THE CAUSATION AND PREVENTION OF ENTERIC FEVER IN MILITARY SERVICE.
WITH SPECIAL REFERENCE TO THE IMPORTANCE OF CARRIERS.

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PART I. CAUSATION OF ENTERIC FEVER BY CARRIERS.

In his preface to Dr. J. C. G. Ledingham's "Report to the Local Government Board on the Enteric Fever 'Carrier,'" Dr. Theodore Thompson uses the following definition:—

"A 'carrier' of enteric fever is a person who, although he may be in good health, carries the infectious material of the fever in his body, from which it may be given off in the stools and urine."

This very complete definition makes two important reservations. The "carrier" may be in good health, but the fact that he may not be so is kept in mind. Thus the definition includes not only the clinically healthy "carrier," whether acute or chronic, but also that highly dangerous person, the atypical and undiagnosed "case." Again "the infectious material . . . may be given off in the stools and in the urine," but the definition does not exclude those persons who, though still harbouring the germ within their bodies, yet cease, during long periods, to excrete it. The latter reservation is one that is often forgotten. In much of the literature on the subject, and worse, in many of the regulations framed to prevent or mitigate the danger to the community from this source, the question of excretion of germs receives more attention than the fundamental fact of carrying.

In fact the question of intermission in excretion, and the causes underlying it, requires thorough elucidation and constitutes one of the most important aspects of the "enteric carrier" question. To the latter point we propose to return later, but for the present will confine ourselves to the proposition that any estimate of the number of typhoid patients who become carriers, based on the positive findings of bacteriological examination of the excreta alone, is likely to be an under-estimate.

As is so often the case in the evolution of scientific opinion, the

1 Parkes Memorial Prize Essay, 1912.
main facts underlying the "typhoid carrier" conception had already been recognized for some time before that conception took definite shape. From the year 1880, when Eberth first discovered the Bacillus typhosus and affirmed that it was the cause of enteric fever, many individual workers had added their quota to the epidemiology of the disease, and the growth of knowledge upon the subject had been uninterrupted. But the knowledge was scattered, not co-ordinated. A salient fact here and there arrested attention, and, examined perhaps without due attention to less conspicuous observations, often served to mislead instead of to guide professional opinion. Thus the correct observation that many epidemics were due to infected water supplies, led to the incorrect inference that enteric fever was almost entirely a water-borne disease. This idea in its turn served to explain the frequent association of outbreaks with infected milk. It was assumed that the milk became infected mainly through dilution with water, and thus the fact of milk-contamination, which might otherwise have led to the search for an infective human being associated with the collection or distribution of the milk, was often considered only as additional proof that water was the main enemy. Up to the time of the South African war it may be truly said that the water-borne theory of enteric fever held the field, although it was not denied that occasional direct infection took place amongst nurses and others intimately associated with cases. The following quotations from a standard textbook on medicine, published in 1901, will serve to give an idea of the average professional opinion in that year:

"Vehicles by which the bacillus is transmitted: Water.—The fact that the poison of typhoid fever is in most cases carried by water has long been recognized. . . . Recently the detection of the specific bacillus in suspected water has completed the chain of evidence. . . ."

Further on it is stated that "typhoid fever is not directly contagious. Cases occurring amongst nurses and attendants can be attributed to want of care in cleansing the hands, or possibly to allowing stools passed in bed to dry on the sheets, and to be inhaled as dust."

Yet side by side with this average professional opinion there was a growing tendency of expert opinion to lay stress on the infective person rather than on the infected thing, to investigate the contagion at its source rather than in its distribution.

In 1900, Horton Smith (Goulstonian Lectures, British Medical Journal, vol i, 1900, p. 827) had cited the presence of the Bacillus
typhosus in the stools early in the disease and in the urine during the later stages, and had expressed the view that infected urine was the chief means by which typhoid fever is spread. He had also actually given expression to the conception of the "chronic faecal carrier" as the term is now understood, for, writing on the subject of typhoid bacilli in the stools, he had said, "but we must be prepared for the possibility of their remaining in some cases a source of danger for long periods after the patient is apparently well—as long, that is to say, as the bile continues to harbour the parasite." Gwyn, too (Johns Hopkins Hospital Bulletin, June, 1899, p. 109) had already found and described a true urinary carrier, while Shakespeare and Reed were engaged in their great investigation of the "Origin and Spread of Typhoid Fever in the United States Military Camps, during the Spanish-American War of 1898" (published in 1904), which demonstrated beyond dispute the enormous importance of contact in the epidemiology of this disease. It remained for Robert Koch to piece together the isolated observations of many workers and to enunciate his belief that "the chief source of typhoid infection is to be found in man himself." As the result of his advice, the campaign against enteric fever in South-West Germany was initiated through the agency of a series of bacteriological stations organized in the infected districts, and it was through the investigations carried out at these stations by Frosch, Klinger, Lentz and others that the conception of the typhoid carrier—both acute and chronic—came to be clearly defined and universally accepted.

To attempt a historical survey of the subject or to gather together a large number of recorded instances where typhoid carriers have been responsible for outbreaks of the disease would be merely to overburden this essay with unnecessary repetition. A very complete precis on these lines already exists in Dr. J. C. G. Ledingham's "Report to the Local Government Board on the Enteric Fever Carrier" (October 5, 1910).

It will be our aim, therefore, to keep, as far as possible, within the limits imposed by our title and to bring forward what experimental work we have at our disposal bearing upon the "causation and prevention of enteric fever in military service," with special reference to the role of "carriers."

Recalling Thompson's definition of a typhoid carrier as "a person who, although he may be in good health, carries the infective material in his body from which it may be given off in the stools and urine," it will be conceded that this definition covers not only the chronic typhoid carrier, but even the acute case, the only
essential being the carrying of infectious material in the body. This is as it should be, and we fully endorse the view that the acute and recognized "case" is a typhoid carrier. From the epidemiological point of view, however, the recognized case should not be very important, as the medical man in charge ought to take steps to safeguard the public from a known focus of infection. In dealing with the causation of enteric fever in military service, especially, the acute recognized case should but seldom be a source of danger to others, as the patient is almost certain to be in hospital, where the risks of transference of infection are reduced to a minimum. Still occasions may arise, especially on active service, where the acute and correctly diagnosed military case may be a serious menace to other soldiers, and we feel obliged to include such cases in the classification of "Typhoid Carriers" given below:

Acute carriers

1. Precocious carriers. Persons who harbour the germs during the period of incubation.
2. Unrecognized cases, atypical, abortive, or ambulatory.
3. Acute diagnosed cases.
4. Persons infected with the typhoid bacillus, but never showing symptoms of illness. "Paradoxical Carriers."
5. Temporary. Persons who, for longer or shorter periods up to three months after an attack, continue to harbour typhoid bacilli.
6. Chronic typhoid carriers. Persons who continue for long periods to carry in their bodies the typhoid bacillus.

We would again insist that the essential point is the fact of carrying or harbouring in the body the infective material. Every "carrier," in this sense of the word, is a potential "excreter," though not necessarily excreting at any given moment.

The endemicity of typhoid fever in certain localities or institutions is usually to be explained by the presence in the vicinity of a chronic carrier. It is to chronic carriers too that we must look for the explanation of the carrying over of typhoid infection from one period of prevalence to another. The actual prevalence of the disease on a large scale, where not due to contamination of water-supplies, is commonly to be explained by the presence of a number of acute and temporary "carriers," persons who have been in contact with the earliest cases, or who are now themselves convalescent from acute attacks.
Military life differs from life under civilian conditions chiefly in the fact that the latter is individualistic while the former is communal. In the Army it is necessary, for purposes of administration and discipline, to aggregate men together in groups, these groups having in common many conditions that are not shared to the same extent in civil life. Thus food is obtained, stored, issued, and prepared on a communal basis, latrines and urinals are arranged, not for individuals, but for groups, and the same applies to living and sleeping rooms. In times of peace and under conditions of service at home, these communal conditions of life can be so carefully supervised that the dangers incident to such close association are to a great extent nullified. Still where some flaw in the arrangements exists, instances occur from time to time to demonstrate that aggregation of men together exposes them to risks which would be much smaller in life under individual conditions. Thus some of the most striking instances of outbreaks of typhoid fever due to the presence of carriers have been recorded from armies under peace conditions and from certain civilian communities where conditions of life similar to those incident to military service exist, such as asylums, schools, reformatories and institutions. We quote, without attempting to describe, the following military instances from the British and other armies:—

(1) An outbreak at Aldershot, described by Major Cochrane, R.A.M.C. (Journal of the Royal Army Medical Corps, February, 1909), where a series of cases was definitely traced to a urinary "carrier."

(2) An epidemic at Kilworth Camp in Ireland, described by Captain J. Dorgan, R.A.M.C. (Journal of the Royal Army Medical Corps, April, 1910), in which the origin of twenty-four cases, occurring from May to August, 1909, was traced to a female urinary carrier employed as dairymaid at the farm whence milk for the troops was obtained. There had been, in addition, fourteen cases amongst civilians associated with this carrier.

(3) Local endemicity of typhoid fever in the Artillery Barracks at Wesel, reported by Niepratschek (Zeitschr. für Hyg., Bd. 64, p. 454), found to be due to the presence of a urinary carrier, Sergeant B.

Instances could be multiplied to the same effect, but the foregoing suffice to demonstrate that the typhoid carrier is a cause of enteric fever in military life, and to illustrate our contention...
that aggregation of men together in barracks exposes them to increased danger. It will be noted also that in all these instances, a chronic carrier was responsible for the outbreak.

In all three instances, therefore, the circumstances were very exceptional, in that some flaw in the sanitary organization co-existed with the presence in the community of that comparatively rare phenomenon, the chronic carrier. In an editorial on the subject of carriers in the Journal of the Royal Army Medical Corps for April, 1910, the opinion is expressed "that if care be taken to exclude them from employment in the preparation of food and drink, infection is not likely to be caused by the presence of a few carriers amongst the community, especially if a water carriage system of sewage disposal be in vogue." With this opinion we are in complete accord, where the implied conditions exist. In peace the danger of carriers is small and would be practically nil if, as the Editorial quoted assumes, their presence were known, and they could be excluded from the preparation of food and drink. Unfortunately their presence is, as a rule, only revealed during the investigation of a series of cases resulting from them, and the same investigation usually calls attention also to some defect in the sanitary routine, without which the carrier would have been powerless to infect others. Even in peace time then, the soldier is exposed by the conditions of his service to some danger from carriers, a danger which can be reduced almost to vanishing point by perfection in sanitary organization. But the Army only exists in peace for its true function, war. In peace time the aggregation of troops into large communities is as far as possible avoided, as a wide distribution of units facilitates maintenance, and places comparatively small bodies of troops at the disposal of the civil power in a large number of localities. In war, where the success of the operations often depends upon numerical superiority at some definite point, aggregation of troops on a large scale is a necessary condition. Further, the fixed sanitary routine, by which excreta are got rid of in peace, gives place in war to a series of improvisations, trench latrines taking the place of "water-carryage" or other systems of a permanent kind. At the same time the troops are exposed to severe exertions, and are liable to be placed on a less satisfactory scale of rations, while the possibility of detecting and isolating carriers, where the presence of these persons is suspected, becomes remote.

Theoretically, then, it is to be expected that the typhoid carrier will be a factor of great importance in the causation of enteric fever.
in armies in the field. Putting aside theory, and turning to the
history of war, we find that enteric fever has, in the past, arisen
amongst armies operating in localities where this disease was not
known to exist before the troops arrived.

The Principal Medical Officer (Surgeon-General W. Taylor,
C.B., A.M.S.) writes as follows concerning enteric fever amongst
troops in India (Army Medical Department Report, 1898):

"The liability of troops on service to enteric fever, even when
encamped on virgin soil, is a fact in regard to which all observers
are unanimous." This officer is driven to the conclusion that
"it may be that the enteric bacillus is more widely distributed in
nature than our present knowledge leads us to suppose." Later,
as Principal Medical Officer (British troops) of the Nile Expedition,
1898, Surgeon-General Taylor is again face to face with the same
problem. He reports of enteric fever that, "This disease was, as
usual, the scourge of the Army in the Sudan . . . It is
responsible for nearly half the mortality of the campaign, and
caused more than double the deaths of both battles put together.
The specific germs are no doubt swarming in both earth and
water . . ." (Army Medical Department Report, 1898). In
both cases we find that recourse is had to "a wide distribution of
the specific organism in nature" to explain the occurrence of this
disease in bodies of troops operating in sparsely populated countries,
where the possibility of infection from outside is otherwise difficult
to account for. In the same year (1898) occurred the Spanish-
American War, with epidemic prevalence of enteric fever amongst
the American troops. In this instance the outbreak assumed such
terrible proportions that three specially qualified officers (W. Reed,
Victor C. Vaughan, and E. O. Shakespeare) were detailed to
investigate it. Their conclusions, published in 1904, mark an
epoch in the history of military hygiene. They abandon the
attempt to explain the outbreak by "infection from without,"
and boldly assert the view that the troops had brought the specific
organism with them in the bodies of certain of the soldiers.
"Infected water," they say, "was not an important factor in the
spread of typhoid fever in the National encampments in 1898.
With typhoid fever as widely disseminated as it is in this country,
the chances are that if a regiment of 1,300 men should be
assembled in any section and kept in a camp, the sanitary con-
dition of which was perfect, one or more cases of typhoid fever
would develop." . . . "When a Command, badly infected with
typhoid fever, changes its location it carries the specific agent of
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the disease in the bodies of the men, in their clothing, bedding, and tentage."

Here are ideas that, applied to the campaigns on the Indian frontier and in the Sudan, would serve to explain the occurrence of enteric fever without the uncomfortable and improbable theory that the germ of the disease is "widely distributed in nature." The importance of "contact" with infected persons, the possibility of carriage of the specific agent in the human body, and the role of flies as transmitters of infection are all clearly brought out in the invaluable report of Reed, Vaughan, and Shakespeare.

"The Medical History of the War in South Africa: An Epidemiological Essay," by Lieutenant-Colonel R. J. S. Simpson, C.M.G., R.A.M.C., is perhaps the most weighty contribution yet made to this subject. This officer, whose valuable paper on the prevalence of enteric fever in Pietermaritzburg (Army Medical Department Report, 1898) shows such a thorough comprehension of the conditions bearing on typhoid fever in South Africa, forms the opinion that the mode of development of the epidemics in the different military groups during the war suggests an auto-infection, and appears not to be explained by any theory of external infection of the mass. Having discussed the possibility of the epidemics having been caused by infected soil or infected water, he concludes that, "None of these external causes suffices to explain the mode of development of these outbreaks. There remains the theory of personal contact, using this term to include direct personal infection from close association, the infection of a commensal, and indirect infection through excreta." It is to this "contact" that the author attributes the prevalence of enteric fever during the South African War, and his careful and unprejudiced analysis of the statistics on which his paper is founded is very convincing.

The deductions of Shakespeare, Vaughan, and Reed, and the close statistical study of enteric fever on active service by Simpson, demonstrate the importance of "contact" in the spread of the disease in military aggregations, but the origin of the early cases from which "contact" was operative might still have been sought in some external location of the specific agent. It remained for Lieutenant-Colonel Sir D. Semple, Captain E. D. W. Greig and their co-workers in India to apply the discoveries of Frosch and Klinger in South-West Germany to a military population and to place beyond doubt the important role of "carriers" in initiating outbreaks of enteric fever. They showed "that the conditions met with outside the human host are not favourable to the prolonged
existence of the \textit{B. typhosus}, and therefore, the persistence of the disease cannot be explained by a hypothesis that postulates long extra-corporeal existence of the bacilli. . . . It is brought home to us more clearly that conditions outside the human body cannot favour the growth of the parasite; at the best they can act only as channels of communication, and these channels soon dry up unless fed from the reservoir, the human host."

The human host then, or in other words, the "carrier"—whether acute or chronic—is the essential factor in the origin and maintenance of enteric fever in armies, whether in peace or in war.

\textbf{Factors in the Causation of Enteric Fever by "Typhoid Carriers."}

In the foregoing paragraphs we have summarized the evidence upon which we base our opinion that carriers are the most important factor in the causation of enteric fever. We shall now attempt to deal with the mechanisms by which these persons infect others, as far as possible systematizing our argument under the four following headings:

(1) Conditions associated with the carrier.
(2) Survival of the \textit{B. typhosus} outside the human body.
(3) Means of transmission from the carrier to the recipient.
(4) Conditions associated with the recipient himself.

\textbf{(1) The Carrier.}

\textit{(a) The Relative Danger of One Variety of Carrier as compared with Another.}—This has been investigated by Klinger ("Epidemiological Observations on the Anti-typhoid Campaign of the German Empire," Dr. Klinger, vide pr\'ecis in \textit{Journal of the Royal Army Medical Corps}, vol. xiv, p. 90), who found that, of 1,397 cases traced to this source, 1,272 were caused by acute carriers—that is to say actual cases—and 125 by chronic carriers. Of 812 cases traced to acute carriers, 183 were infected from persons in the incubation stage, precocious carriers, a further 554 by acute cases in the first five weeks of the attack, and 75 by convalescents up to the end of the tenth week. It may be presumed that the majority of the acute cases would have been sooner or later diagnosed and isolated under military conditions in peace, but it is probable that in war, many of the men would have attempted to "stick it out," and would thus have escaped diagnosis until they reported sick in a condition that precluded further work, probably
early in the second week of illness. It is fair to assume that from 250 to 300 of the above cases were caused by persons who would have been at large and infective under military conditions in peace, equally as in civilian life. But the opportunities for spreading the disease must have been much more restricted in the case of these civilian "acute carriers" than would have been the case on active service, so that the figures certainly under-estimate the "typhoid productivity" of acute carriers in war. All that we can infer from them is that once enteric fever has actually broken out amongst troops in the field, the presence of precocious carriers, atypical and abortive "cases" and infective contacts will serve to maintain, and to increase in geometrical ratio, the amount of infective material menacing the healthy. The extent to which atypical and mild cases may be present during an outbreak of the disease is well shown by an analysis of an epidemic occurring in the French Army in 1909 (Archives de Méd. et de Pharm. militaires, April, 1909), where out of a total of 142 cases, 57 were only discovered by persistent medical inspection, these men not having reported sick. Of the cases so discovered, 39 were so little affected by their illness that it was enough merely to isolate them in barracks, treatment in hospital being unnecessary. The chronic carriers that take the field with every army may be expected to manifest their presence by giving rise to an appreciable number of cases in from forty to sixty days after operations commence. Vaughan, Reed, and Shakespeare found that every unit mobilized during the Spanish-American War had developed cases of typhoid fever by the eighth week. Simpson, in his "Medical History of the South African War" says, "We have seen that in four large bodies of men, an incubation period of the epidemic of enteric fever occurred of about eight weeks' duration, i.e., more than twice the accepted maximum incubation period of the individual, and this was independent of the environment of the troops concerned."

We shall discuss this question—the "eight weeks phenomenon"—later, but would here point out that on active service, we must anticipate that the problem of "acute carriers" will require attention from about the sixth week and onwards. The magnitude of the problem will depend upon how thoroughly we have sought for and dealt with the chronic carriers before taking the field, and upon how completely our field sanitary measures have been carried out during the early weeks of the campaign.

(b) The Number of Germs Excreted by Chronic Carriers.—Intimately associated with the last point dealt with is the number
The charts show a tendency to an inverse relation between *B. coli* and *B. Typhosus.*
of typhoid bacilli excreted by carriers. The average number for a series of observations on two chronic "faecal" carriers, for a period of three months, is given in Chart I, which shows graphically the fluctuations in the excretion of *B. typhosus* and *B. coli* in the two faecal carriers during twenty successive "counts" of the bacterial contents per 1 grm. of faeces. It will be seen at once that individuals differ widely in the numbers excreted, the average for Carrier W. S. being about seven times as great as that for Carrier F. C.

Further, the individual variations for each carrier are very great. The highest count that we have ourselves recorded amounted to over six thousand millions of typhoid bacilli in 1 c.c. of urine in Carrier F. I., and from this enormous figure to one thousand two hundred bacilli per 1 c.c. represents the variation for this urinary carrier during a period of observation covering just over four years. During a series of constant observations for three months, the man being under hospital conditions and not subject to fatigue or exertion, the average number per 1 c.c. was slightly above two and a half millions. While in another urinary carrier observed at the same time the average excretion was under seven thousand bacilli per 1 c.c. Taking the two faecal and two urinary carriers together for three months of consecutive observation, the average excretion per man amounted to nearly eighty million bacilli per gramme or cubic centimetre. Let us try to imagine in terms of contamination power what this means. Suppose that 100 c.c. of infected urine gains access to a collection of water amounting to one million gallons, the water-supply of an army of 500,000 men for one day, and becomes evenly distributed through it, there will be one thousand typhoid bacilli in every pint of the contaminated water. It is not suggested that such a vast body of water could possibly be evenly contaminated by any small addition of fluid, nor that the germs added with this urine would survive for long, but the example may serve to emphasize the potentiality of a single carrier. The great variations in the numbers passed by any one individual at different times give grounds for interesting speculations as to the carrier state. On what do these variations depend? Variations can be brought about under known conditions. It is beyond question that the number of germs excreted is greater after exertion or anything causing fatigue. Carrier F. I., when in hospital, excreted on a much lower level than when out of hospital and at work. Again, the injection of a moderately large dose of antityphoid vaccine will constantly increase the number of bacilli in the urine, a fact first
noted by Irwin and Houston (Lancet, January 30, 1909) and which we observed after every large dose in the case of Carrier F. I. (Table I). Again there appears to be a tendency to an inverse ratio between the excretion of *B. typhosus* and *B. coli* in the case of fecal carriers (vide Chart I), and as will be shown later (page 654) the latter organism is able, within a comparatively short space of time, to completely outgrow and eliminate the former. The more rapid the transit of feces through the bowel, the shorter will be the period of competition with *B. coli* and other organisms, and the more numerous the surviving *B. typhosus*. Where diarrhoea is a prevalent disease, therefore, the excretion of *B. typhosus* by carriers will be, as a rule, increased. It may be expected then that on active service, where over-exertion is the rule and where exposure to chill, bad cooking, and other causes almost invariably leads to diarrhoea,
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the output of infective material by carriers will invariably be much greater than the figures quoted as observed under conditions of rest in hospital.

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<tr>
<th>TABLE II.—COMPARISON OF A &quot;CARRIER&quot; STRAIN (F.C.) WITH (1) A LABORATORY AND (2) A VIRULENT TYPHOID CULTURE, AS TO SERUM-SOLUBILITY.</th>
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<tbody>
<tr>
<td><strong>Experiment 1.</strong></td>
</tr>
<tr>
<td>Emulsion dilutions</td>
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<tr>
<td>Numbers of colonies surviving in each dilution.</td>
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<tr>
<td>Strain &quot;F.C.&quot; + Immune serum</td>
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<td>Laboratory strain + Immune serum</td>
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<tr>
<th>Experiment 2.</th>
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<tbody>
<tr>
<td>Emulsion dilutions</td>
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<tr>
<td>Virulent strain from acute case + Immune serum</td>
</tr>
<tr>
<td>Strain &quot;F.C.&quot; + Immune serum</td>
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<tr>
<td>Laboratory strain + Immune serum</td>
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Equal volumes of immune rabbit serum and each of a series of dilutions of the bacterial emulsions were mixed together and kept at 37° C. for two hours. The mixtures were then blown on to agar slopes, and the latter incubated, the colonies being counted next day.

It will be seen that the "Carrier" strain was much less soluble than the old Laboratory strain, but more so than the fresh virulent strain from an acute case.

(4) The Virulence of the Germs excreted by Carriers.—This question has received considerable attention and the evidence to hand justifies no definite conclusion as to whether carrier strains are more or less virulent than strains isolated from acute cases of enteric fever. Lentz ("Ueber Chronische Typhusbazillenträger," *Klin. Jahrbuch*, Bd. xiv, p. 475) was unable to show any constant difference in virulence between twenty carrier strains and various
other strains of *B. typhosus* at his disposal. Ledingham (Report to the Local Government Board, 1910) failed to find conclusive evidence on this point. We have found that a freshly isolated strain from Carrier F.C. was much less easily killed by normal serum than was an old laboratory culture, but, on the other hand, a strain freshly isolated from the blood of an acute case was more resistant than the carrier strain (Table II).

We regard the question of virulence as more or less academical, since there is ample evidence to prove that carrier strains are able to bring about acute and often fatal infection of healthy persons—a point of much greater significance than any record as to the number of germs necessary to kill a 250-grm. guinea pig on intraperitoneal inoculation.

(2) Survival of the *B. typhosus* Outside the Human Body.

Much work has been done on this subject, and much remains to be done. When an external origin was invoked for every epidemic, it was natural that soil, water and other external objects should be thoroughly examined as to their powers of supporting the life of *B. typhosus*. A great deal of earlier work must be regarded with grave suspicion as the various organisms constituting the typhoid-colon group have only recently been satisfactorily separated, and there seems no doubt that many strains recovered from soil, water and sewage, and confidently regarded as *B. typhosus* by the observers concerned, were in reality other organisms.

As the means of recognition of *B. typhosus* have been perfected, so the period of survival outside the body attributed to it has drawn in and diminished. There is still, however, a decided discrepancy between the work of thoroughly reliable observers. Horrocks and Firth, for instance ("An Inquiry into the Influence of Soil, Fabrics and Flies in the Dissemination of Enteric Infection," *British Medical Journal*, September, 1902), found that soil contaminated with excrement from enteric cases remained infective for a long time, the specific germ being recognized for periods varying from 45 to 74 days, while fabrics such as khaki drill, serge, &c., soiled with typhoid cultures permitted the recovery of the *B. typhosus* for many weeks, and with typhoid feces, to seventeen days. On the other hand, Semple, Greig and their co-workers found that this organism rapidly disappeared from urine and feces outside the body, and that fabrics contaminated with infective stools and exposed to the sun rapidly became sterile. The explanation may be sought in the fact that one series of experiments was
carried out in the South of England, the other in India, where the sun is much more powerful. It may be noted that the latter workers found that "control" fabrics kept in a cupboard where the sun was unable to act on them remained infective up to six days, though not to seventeen.

We may summarize our experiments under the following headings:—

A. Survival of the \textit{B. typhosus} in excreta.
B. " " " on clothes of carriers.
C. " " " on fingers of carriers.
D. " " " in food.

\textbf{A. IN EXCRETA.}

(1) \textit{Survival of the B. typhosus in Urine.}

On several occasions we have recovered \textit{B. typhosus} from the urine of Carrier F. I. up to six and seven weeks after passing. We have made only two attempts to keep it for longer periods. In one the organism was still present in large numbers, and apparently in pure cultures, up to three months. The flask was then emptied by an attendant in error, and further examination was thus stopped; but there seems no reason why this specimen should not have continued to retain the organism for very long periods. On a second occasion a specimen containing enormous numbers of \textit{B. typhosus} was put aside. When examined after two months, no \textit{B. typhosus} was isolated, but a small number of \textit{B. fecalis alkaligenes} had taken its place. These experiments had only an academical value, as urine is unlikely to remain under sterile conditions in nature. It is to be observed that where the \textit{B. typhosus} disappeared, its place was taken by another organism. In nature competition with other organisms is likely to be severe, especially as urine is commonly voided so that it ultimately mixes with fecal sewage. For this reason it appeared important to ascertain the effect of the addition of feces to a sample of urine from a typhoid carrier. The results are shown below:—

(2) \textit{Effect of Fecal Contamination on the Survival of B. typhosus in Urine.}

\textit{Experiment I.}—A sample of urine from Carrier F. I. was divided into two equal parts. To one part was added an emulsion

\footnote{Survival up to one year of \textit{B. typhosus} in urine kept under laboratory conditions has been recorded by Horrocks (\textit{Journal of the Royal Army Medical Corps}, vol. xvi, p. 235).}
of normal faces. The other part was kept unaltered. Both flasks were allowed to remain at room-temperature. Plates were made from each flask daily from October 23 until November 1, 1909.

Result.—(1) B. typhosus was isolated on every day of observation from the uncontaminated urine.

(2) B. typhosus mixed with B. coli was isolated in diminishing numbers from the contaminated urine sample up to October 25. No B. typhosus was found on the plate on October 26. Four colonies of B. typhosus were isolated, amongst many B. coli colonies, on October 27. From that date onwards no B. typhosus could be isolated from the contaminated urine.

Experiment II.—A specimen of urine from Carrier A. was divided into two equal portions. One portion was contaminated by the addition of emulsion of normal faces. The other part was kept unaltered. Both samples were kept at room-temperature from November 3 to November 16, 1909, plates being prepared from each on every day of the experiment except November 8 and November 15.

Result.—B. typhosus was recovered on every day from the uncontaminated sample, the “count” amounting to 91,500 bacilli per cubic centimetre on November 16.

B. typhosus was recovered in decreasing number, mixed with B. coli, from the contaminated sample up to November 14. No B. typhosus could be recovered from the contaminated flask on November 16, by which date B. coli and some streptococci were the only organisms demonstrable in the sample.

In the first experiment B. typhosus disappeared after the fifth day. In the second it was still recovered in small numbers up to the twelfth day, after which it disappeared. The difference is to be explained by the initial number of B. typhosus in the samples used. The “count” of the urine on passing in Experiment I was only 12,600 bacilli per 1 c.c.; while in the second experiment it was 2,691,000 per 1 c.c. In both cases the urine that was left uncontaminated continued to contain the B. typhosus up to the end of the experiment.

(3) Survival of B. typhosus in the faces of a Carrier at Room-Temperature.

Experiment I (Faces).—One sterile watch glass of faces freshly passed by Carrier C. was taken and emulsified in 100 c.c. of sterile water. The mixture was placed in a sterile flask. The presence
The Causation and Prevention of Enteric Fever

of B. typhosus in the emulsion was verified by plating on the first day of the experiment, March 5, 1912.

March 7, 1912: "Plated" and recovered B. typhosus in large numbers, mixed with a few B. coli.

March 8, 1912: Recognized numerous B. typhosus on plates. Few B. coli.

March 15, 1912: Recovered numerous B. typhosus on plates. Few B. coli.

March 26, 1912: Recovered thirty-three colonies of B. typhosus with twelve colonies of B. coli mixed with innumerable small colonies of a streptococcus. The B. typhosus has greatly diminished in number. After this date B. typhosus was not recovered.

(4) Effect of other Organisms on the Survival of B. typhosus in Feces.

Experiment I (Feces).—An emulsion of normal feces was divided into two portions of 50 c.c. each. One portion was "autoclaved" at 115° C. for twenty minutes to render it sterile. The other portion was not sterilized. To each flask was then added an emulsion of one agar slope of B. typhosus in sterile saline. The flasks were kept at 37° C. from September 29, 1910, until completion of the experiment.

On October 1.—Plates prepared from the "sterilized" feces showed enormous numbers of B. typhosus. Plates from the unsterilized feces showed about equal numbers of B. typhosus and B. coli.

By October 3 only B. coli could be isolated from the unsterilized feces, B. typhosus having disappeared.

B. typhosus was isolated in pure culture from the sterile feces on February 15, 1911, after which the experiment was discontinued.

In Experiment I (feces) the B. typhosus started in large excess of B. coli. Perhaps on this account the B. typhosus was able to survive, though in steadily diminishing numbers, for twenty-two days. Here both B. typhosus and B. coli were subjected to destructive competition with organisms not belonging to the typhoid-colon group, such as streptococci and others. Probably the latter grew better at room-temperature than did the "human" parasites, B. typhosus and B. coli. In the second experiment, an attempt was made to put the influence of competitive existence with other organisms on a more certain basis. The influence of the feces itself, as opposed to its living content, was assumed to be operative.
in the portion of the faeces which had been sterilized. In this the
B. typhosus was still living, and abundant four and a half months
after the inception of the experiment. In the flask where the “live”
faeces was used, it was not possible to isolate B. typhosus on the
third day, this organism being completely outgrown by B. coli at
body temperature.

This action of B. coli was regarded as so important that it was
thought worth further investigation, on a quantitative basis, the
results of the foregoing experiments having decidedly pointed to
the significance of the relative numbers of B. typhosus and B. coli
respectively in the original mixtures. The following experiment
was therefore carried out:—

Experiment II.—Emulsions of B. typhosus (a strain from the
blood of an acute case, eighteen months under laboratory conditions
at room-temperature) and of B. coli (isolated a month before from
the urine of a case of chronic pyelitis) were prepared in normal
saline solution, and standardized so that each contained 1,000
million organisms per 1 c.c.

A thick emulsion of normal faeces was prepared, strained to get
rid of lumps, and divided into quantities of 25 c.c. in each of five
flasks. The flasks were then placed in the autoclave for twenty
minutes at 115° C., removed, and allowed to cool. To each flask
was then added a mixture of the two bacterial emulsions as
follows:—

<table>
<thead>
<tr>
<th>Flask</th>
<th>0.9 c.c. typhoid emulsion</th>
<th>0.1 c.c. coli</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0.7 c.c. typhoid</td>
<td>0.3 c.c. coli</td>
</tr>
<tr>
<td>2</td>
<td>0.5 c.c. typhoid</td>
<td>0.5 c.c. coli</td>
</tr>
<tr>
<td>3</td>
<td>0.3 c.c. typhoid</td>
<td>0.7 c.c. coli</td>
</tr>
<tr>
<td>4</td>
<td>0.1 c.c. typhoid</td>
<td>0.9 c.c. coli</td>
</tr>
<tr>
<td>5</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

It will be noted that B. typhosus was in excess in flasks 1 and 2,
B. coli in excess in flasks 4 and 5, while in flask 3 the organisms
were in equal numbers, the whole being placed in a sterile emulsion
of human faeces so that the mise en scène was correct. The flasks
were placed (November 12, 1911) in the incubator at 37° C.

Next day (twenty hours) plates were prepared.

1 For a series of experiments on this point, see Horrocks, Journal of the Royal
Army Medical Corps, vol. xvi, p. 225.
Result:—

From Flask 1

<table>
<thead>
<tr>
<th>Organism</th>
<th>Number of Colonies</th>
</tr>
</thead>
<tbody>
<tr>
<td>B. typhosus</td>
<td>20 colonies</td>
</tr>
<tr>
<td>B. coli</td>
<td>350</td>
</tr>
</tbody>
</table>

From Flask 2

<table>
<thead>
<tr>
<th>Organism</th>
<th>Number of Colonies</th>
</tr>
</thead>
<tbody>
<tr>
<td>B. typhosus</td>
<td>23</td>
</tr>
<tr>
<td>B. coli</td>
<td>154</td>
</tr>
</tbody>
</table>

From Flask 3

<table>
<thead>
<tr>
<th>Organism</th>
<th>Number of Colonies</th>
</tr>
</thead>
<tbody>
<tr>
<td>B. typhosus</td>
<td>8</td>
</tr>
<tr>
<td>B. coli</td>
<td>156</td>
</tr>
</tbody>
</table>

From Flask 4

<table>
<thead>
<tr>
<th>Organism</th>
<th>Number of Colonies</th>
</tr>
</thead>
<tbody>
<tr>
<td>B. typhosus</td>
<td>15</td>
</tr>
<tr>
<td>B. coli</td>
<td>233</td>
</tr>
</tbody>
</table>

From Flask 5

<table>
<thead>
<tr>
<th>Organism</th>
<th>Number of Colonies</th>
</tr>
</thead>
<tbody>
<tr>
<td>B. typhosus</td>
<td>nil</td>
</tr>
<tr>
<td>B. coli</td>
<td>140 colonies</td>
</tr>
</tbody>
</table>

It will be seen that B. coli had already established an excess in all the flasks, and that in flask 5 B. typhosus had disappeared. The excess of B. coli was not, however, in proportion to the relative numbers of each organism in the original preparations, a point which we cannot at present explain. On the fifth day of the experiment (November 17, 1912) plates were again made with the following results:—

B. typhosus was recovered in very small quantities from flasks 1, 2, 3, and 4 only. B. coli was in enormous numbers on all the plates.

On the eighth day (November 20, 1912) the flasks were again examined by plating, two colonies of B. typhosus being isolated from flask 2, and one colony from flask 3, while B. coli was in uncountable numbers on all the plates.

The flasks were then left at room temperature for a week, and again examined. On this final examination, no B. typhosus was isolated from any of the flasks, the B. coli having completely established its ascendancy.

We consider that these experiments, while undoubtedly throwing some light on the fate of B. typhosus when in competition with other organisms, must not be taken to prove that this organism is unable to survive in sewage. In all attempts to recover B. typhosus on plates, the relative numbers of other organisms present must be taken into account. We find it impossible to make accurate "counts" when there are more than approximately a thousand colonies on a 4-in. plate, or say, five thousand colonies on a "Drigalski" plate. Numbers in excess of these invariably lead to fusion of neighbouring colonies. Now fusion of colonies of B. coli leads to a deep red coloration on neutral red lactose plates, or a deep green on "Conradi" or "Fawcus" plates, and on every medium that we have tried, the chances of finding a typhoid colony, where it is outnumbered by coli to the extent of say, 10,000 to 1,
are very small. If the feces be diluted so as to give "countable" plates of \( B. \text{coli} \), the typhoid bacilli may be "diluted out" to vanishing point. In higher concentrations, the fusion of \( B. \text{coli} \) colonies leads to mechanical difficulty in finding such typhoid colonies as may be present.

It may be assumed, then, that failure to find \( B. \text{typhosus} \) on plates is not final proof that it is absent from the feces. There is, on the other hand, some reason to believe that this organism can survive for considerable periods in sewage. Firth reports the case of a boy who appears to have been infected directly from a "Stoddart Filter" septic tank, the sewage treated in which was liable to contain \( B. \text{typhosus} \), as a case was then being nursed in a building within the collection area of the sewage. (Note on an unusual cause of enteric fever infection, Lieutenant-Colonel H. Firth, R.A.M.C., JOURNAL OF THE ROYAL ARMY MEDICAL CORPS, vol. iv, p. 55). Again, Gielt (quoted by Shakespeare, Vaughan, and Reed) calls attention to a series of cases that point strongly to survival of \( B. \text{typhosus} \) for considerable periods in sewage. "A man, away from home, contracted typhoid fever, returning to his village where no cases had occurred for a long time. His feces, not disinfected, were thrown on a dung-heap. Five men carted away the latter some weeks afterwards. Of these men four developed typhoid fever, and one had intestinal catarrh with enlarged spleen. The undisinfected feces from some of these cases were thrown on another dung-heap. A few months later two men removed this collection, and one of these contracted typhoid fever.

The report, as quoted, does not state whether the feces of the convalescents were still being thrown on the dung-heap when the latter was removed, but the implication is that the \( B. \text{typhosus} \) survived for a long time in the dung-heap.

In conclusion, we would lay great stress on the relative numbers of \( B. \text{typhosus} \) in the excrement when passed, and on the temperature prevailing outside the body during subsequent periods. \( B. \text{coli} \) is likely to have a decided advantage at temperatures approximating to body-heat. This may explain why Semple and Greig failed to recover \( B. \text{typhosus} \) from a carrier's feces after the fifth day, in India, while we have recovered it up to the twenty-second day in England, and it may serve to reconcile the apparent divergence between the work of Semple and Greig on the one hand, and of Firth and Horrocks on the other.
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B. SURVIVAL ON CLOTHES OF "CARRIERS."

We consider that sufficient work has been done by others as to the time of survival of B. typhosus on fabrics under various conditions. It seemed, however, to be worth while to attempt to ascertain whether this organism could be isolated from the clothing of carriers.

Experiment I.—The soiled shirts from three "faecal" and two "urinary" carriers were intercepted on their way to the disinfecting apparatus, and treated as follows: Selected portions of the shirts were wetted with sterile water, and wrung out over bile salt lactose plates, which were then incubated. Result: No B. typhosus isolated from any of the plates.

Experiment II.—Pieces of linen were sewn into "selected" positions on the inside of the shirts of the above carriers. After three days' wear these portions were detached and kept for three days at room temperature. They were then cut up into small pieces, and added to bile salt brilliant green peptone water, which was incubated and "plated." Result: Motile Gram-negative rods, not fermenting lactose, were isolated from the clothing from Carriers S. (urinary) and L. (faecal). These colonies proved not to be B. typhosus, but were obviously of excremental origin. Their presence may be taken to show that faecal bacteria will survive on the garments of carriers, a point to be noted in connection with laundry work. We regret that the pieces of linen had to be kept three days at room temperature, as this delay may have conditioned the negative result of the experiment. We had no opportunity of repeating it.

C. SURVIVAL ON THE FINGERS OF CARRIERS.

The following technique was employed in all the experiments:—

The fingers were washed in a small quantity of sterile water in a sterile "Petri" dish, or watch glass; the resulting dirty water was added, as a rule, to bile salt peptone water or else centrifugalized, and the deposit plated. The fingers, too, were passed over the surface of bile salt plates while still moist after washing. The media were then incubated and examined for B. typhosus.


Experiment II.—(November 2, 1909). Fingers of urinary carriers F. I. and S. examined (one hour after passing urine). Result: Carrier F. I., many colonies of B. typhosus both on "direct" plates and from the bile-salt peptone water. Carrier S Negative.
Experiment III.—(November 6, 1909). Two urinary and three faecal carriers were paraded without previous warning, and with no reference to the time since excreta had been voided. The fingers of all were examined as above. All were negative as regards *B. typhosus*. From faecal carrier L., however, a fair number of motile Gram-negative bacilli were isolated, giving the cultural reactions of *B. fecalis alkaligenes*. They were, however, agglutinated completely by a 1 in 100 dilution of anti-typhoid serum, and partially in dilutions up to 1 in 400. It may be added that the strain of *B. typhosus* from Carrier L. was very resistant to agglutination, only reacting completely up to 1 in 200 of the same anti-typhoid serum. Unfortunately, the culture kept for further examination was thrown away inadvertently when changing stations, and we were unable to go more completely into the nature of the interesting organism isolated from the fingers of Carrier L.

The series of experiments quoted serves to prove that *B. typhosus* itself, as well as other faecal organisms, can be isolated from the fingers of "carriers," a fact of great importance in connexion with the contamination of food supplies. The following experiments show the effects of washing and removing the bacilli from infected fingers:

**Experiment IV.**—To ascertain whether a finger infected with urine is easily sterilized. (September 26, 1912.) Dipped the tip of the right index finger in the urine of typhoid carrier A (proved to contain upwards of 3,000 million per cubic centimetre). (a) Rinsed in lysol solution (approximately 2 per cent). (b) Then held the finger under the tap, rinsing first in cold, then in very hot water (temperature not recorded). (c) Washed very carefully in about 0·5 c.c. of sterile water in a watch glass, and plated the whole of the water used for this purpose. Result: Three hundred and thirteen colonies of *B. typhosus* on the plate. (d) After the washing in sterile water mentioned under (c), the tip of the finger was thoroughly soaked in absolute alcohol, allowed to dry, and the washing in sterile water repeated. The "washings" were again "plated." Result: Four colonies of *B. typhosus*.

**Experiment V.**—(October 3, 1912.) Contaminated the tip of left second finger with urine from Carrier A. (a) Allowed the finger to dry; (b) washed very thoroughly with soap and water under a running tap. **Dried thoroughly with a cloth.** "Washed" thoroughly with 0·5 c.c. of sterile water in a watch glass and plated the "washings." Result: No *B. typhosus* isolated. (c) Finally dipped the finger in lysol solution (2 per cent), scrubbed, dried with
a cloth, “washed” as before and plated the “washings.” Result: No B. typhosus.

The first experiment shows that it may be very difficult to free a finger from contamination by B. typhosus. The second attempt was successful, possibly owing to the fact that the fingers were thoroughly dried with a cloth, the mechanical friction apparently helping to remove the bacteria. Even if this were the case, it only means that the cloth became infected, and the danger of contami­nated fingers remains obvious.

**D. SURVIVAL IN FOOD.**

**Milk Experiment I.**—Placed 100 c.c. of fresh unboiled milk in each of two flasks, labelling them A. and B. respectively.

Flask B was then placed in steam at 100°C, for one hour, flask A being left unsterilized.

To each flask was added 0·01 c.c. of an emulsion of B. typhosus (strain F. I.), being the growth from a first subculture from a plate made from carrier F. I.’s urine. This emulsion, counted by plating, was found to contain 5,890 million living B. typhosus per cubic centimetre, so that the amount actually added to each flask was 58,900,000 B. typhosus, making a strength of 589,000 bacilli per 1 c.c. in each flask at the commencement of the experiment on January 15, 1912.

The flasks were left at room temperature for one and a half hours, and then their bacterial content enumerated by plating.

Result: Flask A found to contain—

- *B. typhosus*, 4,580,000 per cubic centimetre.
- *B. coli*, 2,750,000

Flask B.—

- *B. typhosus*, 3,440,000

On January 16, 1912, the bacterial contents of the two flasks were again enumerated.

Result: Flask A.—

- *B. typhosus*, 100,000 per cubic centimetre.
- *B. coli*, 1,300,000

Flask B.—

- *B. typhosus*, 5,100,000

On January 18, 1912, the examination was repeated.

Result: Flask A. No. *B. typhosus*, many *coli*.

Flask B.—*B. typhosus*, 90,000,000 per cubic centimetre.
It will be seen that in the foregoing experiment the presence of B. coli in the unboiled milk was sufficient to get rid of, or at least outgrow B. typhosus in three days. On the other hand, the B. typhosus grew and multiplied freely in the sterilized milk.

Experiment II.—Milk. Took a fresh unboiled sample of milk and examined its contents by plating. Result: B. coli, 15,500 per cubic centimetre. Other bacteria, 8,600 per cubic centimetre.

Immediately after plating the milk, 100 c.c. of the sample was placed in each of two flasks, labelled A and B respectively.

Flask A was left unsterilized.

Flask B was autoclaved for twenty minutes at 115° C. To each flask was added 0.001 c.c. of the urine of carrier F. I. This urine was found by plating to contain something over 3,000 million B. typhosus per cubic centimetre. The total number of bacilli added to each flask was, therefore, three million, making the strength in each flask at commencement of this experiment on September 26, 1912, about 30,000 B. typhosus per cubic centimetre. The flasks were left at room-temperature.

On September 27, 1912, the "content" of each flask was enumerated by plating.

Result: Flask A.—

Per cubic centimetre  
B. typhosus, 1,000 (? approximate)  
B. coli and others, 23,000,000

Flask B.—B. typhosus, 10,000 per cubic centimetre.

On September 28, enumeration showed:

Flask A.—Innumerable B. coli and others.  
No B. typhosus.

Flask B.—B. typhosus, 100,000 per cubic centimetre.

On October 1, 1912, enumeration showed:

Flask A.—B. coli and others, 35,000,000 per cubic centimetre.  
B. typhosus could not be isolated.

Flask B.—80,000,000 B. typhosus per cubic centimetre.

Flask B still contained enormous numbers of B. typhosus in pure culture on December 10, 1912, when last examined. There seems no reason why this culture should not remain alive indefinitely.

This experiment confirmed the previous one, in that B. typhosus disappeared from the unboiled milk by the third day. It had practically disappeared on the second day, but one colony happened
The Causation and Prevention of Enteric Fever

to be discovered on the edge of a "concentrated" plate and led to an approximation to a "count."

The inference is that unboiled milk must be consumed soon after contamination to be infective. Boiled milk might be very dangerous indeed if manipulated by a "carrier" subsequent to boiling. The probability of this occurring, is, however, not very great. The experiments have a decided bearing on the question of the sterilization of milk by heat.

Experiment III.—Soup. On September 26, 1912, 100 c.c. of soup freshly prepared from the "stock pot," was placed in a china bowl, no attempt being made to sterilize the bowl or to cover it from the air. The tip of the experimenter's right index finger was allowed to come in contact with the urine of Carrier A. (proved by plating to contain upwards of 3,000 million B. typhosus per cubic centimetre). The china bowl was then lifted in such a manner that the infected finger came in contact for a moment with the contained soup. The soup was left at room temperature with free access of air and dust to the open bowl. On September 27, enumerated the bacterial contents of the "soup." Result: B. typhosus was present apparently in pure culture, numbering 15,500 per cubic centimetre. The soup was now left until October 1, when it was found to be covered with a thick scum, and to have a sour and putrid smell. On plating and examining the soup as to its bacterial "content," no B. typhosus was isolated, but very many clear colonies partially acidifying lactose, and without any effect on glucose, were found on the plates. No attempt was made to further investigate these organisms owing to lack of time. The salient fact is, that B. typhosus had disappeared.

It is also to be noticed that disappearance was conditioned not by B. coli, but by some other organisms, apparently arriving in the broth from the air. The rapid multiplication of the B. typhosus during the first twenty-four hours leads us to believe that the contamination of soup might be a serious danger in an officers' mess, for example, where a "carrier" waiter might conceivably dip a finger in a tureen or plate.

Food Experiment IV.—Liver and Bacon. On September 26, 1912, obtained a portion of "liver and bacon" as served in an officers' mess. This was minced up and diluted with 60 c.c. of sterile distilled water, the whole being placed in a glass basin with a cover. The tip of the experimenter's right index finger was wetted with urine from Carrier A (the sample being proved to contain upwards of 3,000 million B. typhosus per cubic centimetre).
Afterwards, the contaminated finger was allowed to come in contact with the “liver and bacon” mixture, which was then kept at room temperature.

On September 27 the bacterial content of the mixture was examined by plating. Result: B. coli, 6,900 per cubic centimetre. Non-lactose fermenters, 54,700 per cubic centimetre.

The non lactose fermenters formed opaque colonies and did not acidify glucose. No B. typhosus was isolated from the mixture. On October 1 the mixture, which now smelt very sour and was almost solid from deposit of fat, was again examined. Result: Innumerable B. coli. A few opaque non lactose fermenters, no B. typhosus.

These experiments on the viability of B. typhosus in milk, soup, &c., enable us to form an idea of the conditions under which a “carrier” is likely to infect healthy persons through the manipulation of food. It is obvious that the contamination of food before cooking will usually be rendered harmless when the temperature of the food is raised. Contamination after cooking will be very dangerous even if the food is consumed immediately after the contamination takes place. The fingers of a typhoid carrier may be the vehicles of millions of germs. A single drop of urine—say 5 c.mm., or the two hundredth part of a cubic centimetre—may contain anything from a million to three hundred million typhoid bacilli, and these will multiply in soup at the temperature of serving. Again, soup contaminated, allowed to cool, and “warmed up” again to a temperature just pleasant for drinking, may be very dangerous, especially if the soup has been covered to keep out dust in the interim.

Milk puddings, prepared at a high temperature which will probably sterilize the milk, partly used, perhaps contaminated, and then set aside to be used cold later on, may be a source of extreme danger, as in them the B. typhosus will multiply rapidly. Milk boiled or “pasteurized,” perhaps manipulated for an early meal, and then set aside as more likely to “keep” than unboiled milk, will be an ideal culture medium for the B. typhosus. We are inclined to regard the anti-typhoid activities of B. coli as one of the most important natural safeguards against enteric fever, but would again refer to the difficulty of demonstrating the presence of B. typhosus where it is co-existing with a large majority of B. coli. The possibility that a negative result in the search for B. typhosus may be due to limitations of technique must always be borne in mind.
(3) Means of Transmission from the "Carrier" to the "Recipient."

We do not propose to more than briefly consider this part of the subject, which alone is capable of furnishing material for several essays if treated fully. The preceding pages have all aimed at emphasizing the importance of "contact" in the spread of enteric fever, using the term, as does Simpson, "to include direct personal infection from close association, the infection of a commensal, and indirect infection through excreta." The importance now given to this mode of infection is largely owing to the researches of Reed, Vaughan, and Shakespeare already quoted. "Out of a total of 1,608 cases especially studied, and which were accurately located as to time and place," 35.01 per cent could be directly traced to contact, and 27.79 per cent were indirectly traceable to the same mode of origin—in other words, a total of 62.8 per cent of the cases studied were explained in the terms of "contact." It must be remembered that every case traced to "contact" is traced to a "carrier," either acute or chronic. When the above quoted observers made their celebrated investigation, the conception of the "chronic carrier" had not yet emerged, so that their 62.8 per cent of "contact" infections were presumably all traced to acute carriers. Now, Klinger dealing with 1,397 cases of infection traced to "contact," found that 125 or 8.9 per cent were to be traced to "chronic carriers," and this under the conditions of civil life. It is safe to assume that at least this number would have been traced to "chronic carriers" by Reed, Vaughan, and Shakespeare, had they been equipped with the knowledge which further investigation had placed at the disposal of Klinger. This would have brought their "contact" infections to over 70 per cent. But the rôle of "chronic carriers" will probably be found to be far greater in war than in peace. We propose to deal with this subject when we consider the "typhoid carrier" state, but we may here express our opinion that many persons who, under normal conditions, merely "carry" but do not "excrete" typhoid bacilli will, under the abnormal and exhausting conditions of war, become active "excreters" instead of passive "carriers." Taking the three mechanisms of infection included by Simpson under the name of "contact," direct personal infection will be likely to occur through interchange of articles of clothing, pipes, boots, &c., by fouling of the ground in the vicinity of tents, by urine, which will cause moist and infected earth to adhere to boots or gaiters with the possibility of being, later on, transferred to the mouth on the
fingers; and the "splashing" of urine on the boots and garments of persons using the urinals at the same time as, and in the vicinity of, carriers. The infection of commensals need not be enlarged upon. The risks are obvious and increase enormously if the carrier, instead of being merely a commensal, is actually engaged in the preparation or serving of food. Indirect infection through excreta, however, is probably the most important mechanism of the three. The close association of a high typhoid fever incidence with imperfect systems of sewage disposal is well known both in civil life and in the army under peace conditions. Pringle ("Public Health, London," 1902-3, xy), has shown that in fourteen towns with middens the typhoid rate per thousand was 0.25, while in fourteen towns where water-closets were used it was 0.19. In Birmingham ("Report, Health of Birmingham," 1906 and 1908), the incidence of typhoid fever in the "pail" and water-closet houses respectively was as 65 to 43. Dr. Deane-Sweeting, in an inquiry into the high typhoid incidence amongst colliery-workers at Leigh ("Report of the Medical Officer to the Local Government Board," 1907-8), found that the typhoid rate was four times as high amongst these workers as amongst the rest of the population, and attributed this fact to the filthy conditions of disposal of excreta in the mines. Army Medical Officers have frequently recorded evidence to the same effect, a good instance being that reported by Captain B. B. Burke, R.A.M.C. ("The Importance of Latrine Infection in the Spread of Enteric Fever," Journal of the Royal Army Medical Corps, vol. iv, p. 46, 1905). Instances might be multiplied to the same effect. The point that we wish to emphasize is that in war, the danger from this source is likely to be immensely increased. The necessity for using trench latrines brings into prominence those three great intermediaries in the excremental contamination of food, dust, flies and water. We should prolong this Essay indefinitely were we to enter fully into these large questions. Suffice it to say, that we consider the work of Firth and Horrocks, of Neisser and others conclusive as to the danger of dust in the spread of B. typhosus, while the fly has been definitely incriminated by the work of many recent investigators, and may be regarded as one of the most serious factors in the contamination of food with the specific agent of the disease. We would again refer to our experiments upon the viability of the B. typhosus in food, especially where this has been previously cooked so as to sterilize it. Such cooked food if left uncovered, invites the attention of flies and provides an excellent medium for the multiplication of B. typhosus.
The Causation and Prevention of Enteric Fever

(4) Conditions Associated with the "Recipient" of Infection.

The importance of the "carrier" in the causation of infection will vary with the degree of susceptibility to the disease of the healthy persons in the vicinity. We are, therefore, obliged to deal briefly with this important question because our object is to demonstrate the enormously greater danger of the "carrier" in war than in peace; and we desire to show that, in respect of the relative susceptibility of the troops, war conditions tend to favour the spread of typhoid fever by "carriers." The influence of age and of length of service on the liability to enteric is well known, but there is much reason to believe that the essential point is the length of service in infected localities. Major (now Colonel) Simpson ("Report on the Prevalence of Enteric Fever in Pietermaritzburg appended to Army Medical Department Report, 1898"), dealing with the influence of service, shows that (1) "there is a great difference in the prevalence of enteric fever in units arriving from England, the Cape, and India; that the admission-rate in 4,232 men from England was 39.3 per 1,000, in 3,223 men from the Cape 33.2, and in 3,048 men from India, 23.6 per 1,000; that is, roughly the prevalence in Maritzburg is inversely as the prevalence in their previous stations." Further he shows: (2) "That in 1888 and in 1889, when enteric fever was least prevalent, the garrison was composed of corps which had been at least two years in Natal; and, on the other hand, that an exceptional prevalence has been preceded by the arrival of fresh bodies of men." It would be impossible to make clearer the conditions which conduce to the relative susceptibility of bodies of troops. In war, it will always be necessary to use a large number of men who are at the susceptible age, whose period of service is short, and who have not previously served abroad. It may be assumed, then, that their susceptibility to the disease will be very great. Fatigue, hunger, and over-exertion will also lower the individual resistance; while the minor bowel complaints, incidental to life in camp, will bring about the "local" conditions most favourable to the maturation of the B. typhosus, should this organism gain access to the alimentary canal. At the same time these same conditions, affecting "carriers" who happen to be present amongst the troops, will lower their resistance, and lead to a greater excretion of germs. If it is desired to discover whether a person is a "carrier," or not, it is customary to prescribe a purge and then collect a specimen of the faeces for examination. The increased rapidity
of transit of the bowel-contents brings down the bacilli from the bile-containing areas of the intestine before the competition of other organisms in the large intestine has had time to mask the \textit{B. typhosus}. A diagnosis is thus more easily made.

But an attack of camp diarrhoea should have the same effect. It is worth recalling that the diarrhoea rate usually rises before the enteric rate in armies in the field, a point especially noticed in the South African War (Simpson). This camp diarrhoea is likely to increase the excretion of germs by active "carriers" and to lead to the excretion of bacilli by persons who had previously been passive "carriers" only.

As to the causation of enteric fever we may then conclude:—

1. That owing to service exigencies, especially the aggregation of men in barracks or camps, often in hot climates, the importance of "carriers" will be greater under army conditions than in civil life.

2. That in war:—
   a. The excretion of germs by "carriers" will tend to be increased.
   b. The means of transfer of these germs to the healthy will be enormously facilitated, and
   c. The resistance of the individuals composing the forces will be below normal.

\textit{(To be continued.)}