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Blood-brain barrier traversal by African trypanosomes requires calcium signaling induced by parasite cysteine protease

Olga V. Nikolskaia, ..., Julio Scharfstein, Dennis J. Grab

J Clin Invest. 2008;118(5):1974-1974. https://doi.org/10.1172/JCI27798C1.

Corrigendum Infectious disease

Original citation: J. Clin. Invest.116:2739–2747 (2006). doi:10.1172/JCI27798. Citation for this erratum: J. Clin. Invest.118:1974 (2008). doi:10.1172/JCI27798C1. The Trypanosoma species used in this study included a clinically relevant human CSF isolate and bloodstream form (BSF) from a patient with sleeping sickness. A cloned derivative from this parasite termed "IL1852" was originally identified as a T.b. gambiense and was denoted accordingly in the manuscript. However, the authors recently discovered that IL1852 contains the SRA gene, a characteristic only encountered in T.b. rhodesiense (1). Therefore, because ILRI T.b. gambiense IL2343, a clone derivative of STIB386AA that was derived from TH144/78E(020), was later reclassified as a T.b. rhodesiense (2), the authors have reclassified IL1852 as a T.b. rhodesiense to maintain accuracy. While the reclassification affects certain aspects of the conclusions of this work, it does not invalidate the key finding of a difference in the BBB traversal between human and animal trypanosomes that correlates with the greater incidence of CNS infection in human compared with animal parasites.

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Article amendments



Corrigendum

Blood-brain barrier traversal by African trypanosomes requires calcium signaling induced by parasite cysteine protease

Olga V. Nikolskaia, Ana Paula C. de A. Lima, Yuri V. Kim, John D. Lonsdale-Eccles, Toshihide Fukuma, Julio Scharfstein, and Dennis J. Grab

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The *Trypanosoma* species used in this study included a clinically relevant human CSF isolate and bloodstream form (BSF) from a patient with sleeping sickness. A cloned derivative from this parasite termed "IL1852" was originally identified as a *T.b. gambiense* and was denoted accordingly in the manuscript.

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While the reclassification affects certain aspects of the conclusions of this work, it does not invalidate the key finding of a difference in the BBB traversal between human and animal trypanosomes that correlates with the greater incidence of CNS infection in human compared with animal parasites.

- 1. Welburn, S.C., et al. 2001. Identification of human-infective trypanosomes in animal reservoir of sleeping sickness in Uganda by means of serum-resistance-associated (SRA) gene. *Lancet.* **358**:2017–2019.
- 2. Hide, G., Cattand, P., LeRay, D., Barry, J.D., and Tait, A. 1990. The identification of Trypanosoma brucei subspecies using repetitive DNA sequences. *Mol. Biochem. Parasitol.* 39:213–225.

Expression of concern

HIV-specific cytotoxic T lymphocytes traffic to lymph nodes and localize at sites of HIV-1 replication and cell death

Scott J. Brodie, Bruce K. Patterson, Deborah A. Lewinsohn, Kurt Diem, David Spach, Phillip D. Greenberg, Stanley R. Riddell, and Lawrence Corey

Original citation: J. Clin. Invest. 105:1407-1417 (2000). doi:10.1172/JCI8707.

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In the issue of May 15, 2000, we published a study by Scott J. Brodie and colleagues. According to the report issued by John T. Slattery, Vice Dean of Research and Graduate Education at the University of Washington in Seattle, Washington, "Dr. Brodie was found to have falsified images that appeared as Figure 5A in the publication. These images respectively appeared in JCI as representing neomycin gene–marked CD8⁺ cells before patient infusions, with neo-positive cells showing yellow-red fluorescence and neo-negative cells being purple-blue; in one unfunded NIH grant application labeled as cells harboring HIV DNA (PCR in situ hybridization for gag DNA); and in a second unfunded NIH grant application as depicting alveolar macrophages from HIV+ persons treated with LPS, tuberculin or HIV tat protein-stain for viral RNA. The University's investigative committee concluded that two or more of these images was falsified."

There is an ongoing investigation into potential scientific misconduct in the performance of this study, reportedly by the Office of Research Integrity. We will inform our readers of the outcome of this investigation when it is complete.