I have received two papers for discussion: *Chagas disease: from bush to huts and houses*. It is the case of the Brazilian Amazon? by Prof. Coura and Genetic variability in Brazilian triatomines and the risk of domiciliation, by Dr Borges. I found both papers very interesting, confirming several previous points of the literature. However, I would like to comment on them, firstly on Prof. Coura’s work, in order to clear up some subjects brought up in his paper.

The most plausible hypothesis about the establishment of new foci of Chagas disease in the Amazonian forests is that their origins are in the wild niches of *Rhodnius*, following the invasion and deforestation by man, who uses bushes and straw, infested with such bugs, in order to build his dwelling. Palms, principally *piassava*, are the most common habitat of the triatomines. In fact, the genus *Rhodnius*, comprising several species, probably represents more than 50% of all the triatomines existing in the Amazon forests.

The genera *Belminus*, *Cavernicola*, *Eratyrus*, *Panstrongylus* and *Triatoma*, have been mentioned in the literature, always with an indication of their low numbers. The species which belong to the first three genera listed above do not retain any practical importance for the establishment of anthroponotic foci of *Trypanosoma cruzi*, as they are scarce and do not tend to bite humans. The same can be said about the species of *Panstrongylus* of the region.

Regarding the species of the genus *Triatoma*, *T. maculata* has a low susceptibility to harbor *T. cruzi*; *T. rubrovaria*, a species which was described in the south of Brazil, possibly is registered erroneously as living in the Amazon. If this species had been introduced into the Amazon region, it could not adapt itself to an ecological system quite different to that of its origin, and would consequently disappear or became very rare.

Meanwhile, *T. rubrofasciata* which has adapted itself to a wide range of geographical distribution, has been dispersed from India to several ports around the World, including the Brazilian Amazon. This species is the principal vector of *T. conorhini* among rats. But although it can also be infected with *T. cruzi*, it is not epidemiologically important since it does not generally bite man, preferring to feed on rats.

Apart from the invasion of human dwellings by wild *Rhodnius* in the forests, and the domiciliated *T. rubrofasciata* existing in some city ports in the Amazon, there is no other record of the triatomines responsible for the maintenance of transmission inside houses in some foci of Chagas disease in this region.

The asymptomatic infection with *T. cruzi* in the Amazonian forests surely represents the natural biological behaviour of this trypanosome. This means that *T. cruzi* lives in balanced association with several species of mammals in its natural niches. However one can not exclude the possibility of a different picture in some circumstances, such as the introduction of special strains of *T. cruzi*, or of healthy mammals not yet adapted to that biocenosis. Then the infection can become a true wild zoonosis with pathologic consequences for the infected mammals.

In a similar way, there is also the possibility of infected immigrants being the origin of the epidemic outbreaks of Chagas disease, with the species of *Rhodnius* already existing in the area becoming the principal vectors.

Prof. Coura and cols stressed in their cross sectional studies, carried out in rural areas of Rio Negro, that strong correlation was shown between seroreactivity and the the activity of gathering *piassava* straw fibers.

When wild animals are forced to go to other areas because of deforestation, the triatomines look for alternative blood food sources and new shelters to hide. Then the artificial new ecosystems of the peri and intradomicilary areas become easier
ecotopes with opportunities for abundant blood meals.

So, as suggested by Prof. Coura and cols, precautions must be taken against indiscriminate deforestation and the new settlements must be controlled such that the ecosystem is preserved.

It was interesting that in the work of Prof. Coura and cols, only 1.9% (out of 156 Elisa positive patients performed) were positive using xenodiagnosis. Was this result due to the bugs being fed again on chickens after they had consumed the infective blood meal in the positive patients, or lack of adaptation of the *T. cruzi* Amazonian wild strain to the foreign species of triatomines used in xenos? Or perhaps another species of trypanosome was involved?

There remains the possibility of that the initiation of many foci of Chagas disease in the Amazonian forest could be caused by the infection of man by means of the manipulation or ingestion of infected wild mammals or alternatively, through contact with excretions of these mammals or of triatomines.

In conclusion, I think that the title of the paper of Prof. Coura and cols, could dispense with the sentence “is the case of the Brazilian Amazon?”.

The above arguments point that the picture suggested in the title is not accurate.

Regarding the article of Dr Borges and cols, the authors stressed that those triatomines collected after the sprayings, and identified by genetic, molecular and biochemical techniques, were proven survivors of the original populations and not invaders from the surrounding wild habitats. I think that this observation is only true when the surveys are carried out immediately after the sprayings. Perhaps later, when the residual action of the insecticides is spent, triatomines from the surrounding wild habitats will invade the area.

Until now, it has only been possible to demonstrate eradication of non-indigenous species of vectors, such as *Anophles gambiae*, *Aedes aegypti*, etc. Concerning triatomines specifically, the control of *T. infestans*, a foreign species, is easier than that of the *P. megistus* authocotonous bug. Also, the control of any vector species inside houses is easier than the control of those in the peridomiciliary areas, where the species has more options for escape.