

Chagas disease

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A trypanosome (genus *Trypanosoma*) is a parasitic flagellated protozoan that infects the blood, esophagus and colon of all vertebrate classes, and intestinal cells in invertebrates. Chagas disease (American trypanosomiasis), found mainly in South America, is a sometimes deadly parasitic condition caused by *Trypanosoma cruzi*. It is spread mostly by the triatomine (or kissing) bug of the subfamily Triatominae (figure 1), which feeds mostly on the person's face. The insect takes a blood meal from a vertebrate's host and, as it does, it sheds the infective stage of the parasite onto the host's skin where it can enter the body via the mucous membrane of the eye and bite wounds on the skin. Once in the host the parasites undergo various changes.

Two strains develop: the myotropic strain, which forms pseudocysts in muscle cells, and the reticulotropic strain in white blood cells. Unique trypomastigote forms of the parasite circulate in the blood and are eventually ingested by another

kissing bug and the cycle between bug and human (or animal) continues. Alternate hosts or wildlife reservoirs that can carry this parasite in the United States include opossums, squirrels, armadillos, woodrats, and raccoons.

Endemic areas are South American countries, affecting around 8 million people with an annual rate of 561,000. Non-endemic countries (e.g. the United States) have about 350,000 infected individuals. The worldwide cost of treating this disease is estimated at \$7 billion.

Unique biochemistry and life cycle

Trypanosomes have the uncanny ability to coat themselves with a protein covering that makes them quite invisible to the vertebrate immune system and is the reason why it is so difficult to find a cure for *T. cruzi* infections (as well as African trypanosomiasis, or sleeping sickness). In addition, when they ensconce themselves inside a vertebrate cell, the trypanosomes have a biochemical ability to override the cell's self-destruct mechanism (via an enzyme called Akt), which would otherwise kill the cell and its deadly foreign

cargo. This cell-destructive capacity was originally for regulatory purposes and was a microbe interface system design feature.

Like harmful mutations, creationists in general feel parasitism is part of the Curse, in Genesis 3. Indeed, according to evolutionists, parasitic behaviour, "[is] still an enigma".¹

Complex life cycles of parasites are common. Within these cycles can be found incredible morphological and biochemical transformations. For example, the blood flukes (Digeneans, class Trematoda) have a stage called the cercaria. It is a small, heart-shaped larval phase that undergoes locomotion for about a day in freshwater, seeking a bird or mammal host. The cercaria originates from a structure called the daughter sporocyst within a snail (e.g. *Biomphalaria*). When the cercaria contacts the skin of a host it wiggles vigorously as it sheds its forked tail. After it enters the host's peripheral circulation it is called a schistosomule. The change this tiny entity undergoes is nothing short of amazing. It initially lived in a cold-blooded invertebrate with the ability to evade its immune system; then it enters a cool, freshwater environment. From there it enters a warm-blooded host with a more sophisticated immune system. The number of changes it must immediately undergo in order to survive each new environment is astounding. How could such a series of rapid biochemical changes evolve? It is no wonder an evolutionist stated: "It would be difficult, if not impossible, to explain, step by step, the details of the process of evolution by which some of the highly specialized parasites reached their present condition."²

Things have not changed in the 21st century:

"Hence, tempo and mode of host-parasite co-evolution at the macro-evolutionary scale remains a major challenge to understand."³



image: Glenn Seplak via Flickr

Figure 1. The blood-sucking 'kissing bug', the agent of dispersal of a disease infecting millions

Trypanosome origin

What was the origin of this parasitic trypanosome? What is required for a creature to transition to a parasitic lifestyle from a free-living condition is the loss or modification of anatomical and physiological systems. Poulin has stated that in some metazoan groups “parasitic species have retained some morphological resemblance with their free-living counterparts.”⁴ For example, *T. cruzi* is much like *T. rangeli*, which is not known to be disease-causing in people. *T. rangeli* often coexists with *T. cruzi*. It is common in people, cats, and dogs in South America and is found in triatomine bugs. Is it possible that at one time *T. cruzi* was also a non-pathogen that, through some genetic changes, became the scourge of the Americas? Could the protein coating be a way trypanosome and host could have existed together before the Fall?

In Genesis, when God cursed the earth with weeds and thorns, some non-parasitic protozoa may have lost the ability to live free in the environment and adopted a metazoan host for part—or all—of its life cycle. Perhaps, before the Fall, plant-feeding triatomine bugs had beneficial protozoa such as trypanosomes in their gut. Indeed, some species of trypanosomes are monoxenous, meaning they are found within one arthropod host. There is an intriguing genus of slender, long trypanosome (*Leptomonas*) with a free flagellum (a microscopic whip-like structure). It lives in the hindgut and is unique because the insect is the sole host. The trypanosome living in a mutualistic association within the arthropod host before the Fall would have required a (designed) microbe interface system. The insect and the trypanosome are autonomous entities that are harmonized by this interface arrangement. So, too, humans have associated with microbes (e.g. in their gut) since creation, so this microbial interface system is

a design certainty.⁵ This would answer the difficult question as to the function of the immune system before the Fall. Creation scientists are increasingly looking at this pre-Fall immune system in light of a dynamic host system-to-microbe relationship understood in light of design analysis. Perhaps an understanding of our immune system would be different with a fresh look via design analysis coupled with the work of creationist Joe Francis’ enhanced, co-operative conception of our microbiota.⁶

Mycetomes and triatomines

Some blood-feeding insects have specialized structures, called ‘mycetomes’, that carry endosymbiotic micro-organisms, which, in turn, provide nutrients to the insect. Triatominae and Cimicidae have a partial nutritional dependence on these micro-organisms. Cimicids have two disc-shaped mycetomes beside the gonads, while bloodsucking reduviids have epithelial cells in their gut containing these bacteria. There is good evidence that the triatomine bacteria are crucial for maturation and growth of the insects. Work can be done by creation microbiologists to determine possible links between these endosymbiotic micro-organisms, trypanosomes, and pathogenicity of Chagas disease.

All species of the triatomines are potential vectors of the Chagas disease parasite. Hemiptera generally feed on plant sap, and before the Fall all Hemiptera fed on plants (which are not ‘alive’ in the biblical sense). Perhaps, as mentioned above, the trypanosomes had a mutualistic relationship with the Hemiptera, much like the many hundreds of species of termites that have mutualistic protozoa (e.g. *Monocercomonas*). After the Fall, the transition was made from plants to people and animals. But how many steps would it take for free-living animals to become parasitic? Although

it is true that some life cycles are complex (see reference 2), others may be virtually a one-step process, depending on the animal.

“In fact, free-living species could become parasitic without substantial anatomical or physiological changes.”⁷

Furthermore, it is intriguing that secular biologists, using evidence from molecules, estimate people may have been suffering from Chagas’ disease for at least 4,000 years – which is within the biblical timeframe.⁸

Conclusion

Trypanosoma cruzi is the scourge of South America with a unique biochemistry and evasion that makes cure and control of this protozoan parasite difficult. Secular biologists are unsure as to the origin of parasites whereas creation scientists see parasites as a result of the curse, possibly a transition from a mutualistic to a parasitic existence.

Many questions remain to be answered regarding this devastating parasite. Creation scientists continue to research it and other parasitic diseases from a non-Darwinian perspective.

References

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