EPIDEMIC DIARRHOEA AND THE BACTERIAL CONTENT OF FOOD.

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SINCE it would hardly be possible to conceive a more complex medium for the conveyance of disease than cows' milk, the solution of the problems connected with the origin and source of epidemic diarrhoea is not materially advanced by the general recognition of the fact that in the majority of fatal cases, cows' milk is the vehicle of infection.

In recent years a number of observers have made exhaustive enquiries into the bacteriological and chemical properties of cows' milk. Park (1901, p. 398) has shown that the number of bacteria in milk varies with the temperature at which it is incubated, and that a sample of milk originally containing 30,000 bacteria per c.c., may attain a bacterial content of 25,000,000,000 after 24 hours' incubation at 35° C. Swithinbank and Newman (1903, p. 130) estimated the bacterial content of milk, after 24 hours' incubation at 15° C., at 8,000,000 per c.c., and Delépine (1903, p. 83), of Manchester, found that the pathogenicity of milk for guinea-pigs varied with the mean temperature in the shade.

The striking results of these experiments naturally suggest the question, Are even these enormous numbers of bacteria in milk actually harmful? To this question Park (1901, p. 402) has replied "that children in cities sicken on the milk supplied in summer, that those put on milk which is sterile or contains only a few bacteria as a rule mend rapidly, while those kept on the impure milk continue ill or die." Though not definitely stated, there is no doubt that the illness referred to as produced by impure milk is summer diarrhoea. Dr Newman (Swithinbank and Newman, p. 372) has pointed out "that stale milk contains toxic properties altogether apart from and in addition to actual bacteria. It is possible that the products of organismal action have a much greater effect in the causation of diarrhoea than is generally supposed." In the same connection Delépine (1903, p. 87) has written as follows, ".....long keeping and high temperature are the two impor-

tant factors which determine whether a sample of infected milk will contain a sufficient quantity of bacteria or bacterial products to produce infection."

It is unnecessary to insist any further on the existence of a widespread belief in a more or less intimate relationship between the high bacterial content of milk sold in large towns and the prevalence of diarrhoea amongst those who consume it. Indeed it is now seriously suggested that a numerical bacteriological standard should be instituted as a criterion of the harmful properties of cows' milk. The question is one of some difficulty, and may perhaps be approached with advantage from different points of view.

The causation of diarrhoea by foods other than natural cows' milk.

There is according to Ballard (1887–8, p. 8) no kind of food which is not liable to become infected with the contagium of diarrhoea. Adult patients commonly attribute attacks of diarrhoea to the consumption of stale fish and "tainted" meat, and there is no doubt these foods are frequently the carriers of infection, whilst condensed milk is an even more potent source of diarrhoea than cows' milk, as has been shown by Newsholme (1902–4) of Brighton in his last three annual reports.

The results obtained in Brighton have been confirmed in the borough of Finsbury, where by the courtesy of the Medical Officer of Health, Dr Newman, I have been permitted to make enquiries into the feeding of 554 healthy infants during the first nine months of life and to utilise the records of the Public Health Department, in order to ascertain the diets of all infants dying from diarrhoea before the age of nine months during the years 1903 and 1904.

The results are shown in the following Table from which it will be seen that infants fed on "condensed milk" are three times more liable to fatal diarrhoea than they should be on the assumption of an average distribution of the disease irrespective of diet, whereas the liability of infants fed on cows' milk is less than two and a half times the average.

TABLE I. *Numbers and percentages of infants under nine months, dying or surviving on different diets.*

	Diet:	Condensed milk	Cows' milk	Artificial foods	Total artificial	Human milk alone	Totals
Deaths from Diarrhoea	Numbers	44	63	10	117	22	139
	Percentages	32	45	7	84	16	100
Survivors	Numbers	56	105	11	172	382	554
	Percentages	10	19	2	31	69	100

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The preparation of condensed milk in use was noted as Nestlé's in about 90 per cent. of the fatal cases both in Finsbury and Brighton, so that we are able to narrow the issue down to a consideration of the properties possessed by this particular brand alone. In addition then to a number of solid foods which produce diarrhoea with a frequency that it is not easy to gauge, we have Nestlé's milk, which in proportion to the number of consumers has been ascertained to be even more frequently associated with diarrhoea than cows' milk.

With regard to the number of organisms contained in different articles of diet, it is generally admitted that cows' milk affords an ideal medium for the multiplication of bacteria, which are diffused throughout a fluid food, not only by their own motility, but also by the oscillations and currents in the fluid itself: in a solid medium on the other hand multiplication and the formation of waste products must of necessity be slower. If on these grounds we assume that the bacterial content of solid food is comparatively small, we find a correspondingly small incidence of diarrhoea on adults who live chiefly on this class of diet, and take little milk. There are, however, reasons for believing that adults are constitutionally less susceptible to diarrhoea than milk-drinking infants, and that their freedom from attack is due to natural immunity rather than to the absence of infective properties in the food they consume. The effects of Nestlé's milk, on the other hand, may justly be attributed to its infective properties, since we are able to judge in this case by the incidence of diarrhoea on a class of consumers of the same age and susceptibility as the consumers of cows' milk.

Thirty-three out of thirty-four brands of condensed milk chosen at random were found by Hope (1898, p. 146), of Liverpool, to contain living bacteria, but the actual number of micro-organisms in preserved milk does not appear to have been estimated by actual experiment. Eustace Smith (1889, p. 638) whilst recommending condensed milk for young infants points out that the milk, although still apparently fresh, rapidly breeds bacteria and becomes unfit for the child's consumption; and Cautley (1903, pp. 285, 286) states that tins of condensed milk that have once been opened are liable to decompose rapidly, especially in hot weather. The brands which are liable to these changes are not specified and I have therefore ventured to give the results of an investigation into the rate of multiplication of bacteria in Nestlé's milk and the liability of this particular kind of condensed milk to rapid decomposition.

Methods.

The experiments of Park (1901, p. 398) and of Swithinbank and Newman (p. 130) with cows' milk were taken as a rough basis for comparison. The contamination of the cows' milk used in these investigations was casual and occurred during the process of milking, and for this reason I decided to expose the Nestlé's milk for 24 hours to casual contamination from the air of the laboratory before making the first estimate of the bacterial content.

The alternative plan of taking the initial content of the milk, decanting it into a flask or keeping it in the tin for the daily estimation of the number of bacteria, could hardly have been carried out without accidental contamination, the amount of which would have varied in each experiment.

Between March 6th and May 15th five experiments were made with different samples of Nestlé's milk purchased from grocers in Finsbury. For each experiment, two large tins were opened by turning the tops back as flaps, and the surface of the milk was left exposed to the air of the laboratory for 24 hours, at a temperature which varied between 68° F. in the afternoon, and 45° F. at night. At the end of 24 hours the contents of the two tins were mixed in a beaker and decanted in equal parts into two flasks, which were then incubated at 22° C. and 37° C. respectively, their mouths being closed with plugs of cotton-wool. All the vessels used had been sterilised, but no attempt was made to avoid contamination from the air during the process of mixture and decanting. The numbers of bacteria in the mixed milk prior to incubation, and in the milk in the flasks on successive days, were estimated by plating measured quantities of known dilutions of the milk and counting the resulting colonies. The medium used in making the plates was agar and contained 2% of peptone, ·5% of sodium chloride and was faintly alkaline to litmus. The minimum dilution employed for the first three days was 1–10 and for subsequent days 1–50.

The 1–10 dilutions were made by transferring 5 c.c. of milk in a wide-mouthed pipette to 45 c.c. of sterile water: for the 1–50 dilution, 4 c.c. of milk were added to 196 c.c. of water. Owing to the viscid nature of the milk, the measured quantity was inevitably increased by a certain amount of milk which adhered to, and was transferred on the outside of the pipette. It was to minimise the error arising in this way

that comparatively large quantities of milk were used in making the primary dilutions. The amount of diluted milk added to each plate was 1 c.c., the plates were incubated at 22° C., and the resulting colonies were counted at the end of four days.

In the following instances the methods that have been described were departed from.

Experiment I. The culture medium employed was gelatin containing 2% of peptone, and early liquefaction in certain plates rendered it necessary to count the colonies on the 3rd and 4th days. The minimum dilution employed in plating the milk on the first three days was 1–1000; no colonies grew and thus the bacterial content for these days was not ascertained. On the 4th and 5th days, 1–50 dilutions of the milk incubated at 20° C. only gave one and three colonies in a plate and the experiment with this portion of the milk was abandoned.

Experiments III and IV. The milk was exposed in the usual way, but was incubated in the open tins and remained liable to contamination from the air throughout the experiments. In this way the conditions obtaining in the home of the consumer were more closely imitated and the milk was given every opportunity to develop a high bacterial content. Till the end of the 8th day of incubation the number of bacteria remained exceptionally low, presumably because bacteria alighting on the surface were prevented, by the viscidity of the milk, from growing down into the deeper layers from which the samples for estimation were taken.

Experiment V. In this experiment the milk was diluted about ten times before incubation. One large tin of milk was used and the surface of the milk was only exposed to the air for four hours; 116 grams of the milk were then added to a litre of sterilised water and thoroughly mixed, the mixture being decanted in equal portions into sterile flasks for incubation at 22° C. and 37° C. Before incubation, 1 c.c. of a 1–10 dilution of the mixture was plated in an agar medium, but no colonies grew, so that the bacterial content on the first day was not ascertained, though it was evidently low. The minimum dilution used in plating the milk after two and three days was 1–500, and after four days 1–5000.

In Experiments I and V the acidity of the mixed milks immediately before incubation, and of the milk in the flasks on successive days, was estimated by titrating measured quantities of milk with a decinormal solution of caustic soda; phenophthalein was used as the indicator and the results have been expressed in cubic centimetres of the titrating solution.

In all the experiments the milk was kept until gross physical changes occurred or, in the absence of any marked change, for periods varying between 12 and 19 days.

RESULTS.

The results are summarised in the following Table:

TABLE II.

The Bacterial Content and Acidity of Nestlé's Milk on successive days.

	Milk Undiluted								Milk diluted 1 : 10 with sterile water			
Experiment No....	I.				II.		III.	IV.	V.			
Temperature at which sample was incubated ..	22° C.		37° C.		22° C.	37° C.	22° C.	37° C.	22° C.		37° C.	
	Bacteria per c.c.	Acidity per 20 c.c.	Bacteria per c.c.	Acidity per 20 c.c.	Bacteria per c.c.	Bacteria per c.c.	Bacteria per c.c.	Bacteria per c.c.	Bacteria per c.c.	Acidity per 200 c.c.	Bacteria per c.c.	Acidity per 200 c.c.
Before incubation	...	11·2	...	11·2	80	80	60	80	...	13·6	...	13·6
After 1 day	...	9·8	...	10·0	80	700	60	80	150	11·4	11,072,000	21·0
,, 2 days	...	10·6	5,000	10·4	60	600	100	200	2,320,000	12·0	85,000	59
,, 3 ,,	50	10·4	19,000	10·2	1250	1900	250	600	152,400,000	24	13,000	64
,, 4 ,,	150	10·3	203,000	10·5	350	2200	150	150	803,200,000	50	5,000	68
,, 6 ,,	—	10·8	475,000	10·8	200	200	200	150	—	—	—	—
,, 8 ,,	—	10·5	—	—	200	9900	150	200	—	—	—	—
	No change in odour, taste or consistence, after 15 days.		Curdled and sour after 8 days.		No change after 19 dys. Taste unaltered.	No change after 19 dys. except increased viscidity and odour of coffee.	No change after 12 dys. Taste unaltered.	No change after 12 dys. except increased viscidity and faint odour of wax.	Coagulated and putrid after 4 days		Curdled and musty after 24 hours.	

Three contaminated samples of Nestlé's milk incubated at 22° C. and two incubated at 37° C. showed no signs of decomposition during twelve days and gave a bacterial content of less than 3000 per c.c. at the end of six days, and of less than 10,000 at the end of a week. One of these samples was incubated at 37° C. for as long as 19 days without showing any change except an increased viscidity due to evaporation and the development of an odour resembling that of coffee.

Decomposition only occurred in one specimen of undiluted milk and then not till the end of the 8th day, and this was the only milk in which bacteria multiplied freely, numbering 475,000 per c.c. at the end of 6 days' incubation at 37° C. but giving rise to no increase in acidity. The same milk incubated at 22° C. gave a low bacterial content at the end of 4 days, showed no increase in acidity after 8 days, and at the end of 15 days had undergone no change in taste, odour, or consistence. In a specimen of Nestlé's milk in which the percentage of sugar had been reduced by a tenfold dilution with water, bacteria multiplied with great rapidity, numbering eleven millions on the 2nd day in a portion incubated at 37° C. and two millions on the 3rd day in a portion kept at 22° C. At both temperatures there was a marked rise in acidity which, at 37° C., began to exert a well-marked inhibitory effect on the growth of bacteria after the first 24 hours' incubation.

Thus compared with that of cows' milk the bacterial content of contaminated Nestlé's milk is remarkably low, and remains low for a week or more at the ordinary summer temperature of 70° F. Even at a temperature of 98° F., bacteria do not invariably multiply, but when a rapid increase does take place, there is evidence to show that physical changes occur in the milk which render it obviously unfit for food. The viscidity of Nestlé's and the presence of large quantities of added sugar are probably the two factors which combine to inhibit bacterial growth as is indicated by the striking effects of freely diluting the milk with water before incubation. If now we take into consideration the fact that the youngest infant requires more than one large tin of Nestlé's milk per week, and that many mothers buy the smaller tins, which last only half the time, it becomes evident, if we may judge by laboratory experiment, that even in a hot summer the contents of a tin will have been consumed before large numbers of bacteria have developed and before decomposition has set in. Experimental evidence also tends to show that there is no danger of a mother feeding her child on milk teeming with bacteria, though still apparently fresh, since large numbers of bacteria give rise to changes which would render the milk repugnant

to the infant, even if they were overlooked by the mother. It would thus appear that high bacterial content and liability to decomposition take no part in the causation of diarrhoea by Nestlé's milk, and this being so, it will not be altogether irrelevant to reconsider the significance of the changes which are brought about by large numbers of bacteria in cows' milk.

The bacterial content of cows' milk in relation to epidemic diarrhoea.

Reference has already been made to the experiments of Park and Delépine, in which it was shown that the pathogenicity of cows' milk for guinea-pigs varied with the mean temperature of the atmosphere, whilst the bacterial content varied with the temperature at which the milk was incubated. If then the bacterial content of milk varies with the temperature in an artificial incubator, there is every reason to suppose that in milk stored in a churn or pan the number of bacteria varies in the same way with the mean temperature of the surrounding air, in which case we may assume that the pathogenicity of milk for guinea-pigs varies, not only with the atmospheric temperature, but also with the number of bacteria which the milk contains. Now the pathogenicity of cows' milk, if measured by its power to produce diarrhoea, is not directly influenced by the mean atmospheric temperature, as may be inferred from the following figures extracted from Ballard's Report (1887, p. 77), and the returns of the Registrar-General.

TABLE III.

	Year 1858	
	21st to 24th week	33rd to 36th week
Mean temperature	62·6	60·3
Rainfall	1·91	1·13
Cases of Diarrhoea at all ages	43	130
Cases of Diarrhoea under 1 year	6	27
Deaths from Diarrhoea	197	289
	Year 1865	
	25th to 28th week	33rd to 36th week
Mean temperature	62·1	62·6
Rainfall	2·4	2·28
Cases of Diarrhoea at all ages	320	140
Deaths from Diarrhoea	1109	346

In these tables, the mean temperature is the average of the mean temperatures in four weeks at Greenwich, and the rainfall is also that

recorded at the Greenwich Observatory. The cases of diarrhoea will be found on Charts IV and V in Ballard's Report (1887–8), where they are described as cases "newly occurring in the poor law medical practice and certain relief institutions in Islington." Those under one year of age are given separately for the year 1858 but are not available for 1865. The deaths are deaths from diarrhoea registered in London for periods, beginning and ending, 14 days later than the four weekly terms to which they have been assigned in the table and may be taken as a rough indication of the number of cases beginning in the weeks under consideration and terminating fatally at some later date. The average duration of fatal cases is about eight days and the law compels the registration of a death within five days of its occurrence.

The seasonal incidence occurring in these two years was in no way exceptional, but is merely given as a graphic illustration of Ballard's law that the prevalence of diarrhoea follows the earth temperature and therefore cannot vary with the temperature of the atmosphere. Thus in 1858, a hot period at the beginning of the summer, coincided with less diarrhoea than a colder period at the end of the summer, whilst in 1865, four cold weeks in June and July were accompanied by three times the amount of diarrhoea that occurred in a warmer four-weekly period in August and September.

The great majority of the fatal cases occurred in infants under one year, and the greater number, both of the infants who died and of those returned as "cases of diarrhoea under one year," were undoubtedly fed on cows' milk. In the face of these facts it becomes impossible to avoid the conclusion already set down, that the capacity of cows' milk for producing diarrhoea does not vary with the temperature of the atmosphere. Since moreover there is reason to believe that the atmospheric temperature and the multiplication of bacteria in cows' milk go hand in hand, it follows that during hot weather at the beginning or end of the summer, cows' milk containing enormous numbers of bacteria may be consumed with comparative impunity, and that the incidence of diarrhoea is not directly influenced by the numbers of bacteria occurring in cows' milk, any more than it corresponds with the bacterial content of Nestlé's milk.

It is however true that the incidence of diarrhoea is greatest in the poorer districts where the bacterial content of the milk consumed is highest[1]. Now whether the number of bacteria in milk be taken as an

[1] *Vide* Park's Experiments. This *Journal*, 1901, pp. 395 *et seq.*

index of the quantity of original pollution, or of the operation of heat and time, there is a suggestion in either case that cows' milk, containing specific poisons, only becomes pathogenic if the poisons are present in sufficient quantity. If home influences are excluded, some such intensive theory becomes necessary in order to explain the comparative immunity to diarrhoea enjoyed by the artificially fed infants of the better classes. Although the milk consumed in a wealthy district is comparatively pure, it must receive at every point, from the udder of the cow to the nursery, the same kind of contamination as the milk sold in poorer districts, and the difference between pure and impure milks is a difference in the quantity, rather than in the quality of their pollution. As Delépine (1903, p. 71) has said, it is difficult to conceive how slight faecal contamination during milking can be avoided, and the same remark might be applied to other kinds of pollution such as street dust, which will enter good and bad milk alike, so long as open churns are used in transit. In Finsbury some 50 per cent. of the infants dying of diarrhoea were noted as fed on cows' milk "boiled in bulk on delivery and used as required," and from my own enquiries in the homes of the poor I have learned that it is the usual practice to boil the milk during the hot weather, as without this precaution it turns sour or putrid in a few hours and is wasted. Since, then, both rich and poor alike boil their milk, we can only attribute the immunity of the infants of the wealthier class to the low bacterial content of the milk they consume, if, that is to say, we ignore the operation of home influences.

It thus appears that the reasonable hypothesis, by which the incidence of diarrhoea is held to be related to the high bacterial content of cows' milk, includes the less reasonable assumption that infective bacteria and their toxins may be ingested with impunity, provided they do not exceed a certain number and quantity[1]. In this assumption lies one objection to the theory of bacterial content; in the fact that the greatest incidence of diarrhoea does not always correspond with the greatest number of bacteria in milk we have a second and perhaps graver objection; whilst a third point against the hypothesis is that Nestlé's milk, which has a remarkably low bacterial content, produces diarrhoea with even greater frequency than cows' milk.

[1] "It appears that this infection of food does not generally lead to serious consequences unless the infection is massive from the first or the food is kept for a sufficient length of time and under conditions of temperature favouring the multiplication of bacilli." S. Delépine (1903, p. 90).

The production of diarrhoea by Nestlé's milk.

In the absence of evidence to the contrary, we might either assume that Nestlé's milk contains from the beginning a sufficient quantity of impurities to produce diarrhoea, or that even a small number of infective bacteria may contrive to produce the requisite amount of toxins, during the time that elapses between the preparation of the milk and its consumption. I learn from M. Henri (Nestlé's London manager) that the farms supplying the factory in Vevey are periodically inspected; the milk drawn between 5 and 6 in the morning is in every case delivered before 10 a.m. and immediately scalded and condensed, whilst the factory, until recently, was open to the public and constituted one of the sights of Vevey. The cows in Switzerland are milked in the open, on hill-side upland pastures in cleanly surroundings, and on the whole it seems probable that, in the matter of purity, Nestlé's milk would not suffer by comparison with the milk of a good English company. The bulk of the milk is gathered and prepared in the spring and summer months, the output for the winter months being insignificant. Thus tins purchased in the English summer contain milk collected during the spring, and milk collected during the summer in Switzerland is consumed in England during the winter months.

Under these circumstances assuming that the causative agent of diarrhoea enters the milk during the summer, it gradually loses its virulence and is found to be innocuous when the milk is consumed in the winter months: if the infective matter enters the milk during the spring, it survives in the tin and develops highly virulent properties, not when the temperature of the surrounding atmosphere rises, but when the four-foot earth thermometer reaches the critical temperature of 56° F. And this it is able to do in a viscid medium unfavourable to the growth of the bacteria.

That bacteria survive and retain their infective properties in one medium and lose their infective properties in another is a self-evident truth, but that they should both lose them and retain them in one and the same medium is at least unlikely, even if it cannot be actually disproved, and for this reason I am not inclined to attribute the pathogenicity of Nestlé's milk to the operation of any organism which may enter before the process of condensation and sealing in the tin.

It has been suggested that the sugar in Nestlé's milk is in itself an intestinal irritant, though there seems to be no evidence that this

is the case. Eustace Smith (p. 638) recommends that infants up to the age of six weeks should be fed on "condensed milk," and says it almost invariably agrees well. Cautley (pp. 285, 286) advises it as a temporary food in cases of acute diarrhoea and describes Nestlé's brand as a good variety of condensed milk. From the context it is clear that both authors refer to sweetened milk, and there do not seem to be any valid grounds for suspecting Nestlé's milk of being in itself a cause of diarrhoea.

If Nestlé's milk is not *per se* an irritant and is not liable to changes in composition which might make it so, and if it does not harbour the germs of diarrhoea in the sealed tin, it can only become a cause of diarrhoea by virtue of the presence in it of specific germs which have entered in the home of the consumer after the tin has been opened.

The home infection of Nestlé's milk and other foods.

Having considered some of the objections that may be made to the supposed existence of outside sources of diarrhoea, we are now in a position to turn our attention to evidence in support of the theory of home infection. In the first place diarrhoea is on occasions, if not always, highly infectious. Bruce Low investigated a very remarkable series of 62 cases of diarrhoea, which, with the exception of the first, were all traced to infection from previous cases. In the second place, if personal infection, direct or indirect, is a prominent factor in the spread of diarrhoea, the incidence of the disease will be round foci of infection and will not show a distribution corresponding to a common food supply. In Leicester, Tomkins (1889, p. 181) described the food and water supplies as being practically the same throughout the borough and yet he found that diarrhoea was very largely in excess in certain districts of the borough and comparatively absent in other parts. Conversely milk and foods from widely different sources and prepared in many different ways possess the power in common of producing diarrhoea in a certain class of home, a fact which might be attributed with greater probability to the home infection of the food than to the universal contamination of the sources of all the food supplied to districts where diarrhoea is rife.

Again, the production of diarrhoea by milk that has been boiled and left to stand in an open jug is more probably due to the home infection of milk than to the survival in it of virulent bacteria and toxins.

In his report on the Health of Brighton for 1903, Dr Newsholme

(p. 21) has pointed out that food in the homes of the poor can scarcely escape faecal infection. "The sugar used in sweetening milk is often black with flies, which have come from a neighbouring dust-bin or manure heap or from the liquid stools of a diarrhoeal patient in a neighbouring house. Flies have to be picked out of the half-empty can of condensed milk before its remaining contents can be used for the next meal....When we remember the personal uncleanliness of some mothers, and that they often prepare their infants' food with unwashed hands, the inoculation of this food with virulent colon bacilli of human origin ceases to be a matter of surprise."

The passage I have quoted needs no further emphasis. There is no substance more attractive to flies than human excrement, and no one who has depended on Nestlé's milk in a tropical country will forget the revolting sight presented by an open tin to which flies had gained access. The flies in the fish shops and butchers' shops in poorer neighbourhoods present a no less disgusting spectacle in the summer months, and, when Ballard said he was not prepared to name any kind of food which might not become infected with the contagium of diarrhoea, he might well have added "through the agency of flies."

Without entering into the question as to whether or not the bacteria that produce diarrhoea are colon bacilli, it will be readily conceded that the stools of infected persons are the most obvious and even the most likely source of the specific contagium of diarrhoea. If then we make what seems to be a justifiable assumption and accept the faeces of persons with diarrhoea as infectious, it is no assumption but a fact, that in the summer particles of this infectious matter are constantly being conveyed by flies to the food of healthy persons. It is to the greater attraction for flies presented by sweetened milk compared with cows' milk, that Dr Newsholme (1903, p. 21) attributes the greater prevalence of diarrhoea among infants fed on Nestlé's milk, and I can find no feature in the history of a normal epidemic of summer diarrhoea which cannot be adequately explained by the double supposition that diarrhoea stools are infectious and are frequently conveyed to the food of the healthy by flies. That cholera and typhoid fever have been spread through food contaminated by flies no one will doubt who is familiar with the convincing evidence to this effect collated by Nuttall (1899, pp. 27–31). This evidence is largely derived from countries where human excrement is readily accessible to flies. Since infants are the chief sufferers from epidemic diarrhoea, and since, under the best sanitary systems, their faeces must always be accessible

to flies, there are good grounds for the supposition that in this disease, which in some respects is analogous to typhoid fever and cholera, flies may be carrying agents of the first importance.

Flies as the distributing agents of the contagium of diarrhoea.

One of the most remarkable features in the prevalence of diarrhoea is that it follows the rise and fall of the earth temperature and not that of the air. Hot weather early in the year has no immediate influence, and cold weather at the end of the summer does not produce an immediate diminution in the amount of diarrhoea, the effect only making itself felt when the cold has been prolonged for two or three weeks. It is a matter of common experience that the number of house-flies does not attain its maximum with the first onset of hot weather and from their life-history we would expect their prevalence to follow rather than to coincide with periods of great heat.

Each generation of the common house-fly takes 14 days to mature, a period which may be reduced to five days or less in hot weather or much prolonged in the colder months of the year (Theobald 1904–5, p. 111). Since a notable increase in the prevalence of flies depends, not on the curtailing of the larval and pupal stages of a single generation, but on the rapid succession of one generation by another during a continuous period of several weeks, the effect of a prolonged spell of hot weather is not immediate, but gradual and cumulative. The diminution in the number of flies that follows the onset of cold weather is also brought about slowly and by degrees. In this way it comes about that the prevalence of flies always lags behind the air temperature and persists for a time after the hot weather has ceased, and thus their numbers tend to follow the rise and fall of the earth thermometer rather than the changes in the temperature of the air.

In other words, those meteorological conditions which have been found to increase or diminish the prevalence of diarrhoea, exercise a precisely similar effect on the prevalence of flies.

In the winter when flies are absent, indirect personal infection may still occasionally take place through the agency of the hands of a dairy woman whose infant is suffering from diarrhoea, and in a variety of other ways which must be excluded before we are bound to accept the possibility of an impersonal origin of the disease.

The immunity of well-to-do infants may be explained, partly by the distance that separates the sick from the healthy and partly by the

small number of flies in their neighbourhood. In poorer districts, six or seven babies may occupy the tenements of one house with a common yard where the flies congregate and flit in and out of the open windows, themselves conveying infected excrement to the milk of healthy infants, or depositing the excrement in the dust-bin, whence it may again be conveyed into the house by other flies. Calm weather promotes diarrhoea, and high winds are unfavourable to the spread of diarrhoea and to the active migration of flies alike. Loose soil and fissured rock, containing organic filth in its crevices, favour the spread of diarrhoea and the breeding of flies, whilst solid rock is unfavourable to both. On the circumstances of food keeping and its exposure to emanations from accumulation of domestic filth Ballard laid especial stress, and it is obvious that food under these circumstances would be in the midst of swarms of flies.

Altogether Ballard (1887–8, p. 8) has enumerated eighteen conditions which increase or diminish diarrhoeal mortality, and, with the exception of "Occupation of females from home" and "Illegitimacy," every one may be said to have the same influence on the prevalence of flies. The conditions are well known, and without considering them in detail I will pass on to the provisional conclusions suggested by a survey of some of the factors concerned in the causation of epidemic diarrhoea. To prevent misapprehension it should be made clear that the term diarrhoea has purposely been used in a broad or even loose sense. Clinicians differ among themselves as to what is and what is not epidemic diarrhoea, but it is generally admitted that the bulk of the cases constituting the great wave of diarrhoea that occurs every summer are instances of the same specific disease. It is to these cases that the following conclusions apply, and they must not be taken as denying the occurrence of local outbreaks of diarrhoea which differ fundamentally from the ordinary summer epidemics.

CONCLUSIONS.

1. In proportion to the number of consumers, Nestlé's milk, containing comparatively few bacteria is more frequently associated with diarrhoea than cows' milk in which the number of bacteria is phenomenally high.

2. In certain seasons cows' milk may be exposed to temperatures which favour a high bacterial content and yet not become a frequent source of diarrhoea.

3. The numbers of bacteria in preserved and natural cows' milk have no direct influence on the incidence of diarrhoea.

4. The great majority of cases of diarrhoea are due to the consumption of food which has been infected in the district in which the cases have occurred.

5. The infective matter thus conveyed to food is generally the excrement of some person suffering from diarrhoea.

6. The life-history of house-flies and the facility with which they can convey the faecal excrement of infected infants to the food of the healthy, suggest that the seasonal incidence of diarrhoea coincides with and results from the seasonal prevalence of flies.

In collecting the evidence on which these conclusions have been based I have received assistance from many sources, and in particular I wish to express my gratitude to Dr Newman for placing the records of his office at my disposal, to Dr Garrod and Dr Andrewes, who have permitted me to make use of the laboratories for chemical pathology and bacteriology at St Bartholomew's Hospital, and finally to Mr Theobald and Mr Verrall for their courtesy in furnishing me with the valuable results of their observations on the habits and natural history of flies.

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