Chapter

Introductory Chapter: Biology of *Trypanosoma cruzi*

Wanderley de Souza

1. Introduction

Trypanosoma cruzi, an important zoonotic protozoan that causes Chagas disease, is the focus of this book. There are two major reasons for the significant interest in the study of this protozoan and the disease it causes. First, Chagas disease is an important life-long infection in humans that can be divided into distinct clinical stages: the acute phase, where patient symptoms can vary from asymptomatic to severe; the indeterminate form, which is usually asymptomatic; and the chronic phase, where cardiomyopathy and/or digestive mega syndromes appear. In Latin America, at least 8 million people are infected with *T. cruzi* and 13,000 die each year. In addition, migration patterns are driving the globalization of the disease and there are around 300,000 and 120,000 people infected in the USA and Europe, respectively. Second, *T. cruzi* is an interesting biological model for studying processes such as: (a) cell differentiation, where a non-infective stage transforms into an infective one; (b) cell invasion, where the infective stages are able to penetrate into a mammalian host cell, where they multiply several times and thus amplify the infection; and (c) evasion from the immune system, using several mechanisms.

To better understand the information presented in various chapters of this book, let us review some basic information about *T. cruzi* infection [1]. **Figure 1** shows a general view of the life cycle of *T. cruzi* in both vertebrate and invertebrate hosts [2]. The three basic developmental stages (trypomastigote, amastigote, and epimastigote) are schematically shown in **Figure 2a–c**, based on images obtained using transmission electron microscopy [2]. The various structures and organelles found in the protozoan are indicated. **Figure 3** shows a scheme, where the various phases of the interaction of the trypomastigote form of *T. cruzi* with a host cell are indicated [1, 2]. The process starts with adhesion of the infective stage to the host cell surface followed by parasite internalization with formation of a parasitophorous vacuole (PV), lysis of the PV membrane, division of amastigotes in the cytoplasm of the host cell, and transformation of amastigotes into trypomastigotes that are then released into the intercellular space.

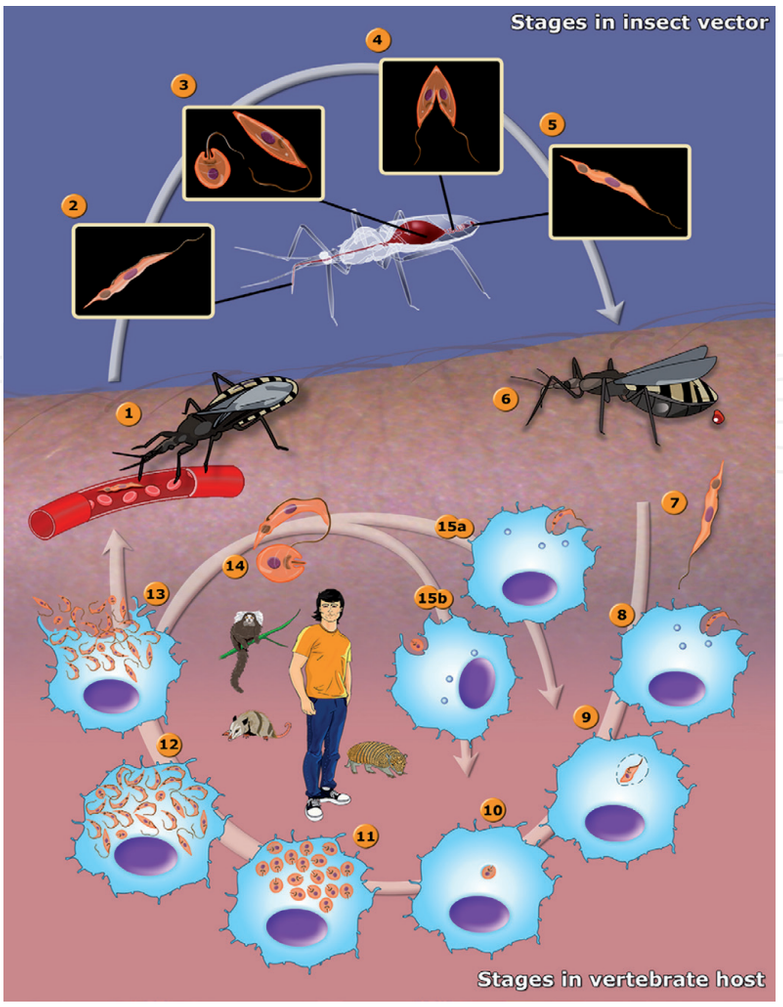

Figure 1.
The life cycle of T. cruzi. 1—The insect vector (female or male) bites a mammalian host and ingests trypomastigotes located in the blood. 2—Metacyclic trypomastigotes. 3—Trypomastigotes transform into epimastigotes and some spheromastigotes. 4—Epimastigotes multiply in the midgut. 5—Epimastigotes transform into metacyclic trypomastigotes in the hindgut. 6—The insect vector passes the metacyclic trypomastigotes in feces near a bite site after feeding on a mammalian host. 7—Metacyclic trypomastigotes form. 8—Metacyclic trypomastigote infects macrophages. 9—Metacyclic trypomastigote transforms into amastigote. 10—Amastigote is released from the parasitophorous vacuole. 11—Amastigotes multiply in the cytoplasm. 12—Amastigotes transform into trypomastigotes. 13—Trypomastigotes burst out of the cell. 14—Amastigotes and trypomastigotes form. 15—(a) Trypomastigotes and (b) amastigotes infect macrophages. In the central portion of the figure, we added the most important animal reservoirs involved in the maintenance of the parasite in the domestic and peridomestic environment (from [2]).

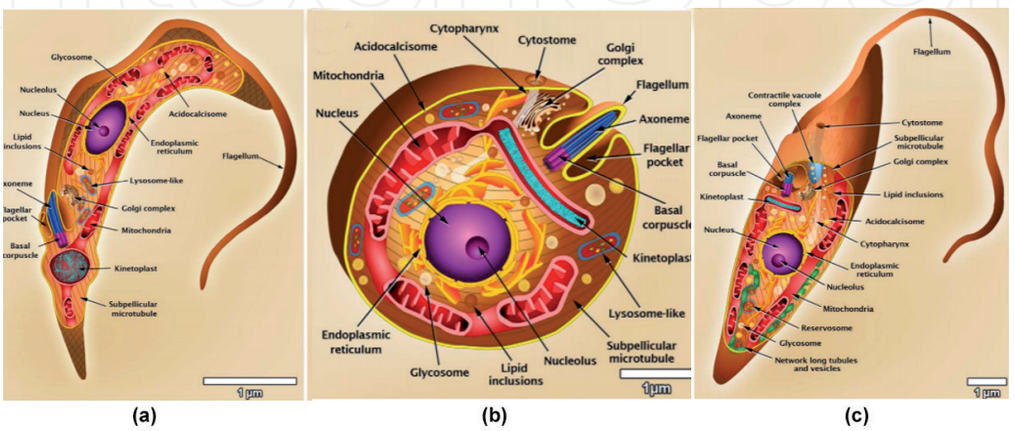

Figure 2.
Schematic representations of T. cruzi (trypomastigote, amastigote, and epimastigote) organelles with 2D models. These images were made based on micrographs of light microscopy as well as scanning and transmission electron microscopy (from [2]).

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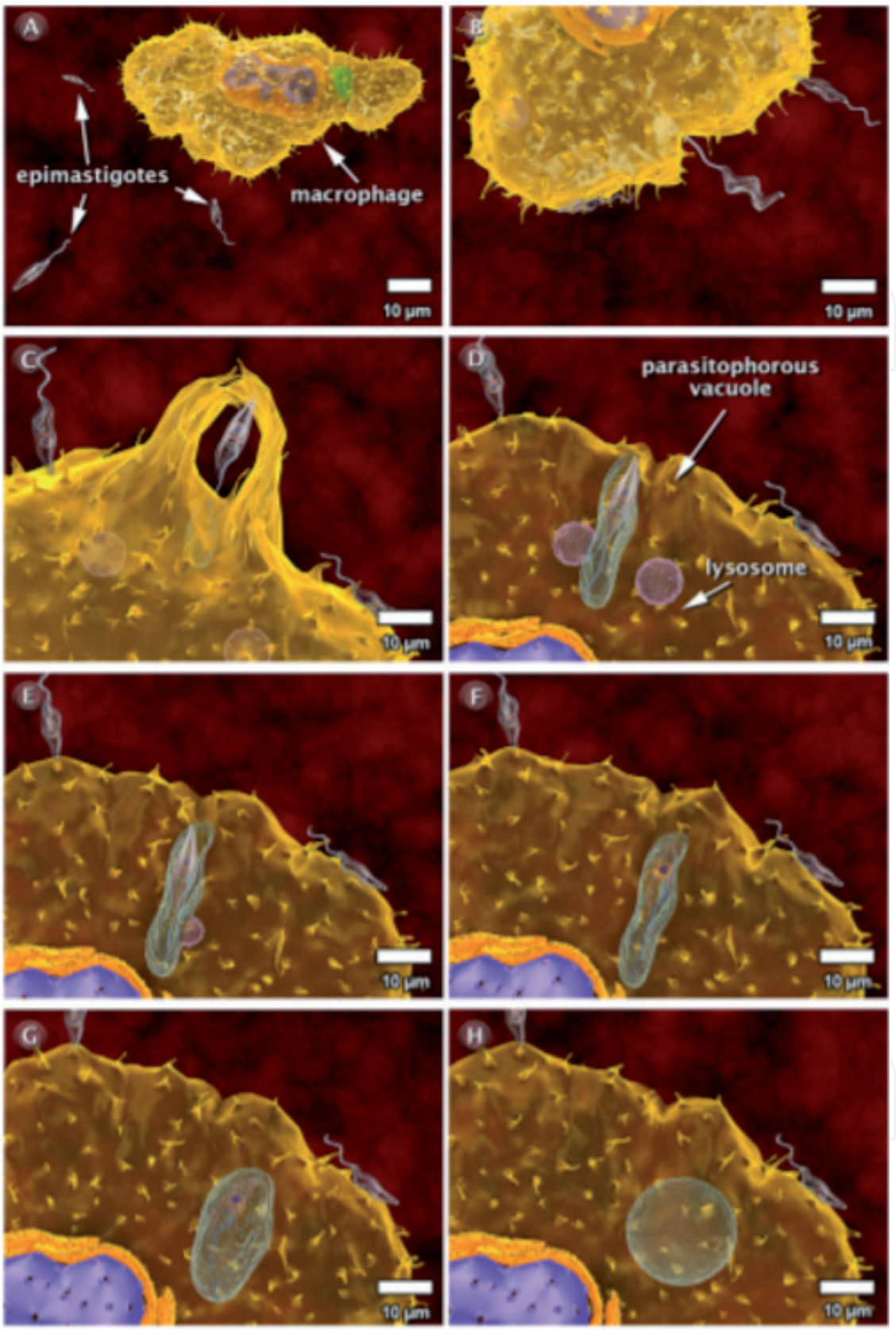

Figure 3.
Schematic representation of the interaction of trypomastigotes with the host cell. The various steps of the interaction are shown with attachment (A, B), internalization with formation of a parasitophorous (PV) (C), lysis of the PV membrane (D), successive division of amastigotes in the cytoplasm (E, F), transformation of amastigotes into trypomastigotes (G) and rupture of the cell releasing a large number of trypomastigotes (H) (from [2]).

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