

Probability of transmission of Chagas disease by *Triatoma infestans* (Hemiptera: Reduviidae) in an endemic area of Santiago del Estero, Argentina

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The daily probability (P) of transmission of Trypanosoma cruzi to a noninfected human host by an infected Triatoma infestans bug was estimated using field data from a 2-year longitudinal study carried out in a rural settlement of 20 households in Amamá, Santiago del Estero, Argentina. The following information was used for this purpose: the bug density and the proportion of infected bugs; the bug biting rate and the distribution of bites between humans and animals; the age-specific seropositivity to T. cruzi of the human population; and the actual number of new cases of human infection. The 2-year accumulated number of infective contacts per house estimated using a binomial model shows a statistically significant logistic correlation with the observed proportion of new cases per house.

An average house where new cases of human infection were registered in the 2-year period had a P value of 0.0012, while an average general house (i.e., with and without new cases) had a P value of 0.0009. The observed range of P is discussed in terms of the chain of factors that affects the individual human risk of acquiring the infection and the possible entomological sampling errors.

Introduction

No field evaluations of the transmission risk of Chagas disease in human populations have been carried out. The potential for transmission of *Trypanosoma cruzi* by different vector species, mainly in terms of their defecation rate, has been evaluated in the laboratory by Zeledón et al. (1) and by Kirk & Schofield (2). However, elucidation of the role played by the vector population in the transmission dynamics of Chagas disease has eluded investigators because extrapolations from animal models to humans are invalid and because assessments of the incidence of the disease in humans have seldom been carried out simultaneously with entomological evaluations. An exception to this is the association between the population density of *Panstrongylus megistus* and the incidence of human cases of Chagas disease established in Brazil by Piesman et al. (3).

Several mathematical models of the epidemiology of Chagas disease have used estimates of the probability of transmission by one infected vector; for example, Rabinovich & Rossell (4) used a probability

of 0.01 and performed sensitivity analyses using probability values between 0.0005 and 0.015 with *Rhodnius prolixus* as the vector. In another study with *R. prolixus*, Rabinovich used 0.0005 for the probability of transmission (5); subsequently Rabinovich & Himschoot used this value in a model study with *Triatoma infestans* as the vector (6).

The results of indirect field measurements, based on cross-sectional or longitudinal surveys, can be used to approximate the risk of infection. For example, in Castro Bahía in Brazil, Mott et al. found that in houses with at least one infected *P. megistus* the household rate of seropositivity to *T. cruzi* in both children and adults was positively correlated with the number of bugs captured (7). However, since the number of infected vectors was not determined the different levels of risk could not be established.

The first attempt to assess the risk of human infection with *T. cruzi* was made by Minter (8) and by Minter et al. (9). These workers proposed that the domestic risk factor (DRF) be expressed as the product of the number of triatomine bugs caught per man-hour of search and the proportion of bugs infected with *T. cruzi*, and compared this with a score that gave evidence of human infection. They found that the DRF index could be used to predict possible future infections and was loosely related to current infection status; although these authors suggested that this should be rigorously tested on a long-term basis, such a study was never carried out.

More recently Piesman et al. reported that in Castro Alvez the rates of infection of *P. megistus* with

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T. cruzi were significantly correlated with the number of seropositive children aged <18 years (10). In the same location Piesman et al. also found, after a 9-year longitudinal study, that the incidence of Chagas disease was significantly correlated with the density of both infected and noninfected bugs (3). These conclusions were based on three cross-sectional studies that involved insecticide spraying; the correlation between disease incidence and bug densities may therefore be spurious and not representative of the process of transmission being evaluated.

In a cross-sectional survey carried out in areas of Argentina with *T. infestans*, Gürtler et al. found statistically significant differences between the infection rates of *T. cruzi* in children and dogs from low-risk (<16 infected vectors) and high-risk houses (>40 infected vectors) (11).

Although all these studies attempted to estimate the *T. cruzi* challenge for human populations, true risk cannot be expressed as a probability. A complete and reliable estimation of the probability of transmission by an infected bug should be based on field data linked to a mathematical transmission model. The field data should include the following estimates for individual houses: bug population densities; proportion of infected bugs; bug biting rate; distribution of biting between humans and animals; age-specific seropositivity to *T. cruzi* in the human population; and the actual incidence of Chagas disease. This paper reports, for the first time, estimates of the daily probability of transmission of *T. cruzi* to a non-infected human host by an infected *T. infestans* bug, as assessed in a highly endemic area of Argentina. This probability, in conjunction with entomological data, should permit estimation of the potential level of risk of transmission in different houses, in order to define priorities for control operations.

Materials and methods

Field data

Raw data. The field estimates used to calculate the probability of transmission involved information reported by Gürtler,^a as well as unpublished data for bug densities, the proportion of infected bugs, and the bug biting distribution between humans and animals. The data that were used on the age-specific seropositivity to *T. cruzi* in humans have been reported by Wisnivesky-Colli et al. (12), while the bug

biting rate was that estimated by Catalá de Montenegro (13). The incidence of Chagas disease could also be obtained because of a delay in spraying the study area with insecticide until 1985 (see below) (13). Following is an outline of the essentials necessary to interpret the data used and the methods applied.

The data stem from a study carried out in Amamá (27° S, 63° W) in the Moreno Department, Province of Santiago del Estero, Argentina. The town is located in an area where the mean rainfall is 740 mm per year and the mean annual temperature, 22°C, with dense xerophytic vegetation dominated by the *quebracho* (*Schynopsis lorentzii*).

Twenty households that had never been sprayed during a Chagas disease control campaign were selected. The settlements, which were distributed over 400 km², were clusters of not more than five houses each and were isolated in the forest. Dwellings consisted mainly of two contiguous rooms (usually bedrooms) with mud and stick (*palo a pique*) walls, and occasionally mud-brick walls; roofs, which consisted of piled branches and mud, extended frontally forming a porch where people and dogs slept most of the year. The following peridomestic structures were surveyed: kitchens, store-rooms, maize store-rooms (*trojas*), and goat-pens.

The heads of each family were informed about the survey, and all of them signed consents. Through informal conversations with almost every member of the family and direct observation of the daily activities, the following information on the householders was registered: name, sex and age of each person, dogs and cats; the period that the family had lived there; the date that the house was constructed and of any recent improvements to it; the sleeping places of people and animals; domestic use of insecticides; and family income and educational level. Although this information was not used directly to estimate the probability of transmission of Chagas disease, most of it was essential for providing data about individual houses.

Two surveys were carried out, in November 1982 and November 1984. Using only forceps and torches, a two-person team collected *T. infestans* bugs in all areas of each house; beds were searched separately. All captured bugs were kept alive in labelled containers. Immediately afterwards, a second collection was undertaken in which bugs were dislodged from house walls and the roof by repeated spraying with a pyrethroid insecticide (0.2% v/v tetramethrin); insects were collected until no more bugs were found (on the average, 4 man-hours of search were spent on each house). For peridomestic structures, the mean searching effort was 1 man-hour. The captured bugs were immediately shipped to the laboratory for faecal examination and blood meal

^a Gürtler, R.E. [*Studies of the transmission of Trypanosoma cruzi in children and dogs in a rural area, with particular attention to the role of canine reserves: a 2-year follow-up study.*] Doctoral thesis, University of Buenos Aires, Buenos Aires, Argentina, 1988, pp. 181 (in Spanish).

studies. Determination of the *T. cruzi* infection status of the bugs was carried out by optical microscopic examination for trypanosomes of the rectal contents mixed with physiological saline at $\times 400$ (14). The origin of blood meals obtained from the promesenteric contents was determined using agar double-diffusion tests with specific antisera prepared in the laboratory (15).

Blood samples for serological testing were obtained by venepuncture from all members of households as well as from all dogs and cats, in both surveys. Since the human population was stable (90% of those surveyed in 1984 had been surveyed in 1982), individuals who were positive in 1984 but negative in 1982 were considered to be new cases and were used to calculate the incidence of Chagas disease. Human sera were mixed 1:1 with buffered glycerol and stored at room temperature. Serological analyses were performed at the National Institute for the Diagnosis and Investigation of Chagas Disease "Dr Mario Fatała Chabén" in Buenos Aires, using the following tests: indirect haemagglutination (IHA), direct agglutination (DA), indirect immunofluorescent antibody (IFA) and, in the case of results that did not agree, enzyme-linked immunosorbent assay (ELISA).

Details of these procedures have been reported by Ruiz et al. (16). The following titres were accepted as positive: 32 (IHA test), 16 (DA test), and 32 (IFA test); an ELISA absorbance of 0.20 was also considered positive. Individuals whose sera were positive in two or more tests were considered seropositive. Xenodiagnosis was performed on all children aged up to 12 years using 20 third or fourth instar *T. infestans* nymphs in two boxes.

The bugs were examined following the procedure described by Cerisola et al. (17). Human subjects were considered to be infected with *T. cruzi* if they were seropositive and/or positive for *T. cruzi* by xenodiagnosis (the latter procedure can detect recent acute cases that are still seronegative). Overall, approximately 51% of the humans were infected, and 41.2% of the children aged 0–9 years were positive. Detailed demographic and serological data have been reported elsewhere (18).

After the first survey in 1982 the area of Amamá and neighbouring houses were not sprayed until 1985 with a pyrethroid insecticide (deltamethrin; 25 mg/m²) by the National Control Agency. When the second survey took place, in November 1984, spraying had therefore not yet occurred; several children had become infected and this permitted calculation of the incidence of the disease in humans.

Criteria for data selection. Some studies (8, 19–21) as well as the data from Amamá and from La Invernada

(a village also in Santiago del Estero) (12) suggest that in endemic areas only children, usually the younger ones, become infected; for example, Lugones et al. have reported that 83% of 1500 acute cases of Chagas disease observed in Santiago del Estero over a period of 8 years were children aged under 10 years (22). Children up to 15 years of age were therefore considered to be susceptibles in the present study.

Data from only 12 of the 20 houses in the field study were included in the analysis. Information from the remaining eight houses was omitted because of one of the following: the house had been abandoned between 1982 and 1984 (house No. 1); the houses were not inhabited permanently between 1982 and 1984 (No. 8, 14, and 19); or the house had no children under 15 years of age (No. 6, 10, and 17).

Although the peridomestic areas (kitchens, store-rooms, maize store-rooms and goat-pens) were surveyed, they were not included in our analysis because about 99% of the bugs were found in the sleeping areas. The entomological data classified the bugs according to whether they were nymphs or adults and included also their sex; however, since no information on the differential biting rate by developmental stage or sex was available, no such discrimination was made in our analysis. Nevertheless, separately collected entomological data for walls and beds partly differentiated between nymphs and adults, since most of the bugs were strongly stratified, the beds being mostly infested with nymphs. Also, since the acquisition of the infection by humans is directly related to the proximity of bugs and people, the entomological data that were used kept beds and walls as a grouping. During the 1982 entomological survey all bugs collected were examined for *T. cruzi* infection; however, this information was available only for November. As the transmission model required data on the proportion of infected bugs for each month, the November 1982 values were assumed to remain constant for the rest of the year because the seasonal variability in the proportion of infected bugs was low: in the Province of Córdoba, an area similar to Amamá, the proportion of bugs that are infected has been reported to oscillate between 25% and 30% over the year (23).

The transmission model

The probability (P) that an infected bug will transmit *T. cruzi* to a human host is related to the risk of transmission (R) through a model of the form:

$$R = 1 - (1 - P)^n \quad (1)$$

where n is the number of infective contacts per person per night.

A contact is equivalent to a bite; however, since

the transmission is not inoculative but occurs through faecal contamination, we have used the term contact here. This equation corresponds to the binomial model, and is based on estimating the accumulated risk of not transmitting $(1 - P)$ the disease after n contacts, which when subtracted from unity gives the accumulated risk of transmission.

The daily number of contacts (n) of an infected bug is calculated using the following expression:

$$n = (V \cdot I \cdot F \cdot f) / H \quad (2)$$

where V is the total number of bugs per house; I the percentage of bugs that are infected, F the fraction of total bites made on humans, f the biting rate (number of bites per bug per night), and H the total number of people per house.

This model assumes that individuals in the group studied have the same susceptibility to *T. cruzi*, irrespective of age and sex, and also that biting is uniformly distributed among all human hosts, i.e., possible differences in human behaviour to biting and attractiveness to bugs were disregarded.

Parameter estimation

A program was written in FORTRAN 77 to estimate the probability of one infected *T. infestans* bug transmitting *T. cruzi*. For each house, the program carried out the following: 1) estimated the accumulated value of n for the 2-year period using data for V , I , F , f , and H obtained from field estimates; 2) assigned a value to the probability of transmission P ; 3) calculated the risk of transmission R ; 4) converted R to an incidence for a 2-year period, by multiplying the risk R by the number of susceptibles in each house; and 5) compared this incidence with that observed for the house in question. Operations 2-5 were carried out reiteratively, modifying the assigned transmission probability P by fixed steps—the process stopped when the difference between the observed and expected incidence of two successive estimates produced values in the χ^2 test that differed by less than 1%. The output of the program was therefore a value for the transmission probability P that minimized the difference between the observed and the expected incidences.

Since the field estimate for the population density of bugs in Amamá was available only for November, the other 11 monthly values were estimated using data on the monthly population distributions of *T. infestans* reported for experimental chicken houses (24). These monthly estimates reflected 4 man-hours' sampling effort and the values were corrected using a bug collection efficiency factor. Each man-hour of collection accounts for approximately 10% of the actual *T. infestans* popula-

tion (R. Chuit, personal communication, 1988) since the man-hour collection of *R. prolixus* domiciliary populations in Venezuela was found to be independent of bug density (J.E. Rabinovich, unpublished data), this factor (10%) was taken to be fixed for all houses. The number of bugs collected was therefore multiplied by 2.5 to arrive at the number of bugs in the house.

Of the 12 houses for which adequate information was obtained, only five (No. 3, 4, 11, 15, and 20) had new cases of human *T. cruzi*, and data from these were used in the above-mentioned procedure. Data from the other seven houses (No. 2, 5, 9, 12, 13, 16, and 18), which had no new cases, were used to fit a logistic regression between accumulated infective contacts and the proportion of new cases per house, and to analyse the distribution of houses with no new cases.

Since in addition to their monthly variations, the populations of bugs were not stable from year to year, the following estimation procedures were used: taking the bug population data for November 1982 and exposing the susceptibles for a 2-year period; the same, but using bug population data that were an average of the November 1982 and 1984 values; and "exposing" the human susceptible population to the November 1982 bug population data for the first year and to the November 1984 data for the second year.

Distribution of houses with new cases

With the assumption that the presence of new cases of Chagas disease in a house is independent of any such cases in another house, the observed frequency distribution of houses with different numbers of new cases per house was fitted to a Poisson distribution. A G goodness-of-fit test was used.

Results

The field data for the surveys in 1982 and 1984, which provide the essential data used in the computer program, are shown in Table 1. Shown also are data for two hypothetical average houses: the first expressed as the mean data for all the houses; and the second as the mean for only those houses that had new cases. In Table 2, data for house No. 5 are used to illustrate the application of equation (2). For example, the data in column (9) were obtained from the following operation:

$$[(\text{column } 5 \times \text{wall blood meal index}) + (\text{column } 7 \times \text{bed blood meal index})] \times (\text{column } 8/H)$$

where $H=10$, the number of people who lived in house No. 5.

Table 1: Field data used to estimate the probability of *Trypanosoma cruzi* transmission by an infected *Triatoma infestans* bug in Amama, Santiago del Estero, Argentina, in 1982 and 1984

House No.	No. of wall bugs ^a	No. of bed bugs ^b	HBI-wall ^c	HBI-bed ^d	No. of people ^e	SUS ^f	INC ^g
1982 Survey							
2	104 (78.9) ^h	16 (35.7)	27.3	60.0	4	1	0
3	86 (72.7)	0	—	—	7	3	2
4	195 (73.3)	0	22.6	—	8	4	2
5	176 (68.8)	36 (69.4)	26.5	18.2	10	2	0
9	69 (61.2)	1 (0)	41.5	—	3	1	0
11	172 (38.4)	14 (38.4)	29.5	—	6	3	2
12	7 (28.6)	0	33.3	—	8	5	0
13	12 (58.3)	0	—	—	9	6	0
15	83 (85.5)	0	43.7	—	9	2	1
16	7 (85.7)	44 (13.2)	—	100.0	6	2	0
18	4 (0)	0	53.3	—	7	2	0
20	77 (64.0)	155 (18.3)	—	—	4	1	1
1984 Survey							
2	60 (98.1)	9 (28.6)	20.4	20.0	4	1	0
3	12 (16.7)	0	—	—	7	3	2
4	131 (88.9)	47 (28.6)	10.2	36.4	8	4	2
5	164 (72.7)	99 (22.2)	22.8	34.4	10	2	0
9	33 (56.8)	0	18.2	—	3	1	0
11	231 (32.3)	74 (30.4)	33.9	67.6	6	3	2
12	147 (50.8)	22 (15.4)	35.2	71.4	8	5	0
13	10 (20.0)	12 (0.0)	100.0	100.0	9	6	0
15	57 (77.8)	13 (16.7)	11.1	40.0	9	2	1
16	46 (21.9)	97 (8.7)	20.0	58.5	6	2	0
18	4 (100.0)	12 (12.5)	—	—	7	2	0
20	19 (25.0)	213 (55.8)	—	64.7	4	1	1
Mean (T) ⁱ	79.3 (58.8)	56.2 (28.5)	33.6	62.2	6.8	2.7	0.6
Mean (+) ^j	106.3 (60.8)	51.6 (31.4)	31.8	61.7	6.8	2.6	1.4

^a No. of bugs collected after 4-man-hours' search of all walls and roofs.

^b No. of bugs collected in beds.

^c Human blood index (% of bugs with human blood in their intestines) for bugs collected on walls.

^d Human blood index for bugs collected in beds.

^e Total number of persons in the household.

^f Total number of human susceptibles (non-infected children up to 15 years of age).

^g Crude incidence (number of susceptible children who became infected between 1982 and 1984).

^h Figures in parentheses are the % of bugs that were infected.

ⁱ Average for a hypothetical house based on all data available (both surveys).

^j Average for a hypothetical house representing only houses with new cases (non-zero incidence).

Addition of these 12 monthly values (after multiplying column 9 by the number of days in each month) gives the accumulated number of infective contacts per person per year in a given house. In turn, multiplication of this value by two produces the total exposure to infective contacts for a 2-year period, which can be correlated with the observed incidence per house in the 1982–84 period (Fig. 1). The data were fitted to a logistic regression, and a statistically significant goodness of fit at the $P=0.05$ level was obtained.

Table 3 shows the calculated probabilities P of transmission of *T. cruzi* to a susceptible human host by an infected *T. infestans* bug, minimizing the difference between the observed and expected incidences

for the set of houses with new cases of Chagas disease. Also shown are the values of P that apply to the two hypothetical average houses in Table 1. P ranged from 0.0006 to 0.0038 for individual houses; for an average house, calculated using the mean of all the data, $P=0.0009$, while for an average house defined as one with new cases, $P=0.0012$.

By fitting the number of houses with new cases of Chagas disease to a Poisson distribution, the average risk of new infections per household per night ($\gamma=0.0007384$) was obtained, while the observed and predicted values for a 2-year period are shown in Table 4. The G test for goodness of fit was 1.323, which was statistically significant at the $P=0.05$ level ($0.75 > P(\chi^2 > 1.323) > 0.5$, with two

Table 2: Results of the monthly calculations used to determine the mean number of potential infective contacts per person per night for house No. 5 in the 1982 survey^a

Month ^b (1)	Goria's series ^c (2)	No. of wall bugs ^d (3)	No. of infected wall bugs ^e (4)	No. infected wall bugs (corrected) ^f (5)	No. of bed bugs ^g (6)	No. of infected bed bugs ^h (7)	Biting rate ⁱ (8)	Mean infective contacts ^j (9)
1	205	97.5	67.1	167.7	19.9	13.8	0.50	2.35
2	385	183.1	126.0	315.0	37.5	26.0	0.45	3.97
3	225	107.0	73.6	184.1	21.9	15.2	0.23	1.19
4	200	95.1	65.4	163.6	19.5	13.5	0.03	0.14
5	180	85.6	58.9	147.3	17.6	12.2	0.01	0.04
6	175	83.3	57.3	143.2	17.0	11.8	0.03	0.12
7	165	78.5	54.0	135.0	16.0	11.1	0.01	0.04
8	160	76.1	52.4	130.9	15.6	10.8	0.17	0.62
9	155	73.7	50.7	126.8	15.1	10.5	0.03	0.11
10	330	157.0	108.0	270.0	32.1	22.3	0.10	0.76
11	370	176.0	121.1	302.7	36.0	25.0	0.17	1.44
12	405	192.7	132.6	331.4	39.3	27.3	0.43	3.99

^a Figures in parentheses are the column numbers. The results (column 9) were obtained using equation (2) and correspond to the parameter *n* in equation (1) in the text.
^b Month 1=January; month 12=December.
^c See ref. (24); used to calibrate the November 1982 data from Amamá.
^d Expected number of bugs on walls found after 4-man-hours' collection.
^e No. of wall bugs × 68.8% (see Table 1, house No. 5).
^f Values in column (4) × 2.5.
^g No. of bugs collected in beds (not affected by correction factor).
^h No. of bed bugs × 69.4% (see Table 1, house No. 5).
ⁱ Estimated number of bites per night per bug (see ref. 13).
^j Average total monthly number of contacts per person, obtained by adding columns (5) and (7), weighted with the corresponding human blood index (in house No. 5, the proportion of bugs with human blood found on walls and in beds was 0.265 and 0.182, respectively).

Fig. 1. Logistic regression fit of the observed 2-year incidence of Chagas disease in susceptibles up to 15 years of age as a function of the accumulated potentially infective contacts.

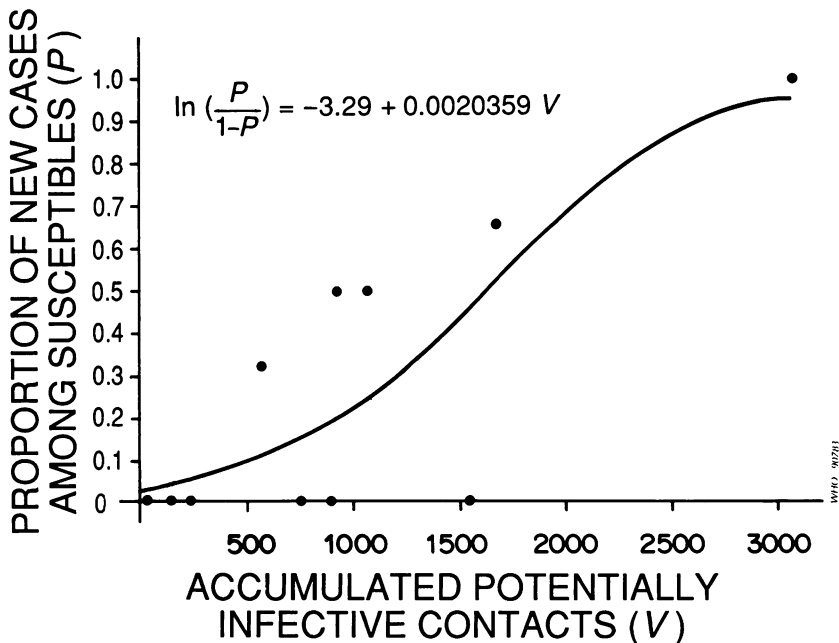


Table 3: Estimated probability of transmission (*P*) of *Trypanosoma cruzi* per bite of an infected *Triatoma infestans* bug (obtained using the data in Table 1)^a

House No.	Probability of transmission using data for:		
	November 1982	Average ^b	Combination ^c
3	0.0007	0.0038	0.0024
4	0.0006	0.0009	0.0009
11	0.0006	0.0007	0.0007
15	0.0007	0.0015	0.0013
20	0.0024	0.0030	0.0024

^a The probability of transmission in the hypothetical house, Mean(T), was 0.0009 and in the hypothetical house, Mean(+), 0.0012 (see Table 1).

^b Determined using an average of the bug population data for November 1982 and November 1984.

^c Determined using bug population data for November 1982 for the first year of exposure and similar data for November 1984 for the second.

Table 4: Observed and expected number of houses (determined using a Poisson distribution) with new cases of Chagas disease over a 2-year period

No. of new cases per household	No. of houses	
	Observed	Expected
0	7	7
1	3	3.77
2	2	1.02
3	0	0.18

degrees of freedom). A Poisson distribution requires that the average number of new cases per household in 2 years be $2 \times 365 \times \gamma = \ln(7/12) = 0.539$, which agrees well with the observed average number of new cases per household of 0.583 over 2 years for the seven new cases in 12 houses; see column "INC" in Table 1. Since the average number of susceptible individuals per household was 2.7 (see row "Mean (T)" of column "SUS" in Table 1), the average daily risk per susceptible individual is $\gamma/2.7 = 0.000274$, and the average risk per susceptible individual per infective contact (equivalent to our previous definition of the probability *P* of transmitting infection through one infective contact) is $0.000274/1.35685 = 0.000202$, where 1.35685 is the average daily number of infective contacts from all houses (except house No. 18, which had only four bugs).

Discussion

The six basic items of field data (see Introduction) that were used to assess the probability of an infected

bug transmitting *T. cruzi* were estimated with different degrees of reliability, as discussed below.

The estimates of the bug population density were based on the following assumptions: there is a linear relationship between the sampling efficiency and total capture effort—based on an efficiency of 10% for 1-man-hour collecting effort, we concluded that an efficiency of 40% could be used for a 4-man-hour effort; the correction factor of 0.4 between a 4-man-hour collection and the actual population of wall bugs was taken to be independent of bug population density and the age of the house; the bug population densities for the months with no field estimate were determined assuming that the same seasonal variation found for an experimental chicken house applies also to households—although this may be questionable, the climatic conditions in Córdoba are similar to those in Santiago del Estero.

The proportion of infected bugs was assumed to remain constant all year-round at the value of the November estimate. Although this proportion could vary with time, data from Córdoba indicate that the proportion of infected *T. infestans* bugs shows little seasonal variation (23). However, the estimate for November corresponds to one of the highest annual rates of infection, since by late spring adult and elder nymphs are the most abundant bugs (24). The effect of this assumption is to decrease the final estimate of *P*; however, the magnitude of the underestimation is of minor importance because those months when the proportion of infected bugs is lower are also those with lower bug population densities.

Estimates of the monthly biting rate made in Córdoba (13) were for the same experimental chicken houses from which the bug population densities were obtained; the rates ranged from about once every 2 days in summer (December–February) to about once every 100 days in winter (May–July). These values are reasonable since the biological activity of the bugs is almost completely arrested in winter (25). In this respect, any differences between the two sites would be restricted to April, which is warmer in Amamá than in Córdoba, and would produce a corresponding higher biting rate.

The seasonal variation in the feeding profile of the bugs is restricted to a change in the distribution of their feeds on different animals, with the number of feeds on humans being essentially the same in summer and winter (C. Wisnivesky-Colli, unpublished data). We therefore assigned to each house the November estimate of the human blood index.

The serological information and the detection of new cases are based on standardized techniques and constitute probably the most reliable estimates used to evaluate *P*, the probability of transmission.

The estimate of the mean number of potential

infective contacts per person per night for each month exhibits an interesting seasonal distribution. For example, for house No. 5 (Table 2) 93% of all potential infective contacts occurred during the 6 months October–March. This is consistent with field observations in Argentina that most acute cases of Chagas disease appear between September and March (21, 26).

The logistic regression between the 2-year accumulated potential infective contacts and the proportion of new cases per house (Fig. 1) has an overall sigmoid form, but some of the observed values deviate markedly from the curve. Infection is a discrete process and with a sample of only 11 houses such deviations are to be expected; the logistic regression is therefore a good statistical representation of the nature of the infective process. With no new cases the number of infective contacts becomes zero, while at very high numbers of infective contacts the curve asymptotically approaches unity. We therefore feel justified in ascribing the deviations in the observed values from the fitted regression to sampling errors. By fitting a Poisson distribution to the frequency of houses with new cases, the average risk per susceptible individual per infective contact (equivalent to the probability, P , of transmitting infection through one infective contact) was found to be 0.0002, which is reasonably close to the P value of 0.0009 estimated using the binomial model with average values for all houses (Mean (T) in Table 3). Thus, using two separate methods to analyse the data, we arrive at the conclusion that approximately 1000–2500 infective contacts are necessary to produce an infection in a susceptible human aged 0–15 years.

Our estimate of the value of P ranges from 0.0006 to 0.0038; however, by combining the data for 1982 and 1984 the range of values was reduced considerably (see Table 3). A word of caution is necessary when analysing these results: the value of P calculated is a probability resulting from a process involving a complex chain of biological events that were not evaluated independently. They entail the following: whether the bugs' faeces are or are not deposited on the human host; where on the body the faeces are deposited; the number of infective metacyclic trypanosomes in the faeces of the bugs; the possible existence of differential human susceptibility to *T. cruzi*; and the possible development of human resistance to infection with increasing age. These factors, which varied in different ways in each house, combined with entomological sampling errors, may well account for the wide range in the values of P estimated here.

Additionally, although differential human susceptibility to *T. cruzi* has not been demonstrated, our results show that under similar epidemiological con-

ditions the individual risks of acquiring the infection appear to be highly variable. This, however, requires further investigation.

Since the exact date of human infection was not known in the study, we assumed that infections took place at the end of the 2-year period (November 1982 to November 1984). Thus, if in a particular house a new case of human infection occurred in a shorter period than this, it would result in an underestimation of the probability of transmission, P .

Although the analysis tended to underestimate P , and the values obtained exhibited a fairly wide range, our results show that the probability of transmission is very low, i.e., that the vector is very inefficient. Our estimates of P should therefore be taken into consideration in the design of control operations. For vector control programmes, the objective thus need not be total eradication of the bugs, provided adequate surveillance measures following a satisfactory spraying campaign result in sufficiently low bug population densities to interrupt disease transmission.

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Résumé

Probabilité de transmission de la maladie de Chagas par *Triatoma infestans* (Hémiptère: Reduviidae) dans une région d'endémie de Santiago del Estero, Argentine

On a estimé la probabilité (P) de transmission de *Trypanosoma cruzi* par *Triatoma infestans*, un réduve, au moyen des données recueillies sur le terrain au cours d'une étude longitudinale de deux ans (novembre 1982 à novembre 1984), menée dans une localité rurale comprenant 20 foyers; les habitations n'avaient jamais reçu de pulvérisation dans le cadre d'une campagne de lutte contre la maladie de Chagas et étaient réparties sur 400 m² dans la localité d'Amamá, Santiago del Estero (Argentine). P a été calculé à l'aide d'un modèle binomial de transmission; un programme infor-

matique a été mis au point en FORTRAN 77 et a permis d'estimer P en minimalisant la différence entre le nombre de nouveaux cas observés et le nombre de nouveaux cas attendus dans chaque foyer.

Le modèle de transmission utilise les informations suivantes: densité des réduves par maison, estimée par une recherche de 4 heures-homme en moyenne par maison; pourcentage de réduves infestés, déterminé par examen microscopique direct (au grossissement 400 ×) du contenu rectal, à la recherche de trypanosomes; taux de piqûres donné par la littérature; répartition des piqûres entre l'homme et l'animal, déterminée par double diffusion en gélose avec les repas de sang obtenus à partir du contenu du proventricule; sérologie par âge de la population humaine, par hémagglutination indirecte (IHA), agglutination directe (AD), immunofluorescence indirecte (IFI) et, en cas de résultats discordants, par une méthode immunoenzymatique (ELISA); enfin, incidence chez l'enfant de 15 ans et moins (les sujets ont été considérés comme infestés par *T. cruzi* s'ils étaient séropositifs et/ou positifs pour *T. cruzi* par xéno-diagnostic).

L'emploi d'un modèle binomial de transmission a donné des estimations de P allant de 0,0006 à 0,0038 dans les cinq maisons où des nouveaux cas sont apparus; pour ces maisons P est en moyenne de 0,0012, alors qu'en général (c'est-à-dire sur l'ensemble des maisons avec et sans nouveau cas) la valeur obtenue est de 0,0009. Toutes ces valeurs expriment le risque par sujet sensible par contact potentiellement infestant. Théoriquement, P devrait être constant; l'intervalle de variation observé est probablement dû à la chaîne complexe de facteurs qui entrent en jeu dans le risque individuel de contracter cette infestation. Par exemple, les déjections de l'insecte sont-elles ou non déposées sur l'hôte humain? à quel endroit? quel est le nombre de trypanosomes au stade métacyclique (infestant) présents dans ces déjections? existe-t-il différentes sensibilités à *T. cruzi* chez l'homme? y a-t-il émergence d'une résistance humaine à l'infestation avec l'âge?

Outre le modèle binomial de transmission, la distribution observée de la fréquence des maisons présentant des nombres différents de nouveaux cas a été ajustée à une distribution de Poisson au moyen d'un test G . Les résultats donnent un risque moyen par sujet sensible par contact infestant de 0,0002, ce qui est inférieur mais pas trop éloigné de la valeur 0,0009 de P estimée avec le modèle binomial. Ainsi, par deux méthodes différentes appliquées à la même information de base, peut-on conclure qu'il faut entre 1000 et 2500 contacts

infestants pour produire une infestation chez un sujet sensible de la tranche d'âge 0-15 ans.

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