Ana Godinho-Santos et al., J Antivir Antiretrovir 2015, 7:4
http://dx.doi.org/10.4172/1948-5964.C1.025

OMICS International
conferenceseries.com

5th World Congress on
Virology

December 07-09, 2015 Atlanta, USA

CIB1 and CIB2 are HIV-1 helper factors and their modulation influences envelope-mediated viral entry

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Human Immunodeficiency Virus type 1 (HIV-1) relies on the host cell machinery to complete its life cycle. Several helper factors have been identified in expectation that a better understanding of host-HIV interactions would lead to novel antiviral targets. As the characterization for many of these proteins is still limited, we aimed to depict the contribution of CIB2, previously identified as helper factor, as well as the potential contribution of its homolog, CIB1. Knockdown of both CIB1 and CIB2 in shRNA-transduced cell populations strongly impaired viral replication in target cells, recognizing these proteins as non-redundant HIV-1 helper factors. Also, a single-round cycle assay demonstrated that normal levels of CIB1 and CIB2 are required for HIV-1 envelope-mediated process. In fact, both X4 and R5 viral strains seem to be dependent on CIB1 and CIB2 expression levels, since infectivity of X4- and R5-enveloped particles was affected. Moreover, CIB1- and CIB2-knockdown populations displayed reduced viral fusion efficiency when compared to untransduced population, indicating virus-free entry impairment. Furthermore, virus-transfer through cell-cell contacts was decreased after co-culture of infected donor cells with CIB1- and CIB2-knockdown target cells. Flow cytometry showed that surface expression of some key players of both routes of viral entry was altered in CIBs-depleted populations, namely co-receptor CXCR4 and integrin α4β7. Taken together, these studies revealed for the first time that CIB1 is a HIV-1 helper factor along with CIB2, and suggest that both CIB1 and CIB2 facilitate HIV-1 entry in natural target cells possibly through modulation of CXCR4 and α4β7.

Biography

Ana Godinho-Santos has a degree in Microbiology and a Master's degree in Applied Microbiology. She is currently a Doctoral candidate at Faculty of Pharmacy - University of Lisbon, expected to complete the PhD degree requirements by March 2016.

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