

CHAPTER 24

Vector Control by Surveillance Networks: The ECLAT Program and Chagas

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24.1 INTRODUCTION

Chagas disease is a serious problem for public health in Latin America. In 1984, it was estimated that over 24 million people were infected [58], with a further 100 million people at risk. With extensive vector control programs during the last decade, these figures have been revised downward to around 12 million people infected [48], although large-scale control and surveillance programs are still lacking in several countries.

Chagas disease takes its name from Brazilian clinician Carlos Chagas, who first described it in 1909. It is a parasitic disease caused by *Trypanosoma cruzi* (Kinetoplastidae), producing a wide range of pathology from clinically undetectable lesions (60% of cases) to serious chronic problems of heart and digestive tract that can be fatal. Humans mainly become infected through contact with blood-sucking insects of the subfamily Triatominae (Hemiptera, Reduviidae) (see Fig. 24.1) those often colonize houses in rural areas [25]. Other mechanisms of infection include blood transfusion from infected donors, and occasional congenital transmission, oral contamination, or laboratory accident. Treatment (using benznidazole or nifurtimox) is effective only in the early acute stage of infection, where unfortunately the diagnostic is frequently overlooked, and frequently leads to undesirable side effects—especially in adults. The drugs are better tolerated in children, and treatment may be given to under-14s even in the chronic stage of infection, with the idea that this may impede the development of chronic lesions later in life [56]. Considered at the continental level,

the debilitation caused by chronic infections makes Chagas disease one of the most expensive public health problems in the world in term of loss of productivity (expressed as disability-adjusted life-years) [60] although it is relatively simple to halt transmission by eliminating the domestic insect vectors.

In the absence of vaccine [9], and because of difficulties in curative treatment, control of Chagas disease relies primarily on measures directed against the insect vectors. These are large blood-sucking bugs (Triatominae) that are generally associated with small mammals, birds, or reptiles. But some species of Triatominae have become adapted to colonize human dwellings, where they feed predominantly on the people and their domestic animals. In houses, these bugs can develop abundant populations, often numbering several thousand individuals, causing significant nuisance to the householders and their animals. And because these bugs can take substantial quantities of blood during each meal (up to 0.5 mL per meal for many adult Triatominae), they are also believed to contribute to chronic iron-deficiency anemia, as well as transmitting *T. cruzi* [41,49].

Elimination of domestic populations of Triatominae can generally be achieved by a thorough application of a modern pyrethroid insecticide (Table 24.1). If done on a small scale, however, the treated premises may be quickly reinfested by bugs accidentally carried in from untreated foci. For this reason, current control initiatives emphasize very wide area coverage—preferably over the entire geographical distribution of the target species, and generally involving several countries in



Fig. 24.1. Faeces of *Rhodnius prolixus* (here a fifth stage nymph of *R. prolixus*) left on the skin after feeding; if the bug is infected by *Trypanosoma cruzi*, this is the way the vector may infect its host: by depositing an infected drop of faeces. The parasite then enters actively through the mucosa or through the abraded skin.

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a simultaneous campaign. For example, the Southern Cone Initiative launched in 1991 now covers the entire distribution of *Triatoma infestans* in Argentina, Bolivia, Brazil, Chile, Paraguay, southern Peru, and Uruguay. Similarly, the Central American Initiative launched in 1997 is primarily directed against *Rhodnius prolixus* in Guatemala, Honduras, El Salvador, and Nicaragua. In both these initiatives, the vector control measures—coupled with measures to improve blood transfusion control—have been highly successful, with Chagas disease transmission already interrupted in Uruguay, Chile, and large parts of Brazil, Argentina, Paraguay, Bolivia, and the Central American Countries [35,52]. Further regional initiatives are being developed for Mexico, Andean Pact countries, and the Amazon region. In all cases, however, it is recognized that extensive long-term vigilance will be required to avoid recolonization of dwellings in treated areas, either by the original species or by another species adapting from a silvatic habitat to colonize the houses. Here, we argue

TABLE 24.1. Pyrethroid Insecticides Used Against Domestic Triatominae

Insecticide	F	R (rate, mg.a.i./sq.m)	MS
Deltamethrin	SC	25	Aventis/Bayer
Lambda-cyhalothrin	WP	30	Zeneca/Syngenta
Cyfluthrin	WP	50	Bayer
Beta-cyfluthrin	SC	25	Bayer
Cypermethrin	WP	125	Various

F, formulation; R, recommended doses; MS, main supplier.

that epidemiological vigilance, when connected with international research, can be a fruitful activity on scientific ground with very positive public health output. The ECLAT network, by creating an international link between scientific research and operational activities, could maintain an efficient and continuous surveillance network during years. This partnership gave scientific research the opportunity to focus on relevant topics of immediate interest for public health, and it allowed health authorities to be aware of new techniques or new discoveries otherwise restricted to highly specialized literature. Its international nature also increased motivation and helped maintain continuity both of the research and surveillance activities.

24.2 ORIGIN AND SPREAD OF HUMAN CHAGAS DISEASE

T. cruzi is a widespread parasite of small mammals in the Americas, with a distribution roughly from the great lakes of North America to southern Patagonia. Although it may originally have been transmitted directly between marsupial hosts (especially didelphid opossums) [51], it is now almost entirely transmitted by various species of Triatominae—especially those that colonize small mammals nests and marsupial lodges. Well over 130 species of Triatominae are now recognized. Most are of silvatic habit, but may occasionally fly into houses. Some also colonize peridomestic habitats such as chicken coops and goat corrals, and a few species colonize human dwellings—especially in rural areas. It is these “domestic” species—especially *T. infestans* in the Southern Cone Region, and *R. prolixus* in Colombia, Venezuela, and parts of Central America—that are of greatest public health importance and epidemiological significance as vectors of *T. cruzi* to humans.

Archaeological studies indicate the presence of *T. cruzi* in pre-Colombian mummies in the Andean region of South America (Chinchorro culture of northern Chile and southern Peru) dated up to 9000 years BP [5].

However, historical reconstruction suggests that the main expansion of human Chagas disease was in post-Colombian times, particularly during the last 150 years, associated with human migrations and accidental transport of “domesticated”¹ Triatominae [50].

Genetic studies on different geographical populations of the target vectors have confirmed the historical records suggesting their progressive but fast, recent spread largely due to human activities. These studies led to a predictive model explaining the spread of the human disease, that is, the birth of the disease itself [22]. Steps of this model are (i) adaptation of the insect to domestic conditions, (ii) passive dispersion of

¹ Synanthropic Triatominae are usually called “domestic” and/or “peridomestic” Triatominae by field entomologists; this expression will be used in this text

these domestic forms with humans, (iii) isolation from their original, wild foci, and (iv) lack of entomological surveillance or preemptive control response. In a few decades, this mechanism may contribute to the installation of a widely extended domestic vector, leading to widespread transmission of the disease [22]. Similar problems could arise with the same or with other vectors, to which the appropriate public health response is through an active surveillance network. To some extent, this surveillance requirement has been addressed by a combination of active surveillance by the public health authorities, coupled with community-based surveillance by the householders in endemic regions.

24.3 THE DISPERSAL OF THE MAIN VECTORS

Prior to the Southern Cone Initiative launched in 1991, primarily against *T. infestans*, this species was considered responsible for more than half of all transmission of Chagas disease to humans—responsible for infecting some 12 million people in the seven southernmost countries of Latin America: Argentina, Bolivia, Brazil, Chile, Paraguay, Peru (Arequipa Province), and Uruguay [25]. *T. infestans* is an efficient and widespread vector, extended over large geographic areas, highly dependent of domestic structures, but also found in silvatic habitats under rockpiles and parts of Central Bolivia [29]. Historical reconstruction suggests these silvatic populations first entered domestic habitats in pre-Colombian times—possibly associated with the hunting and domestication of its wild guinea pig hosts [50]. These domestic populations were then spread—probably by accidental carriage with migrating humans—mainly during the last century [13,31,34,38], reaching their maximum extension in northeastern Brazil just prior to 1981 [4,6,47]. In Central Brazil (states of Goiás, Minas Gerais, and Bahia), *T. infestans* was unknown until the 1930s, but progressively replaced the local domestic vector—*Panstrongylus megistus*—that had originally been incriminated by Carlos Chagas two decades earlier. Genetic studies confirmed the scenario of a recent and rapid geographical spread of *T. infestans*, by showing low genetic heterogeneity of its populations [27,28,32,36], and suggesting a cline of decreasing variability from Bolivia to other countries [26]. Together with these genetic studies, the presence of silvatic foci only in Bolivia strongly suggested a Bolivian origin [29]. Further cytogenetic studies now suggest this expansion was completed in a two steps process, a first Andean expansion, followed later by a lowland expansion probably contemporary to Spanish conquest [39].

In Venezuela, Colombia, and parts of Central America, the most important vector of Chagas disease is *R. prolixus* [25]. Again, historical reconstruction and genetic comparisons of different populations suggest a relatively recent spread from an original domestic focus probably in Venezuela [17,55]. The spread of *R. prolixus* into Central Colombia may have been in association with Spanish expeditions from Venezuela in the sixteenth century. However, its spread into Central America



Fig. 24.2. Adult specimen of the main vector in the seven southernmost countries of Latin America: *Triatoma infestans*. Photo by Marcia Gumiel, Bolivia.

seems to have been much more recent and possibly due to an escape from a laboratory colony in San Salvador in 1913 [23]. By 1915, *R. prolixus* had spread into rural houses in El Salvador [37], and then into neighboring countries—reaching its maximum Central American distribution during the 1950s. This included parts of Southern Mexico, Guatemala, Honduras, El Salvador, and Nicaragua [19]. These Central American populations of *R. prolixus* reached northern Costa Rica in 1953 (probably in association with migrant workers from Nicaragua) but were quickly eliminated by insecticide spraying [45]. Since then, its distribution has been progressively reduced, particularly as a result of concerted action through the Central American Initiative launched in 1997, and now includes just a few remaining foci in eastern Guatemala, Honduras, and western Nicaragua. Venezuelan populations of *R. prolixus* were also reduced through the national campaign (1966–1972), and some progress has been made in eliminating this species from houses in parts of central Colombia.

The distribution of other domestic Triatominae also seems to have been influenced by accidental carriage in association with human migrations. *T. dimidiata*, for example, is the second most important domestic vector of Chagas disease in southern Mexico and Central America, where it also maintains widespread silvatic populations. But in Ecuador and parts of northern Peru (Tumbes), *T. dimidiata* seems exclusively domestic and is the main vector in these regions. Again, historical reconstruction combined with genetic comparisons of different populations indicates that *T. dimidiata* was first domesticated in the Tehuantepec region of Central America, and that a subset of these domestic populations was then accidentally carried to the port of Guayaquil in Ecuador following well-established pre-Colombian maritime trade routes [1]. A more recent example concerns *Rhodnius ecuadoriensis*—of little epidemiological significance in its native Ecuador where it primarily inhabits palm tree crowns, but a major domestic vector in parts of northern Peru (La Libertad) where it is exclusively domestic. In this case—although the evidence

requires further confirmation—it is suspected that *R. ecuadoriensis* has been accidentally brought from Ecuador to Peru in lorries returning after delivering the Peruvian grape harvest [55].

Parallels in the adaptive history of these important vector species support the idea of a common strategy for their control. Silvatic populations of these species can be considered to have a “natural range”—albeit probably mediated by passive carriage with their silvatic vertebrate hosts. But an original domestication event, followed by dispersal of the domestic populations in association with human movements, has led to domestic populations of these species occurring well outside their original range—and it is these domestic populations that are of greatest epidemiological significance as vectors of Chagas disease to humans. Moreover, this process seems to involve genetic bottlenecks, founder effects and genetic drift [22], together with selection for the optimum genotypes for domestic habitats [54], so that the domestic populations tend to be of reduced genetic variability and consequently more vulnerable to available control methods. The combination of feasibility to eliminate domestic populations with the idea of “rectifying” the accidental transport of these populations outside their original range forms a compelling argument in favor of large-scale elimination of these domestic Triatominae. However, the capacity for domestication and passive dispersal may be similarly shared by many other species of Triatominae. The resulting working hypothesis is that passive and important migrations outside the current range of its original, wild foci, would be a trait specific to any other species becoming highly dependent to human environment [25].

24.4 FROM DISEASE TO PUBLIC HEALTH PROBLEM

24.4.1 The Nature of the Disease

As a human suffering, Chagas disease is not well known, and is presently classified as “neglected disease” by the European Commission. The acute phase of the disease lasts a few weeks, corresponding to the diffusion of the parasite into blood circulation and its entry to muscular cells, frequently cardiac cells, and hearth RX may show enlargement of the organ. Although this acute phase may be lethal, it generally happens without serious consequences, often being comparable to a bad cold. During the chronic phase, however, and after a few decades, up to 40% of patients may develop a severe disease. A proportion of them suffers from cardiopathy ranging from arrhythmias to complete bundle-branch blocks requiring pacemakers implant. Cardiac aneurysm can also occur, leading to cardiac rupture (sudden dead syndrome) on exercise. Another proportion of infected people may develop megaorgans of the digestive tract, with intestinal peristalsis interrupted in severe cases, leading to difficulties in swallowing in the case of megaesophagus, and inability of stool transit in the case of megacolon.

The clinical outcome of Chagas disease, asymptomatic or lethal, heart or digestive disease, remains unpredictable. Geographic variation is obvious [43]. It has been attributed to differences in human genotypes and to differences in parasite genotype [3,57], virulence, growth rate, and tissue tropisms. In genetic terms, there appears to be two main lineages of *T. cruzi*—now denoted *cruzi 1* and *cruzi 2*—that can be characterized by isoenzymes and DNA sequence differences. Of these, *cruzi 1* seems more homogeneous and has been regarded as the more primitive form [51]; in humans, *cruzi 1* tends to be associated with cardiac lesions, but rarely—if ever—with the lesions and dilations of the digestive tract known as “megs.” By contrast, *cruzi 2* is more heterogeneous, and although infections with forms of *cruzi 2* are also typically associated with cardiac lesions, they are also frequently associated with megas (particularly megaesophagus and megacolon) especially in the Southern Cone countries. In neither case, however, is the pathogenesis clearly understood [10,42,44], although the cardiac lesions are often attributed to parasite stimulation of a host autoimmune response, and the digestive tract lesions are often attributed to progressive neuronal destruction of the peripheral sympathetic nervous system.

As a research topic, the pathogenesis of Chagas disease remains a major challenge [40],—especially in understanding prognosis and determining which factors will contribute to the development of chronic lesions. One feature that has recently come to light is that the vector control programs themselves may have an impact in progression of the disease—even amongst those people already infected [21]. Since the launch of the Southern Cone Initiative against Chagas disease in 1991, clinicians report an apparent decline in the average severity of chronic lesions amongst those infected prior to the vector control campaign. This could suggest that a reduction in the rate of reinfection of chronically infected people (due to elimination of the insect vector) has contributed to a reduction in the severity of their disease—an idea subsequently supported by experimental studies of reinfections in mice, and already suspected by clinicians [18].

24.4.2 The Disease of Poverty

As for many parasitic diseases, Chagas disease mainly affects people of low economic status, and there is a broad correlation between the occurrence of the disease and poor quality housing [12]. Traditional rural dwellings of earth, sticks and palm thatch, generally provide suitable shelter for domestic Triatominae, and rural houses are often close to silvatic foci of these bugs, so that they tend to be the first to be colonized. But even for human dwellings with higher standards of construction, the vectors easily colonize chicken houses and other domestic animals enclosures. With the development of large cities with surrounding precarious constructions, Chagas disease also became periurban in many Latin American countries [14].

Actually, even cities are not protected against some vectors [8,30,46]. Not only the habitat provides shelter to the insect

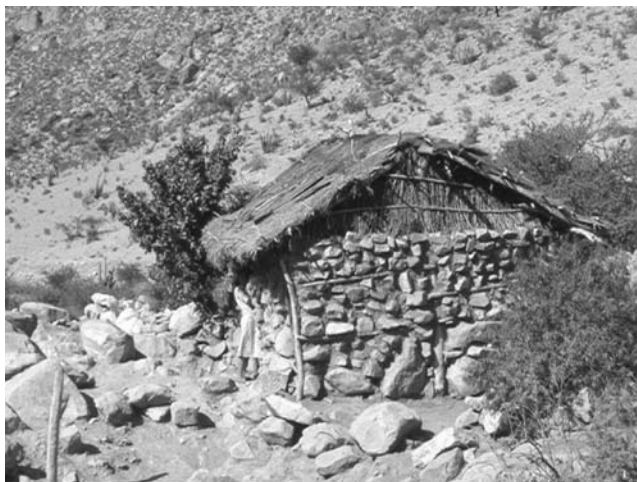


Fig. 24.3. Rural house in the Chilean Andes, infested with *Triatoma infestans* (Fig. 24.24.2).

but also this latter has more or less preference for it, according to the species. Some authors have suggested classifying vector species of Triatominae according to their ability to colonize houses, going from completely silvatic to highly domestic species [50].

The importance of the domestic habitat in terms of vector colonization and transmission of Chagas disease has been well recognized—even since the earliest studies of Carlos Chagas himself [15,16]. And this has led to a number of calls—and many projects—to combat the disease through programs of improved rural housing. Such programs have met with various levels of success [11,53] but all have been limited in scale both by availability of funds and by different levels of community acceptance. House improvement programs tend to be considerably more expensive than vector

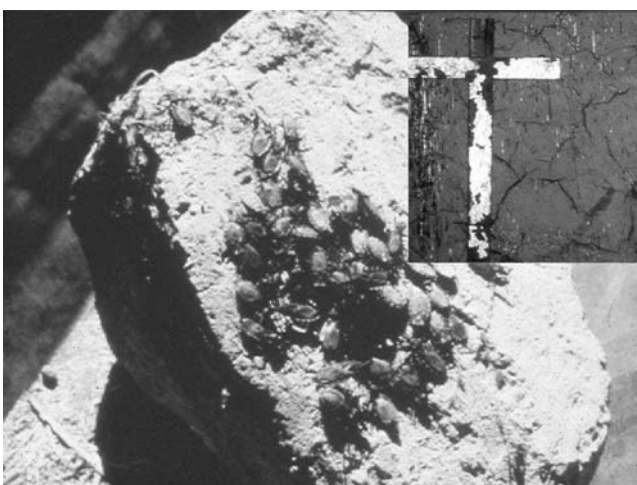


Fig. 24.4. One stone from the house wall is turned over to reveal many *Triatoma infestans* adults and nymphs; top right: the faeces of *T. infestans* (Fig. 24.24.2) streak down the house wall.



Fig. 24.5. Typical house in Venezuela, infested with *Rhodnius prolixus*.

elimination by insecticide spraying, and the proposed improvements do not always meet with the approval of local communities. They are also relatively slow to implement, and often require a higher level of subsequent maintenance than the local communities are able to carry out. There are also practical difficulties in the sense that house improvements alone are rarely sufficient to extinguish an existing infestation with Triatominae, and ethical considerations in the sense that presence of domestic Triatominae should not be the primary criterion for house improvement (because this may disfavor poor families that do not have domestic Triatominae). It is generally recommended, therefore, that rural house improvement should be considered an independent developmental goal, irrespective of the presence or absence of domestic Triatominae, whereas programs to eliminate these insects should proceed independently.

24.4.3 Socioeconomic Impact

At a societal level, the social and economic importance of Chagas disease derives both from its high prevalence and the severity of its symptoms—especially amongst the most productive age-classes between 20 and 40 years. Overall prevalence rates of around 5–6% were typical of most endemic countries prior to the current large-scale control initiatives, although local prevalence rates often exceeded 50% in some areas. In the initial acute phase of infection, which can be fatal without treatment, the patient may be incapacitated for 2–3 weeks due to fever, diffuse chest pain, insomnia, and general discomfort. During the chronic phase, however, up to 40% of surviving patients become incapacitated because of either severe cardiopathy or digestive tract megasyndroms. Costs of diagnosis, clinical follow-up, and supportive treatment are high—over US\$ 120 per year even in the case of clinically asymptomatic patients [7], but rising to several thousand dollars where cardiac pacemaker or corrective surgery are required. In Bolivia, even considering that only 10% of

infected people had access to medical care, USAID estimated the annual loss due to Chagas disease at 100 millions of US dollars—then over twice the national health budget for the country. Estimates by the World Bank (1993) prior to the current large-scale control initiatives, ranked Chagas disease as by far the most serious parasitic disease of the Americas—far outranking even the combined socioeconomic impact of other parasitic diseases such as malaria, schistosomiasis, and leishmaniasis [52]. At the level of the individual, however, the impact of the infection may be equally as severe as that of the disease. Diagnosis of a chronic infection—even if asymptomatic—can lead to severe stress and confusion. Some countries continue (by law) to refuse employment to those diagnosed as infected, even when no disease is apparent. Suicides have been reported, following such diagnosis, assumed due to the difficulties of reconciling the idea of living with an untreatable infection that may (or may not) develop into a life-threatening disease. In poorer rural communities, infection and incapacity of the head of the household can lead to hardship for the rest of the family. In parts of Central America prior to the current control initiatives, we have seen testaments from chronic Chagas disease cases bequeathing their pacemakers to the eldest son—presumably in the sad expectation that he too will develop a similar need in later life.

24.5 CONTROL AND SURVEILLANCE

24.5.1 Control Strategies

The essential rationale of Chagas disease control is to halt transmission, and to provide treatment and support for those already infected. Interruption of transmission includes serological screening of blood donors to reduce the risk of transfusional transmission, but relies most heavily on elimination of domestic populations of the Triatominae vectors. As with other vector disease vectors, control of Triatominae on a small scale is difficult to sustain due to reinvasion of vectors from untreated foci. This sets the increasingly perceived need for large-scale initiatives and, because it is difficult to maintain control interventions indefinitely, there is also a need to design strategies that can reach a sustainable end point [33]. In the case of Chagas disease, the control strategy is, therefore, based on elimination of existing domestic vector populations—generally achieved through a single thorough insecticide application in each infested house (Table 24.1)—followed by continuous vigilance both by the local communities and by the public health services, with selective interventions wherever new domestic infestations are suspected. This basic control strategy is now being progressively implemented through large-scale initiatives involving most countries of Latin America.

The essential control strategy is focused on the elimination of all domestic populations of the insect vectors, and prevention of recolonization by (a) eliminating neighboring domestic and peridomestic colonies that could serve as foci for reinfestation, and (b) sustained entomological vigilance coupled with selective retreatment of houses if recolonization

is detected. For operational purposes, the control programs work within the context of existing administrative units (localities or villages, within municipalities/departments/veredas—according to local administrative systems). Each locality is first mapped to establish the number and distribution of houses, with a search of each house to establish whether or not the target vectors are present. Where the target vector species is considered a feasible target for local elimination—as in the case of *T. infestans* in the Southern Cone countries, or *R. prolixus* in Central America—then all houses of each locality where one or more of these insects is detected will be sprayed, regardless of whether an individual house is infested or not. The reasoning is that because available sampling methods are imprecise, it is considered more effective to spray houses even if they are not actually infested, rather than risk not spraying a house when it is in fact infested. For other vector species of Triatominae, the decision to spray all houses, or only those that are apparently infested, is taken on the basis of the overall house infestation rate for that locality. In the case of *T. brasiliensis* in northeastern Brazil, for example, all houses are sprayed in localities showing 20% house infestation rates or more, whereas in localities with less than 20% house infestation rates spraying is confined only to those houses where infestation is confirmed. This survey and spraying cycle is generally repeated at 6-monthly or annual intervals, until all houses in the locality appear uninfested and that locality is then declared to be under the vigilance phase.

24.5.2 Vigilance Strategies

Vigilance, or epidemiological surveillance, represents a series of measures designed to detect—and respray if necessary—any new or surviving colonization of houses by vector Triatominae. In very general terms, this may involve house surveys by trained personnel of the operational vector control services (sometimes known as “active surveillance”) and/or community-based vigilance whereby householders are trained and requested to report the finding of any bugs in



Fig. 24.6. In Paraguay, after the vector control program, this family can now sleep peacefully, without being bitten by Triatominae.

their houses. Several “tools” are available to assist in this, including visual inspection of the house structure (using a torch to see into cracks, and long blunt forceps to withdraw any bugs found), spraying with irritant nonresidual pyrethroids (e.g., 0.2% aqueous tetramethrin) to dislodge any bugs that may be present, artificial refuges pinned to the house walls where bugs may collect, sheets of white paper, or calendars pinned to the house walls that may reveal streaks of recent bug feces, and householder information notices together with a self-sealing plastic bag in which householders can put any bugs they encounter. In addition, health education is now widely offered in schools, clinics, and others, to help ensure that householders are aware of Triatominae and Chagas disease, and willing to help in the community-based vigilance. In most cases, active vigilance is maintained for the first year following the initial control interventions, but progressively greater reliance is then given to community-based vigilance. This can be effective, but requires continual reinforcement (e.g., periodic community discussions) and a well-organized system of response to any notifications of possible new infestations. Communities that feel their notifications are ignored can quickly lose interest in collaborating with the public health services.

24.6 VIGILANCE AND RESEARCH

24.6.1 Research and Vigilance

For much of the last century, research on Chagas disease was focused on understanding the epidemiological problem and seeking ways to control the domestic vectors. These aspects are now sufficiently understood to provide the social and biological rationale for large-scale control interventions, together with demonstrable evidence of the success of well-organized interventions—both for blood-donor screening and for halting transmission by elimination of domestic vector populations. The research focus is, therefore, changing to concentrate much more on epidemiological and entomological vigilance, especially factors that influence vector domestication, and ways to sustain adequate vigilance in the face of changing epidemiological patterns. This requires a much greater understanding of Triatominae populations that currently have silvatic habits, but may—under certain circumstances—invade domestic premises and potentially form new domestic colonies.

24.6.2 Endangered Continuity

In many countries, especially in parts of the Southern Cone Region and Central America, the success of the vector control interventions has led to a decline in political priority for these interventions. This apparent paradox—known in Spanish as “el castigo del éxito” (lit: the punishment of success, i.e., self-defeating success)—derives from the widespread view that resource allocation for public health should be proportional to the perceived severity of the problem. Thus, where successful control interventions reduce this perceived

severity, then the premature assumption is made that the problem no longer requires continuing investment. Such a view risks future recrudescence of transmission, and takes no account of the marginal costs of reaching a sustainable end point after the initially successful interventions [2,21]. Moreover, with the ideological drive to decentralized public health services, irrespective of the biological and operational constraints of particular epidemiological patterns, it becomes even more urgent to conceptualize a future scenario in which disease transmission can be maintained at levels low enough to be compatible with projected health service capacity, but also with adequate provision for any new cases that may occur. For Chagas disease, control has long been conceptualized on the basis of intervention followed by various levels of surveillance (also known as “vigilance”), in which the most successful long-term programs have progressively adapted the surveillance strategy in accordance with changing epidemiological patterns [59].

24.6.3 The Role of Research

Even with progressive adaptations, long-term epidemiological surveillance becomes unsustainable unless it can be integrated with other community health care activities, which in turn risks losing the specialist expertise required, for example, in vector and parasite identification, and interpretation of epidemiological patterns and trends. Yet that specialist expertise is present, continually developing and, to a large extent, independently financed—in the research community. Potentially, therefore, it is to the benefit of all to develop closer links between the executive health services and the research community, such that the research becomes more focused on epidemiological surveillance, and the results of that research can be more readily assimilated within the health services themselves. The need for research to sustain the control initiatives has been well recognized—partly to provide the medical, social, and biological rationale underlying the political decisions, but also to interpret the changing epidemiological patterns and help to resolve operational problems. In Brazil, the research community played a key role in providing the social and biological arguments that prompted the first national campaign against Chagas disease during the 1980s [24,52].

24.6.4 The ECLAT Network

During the Southern Cone Initiative, the Andean Pact Initiative and the Central America Initiative, surveillance activities have been backed by development of the ECLAT network as a consortium of entomologists, geneticists, and control service personnel linked to the monitoring activities of the public health authorities, including preemptive studies of candidate or potential new domestic vectors. Through this mechanism, coordinated investigation projects were set up in almost every country of Latin America in collaboration with local health authorities, in a way that enabled a wide range of analytical techniques to be applied over the entire geographic range of each vector species. Network workshops provided

a forum for discussion of results with scientists and decision makers at the highest national level, as well as international observers. These contacts allowed scientists to turn their curiosity to some problems identified by health authorities, and these later were informed about new advances in the entomological or epidemiological knowledge. These meetings always gave rise to new research and collaborative projects, stimulating and improving what would have been otherwise only routine activities. Analysis of this paradigm—linking research and public health interventions—shows that it has worked well both to promote the control interventions themselves [20,21], and also to promote the essential research. At the international level, the research network has also helped provide greater continuity of action—even if only because the research community is generally less subject to the political changes that affect Health Ministry personnel and policies. But integration of similar networks at national levels has yet to be fully developed. In many countries, national research activities are largely independent of the national public health intervention services, with their results presented in scientific congresses and journals—rather than being also accepted as an integral part of the public health service itself. So a national research activity may reach the international scientific media, and only from there—sometimes—become incorporated back into the national public health services.

24.6.5 The ECLAT Lesson

From our experience (the authors are joint coordinators of the ECLAT network), we deduce a need within each endemic country to develop greater integration between the research scientists working on Chagas disease and its vectors, and the public health services with responsibility for Chagas disease surveillance and control. We base this conclusion on the following logic:

1. Chagas disease can be controlled by elimination of domestic vector populations using currently available techniques.
2. It is appropriate to do this, on medical, social, and economic grounds, in the sense that such interventions have high cost–benefit ratios, and high social value.
3. It is also appropriate to do this because it is biologically and operationally feasible to do so.
4. Successful control interventions will inevitably lead to a decline in resource allocation for Chagas disease surveillance and control.
5. Surveillance procedures focused specifically on Chagas disease vectors can be progressively adapted over the short to medium term, in accordance with changing epidemiological patterns. However, the quality of such surveillance will inevitably decline with success (i.e., declining likelihood of detecting new domestic populations) and also due to declining resource allocation.
6. The risk of new domestic infestations will remain as long as there are silvatic species of Triatominae that may adapt

to domestic conditions; but this adaptation represents an intriguing challenge for research scientists.

7. New domestic adaptations may become of continental health importance if the domestic species is (passively) spread over large geographical areas, as it probably has been the case for the present main targets (*T. infestans*, *R. prolixus*, and *T. dimidiata*).
8. Research on silvatic Triatominae can be promoted by the need for surveillance, and may be independently financed through research grants.
9. Scientists involved with such research are generally less subject to political changes, compared to their counterparts in Health Ministries, and may, therefore, provide greater continuity of investigation.
10. Research on silvatic Triatominae should, therefore, be closely linked with the public health authorities, and conceived as an independent but necessary component of national epidemiological surveillance.
11. National research councils and international research funding organizations should, therefore, seek to promote greater integration—and possible cofinancing—of research and surveillance activities in areas where Chagas disease transmission remains a potential threat.

24.7 CONCLUSION

For Chagas disease control and surveillance, we may conceptualize an end point at which existing domestic infestations of Triatominae have been eliminated. Such an end point would not be universally sustainable, except with a high degree of entomological surveillance coupled with selective interventions wherever necessary. But such surveillance would itself be unsustainable if successful, and so would tend to decline in interest, quality, and resource allocation. We propose, therefore, that the national scientific community be encouraged to play a closer role in entomological surveillance, providing continuity and detailed focal studies, together with a degree of interpretation from which additional surveys and interventions would be proposed where and when necessary.

We must recognize, however, that although this end point may include elimination of many populations of Triatominae, it cannot contemplate eradication either of all species of Triatominae or of the causative agent—*T. cruzi*. The parasite and its silvatic vectors will continue to exist throughout the Americas, and this may lead to occasional contact with humans and transmission of a new case of human infection. This component of our proposed end point has been termed—The Acapulco Syndrome—[24], whereby occasional transmission can be expected due to adventitious silvatic bugs that occasionally enter houses but do not establish domestic colonies. In such situations, vector control becomes largely irrelevant, and the main surveillance imperative rests with the clinical health services, because of the need for swift parasitological diagnosis and treatment of the acute infection.

REFERENCES

1. Abad Franch F, Paucar A, Carpio C, Cuba CAC, Aguilar HM, Miles MA. Biogeography of Triatominae (Hemiptera: Reduviidae) in Ecuador: implications for the design of control strategies. *Memorias do Instituto Oswaldo Cruz* 2001;**96**(5):611–20.
2. Akhavan D. Análise de custo-efetividade do programa de controle da doença de Chagas no Brasil. Report to the Ministerio da Sade (FNS), Brasília, Brazil, 1997, 28 pp.
3. Apt W, Aguilera X, Arribada A, Gomez L, Miles M, Widmer G. Epidemiology of Chagas Disease in Northern Chile: isozyme profiles of *Trypanosoma Cruzi* from domestic and sylvatic transmission cycles and their association with cardiopathy. *Am J Trop Med Hyg* 1987;**37**(2):302–7.
- [Q2] 4. Aragao MA. Sobre a dispersao do *Triatoma infestans*. *Rev Soc Bras Med Trop* 1971;**4**(4):183–91.
- [Q3] 5. Aufderheide AC, Salo W, Madden M, et al. A 9,000-year record of Chagas disease. *Proc Natl Acad Sci* 2004;**101**:2034–9.
6. Barrett TV, Hoff R, Mott KE, Guedes F, Sherlock IA. An outbreak of acute Chagas disease in the Sao Francisco valley region of Bahia, Brazil: triatomine vectors and animal reservoirs of *Trypanosoma cruzi*. *Trans R Soc Trop Med Hyg* 1979;**73**:703–9.
7. Basombrio MA, Schofield CJ, Rojas CL, Del Rey EC. A cost-benefit analysis of Chagas disease control in northwest Argentina. *Trans R Soc Trop Med Hyg* 1998;**92**:137–43.
8. Brazil RP, Da Silva AR. Triatomine vectors of *Trypanosoma cruzi*. Trypanosomes in urban areas of Sao Luiz, Maranhao Brasil. *Trans R Soc Trop Med Hyg* 1983;**77**(4):568.
9. Brener Z. Why vaccines do not work in Chagas disease? *Parasitol Today* 1986;**2**(7):196–7.
10. Brener Z. Pathogenesis and immunopathology of chronic Chagas disease. *Memorias do Instituto Oswaldo Cruz, Rio de Janeiro* 1987;**82**(Suppl.):205–86.
11. Briceño Leon R. Rural housing for control of Chagas disease in Venezuela. *Parasitol Today* 1987;**3**(12):384–7.
12. Bucher EH, Schofield CJ. Economic assault on Chagas disease. *New Scientist* 1981;**92**:321–4.
13. Casini CE, Dujardin JP, Martinez M, Pereira AB, Salvatella R. Morphometric differentiation evidenced between two geographic populations of *Triatoma infestans* in Uruguay. *Res Rev Parasitol* 1995;**55**(1):25–30.
14. Cattán PE, Pinochet A, Botto-Mahan C, Acuna MI, Canals M. Abundance of *Mepraia spinolai* in a periurban zone of Chile. *Memorias do Instituto Oswaldo Cruz* 2002;**97**(3):285–7.
15. Chagas C. Nova tripanosomiase humana. *Gaceta Médica da Bahia* 1909;**40**:440–53.
- [Q4] 16. Chagas C. A doença de Chagas. *Arch Bras Med* 1924;**14**:52–88.
17. Chavez T, Moreno J, Dujardin JP. Isoenzyme electrophoresis of *Rhodnius* species: a phenetic approach to relationships within the genus. *Ann Trop Med Parasitol* 1999;**93**(3):299–307.
18. Davila H, Beloscar JS, Bottasso OA, Morini JC. Alteraciones electrocardiográficas en individuos infectados con *Trypanosoma cruzi* con distinto tiempo de residencia en areas de alta endemicidad. *Medicina (Buenos Aires)* 1987;**47**:154–8.
19. Dias E. Doença de Chagas nas Américas. III América Central. *Rev Bras Malariol Doenças Tropicais* 1952;**4**:75–84.
20. Dias JCP, Schofield CJ. The evolution of Chagas disease (American trypanosomiasis) control after 90 years since Carlos Chagas discovery. *Memorias do Instituto Oswaldo Cruz* 1999;**94**(Suppl 1):103–21.
21. Dias JCP, Silveira AC, Schofield CJ. The impact of Chagas disease control in Latin America. *Memorias do Instituto Oswaldo Cruz* 2002;**97**:603–12.
22. Dujardin JP. Population genetics and the natural history of domestication in Triatominae. *Memorias do Instituto Oswaldo Cruz* 1998;**93**(Suppl II):34–6.
23. Dujardin JP, Muñoz M, Chavez T, Ponce C, Moreno J, Schofield CJ. The origin of *Rhodnius prolixus* in Central America. *Med Vet Entomol* 1998;**12**:113–5.
24. Dujardin JP, Schofield CJ. *The Trypanosomiasis*, chapter Triatominae: systematics, morphology and population biology. *CAB Int* 181–201, 2004.
25. Dujardin JP, Schofield CJ, Panzera F. *Los Vectores de la Enfermedad de Chagas. Investigaciones taxonomicas, biologicas y geneticas*. Académie Royale des Sciences d'Outre-Mer, Classe des Sciences naturelles et médicales, Traduction espagnole, 2002.
26. Dujardin JP, Schofield CJ, Tibayrenc M. Population structure of Andean *Triatoma infestans*: allozyme frequencies and their epidemiological relevance. *Med Vet Entomol* 1998;**12**:20–9.
27. Dujardin JP, Tibayrenc M. Etude de 11 enzymes et données de génétique formelle pour 19 loci enzymatiques chez *Triatoma infestans* (Hemiptera: Reduviidae). *Ann Soc Belge Med Trop* 1985;**65**:271–80.
28. Dujardin JP, Tibayrenc M. Etudes isoenzymatiques du vecteur principal de la maladie de Chagas: *Triatoma infestans* (Hemiptera: Reduviidae). *Ann Soc Belge Méd Trop* 1985;**65**(1):165–9.
29. Dujardin JP, Tibayrenc M, Venegas E, Maldonado L, Desjeux P, Ayala FJ. Isozyme evidence of lack of speciation between wild and domestic *Triatoma infestans* (Heteroptera: Reduviidae) in Bolivia. *J Med Entomol* 1987;**24**(1):40–5.
30. Feliciangeli MD, Dujardin JP, Bastrenta B, et al. Is *Rhodnius robustus* (Hemiptera: Reduviidae) responsible for Chagas disease transmission in Western Venezuela? *Trop Med Int Health* 2002;**7**(3):280–7.
31. Gaminara A. *Notas sobre Triatomas uruguayas, Sociedad Argentina de Patología regional del norte*, 1927.
32. Garcia BA, Soares Barata JM, Blanco A. Enzyme polymorphism among *Triatoma infestans* (Hemiptera: Reduviidae) colonies. *J Med Entomol* 1995;**32**(2):126–37.
33. Ken-Hong T. *Area-Wide Control of Fruit Flies and Other Insect Pests*, Penerbit Universiti Sains Malaysia, 2000, 782 pp.
34. Lumbreras H. El problema de la enfermedad de Chagas en los diferentes departamentos del Per. *Separata de la Rev Viernes Medico* 1972;**XXXIII**(1):43–77.
35. Moncayo A. Chagas disease: current epidemiological trends after the interruption of vectorial and transfusional transmission in the Southern Cone countries. *Memorias of Instituto Oswaldo Cruz* 2003;**98**(5): 577–91.
36. Monteiro FA, Perez R, Panzera F, et al. Mitochondrial DNA variation of *Triatoma infestans* populations and its implication on the specific status of *T. melanosoma*. *Memorias of Instituto Oswaldo Cruz* 1999;**94**(Suppl I):229–38.
37. Neiva A. Contribuição para o conhecimento dos hemipteros hematofagos de America Central. *Brasil Médico* 1915;**29**:1–3.
38. Osimani JJ. Enfermedad de Chagas: Importante flagelo de las zonas rurales del Uruguay. *Separata da Rev Goiana Med* 1959;**5**:339–56.

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39. Panzera F, Dujardin JP, Nicolini P, et al. Genomic changes of Chagas disease vector, South America. *Emerg Infect Dis* 2004;438–446.
40. Petry K, Eisen H. Chagas disease: a model for the study of autoimmune diseases. *Parasitol Today* 1989;5(4):111–6.
41. Rabinovich JE. Vital statistics of Triatominae (Hemiptera: Reduviidae) under laboratory conditions. *J Med Entomol* 1972;9(4):351–70.
42. Ribeiro Dos Santos R. Chagasic cardiopathy: a disease reflecting imbalance in the host-parasite relationship. *Memorias do Instituto Oswaldo Cruz, Rio de Janeiro* 1984;(Suppl. 79):67–8.
43. Rodrigues Coura J, Borges Pereira J. A follow-up evaluation of Chagas disease in two endemic areas in Brazil. *Memorias do Instituto Oswaldo Cruz, Rio de Janeiro* 1984(Suppl 79):107–12.
- Q5 44. Rothhammer F, Llop E, Acuna W. Is Chagasic cardiopathy associated with HLA haplotype? *Parasitol Today* 1986; 2(3):76.
45. Ruiz H. *Rhodnius prolixus* en Costa Rica. *Rev Biol Trop* 1953;1:139–240.
46. Sampson Ward L, Urdaneta Morales S. Urban *Trypanosoma cruzi*: Biological characterization of isolates from *Panstrongylus geniculatus*. *Ann Soc Belge Med Trop* 1988;68:95–106.
47. Santos D, Marcondes CB, Elesbao MAS, Madruga JP. Observacoes sobre a doenca de Chagas na Paraiba, Brazil. I. Primeiro encontro de *Triatoma infestans* (Klug) no estado, no municipio de Ouro Velho. *Cienc Cult Saude* 1981;(3): 15–7.
48. Schmunis GA. Iniciativa del Cono Sur. In: *Proceedings of the Second International Workshop on Population Biology and Triatominae, Tegucigalpa, Honduras* (C.J. Schofield and C. Ponce, eds), INDRÉ Mexico City, 1999, pp. 26–31.
- Q6 49. Schofield CJ. Chagas disease, triatomine bugs and blood-loss. *Lancet* 1981;1:1316.
50. Schofield CJ. Biosystematics of the Triatominae. In: *Biosystematics of Haematophagous Insects*, Vol. 37 (M.W. Service, ed.), Clarendon Press, Oxford, 1988, pp. 285–312. [Q7]
51. Schofield CJ. *Trypanosoma cruzi*—the vector-parasite paradox. *Memorias do Instituto Oswaldo Cruz* 2000;95:535–44.
52. Schofield CJ, Dias JCP. The Southern Cone Initiative against Chagas disease. *Adv Parasitol* 1998;42:1–27.
53. Schofield CJ, Dias JPC. A cost-benefit analysis of Chagas disease control. *Memorias do Instituto Oswaldo Cruz, Rio de Janeiro* 1991;86(3):285–95.
54. Schofield CJ, Diotaiuti L, Dujardin JP. The process of domestication in Triatominae. *Memórias do Instituto Oswaldo Cruz* 1999;94:375–8.
55. Schofield CJ, Dujardin JP. Theories on the evolution of *Rhodnius*. *Actualidades Biologicas* 1999;21:183–97.
56. Sosa ES, Segura EL, Ruiz AM, Velazquez E, Porcel BM, Yampotis C. Efficacy of chemotherapy with benznidazole in children in the indeterminate phase of Chagas disease. *Am J Trop Med Hyg* 1998;59:526–9.
57. Tibayrenc M, Ward P, Moya A, Ayala FJ. Natural populations of *Trypanosoma cruzi*, the agent of Chagas disease, have a complex multiclinal structure. *Proc Natl Acad Sci USA* 1986;83:115–9.
58. Walsh JA. Estimating the burden of illness in the tropics. In: *Tropical and Geographical Medicine* (K.S. Warren and A.A.F. Mahmoud, eds) McGraw-Hill, USA, 1984, pp. 1073–85.
59. Wanderley DMV. *Perspectivas de controle da doenca de Chagas no estado de Sao Paulo*. Thesis, University of Sao Paulo (Brazil), 1994, 161 pp.
60. World Bank. *World Development Report: Investing in Health*, Oxford University Press, New York, 1993, 329 pp.



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