INFECTIOUS AGENTS AND CANCER

MEETING ABSTRACTS Open Access

EBV-induced miR-34a functions to stimulate transformed B cell growth

Eleonora Forte[1], Christina Chang[1], Jason Tourigny[1], Eva Gottwein[1], Bryan Cullen[1], Sandeep Dave[2], Micah Luftig[1*]

From 12th International Conference on Malignancies in AIDS and Other Acquired Immunodeficiencies (ICMAOI)
Bethesda, MD, USA. 26-27 April, 2010

Background

Epstein-Barr virus (EBV) is a member of the γ-herpesvirus family estimated to infect 90% of the world's population. Despite the high prevalence of infection, EBV-associated malignancies are largely kept in check by a strong cytotoxic T cell immune response. However, EBV causes lymphoproliferative disease in immune-deficient individuals and plays a role in the pathogenesis of African Burkitt lymphoma, Hodgkin's disease, and nasopharyngeal carcinoma. In vitro, EBV infection of B cells results in proliferation and outgrowth of indefinitely proliferating lymphoblastoid cell lines (LCLs). Thus, LCLs represent a viable model for the pathogenesis of EBV-associated malignancies.

microRNAs are small noncoding RNAs that post-transcriptionally regulate gene expression to control a variety of processes including development, cell cycle, and immunity. Their role in EBV transformation and lymphomas is currently not well understood.

Results

Using a miRNA microarray, we identified a number of cellular miRNAs that were over- or under-expressed comparing resting CD19+ B cells to EBV-infected, proliferating B cells and immortalized LCLs. In particular, we focused on miR-34a, whose expression was induced by EBV. This miRNA has been previously reported to be a pro-apoptotic target of p53 implicated in the response to DNA damage. Surprisingly, contrary to its regulation in other cell types, miR-34a was not found to be p53 responsive in LCLs. In order to understand the functional role of this miRNA in EBV transformation, we constructed a miRNA sponge. miR-34a knockdown in LCLs showed that these cells depend on normal miR-34a expression to proliferate and to aggregate.

Conclusions

miR-34a is important for efficient growth and survival of EBV-transformed cells, in contrast to its tumor suppressive role in carcinoma and sarcoma-derived cell lines.

Acknowledgements

This article has been published as part of *Infectious Agents and Cancer* Volume 5 Supplement 1, 2010: Proceedings of the 12th International Conference on Malignancies in AIDS and Other Acquired Immunodeficiencies (ICMAOI). The full contents of the supplement are available online at http://www.biomedcentral.com/1750-9378/5?issue=S1.

Author details

[1]Department of Molecular Genetics and Microbiology, Center of Virology, Duke University School of Medicine, Durham, NC 27712, USA. [2]Duke Institute for Genome Sciences and Policy, Duke University, Durham, NC, USA.

Published: 11 October 2010

doi:10.1186/1750-9378-5-S1-A23
Cite this article as: Forte *et al.*: EBV-induced miR-34a functions to stimulate transformed B cell growth. *Infectious Agents and Cancer* 2010 5(Suppl 1):A23.

* Correspondence: micah.luftig@duke.edu
[1]Department of Molecular Genetics and Microbiology, Center of Virology, Duke University School of Medicine, Durham, NC 27712, USA
Full list of author information is available at the end of the article