Why Studies on Invasion of Host Cell by *Trypanosoma cruzi* Using Stablished Cell Lines or Primary Cell Cultures Give Conflicting Results?

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Key words: *Trypanosoma cruzi* - invasion - Chagas disease

Studies *in vitro* of the interaction of *Trypanosoma cruzi* with host cells did not always involve cells that are *in vivo* targets of infection. Tumoral cells and cells lines of different origins, which are commonly used, do not represent the real possibility of the interaction of the parasite within mammal host. In the last 15 years our group has been using primary cultures of heart and skeletal muscular cells, the main target cells during evolution of Chagas disease, to approach the biology of the *T. cruzi*, the molecular events of the parasite interaction, the formation of the parasitophorous vacuole and the intracellular fate of the parasites (Meirelles et al. 1986, Araújo-Jorge et al. 1992, Barbosa & Meirelles 1992, 1993). One of the points that remains under discussion, mainly in the last 10 years, has been the mechanism of invasion of phagocytic and non-professional phagocytic cells by *T. cruzi*. A series of papers have reported that cytochalasins B (CB) and D (CD) block the entry of epimastigotes and trypomastigotes into macrophages, Vero cells and fibroblasts (Alexander 1975, Nogueira & Cohn 1976, Ebert & Barbosa 1981, Henriquez et al. 1981, Meirelles et al. 1982, Zenian & Kierszenbaum 1983), while others have reported active penetration of trypomastigote forms into CB-treated fibroblasts, MDCK and HeLa cells (Schenkman et al. 1991, Schenkman & Mortara 1992). Amastigote forms, on the other hand, invade HeLa cells after association with surface microvilli and mobilization of actin microfilaments (Mortara 1991). CD treatment has been also shown to enhance *T. cruzi* invasion of rat kidney epithelial cells, and the with disruption of cell microfilaments facilitates the access of lysosomes to the adhesion site (Tardieux et al. 1992). Our results obtained with cardiomyocytes showed that the infection rate ranges from 65 to 75% when CB and CD are used. Ultrastructural analysis on the first 30 min of interaction showed that pseudopodia-like expansions of the host cell membranes occur in the adhesion step of the parasite, which are later enclosed by projections of the host cell membrane. Infected cells treated with Triton X-100 demonstrated active mobilization of cytoskeleton filaments at the site of parasite invasion and "sleeve-like" membrane extensions around the parasites. Fixed parasites were never seen inside cardiomyocytes, neither live parasites did invade fixed cells. Our data do not preclude the possibility of additional mechanism(s) of penetration that might require more active participation of the parasites for complete invasion to occur, but indicate that endocytosis is the main process involved in the uptake of metacyclic forms of *T. cruzi* by cardiomyocytes (Barbosa & Meirelles 1995). In a recent paper, De Souza et al. (1998) suggested that both active penetration and typical phagocytosis can be used by the parasites to invade macrophages and Vero cells, and that both process can occur in the same cell.

Does the use of different host cells, as well as different strains and evolutive forms of the parasites increase the knowledge on the biology of the parasite or does it amplify the differences in results?

This question has been partially answered during Dr Mortara presentation in this round-table: the distribution of different host cell components during the parasite invasion is dependent on the infective forms and also on the host cells, which has been demonstrated by the recruitment of extracellular matrix components, integrin receptors and cytoskeleton elements of HeLa and Vero cells (Procópio et al. 1998).

From the above remarks, a question still unsolved: Can the mechanisms of *T. cruzi* invasion described with cell lines and tumor cells be con-

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Received 9 June 1999
Accepetd 9 August 1999

sidered as universal, despite the fact that these cells are not involved in the *in vivo* system during the Chagas disease?

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