

***Endemic malaria in San Antonio de Pintuyacu,
Loreto, Peru in 2002:
Behavioral factors that make a difference***

by Christine Beier

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to Dr. Claud Bramblett

*ANT 392: Graduate Physical Anthropology
University of Texas at Austin*

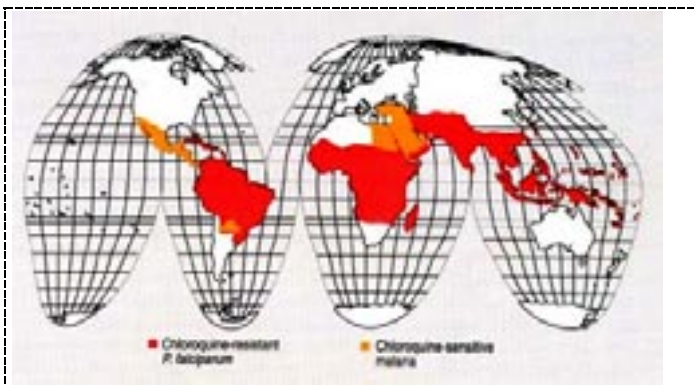
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http://www.iqito.org/malaria_2002.pdf

States, through USAID, gave \$790 million to the Global Eradication of Malaria Program. By 1967, however, the WHO realized that “the global eradication of malaria was impossible for a variety of reasons” and the focus shifted to control of the disease. (Desowitz 1991)

The impact of the WHO’s eradication program on malaria was significant, but the disease and its mosquito vector have steadily regained strength since the 1970s. In fact, the patterns of use and misuse of DDT and antimalarial drugs during the course of the second half of the twentieth century have directly led to the emergence of more virulent, drug-resistant strains of the disease and insecticide-resistant mosquitoes around the globe. By the 1990s, malaria had once again become a focal issue in world health, leading to public health crises throughout Africa as well as in other tropical zones including lowland Peru. In 1998, the WHO initiated the “Roll Back Malaria” program, which is described as a “global partnership to halve the world’s malaria burden by 2010”. (<http://mosquito.who.int>)

International attention to malaria, in the forms of both research and resources – specifically of health agencies and programs such as the World Health Organization (WHO), the International Development Research Centre (IDRC), the Centers for Disease Control and Prevention (CDC), and the “Roll Back Malaria” initiative – is overwhelmingly focused on Africa, and rightly so. Fortunately, while many epidemiological factors between any two malarial zones are non-identical, many strategies developed in and for Africa are applicable in malarial zones in tropical South America *if* new information from Africa has some way of traveling between the two continents.

In addition to discussing malaria’s abstract existence as a series of facts and statistics about a worldwide phenomenon, in this paper I would like to focus our attention on the lived reality of malaria in the northern Amazon Basin of Peru. In one sense – the sense you might derive from doing online research on malaria – malaria wreaks havoc on 300 to 500 million nameless people every year and is the focus of elaborate global institutional analyzing and strategizing. In another sense, however, malaria impacts individual lives, affecting families and communities in a variety of ways that usually includes days or weeks of illness, but also includes the deaths of babies and elderly people as well as the disruption of subsistence activities for entire economic units: people sick with malaria can’t work, men can’t hunt, women can’t care for children, children can’t attend school.



© www.malariasite.com/malaria/whatismalaria.htm

1. From the global...

According to current statistics, the World Health Organization (WHO) estimates that at least 300 million people are afflicted with malaria annually and at least 1 million people die from the disease each year. Due to the impact of malaria on families and communities, malaria is estimated to impact 40% of the world’s population. At present, an estimated 90% of deaths from malaria occur in sub-Saharan Africa. The majority of those who die from malaria are children under five years of age, and among those the vast majority are children who live in poverty. In the last decade, incidence of malaria has increased dramatically in all tropical countries. (cf. <http://www.who.int>)

Malaria is thought to have emerged in human beings in Africa and has afflicted us for thousands of years. An intensive worldwide malaria eradication program launched by the WHO in the mid 1950s substantially decreased incidence of malaria through the spraying of the insecticide DDT. According to malaria historian Robert Desowitz, between 1956 and 1969 the United

In the range of discourse from political speeches to personal conversations, people throughout the world often characterize malaria as a ‘social’ phenomenon, a problem whose solution must be brought about by national ministries of health, regional hospitals, community health workers, and/or other representatives of governmental institutions. To a significant degree, this characterization is accurate; without large-scale interventions by governing entities, malaria cannot be kept under control. (cf. www.minsa.gob.pe) In particular, as both human beings and disease vectors like mosquitoes become more mobile as a result of ‘globalization’ in the ‘jet age,’ the scope of the problem of infectious diseases realistically includes the entire planet as a single ecosystem. At the same time, however – and this is my point of departure for the rest of this paper – malaria is still suffered by individual human beings; and therefore the individual is still and always capable of local interventions against the spread of malaria.



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2. ... to the local

The geographical center of this paper is San Antonio de Pintuyacu, located on the Pintuyacu River in the department of Loreto in the northern region of Peruvian Amazonia. San Antonio is an indigenous Iquito community with a population of approximately 200 residents; the greater San Antonio area, so to speak, has a population of approximately 500 people¹. San Antonio

¹ It is estimated that there were at least 10,000 Iquito people in Loreto at the time of the arrival of Europeans in that region in the seventeenth century. Since then, disease and genocide resulting from



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was founded at the turn of the twentieth century and is now the only Iquito community in Loreto.

The motivation for the present study of malaria in San Antonio results from my personal experiences in San Antonio de Pintuyacu in July of 2001 and between June and August of 2002. San Antonio is the site of the Iquito Language Documentation Project, a long-term language documentation and revitalization project of which I am a co-founder. Together with my research partner Lev Michael, I first visited San Antonio in July of 2001 to initiate a relationship with the community and to investigate the possibility of launching a language revitalization project there. As a result of those initial investigations and negotiations in 2001, Lev and I returned to San Antonio in June 2002 along with two other graduate students from the University of Texas at Austin and spent an intensive eight weeks carrying out the first phase of the Iquito Language Documentation Project.

Malaria is endemic to San Antonio at present. During my eight-week stay in 2002, it was the principal illness affecting community members, afflicting about half of the adults with whom our research team had regular contact. Community members recounted to me that several children had died in recent years of cerebral malaria, and in the mid 1990s about half of the elderly fluent Iquito speakers died in an unanticipated and severe outbreak of malaria. In addition, one of our project’s four team members, Lev, fell ill with malaria while in San Antonio, driving home to me the gravity of the present health situation in the community. Below as I develop my discussion, I will refer to Lev’s set of

missionization and slavery have reduced the number of ethnic Iquitos to approximately 500 people.

experiences with malaria as a key source of data for several assertions I make.

The explicit purpose of this paper is to generate concrete strategies to reduce the incidence of malaria in San Antonio de Pintuyacu, focusing on behavioral strategies that can realistically be implemented by me, the ILDP research team, and residents of San Antonio, with minimal expense and maximal probability of success, during the two eight-week phases that the ILDP team will spend in San Antonio between June and August of 2003 and 2004.

3. The disease

According to the International Development Research Centre, “Deadly fevers – probably malaria – have been recorded since the beginning of the written word (6000-5500 B.C.)... There are no references to malaria in the “medical books” of the Mayans or Aztecs. It is likely that European settlers and slavery brought malaria to the New World and the awaiting *anophelines* within the last 500 years.” Not until 1880 was the malaria parasite first seen under a microscope. In December of 1897, the discovery of “malaria cysts in the stomach wall of *anopheline* mosquitoes” was first reported and by July of 1898 malaria transmission through the mosquito was established.” (www.idrc.ca/books/reports/1996/o1-05e.html)

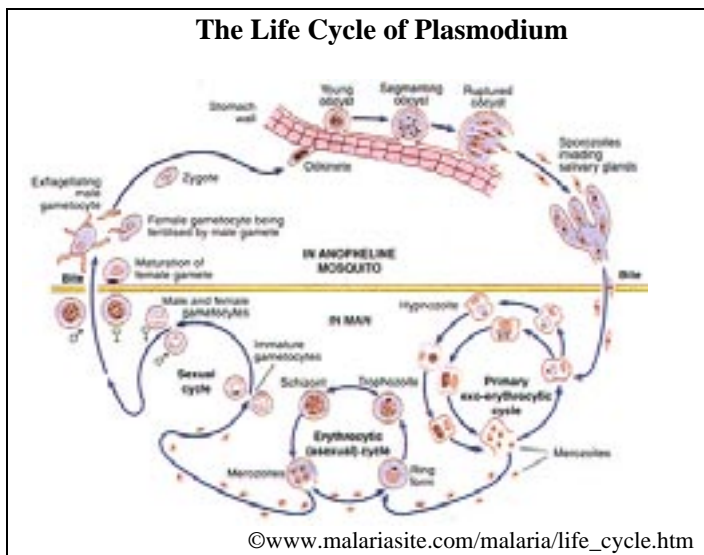
malariae species, but the vast majority of slide-positive diagnosed cases are of the first two species. For example, in 1997, of 158,115 reported cases of malaria, only 44 were reported as *P. malariae*, the rest being roughly half *P. vivax* and half *P. falciparum* (Aramburú et al 1999).

After a person is infected through the bite of an anopheles mosquito, the Plasmodium parasite incubates in the human host for 8 to 25 days (incubation period varies across species) before symptoms appear. The high fever associated with malaria kills the blood phase of Plasmodium. While the malaria patient is in fever, the blood is full of Plasmodia; this is the only phase of the disease in which it may be transmitted to another person if the malaria patient is bitten by another mosquito. (http://www.malariasite.com/malaria/life_cycle.htm)

The most common symptoms of malaria are severe headaches accompanied by bouts of chills followed by high fever (usually 40° C or more). Malaria has a distinctive fever pattern that makes it relatively easy to recognize: a short bout of chills is followed by a high fever and delirium. After several to many hours, the person begins to sweat profusely and the fever drops abruptly. Once the fever breaks, the person feels weak but relatively well. A day or more may pass before the next bout of headache, chills, and high fever. Between attacks, the person often does not feel ill (cf. www.cdc.gov; Werner 1997).

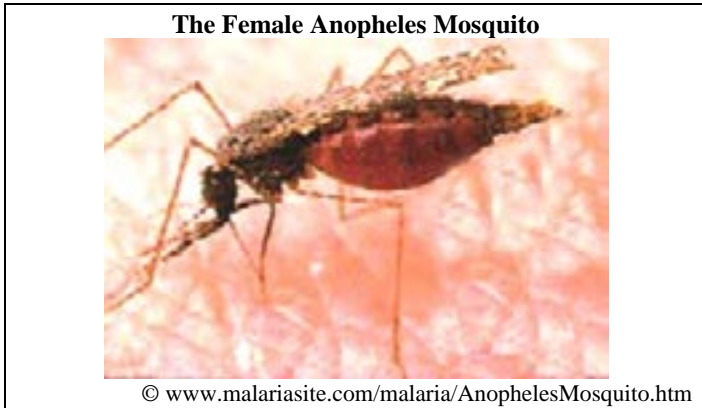
Despite the fact that the pattern described here is very common in malaria attacks, both the onset and the progress of the disease can vary significantly. Therefore, any person with a high fever in a malarial zone should be tested for malaria. If left untreated, *P. vivax*, *P. ovale*, and *P. malariae* may recur for months or years. Severe *P. falciparum* can cause kidney failure, liver failure, convulsions, coma, and death. (cf. www.cdc.gov, UT-Austin University Health Services). Children are particularly at risk of contracting cerebral malaria. According to Dr. B. S. Kakkilaya, “The case fatality of *P. falciparum* malaria is around 1 per cent and this accounts for 1 to 3 million deaths per year all over the world. 80% of these deaths are caused by cerebral malaria.” (<http://www.malariasite.com/malaria/Complications2.htm>)

The best and most common method for diagnosing malaria is the MP smear, known as *gota gruesa* in San Antonio. A small sample of blood is taken from the patient and examined under a microscope. The trained technician will see and count the number of Plasmodium parasites on the slide and thereby diagnose the species of Plasmodium and the severity of the case depending on the concentration of parasites in the smear. A number of



Malaria is an infectious vector-borne disease caused by parasites called Plasmodia. Four species of Plasmodia that cause human malaria have been identified: *Plasmodium vivax*, *P. falciparum*, *P. ovale*, and *P. malariae*. The department of Loreto in Peru is demonstrated to have the *P. vivax*, *P. falciparum*, and *P.*

factors can affect the accuracy of this test and false negatives are not uncommon; however, the MP smear is still considered far more reliable than any competing diagnostic. (<http://www.malariasite.com/malaria/diagnosisofmalaria.htm>)



4. The vector

All vector-borne diseases “require an intermediate living agent for their transmission.” (Brêtas 1996). The necessary vector for malaria is a female mosquito of the genus *anopheles*. Of the 380 known species of the genus *anopheles*, 60 species are known to transmit malaria among humans. Of the six known anopheline vectors in Loreto, *Anopheles darlingi* is the most common; in 1997, *A. darlingi* represented more than 90% of the anophelines found in the Iquitos area during the rainy season. (Aramburú et al 1999)

Mosquitoes pass through four distinct phases in their life cycle: egg, larva, pupa, and adult. The adult mosquito lives for two to three weeks, although some species are said to live four to eight weeks. The female anopheles mosquito bites human beings for the blood meal she needs to nourish her eggs. Anophelines lay their eggs in virtually any collection of standing or slow moving water. The eggs develop into larvae, then pupae, then adults in the water environment in 7 to 10 days. Crucially, *only* the adult female anopheline mosquito carries the malaria parasite as a result of feeding on an infected human being², therefore controlling mosquitoes’ access to people with malaria is a decisive factor in reducing transmission of the disease. Antilarval interventions are considered to be the most effective means for reducing the mosquito population. Efforts to reduce the availability of appropriate water environments are widespread in malarial zones, but in Peruvian Amazonia and in San Antonio in particular,

² Male anophelines do not feed on blood; they feed on plant nectar.

slow moving river water is a permanent, unchangeable, and highly attractive breeding site for anophelines.

Anopheles mosquitoes are active at night and hide during the day. Though they are most visible at dusk, when seeking out and entering human dwellings, most of their feeding activity takes place between 10 pm and midnight. According Aramburú et al, “*A. darlingi* in the Iquitos area bite indoors or outdoors from dusk to midnight, with a small dawn peak.” (Aramburú et al 1999) Bed nets are therefore a crucial intervention in preventing mosquitoes’ access to human beings.

Annual Parasite Index in Loreto by district per 1000 population

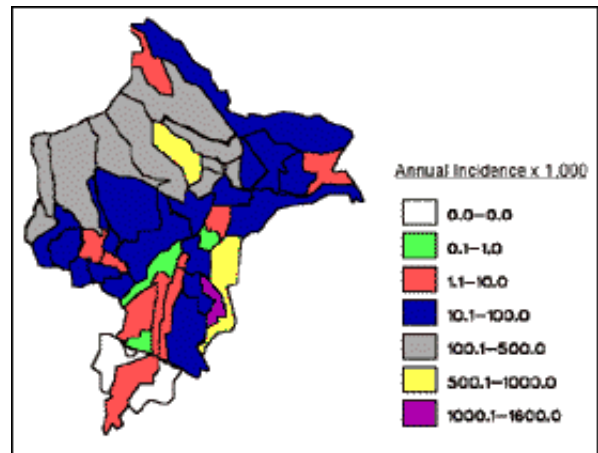


Image source: Aramburú et al 1999

Malaria Incidence in Loreto, 1992-1997

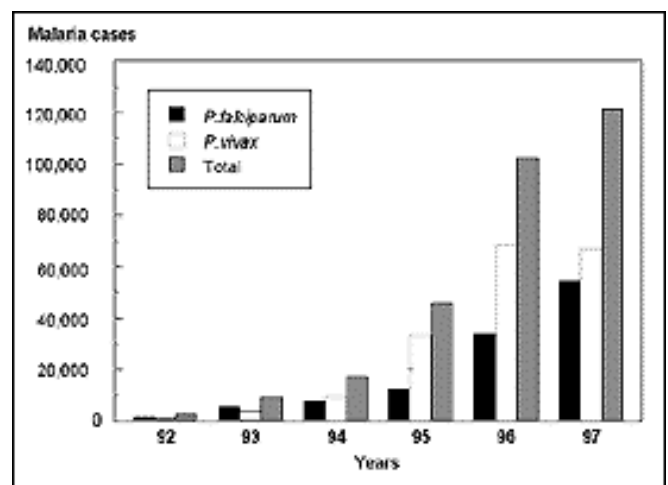


Image source: Aramburú et al 1999

5. The return of malaria and the epidemic in Loreto

After intensive mosquito eradication efforts largely based on the spraying of DDT in Peru in the 1950s and 1960s, incidence of malaria in Peruvian Amazonia remained relatively low through the 1970s and 1980s.

(www.minsa.gob.pe) According to Aramburú et al, in “1988, no cases of *P. falciparum* were reported in Loreto. In 1991, 140 cases were reported...the number of malaria cases in 1997 reported by slide or clinical definition was 158,115.” “From 1992 to 1997, malaria increased 50-fold in Loreto” and increased fourfold in Peru as a whole. (Aramburú et al 1999) In Peru as around the world, mosquito eradication measures were thought to have ‘won the battle’ against malaria, and attention to both mosquito control and malaria prevention dwindled. The overwhelming resurgence of malaria in Loreto in the five years between 1992 and 1997 is evidence of the lack of preparedness of the Peruvian Ministry of Health to deal with the resurgence of malaria. (cf. www.minsa.gob.pe) On the local level, the malaria crisis that began in the mid 1990s has taken a tremendous toll on the community of San Antonio and has yet to be resolved.

A core set of factors has been identified that have led to the increase of malaria around the world. Brêtas 1996 lists the following factors: “mosquito resistance to pesticides, parasite resistance to drugs, changes in land-use patterns, and reductions in funding and manpower dedicated to control activities. Most of the determinants are heterogeneously distributed, changing over both space and time.” *Fact sheet #94* published by the WHO’s Roll Back Malaria Program states,

“Factors such as topography, temperature, rainfall, land use, population movements, and degree of deforestation have a profound influence on the temporal and spatial distribution of malaria vectors and malaria. Factors which may precipitate a malaria epidemic fall into two categories: natural (climatic variations, natural disasters), and man-made (conflict and war, agricultural projects, dams, mining, logging). Most of these factors modify the physical environment, and increase the capacity of mosquitoes to transmit malaria. Some factors also result in massive population movements that expose non-immune populations to malaria infection.” (<http://www.who.int/inf-fs/en/fact094.html>)

At www.malariasite.com, Kakkilaya summarizes the following set of factors that facilitate the spread of malaria:

<i>Manmade reasons</i>	Complacency and laxity in anti malarial campaigns; conflicts and wars; migrations; deteriorating health [care] systems; poverty
<i>Parasite</i>	Drug Resistance
<i>Vector</i>	Insecticide Resistance and ? ban on DDT
<i>Environment</i>	Global Warming – increased breeding and life span of the insect vector
<i>Jet Age</i>	Shrinking World–spread of malaria from endemic areas to all other parts of the world

Most of these various factors are relevant in the case of Loreto in the last decade, with the exception of war and dam-building. I would like to focus on two unique manifestations of these general trends that are pertinent in the case of Loreto. First, lowland tropical rainforest is a mosquito’s paradise. Slow-moving rivers like the Pintuyacu provide excellent breeding grounds for anophelines³ and the long seven to eight months of the equatorial rainy season are always accompanied by increases in malaria and other infectious diseases. Even more clearly than in many regions, mosquito eradication is an impossibility in San Antonio – nor would it be desirable, due to the devastating long-term impact mosquito eradication would have on other species in the ecosystem.

Second, in Peruvian Amazonia, a major factor in the spread of malaria is the mobility of workers in the natural resource extraction industries. These are mostly men working as woodcutters, in petrochemical extraction, or in gold dredging. A number of doctors and nurses working in healthposts in Peru’s Amazonian regions have explicitly told me that many outbreaks in indigenous communities of malaria and other infectious diseases including influenza, uta or leishmaniasis, bartonellosis, hepatitis and cholera (in the 1980s and 1990s), have been traced to contact between visiting laborers and local residents. In San Antonio’s case, over the last several years, Brazilian gold-dredgers have been visiting the community on a regular basis, on their trips up to and down from the headwaters of the Pintuyacu.

6. A brief history of chemical antimalarials

The group of antimalarial drugs most commonly used by Europeans and their descendants consists of quinine and the synthetic drugs based on quinine. Quinine is a “toxic plant alkaloid made from the bark of the Cinchona tree” that is indigenous to South America. According to the IDRC, “Jesuit missionaries in South America learned of the antimalarial properties of the bark of the Cinchona tree and had introduced it into Europe by the 1630s and into India by 1657.” Later, the “Dutch bought Cinchona seeds from a British trader...who brought them from Peru. They established Cinchona plantations in Java (Indonesia) in the mid 1800s and soon had a virtual monopoly on quinine.” (Desowitz 1991)

³ The Pintuyacu is a blackwater river. Blackwater rivers are somewhat less habitable for insect eggs and larvae than the whitewater rivers of Amazonia, due to high levels of tannins leached from plant matter into the river.

When Java was captured by the Japanese in the second World War, quinine effectively became unavailable to Europeans. This spurred research on synthetic antimalarial drugs and by 1943, Chloroquine became available. Chloroquine is a synthetically manufactured drug of “the class of compounds known as 4-amino quinolines” and it quickly became the most commonly used antimalarial drug. Worldwide use, and misuse, of chloroquine led to the appearance in the 1960s of chloroquine-resistant strains of *P. falciparum* malaria. Eventually quinine was completely synthesized; Mefloquine is the “synthetic analogue” of quinine. (Desowitz 1991) Primaquine was first synthesized in 1926 and is effective against malaria only when used in conjunction with other antimalarial drugs. (cf. www.inchem.org)

According to the International Programme on Chemical Safety (IPCS at www.inchem.org), “Chloroquine and its 4 amino-quinoline congeners block the enzymatic synthesis of DNA and RNA. Chloroquine is an excellent blood schizonticide but has no effect on secondary tissue schizonts and on sporozoites. It inhibits the erythrocytic stage of development of plasmodia. Thus relapses may occur after cessation of the treatment.” According to IPCS, Primaquine is effective for “elimination of primary and secondary exoerythrocytic stages of *P. vivax*, *P. malariae* and *P. ovale* and the primary exoerythrocytic forms only of *P. falciparum*. Primaquine must always be given in conjunction with full doses of a 4-aminoquinoline... [for] the elimination of gametocytes of all species.”

Doxycycline (a form of tetracycline) is a commonly used and effective antimalarial for chloroquine-resistant strains of Plasmodia. “Tetracyclines are bacteriostatic agents, supposedly acting by inhibiting protein synthesis” in the blood. Its side effects are few, it is relatively inexpensive, and it is widely available, making it an excellent choice for chemoprophylaxis; its main disadvantage is the dosing regimen, which requires daily oral ingestion. It is not generally used for treatment of malaria because it is a slow-acting drug. (<http://www.malariasite.com/malaria/tetracyclines.htm>)

Mefloquine, available under the trade name Lariam®, was approved by the FDA for use in the prevention and treatment of malaria in 1989. It is one of the most commonly used antimalarial chemoprophylactics and treatments in regions where chloroquine-resistant Plasmodia are demonstrated. No information on mefloquine is available from IPCS. According to product information from Roche Pharmaceuticals, “Mefloquine hydrochloride is a 4-

quinolinemethanol derivative...which acts as a blood schizonticide. Its exact mechanism of action is not known.” Roche also explicitly states that “Patients with acute *P. vivax* malaria, treated with Lariam, are at high risk of relapse because Lariam does not eliminate exoerythrocytic (hepatic phase) parasites. To avoid relapse...patients should be subsequently treated with an 8-aminoquinoline (eg, primaquine).” (<http://www.roche.usa.com/products/lariam/pi.pdf>) Unfortunately, Lariam is widely known to have severe side effects when taken for extended periods of time (cf. Holder 1997) and is very expensive, making it an unappealing chemoprophylaxis for extended use.

Another alternative drug treatment for chloroquine-resistant *P. falciparum* malaria is Malarone. According to the CDC, “Malarone™ (a fixed combination of atovaquone and proguanil hydrochloride) is an antimalarial drug approved in the United States in July 2000 for both treatment and prophylaxis of malaria.” Malarone attacks the blood phase of the disease and inhibits the reproduction of the Plasmodium parasite. (cf. www.malarone.com) Thus far, it is considered to have few side effects, but like Lariam® it is very expensive.

Contemporary research on antimalarial drugs focuses on artemisinins. Artemisinins are derived from the Chinese herb qinghao, or sweet wormwood, which has been used in China to treat malarial fevers for more than 2,000 years. The World Health Organization is enthusiastic about the prospects of treating chloroquine-resistant strains of malaria with Artemisinin-Based Combination Therapies (ACTs) because these combination drugs “kill the malaria parasite very fast, allowing the patient to recover rapidly, and with very few side effect. Because ACTs combine two medicines which work in different ways, it is unlikely that the malaria parasite...would evolve to resist these medicine combinations.” (<http://www.who.int/inf/en/pr-2002-31.html>)

Dr. Kakkilaya stresses that “Primaquine is the essential co-drug with chloroquine in treating all cases of malaria. It is highly effective against the gametocytes of all plasmodia and thereby prevents the spread of the disease to the mosquito from the patient. It is also effective against the dormant tissue forms of *P. vivax* and *ovale* malaria, and thereby offers radical cure and prevents relapses.” He goes on to state, “At present, Primaquine is the only drug available for tissue schizonticidal activity in *P. vivax* malaria and gametocytocidal activity in *P. falciparum* infection. Therefore, it must be used in both these infections. Therefore, at present there are no alternatives to primaquine. Newer antimalarials like mefloquine or

artemisinin derivatives are NOT substitutes for primaquine!" (<http://www.malariasite.com/malaria/primaquine.htm>)

Note that antimalarial chemoprophylaxis is not a viable option for residents of malarial regions for a number of reasons including cost, long-term side effects, and the rapid emergence of resistant parasites. The efficacy of antimalarials as radical cure treatments, however, is highly useful information for residents of malarial regions. The primary benefit of the present research on antimalarial drugs for residents of San Antonio will be my own ability to provide information to community members and healthcare workers on treatment options when the sharing of such information is appropriate. This research will also be extremely useful in educating and preparing my own research team for our stays in San Antonio, thereby minimizing the possibility that we become implicated in the transmission of malaria in the community.

7. *Antimalarial drug treatment in Peru*

In areas of Peru where malaria is not chloroquine resistant, the most common and least expensive treatment used for either *P. vivax* or *P. falciparum* is chloroquine to kill the blood phase of the Plasmodium parasite. Often – but not always – chloroquine is combined with primaquine to kill the encysted phase of *P. vivax* and the Plasmodium gametocyte. In San Antonio, all strains of *P. falciparum* are presumed to be chloroquine resistant and are presently treated with a combination of artesunate (an artemisinin) and mefloquine.

Reading through the official Peruvian Ministry of Health documents available online, (<http://www.minsa.gob.pe/infovigia/normas.htm>) I learned that essentially all treatment regimens of malaria are mandated to include primaquine. I must deduce that primaquine is not presently used to treat *P. falciparum* in San Antonio either because it is not available or because the healthpost staff chooses not to prescribe it. My personal experiences over the years in Peru suggest to me that it is not common knowledge among Peruvians – either laypersons or healthcare workers – that primaquine should be taken in all cases. In general, if people know about primaquine, they seem to consider it a non-essential part of the chloroquine regimen.

In 1994 the Peruvian Ministry of Health launched a new national antimalarial program, intended to provide free malaria treatment throughout Peru. This program combines educational activities with mosquito control efforts, free MP smear testing, and free drug treatment.

A somewhat bizarre side effect of the MINSA program is the present relative scarcity of chloroquine and primaquine for purchase anywhere in the nation, which in turn means that people sick with malaria cannot purchase these medicines. Because many people who suffer from malaria live at prohibitive distances from MINSA healthposts and because MINSA healthposts are chronically undersupplied with medicines, the MINSA program in some instances actually prevents the treatment of malaria!

On November 21, 2002 I received an email message from a friend who is a medical doctor in Peru and who is presently working at a PlusPetrol fieldsite in the Urubamba River valley in southern Peru. In his brief update on the local situation, Dr. Pacheco commented,

“it is now the rainy season here... there was an outbreak of malaria at the Techint fieldsite, more than 50 cases... it would be recommendable that you coordinate the donation of antimalarials to the Camisea communities... the government, through the ministry of health, promises much, but in the end provides nothing; can you believe that in all of Perú there is no primaquine? It is the extreme of careless, but you know that...” (personal communication; my translation from Spanish).

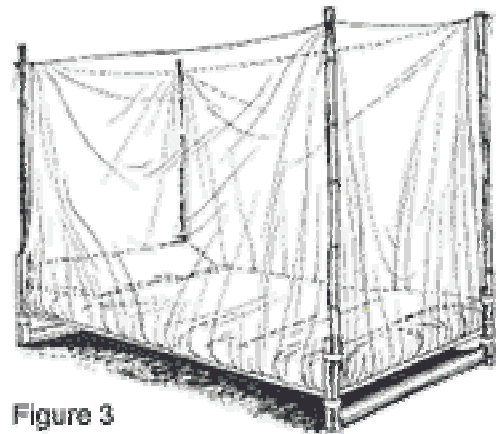


Figure 3

Image source: <http://mosquito.who.int/docs/healthworkers3.htm>

8. *Individual prevention strategies: bed nets*

A major component of the campaign to combat malaria in Africa promotes the use of insecticide-treated bed nets. According to IDRC, “Studies have shown a 20 to 63% reduction in malaria following the introduction of insecticide-treated nets (ITNs).”⁴ ITNs are considered

⁴ “Preventing Malaria: Mixing Public Health Interest with Private Profit.” http://www.idrc.ca/reports/prn_report.cfm?article_num=362

especially important in reducing the incidence of malaria in children, since they are both more vulnerable to malaria and are often sleeping during the hours of greatest mosquito feeding activity. ITNs are treated with any of several biodegradable pyrethroid insecticides that are believed to be harmless to human beings and safe for the environment when used appropriately. ITNs must be retreated with insecticide every six months in order to retain their effectiveness.

Dr. Catherine Reed of PATH Canada, an institution that does research on ITN use in Africa, points out that “the popularity of nets seems to have less to do with a country’s wealth than with local traditions” and as such could successfully be linked to Africans’ purchasing decisions. In addressing the linked problems of the distribution and the retreatment of ITNs, Reed advocates introducing private sector interests into antimalarial strategies, essentially creating and then meeting a consumer demand for nets and insecticides through targeted marketing and advertising efforts.

Fortunately, the use of bed nets is already ‘traditional’ in San Antonio de Pintuyacu. To the best of my knowledge, the only reason that a community member would not use a bed net is not having one. Treating bed nets with insecticide, however, is not presently done in San Antonio; I would guess that community members simply do not know about this strategy and do not have the resources to initiate such a practice without outside assistance.

African women treating bednets with non-toxic antimalarial insecticides



Image Source: Bourgoing 1996

8. Infrastructural resources available in San Antonio

The village of San Antonio has a Peruvian Ministry of Health healthpost which serves the entire population of the greater San Antonio area. Prior to August 2002, San Antonio’s healthpost had a single paid employee who was trained as a *tecnico medico* – roughly the equivalent of a physician’s assistant here in the US.

In July of 2002, a new healthpost was inaugurated in San Antonio. The construction of this building was begun in 1999, funded by a special project of the Presidencia de la Republica, but residents of San Antonio explain that the director of the project extorted and disappeared with several thousand dollars in funds necessary to complete construction; so the building sat nearly completed but unused for more than a year until the community secured additional funds to complete building and equipping the healthpost. Conversations I had with residents of San Antonio led me to believe that the malaria crisis in the community in the mid 1990s was the main reason the government approved their request for the new and greatly improved healthcare facility. The community’s single *tecnico medico* relocated his practice to the new building in July of 2002, but as of August 2002, the healthpost still lacked medical equipment, including the microscopes necessary to read *gota gruesa* slides for malaria, and the trained personnel necessary to operate this equipment.

As with all community healthposts in Peru, San Antonio’s healthpost includes a small pharmacy. However, because Ministry of Health policy dictates that the healthpost staff must purchase medicines for the pharmacy with the money they collect selling medicines, community pharmacies including this one frequently do not have many necessary medicines. The lack of funds and local transportation cause chronic problems in stocking community pharmacies, which in turn severely impacts a healthpost’s success in treating the illnesses – including malaria – that are diagnosed there (see section 7 above).

9. Case study: Lev Michael

During our first visit to the community in 2001, Lev Michael and I learned that malaria was endemic in and around San Antonio. As part of our preparations to return to San Antonio for eight weeks in 2002, we determined that both *P. falciparum* and *P. vivax* are demonstrated in San Antonio and that both species are considered chloroquine resistant. Further research – which included consultations with doctors in Peru and with the travel health clinic at UT-Austin – led us to conclude that, given the length of our intended stay in

San Antonio, the most appropriate antimalarial chemoprophylaxis for us to use was doxycycline.

As an antimalarial, 100 mg. of doxycycline is taken orally once daily beginning one week prior to entering the malarial zone and for four weeks after leaving. In June 2002, all four members of our research team began the regimen as directed and adhered to it during our stay in San Antonio.

Nonetheless, about a month after arriving in San Antonio, Lev began to experience symptoms of malaria. Having had the disease twice prior, he said he “knew right away” that he had malaria, describing the symptoms as “unmistakable”. After his first high fever, I immediately took him to the healthpost. After we described Lev’s symptoms, the *tecnico* took an MP smear from both of us and immediately prescribed Lev treatment for *P. falciparum*. At that point, all MP smears were read in the city of Iquitos, so all suspected cases of *P. falciparum* were treated immediately. We had to wait two weeks until the test results returned; when our MP smears came back from Iquitos, Lev’s slide read positive for *P. falciparum*.

As the *tecnico* explained, the treatment Lev took is “experimental” – that is, not yet officially sanctioned and authorized by MINSA. Nonetheless, the experimental drugs are provided to San Antonio by MINSA; no doubt this reflects the fact that governmental bureaucracy moves more slowly than infectious disease. Lev’s treatment involved 5 artesunate tablets for each of three days combined with 3 tablets of mefloquine on the last two days. No primaquine was prescribed. Many malaria patients in San Antonio describe this treatment as “worse than the disease,” complaining that it aggravates the already intensely painful headaches that are associated with malaria and causes extreme dizziness and light-headedness. Overall, Lev found the treatment no worse than the illness, although on the first night of his treatment his head ached so badly as to bring him to tears and his body temperature reached 40.3° C, or almost 105° F. But after that first night, Lev felt much better and he was able to return to normal activity on the second day of his treatment. Despite our anxieties, none of the rest of the team fell ill, and a week later we all considered the malaria case “closed.”

Our whole research team returned to Austin in good health in mid-August. On Thursday, September 19th, Lev had a low (38°C) fever. On the 20th, however, he felt fine. Then on September 21st he again ran a fever, but on the 22nd felt fine. Because he had no other symptoms, he assumed he was fighting a local virus or allergies. Only on Monday the 23rd, when his body

temperature reached 40° C (104°F) and he experienced a headache was he convinced that he was really sick and should visit the health clinic. To make a long story short, finally on Friday, September 27th, he had an MP smear blood test and was diagnosed with malaria. Suffice it to say that it is quite difficult persuade Austin healthcare workers that you have malaria – until your temperature reaches 105° while you are waiting in the examination room. Fortunately the doctor who attended Lev on Friday was exceptionally sensitive and receptive to Lev’s experiential knowledge. Dr. M. suggested that he simply repeat the treatment Lev had received in San Antonio, but Dr. M quickly learned that artesunate is unavailable in the United States. Therefore, presuming Lev to be suffering *P. falciparum*, he treated Lev immediately with mefloquine and doxycycline. A few days later, Lev received the results of the MP smear, which indicated that in fact he had *P. vivax*. At that point, Dr. R treated Lev with chloroquine and primaquine. This second bout of malaria left Lev quite anemic and weak, but he has felt well since early October.

I would like to focus in on the most important and surprising fact in this case history. Lev’s MP smear taken in Austin demonstrated that he had *P. vivax*, not *P. falciparum*. We deduce that the artesunate and mefloquine treatment administered in San Antonio had eliminated the blood phase of the disease, but had not eliminated the liver phase of the *P. vivax* parasite – for which primaquine is necessary. We also deduce that Lev had been infected with both *P. falciparum* and *P. vivax* while in San Antonio and after about six weeks, the liver phase of *P. vivax*, which had escaped untreated, ruptured and infected him again.

This case study has led me to the following crucial realization: a person suffering malaria symptoms in San Antonio de Pintuyacu may be infected with more than one species of Plasmodium parasite and therefore the treatment prescribed should attack both *P. falciparum* and *P. vivax*, not just one or the other.

10. Strategies for reducing the incidence of malaria in San Antonio in 2003 and 2004

Prior to this point, I have discussed various facts and factors that are implicated in the present incidence of malaria in San Antonio de Pintuyacu. In this section I will outline three key strategies that have emerged from my research that are likely to be both *attainable* during my stays there between June and August of 2003 and 2004 and *effective* in reducing the incidence of malaria in San Antonio.

10.1 Key strategy #1

First, my research (see sections 7 and 9) has convinced me that treating all cases of malaria with primaquine is an absolute necessity. Therefore, I will enlist the professional assistance of doctors and MINSA representative that I know in Peru, and also present key official MINSA documents regarding malaria, to convince the healthcare staff in San Antonio to begin this practice. I will also acquire an ample supply of primaquine and donate it to the community so that the chronic scarcity of primaquine in Peru is not an obstacle in San Antonio. My experience providing medical supplies to isolated indigenous communities in southern Peru will facilitate this step. I will also provide community members with basic information concerning the necessity of treating all cases of malaria with primaquine in order to promote a common and widespread understanding of *why* primaquine is a necessary facet of all malaria treatment.

10.2 Key strategy #2

Second, the use of ITNs, particularly for children and elderly persons, could greatly reduce the transmission of malaria in San Antonio. (see section 8) The use of bed nets is already common in San Antonio; therefore, introducing the use of insecticide-treated nets by community members would build on an existing preventative measure.

I will bring with me to San Antonio the information necessary for the safe use of pyrethroid insecticides to treat bednets. (see Appendix A) that I discovered in researching this paper. I will find a reliable source for an appropriate pyrethroid insecticide in Iquitos and donate a one year's supply of insecticide to the community. Then I will hold a 'workshop' in San Antonio for interested community members in which, as a group, we will treat a supply of bednets. In addition, if possible I will bring a supply of new nets that can be treated in the community workshop and then distributed to households that lack them.⁵

10.3 Key strategy #3

Third, when residents of San Antonio are in fever, they typically do not confine themselves to bed nets in order to prevent the transmission of Plasmodium parasites to others. I intend to encourage people to quarantine themselves or family members while in fever inside of insecticide-treated nets. People sick with malaria already

are overwhelmingly likely to be lying down while in fever; they simply need to add the habit of confining themselves to the bed net.

10.4 Key strategy #4

This research paper has revealed to me the most important details of human behavior in San Antonio that affect the incidence of malaria there. Therefore, I will prepare a small set of focused materials in Spanish to offer to community members in order to facilitate the implementation of the strategies mentioned in sections 10.1, 10.2, and 10.3. I know that people in San Antonio do not want to suffer malaria; my goal is to make it easier for them to avoid suffering malaria by making a few simple and sensible behavioral changes.

Chris Beier is a graduate student in linguistic anthropology at UT Austin and is also co-founder of Cabeceras Aid Project, a non-profit organization that carries out linguistic and anthropological research within the context of humanitarian projects in indigenous communities in Peruvian Amazonia. For more information, visit www.cabeceras.org or www.iquito.org, or contact chris@cabeceras.org

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⁵ The limiting factor on strategy #2 will be the amount of money available for purchasing supplies.

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Appendix A.

WHO recommended insecticides for treatment of mosquito nets for malaria vector control.
 Source: www.who.int/ctd/whopes/sptmn_eng.pdf

INSECTICIDE	FORMULATION ¹	DOSAGE ²
Alpha-cypermethrin	SC 10%	20-40
Cyfluthrin	EW 5%	50
Deltamethrin	SC 1% & WT 25% ³	15-25
Etofenprox	EW 10% ³	200
Permethrin	EC 10%	200-500

Notes:
 1 SC= suspension concentrate; EW=emulsion, oil in water; WT=water dispersible tablet; EC: emulsifiable concentrate.
 2 Milligrams of active ingredient per square metre of netting.
 3 WHO specifications under development