linquish his duties on account of poor health. In the spring of 1864 his health gave way. Fortunately after a sojourn in Wisconsin and Minnesota it was so far restored that in the fall of 1865 he was again able to resume his duties at the university. But he devoted himself so assiduously to his duties that his health again failed and he was obliged in 1869 to give up his work. This time the effort to regain his health was in vain. He passed a summer in Colorado and Minnesota, the following winter in Philadelphia and the spring in South Carolina. Returning to St. Louis and then to Minnesota, "he finally closed a laborious, useful life at St. Paul, Minnesota, on December 13, 1870, in the 51st year of his age." He was buried in Bellefontaine Cemetery in St. Louis.

In his family the warmest traits of character were constantly exhibited. In 1842 soon after taking charge of the Naval School in Philadelphia, he married Miss Catherine Hemple of that city. Even in his most laborious days, he found time to join in the sports and amusements of his children and in later years to guide their reading and studies. He constantly manifested the religious faith which he professed, but he never obtruded the peculiarities of his faith on those who differed from him.

Professor Chauvenet was honored by being elected to the American Philosophical Society and to the American Academy of Arts and Sciences. In 1860 St. John's College, Annapolis, Maryland, conferred on him the degree LL.D. At the formation of the National Academy of Sciences he was one of the prominent members. He served on a number of its committees and was its vice president at the time of his death. As already stated, he was one of the first members of the American Association for the Advancement of Science. In 1859 he was elected general secretary and in 1869 at the Salem meeting he was elected president. At the next meeting in 1870, in the absence of Professor Chauvenet, who was too ill to attend, the vice president, Thomas Sterry Hunt, presided. At the following meeting, the president, Mr. Hunt, in announcing the death of Professor Chauvenet, said in part:

It was already feared at the time of his election to the presidency that failing health would prevent his presence in 1870, as was the case. He died at the age of fifty leaving behind him a record of which science and his country may be proud. During his connection of fourteen years with the Naval Academy he was the chief instrument in building up that institution, which he left in 1859 to go to St. Louis. It is not for me to pronounce a eulogy, to speak of his profound attainment in astronomy and mathematics or of his published works which have taken rank as classics in the literature of these sciences. Others more familiar with his field may in proper time and place attempt the task.

All who knew him can, however, join with me in testifying to his excellences as a man, instructor and friend. In his assiduous devotion to scientific studies he did not neglect the more elegant arts, but was a skillful musician and possessed of great general culture and refinement of taste. In his social and moral relations, he was marked by a rare elevation and purity of character, and has left to the world a standard of excellence in every relation of life which few can hope to attain.[7]

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THE TRANSMISSION OF HUMAN PROTOZOA[1]

EVERY class in the phylum Protozoa contains species that live in man, and some of these are of considerable importance in various parts of the world as disease-producing agents.

One of the most interesting phases in the life cycle of these parasitic Protozoa is that during which they are transmitted from one host to another, either directly or through an intermediate host. It is at this time that measures for prevention and control can most successfully be applied, measures which, from the standpoint of personal hygiene, protect the individual from infection, and, from the standpoint of public health, protect the general population, either rural or urban, from infection. The twenty-five species of human Protozoa that are usually recognized by protozoologists at the present time may be classified as follows with respect to their habitat within the body and the method by which they are transmitted.

I. *Intestinal Protozoa:*
- (1) Species transmitted by the contamination of food or drink by cysts.
  - (a) Intestinal Amœbae.
    - (1) *Endamœba histolytica,* the organism of amœbic dysentery and amœbic liver abscess.
    - (2) *Endamœba coli,* a harmless commensal living in the large intestine.
    - (3) *Endolimax nana,* similar to (2).
    - (4) *Iodamœba williamsi,* similar to (2).
    - (5) *Dientamœba fragilis,* similar to (2).
  - (b) Intestinal Flagellates.
    - (6) *Chilomastix mesnili,* a possible causative organism of flagellate diarrhea.
    - (7) *Embadomonas intestinalis,* probably a harmless commensal.

[7] Proceedings of the American Association for the Advancement of Science (1871) Vol. XX.

[1] From the Department of Medical Zoology, School of Hygiene and Public Health, the Johns Hopkins University and the London School of Hygiene and Tropical Medicine. This paper is an abstract of three lectures delivered at the London School of Hygiene and Tropical Medicine on April 18, 19 and 20, 1926.

(8) *Tricercomonas intestinalis,* also probably a harmless commensal.

(9) *Giardia lamblia,* accused of causing flagellate diarrhea.

(c) Coccidia.

(10) *Isospora hominis,* the agent of a type of diarrheic infection known as coccidiosis.

(d) Infusoria.

(11) *Balantidium coli,* the ciliate responsible for balantidial dysentery.

(2) Species transmitted by the contamination of food or drink by trophozoites.

(12) *Trichomonas hominis,* another flagellate accused of causing diarrhea.

(3) Species transmitted by contact in the trophozoite stage.

(13) *Endamœba gingivalis,* an amœba that lives in the mouth and that has been accused, probably unjustly, of causing pyorrhea.

(14) *Trichomonas buccalis,* apparently a harmless flagellate living in the mouth.

(15) *Trichomonas vaginalis,* a flagellate living in the vagina that may be pathogenic.

II. *Blood-inhabiting Protozoa:*

(1) Species transmitted by tsetse flies.

(16) *Trypanosoma gambiense,* the organism of Gambian sleeping sickness.

(17) *Trypanosoma rhodesiense,* the organism of Rhodesian sleeping sickness.

(2) Species transmitted by triatoma bugs.

(18) *Trypanosoma cruzi,* the organism of Chagas's disease or South American trypanosomiasis.

(3) Species transmitted (probably) by phlebotomus sand flies.

(19) *Leishmania donovani,* the agent of kala-azar.

(20) *Leishmania tropica,* the agent of oriental sore.

(21) *Leishmania americana,* the agent of espundia or South American leishmaniosis.

(4) Species transmitted by anopheline mosquitoes.

(22) *Plasmodium vivax,* of benign tertian malaria.

(23) *Plasmodium malariæ,* of quartan malaria.

(24) *Plasmodium falciparum,* of estivoautumnal or malignant tertian malaria.

III. *Tissue-inhabiting Protozoa:*

(25) *Sarcocystis* sp. (?), a parasite rarely reported in the muscle of man.

TISSUE-INHABITING PROTOZOA

For the sake of convenience the last-named parasite will be considered first. Protozoa of the genus *Sarcocystis* are frequent inhabitants of muscle in sheep, mice and many other lower animals in which they often produce diseased conditions that may prove fatal. Not more than ten authentic cases of sarcosporidiosis have been reported from man. The method of transmission in nature either to man or to the lower animals has never been determined. Laboratory experiments have demonstrated that mice become infected if fed on muscle from other mice containing spores (T. Smith, 1901),[2] that mice may likewise be infected if fed on diseased muscle from sheep (Erdmann, 1910),[3] that guinea pigs may be infected by feeding them on diseased muscle from mice (Negri, 1908;[4] Darling, 1910),[5] and that the infection may be transmitted if mice are fed the feces from diseased mice (Negri, 1910).[6] These results suggest that carnivorous and omnivorous animals may become parasitized either by eating diseased muscle or by ingesting food or drink contaminated by feces from diseased animals. Herbivorous animals, such as sheep and cattle, may be infected by the latter method but hardly by eating diseased tissue. The host-parasite specificity, that is, the rigidity with which one species of parasite is limited to one species of host, must be very weak in the case of *Sarcocystis,* since it can apparently live in several vertebrate hosts. This fact renders the "blind-alley theory" quite reasonable. This theory holds that the organisms found in man are really sarcosporida from lower animals that accidentally set up an infection in a human host from which they are unable to escape and hence have encountered a blind alley. *Sarcocystis* is not and probably never will be a successful human parasite, but its method of transmission remains one of the least known of any of the important protozoan parasites.

TRANSMISSION BY CONTACT

The two species of Protozoa that occur in the human mouth are apparently widespread, having been found practically wherever looked for. They likewise are known to be present in a large percentage of the general population. The amœba, *Endamœba gingivalis,* lives in the tartar of the teeth and in the materia alba around them. Probably at least one half of the general population is infected. It was once thought to be the causative agent of pyorrhea

[2] Smith, T., 1901, "The Production of Sarcosporidiosis in the Mouse by feeding Infected Muscular Tissue," *Jour. Exp. Med.,* 6: 1–21.

[3] Erdmann, R., 1910, "Beiträge zur Morphologie und Entwicklungsgeschichte des Hammelsarkosporids in der Mans." *Centrlb. Bact. Orig.,* 53: 510.

[4] Negri, A., 1908, "Beobachtungen über Sarkosporidien." *Centrlb. Bakt. Orig.,* 47: 56–61, 612–622.

[5] Darling, S. T., 1910, "Sarcosporidiosis in the opossum, etc.," *Bull. Path. Exot.,* 3: 513–517.

[6] Negri, A., 1910, "Beobachtungen über Sarkosporidien," *Centrlb. Bakt. Orig.,* 55: 373.

but is now considered harmless. *Trichomonas buccalis* the other mouth-inhabiting protozoon, is likewise widespread and occurs in perhaps a third or more of the general population. It lives in the mucus and tartar between the teeth and is probably harmless. Both of these species are no doubt transferred from one person to another by kissing.

The vaginal flagellate, *Trichomonas vaginalis*, lives in vaginal mucus, but has been reported also from the urine of men (Hegner and Taliaferro, 1924).[7] It apparently does not occur in young girls (Pronoschina, 1923)[8] but has been recorded in from ten to fifty per cent. of adult women (Brumpt, 1913;[9] Hegner, 1925).[10] Whether it is a pathogenic protozoon or not is still undecided. Transmission probably takes place during coitus.

Transmission of Trichomonas Hominis in the Trophozoite Stage by the Contamination of Food or Drink

No cysts are known in the life history of this intestinal flagellate, hence it must be transmitted in the trophozoite stage. The only conceivable method of reaching the intestine appears to be by the fecal contamination of food or drink that is ingested by the host. The first question that arises is whether trophozoites are able to withstand the action of the digestive juices during their passage through the stomach and small intestines. It has been proved that *Trichomonas muris* is able to do so in the rat and there is every reason to believe that *Trichomonas hominis* can in man (Hegner, 1924).[11] The next problem is whether *Trichomonas* is able to live outside of the human body in fecal material long enough for food and drink to become contaminated. Experiments involving the cultivation of the flagellate in test tubes indicate that they are (Hegner and Becker, 1922).[12] An ovomucoid medium was used; a better solution has since been evolved known as the serum-saline-citrate medium. This consists of water 100 cc., NaCl 0.7 gms., Na Cit 1.0 gms. and Loeffler's dehydrated blood serum 0.5 gms. This mixture is placed in test tubes, about 10 cc. in each; then a sample of fecal material the size of a pea is obtained on the end of a toothpick and both toothpick and sample dropped into the tube; after cultivation for twenty-four hours at about 36° C. a drop from the top of the culture is placed on a slide and examined. Hegner and Becker found eight out of 110 persons positive by this method, whereas only two of these were discovered by direct fecal examination. The results of more recent work on a large scale are even more striking. They also found by making cultures at frequent intervals from a stool known to be infected that *Trichomonas hominis* remains alive at least seventy-nine hours in undiluted fecal material. That this species encounters considerable difficulty in reaching new hosts is indicated by the small percentage (about 3 per cent.) of persons infected. How food and drink become contaminated by these and other flagellates will be discussed later.

Transmission of Intestinal Protozoa in the Cyst Stage by the Contamination of Food or Drink

This is the most common method of transmission and is the way in which eleven of the fifteen human intestinal species succeed in invading new hosts. The percentages, in round numbers, of the general population that are estimated to be infected by these organisms, which give some idea of the effectiveness of this method of transmission, are as follows (the number of infections reported for those species for which percentages are not given is too small to furnish reliable figures):

	Per cent.		Per cent.
Endamœba histolytica	10	*Embadomonas intestinalis*	—
Endamœba coli	50	*Tricercomonas intestinalis*	—
Endolimax nana	25	*Giardia lamblia*	15
Iodamœba williamsi	10	*Isospora hominis*	—
Dientamœba fragilis	10	*Balantidium coli*	—
Chilomastix mesnili	10		

There is some evidence that racial differences exist with respect to susceptibility to infection with the various species listed; and there is no doubt that age has a distinct influence, since children are more often infected than adults. For example, about 40 per cent. of children have been found to be parasitized by *Giardia lamblia* and only about 15 per cent. of adults. Laboratory experiments likewise prove that the young of lower animals are more susceptible than adults of the same species.

The success of transmission depends to a considerable degree on the number of cysts discharged by the hosts and the ability of these cysts to live outside of the body until they are ingested by new hosts. The

[7] Hegner, R. W., and Taliaferro, W. H., 1924, "Human Protozoology." New York.

[8] Ponoschina, V. G., 1923, "On the Rôle of *Trichomonas vaginalis* in Human Pathology," *Russian Jour. Trop. Med.*, No. 1: 27–30.

[9] Brumpt, E., 1913, "Précis de Parasitologie." Paris.

[10] Hegner, R. W., 1925, "*Trichomonas vaginalis* Donné," *Amer. Jour. Hyg.*, 5: 302–308.

[11] Hegner, R. W., 1924, "Infection Experiments with *Trichomonas*," *Amer. Jour. Hyg.*, 4: 143–151.

[12] Hegner, R. W., and Becker, E. R., 1922, "The Diagnosis of Intestinal Flagellates by Culture Methods," *Jour. Parasit.*, 9: 15–23.

reproductive powers of parasites, including Protozoa, are enormous; for example, it has been estimated that a single person may pass several hundred million cysts of *Endamœba histolytica* in a single day. The viability of these cysts has been tested by the eosin method. One drop of one per cent. eosin solution is added to one drop of washed cysts; if the cysts take the stain, they are considered dead, if not, they are probably alive. It has been found that the cysts of *Endamœba histolytica* die within fifteen minutes if they are allowed to dry (Kuenen and Swellengrebel, 1913);[13] that they will live from sixteen days (Thomson and Thomson, 1916)[14] to one month (Wenyon and O'Connor, 1917)[15] in feces that are kept moist; that they will live for five months at room temperature if washed and kept in distilled water (Boeck, 1921);[16] that they will remain alive in the intestine of houseflies for about two days (Root, 1921);[17] and they will bring about infection in kittens after being stored at 2° C. for six days, but not after storage for two weeks (Sellards and Theiler, 1924).[18]

Our information regarding the cysts of other species is not so extensive, but we know that washed cysts of *Endamœba coli* will live for eight months; those of *Chilomastix mesnili* for six months; and those of *Giardia lamblia* for at least one month (Boeck, 1921);[17] and that the cysts of these species will remain alive within the intestines of flies for from sixteen to eighty hours (Root, 1921).[18] Furthermore, the thermal death point of these cysts has been found to range between 64° and 76° C., which is much above any temperature these organisms are subjected to in nature. Flies feed regularly on fecal material and the living cysts may pass through their digestive tract and be deposited on food or drink as early as seven minutes and probably up to three days after they are ingested.

[13] Kuenen, W. A., and Swellengrebel, N. H., 1913, "Die Entamöben des Menschen und ihre praktische Bedeutung," *Centrlb. Bakt. Orig.*, 71: 378–410.

[14] Thomson, J. G., and Thomson, D., 1916, "Some Observations on the Effect of Emetine Administration on the Free Vegetative Forms and Cysts of *Entamœba histolytica* and *Entamœba coli*," *Jour. Roy. Army Med. Corp.*, 26: 683–694.

[15] Wenyon, C. M., and O'Connor, F. W., 1917, "Human Intestinal Protozoa in the Near East," London.

[16] Boeck, W. C., 1921, "On the Longevity of Human Intestinal Protozoan Cysts," *Amer. Jour. Hyg.*, 1: 527–540.

[17] Root, F. M., 1921, "Experiments on the Carriage of Intestinal Protozoa of Man by Flies," *Amer. Jour. Hyg.*, 1: 131–153.

[18] Sellards, A. W., and Theiler, M., 1924, "Investigations concerning Amœbic Dysentery," *Amer. Jour. Trop. Med.*, 4: 309–330.

These data all favor the conclusion that there are sufficient cysts produced and that these are capable of withstanding temperature and other conditions outside of the host so that enough of them succeed in contaminating the food and drink of man to initiate new infections and thus keep the race from dying out. Moist conditions, an equable temperature and bad sanitation thus offer the most favorable conditions for the spread of intestinal Protozoa. These conditions occur more often among rural than among urban populations and most frequently in the tropics where soil pollution is customary.

The prevention of transmission of intestinal Protozoa is largely a sanitary problem. There is a tendency for these organisms to spread in families which is suggestive. Soiled hands, the common towel, food that is insufficiently cooked after becoming contaminated by food handlers in markets, restaurants and private homes all favor infection. Recently the thorough washing of uncooked food and its submersion for thirty seconds in water at 80° C. has been recommended as preventive measures for visitors to China (Mills, Bartlett and Kessel, 1924).[19] Efforts to control should be directed toward the elimination of soil pollution, the better disposal of human excrement, the screening of latrines from flies and the destruction of these and other insects, such as ants and cockroaches, that may be responsible for the contamination of food by fecal material containing cysts.

One fact of considerable biological interest brought out by these studies is the active rôle played by the host and the passive rôle of the parasite in transmission. We are accustomed to speak of these Protozoa as invading the host, but, as a matter of fact, they depend entirely on the behavior of the host for their transfer from man to man. Human beings not only contaminate their own food and drink, but carry the parasites by peristalsis into the large intestine, which is the normal habitat of all but *Giardia lamblia* and *Isospora hominis*. *Giardia lamblia* overcomes the action of peristalsis by seeking refuge among the villi of the duodenum, where it clings to the cells by means of its sucking disc, and the sporozoites of *Isospora hominis* escape from the current within the lumen of the intestine by penetrating cells in the intestinal wall.

Blood-inhabiting Protozoa

So far as is known all the blood-inhabiting Protozoa of man are transmitted by insects and each species of protozoon is carried by one or a very few species of insects. This has an important influence on the geographical distribution of this type of proto-

[19] Mills, R. G., Bartlett, and Kessel, J. F., 1924, Weekly calendar, Peking Union Medical College, June 3, 1924.

zoon. Intestinal species have been found wherever man exists, but blood-inhabiting species are restricted in their distribution to the habitats of their insect vectors. That part of this subject concerned more particularly with the entomological aspects of transmission is omitted from the following account and emphasis placed on the parasite itself.

TRYPANOSOMES

Trypanosoma gambiense and *Trypanosoma rhodesiense,* the organisms of African sleeping sickness, are usually transmitted by tsetse flies belonging to the species *Glossina palpalis* and *Glossina morsitans,* respectively. Other species of the genus *Glossina* may perhaps serve as vectors under favorable circumstances. If one of these flies bites an infected host and then within two hours bites a susceptible host, the latter may become infected by direct mechanical transfer, and biting flies of other genera may also bring about infection in this way; but the usual method of transmission involves a cycle of development in the body of the tsetse fly during which the trypanosomes are not infective. At the end of this cycle, which requires for *Trypanosoma gambiense* about three weeks and for *Trypanosoma rhodesiense* about two weeks, infective trypanosomes reach the salivary glands, where they remain until they are injected into a new host by the fly. Many lower animals, as well as man, are susceptible to infection with these species of trypanosomes and wild game, especially antelopes, are supposed to serve as reservoirs from which the tsetse flies may acquire their infection.

The third species of trypanosome of man, *Trypanosoma cruzi,* the organism of Chagas' disease in South America, is transmitted by a large bug, *Triatoma megista,* which lives in the crevices in the mud walls of native huts during the day and comes out to feed, usually on the face of the victim at night. Several other species of the genus *Triatoma* may act as vectors and a species of another genus, *Rhodnius prolixus,* may also be guilty, since it is known to be infected in nature. Just as in the other trypanosomes, *Trypanosoma cruzi* undergoes a cycle of development in the bug ending in infective forms; but these are located in the rectum and are voided by the insect at the time it bites. The person bitten gets some of this material into his mouth and ingests it or else the parasites gain entrance through the wound or through the mucosa of the nose and eyes. After much effort specimens have been found in the salivary glands of the bug, but it seems probable that infection very seldom occurs by inoculation.

LEISHMANIAS

The literature regarding the transmission of the *leishmania* Protozoa that cause kala-azar, oriental sore and espundia is very extensive, but most of it is inconclusive. Bedbugs, fleas and sandflies have been suspected, but at the present time it can only be stated that sandflies of the genus *Phlebotomus* are known to transmit *Leishmania tropica* of oriental sore and probably transmit those of the other two diseases as well.

MALARIAL ORGANISMS

The three species of malarial organisms are transmitted only by female mosquitoes of the genus *Anopheles.* A cycle of development takes place in the stomach cavity and stomach wall; and finally the infective stages, sporozoites, become lodged in the salivary glands and are injected into new hosts when the mosquito bites. The temperature of the air has a considerable influence on the length of the cycle in the mosquito. For example, the benign tertian parasite, *Plasmodium vivax,* completes its cycle in eight or nine days at the optimum temperature of 25 to 30° C; it requires ten to twelve days at 24° C; eighteen to nineteen days at 18 to 22° C and develops abnormally at 16° C and 35° C. The quartan parasite, *P. malariæ,* develops best at 22° C in eighteen to twenty-one days; and the estivoautumnal or malignant tertian parasite at 30° C in ten to eleven days. It is evident that if the temperature is too high (35° C +) or too low (16° C −) the mosquitoes do not become infected. This has an important bearing on transmission, since in countries where a temperature of 16° C or below prevails during the winter the malarial organisms are not carried over from the previous autumn in mosquitoes, but in man, which constitutes their only reservoir. It is therefore from man that mosquitoes receive their infection in the spring and if human reservoirs could be eliminated transmission would be impossible and malaria quickly stamped out.

Mosquitoes do not become infected unless they ingest a large number of the sexual stages in the life cycle of the parasite. A sufficient number of these would not usually be present if it were not for the phenomenon of relapse. The usual course of a malarial infection includes an acute period, during which the parasites multiply until enough are present in the blood to bring about symptoms; then a decrease in the number of parasites until none can be found in the blood by ordinary routine measures. This latent period may last for weeks or months; but often, because of some change in the host-parasite relationship, this period gives way to a period of relapse during which the parasites again increase rapidly in numbers and the patient again exhibits symptoms. A person may remain infected for years, suffering relapses from time to time. In many cases these relapses occur in

the spring just as the mosquitoes become active. It is obvious then that relapses are largely responsible for the transmission of malaria and for this reason the cause of relapse is the most important problem in malaria.

The three principal theories to acoount for relapse are: (1) the undeveloped sexual forms of the parasite (gametocytes) that remain in the blood after symptoms disappear are stimulated to develop parthenogenetically, thus bringing about an increase in the numbers of parasites; (2) resistant forms of the parasite lie dormant somewhere within the body until some change in their habitat awakens them to renewed activity; and (3) asexual reproduction continues, but most of the offspring are destroyed as soon as formed until the physiological state of the host becomes modified in some way that allows a greater number to survive than are destroyed, the result being an increase in numbers ending in another clinical attack. The third theory has considerable evidence in its favor, some of which will now be presented.

The organism of bird malaria, *Plasmodium præcox,* with which Sir Ronald Ross first demonstrated the transmission of malaria by mosquitoes, is a favorable species with which to carry on malaria studies because it can be grown *in vivo* in canaries and is easily transferred from one canary to another. Experimental work has been in progress on this species in the laboratory of medical zoology at the School of Hygiene and Public Health of the Johns Hopkins University since the school was established in 1918 (Hegner, 1926).[20] Our method of infection is to prick a vein in the leg, suck up a few drops of infected blood into a syringe containing saline or sodium citrate solution and inject it into the breast muscle or peritoneal cavity of a fresh bird. On the average, parasites appear in the blood of this bird about five days later; they increase for about five days when the maximum number is reached; and then decrease until in about five days more they again disappear from the blood.

From that time on the bird remains infective to other birds if its blood is used for inoculation, but the parasites are so few in the blood that they are not revealed by ordinary routine examination. Whitmore (1918)[21] had one canary that remained infective for twenty-nine months and Mazza (1924)[22] has recently reported a bird from which he obtained new infections after a period of four years and two months. Apparently the infection continues as long as the bird lives.

That asexual reproduction continues during the latent period after the acute infection subsides is evident from studies made by Ben-Harel (1923),[23] who was able to find stages undergoing asexual multiplication in spleen and bone marrow in five birds that were sacrificed at intervals of from two weeks to six months after the beginning of the latent period.

Further evidence that the strain is maintained during the period of latency by asexual reproduction was furnished by L. G. Taliaferro (1925),[24] who by patient search and statistical methods demonstrated that a periodicity exists in the asexual cycle of bird malaria; the cycle is twenty-four hours long in a strain obtained by Hartman in Baltimore. Not only does this periodicity exist during the acute phase of the infection but also during the latent period and throughout subsequent relapses. Segmentation in this strain took place at the same time every day (about 6 p. m.). Latency appears to be due, therefore, not to a retardation of asexual reproduction but to the destruction of young parasites; and relapse is due on the other hand to some change that prevents this destruction.

What conditions within the host are responsible for the changes that bring on relapses are now under investigation. We view the blood stream of the bird (and of man) as a culture medium and are attempting to modify infections by changing this medium. The fact that malarial organisms will live in artificial cultures only in the presence of sugar led us to attempt to increase and decrease the sugar content of the blood (1) by feeding sugar to increase the amount and (2) by treating birds with insulin to decrease the amount present. Our results so far (Hegner and MacDougall, 1926)[25] have been surprisingly successful. Preliminary experiments indicate that in birds that are given daily doses of insulin the infection is not so severe as in controls, and that when sugar is regularly fed to birds each day during the hours when segmentation is going on the parasites continue to in-

[20] Hegner, R. W., 1926, "Studies on Bird Malaria," *South. Med. Jour.*, May, 1926.

[21] Whitmore, E. W., 1918, "Observations on Bird Malaria and the Pathogenesis of Relapse in Human Malaria," Johns Hopkins Hospital Bull., 29: 62–67.

[22] Mazza, S., 1924, "On the Duration of Relative Immunity in Malaria of Birds," *Jour. Trop. Med. and Hyg.*, 27: 98–99.

[23] Ben-Harel, S., 1923, "Studies of Bird Malaria in Relation to the Mechanism of Relapse," *Amer. Jour. Hyg.*, 3: 652–685.

[24] Taliaferro, L. G., 1925, "Infection and Resistance in Bird Malaria with Special Reference to Periodicity and Rate of Reproduction of the Parasite," *Amer. Jour. Hyg.*, 5: 742–789.

[25] Hegner, R. W., and MacDougall, Mary S., 1926, "Modifying the Course of Infections with Bird Malaria by changing the Sugar Content of the Blood," *Amer. Jour. Hyg.*, 6: July, 1926.

crease beyond the maximum number characteristic of normal infections and the infection eventually ends in the death of the birds. No analyses have yet been made of the sugar content of the blood of these birds and other factors no doubt play a rôle in relapse, but our experiments are very suggestive and we hope when carried further will help solve the problem of relapse, which, because of its bearing on transmission, is responsible for the continued existence of malaria.

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LONDON.

SCIENTIFIC EVENTS

THE EXPEDITION OF THE CALIFORNIA ACADEMY TO THE REVILLAGIGEDO ISLANDS

THE 1925 Expedition of the California Academy of Sciences to the Revillagigedo Islands, Mexico, is reported to have been most successful in every way. In addition to the collections made covering practically every phase of life on these islands and points on the mainland, there were several items of more than passing interest, notably the establishing of seven new geographic names. These names have been adopted by both the United States and Mexican governments and are as follows:

Angulo Rock.—A small, outlying, flat-topped rock immediately northeast of Asuncion Island, Lower California. It is named for Captain Victor Angulo, Commander of the Mexican National Patrol Vessel, *Presidente.*

Mount Gallegos.—The highest mountain on Clarion Island of the Revillagigedo Group. Chart No. 1688 of the United States Hydrographic Office gives the elevation of this mountain as 1,100 feet. It is named in honor of the late Professor Jose M. Gallegos, explorer for the government of Mexico and a member of the party which, in 1925, explored this mountain.

Mount Evermann.—The central peak of Socorro Island of the Revillagigedo Group. Named for Dr. Barton Warren Evermann, the distinguished director of the California Academy of Sciences and the organizer of this and many other expeditions in which the academy has actively cooperated with the government of Mexico.

Grayson's Cove.—There is a little cove at the west end of Cornwallis Bay, Socorro Island, as shown on Chart No. 1687, of the United States Hydrographic Office. Here, in 1867, Colonel A. S. Grayson's sloop was wrecked. It is the only known supply of fresh water on the island and the suggestion has been made that it be so marked on future charts.

Point Old Man of the Rocks.—This name was given by Colonel Grayson to the point of rocks which formed the eastern boundary of the little cove when he found fresh water.

Ash Heap.—At the south end of San Benedicto Island the highest elevation is attained, 975 feet. This elevation or peak is composed almost entirely of soft volcanic ashes, hence the name.

Herrera Crater.—The central peak of San Benedicto Island is indicated on Chart No. 1687 of the United States Hydrographic Office as being 683 feet high. This peak is named in honor of Professor Alphonso Herrera, the director of the National Museum of Mexico. Professor Herrera took an active part in the expedition.

HAWAIIAN ACADEMY OF SCIENCE

FOLLOWING the Pan-Pacific Food Conservation Conference, held in Honolulu in August, 1924, a committee was appointed by the American Association for the Advancement of Science, with Dr. L. O. Howard as chairman, to consider some form of cooperation between that organization and the Pan-Pacific Union, under whose auspices the conference was held. In accordance with the recommendations of this committee, a meeting was called of the members of the association residing in Hawaii, to consider the formation of a local organization. A committee was appointed at this meeting to formulate means for a permanent organization.

After some correspondence with the committee of the American Association for the Advancement of Science in Washington, and several meetings of the local members of the American Association for the Advancement of Science, the Hawaiian Academy of Science was organized on July 23, 1925, and a constitution was adopted. The following officers were elected at that time:

President, Dr. Frederick C. Newcombe.
Vice-president, Dr. C. Montague Cooke, Jr.
Secretary-Treasurer, Mr. Edward L. Caum.
Councilors, Mr. Otto H. Swezey, Professor Frederick G. Krauss.

During the year 1925–26 three public meetings of the academy were held, to hear visiting scientists. On November 9, 1925, Dr. C. P. Berkey, geologist of the American Museum of Natural History's Third Asiatic Expedition, spoke on "Evidence of Change of Climate in the Gobi desert." On January 7, 1926, Dr. Edwin G. Conklin, of Princeton University, spoke on "The Mechanism of Evolution." On March 29, 1926, Dr. Carl M. Meyer, of the Hooper Foundation, San Francisco, spoke on "Food Poisoning and Food Infection."

The First Annual Meeting was held May 19 to 22, 1926. Dr. Newcombe gave the presidential address on "A Field for the Hawaiian Academy of Science" and a program of forty scientific papers was presented.

Following the program on May 22, a business meeting was held at which five resolutions were adopted,