

## Epidemiological Section.

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### The Bacteriology of Summer Diarrhœa.

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WE propose in this communication to give a brief review of our knowledge of the bacteriology of summer diarrhœa, dealing more particularly with the results obtained during the last four years by Morgan and other workers in the Lister Institute. Details of bacteriological technique will in great part be dispensed with, as our main object is to elicit discussion on the possibility of correlating current epidemiological opinion on this question with bacteriological findings.

The difficulties experienced by clinicians in accurately differentiating the true summer diarrhœa of children from others occurring during the epidemic season are felt also by the bacteriologist who naturally desires to investigate a series of selected cases of the disease concerning which clinical opinion is unanimous. Unfortunately, this desideratum can never be completely realized. The cases vary greatly in severity in the same season and in different seasons, and accordingly the bacteriologist has to control the results obtained in a selected group of clinically typical cases with those obtained in extensive control series of less severe diarrhœas, diarrhœas occurring as complications of other diseases, and, finally, in a series of apparently healthy children.

The first attempt on a large scale to investigate the bacteriology of summer diarrhœa was made as long ago as 1887 by Booker in America. Up to that time the flora of the intestinal tract had received but scanty attention. Only two years before, in 1885, the *Bacillus coli communis* and the *Bacillus lactis aerogenes* had been described by Escherich and

for some time the importance of these micro-organisms was considered to be a dominant one.

The work done by Booker was very complete so far as bacteriological methods permitted, but with our present knowledge little or no etiological significance can be attached to the micro-organisms isolated by him in summer diarrhœa. Booker himself felt that no one micro-organism of the many varieties recovered from his cases could be considered as the specific agent. In an attempt to correlate his bacteriological findings with clinical phenomena he adopted the following grouping:—

Group I, 35 cases: The patients in this group are emaciated and exhibit toxic phenomena. The stools are at times frequent and at other times infrequent. Among other bacilli isolated (including the *Bacillus coli communis*) the *Bacillus proteus vulgaris* appears to occupy an outstanding position.

Group II, 27 cases: The cases are serious and show general toxic disturbance. The stools are fluid or soft, often green and slimy, and vary in frequency from three to twenty in twenty-four hours. Micrococci (streptococci) preponderate in these stools, in conjunction with pus-cells.

Group III, 6 cases: The cases are serious, the stools vary in frequency and often have a putrid odour. The *Bacillus coli communis* is numerous in all these cases along with the *Bacillus lactis aerogenes*. A large number of inconstant varieties are also found.

Group IV, 24 cases: The cases are mild and of short duration. The *Bacillus coli* predominates in all cases and occurs sometimes in pure culture.

Some general observations made by Booker with regard to the distribution of coliform organisms in the intestinal tract and in the fæces are of interest. He noted that while the *Bacillus coli communis* and the *Bacillus lactis aerogenes* do not altogether disappear from diarrhœal fæces, they occur in much fewer numbers than in the fæces of healthy milk-fed children, and, in fact, tend to diminish in proportion to the severity of the case, their place being taken by varieties which occur inconstantly in the healthy intestine. This substitution of the normal flora by inconstant varieties in diarrhœal fæces will be referred to later.

In considering the work of American authors on the diarrhœas of children one must pay close attention to the nature of the cases they dealt with. It will be mentioned below that the clinical picture of

summer diarrhoea in many of the epidemics investigated in America is entirely different from that which prevails in this country. One very crucial point of distinction is the condition of the stools. In cases met with in London the stools may be characterized generally as green, watery, slimy and putrid with varying acid or alkaline reaction. Blood and mucus are rarely, if ever, found. Unlike most of the cases subsequently investigated in America, Booker's cases did not present blood or mucus in the stools, and they are, accordingly, clinically comparable with our summer cases in London. As, however, the dysentery bacillus of Shiga was not described until 1898, it is impossible now to know whether Booker's cases may have been associated with bacilli of the dysentery group.

The next advance made in the bacteriology of infantile diarrhoea was made in 1903 in America, when the subject was investigated by a number of observers, notably Wollstein, Park, Collins and Goodwin, Flexner, Duval and Schorer, Weaver and Tunnicliffe. The Flexner type of the dysentery bacillus was discovered in 1899, and was at this time not completely differentiated from that of Shiga described in 1898. The object of the investigations above referred to was to determine whether these infantile diarrhoeas were also associated with dysentery organisms. The clinical features in most of the epidemics recorded were very similar. The stools invariably contained mucus, and as a rule an admixture of blood.

Wollstein examined the stools of 114 cases of infantile diarrhoea, and isolated the dysentery bacillus (Flexner type) in 39 cases. From all those cases which showed both blood and mucus in the stools, the dysentery bacillus was recovered. Agglutination reactions with the sera of the patients were obtained in a large percentage of the positive cases, but not during the first week of the disease. In autopsies the dysentery bacillus was never found outside the intestinal tract.

In the same year (1903) Park Collins and Goodwin examined cases of summer diarrhoea associated with excessive mucus and occasional blood. The majority of such dysenteric cases showed Flexner bacilli in the stools. Duval and Schorer (1903) reported the finding of dysentery bacilli in 94 per cent. of summer diarrhoea cases, including all grades of severity. They did not believe that the character of the stools was a certain indication of the presence of dysentery bacilli, but they admitted that these bacilli were most likely to be present in cases accompanied by bloody mucus. In opposition to Wollstein they also isolated dysentery bacilli from the stools of normal infants, but gave no figures in support of their statement.

Although normal children living amongst infected cases might quite conceivably harbour dysentery bacilli, we must also reckon with the possibility that these dysentery bacilli from normal infants may not have been true dysentery bacilli, but paradysentery bacilli similar to those occasionally got by Morgan, and to be later referred to. Other American epidemics associated with dysentery bacilli were reported by Cordes, Weaver, Tunncliffe, and others. It may be noted that Cordes got a positive agglutination reaction (1 in 40 to 1 in 50) with the sera of 22 per cent. of his cases.

That the dysenteric cases of summer diarrhœa occurring in America are due to infection by bacilli of the dysentery group is thoroughly established. Only scanty attention, however, has been paid to the bacteriology of the non-dysenteric cases (so-called cholera infantum) similar to those occurring in this country. Park, Collins and Goodwin examined some cases of cholera infantum, but failed to isolate dysentery bacilli. They also failed to recover the dysentery bacillus from cases of subacute diarrhœa in institutions where no cases of the dysentery type existed. Also, the blood-serum of cases of cholera infantum has no agglutinating effect on dysentery bacilli in dilutions higher than 1 in 10.

Schwarz confined his attention to cases of typical summer complaint which were characterized by profuse watery, green stools with little or no mucus. Dysentery bacilli were not recovered from any one of the thirty such cases examined, and the serum reactions were all negative.

In 1905 Jehle and Charleton, in Vienna, examined numerous cases of acute gastro-enteritis (cholera infantum) during the hot weather, but never found dysentery bacilli. Agglutination tests also proved negative.

In this survey we have thought fit to mention only those bacteriological investigations on summer diarrhœa which deserve attention, from the fact that they have been made on an extensive scale and scientifically worked out. Isolated observations on single cases or small groups of cases are of little value, and will not be referred to.

#### SUMMER DIARRHŒA IN LONDON.

We shall now proceed to an analysis of the results obtained by Morgan and others in the bacteriological investigation of summer diarrhœa in London during the four years 1905-1908. In these researches chief attention has been paid to that group of the intestinal bacteria which does not ferment lactose. To this group all the pathogenic bacteria associated with affections of the intestinal tract belong—viz., the typhoid and paratyphoid bacilli, the organisms of the food-poisoning group and the dysentery group.

Reports of Morgan's work during the epidemic seasons of 1905 and 1906 have already been published, but it will be convenient to discuss the results of these two years with those of 1907 and 1908, when researches on an extensive scale were carried out, demanding the labour of many laboratory workers and the kindly co-operation of hospital physicians and health authorities.

#### SOURCE AND AMOUNT OF MATERIAL EXAMINED DURING THE FOUR YEARS.

1905: The fæces of 58 cases of infantile diarrhœa under treatment at the Hospital for Sick Children, Great Ormond Street, and the Victoria Hospital, Chelsea, were examined bacteriologically, along with any available post-mortem material. Of these 58 cases the clinical diagnosis in 28 was acute infective diarrhœa, and in 30 catarrhal diarrhœa, but the stools in the latter cases did not differ appreciably in appearance from those of the acute cases.

1906: During the epidemic season the fæces and autopsy material of 54 cases from the special ward set apart for diarrhœa at the Hospital for Sick Children, Great Ormond Street, were investigated.

1907: Preparations were made this year for a very extensive investigation of the subject, but, unfortunately for the purposes of research, very few cases occurred, and these, as the clinicians agreed, were atypical and of only moderate severity. The material came from Paddington Green, Shadwell, Great Ormond Street, and Victoria Hospitals; 191 cases in all were examined, both in-patients and out-patients. Material from ten autopsies came also under review. Many of the cases were examined repeatedly.

1908: One hundred and sixty-six selected cases of summer diarrhœa occurring at Shadwell and Victoria Hospitals were investigated. When possible the stools of each patient were taken for examination on three consecutive days, and in the event of the patient's death complete bacteriological examination of the organs was made. Material from thirty-three autopsies was available.

#### BRIEF DESCRIPTION OF TECHNIQUE.

Whatever the nature of the material, the technique followed remained essentially the same. In the case of fæces, intestinal scrapings at autopsies, portions of various organs, &c., an emulsion of the material was made in sterile broth. A loopful of the emulsion was then spread

over three plates containing MacConkey's bile-salt-lactose neutral red agar. The colourless (or non-lactose) colonies which developed were picked off and submitted to a series of biological and chemical tests—viz., indol-formation, liquefaction of gelatin, motility and fermentation reactions on various sugar media. In 1908 mannite-bile-salt agar was employed for reasons stated below (*see* Tables I and II), pp. 148 and 149.

The annexed table (Table I) shows all the varieties of non-lactose, non-liquefying micro-organisms met with in the fæces of children suffering from summer diarrhœa, and the percentages in which they occur. The biological characters of these micro-organisms have been completely worked out, and they have been classified according to their affinities to well-known pathogenic members of this group. During the years 1905, 1906, 1907, the exact percentages of these micro-organisms were ascertained, but during 1908, owing to the employment of a modified medium (mannite-bile-salt agar), which was justified by previous experience of the predominance of Morgan's bacillus, only the non-mannite group, to which this bacillus belongs, came under review. We do not propose to discuss in detail all these various micro-organisms, many of which may be familiar to those experienced in the bacteriological examination of excreta. Some special points, however, deserve attention.

In normal fæces the non-lactose group lags in frequency far behind the lactose-fermenting group, of which the *Bacillus coli communis* may be taken as a type. During the investigation of fæces from series of normal persons, adult and infantile, one finds colourless or non-lactose colonies only in a small proportion of the cases. The predominant group, at least in normal stools, is that of the lactose fermenters.

In diarrhœal cases, on the other hand, the lactose-fermenting group is still met with, but in less marked frequency; while the non-lactose varieties relatively increase, and may even in rare cases lead to the complete exclusion of the coliform group.

It will be seen, from the columns giving the percentages of the various non-lactose fermenters met with in the four years' work, that the bacillus designated No. 1, which will be hereafter referred to as Morgan's bacillus, predominates conspicuously over all the other varieties. The group to which it belongs—*i.e.*, the non-mannite, non-liquefying group—is an exceedingly small one. The only known pathogenic member of this group is the dysentery bacillus of Shiga, which, of course, is readily distinguishable from Morgan's bacillus by at least three important characteristics—viz., absence of motility and absence of indol-formation and gas-production.

The second group possesses some affinities to the Flexner type of the dysentery bacillus and to the *Bacillus typhosus*—at any rate in so far as the fermentation reactions on glucose and mannite are concerned—but their reactions on litmus-milk, and their properties with regard to indol-formation, serve to differentiate them readily.

(Table II shows the reactions of the well-known pathogenic members of the non-lactose group.)

The Bacillus No. 3, unlike Flexner's, has no action on litmus-milk, and it produces acid in sorbite, which the Flexner bacillus never does. From the small percentages in which these paradysentery bacilli occur, there is no evidence that dysentery or pseudodysentery bacilli have any significance in the etiology of summer diarrhœa, as it is met with in London.

The Gaertner group, of which there are several varieties (judging by fermentation reactions alone), is headed by the true *Bacillus enteritidis* of Gaertner (isolated once in 1905, four times in 1906, and three times in 1907).

The remaining groups require little attention. Groups X, XII, XIV, and XV are common frequenters of normal and adult stools, and Group X is often isolated from drinking water. Group XV, a variety which does not possess any action on sugars, corresponds to the *Bacillus fœcalis alkaligenes* of Petruschky.

#### OBSERVATIONS ON THE EPIDEMIC SEASONS.

It will be apparent that Morgan's bacillus occupies a predominant place among the non-lactose fermenters in epidemic diarrhœa. The fact that this bacillus was so conspicuous in the years 1905-6 led to the extensive investigations of 1907-8. So far as mere association of the bacillus with the disease is concerned, this point was proved absolutely, as the figures show. The epidemic of 1907 deserves special mention. The research in that year extended from the first week of August to the first week of October. During the first three weeks only six cases were examined, and Morgan's bacillus was not isolated. It was not till the ninth week of the research (last week of September) that Morgan's bacillus began to be isolated with frequency from the cases examined. The percentage of success in that week reached 56·5. An interesting point in this connexion is the fact that the mortality curve for summer diarrhœa in London (1907) gradually rose to a maximum during the last week of September, whereas for the preceding five years the average

mortality curve showed a maximum rise in August. Clinicians were also agreed that this last week of September was the only true epidemic period in the summer of 1907.

#### EXAMINATION OF AUTOPSY MATERIAL.

In 1906 Morgan's bacillus was isolated from the intestinal tract in three cases, and once from the mesenteric glands. It was never isolated from the spleen in 1906. (N.B.—Invasion of the spleen occurred in feeding experiments on young rats and young rabbits, but not in monkeys.) In 1907 material was obtained from ten autopsies. In two only of these cases had Morgan's bacillus been found in the *fæces intra vitam*, and it was in one of these two that Morgan's bacillus was recovered from the spleen. The others were negative. In 1908 Morgan's bacillus was recovered in sixteen autopsies out of thirty-three. It may be convenient to detail the sites in which the bacillus was found: jejunum once, colon thirteen times, liver twice, spleen, kidney and mesenteric glands three times, lung once, bile twice, urine three times, and heart blood three times. In some cases, therefore, it would appear that Morgan's bacillus may give rise to a septicæmia.

#### EXAMINATION OF NORMAL FÆCES OF ADULTS AND CHILDREN.

Throughout all four seasons the possibility of the presence of Morgan's bacillus in the *fæces* of other than diarrhœal cases has been borne in mind, and numerous control series have been investigated. The results in these cases may be briefly summarized. In 1905, during the winter, 20 normal children were examined, and Morgan's bacillus was found in one case. In 1908, during the epidemic season, 99 normal children were examined and the bacillus was isolated twelve times (12 per cent.). Of these cases, 60 were examined by Dr. Eyre and Dr. Minett at Guy's Hospital during the season of 1908, and four positive cases were found (= 6.6 per cent.). The remaining 39 cases came from the Paddington district, and of these eight harboured Morgan's bacillus (= 20 per cent.). Adults: 90 cases were examined during the winter of 1907-8, and the bacillus was obtained in one case only; 33 cases were examined in the spring of 1908, and the bacillus was recovered once.



## CHILDREN DYING FROM OTHER DISEASES.

During the winter 1907-8 post-mortem material from 30 cases was examined, and Morgan's bacillus was recovered in four. The cause of death was broncho-pneumonia, and two of these four had had diarrhoea.

CHILDREN SUFFERING FROM OTHER DISEASES, WITH DIARRHŒA AS  
A COMPLICATION.

In the season of 1908 22 such cases were examined, and the bacillus was recovered in two cases. Morgan's bacillus is therefore met with in a small percentage of cases of healthy children and of children suffering from other diseases complicated by diarrhoea. Adults only very rarely harbour the bacillus. It is to be noted, also, that the percentage of carrier cases is less during the winter season than during the epidemic season, when, it may be presumed, facility of contact is much greater.

EXAMINATION OF MILK, FLIES AND OTHER MATERIAL WHICH MAY ACT  
AS VEHICLES OF INFECTION.

*Milk.*—In view of the fact that milk has been considered by many observers to be the vehicle for the transference of the infection of summer diarrhoea to children, attempts have frequently been made on an extensive scale to isolate Morgan's bacillus from milk samples. In the beginning of the season 1907 samples of milk from feeding-bottles, swabs from rubber teats and comforters were examined, but negative results were invariably obtained. During the season 1908 samples of milk from twenty-nine infected houses in Paddington and from twenty-six uninfected houses in the same district were examined, but only in one instance (from an infected house) was Morgan's bacillus isolated. Such a result was puzzling, especially when one considered the prevailing view of the transference of infection by milk. Some laboratory experiments made in this connexion throw a little light on this matter. When unsterilized milk was inoculated with Morgan's bacillus and incubated at room temperature for six, twelve, twenty-four hours, it was impossible to recover the bacillus by the usual methods. In sterilized milk the bacillus grew readily and was easily recovered. Similar experiments with condensed milks have not so far been made.

*Flies.*—A good deal of attention has been paid within the last two years to the conveyance of infection by the common house-fly. The matter was considered in detail by the London County Council in 1907 and reported on by Dr. Hamer in 1908. He came to the conclusion that flies could not be the sole agents for the conveyance of summer diarrhœa from patient to patient because flies were found in houses in the late autumn after the epidemic had ceased. It must be remembered, however, that, with the onset of cold weather, flies become much more sluggish in their movements and would therefore be less likely to spread infection rapidly. Batches of flies came for examination from infected and uninfected houses in Paddington and from a country house situated many miles from London, where no cases of diarrhœa had occurred, at any rate within a radius of two miles. The flies were killed with ether vapour and crushed with a sterile rod in peptone broth. The result was that Morgan's bacillus was isolated from nine of the thirty-six batches from infected houses and from one of the thirty-two batches from uninfected houses. It was also got in five out of twenty-four batches from the country house.

*Dust.*—Samples of dust (from ashbins) collected from eleven infected houses in Paddington were examined, with negative results.

*Cow and Horse Fæces.*—Morgan's bacillus was isolated once from fresh cow's fæces (eighteen animals examined). It has not been recovered from the fæces of the horse (thirteen animals examined).

#### PATHOGENICITY OF MORGAN'S BACILLUS FOR LABORATORY ANIMALS.

In 1905 feeding experiments were carried out with young rats and young rabbits. Death took place within twenty-four hours and was preceded by violent diarrhœa. The bacillus was recovered from the spleen and sometimes from the heart blood.

In 1906 Morgan's bacillus was again virulent for rats by feeding. Four small but full-grown monkeys received with their food one agar tube each of Morgan's bacillus. All four died after a period of diarrhœa which came on from two to twelve days after feeding. The diarrhœa was an acute and progressive one and was followed by rapid emaciation and death. No vomiting was observed.

The strains of Morgan's bacillus isolated during 1907 and 1908 were, for some reason still unexplained, much less virulent for experimental animals by feeding than those recovered in previous years. The monkeys

which were fed with Morgan's bacillus in 1908 developed diarrhoea which lasted seven to eight days, but all ultimately recovered.

#### TOXIN PRODUCTION.

This subject will be only very briefly referred to. Filtrates of broth cultures of Morgan's bacillus grown for one month are toxic for the guinea-pig, the minimum lethal dose by intraperitoneal injection being 5 c.c. The toxin can be concentrated by precipitation, so that the minimum lethal dose becomes 0.5 c.c. to 1 c.c. Great difficulties have been experienced in the immunization of the horse and donkey with Morgan's bacillus, but a serum has been obtained which possesses marked antitoxic properties.

#### AGGLUTINATION REACTIONS WITH THE SERA OF PATIENTS.

During the first season, 1905, forty-four samples of serum were tested against known micro-organisms like the *Bacillus typhosus* and the *Bacillus enteritidis* of Gaertner. In two cases only was a reaction obtained (1 in 60) with these micro-organisms. Also one case in which Morgan's bacillus was present gave a reaction (1 in 30) with the *Bacillus typhosus*.

In 1908 this question was attacked in a more extensive fashion. Samples of serum were taken at various stages of the disease and during convalescence. Each specimen was tested against various strains of Morgan's bacillus, including the homologous strain when possible, and also against two or three strains of the *Bacillus proteus vulgaris*. Sixty-five patients in all were examined, and thirty of these gave a positive reaction with Morgan's bacillus. One gave a reaction with the *Bacillus proteus vulgaris*. Of twelve healthy control children only one gave a slight reaction (1 in 10) with Morgan's bacillus. The dilution in which a reaction was obtained never exceeded 1 in 40, and as a rule 1 in 20 was the limit. The best results were obtained at or near convalescence. Of nineteen patients at this stage fifteen gave a positive reaction (78.9 per cent.). Of twenty-seven sera taken within a few days of admission only five gave a positive reaction. At this early stage agglutinin-development is, as one might expect, slight. In a few instances where repeated samples of blood were taken during the progress of the disease, it was noticed that agglutination occurred in

gradually higher dilutions as convalescence approached, and in gradually decreasing dilutions after the child had left hospital till a reaction was no longer obtained. The longest period after convalescence at which a reaction appeared was thirty days, and in one case it had disappeared thirty-seven days after convalescence. Of the sixty-five patients examined, Morgan's bacillus was recovered in forty-two, and of these forty-two cases a reaction was obtained in twenty-four. Of the remaining twenty-three cases from which Morgan's bacillus was not isolated six only gave a reaction. It should be noted that those cases which gave no reaction with the homologous strains frequently agglutinated stock strains of the bacillus, and vice versa.

#### THE QUESTION OF A FILTER-PASSER AS THE INFECTIVE AGENT.

During the season 1908 some preliminary experiments were performed with the view of testing this hypothesis. The blood and organs from three typical fatal cases of summer diarrhœa were mashed up in salt solution and passed through a Berkefeld filter. With this material feeding experiments in young cats and inoculation experiments in rats were made. The results were invariably negative.

#### GENERAL SUMMARY AND CONCLUSIONS.

We have, in the above statement, attempted to give a brief sketch of the very laborious researches which Morgan and other workers have carried out during the last four years on the bacteriology of summer diarrhœa. The necessity for making a careful research in the non-lactose group for some specific micro-organism in this disease was, as we have already indicated, justified by the knowledge which has accumulated concerning typhoid, paratyphoid, dysentery and food-poisoning bacilli. The outcome of this search has been that a certain bacillus (Morgan's bacillus) has been proved to occupy a predominant position among the non-lactose fermenters in the excreta of summer diarrhœa patients. In selected cases of the disease it has been isolated in the very high percentage of 63—a percentage which compares very favourably with that obtained—*e.g.*, in typhoid. Further, in the course of this work a large mass of information has been obtained concerning a very important group of the intestinal flora, and this, it is hoped, will prove of great value to future workers on these lines. To one searching for

well-known members of the pathogenic non-lactose group in the excreta, the convenience of having at one's disposal such a table as that shown you for constant reference is obvious. The association of Morgan's bacillus with summer diarrhoea has, we think, been abundantly established. Mere association, however, does not necessarily prove etiological connexion, though it must form a very important link in any chain of evidence connecting a bacillus with an infection. The fact that this bacillus is found in a small percentage of healthy children both during and apart from the epidemic season, and in a still smaller percentage of adults, does not conflict with present-day notions regarding the natural history of pathogenic micro-organisms affecting the intestinal tract. That acute diarrhoea of the summer type may also occur sporadically during the winter season is well known, and it seems quite reasonable to attribute such cases to contact infection from carriers. Extended observations on the duration of the bacillus in the intestine of convalescents are at present being made, and they are expected to yield valuable results.

With regard to the pathogenicity of Morgan's bacillus for experimental animals, we have found that rats and monkeys are susceptible to infection by feeding and, after a period of diarrhoea, succumb. Some strains of the bacillus appear to be much less virulent than others in this respect. Whether the virulence of the strain can be correlated with the severity of the epidemic, we are not yet in a position to decide. By experimental inoculation, both the bacillus and its toxin are markedly pathogenic for rats, guinea-pigs and goats.

It is, however, on the score of association, pathogenicity by feeding and otherwise (especially the results with monkeys), and the serum-reactions on the part of the infected hosts, that the claim of Morgan's bacillus to be considered an important etiological factor in summer diarrhoea mainly rests. Whether that claim will ultimately find general acceptance it is not for us to predict. We are fully alive to the many problems which its habitats outside the infected host suggest. At any rate, this bacillus is in no danger of dying out for want of an animal host to carry it on from one season to another.

The presence of the bacillus in flies from infected houses has an importance which is not necessarily lessened by the fact that flies, in uninfected houses of the same district, may also harbour the bacillus. The intercourse that may occur between flies belonging to the same district is a question on which one cannot dogmatize.

We are at present unable to offer any explanation of the positive

results obtained in flies from the country, though there is some evidence that Morgan's bacillus may be found in the bovine intestine. If this proves to be the case, the question of milk infection will assume another phase.

From the actual bacteriological results obtained, we are inclined to the view that during the epidemic season direct infection by contact with carriers or infected flies may play as important a role as infection through the medium of the milk. Sporadic cases in the winter can only be explained on a carrier hypothesis.

In conclusion, we desire to thank all those physicians, medical officers of health, nurses and sanitary inspectors whose generous co-operation and interest made these researches possible.

Our special thanks are due to Dr. Dudfield, Medical Officer of Health of Paddington, for the very valuable assistance he has given us in the collection of specimens.

It may be mentioned that a detailed report of the bacteriological work in connexion with the investigation of summer diarrhoea will appear later in the *Journal of Hygiene*.

Dr. MORGAN: I have practically nothing to add to Dr. Ledingham's and my joint communication, for although it is only a sketch of the results of four years' work I think it covers nearly all the ground. As regards the association of Morgan's bacillus with the disease, we have found that it is isolated much more frequently from severe cases of summer diarrhoea than it is from mild ones. This was shown by the low percentage in which it was isolated in 1907, when the epidemic was of a mild type. It is still more forcibly shown by the figures obtained during 1908: Seventy-four severe cases were examined from Shadwell Hospital with a mortality of 45·9 per cent., and from these Morgan's bacillus was isolated in 63·5 per cent., whereas from the fifty mild cases from Paddington, of whom none died, this bacillus was only isolated in 34 per cent. Of course, there still remains a great deal of work to be done in relation to this bacillus; at present we are investigating the "carrier" question—in other words, we are examining the stools of the children from whom we isolated Morgan's bacillus last summer and who recovered, to see whether the bacillus is still present; we are also trying to find the best method of producing an efficient antitoxin. The main object, however—that of stamping out the disease—can only be effected by the co-operation of the various sanitary authorities, and it is to them we look for help, not without confidence.

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TABLE I.—BACILLI THAT NEITHER FERMENT LACTOSE NOR LIQUEFY GELATINE.

Bacilli	Morphology	Glucose	Mannite	Dulcicite	Lactose	Cane sugar	LITMUS-MILK			Indol	Sorbitol	Gelatine	PERCENTAGE IN			
							First day	Third day	Fifteenth day				1905	1906	1907	1908
Type I	1	M.B.	A.G.	—	—	—	O.	O.	Alk.S.	+	—	—	48.2	55.8	16.2	53.0
	1A	N.M.B.	A.G.	—	—	—	O.	O.	Alk.S.	+	—	—	0.0	0.0	0.5	
	2	N.M.B.	A.G.	—	—	—	O.	O.	Alk.S.	—	—	—	5.6	0.0	4.7	
Type Flexner	3	N.M.B.	A.	A.	—	—	O.	O.	O.	+	A.	—	8.6	8.8	5.2	
	4	N.M.B.	A.	A.	—	—	O.	O.	O.	—	—	—	3.4	2.9	1.05	
	4A	N.M.B.	A.	A.	—	—	A.	A.	A.C.	—	—	—	0.0	14.7	6.8	
	4B	M.B.	A.	A.	—	—	A.	O.	Alk.	+	—	—	0.0	2.9	0.0	
	4C	M.B.	A.	A.	—	—	A.	O.	Alk.	—	—	—	0.0	2.9	0.0	
Type V	5	M.B.	A.	—	—	—	O.	O.	Alk.S.	+	—	—	6.9	11.7	8.3	
	5A	N.M.B.	A.	—	—	—	O.	O.	Alk.S.	+	—	—	0.0	0.0	3.6	
	5B	M.B.	A.	—	—	—	A.	Alk.S.	Alk.	+	—	—	0.0	0.0	0.5	
Type Gaertner	6	M.B.	A.G.	A.G.	—	—	A.	O.	Alk.	—	A.G.	—	1.7	11.7	1.6	
	7	M.B.	A.G.	A.G.	—	—	A.	Alk.	Alk.	+	—	—	3.4	11.7	1.6	
	7A	N.M.B.	A.G.	A.G.	—	—	A.	O.	A.C.	+	—	—	0.0	2.9	2.6	
	7B	N.M.B.	A.G.	A.G.	—	—	A.	O.	Alk.	+	—	—	0.0	0.0	5.7	
	7C	M.B.	A.G.	A.G.	—	—	O.	A.C.	A.C.	+	—	—	0.0	0.0	3.6	
	8	N.M.B.	A.G.	A.G.	—	—	A.	O.	A.	—	—	—	1.7	0.0	0.0	
	9	N.M.B.	A.G.	A.G.	—	—	A.	A.	Alk.	—	—	—	1.7	0.0	0.0	
	10	N.M.B.	A.G.	A.G.	—	—	A.G.	A.	A.C.	+	—	—	6.9	2.9	0.0	
	11	M.B.	A.G.	A.G.	—	—	A.G.	O.	Alk.	+	—	—	5.1	5.8	3.6	
Type X	11A	M.B.	A.G.	A.G.	—	—	A.	A.	Alk.	+	—	—	0.0	8.8	0.0	
	11B	M.B.	A.G.	A.G.	—	—	A.	A.	A.C.	+	—	—	0.0	2.9	0.0	
	11C	N.M.B.	A.G.	A.G.	—	—	A.G.	O.	Alk.	+	—	—	0.0	0.0	4.2	
Type XII	12	M.B.	A.G.	A.G.	—	—	A.	A.	A.C.	—	—	—	3.4	0.0	0.0	
	13	M.B.	A.G.	A.G.	—	—	A.	A.	A.C.	+	—	—	1.7	8.8	0.0	
	13A	N.M.B.	A.G.	A.G.	—	—	A.G.	A.	A.C.	+	—	—	0.0	5.8	0.0	



TABLE II.—SOME KNOWN PATHOGENIC INTESTINAL BACTERIA.

Type	Bacillus	Morphology	Glucose	Mannite	Dulcite	Lactose	Cane sugar	LITMUS-MILK			Indol	Sorbitol	Gela- tine
								First day	Third day	Fifteenth day			
Type XIV	14A M.B.	A.G.	-	A.	A.	Alk.S.	+	-	-	8.6	2.9	1.05	
	14B N.M.B.	A.G.	-	A.	Alk.	Alk.	+	-	-	0.0	8.8	3.2	
	14C M.B.	A.G.	-	A.	A.	A.C.	+	-	-	0.0	8.8	2.1	
	14D M.B.	A.G.	-	A.	O.	A.C.	+	-	-	0.0	5.8	1.05	
Type XV	15 N.M.B.	-	-	Alk.S.	Alk.S.	Alk.	-	-	-	?	?	0.5	
	15A M.B.	-	-	Alk.	Alk.	Alk.	-	-	-	?	?	1.6	
	15B N.M.B.	-	-	O.	O.	O.	-	-	-	?	?	2.1	
	<i>Bacillus dysenteriae</i> (Flexner, Philip- pines) ...	N.M.B.	A.	A.	-	-	-	-	-	Alk.S.	+	-	
	<i>Bacillus dysenteriae</i> (Shiga) ...	N.M.B.	A.	-	-	-	-	-	-	Alk.S.	-	-	
	<i>Bacillus typhosus</i> ...	M.B.	A.	A.	-	-	-	-	-	A.	-	A.	
	<i>Bacillus enteritidis</i> (Gaertner) ...	M.B.	A.G.	A.G.	A.G.	-	-	-	-	Alk.	-	A.G.	
	<i>Bacillus paratyphosus</i> A (Schot- müller) ...	M.B.	A.G.	A.G.	A.G.	-	-	-	-	A.	-	A.G.	
	<i>Bacillus paratyphosus</i> B (Schot- müller) ...	M.B.	A.G.	A.G.	A.G.	-	-	-	-	Alk.	-	A.G.	
	<i>Bacillus hog cholera</i> (McFadyean) ...	M.B.	A.G.	-	-	-	-	-	-	A.	-	-	

M.B. = Motile bacillus. N.M.B. = Non-motile bacillus. A. = Acid. Alk. = Alkaline. S. = Slight. G. = Gas.

## DISCUSSION.

The PRESIDENT (Dr. Newsholme) conveyed the thanks of the Section to the authors for their valuable paper. After the last meeting it required some courage to present such a paper, but their courage was justified by results, for the paper had brought them nearer a bacteriological solution of the problem of epidemic diarrhoea. Morgan's bacillus seemed to be likely to have a future before it. The authors of the paper showed great wisdom in realizing the importance of fitting together evidence from the bacteriological and epidemiological points of view, and from both these there was ample material for a good discussion.

Dr. NASH sent the following contribution, which was read by Dr. Hamer: Dr. Morgan and his coadjutors have made out a good case for the bacillus first isolated and fully described by Morgan as one of the organisms probably responsible for a large percentage of the cases of summer diarrhoea. If the definition of epidemic diarrhoea is to be limited to cases of diarrhoea where the stools have certain well-defined characteristics they might even have grounds for claiming that Morgan's bacillus is the organism which causes that form of diarrhoea. To my mind the paper contributed by Dr. Morgan and Dr. Ledingham adds additional support to the views I hold, and which I have urged since the year 1902 on many different occasions in various contributions to the medical Press, as well as in special reports on epidemic diarrhoea and in annual health reports. Briefly, I urge two main points: (1) that there is no one and only bacterial organism responsible for *all* cases of epidemic diarrhoea. In my opinion the diarrhoea-causing organism in one locality may differ in some small degree or "in toto" from the diarrhoea-causing organism in another locality. Further, I hold that ordinary putrefactive bacteria which, taken in smaller numbers in colder weather, are neither active enough nor numerous enough to be pathogenic, on the other hand in warm weather multiply rapidly, and withal become more active and produce larger quantities and more powerful toxins, and hence tend to become pathogenic in summer. Even Morgan's bacillus, though found in a larger percentage of cases examined than any other one particular bacillus, was *not* found in nearly 40 per cent. of the cases so carefully investigated. (2) I have held on the grounds of careful observation, epidemiological investigation, by process of exclusion, and by the doctrine of probabilities, that flies—more especially house flies—are among the main agents or factors in the spread of epidemic diarrhoea in warm weather. I venture to think the facts adduced in the paper support that doctrine also. To my mind it is so obvious a mode of spread—if we believe in bacterial infection at all—that the scientific proof of flies carrying the organisms is almost an insult to common sense, and may be likened almost to proving that the sun is the source of daylight. Only those who have worked at the isolation, cultivation and study of bacteria can fully realize the arduous labours which the authors of the paper

have undertaken, and I would only wish in conclusion to congratulate them on having achieved so much good and useful work.

Dr. RANSOME expressed his high appreciation of such a valuable paper. It showed not only great labour, but extreme candour. He did not notice that anything was said about the natural history of the bacillus held to be the cause of diarrhoea; more especially, no mention had been made of facts which would account for the appearance of the disease after a certain temperature had been reached. The prevalence of a mean temperature of 60° F. for a week was the point which seemed to start an epidemic. Had the authors' experiments with the bacillus enabled them to account for that fact? Flies would only partially explain the temperature coincidence.

Dr. TWORT said he desired to speak of the diagnosis of so-called new organisms by minute differences in their fermentative action on various sugars. The chief difference given on the chart between Morgan's bacillus and the dysentery bacillus of Kruse, so far as its fermentative action on the sugars was concerned, was that it produced acid and gas in glucose instead of acid only. Going down the list of bacilli and their reactions which was exhibited, one found a large number of small reaction variations, and he did not think there was any hard and fast line of demarcation between the various members of that group. The artificial line of demarcation employed resulted in groups and subgroups; and the latter were subdivided into numerous varieties, so that the typhoid were subdivided into typhoid and paratyphoid, the paratyphoid into paratyphoid A and B and so on. He believed that no one was justified in asserting that a certain organism did ferment a particular sugar and another did not; it was purely a matter of degree. When tested, variations were found which were left out in any chart; the reaction was simply put down as acid, + or -, as indicated by litmus, the quantity of acid produced, the time taken, and the various amounts of alkaline products which at the same time were being formed by the breaking up of the peptone varied with each organism tested, and also with the exact composition of the medium used, yet all these points seemed to be ignored. It was well known that the quantity of alkaline products produced might mask a slight acid reaction and so entirely alter the chart grouping of the organism. There appeared to him to be no scientific reason why, for instance, an organism showing marked acidity in glucose, after some days' growth, should be placed in the same group as an organism showing marked acidity in twenty-four hours, while at the same time they refused to admit into that group another organism producing a slightly less quantity of acid which might be neutralized by the alkaline products of the peptone; or, on the other hand, for refusing to admit into the group another organism which might easily be made to acquire the power of fermenting the sugar. It was probable that in many cases the difference was just as great between vigorous and slight acid producers as between slight acid producers detected by litmus solution and those in which a still less quantity of acid was formed which was masked by the alkaline products of the peptone. He had found that an organism which was supposed not to ferment a particular sugar acquired the power to do so

if it were cultivated for a sufficient number of generations on that particular sugar. The line of demarcation shown in the charts was an absolutely artificial one. If the sugars were replaced by various other substances the whole of the grouping shown on the chart would probably be altered. He had found, for instance, that by using glucosides instead of the usual sugars some of the organisms of the Friedländer group came out of that group and entered another group by themselves; at the same time other new groups were formed which included organisms belonging to several of the existing groups. There was no logical justification for choosing one particular substance and saying that should be the medium for the differentiation of bacilli. He maintained that the particular sugars used were used because they were easily obtainable and not from any scientific standpoint. Change the substances tested and probably the so-called Morgan's bacillus would show a large number of varieties, change the substances again and they might give reactions similar to the dysentery bacillus of Kruse. In testing the organisms of the Friedländer group he did not think there were any two organisms which showed exactly the same characters. He had done some work in connexion with the power of organisms to ferment sugars. He grew a particular organism on a sugar which it would not ferment, to see if, by subculturing it for a sufficient number of generations, it could be made to acquire the power to ferment that sugar. He found that the typhoid and dysentery organisms acquired the power to ferment lactose with the production of acid and still showed considerable agglutination with their respective agglutinating sera. It was probable that any organism could lose as well as gain characters. He believed that any particular organism, when grown outside the body, would gradually acquire the power of fermenting sugars and other carbohydrates; that it gained the power of splitting up and utilizing simple products, when this would help it to survive, and when its energies were not required to act against the tissues and products of the human or animal body. He believed that an organism would become pathogenic far more rapidly than it would lose its fermentative properties. It was well known that the virulence of some colon bacilli, as estimated by dosage, could be increased enormously in a very short time by passage through the body of a guinea-pig for about twelve or eighteen generations, but the fermentative powers of such bacilli would not change in so short a time; from this it could be understood that if an organism supposed to be non-pathogenic were suddenly thrown on to an individual who was feebly resistant, and other conditions were favourable to its multiplication, then that organism might cause a low type of diarrhoea or dysentery. He maintained, further, that having once obtained a hold it would probably gain virulence, and if now its environment were such as to favour its survival and dissemination it would probably attack other members of the community, and so give rise to a small epidemic. This organism might belong to any of the fermentation groups, and since its fermentations would be modified slightly after passing for a number of generations through the human body, certain bacteriologists investigating the outbreak would describe it as a new organism; if, on the other hand,

a non-fermenting organism cast off from the human body grew for a large number of generations outside the body, then certain fermentative powers would be acquired, as had been proved to take place in the laboratory under certain circumstances; if, again, this organism were to pass through the human body and give rise to an epidemic of diarrhœa or dysentery, again it would be described as a new species of dysentery. He maintained that if every micro-organism of the colon group showing slight differences in fermentative powers was going to be described as a new species, then the time would come when the so-called Morgan's bacillus would be tabulated into hundreds of varieties. First we would have a Para-Morgan, then Para-Morgan A, B, C, &c.; then Para-Morgan A, B, C, 1, 2, or 3, until eventually we arrived at Para-Morgan Z 99; and still the division into varieties and species would go on. He thought agglutination was not an absolute test by which one could say that a particular organism was, or was not, the cause of dysentery. Morgan's bacillus gave an agglutination of 1 in 40, but the serum of that patient, in one or two cases, gave an agglutination for typhoid of 1 in 60. If, then, the serum of a patient could agglutinate a foreign organism in 1 in 60, he failed to see that an agglutination of 1 in 40 to Morgan's bacillus was evidence that the latter was the specific cause of summer diarrhœa. Another point concerned the breaking up of the various proteid substances. It was easy to work on the sugars; one tested either for acid or gas, or both; but there was no such nice way of testing the products of peptone decomposition. He believed that micro-organisms could acquire or lose their particular powers to act on the proteid substances in the same way that they acquired or lost their powers of fermenting sugars. He did not agree that the more delicate organisms, such as typhoid, could not live long outside the human body in material containing more vigorous fermenters—*i.e.*, in such substances as water or soil. He placed the typhoid bacillus in a medium containing dulcete, a substance which it could easily acquire the power to ferment, and inoculated with it another organism—*Bacillus capsulatus*—which, although a vigorous fermenter of most sugars, could not easily acquire the power to ferment dulcete. According to the prevalent views one would have expected the typhoid bacillus to have died off in a few days or weeks. But there it soon acquired the power of fermenting dulcete, and although subcultures were taken from the mixture on to a fresh dulcete tube every two or three weeks the typhoid was found to be active at the end of fifteen months, still growing side by side with the *Bacillus capsulatus*. A short time ago he found gas produced in the particular tubes in which he was growing them, and the question arose as to whether it was the typhoid bacillus which was producing the gas as well as the acid, or whether the *Bacillus capsulatus* had also acquired the power of fermenting dulcete with the production of gas. Therefore he isolated the organisms out, and he was somewhat surprised to find that the typhoid produced acid only in the dulcete, which it had acquired the power of fermenting, while the *Bacillus capsulatus* colonies isolated produced no acid or gas in dulcete, so the question was—Had he failed to isolate the organism which produced the gas, or was it that one of the organisms could only produce gas in

dulcite when mixed with the other? He therefore took those two organisms and mixed them together again in another dulcite tube, and found they showed marked acid and gas production as before. He found the normal typhoid produced no acid in dulcite, or only very slowly after it had been grown outside the body for a long time. He found that the experimental capsulatus mixed with normal typhoid could not produce gas in dulcite; from this it was clear that the *Bacillus capsulatus* could only produce gas in dulcite when acting in combination with the changed *Bacillus typhosus*. It appeared, in fact, to be a case of acquired symbiosis, and if a highly pathogenic micro-organism could, by becoming modified, act in symbiosis with another modified but less pathogenic micro-organism in the laboratory, then there seemed to be no reason why it should not do so when in such situations as water and soil, in which case it might survive for a very long time, and not be killed out as most bacteriologists thought. He thought that if Morgan's bacillus could be proved to be the chief causative organism of summer diarrhœa, then it would be better to describe it as a variety of dysentery. He thought the differentiation of such organisms into different species, relying upon slight differences in fermentative powers, had gone far enough, if not too far, and there was no justification for putting Morgan's bacillus into a new group simply because it produced acid and gas in glucose instead of producing acid only, as is done by the dysentery bacillus.

Dr. COPEMAN congratulated the authors on their laborious research. The difficulty in regard to summer diarrhœa had always been that practically nothing was known of its bacteriology. Many micro-organisms, both pathogenic and those which under ordinary circumstances were not so, had been laid under contribution as the cause, and he wished to ask whether there was a possibility that certain anaerobes and other micro-organisms, for instance moulds, or even higher forms such as amœbæ, had anything to do with this epidemic diarrhœa. He thought the evidence seemed to show that various micro-organisms isolated by Metchnikoff and other observers might, on occasion, have some etiological relationship to the disease. The authors said the association of Morgan's bacillus with the disease had been absolutely proved. His own view was that it was not absolutely proved, because they found it in cases which were not of the epidemic variety, and they did not find it in some cases which, clinically, were that disease. They referred to the association in 63 per cent. of selected cases; but they had not, perhaps, heard Dr. Hamer's crushing indictment of the typhoid bacillus itself at the previous meeting. Mere association did not necessarily mean anything in the etiology of a disease. Few people, if any, now thought the *Micrococcus neoformans* had anything to do with the origin of cancer, yet Dr. Lazarus Barlow said that in the last four years it had been isolated in at least 90 per cent. of the cases of malignant growths at the Middlesex Hospital examined in his laboratory. But with regard to the spread of the disease, the authors had exploded one or two bombs among epidemiologists. Most writers had held that milk was the chief carrier of epidemic enteritis, and the provision of sterilized milk was said to have reduced the infant mortality from the disease. It was extraordinary

to hear that Morgan's bacillus died out in milk which had been kept at room temperature for as short a period as six hours. Was it likely that members of an artisan's family, for instance, would get their milk within that time? If that statement was correct, milk could not be an important carrier of the disease, but he would be surprised if milk really had nothing to do with the matter, since investigation by many skilled observers had apparently shown, very definitely, that epidemic enteritis was less frequent among children who were breast-fed than among those who had been fed with cow's milk or artificial food. The authors had said there was some evidence that Morgan's bacillus might be found in the bovine intestine, and that if such proved to be the case the question of milk infection would assume another phase. But what phase would it assume? Because if the organism could not live in unsterilized milk, how was the milk infection question altered by the sterilization of milk? Possibly the cow might occasionally yield sterile milk, but he had never heard the suggestion that it could yield sterilized milk, and therefore, according to the authors' own showing, its milk could hardly be responsible for infection from Morgan's bacillus derived from its own intestine and carried into the milk during the operation of milking. He had been entrusted by the Local Government Board with an investigation into the possible carriage of infection by flies, and he had had the advantage of the co-operation of Dr. Hamer in connexion with the work carried out by that gentleman for the County Council. He was interested to know that the suggestion he made then commended itself to the present authors—namely, that owing to the fact that flies were more sluggish in the latter portion of their existence, they were less likely to infect either milk or the infant. In Egypt ophthalmia was undoubtedly carried direct from one individual to another by flies, and possibly epidemic enteritis might be carried in similar fashion by the same agents. The question of flies collected from infected houses containing organisms, and flies not containing them from uninfected houses, he did not think of much importance, as the authors had suggested, because the flies from uninfected houses might previously have come from infected houses or direct from dustbins. And the authors did not say whether they thought the carriage of infection by the fly was caused by contagion on the surface of its body or in its intestinal tract. As the investigators, in making their cultivations, broke the flies up in nutrient broth, it might be either. He had found that the colon bacillus would survive for a long time in the fly's intestine, as also, apparently, in its dejecta, and it would be interesting to know whether the same was true of Morgan's bacillus. Except in a very heavy wind, he did not think flies were able to traverse a distance of two miles. In some experiments which Dr. Hamer and he carried out with flies, from a glue factory at Bermondsey, which, owing to their green-coloured body, were easily recognizable, it was found that these flies were not to be identified at a greater distance from that factory than 250 yards. The authors said "The intercourse that may occur between flies belonging to the same district is a question on which we cannot dogmatize." He was tempted to ask whether that intercourse was social or

sexual. No doubt the answers to some of the questions asked would be evolved in the later steps of the research.

Dr. BULLOCH said there had been some iconoclastic suggestions from Dr. Twort. If they had been forthcoming from an amateur they would have been put down as foolish; but under his own observation Dr. Twort had carried out the work, and with extraordinary care, and had submitted the results to bacteriologists in Europe and in this country. Personally, he (Dr. Bulloch) had very slight knowledge of that particular group, and he felt glad that he had not wasted his time in studying things which were beyond him. With regard to bacteria acquiring fermentative qualities, Dr. Twort had recently sent a culture of typhoid which fermented lactose to Dr. Ledingham, and he wished to ask Dr. Ledingham whether he considered the culture which he obtained from Dr. Twort was typhoid or not.

Dr. HAMER said he desired to draw the attention of the Section to an interesting paper on "Summer Diarrhœa," in the last issue of the *Zeitschrift für Hygiene* (1909, lxii, p. 199), by Dr. Liefmann, of Halle. The paper dealt more particularly with the use of sterilized milk. Experience at Halle showed that the children fed on sterilized milk suffered from summer diarrhœa to as large an extent as did the children not so fed. Some interesting statements were incidentally made bearing on the bacteriology of summer diarrhœa. Dr. Liefmann observed that all attempts to demonstrate a causal organism had proved unavailing, and he quoted the dictum of Pfaundler that children sickened *ex alimentatione*, and that they died *ex infectione*. This consideration led him to advert to the importance of secondary invaders, which as he said might be spoken of as "nosoparasites," or, again, there might be applied to them the designation "facultative pathogenic organisms" to distinguish them from "obligate pathogenic organisms." Dr. Liefmann argued that in any case one had to recognize the existence of transitional forms between one group and the other, and germs belonging to one and the other group occurred side by side in "mixed infections." If possibilities such as these had been submitted for their consideration at a meeting of the Epidemiological Section there would doubtless have been manifestations of dissent and expressions of incredulity. One member would have complained that the facts of disease were being made more difficult to understand, and another would have threatened them with the razor of Occam which frightened them at their last meeting. The interest of the utterances was, however, that they came not from a mere epidemiologist, but from an expert bacteriologist, and, further, that they appeared in the *Zeitschrift* of Robert Koch himself.

The PRESIDENT remarked that it was unfortunate that the work of the authors had not yet extended to the subject of condensed milk. Some years ago he found—and the observation had been confirmed by Dr. Richards and others—that in proportion to the total number in each group there was a larger amount of summer diarrhœa among the babies who were fed solely on condensed milk than among those fed on fresh cow's milk. That was



important from an epidemiological point of view, because from the standpoint of the present paper either one must conclude that Morgan's bacillus had remained in the condensed milk, or that it had got into it in domestic life after the opening of the tin of milk. In either case the importance of domestic infection and of domestic cleanliness was largely enhanced, so far as the disease now being discussed was concerned, by this consideration. It would thus be most important that the incidence of Morgan's bacillus in domestic milk or in domestic dirt should be further investigated. It was interesting, but puzzling, to find that Morgan's bacillus was present in flies in the open country.

Dr. LEDINGHAM, in reply, said they realized the importance of investigating condensed milk, but last year they had too much work on hand to do it. They hoped this year to be in a position to ascertain whether Morgan's bacillus would remain alive in condensed milk as well as in sterilized milk. Further experiments with unsterilized milk were also indicated, as there always remained the possibility that Morgan's bacillus was inhibited in its growth and not actually killed by other micro-organisms in the milk. With regard to Ballard's views on the relation of the ground-soil temperature to outbreaks of summer diarrhoea, he wished to point out that this temperature relationship was a very general one so far at least as infections of the intestinal tract were concerned. Dysentery, cholera, and to a less extent typhoid, occurred mostly during the warm season, probably because the organisms causing these affections attained their maximum growth at that period. So far, the bacteriological results did not explain the exact correspondence which had been stated to subsist between the attainment of a certain ground-soil temperature and the outbreak of summer diarrhoea. Dr. Twort's views on the fermentation reactions of intestinal bacteria and the question of mutation were very valuable, though they were not quite relevant to the matter at issue that evening. He had been familiar with these opinions of Dr. Twort for some considerable time, but, after all, the lively speculations in which Dr. Twort had indulged rested on an extremely small basis of actual fact. Whether these organisms could be trained to mutate or not did not affect the main thesis that the fermentation reactions of these organisms as found in nature were remarkably constant. When Dr. Twort had the courage to search in water or elsewhere for the *Bacillus typhosus* disguised as *Bacillus coli* and succeeded in unmasking it, he would have even greater respect for his views than he had now. Dr. Twort had stated his belief that Morgan's bacillus might be a dysentery bacillus. This was comforting, but why it should be a dysentery bacillus rather than a typhoid bacillus or a coli bacillus he (Dr. Ledingham) could not understand. In reply to Dr. Bulloch, he could not yet give a definite answer to the question whether the organism sent him by Dr. Twort was a changed typhoid bacillus having the power of fermenting lactose. It was being investigated by a colleague, and so far it appeared to produce a reaction on lactose, but that was not the whole question. With regard to dulcitate referred to by Dr. Twort, there appeared to be some evidence that the typhoid bacillus had the power

of fermenting dulcete (late reaction) without any special training. Dr. Copeman's remarks on the milk question and the fly question in relation to Morgan's bacillus were very apt. Certainly the fact that they had had so little success in the isolation of Morgan's bacillus from milk samples was surprising, but it had to be remembered that the bactericidal property of fresh milk and the presence of other micro-organisms were factors which they could not yet estimate accurately. As Dr. Copeman indicated, horse-dung and not cow-dung formed the favourite breeding-ground of the domestic fly, and it was in the latter that Morgan's bacillus was found. More extensive investigations on these special points were necessary. So far they could not say whether Morgan's bacillus was present in the intestine or on the exterior of the fly. Dr. Hamer had referred to a recent paper by Liefmann on the conditions of summer diarrhoea in Halle and Leipzig. He (Dr. Ledingham) had also read that paper. It contained no contributions to the bacteriology of the subject. What had impressed him (Dr. Ledingham) most was the author's conclusion that infection was as liable to occur from contact as from milk, and that the mortality among children fed on sterilized milk was probably as great as among those not so fed.