Virus Research 160 (2011) 1–14

Contents lists available at ScienceDirect

Virus Research

journal homepage: www.elsevier.com/locate/virusres

Review

Recombination-dependent concatemeric viral DNA replication

Ambra Lo Piano, María I. Martínez-Jiménez, Lisa Zecchi, Silvia Ayora*

Departamento de Biotecnología Microbiana, Centro Nacional de Biotecnología, CSIC, Campus de la Universidad Autónoma de Madrid, C/Darwin 3, Cantoblanco, 28049 Madrid, Spain

ARTICLE INFO

Article history:
Received 28 April 2011
Received in revised form 7 June 2011
Accepted 10 June 2011
Available online 17 June 2011

Keywords:
Break-induced replication
Primosome assembly
Replication fork reversal
Concatemeric DNA synthesis

ABSTRACT

The initiation of viral double stranded (ds) DNA replication involves proteins that recruit and load the replisome at the replication origin (*ori*). Any block in replication fork progression or a programmed barrier may act as a factor for *ori*-independent remodelling and assembly of a new replisome at the stalled fork. Then replication initiation becomes dependent on recombination proteins, a process called recombination-dependent replication (RDR). RDR, which is recognized as being important for replication restart and stability in all living organisms, plays an essential role in the replication cycle of many dsDNA viruses. The SPP1 virus, which infects *Bacillus subtilis* cells, serves as a paradigm to understand the links between replication and recombination in circular dsDNA viruses. SPP1-encoded initiator and replisome assembly proteins control the onset of viral replication and direct the recruitment of host-encoded replisomal components at viral *ori*L. SPP1 uses replication fork reactivation to switch from *ori*-dependent θ-type (circle-to-circle) replication to σ-type RDR. Replication fork arrest leads to a double strand break that is processed by viral-encoded factors to generate a D-loop into which a new replisome is assembled, leading to σ-type viral replication. SPP1 RDR proteins are compared with similar proteins encoded by other viruses and their possible *in vivo* roles are discussed.

© 2011 Elsevier B.V. All rights reserved.

Contents

1. Introduction 2
2. Viral recombination proteins 3
   2.1. Recombinase 3
      2.1.1. T4-UvsX 3
      2.1.2. T7-gp2.5 3
      2.1.3. RecT-like recombinases (RecT, Redβ, G35P) 3
      2.1.4. HSV-1-ICP8 and baculovirus LEF-3 4
      2.1.5. Vaccinia-E9L 4
   2.2. Recombinase mediators 4
   2.3. Exonuclease 4
      2.3.1. T4-gp46/gp47 4
      2.3.2. T7-gp6 exonuclease 5
      2.3.3. RecE and related exonucleases 5
      2.3.4. HSV-1-UL12 5
   2.4. SSB 5
      2.4.1. T4-gp32 5
      2.4.2. SPP1-G36P 5
      2.4.3. Vaccinia-I3L 6

Abbreviations: DA, DNA arrested; DSB, Double Strand Break; dsDNA, double-stranded DNA; HJ, Holliday junction; HSV-1, Herpes-simplex virus type 1; MRN, Mre11–Rad50–Nbs1 complex; *ori*, origin of replication; OB-fold, oligosaccharide/oligonucleotide binding fold; *pac*, packaging site; PAI, Pathogenicity Island; RDR, recombination dependent replication; SaPI, *Staphylococcus aureus* pathogenicity island; SSA, single-strand annealing; SSB, single-stranded DNA binding protein; ssDNA, single-stranded DNA; TR, terminal repeat; VLF-1, baculovirus very late expression factor 1.

* Corresponding author. Tel.: +34 91585 5450; fax: +34 91585 4506.
E-mail address: sayora@cnb.csic.es (S. Ayora).

0168-1702/$ – see front matter © 2011 Elsevier B.V. All rights reserved.
doi:10.1016/j.virusres.2011.06.009

2.5. HJ-resolving enzymes 6
2.5.1. T4-Endonuclease VII 6
2.5.2. T7-Endonuclease I 6
2.5.3. Lambdoid Rap/RusA endonucleases 6
2.5.4. Vaccinia virus HJ resolvase 6
2.6. DNA helicases 7
3. Biological significance of RDR 7
3.1. Replisome assembly via RDR 7
3.2. RDR of viruses with linear genomes 8
3.3. RDR of viruses with circular genomes 8
3.3.1. Shift from θ to σ by replication re-start in phage SPP1 9
3.3.2. Shift in phage λ as a prototype of Proteobacteria infecting viruses 9
3.3.3. RDR in HSV-1 10
3.4. Formation of transducing plasmid molecules 11
3.5. Amplification of pathogenicity islands 11
4. Conclusions 11
Acknowledgements 11
References 11

1. Introduction

DNA replication is established from a single origin in bacterial genomes and in many double-stranded (ds) DNA viruses with circular genomes, but from multiple origins in linear chromosomes of some viruses, or in eukaryotic and archaeal cells. In bacteria multi-level control mechanisms ensure the assembly of a replication fork at the origin region, only once per cell cycle, and such a control does not exist in lytic viruses (Zakrzewska-Czerwinska et al., 2007; Kaguni, 2006; Weigel and Seitz, 2006). Initiation of *ori*-dependent θ (circle-to-circle) DNA replication has been extensively studied in γ-Proteobacteria, with *Escherichia coli* and its viruses as model systems (Kaguni, 2006; Kornberg and Baker, 1992; Weigel and Seitz, 2006). It has been shown that the key steps identified are common in many bacteria and phages, although with different number of players. Initiation of DNA replication in Firmicutes (*e.g.*, *Bacillus subtilis*) and their viruses (*e.g.*, virus SPP1) requires first the wrapping of the origin region (*oriC* in bacteria or *ori*L in SPP1) by the origin recognition protein (*e.g.*, DnaA or G*38*P) followed by localized unwinding of the adjacent AT-rich region, where the replicative hexameric DNA helicase (*e.g.*, DnaC in *B. subtilis* or G*40*P in SPP1) bound to its loader (*e.g.*, DnaD-DnaB and DnaI in *B. subtilis* or G*39*P in SPP1) is delivered (Ayora et al., 1999; Zakrzewska-Czerwinska et al., 2007). Then the activated helicase coordinates all events at the fork. The viral hexameric replicative DNA helicase G*40*P directly loads the host encoded DnaG primase (Ayora et al., 1998; Barcena et al., 1998; Lecointe et al., 2007), and upon interaction with the τ subunit of the clamp loader it loads the polymerase (Martínez-Jiménez et al., 2002). This holoenzyme [PolCDnaE(β_2)($\tau_4\delta\delta'$)] is composed by two polymerases [PolC and DnaE, (Dervyn et al., 2001)], the sliding clamp (β_2), and the clamp loader complex [including τ, δ, and δ′ subunits; Sanders et al., 2010]. Bacterial and SPP1 *ori*-dependent replication proceed by the θ (circle-to-circle) mechanism. In general, these steps of replication initiation are conserved in other circular viruses that replicate by a θ mechanism, just the number of virus-encoded functions may vary. As an example, the genome of herpes simplex virus type 1 (HSV-1) encodes seven essential replication proteins (reviewed by Muylaert et al., 2011). These include an *ori* binding protein (UL9), and six core replication proteins including a DNA polymerase composed by two subunits (UL30 and UL42), ICP8, a single-stranded DNA binding protein (SSB), and a helicase/primase complex composed of three subunits (the helicase UL5, the UL52 primase and the accessory protein UL8). The UL8 accessory protein interacts with the UL5/52 complex, and also with UL9, ICP8 and DNA polymerase (Muylaert et al., 2011). UL9 binds to the *ori*, distorts it, and recruits additional factors, as ICP8 to assemble the replisome. Subsequently, UL9, through its interaction with the UL8 loading protein may recruit the viral helicase–primase to initiate primer synthesis and to establish a bi-directional replication fork. The viral DNA polymerase may be recruited to the site of initiation either by the interaction of its catalytic subunit (UL30) with UL8, or via an interaction between its processivity factor (UL42) and UL9 (reviewed in Boehmer and Nimonkar, 2003).

Initiation of replication of dsDNA viruses with linear genomes does not completely follow these general rules. In phage T4, no origin binding protein exists. In addition, T4 contains several replication origins (Brister and Nossal, 2007). Though few obvious sequence characteristics are shared between them, all of the T4 origins are thought to facilitate formation of RNA primers used to initiate leading strand DNA synthesis. The presence of an R-loop presumably holds the origin duplex in an open conformation, giving the T4 gp41/61 primosome complex access to allow extensive parental DNA unwinding and priming (reviewed in Kreuzer and Brister, 2010). Several viral proteins, in addition to the helicase/primase are required for significant replication of these R-loop substrates: DNA polymerase (gp43), polymerase clamp (gp45), clamp loader (gp44/62), and SSB (gp32). While the gp41 helicase can be loaded onto DNA without the helicase loader (gp59), the presence of gp59 greatly accelerates the process. The phage SSB, gp32, seems to help also in the recruitment of the gp41 helicase, but also recruits, by direct protein–protein interaction another helicase, Dda (Ma et al., 2004). The Dda helicase, seems to be also involved in initiation of replication, although it is not an essential component (Jongeneel et al., 1984).

During the replication process there is a trade-off between transcription, replication, and other DNA transactions that could lead to the stall or collapse of the replication machinery. In bacteria, re-start of replication from a fortuitous or programmed replication fork barrier might simply involve resumption of DNA synthesis by a PriA-mediated replisome reassembly system without necessarily invoking a recombination event and a switch in the replication mode (reviewed in Cox et al., 2000; Gabbai and Marians, 2010; Heller and Marians, 2006; Kreuzer, 2000; Michel et al., 2004). In viruses, recombination-dependent reassembly of the replisome is needed to resuscitate a replication fork with a switch from θ to RDR in circular genomes, which is visualized as a Greek "σ" letter by electron microscopy, or from early to late replication in linear viruses. Both, σ type or late viral replication, proceed via RDR. This replication mode generates the DNA substrate (concatemeric viral DNA) to be encapsidated into an empty prohead to generate a mature viral particle. Functions usually involved in

Download English Version:

https://daneshyari.com/en/article/6143338

Download Persian Version:

https://daneshyari.com/article/6143338

Daneshyari.com