If we make a mistake we suffer for it, and can blame no one but ourselves. But why should we be called upon to suffer for the fads and mistakes of people at home? We hold that further temporizing, in the light of our present knowledge, is nothing short of criminal folly.

With reference to Dr. Neave's suggestion that the game within a certain area should be impounded and destroyed, I may say that this suggestion was made fully two years ago. We asked, when the first epidemic broke out in Nyasaland, to have the game destroyed. This was refused, on the grounds, among other reasons, that the outbreak was distinctly localized, and that any interference with the game might cause the animals to spread out and carry disease into clean districts. On the strength of this statement I suggested to the Acting Governor that he should impound all the animals within the area mentioned, and once and for all stamp out the disease by exterminating the animals. To this proposal I got a reply that while His Excellency appreciated my good intentions he did not regard the proposal as practicable.

In conclusion, I should like to express my appreciation of and gratitude for the work of the Liverpool Commission, which has settled once and for all many hitherto hotly disputed points, and also my gratitude to the BRITISH MEDICAL JOURNAL for the publicity it is giving to the work of Dr. Warrington Yorke.

A STUDY OF EPIDEMIC DYSENTERY IN THE FIJI ISLANDS,

WITH SPECIAL REFERENCE TO ITS EPIDEMIOLOGY AND TREATMENT.

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THE observations recorded in this paper were made in the Fiji Islands during the year 1910. This group of islands, situated between lat. 17–19 degrees S. and long. 177–179 degrees E., was ceded to Great Britain in 1874, and now constitutes a Crown Colony.

According to Dr. B. G. Corney, I.S.O., a great authority on all matters appertaining to the Pacific, there is no evidence that dysentery was originally introduced by the early European settlers,* but it is known that since the early days epidemics of dysentery have been of annual occurrence, and have levied a heavy toll of life, not only among the European settlers and the imported indentured Indian and Solomon Island labourers, but also among the Fijian natives themselves, and have constituted the chief scourge of the colony, so healthy in other respects.

In former days these dysentery epidemics were of great virulence; the newly imported Solomon Island labourers were specially attacked; thus Daniels[1] reported one such epidemic in which the death-rate was 48 per cent.

At the present day, probably owing to better medical service and hygienic conditions, the case mortality from dysentery of all kinds is not nearly so high as formerly, and amongst the Fijians, according to the official records, is about 16 per cent.

During the year 1910 some 170 well-marked dysentery cases were studied in detail at the Colonial Hospital, Suva, the capital of the group; of these only 11 were found to be of amoebic origin—that is to say, vegetative amoebae bearing the characteristics of the *Amoeba histolytica* of Schaudinn were found in the stools. In the dysenteric discharges of the other 159 cases no amoebae were ever found.

The epidemic dysentery affected members of the various nationalities—Indian, Fijian, Solomon Islanders, Europeans, and half-castes—to an equal degree as far as could be ascertained.

In Fiji, as in more temperate zones, epidemic dysentery is an institutional disease; some 53 per cent. of the patients under observation were prisoners undergoing sentences in either of the prisons of Suva.

* The Fiji Islands were discovered in 1643 by Tasman, who, however, did not land there. The first Europeans who had intercourse with the natives were the mutineers of the *Bounty*, who had crossed over from Tahiti in 1791.

The Bacilli.

By plating out the mucus diluted in sterile broth on Conradi-Drigalski medium (litmus-lactose-nutrose-crystal-violet agar), dysentery bacilli were isolated from the stools in 35 cases and twice from the intestinal mucosa *post mortem*. The bacilli were recognized by their morphology, by their reactions with 1 per cent. solution of various sugars in litmus peptone water, by their agglutination reactions with high dilutions (1 in 50) of a polyvalent anti-dysenteric serum (Lister Institute), and further by their toxicity to guinea-pigs on intraperitoneal injection.

The dysentery bacilli isolated mainly conformed to the well-known types, the one most frequently encountered being the Shiga-Kruse bacillus, but a bacillus identical with the Hiss and Russel Y bacillus was also almost as common. In addition to these, other bacilli, morphologically and culturally similar, but giving atypical reactions with solutions of certain sugars such as saccharose and maltose, while agglutinating with the same polyvalent immune serum, were also encountered, and in these reactions agreeing with Strong's[2] original Philippine strain.

In the varieties of bacilli encountered the results, in the main, are in agreement with those of Shiga,[3] who now recognizes five main types of dysentery bacilli as differentiated by their sugar reactions.

On three occasions bacilli giving identical reactions to those cited above, but in addition exhibiting a feeble lactose fermenting power, were encountered. Ohno,[4] during his extensive study of 74 strains of the dysentery bacillus, also appears to have encountered this variety, to which he assigns 3 out of his 15 types.

Table of Reactions of Main Types of Dysentery Bacilli in Litmus Peptone Water containing 1 per Cent. of Sugars after Twenty-four Hours' Incubation at 37° C.

	Shiga-Kruse Type.	Hiss and Russel's Type Y.	Ohno Type M.
Mannite ...	−	+	+
Lactose ...	−	−	+
Saccharose ...	−	−	−
Maltose ...	−	=	+
Dulcite	−	−	−
Dextrose ...	+	+	+
Milk	Acid at first, returned to alkaline second day	Acid at first, returned to alkaline second day	Acid at first, returned to alkaline fourth day

The production of indol by these bacilli was very variable. In the true Shiga-Kruse type none was produced as a rule, even after lengthy incubation.

After long subculture over a period of nine months, and after the sugar reactions had been frequently tested, certain minor variations of certain strains were noted, especially with regard to the fermentation of maltose and saccharose; nevertheless, the reactions of the non-acid or Shiga-Kruse and the acid or Flexner type of bacillus to dextrose and mannite respectively remained constant throughout.

Clinical Observations.

Cases from which the dysentery bacilli were isolated varied greatly in their clinical manifestations—from a mild catarrhal condition, in which the stools were solid and coated with a small amount of blood and mucus, to a fulminating one, in which the call to stool was incessant and in which the patient, overwhelmed with pain, physical exhaustion, and toxic absorption, would succumb in from two to three days from the commencement of the attack. The temperature also varied within wide limits; cases were encountered in which it was subnormal throughout, others in which it reached 103° F. and over. No special dysentery bacillus was found to be associated with any clinical type of case.

No hepatic symptoms in life or suppurative lesions of this organ *post mortem* were ever encountered in this infection. Only one case of bacillary dysentery of a relapsing nature was encountered amongst the whole series. The serum of 74 per cent. of the bacillary cases agglutinated—in a dilution as high as 1 in 200 in some

instances—one or other of the dysentery organisms. A positive agglutination did not take place until six days after the commencement of symptoms.

The eleven amoebic cases, on the other hand, gave a previous history of a much more subacute and relapsing character. Symptoms of acute hepatitis were present in four instances, and in another multiple miliary abscesses of amoebic origin were found *post mortem*.

In making a diagnosis of amoebic dysentery from the microscopic examination of the stools alone care must be exercised so as not to mistake certain round refractile cells, probably representing disintegrated intestinal epithelium and leucocytes, and in which red blood cells are often enclosed; these in some instances certainly bear a close resemblance in size and form to the pathogenic amoeba, and are at present in considerable numbers in the stools of bacillary cases as well as in those of the more chronic amoebic disease.

Epidemiology.

In studying the epidemics of dysentery which have occurred in Suva in recent years one is struck by the fact that the maximum incidence of the disease occurs during the months of December, January, February, March, and April, the period of the highest mean temperature and of the largest rainfall.

In searching for an explanation of this fact I was led to inquire into the possible sources of the bacillary infection.

From what we know of the bacteriology of acute dysentery it is right to assume that as the bacillus escapes from the body in the stools these, as in the case of typhoid, act as the primary source of infection, and, further, that the alimentary route is probably the one by which a fresh infection takes place. The water supply of the town of Suva is beyond reproach, therefore any suspicion of its contamination by infected faecal matter can be dismissed.

There is, in addition, no evidence that the direct infection of foodstuffs plays any part in the spread of the disease, since acute dysentery occurs amongst all races in Fiji, though their respective dietaries are entirely dissimilar. The Fijians for the most part live on cooked farinaceous foods, such as the yam and the dalo (*Calocasia esculenta*), whilst the Indian subsists, as elsewhere, on boiled rice and pulse; the Europeans on a mixed diet, in which fresh meat plays an important part.

In considering the indirect methods by which infection could be conveyed to foodstuffs suspicion fell on the house-fly. Any visitor to the islands will bear me out when I say that the house-flies (*Musca domestica*) constitute a plague of considerable importance. They swarm in the bush and on the roadside, covering the traveller from head to foot. In the houses every article of food must be protected from their attentions, and it is a common occurrence during meals to place a fly-paper on the table in order to draw away the flies from the meal. I need hardly add that in the rather insanitary kitchens they are specially abundant.

The abundance of flies in the cultivated districts can probably be explained by the fact that numerous favourable breeding places are afforded by heaps of decomposing sugar cane, and in the uncultivated districts by the faecal accumulations which generally mark the proximity of native villages.

There can be little doubt that in Suva, at any rate, there is a definite seasonal variation in the number of house-flies, and that this season corresponds with the conditions most favourable for their propagation, that is the season of highest rainfall and atmospheric temperature, and as I have previously pointed out, it is just during these months that the maximum number of cases of acute dysentery occur.

Investigations on the bacteriological flora of flies captured in a dysentery ward of the Colonial Hospital, Suva, were undertaken. A number of attempts were made but on two occasions only did I succeed in isolating the typical Shiga-Kruse bacillus from the lower intestinal tract of the fly.*

The technique adopted was based on the work of Graham-Smith;[5] the suspected flies were caught in batches of four in the vicinity of a patient from whose stools the Shiga-Kruse bacillus had already been isolated. The wings and legs of the fly were first removed, the body, sterilized as much as possible by passing it rapidly through a flame, was then placed on a sterilized slide together with a drop of sterile saline solution. The abdominal segments were next separated by means of sterile needles, thus exposing the stomach and intestinal tract; a portion of the latter was then removed on a platinum rod, emulsified in sterile broth, and plated on Conradi-Drigalski medium. The Shiga-Kruse bacillus was recognized by the tests already given.

In view of the incomplete data existing at present connecting "wild flies" with the spread of disease, this result may be considered of some value when taken in conjunction with the seasonal distribution of the disease in Fiji and the concomitant prevalence of the house-fly.

A number of Gram-negative non-motile bacilli were also isolated from the intestines of these flies. These bacilli do not ferment the sugars with gas production, and, as has been pointed out by Graham-Smith, are probably normal inhabitants of the intestinal tract of the fly. They, however, are important in that they are liable to be mistaken for pathogenic organisms, especially the dysentery bacillus.

In his interesting book, *Flies and Disease*,[6] Graham-Smith has detailed this subject at great length. These bacilli which do not liquefy gelatin are divided into lactose and non-lactose fermenting groups. Of these, the latter group, from my point of view, are the most important. Graham-Smith found non-lactose fermenting bacilli in 32 per cent. of flies from diarrhoea-infected houses.

The Three Main Varieties of Non-lactose Fermenting Bacilli found in the Intestines of Flies caught in Fiji.

	Mannite.	Lactose.	Saccharose.	Maltose.	Dulcite.	Dextrose.	Milk.
1	A.	—	A.	A.	—	A.	Acid; alkaline fourth to fifth day.
2	—	—	A.	A.	—	A.	Alkaline.
3	A.	—	—	A.	—	A.	Acid; alkaline third to fifth day.

These probably correspond to Group C, Subgroup *b*, Group B, Subgroup *j*, and Group C, Subgroup *a*, respectively, in Graham-Smith's classification.

These bacilli were further differentiated from the true dysentery organism by their failure to agglutinate with a polyvalent antidysenteric serum.

Of the bacilli fermenting lactose without gas production and forming permanent acidity and occasionally clot in milk a great number were found. To these bacilli I need not refer at any length, as they are of little scientific interest in this connexion. All were tested with a polyvalent antidysenteric serum, but with a negative result.

Later experiments on the transmission of the dysentery bacillus through the intestinal tract of the house-fly were undertaken at the London School of Tropical Medicine. They were conducted in a glass house in which the temperature approached that of the tropics, reaching as high as 91° F. in the daytime. Before experiments were commenced the house was thoroughly washed out with lysol, and all stray flies and other insects were rigidly excluded. The flies were bred and hatched in captivity under as sterile conditions as possible. The pupae were washed in dilute mercury perchloride solution (1 in 1,000) and placed in a sterile cage till the flies emerged. They were then transferred in batches of twenty in sterile test tubes to sterile bottles fitted with a gauge sleeve in the manner recommended by Graham-Smith, and fed with an emulsion of sterile bread and water introduced into the bottles by means of a sterile spoon. The flies were infected by means of bread soaked in a broth culture of the dysentery organism. Over forty experiments were conducted in this manner, but for some reason or other not all were successful.

Two strains of bacilli belonging to the non-acid and the acid or Flexner group were used for infecting the flies—that is, the true Shiga-Kruse bacillus and the Y bacillus—

*A summary of these observations has already been published in Supplement No. 2 of the *Journal of the London School of Tropical Medicine*. Witherby and Co., London. 1912.

bacilli which after frequent testing and subculture over a period of nine months had given constant fermentation reactions with the sugars. The infected material, after being left in contact with the flies for twenty-four hours, was removed and sterile food was given as before.

At varying periods batches of flies were dissected, and the contents of the lower intestine were plated out in the manner described above, with the result that neither of these organisms could be recovered from the flies' intestines after the fifth day. The bacilli were recognized by the usual tests.

I was unable to obtain any evidence of the multiplication of these organisms in the fly.

Somewhat similar results have been obtained by Graham-Smith in his experiments with typhoid bacillus and *B. enteritidis*.

Naturally in these experiments the possibility of the flies being infected with the dysentery bacillus in the larval state cannot be excluded; but against this probability are the experiments of Bacot[7] and Graham-Smith, who found that the non-sporing saprophytic organisms only rarely survived in the intestines throughout the larval and pupal stage, and that cultivated strains of *B. typhosus* and *B. enteritidis* fed to larvae do not survive. Lately Tebbutt,[8] in a series of experiments in which larvae were fed with cultures of *B. dysenteriae*, found that the organism could only be recovered from the pupae and imagines in a small number of cases, but concluded that the possibility of infection of flies by these organisms in their breeding grounds may be considered a very remote one.

The dysentery bacillus thus recovered in the majority of instances gave, when tested on solutions of the sugars, the same reaction as the original bacillus with which the flies were infected; there were, however, several exceptions. A maltose fermenting strain was obtained from a fly originally fed with the Shiga-Kruse bacillus. On further subculture, over a period of fifteen days, this property was lost.

From flies infected with the Y bacillus a bacillus was recovered with such feeble power of fermenting mannite that acid was only produced in this sugar after four days' incubation. This property it retained for two months after isolation, but subsequently lost. Other bacilli capable of fermenting saccharose in addition to mannite and dextrose were also obtained; these, however, are not of such great interest.

A Flexner bacillus exhibiting but a poor reaction to mannite has been found by Shiga in the stools of acute dysentery; according to the published descriptions, this bacillus gives a similar reaction to that I have just recorded. Shiga considers that this bacillus occupies a place halfway between the acid and non-acid groups.

That a bacillus can acquire these characteristics by a short sojourn in the intestinal tract of the fly certainly suggests that under certain conditions dysentery bacilli of one group can acquire the characteristics of the other. It may be that this is an instance of an adaptation of the bacillus to its surroundings comparable to the mutation of the *B. typhosus* when grown for a long period on lactose-containing media, according to the researches of Penfold[9] and Twort.[10] My results are certainly suggestive of some such process, but it must be noted that the acquired character of this bacillus was not retained for many generations; it is therefore necessary that these experiments should be repeated on a more extensive scale.

Treatment of Acute Dysentery.

In a disease characterized by clinical forms of such varying severity, especially in native patients belonging to races differing widely in physique and disease-resisting powers, it is difficult to compare the results of various forms of treatment. In every series, however selected, there will necessarily be a number of mild cases in which the constitutional disturbances are so slight as to cause the minimum amount of inconvenience, and which, if untreated, would recover in a few days. The only reliable method would seem to be to compare the results of treatment by different methods of a parallel series of consecutive cases all approximately of equal severity.

The first 53 consecutive cases, of which 41 per cent. had marked constitutional symptoms, were treated by the routine treatment adopted in the Colonial Hospital, Suva. This treatment consisted of the administration of sodium sulphate in drachm doses given every hour for the first twenty-four hours, and subsequently every four hours. The case mortality in this series was 13.2 per cent.

The next series consisted of 106 cases, of which 42 per cent. had marked constitutional symptoms. Some 34 of these were treated by salines combined with cyllin in gelatin capsules (palatinoids) in doses of twenty to thirty in the twenty-four hours (m 60 to 90 pure cyllin); the remaining 72 cases received in addition to this intravenous injections of a polyvalent antidysenteric serum (kindly supplied by the Lister Institute). A dose of 20 c.cm. was given to adults, 10 c.cm. or less to children.

In apparently hopeless cases injection of 50 to 70 c.cm. of this serum in the first twenty-four hours after admission was followed by remarkable improvement. After such injections no deaths occurred in a series of five cases, in whom the disease was of the severest type, as evidenced by the passage of gangrenous stools and the toxic condition of the patient.

In this series of 106 cases there were only two deaths—a mortality rate of 1.8 per cent. Neither of these deaths could be ascribed to the dysenteric lesions—one, a Fijian child, succumbed to an intercurrent attack of bronchopneumonia; the other, an Indian woman heavily infected with ankylostomes, died in uraemic convulsions. Attention has been drawn to the occurrence of death from nephritis after massive doses of a polyvalent antidysenteric serum by Savage in his El Tor series.

In this instance not only was the average stay in hospital of cases of moderate severity considerably shortened, but it was also found that the stools resumed their normal faecal consistency in a much shorter average space of time—that is, after five days, as compared with eight on the sodium sulphate treatment alone. These favourable results have been confirmed by Willmore[11] and Savage working with a much more extended series of cases. The case mortality rate of dysentery in Egypt seems to have been abnormally high (70 per cent.), but under antiserum treatment in 1912 to 1913 it was reduced to 12 per cent.

The oral administration of salines and intestinal antiseptics, together with the intravenous injections of antiserum, appears to me to rest on a rational basis when the following facts are considered.

The lesions of acute dysentery are confined to the large intestine, and from it the dysenteric toxins are absorbed; it is probably the absorption of these toxins which is responsible for the collapse so often encountered in this affection, especially in children. To counteract the deleterious effects of this toxin, antiserum is injected. The administration of sodium sulphate is a necessary measure to cleanse the lower bowel of faecal matter and thereby to accelerate the repair of the ulcerated mucous membrane. The administration of cyllin, on the other hand, in such large doses must certainly tend to inhibit the multiplication of the dysentery bacilli and other organisms in the intestinal contents.

It must be borne in mind that a very acute dysentery of primary amoebic origin, associated with the passage of gangrenous stools, in which amoebae, though present in the intestinal lesions *post mortem*, cannot be found in the stools in any numbers during life, is also sometimes met with. In Fiji I encountered one such case. Therefore, in any given case of an apparently desperate nature and in which there is no time to make a diagnosis by cultural methods, it is advisable to combine the specific treatment of both the amoebic and bacillary forms. If the case be not of primary amoebic origin, the hypodermic injections of emetine, if inefficacious, is completely harmless.

The combined emetine and antiserum treatment in any case of doubtful origin is therefore the most rational to adopt in the circumstances.

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