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# Interactions Between African Swine Fever Virus and Host Cells: Mechanisms and Outcomes

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**Abstract** This study explores the complex interactions between ASFV and host cells, with a focus on the mechanisms of virus entry and replication, as well as the cellular and systemic outcomes of infection. It emphasizes how ASFV utilizes host cell mechanisms to manipulate immune responses and induce cytopathic effects, ultimately leading to disease progression. Future research priorities, including technological advancements and global management strategies, are discussed, emphasizing the importance of comprehensive approaches to mitigate the impact of African swine fever. A case study of a regional epidemic provides insights into host response and broader impacts on pig health, emphasizing the challenges of current control measures and the necessity of innovative vaccines and treatment strategies. This review aims to deepen our understanding of the biology of African swine fever virus and provide information for developing effective control and prevention measures.

**Keywords** African swine fever virus (ASFV); Viral-host interactions; Immune evasion; Cytopathic effects; Disease control strategies

## 1 Introduction

African swine fever virus (ASFV) is a highly contagious and lethal virus affecting domestic pigs and wild boars, causing a disease known as African swine fever (ASF) (Alonso et al., 2012; Zhang et al., 2021). ASFV is a member of the Asfarviridae family and is characterized by its large, double-stranded DNA genome, which encodes a complex array of structural and nonstructural proteins (Galindo et al., 2015). The virus primarily targets macrophages, utilizing mechanisms such as clathrin-mediated endocytosis for entry into host cells (Chen et al., 2023). ASFV's ability to evade host immune responses and its high mortality rate, often reaching 100%, make it a significant threat to the global swine industry (Zhu et al., 2019).

Research on ASFV has been intensifying due to its severe impact on the swine industry and the lack of effective vaccines or antiviral treatments (Dixon et al., 2019). Recent studies have focused on understanding the virus-host interactions, particularly the protein-protein interactions (PPIs) that facilitate viral entry, replication, and immune evasion (Yang et al., 2021b; Dolata et al., 2023). Investigations have revealed that ASFV manipulates host cellular pathways, such as endocytosis and immune signaling, to establish infection and evade immune responses (Guo et al., 2021). Despite these advances, the molecular mechanisms underlying ASFV pathogenesis and immune evasion remain incompletely understood, highlighting the need for further research (Chen et al., 2022).

This study provides a comprehensive overview of the interactions between ASFV and host cells, with a focus on the mechanisms and outcomes of these interactions. By synthesizing current research results, it elucidates the molecular pathways utilized by ASFV during infection and identifies potential targets for therapeutic interventions, covering the entry mechanisms of the virus, manipulation of host cell processes, and their impact on cell survival and immune response. This study aims to contribute to the development of effective vaccines and antiviral strategies against ASFV.

## 2 Mechanisms of ASFV Entry into Host Cells

### 2.1 Viral attachment to host receptors

African swine fever virus (ASFV) initiates infection by attaching to host cell receptors. The main capsid protein, p72, plays a crucial role in this process by binding to the host protein CD1d, which facilitates ASFV entry into

host cells via clathrin-mediated endocytosis (Figure 1) (Chen et al., 2023). This interaction is essential for the virus to attach and subsequently internalize into the host cell. The specific receptors and attachment factors involved in ASFV entry are still being studied, but CD1d has been identified as a significant host-entry factor.

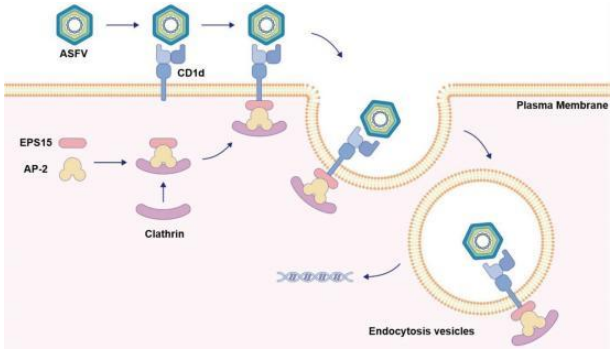


Figure 1 Schematic illustration of CD1d in enhancing ASFV endocytosis as a host factor (Adopted from Chen et al., 2023)
Image caption: ASFV virions enter host cells via host factor CD1d on the cell membrane. The p72 on the surface of the ASFV virion binds with CD1d on the cell membrane. The signal is then transmitted to EPS15 in the cytoplasm to form the p72-CD1d-EPS15 axis, where CD1d recruits EPS15 through the interaction between CD1d and EPS15 intracellular domains. The process promotes the formation of EPS15-AP-2-clathrin complexes and aggregation, resulting in ASFV virions endocytosis during ASFV infection (Adopted from Chen et al., 2023)

### 2.2 Internalization and endosomal trafficking

Once attached, ASFV enters host cells primarily through clathrin-mediated endocytosis, a process that is dynamin-dependent and requires cellular cholesterol (Galindo et al., 2015). ASFV can also utilize macropinocytosis, a form of endocytosis characterized by actin reorganization and membrane ruffling, to enter host cells (Sánchez et al., 2012). After internalization, ASFV traffics through the endolysosomal system, where it undergoes disassembly in acidic late endosomes, a process crucial for successful infection. The virus's internalization and trafficking are tightly regulated by host cell factors, including small GTPases and phosphoinositide signaling.

### 2.3 Escape to the cytoplasm

The escape of ASFV to the cytoplasm involves the fusion of the viral membrane with endosomal membranes, a process facilitated by viral proteins such as pE199L. This protein is necessary for the fusion event that allows the viral core to penetrate the host cell cytoplasm, enabling the release of the viral genome. The uncoating process involves the loss of outer capsid layers and the fusion of the inner viral membrane with endosomes, which is essential for the virus to establish infection within the host cell (Sánchez et al., 2017).

## 3 ASFV-Host Interactions During Replication

### 3.1 Hijacking host transcriptional machinery

African swine fever virus (ASFV) employs sophisticated mechanisms to manipulate host transcriptional machinery to favor its replication. ASFV regulates the activation of several transcription factors, thereby controlling the expression of specific target genes during infection (Sánchez et al., 2013). This regulation includes the compartmentalization of viral mRNA and ribosomes with cellular translation factors within the virus factory, which facilitates the preferential expression of viral genes over host genes. Additionally, ASFV infection leads to the dysregulation of host metabolism, which further promotes viral replication at the transcriptional level (Ju et al., 2021).

### 3.2 Manipulation of host immune responses

ASFV has evolved multiple strategies to evade and manipulate host immune responses, ensuring its survival and replication. The virus interacts with host innate immune factors, inhibiting and inducing various signaling pathways such as cGAS-STING, NF-κB, and TGF-β (Figure 2) (Afe et al., 2023). ASFV also downregulates host immune-related genes and microRNAs that target viral genes, thereby suppressing the host's antiviral response.

Furthermore, ASFV encodes several apoptosis inhibitors, which help in evading host defenses and prolonging the survival of infected cells (Dixon et al., 2019). The virus's ability to modulate immune responses is crucial for its persistence in natural hosts and domestic pigs (Netherton et al., 2019).

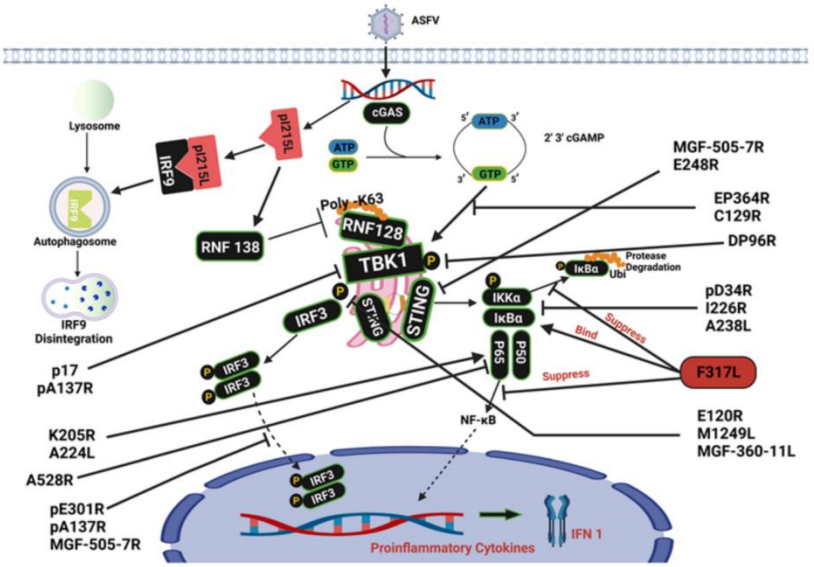


Figure 2 ASFV proteins modulating the cGAS/STING and NF-κB signaling pathway (Adopted from Afe et al., 2023)

### 3.3 Resource reallocation for viral assembly

ASFV reprograms host cellular machinery to allocate resources for viral assembly. The virus infects macrophages, utilizing clathrin-and cholesterol-dependent endocytosis for entry, which is essential for a productive infection (Galindo et al., 2015). ASFV also influences the host's cellular machinery of protein synthesis, ensuring that viral proteins are synthesized efficiently. This reallocation of resources is critical for the assembly and maturation of new viral particles, facilitating the spread of the virus within the host (Dolata et al., 2023).

## 4 Outcomes of ASFV Infection

### 4.1 Cytopathic effects on host cells

African swine fever virus (ASFV) primarily targets porcine macrophages, leading to significant cytopathic effects. The virus enters these cells through clathrin- and cholesterol-dependent endocytosis, which is crucial for successful viral replication (Galindo et al., 2015). ASFV infection results in the modulation of host cell gene expression, including the upregulation of pro-inflammatory cytokines and the downregulation of anti-inflammatory cytokines, contributing to excessive tissue inflammation and apoptosis (Yang et al., 2021a). The virus also affects cellular homeostasis by interacting with host proteins, which can lead to cell death and tissue damage (Guo et al., 2021).

### 4.2 Immune system evasion and suppression

ASFV has developed sophisticated mechanisms to evade and suppress the host immune system. It inhibits MHC Class II antigen processing and presentation, thereby avoiding detection by CD8+T effector cells (Zhu et al., 2019). The virus also suppresses macrophage activation and induces immune-suppressive cytokines, which help it evade both innate and adaptive immune responses (Afe et al., 2023). ASFV encodes several apoptosis inhibitors, which prevent the host from effectively clearing the infection (Dixon et al., 2019). Additionally, ASFV can modulate host immune signaling pathways, such as cGAS-STING and NF-κB, to further suppress antiviral responses.

### 4.3 Impacts on swine physiology and disease progression

ASFV infection in domestic pigs leads to acute hemorrhagic fever with a high mortality rate, often reaching 100% in naive herds (Netherton et al., 2019). The virus causes severe physiological disruptions, including elevated body temperature, bleeding, and ataxia. ASFV's ability to modulate host immune responses and induce apoptosis

contributes to rapid disease progression and high mortality (Wu et al., 2021). The virus's interaction with host metabolic pathways also promotes its replication, exacerbating the disease's impact on swine physiology (Ju et al., 2021).

## 5 Case Study

### 5.1 Background of the selected outbreak

The African swine fever virus (ASFV) has been a significant threat to the global pig industry, with outbreaks causing severe economic losses. A notable outbreak occurred in China in 2018, marking the first report of ASFV in the region. This outbreak involved both high-virulence strains, which can cause up to 100% mortality, and low-virulence genotype I strains, complicating prevention and control efforts (Wang et al., 2022). The virus's ability to spread rapidly and its high mortality rate in domestic pigs have made it a critical concern for the swine industry worldwide (Afe et al., 2023).

### 5.2 Host cell responses during the outbreak

During the outbreak, ASFV demonstrated its capacity to manipulate host cell responses to facilitate its replication and evade the immune system. The virus interacts with host proteins to suppress immune responses, such as inhibiting interferon expression and modulating cytokine production. ASFV proteins, like MGF110-7L, can induce stress responses in host cells, leading to translation suppression and stress granule formation, which are crucial for viral survival and replication (Figure 3) (Zhong et al., 2022). Additionally, ASFV exploits host cell machinery, such as the clathrin-mediated endocytosis pathway, to enter cells and establish infection (Chen et al., 2023).

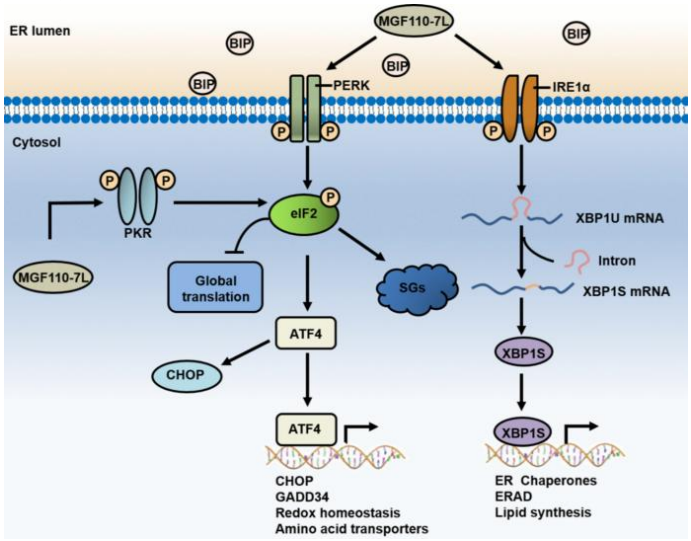


Figure 3 Schematic representation of the proposed role of ASFV MGF110-7L in subversion of the host protein translation (Adopted from Zhong et al., 2022)

Image caption: ASFV-encoded MGF110-7L could trigger ER stress and activate the PERK and IRE1α-XBP1 branches of the UPR. The ER stress and UPR induction promotes the phosphorylation of eIF2α by activating the kinases PERK and PKR, resulting in host translation arrest and SG formation (Adopted from Zhong et al., 2022)

### 5.3 Outcomes and lessons learned

The outbreak highlighted the need for improved understanding of ASFV-host interactions to develop effective control measures. The virus's ability to evade immune responses and manipulate host cell processes underscores the complexity of developing vaccines and antiviral strategies. Research into ASFV's interaction with host proteins has provided insights into potential targets for therapeutic intervention, such as the identification of host proteins involved in viral entry and replication (Chen et al., 2022). The outbreak also emphasized the importance of rapid response and containment measures to prevent the spread of ASFV in new regions. Overall, the lessons learned from this outbreak can guide future research and policy decisions to mitigate the impact of ASFV on the swine industry.

## 6 Implications for Disease Control and Vaccine Development

### 6.1 Challenges in current ASFV control measures

African swine fever virus (ASFV) presents significant challenges for disease control due to its complex interactions with host cells and its ability to evade the host's immune system. The virus primarily targets macrophages and monocytes, modulating these cells to evade immune responses, which complicates the development of effective vaccines. The lack of a commercial vaccine is a major hurdle, as the virus's ability to inhibit interferon signaling and manipulate inflammatory pathways allows it to persist and spread rapidly among swine populations (Orosco, 2024). Additionally, the intricate entry mechanisms of ASFV, involving clathrin-mediated endocytosis and other pathways, further complicate the development of antiviral strategies.

### 6.2 Advances in ASFV research and therapeutics

Recent research has made significant strides in understanding ASFV-host interactions, which is crucial for developing effective vaccines and therapeutics. Studies have identified key viral proteins that interact with host proteins, providing insights into the molecular mechanisms of ASFV infection. Advances in vaccine development include the exploration of live attenuated vaccines (LAVs) and subunit vaccines, which have shown promise in providing homologous and partial heterologous protection (Arias et al., 2017). The identification of specific virulence genes that can be deleted to create attenuated strains is a promising avenue for vaccine development.

### 6.3 Integration of research insights into control strategies

Integrating recent research insights into ASFV control strategies is essential for developing effective measures against the virus. Understanding the virus's immune evasion tactics, such as its ability to inhibit antigen presentation and induce non-neutralizing antibodies, can inform the design of vaccines that elicit a more robust immune response (He et al., 2022). Additionally, targeting the virus's entry mechanisms and its interactions with host proteins could lead to novel antiviral therapies. By leveraging these insights, researchers can develop more targeted and effective control strategies, potentially leading to the eradication of ASFV in affected regions.

## 7 Future Directions

### 7.1 Research priorities for understanding ASFV biology

Understanding the complex interactions between African swine fever virus (ASFV) and host cells remains a critical research priority. Recent studies have highlighted the intricate relationship between ASFV and host immune responses, particularly the virus's ability to manipulate host cell pathways such as the NF-κB signaling pathway and cytokine-cytokine receptor interactions (Li et al., 2022). Further research is needed to elucidate the multifunctional roles of ASFV proteins in these interactions, which could provide insights into viral pathogenesis and immune evasion strategies (Chen and Lin, 2024). Additionally, exploring the virus-host protein-protein interactions (PPIs) can reveal potential targets for antiviral strategies.

### 7.2 Technological advances in ASFV research

Technological advancements such as RNA sequencing and proteomics have significantly enhanced our understanding of ASFV biology. Transcriptome profiling has been instrumental in identifying differentially expressed genes and pathways affected by ASFV infection, providing a comprehensive view of the host's response to the virus (Li and He, 2024). Moreover, the use of yeast two-hybrid assays and mass spectrometry has facilitated the mapping of ASFV-host protein interaction networks, which are crucial for identifying key regulatory proteins involved in viral replication and pathogenesis (Chen et al., 2022). These technologies will continue to play a pivotal role in advancing ASFV research and developing novel therapeutic approaches.

### 7.3 Strategies for global ASFV management

Effective global management of ASFV requires a multifaceted approach, including the development of vaccines and antiviral drugs. Although there are no commercially available vaccines, research into live-attenuated viruses and the identification of non-essential viral genes offers promising avenues for vaccine development (Gallardo et al., 2018; Ramírez-Medina et al., 2020). Additionally, understanding the mechanisms of ASFV entry and replication in host cells can inform the design of antiviral drugs that target specific stages of the viral life cycle.

Collaborative international efforts are essential to control the spread of ASFV and mitigate its economic impact on the swine industry.

## 8 Concluding Remarks

The interaction between African swine fever virus (ASFV) and host cells is a complex process that involves multiple pathways and mechanisms. ASFV primarily targets macrophages, entering these cells through clathrin- and cholesterol-dependent endocytosis, which is crucial for successful viral replication. The virus-host interaction network is intricate, with ASFV proteins such as MGF360-9L and p30 interacting with host proteins involved in protein binding, metabolism, and immune responses. ASFV's ability to evade host immune defenses is facilitated by its modulation of cytokine expression and inhibition of apoptosis and autophagy in infected cells. These interactions are essential for understanding ASFV pathogenesis and developing effective antiviral strategies.

Future research should focus on further elucidating the molecular mechanisms of ASFV entry and replication within host cells. Identifying key viral and host protein interactions can provide targets for novel antiviral drugs and vaccine development. Additionally, understanding the role of specific host proteins, such as CD1d, in facilitating ASFV entry could lead to new therapeutic interventions. Policymakers should prioritize funding for ASFV research to accelerate the development of effective vaccines and treatments, which are currently lacking. Implementing biosecurity measures and surveillance systems in pig farming can help control the spread of ASFV and mitigate its economic impact.

The ongoing research into ASFV-host interactions is crucial for combating this devastating disease. By advancing our understanding of the virus's mechanisms of infection and immune evasion, we can develop more effective strategies to prevent and treat ASFV infections. Collaborative efforts between researchers, policymakers, and the swine industry are essential to address the challenges posed by ASFV and protect global pig populations.

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### Conflict of Interest Disclosure

The author affirms that this research was conducted without any commercial or financial relationships that could be construed as a potential conflict of interest.

### References

Afe A., Shen Z., Guo X., Zhou R., and Li K., 2023, African swine fever virus interaction with host innate immune factors, Viruses, 15(6): 1220.
https://doi.org/10.3390/v15061220

Alonso C., Galindo I., Cuesta-Geijo M., Cabezas M., Hernáez B., and Muñoz-Moreno R., 2012, African swine fever virus-cell interactions: from virus entry to cell survival, Virus Research, 173: 42-57.
https://doi.org/10.1016/j.virusres.2012.12.006

Arias M., De La Torre A., Dixon L., Gallardo C., Jori F., Laddomada A., Martins C., Parkhouse R., Revilla Y., Rodríguez F., and Sánchez-Vizcaíno J., 2017, Approaches and perspectives for development of african swine fever virus vaccines, Vaccines, 5(4): 35.
https://doi.org/10.3390/vaccines5040035

Chen H.Y., and Lin X.F., 2024, ASFV proteins as drug targets: insights from genomic and proteomic studies, Bioscience Evidence, 14(5): 229-237.
https://doi.org/10.5376/be.2024.14.0024

Chen X., Chen X., Liang Y., Xu S., Weng Z., Gao Q., Huang Z., Zhang G., and Gong L., 2022, Interaction network of African swine fever virus structural protein p30 with host proteins, Frontiers in Microbiology, 13: 971888.
https://doi.org/10.3389/fmicb.2022.971888

Chen X., Zheng J., Liu C., Li T., Wang X., Li X., Bao M., Li J., Huang L., Zhang Z., Bu Z., and Weng C., 2023, CD1d facilitates African swine fever virus entry into the host cells via clathrin-mediated endocytosis, Emerging Microbes and Infections, 12(2): 2220575.
https://doi.org/10.1080/22221751.2023.2220575

Dixon L., Islam M., Nash R., and Reis A., 2019, African swine fever virus evasion of host defences, Virus Research, 266: 25-33.
https://doi.org/10.1016/j.virusres.2019.04.002

Dolata K., Pei G., Netherton C., and Karger A., 2023, Functional landscape of African swine fever virus-host and virus-virus protein interactions, Viruses, 15(8): 1634.
https://doi.org/10.3390/v15081634

Galindo I., Cuesta-Geijo M., Hlavová K., Muñoz-Moreno R., Barrado-Gil L., Domínguez J., and Alonso C., 2015, African swine fever virus infects macrophages, the natural host cells, via clathrin- and cholesterol-dependent endocytosis, Virus Research, 200: 45-55.
https://doi.org/10.1016/j.virusres.2015.01.022

Gallardo C., Sánchez E., Pérez-Núñez D., Nogal M., De León P., Carrascosa Á., Nieto R., Soler A., Arias M., and Revilla Y., 2018, African swine fever virus (ASFV) protection mediated by NH/P68 and NH/P68 recombinant live-attenuated viruses, Vaccine, 36(19): 2694-2704.
https://doi.org/10.1016/j.vaccine.2018.03.040

Guo Z., Zhuo Y., Li K., Niu S., and Dai H., 2021, Recent advances in cell homeostasis by African swine fever virus-host interactions, Research in Veterinary Science, 141: 4-13.
https://doi.org/10.1016/j.rvsc.2021.10.003

He W., Yuan J.Y., Zhao C., Yang Z., Zhang Y., Han S., Wan B., and Zhang G., 2022, Modulation of host antiviral innate immunity by African swine fever virus: a review, Animals, 12(21): 2935.
https://doi.org/10.3390/ani12212935

Ju X., Li F., Li J., Wu C., Xiang G., Zhao X., Nan Y., Zhao D., and Ding Q., 2021, Genome-wide transcriptomic analysis of highly virulent African swine fever virus infection reveals complex and unique virus host interaction, Veterinary Microbiology, 261: 109211.
https://doi.org/10.1016/j.vetmic.2021.109211

Li J., and He J., 2024, Eliminating porcine pathogens: the role of genetic modifications in enhancing biosafety of transplantable pig organs, Bioscience Methods, 15(4): 162-172.
https://doi.org/10.5376/bm.2024.15.0017

Li Z., Chen W., Li X., Wu K., Wang X., Wang W., Li Y., Yi L., Zhao M., Ding H., Fan S., and Chen J., 2022, Transcriptome profiling in swine macrophages infected with African swine fever virus (ASFV) uncovers the complex and close relationship with host, Pathogens, 119(19): e2201288119.
https://doi.org/10.3390/pathogens11121411

Netherton C., Connell S., Benfield C., and Dixon L., 2019, The genetics of life and death: virus-host interactions underpinning resistance to african swine fever, a viral hemorrhagic disease, Frontiers in Genetics, 10: 402.
https://doi.org/10.3389/fgene.2019.00402

Orosco F., 2024, African swine fever virus proteins against host antiviral innate immunity and their implications for vaccine development, Open Veterinary Journal, 14: 941-951.
https://doi.org/10.5455/OVJ.2024.v14.i4.1

Ramírez-Medina E., Vuono E., Velazquez-Salinas L., Silva E., Rai A., Pruitt S., Berggren K., Zhu J., Borca M., and Gladue D., 2020, The MGF360-16R ORF of African swine fever virus strain georgia encodes for a nonessential gene that interacts with host proteins SERTAD3 and SDCBP, Viruses, 12(1): 60.
https://doi.org/10.3390/v12010060

Sánchez E., Pérez-Núñez D., and Revilla Y., 2017, Mechanisms of entry and endosomal pathway of African swine fever virus, Vaccines, 5(4): 42.
https://doi.org/10.3390/vaccines5040042

Sánchez E., Quintas A., Nogal M., Castello A., and Revilla Y., 2013, African swine fever virus controls the host transcription and cellular machinery of protein synthesis, Virus Research, 173(1): 58-75.
https://doi.org/10.1016/j.virusres.2012.10.025

Sánchez E., Quintas A., Pérez-Núñez D., Nogal M., Barroso S., Carrascosa Á., and Revilla Y., 2012, African swine fever virus uses macropinocytosis to enter host cells, PLoS Pathogens, 8(6): e1002754.
https://doi.org/10.1371/journal.ppat.1002754

Wang Z., Ai Q., Huang S., Ou Y., Gao Y., Tong T., and Fan H., 2022, Immune escape mechanism and vaccine research progress of African swine fever virus, Vaccines, 10(3): 344.
https://doi.org/10.3390/vaccines10030344

Wu L., Yang B., Yuan X., Hong J., Peng M., Chen J., and Song Z., 2021, Regulation and evasion of host immune response by African swine fever virus, Frontiers in Microbiology, 12: 698001.
https://doi.org/10.3389/fmicb.2021.698001

Yang B., Shen C., Zhang D., Zhang T., Shi X., Yang J., Hao Y., Zhao D., Cui H., Yuan X., Chen X., Zhang K., Zheng H., and Liu X., 2021a, Mechanism of interaction between virus and host is inferred from the changes of gene expression in macrophages infected with African swine fever virus CN/GS/2018 strain, Virology Journal, 18: 1-16.
https://doi.org/10.1186/s12985-021-01637-6

Yang B., Zhang D., Shi X., Shen C., Hao Y., Zhang T., Yang J., Yuan X., Chen X., Zhao D., Cui H., Li D., Zhu Z., Tian H., Yang F., Zheng H., Zhang K., and Liu X., 2021b, Construction, identification and analysis of the interaction network of african swine fever virus MGF360-9L with host proteins, Viruses, 13(9): 1804.
https://doi.org/10.3390/v13091804

Zhang K., Li S., Liu S., Li S., Qu L., Gao G., and Qiu H., 2021, Spatiotemporally orchestrated interactions between viral and cellular proteins involved in the entry of African swine fever virus, Viruses, 13(12): 2495.
https://doi.org/10.3390/v13122495

Zhong H., Fan S., Du Y., Zhang Y., Zhang A., Jiang D., Han S., Wan B., and Zhang G., 2022, African swine fever virus MGF110-7L induces host cell translation suppression and stress granule formation by activating the PERK/PKR-eIF2α pathway, Microbiology Spectrum, 10(6): e03282-22. https://doi.org/10.1128/spectrum.03282-22

Zhu J., Ramanathan P., Bishop E., O'Donnell V., Gladue D., and Borca M., 2019, Mechanisms of African swine fever virus pathogenesis and immune evasion inferred from gene expression changes in infected swine macrophages, PLoS One, 14(11): e0223955. https://doi.org/10.1371/journal.pone.0223955