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INFLUENCE OF HUMANS AND DOMESTIC ANIMALS ON THE HOUSEHOLD PREVALENCE OF *TRYPANOSOMA CRUZI* IN *TRIATOMA INFESTANS* POPULATIONS IN NORTHWEST ARGENTINA

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Abstract. In three rural villages of northwest Argentina, the overall proportion of domiciliary *Triatoma infestans* infected with *Trypanosoma cruzi* was 49% among 1,316 bugs individually examined for infection in March and October 1992). Most of the variation among individual households in the proportion of infected triatomines was explained by variations among houses in the proportion of bugs that fed on dogs or cats, the prevalence of infected dogs or cats, and the proportion of bugs that fed on humans, according to a logistic multiple regression analysis. The effects of human infection rates on bug infection rates were not statistically significant. After adjusting for the effects of other predictors, the presence of chickens in bedroom areas had negative and significant effects on the proportion of infected *Triatoma infestans*, and positive and significant effects on the number of *T. cruzi*-infected triatomines collected per person-hr per house. Dog or cat infection rates and the proportion of bugs that fed on dogs or cats and on chickens explained 80% of the total variance of infected-bug numbers in a linear multiple regression model. This is the first study to use detailed field data to show that variations in triatomine infection rates depend on bug host feeding patterns and dog or cat infection rates, while the presence of chickens in bedroom areas exerts opposite effects on the proportion and number of infected triatomines. Domestic animals play a crucial role in the domiciliary transmission of *T. cruzi*.

Triatoma infestans Klug, probably the most important and widespread domiciliary vector of *Trypanosoma cruzi*,[1] showed one of the highest rates of infection with *T. cruzi* (median = 34%, range = 12–68%) in extensive surveys carried out when insecticidal campaigns were nil or at an early stage in several countries.[2] For young men drafted into military service from all over Argentina, the Province of Santiago del Estero (northwest Argentina) had the highest prevalence rate of seropositivity for *T. cruzi* among all province-wide prevalence rates in the period 1964–1969 when the first screening took place, and the third highest prevalence rate in 1981.[3] In this province, the percentage of *T. cruzi*-infected *Triatoma infestans* collected from human sleeping quarters (hereafter domiciliary areas) was high (44%) and widely variable (range = 21–82%) from district to district.[4] More recently, domiciliary vector infection rates were 31% more than 10 years after the first spraying of houses with insecticides in northwest Santiago del Estero.[5] Explaining such variable infection rates of domiciliary triatomines within a given community or province may increase our understanding of the transmission dynamics of *T. cruzi*.

Vector feeding patterns link hosts, vectors, and the transmission of parasites in either direction between hosts and vectors. Dogs, chickens, and humans are the most important host blood sources of domiciliary *Triatoma infestans* throughout its range.[6] In northwest Argentina, the feeding pattern of domiciliary *Triatoma infestans* varied seasonally, with higher frequencies of feedings on chickens during spring–summer due to the presence of hens nesting indoors.[7] In spring and summer bug collections, chickens and dogs were preferred to humans with respect to their relative numbers; the more the bugs fed on chickens or dogs, the less they fed on humans.[8] Dogs also were the principal domestic reservoir of *T. cruzi*.[9] This explains why in previous studies, vector infection rates increased with the number or proportion of infected dogs[10] or infected dogs or cats[11] in the house. However, these previous studies did not adjust for differing age distributions of the bug populations from different houses, and did not consider variability among houses and vector feeding patterns, to establish a causal link between host and vector infection rates.

Chickens and other birds are not susceptible to *T. cruzi*, and avian blood exerts no deleterious (or positive) effect on an established infection within the vector.[12] The effects of chickens on the domiciliary transmission of *T. cruzi* are still unresolved. Minter[13] stated: ''It's not yet clear whether the presence of chickens in houses is beneficial (reduced overall *T. cruzi* bug infection rate, some predatory activity) or the reverse (support a larger total bug population).'' Recently, Cecere and others,[14] using multiple regression analysis, showed that the domiciliary density of *Triatoma infestans* significantly increased both with the proportion of bugs that fed on chickens and with the house-dwellers' habit of letting hens nest in bedroom areas. Up to now, the only published data on the effects of the presence of chickens on the transmission of *T. cruzi* within domiciliary triatomine populations were taken at a single house.[15]

The prevalence of infection among vector populations is closely connected to the risk of human infection.[16] In a mathematical model for the African trypanosomiases that includes animal and human hosts,[17] the equilibrial vector prevalence of infection depends on human and animal infection rates and their contact rates with vectors, among other factors. For Chagas' disease, the number of domiciliary triatomines infected with *T. cruzi* collected per person-hr (hereafter, infected-bug density) was closely related to the human prevalence and incidence of infection.[18] As part of a larger project aimed at building an empirically based mathematical model of *T. cruzi* transmission, we use logistic multiple regression analysis to explain variations among houses in vec-

tor infection rates and infected-bug densities as dependent variables, using data on bug feeding patterns and host infection rates as independent variables. To our knowledge, this is the first study to use such detailed field data to study variations in triatomine infection rates. We also investigated whether the presence of chickens in bedroom areas modified bug infection rates with *T. cruzi* or the density of infected *Triatoma infestans.* Our test controls statistically for the effects of infected humans and dogs or cats in the house.

MATERIALS AND METHODS

Study area. Studies were undertaken in the rural villages of Amama, Trinidad, and Mercedes in the province of Santiago del Estero, Argentina. The villages were situated in a semiarid, hardwood forest habitat at 27°12′S, 63°2′W.[10, 19] All houses in Amama were sprayed once with deltamethrin for the first time in October 1985. This spraying eliminated domiciliary infestations by *Triatoma infestans* for an average of three years. Trinidad and Mercedes had never been sprayed with insecticides before the present study, and because they are adjacent and roughly similar, they were treated as one village.

Study design. A house-to-house census of people, dogs, and cats, and a serologic survey were carried out in March 1992.[9] Entomologic surveys were conducted in March[20] and October 1992. In March 1992, two experts from the National Chagas Disease Service searched all human sleeping areas (1 person-hr) and peridomestic sites (0.5 person-hr/house) for live *Triatoma infestans.* Peridomestic sites included all outbuildings, such as toolsheds and animal shelters. Roofs and walls were sprayed with an aerosol flushing-out agent (0.2% tetramethrin; Icona, Icona, Buenos Aires, Argentina) to augment bug captures. As part of a comparative study on triatomine sampling methods, an average of three sensor boxes (Biosensor Detector de Vinchucas, Biocientifica de Avanzada®; Buenos Aires, Argentina) placed on bedroom walls 30 days previously were inspected for hidden triatomines and other signs of infestation in Amama only. After flushing-out collections were completed, insecticide fumigant canisters were applied to demonstrate whether flushing-out collections were a valid index of bug density and to reduce heavy bug infestations until more effective methods were applied.

In October 1992, all houses were treated with deltamethrin.[21] As a by-product, the triatomines knocked down during the first 24 hr after spraying were collected by house-dwellers and the research team to enlarge the bug sample size at each house for analyses of infection by season.

All bugs collected at each house were stored separately according to the site and method of capture. The bugs were stored in labeled plastic bags with folded filter paper inside. Bug species were identified and instar frequencies were counted at the field laboratory. Live or moribund third instar nymphs and older stages were individually examined for infection within 5–10 days of capture. First and second instar nymphs were seldom captured, and because they had low or nil infection rates in previous studies, were excluded from infection analyses. The sampling fraction varied with the capture month. In March bug collections, when the total number of triatomines captured by the flushing-out or sensor boxes at each house was less than 50, all were examined for infection; when more than 50 bugs were captured by the methods at each house in March, approximately 30–50% of each instar collected were examined. In the abundant October collections obtained by knockdown, approximately 20% of all triatomines were examined for infection. All specimens to be examined for infection were chosen from among the plastic bags in which bugs were stored in each house without regard to whether the insects were engorged with blood. Fecal drops obtained by abdominal compression to each bug were diluted with one drop of saline solution, covered with 22 × 22 mm coverslips, and thoroughly examined for active trypanosomes at 400×.

The methods used in the dissection of triatomines and identification of blood meals have been previously described.[7] The individual blood meal sources of all bugs were identified by agar double-diffusion tests using five antisera representing the most common domiciliary hosts (humans, dogs, cats, chickens/ducks, and goat/sheep). Mixed blood meals are those in which at least two types of hosts can be recognized. Unmixed blood meals are those in which only one host type is recognized, although the bug may have fed multiple times on the same type of host. A reactive bug is one that showed a positive result to at least one of the antisera used. For a given blood meal source, we calculated the proportion of reactive *Triatoma infestans* that fed on that blood source (the host blood index), whether or not the bugs had fed on any other host.

Serology and xenodiagnosis. The Ethical Review Committee of the National Chagas Institute ''Dr. Mario Fatala Chaben'' of the Argentine Ministry of Health and Social Welfare reviewed and approved the procedures of the project in 1992. The objectives and activities of the entire research project were explained to the head of each family, and signed written informed consent was obtained.[9] Blood samples from humans and dogs were obtained by venipuncture and tested by the indirect hemagglutination (IHA) test, the indirect immunofluorescent antibody test (IFAT), and an ELISA at the Instituto Nacional de Chagas following standard procedures.[9] Titers (IHA and IFAT) of 1:32 or greater (for human sera), or 1:16 or greater (for dog sera), or an optical absorbance of 0.2 or greater for the ELISA (for both species), were considered positive for *T. cruzi* infection. For both human and dog sera, seropositive refers to samples reactive by at least two different serologic tests. To complete and refine the data beyond those already published,[9] new blood samples obtained in 1993 and 1994 (after deltamethrin spraying) were used for a definitive diagnosis of a few cases that had been serologically discordant or missing in March 1992. To assess the probability of infection of *Triatoma infestans* after a single blood meal on an infected host,[9] xenodiagnosis of a sample of the seropositive humans and seropositive dogs was carried out from October to December 1992. Cats were tested by xenodiagnosis, but not serologically, due to the difficulties in handling and bleeding them. A careful record of humans, dogs, and cats in each household was kept.

Data analysis. Using logistic multiple regression analysis implemented in Statistica (Statsoft, Seattle, WA) for Windows (release 4.3), we investigated the variation in the proportion of infected triatomines as a function of the variation

in the proportion of infected hosts of each type and host-vector contact, as measured by host-feeding patterns. Logistic regression analysis was preferred to standard linear regression because an individual bug is either positive or negative for *T. cruzi* infection and a sample of bugs has a fraction positive between 0 and 1 that has a binomial distribution. The fitting of the model was carried out iteratively by maximum likelihood. Because domiciliary *Triatoma infestans* populations have a low rate of active dispersal, we consider each house as the relevant sampling unit where most of the transmission of *T. cruzi* takes place. For a meaningful analysis of proportions of infected triatomines, we selected houses with at least 10 bugs examined for infection and at least 10 reactive bugs. This restrictive criterion, selecting for 22 houses, was later relaxed to enlarge the study base and see how this restriction affected the outcome of regression analyses. Most *Triatoma infestans* examined for infection and blood meals were collected in March 1992 and then pooled with additional collections made in October 1992 to increase sample size. Results reflect rates of bug infection for the period March–October 1992.

Before analysis began, we calculated a directly age-standardized proportion of infected bugs at each house to adjust for variations in age distributions among houses. To this end, we multiplied the instar-specific infection rate at each house times the instar-specific total number of bugs examined for infection in the data base divided by the total number of bugs examined. The relationship between the age-standardized proportion of infected bugs (PINFBUG) (the dependent variable) and the contributions due to the various factors was initially assumed to be logit PINFBUG = $b_0 + b_1 \times$ DCBI $+ b_2 \times$ INFDC $+ b_3 \times$ HBI $+ b_4 \times$ INFHU (equation 1), where b_0–b_4 are regression coefficients, DCBI is the proportion of reactive *Triatoma infestans* with dog or cat blood meals (dog-cat blood index), INFDC is the proportion of infected dogs or cats (by serology and/or xenodiagnosis), HBI is the proportion of reactive *Triatoma infestans* with human blood meals (human blood index), and INFHU is the proportion of humans seroreactive to *T. cruzi.* Cats were fewer than dogs; therefore, they were pooled with dogs to avoid creating a new variable. For similar reasons, cat and dog blood meals were pooled, and only some interactions for main effects variables and for biologically plausible interactions (DCBI with INFDC and HBI with INFHU) were added to the model. Missing data for the number of infected humans or dogs in two houses and missing data on bug feeding patterns at several houses were replaced by the observed conditional means. Backward stepwise elimination of nonsignificant variables was used to obtain the most parsimonious model that retained all significant independent variables. A maximum likelihood χ^2 was computed as twice the difference between the log-likelihoods of the fitted and the null model. This general procedure was repeated using the number, instead of the proportion, of infected hosts as independent variables in equation 1. Residuals were visually inspected for consistent deviations from normality and presence of outliers. The fitted coefficients were used to compute adjusted odds ratios and confidence intervals.

The basic equation 1 was then extended by including another term representing the effects of the presence of chickens in domiciliary areas. This variable was alternatively the chicken blood meal index (CKBI, proportion of reactive triatomines with chicken blood meals, regardless of whether bugs gave evidence of meals on other hosts), the chicken blood meal index only (CKBI ONLY, proportion of reactive bugs with only chicken blood meals), and the reported or observed presence of hens brooding indoors.[14]

Variation in the number (per person-hr of searching) of infected domiciliary *Triatoma infestans* per house in relation to host infection rates and host blood indices (the independent variables) was studied by multiple linear regression analysis and backward stepwise elimination. We initially used the same house selection criteria as before; however, because the dependent variable was the number of infected triatomines, for this particular analysis we also selected houses with at least five or 10 *Triatoma infestans* collected by flushing-out in March 1992. The dependent variable NINFBUG was calculated as the number of *Triatoma infestans* collected per person-hr in March 1992 times the age-standardized proportion of infected *Triatoma infestans* in March and October 1992 (x), transformed to logarithms to the base 10 (log[x + 1]). The regression equation was NINFBUG = $b_0 + b_1 \times$ DCBI $+ b_2 \times$ INFDC $+ b_3 \times$ HBI $+ b_4 \times$ INFHU $+ b_5 \times$ CKBI (equation 2).

RESULTS

Community–wide data. Overall, *Triatoma infestans* was collected in 92% of the examined houses in the three villages. Domiciliary *Triatoma infestans* fed on humans (53%), dogs (48%), chickens (38%), and cats (12%) in March and October 1992.[7] The overall proportion of domiciliary *Triatoma infestans* infected with *T. cruzi* was 49% among 1,316 triatomines individually examined for infection in March and October 1992 (Table 1). Trinidad–Mercedes had a significantly higher bug infection rate (56%) than Amama (46%) when stratified by instar. In Amama, infection rates significantly increased from 41% in bugs collected in March to 65% in October 1992, whereas a significant decrease from 62% to 48% occurred in Trinidad–Mercedes (Figure 1). The distribution among households of bug infection rates in both villages was unimodal, but in Amama it was more skewed to the right than in Trinidad–Mercedes. The infection rates of domiciliary *Triatoma infestans* increased with instar from approximately 23–30% in third instar nymphs to 45–70% in adult triatomines regardless of the methods (flushing-out combined with sensor boxes, knockdown) and dates (March, October) of bug collections (Figure 1).

The overall prevalence of *T. cruzi*-infected *Triatoma infestans* collected in peridomestic sites was only 3% among 335 bugs examined for infection. The infection rates of peridomestic bugs did not differ significantly between Amama (3.5% of 289 bugs) and Trinidad–Mercedes (6.5% of 46 bugs) ($\chi^2 = 1.0$, degrees of freedom [df] = 1, $P > 0.3$). The infected insects were fourth or higher instars collected from kitchens and storerooms (but not from corrals), which were located 10–20 m away from bedrooms and frequently used by dogs and cats as a resting place.

The age-specific prevalence of seropositivity for *T. cruzi* and parasitemia of humans and dogs were reported elsewhere.[9] In Amama, seropositivity was detected in 34% of 225 people and in 65% of 83 dogs tested; parasitemia was

TABLE 1

Trypanosoma cruzi infection rates of domiciliary *Triatoma infestans* collected by different methods in bedroom areas from Amama, Trinidad–Mercedes, Argentina, March and October 1992

Community	Date	Collection method	No. of houses*	No. of bugs			% infected†
				Collected	Examined	Infected	
Amama	March	Flushing–out‡	34	1,299	720	294	41
	October	Knockdown	27	909	196	128	65
	Total			2,208	916	422	46
Trinidad–Mercedes	March	Flushing–out	20	261	239	147	62
	October	Knockdown	18	720	161	77	48
	Total			981	400	224	56
Overall				3,189	1,316	646	49

* Houses where bugs were collected.
† Differences analyzed by Mantel–Haenszel test stratifying for fourth instars, fifth instars, and adult bugs (degrees of freedom = 1): between villages aggregated over time: $\chi^2 = 7.14$, $0.001 < P < 0.01$; March versus October differences within villages: Amama, $\chi^2 = 24.3$, $P < 0.001$; Trinidad–Mercedes, $\chi^2 = 13.0$, $P < 0.001$.
‡ Includes bugs concurrently collected from inside sensor boxes.

detected by xenodiagnosis in 29% of 41 seropositive persons and in 85% of 34 seropositive dogs.

Household data (Appendix 1). The age-standardized proportion of infected *Triatoma infestans* (in March and October 1992) correlated significantly with the log-transformed number of triatomines collected by flushing-out (Figure 2A) or knockdown ($r = 0.57$, $N = 34$, $P < 0.001$) in March 1992. Similarly, the age-standardized number of infected bugs significantly increased with the log-density of bugs assessed by flushing-out in all villages ($r = 0.75$, $N = 57$, $P < 0.001$), or by knockdown in Amama (Figure 2B).

The feeding patterns of triatomines at each house and the relation between *T. cruzi* infection and host blood source were scrutinized for discrepancies with census and interview records. Two households that reported that they did not own any dogs or cats (A-06 and A-102) in fact provided part-time housing for these animals owned by relatives located 10–50 m from their houses. In both houses there were *Triatoma infestans* that had fed on dogs or cats and also showed a high rate of *T. cruzi* infection, indicating the presence of infected animals. Therefore, in these houses only, dog and cat infection data were corrected using the number of infected animals in the relatives' house.

We carried out logistic multiple regression analyses of the

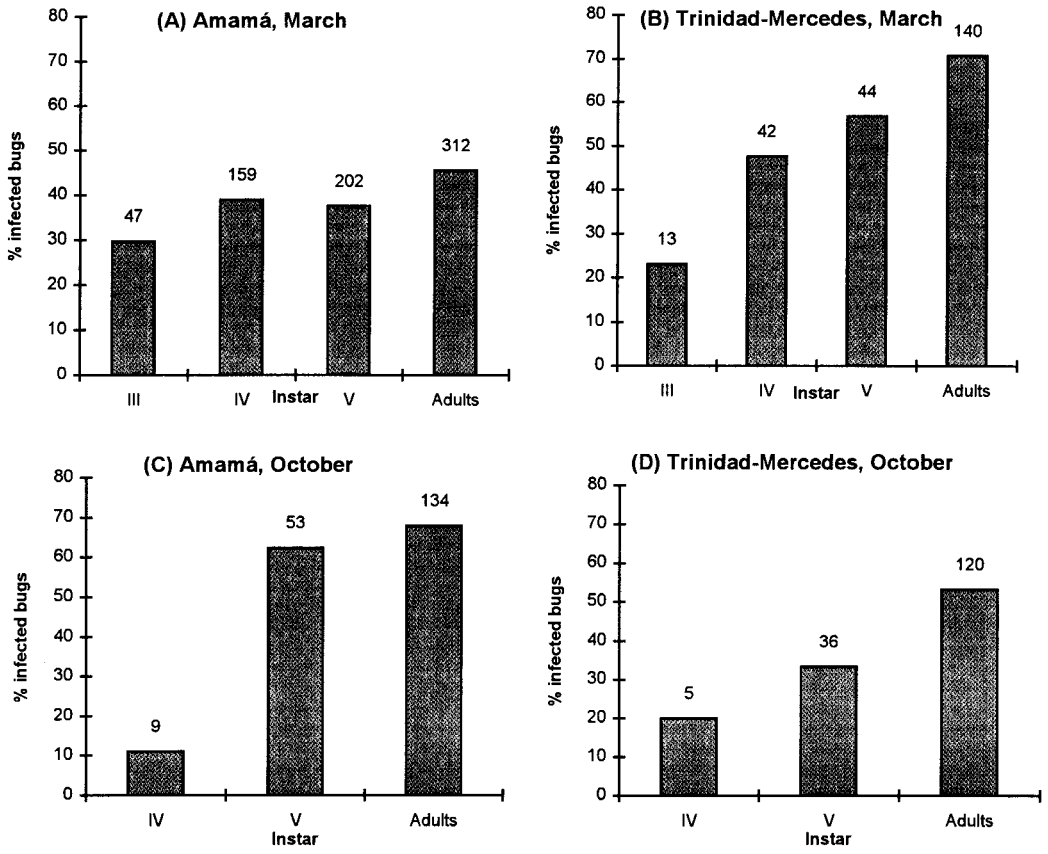

FIGURE 1. Instar-specific percentages of domiciliary *Triatoma infestans* infected with *Trypanosoma cruzi* collected by different methods. Flushing-out collections in March 1992 at **A,** Amama or **B,** Trinidad-Mercedes; knockdown collections after spraying of deltamethrin in October 1992 at **C,** Amama or **D,** Trinidad-Mercedes. The numbers on top of the bars indicate numbers of bugs examined for infection.

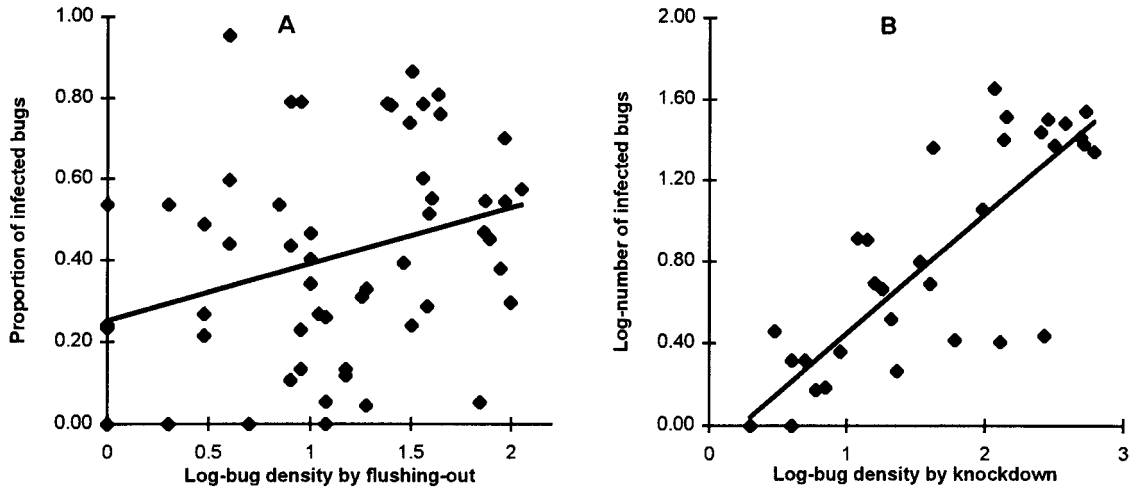

FIGURE 2. Age-standardized proportion of domiciliary *Triatoma infestans* infected with *Trypanosoma cruzi* or $\log_{10}$-transformed number of infected bugs (y) in relation to the $\log_{10}$-transformed number of triatomines collected (x) by flushing-out in all villages (**A,** $y = 0.25 + 0.14x$, $N = 57$, $r^2 = 0.1$, $P = 0.02$) or by knockdown in Amama (**B,** $y = -0.13 + 0.58x$, $N = 34$, $r^2 = 0.68$, $P < 0.001$) in Amama and Trinidad-Mercedes, March and October 1992.

age-standardized proportion of infected domiciliary *Triatoma infestans* on host infection rates and host blood indices in 22 houses with at least 10 bugs examined for infection and at least 10 bugs with identified blood meals. Most of the variation in bug infection rates can be explained by variations in the proportion of bugs that fed on dogs or cats and their prevalence rates of infection ($P < 0.001$; Figure 3). Among the significant predictors, the HBI was the least significant ($P = 0.04$) and the proportion of infected humans was not statistically significant ($P = 0.13$). The regression was statistically significant ($\chi^2 = 99.8$, df = 4, $P < 0.001$), and no interaction term was significant. The equation was LOGIT (PINFBUG)= $-3.07 + 2.73 \times$ DCBI $+ 2.10 \times$ INFDC $+ 0.83 \times$ HBI $- 0.75 \times$ INFHU. The odds ratio of being infected for a bug that fed on dogs or cats was 15.3 per unit increase in the DCBI (95% confidence interval [CI] = 7.1–32.91), 8.2 (4.3–15.6) per unit increase in the proportion of infected dogs or cats, and 2.3 (1.1–4.8) per unit increase in the HBI. When we fitted the proportion of infected triatomines to the regression model that included host blood indices and numbers, instead of proportions, of infected hosts in equation 1, all four predictors had significant and positive effects on bug infection rates. The interaction terms were not statistically significant, and neither was a dummy variable that represented the presence of children who had seroconverted for *T. cruzi* in the last three years[10] or the log-density of triatomines captured by flushing-out.

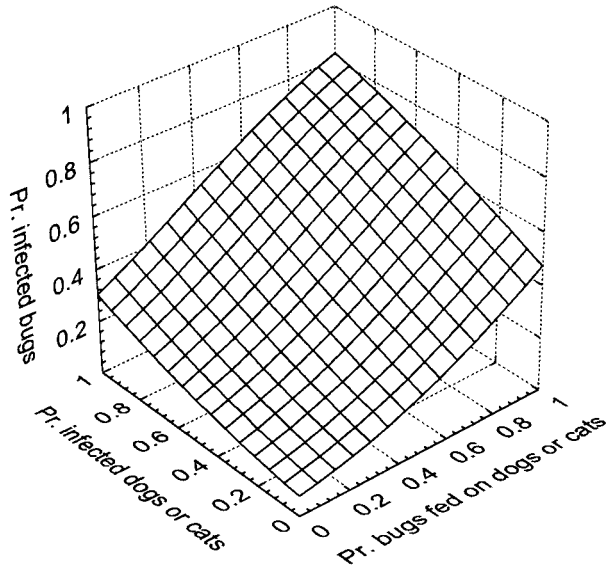

FIGURE 3. Proportion (Pr.) of domiciliary *Triatoma infestans* infected with *Trypanosoma cruzi* in relation to the prevalence of infection in dogs or cats and the proportion of bugs that fed on dogs or cats in Amama, March and October 1992.

We changed the criterion for including study houses to see how this affected the selection of significant variables. Successive analyses including houses with at least five *Triatoma infestans* examined for infection and with at least five identified blood meals, or one bug examined for infection, increased the sample size to 28 and 43 houses, respectively. All regression equations were statistically significant. In both cases, the proportion of infected dogs or cats, the DCBI and the HBI were all highly significant predictors with positive effects on bug infection rates.

Table 2 summarizes the results of regression analyses that included a term for the effects of chickens on the proportion of *T. cruzi*-infected bugs in equation 1. In houses with 10 bugs examined for infection and with identified blood meals, after backward elimination of nonsignificant variables, the infection rates of *Triatoma infestans* were significantly and negatively related to the CKBI and positively related to DCBI and INFDC (Figure 4A and B). Figure 4C shows a moderately good fit between observed bug infection rates and those predicted by the backward elimination model, although the predicted values tend to be slightly too high at low values, and slightly too low at high values. Similar results were obtained with the CKBI ONLY. Using all 43 houses in the study base, bug infection rates were significantly and negatively related both to the CKBI or the CKBI

TABLE 2

Logistic or linear multiple regression analysis and backward elimination of nonsignificant variables for the model that regressed the proportion (PINFBUG), or the log-number (NINFBUG), of domiciliary *Triatoma infestans* infected with *Trypanosoma cruzi* (the dependent variables) on the dog–cat blood index (DCBI), the proportion of infected dogs or cats (INFDC), the human blood index (HBI), the proportion of infected humans (INFHU), and the chicken blood index (CKBI) (independent variables). Data taken at Amama in March and October 1992*

Dependent variable	Independent variables	Coefficient†	SE‡	*P*§	Adjusted odds ratio	Confidence interval
PINFBUG	Intercept	−1.89	0.81	0.02		
	DCBI	2.07	0.54	<0.001	7.9	2.7–22.8
	INFDC	1.87	0.36	<0.001	6.5	3.2–13.1
	HBI	−0.01	0.62	0.98	1.0	0.3–3.3
	INFHU	−0.48	0.51	0.36	0.6	0.2–1.7
	CKBI	−0.89	0.52	0.10	0.4	0.1–1.1
		$\chi^2 = 102.7$ with 5 df, $P < 0.001$				
Addition of interaction terms: not significant						
Backward model logit (PINFBUG) = −1.84 + 1.93 × DCBI + 1.69 × INFDC − 1.04 × CKBI						
		$\chi^2 = 101.8$ with 3 df, $P < 0.001$				
NINFBUG	Intercept	−0.44	0.35	0.23		
	DCBI	1.01	0.21	<0.001		
	INFDC	1.34	0.13	<0.001		
	HBI	0.30	0.29	0.30		
	INFHU	−0.44	0.19	0.03		
	CKBI	0.72	0.23	<0.01		
	$r^2 = 0.80$	F-ratio = 30.6 with 5 and 38 df, $P < 0.0001$				
Backward model NINFBUG = −0.09 + 0.86 × DCBI + 1.30 × INFDC + 0.52 × CKBI − 0.46 × INFHUM						
	$r^2 = 0.80$	F-ratio = 37.9 with 4 and 39 df, $P < 0.001$				

* df = degrees of freedom.
† *y*-intercept and regression coefficients.
‡ Standard error of coefficients
§ t values computed as regression coefficients on standard error.

ONLY ($P < 0.001$) before and after stepwise elimination. No significant effects of indoor-brooding hens on bug infection rates were detected.

The regressions of the number of infected *Triatoma infestans* collected per person-hr per house (NINFBUG) on human and dog or cat prevalence rates of infection, and human, dog or cat, and chicken blood indices (equation 2) in the 22 houses are shown in Table 2 and Figure 5. Significant predictors of infected bug density were the proportion of infected dogs or cats, the DCBI and the CKBI; all three showed positive effects and explained 80% of the total variance. The positive effects of chickens were highly significant ($P < 0.001$) for 27 or 33 houses with at least 10 or five bugs collected by flushing-out, respectively.

DISCUSSION

This study is the first to show that 1) the prevalence of infected dogs or cats and their degree of contact with domiciliary *Triatoma infestans* explains most of the variation among the households in the proportion of triatomines infected with *T. cruzi,* and 2) the presence of chickens in bedroom areas decreased significantly the effects of dogs or cats on domiciliary bug infection rates, but at the same time significantly increased the infected bug density per house.

The proportion of domiciliary *Triatoma infestans* infected with *T. cruzi* was significantly higher in Trinidad–Mercedes than in Amama, possibly because the former villages were never sprayed with insecticides. In Amama, bug infection rates decreased from 51–63% before the first house spraying with insecticides ever made in the area in 1985 to 22% in March 1989, and increased to 41% in March 1992, while densities of infestation recovered and the prevalence rate of infection among dogs increased from 40% to 65%.[9, 10] In contrast, in unsprayed Trinidad–Mercedes, domiciliary bug infection rates varied little (from 42% in September–December 1988 to 48% in October 1992). As shown in Figure 2B, there is a direct relationship between the density of triatomines and the density of *T. cruzi*-infected bugs in the same houses, even though both samples of bugs were collected by different methods and at different times within the same year. However, because the slope of this relationship is less than 1, the plot on an arithmetic scale would be concave, suggesting a density-dependent saturation effect on infected bug density.

Few studies have sought to assess seasonal variation in the proportion of domiciliary triatomines infected with *T. cruzi* in a well-defined population. The comprehensive review by Zeledon and Rabinovich[2] mentioned no study on seasonal variation of triatomine infection rates with *T. cruzi.* Other studies (Gurtler RE and others, 1983, unpublished data)[22, 23] noted seasonal fluctuations, but found no consistent explanation for these variations. In the present study, bug infection rates stratified by instar significantly differed between March and October collections, but the sign of the differences was exactly reversed in the two communities although sampling methods were consistent over villages in March and October. We have no explanation for such differences.

In principle, seasonal variation in domiciliary bug infection rates may not be as marked as that reported for sylvatic triatomines. Domiciliary triatomines are possibly longer-lived insects that thrive in a more stable environment with a more stable and abundant supply of diverse hosts (including infected hosts) when compared to sylvatic triatomines. *Trypanosoma cruzi* infection in triatomines is mostly irreversible and causes no significant adverse effects on survival, development, and reproduction of regularly fed insects.[24]

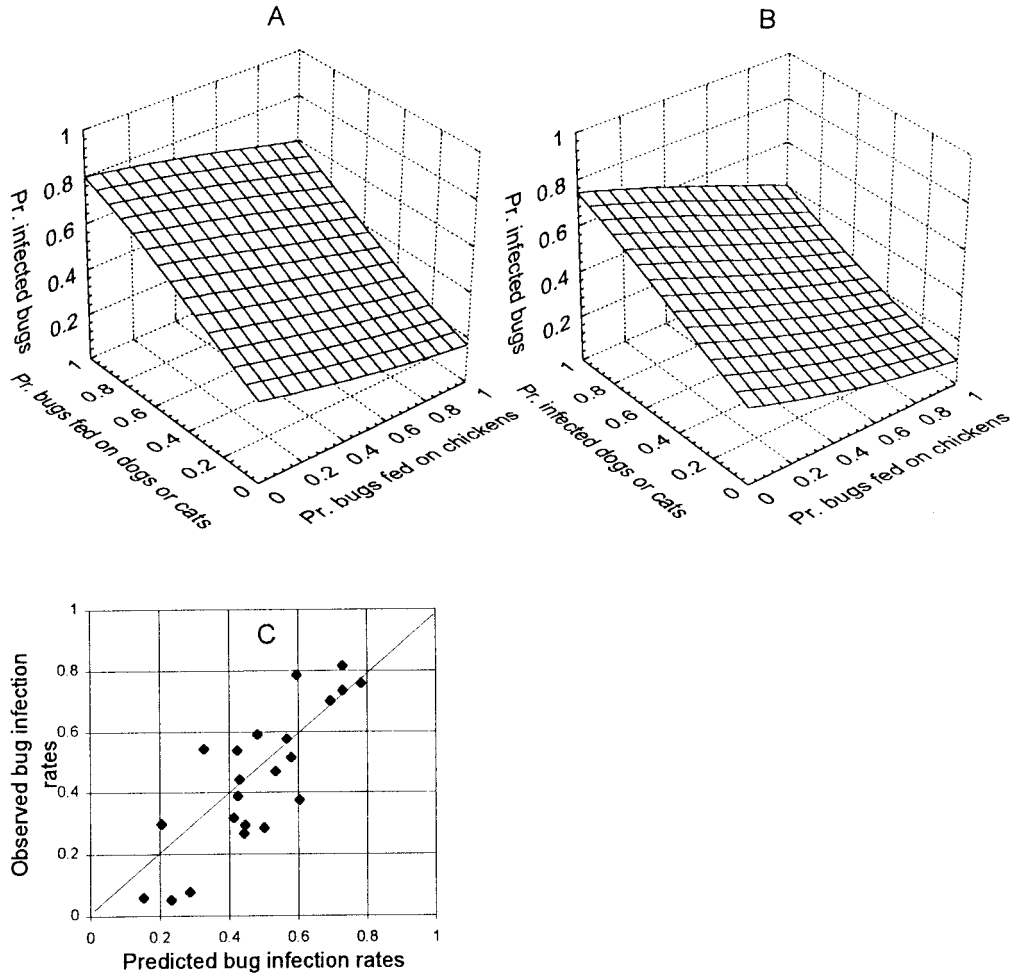

FIGURE 4. Proportion (Pr.) of domiciliary *Triatoma infestans* infected with *Trypanosoma cruzi* in relation to the proportion of bugs that fed on chickens and the proportion of bugs that fed on dogs or cats **(A)** or the dog or cat infection rate **(B)**. Predicted and observed proportions of infected bugs for the backward elimination logistic model that included dog or cat infection rates, and proportions of bugs that fed on chickens, or on dogs or cats **(C)**. Amama, March and October 1992.

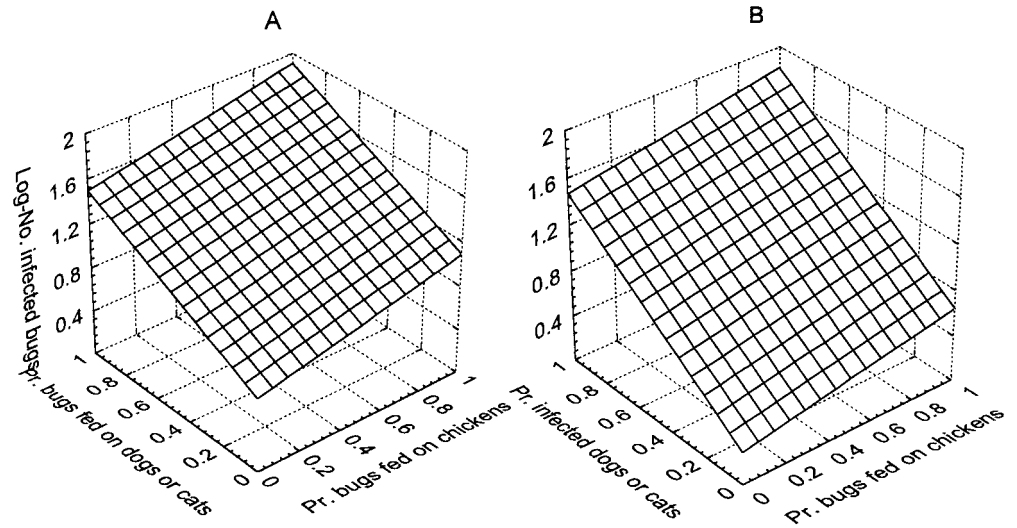

FIGURE 5. Linear multiple regression analysis of the $\log_{10}$-transformed number of domiciliary *Triatoma infestans* infected with *Trypanosoma cruzi* collected per person-hr per house in relation to the proportion of bugs that fed on chickens and the proportion of bugs that fed on dogs or cats **(A)** or the dog or cat infection rate **(B)** in Amama, March and October 1992.

These characteristics and the increasing blood meal size with instar, coupled with more feeding opportunities, may account for the usual finding of increasing vector infection rates with instar. The probability that an uninfected *Triatoma infestans* acquires a *T. cruzi* infection after a single blood meal on a dog naturally infected with *T. cruzi* did not differ significantly between seasons (unpublished data), but there is a well-known increase in the frequency of human acute cases (the most infectious) during the hot season.[25] An increase in the hatching of susceptible triatomines during spring and summer may swell the ranks of the uninfected fraction of the population and decrease the proportion of infected bugs. However, the increased vector biting rate due to higher temperatures is expected to increase the chance of bug infection. It is difficult to predict whether the opposing effects of the hatching and biting rates, in combination with a greater likelihood of acute cases in the hot season and other characteristics of local transmission, will increase or decrease bug infection rates; indeed, the balance may fluctuate randomly. In summary, the infection rates of domiciliary *Triatoma infestans* do not show a clear-cut, consistent pattern of seasonal variation.

Bugs infected with *T. cruzi* were concentrated in bedroom areas, in close association with the presence of dogs, cats, and humans. Few infected *T. infestans* were collected in peridomestic sites, and the origins of their infection may likely be traced to feedings on dogs or cats.[14] In northwest Argentina, the peridomicile is more important as a source of *Triatoma infestans* for domiciliary recolonization than as a source of *T. cruzi*-infected bugs.[21]

Using vector feeding patterns and host infection rates incorporated in a logistic multiple regression model, we have reinforced previous evidence showing that bug infection rates were closely related to the prevalence of *T. cruzi*-infected dogs or cats diagnosed by serology or xenodiagnosis.[10, 11] In the present study, for the first time the degree of contact between vectors and dogs or cats was identified as a significant predictor of triatomine infection rates.

In the presence of strong effects of dogs or cats on the proportion of domiciliary *Triatoma infestans* infected with *T. cruzi,* the number or relative abundance of infected humans had no significant effect on bug infection rates. This is to be expected since the infectivity of dogs seropositive for *T. cruzi* to third or fourth instar nymphs was 12 times higher than that of seropositive children, and 100 times higher than that of seropositive adults.[9] These results should not be interpreted to suggest that infected humans play no role in the transmission dynamics of *T. cruzi.* For example, 6–14% of domiciliary triatomines were infected with *T. cruzi* in houses with only infected adults detected.[10, 26, 27] In xenodiagnostic studies of adults seropositive for *T. cruzi,* patients showed a rather constant pattern of high, intermediate, or low infectivity to triatomines in a 30 month follow-up.[28] On the average, the potential contribution of chronically infected adults to *T. cruzi* transmission is much lower than that of dogs or children, but given its variability it may not be negligible in all circumstances.

The effect of chickens is clearly connected to the issue of zooprophylaxis, defined as the use of wild or domestic animals, which are not the reservoir hosts of a given disease, to divert the blood-seeking mosquito vectors from the human hosts of that disease.[29] Zeledon and Rabinovich[2] stated ''. . . The rates of infection by *T. cruzi*. . . decrease considerably when the insects are closely associated with refractory animals such as birds.'' However, at least one detailed study, based on a single house, was inconclusive on this point because of the confounding effect of differing age structures of bug populations between surveys.[15]

Our multiple regression analysis suggests that the presence of chickens in bedroom areas modified significantly the statistical relationship among bug infection rates with *T. cruzi,* host infection rates, and host–vector contact in infested houses. However, as shown by the slightly tilted slope of the fitted surface in Figure 4, the depressing effect on bug infection rates was limited. Domiciliary *Triatoma infestans* that fed exclusively on chickens (according to our means of detecting blood sources) had a lower infection rate (27%) than those that fed on dogs (58%) or humans (35%), whereas bugs that at least fed on chickens (regardless of other host blood sources) had only a slightly lower infection rate (41%) than those that fed on dogs (56%) or humans (49%) (Gurtler RE and others, unpublished data). The most likely explanation for the high frequency of infected chicken-fed bugs is that during its relatively long life cycle, *Triatoma infestans* shifted repeatedly from chickens to humans or dogs in the hot season, as shown by the increasing frequency of mixed blood meals with instar.[7] In addition, brooding hens usually were indoors for a limited period of time, mostly in spring and less in the hot summer months.[14] When chickens were not present, the increased bug population that fed on them likely shifted to humans or dogs. In the presence of highly infectious reservoirs, such as dogs, the effects of chickens on bug infection rates were blurred. A depressing effect of chickens on bug infection rates may possibly be more significant during spring or when the bugs are stably aggregated around a chicken nest, but when measured at the end of the bugs' reproductive season (from August to April) in the presence of infectious hosts of *Triatoma infestans,* the combined effects of chickens on bug infection rates were not detectable.

When we focused on the effects of domiciliary hosts on the density, instead of the proportion, of infected triatomines per house, a different picture emerged. After allowing for the presence of infected dogs or cats and the bugs feeding on dogs or cats, the density of infected triatomines increased significantly with more bugs feeding on chickens. The domiciliary density of *Triatoma infestans* was significantly higher in houses in which hens usually nested indoors than in houses in which they did not, and increased linearly and significantly with the percentage of domiciliary bugs that fed on chickens, while allowing for the statistical effects of the number of other resident hosts and house construction features.[14] We conclude that the presence of chickens in domiciliary areas most likely increased the basic reproduction number of *T. cruzi* in *Triatoma infestans* populations from our study area. The relationship between these factors and the human incidence and prevalence of *T. cruzi* will be treated separately.

Several assumptions and problems limited interpretation of the data. First, all our models, exemplified by equations 1 and 2, treat the explanatory variables, such as the proportion of infected dogs or cats (INFDC) and the proportion of

humans seroreactive to *T. cruzi* (INFHU), as if they were independent of the dependent variables, the proportion or density of infected *Triatoma infestans.* Because *T. cruzi* is transmitted cyclically by infected bugs from one susceptible host to another, the proportion of infected dogs or cats, for example, depends on the density of infected bugs. The models analyzed here thus focus on only one part of the transmission cycle, from hosts to bugs, and do not attempt to represent the entire feedback system.

Second, in our framework we assumed that the transmission system was at a steady state and therefore the measured variables were not varying over time. However, the time series of bug and dog infection rates from 1985 to 1992 indicated that the highest levels of infection observed before spraying had not returned seven years later, whereas the percentage of infested houses and the domiciliary density of *Triatoma infestans* were qualitatively similar to baseline conditions. In summary, the system was close to, but had not yet fully returned to, a presumed equilibrium that it may have occupied before interventions began in 1985.

Third, the timed manual capture of triatomines selects for increasingly larger (and older) instars,[30] which have a greater likelihood of being infected than younger instars. The age-standardization procedure adjusted for differing age distributions of bug populations among houses, not for biased vector captures. Therefore, our standardized bug infection rates may overestimate the true rates. On the other hand, detection of *T. cruzi* infection in triatomine feces obtained by abdominal compression is expected to miss some infections.[31]

Fourth, very few human residents, mostly adults, were not tested for *T. cruzi* infection; their impact on bias was probably negligible. Dog or cat residents that escaped diagnosis were also few, but due to their high infectivity to bugs, their effect might be important. Dogs or cats that recently died, were lost, or were borrowed from neighbors may have contributed to bug infection rates but their effects were not represented in the data base. We partially corrected for these biases through interviews and by verifying the consistency between the census of hosts and the vector feeding patterns within each house.

Fifth, our data on cats examined for infection and on cat blood meals were sparse; thus, we pooled them with those from dogs. However, the role of cats in *T. cruzi* domiciliary transmission is certainly not as marked as that of dogs because cats probably have a shorter residence period in the house than dogs and weaker links to bedroom areas when they reach adulthood. Because adult cats are nocturnally active, they may be less exposed than dogs to domiciliary triatomines, which seek hosts mostly at night.

Finally, our analyses did not consider the effects of differential infectivity to bugs of infected humans and dogs or cats, varying with host age, and differential exposure to bugs among individual dogs, cats, and humans within and among households; these factors may help explain the variability observed. For our purposes, we considered negligible and thus ignored the possible invasion by flight of adult *Triatoma infestans* (infected or not) from other foci, and the potential contribution to domiciliary bug infection of other peridomestic or sylvatic reservoirs of *T. cruzi*, such as rodents (which were scarce in peridomestic areas and reportedly almost absent in bedroom areas) and opossums; these hosts were imputed as exceptional blood sources of domiciliary *Triatoma infestans.*[6]

Domestic animals play a crucial role in the domiciliary transmission of *T. cruzi.* The existing mathematical models of the transmission of Chagas' disease do not explicitly include the existence of highly infective domestic animal reservoirs of *T. cruzi* and seasonality.[32, 33] Because *T. infestans* feed preferentially and more frequently on dogs and chickens during the hot weather (thus boosting the bug population) and dogs are highly infective reservoirs, the combined effects of chickens, dogs, and cats on the domiciliary transmission dynamics may overwhelmingly dominate the contribution to transmission due solely to humans in settings such as the villages we studied.

The methods developed in our study may be applied to other domiciliary triatomine species and other domestic animal reservoirs. For example, in some situations,[13, 34–36] rodents and opossums show long-lasting parasitemia[37, 38] and seem to play a role analogous to that of dogs in northwest Argentina. Their removal is expected to bring a decrease in bug infection rates.

Our present observations show that making dogs, cats, and chickens sleep in outhouses or enclosures would reduce the size of both the infected domiciliary bug population and the proportion infected with *T. cruzi.* Excluding these domestic animals from areas where humans live and sleep would be expected to reduce, first, the probability that bugs acquire the infection, and next, the probability that dogs, cats, and humans become infected. In the context of an integrated approach to the control of Chagas' disease in Argentina, the preventive management of domestic animals is an essential adjunct to insecticide spraying.

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APPENDIX 1

Domiciliary *Triatoma infestans* abundance, infection, and feeding patterns, and *Trypanosoma cruzi* infection of humans, dogs, or cats by house; Amama, Trinidad–Mercedes, March and October 1992

No. of house*	No. of bugs				No. of bugs fed on				No. of humans		No. of dogs or cats		Domiciliary brooding hens**
	Captured†	Examined‡	Infected§	Reactive¶	Humans	Dogs or cats	Chickens	Chickens only	Examined	Infected#	Examined	Infected#	
A–004	1	1	0	1	1	1	0	0	2	0	2	2	0
A–005	73	54	29	35	10	11	26	19	6	4	3	3††	1
A–006	72	49	23	36	28	10	9	4	5	2	0	0††	1
A–008	28	18	7	10	10	2	0	0	1	0	0	0	0
A–010	17	17	5	16	13	8	3	1	3	1	2	1	0
A–011	87	64	24	61	28	34	30	18	8	5	2	2	1
A–012	43	58	44	39	27	31	4	0	3	1‡‡	1	1	0
A–013	11	9	0	5	1	5	0	0	4	1	1	1	0
A–015	91	30	21	26	10	24	7	0	0	0	0	0	0
A–016	8	8	1	4	2	2	2	2	7	2	2	0	1
A–018	35	44	27	25	17	14	4	2	2	1	2	1	1
A–019	92	46	25	38	15	12	31	18	8	4	5	4	1
A–020	8	10	2	7	2	5	4	2	4	2	1	1	1
A–021	37	37	11	21	14	3	13	7	1	0	2	1	1
A–024	11	15	4	10	6	5	2	1	4	3	2	1	1
A–026	14	12	2	8	7	0	2	1	10	6	2	2	1
A–027	42	38	31	44	20	36	1	0	8	4	4	3	1
A–028	7	5	4	4	2	2	2	1	3	1	2	1	0
A–029	23	28	22	21	15	13	2	0	9	1	3	2	1
A–030	14	13	2	11	10	0	1	1	12	3‡‡	5	3	1
A–031	18	22	7	17	6	11	10	3	2	1‡‡	2	1	0
A–032	98	35	10	29	3	13	20	15	2	1	3	3	1
A–034	3	6	6	2	1	2	0	0	1	1	0	0	0
A–037	11	4	0	3	2	2	0	0	5	2	3	2	1
A–044	2	4	1	2	0	1	1	0	6	4	3	2	0
A–046	0	1	1	0	0	0	0	0	2	1	1	0	0
A–100	0	1	0	1	0	1	0	0	10	1	2	0	0
A–101	69	34	2	17	5	8	13	8	4	0	5	0	1
A–102	39	35	23	56	39	40	13	1	6	2	1	0††	1
A–103	9	9	4	6	2	6	2	0	5	1	5	3††	1
A–104	111	59	34	42	14	25	30	13	8	6	3	3	1
A–105	2	6	1	1	1	1	0	0	9	3	2	2	0
A–107	18	19	1	16	12	8	5	2	4	1	2	0	0
A–108	0	1	1	1	1	1	1	0	4	2	1	1	0
A–109	77	70	32	48	8	31	35	15	4	2	8	5	1
A–111	0	8	2	7	3	3	5	1	3	2	1	1	1
A–113	4	4	0	4	2	0	4	0	3	1	0	0	1
T–001	3	9	5	4	4	2	2	0	3	3	5	5	0
T–002	24	18	14	4	2	1	1	1	7	5	5	5	1
T–003	8	9	7	0	0	0	0	0	2	0	2	2	0
T–004	7	11	5	2	2	0	0	0	6	2††	3	2	0
T–008	31	33	24	8	8	6	0	0	8	5	3	1	0
M–016	35	64	50	11	4	7	1	1	8	3	3	3	1

* A = Amama; T = Trinidad; M = Mercedes.
† No. of bugs collected by flushing-out in one person-hour per house in March 1992.
‡ Bugs collected in March and October 1992 by different methods and examined for *T. cruzi* infection by microscopic examination of diluted bug feces.
§ Age-standardized number of infected bugs in March and October 1992.
¶ Reactive bugs giving a positive reaction against at least one of the antisera used.
# For humans and dogs, seropositive for at least two different serologic techniques. For cats, with a positive xenodiagnosis.
** Habit of keeping brooding fowls indoors or in gallery areas, as reported by house-dwellers and concurrent observations during vector surveys, takes the value of 1.
†† Dogs were shared between A–005 and A–006, and between A–102 and A–103; thus dog or cat infection data in houses A–006 and A–102 were corrected using the number or proportion of infected animals in the neighboring house.
‡‡ One individual with discordant serologic test result, considered as seronegative.