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Intracellular dynamics of actin affects Borna disease virus replication in the nucleus

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ABSTRACT

Borna disease virus (BoDV) is a nonsegmented, negative-strand RNA virus that uniquely replicates and establishes persistent infection in cell nucleus. Recent studies have demonstrated the presence of actin in the nucleus and its role in intranuclear phenomena such as transcription and DNA repair. Although nuclear actin is involved in the life cycle of some intranuclear DNA viruses, the interaction between BoDV and nuclear actin has not been reported. In this study, we show that the inhibition of the nucleocytoplasmic transport of actin affects the replication of BoDV in the nucleus. The knockdown of a nuclear export factor of actin, exportin 6, results in the induction of structural aberration in intranuclear viral factories of BoDV. Furthermore, the inhibition of the nuclear export of actin promotes accumulation of viral matrix protein in the cytoplasm and periphery of the infected cells. These results suggest that the dynamics of actin affect the replication of BoDV by disturbing the structure of viral factories in the nucleus.

1. Introduction

Actin is a cytoskeletal protein that is mostly localized in the cytoplasm and forms microfilaments. It is involved in the maintenance of various cell dynamics such as cellular structure, cell movement, and intracellular transport (Lee and Dominguez, 2010). Recent studies have demonstrated that actin is also localized in the nucleus and plays fundamental functions related to nuclear dynamics of the cells. For instance, actin is present in a chromatin remodeling complex termed as the BAF complex and is required for its ATPase activity (Zhao et al., 1998). Actin participates in the transcription machineries of RNA polymerase I, II, and III, resulting in the regulation of gene expression (Hofmann et al., 2004; Hu et al., 2004; Philimonenko et al., 2004). Furthermore, the association of actin with several heterogeneous nuclear ribonucleoproteins (hnRNPs) has been demonstrated (Percipalle et al., 2002, 2001) and the involvement of nuclear actin in DNA repair was recently studied. Caridi et al. (2018) revealed that nuclear actin filaments and myosin induce directed motion of heterochromatic repair sites to nuclear periphery (Caridi et al., 2018). It was shown that the mobility of the sites of DNA double-strand breaks directed by nuclear actin and its nucleators is essential for homology-directed repair (Schrank et al., 2018).

Recent studies have suggested the association between nuclear actin and several DNA viruses replicating in the nucleus. It was demonstrated that pseudorabies virus and herpes simplex virus type 1 infections induce the formation of nuclear actin filaments and that the viral capsid protein VP26 co-localized with the actin motor protein, myosin V (Feierbach et al., 2006). Furthermore, nuclear F-actin is shown to promote the movement of human cytomegalovirus to the nuclear periphery and facilitated its nuclear egress (Wilkie et al., 2016). In human adenovirus type 5 (Ad5) infection, nuclear actin re-localizes to the viral transcription sites during an intermediate stage of viral replication (Fuchsova et al., 2015). These observations suggest that DNA viruses replicating in the nucleus rely on nuclear actin at several stages of their

Abbreviations: BoDV, Borna disease virus; RNP, ribonucleoprotein; IPO9, importin 9; XPO6, exportin 6; vSPOT, viral speckle of transcript; Ad5, adenovirus type 5; siRNA, small-interfering RNA

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life cycles.

Borna disease virus (BoDV) is a nonsegmented, negative-strand RNA virus that belongs to the family *Bornaviridae* (Tomonaga et al., 2002). Among the six proteins coded by its 8.9 kb genome, nucleoprotein (N), phosphoprotein (P), and RNA-dependent RNA polymerase (L) comprise an RNP along with its genomic RNA. Matrix protein (M) and glycoprotein (G) are the viral structural proteins. Although most RNA viruses replicate in the cytoplasm, BoDV replicates in the nucleus (Briese et al., 1992). We have previously shown that BoDV RNP forms cage-like membrane-less viral factories in the nucleus, termed as viral speckle of transcripts (vSPOTs) that are tethered to the host chromatin throughout the cell cycle to maintain persistent infection (Hirai et al., 2016; Matsumoto et al., 2012). As the function of nuclear actin is related to chromatin remodeling, it is possible that the dynamics of nuclear actin may radically influence the intranuclear life cycle of BoDV. In the present study, we show that nuclear transport factors of actin affect the replication of BoDV. We found that the knockdown of a nuclear export factor of actin, exportin 6 (XPO6), causes structural aberration in vSPOTs, resulting in the upregulation in BoDV replication in the nucleus and accumulation of vial matrix protein in the cytoplasm. These results suggest that the dynamics of actin may affect the replication of BoDV through the disturbance in the structure of intranuclear viral factories.

2. Materials and methods

2.1. Cell lines and viruses

The uninfected OL cells and the BoDV-infected OL cells, the OL cells that were persistently infected with BoDV strain huP2br (Nakamura et al., 2000), were cultured in Dulbecco's modified Eagle's medium (DMEM; Thermo Fisher Scientific) supplemented with 5% fetal bovine serum (FBS). Vero cells were cultured in DMEM supplemented with 2% FBS.

2.2. Antibodies, DNA constructs and siRNAs

Anti-N, anti-P, and anti-M antibodies were used as previously described (Hirai et al., 2016). Anti-IPO9 antibody was purchased from Abcam (ab124710) and anti-XPO6, from Proteintech (11408-1-AP). Anti-alpha-tubulin antibody was obtained from Sigma (T5168). A cDNA fragment encoding β-actin was amplified by PCR and cloned into the pEGFP-C1 vector (Clontech). siRNAs targeting IPO9 (SI00448217) and XPO6 (SI00764099) and control siRNA (SI03650318) were supplied by Qiagen. The cells were transfected with siRNAs or co-transfected with siRNAs and the plasmid DNA using Lipofectamine 2000 (Thermo Fisher Scientific).

2.3. Immunofluorescence staining and microscopy

Cells cultured on coverslips were fixed with 4% paraformaldehyde for 10 min, blocked with 10% normal goat serum containing 0.5% Triton X-100 for 15 min, and probed with anti-N, anti-P, and anti-M antibodies for 2 h at room temperature. The cells were washed twice with phosphate-buffered saline (PBS) and incubated with secondary antibodies (Alexa Fluor® 488-conjugated goat anti-rabbit IgG and Alexa Fluor® 568-conjugated goat anti-mouse IgG) and 4′,6′-diamidino-2-phenylindole (DAPI) for 1 h at room temperature. The cells were washed thrice with PBS and mounted in ProLong™ Gold Antifade Mountant or ProLong™ Diamond Antifade Mountant (Thermo Fisher Scientific). Confocal microscopy was performed using a C1 confocal laser-scanning microscope (Nikon) with a CFI Apo Lambda S 60× objective lens (N.A. = 1.4) or a LSM 700 laser scanning confocal microscope (Carl Zeiss) with a Plan-Apochromat 63× objective lens (N.A. = 1.4). Image analyses were performed using ImageJ.

2.4. RNA preparation and quantitative PCR (qPCR)

Total RNA was isolated from BoDV-infected OL cells using TRIzol reagent (Thermo Fisher Scientific) according to the manufacturer's protocol. Reverse transcription was performed with Verso cDNA Synthesis Kit (Thermo Fisher Scientific) using oligo-dT primers or BoDV genome-specific primer (5′-TGTTGCGCTAACAACAAACCAATCAC-3′). qPCR was performed with SYBR-Green PCR Assay (Toyobo) in a 20 µl reaction volume, and products were detected with a Rotor-Gene Q System (Qiagen). PCR reactions were incubated at 95 °C for 30 s, followed by 40 amplification cycles of denaturation at 95 °C for 5 s and annealing and extension at 60 °C for 30 s. BoDV RNA was quantified with primers (5′-ATGCATTGACCCAACCGGTA-3′ and 5′-ATCATTCGAT AGCTGCTCCCTTC-3′). Glyceraldehyde-3-phosphate dehydrogenase (*GAPDH*) mRNA was quantified with primers (5′-AGCGAGATCCCTCC AAAATC-3′ and 5′-AAATGAGCCCCAGCCTTCTC-3′) to normalize the total amount of cDNA. qPCR data were analyzed with Rotor-Gene Q software (Qiagen, version 2.1).

2.5. BoDV titration

Vero cells were inoculated with 100 µl of the supernatants of BoDV-infected OL cells in 24-well plate. After incubating the cells for 1 h at 37 °C, the cells were washed with the fresh medium and cultured for 5 days. The cells were subjected to immunofluorescence staining using an anti-BoDV N antibody and the focus forming units (FFU)/100 µl was counted.

3. Results

3.1. Intranuclear level of actin affects the replication of BoDV

Actin is actively imported to and exported from the nucleus by the actin-specific nuclear import and export factors, importin 9 (IPO9) and XPO6, respectively, both of which belong to a karyopherin beta superfamily (Dopie et al., 2012; Stüven et al., 2003). To understand the involvement of nuclear actin in BoDV replication, we knocked down IPO9 and XPO6 using small-interfering RNAs (siRNAs), because karyopherin beta superfamily proteins, IPO9 and XPO6, regulate direct nucleocytoplasmic shuttling of actin through a nuclear pore complex. The knockdown of IPO9 and XPO6 resulted in the cytoplasmic and nuclear accumulation of actin, respectively (Dopie et al., 2012) (Fig. 1A and B). As shown in Fig. 1B, the prominent filaments of GFP-actin were observed in the basal plane of the cells in the IPO9 knockdown condition (Fig. 1B, arrowheads), and the nuclear accumulation of GFP-actin was observed in the middle plane of the cells in the XPO6 knockdown condition (Fig. 1B, arrow). To examine BoDV replication in the nucleus, we measured the levels of BoDV mRNA and genomic RNA with real-time quantitative polymerase chain reaction (RT-qPCR). As shown in Fig. 1C, IPO9 knockdown decreased the levels of mRNA of BoDV, although the genomic RNA was not significantly decreased by IPO9 knockdown (Fig. 1C and D). On the contrary, the levels of both BoDV mRNA and genomic RNAs were significantly upregulated following treatment with XPO6 siRNA (Fig. 1C and D). Fluorescence in situ hybridization analysis also suggested that BoDV replication was upregulated by XPO6 knockdown (Supplementary Fig. S1). These results suggest that intranuclear level of actin affects the replication of BoDV in the nucleus.

3.2. XPO6 knockdown induces structural aberration in vSPOTs and cytoplasmic accumulation of BoDV proteins

BoDV forms intranuclear replication factories, vSPOTs, that use the host chromatin as a structural scaffold in the nucleus (Matsumoto et al., 2012). To examine whether the disruption of the nuclear transport of actin influences the structure of vSPOTs, we visualized vSPOTs using

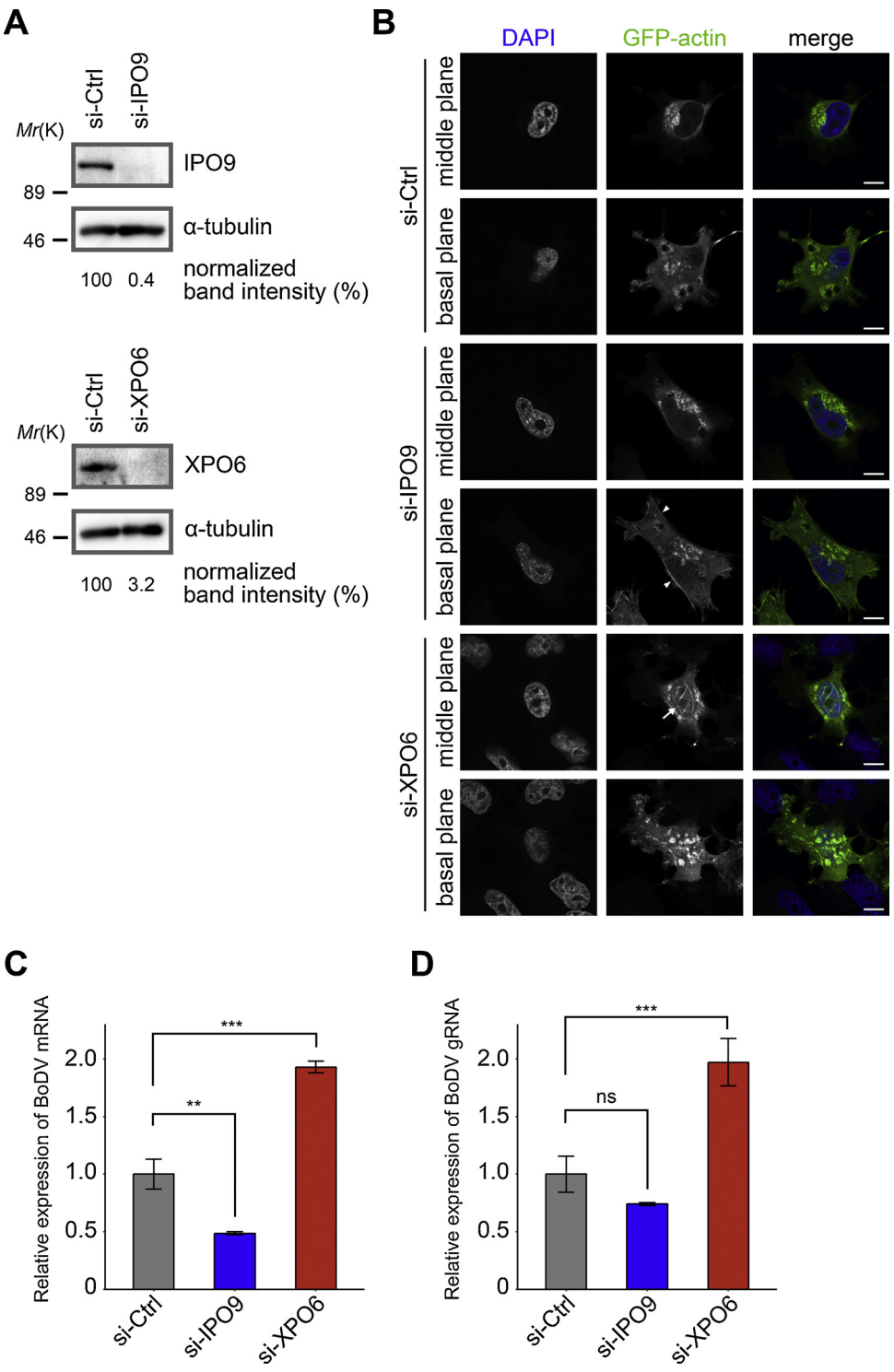

Fig. 1. Changes in the transcripts and replicates of BoDV after IPO9 and XPO6 knockdown.

(A) BoDV-infected cells were transfected with siRNAs against IPO9 and XPO6. The whole cell lysates of the transfected cells were subjected to SDS-PAGE, followed by western blotting. The band intensities of IPO9 (top) and XPO6 (bottom) were normalized by the band intensity of α-tubulin and are presented as the ratio to the si-Ctrl (si-Ctrl = 100). (B) Uninfected cells were co-transfected with the plasmid DNA encoding GFP-actin and siRNAs against IPO9 and XPO6. The transfected cells were subjected to confocal microscopy. Bars: 10 μm. (C and D) The relative expression levels of BoDV mRNA (C) and genomic RNA (gRNA) (D) after IPO9 or XPO6 knockdown were monitored by qPCR and are presented as the ratio to the si-Ctrl (si-Ctrl = 1.0). Bars represent mean ± SEM (n = 3). Statistical significances were calculated with one-way ANOVA, followed by Tukey's honestly significant difference (HSD) post-hoc test. **P < 0.01, ***P < 0.001, ns: not significant.

anti-N and anti-P antibodies in IPO9 and XPO6 knockdown cells. While the knockdown of IPO9 had no apparent influence on the structure of vSPOTs in the infected nuclei, the treatment of cells with XPO6 siRNA resulted in structural aberration in vSPOTs (Fig. 2A, arrows). The measurement of the size and circularity of speckles revealed that the knockdown significantly changed the size and shape of vSPOTs in the nucleus (Fig. 2B and C).

XPO6 knockdown seemed to be enhanced the fluorescence intensities of viral N and P proteins in the cytoplasm, as evident in immunofluorescence assay (Fig. 2A). Furthermore, the staining intensity for M protein also increased in the cytoplasm of XPO6 knockdown cells. The accumulation of M protein around the cell surface or the border of the cell was clearly observed (Fig. 2D), suggesting that enhanced transcription of BoDV by XPO6 knockdown induces increased expression of the viral proteins in the cells. Considering no significant difference in the infectious titers between the supernatants of XPO6 knockdown and control cells (Fig. 2E), the knockdown cells resulted in the accumulation of the proteins without enhancing the viral particle production from cell.

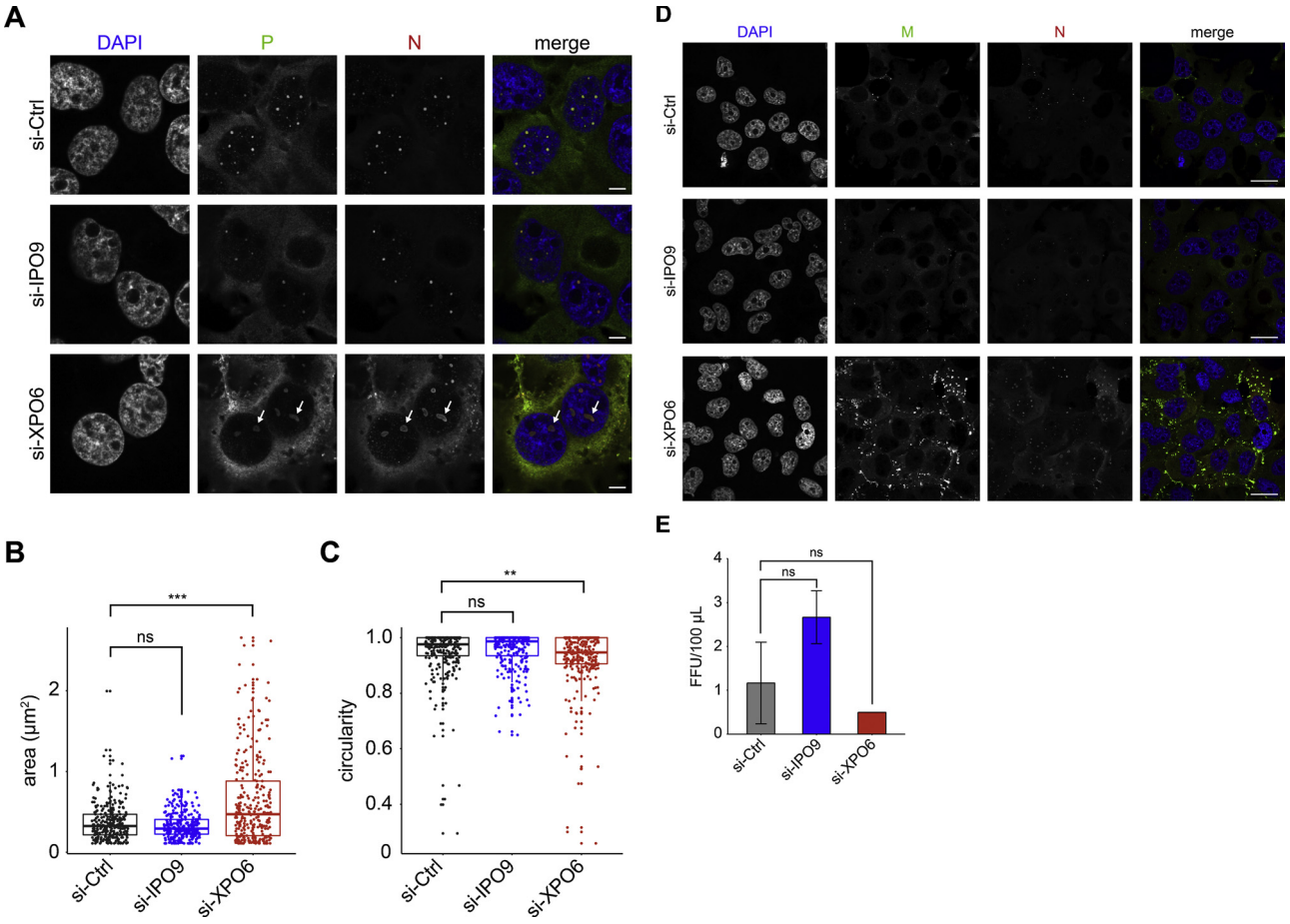

Fig. 2. Structural changes in vSPOTs after IPO9 and XPO6 knockdown.
BoDV-infected cells were transfected with siRNAs against IPO9 and XPO6. After knockdown, the cells were subjected to immunofluorescence staining using anti-N and anti-P antibodies, followed by confocal microscopy. Bars: 5 μm. (B and C) Box plots show the area (B) and circularity (C) of each vSPOT. n = 266–282 from six independent experiments; one-way ANOVA, followed by Tukey's HSD post-hoc test. **P < 0.01, ***P < 0.001, ns: not significant. (D) BoDV-infected cells were transfected with siRNAs against IPO9 and XPO6. After knockdown, the cells were subjected to immunofluorescence staining using anti-N and anti-M antibodies, followed by confocal microscopy. The intensities of the images were best-fitted to the si-Ctrl panels, resulting in the saturated images in the si-XPO6 panels. Bars: 20 μm. (E) The BoDV titers in the cell culture supernatants of BoDV-infected cells transfected with siRNAs were measured. The FFU is the duplicate in each knockdown condition. Bars represent mean ± SEM (n = 3). Statistical significances were calculated with one-way ANOVA, followed by Tukey's HSD post-hoc test. ns: not significant.

4. Discussion

In the present study, we demonstrate that the knockdown of the nuclear transport factors of actin affects the transcription and replication of BoDV in the nucleus (Fig. 1 and Supplementary Fig. S1). Interestingly, the knockdown of IPO9 and XPO6 resulted in opposite effects on BoDV replication; while IPO9 knockdown decreased BoDV transcription, inhibition of XPO6 expression resulted in an increase in viral transcription and replication. The effect of IPO9 siRNA on BoDV replication was not significant, suggesting that nuclear actin is involved in BoDV RNA synthesis.

Fuchsova et al. (2015) revealed the recruitment of actin to the transcription site of Ad5 and cellular transcription sites. These authors found that the treatment of cells with an actin polymerization inhibitor and overexpression of the monomeric form of nuclear actin resulted in the inhibition of Ad5 proliferation (Fuchsova et al., 2015). Together with our observation, these results suggest the involvement of nuclear actin in viral replication in the nucleus.

In the present study, we were unable to investigate the detailed relationship between BoDV replication and nuclear actin. One possible mechanism underlying the effects of actin on the transcription and replication of BoDV is through the influence on the host chromatin environment. It has been shown that nuclear actin is involved in chromatin-based events such as chromatin remodeling and transcription through cellular RNA polymerases; actin is present in the INO80 chromatin remodeling complex and regulates its chromatin remodeling activity (Kapoor et al., 2013). Furthermore, actin is known to associate with rDNA and is essential for RNA polymerase I-mediated transcription (Philimonenko et al., 2004). β-Actin was shown to interact with RNA polymerase II and is known to be important for its transcription activity (Hofmann et al., 2004). Moreover, β-actin distributes at an active U6 promoter site in association with RNA polymerase III and is required for its transcription (Hu et al., 2004). These facts suggest that a quantitative change in actin level in the nucleus may influence the chromatin structure. In previous studies, we have reported that BoDV RNP forms vSPOTs using chromatin as a structural scaffold and starts replication in speckles (Matsumoto et al., 2012). BoDV RNP associates with the host chromatin, and the knockdown of a chromatin-binding protein, high mobility group box 1 (HMGB1), was shown to reduce the transcription and replication of BoDV (Matsumoto et al., 2012). Thus, it is likely that the change in the chromatin structure induced by XPO6 knockdown may cause instability in vSPOTs, consequently leading to abnormalities of viral replication. We have shown that the structure of vSPOTs becomes distorted after XPO6 knockdown (Fig. 2). We could not report any apparent change in vSPOT structure in response to IPO9 knockdown, although IPO9 siRNA decreased the RNA synthesis of

BoDV. This observation suggests that the structure of vSPOTs may be affected after the relaxation of the chromatin structure through the accumulation of actin in the nucleus. It is possible that actin may affect viral replication independent of chromatin. Considering that XPO6 specifically exports actin-profilin complexes from the nucleus (Stüven et al., 2003), it may be suggested that actin-profilin complexes directly stimulate the transcription of BoDV in the nucleus. Further studies are warranted to clarify the direct and indirect role of nuclear actin on the transcription of BoDV.

We found that viral proteins were accumulated in the cytoplasm after XPO6 knockdown (Fig. 2) probably because these proteins were overproduced through an increase in the viral transcription in the knockdown cells. Despite the accumulation of viral proteins, no increase in the release of infectious virion was observed in the knockdown cells; this observation suggests the disruption of the intracellular transport or release pathway involved in viral particle production by XPO6 siRNA in the cytoplasm.

The present study demonstrates the relationship between intracellular dynamics of actin and replication of a highly nucleus-associated RNA virus, BoDV. Although the exact mechanism of action of actin on BoDV replication is unknown, the interaction between nuclear actin and host chromatin suggests the influence of actin on the formation of virus factories in the nucleus. Further studies are necessary to reveal the correlation between role of nuclear actin and BoDV replication.

Disclosure

The authors declare no conflict of interest.

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Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:https://doi.org/10.1016/j.virusres.2019.02.004.

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