Trypanosoma Cruzi Affects The Sensory Biology of Triatoma Dimidiata (Hemiptera: Reduviidae).

Irving J. May-Concha (irving.may@correo.uady.mx)
Regional Centre of Research Dr Hideyo Noguchi: Centro de Investigaciones Regionales Dr Hideyo Noguchi

Maryrose J. Escalante Talavera
Regional Centre of Research Dr Hideyo Noguchi: Centro de Investigaciones Regionales Dr Hideyo Noguchi

Jean-Pierre Dujardin
Institut de recherche pour le développement: Institut de recherche pour le développement

Etienne Waleckx
Regional Centre of Research Dr Hideyo Noguchi: Centro de Investigaciones Regionales Dr Hideyo Noguchi

Research

Keywords: Triatominae, Chagas disease, Antennal phenotype, Physiology changes, Sensory organs, Phenotypic plasticity

DOI: https://doi.org/10.21203/rs.3.rs-589244/v1

License: © This work is licensed under a Creative Commons Attribution 4.0 International License. Read Full License
Abstract

Background: *Triatoma dimidiata* is a vector of the protozoan parasite *Trypanosoma cruzi*, the etiologic agent of Chagas disease. Phenotypic plasticity allows an organism to adjust its phenotype in response to stimuli or environmental conditions. Understanding the effect of *T. cruzi* on the phenotypic plasticity of its vectors, known as triatomines, has attracted great interest because of the implications of the parasite-triatomine interactions in the eco-epidemiology and transmission of the parasite. We investigated whether the infection of the vector with *T. cruzi* can change the antennal phenotype of sylvatic, domestic, and laboratory-reared populations of *T. dimidiata*.

Methods: The abundance of each type of sensillum (bristles, basiconic, thick- and thin-walled trichoid) on the antennae of *T. cruzi*-infected and non-infected *T. dimidiata* reared in the laboratory or collected in sylvatic and domestic ecotopes were measured under light microscopy and compared using Kruskal–Wallis non-parametric tests and Permutational Multivariate Analysis of Variance.

Results: We found significant differences between sensilla patterns of infected and non-infected insects within sylvatic and domestic populations. Conversely, we found no significant differences between sensilla patterns of infected and non-infected insects within the laboratory-reared population. Besides, our results show that the infection with *T. cruzi* affects the sexual dimorphism linked to antennal phenotype in sylvatic and domestic populations.

Conclusion: These differences could be linked, for infected insects, to higher efficiency in the perception of odor molecules related to the search of distant mates and hosts and for flight dispersal in search of new habitats, and the possibility of a positive effect on population dynamics and on the vectorial transmission of *T. cruzi*.

1. Background

Phenotypic plasticity is of great interest in ecology and evolution because it allows an organism to actively adjust its phenotype in response to stimuli or environmental conditions [1–8]. The response may or may not be adaptive, and it may involve changes in morphology, physiological state, behavior, or some combination of these [9]. Besides, phenotypic plasticity is also widely recognized as an important factor for the evolution, population biology, and ecological interactions of many species [10–13]; thus, it is a major mechanism of ecological adaptation [14]. Most information on phenotypic plasticity comes mainly from social insects [14–16], triatomines [17–20], grasshoppers [21, 22] and butterflies [13, 23].

In insects, the olfactory system plays an important role in many behavioral contexts, such as food, refuges and mate finding, alarm and aggregation behaviors, as well as avoidance of natural enemies [24]. In triatomines, the antennal phenotype (AP) comprises the type and number of sensilla (classified as mechanoreceptors and chemoreceptors) distributed on the antennae. Sensilla act as an interface between the external and internal environments of insects (inter and intraspecific communication), capturing different stimuli from the external environment and directing them to the central nervous
This then triggers specific behavioral responses, such as the selection of a host for feeding, oviposition behavior, mate finding, as well as alarm and aggregation behaviors [27–33].

AP has been widely used to analyze genetic diversity, as well as environmental influences on populations [33–35]. In certain species or complexes, AP analysis complements other phenotypic and genetic characteristics [34, 36–39] or provides evidence for species´ differentiation [40, 41]. On the other side, previous studies have established that the types of sensilla on an insect’s antennae may show a degree of morphological plasticity between populations that seems associated with adaptations to sensorial requirements of different habitats [17, 37]. Besides, the number of sensilla may also vary because of selection pressure, sex, infection by a microorganism, and feeding habits [37, 39, 42–46]. Such changes show the degree of phenotypic plasticity exhibited by the species [17].

As vectors of *Trypanosoma cruzi*, the causal agent of Chagas disease, the insects of the subfamily Triatominae (Hemiptera: Reduviidae), have special relevance in Latin America [47]. The parasite is transmitted to humans and other animals when feces or urine of infected insects come into contact with mucous membranes or damaged areas of mammal skin [48]. The co-evolution between triatomines and *T. cruzi* has promoted the development of powerful and sophisticated strategies, which can modify a wide range of physiological processes of the insects, including those related to the input, development, and discharge of the parasite [49]. The existence of these modifications as a characteristic of an association between *T. cruzi* and triatomines could be the consequence of different adaptive or nonadaptive scenarios (e.g., adaptive host manipulation) [50–51]. While several works have analyzed the mechanisms associated with *T. cruzi*-vector dynamics (e.g., biotic and abiotic factors) to understand the *T. cruzi*-triatomine interactions, under a co-evolutionary scenario [52], literature about how the parasites may influence the insects is more limited, and the studies have only been focused on the parasite’s effects on four patterns of the vector's behavior: life-history traits, feeding, defecation, and dispersion/locomotion [53]. Different studies have found negative effects of *T. cruzi* infection on vector’s survival [54–57], fecundity [57, 58], post-embryonic development [57, 59, 60], behavior [53, 61–65], and physiological processes [54, 58, 66–68], while other studies have not identified these effects on patterns of alimentation/defecation [54, 69], development and reproduction [70–72]. Overall, most of these studies determined that the effects of *T. cruzi* are species-dependent, age-dependent, sex-dependent, and even environment/physiology-dependent.

Although the AP, infection with *T. cruzi*, and phenotypic plasticity of the triatomines have been extensively studied [17, 34, 52], the phenotypic plasticity linked to the infection with *T. cruzi* in triatomines has not been investigated so far. In this study, we performed a series of analyses to test whether the infection with *T. cruzi* modifies the AP of *T. dimidiata* from Yucatan, Mexico. More specifically, we investigated whether the infection with *T. cruzi* modifies the AP and sexual dimorphism of sylvatic, domestic and laboratory-reared *T. dimidiata* populations.

### 2. Methods
2.1. Insects

Laboratory-reared *T. dimidiata* came from a colony maintained for the past 10 years at the Parasitology Laboratory of the Regional Research Center Dr. Hideyo Noguchi, Autonomous University of Yucatan. New insects have been periodically added to this colony to avoid inbreeding depression. The colony is reared and maintained at 27 ± 1°C, 70 ± 5% RH, a photoperiod of 12: 12 (L: D) h, and insects are fed on immobilized pigeons (*Columba livia*). The domestic and sylvatic populations were composed of insects collected during entomological surveillance inside and outside human dwellings of the rural village of Teya (21.05º N, 89.07º W), Yucatan, Mexico, and in the sylvatic habitat surrounding this village, respectively [73–74]. The study was approved by the Institutional Bioethics Committee of the Autonomous University of Yucatan.

2.2. Trypanosoma cruzi

For infection of triatomines, the “V strain”, a Tcl strain of *T. cruzi* maintained in the laboratory by cyclical passages in BALB/c adult mice was used.

2.3. Infection of the laboratory-reared triatomines

After a two-week starvation period, the initial infection of the laboratory-reared triatomines was carried out with nymphs that had just molted to their 5th instar. Nymphs were fed ad libitum on BALB/c mice 15 days after these were infected with $1 \times 10^6$ parasites ml$^{-1}$ of blood (i.e., during the parasite's exponential stage of growth; [63]). Approximately 30 days after infection, we corroborated infection status through examination of a fecal drop observed under a light microscope at 40× magnification. Control group insects were fed under the same conditions on non-infected mice. The nymphs of both groups were maintained under rearing conditions and were fed fortnightly on infected/non-infected mice until they molt to the adult stage. For *T. dimidiata* collected in natural conditions (i.e. domestic and sylvatic populations), *T. cruzi* infection status was assessed by amplifying parasite DNA from each bug midgut by PCR using TCZ primers, as previously described [75].

2.4. Antennal preparation

We examined a total of 130 antennae of *T. cruzi*-infected and non-infected females and males from the sylvatic, domestic and laboratory-reared populations of *T. dimidiata* (Table 1). One antenna per specimen was removed using fine forceps and scissors. Antennae were processed with sodium hydroxide 4% for 6 hr at 60°C and then neutralized with glacial acetic acid 5% for 2 min. This procedure allowed cuticle diaphanization and allowed the identification and counting of the sensilla using a stereo microscope Zeiss Primostar® at 400×. The number and type of sensilla on antennal segments was counted manually using a procedure reported previously [33]. The ventral side of the three distal segments of the antennae (P: pedicel, F1: flagellum 1, and F2: flagellum 2) was evaluated, by identifying and counting the following sensilla: bristles (BR), thin-walled trichoid (TH), thick-walled trichoid (TK), and basiconic (BA) (nomenclature according to Catalá and Schofierl [36]), thus giving a total of 12 morphological variables.
Table 1  
Number of *T. dimidiata* specimens used in this study. Population, sex and infection status of the specimens are indicated.

| Populations     | Infected | Non-infected | Overall |
|-----------------|----------|--------------|---------|
| Laborator-y-reared | 10       | 10           | 20      |
| Domestic        | 10       | 10           | 20      |
| Sylvatic        | 10       | 13           | 23      |
| Overall         | 30       | 33           | 63      |

### 2.5. Data analysis

Differences on the AP between *T. cruzi*-infected (I) and non-infected (NI) insects were explored overall populations, within each sex, within each population, and within each sex within each population using univariate and multivariate analyzes. Means and standard deviations of abundance were calculated for each type of sensilla (chemoreceptors: BR, TH, TK, and mechanoreceptors: BA) and antennal segment (pedicel, flagellum 1 and flagellum 2). As original data and their transformations were not normally distributed using Shapiro-Wilk tests [76]. Kruskal – Wallis non-parametric tests were thus used for univariate analyses. Data were analyzed with the MINTAB Statistical Software, version 17 (Minitab Inc., PA, U.S.A.). In all cases, *P* < 0.05 was considered statistically significant. Moreover, the sources of variation of the AP were assessed using two-way Permutational Multivariate Analysis of Variance (PERMANOVA) on Bray-Curtis similarity matrices of square-root with 9999 permutations. These analyses were conducted in PAST version 3.05.

### 3. Results

#### 3.1 Overall data

Abundances of the sensilla found for all the *T. dimidiata* specimens included in this study are shown in Table A1, Supplementary data. All the insect’s antennae presented three types of chemoreceptors (TH, TK, and BA) and one mechanoreceptor (BR) on the three segments. The average number of sensilla per insect was of 669.52 ± 176.45. Overall, the TH sensillum of the pedicel (P-TH) was the more abundant (183.42 ± 92.70) while the BR sensillum of the flagellum 2 (F2-BR) was the less abundant (17.45 ± 12.51). The pedicel was the segment with the highest number of sensilla (322.42 ± 115.54) while the flagellum 2 was the segment with the lowest number of sensilla (149.63 ± 54.43).

#### 3.2. Effect of the infection with *T. cruzi* on the AP of *T. dimidiata*
Differences of each sensillum on the three antennal segments between infected and non-infected insects’ overall populations, within each sex, within each population, and within each sex within each population, are summarized in Table 2.
Table 2
Comparisons of the abundances of each sensillum between infected and non-infected insects overall populations, within each sex, within each population, and within each sex within each population of *Triatoma dimidiata*. BR: bristles; BA: basiconic; TH: thin-walled trichoid; TK: thick-walled trichoid. F: female and M: male. I: infected; NI: non-infected. D: domestic, S: Sylvatic, and L: Laboratory reared. Asterisks represent a significant difference between infected and non-infected insects (P < 0.05*; P < 0.01**; P < 0.001***; – no difference).

| Factor                                           | Pedicel | Flagellum 1 | Flagellum 2 |
|--------------------------------------------------|---------|-------------|-------------|
| Overall populations (I vs NI)                    | -- **   | --          | --          |
| Whitin females (I females vs NI females)          | -- --   | *           | --          |
| Whitin males (I males vs NI males)                | -- --   | --          | **          |
| Whitin domestic insects (I D vs NI D)             | * --    | ***         | * --        |
| Whitin sylvatic insects (I S vs NI S)             | -- ***  | --          | *** ** --   |
| Whitin laboratory-reared insects (I L vs NI L)    | -- --   | --          | --          |
| Whitin females of the domestic population (F D I vs F D NI) | -- --   | *           | --          |
| Whitin females of the sylvatic population (F S I vs F S NI) | -- ***  | --          | **          |
| Whitin females of the laboratory-reared population (F L I vs F L NI) | -- --   | --          | --          |
| Whitin males of the domestic population (M D I vs M D NI) | * --    | ***         | --          |
When infected and non-infected insects were compared, significant increases in the numbers of BA sensilla on pedicel (P-BA) and TK sensilla on flagellum 1 (F1-TK) were observed in infected insects (Kruskal–Wallis test, $P = 0.007$ and $P = 0.01$, respectively).

Conversely, a significant increase in the number of TK sensilla on flagellum 1 (F1-TK) was observed in infected males compared to non-infected males (Kruskal–Wallis test, $P = 0.008$).
3.2.3. Within each population. In the domestic population, when infected and non-infected insects were compared, a significant increase in the number of BR sensilla on pedicel (P-BR) was observed in infected insects (Kruskal–Wallis test, $P = 0.01$). On the other side, significant decreases in the number of TH and TK sensilla on pedicel; BR, BA, TK sensilla on flagellum 1, and BR, BA, TH sensilla on flagellum 2 were observed in infected insects compared to non-infected insects (Kruskal–Wallis test, $P < 0.05$ in all cases). Additionally, the two-way PERMANOVA test revealed that the infection with *T. cruzi* affected the AP of the domestic population ($F = 7.15; P = 0.0001$), while the sex and the interaction infection*sex did not have significant effects ($F = 1.51; P = 0.177$ and $F = 1.188; P = 0.299$, respectively; Table 3A).

In the sylvatic population, when infected and non-infected insects were compared, significant increases in the number of BA sensilla on pedicel; BR, BA, and TK sensilla on flagellum 1; BR, BA, TH and TK sensilla on flagellum 2 were observed in infected insects (Kruskal–Wallis test, $P < 0.05$ in all cases). The two-way PERMANOVA test revealed that the infection with *T. cruzi* and the sex affected the AP of the sylvatic population ($F = 7.41; P = 0.0001$ and $F = 4.28; P = 0.002$, respectively), while the interaction infection*sex did not have significant effect ($F = 0.368; P = 0.125$; Table 3B).

Finally, in the laboratory-reared population, when infected and non-infected insects were compared, no difference in the number of sensilla were observed (Kruskal–Wallis test, $P > 0.05$ in all cases). In the same way, the two-way PERMANOVA test did not reveal significant effects of the infection with *T. cruzi*, of the sex and of the interaction infection*sex on the AP of laboratory-reared insects ($P > 0.05$; Table 3C).

3.2.4. Within each sex within each population. Differences in the abundances of each sensillum on the three antennal segments between infected and non-infected insects within each sex within each population are shown in Table A1 (Supplementary data) and are summarized in Table 2.

**Domestic population.** The antennae of infected females of the domestic population showed a significant decrease in the number of TH sensilla on pedicel and flagellum 2, and in the BA sensilla on flagellum 2, compared to non-infected females (Kruskal–Wallis, $P < 0.05$ in all cases). On the other side, infected males of the domestic population showed an increase in the number of BR sensilla on pedicel (P-BR), compared to non-infected males (Kruskal–Wallis test, $P = 0.01$). Moreover, infected males of the domestic population showed a decrease in the BR sensilla on flagellum 1 and flagellum 2, and in the BA sensilla on flagellum 2, compared to non-infected males (Kruskal–Wallis, $P < 0.05$ in all cases).

**Sylvatic population.** The antennae of infected females of the sylvatic population showed a significant increase in the number of BA sensilla on the three segments of the antennae, in the BR sensilla on flagellum 1 and flagellum 2, and in the TK sensilla on flagellum 2, compared to non-infected females (Kruskal–Wallis test, $P < 0.05$ in all cases). On the other side, infected males of the sylvatic population showed an increase in the BA sensilla on pedicel, in the BR and TK sensilla on flagellum 1, and in the BR and TH sensilla on flagellum 2, compared to non-infected males (Kruskal–Wallis test, $P < 0.05$ in all cases).
Laboratory-reared population. In the laboratory-reared population, there were no differences in the number of sensilla between infected and non-infected females and males (Kruskal–Wallis test, $P > 0.05$).

3.3. Effect of the infection with *T. cruzi* on the sexual dimorphism of *T. dimidiata*.

Differences in the abundances of each sensillum between non-infected females and males, and between infected females and males overall populations, and within each population, are summarized in Table 4.
Table 4
Comparisons of the abundances of each sensillum between infected females and males and between non-infected females and males overall populations, and within each population of *Triatoma dimidiata*. BR: bristles; BA: basiconic; TH: thin-walled trichoid; TK: thick-walled trichoid. F: female and M: male. I: infected; NI: non-infected. D: domestic, S: Sylvatic, and L: Laboratory reared. Asterisks represent a significant difference between infected and non-infected insects (P < 0.05*; P < 0.01**; P < 0.001***; -- no difference).

| Factor                                                                 | Pedicel | Flagellum 1 | Flagellum 2 |
|------------------------------------------------------------------------|---------|-------------|-------------|
|                                                                       | BR      | BA          | TH          | TK          | BR      | BA          | TH          | TK          | BR      | BA          | TH          | TK          |
| Overall non-infected insects (NI females vs NI males)                   | -       | -           | -           | -           | -       | -           | -           | -           | -       | -           | -           | -           |
| Overall infected insects (I females vs I males)                        | -       | -           | **          | -           | -       | -           | -           | -           | -       | -           | -           | -           |
| Whitin non-infected domestic insects (F D NI vs M D NI)                | -       | -           | -           | -           | -       | -           | -           | -           | -       | -           | -           | -           |
| Whitin infected domestic insects (F D I vs M D I)                      | -       | -           | -           | -           | -       | -           | -           | -           | -       | -           | -           | -           |
| Whitin non-infected sylvatic insects (F S NI vs M S NI)               | -       | -           | *           | -           | -       | -           | -           | -           | -       | -           | -           | -           |
| Whitin infected sylvatic insects (F S I vs M S I)                     | -       | **          | *           | -           | -       | -           | -           | -           | -       | -           | -           | -           |
| Whitin non-infected laboratory-reared insects (F L NI vs M L NI)       | -       | -           | -           | -           | -       | -           | -           | -           | -       | -           | -           | -           |
| Whitin infected laboratory-reared insects (F L I vs M L I)             | -       | -           | -           | -           | -       | -           | -           | -           | -       | -           | -           | -           |

1. **3.3.1. Overall populations.** When non-infected females and males were compared, no significant difference in the number of sensilla was observed (Kruskal–Wallis test, \(P > 0.05\)). However, when infected females and males were compared, a significant difference in the number of TH sensilla on pedicel (P-TH) was observed (Kruskal–Wallis test, \(P = 0.002\)).
2. **3.3.2. Domestic population.** When non-infected females and males were compared, no significant difference in the number of sensilla was observed (Kruskal–Wallis test, \( P > 0.05 \)). However, when infected females and males were compared, a significant difference in the number of TH sensilla on flagellum 1 (F1-TH) was observed (Kruskal–Wallis test, \( P = 0.01 \)).

3. **3.3.3. Sylvatic population.** When non-infected females and males were compared, significant differences in the number of TH sensilla on pedicel (P-TH) and flagellum 1 (F1-TH) were observed (Kruskal–Wallis test, \( P = 0.02 \) and \( P = 0.003 \), respectively). When infected females and males were compared, the significant difference in the numbers of TH sensilla on pedicel (P-TH) was still observed (Kruskal–Wallis test, \( P = 0.04 \)), while the difference in the number of TH sensilla on flagellum 1 (F1-TH) was not observed anymore. However, significant differences in the numbers of BA sensilla on pedicel (P-BA) and flagellum 1 (F1-BA) were observed (Kruskal–Wallis test, \( P = 0.003 \) and \( P = 0.04 \), respectively).

3.3.4. **Laboratory-reared population.** In the laboratory-reared population, there was no sexual dimorphism in infected and non-infected insects (Kruskal–Wallis test, \( P > 0.05 \)).

4. **Discussion**

Phenotypic plasticity has been analyzed in different triatomine species in response to ecological factors [17, 35, 78], or to assess the effect of ecotope [18], food source [19], environment [35, 43, 79, 80], and sex [78]. The present study is the first to analyze phenotypic plasticity related to the infection with *T. cruzi* in domestic, sylvatic, and laboratory-reared populations of *T. dimidiata*.

Our results demonstrate that the infection with *T. cruzi* modifies the AP of *T. dimidiata*. We observed that infected and non-infected insects from the domestic and sylvatic populations showed significant differences in the number of some sensilla types. Besides, our results show that the sexual dimorphism tended to increase in *T. cruzi*-infected populations. Importantly, these differences and the effect of *T. cruzi* infection in the sexual dimorphism was not observed in the laboratory-reared population. Because in this study, the laboratory-reared insects were infected during their 5th development stage, this suggests that we should have established the infection in the earliest development stages to see the effect of *T. cruzi* on the AP. Indeed, insects infected in early development stages are more likely to be manipulated as suggested by Poulin et al. [81]. However, this would need to be further investigated in the current case.

Infected insects of the domestic and sylvatic populations showed, in general, a significant increase in the numbers of BR sensilla compared to non-infected insects. These mechanoreceptors allow insects to perceive contact stimuli, vibratory signals (through stridulation) during mating, and variations in the air current [82–84]. Besides, these play an important role in orientation towards odor-laden currents [85]. Various studies have determined that the infection by *T. cruzi*, can impair the fecundity, fertility, and mating performance of triatomines (e.g., Fellet et al. [58]). Hence, the increase in these mechanoreceptors in infected insects may help limiting this phenomenon, which is disadvantageous for the parasite. However, this deserves to be further investigated.
Concerning the chemoreceptors (BA, TH and TK), it has previously been reported that BA sensilla have an olfactory and/or gustative function for detection of habitats, shelters, hosts, and couple [86, 87]. Besides, these sensilla seem involved in the detection of presumed pheromones in conspecific feces [88, 89]. TH sensilla have an olfactory function for the detection of new habitats, sexual pairs, and hosts [34, 36, 90, 91]. On the other side, although TK sensilla have been shown to predominate in triatomines [34], their chemosensory function has not been confirmed [86, 92]. However, it may be related to the detection of a pheromone by contact, thus acting as olfactory sensilla [93], as has been shown in the insect *Cimex lectularius* L. [94]. In our study, variations in the olfactory sensitivity because of *T. cruzi* infection in the domestic and sylvatic populations is suggested. Indeed, in these populations, infected and non-infected insects showed significant differences in the number of some specific chemoreceptors. Surprisingly, infected insects of the domestic populations showed a decrease in some chemoreceptors. Conversely, in the sylvatic population, the infection with *T. cruzi* increased in all cases the number of chemoreceptors. If these differences between populations are not easy to explain, in general, the infection with *T. cruzi* significantly increased the number of chemoreceptors, which may be linked to an improved capacity for dispersal and invasion of different habitats [79, 90], and better efficiency in the perception of odor molecules in the search of distant mates and hosts and for flight dispersal in search of new habitats, as it has been suggested by other authors [80, 95–98], thus conferring an advantage to *T. cruzi*.

Although several studies show the effect of *T. cruzi* on behavioral changes [53, 61–65], so far, no study has analyzed whether there are differences in the olfactory physiology between infected and non-infected and a possible correlation of behavioral changes with their AP. In this context, it is possible to ask if the behavioral changes may be due to the AP modification in infected insects. Although the present work did not aim to answer this question, considering the recently reviewed information and the results of this study, we could suggest a possible correlation of behavioral changes with the AP as it has been suggested by May-Concha et al. [34]. Therefore, it could be interesting to evaluate the olfactory system of infected insects and non-infected towards chemical cues to find components of attractive blends that could contribute to the list of volatile compounds that modulate the behavior between infected and non-infected insects, to design better strategies for behavioral manipulation of this triatomines.

Several studies have provided information about the sexual dimorphism in non-infected triatomines from different species, populations, rearing, and ecotopes [38, 78, 99]. However, our study reports for the first time the sexual dimorphism in the AP of infected insects of *T. dimidiata*. In general, the sexual dimorphism observed in infected insects of *T. dimidiata* was based on an increase in the number of TH sensilla in infected males and/or an increase of BA sensilla in infected females. These chemoreceptors have an olfactory function for the detection of sexual pairs, habitats, hosts as mentioned above. Evidence of this study and previous works [79, 100, 101] suggest that the sexual dimorphism in the AP may be linked to the perception of molecules related to sexual behavior and to differences in sensing sexual pheromones, as it has been suggested by other authors (e.g., May-Concha et al. [34]; Souza et al. [80]). May-Concha [102] provides information on a chemical signal produced during *T. dimidiata* mating since fewer mating attempts were observed when the opening of female glands was occluded. Besides, that study describes a chemical signal which promotes the attraction of males to volatiles emitted by females
and to mating couples [30]. On the other side, based on previous works on olfactory receptors [24–26], we propose that the increased number of TH chemo-sensilla in infected males could suggest a greater efficiency in the perception of odor molecules involved in sexual communication compared with infected females. In contrast, the increased number of BA chemo-sensilla in infected females could suggest a greater efficiency in the perception of host odors compared with infected males. Therefore, the increase in the odor perception in infected insects probably elicits a positive effect on population dynamics and could affect the vectorial transmission of T. cruzi. However, to achieve depth in the knowledge on this subject, studies on the effects of the infection with T. cruzi on the behavior of aggregation, alarm, sexual pair, feeding, excretion/defecation, and host foraging in T. dimidiata should be carried out.

5. Conclusion

This is the first report of the effect of the infection with T. cruzi on the antennal phenotype of T. dimidiata. Our study shows that the infection with this parasite modifies the antennal phenotype of this vector and reveals a significant difference between infected and non-infected insects within natural populations of T. dimidiata. Overall, the increased number of some sensilla in infected insects suggests a greater contact/vibratory stimuli perception and olfactory perception compared to non-infected insects. Besides, the increase in these perceptions in infected insects probably elicits a positive effect on population dynamics which could favorize the vectorial transmission of T. cruzi.

Abbreviations

AP: antennal phenotype; P: pedicel; F1: flagellum 1; F2: flagellum 2; BR: bristles; TH: thin-walled trichoid; TK: thick-walled trichoid; BA: basiconic; I: T. cruzi-infected; NI: T. cruzi non-infected.

Declarations

Acknowledgements

We are grateful for the technical assistance of Bachelor of Biology Salma Uc Diaz and Victor Garrido Gonzalez, and the statistical advice of Master of Science Joel Moo-Millan.

Authors’ contributions

IJMC and EW contributed to the design of the Project. IJMC and MJET contributed to sample collection and laboratory analysis. IJMC, EW, MJET and JPD analyzed the data. IJMC, EW and JPD wrote the manuscript. All authors read and approved the final manuscript.

Funding
This research was supported by Consejo Nacional de Ciencia y Tecnología de México (CONACyT) IJMC/CVU: 272733. This work received financial support from CONACYT (National Council of Science and Technology, Mexico) Basic Science (Project ID: CB2015-258752) and National Problems (Project ID: PN2015-893) Programs attributed to EW.

**Availability of data and materials**

The datasets used and/or analyzed during the present study available from the corresponding author upon reasonable request.

**Ethics approval and consent to participate**

Not applicable.

**Consent for publication**

Not applicable.

**Competing interests**

The authors declare that they have no competing interests.

**References**

1. Via, S., Gomulkiewicz, R., De Jong, G., Scheiner, S.M., Schlichting, C.D., Van Tienderen, T.H., 1995. Adaptive phenotypic plasticity: consensus and controversy. Trends Ecol. Evol. 10, 212–217.
2. West-Eberhard, M.J., 2003. Developmental Plasticity and Evolution. Oxford University Press.
3. Pigliucci, M., 2005. Evolution of phenotypic plasticity: where are we going now? Trends Ecol. Evol. 20, 481–486.
4. Laland, K., Uller, T., Feldman, M., Sterelny, K., Müller, G.B., Moczek, A., Jablonka, E., Odling-Smee, J., Wray, G.A., Hoekstra, H.E., Futuyma, D.J., Lenski, R.E., Mackay, T.F., Schluter, D., Strassmann, J.E., 2014. Does evolutionary theory need a rethink? Nature 514, 161–164.
5. Lande, R., 2015. Evolution of phenotypic plasticity in colonizing species. Mol. Ecol. 24, 2038–2045.
6. Beaman, J.E., White, C.R., Seebacher, F., 2016. Evolution of plasticity: Mechanistic link between development and reversible acclimation. Trends Ecol. Evol. 31, 237–249.
7. Gadenne, C., Barrozo, R.B., Anton, S., 2016. Plasticity in insect olfaction: To Smell or Not to Smell? Annu. Rev. Entomol. 61, 317–333.
8. Colautti, R.I., Alexander, J.M., Dlugosch, K.M., Keller, S.R., and Sultan, S.E., 2017. Invasions and extinctions through the looking glass of evolutionary ecology. Philos. Trans. R. Soc. B Biol. Sci. 372, 20160031.
9. West-Eberhard, M.J., 2008. Phenotypic plasticity. In Encyclopedia of Ecology, ed. E. Jørgensen, B. Fath, pp. 2701–7. Amsterdam: Elsevier Sci.

10. Baldwin, J.M., 1896. A New Factor in Evolution. Am. Nat. 30: 441–451, 536–553.

11. West-Eberhard, M., 1989. Phenotypic plasticity and the origins of diversity. Annu. Rev. Ecol. Syst. 20, 249–278.

12. Weiss, L.C., 2019. Sensory Ecology of Predator-Induced Phenotypic Plasticity. Front. Behav. Neurosci. 12, 330.

13. Bhardwaj, S., Jolander, L.S., Wenk, M.R., Oliver, J.C., Nijhout, H.F., Monteiro, A. 2020. Origin of the mechanism of phenotypic plasticity in satyrid butterfly eyespots. eLife, 9, e49544.

14. Manfredini, F., Arbetman, M., Toth, A.L., 2019. A Potential Role for Phenotypic Plasticity in Invasions and Declines of Social Insects. Front. Ecol. Evol. 7, 375.

15. Stern, D.L., Foster, W.A., 1996. The evolution of soldiers in aphids. Biol. Rev. 71, 27–76.

16. Moczek, A.P., 2010. Phenotypic plasticity and diversity in insects. Philos. Trans. R. Soc. B Biol. Sci. 365, 593–603.

17. Dujardin, J.P., Panzera, P., Schofield, C.J., 1999. Triatominae as a model of morphological plasticity under ecological pressure. Inst. Oswaldo Cruz 94, 223–228.

18. Batista, V.S.P., Fernandes, F.A., Cordeiro-Estrela, P., Sarquis, O., Lima, M.M., 2012. Ecotope effect in Triatoma brasiliensis (Hemiptera: Reduviidae) suggests phenotypic plasticity rather than adaptation. Vet. Entomol. 27, 247–254.

19. Nattero, J., Malerba, R., Rodríguez, C., Crocco, L., 2013. Phenotypic plasticity in response to food source in Triatoma infestans (Klug, 1834) (Hemiptera, Reduviidae: Triatominae). Gen. Evol. 19, 39-44.

20. Nattero, J., Dujardin, J-P., Fernández, M.P., Gürtler, R.E., 2015. Host-feeding sources and habitats jointly affect wing developmental stability depending on sex in the major Chagas disease vector Triatoma infestans. Inf. Gen. Evol. 36, 539–546.

21. Bernays, E.A., Chapman, R.F., 1998. Phenotypic plasticity in numbers of antennal chemoreceptors in a grasshopper: effects of food. J. Comp. Physiol. A. 183, 69–76.

22. Hochkirch, A., Deperman, J., Gröning, J., 2008. Phenotypic plasticity in insects: the effects of substrate colour on the colouration of two ground-hopper species. Evol. Dev. 10, 350–359.

23. Jorge, L.R., Cordeiro-Estrela, P., Klaczko, L.B., Moreira, G.R.P., Freitas, A.V.L., 2011. Host-plant dependent wing phenotypic variation in the neotropical butterfly Heliconius erato. J. Linn. Soc. 102, 765–774.

24. May-Concha, I.J., Guerenstein, P.G., Malo, E.A., Catalá, S., Rojas, J.C., 2018a. Electroantennogram responses of the Triatoma dimidiata complex to volatiles produced by its exocrine glands. Acta Trop. 185, 336–343.

25. Guidobaldi, F., May-Concha, I.J., Guerenstein, P.G., 2014. Morphology and physiology of the olfactory system of blood-feeding insects. J. Insect Physiol. 106, 96–111.
26. Pontes, G., Minoli, S., Ortega Insaurralde, I., de Brito Sanchez, M.G., Barrozo, R.B., 2014. Bitter stimuli modulate the feeding decision of a blood-sucking insect via two sensory inputs. J. Exp. Biol. 217, 3708–3717.

27. Guerenstein, P.G., Guerin, P.M., 2001. Olfactory and behavioural responses of the bloodsucking bug *Triatoma infestans* to odours of vertebrate hosts. J. Exp. Biol. 204, 585–597.

28. Guidobaldi, F., Guerenstein, P.G., 2015. Oviposition in the blood-sucking insect *Rhodnius prolixus* is modulated by host odors. Parasit. Vectors 8, 265.

29. Guidobaldi, F., Guerenstein, P.G., 2016. A CO2-free synthetic host odor mixture that attracts and captures triatomines: effect of emitted odor ratios. J. Med. Entomol. 53, 770-775.

30. May-Concha, I.J., Rojas, J.C., Cruz-López, L., Millar, J.G., Ramsey, J.M., 2013. Volatile compounds emitted by *Triatoma dimidiata*, a vector of Chagas disease: chemical identification and behavioural analysis. Med. Vet. Entomol. 27, 165–74.

31. May-Concha, I.J., Rojas, J.C., Cruz-López, L., Ibarra-Cerdeña, C.N., Ramsey, J.M., 2015. Volatile compound diversity and conserved alarm behaviour in *Triatoma dimidiata*. Parasit. Vectors 8, 84–98.

32. May-Concha, I.J., Loobia, P.A., Mougbabure-Cueto, G., 2018b. Interaction between two aggregation chemical signals in *Triatoma infestans* (Hemiptera: reduviidae). J. Insect Physiol. 109, 79–84.

33. May-Concha, I.J., Cruz-López, L.C., Rojas, J.C., Ramsey, J.W., 2018. “Sweeter than a rose”, at least to *Triatoma phyllosoma* complex males (Triatominae: Reduviidae). Parasit. Vectors 11, 95.

34. May-Concha, I.J., Guerenstein, P.G., Ramsey, J.M., Rojas, J.C., Catalá, S., 2016. Antennal phenotype of Mexican haplogroups of the *Triatoma dimidiata* complex, vector of Chagas disease. Genet. Evol. 40, 73–9.

35. Müller, J.N., Gonçalves, T.C.M., Ricardo-Silva, A.H., Souza, C.A., Santos, F.M., Santos, R., Coelho-Vargas, N., Macedo-Lopes, C., Carbajal-de la Fuente., A.L., 2019. Does antennal sensilla pattern of different populations of *Triatoma maculata* (Hemiptera: Reduviidae) reveal phenotypic variability?. Parasit. Vectors 12, 602.

36. Catalá, S., Schofield, C.J., 1994. The antennal sensilla of Rhodnius. J. Morphol. 219, 193–203.

37. Catalá, S., 1997. Antennal sensilla of Triatominae. A comparative study of five genera. Int. J. Insect Morphol. Embryol. 26, 67–73.

38. Carbajal de la Fuente, A.L., Noireau, F., Catalá, S.S., 2008. Inferences about antennal phenotype: the “*Triatoma maculate* complex” (Hemiptera: Triatominae) is valid? Acta Trop. 106, 16–21.

39. Hernández, M.L., Abrahan, L., Moreno, M., Gorla, D., Catalá, S., 2008. Phenotypic variability associated to genomic changes in the main vector of Chagas disease in the southern cone of South America. Acta Trop. 106, 60–67.

40. Catalá, S., Torres, M., 2001. Similitude of the patterns of sensilla on the antennae of *Triatoma melanosoma* and *Triatoma infestans*. Trop. Med. Parasitol. 95, 287–295.
41. Martínez-Hernández, F., Martínez-Ibarra, J.A., Villalobos, G., De la Torre, P., Laclette, J.P., Alejandré-Aguilar, R., Catalá, S., Espinoza, B., 2010. Natural crossbreeding between sympatric species of the Phyllosoma complex (Insecta: Hemiptera: Reduviidae) indicate the existence of one species with morphologic and genetic variations. Am. J. Trop. Med. Hyg. 82, 74–82.

42. Chapman, R., 1982. Chemoreception: the significance of receptors numbers. Insect Physiol. 16, 247–356.

43. Catalá, S.S., Maida, D.M., Caro-Riaño, H., Jaramillo, N., Moreno, J., 2004. Changes associated with laboratory rearing in antennal sensilla patterns of *Triatoma infestans*, *Rhodnius prolixus*, and *Rhodnius pallescens* (Hemiptera, Reduviidae, Triatominae). Mem Inst Oswaldo Cruz. 99, 25–30.

44. Abraham, L., Hernández, L., Gorla, D., Catalá, S., 2008. Phenotypic diversity of *Triatoma infestans* at the microgeographic level in the Gran Chaco of Argentina and the Andean valleys of Bolivia. Med. Entomol. 45, 660–666.

45. Dujardin, J.P., Costa, J., Bustamante, D., Jaramillo, N., Catalá, S., 2009. Deciphering morphology in Triatominae: The evolutionary signals. Acta Trop. 110, 101–111.

46. Cantillo-Barraza O, Garcés E, Gómez-Palacio A, Cortés LA, Pereira A, Marcet PL, Jansen, A.M., Triana-Chavez, O., 2015. Eco-epidemiological study of an endemic Chagas disease region in northern Colombia reveals the importance of *Triatoma maculate* (Hemiptera: Reduviidae), dogs and *Didelphis marsupialis* in *Trypanosoma cruzi* Parasit. Vectors 8, 482.

47. Guhl, F. 2009. Enfermedad de Chagas: Realidad y perspectivas. Revista Biomedica, 20, 228-234.

48. Silva-Neto, M.A.C., Fampa, P., Caiaffa, C.D., Carneiro, A.B., Atella, G.C., 2010. Cell signaling during *Trypanosoma cruzi* development in triatominae. Open Parasitol. J. 4, 188–194.

49. Libersat, F., Delago, A., Gal, R., 2009. Manipulation of host behavior by parasitic insects and insect parasites. Annu. Rev. Entomol. 54, 189–207.

50. Poulin, R., 2010. Parasite manipulation of host behavior: an update and frequently asked questions. Adv. Study Behav. 41, 151–186.

51. Heil, M., 2016. Host manipulation by parasites: cases, patterns, and remaining doubts. Ecol. Evol. 4, 80.

52. De Fuentes-Vicente, J.A., Gutiérrez-Cabrera, A.E., Flores-Villegas, A.L, Lowenberger, C., Benelli, G., Salazar-Schettino, P.M., Córdoba-Aguilar, A., 2018. What makes an effective Chagas disease vector? Factors underlying *Trypanosoma cruzi* triatomine interactions. Acta Trop. 183, 23–31.

53. Depickère, S., Ramírez-Ávila, G.M., Deneubourg, J., 2019. Alteration of the aggregation and spatial organization of the vector of Chagas disease, *Triatoma infestans*, by the parasite *Trypanosoma cruzi*. Sci Rep 9, 17432.

54. Elliot, S.L. Rodrigues, J. de O., Lorenzo, M.G., Martins-Filho, O.A., Guarneri, A.A., 2015. *Trypanosoma cruzi*, etiological agent of Chagas disease, is virulent to its triatomine vector *Rhodnius prolixus* in a temperature-dependent manner. PLoS Negl. Trop. Dis. 9, e0003646.

55. Hinestroza, G., Ortiz, M.I., Molina, J., 2016. Behavioral fever response in *Rhodnius prolixus* (Reduviidae: Triatominae) to intracoelomic inoculation of *Trypanosoma cruzi*. Rev. Soc. Bras. Med.
Trop. 49, 425–432.
56. Peterson, J.K., Graham, A.L., Elliott, R.J., Dobson, A.P., Chávez, O.T., 2016. *Trypanosoma cruzi–Trypanosoma rangeli* co-infection ameliorates negative effects of single trypanosome infections in experimentally infected *Rhodnius prolixus*. Parasitology 143, 1157–1167.

57. Cordero-Montoya, G., Flores-Villegas, A.L., Salazar-Schettino, P.M., Vences-Blanco, M.O., Rocha-Ortega, M., Gutiérrez-Cabrera, A.E., Rojas-Ortega, E., Córdoba Aguilar, A., 2019. The cost of being a killer's accomplice: Trypanosoma cruzi impairs the fitness of kissing bugs. Parasitol. Res. 118, 2523–2529.

58. Fellet, M.R., Lorenzo, M.G., Elliot, S.L., Carrasco, D., Guarneri, A.A., 2014. Effects of infection by *Trypanosoma cruzi* and *Trypanosoma rangeli* on the reproductive performance of the vector *Rhodnius prolixus*. PLoS One 9, e105255.

59. Eichler, S., Schaub, G.A., 2002. Development of symbionts in triatomine bugs and the effects of infections with trypanosomatids. Exp. Parasitol. 100, 17–27.

60. Botto-Mahan, C., 2009. *Trypanosoma cruzi* induces life-history trait changes in the wild kissing bug *Mepraia spinolai*: implications for parasite transmission. Vector Borne Zoonotic Dis. 9, 505–510.

61. Schaub, G.A., 2006. Parasitogenic alterations of vector behaviour. Int. J. Med. Microbiol. 296, 37–40.

62. Botto-Mahan, C., Cattan, P.E., Medel, R., 2006. Chagas disease parasite induces behavioural changes in the kissing bug *Mepraia spinolai*. Acta Trop. 98, 219–223.

63. Pereyra, N., Lobbia, P.A., Mougalure-Cueto, G., 2019. Effects of the infection with *Trypanosoma cruzi* on the feeding and excretion/defecation patterns of *Triatoma infestans*. Entomol. Res. 24, 1–8.

64. Ramírez-González, M.G., Flores-Villegas, A.L., Salazar-Schettino, P.M., Gutiérrez Cabrera, A.E., Rojas-Ortega, E., Córdoba-Aguilar, A., 2019. Zombie bugs? Manipulation of kissing bug behavior by the parasite *Trypanosoma cruzi*. Acta Trop. 200, 105177.

65. Uc-Diaz, S.S., 2020. Efecto de *Trypanosoma cruzi* sobre la selección de hospederos sanguíneos por *Triatoma dimidiata*. Universidad Autónoma de Yucatán, Campus de Ciencias Biológicas y Agropecuarias. Pp. 65 (BcB Thesis).

66. Vallejo, G., Guhl, F., Schaub, G., 2009. Triatominae- *Trypanosoma cruzi*/? Rangeli: vector–parasite interactions. Acta Trop. 110, 137–147.

67. Oliveira, T., Carvalho-Costa, F., Gomes, T., Sarquis, O., Sposina, R., Lima, R., 2010. Developmental and reproductive patterns of *Triatoma brasiliensis* infected with *Trypanosoma cruzi* under laboratory conditions. Mem. Inst. Oswaldo Cruz 105, 1057–1060.

68. Marliére, N.P., Latorre-Estivalis, J.M., Lorenzo, M.G., Carrasco, D., Alves-Silva, J., Rodrigues, J.O., de Lima Ferreira, L., de Melo Lara, L., Lowenberger, C., Guarneri, A.A., 2015. Trypanosomes modify the behavior of their insect hosts: effects on locomotion and on the expression of a related gene. PLoS Negl. Trop. Dis. 9, e0003973.

69. Takano-Lee, M., Edman, J.D., 2002, Lack of manipulation of *Rhodnius prolixus* (Hemiptera: Reduviidae) vector competence by *Trypanosoma cruzi*. Med. Entomol. 39, 44–51.
70. Zeledón, R., Guardia, V.M., Zúñiga, A., Swartzwelde, J.C., 1970. Biology and ethology of *Triatoma dimidiata* (Latreille, 1811). II. Life span of adults and fecundity and fertility of females. Med. Entomol. 7, 462–469.

71. Schaub, G.A., 1988. Developmental time and mortality of larvae of *Triatoma infestans* infected with *Trypanosoma cruzi*. Trans. R. Soc. Trop. Med. Hyg. 82, 94–97.

72. Lima, M.M., Borges-Pereira, J., Albuquerque Dos Santos, J.A., Teixeira-Pinto, Z., Vianna-Braga, M., 1992. Development and reproduction of *Panstrongylus megistus* (Hemiptera: Reduviidae) infected with Trypanosoma cruzi, under laboratory conditions. Ann. Entomol. Soc. Am. 85, 458–461.

73. Waleckx E, Camara-Mejia J., Ramirez-Sierra M.J., Cruz-Chan V., Rosado-Vallado M., Vazquez-Narvaez S., Najera-Vazquez R., Gourbière S., Dumonteil E., 2015. An innovative ecohealth intervention for Chagas disease vector control in Yucatan, Mexico. R. Soc. Trop. Med. Hyg. 2, 143-9.

74. Waleckx, E., Pérez-Carrillo, S., Chávez-Lazo, S., Pasos-Alquicira, R., Cámara-Heredia, M., Acuña-Lizama, J., Collí-Balám, F., Cámara-Mejía, J., Ramírez-Sierra, M. J., Cruz-Chan, V., Rosado-Vallado, M., Vázquez-Narvaez, S., Najera-Vázquez, R., Gourbière, S., Dumonteil, E., 2018. Non-randomized controlled trial of the long-term efficacy of an Ecohealth intervention against Chagas disease in Yucatan, Mexico. PLoS Negl Trop Dis, 12 e0006605.

75. Moo‐Millan, J.I., Arnal, A., Pérez‐Carrillo, S., Hernandez‐Andrade, A., Ramírez‐Sierra, M.J., Rosado‐Vallado, M., Dumonteil, E., Waleckx1, E., 2019. Disentangling *Trypanosoma cruzi* transmission cycle dynamics through the identification of blood meal sources of natural populations of *Triatoma dimidiata* in Yucatán, Mexico. Parasit. Vectors 12, 572.

76. Sokal, R.R., Rohlf, F.J., 2009. Introduction to Biostatistics, 2nd ed. Dover Publications Inc., New York, pp. 384.

77. Dujardin, S., Dujardin. J.P., 2019. Geometric morphometrics in the cloud. Infect. Genet. Evol. 70, 189–196.

78. Moreno, M.L., Gorla, D., Catalá, S., 2006. Association between antennal phenotype, wing polymorphism and sex in the genus Mepraia (Reduviidae: Triatominae). Infect. Genet. Evol. 6, 228–234.

79. Catalá, S., Sachetto, C., Moreno, M., Rosales, R., Salazar-Schettino, P.M., Gorla, D., 2005. Antennal phenotype of *Triatoma dimidiata* populations and its relationship with species of phyllosoma and protracta Complexes. Med. Entomol. 42, 719–725.

80. Souza, A.C., Catalá, S., Carbajal-de-la Fuente, A.L., Junqueira, A., 2017. Phenotypic variability of the Amazonian species *Rhodnius brethesi* (Hemiptera: Reduviidae). J. Med. Entomol. 54, 909–16.

81. Poulin, R., Brodeur, J., Moore, J., 1994. Parasite manipulation of host behaviour: should hosts always lose? Oikos 70, 479–484.

82. Wigglesworth, V.B., Gillett, J.D., 1934. The function of the antennae in *Rhodnius prolixus* Hemiptera and the mechanism of orientation to the host. J. Exp. Biol. 11, 120–139.

83. McIver, S.B., Siemicki, R., 1984. Fine structure of antennal mechanosensilla of adult Rhodnius prolixus Stål Hemiptera: Reduviidae. J. Morphol. 180, 19–28.
84. Lazzari, C.R., Nuñez, J.A., 1989. The response to radiant heat and the estimation of the temperature of distant sources in *Triatoma infestans*. J. Ins. Physiol. 35, 525–529.

85. Barrozo, R.B., Reisenmann, C.E., Guerenstein, P., Lazzari, C.R., Lorenzo, M.G., 2016. An inside look at the sensory biology of triatomines. J. Insect Physiol. 97, 3–19.

86. Bernard, J., 1974. Études Electrophysiologiques des Récepteurs Impliqués dans la Orientation vers l'Hôte et dans l'Acte Hematophage chez un Hémiptère *Triatoma infestans*. Université de Rennes, France, p. 285 (PhD Thesis).

87. Guerenstein, P., Lazzari, C., 2009. Host-seeking: how triatomines acquire and make use information to find blood. Acta Trop. 110, 148–158.

88. Taneja, J., Guerin, P.M., 1995. Oriented responses of triatomines bugs *Rhodnius prolixus* and *Triatoma infestans* to vertebrate odours on a servosphere. J. Comp. Physiol. A 176, 455–464.

89. Taneja, J., Guerin, P.M., 1997. Ammonia attracts the haematophagous bug *Triatoma infestans*: behavioural and neurophysiological data on nymphs. J. Comp. Physiol. A. 181, 21–34.

90. Arroyo, C.M., Esteban, L., Catalá, S., Angulo, V.M., 2007. Variación del fenotipo antenal de poblaciones del domicilio, peridomicilio y silvestres de *Triatoma dimidiata* (Hemiptera: Reduviidae) en Santander, Colombia. Biomed. 27, 92–100.

91. Lorenzo-Figueiras, A.N., Manrique, G., Lorenzo, M.G., Lazzari, C.R., Schilman, E., 1999. Sensory ecology. B: Communication. In: Carcavallo, R.U., Galíndez Girón, I., Jurberg, J., Lent, H. (Eds.), Atlas of Chagas’ Disease Vectors In The Americas, vol. 3. pp. 1089–1104.

92. Guerenstein, P.G., 1999. Sensory and behavioural responses of Triatoma infestans to host and conspecific odours. University of Neuchâtel, Switzerland, Pp. 137 (Ph.D. Thesis).

93. Steinbrecht, R.A., Stankiewicz, B.A., 1999. Molecular composition of the wall of insect olfactory sensilla: the chitin question. J. Insec. Physiol. 45, 785–790.

94. Steinbrecht, R., Muller, B., 1976. Fine structure of the antenna1 receptors on the bed bug, *Cimex lectularius* Tissue Cell 8, 615–636.

95. Dumonteil, E., Gourbière, S., Barrera-Perez, M., Rodriguez-Felix, E., Ruiz-Piña, H., Baños-López, O., Ramírez-Sierra, M.J., Menu, F., Ravinovich, J.E., 2002. Geographic distribution of *Triatoma dimidiata* and transmission dynamics of *Trypanosoma cruzi* in the Yucatan peninsula of Mexico. Am. J. Trop. Med. Hyg. 67, 176–183.

96. Guzmán-Tapia, Y., Ramírez-Sierra, M.J., Dumonteil, E., 2007. Urban infestation by *Triatoma dimidiata* in the city of Mérida, Yucatán, México. Vector Borne Zoonotic Dis. 7, 597–606.

97. Ibarra-Cerdeña, C.N., Zaldivar-Riveron, A., Peterson, A.T., Sanchez-Cordero, V., Ramsey, J.M., 2014. Phylogeny and niche conservatism in north and Central American triatomine bugs (Hemiptera: Reduviidae: Triatominae), vectors of Chagas' disease. PLoS Negl. Trop. Dis. 8, e3266.

98. López-Cancino, S.A., Tun-Ku, E., De la Cruz-Felix, H.K., Ibarra-Cerdeña, C.N., Izeta-Alberdi, A., Pech-May, A., Mazariegos-Hidalgo, C., Valdez-Tah, A., Ramsey, J., 2015. Landscape ecology of *Trypanosoma cruzi* in the southern Yucatan Peninsula. Acta Trop. 151, 58–72.
99. Catalá, S., Dujardin, J.P., 2001. Antennal sensilla patterns indicate geographic and ecotopic variability among *Triatoma infestans* (Hemiptera: Reduviidae) populations. J. Med. Entomol. 38, 423–428.

100. Catalá, S., Carbajal, A., Torres, M., Moreno, M., Ordoñez, R., Montaña, F., Esteban, L., Gonzalez, N., 2000. La antena de los triatominae: caracteres ancestrales y marcadores funcionales. In: Schofield, C., Ponce, C. (Eds.), Proceedings IV International Workshop on Population Genetics and Control of Triatominae. European Community, Cartagena de Indias. Colombia, pp. 80–81 (INDRE. Mexico).

101. Carbajal de la Fuente, A.L., Catalá, S., 2002. Relationship among the habitat and the antenal sensilla pattern of six species of Triatominae (Hemiptera: Reduviidae). Inst. Oswaldo Cruz 97, 1073–1077.

102. May-Concha, I.J., 2010. Compuestos volatiles emitidos por adultos en disturbio y copula de *Triatoma dimidiata* (Hemiptera: Reduviidae), vector de la enfermedad de Chagas. Instituto Nacional de Salud Publica, Tapachula, pp. 1–27 (MSc Thesis).

**Tables**

**Table 1.** Number of *T. dimidiata* specimens used in this study. Population, sex and infection status of the specimens are indicated.

| Populations       | Infected | Non-infected | Overall |
|-------------------|----------|--------------|---------|
| Laboratory-reared | 10       | 10           | 11      |
| Domestic          | 10       | 10           | 10      |
| Sylvatic          | 10       | 10           | 13      |
| Overall           | 30       | 30           | 33      |

**Table 2.** Comparisons of the abundances of each sensillum between infected and non-infected insects overall populations, within each sex, within each population, and within each sex within each population of *Triatoma dimidiata*. BR: bristles; BA: basiconic; TH: thin-walled trichoid; TK: thick-walled trichoid. F: female and M: male. I: infected; NI: non-infected. D: domestic, S: Sylvatic, and L: Laboratory reared. Asterisks represent a significant difference between infected and non-infected insects (P < 0.05*; P < 0.01**; P < 0.001***; – no difference).
| Factor                                      | Pedicel | Flagellum 1 | Flagellum 2 |
|---------------------------------------------|---------|-------------|-------------|
| Overall populations (I vs NI)               |         |             |             |
| Whitin females (I females vs NI females)    |         |             |             |
| Whitin males (I males vs NI males)          |         |             |             |
| Whitin domestic insects (I D vs NI D)       | *       |             |             |
| Whitin sylvatic insects (I S vs NI S)       |         | ***         | ***         |
| Whitin laboratory-reared insects (I L vs NI L) |         |             |             |
| Whitin females of the domestic population (F D I vs F D NI) |         |             |             |
| Whitin females of the sylvatic population (F S I vs F S NI) |         | ***         | ***         |
| Whitin females of the laboratory-reared population (F L I vs F L NI) |         |             |             |
| Whitin males of the domestic population (M D I vs M D NI) | *       |             | ***         |
| Whitin males of the sylvatic population (M S I vs M S NI) |         | *           | ***         |
| Whitin males of the laboratory-reared population (M L I vs M L NI) |         |             |             |

**Table 3.** Two-way PERMANOVA based on Bray-Curtis distance matrix assessing the sources of variation of the AP of *T. dimidiata* populations. P-values are based on 9999 permutations.
A) Domestic population

| Source of variation | Sum of squares | Mean square | F    | P      |
|---------------------|---------------|-------------|------|--------|
| Infection           | 0.188         | 0.188       | 7.151| 0.0001 |
| Sex                 | 0.039         | 0.0.39      | 1.5179| 0.1776 |
| Interaction         | 0.031         | 0.031       | 1.1887| 0.2993 |

B) Sylvatic population

| Source of variation | Sum of squares | Mean square | F    | P      |
|---------------------|---------------|-------------|------|--------|
| Infection           | 0.209         | 0.209       | 7.418| 0.0001 |
| Sex                 | 0.121         | 0.121       | 4.288| 0.0021 |
| Interaction         | 0.103         | 0.013       | 0.368| 0.125  |

C) Laboratory-reared

| Source of variation | Sum of squares | Mean square | F    | P      |
|---------------------|---------------|-------------|------|--------|
| Infection           | 0.031         | 0.031       | 0.708| 0.569  |
| Sex                 | 0.083         | 0.083       | 1.869| 0.104  |
| Interaction         | 0.052         | 0.052       | 0.126| 0.473  |

Table 4. Comparisons of the abundances of each sensillum between infected females and males and between non-infected females and males overall populations, and within each population of *Triatoma dimidiata*. BR: bristles; BA: basiconic; TH: thin-walled trichoid; TK: thick-walled trichoid. F: female and M: male. I: infected; NI: non-infected. D: domestic, S: Sylvatic, and L: Laboratory reared. Asterisks represent a significant difference between infected and non-infected insects (P < 0.05*; P < 0.01**; P < 0.001***; – no difference).
| Factor                                           | Pedicel | Flagellum 1 | Flagellum 2 |
|-------------------------------------------------|---------|-------------|-------------|
|                                                 | BR      | BA          | TH          | TK          | BR | BA | TH | TK | BR | BA | TH | TK |
| Overall non-infected insects (NI females vs NI males) | –       | –           | –           | –           | –  | –  | –  | –  | –  | –  | –  | –  |
| Overall infected insects (I females vs I males)  | –       | –           | **          | –           | –  | –  | –  | –  | –  | –  | –  | –  |
| Whitin non-infected domestic insects (F D NI vs M D NI) | –       | –           | –           | –           | –  | –  | –  | –  | –  | –  | –  | –  |
| Whitin infected domestic insects (F D I vs M D I) | –       | –           | –           | –           | –  | –  | –  | –  | *  | –  | –  | –  |
| Whitin non-infected sylvatic insects (F S NI vs M S NI) | –       | –           | *           | –           | –  | –  | –  | –  | ** | –  | –  | –  |
| Whitin infected sylvatic insects (F S I vs M S I) | –       | **          | *           | –           | –  | –  | –  | –  | *  | –  | –  | –  |
| Whitin non-infected laboratory-reared insects (F L NI vs M L NI) | –       | –           | –           | –           | –  | –  | –  | –  | –  | –  | –  | –  |
| Whitin infected laboratory-reared insects (F L I vs M L I) | –       | –           | –           | –           | –  | –  | –  | –  | –  | –  | –  | –  |

**Supplementary Files**

This is a list of supplementary files associated with this preprint. Click to download.

- GraphicalabstractMayConchaetal2021.tif
- SupplementarymaterialMayConchaetal2021.docx