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INTERACTION OF TPA-TREATED TRICHOMONADS WITH FIBRONECTIN-COATED SUBSTRATA

VERA LÚCIA BONILHA & FERNANDO COSTA E SILVA FILHO

Departamento de Parasitologia e Biofísica Celular,
Instituto de Biofísica Carlos Chagas Filho, CCS-Bloco G,
21941 Ilha do Fundão, Rio de Janeiro, RJ, Brasil

Tritrichomonas foetus and *Trichomonas vaginalis* are parasitic protozoa naturally occuring in the urogenital tract of bovines where it display serious symptoms characterizing the bovine and human trichomoniasis, respectively (B. M. Honigberg, 1978, *Parasitic Protozoa, 2*: 164-273 8275-454). The etiology of the disease seems to be intimaly related to the adhesion of the parasite to the host epithelial cells (J. Kulda & B. M. Honigberg, 1969, *J. Protozool., 16*: 479-495). During last years some reports have pointed out the adhesion exerted by the parasite to both living and non-living surfaces as a very relevant subject of investigation (F. C. Silva Filho & W. de Souza, 1988, *Cell Struct. Funct., 52*: 362-380; F. C. Silva Filho & V. L. Bonilha, in preparation). In addition, it has also been found that cell matrix glycoproteins such as laminin, may provide a suitable surface for parasite adhesion (F. C. Silva Filho et al., 1988, *Proc. Natl Acad. Sci. USA, 85*: 8042-8046). Some cellular processes described in cell adhesion, such as attachment and spreading, are Ca^{2+} regulated processes, which in turn is modulated by protein kinase C (R. L. Juliano, 1987, *Biochim. Biophys. Acta, 907*: 261-278). This membrane-bound enzyme is highly sensitive to tumour promoters such as the phorbol ester 12-0-tetradecanoylphorbol 13-acetate (TPA) (R. Gopalakristina & S. H. Barry, 1988, *Proc. Natl Acad. Sci. USA, 85*: 612-616).

Parasites grown in a medium (L. S. Diamond, 1957, *J. Parasitol., 43*: 488-490) supplemented with nanomolar concentrations of TPA (V. L. Bonilha & F. C. Silva Filho, submitted) were allowed to interact with 30 μg ml^{-1} fibronectin-coated or uncoated polystyrene substrata. Treatment with TPA became parasites morphologically altered; many rounded forms of the parasite exibiting fillopodia-like projections could be found. These activated regarding forms of *I. foetus* firmly adhered to either uncoated as fibronectin-coated substrata, being that a sequencial step of de-adhesion was observed by using the last substrate. The exact mechanism by which TPA interfere with fibronectin-binding surface components of *parasites* is currently under investigation. However, some beginning data permit us to suggest the occurence of shedding or endocytosis of the fibronectin-binding surface components concomitantly to the activation of a surface protease of *T. foetus*, as consequence of the TPA treatment.

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