A NEW FACTOR IN THE ETIOLOGY OF MALARIAL FEVER, INDICATING NEW METHODS OF TREATMENT.

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The now well-known facts that malarial fever is a parasitic affection and that its paroxysms are produced by the sporulation of successive groups of parasites in the blood compel us to reconsider some of our former ideas as to the influence of meteorological factors in the etiology of the disease. Especially is this true with regard to the *heat* of the sun, which has been so long accorded paramount importance as an etiological factor. That solar heat generates fever "miasma" by evaporating swamps, etc., is no longer to be admitted. The dreadful mortality among Europeans—especially of European soldiers marching in the sun—in tropical regions, may have been *partly* due to malarial fever, but no doubt *largely* due to "thermic fever" or insolation. Which of these two components has been most fatal in the past must remain unknown. During the month of August, 1896, there were more than two thousand deaths from sunstroke in nine cities of the United States.[1] The mortality in the tropics must be still greater.

That a certain elevation of temperature is necessary to keep alive and maintain the inoculating activity of infected mosquitoes is true for all seasons and places; but, further than this, there seems to be no scientific evidence that solar heat is in any way a factor in the etiology of malarial disease. The malarial parasite cannot be affected by external temperature, for it is submerged in a fluid—really a blood bath—of 98.2° F. In a healthy human "host;" and in a malarial patient, while the temperature may reach 104°–106° F. during the few hours of the cold and hot stages of a febrile paroxysm, it is, during the much longer period of the intermission, usually *sub*normal, between 97° and 98° F., or thereabout.

The white man is most liable to this disease, the black least so; but the black skin of the negro absorbs heat like a black coat; thus there occurs the anomaly of the hottest individual being the fittest to survive in regions liable to fever from alleged solar heat.

The island of Tahiti is situated almost under the thermal equator and only 18 degrees from the geographical equator; yet the Tahitians are "free from marsh fevers."[2]

Dr. Manson,[3] in his excellent treatise, says the more we know of these diseases the less apparent becomes the rôle of temperature *per se* as a pathogenic factor and the more important the tropical fauna.

Dr. Celli,[4] in his recent (1900) work, observes that temperature and malarial fever do not run exactly parallel; and he reproduces a table

of Tacchini's covering twelve years, from 1871 to 1882 inclusive, regarding fever and weather factors, in which we see "that in the year 1879, when a true malarial pandemic raged, the mean temperature for the months of July and August was the *lowest* of these years."

If the solar heat theory be untenable, may we not explain the undeniable relation between hot climates and malarial fever by eliminating the term "heat" and substituting that of "light?" Suppose we say it is the light of the sun, not its heat, that determines the periodic sporulation of parasites in the blood? I propose this idea as a working hypothesis, and the evidence in support of it will be comprised in the following statements:

1. *The accumulated experience and observations of centuries, which have been held to prove the agency of solar heat in causing malarial fever, may be held to prove the agency of solar light.* The light and heat of the sun are inseparable. The "burning" sands of Africa must also be "dazzling" sands. So with hot seasons; the cloudless skies of a hot, dry summer diffuse an intense and brilliant light. We cannot conceive how external heat can warm the parasite in human blood; but that light can penetrate the skin and act upon the plasmodium to promote its development—as light is known to act on other amœbæ—is quite intelligible.

2. *Paroxysms of intermittent fever will not, as a rule, take place at night, in the dark.* Numerous authors support this statement. Wood[1] says: "It is worthy of observation that when the anticipating and advancing paroxysm reaches the period of darkness, it is either apt to be arrested in its course or to leap over the night backward into the evening or forward into the morning."

Flint[4] remarks: "Paroxysms may occur at any time of the day. They very rarely occur during the night."

"It is *entirely unknown*," says Fordyce,[3] "upon what this depends; indeed, the observation has been little attended to by any author who has not frequently seen the disease." The successive paroxysms getting an hour or two later may, however, be explained by the patient (languid from the paroxysm of the previous day) lingering in bed an hour or two later than usual, thus depriving the parasites of an hour or two of morning sunlight; as Flint[4] tells us, "a paroxysm is sometimes warded off by taking to the bed before the hour."

3. *The relative liability and relative immunity of different races of men to malarial fever depend upon the relative translucency or non-translucency of their skin, and probably of their blood.* All agree that dark-skinned peoples, in which Welch[5] includes "Negroes, Arabs, Indians, and Tamils," enjoy a relative immunity; and this has never been explained except by "specific idiosyncrasy" (a term without meaning) or by acclimation. Moreover, hundreds of negroes in the United States have the

disease, notwithstanding their alleged idiosyncrasy. Will the relative transmissibility of the skin to light clear up this difficulty?

In recently examining over a hundred negroes of different colors in Washington, to determine the translucency of their skin (easily done by passing the rays from a pocket electric flash-light through the external ear), I was surprised to find that *light passed through the skin almost as readily as through the skin of a white man.* In only three of the whole lot did I find the skin *absolutely impenetrable* to light. These were very dark, though not as black as genuine Africans, and, while they had lived in malarial places and had been bitten by mosquitoes, they had never in their lives (all were middle-aged) had ague. The affirmative evidence of these few cases, however, becomes of little real value, since there were other individuals with *translucent* skins who had *also* never had the disease.

It appears, therefore, that some negroes are not more immune than white people, because their skin, though of darker hue to an outside observer, is really not much more impenetrable to light than the skin of a white man. If my theory be correct, future experiment in tropical countries, etc., may be expected to demonstrate that individuals enjoying immunity will be found to have skins that will not allow the transmission of light.

White men who become black from malarial melanosis (cases are reported by Fails[10] and Gordon[11]), and white races becoming black after living for generations in malarial countries, may be considered instances of conservative structural modification to suit new and abnormal conditions, thus conforming to the general principle of adaptation to environment common to living organisms.

Finally, no white skin is impenetrable to light, and immunity in a white person is, so far, unknown.

Whether the *blood* of the negro is too dark, in the tropics, to admit light may be worth investigation. Cartwright[12] gives numerous authorities who affirm that the blood of negroes *is* blacker than that of whites.

4. *In places where malarial fever prevails the disease is increased by bright, sunny weather and lessened by clouded skies.* Observations on this point are plentiful, but they have been ascribed to "salutary rains" instead of to a clouded sky. Jackson[13] speaks of fever-stricken troops being benefited by three days of rain, and of a camp "over which *fog hung* until late in the day being better off than another placed in a dry and elevated situation." Of Trinidad, Ferguson[14] says: "It always rains nine months in the year; if it only *rained* eight, or if at any time there was a cessation of the preserving rains, the worst kind of fevers were sure to appear." In Tacchini's table, quoted by Celli, the year 1879, in which the percentage of fever was *greater* than in any other of the twelve years tabulated, is shown also

to have had the *least cloudiness* of all those years. It appears, therefore, that when the light of the sun is veiled by rain-clouds and fog, the parasites get less light and their sporulation is retarded. Conversely, the brighter the light the greater the liability to fever; hence the great liability of "sailors and fishermen" (Osler[14]) and of "berry-pickers" (Welch[15]).

5. *It has long been a popular tradition that to prevent the occurrence of ague, or to forestall its recurrence when it has once occurred, it is advisable to keep in the shade and avoid sunlight.* Persistent traditions have a certain amount of evidential value. Manson tells us the peasants of Italy and natives of German East Africa believed *for centuries* that fevers were caused by the bites of mosquitoes. So, now, the popular idea that sunshine will bring on a "chill," and shade prevent it, may be really true.

Osler, Thayer, and others affirm that intermittent fever is spontaneously curable without medicine. But in these cases of spontaneous recovery the patients were, presumably at least, shaded by their hospital surroundings from the light of the sun. That rest, food, and improved nutrition on the part of a human "host" should kill parasites in the blood seems unreasonable; and that being housed in bed prevents sporulation of the parasite, because of protection from the sun's heat, would force us to the admission that while so housed a man's blood is *too cold* to allow sporulation. Protection from *light* seems to be the only constant factor by which the spontaneous recoveries become explicable.

6. *The malarial parasite is a naked amœba. Red light promotes the vital activities of amœbæ, while violet or purple light restricts them. The color of the light diffused through the blood is necessarily red.* Experiments of Harrington and Lenning[16] on the common amœba proteus show that its protoplasm may be set in motion, or brought to rest, by varying the *color* of the light to which it is exposed. Their concluding summary is as follows:

1. Amœba streams in the presence of red light.

2. Streaming is retarded, stopped, or reversed by rays from the violet end of the spectrum.

3. Further, the effectiveness of the following kinds of light as inhibiters of protoplasmic flow diminishes in the order named—white, violet, red.

4. Enucleated amœbæ stream in red light and cease to stream in violet or white light.

If the plasmodium malariæ be a light-loving organism, red light would seem to be its natural requirement while in the blood, and we might expect to find its activities inhibited, like amœba proteus, by light from the violet end of the spectrum. This, in fact, we *do* find. Prussian blue (an old remedy for ague), and recently methylene blue, seem so far to inhibit protoplasmic movement in the malarial parasite as to pre-

vent its sporulation and thus cut short the ague paroxysms. Cases have been reported[11] by Mya, Thayer, Hoinci, Thintignan, Huddleston, of New York, and Mühll, of Basle, and others. If methylene blue stains the parasite or its nucleus, this blue, commingling with the red of the blood, would produce the (to the parasite) disastrous violet or purple.

Possibly the (hitherto enigmatical) curative action of sulphate of quinine may be due to its remarkable *fluorescence*. In solution it intensifies the violet, and even renders the ultra-violet rays of the spectrum perceptible to human vision.

Of the few vegetable products having this quality of fluorescence, another one is esculin, the bitter principle of the horse-chestnut tree bark (*Æsculus hippocastanum*). But this also has been successfully used as an antiperiodic for intermittent fever.[12] Do these fluorescent substances act by intensifying violet rays in the blood?

If the etiology now given be correct, treatment is self-evident, viz., keep malarial patients in the dark, or in rooms with purple or indigo windows, and clothe them with garments impenetrable to light; in the tropics, with white clothing lined with purple or black. Give drugs that darken the blood or render it violet, or lessen its translucency.

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ANGINA PECTORIS.

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Angina pectoris, in its typical form, is in my experience a very rare disease. Pseudo-angina, or what resembles it at times, cardiac asthma, is not infrequent. Although angina pectoris is described among the neuroses of the heart by authors, this view in my judgment