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A COST-BENEFIT ANALYSIS OF CHAGAS DISEASE CONTROL

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Chagas disease transmission can be effectively interrupted by insecticidal control of its triatomine bug vectors. We present here a simple model comparing the costs and benefits of such a programme, designed to eliminate domestic populations of Triatoma infestans throughout its known area of distribution over the seven southernmost countries of Latin America. The model has been simplified to require only four financial estimates relating to the unit cost of housing spraying and benefits due to avoidance of premature death in the acute phase of the disease, avoidance of supportive treatment and care in the chronic phase of the disease, and avoidance of corrective digestive and cardiac surgery. Except for these direct medical costs, all other potential benefits have been ignored. Nevertheless, the model shows that the direct financial benefits of such a programme would far outweigh the costs, and the project would support a remarkably high internal rate of return under the least optimistic estimates.

Key words: Chagas disease - *Triatoma infestans* - cost-benefit - vector control

Chagas disease, also known as South American trypanosomiasis, is endemic throughout Latin America, from northern Mexico to southern Argentina and Chile. The disease is caused by infection with a protozoan parasite, *Trypanosoma cruzi*, transmitted in the faeces of blood-sucking reduviid bugs of the subfamily Triatominae. Of those infected, up to 10% succumb to the acute phase of the disease during the first few weeks after infection. The remainder, who may or may not experience acute phase symptoms, progress to an indeterminate symptomless phase lasting for several years, which may persist or, in about 40% of cases, develops to the debilitating chronic form of the disease. The chronic phase of the infection is often characterized by irreversible damage to heart muscle and digestive tract; in such cases the patient becomes progressively weaker and may die from heart failure or digestive complications. Severe cardiac complications requiring a cardiac pacemaker occur in about 0.2% of those infected, while severe digestive problems requiring corrective surgery occur in 3-4% (Dias, 1982).

Current WHO estimates suggest that 16-18 million people in Latin America are infected with *T. cruzi*. For practical purposes, chronic Chagas disease is incurable. Two drugs, nifurtimox and benznidazole can be used for very early infections, but early diagnosis is difficult and adverse side-effects can occur. Moreover, because *T. cruzi* antigens may stimulate autoimmunity (immune attack on host tissues) the likelihood of a safe effective vaccine now seems very remote. Control therefore relies primarily on the use of residual insecticides to kill the triatomine bugs in houses and so prevent them from transmitting the parasite. House improvement is another approach to rendering houses unsuitable for colonisation by triatomine bugs (Schofield & White, 1984; Briceno-Leon, 1987, 1990) but is not considered here because this approach is not widely used by control services in Latin America as it is considered too expensive as a public health intervention (Cedillos, 1988).

Of the 118 recognized species of Triatominae (Schofield & Dolling, in press), a relatively small number of species are epidemiologically significant as vectors of *T. cruzi*. These are species that colonise poorer quality rural houses, living in the cracks and crevices of walls and roof, and emerging at night to feed and defaecate on the sleeping

occupants. The most important vector species is *Triatoma infestans*, which is widely distributed in the seven southernmost countries of Latin America, and is considered responsible for most of the cases of Chagas disease occurring in those countries (Schofield, 1988). Unlike most other triatomine species, *T. infestans* is almost entirely restricted to domestic and peridomestic environments, and sylvatic colonies of these bugs have been confirmed only in small foci in the Cochabamba region of Bolivia (Dujardin et al., 1987). Because of its high level of domesticity, eradication of *T. infestans* is seen as a feasible target by the control authorities of some affected countries such as Brazil (Dias, 1987a, 1988).

This paper develops a very simple model to compare the costs and benefits of Chagas disease vector control throughout the known area of distribution of *T. infestans*. Such an analysis could become highly complicated due to regional differences over the large geographic area considered, and because of the inherent difficulties of assigning a value to healthy lives gained. The affected communities include a high proportion of subsistence farmers and labourers who are often considered to be only marginally productive. For these reasons, several simplifying assumptions have been made, any benefits due, for example, to "quality adjusted life years" have been simply ignored. In spite of this, the analysis suggests that even by considering benefits only in term of potential savings in medical costs, the vector control programme would show a substantial positive rate of return.

MATERIALS AND METHODS

Geographic area, affected communities and control strategy — The area under consideration is huge — over 6 million km² — representing the known distribution of *T. infestans*. It includes parts of seven countries — Argentina, Brazil, Bolivia, Chile, Paraguay, Peru and Uruguay (Fig. 1). Within this area, there are some other target species of Triatominae, notably *T. sordida*, *T. brasiliensis* and *Panstrongylus megistus*. But although these species differ in their relative susceptibility to different insecticides, domestic populations of these species would probably succumb to the same control measures applied against *T. infestans*.

The total population of the affected area is not known precisely, but can be estimated from available demographic data (UN, 1985) combined with

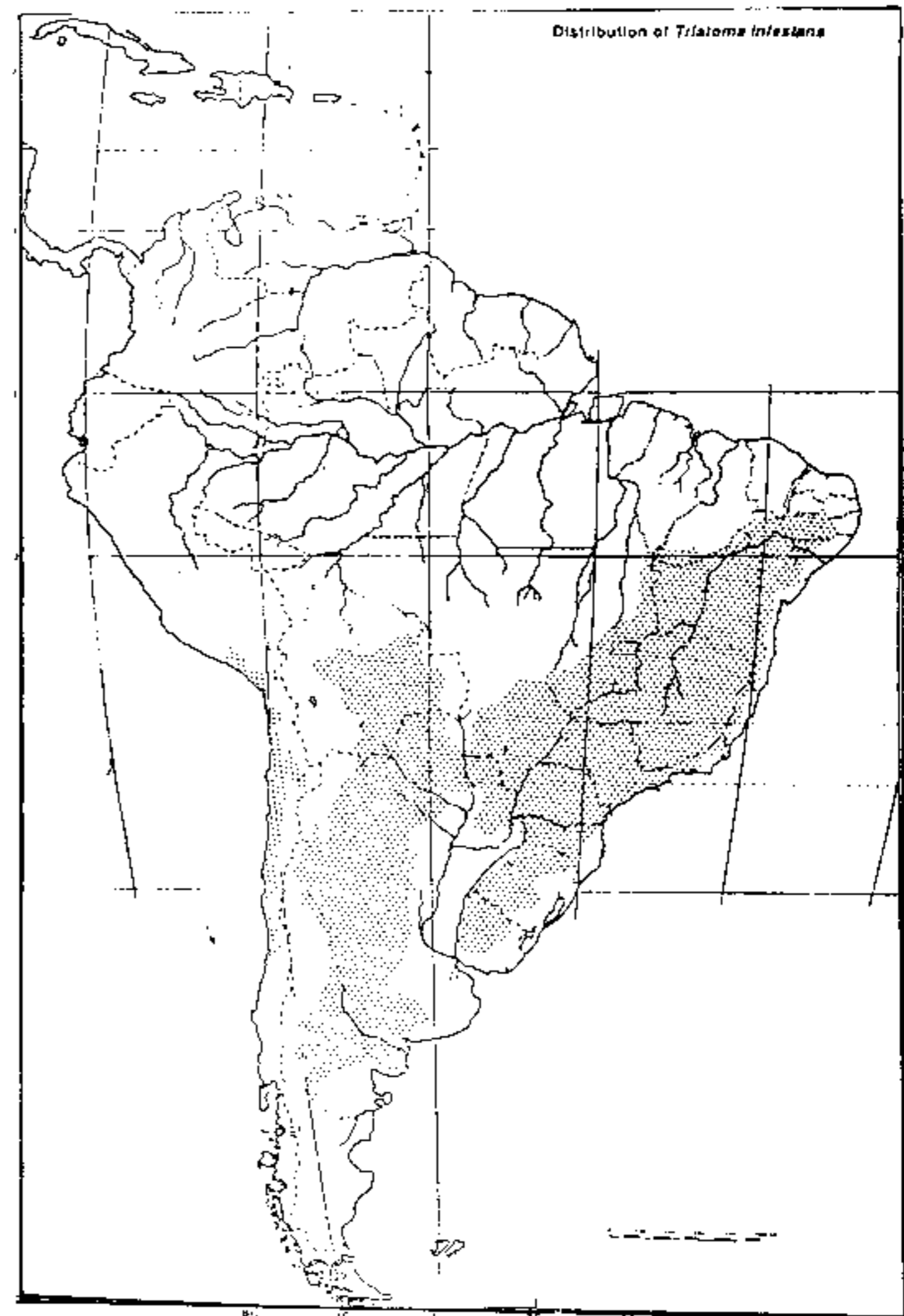


Fig. 1: approximate distribution of *Triatoma infestans*.

estimates of the proportion of people in the rural zone of each of the above countries (Table I). At an average of two adults and three children in each house, the population estimate then allows an estimate of the total number of houses to be treated.

The control strategy involves three phases — attack, consolidation and vigilance. This strategy follows that currently employed in Brazil and parts of other Latin American countries using currently available residual pyrethroid insecticides such as cypermethrin, cyfluthrin, deltamethrin, lambda-cyhalothrin etc. (Dias, 1987a, 1988). The attack phase involves spraying all houses in each locality, regardless of whether or not an individual house is known to be infested. This is for two reasons, firstly because of the relatively high cost of visiting and recording infestations in each house, and secondly because available sampling methods for domestic bug populations are very imprecise (Schofield, 1978; Pinchin et al., 1981) and so there is a risk that untreated houses many remain as foci from which bugs could reinfest treated houses (Schofield, 1985).

The consolidation phase envisages visiting each house after one year to assess the presence of

bugs, with a selective respray of all houses reported to be still infested, together with all neighbouring houses within a 200 meters radius in areas where *T. infestans* is prevalent. This is to ensure that no infested house might have given rise to other infestations in neighbouring houses, for example by active dispersal of the bugs (Schofield & Matthews, 1985). The vigilance phase, however, relies strongly on community participation, by which householders report the presence of any bugs in their houses to a local voluntary community worker, who reports periodically to a visiting inspector (Garcia-Zapata, 1985; Dias, 1987a). Houses are then resprayed as in the consolidation phase if the report is confirmed (i.e. the bugs found by the householders are indeed Triatominae).

The proposed duration of the model control programme is 10 years — a time period endorsed both by computer simulations (eg. Rabinovich, 1981, 1984) and by the experience accumulated in control trials in areas of Argentina and Brazil. However, because of the very large area to be covered, it would not be possible to mount the attack phase simultaneously over the whole area. For this reason, our model assumes that the attack phase is spread over the first three years, followed in each case by one year's consolidation phase, and then by the community-based vigilance.

Calculation of costs — Published data is available on the unit cost of house spraying in different countries of Latin America, both for Chagas disease vector control and for malaria control (eg. Rocha e Silva & França, 1965/66; Sherlock, 1979; Marsden, 1981; Dias, 1982; Marsden et al., 1983; SUCAM, 1987; Oliveira Filho, 1989). Estimates for Chagas disease range from a low of US\$4.00 per house for a small trial using BHC in Bahia, Brazil in 1978 (Sherlock, 1979), to around US\$66.00 per house for a more extensive trial in Chile in 1984 using propoxur. For these estimates, around 60-90% of the unit cost was in the delivery, with the remainder being the cost of the product applied. However, using organochlorines such as BHC, or carbamates such as propoxur, usually means that more than one spray per year is required for effective control of domestic Triatominae. In contrast, although synthetic pyrethroids are more expensive, their greater residual effect means that a single treatment may remain active for over a year. In the current study therefore, we have assumed exclusive use of synthetic pyrethroids, and followed the data of Oliveira Filho (1989) to estima-

te the average unit cost of house spraying at about US\$30.00 (Table II). In this we have included estimates of direct administrative overhead, as well as transport and maintenance costs, wages and per diems for spray teams and supervisors, and the cost of the insecticide itself. Figures published by the Brazilian Ministry of Health indicate similar unit costs of house spraying between US\$16.00 for BHC, and US\$18.43-27.25 for pyrethroids (SUCAM, 1987). However, variation in operational costs (particularly fuel prices) and in the prices paid for the insecticides (see Table III), shows that unit costs could rise as high as US\$70.00-90.00 (Oliveira Filho, 1989), which provides an upper cost limit for our sensitivity analysis.

In our estimate for programme costs, we have therefore taken US\$30.00 as the base cost of spraying one house during the attack phase, of which 40% represents the cost of the insecticide. To derive the cost of the consolidation phase, we assume that all houses will be visited and checked for bugs, but that the teams would be able to check the houses at twice the rate that they would spray them. Thus the unit cost would be US\$30.00 minus the cost of the insecticide, divided by two. However, we also assume selective respray of 10% of the houses (i.e. at the same cost as the attack phase). To derive the cost of the vigilance phase, we note that visits would be made only to those houses reporting infestation, and, taking experience from the vigilance phase of the current Brazilian programme, this would represent less than 1% of treated houses. However, assuming that at least some of these reports may be confirmed and require respraying, we have set the unit cost of vigilance at 2% of the attack cost. The robustness of all the above cost estimates is tested by sensitivity analysis of the complete model.

Thus, if the unit cost of respraying during the attack phase is CA, then the unit cost of the consolidation phase (CV1) is given by:

$$CV1 = (.1*CA) + (.27*CA)$$

and the unit cost of the vigilance phase (CV2) is given by:

$$CV2 = .02*CA$$

Total discounted costs (DC) are therefore given by:

$$DC = \sum_{I=1}^{I=10} H*((CA*A(I) + (CV1*V1(I)) + (CV2*V2(I)))/(1 + R)^I$$

where H is the total number of houses to be treated, $A(I)$ is the proportion in the attack phase in year I , $V1(I)$ is the proportion in the consolidation phase, $V2(I)$ is the proportion in the vigilance phase, and R is the discount rate. Note that the sum $(A(I) + V1(I) + V2(I))$ represents the proportions of people protected from infection during year I .

Calculation of benefits — We have assumed only three sources of benefit from Chagas disease vector control. Firstly, benefits due to avoidance of early death during the acute phase of the disease, secondly benefits accruing from avoidance of medical consultation and care for those in the chronic phase, and thirdly benefits due to avoiding the need for cardiac pacemakers or corrective intestinal and/or oesophagic surgery in the severely ill chronic patients. It should be emphasised however, that these categories do not include any estimates of non-financial benefits of health, and nor do they even include all the financial benefits that could accrue. For example, we have not included any estimate of the fraction of acute patients who receive medical consultation, care and treatment. Also, because of the effect of our discount rates (see below) we have not included any estimates or benefits due to averted deaths amongst chronic phase patients. Nor have we included any estimate of the benefits or avoidance of transfusional transmission of the disease in non-endemic areas.

In each year from the start of the programme, benefits accrue only in respect of that cohort of people who would have become infected during that year in the absence of vector control. Estimates of the annual incidence of infection for each of the countries involved are taken from the model of Hayes & Schofield (1990) (see Table I).

In calculating our three classes of benefits, we have assumed that the average age of infection ($X1$) is 15 years, and that the average of death ($X2$) is 52 years (Puffer & Griffith, 1968; Dias, 1982, 1987b; UN, 1985). Thus, for each year I , benefits accruing to avoidance of early death ($B1$) are given as follows:

$$B1(I) = IN * D * PP(I) * (CD - CD / (1 + R)^{(X2 - X1)})$$

where IN is the total number of individuals who would have been infected (i.e. the annual incidence of infection in the absence of control), D is the expected death rate due to acute disease, PP is the proportion protected by control programme, CD is the average financial cost of a death, and R is the discount rate.

TABLE I

Population and incidence of Chagas disease in the seven countries of Latin America where *Triatoma infestans* is the principal vector

	Popn (millions)	Popn in endemic region	Target popn	Annual incidence ^a
Argentina	28.24	0.3	8.52	63,888
Bolivia	5.60	0.5	2.80	86,676
Brazil	121.29	0.2	24.26	202,880 ^b
Chile	11.10	0.2	2.22	8,843
Paraguay	3.17	0.3	0.95	14,680
Peru	17.30	0.1 ^c	1.73	8,107 ^c
Uruguay	2.91	0.3	0.87	5,124
			41.35	332,698

^a: from Hayes & Schofield (1990).

^b: for Brazil, this estimate of incidence does not take account of the impact of the recent control programme.

^c: for Peru, one about one third of the total Chagas disease is considered due to transmission by *Triatoma infestans*. Other vectors such as *T. dimidiata*, *Rhodnius ecuadoriensis* and *Panstrongylus herreri* are important in central and northern parts of the Country. Thus, the estimated incidence for Peru given by the model of Hayes & Schofield (1990) has here been divided by three.

Similarly, annual benefits accruing to future avoidance of medical costs in the chronic phase ($B2$), are given by:

$$B2(I) = \sum_{J=X1+10}^{J=X2} (IN * S * PP(I) * CS) / (1 + R)^J$$

where S is the proportion of people who would have developed chronic phase symptoms, and CS is the annual cost of consultation, care and supportive treatment for those with chronic phase symptoms. These benefits accrue only for the period during which chronic phase symptoms would be apparent, i.e. for the J years between appearance of symptoms about 10 years after infection, and death.

The average cost of consultation, care and supportive treatment for chronic Chagas disease patients (CS) is here taken as US\$1,000 per year, based on figures published in Brazil. These figures include an average of 25 days hospitalisation per cardiac patient per year at a minimum cost of US\$30.00 per patient per day, plus an annual cost of US\$185.00 for treatment with the anti-arrhythmic drug amiodarone (Dias, 1987b; SUCAM, 1987).

Annual benefits due to avoidance of the needs for cardiac pacemakers or digestive surgery (B3) accrue only in respect of the small proportion of very serious cases (P). For these people, the benefits would arise on average some 20 years after infection, and are given by:

$$B3(I) = (IN * PP(I) * P * CP) / (1 + R)^{(X1 + 20)}$$

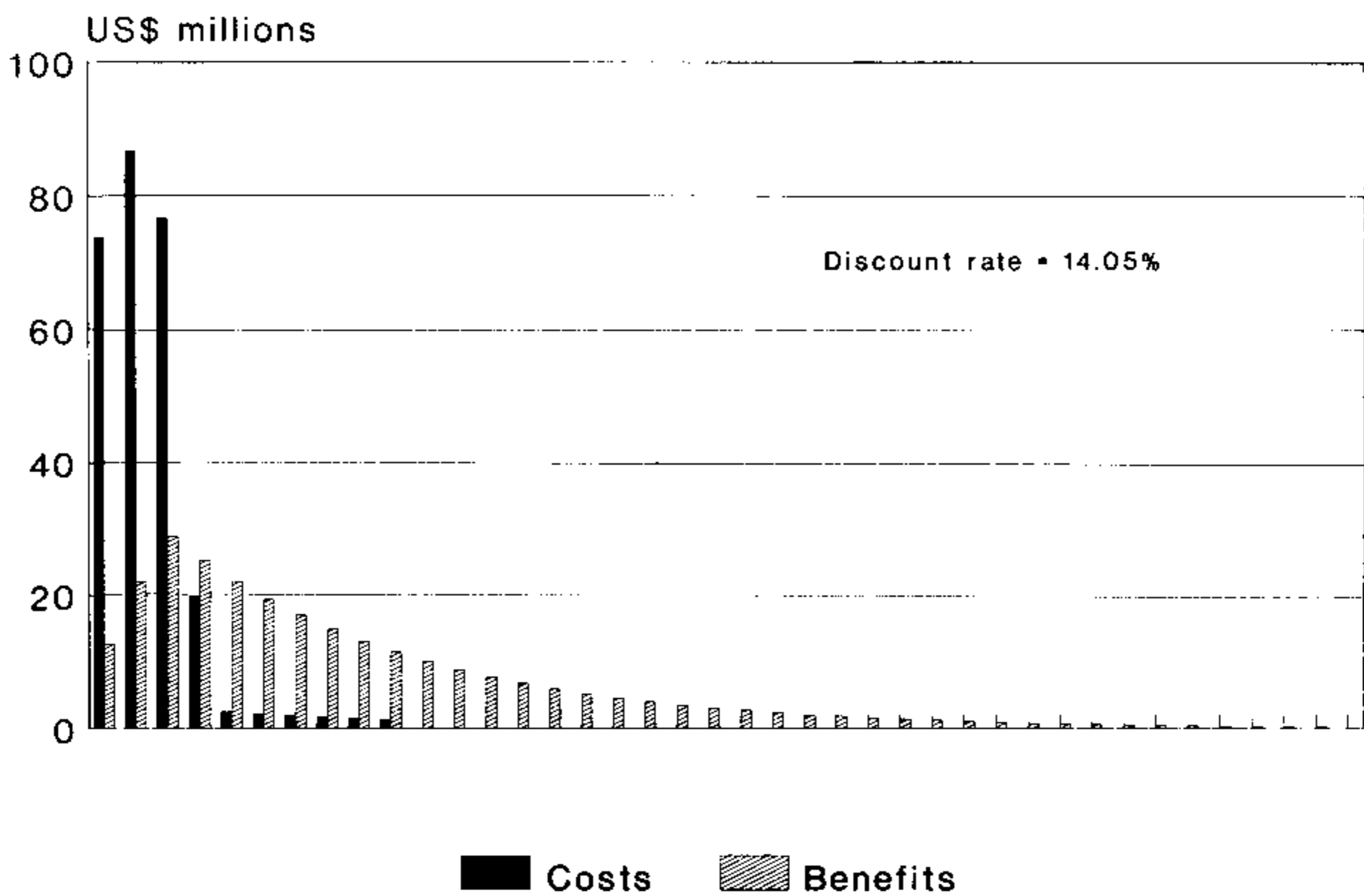
where CP is the average cost of pacemaker implant or corrective digestive surgery. Estimates for these costs are given by Dias (1987b) as follows (minimum prices): US\$2,500 for implantation of cardiac pacemaker (required by 0.2% of those infected in Brazil); US\$1,750 for corrective surgery for

grade III mega-oesophagus or mega-colon (required for 3-4% of those infected). The average cost is therefore taken as US\$1,790. Note however, that this is probably an underestimate given that the Brazilian Ministry of Health has estimated the average cost of pacemaker implant to be as high as US\$6,118 (SUCAM, 1987).

The total discounted benefits from these three categories (BB) is then given by:

$$BB = \sum_{I=X1}^{I=X2} (B1(I) + B2(I) + B3(I)) / (1 + R)^I$$

The above model, written in BASIC (see Annex), was run at various discount rates (R) to find at what rate the net present value (i.e. BB-CD) is approximately zero. This then represents the internal rate of return of the project in inflation-free terms, and is the opportunity cost of capital that could be supported (Mishan, 1976; Drummond et al., 1987). Using the base parameter values as shown in Table IV, the internal rate of return is 14.05% on a total discounted cost of US\$268.9 million (Fig. 2). [Actual costs (i.e. using zero discount rate) are US\$374.7 million].



RESULTS

Sensitivity analysis — With this model, costs are high during the first three years (peaking in the second year) due to the relatively high cost of the attack phase. Costs then decline steadily to the end of the intervention cycle after 10 years (Fig. 2). However, the model is quite robust to changes in the cost settings, such that a 20% decrease in the unit cost of the attack (which is then reflected in the cost of consolidation and vigilance) raises the internal rate of return only from 14.05% to 14.8%.

Benefits would accrue very quickly to the project, surpassing annual costs after three years (Fig. 2). The most significant of the three classes of benefits is the benefit that accrues to savings in consultation, care and supportive treatment of chronic patients. Benefits due to deferred death from the acute disease, and from avoidance of the need for cardiac pacemakers or digestive surgery, contribute a maximum of around 1% of the total benefits accruing in any one year. Moreover, in areas where a higher proportion of people enter the chronic phase, the benefits are improved — if the proportion of chronic patients is increased from 40% to 50%, then the internal rate of return is increased from 14.05% to 14.7%. Again however, the model is very robust even to changes in the es-

timated annual cost savings in respect of chronic phase patients. Halving the unit savings decreases the internal rate of return only from 14.05% to 12.08%.

On a “best” and “worst” comparison (Table IV), the model again shows its robustness. Using worst case figures (i.e. highest expected costs with lowest expected benefits) the internal rate of return falls to 8.2%, while with best case figures (i.e. lowest expected costs with highest expected benefits) the internal rate of return rises to 17.8%.

Variation in insecticide price — Since the model shows an attractive financial rate of return even on the least optimistic assumptions, the key parameter from the point of view of implementation becomes the price of the insecticide. For the seven countries considered here, most or all of the insecticide would be imported from elsewhere, implying payment in “hard” currency. However, the market price for suitable insecticides shows considerable variation — as illustrated by data from Brazilian Ministry of Health for 1985 and 1988 (Table III). Moreover price tariffs vary considerably between these countries, as do bulk freight costs. In addition, there are economies of scale that would be applicable to a project of this scope, such that a 10-fold increase in quantity purchased could lead to a discount of around 25% on the price per kilo.

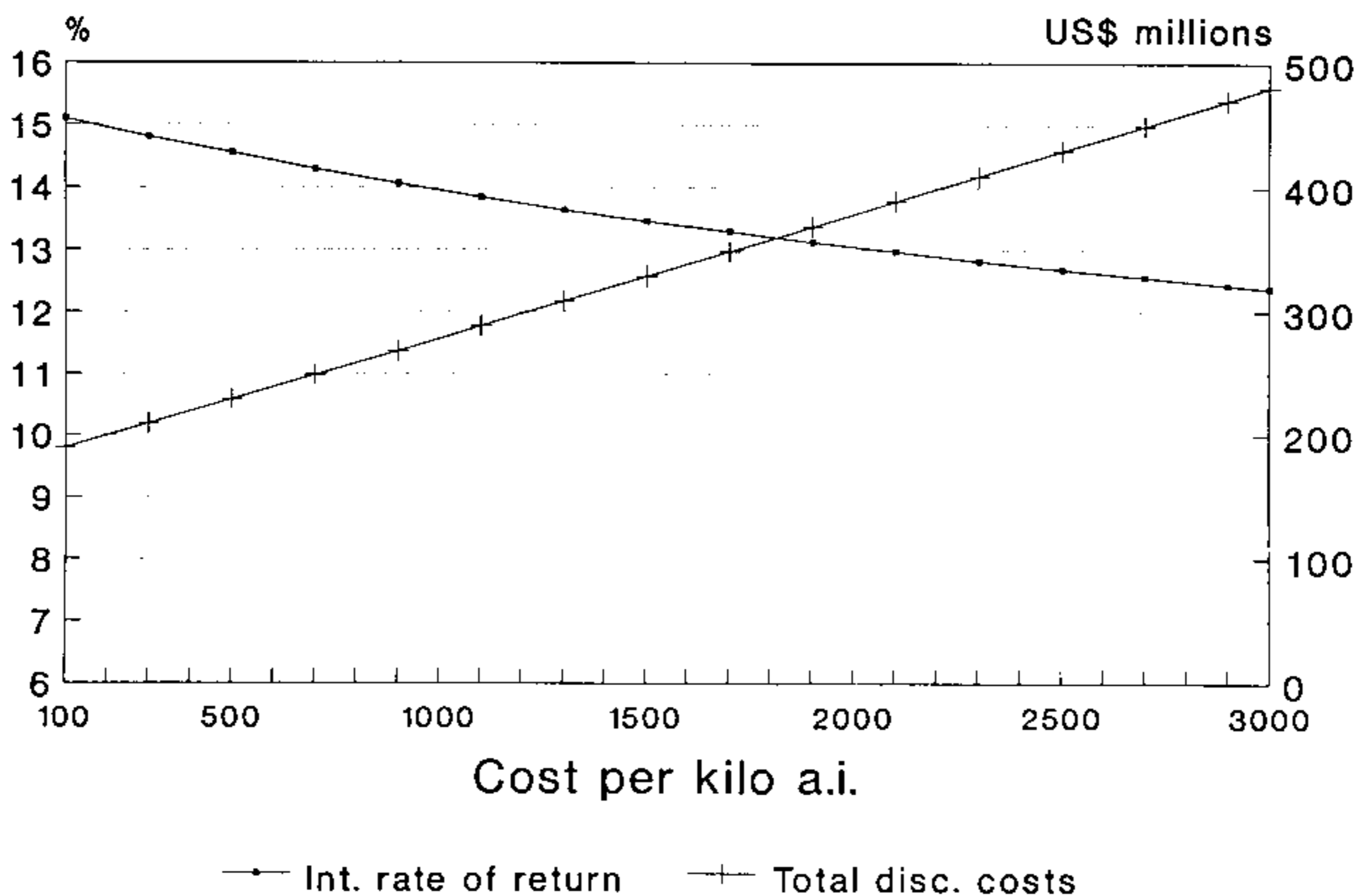


Fig. 3: variation in internal rate of return and total discounted costs at that discount rate, according to different insecticide prices.

TABLE II

Estimated unit costs (US\$) of house spraying against *Triatoma infestans*^a

Operational costs:	
Number of houses sprayed monthly/field team	240
Field team salaries and per diems (1)	1950
Supervision and technical support (2)	488
Administration and overhead (3)	360
	4448
Unit operational cost (4448/240) = US\$18.53	
Insecticide costs:	
Target spray rate	50 mg a.i./m ² ^b
Average surface area per house	250 m ²
Average deposit per house (250x50mg)	12.5 gm
Average cost per kilo (see Table III)	US\$921.00 ^c
Unit insecticide cost = US\$ 11.51	
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US\$ 30.04	

a: estimates follow those of Oliveira Filho (1989), except increased salaries and allowances, and updated transport costs. We have also included an extra allowance for administrative overhead.

b: can vary between 25-420 mg a.i./m² depending on insecticide used.

c: depends on recommended spray rate.

(1) : each field team of driver, inspector and three spray men, each paid US\$150/month, plus 20 days per diem (US\$ 12 per day); (2) : each team covers 100km per day; (3) : fixed overhead, equivalent to US\$1.5/house during the attack phase.

TABLE III

Pyrethroid insecticides — costs per kilo of active ingredient^a

	1985	1988
Cyfluthrin (Bayer)	474	1050
Cypermethrin (ICI)	142	344
Delta'methrin (Roussel-Uclaf)	640	2880
average (1985/1988) = US\$921.00		

a: from prices offered to SUCAM, Ministry of Health, Brazil.

TABLE IV

Definition of parameter values for the cost-benefit model

	Baseline value	Worst case	Best case
Annual incidence (IN)	332,698		
Number of houses (H)	827,0000		
Average age at infection (X1)	15		
Average age at death (X2)	52		
Propn dying in acute phase (D)	0.1	0.05	0.15
Propn developing chronic disease (S)	0.4	0.3	0.5
Propn requiring surgery (P)	0.037	0.018	0.075
Cost of death (\$) (CD)	100	50	150
Cost of chronic disease (\$) (CS)	1000	500	1500
Cost of surgery (\$) (CP)	1790	700	3500
Unit cost of attack phase (\$) (CA)	30	90	20

Worst case is defined as highest expected costs with lowest expected benefits.

Best case is defined as lowest expected costs with highest expected benefits.

Because of these complications and the difficulty of obtaining guide prices from different producers, we have used our model only to illustrate how changes in the price per kilo would influence total discounted costs (assuming that other operational costs remained unaffected) (Fig. 3). This analysis shows that the internal rate of return for the project remains attractive over the range of prices considered, although the steep slope of the total cost curve implies that increasing prices for the insecticide would be a constraint on such a project.

TABLE V

Estimates of working years lost due to premature deaths from acute and chronic Chagas' disease

	Target popn ^a	Working years lost ^a	GNP per capita ^c	Value (US\$ millions) ^d
Argentina	8.52	154,979	2,130	330.1
Bolivia	2.80	50,932	470	23.9
Brazil	24.26	441,289	1,640	723.7
Chile	2.22	40,381	1,440	58.1
Paraguay	0.95	17,280	940	30.2
Peru	1.73	31,469	960	16.2
Uruguay	0.87	15,825	1,660	26.3
				1208.5

a: from Table I.

b: from Pereira (1984), who estimated from mortality statistics in Brasília, the average number of working years lost per year due to premature death from Chagas disease as 2275/100000 population for males and 1363/100000 females. This implies an average of 1819/100000.

c: 1985 figures, from *The World Bank Atlas* (1987).

d: column b x column c.

DISCUSSION

Since 1947, when Dias & Pellegrino (1948) carried out the first trials of Chagas disease vector control using BHC in Minas Gerais, Brazil, there have been several Chagas vector control trials and campaigns in different parts of Latin America. Until the late 1970s these campaigns relied on organochlorine and carbamate insecticides (mainly BHC, dieldrin and propoxur), usually sprayed twice per year at a target dose of 0.5 to 2.0 grams a.i. per m². However, during the 1970s and 1980s, trials with highly residual synthetic pyrethroids such as deltamethrin and cypermethrin showed that these compounds were very effective against domestic Triatominae at very low doses (50 to 125 mg a.i. per m²), and retained residual activity — as indicated by bioassay on house walls — of up to 12 months or more (Dias, 1987; Oliveira Filho, 1989). Thus, although the pyrethroids were often more than 10 times the price of organochlorines per tonne), the lower doses used and consequently reduced freight and delivery costs, coupled with the need for only a single spraying, made them economically attractive.

The various insecticide trials and campaigns against triatomine bugs provide a good basis for the cost estimates used here. However, our estimates of benefits may be subject to some criticism. The cost-benefit model argues that control could be justified by comparison with benefits that accrue in respect of avoidance of costs attributable to the disease — most of which are due to the costs of medical consultation and care of chronic patients. Some might argue that such benefit estimates are unrealistic because most chronic patients do not have access to the care and attention they need. For example, very few of those requiring cardiac pacemakers would actually receive them. Precise estimates of actual expenditures in relation to the medical costs of the disease are not available. However, Bryan & Tonn (1990) cite data suggesting the estimated annual cost of medical care due to Chagas disease in Brazil alone is US\$250 million. This is considerably more than the annual benefits estimated here for the whole seven-country region (Fig. 2), suggesting that our estimates are very conservative. Bryan & Tonn (1990) also state that Brazil 'loses an additional US\$5000 million a year due to absenteeism caused by Chagas' disease'. In addition, Pereira (1984) has estimated an average of 1819 working years lost per 100,000 population per year due to premature deaths from Chagas disease amongst the working population. This, for the target population and average GNP *per capita* of the countries concerned, would translate to an annual loss of US\$1,208 million (Table V). Our model has not attempted to include such benefits, which, at any level, would add to the financial attractiveness of the vector control project.

The various campaigns against domestic Triatomines met considerable financial and organizational constraints, which seem fairly typical of control campaigning against other vector-borne diseases (cf. Arcoverde de Freitas, 1974/75). Often there were delays in importing the insecticides, thus curtailing the impetus and coverage of the initial attack phase. Funds and personnel were sometimes diverted into other priority tasks, such as control of mosquito vectors of dengue and yellow fever in outbreak areas — a problem faced by the current Brazilian Chagas control programme during 1986-87 (Dias, 1987a, 1988). But perhaps the most serious constraint was the lack of long-term continuity, especially in the vigilance phase of the programme. Thus even after a successful attack phase, the lack of continued vigilance allowed domestic infestations of vectors to recover, restoring transmission to the previous rates.

The rate of recovery of domestic triatomine populations following a control trial, has been subject of considerable research (cf. Schofield, 1985; Gorla, 1988). The process is complex but appears to depend on two key factors: (1) the intrinsic rate of population increase of the target vector species, and (2) the degree of successful control achieved (i.e. the number and distribution of houses and/or sylvatic and peri-domestic ecotopes in the case of vectors other than *T. infestans*, that retain a residual bug population). We can therefore model this by making two simplifying assumptions for the present analysis — by assuming that the distribution of susceptible houses is contiguous (i.e. there are no “barrier zones” that prevent active or passive dispersal of the bugs) and that any residual house infestations are randomly distributed amongst those susceptible houses*.

The cost implications of having no vigilance phase are minor but the implications for benefits are quite severe. Using our baseline parameter values (Table II) with a discount rate of 14.05%, then abandoning the vigilance phase reduces overall discounted costs by only 5.4%, but reduces overall benefits by 33%, with the stream of benefits falling to zero after 11 years. Moreover, although a programme of attack phase and consolidation, without vigilance, would still give an internal rate of return of 12.8% with our baseline parameter values, this drops to 6.4% on our “worst case scenario”, which is below the range generally considered acceptable for this type of project (Drummond et al., 1987).

The importance of the vigilance phase is also clear in biological terms. Our target vector species is *T. infestans*, not only the most important vector species in Latin America because of its high domesticity and wide distribution, but also the most vulnerable to available control measures. *T. infestans* is confined to the domestic and peri-domestic environments throughout its range, with sylvatic foci known only from the Cochabamba region of Bolivia — which is thought to represent

the original focus of the species (Dujardin et al., 1987; Schofield, 1988). Thus, the attack, consolidation and vigilance phases could be expected to reach all current foci of infestation. Moreover, *T. infestans* is a slowly reproducing species, with only two generations per year in the warmer areas of its distribution such as central Brazil, and only one generation per year in areas with cold winters such as central Argentina (Gorla & Schofield, 1989). As a result, its population recovery rate after control is low, so that continued vigilance over a 10 years period can be expected to eliminate all residual domestic infestations. Also, the low rate of reproduction — implying a low rate of genetic rearrangement — suggests that selection for insecticide resistance would be most unlikely over this time period.

Elimination of *T. infestans* as a domestic vector is thus seen as a feasible target by many authorities. There would still be some transmission of *T. cruzi*, partly by non-vectorial routes such as blood transfusion and congenital transmission, but these would decline rapidly due to the marked reduction in the incidence of infection due to successful vector control. Some transmission could also be expected due to sylvatic species of Triatominae occasionally invading houses, but experience in areas where sylvatic bugs are common but domestic bugs do not occur (such as the Amazon region) suggests that such occurrences would be rare. Thus, this analysis, taking a conservative approach to the limited data available and making a number of simplifying assumptions, suggests that elimination of *T. infestans* from the seven countries where it occurs would be an economically attractive project, both from a societal and government point of view.

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* With this assumptions, the model of the spread of infestation between houses simplifies to a trivial exercise of time-lagged exponential spread, depending only on the number of houses infested and the rate of increase of the bug populations (For a more detailed model which incorporates known rates and patterns of bug dispersal by flight and estimates of passive carriage by vertebrates, see Jedwab, 1986).

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ANNEX

```

10 H = 8.27
20 IN = 332698!
30 D = .1
40 S = .4
50 P = .037
60 X1 = 15
70 X2 = 52
80 DIM PP(X2),A(X2),V1(X2)
90 INPUT "r=";R
100 REM attack rate
110 A(1) = .34
120 A(2) = (1-A(1))/2
130 A(3) = A(2)
140 REM costs
150 CA = 30
160 CV1 = (.1*CA) + (.27*CA)
170 CV2 = .02*CA
180 CD = 100
190 CS = 1000
200 CP = 1790
210 REM calculate proportion protected/year
    
```

```

220 FOR I = 1 TO X2
230 V1(I) = A(I-1)
240 V2 = V2 + V1(I-1):IF V2 > 1 THEN V2 = 1
250 PP(I) = A(I) + V1(I) + V2
260 REM calculate costs per year
270 CY = 0:IF I > 10 THEN 310
280 CY = CA*A(I)*H
290 CY = CY + (CV1*V1(I)*H)
300 CY = CY + (CV2*V2*H)
310 CC = CY/(1 + R) I:REM discounted costs/year
320 DC = DC + CC:REM total discounted costs/year
330 REM calculate bens for deferred death
340 B1 = IN*D*PP(I)*(CD-CD/(1 + R) (X2-X1))
350 REM calculate bens for care of symptomatics
360 B2 = 0
370 FOR J = (X1 + 10) TO X2
380 B2 = B2 + (IN*S*PP(I)*CS)/(1 + R) J
390 NEXT J
400 REM calculate bens for pacemakers
410 B3 = (IN*PP(I)*P*CP)/(1 + R) (X1 + 20)
420 BY = (B1 + B2 + B3)/1000000!
430 BY = BY/(1 + R) I:REM discounted benefits/year
440 BB = BB + BY:REM total discounted benefits
445 PRINT CC, BY
450 NEXT
460 PRINT DC, BB
470 PRINT"-----"
480 DC = 0:BB = 0
490 V2 = 0
500 GOTO 90
    
```