact that none or only a trace of arsenic, such as one part per million to one part per ten million, has been found in numerous samples of ginger examined should be sufficient to render untenable the arsenic theory. If by some remote chance the implicated ginger which, for the sake of argument, escaped the chemists' notice, became heavily contaminated with arsenic, large doses of the ginger, which many victims admit having drunk, should certainly have resulted in some deaths with clinical manifestations of arsenic poisoning other than the paralysis. Lastly, the absence of sensory disturbances, cutaneous and other clinical manifestations characteristic of arsenic neuritis must be quite sufficient definitely to eliminate arsenic as the etiologic factor.

By a similar process of reasoning, lead and other heavy metals may be eliminated from the discussion, since no appreciable amounts of lead or other poisonous metals have ever been found in any of the suspected samples of ginger, nor have any of the patients shown any clinical symptoms of lead poisoning other than the paralysis.²

We are thus left with two possibilities which merit consideration: Either a shipment of ginger root, imported through other than the

¹ A splendid description of the clinical picture with reports of cases of "ginger paralysis" is given in recent publications by C. R. Bennett (1) and S. Harris (2).

³ Very careful chemical examination of the urine and feces for lead by Doctor Kehoe in a series of ginger paralysis cases at the Cincinnati General Hospital gave completely negative results. Similar studies for arsenic have likewise yielded negative results. Personal communication.

regular channels, was contaminated, probably through carelessness or ignorance or both, with a root resembling ginger superficially but having remarkable pharmacologic properties of affecting the peripheral motor nerves in selected areas; or some known poison or derivative thereof got into a manufactured lot of the ginger beverage. accidently or in some other manner, and its pharmacologic properties were so altered through the ginger or alcohol, or both, so as to be no longer recognizable. In favor of the first alternative there is some information to the effect that an obscure paralytic disease of livestock, known among the natives as "derringada," occurs in some parts of South America. The plant that is probably responsible for this, and known as "derringue" or "jaqua," appears to be a tree, and it is not clear how this could be confused with ginger root. There is also a plant in Porto Rico and Cuba that has a reputation of being poisonous to cattle, commonly known as "Tibey" and scientifically as Isotoma longiflora.3 From a paper by Plugge (3) on the toxic action of the alkaloid of this plant, isotomin, it appears that the toxic nature of the plant is such that it could not possibly account for the clinical picture of paralysis that the ginger has caused in man. Furthermore, the results of our pharmacological studies with the suspected ginger, in animals, which will be detailed below, pretty well eliminate Isotoma longiflora as the offending agent and also probably eliminate to a large extent, though not completely, the possibility of other rare plants that might bear an unknown and specific poison.

We must now consider the other possibility of some known or only partially known poison with its pharmacologic properties so altered as to produce a condition in man heretofore virtually unknown. From the very nature of the problem it would seem not improbable that the suspected ginger contained some denaturant, since denatured alcohol might very well have been and probably was used in the manufacture of some of the beverage, or that it contained some adulterant, since it is known with certainty that adulterants of various kinds have been used for several years in the manufacture of the ginger beverage. In the following we submit chemical and pharmacological evidence which, though by no means complete, seems to indicate that the latter explanation appears to be indeed the correct one, though the mechanism of action of the suspected and partially identified adulterant is as yet not clear.

PHARMACOLOGICAL AND CHEMICAL EVIDENCE

Through a careful and painstaking search for samples of ginger in any way related to the epidemic, we succeeded in securing 13 such samples, a brief description of which is given below.

³ Personal communication from Dr. D. H. Cook, acting director, School of Tropical Medicine, San Juan. P. R.

- 1. Hub Distributing Co. Seized by prohibition officers April 17 in a warehouse where it was stored in bulk.
- 2. Queen City brand. Seized by prohibition officers. This was bottled in 2-ounce bottles.
 - 3. Davis & Co. Seized by prohibition officers. Bottled as above.
- 4. Davis & Co. Shipped in bulk to H. C. Guernsey, Seminole, Okla., who drank of it and developed paralysis.
- 5. Queen City brand, bottled. A shipment to a dealer in Johnson City, Tenn., under date of January 18 and seized by the local prohibition officer.
 - 6. Same as above. Shipped under date of February 18.
 - 7. Same as above. Shipped under date of February 24.
- 8. Small sample, about 50 c. c. Q. C. brand obtained from the commanding officer, Soldier's Home, Johnson City. This was a sample of a lot that is said to have produced paralysis in some of the inmates.
- 9. Sample similar to above, Land brand, said to have been drunk by some of the inmates with no ill effects.
- 10. A sample of B & L received from Dr. W. M. Simpson, Miami Valley Hospital, Dayton, Ohio. This represented part of several 2-ounce bottles consumed by a patient of Dr. G. P. Tyler, jr., of Ripley, Ohio, resulting in paralysis.
- 11. A sample of B & L obtained from Dr. G. P. Tyler, jr., of Ripley, Ohio, who is fairly certain it produced paralysis.
- 12. A sample of Peer brand obtained from Doctor Tyler as probably harmless.⁴
- 13. A sample of B & L obtained from a colored resident in Ripley with the assurance that a friend had acquired paralysis from drinking several bottles of similar material.

It will be seen that of these 13 samples, Nos. 4, 8, 10, 11, and probably 13 may be considered as almost certainly paralytic, while Nos. 9 and 12 as almost certainly harmless. The others may be said to be uncertain with the exception of Nos. 5, 6, and 7, which from the epidemiological evidence of the prevalence of the disease in eastern Tennessee, and especially in Johnson City, would make it appear very probable that No. 5 would be the most likely and No. 7 the least likely shipment to have caused paralysis.

The chemical evidence of a positive nature which we have secured concerns the test for phenols which, so far, has resulted positively in the cases of all specimens of ginger that have caused paralysis either definitely or with a high degree of probability, and negatively in the

⁴ Ripley has a population of about 1,500 with over 100 cases of paralysis, according to Dr. G. P. Tyler, ir.

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cases of ginger preparations that were definitely or probably harmless. The test for phenols was carried out as follows:

Five c. c. of the sample was placed in a 250-c. c. distilling bulb. It was made alkaline by the addition of 25 c. c. N/10 NaOH, and 20 c. c. of distillate was collected. The residue in the flask was then diluted with 10 c. c. of distilled water and acidified by addition of 10 c. c. N/1 H₂SO₄. It was again boiled and 20 c. c. of distillate collected. Ten c. c. of the latter distillate, after mixing, was tested for phenols by treating with 5 c. c. of Millon's reagent.⁵

The pharmacologic evidence supporting and supplementing this chemical finding is shown by the experiments upon rabbits, wherein every sample of ginger giving a positive test for phenols has produced upon oral administration a symptom complex characterized by muscular tremors, hyperexcitability, spastic rigidity, followed by general muscular weakness, and generalized flaccid paralysis of all the extremities and finally death from respiratory failure. The condition produced in rabbits by the suspected ginger may be briefly described as that resembling systemic phenol or cresol poisoning, with the difference that the stimulating action upon the spinal cord was somewhat more pronounced than with pure phenol or tricresol in alcohol similarly administered, the end result, however, being the same in all cases—viz, generalized flaccid paralysis for several hours or days preceding death due to respiratory failure.

Table 1.—The presence of a phenolic compound in certain adulterated fluid extracts of gingers, its close association with paralysis in man, and phenol-like toxicity in rabbits.

			Pharmacologic action in rabbits						
Sample Paralysis in in man		Phenol re- action	Number		se adminis- red	Down to			
			rabbits	Mini- mum	Maxi- mum	Result			
1 2 3 4 5 6 7 8 9 10 11 12	Not known do	Positive Negative Positive do do Positive Positive Negative Positive Negative Positive	9 5 2 2 2 2 (*) (*) 1 1	C. c. per kilo 6 30 12 12 25 8 64	C. c. per kilo 24 48 24 18 40 12 64	All died with typical symptoms, Survived. No symptoms. Died with typical symptoms. Do. Do. Survived. No symptoms. Died with typical symptoms. Died with typical symptoms.			

[•] Insufficient for pharmacological test.

⁵ The Millon reagent was prepared and the test carried out in accordance with the directions given in Hygienic Laboratory Bulletin No. 110, pp. 25-33 (1917).

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These preliminary findings are given in Table 1. The data therein are self-explanatory and require but little comment. The ginger was administered to the rabbits by stomach tube after dilution with water so that the alcohol concentration was about 25 per cent. Eight c. c. per kilo of 80 per cent alcohol is close to the maximum tolerated dose in the rabbit; hence the amount of ginger administered at any one time never exceeded this, and only the nontoxic gingers were administered in such large daily doses. The toxic gingers were administered in daily doses of from 2 to 6 c. c. per kilo until definite symptoms of tremors or spastic rigidity developed, when the treatment was generally discontinued.

Summarizing the results detailed in Table 1, it appears that samples of ginger which were definitely or probably paralytic in man gave a positive reaction for phenols and produced in relatively small doses a phenol-like symptom complex in rabbits terminating in medullary paralysis, while ginger samples that in so far as we know were harmless in man, gave no such phenol reaction, and had no toxic effects in rabbits when administered in moderately large doses.

THE PROBABLE NATURE OF THE PHENOLIC COMPOUND

Important information on this phase of the problem was gleaned from some experiments on monkeys and dogs. At the very outset it was felt, for obvious reasons, that the monkey would probably be the most useful experimental animal in this problem. Contrary to expectations, however, it was found that no symptoms of any description other than those produced by the alcohol could be elicited from the oral administration of the suspected gingers. If the ginger was administered daily, there soon developed a tendency toward vomiting. When administered every other day, the animals generally tolerated it well. The gingers were given in doses of 8 to 10 c. c. per kilo, or the equivalent of the maximum tolerated dose of alcohol. To our great astonishment, not the slightest symptoms could be elicited. The same results were noted in several experiments upon dogs.

Table 2 is presented to show the peculiar and almost absolute immunity of the monkey to the phenolic substance demonstrated in the suspected gingers. Only a few of the most striking experiments are given in this table, which, however, show sufficiently conclusively that for some, at that time, obscure reason the monkey was extremely refractory.

⁶ There was one exception to this in the series of 17 monkeys used in this work: Monkey No. 3, weighing 3.2 kflos, developed what appeared to be a typical case of flaccid paralysis of the upper and lower extremities within two days of the oral administration of two doses of 5 c. c. per kflo of ginger (sample No. 1). This condition lasted for about 10 days and was followed by nearly complete recovery. The animal was then given several more doses of 8 c. c. per kflo of the same material, but failed to show anything further. We have never known how to explain this apparent exception and the failure to reproduce the condition in the same animal with the same material.

Sam- ple No.	Phenol reaction	Mon- key No.	Weight, (kilo- grams	Total dose (e.c. per kilo)	Result	Effect in Rabbits
1 5 6 11 4	Positive dododo	{ 1 2 4 4 15 11 8 17	4.0 8.3 8.3 8.0 8.0 8.4	89 49 42 42 56 40 72	No effect do	Killed rabbits with typical phenolike symptoms in doses of from 6 to 24 c. c. per kilo. Killed in 25 to 40 c. c. per kilo. Killed in 8 to 12 c. c. per kilo. Killed in 12 c. c. per kilo. Killed in 12 to 18 c. c. per kilo.

TABLE 2.—Comparative effects of suspected ginger in monkeys and rabbits

It was thought that the immunity is only a relative one, and by concentrating the ginger through the removal of alcohol it might be possible to elicit some symptoms upon the administration of relatively large doses.

Experiments were therefore performed in which the alcohol and water were removed with the greatest care to avoid as much as possible the chemical breakdown of the constituents of the "fluid extract," and concentrates, the equivalent of 500 to 1,000 c. c. of sample No. 1, were administered. There were no effects either immediate or remote.

These negative experiments then made it appear likely that the substance in the suspected ginger which is toxic in rabbits and which may or may not be identical with the substance which produced paralysis in man is in some peculiar combination, so that it can not exert its action in the monkey. Experiments were then made in an attempt to recover the suspected material by fractional distillation. A liter of ginger (sample No. 1) was freed of alcohol and water and subjected to partial vacuum distillation. A small amount, about 2 c. c., of volatile oil came over at a temperature below 195° C. material injected intramuscularly into a monkey had no effect. further distillation a considerable amount, about 10 c. c., came over at a temperature of from 195° to 205° C. This was followed by a drop in temperature suggesting decomposition, then another distillate of a few cubic centimeters was collected at 100° to 160° C. The last two fractions gave a strongly positive Millon reaction for phenols. two distillates were injected intramuscularly into monkey No. 10. In about 15 minutes there were typical symptoms of systemic phenolpoisoning, with fine and coarse muscle tremors, loss of reflexes, and The animal recovered within 18 hours, but showed pronounced flaccid paralysis of the extremities, with great difficulty of locomotion. This condition persisted for three days, when the animal died. histologic findings of this and many other animals that came to autopsy in this work will be reported later if the results so warrant.

This experiment was repeated with essentially the same results in monkey No. 20, which likewise developed a flaccid motor paralysis

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of the upper and lower extremities. In this case the material recovered from 1,000 c. c. of the same ginger was injected subcutaneously and intramuscularly in divided doses over a period of five days so that acute symptoms of phenolic poisoning were never elicited. The paralytic symptoms developed on the fourth day following the first injection and lasted five days, when death supervened. Control experiments on monkeys with similar fractions obtained from U. S. P. ginger or adulterated ginger not giving the phenol reaction (ginger No. 2) gave negative results.

These experiments on monkeys taken in conjunction with the earlier negative ones clearly verified our assumption that the phenolic compound in the suspected ginger must be an extremely stable substance, resisting decomposition in the body of the monkey or dog and, therefore, harmless. These experiments further suggested another line of investigation with the aim of using more drastic chemical treatment but at lower temperatures, whereby more complete decomposition of the phenolic compound could be effected without at the same time obtaining organic decomposition products as the result of the high temperatures.

The following procedure was then adopted. Four hundred c. c. of the suspected ginger (sample No. 1) was carefully freed of its alcohol and water. The residue was acidified with H2SO4 and extracted with ether. The ether extract 7 was treated with 25 c. c. of a 25 per cent solution of NaOH at room temperature to remove resin acids and free phenols if present. Subsequent acidification of this aqueous solution and distillation showed, however, that there were no free phenols. The ether extract was freed of its ether and the residue saponified with 25 per cent NaOH at 100° C. for 1 to 2 hours. This was then acidified with H₂SO₄ and distilled, whereupon phenols (probably cresols) were recovered in amounts corresponding roughly to about 1 per cent of the suspected ginger. The chemical work on the identification of the phenols is still in progress. The pharmacologic evidence, however, seems conclusive, for the oral administration of this material in alcohol, divided in two doses produced in a monkey (No. 14) the immediate effects of systemic phenol poisoning, including generalized muscular tremors, muscular weakness, and coma, followed by complete recovery within 24 hours. Indeed the symptoms were identical qualitatively with those produced by the administration of 5 c. c. per kilo of a 5 per cent phenol solution in 95 per cent alcohol.

The exact nature of the phenolic compound which we have found uniformly to be present in suspected ginger and absent in unsuspected ginger is as yet unknown. From its chemical behavior it appears to

⁷ In one experiment the ether extract representing 2,000 c. c. ginger was evaporated at this point, residue treated with H₂SO₄, and distilled, whereupon 4 c. c. of volatile oil was collected, probably ginger oil. This was recently injected intramuscularly to a monkey (No. 12) with no effects whatever so far.

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resemble a phosphoric acid ester of one or more of the cresols. The strong alkali and heat required for its saponification and the fact that phosphate has been found in the suspected gingers would make it very probable that it may indeed be the ester suggested.

We also have the following pharmacologic evidence for the above suggestion:

1. An adulterated ginger prepared from U. S. P. fluid extract of ginger made to approximate in composition the suspected gingers behaved exactly like the suspected gingers in rabbits and monkeys. The adulterated ginger so prepared had the following composition:

	C. c.
Fluid-extract ginger, U. S. P.	. 30
Oleo resin ginger	. 10
Castor oil	
Tricresyl phosphate (technical)	. 24
Water	. 50
Alcohol	770
Total	900

The above sample of ginger was tested on 12 rabbits in daily doses of from 2 to 6 c. c. per kilo, with the result that in every case the typical symptom complex obtained with the suspected gingers followed, with ultimate death from respiratory failure. The minimum total dose of this ginger that killed rabbits was 6 c. c. per kilo, and the maximum 15 c. c. The same ginger given to a monkey (No. 11) in five doses of 10 c. c. per kilo, each given every other day, had no effects whatever.

- 2. A 2½ per cent solution of tricresyl phosphate (technical) in 80 per cent alcohol administered in daily doses of 5 c. c. per kilo to three rabbits produced the same typical symptom complex, ending fatally in every case. The minimum total lethal dose of this solution was 10 c. c. per kilo, and the maximum 15 c. c. per kilo.
- 3. Technical tricresyl phosphate administered orally to monkeys (Nos. 7 and 23) in huge doses of 10 and 15 c. c. per kilo with or without alcohol had no effects whatever.
- 4. The same tricresyl phosphate saponified with NaOH and heat, acidified and distilled, yielded phenols similar to those obtained from suspected gingers with similar treatment. This material administered in alcohol orally to a monkey (No. 13) in a dose equivalent to 1 c. c. per kilo of the tricresyl phosphate produced very marked typical symptoms of systemic phenolic poisoning with tremors, coma, etc. The animal died within four hours of respiratory failure.

CONCLUSIONS

If we consider the problem in the light of all the experiments performed, of which only the essential ones are detailed in this paper, the following conclusions may be drawn at this time:

- 1. Adulterated gingers with a reasonably certain or highly probable history of paralysis in man have yielded distillates, upon saponification and subsequent acidification, giving a positive reaction for phenols; while unsuspected adulterated gingers, as well as U. S. P. fluid-extract of ginger, treated similarly, failed to give such a reaction.
- 2. Suspected adulterated gingers have invariably proved toxic in rabbits in moderate doses; death, which is due to respiratory paralysis, is preceded by a symptom complex resembling very closely in its essentials, though not absolutely, systemic phenol poisoning. Unsuspected adulterated gingers in large doses, as well as U. S. P. fluid extract of ginger, failed to produce such effects.
- 3. All adulterated gingers examined, including the suspected ones giving a positive reaction for phenols, proved practically uniformly harmless in monkeys. A few experiments on dogs were likewise essentially negative.
- 4. Chemical and pharmacological evidence indicate that the phenolic substance in the suspected gingers is a stable combination of phenols, probably in the form of a phosphoric acid ester or some related substance, which resists hydrolysis and requires drastic treatment with alkali and heat to effect complete saponification. The pharmacologic experiments furthermore indicate that this stable phenolic compound breaks down with great ease in the rabbit and apparenty not at all in the monkey. The few observations we have in the dog show that it, too, is unable to liberate the phenols from this firm combination.
- 5. The precise relation of this phenolic compound either by itself or in combination with the other ginger constituents to the multiple neuritis in man is as yet not clear. Before we can be certain of the etiologic relationship it will be necessary to find means of reproducing the human disease in animals more faithfully than we have been able to do so far. The remarkable difference in species susceptibility we have observed tempts one to venture the suggestion that as regards susceptibility man may stand in some intermediary position between the rabbit at the one extreme and the monkey at the other. Until some satisfactory explanation of this difference in species susceptibility becomes available, the suggestion must be considered as purely speculative. We may express the hope, however, that with more chemical information on this phenolic compound and a better knowledge of its action in the animal body its etiologic relationship to the human disease may become more apparent.

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Addendum.—Since the foregoing was written, an important experiment has been performed upon calves which proves almost conclusively our tentative conclusions as to the etiologic relationship of the phenolic ester to the multiple neuritis in man. A description of this experiment follows:

On June 3 three male calves of approximately the same age and weight were selected, and ginger, diluted with equal parts of water, was administered by stomach tube as follows:

Calf No. 1 (identification No. 1652), Jersey, 3 months old, weighing 60 kilos, received 5 c. c. per kilo ginger sample No. 7. This, it will be remembered, was nontoxic in rabbits and gave no phenol test chemically.

Calf No. 2 (identification No. 1651), black and white, 3 months old, weighing 80 kilos, received 5 c. c. per kilo ginger sample No. 1, which gave a positive test for phenols and proved toxic in rabbits.

Calf No. 3 (identification No. 1654), brown and white, 2 months old, weighing 86 kilos, received 5 c. c. per kilo of U. S. P. fluid extract of ginger adulterated by dilution with alcohol and the addition of 2.5 per cent technical tricresyl phosphate, castor oil, and a small amount of oleoresin ginger. (For complete formula see text.)

Moderate alcoholic intoxication followed in all cases with complete recovery within 24 hours.

On June 9 a second treatment was administered to the calves as follows:

Calf No. 1, six c. c. per kilo of ginger sample No. 7.

Calf No. 2, six c. c. per kilo of ginger sample No. 4. (This, like No. 1, gave a positive phenol test, proved toxic to rabbits, and is almost certainly known to have caused paralysis in man.)

Calf No. 3, six c. c. per kilo of U. S. P. fluid extract of ginger adulterated by dilution with alcohol and addition of 2.5 per cent tricresyl phosphate (technical) and crude resin oil instead of the oleoresin ginger and castor oil.

Calves Nos. 1 and 2 showed moderate alcoholic intoxication and recovered the following day. Calf No. 3 was markedly depressed and was unable to get up for two days. There were no tremors or other evidence of phenol poisoning. On examination on June 13 the three calves appeared normal. Nothing unusual was noted about them on June 20. The animals were not examined closely between this date and July 5. Examination on July 5 revealed distinct weakness of the hind legs in calves Nos. 2 and 3. This was noticed especially when the animals were made to run, when they would stumble frequently, with bending and dragging of the hind feet and hoofs. The anterior extremities appeared normal. The control calf No. 1 was normal. A second examination on July 7 found calves Nos. 2 and 3 in the same general condition, the weakness in

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the hind legs being more pronounced. The deep reflexes of the anterior extremities appeared normal, while those of the posterior extremities were much reduced or absent.

The progress of this experiment is being followed and further experiments are being planned. Barring the remote possibility of some of the other ginger constituents or some impurity in the technical tricresyl phosphate, having something to do in a supplementary manner with the paralytic disease, it appears almost certain that the cresol-phosphoric acid-ester postulated earlier in the paper is indeed the etiologic factor of the epidemic of so-called ginger paralysis. Further pharmacologic work will be needed to elucidate the singular and highly specific action of this unique poison in man and in some of the lower animals, and its remarkably different behavior in different species of animals.

We are greatly indebted to Dr. W. E. Cotton of the Bureau of Animal Industry, Department of Agriculture, for the facilities given us to carry out the calf experiments.

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- (2) Harris, S.: Ibid, p. 375.
- (3) Plugge, P. C.: Arch. Exp. Path. Pharm. (1892) 23, 266.

RELATION BETWEEN TRYPANOCIDAL AND SPIROCHETI-CIDAL ACTIVITIES OF NEOARSPHENAMINE

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The desirability of a test for therapeutic potency of the antisyphilitic remedies of the arsphenamine type has led to the development of the trypanocidal activity test, and later to the spirocheticidal test in experimental syphilis in rabbits. The early reports indicated that the former test was a satisfactory means of establishing the therapeutic efficiency of the arsenicals, but recent reports of the spirocheticidal test in rabbits have weakened this view.

Schamberg, Kolmer, and Raiziss (1), Dale and White (2), and Voegtlin (3) favored the adoption of the trypanocidal test as a routine centrol measure for the arsenicals. Numerous clinical workers, seeing the desirability of a potency or efficiency test, have supported this view. The recommendation of the Second International Conference on the Biological Standardization of Certain Remedies (4) that the therapeutic potency of arsenicals be tested by the trypanocidal test, as well as the official requirement of this test by England (5) and Germany (6), have materially strengthened this position. However, the maxim

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laid down by Voegtlin and Dyer (19), Kolmer (7), and by Wakerlin (8) is that the final analysis of the evaluation of the antisyphilitic property of drugs must be ascertained on experimental syphilis in rabbits. It was therefore decided to ascertain the relation between the therapeutic activity of neoarsphenamine as measured by the trypanocidal test, using rats, and as indicated by the spirocheticidal test, using rabbits.

TRYPANOCIDAL ACTIVITY OF NEOARSPHENAMINE

Schamberg, Kolmer, and Raiziss (9) reported in 1920 that the smallest effective dose of different preparations of neoarsphenamine varied from 20 to more than 40 mg. per kilogram in the albino rat. The investigation included 22 preparations from six manufacturers. Voegtlin and Miller (10), 1922, reported an even greater variation in the products of five manufacturers, showing the most efficient to require 9.32 mg. while the least efficient required 30.08 mg. per kilogram.

Kolmer (11), in the comparison of the trypanocidal and spirocheticidal properties of arsphenamine and neoarsphenamine, showed variations of from 2 to 10 mg. per kilogram in six different neoarsphenamines. It is not reported whether the material was from the same or from different manufacturers. Dale and White (12), 1922, reporting on the trypanocidal activity of neoarsphenamine of British and German manufacturers, found the minimum effective dose of the former to be two to three times that of the latter.

SPIROCHETICIDAL PROPERTIES OF NEOARSPHENAMINE

Nichols and Walker (13), and Voegtlin, Armstrong, and Dyer (14), in 1923, reported the sterilizing dose of neoarsphenamine to be 15 mg. per kilogram for syphilis in rabbits after one treatment. The data reported, however, were on one animal only in each case. Pierce and Brown (15), 1922, previously reported that one treatment of 9 mg. per kilogram failed to sterilize. Kolmer (16), 1926, found the curative dose of six neoarsphenamines to vary between 8 and 12 mg. per kilogram. Voegtlin and Dyer (17), 1927, reporting on the sterilizing efficiency of arsphenamine, neoarsphenamine, and sulpharsphenamine in experimental syphilis, found that the minimum sterilizing dose was identical in terms of absolute amount of arsenic. They reported the sterilizing dose of neoarsphenamine after one treatment as 40 mg. per kilogram.

COMPARISON OF THE TRYPANOCIDAL AND SPIROCHETICIDAL PROPERTIES OF THE ARSENICALS

In 1922, Voegtlin (18) reported that the objection to the use of the trypanocidal test on the ground that it does not establish the relaJuly 25, 1930 1718

tive efficiency of arsphenamine and neoarsphenamine with regard to *Treponema pallidum* is not justified, for the reason that the curative ratio of the two drugs as established by Ehrlich and Hata and Castelli in spirochete infections, including rabbit syphilis, is practically the same as that determined by Voegtlin and Smith, 1921, by means of the trypanocidal test.

Voegtlin (19), in light of work reported in 1925 on sulpharsphenamine, called attention to the fact that this drug in spite of a relatively low trypanocidal action, is as effective as arsphenamine and neo-arsphenamine with regard to the healing of syphilitic lesions and freedom from clinical relapse.

Kolmer (20) accepts the trypanocidal tests as of distinct worth in evaluating the properties of arsphenamine and neoarsphenamine, although he states that the relation between the trypanocidal activity in the rat and the spirocheticidal activity in the rabbit is not definite and constant, but only broad and general.

The most interesting comparison of the trypanocidal activity in animals with spirocheticidal properties in man was reported by Dale and White (21) in 1922. This report compares the trypanocidal activity of neoarsphenamine with the dose necessary to free the chancre of *T. pallidum* in 18 to 20 hours. A parallelism was found to exist between the trypanocidal properties and the clinical effect on a primary lesion in man. It is reported that the results obtained justify the conclusion that the trypanocidal test is a very valuable index, if not an accurate quantitative measure, of the therapeutic activity of different samples of a preparation such as neoarsphenamine on syphilis in man.

TYPES OF NEOARSPHENAMINE

As pointed out by Dale and White (22) in 1922, there are two types of neoarsphenamine—one of slow solubility with relatively high toxicity, and the opposite type with rapid solubility and low toxicity. The trypanocidal activity of each group was found to parallel the toxicity of the product.

There has since been developed a new type of this product which has the qualities of the latter group and is as trypanocidally active as the former.

The classification of the two major types is represented by Groups E and F, respectively.

TRYPANOCIDAL ACTIVITY TEST

The procedure outlined by Voegtlin and Miller (23), using albino rats inoculated with *Trypanosoma equiperdum*, was followed, except that the amount of neoarsphenamine indicated by the reading which gave negative findings in 80 per cent of the rats and permitted 20

per cent to have a trace of infection was accepted as the minimum effective dose. It will be seen from Table 1, on the trypanocidal activity reported on 32 batches of neoarsphenamine from eight manufacturers, that the effective dose ranges from 10 to 15 mg. as the most efficacious, to 25 to 35 mg. as the least effective.

TABLE 1.—Trypanocidal activity test—Neoarsphenamine

Products	A	В	o	D	E	F	G	н
Number examined	3	2	4	2	7	6	3	5
M. E. D. (mg. per kg.)	15	15	15	15	10–15	25-35	15-20	15

The products represented by E and F, Table 1, were accepted as representing the most effective and the least effective trypanocidal activity of the neoarsphenamines.

TABLE 2.—Trypanocidal activity of neoarsphenamines E and F

	Neo	arsph	enami	ne E				Ne	oarsp	benar	nine :	F		
Dose (mg. per	Lot					Dose (mg. per	Lot							
Dose (mg. per kg.)	1	2	3	4	5	6	7	kg.)	1	2	3	4	5	6
7	2+ 2+ + -	4+ 4+ - -	2+ 2+ 2+ +				D. D. D.	10			3+ 2+ 2+ 2+ +	2+ 2+ + Tr.		
10	++++	=	Tr. Tr. Tr.	Tr. Tr.	++++++	+++++	4+ 4+ - -	15	D. D. D. D.	D. D. D. D.	Tr. Tr. Tr. Tr. Tr.	Tr. Tr. Tr.	D. 4+ + -	DDD 44
15			=	=	- - - Tr.	- - - Tr.	- - -	25	D. D. 2+ -	=	=	=		
20				-		- - -		35	-	11111			11111	
M. E. D. (mg. per kg.)	15	10	15	15	15	15	15	M. E. D. (mg. per kg.)	35	25	25	25	25	25

D=dead. Tr.=trace.

The reported variations in the trypanocidal activity of neoarsphenamine is quite apparent, as indicated in the accompanying tables. There is, however, a striking uniformity in the efficacy of the products of the same manufacturer. This would indicate that the trypanocidal activity test is valuable in ascertaining the uniformity of the therapeutic efficacy of the same product. July 25, 1980 1720

EXPERIMENTAL SYPHILIS IN RABBITS

The rabbits were inoculated in the left scrotum with 0.3 c. c. of testicular emulsion of Nichols's strain of Treponema pallidum. Only animals which developed a dark field positive typical primary lesion were used. The report of the size of the lesion as shown in the tables is the area of the chance recorded in centimeters.

Treatment consisted of one intravenous injection of the dose of the arsenic preparation shown in the protocels. For convenience the observation is divided into pre-treatment and post-treatment periods, and recorded in days. The progress of the disease and the effect of the treatment are reported by measurements of the lesion, by examination by dark field, and by the quantitative Kahn test.

The evaluation of the therapeutic efficiency of the preparation was based upon the minimal dose which caused rapid disappearance of the spirochetes from the primary lesion and rapid healing of the lesion without clinical relapse—the so-called therapeutic dose. The choice of the products for the spirocheticidal test is based on the results obtained in the trypanocidal test. (Tables 1 and 2.) Neoarsphenamine brand E represented the most effective in trypanocidal activity, and brand F proved to be the least efficient.

Table 3.—Spirocheticidal activity of neoarsphenamine, products E 1 and F 5

THERAPEUTIC EFFECT AT 15 MG. PER KG.

1		Kahn	88	8	*	834	
	67 days	Dark field	1.1			1+1	
	8	Lesion	11	ı	1	1 2 1	
		Kahn	83	*	4	888	
	59 days	Dark	11	1	ī	1+1	
	25	noiss-1	11	1		1.32	e
	50	Карп	886	S	4	484	
	52 days	Dark field	116	DT.		1+1	1
	22	Tesion.	116	ΕI	ı	1.57	ı
		Каћп	444	. 4	4	₹ 88€	্ন
	46 days	Dark	11		ı	1+15	21
	\$	Lesion	111	1	ı	1.16	1
nent		Каћп	48.	* \$	4	ន្តន្តន	'ম
reatn	38 days	Dark field	111	I	1	1.+1.1	1
Post-treatment		noi29-J	111	1	ı	18,11	1
	32 days	Каћп	48.	"ଛଚ୍ଚ	4	888	'ଛ
		Dark	11	118	1	1+11	1
		noisəa	11	116	۱(1111	ı
	24 days	Kabn	848	388	4	ลลลา	'ଛ
		Dark	111	11	1	1111	ı
		Lesion	111	111	ı	1111	1
	17 days	Ksbn	844	88	8	នននន	ន
		Dark field	111	11	1	1111	<u> </u>
	H	Lesion	111	111	0.21	1118	
		пляЖ	\$ 46	33	4	4446	ล
	days	Dark field	111			1111	1
	1	nois9/1	e 843	25.53	.28	8888	8
ent 1		Kahn	888	5점	4	8448	3
estm	56 days	Dark	+++	++	+	++++	
Pre-treatment	8	noisə.1	0.69			<u>क्</u> ट≋्र	
	Rabbit No.			288	84	71. 72. 73.	76
	Product					F 5	

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11++11	1++111
11.8811	1.1.18
583884	8833444
+1++11	1++111
0.97	1.1.82
884884	883484
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Dead Oct. 27, 1928.
 Dead Nov. 6, 1928.
 Dead Nov. 19, 1928.
 Dead Nov. 11, 1928.

1 Total period of pre-treatment, 59 days. Treated Sept. 14, 1928.

3 Dead Nov. 2, 1928.

4 Ordhitis.

Table 3.—Spirocheticidal activity of neoarsphenamine, products E 1 and F 5—Continued

THERAPEUTIC EFFECT AT 5 MG. PER KG.

١	. 1	Kabn	3 555388	8 8 8 8
	days	Dark	+++1+1	+ +11+
	6	noissal	22.20 2.75 1.67 1	4.75 14.1 0
	_	Kshn	888848	8 888
	59 days	Dark field	+++111	+ +11+
	20 d	поізэЛ	1.15 1.15	.38
		Каћп	200 160 120 120 120	200 200 80 80 80
	days	Dark field	+++11+	+ ++++
	29	nolzsal	8482 3	3. 57 88
	•	падеж	240 200 200 160 160	8 55 58
	46 days	Dark field	+++111	+ ++1+
	\$	noise.I	3.21 1.253 1.73 2.61	8 8 17 1 3
ent		Каћп	338348	දීවදිදියි
estm	38 days	Dark	++++1+	+9++++
Post-treatment		noisal	3.91 1.76 2.41 .91	S 10 28 28 28 28 1 2
-	32 days	плаЯ	888848	256583
		Dark	+++11+	+++++
		Tois9-J	3. 1. 1. 1. 1. 1. 1. 1. 1. 1. 1. 1. 1. 1.	31 55 35 15 5 5 5 5 5 5 5 5 5 5 5 5 5 5 5 5 5 5
	24 days	Каћп	888884	888348
		Dark field	++++1+	++++++
		noised	3.58 1.15 1.19 1.41 2.61	38.72 88.72 88.32 38.72
		Кярп	883343	538488
	17 days	Dark	+++11+	++++++
	17	noise.I	58531 Z	1. 13 2. 41 1. 29 1. 29 3. 13
		Kahn	888488	888888
	days	A18(I blañ	+1+11+	11++1+
	1	Lesion	1. 1. 1. 1. 1. 1. 1. 1. 1. 1. 1. 1. 1. 1	
lent		пдвЯ	888388	888883
reatn	56 days	Dark	+++++	+++++
Pre-treatmen	28	noies-J	€.55 \$25 \$35 \$35 \$35 \$35 \$35 \$35 \$35 \$35 \$35 \$3	1.62 1.85 1.85 1.57 1.57
	Rabbit No.		7828 78	84272888
	:	Product	E 1	10 f4

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+++++++	
2.07 2.29 2.67 5.15 8.23 1.92	
250 250 250 250 250 250 250 250 250 250	
+++++++	
1.28 2.28 1.03 1.03 7.45 1.75 1.86	1928.
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+++++++	ğ
0.72 1.16 1.16 3.02 8.80 1.20	Dead
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Desd Oct. 21, 1928.

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The therapeutic effect of neoarsphenamine E 1 and F 5 at 15, 10, and 5 mg. per kilogram on experimental rabbit syphilis is reported in Table 3. The animals were given one treatment 59 days after inoculation. In this series the therapeutic dose was established at 15 mg. for both products. At this dose all animals on E 1 became dark-field negative; the chancres healed and remained negative throughout the observation period. The findings on F 5 were comparable, except that one rabbit, No. 72, had relapsed 32 days after treatment.

The results obtained with the 10 mg. dose indicate that both products are ineffective. This conclusion is inevitable from the fact that while all rabbits on lot E 1 became dark-field negative and the chancres healed, there were three relapses; and, further, that, although four animals on F 5 remained negative throughout the observation period, two showed relapses.

As would be expected, the effect of 5 mg. was very feeble. Except for one animal on each product, which was negative, the results very closely paralleled the control group.

Of the control (no treatment) group of eight rabbits, one died rather early, three became negative approximately 97 days after inoculation, about 38 days after administration of the drug in the treated group, and four were positive on discharge 126 days after inoculation.

Table 4.—Spirocheticidal activity of neoarsphenamine, products B 7 and F 6

16 MG. PER KG.

1		Кећи	11	44	2084 0
	88 days	Diet	T	iiII	
	8	Lesion	-	11	11111
	68 days	Kahn	#	44	4084 0
		blen	T		
		Dark			
		I.esion	1	111	1111
	2	Dark			
	68 days	noise.I	ŀ	111	1111
		Ksha	4	448	4084 0
	838	Dark fleld			
	47 days	<u> </u>			
		nois9.1	ı	+ + + +	1111
nent	pa .	Dark fleld			11
Post-treatment	40 days	Leston	ı	111	1111 1
A		Kshn	4		4484 4
	29 days	ppg			1+1
	83	Lesion Instr		101	1 1 1
		Dark		111	1111
	25 days	Lesion		101	18,88
-		Kshn		483	445884
	18 days	Dark	1	111	111111
	8	InoissAI		88.	1 . 8 . 1 1 1
		feld		1111	111111
	11 days	Dark		828	88222
	=	noiss-I	€	⊕∺∺ .	. 91.01.0
	days	Dark field	1 1	72888 1111	832228
	7 d	noissA	ହା	2001 1 2 2 2 2 2 2 2 2 2 2 2 2 2 2 2 2 2 2	ಚನ್ನಾಗ್ನ
But 1		Каћп	88	88838	888884
atm.	65 days	Dark	++	++++	+++++
Pre-treatment 1	8	noissal	(S)	82285 8282	200.000 200.000 200.000 200.000 200.000
	Prod. Rabbit No.			165 170 183	141 145 146 148 181
	t d				9
	Pg				is I

12.5 MG. PER KG.

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111111	11111
<u>व्यक्त</u>	44488
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7.7. 6. 5. 8. 8. 1. 8. 9. 1. 1. 1. 1. 1. 1. 1. 1. 1. 1. 1. 1. 1.
F-50 600
144 183 185 187 182 172

1 Treated on sixty-ninth day. L-left scrotum.

R-right scrotum.

The products reported in Table 4 represent two other lot numbers of the same manufacturer's neoarsphenamine as that reported in the previous table. These lots, E 7 and F 6, were tested at 15 and 12.5 mg. per kg., with one treatment 69 days after inoculation. There is no apparent difference in the effect on the lesions, as all animals became dark-field negative, the chancres healed, and there was no clinical relapse. The therapeutic dose is indicated to be 12.5 mg. or less per kg., but it is impossible to state definitely, as lower dosage was not included in this series.

The strength of the Kahn reactions definitely paralled the early syphilitic involvement. This is very noticeable in the successfully treated group, where there is a very rapid disappearance of the chancre, accompanied by a reversal of the Kahn reaction. This parallelism of the Kahn reaction with the primary syphilitic lesion, and the reversal of the Kahn test accompanied by healing of the chancre in the treated animals, indicates no apparent difference in the serological results of the two neoarsphenamines tested. These results agree with the reported findings of Wakelin et al. (24) that there is a definite parallelism between the Kahn reaction with the intensity of the experimental syphilitic involvement.

The disappearance of the organism from the chancre does not indicate the efficacy of the drug. This will be seen in the 10-mg. dose, Table 3, which gave negative dark-field results in all animals, but clinical relapse occurred in 5 of the 12 rabbits. Even at 5 mg. 3 rabbits treated with each product were dark-field negative on the first posttreatment observation, but 2 of each group relapsed.

Table 5.—Duration of chance and presence of Treponema after certain periods of treatment (average, in days)

Product	Number	Dose	Duration of (in days)—		
Product	of rabbits	(mg. per kg.)	Chancre	Trepo- nema	
F 5	5 6 6	15 15 12, 5	18, 4 25, 8 16, 8	7 7 7	
E 1	6 4 6	15 15 12. 5	18 25. 2 21. 5	7 7 7	
Controls	7 6		>74 51	>56 43	

It is evident (Table 5) that there is no noteworthy difference in the power of these products to cause rapid disappearance of spirochetes from the chancre and rapid healing of the lesion without clinical relapse, though, as stated above, there is a very pronounced difference in trypanocidal activity. The chancre disappeared after an average of 19.8 days after treatment with product F and after 21.2 days

when product E was used. These figures agree with those obtained by Wakelin, Lorenz, and Lovenhart (25) in 1925, on a series of nine rabbits receiving three doses of neoarsphenamine of 1 to 4 ratio to the tolerated dose (50 to 75 mg. per kg.). They reported the average duration of the chance from the institution of treatment as 24 days.

TABLE 6.—The trypanocidal and spirocheticidal properties of neoarsphenamine—
per cent of efficacy

		Trypan	ocidal te	st	Spirocheticidal test					
Product	Dose	Dose (mg. per kg.) M. E			Dose (mg. per kg.)				Effective	
	35	25	15	(mg. per kg.)	15	12.5	10	5	dose (mg. per kg.)	
F5F6	Per cent 100 100	Per cent 100 100	Per cent 40 000	25 25	Per cent 80 100	Per cent	Per cent 66	Per cent 17	>15 >12.5	
	15	10	7 ·							
E 1	100 100	0 60	25 0	15 15	100 100	100	50	17	>15 >12.5	

Table 6 was prepared for convenience in order that the trypanocidal and spirocheticidal activity might be readily compared. The results are evident and need no further comment.

CONCLUSION

From the limited data presented here two brands of neoarsphenamine varying markedly in their trypanocidal activity have shown approximately the same ability (1) to cause the rapid disappearance of spirochetes from the chancre, (2) to cause the rapid healing of the lesion with freedom from clinical relapse, and (3) to influence the Kahn reaction in experimental rabbit syphilis over periods of 67 to 88 days.

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- (24) Wakerlin and Horrall: Arch. Dermatol. and Syphilol., vol. 18 (1928), p. 539.

COURT DECISION RELATING TO PUBLIC HEALTH

Compensation under workmen's compensation act awarded for injury through infection following vaccination.—(Texas Court of Civil Appeals: Texas Employers' Insurance Association v. Mitchell, 27 S. W. (2d) 600; decided Apr. 15, 1930.) In March, 1928, a number of cases of smallpox developed in the city of Sherman. The employees of a company in that city were directed by the company's manager to be vaccinated or to bring a physician's certificate stating that vaccination was unnecessary. This direction was coupled with the ultimatum that, unless they did so, they could not work for the company until after the smallpox epidemic was over. No member of the State or city board of health, acting as a public agency for the public interest, in any wise directed or caused the vaccination of the employees. One of the employees who was vaccinated suffered injury because of infection following the vaccination. Her vaccination was done by a physician who was suggested to her and the cost of the vaccination was taken out of her pay check. The physician received payment from the company.

Compensation under the workmen's compensation act was granted to the injured employee and the awarding of compensation for such injury was upheld by the court of civil appeals. The following are excerpts from the appellate court's opinion:

The order for vaccination was given on Thursday, March 22, and the vaccination was to be done "Friday," or before Monday morning, March 26. The circumstances do not reflect the purpose of the manager in so peremptorily ordering the vaccination of the employees to have been to discharge a purely moral obligation to provide for medical attention or to further the personal welfare of the employees. Neither do they reflect his intention to require the vaccination to have been an act entirely outside the range of the employees' service to their employer. The circumstances strongly point to the view that in the emergency of the smallpox epidemic the vaccination was for the purpose of furthering the work or business of the factory by having the employees made immune to smallpox as a precaution against suspension or interruption through smallpox of the regular work or business of the factory. * * * Compliance with the special order was intended to operate as an obligation of employment on the part of the employees, and noncompliance was intended to operate as an act inconsistent with the relation of master and servant and incompatible with the faithful performance of duty owing the employer.

* * In the present case the employer himself, through the manager, in furtherance of his business, and not as a State or public agency, ordered the employees to be vaccinated; and the vaccination wound received in the act of vaccination came in direct contact with infectious or poisonous matter, resulting in the injury complained of.

DEATHS DURING WEEK ENDED JULY 12, 1930

Summary of information received by telegraph from industrial insurance companies for the week ended July 12, 1930, and corresponding week of 1929. (From the Weekly Health Index, July 16, 1930, issued by the Bureau of the Census, Department of Commerce)

	Week ended July 12, 1930	Corresponding week, 1929
Policies in force	76, 067, 749	74, 515, 561
Number of death claims	13, 433	12, 174
Death claims per 1,000 policies in force, annual rate_	9. 2	8. 5

Deaths from all causes in certain large cities of the United States during the week ended July 12, 1930, infant mortality, annual death rate, and comparison with corresponding week of 1929. (From the Weekly Health Index, July 16, 1930, issued by the Bureau of the Census, Department of Commerce)

		ided July 1930	Annual death rate per	Deaths	Infant mortality	
·· City	Total deaths	Death rate ¹	1,000, corre- sponding week, 1929	Week ended July 12, 1930	Corresponding week, 1929	rate, week ended July 12, 1930 ²
Total (65 cities)	6, 507	11.4	11. 2	616	615	* 55
Akron	19 41 88 45 43 189 28 133 170 29 128 182 222 227 175 86 56 41 15 30 30 102 31 31 32 32 32 33 30 45 22 22 23 38 30 30 31 31 31 31 31 32 33 30 30 31 31 31 31 32 33 30 30 31 31 31 31 31 32 33 33 30 30 31 31 31 31 32 33 30 30 31 31 31 31 31 31 31 31 31 31 31 31 31	17. 8 18. 0 (9) 11. 9 13. 8 (4) 11. 1 12. 0 7. 5 9. 8 9. 5 9. 5 9. 0 11. 9 13. 4 (6) 11. 0 18. 1 10. 6 14. 7 14. 7 13. 3 8. 5 7. 7 11. 6 (9) 10. 2	13. 9 14. 5 (9) 13. 1 (10. 6 7. 9 10. 6 7. 9 10. 4 7. 6 10. 9 10. 5 11. 0 12. 6 12. 2 (5) 8. 5 11. 0 (9) 8. 6 (9) 13. 5 11. 0 (10. 6) 13. 5 14. 5 15. 9 15. 9 16. 6 16. 6 17. 9 18. 6 18. 5 19. 6 19.	3 0 0 11 4 7 7 11 9 12 4 5 5 2 2 3 3 19 2 2 5 7 7 7 0 0 1 16 2 2 6 5 5 1 5 7 5 2 2 1 1 0 6 5 5 5 5 5 5 5 5 5 5 5 5 5 5 5 5 5 5	4 0 0 10 4 6 20 9 1 1 9 6 6 3 6 6 9 1 1 3 5 4 1 1 1 6 6 6 4 4 4 0 9 6 3 3 8 6 2 6 4	27 00 116 127 1111 65 60 81 47 31 71 54 67 37 36 0 50 59 57 49 15 167 27 128 46 0 0 43
White	24 7 97	(5) 12.9	(5) 13. 9	1	3 1 11	106 217 70

See footnotes at end of table.

1730 July 25, 1980

Deaths from all causes in certain large cities of the United States during the week ended July 12, 1930, infant mortality, annual death rate, and comparison with corresponding week of 1929. (From the Weekly Health Index, July 16, 1930, issued by the Bureau of the Census, Department of Commerce)—Continued

		ided July 1930	Annual death rate per 1,000,		under 1	Infant mortality rate, week
City	Total deaths	Death rate 1	corre- sponding week, 1929	Week ended July 12, 1930	Corresponding week, 1929	ended July 12, 1930 3
Knoxville	30	14.8	18.8	8	10	70
White	24			3	7	70 78
Colored	6	(4)	(4)	.0	3	0
Los AngelesLouisville	249 74	11.7	13. 9	28 3	17	85
White	57	11.7	10.9	9	3	26 30
WhiteColored	17	(5)	(4)	3	.3	0
Lowell	26			2	4	47
Lynn	11	5.4	9. 9	0	4 3 5	47 0
Memphis	78	21. 4	15.4	7		83
White	41			5 2	1	92
Colored Milwaukee	37 101	(5) 9. 7	(5) 8.1	2	4	67
Minneapolis	101	9. 7 12. 2	8.1 9.3	10 9	13	50 58
Nashville.	73	27. 3	9. 3 22. 8	9	8 10	58 46
White	41	21.0	44.0	3 2	10	41
Colored	32	(5)	(5)	ī	ĭ	63
New Bedford	21			5	2	128
New Haven	30	8.3	9.7	1	2 0	19
New Orleans	155	18.8	14. 2	15	13	87
White	97			11	7	97
Colored	58	10.8	(9)	4	.6	67
Bronx Borough	1, 248 178	9.8	11. 0 7. 3	125	93	53
Brooklyn Borough	412	9.3	9.7	8 51	15 35	19 54
Manhattan Borough	462	13. 7	15.8	54	36	89
Queens Borough	149	9.1	7.8	7	3	20
Richmond Borough	47	16.3	16.3	5	ă	93
lewark, N. J.	101	11. 1	10.7	9	12	47
Oakland	46	8.8	9.7	5	1	60
okland klahoma City maha	53			17	5	334
aterson	65 37	15. 2 13. 3	14.0	5	4	57
hiladelphia	391	9.9	11.5 11.7	6 31	7 46	104 46
ittsburgh	152	11.8	10.8	16	15	59
Pittsburgh Portland, Oreg	79		10.0	2	3	25
rovidence	56	10. 2	12.0	6	5	55
ichmond	45	12.1	16.4	2 0	4	3 0
White	29			0	1	.0
ochester	16	(9), ,, !	(9)	2	3	87
. Louis	61 236	(5) 9. 7 14. 5	(5) 11. 0 12. 7	3 19	.9	27 62
t. Paul	59	12.0	12. /	19	14 2	62 10
alt Lake City	31	11.7	12.8	4	4	63
alt Lake City 4an Antonio	67	16.0	12.4	16	15 .	
an Diego	49			ĭl	2	21
an Francisco	179	15. 9	8. 9	6	2 7	41
chenectady	18 77 21 26	10. 1	13.4	i	4	31
eattleomerville	77	10.5	7.8	1	3	10
nokana	21	10.7 12.4	7. 1 7. 2	1	2 2	33 26
pringfield, Mass	34	11.8	11.8	1 6	ı i	20 95
pokane pringfield, Mass yracuse acoma	27 24	7.1	9.9	21	2	50
acoma	24	11.3	8.0	āl	. ő l	77
01600	88	14.7	13.0	4 3 5	ğ	46
renton	30	11.3	18. 4	4 2 17	4	74
tica ashington, D. C	25	12.5	15.0	2	3	57
White	142 92	13. 4	12.2	17	16	99
Colored	50	(4)		7 10	8	60 177
aterbury	16	(7)	(4)	2	8	177 51
aterbury ilmington, Del	19 20	8.1	13.0	ő	0	0
orcester	45	11.9	12.7	ĭ	5	13
onkersoungstown	14 38	6.0	6.0	î	ĭ	24

¹ Annual rate per 1,000 population.
2 Deaths under 1 year per 1,000 births. Cities left blank are not in the registration area for births.
3 Data for 73 cities.
4 Deaths for week ended Friday.
4 To the cities for which deaths are shown by color, the colored population in 1920 constituted the for Deaths for week ended Friday.

In the cities for which deaths are shown by color, the colored population in 1920 constituted the following percentages of the total population: Atlanta, 31; Baltimore, 15; Birmingham, 39; Dallas, 15; Fort Worth, 14; Houston, 25; Indianapolis, 11; Kansas City, Kans., 14; Knoxville, 15; Louisville, 17; Memphis, 88; Nashville, 30; New Orleans, 26; Richmond, 32; and Washington, D. C., 25.

PREVALENCE OF DISEASE

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

UNITED STATES

CURRENT WEEKLY STATE REPORTS

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

Reports for Weeks Ended July 12, 1930, and July 13, 1929

Cases of certain communicable diseases reported by telegraph by State health officers for weeks ended July 12, 1930, and July 13, 1929

	Diph	Diphtheria Influenza Measles Meningoc meningi						
Division and State	Week ended July 12, 1930	Week ended July 13, 1929	Week ended July 12, 1930	Week ended July 13, 1929	Week ended July 12, 1930	Weck ended July 13, 1929	Week ended July 12, 1930	Week ended July 13, 1929
New England States: Maine	10 2 37 2 1	6 1 1 60 3 17	1	3	42 10 10 440 11 20	34 12 263 44 36	1 0 0 2 1	1 0 0 1 0
Middle Atlantic States: New York New Jersey Pennsylvania East North Central States:		181 75 83	1 1 2	1 5	1, 075 535 638	403 75 427	11 2 1	26 3 8
Ohio Indiana Illinois Michigan Wisconsin	42 10 113 54 12	45 11 148 74 13	3 1 2	10 2 6	194 53 138 266 54	439 43 560 309 482	5 4 8 6 0	12 1 7 17 3
West North Central States: Minnesota Iowa Missouri North Dakota South Dakota	10 4 22	11 3 34 5 7		1	99 53 43 4 50	79 21 21 25 4	1 1 3 1	2 3 4 0
Nebraska	10 7	3 3 2 9	3	5	10 63 7 18	57 114 2 12	0 0 0	0
Maryland ¹ District of Columbia West Virginia North Carolina South Carolina	5 4 18 2	4 8 26 15	9 5 52	104	22 20 30	7 64 2	0 0 0 2	0 0 0
Georgia	4 4	6 3	6 2	5 1	10 16	4	1 0 1 3	3 3 0 2
Alabama Mississippi	6	7 12	ĭ	4	36	10	0 2	0 0

¹ New York City only.

³ Week ended Friday.

Cases of certain communicable diseases reported by telegraph by State health officers for weeks ended July 12, 1930, and July 13, 1939—Continued

	Dipl	ntheria	Infl	uen sa	Measles		Menin meni	gococcus ingitis
Division and State	Week ended July 12, 1930	Week ended July 13, 1929	Week ended July 12, 1930	Week ended July 13, 1929	Week ended July 12, 1930	Week ended July 13, 1929	Week ended July 12, 1930	Week ended July 13, 1929
West South Central States: Arkansas. Louisiana. Oklahoma 3. Texas. Mountain States:	1 19 6 10	1 12 10 17	7 3 4 1	5 18 8	4 1 16 14	6 8 14 34	0 1 1 1	0 3 2 0
Montana Idaho Wyoming Colorado New Mexico Arizona Utah ¹	1 1 6 3	10	1	2	2 4 10 68 13 61 19	7 6 7 7 5 4	1 2 0 0 0 1 4	2 1 0 0 1 0
Pacific States: Washington Oregon California	6 5 53	7 2 48	3 3 19	6 6	192 32 552	56 28 76	1 0 4	2 5
	Poliomyelitis		Scarle	t fever	Sma	llpox	Typhoi	id fever
Division and State	Week ended July 12, 1930	Week ended July 13, 1929	Week ended July 12, 1930	Week ended July 13, 1929	Week ended July 12, 1930	Week ended July 13, 1929	Week ended July 12, 1930	Week ended July 13, 1929
New England States: Maine New Hampshire Vermont Massachusetts Rhode Island Connecticut	000	0 0 0	19 1 0 73 6 7	11 6 0 78 3 19	0 0 0	0 0 1 0 0	0 0 0 8 1	6 0 0 1 0 2
Middle Atlantic States: New York New Jersey Pennsylvania East North Central States:	10 0 1	12 1 1	121 54 126	126 38 112	13 0 0	1 0 0	22 8 12	26 10 15
Ohio Indiana Illinois Michigan Wisconsin West North Central States:	1 5 3 1 0	1 0 1 0 0	121 42 146 99 40	146 46 111 170 52	51 76 34 40 10	43 31 27 42 8	21 11 26 4 0	23 3 16 1 2
Minnesota. Lowa	6 2 0 0 1 0	0 0 0 0 0 0	38 14 32 3 4 5	37 26 14 3 2 10	1 48 12 10 41 10 21	21 6 3 13 20 18	2 0 13 4 0 0	3 4 11 1 0 1 10
South Atlantic States: Delaware. Maryland ² District of Columbia. West Virginia. North Carolina. South Carolina. Georgia. Florida.	0 0 0 0 6 1 1	0 0 0 0 6 4 1	9 18 6 9 21 1 4	1 10 7 11 20 7 12	0 0 0 17 13 0 0	0 0 0 16 17 1 0	1 8 1 11 58 59 59	1 26 0 12 45 87 54
East South Central States: Kentucky Tennessee	0 1 3 1	0 4 1 0	18 7 2 4	24 3 15	0 10 0 1	5 1 1 1	22 56 24 58	7 43 24 40

³ Week ended Friday.

^{*} Figures for 1930 are exclusive of Oklahoma City and Tulsa.

Cases of certain communicable diseases reported by telegraph by State health officers for weeks ended July 12, 1930, and July 13, 1929—Continued

	Polion	Poliomyelitis		Scarlet fever		Smallpox		id fever
Division and State	Week ended July 12, 1930	Week ended July 13, 1929						
West South Central States: Arkansas Louisiana Oklahoma 3 Texas Mountain States:	1 29 14 1	0 0 0 1	4 12 8 5	1 9 10 22	12 1 34 24	0 1 13 24	39 34 16 16	29 33 9
Montana Idaho Wyoming Colorado New Mexico Arizona Utah ³		0 0 0 1 1 0	23 0 2 8 5 2 2	6 2 7 9 2 3	5 3 1 5 2 0	2 11 5 17 0 0 4	1 0 0 3 9 10	1 1 8 5 0
Pacific States: Washington	2 0 99	0 1 6	25 7 50	10 4 98	43 9 33	12 27 14	2 7 19	10 4 13

² Week ended Friday.

SUMMARY OF MONTHLY REPORTS FROM STATES

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

State	Cere- bro- spinal menin- gitis	Diph- theria	Influ- enza	Ma- laria	Mea- sles	Pel- lagra	Polio- mye- lįtis	Scarlet fever	Small- pox	Ty- phoid fever
April, 1930 Massachusetts May, 1930	18	284	45	2	5, 751		3	1, 206	0	20
Delaware		8			50		0	38	0	0
Iowa Massachusetts Nebraska New Jersey New Mexico North Dakota Porto Rico South Carolina Tennessee Vermont	5 16 2 16 6 2 	18 203 20 341 37 15 20 67 23	8 14 2 1 115 549 45	7 1 1 17 594 1,686 243	360 4, 227 279 4, 268 156 56 55 149 349 196	2 4 1,661 66	0 3 0 1 1 4 1 7 5	111 603 90 467 20 53 14 100 25	427 0 140 0 21 87 0 7 49	10 17 7 23 6 3 42 239 119 0

³ Figures for 1930 are exclusive of Oklahoma City and Tulsa.

April, 1930		Mumps:	Case
Massachusetts:	Cases	Iowa	4
Anthrax	4	Massachusetts	
Chicken pox		Nebraska	_
Dysentery		New Mexico	
Lethargic encephalitis		North Dakota	
Mumps	•	South Carolina	
Ophthalmia neonatorum		Tennessee	
Septic sore throat		Vermont	
Tetanus		Ophthalmia neonatorum:	
Trachoma	. 3	Massachusetts	103
Whooping cough	1, 315	New Jersey	:
May, 1930		Porto Rico	4
Delaware:		South Carolina	
Anthrax	1	Tennessee Paratyphoid fever:	
Chicken pox	19	South Carolina	,
Mumps		Puerperal septicemia:	•
Whooping cough	21	Porto Rico	10
June, 1930		Tennessee	1
Anthrax:		Rabies in animals:	
Massachusetts	1	South Carolina	8
New Jersey	1	Septic sore throat:	
Porto Rico	1	Massachusetts	19
Chicken pox:		Nebraska	3
Iowa	74 876	Tennessee	2
Massachusetts	113	Massachusetts	4
New Jersey	613	North Dakota	1
New Mexico	33	Porto Rico	8
North Dakota	15	South Carolina	1
South Carolina	209	Tetanus (infantile):	
Tennessee	83	Porto Rico	50
Vermont	96	Trachoma:	
Dengue:	_	Massachusetts	4
Porto Rico	1	New Jersey	5
South Carolina	5	Porto Rico	11
Porto Rico.	1	Trichinosis:	•••
South Carolina.	_	Massachusetts	3
Dysentery:	-,	Tularæmia:	
Massachusetts	1	Tennessee	1
New Jersey	1	Typhus fever:	
Porto Rico	7	New Jersey	1
Tennessee	63	South Carolina	1
Filariasis:		Undulant fever:	24
Porto Rico	1	Iowa Nebraska	24
Massachusetts	820	New Mexico	1
New Jersey	453	South Carolina	1
New Mexico	3	Vincent's angina:	
South Carolina	11	Iowa	3
Hookworm disease:		North Dakota	14
South Carolina	118	Tennessee	3
Impetigo contagiosa:	_	Whooping cough:	•0
Tennessee	1	Iowa Massachusetts	58 773
Lead poisoning:		Nebraska	33
Massachusetts New Jersey	4	New Jersey	316
Leprosy:	•	New Mexico	13
Porto Rico	1	North Dakota	89
Lethargic encephalitis:	-	Porto Rico	69
Massachusetts	3	South Carolina	400
New Mexico	1	Tennessee	122
North Dakota	2	Vermont	62
South Carolina	2		
Tennessee	1]		

GENERAL CURRENT SUMMARY AND WEEKLY REPORTS FROM CITIES

The 95 cities reporting cases used in the following table are situated in all parts of the country and have an estimated aggregate population of more than 31,475,-000. The estimated population of the 88 cities reporting deaths is more than 29,880,000. The estimated expectancy is based on the experience of the last nine years, excluding epidemics.

Weeks ended July 5, 1930, and July 6, 1929

	1930	1929	Estimated expectancy
. Cases reported			
Diphtheria:			!
46 States	650	968	
95 cities	356	543	568
Measles:			ľ
45 States	5, 538	5, 001	
95 cities	1, 695	1, 187	
Meningococcus meningitis:	· 1	•	
46 Štates	70	137	
95 cities	23	71	
Poliomyelitis:	1		
46 States	173	29	
Scarlet fever:			
46 States	1, 136	1, 383	1
95 cities	466	526	478
Smallpox:			1
46 States	770	647	ł
95 cities	40	92	23
Typhoid fever:			1
46 States	532	515	İ
95 cities	62	55	80
Deaths reported			
Influenza and pneumonia:			ľ
22 nitige	336	359	
88 cities	330	309	
88 cities	ام	•	1
00 010100	0	0	

City reports for week ended July 5, 1930

The "estimated expectancy" given for diphtheria, poliomyelitis, scarlet fever, smallpox, and typhoid fever is the result of an attempt to ascertain from previous occurrence the number of cases of the disease under consideration that may be expected to occur during a certain week in the absence of epidemics. It is based on reports to the Public Health Service during the past nine years. It is in most instances the median number of cases reported in the corresponding weeks of the preceding years. When the reports include several epidemics, or when for other reasons the median is unsatisfactory, the epidemic periods are excluded, and the estimated expectancy is the mean number of cases reported for the week during nonepidemic years.

If the reports have not been received for the full nine years, data are used for as many years as possible, but no year earlier than 1921 is included. In obtaining the estimated expectancy, the figures are smoothed when necessary to avoid abrupt deviation from the usual trend. For some of the diseases given in the table the available data were not sufficient to make it practicable to compute the estimated expectancy.

		Diph	theria	Influ	ienza			
Division; State, and city	Chicken pox, cases reported	Cases, estimated expect- ancy	Cases reported	Cases reported	Deaths reported	Measles, cases reported	Mumps, cases reported	Pneu- monia, deaths reported
NEW ENGLAND								
Maine: Portland New Hampshire: Concord	0	0	0		0	0	6	0
NashuaVermont:	ŏ	ŏ	ő		ŏ	0 5	0	0
Barre Burlington	0	0	0		0 0	5 0	0	2 0

City reports for week ended July 5, 1930—Continued

Division, State, and city Chicken pox, cases reported Cases, cases reported Cases, cases reported Cases reported	183	deaths reported
Massachusetts: 22 27 17 1 152 Fall River 3 2 3 0 1 Springfield 3 1 1 0 4 Worcester 2 1 0 0 43 Rhode Island: 2 0 0 0 0 0 Providence 9 3 2 0 17 Connecticut: 3 2 0 0 0 Bridgeport 1 3 0 0 0 Hartford 0 2 0 0 0	010000000000000000000000000000000000000	0 0 0 1 2
Boston	010000000000000000000000000000000000000	0 0 0 1 2
Fall River	010000000000000000000000000000000000000	0 0 0 1 2
Rhode Island:	0	. 0 0 1 2
Rhode Island:	0	1 2
Providence 9 3 2 0 17 Connecticut: Bridgeport 1 3 0 0 0 Hartford 0 0 0	0	2
Connecticut: Bridgeport 1 3 0 0 0 Hartford 0 0 0	0	1
Hartford 0 2 0 0 0	0	
		0
	_	ő
MIDDLE ATLANTIC		
New York:		
Buffalo	5 47	7 72
Rochester 4 5 3 1 1	1	1
Syracuse 0 33 New Jersey:	1	. 2
Camden	0	0
Newark 8 10 8 1 0 26 Trenton 4 1 0 1 0 9	9	4 0
Pennsylvania:		1
Philadelphia 31 39 12 1 95 Pittsburgh 14 14 18 1 77	26 3	23
Reading	6	1
Scranton 2	0	0
1 1. 1 1 1 1		1
Ohio: Cincinnati 0 4 2 0 37	8	
Cleveland 89 19 7 0 8	11	5 6
Columbus 11 3 2 2 0 10 Toledo 23 3 0 3	6 10	1 0
Indiana:		ľ
Fort Wayne 2 2 0 0 1 Indianapolis 5 2 0 0 10	0	0 9
South Bend 0 0 0 3	Ō	2
Illinois:	0	1
Chicago	44	22
Michigan:	0	0
Detroit 29 32 31 1 0 65 Flint 0 42	16	10
Grand Rapids 3 1 1 1 3	8	2 0
Wisconsin: Kenosha 4 0 0 0 2	7	0
Madison	ó	0
Milwaukee 62 9 2 0 22 Racine 0 11	19	7
Superior 0 0 0 0	ŏ	ŏ
WEST NORTH CENTRAL	+	
Minnesota:		
Duluth 0 0 0 0 5 Minneapolis 25 9 0 15	1	0 2
St. Paul	3	1
Iowa: Des Moines 0 1 0 0	0	
Sioux City 1 0 0 5	1	
Waterloo 0 0 0 0 0	0	
Kansas City		
St. Joseph 0 0 0 0 1 St. Louis 23 17 14 30	8	1
North Dakota:	- 1	
Fargo	8	1

City reports for week ended July 5, 1930—Continued

		Diph	theria	Infl	uenza				
Division, State, and city	Chicken pox, cases reported	Cases, estimated expect- ancy	stimated Cases expect- reported		Deaths reported	Measles, cases reported	Mumps, cases reported	Pneu- monia, deaths reported	
WEST NORTH CEN-									
South Dakota:	l .			1					
Aberdeen Sioux Falls	1 0	0	0			25	0		
Nebraska: Omaha	1 1	2	1			2	0	8	
Kansas: Topeka	2	0	0		0	3	2	1	
Wichita	ő	ŏ	ŏ		ŏ	5	ő	2	
SOUTH ATLANTIC									
Delaware: Wilmington	3	1	1		0	0	0	4	
Maryland:					İ			_	
Baltimore Cumberland	21 1	12 0	4 0		0	7 0	8	8 0	
Frederick	0	0	0		0	0	0	0	
Washington	5	4	5		1	43	0	3	
Virginia: Lynchburg	4	0	1		0	6	0	0	
Norfolk Richmond	1 3	0	0		0	3 8	0	1	
Roanoke	2	ō	Ŏ		ĭ	12	2	1	
West Virginia: Charleston	0	0	1		o	. 1	0	1	
Wheeling North Carolina:	1	0	0		0	1	0	0	
Raleigh	0	0	0		0	0	0	0	
Wilmington Winston-Salem	1 2	ŏ	ŏ		ŏ	1	2	ŏ	
South Carolina: Charleston	ol	ol	0	10	. 0	0	1	1	
Columbia Georgia:	i	Ō	Ō		Ō	1	1	Ō	
Atlanta		2							
Brunswick Savannah	i	0 1	0		·ō		3	i	
Florida: Miami	o	1	o		o	0	1	1	
St. Petersburg		0 .			0			0	
Tampa	0	0	. 0		0	6	0	2	
EAST SOUTH CENTRAL	i						Į		
Kentucky:	ا	ام			ام		ا	4	
Covington Tennessee:	0	0	3		0	1	0	-	
Memphis Nashville	1 2	8	0		0	1 12	8	7	
Alabama:	1	1	2	1	1	7	1	9	
Birmingham Mobile	0	0	0		ő	0	0	i	
Montgomery	1	٥١	1			0	1		
WEST SOUTH CENTRAL									
Arkansas: Fort Smith	0	o	0			0	0		
Little Rock Louisiana:	Ō	Ō	0		0	Ō	0	0	
New Orleans	o l	5	4	1	3	1	0	9	
Shreveport Oklahoma:	0	0	0		0	1	3	2	
Tulsa Texas:	3	0	1			0	0 -		
Dallas	o l	3	2		0	2	0	4	
Fort Worth Galveston	0	3 1 0 2 1	0 .		0	0 0 3	0	ģ	
Houston	8	1	7		0	3	0	0 4 3	
	•	•	•	-,	•	•	•		

City reports for week ended July 5, 1930—Continued

		Diph	theria	Influ	lenza				
Division, State, ar	Chicken pox, cases reported	Cases, estimated expectancy		Cases reported	Deaths reported	Measles, cases reported	Mumps, cases reported	Pneu- monia, deaths reported	
MOUNTAIN									
Montana: Billings Great Falls Helena Missoula	0	0	0		0	2 0 1	0 2 0 1	, 0	
Idaho: Boise	I	0	o		0	2	0	1	
Denver Pueblo New Mexico:		'7 1	1 0		0	25 32	3 6	4	
Albuquerque Arizona:	1 1	0	0		0	0	0	0	
Phoenix Utah:	0	0	0		0	1	0	4	
Salt Lake City_ Nevada:	4	3	0		0	21	4	2	
Reno	0	0	0		0	0	0	0	
Washington: Seattle Spokane Tacoma Oregon:	3	3 1 2	0 0 0		0	53 15 24	31 0 0	<u>-</u> 2	
Portland		5 0	2 0		0	12 0	3	7 0	
Los Angeles Sacramento San Francisco	17 0 9	35 2 10	13 2 1	7	2 0 1	103 12 16	28 0 16	17 1 1	
	Scarlet fever	81	nallpox	Tuber	Typi	noid fever	Whoon	J	

	Scarlet fever Smallpox			Tuber-	T	rphoid f	Whoop-				
Division, State, and city	Cases, esti- mated expect- ancy	Cases re- ported	Cases, esti- mated expect- ancy	Cases re- ported	Deaths re- ported	culo- sis, deaths re- ported	Cases, esti- mated expect- ancy	Cases re-	Deaths re- ported	ing cough, cases re- ported	Deaths, all causes
NEW ENGLAND											
Maine: Portland New Hampshire:	0	3	0	0	0	3	1	0	0	12	16
Concord	0	Q	o	o	0	0	0	0.	0	0	7
Nashua Vermont:	0	0	0	0	0	0	0	0	0	0	0
Barre Burlington Massachusetts:	0	0	0	0	0	0	0	0	0	0	7 6
Boston Fall River Springfield	31 1	15 2	0	0	0	16 2	2 1	0	0	41 4	187 24
Worcester Rhode Island:	3	. 1 2	0	0	0	0 2	0	0	8	3 4	22 43
Pawtucket Providence Connecticut:	3	0	0	8	8	1 2	0	8	8	0 2	9 53
Bridgeport Hartford New Haven	3 2	1· 2 0	0	0	0	3	0	0 2	0	0	29 36
MIDDLE ATLANTIC	1	١	0	0	0	2	0	0	0	٥	31
New York: Buffalo	12	6	0	0	0	11	1	0	0	17	119
New York Rochester Syracuse	83 3 3	48 3 4	0	0	0	72 2 3	16 0 0	8 0 0	0	68 2 34	1, 159 57 34
New Jersey: Camden Newark Trenton	1 10 1	2 8 5	0	0	0 0	0 14 4	0 1 0	0 0 2	0	0 16 0	15 87 27
Pennsylvania: Philadelphia_PittsburghReadingScranton	37 15 1	25 17 2 1	0 0 0	0	0 0 0	36 5 2 0	4 1 0 0	1 0 1 0	0	16 25 9 5	421 136 23

			1			1	1				
	Scarle	t fever		Smallp	OX .	Tuber-	T	phoid f	lever	Whoop	
Division, State, and city	Cases, esti- mated expect- ancy	Cases re- ported	Cases, esti- mated expect- ancy	Cases re- ported	Deaths re- ported	culo- sis, deaths re-	Cases, esti- mated expect- ancy	Cases re- ported	Deaths re- ported	ing cough, cases re- ported	Deaths, all causes
EAST NORTH CENTRAL											
Ohio: Cincinnati Cleveland Columbus Toledo Indiana:	6 20 3 5	7 19 4 9	0	0 0 0 2	0 0 0 0	12 12 1 5	1 2 0 0	0 1 0 1	000	8 50 3 0	102 151 68 52
Fort Wayne Indianapolis South Bend Terre Haute Illinois:	1 3 1 0	1 2 0 0	3 0 0	2 4 0 0	0 0 0	0 4 0 0	0 0 0	0 0 0	1 0 0 0	1 8 0 0	20 19 10
Chicago Springfield Michigan:	55 1	104 0	2 0	0	0	45 1	3 0	0	1 0	54 1	590 16
Detroit Flint Grand Rapids. Wisconsin:	42 4 5	29 6 3	2 0 0	0 0 1	0	23 2 0	3 0 1	0 0 0	1 0 0	57 13 3	219 18 25
Kenosha Madison Milwaukee Racine Superior	1 0 11 2 2	0 2 10 0 0	0 0 1 0 0	0 0 1 0 0	. 0 0 0	0 1 1 0 0	0 0 0 0	0 0 0 0	0 0 0 0	13 9 51 4 0	2 26 79 5 7
WEST NORTH CENTRAL						•					
Minnesota: Duluth Minneapolis St. Paul	4 15 9	. 0 7 6	0 1 0	0 0 0	0	2 0 1	0 1 1	1 2 0	0	8 1 2	16 71 34
Iowa: Des Moines Sioux City Waterloo Missouri:	3 1 1	1 1 0	0 0 1	12 1 3			0 0 0	0 0 0		0 2 1	30
Kansas City St. Joseph St. Louis North Dakota:	3 0 10	6 22	0	0 1	0	1 6	1 0 3	0	0	0 21	18 170
Fargo	0	0	0	1 4 5	0	1	0	0	0	0 0 3	14
Sioux Falls Nebraska: Omaha	ŏ	ŏ 5	0	ŏ	0	0	ŏ	ŏ	0	ŏ	6 59
Kansas: Topeka Wichita	0	4 0	0	0	0	1 2	0	0	0	10 2	15 41
SOUTH ATLANTIC			İ		l	İ				l	
Delaware: Wilmington Maryland:	1	3	0	0	0	1	0	0	0	2	23
Baltimore	10 0 0	16 0 0	0	0	0	11 0 0	3 0 0	3 0 0	1 0 0	31 2 0	158 12 5
lumbia: Washington Virginia:	7	4	0	0	0	12	1	o	0	9	126
Lynchburg Norfolk Richmond Roanoke	0 0 1 0	0 0 -	0 1 0	0	0	0 1 5 1	0 1 1 0	3 0 4 0	0 0 0	2 4 0 0	15 46 18
West Virginia: Charleston Wheeling	0	0	1 0	8	0	0	1 1	0	0	4 0	12 14
North Carolina: Raleigh Wilmington Winston-Salem	0	0 0 1	0 0 1	1 0 0	0	0 0 2	0 0 1	0 0 1	1 0 1	0	10 14 26

	Scark	st fover		Smallp	ox	Tuber	T;	yphoid i	lever	Whoop	
Division, State, and city	Cases, esti- mated expect- ancy	Cases re-	Cases, esti- mated expect- ancy	Cases re- ported	Deaths re- ported	culo- sis, deaths	Cases, esti- mated expect- ancy	Cases re- ported	Deaths re- ported	ing cough, cases re- ported	lane
SOUTH ATLANTIC— continued											
South Carolina: Charleston Columbia Georgia: Atlanta	0 0 2	1 1	0 0 1	0	0	5	1 1 3	0 1	0	0 2	19
Brunswick	Ó		Ō				0				
Savannah Florida:	1	0	0	0	0	2	2	1	0	0	26
Miami St. Petersburg_ Tampa	0 0 1	0 0	0	0	0	3 0 2	1 0 1	0	0 0 0	<u>0</u>	29 13 9
EAST SOUTH CENTRAL Kentucky:											
Covington	0	0	0	0	0	0	0	0	0	0	20
Tennessee: Memphis Nashville Alabama:	2 1	0	0	0 3	0	5 2	6 5	4 2	0	9	91 42
Birmingham Mobile	8	2	1 0	8	0	2	3 0	7	2	0	88
Montgomery WEST SOUTH CENTRAL	ŏ	ŏ	ŏ	ŏ			ĭ	ô		ŏ	23
Arkansas:							1				
Fort Smith Little Rock Louisiana:	0	0	8	0	0	0	0	0	0	0	
New Orleans Shreveport Oklahoma:	3	10	0	0	0	9	3	8	1 1	0	129 37
Tulsa Texas:	0	0	1	2			2	0 -		0	
Dallas Fort Worth	2	0	0	8	0	4	2	0	0	8	55 37
Galveston Houston	0	0	0	0	0	0 3	0	1	0	0	15 82
San Antonio MOUNTAIN Montana:	°	2	. 0	°	0	7	0	1	0	٥	69
Billings	0	0 13	0	0	0	1 0	0	0	8	0	4
Helena Missoula Idaho:	1 0	1 0	0	8	8	0	ö	0		1 3	10 2 3
BoiseColorado:	0	0	0	0	0	0	0	0	0	1	3
Denver Pueblo	6	1	8	0 5	0	12	0	0	0	49	81 7
New Mexico: Albuquerque	0		0			2	0				5
Arizona: Phoenix	0	٥	o	0	0	3	0		2		22
Utah: Salt Lake City	1	4	0	0	0	0	o	0	0	28	27
Nevada: Reno	0	0	0	1	0	0	o	0	0	0	3
PACIFIC Washington:					l]			
Seattle Spokane	4 2	1 0	1	2			1 0	8		10 -	
TacomaOregon:	í	ĭ	2	3	0	0	ŏ	8	0	10	29
Portland Salem California:	0	0	7	0	0	0	0	1 0	0	8	62
Los Angeles Sacramento San Francisco.	15 1 8	8 1 8	3 0 0	6 2 0	0	29 0 15	2 1 0	1 1 0	0	27 1 4	247 28 160

	Menin meni	gococcus ngitis		rgic en- alitis	Pell	lagra	Poliom	yelitis (i paralysis	infantile)
Division, State, and city	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases, esti- mated expect- ancy	Cases	Deaths
NEW ENGLAND									
Connecticut: New Haven	` 1	o	0	o	0	o	0	0	0
MIDDLE ATLANTIC									
New York: Buffalo New York Rochester	1 2 1	1 6 0	0 0 0	0 2 0	0	0 0 0	0 4 0	0	0
Pennsylvania: PhiladelphiaPittsburgh	0	2 2	0	0	0	0	0	. 1 0	0
EAST NORTH CENTRAL									
Ohio: Cincinnati ¹	1	1	0	0	0	o	0	0	. 0
Indiana: IndianapolisIllinois:	1	1	0	0	0	0	0	1	0
Chicago	3	. 2	0	0	0	0	1	. 0	0
Detroit	3	1	1	0	0	0	0	1	0
WEST NORTH CENTRAL Missouri:									
St. Joseph St. Louis North Dakota:	1	0	0	0	0	0	0	0 1	0
North Dakota: Fargo Nebraska:	0	0	2	.0	0	0	0	0	0
Omaha Kansas:	1	0	0	0	0	0	0	0	0
Wichita	0	0	0	0	0	0	0	1	0
SOUTH ATLANTIC						Ī	1		
District of Columbia: Washington Virginia:	1	0	0	0	0	0	0	0	0
Norfolk Richmond	2 0	0	8	0	0	0	0	1 0	0
North Carolina: Wilmington	0	0	0	0	2	1	0	0	0
Winston-Salem South Carolina: Charleston	0	0	0	0	8 12	0	0	0	0
Georgia: Savannah				. 0	1	1	0	0	0
EAST SOUTH CENTRAL		1			l				
Tennessee: Memphis Nashville	4 0	2 0	0	0	0	1 0	0	0	0
Alabama: . Birmingham Mobile	0	1 0	0	1 0	0	0 2	0	0	0
WEST SOUTH CENTRAL			- 1					į	
Arkansas: Little Rock		اه	o	o	0	1	0	o	0
New Orleans	0	0	0	0	1	1	0	0	0
Shreveport Oklahoma: Tulsa	0	1 0	0	0	0	4	0	1	0

Rabies (in man): 1 case and 1 death at Cincinnati, Ohio.
 Typhus fever: 2 cases at Savannah, Ga.

	Menin meni	gococcus ngitis	Letha: . ceph	rgic en- malitis	Pel	lagra	Poliomyelitis (infantile paralysis)			
Division, State, and city	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases, esti- mated expect- ancy	Cases	Deaths	
WEST SOUTH CENTRAL— continued										
Texas: Dallas Fort Worth Houston	0	0 0 0	0	0 0 0	1 0 0	4 4 2	1 0 0	0	0 0 0	
MOUNTAIN						ĺ				
Colorado: DenverArizona:	0	1	0	0	0	0	0	. 0	0	
Phoenix	2	0	0	0	0	0	0	0	0	
PACIFIC					-					
California: Los Angeles Sacramento	1 0	0	0	0	0	0 1	0	49 0	4 0	

The following table gives the rates per 100,000 population for 98 cities for the 5-week period ended July 5, 1930, compared with those for a like period ended July 6, 1929. The population figures used in computing the rates are approximate estimates, authoritative figures for many of the cities not being available. The 98 cities reporting cases have an estimated aggregate population of more than 32,000,000. The 91 cities reporting deaths have more than 30,500,000 estimated population.

Summary of weekly reports from cities, June 1 to July 5, 1930—Annual rates per 100,000 population, compared with rates for the corresponding period of 1929 ¹

DIPHTHERIA CASE RATES

		Week ended—										
	June 7, 1930	June 8, 1929	June 14, 1930	June 15, 1929	June 21, 1930	June 22, 1929	June 28, 1930	June 29, 1929	July 5, 1930	July 6, 1929		
98 cities	76	110	80	106	68	112	67	110	2 59	8		
New England	86	72	35	79	35	74	62	94	51	7		
Middle Atlantic	72	148	82	131	81	125	65	144	59	10		
East North Central	113	123	129	145	93	165	98	131	91	12		
West North Central	51	96	59	65	34	87	70	85	1 33	7		
South Atlantic	49	54	40	64	33	64	24	34	4 24	3		
East South Central	13	21	13	41	13	34	13	34	40	2		
West South Central	41	88	86	84	86	65	37	69	52	7		
Mountain.	17	61	34	35	9	26	Ö	26	9	2		
Pacific	76	56	43	34	54	58	64	84	38	4		

MEASLES CASE RATES

•										
98 cities	955	734	833	483	656	423	500	267	2 281	195
New England	1,462	602	1,415	337	1,048	391	762	211	498	209
Middle Atlantic	1,076	169	1.089	143	818	123	640	99	339	76
East North Central	517	1,827	457	1, 152	381	1.010	334	620	170	474
West North Central	412	1,060	362	581	658	504	264	256	3 154	114
South Atlantic	478	238	362	242	375	129	234	137	4 175	73
East South Central	418	41	182	41	270	41	256	7	142	27
West South Central	123	400	101	209	82	183	19	156	26	69
Mountain	5. 518	192	3, 321	261	2,617	218	1,416	148	712	148
Pacific	2, 220	408	1, 564	384	1, 247	352	931	208	527	128
							•			

Summary of weekly reports from cities, June 1 to July 5, 1930—Annual rates per 100,000 population, compared with rates for the corresponding period of 1929 — Continued

SCARLET FEVER CASE RATES

				,	Week er	nded→						
	June 7.	June 8.	June 14,	June 15,	June 21,	June 22, 1929	June 28,	June 29,	July 5, 1930	July 6, 1929		
	7, 1930	8, 1929	14, 1930	15, 1929	21, 1930	1929	1930	1929	1930	1929		
98 cities	213	209	192	188	145	148	109	112	2 77	88		
New England	230 196	191 135	199 155	204 129	115 118	159 100	124 89	119 72	66 57	90		
Middle Atlantic East North Central	296	321	334	322	229	260	184	191	116	17		
West North Central	260 156	165 300	233 145	110 133	148 97	77	97 62	104 62	3 114 4 55	173 34 5 5 24 13		
South AtlanticEast South Central	108	96	54	75	67	89	61	34	13	Š		
West South Central Mountain	78 189	76 78	37 129	107 70	105 197	88 96	41 60	42 70	49 163	4		
Pacific	109	270	113	251	85	210	57	164	45	13		
SMALLPOX CASE RATES												
98 cities	20	8	15	16	10	9	13	15	27	1.		
New England	0	0	0	0	0	0	0	0	0			
Middle Atlantic East North Central	1 8	17	0 11	28	0 8	0 18	0 10	38	0 5	4		
West North Central	116	12	53	12	30	6	51	19 2	¹ 13	4 1 2 1		
South AtlanticEast South Atlantic	4 34	2 14	7 40	4 55	2 20	6	9 7	7	20	2		
West South Central	22	8	22	42	26 34	61	22 51	113	0 51	1		
MountainPacific	60 68	52 14	34 57	44 46	43	31	50	14	38	3		
TYPHOID FEVER CASE RATES												
98 cities	8	8	9	9	8	8	13	12	³ 10	10		
New England	4	7	9	11	0	4	9	9	7	•		
Middle AtlanticEast North Central	6	5 3	8 4	3 4	3	2 4	5 10	7 3	6			
West North Central	9	8	6	17		19	13	15	17	1		
South Atlantic East South Central	20 13	17	15 27	11 34	22 54 26	13 55	37 67	30 34	4 28 94	3		
West South Central	37	27 27	19	19	26	34	34	34 34	49			
Mountain	0 2	0 12	9 19	9 19	9 7	9 5	34 5	52 19	0 5	1: 3: 4!		
1 80000		<u>'</u>	ENZA :		I RAT	ES			1			
91 cities	5	7	6	6	4	6	3	5	24			
New England	- 0		- 3	7		2	0	2	2			
Middle Atlantic East North Central	4	2 5 6	5	4	2 5	1 3	2 3	4	4 2	1		
East North Central West North Central	12	6 3	6 15	8 9	4	8	0	4 0	1 30			
South Atlantic East South Central	9	3 7	15 15	9 2 7	2	6	5	4	44			
East South Central	15 11	22 16	15 27	7 12	15 8	15 16	15 11	15	7 15	1		
West South Central Mountain	9	35	0	0	0	1 0	0	44	0			
Pacific	3	16	6	6	0	6	8	3	9			
	P	NEUM	ONIA	DEAT	H RAT	ES						
91 cities	86	90	85	86	74	81	68	64	2 55	63		
New England	73 106	65 105	82 101	85 98	69 82	56 89	49 75	58 65	29 58	49		
East North Central	59	96	67	82	53	76	56	69	41	6		
West North Central	130	81	77	54	109	48	86	48	\$ 62 4 51	6 6 7 10		
South AtlanticEast South Central	93 81	67 60	73 110	88 104	64 133	84 119	66 103	62 75	4 51 162	7		
West South Central	84	90	107	62	69	82	92	66	84	10		
Mountain	112	61 69	86 71	113 60	129 74	78 104	77 55	104 38	60 64	61		
Pacific	40											

¹ The figures given in this table are rates per 100,000 population, annual basis, and not the number of cases reported. Populations used are estimated as of July 1, 1930, and 1929, respectively.

3 Kansas City, Mo., Atlanta and Brunswick, Ga., not included.

4 Kansas City, Mo., not included.

4 Atlanta and Brunswick, Ga., not included.

FOREIGN AND INSULAR

CANADA

Provinces—Communicable diseases—Week ended June 28, 1930.— The Department of Pensions and National Health reports cases of certain communicable diseases in Canada for the week ended June 28, 1930, as follows:

Province	Cerebro- spinal fever	Influ- enza	Polio- myelitis	Small- pox	Typhoid fever
Prince Edward Island 1				`	
Nova Scotia		4			
New Brunswick					5
QuebecOntario	1 2			10	11
Manitoba			l	10	1
Saskatchewan					î
Alberta	1				
British Columbia					1
Total	4	8	1	10	23

¹ No case of any disease included in the table was reported during the week.

Quebec Province—Communicable diseases—Week ended July 5, 1930.—The Bureau of Health of the Province of Quebec, Canada, reports cases of certain communicable diseases for the week ended July 5, 1930, as follows:

Disease	Cases	Disease	Cases
Chicken pox Diphtheria. German measles Influenza Lethargic encephalitis Measles Mumps	34 31 3 1 1 36 15	Ophthalmia neonatorum Poliomyelitis Scarlet fever Smallpox Tuberculosis Typhoid fever Whooping cough	2 1 30 3 60 9 12

Ontario Province—Communicable diseases (comparative)—Four weeks ended June 26, 1930.—The following table shows the number of cases of certain communicable diseases, with deaths therefrom, reported in the Province of Ontario, Canada, for the four weeks ended June 26, 1930, as compared with the corresponding period of 1929:

	4 week	rs, 19 29	4 weeks, 1930		
Disease	Cases	Deaths	Cases	Deaths	
erebrospinal meningitis	4	5	11		
hancroid		Ŏ	6	1	
hicken pox		l ŏ	836	Ì	
)iphtheria		14	237	1 1	
rysipelas	2		2		
lerman measles	36		429		
ioi tre	1		1		
lonorrhea.	189	İ	130	l	
nflue nza	17	2	13	2	
ethargic encephalitis	2	1	1	1 1	
Measles	3, 077	2	1, 319		
lumps	463	0	130		
Paratyphoid fever	2	l	5		
neumonia		158		130	
oliomyelitis		2	2		
uerperal septicemia		1	0	2	
carlet fever	446	2	511	2	
eptic sore throat			1		
mallpox		0	1 47	0	
yphilis	158	2	122		
etanus				1	
rachoma			1		
uberculosis		44	129	48	
yphoid fever	66		30	<i>-</i>	
ndulant fever			11	! -	
hooping cough	550	1	232	! 	

¹ Cases of smallpox for this period were distributed as follows: Ottawa, 10; Nairn, 12; Sudbury, 6; Toronto, 3; Welland, 3; 1 case in each of the following places, Guelph, Espanoia, Sullivan, Napean.

CUBA

Habana—Communicable diseases—June, 1930.—During the month of June, 1930, certain communicable diseases were reported in Habana, Cuba, as follows:

Disease	Cases	Deaths	Disease	Cases	Death
Chicken pox	11 12 2 13	1	Measles Scarlet fever Tuberculosis Typhoid fever	1 8 41 15	9 2

Provinces—Notifiable diseases—Four weeks ended June 7, 1930.— During the four weeks ended June 7, 1930, cases of certain diseases were reported in Cuba as follows:

Disease ·	Pinar del Rio	Habana	Matan- zas	Santa Clara	Cama- guey	Oriente	Total
Cancer Chicken pox Diphtheria Malaria Measles	36 1	41 17 7	4	1 6 1 1 10	4 1 7	3 1 39	5 90 21 54 16
Paratyphoid fever Scarlet fever Tetanus (infantile)	2	7 22	2 1	1 1		7	17 24 2
Typhoid lever	7	29	9	41	7	12	105

1746

MEXICO

Tampico—Communicable diseases—June, 1980.—During the month of June, 1930, certain communicable diseases were reported in Tampico, Mexico, as follows:

Disease	Cases	Deaths	Disease	Cases	Deaths
Diphtheria. Enteritis (various)	3 1 63	1 51 13	Measles Smallpox Tuberculosis Typhoid fever Whooping cough	4 2 25 25	21 5

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

From medical officers of the Public Health Service, American consuls, International Office of Public Hygiene, Pan American Sanitary Bureau, health section of the Loague of Nations, and other sources. The reports contacting in the following tables must not be complete or final as regards either the list of countries for which reports are given.

CHOLERA

									A	Week ended-	Î						
Place	Jan. 12- Feb. 8,	Feb. 9- Mar. 8, 1930	Feb. 9- Mar. 9- Mar. 8, Arr. 5, 1930		April, 1930	Q		4	May, 1980				June, 1980	0861	-	July, 1980	98
	3			13	19	88	60	01	11	74	31	7	14	21	8	8	21
Afghanistan China:									-		-		6				A
	8,461 906	1 5,914 3,371	10, 817 5, 866	4,947 2,924	7, 436	15, 870 10, 403	13. 209 10, 234	15, 596 12, 782	14, 600 11, 882	12, 468 9, 756	100	4	60				
			•	00 00			~		40			Ħ	$\dagger \dagger$	$\dagger \dagger$	$\dagger \dagger$	$\dagger \dagger$	Ì
Calciuta, Calciuta D Neganatam	25 25 25 21	289 153	**************************************	137 85	165 118	165 118	85 88	194 125	175 107	142 83	24.88	% ‡	282	<u>2</u> 28	83		
Rangoon	4000	e -	6161						91	7	6161			8-	8-		
India (French): Chandernagor C Karikal	-	404	62	1	8 -	4	∞-1	21	614	1					8-		
	120000	. 07-24	o 646	12	1 12 1	2 62	88	1 88	2402	2,42	131	17.12	5455	2	700		

¹ An outbreak of cholers was reported in June, 1930, in Afghanistan.

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

CHOLERA—Continued

									We	Week ended—	<u>T</u>						
Place	12- F 12- F Feb. 8, N	Feb. 9- Mar. 9- Mar. 8, Apr. 5, 1930 1930	Mar.9 Apr. 5 1930		April, 1930	00		M	May, 1930				June, 1930	88		July, 1930	8
	3			13	19	88	8	01	11	*	E S	7	71	12	88		2
Philippine Islands: Bullecan Province— Malolos												-			-		1
South Mondo									Ī				+		+		
Cebu				<u> </u>							İ		9	8	- s	1 2	1
Barili												900	9	88	32-	5 co -	#
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Masbate Province— Catbabayn— D Cataingan Misami Province—San Miguel D	Binalbagan Cadir Calatrava Isabaltava			Vula Herinosa Pampanga Provinc o Angeles Bacolor Lubao. Pangasinan Province—Binmaloy Rital Province—Navotas.

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

CHOLERA—Continued

									₩	Week ended-	1						
Place	F. 65.	12- Feb. 9- Mar. 9, Apr. 5, 1930	Mar.9- Apr. 5, 1930		April, 1930			M	May, 1930				June, 1930	0261		July, 1930	088
i				13	19	8	60	92	11	8	31	7	71	21	88	2	12
Bangkok	00000 000000	P40 11 11	1 91	8644	10000	© 03 4 11 4 4 43	80 N CO CO	62	40	6060	4000	11		© 4'®			
	Десет.	January	Febr		Maı	March, 1930	6	•	April, 1930	330		Ms	May, 1930		- F	June, 1930	8
Fiace	ber, 1929	1930	ary, 1930		1-10	11-20	21-31	1-10	11-20	21-30	<u> </u>	1-10	11-20	21-31	1-10		11-20
Indo-China (French) (see also table above): Annam . Cambodia !	17.84	147		4.88	64	2222	55	81.83	8			288	ន្តដូ	259 259		2821	188 2
1 Dans de la complete																	

¹ Reports incomplete.

PLAGUE [C indicates cases; D, deaths; P, present]

	Jan.	Feb.	Mar.						W	Week ended-	l be						
Place	Feb. 8,	Agr.	A pr.	ΨĎ	April, 1930	-		May	May, 1930			2	June, 1930		Ju.	July, 1930	ا ـ ا
,	1930	1930	1930	12	19	8	ဗ	10 1	17 2	24 31	1		14 21	88	80	21	, ,
Algeria: Aktiers Constantine Argentina:															1	:	
Andalgala.¹ Rosario Santo Santa Fe Villa Lia Azores: Ponta Delgada.	Р	81			T	F-1			$\dashv \dag \dag$		$\frac{1}{1}$::::
Belgian Congo B						•										88	: ::
Brazil: Rio de Janeiro																	
oelow):		7			=	8											· •
UgandaO	82	47	88	27	282	522	38	23	288	╫	₩	₩					: : :
Ceylon: Colombo Plague-infected rats Colile: Antologasta	4488	∞∞∞	4461-6	63				41.00			i						11111
Dutch East Indies: Batavia and West JavaD Plague-infected rata		25 25 8	121 122 8	22,	16 16 16	೩೩೪	222	82 82	88	17 17 8	62	60					1111
D Java and Madura	317	288	223	88	3	35	8	88	74	38	$\frac{+1}{11}$	$^{\rm H}$	∦	₩	∦	╣	: :
1 On Mar. 11, 3 deaths from bubonic plague were reported in Andalgala, Catamarca Province, Argentina, since Feb. 5, 1930. 21 cases of plague with 8 deaths reported Jan. 29, 1830, in the State of Sec Faulo, Bratli, 15 of these cases were in the city of Sec Faulo.	in Andal the Stat	gala, Cat s of Sao I	amarca aulo, B	Provinc razil; 14	e, Arg	entina, se case	since F	eb. 5, 1 n the c	930. Ity of E	80 Pat	형						

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

PLAGUE-Continued

[C indicates cases; D, deaths; P, present]

	Jan.	Feb.	Mar.						P	7eek ei	Week ended-						
Place	Feb.	Mar. 8	Apr.	ΨÞ	April, 1930			Ma	May, 1930			•	June, 1930	020		July, 1930	98
	1930	1830	1930	13	81	8	80	91	- 11	*	ឌ	-	7		83		22
Ecuador (see table below). Egypt:	•	-	•			-	-	6	«	65	· ·	~	•	•	•	•	
Assiout						1-1-		. 69	9	· ~ ~	969	i ~	[0]	900	110	967	
	2		7			~	~		69	-	-		-		69		
Beni Suef. C. Dakahlieh. C. C.		œ	40	67		-	64	100	-	61	10	F	H	Ħ	Ħ	Ħ	
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Girea				F	T	-		$\dagger \dagger$	$\dagger \dagger$			11-	╫	Ħ	Ħ	Ì	
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Piraeus	-		1			$\frac{1}{1}$	$\overline{\parallel}$	Ħ	Ħ	Ħ	$\dagger \dagger$	$\frac{11}{11}$	T	+	Ħ	Ħ	
ii: Plague-infected rats.	4.814	5.639	4.087	917	343	536	8	158	- <u>s</u>	141				-			
Bassein	3,308 1	3, 940	3,344	753	623	517	\$	271	<u>S</u>	8							
*		-	-1		4.	†ŀ	Ħ	4.	-	$\dagger \dagger$	-	-	$\frac{1}{11}$	<u>~</u>	T		
Plague-infected rats	785	7 E &	86 157	-82	. % 15	4. ¹⁵ C	155 œ	-82%	*8	9	61	0	7	-∞≈	140		
Rangoon	က္ကက	140		9	သက	4 -	- 63			4 10	$\dagger \dagger$	Ħ	$\dagger \dagger$	İT		II	
Plague-infected rats India (Portuguese)	1-	7	9	-	101	1	1-		160					-	1		

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

PLAGUE—Continued

Place	Janu- gry, 1930	Feb- ru- ary, 1930	March, April, May, 1930 1930 1930	April, 1930	May, 1930	June, 1930	Place	Janu- ary, 1930	Feb- ru- ary, 1930	March, April, May, 1930 1930 1930	A pril, 1930		June, 1930
Eritish East Africa (see also table above): Kenya. Kenya. Uganda. Cusador: Guayaquil. Plague-infected rats. Cuador (outside of Guayaquil). Cuece (see also table above). Ambositra Frovince. Ambositra Province. Itasy Province. Maiafinarivo Province. D Miarinarivo Province.	44824 2888 2888 2888 2888 2888 2888 288	88000 8 3122 22	6 6 88 88 4 4 4 4 4 4 4 4 4 4 4 4 4 4 4	000 14 4200	000 44 4266 44 41		Madagascar—Continued. Moramanga Province	<u> </u>	222 223 33 33 100 110 1 3 1 3 1 3 1 3 1 3 1 3 1 3 1 3 1 3 1 3	25 8 8 8 1 1 1 2 2 1 2 8	88 42448511988	2122222088	

¹ Incomplete reports.

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1	1930	22													
	July, 1930	9									1	8			
		88								9	-	*			
	1930	21	1						7	13	∞ - -	01			•
	June, 1930	14							1	9		12			
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Week ended—		31	7	T	276	24	89		~~	ล	- 2	69			
Week		22			58	æ	3,∞		42	2	7-80	020			
	1930	17	-		*	·			İ	8	2	9			
	May, 1930	10		$\frac{11}{11}$	8	e	84		$\frac{1}{11}$	41	- 60	ล	i		
		8		 		4	*		60	2	2	22	ω.	•	-
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	April, 1930	19				$\frac{1}{1}$		-	-	ຂ	4	10		Ħ	-
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Feb	P.Σ.∞	 1930 1930		<u> </u>							808			in	· }
Ian	Feb.	1930	91	20-	4 10			<u> </u>		·				<u> </u>	
Dec.	1929- Jan.	1830		1	7 7	۵	86	, 91	272	, <u>1</u>	7	8 5 8			<u> </u>
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	Place		eria: Affers. Constantine	Oran abia: Aden livia: La Paz. ¹	azii: Rio de Janeiro. itish Borneo: Sarawak itish East Africa (see also table below): Tanganyika.	itish South Africa: Northern Rhodesia	Southern Rhodesia	nada: Alberta	Edmonton British Columbia—Vancouver	Ontario Ontario Fort William	North Bay. Ottawa. Toronto	Quebec Montreal Saskatchewan Regina	eylon: Angoda, Western Province	Colombo	

¹ From Jan. 1 to May 31, 1930, 44 deaths from smallpox were reported in La Paz, Bolivia.

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

SMALLPOX—Continued

	Ã										Weel	Week ended-	,					
Place	1829- Jan.	7. Feb.	a Ž	A pr		April, 1930	8		M	May, 1930				June, 1930	088		July, 1980	8
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China: Canton.	<u> </u>	100				-							-					
Chungking C Foochow.	<u>:</u>	P.A.		∞ын			Ы	ው	P	А	Ь	ММ	А			$\overline{\Pi}$	TH	
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Manchuria— Harbin————————————————————————————————————				160				-	-	9	ör.			Ħ	20.	ii	00	
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n s table below).			• 	о н	000						1	*	-	$\overline{ }$				
Colombia: Buenquilla. C Buenaventura.		13		 102	41	10	63		63		1		-	-	T			
Costa Rica: Port Limon		+	+			-	61	~~				8	8		\uparrow			
Curacao (alastrim). Curacao (alastrim). Curacao (alastrim). Curacao (alastrim).					14	<u> </u>	-	-	C4					-				
Dutch East Indies: Belawan Deli		1 1 1		185	5 17 2 16	12	88	01		10	4							
Batavia and West JavaD		10 10	14	71.	878 08.00		••••				10.09	-6						
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Sanggi Islands	Great Britain: England and Wales Ashton under Lyne Cardiff	Leeds London London and Great Towns shownid	Stoke-on-Trent Scotland	Hedjar India	Bombay	Cochin Karachi	Madras. Moulmein	Negapatam Rangoon.	Tuticorin. Vizagapatam	India (French): Chandernagor Karikal	Pondicherry Province India (Portuguese)

2 6 cases of smallpox were reported Apr. 14, 1930, in Costa Rica outside of city of San Jose.

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

SMALLPOX—Continued [C indicates cases; D, deaths; P, present]

			•															١
	Dec.	Jan	Feb.	Mar.							Week	Week ended-	,					
Place	152 E	45°	P. K.	9 Å	4	April, 1930	Q		Ms	May, 1930		·		June, 1930	086		July, 1930	1930
•	1930	1930	1930	1930	12	19	36	8	or	11	72	E S	-	3	z l	88	20	2
Indo-China (see also table below): Pnompenh						-	-				-	-	-					
Saigon and Cholon	40		40	∞ ⊣		• -	40		-				-					
Iraq: BaghdadC					-	8	60	8-	-				-				-	
Basra			1-20			ង្គ«		·	1 22 6		æ-					80	79	
Ivory Coast (see table below). Jamaica			•			<u> </u>		T,				63						
		<u> </u>	63	80	-				1			Ħ	Ħ	Ħ		$\overline{1}$	Ħ	
Mexico (see also table below): Jalisco (State): Guadalajara				ឌ្គ	. •	*	8	7			9	-	*		20-	•	**	
Mexico City and surrounding territory 1 D		181	-85	8	87	200	87	8 =	జ్ఞ	125	17.00	<u> </u>			•			
Morelos State.														i	F			
San Luis Potosi			<u> </u>	\coprod							1			İ				
Morocco (see table below). Netherlands: Rotterdam																ľ		
Nigeria (soe also table below): Lagos D		70	~		_										T	1	$\overline{\parallel}$	
Persia (see table below): Philippine Islands: Sarangani and Balut Islands D		18						14				2						
			·		2 2	60	100		0		67		-		63	7		

_	42	-	2	-	-2	2	7	7	+	+	+	1	-	+	+
a v	C	32-	19	2											
A C		∞	67	9											
Straits Settlements	<u> </u> 	2		9	2		2		60	2	19	-10	-	8	
Α		-	;	_		/			_	_	_		-		
Sudan (Anglo-Egyptian) C 29	290	230	<u>6</u> ه	8 4	~-	933			22	<u>-</u>		œ	-	Z "	
table below).			·	•								-			·
C	22	_	က	8	-	-	60		-	-			67	-	!
Turkey (see table below). Union of South Africa:															
00	A.	PI F	A.F	P. F	<u>H</u>	<u>-</u>		Д	д	Д	Ы	-	-	<u> </u>	-
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S. S. Tairoa, at Liverpool, from London C	-	-	-		-	-			-	-	-			-	
S. S. Karagola, at Zanzibar, from India C	<u> </u>	4	1	+	-	:			i	+	1	-	-	1	!
India	_	_	-	_							_			_	-
Bombay	+	+	+	+	-	1		ŀ		+	+		1	-	-
om Honolulu to San Fran-		_	<u> </u>	_			<u> </u>	-		-	<u> </u>	<u> </u>		-	
		\parallel	$\left. \cdot \right $								-			-	
			Janu-	Febru-		March, 1930	0		April, 1930	08		May, 1930	8	June	June, 1930
Fiate		1929	1930 1930	1830.	1-10	11-20	21-31	1-10	11-20	21-30	1-10	11-20	21-31	1-10	11-20
Belgian Congo	0	47													
	AC	45			-	-			-	-					!
Indo-China (see also table above)	000	142	460	\$			26	261				22	132	8	133
Sudan (French)	: 	17	229	213		- 8	<u>!</u>			150	9	28	_		
Orașio Dolmit	בי בי	ě	38	===	!	æ°	33	85		_	_		81	•	
Taiwan: Taihoku	90	3	?	4 2	33,4	21			***					•	1
	-]	_				-				

* During the month of March, 1930, 100 cases of smallpox were reported in Mexico City, Mexico, and surrounding territory.

* Newspaper reports of Feb. 4 show an epidemic of smallpox in Ionacatepee, Morelos State, Mexico, and vicinity, giving 600 deaths in preceding 2 weeks.

* On Feb. 1, 1930, 317 cases of smallpox with 102 deaths were reported to that date in the Sarangani and Balut Islands.

CHOLERA, PLAGUE, SMALLPOX, TYPHUS FEVER, AND YELLOW FEVER-Continued SMALLPOX—Continued

	(ay,	18 16			July 5,	8	
	March, April, May, 1930 1930 1930	401 6			Ju	-T -83	- (1) - (4)
	Prch, A	100			930	21	
	Feb- ru- ary, 1930	e7 114 142			June, 1930	14	
		822 822			-	1	21
	1- Jan- 1- uary, 19 1930			ı		31	80,60
	De- cem- ber, 1929	4285 485		ended		77	4
				Week ended-	1930		
		Mexico: Durango (see also table above) Morocco		•	May, 1930	0 17	1. 6.4
		Mexico: Durango (see also table abov Morocco. Nigeria. Persia. Turkey.				10	1
	Place	ee also				60	1 1
[C indicates cases; D, deaths; P, present]		s) oầu	sent		1930	8	6169
		Dura	P, pre		April, 1930	22	
		exico: orocco igeria- arsia- urkey-	EVER eaths:			12	614
D, &	, o		TYPHUS FEVER [C indicates cases; D, deaths; P, present	Dec. 16, Jan. 12- Feb. 9- Mar. 9- Jan. 11, Feb. 8, Mar. 8, Apr. 6, 1830 1930			61 11
C8.368;	l, May,		TYPH ss case		65. 9- 1930 8. 8.		410
licates	, Apri 1930	174	dicate	808 N. N.			
[C Ind	March, April, 1930	175	[C tr		, Feb.		6040
	Feb- ru- ary, 1930	2100 2118 201 201 201 201 201 201 201 201 201 201			1929 Jan. 1		14 2
	Jan- uary, 1930	12 184 155 1 1					A0000
	De- cem- ber, 1929	168					
	Place	British East Africa (see also table above): Kenya			Place		Algeria: Algiers Constantine Department Oran. Arabia: Aden

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Chile:
Talcahuano

Valparaiso

C

China:
Manchuria—Harbin.
Shanghai
Tientsin.

zil: Porto Alegre C garla. D D D D

Bolivia: La Paz.¹ Brazil: Porto Alegre Bulgaria.

Chosen (see table below). Czechoslovakia (see table below). Egypt:																_
Assumuting Assumpting Bahaira Province	6-1	7		64			7 6	6	2	0	9	17	9			• • • •
			12					4	4	4	-	-	-	<u> </u>	<u> </u>	111
		67	1		$\frac{11}{11}$		$\frac{\parallel}{\parallel}$			$\dagger \dagger$	$\frac{++}{11}$	#	╫	#	#	!!
Great Britain: Scotland—Glasgow		- 				$\frac{\square}{\square}$	$\frac{\parallel}{\parallel}$	<u> </u>		$\dagger \dagger$	H		$\frac{ \cdot }{ \cdot }$	-		111
Greece (see table below). Iraq: Baghdad Liwa				61												11
Ireland: Irish Free State	5.555										63		-			
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cipalities in Foder-	000	548	9	38 48	888		60		8 8	-	40-			90	90	111
			183	23.5.7	8 8	20	53 67	110	64 PG 6	4	88-	6		11	25.	" ;;;
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Value of South Africa: Cape Province Cape Province Cape Province	444 000	₽ -₽		ድድ	ር ር	다다	Д,		ር ር	P4 P	בי בי					!!!
				'	А	Ъ	ь						+		\dashv	: 1

1 12 deaths from typhus fever were reported in La Paz, Bolivis, from Jan. 1 to May 31, 1930.

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

TYPHUS FEVER—Continued

May, 1930	27 16 16	Cases
April, 1930	£4€2 4	
De- Janu- Feb- March, April, May, ber, 1929 1930 1930 1930	24142	
Feb- ru- ary, 1930	52283	
Janu- ary, 1930	64 CA SE	
De- cem- ber, 1929	≈-4-6-1	
Place	Lithuania C Turkey C Yugoslavia C D Turkey C D D D D D D D D D D D D D D D D D D D	YELLOW FEVER Case Gold Coast: Ost Dec. 21, 1929 Dec. 21, 1920 Dec. 21, 1930 De
May, 1930	3	Cases Oy, 2 2
April, 1930	20 3	YE
Janu- Feb- ary, ary, 1930 1930 1930 1930 1930	42	and Nic
Feb- ru- ary, 1930	17 8	aneiro
Janu- ary, 1930	10 12 18	de J
96. 1929	1 108	en Ri
Place	Chosen: Seoul	Brazil: Mage, on the Leopoldina Rallway, between Rio de Janeiro and Nictheroy, Apr. 22, 1830. Campos, Rio de Janeiro Province, May 23, 1930. X