The Relation of the Bacillus typhosus to Typhoid Fever.

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A marked change of mental attitude towards the problems of typhoid fever has been brought about by recent bacteriological work, which has led, *inter alia*, to adoption by many of the view that "the causal organisms multiply only within the human body and cannot carry on a saprophytic existence outside it."[1] It is urged by those who think thus that exaggerated importance has been attached in the past to water, milk and food as vehicles of infection, and it is claimed that attention should be more perseveringly directed to the question of "contact infection."

When it was realised that the excretions of persons attacked by typhoid often contain for weeks, and it may be for months—it is said, in exceptional instances, even for thirty or forty years—vast numbers of bacilli, it was at once declared that this must mean mischief, and evidence of harm so caused was looked for, first in one and then in another direction. As it was found that little or nothing came of the search, one theory after another was propounded to explain this want of success, and new branches of inquiry were successively exploited. Thus one of the first points to receive attention, when infectivity from person to person is in question, is whether there is a special incidence upon particular houses.[2] Then secondly, in the absence of obvious "contact infection," there still remains the possibility that the disease may be spread by apparently healthy persons, the fire thus smouldering unrecognised. In the light of Koch's observations regarding malaria, children naturally fell under suspicion, and it was conjectured that, in particular areas, it would be found the native-born adult had been already rendered immune, while newcomers more especially suffered. Suggestions of this sort prompted inquiry into conditions obtaining in endemic areas of prevalence, both as to the immunity of particular

[1] Vorwort. *Arb. a. d. kaiserl. Gesund.*, 1906, xxiv., p. 1.

[2] It should be noted that, even if the existence of typhoid houses were demonstrated, it would not necessarily follow that personal infection afforded the only possible explanation of such occurrence.

sections of the population and as to the ability of apparently healthy individuals to spread infection. Thus Frosch (Koch's *Festschrift*) observes that certain epidemiological problems necessarily now come to the front, and, as he says, they cannot be settled in the clinic or the laboratory. He mentions "typhoid houses" and "regional immunity." Quite recently Klinger[1] has emphasised the importance of the "chronic bacillus-carrier," and he notes that, for solution of the problems which arise in this connection, we are, since pathogenicity for the guinea-pig does not necessarily imply pathogenicity for man, "unfortunately compelled to fall back upon epidemiological observations." There is, then, an appeal to the observer in the field with regard to these three questions.

(1) *Typhoid Houses.*—Some writers[2] go so far as to refer cases for which no other etiology can be discovered to earlier cases in the same house *or in a neighbouring house*, and this *after an interval of years;* Frosch (1907) describes "Die Wanderung des Typhus eine Strasse entlang von Haus zu Haus." The facts recorded by von Dönitz, in Berlin (1903), are more in accord with those ascertained in this country. Niven insisted ten years ago, in this connection, upon the need of determining whether an observed incidence is any more remarkable than mere chance will explain, and when this test is applied the case for "typhoid houses" breaks down, just as that for "cancer houses" (Behla) has been found to do by Symons and others.[3]

A particular instance of employment of the house incidence test in typhoid fever may, however, be mentioned, as the outbreak presents special points of interest. It occurred in Beuthen, in Upper Silesia,[4] and there were two waves of disease; the first prevalence reached its height in May, the second at the end of July. Contact infection is said to have been responsible for the double outbreak, and this is held to be proved by the occurrence of a large number of secondary cases in houses. The report is a full one; it is open to the criticism that no attempt is made to separate primary from secondary cases, but this, it may be said, is a *petitio principii.* At least it may be urged, however, that the early and later cases in Beuthen occur in a way which suggests that the

[1] *Arb. a. d. kaiserl. Gesund.*, 1906, xxiv., p. 35.

[2] Richter, *Zeit. f. Medizinal-beamte*, 1904.

[3] In an interesting study of the house incidence of *phthisis* in Posen, by Wernicke (Koch's *Festschrift*), the point is made that, in certain streets, some houses suffer severely while many escape altogether. There is, however, no reference to the a priori probability of occurrence of the phenomena observed. If the numbers of susceptible persons, in the houses in question, may be assumed to have varied within the usual wide limits, I find that chance, and chance alone, would explain the results exhibited by the Posen figures.

[4] *Zeit. f. Hyg.*, 1901.

separation should have been made.[1] The report is open to the further criticism that, arithmetically considered, the later incidence upon houses originally invaded is no more remarkable than chance would explain. The houses, we are told, are massive, two or three storeys high, and each contains four to ten families. The inhabitants of any one house thus constitute a not inconsiderable fraction of the total population at risk. The probability of occurrence, as a mere coincidence, of later cases in houses already attacked is therefore appreciable, and as no regard is paid in the report to this fact, the argument is vitiated.

Particularly noteworthy, in connection with study of "contact infection," is the explanation given of the double prevalence. We are told how, in the May outbreak, step by step a contact epidemic was built up; the authorities, it appears, were then stimulated to undertake energetic measures (of isolation and disinfection), with the result that slackening followed in June. Surely, however, the report says, contact infection in the dwelling again comes into operation; . . . numerous convalescents, too, are discharged from hospital, pressure upon accommodation leads to early return of cases to their homes, and these spread abroad contagion in the disinfected houses. Now, too, those affected in the first outbreak and not removed to hospital again begin to mix with their fellows . . . and so on.

It is perhaps unnecessary to pursue the "house incidence question" further, as during the last few years it has been practically abandoned in favour of more novel lines of investigation.

(2) *Regional Immunity.*—On turning now to the study of the smouldering of typhoid in particular communities, or in sections of the population, a most instructive case is that presented by Conradi.[2] This observer had published an earlier paper on dysentery in and near Metz, giving an account of an outbreak of 70 cases, in which not one single native-born inhabitant more than 22 years old was attacked, while twenty-five strangers, whose ages exceeded 25 years, suffered.[3] The numbers, it will be felt, are too small to found conclusions upon, but the

[1] Examination, for example, of Barry's (Tees Valley, 1890-91) figures show that the percentage of later or secondary attacks in houses to total attacks rose from quite a small to quite a large percentage as the outbreak developed (3 per cent. in the first, to 35 per cent. in the sixth fortnight). The Beuthen investigator draws no distinction between earlier and later stages of prevalence; indeed, when he shows that second cases in houses are common, he takes periods subsequent to those of development of the outbreaks. He would, of course, have obtained results such as those he exhibits by treating in a similar way figures relating to almost any food or water epidemic.

[2] *Arb. a. d. kaiserl. Gesund.*, Berl., 1906, xxiv., p. 97.

[3] The main group of cases occurred in the Hauptstrasse of Moulins, where there were six cases in one house, four in each of two others, and so on. Clearly generalisations from such figures are unwarranted. Moreover, it appears (*see* Koch's *Festschrift*) that there was "avoidance of medical treatment" and "concealment of cases." May not this "avoidance" and "concealment" have been especially manifested by Metz-born adults? Again, it transpires that six native-born persons, whose ages ranged from 16 to 22, were attacked.

experience is interesting as it no doubt led to the main inquiry to be now referred to. Conradi's thesis is that typhoid has prevailed from time immemorial in Metz, and that it ever smoulders, mainly among the babies and young children of the town, the native population at higher ages being thus rendered immune. The disease spreads, however, to newcomers and hence to the soldiers; this especially occurs during campaigns; it did so very notably at the time of the siege of 1870, when upwards of 30,000 cases occurred in Metz and its near neighbourhood; but the mischief continually operates and interferes, year in and year out, with military efficiency.

The thesis is supported by a comparison of certain percentages, of total deaths and of typhoid deaths, contributed by native-born persons.

Figures are given showing deaths at age-periods (1880 to 1904). There were, it transpires, 382 deaths from typhoid among immigrants (173 among soldiers, 209 among civilians), and only 45 among the native-born population. Unfortunately the proportions of these two classes of the inhabitants cannot be stated.[1]

Conradi, however, calls particular attention to the fact that up to 15 years of age natives and immigrants suffered from typhoid to something like an equal extent; but over 25 there were only four natives as contrasted with 119 immigrants. He says it may be suggested that this striking difference finds explanation in the fact that, following upon the events of 1870, the native-born element in Metz underwent an enormous diminution; but he disposes, as he believes, of this suggestion by giving figures from which he estimates the proportions of natives and immigrants respectively among the older people now living in Metz. At ages over 30 there were, in 1890, 409 deaths, from all causes, among immigrants and 120 among native-born persons. The argument thus assumes this form:—

At ages over 30, in 1890, 22 per cent. of the total deaths occurred among native-born persons. At ages over 25, taking a period of twenty-five years (1880 to 1904), only 3 per cent. of the typhoid deaths occurred among native-born persons. Hence, &c. Obviously these percentages should not be compared with one another; the first is based on a single year, and that, it appears, was an exceptional one; further, a rate in 1890 is not comparable with an average rate taken over the period 1880 to 1904, having in view the admitted remarkable changes of population in Metz. But the most important point is that, of the 120 deaths of Metz-born persons over 30 years old (upon which the first percentage is based), no fewer than 79 occurred at over 60, and actually 99 at over 50 years of age.

Clearly the extent to which Metz-born persons are included among

[1] In 1895, however, out of 45,480 persons, only 16,484 were native-born; 28,996 were civilian immigrants, to whom must be added 16,776 soldiers, making 45,772 total immigrants.

those dying at advanced ages is not much to the point. We have to deal, if the comparison of percentages is to be precisely made, with the typhoid ages. We cannot obtain from Conradi's tables the percentage of native-born persons attacked at the ages of special typhoid incidence, but it must be considerably less than the percentage (14 per cent.) at the ages 30 to 50. From the rates given in the tables we may conclude with some degree of certainty that there were not, in 1903, more than 4,000 or 5,000 Metz-born persons in Metz at ages exceeding 20 years, and on this basis there cannot have been at the ages 20 to 45 more than one Metz-born to some ten or twelve non-Metz-born persons. This fairly corresponds with the proportion of Metz-born to non-Metz-born persons among those attacked by typhoid at those ages in 1903. (The figures are given in Table V.[1]) Thus the supposed immunity of native-born persons proves to result from a mere trick of the figures; and as the native-born persons at the ages in question are in actual fact an insignificant minority, there is no need to assume that their failure to appear in larger numbers, in mortality returns relating to typhoid, can only be explained by adopting the hypothesis that they are immune to that disease.

It is especially deserving of note that the garrison, consisting of men at susceptible ages, and constituting more than one-quarter of the total population of Metz, is entirely immigrant—the Metz-born lads as they reach the age of military service are apparently removed from the town. Moreover, the special influence of the year 1870 needs to be considered. The tables given by Conradi supply evidence that few Metz-born babies and children remained in Metz after the siege; hence it is no matter for surprise that typhoid mortality figures relating to 1880 to 1904, *i.e.*, to a period from some years after to upwards of thirty years after the war, yield scanty evidence of the presence in Metz of native-born persons between 15 and 35 years of age.

Another interesting contribution to the "regional immunity" question is that of Frosch.[2] The paper has been much quoted in Germany, but the figures are too small to carry weight. In a population (of apparently 400 to 500 persons) occupying sixty-six houses, in Wittlich, a town of 3,600 inhabitants, a water-borne epidemic of typhoid occurred in 1895. Subsequently (1896 to 1903) further cases occurred in Wittlich, and in

[1] This correspondence is observed on comparing an estimated population in 1903 with cases in that year. Comparison of total deaths in a number of years with the deaths or population of a particular year is, as already remarked, open to obvious objections.

[2] Koch's *Festschrift*, 1904.

the town generally new arrivals and natives of the place were indifferently attacked; in the special area of the water epidemic no natives suffered, but "several cases" occurred among strangers. According to the case-rates obtaining in Wittlich, only three or four cases would, on the law of chance, have to be allotted, for the period 1896 to 1903, to the special area. The fact that no native of the area was attacked does not, of course, under these circumstances, furnish material upon which to generalise.

Conradi has made an elaborate study [1] of this question of "regional immunity" in Ottweiler. This town (5,028 inhabitants) suffered from an extensive outbreak of typhoid (353 cases) in 1891 to 1892. Conradi finds that 75 further cases occurred, during the succeeding fourteen years, *in natives who were, at the time of the outbreak of* 1891 *to* 1892, *living in the epidemic area, and had since continued to do so.* Clearly, as Conradi says, more evidence is required before the doctrine of regional immunity can find general acceptance.

(3) *Chronic Carriers.*—Study of chronic bacillus carriers, however, has brought new problems to the front. If persons who have undergone attack by typhoid be examined after a short interval of time, 3 per cent. or 4 per cent. are found to have typhoid bacilli in their excretions. When this was first demonstrated it was assumed that all difficulties with regard to the origin of cases of the disease were at an end. The facts did not, however, fit into their places in the way anticipated. Thus, in the first place, there is apt to be trouble as regards discovering chronic carriers under circumstances in which they might be expected to be found. Brummund [2] could not detect one carrier among 160 persons examined two or three years after attack, and he says: "Es ein besonders glücklicher Zufall ist bei einem chronischen Typhusträger bazillushaltigen Stuhl zu bekommen." Kayser, too, has pointed out the remarkable intermittency of appearance of the bacilli ("exquisit schubweisen Darmentleerung"). Again, when chronic carriers are actually kept under observation, no contact cases (Klinger) are found to occur, but this is, perhaps not unnaturally, explained as being due to the fact that proper precautions are taken.[3]

[1] "Klin. Jahrb.," Bd. xvii., H. 2.

[2] *Zeit. f. Hyg.*, Leipz., 1907, lvi., p. 425.

[3] Dr. Geo. Dean (*Brit. Med. Journ.*, 1908, i., p. 562) has recorded a case of a carrier of twenty-nine years standing from whom, so far as is known, no case has originated. Kirchner ("Klin. Jahrb.," Bd. xvi.) has shown that when a cholera epidemic is declining the primary bacillus carriers increase in numbers. Conradi thinks that in typhoid, too, the appearance of carriers indicates loss of virulence of the organism. In point of fact, the practical outcome of this teaching appears to be that in a multitude of bacillus carriers there is safety.

A curious explanation of this absence of contact cases is given by Brummund. The outbreak recently investigated by him at Mulsum was caused by milk, and among those who did not consume the infected supply there were no contact cases. Brummund points out that the inhabitants of the place were divided into two hostile camps, according to their source of milk supply. "Die Mulsumer gewissermassen in zwei feindliche Heerlager mit dem Devisen; hie Mulsumer, hie Kutenholzer Molkerei, gespalten sind, die sich grimmig befehden und jedenfalls allen freundschaftlichen Verkehr miteinander vermeiden." In the schools, it is true, the children drinking one supply sat side by side with those drinking the other supply, but here, we are told, the insistence of the school teacher upon the risk of infection impressed the scholars and led to proper precautions being taken.

The need for reconsideration of the whole position, in the light of the facts ascertained with regard to "carriers," is urged by Conradi.[1] He now contends that typhoid is especially infective quite early in the disease, even in the incubation period. Bacillus carriers, he says, are of three kinds (primary, secondary and tertiary), and only the last kind really constitutes a source of danger, and that only under exceptional circumstances. Contact infections arise, as his detailed examinations show, within the first or second weeks of illness of the original cases; late contact cases rarely occur. Sporadic cases, however careful the investigation made, cannot, as a rule, be traced to previous cases, and these sporadic cases, even in the absence of isolation, disinfection, &c., do not give rise to secondary cases. He asks: "Why are outbreaks thus limited to single cases, which again on their part give rise to no further infection?" Too much reliance must not be placed, he adds, in linking up cases of infection, upon the healthy "carrier." "It is difficult to prove that he is infectious, as we discover him only because of the fact that he lives in infected surroundings. *Post hoc ergo propter hoc.*" Having carefully watched bacillus carriers, he finds they are not, as a rule, infective; nothing is known concerning the virulence of the organisms they excrete, and he concludes that it can only be under exceptional circumstances that they are a source of risk.

In striking contrast to this attitude stands that of Kayser, who has from the first claimed that the *rôle* of the chronic "carrier" is an extremely important one. In 1906 he wrote his "Milch und Typhusbazillenträger," and in the same year, in his "Gefährlichkeit von Typhusbazillenträgern," he recounted the case of the Bäckermeisterin, who ten years previously had typhoid, and whose young assistants, year after year, suffered from gastro-intestinal disorders, until at length, upon

[1] "Ueber die Kontagiosität des Typhus," "Klin. Jahrb.," Bd. xvii., H. 2.

one of them developing typhoid, the Bäckermeisterin herself came under suspicion, and the *Bacillus typhosus* was demonstrated in her stools. Again, he gives the case of Frau M., who, having suffered from typhoid thirty years before, was at length, on the occurrence of the disease in her neighbours, proved to be a bacillus carrier. It is, of course, always possible, working on these lines, to show that any given bacillus carrier has suffered from, or has been in contact with, a case of the disease, for we have thirty years at our disposal. While the significance of the presence of *Bacillus typhosus* in the stools is made clear, the method of proof adopted suspiciously resembles argument in a circle.

In the last year or two many further cases of chronic carriers have been reported. One of the most remarkable is that from New York, recorded by Soper.[1] The history of the carrier, a cook, is traced for ten years, and in seven "families" (in this designation visitors, gardeners and laundresses are included) with whom she lived during that time a case or cases of typhoid are said to have occurred. The difficulties in obtaining this record must have been great, as the cook herself resolutely declined all information, and admittedly there is doubt about certain particulars. Assuming, however, the substantial correctness of the story, it cannot be regarded as finally conclusive. Five of the outbreaks—and they include those best attested—occurred at seaside resorts near New York. Little is said about possible sources of infection other than the cook, but in one outbreak there was strong suspicion that shellfish and in another that water was at fault. "The families were of ample means and accustomed to living well." They constituted, in fact, a selected population. The problem assumes the form—Given a person, associated with a number of selected persons, first at one and then at another seaside resort, during a period of ten years, to find the chance that at the end of that time inquiry will elicit expression of a belief that cases of typhoid have occurred in correspondence with such association. If the seaside resorts were, as is probable, also more or less selected, and if at all of them, as we are told was the case at one, sewage-polluted soft clams and oysters were consumed by the "families," the a priori chance of the occurrence, apart from infectivity of the cook, of the sequence of events recorded is far from being a negligible one.

There are, moreover, certain asylum observations to which great interest attaches. Those recorded by Nieter, and by Nieter and

[1] *Journ. Amer. Med. Assoc.*, 1907, xlviii., p. 2019.

Liefmann,[1] furnish instances of successful search being made, on the occurrence of cases of typhoid, for a bacillus carrier, among a considerable number (perhaps some hundreds) of persons more or less associated with the cases. Facts of a similar sort have been recently reported by the Ledinghams.

The question here arises as to the standard to be applied in such investigations. What percentage of carriers may be expected in an asylum, apart altogether from occurrence of typhoid in the institution at the time of the examination? Klinger (1906), at the Strasburg typhoid station, found eleven bacillus carriers among 1,700 healthy individuals —0·6 per cent.[2] Minelli,[3] in a prison where there had been no typhoid for three years, found 1 in 250 (0·4 per cent). It is noteworthy that bacillus carriers are more common among females than males, and there is a remarkable association of liability to excrete typhoid bacilli with certain pathological conditions (*e.g.*, gall-stones). The age-distribution of a population examined may be also expected to have influence. Clearly, therefore, provided standards such as those just quoted are to be applied, it must be held there is nothing much out of the common if, out of a hundred or so individuals submitted to examination (by Nieter and Liefmann or in the Ledinghams' cases), a bacillus carrier or two be found. There is a further point which should be kept in mind. Inasmuch as chronic carriers only excrete bacilli intermittently ("schubweise"), repeated examinations as a rule have to be made. If, however, a suspect be again and again examined we cannot take as our standard 1 in 100 or 1 in 200, as above; we require, of course, to know what the ultimate result would prove to be were the control population submitted to equally searching and persistent examination.

Nieter and Liefmann found thirteen carriers among 900 female lunatics—1·5 per cent.; or, if consideration be limited to a particular part of the asylum, used for some years previously for isolating typhoid and dysentery, they found seven carriers among 250 persons examined, *i.e.*, 2·8 per cent. They point out:—

(1) That their patients were women, among whom, according to Klinger, carriers are three times as common as among men.

[1] *Münch. med. Wochenschr.*, 1906, liii., p. 1611, and 1907, liv., p. 1622.

[2] This is the figure sometimes quoted, but as a matter of fact, Klinger has found 27 cases among 1,800 examined—1·5 per cent. In some of these there was a history of having in the past suffered from typhoid, and for this reason, apparently, they are excluded from consideration.

[3] *Centralbl. f. Bakt.*, Jena, 1906, xli., p. 406.

(2) That the opportunities for spread of infection are especially great in a lunatic asylum.

(3) That the 250 inmates occupied a building to which typhoid cases had for years been sent from all parts of the asylum.

Again, they emphasise the fact that their tests were again and again repeated, and it was only as successive examinations were made that the percentage of carriers grew, for some patients who gave a negative result on a first examination yielded a positive one later. As the ultimate percentage reached was only 2·8, there seems no reason for concluding that the proportion of carriers was higher, in this institution, than it might be expected to be, on the facts at present known, in any other asylum for female lunatics in this country or in Germany.

Nieter and Liefmann themselves say: "Our observations contribute little towards determining the important and interesting question how great the risk from association with bacillus carriers is. Cases of typhoid were developed in wards in which we were able to demonstrate the presence of chronic carriers, but the transference of patients from ward to ward was such a common occurrence that too much stress should not be laid upon the local distribution of these cases."

The Ledinghams' cases and, so far as figures are given, those from Merzig, Hördt, Saargemünd and Klingemünster, referred to by Nieter, tend to confirm a belief, which may now perhaps increasingly be entertained, that the appearance of typhoid in an asylum by no means necessitates a higher percentage of chronic carriers being found among the inmates than might be expected in any similar population free from typhoid.

Nieter also quotes Friedel's case. Only one carrier was found here,[1] but stress is laid on the fact that this carrier handled uncooked vegetables in the asylum kitchen; no attempt is made to differentiate, between those attacked and those not attacked by the disease, as regards consumption of such vegetables. In gauging the significance to be attached to discovery of a chronic bacillus carrier among those handling the foodstuffs consumed in an asylum it is important, of course, to remember that many persons are more or less concerned with the supply of food to large institutions, and that the chance of discovering a bacillus carrier is enhanced *pari passu* with increase in the number of individuals submitted to bacteriological investigation.

[1] Examination of the excreta of all those employed in the asylum kitchen, scullery, milk and laundry services was undertaken. The only positive result was in a woman, aged 65, who had been for many years under observation in the asylum, and was believed not to have had typhoid.

Murchison records the fact that the President of the Society of Engineers of the day, in a letter to the *Times*, December 4, 1871, stated that "having examined many houses in which enteric fever had occurred he had in every instance been able to trace the outbreak to some unlooked-for defect in the drainage." Obviously, in connection with such experiences, as with inquiries made at the present time also, it is necessary to have regard to control observations. If a milk supply or water supply be suspected, the investigator does not content himself with discussing whether individual persons attacked had consumed the milk or water; the fact that any particular percentage of the sufferers did so does not interest him, unless he be satisfied there was a greater incidence upon consumers of the supplies in question than those supplies were entitled to show. He would, of course, estimate how many consumers of the milk or water might a priori (having regard to all the facts as to the distribution of the milk or water) be expected to be attacked, and only if that number was exceeded[1] would he attach importance to the degree of special incidence observed. This plan of procedure is not adopted in dealing with bacillus carriers in institutions; yet milk and water are known to have communicated typhoid; while Conradi tells us we cannot say whether the bacilli excreted by a healthy carrier are capable of conveying infection.

On a review of the facts now known the conclusion may clearly be formulated that the chronic carrier is not, as a rule, a source of mischief. It may be, as Conradi conjectures, that there are special circumstances under which he becomes dangerous, and the question in that case is, What are those circumstances? On the other hand, as the evidence at present stands, we cannot lose sight of the possibility that the chronic carrier may not be immediately concerned with transmitting typhoid fever at all. Two further questions then arise. Does the *Bacillus*

[1] As illustration of the need for laying stress on considerations of this sort may be mentioned the fact that, in an account of a recent inquiry as to the origin of 125 cases of *phthisis*, it is stated that in 13 cases out of the total number infection was probably contracted from a relative. The number of cases in which it might be anticipated there would be the appearance of such supposed transference of infection as result of chance coincidence (assuming for the sake of argument that phthisis is never directly transmitted from one human being to another) can be estimated arithmetically. I find that more than thirteen such instances might be expected to present themselves in 125 cases examined, as a matter of pure chance, provided it be taken for granted that the proportion of the population at the phthisis ages who have within the last five years manifested symptoms is not less than 3 per cent. The number (13), therefore, to which the casual reader might be disposed to attach special significance, is really just what might be expected as result of mere coincidence. Obviously it is unnecessary to seek explanation of the infection by an appeal to relatives in all, if in any, of the 13 cases.

typhosus represent a particular phase in the life-history of the specific organism of typhoid? Or, on the other hand, is the *Bacillus typhosus* merely a "secondary invader"?

Many observers, having regard to the facts that the disease is "constantly springing up in isolated cases without any possible communication," and that, when well-defined outbursts occur, the number of secondary cases is comparatively speaking small, are inclining to the view that contact from man to man plays no great part in causing spread of the disease. Arguing, moreover, from conclusively demonstrated instances of origin of typhoid from consumption of polluted water and sewage contaminated shellfish, from the involvement of dwellers near foul foreshores, and from the incidence observed in Germany and in this country upon certain riverside populations, it is natural to seek explanation of such associated circumstances in a hypothesis that the disease is, as a rule, in a wide sense of the word, "water-borne." It might then, further, be suggested that the typhoid organism has two phases in its life-history, and that in one phase it is apt to produce typhoid fever; while in the second, the *Bacillus typhosus* phase, it is only exceptionally, if at all, capable of causing extension of disease by direct contact. On the other hand, the causal organism of typhoid may be in no way connected with the *Bacillus typhosus;* and, indeed, apart from considerations already adverted to, there are several reasons for advocating a critical re-examination of the relationship of the *Bacillus typhosus* to typhoid fever. The following more particularly deserve mention:—

(1) There are the difficulties which are referred to from time to time by bacteriologists. Numerous anomalies and exceptional results have been recorded with regard to agglutination (*see*, for example, papers which have appeared in the last two or three years by Zupnik, Poggenpohl, Gaehtgens, and others). There is, further, a difficulty owing to the close resemblance—it may be the actual interchangeability—of the typhoid bacillus and closely allied organisms, notably those known as paratyphoid bacilli. Mixed infections of typhoid and paratyphoid occur, not only in individual cases, but in epidemics.[1] Fornet and Levy, and Gaehtgens, in recent papers,[2] have demonstrated remarkable epidemiological relationships ("epidemiologische Beziehungen zwischen Typhus und Paratyphus B."). Smallman and MacConkey have obtained results which might naturally be

[1] Paper by Thomas, "Klin. Jahrb.," Bd. xvii., H. 2.

[2] *Arb. a. d. kaiserl. Gesund.*, 1907, xxv., pp. 247, 250.

explained by assuming that one bacillus was changed into the other in the animal body.[1]

The bacillus is not, as has been seen, always possessed of pathogenic property. It was found in the public water supply of Detmold (by Beck and Ohlmüller) in 1904, about a month after the cessation of an outbreak, and was not then causing inconvenience. The question whether the disease can be experimentally produced in animals has never been satisfactorily cleared up.[2] The recorded instances of accidental laboratory infection in man may have been due to causes other than ingestion of *Bacillus typhosus*, and the swallowing of cultures has given, at any rate in some cases, a negative result.

(2) A number of organisms, believed at one time to be "causal," are now classed as "secondary invaders." Particular interest attaches here to the case of the hog cholera bacillus (a near relation of the *Bacillus typhosus*), an organism long supposed to be the cause of hog cholera. It is now stated, however, that while cultures of the hog cholera bacillus produce on inoculation a disease closely resembling hog cholera, the blood of animals thus inoculated is not, while the blood of animals which have acquired the disease in a natural way is, infective. The hog cholera bacillus is regarded in America as an inhabitant of the intestine of the normal hog. Dorset, Bolton and McBryde[3] point out that this state of things "is entirely analogous to the condition under which many pathogenic organisms exist—as, for example, the pneumococcus in the mouth of healthy individuals, and the swine plague bacillus (*Bacillus suisepticus*) on the tonsils of healthy hogs. . . ." They add: "*Bacillus coli communis* is a familiar example of an organism constantly present in health and yet assuming under certain conditions very great pathogenic power."

(3) The known behaviour of the bacillus, as observed in the laboratory, does not altogether accord with field observations, and

[1] See, moreover, Savage's observations on "variant forms" of *Bacillus coli*; and the work of Twort, who has shown that sugar-fermenting powers may undergo change; while Stephens has made it clear that non-motility and the non-flagellate condition may be assumed by *Bacillus typhosus* under certain conditions of growth.

[2] Grünbaum (*Brit. Med. Journ.*, 1904, ii., p. 817) published a "preliminary communication," containing an account of the appearances produced in the small intestine of the chimpanzee by feeding experiments. His observations, he considers, "assist in fulfilling Koch's third postulate as applied to Eberth's bacillus."

[3] Annual Report of the Bureau of Animal Industry, 1904.

with these the typhoid organism must comply. Thus Dr. Barry, in giving evidence before the Water Commission (1892 to 1893) said: "If it is found that what is called the specific bacillus of typhoid fever will not live under particular circumstances, as has seemed to be shown in laboratory experiments, then I think that possibly the true bacterium has not been found." During the last fifteen years a new difficulty has arisen, inasmuch as it has been ascertained that large communities can with impunity be supplied with polluted river water, for periods of some years, without manifest injury, provided certain precautions are adopted, and these, it would seem, are not of a kind which necessarily preclude the bacillus from obtaining access to household supplies.

It may be urged, on the other hand, how can the common association of a "specific organism" like the *Bacillus typhosus* with a particular "symptom complex," not only in sporadic cases but also in outbreaks of disease, be explained if the organism be a mere "secondary invader"?

To this it may be replied that for many years the bacillus was held not to occur in the blood; now it is readily demonstrated there. At one time it was believed to be impossible to single it out from the fæces; now it can be isolated from material in which there is only one typhoid bacillus to hundreds of other bacilli. With perfecting of bacteriological methods bacilli, which are now supposed to be very particular as to the company they keep, may turn out to be travelling incognito in all sorts of places Study, at a time when there is no cholera, of material from the intestines of pilgrims at El Tor, reveals the presence of the cholera vibrio. Pratt, Peabody and Long[1] hold that the typhoid bacilli in the alimentary tract come chiefly from the bile, and that they are speedily destroyed in the intestine. Neufeldt[2] explains the El Tor phenomenon by assuming that the pilgrim suffered from cholera long ago, and that the vibrio has for years ceased to be quite at home in its host's intestines, but owing to slight dysenteric or other disturbances it is enabled ultimately to again come to the front. In the same way, says Neufeldt, typhoid "bacillus carriers" come under notice owing to rapid multiplication of the bacillus as result of their suffering from some derangement of the biliary secretion (gall-stones, &c.). The cholera or typhoid bacilli are always there, but the dysentery or biliary colic places them in evidence; they are representatives of the ordinary flora of the intestine brought into prominence

[1] *Journ. Amer. Med. Assoc.*, 1907.

[2] *Arb. a. d. kaiserl. Gesund.*, Berl., 1907, xxv., p. 164.

by special favouring circumstances. Why, then, should not typhoid fever also be competent to place typhoid bacilli in evidence? In other words, why continue to regard the presence of typhoid bacilli as necessary for the production of typhoid fever?

However this may be, some discrimination is necessary in attributing outbreaks of typhoid to contact infection. Murchison held all supposed contact cases were "as readily explicable on the supposition that the disease has had a local origin as upon that of contagion." Without accepting quite such an extreme view it is at least well to remember the caution of Bulstrode[1] that we should always, in connection with supposed contact infection in households, keep in mind the possible "continued operation of the cause, which may have given rise to the first case in any house where many cases arose."

DISCUSSION.

The PRESIDENT (Dr. Newsholme) spoke sympathetically of the absence, through illness, of Dr. Seaton. The Section, he said, had had the great advantage of listening to papers on the pathogenicity and specificity of the typhoid bacillus and the important question of carrier cases. On the one hand an able agnostic in this matter, like Dr. Hamer, said the typhoid bacillus was possibly not the bacillus of typhoid fever, and suggested that carriers of the disease did not frequently occur; while, on the other hand, Dr. Davies had given a most lucid description of an outbreak in which carrier cases figured, and in which the connection between these cases and the subsequent outbreaks was practically demonstrated. It would be difficult to imagine circumstances under which a more interesting discussion might be expected.

Dr. E. W. GOODALL said that several very important questions concerning the etiology of typhoid fever had been raised in the papers read that evening, but he would confine his remarks to three of them. The first, a fundamental one, had been raised by Dr. Hamer, who appeared to have doubts whether the *Bacillus typhosus* was the cause of typhoid fever. Nor were his doubts without support, for Dr. C. J. Martin was reported to have stated before the Royal Commission on Vivisection that the evidence that the bacillus was the cause of typhoid fever was not absolutely complete. He (Dr. Goodall) was very much surprised to read that statement, for he had been led to suppose that the experimental evidence, the unfortunately only too frequent occurrence of laboratory infection, and cases such as that of the Paris nurse who developed typhoid fever

[1] "Whitehaven Report," 1903.