World Journal of Virology

Online Submissions: http://www.wjgnet.com/esps/
wjv@wjgnet.com
doi:10.5501/wjv.v2.i2.16

World J Virol 2013 May 12; 2(2): 16-17
ISSN 2220-3249 (online)

EDITORIAL

Reprogramming the host: Modification of cell functions upon viral infection

Gualtiero Alvisi, Giorgio Palù

Gualtiero Alvisi, Giorgio Palù, Department of Molecular Medicine, Microbiology Section, 35121 Padua, Italy
Author contributions: Both the authors contributed to this paper.
Correspondence to: Gualtiero Alvisi, PhD, Assistant Professor, Department of Molecular Medicine, Microbiology Section, Via Gabelli 63, 35121 Padua, Italy. gualtiero.alvisi@unipd.it
Telephone: +39-49-8272353 Fax: +39-49-8272355
Received: April 22, 2013 Revised: April 27, 2013
Accepted: May 3, 2013
Published online: May 12, 2013

Abstract

Viruses and their hosts have co-evolved for million years. In order to successfully replicate their genome, viruses need to usurp the biosynthetic machinery of the host cell. Depending on the complexity and the nature of the genome, replication might involve or not a relatively large subset of viral products, in addition to a number of host cell factors, and take place in several subcellular compartments, including the nucleus, the cytoplasm, as well as virus-induced, rearranged membranes. Therefore viruses need to ensure the correct subcellular localization of their effectors and to be capable of disguising from the cellular defensive mechanisms. In addition, viruses are capable of exploiting host cell activities, by modulating their post-translational modification apparatus, resulting in profound modifications in the function of cellular and viral products. Not surprisingly infection of host cells by these parasites can lead to alterations of cellular differentiation and growing properties, with important pathogenic consequences. In the present hot topic highlight entitled "Reprogramming the host: modification of cell functions upon viral infection", a number of leading virologists and cell biologist thoroughly describe recent advances in our understanding of how viruses modulate cellular functions to achieve successful replication and propagation at the expenses of human cells.

Key words: Virus-host interaction; Pathogenesis; Post translational modification; Viral factories; Cancer; Differentiation; Human immunodeficiency virus; Hepatitis C virus; RNAi

Core tip: Viruses are obliged intracellular parasites causing million causalities every year. In order to successfully replicate their genome, viruses need to usurp the biosynthetic machinery of the host cell. Depending on the complexity and the nature of the genome, replication might involve or not a relatively large subset of viral products, in addition to a number of host cell factors, and take place in several subcellular compartments, including the nucleus, the cytoplasm, as well as virus-induced, rearranged membranes. We describe recent advances in our understanding of how viruses modulate cellular functions to successfully replicate at the expenses of human cells.

Alvisi G, Palù G. Reprogramming the host: Modification of cell functions upon viral infection. *World J Virol* 2013; 2(2): 16-17 Available from: URL: http://www.wjgnet.com/2220-3249/full/v2/i2/16.htm DOI: http://dx.doi.org/10.5501/wjv.v2.i2.16

Viruses and their hosts have co-evolved for million years. In order to successfully replicate their genome, viruses need to usurp the biosynthetic machinery of the host cell. Depending on the complexity and the nature of the genome, replication might involve or not a relatively large subset of viral products, in addition to a number of host cell factors, and take place in several subcellular compartments, including the nucleus, the cytoplasm, as well as virus-induced, rearranged membranes. Therefore viruses need to ensure the correct subcellular localization of their effectors and to be capable of disguising from the cellular defensive mechanisms. In this hot topic highlight entitled "Reprogramming the host: Modification of cell functions upon viral infection", we describe recent advances in our

understanding of how viruses modulate cellular functions to achieve successful replication and propagation at the expenses of human cells.

The first review of this issue, by Amberkar *et al*[1] "High-throughput RNA interference screens integrative analysis: Towards a comprehensive understanding of the virus-host interplay", based on bioinformatic and statistical approaches, explains how high throughput technologies can help unveiling the complex relationship between viruses and host cell proteins, which might represent targets for potential therapeutic intervention.

In "Architecture and biogenesis of plus-strand RNA virus replication factories", Paul *et al*[2] propose an innovative classification of positive strand RNA viruses according to the morphology of membrane rearrangements they are able to induce, and on which genome replication is believed to take place. The interplay of viral and cellular factors in the biogenesis of these replication factories is discussed.

The relationship between viruses and the host cell defensive system is the particular focus of the two following reviews "Innate host responses to West Nile virus: Implications for central nervous system immunopathology", by Rossini *et al*[3] and "Paramyxovirus evasion of innate immunity: Diverse strategies for common targets" by Audsley *et al*[4], which both provide a simultaneously accurate and concise summary of viral strategies to subvert the innate response at the molecular level, and the implication thereof in viral mediated pathogenesis.

In the following review "Viral proteins and Src family kinases: Mechanisms of pathogenicity from a 'liaison dangereuse'", Pagano *et al*[5], describe the mechanisms by which several viruses exploit protein-protein interactions to modulate the subcellular localization and enzymatic activity of these cellular enzymes, thus promoting their replication and regulating cell survival. Indeed, viruses are known to efficiently modulate the cell post translational machinery for their own benefit. Similarly, Mattoscio *et al*[6], in "Viral manipulation of cellular protein conjugation pathways: The SUMO lesson", review the relationship between the Small Ubiquitin like MOdifier apparatus and a number of DNA and RNA viruses.

"Effects of human immunodeficiency virus on the erythrocyte and megakaryocyte lineages" by Gibellini *et al*[7], deals with the ability of human immunodeficiency virus-1 infection to affect the differentiation potential of different cellular types, including osteoclast and vessel stem cells, and its implication in pathogenesis.

Finally, the hot topic highlight is closed by an intriguing hypothesis formulated by Avanzi *et al*[8], "How virus persistence can initiate the tumorigenesis process" describing in detail how infection-reinfection/reactivation cycles of viruses might contribute to the initiation of tumorigenesis.

REFERENCES

1 **Amberkar S**, Kiani NA, Bartenschlager R, Alvisi G, Kaderali L. High-throughput RNA interference screens integrative analysis: Towards a comprehensive understanding of the virus-host interplay. *World J Virol* 2013; **2**: 18-31 [DOI: 10.5501/wjv.v2.i2.18]

2 **Paul D**, Bartenschlager R. Architecture and biogenesis of plus-strand RNA virus replication factories. *World J Virol* 2013; **2**: 32-48 [DOI: 10.5501/wjv.v2.i2.32]

3 **Rossini G**, Landini MP, Gelsomino F, Sambri V, Varani S. Innate host responses to West Nile virus: Implications for central nervous system immunopathology. *World J Virol* 2013; **2**: 49-56 [DOI: 10.5501/wjv.v2.i2.49]

4 **Audsley MD**, Moseley GW. Paramyxovirus evasion of innate immunity: Diverse strategies for common targets. *World J Virol* 2013; **2**: 57-70 [DOI: 10.5501/wjv.v2.i2.57]

5 **Pagano MA**, Tibaldi E, Palù G, Brunati AM. Viral proteins and Src family kinases: Mechanisms of pathogenicity from a "liaison dangereuse". *World J Virol* 2013; **2**: 71-78 [DOI: 10.5501/wjv.v2.i2.71]

6 **Mattoscio D**, Segré CV, Chiocca S. Viral manipulation of cellular protein conjugation pathways: The SUMO lesson. *World J Virol* 2013; **2**: 79-90 [DOI: 10.5501/wjv.v2.i2.79]

7 **Gibellini D**, Clò A, Morini S, Miserocchi A, Ponti C, Carla Re M. Effects of human immunodeficiency virus on the erythrocyte and megakaryocyte lineages. *World J Virol* 2013; **2**: 91-101 [DOI: 10.5501/wjv.v2.i2.91]

8 **Avanzi S**, Alvisi G, Ripalti A. How virus persistence can initiate the tumorigenesis process. *World J Virol* 2013; **2**: 102-109 [DOI: 10.5501/wjv.v2.i2.102]

P- Reviewer Zhong ZH **S- Editor** Song XX
L- Editor A **E- Editor** Zheng XM