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Review

Insights into mosquito-borne arbovirus receptors

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A B S T R A C T

The increasing global prevalence of mosquito-borne viruses has emerged as a significant threat to human health and life. Identifying receptors for these viruses is crucial for improving our knowledge of viral pathogenesis and developing effective antiviral strategies. The widespread application of CRISPR-Cas9 screening have led to the discovery of many mosquito-borne virus receptors. The revealed structures of virus-receptor complexes also provide important information for understanding their interaction mechanisms. This review provides a comprehensive summary of both conventional and novel approaches for identifying new viral receptors and the putative entry factors of the most prevalent mosquito-borne viruses within the *Flaviviridae*, *Togaviridae*, and *Bunyavirales*. At the same time, we emphasize the common receptors utilized by these viruses for entry into both vertebrate hosts and mosquito vectors. We discuss promising avenues for developing anti-mosquito-borne viral strategies that target these receptors. Notably, targeting universal receptors of specific mosquito-borne viruses in both vertebrates and mosquitoes offers dual benefits for disease prevention. Additionally, the widespread use of AI-based machine learning and protein structure prediction will accelerate the identification of new viral receptors and provide new avenues for antiviral drug discovery.

1. Introduction

Mosquito-borne arboviruses are a major global public health concern due to their potential to cause widespread outbreaks and severe disease in humans (Sukhralia et al., 2019). The prevalence of mosquito-borne viral infectious diseases is continually worsening, with an estimated 80% of the global population residing in areas threatened by these viruses each year (Franklinos et al., 2019). These viruses are primarily classified into 14 families, with the most notable being *Flaviviridae*, *Togaviridae*, and several families within *Bunyavirales* (Braack et al., 2018). Specifically, viruses in the Flaviviridae family, such as dengue virus (DENV), Zika virus (ZIKV), West Nile virus (WNV), yellow fever virus (YFV) and Japanese encephalitis virus (JEV) have become widely prevalent globally. These viruses cause over 400 million infections annually and lead to hundreds of thousands of severe illnesses or deaths, posing a significant threat to public health (Pierson & Diamond, 2020). Viruses in the *Togaviridae* family, such as chikungunya virus (CHIKV), Eastern equine encephalitis virus (EEEV), have disseminated to over 100 countries and regions across Asia, Europe, and the Americas, infecting millions of individuals. These infections manifest in severe symptoms, including chronic infectious arthritis and neurological encephalitis, resulting in long-term and persistent harm to human health (Azar et al., 2020; Rezza, 2014; Schuffenecker et al., 2006). Rift Valley fever virus (RVFV), a member of the *Phenuiviridae* family in the *Bunyavirales* order, is transmitted by mosquitoes and can cause severe hemorrhagic fever in humans, with a mortality rate that can reach up to 50% in severe cases. The virus can also lead to encephalitis and ocular complications (WHO, 2018). The World Health Organization (WHO) has listed it as one of the infectious diseases of great concern for the future. Since 2023, the Oropouche virus (OROV), a member of the *Peribunyaviridae* family in the *Bunyavirales* order, has caused a significant outbreak in South America, particularly in Brazil. This outbreak has resulted in more than 500,000 diagnosed cases, with the virus also being detected in Italy for the first time, indicating its potential for global spread (Castilletti et al., 2024;

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Moutinho, 2024; Zhang, Liu, et al., 2024). Given that most mosquito-borne viruses lack effective vaccines or medications, mosquito-borne viral infectious diseases continue to be one of the greatest threats to human health (Soni et al., 2023).

The transmission cycle of mosquito-borne viruses involves both vertebrate hosts and mosquito vectors. For these viruses to persist in nature, they must be efficiently transmitted between these two evolutionarily distant species (Weaver & Barrett, 2004). Mosquito-borne viruses typically possess an enveloping membrane, with surface proteins on this envelope responsible for binding to cell receptors. This interaction facilitates viral attachment to the cell membrane and subsequently triggers cellular entry through complex mechanisms, including endocytosis and membrane fusion (Carro & Cherry, 2020; Kim & Diamond, 2023). Receptors are the primary and most critical determinants for viral entry into host cells, serving as essential gateways for subsequent viral replication and reproduction. Virus receptors can be divided into two categories: attachment factors and entry receptors. Attachment factors primarily increase the binding affinity of the virus to the host cell surface but may not directly facilitate entry. Entry receptors are proteins that directly bind and facilitate viral entry into host cells, often triggering conformational changes or signaling pathways that enable internalization. Blocking these receptors with antibodies or decoys can effectively inhibit infection, while overexpression of the receptors in cells or animals should promote viral infection (Marsh & Helenius, 2006; Mercer et al., 2020; Zimmerman, Holmes, et al., 2023). Numerous receptors for mosquito-borne viruses have already been identified, some of which have homologous family members present in both vertebrates and mosquitoes, while others function exclusively in one of these species. Understanding the common features of receptors for mosquito-borne viruses across different types of hosts and vectors will facilitate the identification of potential new viral receptors, while also providing a foundation for developing antiviral strategies and drugs. In this review, we summarize the existing methods for identifying viral receptors, outline the receptors identified for mosquito-borne flaviviruses, alphaviruses, and bunyaviruses (Fig. 1 and Table 1), and discuss common receptors and patterns among them. Additionally, we explore potential virus control strategies based on these receptors.

2. Approaches for the identification of mosquito-borne viral receptors

The identification of viral receptors remains a critical challenge in virology research. Conventional methodologies for receptor identification often involve the use of the virus or its surface proteins as bait to capture potential receptor molecules, followed by mass spectrometry for protein identification (Moutinho, 2024). Additionally, display library methods using systems such as bacteria, yeast, and phages can express receptor proteins or peptides on cell surfaces, allowing the selection of potential viral receptors based on binding interactions with the virus or its surface proteins (Sheehan & Marasco, 2015). However, these static protein-protein interaction methods have limitations, as functional receptors may exhibit transient and weak interactions with viral proteins, complicating their identification. Proximity labeling, which involves fusing a label enzyme with the viral protein as bait (V'Kovski et al., 2019), followed by affinity purification and mass spectrometry to capture transiently interacting proteins, may address the aforementioned issues, but is often restricted by a lack of specificity and high background noise, limiting its application for viral receptor identification.

A biochemical approach, named VOPBA (virus overlay protein binding assay), has also been employed for viral receptor identification. In this method, cell membrane protein fragments from permissive cells are electrophoresed, transferred to a nitrocellulose membrane, and

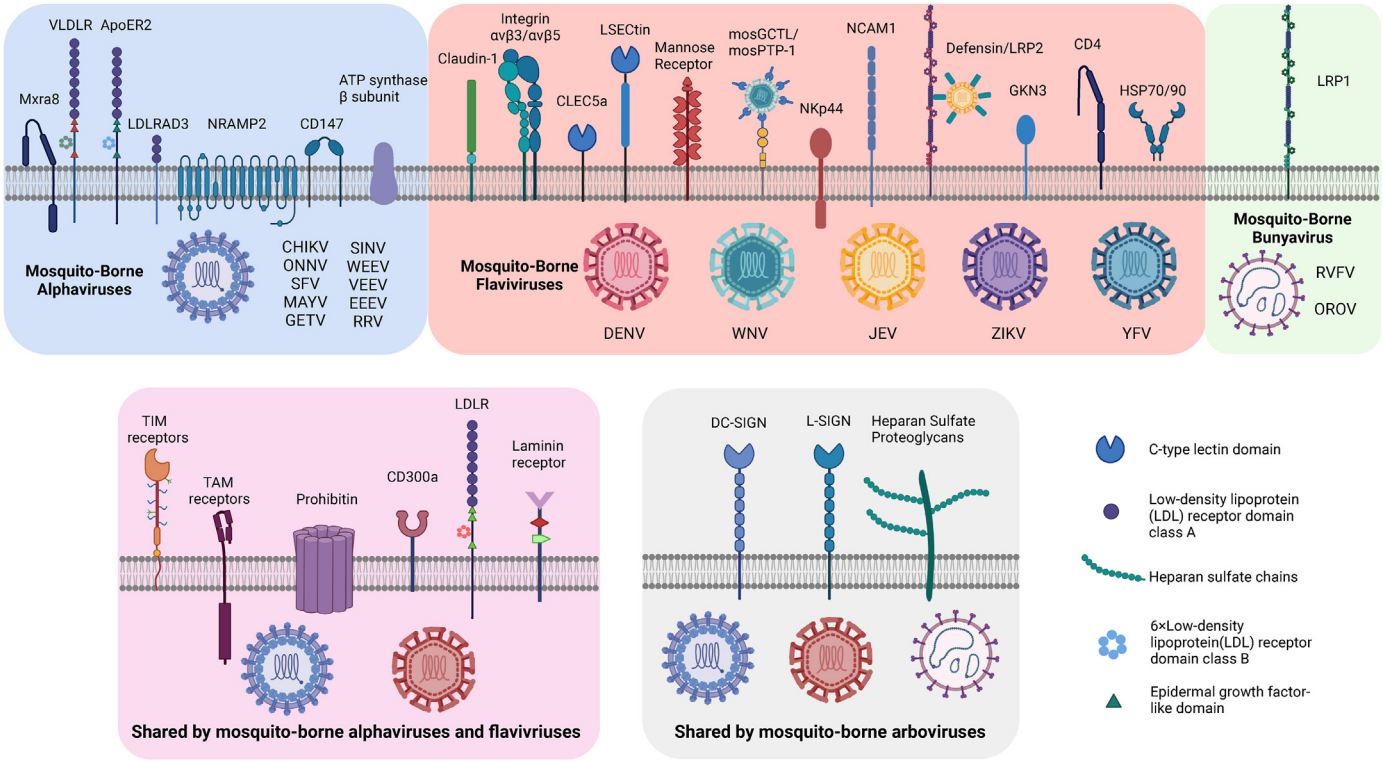

Fig. 1. Putative receptors of mosquito-borne arboviruses. Alphaviruses: Multiple functional entry receptors such as MXRA8, LDLR family members, NRAMP2, CD147 have been identified to facilitate virus entry; Flaviviruses: Molecules including C-type lectins, phosphatidylserine receptors, integrins, heat shock proteins, Claudin-1, NACM1, NKp44, GKN3, CD4 may be employed for virus attachment or entry; Bunyaviruses: LRP1 facilitates the entry of mosquito-borne bunyaviruses such as RVFV, OROV. Shared receptors: Some receptors are shared by both mosquito-borne alphaviruses and flaviviruses, including TIM receptors, TAM receptors, Prohibitin, CD300a, LDLR, and the Laminin receptor. Additionally, receptors like DC-SIGN, L-SIGN, and Heparan Sulfate Proteoglycans (HSPGs) are utilized by a broad range of mosquito-borne arboviruses to enhance viral attachment or entry. Created with BioRender.com.

Table 1
Potential receptors of mosquito-borne arboviruses.

Arboviruses	Molecules	Properties	Viruses	Cell types	Approaches used to identify/ verify the receptors	Interacted viral components	References
Mosquito-Borne Flaviviruses	DC-SIGN/L-SIGN/ LSECtin	C-type lectins	DENV, WNV, JEV, ZIKV	Mammalian	Binding assays; Immunofluorescence; RNAi assays; Antibody blocking assay; Infection assays in overexpressed non-permissive cell lines; Cryo-EM	prM/E proteins	(Davis et al., 2006; Dejnirattisai et al., 2011; Liu, Ridilla, et al., 2017; Pokidysheva et al., 2006; Wang et al., 2016)
	Mannose receptor		DENV	Mammalian	Binding Assays; Blot overlay; Immunofluorescence	E protein	McFadden et al. (2008)
	CLEC5A		DENV, JEV, ZIKV	Mammalian, Insect	Infection assays in overexpressed non-permissive cell lines; Immunofluorescence; Antibody blocking assay in cell lines and animal models; Flow cytometry	E protein	(Chen et al., 2008, 2012)
	mosGCTL-1/3/7		WNV, DENV, JEV	Insect	Genome-wide RNAi screening; RNAi assays; immunofluorescence; CO-IP	E protein	(Cheng et al., 2010; Liu et al., 2014, 2017a)
	TIM receptors	Phosphatidylserine receptors	DENV, WNV, JEV, ZIKV, YFV	Mammalian	Gain-of-function cDNA screening; Infection assays in overexpressed non-permissive cell; Flow Cytometry; Antibody blocking assay; Immunofluorescence	E protein	(Hamel et al., 2015; Meertens et al., 2012; Niu et al., 2018)
	TAM receptors		DENV, ZIKV	Mammalian	Gain-of-function cDNA screen; RNAi assays; Infection assays in overexpression/ knockout cell lines; Flow Cytometry; Antibody blocking assay; Immunofluorescence	E protein	(Meertens et al., 2012, 2017; Richard et al., 2017)
	CD300a		DENV, YFV	Mammalian	cDNA library screening; Infection assays in overexpressed cell lines; Flow Cytometry; RNAi assays; Antibody blocking assay	E protein	Carnec et al. (2016)
	Integrin αvβ3	Integrin family	WNV, JEV	Mammalian	VOPBA; Antibody blocking assay; RNAi assays; Immunofluorescence; Flow Cytometry	E protein	(Chu and Ng, 2004; Chu & Ng, 2004; Fan et al., 2017; Schmidt et al., 2013)
	Integrin αvβ5		ZIKV	Mammalian	CRISPR-Cas9 screening; Infection assays in knockout/ overexpression cell lines assays; Immunofluorescence; Antibody blocking assay	Viral particle	Wang et al. (2020)
	HS/HSPG	Glycosaminoglycans	DENV, JEV, ZIKV, YFV	Mammalian	Binding assays; GAG-deficient cell infection assays; In vivo bioluminescence imaging; RNAi assays	E protein	(Gao et al., 2019; Germi et al., 2002; Okamoto et al., 2012; Su et al., 2001)
	37kDa/67 kDa Laminin receptor	High-affinity laminin receptor	DENV, JEV	Mammalian, Insect	VOPBA; Binding assays; Liquid chromatography-mass spectrometry (MS); Antibody blocking assay	E protein	(Thepparit & Smith, 2004; Thongtan et al., 2012)

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Table 1 (*continued*)

Arboviruses	Molecules	Properties	Viruses	Cell types	Approaches used to identify/ verify the receptors	Interacted viral components	References
	GRP78/HSP70/90	Heat shock protein family	DENV, ZIKV, JEV	Mammalian, Insect	VOPBA; Cell heat shock assay; Antibody blocking assay; RNAi assays; Immunofluorescence; Immunolocalization	E protein	(Das et al., 2009; Hung et al., 2011; Pujhari et al., 2019; Reyes-del Valle et al., 2005)
	NKp44	Natural cytotoxicity receptor family	DENV, WNV	Mammalian	Elisa; Antibody blocking assay; Flow cytometry; Cytotoxicity assays	E protein	Hershkovitz et al. (2009)
	Prohibitin-1	Prohibitin family	DENV	Mammalian, Insect	VOPBA; RNAi assays; Antibody blocking assay; Immunofluorescence	E protein	(Kuadkitkan et al., 2010; Sharma et al., 2020)
	Claudin-1	Claudin family	DENV	Mammalian	Yeast two-hybrid (Y2H) screening; Pull-down assays; RNAi assays	prM/M protein	Che et al. (2013)
	nLc4Cer/GM1a/GM3	Glycosphingolipid	DENV	Mammalian, Insect	TLC/virus-binding and TLC immunostaining assays; Confocal microscope imaging; MS analysis; ITC; RNAi assays	E protein	(Tantirimudalige et al., 2022; Wang et al., 2016; Wichit et al., 2011)
	NCAM1	NCAM family	ZIKV	Mammalian	Proximity labeling using chemical labeled ZIKV particles; LC–MS/MS analysis; Immunoprecipitation; CRISPR-Cas9 knockout assays; Immunofluorescence	Viral particle	Srivastava et al. (2020)
	LDLR	LDLR family	JEV	Mammalian	RNAi assays; CO-IP; Immunofluorescence; Antibody blocking assay; animal assays	E protein	Huang et al. (2021)
	Defensin/LRP2	Antimicrobial peptides	JEV	Insect	RNAi assays; Immunofluorescence; Antibody blocking assay	E protein	Liu et al. (2020)
	Vimentin	Intermediate filament protein	JEV	Mammalian	Entry blocking assays; Pull-down	E protein	(Zhang et al., 2024)
	CD4/CD14	Transmembrane glycoprotein	JEV	Mammalian	VOPBA; Binding assays; LC-MS; Antibody blocking assay	E protein	(Thongtan et al., 2012; Wang et al., 2023)
	PLVAP/GKN3	Transmembrane glycoprotein/ Gastrokine family	JEV	Mammalian	Pull down with 2-DE separation and MS; Infection assays in overexpression cell lines; RNAi assays; Antibody blocking assay; Immunofluorescence	E protein	Mukherjee et al. (2018)
Mosquito-Borne Alphaviruses	LDLRAD3	LDLR family	VEEV	Mammalian	CRISPR–Cas9 screening; Infection assays in knockout/ overexpressed cell lines and animal models; Surface plasmon resonance; In vivo imaging; Cryo-EM; Site-directed mutagenesis	E2–E1 heterodimers	(Basore et al., 2021; Ma et al., 2020, 2021)
	VLDLR		SFV, EEEV, SINV	Mammalian, Insect	CRISPR-Cas9 screening; Infection assays in overexpressed cell lines; Entry blocking assays; Biolayer interferometry binding	SFV E1 protein, EEEV E2 protein	(Adams et al., 2024; Cao et al., 2023; Clark et al., 2021)

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Table 1 (*continued*)

Arboviruses	Molecules	Properties	Viruses	Cell types	Approaches used to identify/ verify the receptors	Interacted viral components	References
	ApoER2		SFV, EEEV, SINV	Mammalian	CRISPR-Cas9 screening; Cryo-EM assays; Confocal microscopy; Infection assays in knockout/ overexpressed cell lines; Entry blocking assays; Binding assays; Confocal microscopy	Viral particle	Clark et al. (2021)
	LDLR		EEEV, WEEV, SFV, RRV, GETV, BEBV	Mammalian	Host membrane protein expression library screen; Infection assays in knockout/ overexpression cell lines assays; Co-IP; Binding assays; Entry blocking assays	E1 and E2 proteins	(Ma et al., 2024; Zhai et al., 2024)
	MXRA8	Transmembrane glycoprotein	CHIKV, RRV, ONNV, MAYV, SINV, WEEV, GETV	Mammalian, Avian	CRISPR-Cas9 screening; Co-IP; Mass Spectrometry; Binding assays; Infection assays in knockout/ overexpression cell lines and animal models; X-ray Crystallography; Cryo-EM	E1 and E2 proteins	(Basore et al., 2019; Kim et al., 2020; Song et al., 2019; Zhang et al., 2018; Zimmerman et al., 2023)
	NRAMP/NRAMP2	NRAMP family	SINV	Mammalian, Insect	RNAi assays; Immunofluorescence	Viral particle	Rose et al. (2011)
	CD147	Transmembrane glycoprotein	EEEV, WEEV, SINV, CHIKV, RRV	Mammalian	CRISPR-Cas9 screening; CRISPR-Cas9 knockout and complement assays	E2 protein	De Caluwé et al. (2021)
	Prohibitin-1	Prohibitin family	CHIKV	Mammalian	2D-VOPBA; Antibody blocking assay; CO-IP; Immunofluorescence; RNAi assays	E2 protein	Wintachai et al. (2012)
	37kDa/67 kDa Laminin receptor	High-affinity laminin receptor	SINV, VEEV	Mammalian	Antibody blocking assay; Immunoprecipitation; Flow cytometry	E2 protein	(Malygin et al., 2009; Wang et al., 1992)
	TIM receptors	Phosphatidylserine receptors	CHIKV, RRV, SINV, EEEV	Mammalian	Infection assays in overexpressed non-permissive cell lines; Antibody blocking assay; Single particle tracking; Confocal microscope imaging	E2 protein	(Kirui et al., 2021; Moller-Tank & Maury, 2014; Pierson et al., 2020)
	TAM receptors	Phosphatidylserine receptors C-type lectins	RRV, SINV	Mammalian	Infection assays in overexpressed non-permissive cell lines; Entry blocking assays; Flow cytometry	Viral particle	(Pierson et al., 2020)
	CD300a		SINV	Mammalian	cDNA library screening; Infection assays in overexpressed non-permissive cell lines	Viral particle	Carnec et al. (2016)
	DC-SIGN/L-SIGN		SINV	Mammalian, Insect	Flow cytometry; Immunofluorescence; Infection assays in overexpressed non-permissive cell lines	E1 and E2 proteins	Klimstra et al. (2003)
	HS/HSPG	Glycosaminoglycans	SINV, CHIKV, SFV, RRV, EEEV, VEEV	Mammalian	Binding assays; Infection assays in HS/GAG-deficient cell lines; In vivo imaging	E2 protein	(Gardner et al., 2011; McAllister et al., 2020; Ryman et al., 2007; Smit et al., 2002)
	Integrin α1β1	Integrin family	RRV	Mammalian	Antibody blocking assay; Elisa	E2 protein	La Linn et al. (2005)

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Table 1 (*continued*)

Arboviruses	Molecules	Properties	Viruses		Cell types	Approaches used to identify/verify the receptors	Interacted viral components	References
	ATP synthase β subunit	ATPase alpha/beta chains family	CHIKV		Insect	VOPBA; Mass Spectrometry; Antibody blocking assay; RNAi assays; Confocal microscope imaging	E2 protein	Fongsaran et al. (2014)
Mosquito-Borne Bunyaviruses	LRP1	LDLR family	RVFV, OROV		Mammalian	CRISPR-Cas9 screening; Infection assays in overexpression/knockout cell line assays; Antibody blocking assay; Biolayer interferometry binding assays; Immunofluorescence	Gn glycoproteins	(Ganaie et al., 2021; Schwarz et al., 2022)
	DC-SIGN/L-SIGN	C-type lectins	RVFV, LACV, OROV		Mammalian	Binding assays; Immunofluorescence; Infection assays in overexpressed non-permissive cell lines; Flow cytometry	Gn/Gc glycoproteins	(Léger et al., 2016; Lozach et al., 2011)
	HS/HSPG	Glycosaminoglycans	RVFV		Mammalian	Infection assays in HS/GAG-deficient cell lines; Immunofluorescence; Binding assays	Viral particle	(de Boer et al., 2012; Riblett et al., 2016)

exposed to viral particles. The binding fractions are then analyzed by mass spectrometry, but only proteins that bind under denatured conditions can be isolated (Zimmerman, Holmes, et al., 2023). The antibody blockade method screens and identifies viral receptors by using specific monoclonal antibodies to block potential receptor-virus interactions. However, this approach is costly due to the high price of quality monoclonal antibody libraries, and nonspecific antibody binding may also cause interference.

The CRISPR-Cas9 single-guide RNA (sgRNA) knockout library technique is a novel method for discovering potential viral receptors. This approach creates a cell library with thousands of single-gene knockouts. Upon infection with viruses, cells resistant to infection or showing different fluorescence intensities are sorted by FACS. High-throughput sequencing is then used to identify the absent genes in these enriched cell populations (Shalem et al., 2014). The CRISPR gene knock-in method can overexpress a set of library proteins in individual cells. Cells with high infectivity, usually indicated by higher fluorescence when infected with a non-replicating virus, are isolated and identified through sequencing. Using these advanced methods, several viral receptors have been successfully identified in recent years (Clark et al., 2021; Ganaie et al., 2021; Ma et al., 2020, 2024; Zhai et al., 2024; Zhang et al., 2018).

Notably, with advancements in artificial intelligence (AI) and big data computation, AI-driven approaches like AlphaFold now enable more accurate predictions of unknown protein structures. By combining these predictions with protein-protein interaction docking methods, we can significantly narrow down candidate genes that bind to specific viral proteins, greatly enhancing receptor identification efficiency and reducing costs. It should be noted that, regardless of the methods used, identified receptors need be functionally validated in cell and animal models to confirm their role as viral attachment or entry factors.

3. The potential entry receptors of mosquito-borne arboviruses

3.1. The receptors of mosquito-borne flaviviruses

The envelope (E) protein, arranged as trimers on the envelope of flaviviruses, is the key protein responsible for binding to receptors and mediating viral entry. Research on the receptors of mosquito-borne flaviviruses has been ongoing for many years, but the putative entry receptors have not yet been fully elucidated (Oliveira & Peron, 2019). Many proteins have been identified as involved in the entry process of viruses such as DENV, ZIKV, WNV, JEV, and YFV (Anwar et al., 2022). However, many of these proteins merely mediate virus-cell binding, acting as ‘attachment factors' rather than facilitating entry. Additionally, some of these proteins function as entry receptors only in specific cell types, possibly due to the presence of alternative receptors in these cells, with downregulation or knockout in other cell types or animals not significantly affecting viral infection and replication. Current research suggests that for most flaviviruses, multiple proteins on the cell membrane are likely involved in the virus's binding and entry process. The absence of a universal dominant entry receptor for flaviviruses presents a significant challenge for studies on viral pathogenesis and antiviral drug discovery. The ongoing identification of putative entry receptors is gradually unraveling the mechanisms of flavivirus entry into cells, providing crucial insights into the common receptor patterns of flaviviruses.

3.1.1. C-type lectins

C-type lectins (CTLs) are carbohydrate-binding proteins characterized by their calcium-dependent binding to specific sugar moieties, playing a crucial role in inducing the host immune response to fungal, bacterial, and viral infections (Hoving et al., 2014). During flavivirus infection, certain CTLs are recruited as entry factors to facilitate infection. Specifically, DC-SIGN (CD209) and L-SIGN (CD209L) have been identified as an attachment or entry receptor exploited by WNV, JEV, DENV and ZIKV to invade dendritic cells or liver cells (Davis et al., 2006; Dejnirattisai

et al., 2011; Hamel et al., 2015; Liu, Ridilla, et al., 2017; Pokidysheva et al., 2006; Shimojima et al., 2014; Tassaneetrithep et al., 2003; Wang et al., 2016). Additionally, the mannose receptor (MR) and CLEC5A can promote DENV infection in macrophages (S.-T. Chen et al., 2008; S.-T. Chen et al., 2012; McFadden et al., 2008; Pöhlmann et al., 2016). In mosquitoes, mosquito galactose-specific C-type lectin 1 (mosGCTL-1) and C-type lectin 7 (mosGCTL-7) serve as functional receptors for WNV and JEV infections, respectively. These secretory C-type lectins bind to WNV or JEV and are captured by a CD45 phosphatase homolog (mosPTP-1), facilitating viral entry in mosquitoes (Cheng et al., 2010; Liu, Qian, et al., 2017). Besides, mosGCTL-3 can interact with DENV-2 surface envelop protein and facilitate virus infection (Liu et al., 2014). These data indicate that C-type lectins may serve as important entry receptors for the flavivirus family. However, to date, only the structure of DC-SIGN has been described (Pokidysheva et al., 2006), and the overall binding patterns between C-type lectins and viruses remain unclear.

3.1.2. Phosphatidylserine receptors

Phosphatidylserine (PS) receptors are a group of receptors that recognize and bind to phosphatidylserine, a phospholipid that translocate from the inner to the outer leaflet of the plasma membrane during apoptosis or cell activation. The TIM and TAM proteins, which mediate PS-dependent phagocytic removal of apoptotic cells, also facilitate the entry of flaviviruses, including DENV, WNV, and YFV (Meertens et al., 2012). TIM-1 has been reported to enhance JEV and ZIKV infection in 293T and A549 cells and (Carnec et al., 2016; Niu et al., 2018), while AXL and Tyro3, members of the TAM family, has been reported to mediate viral entry in human glial cells and fetal endothelial cells (Carnec et al., 2016; Meertens et al., 2012, 2017; Richard et al., 2017). CD300a, another phospholipid receptor, facilitates the infection of all DENV serotypes (DENV I-IV), as well as some alphaviruses (Carnec et al., 2016). These data suggest that PS receptors may have a universal role in interacting with multiple mosquito-borne viruses.

3.1.3. Integrins and glycosaminoglycans

Integrins are transmembrane receptors that facilitate cell-extracellular matrix (ECM) adhesion, while glycosaminoglycans (GAGs) are long, unbranched polysaccharides in the ECM. Both cell membrane proteins play crucial roles in various physiological processes and diseases. Integrins may serve as common entry receptors for several mosquito-borne flaviviruses. Blocking $\alpha v\beta_3$ integrin with antibodies reduces the infection of WNV and JEV, but not DENV (Chu & Ng, 2004; Fan et al., 2017; Schmidt et al., 2013). Based on CRISPR-Cas9 screening, integrin αvβ5 was identified as an internalization factor for ZIKV. Blocking αvβ5 with antibodies alleviates viral infection (Wang et al., 2020). The GAG family members, heparan sulfate (HS) and heparan sulfate proteoglycan (HSPG), are also common attachment factors for flaviviruses. Several early studies indicated that heparin, a highly sulfated heparan sulfate, facilitates DENV, JEV, and YFV infections. Desulfation of the cell surface or using heparin competitors significantly reduces the invasion of these flaviviruses (H.L. Chen et al., 2010; Y. Chen et al., 1997; Gao et al., 2019; Germi et al., 2002; Okamoto et al., 2012; Su, Liao, Lee, & Lin, 2001). Further mechanism studies indicate that the 261–402 amino acid region of the JEV E protein may contribute to the viral binding to heparan sulfate (Chen et al., 2010). It should be noted that these extracellular matrix proteins can bind to various viral particles, not just mosquito-borne viruses. However, most of them likely function only as attachment factors and do not mediate viral internalization.

3.1.4. Laminin receptor and heat shock protein family

Laminin is a large glycoprotein in the extracellular matrix, primarily in the basement membrane. Some laminin proteins have non-classical transmembrane regions, while others may bind to co-receptors or be anchored to the cell membrane (DiGiacomo & Meruelo, 2016). Heat Shock Proteins (HSPs) are abundantly expressed under stress conditions to maintain protein homeostasis and can appear on the cell membrane surface under stress and certain pathological conditions (De Maio & Hightower, 2021). Using the virus overlay protein binding assay (VOPBA) and affinity chromatography, a high-affinity laminin receptor and heat shock protein 90/70 was reported to serve as a DENV receptor (Reyes-del Valle et al., 2005; Thepparit & Smith, 2004). The 37/67-kDa high-affinity laminin receptor promote the JEV infection in mouse microgila BV-2 cell (Thongtan et al., 2012). Several studies suggest that HSP70, HSP90 and Glucose Regulated Protein 78 (GRP78, a member of the HSP family) may contribute to the entry of JEV (Das et al., 2009; Hung et al., 2011; Nain et al., 2016; Thongtan et al., 2012). HSP70 has also been reported to mediate ZIKV entry and contribute to viral replication (Pujhari et al., 2019). Although HSPs and Laminin receptors are not resident proteins on the cell membrane, they play important roles in viral entry but often require co-receptors to facilitate internalization.

3.1.5. Other putative cellular receptors

Besides the common receptor families for multiple flaviviruses, many specific receptors for certain flaviviruses have also been identified. The NK-activating receptor NKp44 directly interacts with domain III of the envelope protein of WNV and DENV on NK cells, resulting in NK degranulation (Hershkovitz et al., 2009). The Prohibitins (PHB) were demonstrated to facilitate the entry of DENV-2 into insect cells (Kuadkitkan et al., 2010). Another study indicates that Prohibitin-1/2 mediates the entry of DENV-3 into neuronal cells (Sharma et al., 2020). Claudin-1, a transmembrane tight junction protein, interacts with prM/M proteins for DENV entry, despite the M protein having relatively little exposure on the viral particle's surface (Che et al., 2013). One study suggested that DENV-2 may recognize neutral glycosphingolipids, nLc4Cer, in both mammalian and mosquito cells (Wichit et al., 2011). Using chemical proteomics, the Neural Cell Adhesion Molecule (NCAM1) was identified as a potential ZIKV receptor, facilitating viral infection in glioblastoma and U-251 cells (Srivastava et al., 2020). For JEV, several proteins have been identified as entry factors. A recent study indicates that LDLR, a well-known receptor for alphaviruses, also facilitates JEV infection in mammalian cells (Huang et al., 2021). Defensin in mosquitoes binds to the viral E protein and mediates viral entry with the lipoprotein receptor-related protein 2 (LRP2) on the cell surface (Liu et al., 2020). The anti-CD4 antibody impairs JEV entry into microglial cells, indicating that CD4 may be involved in JEV infection (Thongtan et al., 2012; Wang et al., 2023). Vimentin, PLVAP (Plasmalemma vesicle-associated protein), and GKN3 (Gastrokine 3) have been identified as entry factors for JEV through pull-down experiments. The vimentin-Fc fusion protein suppresses JEV infection by competition (Zhang, Chen, et al., 2024). Overexpression of these genes increases viral load, while silencing them reduces it (Liang et al., 2011; Mukherjee et al., 2018)

3.2. The receptors of mosquito-borne alphaviruses

Alphaviruses, belonging to the *Togaviridae* family, are another major group of mosquito-borne viruses. The E1 and E2 proteins of alphaviruses forms heterodimers and trimers on the viral membrane, which can be responsible for receptor binding as a whole or provide residues for interaction alone (Cao et al., 2023; Kim & Diamond, 2023). Compared to flaviviruses, research on mosquito-borne alphavirus receptors is more enveloped. Numerous alphaviruses have identified receptors or receptor families, and the structures of viral surface protein-receptor complexes have been elucidated, providing valuable insights into alphavirus biology and aiding in the development of targeted therapies.

3.2.1. LDLR family

The Low-Density Lipoprotein Receptor (LDLR) family consists of cell surface receptors that play a crucial role in the uptake of cholesterol and other lipids into cells. These proteins are essential for maintaining cholesterol homeostasis and are involved in various cellular processes and pathogen invasions (Go & Mani, 2012). Recently, multiple members of the LDLR family have been identified as receptors for various

alphaviruses. The Low-Density Lipoprotein Receptor Class A Domain-Containing (LDLRAD3) has been identified as an attachment and entry receptor for Venezuelan equine encephalitis virus (VEEV) (Ma et al., 2020, 2021). Slightly later, the very-low-density lipoprotein receptor (VLDLR) and the apolipoprotein E receptor 2 (ApoER2) were identified as new entry receptors for Semliki Forest virus (SFV), EEEV and Sindbis virus (SINV). The interaction involves multiple LDL type A (LA) domains binding to the surface E proteins, and a receptor-associated protein (RAP) decoy can block virus infection (Adams et al., 2024; Cao et al., 2023; Clark et al., 2021). Based on CRISPR-Cas9 screening, in addition to the previously mentioned LDL family protein, the low-density lipoprotein receptor (LDLR) was also identified as a candidate receptor for EEEV, Western equine encephalitis virus (WEEV) and SFV. LDLR facilitates the release of virions for endosomal fusion, enabling penetration into the cytosol (Ma et al., 2024). In another recent publication, the LDLR is identified as a common receptor for multiple alphaviruses, including Getah virus (GETV), SFV, Ross River virus (RRV) and Bebaru virus (BEBV) (Zhai et al., 2024). The class A domains of LDL family proteins exhibit considerable conservation in their binding with alphaviruses. They can serve as receptors for various alphaviruses in both vertebrate hosts and mosquito vectors, suggesting a general binding pattern between LDL proteins and alphaviruses.

3.2.2. MXRA8 receptor

Besides the LDLR family proteins, matrix remodeling-associated 8 (MXRA8), is another important entry receptor for multiple alphaviruses. Mammalian MXRA8 serves as an entry receptor for CHIKV and several other arthritogenic alphaviruses, which are classified into the Semliki Forest (SF) antigenic group, including Mayaro virus (MAYV), SFV, O'nyong'nyong virus (ONNV), and RRV (Basore et al., 2019; Song et al., 2019; Zhang et al., 2018). Interestingly, alphaviruses in the WEE antigenic group, including WEEV and SINV, employ avian MXRA8 as an entry receptor but do not interact with mammalian MXRA8 (Zimmerman, Zimmerman, et al., 2023). The binding models and regions revealed by the structures of antigen-receptor complexes are distinct in these two cases (Basore et al., 2019; Kim et al., 2020; Song et al., 2019; Zimmerman, Zimmerman, et al., 2023).

3.2.3. Phosphatidylserine receptors

The phosphatidylserine receptor also contributes to the binding of multiple alphaviruses. A study generated virus-like particles (VLPs) pseudotyped with surface invasion proteins from various viruses on a retrovirus backbone to assess their cell entry abilities. The results showed that EEEV, SINV, and RRV had increased entry ability in TIM-1 overexpressing HEK293 cells. Overexpression of Axl (a TAM receptor) promoted CHIKV and EEEV infection, while TIM3/4 only enhanced EEEV entry (Jemielity et al., 2013; Moller-Tank & Maury, 2014). A recent study demonstrated that TIM-1, but not Axl, facilitates CHIKV entry in HEK293T cells. TIM-1 directly binds to CHIKV, and its overexpression or silencing enhances or reduces infection accordingly (Kirui et al., 2021). CD300a, a member of the PS family, has also been reported to bind to pseudotype lentivirus with the envelope proteins from SINV (Morizono & Chen, 2014).

3.2.4. Integrins and glycosaminoglycans

Few studies have explored the binding between integrins and alphaviruses. An early study indicates that RRV utilizes the collagen-binding α1β1 integrin as a cellular receptor. Soluble α1β1 integrin binds to RRV, and infection can be inhibited by competitor collagen IV (La Linn et al., 2005). Glycosaminoglycans, including HS and HSPG, have been reported as common attachment factors for multiple alphaviruses. When SINV and SFV are passaged on BHK-21 cells, they gain high affinity for heparan sulfate, suggesting they may utilize HS for infection (Ryman et al., 2007; Smit et al., 2002). Heparan sulfate has been shown to bind with EEEV, enhancing neurotoxicity in mice. Additionally, HS exhibits a weak interaction with WEEV and no interaction with RRV (Gardner et al., 2011) A glycan microarray analysis revealed that CHIKV binds to longer GAG chains of heparin and heparan sulfate (McAllister et al., 2020). Another study indicates that heparan sulfate may serve as an independent receptor for CHIKV in HAP1 cells (Ballista et al., 2023).

3.2.5. Other putative cellular receptors

The C-type lectin family proteins, which exhibit wide-ranging interactions with flaviviruses, show rare connection with alphaviruses. Only one early study indicated that overexpression of DC-SIGN and L-SIGN enhances the binding of SINV to THP1 cells (Klimstra et al., 2003). Also, the Natural Resistance-Associated Macrophage Protein (NRAMP) was identified as an entry receptor for SINV infection in Drosophila cells, while its homolog, NRAMP2, mediates binding and infection in mammalian cells (Rose et al., 2011). Laminin receptors facilitate SINV infection in hamster cells. Anti-laminin antibodies block SINV binding to mosquito cells, suggesting laminin may serve as a broad-range receptor (Wang et al., 1992). Also, The C-terminal of human laminin-binding protein (LBP) binds to the VEEV E2 protein, and related antibodies reduce viral replication (Malygin et al., 2009). Using a two-dimensional virus overlay assay, prohibitin (PHB) was identified as a binding factor of CHIKV in microglial cells, confirmed by RNAi and antibody blocking experiments (Wintachai et al., 2012). The CD147 complex, a member of the immunoglobulin (Ig) superfamily with similar domains to MXRA8, has been reported to be involved in the entry of CHIKV and several other alphaviruses in HEK293T cells, as identified by affinity purification mass spectrometry (De Caluwé et al., 2021). The ATP synthase β subunit (ATPSβ), one component of the F_1-F_o ATP synthase, was found as one of the CHIKV E2 binding protein in C6/36 cells through VOPBA assay. The researchers used RNAi, antibody inhibition assay and confocal microscope imaging to revealed the receptor functions (Fongsaran et al., 2014).

3.3. The receptors of mosquito-borne Bunyavirus

Arboviruses in the *Bunyavirales* order are transmitted by various invertebrate vectors, including mosquitoes, ticks, and midges and other arthropods. Although not the unique vectors, mosquitoes play a significant role in the persistence of these viruses in nature. The primary surface proteins of Bunyavirus responsible for cell entry are Gn and Gc, which form heterodimers and are tightly and orderly arranged on the surface of viral particles. Current studies on receptor identification for mosquito-borne viruses in *Bunyavirales* are limited.

The common viral attachment factor heparan sulfate (HS) also serves as a receptor for RVFV. Lacking, removing, or competitive inhibition of HS on CHO cells impairs RVFV infection (de Boer et al., 2012). Disruption of the biogenesis and transport of HSPG confers resistance to RVFV infection, indicating HSPG's positive role in RVFV entry, but this phenomenon is restricted to certain cell types (Riblett et al., 2016).The low-density lipoprotein receptor-related protein (LRP1), a member of the LDLR family, has been identified as interacting with RVFV and promoting viral infection. The Gn protein of RVFV could directly binds to LRP1 (Ganaie et al., 2021). LRP1 may also be a receptor for OROV; lacking LRP1 impairs viral infection in cells and treating cells with the high-affinity LRP1 ligand RAP protein reduces OROV infection (Schwarz et al., 2022). DC-SIGN has been demonstrated as the receptor for multiple phleboviruses. RVFV is the most well-known mosquito-transmitted phlebovirus, and DC-SIGN directly binds to its surface glycoproteins (Lozach et al., 2011). L-SIGN, a homolog of DC-SIGN, also plays a role in RVFV infection. HeLa cells overexpressing L-SIGN exhibit high infectivity to RVFV (Léger et al., 2016).

4. The receptors shared in mosquitoes and vertebrates

The life cycle of mosquito-borne viruses requires cross-species transmission between invertebrate vectors and vertebrate hosts. When infected mosquitoes bite vertebrates, these viruses must quickly enter

primary target cells, such as Langerhans cells, macrophages, and dendritic cells, through specific mammalian receptors. Conversely, when mosquitoes acquire viruses from an infected vertebrate, the viruses must establish an initial infection in the midgut by binding to specific mosquito receptors (Ruckert & Ebel, 2018). To facilitate cross-species transmission, it is reasonable that mosquito-borne viruses share a common protein family as their receptor in both vertebrates and mosquitoes. Indeed, several identified mosquito-borne virus receptors have homologs in both vertebrates and mosquitoes, functioning as entry receptors.

For the alphaviruses, the homologs of VLDLR and ApoER2 in mosquitoes, which belong to the LDLR family, also facilitate SFV and EEEV infection in mosquito cells (Clark et al., 2021). LDLR family members are large proteins with multiple domains, including LDL-class A (LA) and LDL-class B (LB). Currently, only LDL-class A domains exhibit binding affinity to mosquito-borne viruses. Structural information on LDLR protein-alphavirus complexes reveals that binding affinity between LDLR domains and viral surface proteins varies significantly, with a single amino acid substitution determining the interaction (Adams et al., 2024; Basore et al., 2021; Cao et al., 2023; Ma et al., 2021). LDLR family proteins are widespread in various mosquito species and vertebrates, with their LDLR LA domains exhibiting only minor differences. This variation in LDL domains may contribute to the preference of different mosquito-borne alphaviruses for different hosts. This hypothesis still requires experimental evidence but uncovering the patterns among hundreds of LA domains and various alphaviruses will greatly enhance our understanding of mosquito-borne virus entry and promote the discovery of new LDL receptors.

Another large protein family, the C-type lectins (CTL), also serves as receptors for flaviviruses in both mosquitoes and vertebrates. DC-SIGN, L-SIGN, and mannose receptors are important entry receptors for multiple flaviviruses and several alphaviruses. Mosquito C-type lectins (mosGCTLs) interact with various flaviviruses, including WNV, DENV, and JEV, serving as intermediate receptors to bring the virus to the target cell membrane. The glycans on the surface of flaviviral E proteins contribute to the interaction with receptors, as shown by the resolved structure between DC-SIGN and DENV (Pokidysheva et al., 2006). This suggests that specific glycosylation patterns may determine the interaction between various flaviviruses and their C-type lectin receptors. Besides the LDLR and CTL families, the highly glycosylated heparan sulfate proteins and laminin receptors may also serve as attachment factors in both mosquitoes and vertebrates. However, these factors typically enhance viral particle capture by cells without triggering entry, while co-receptors or specific entry receptors are needed for viral entry.

Despite the increasing discovery of shared receptors between mosquitoes and vertebrates, many receptors identified in mammals do not have homologs in mosquitoes. This suggests that these viruses may employ completely different strategies to enter these specific hosts. On the other hand, current investigations into protein homologs mostly rely on amino acid sequence identity or similarity. However, entirely different peptides may generate similar protein structures and perform the same functions over millions of years' evolution. Utilizing AI-based protein structure prediction tools may discover novel "functional homologs" in mosquitoes and vertebrate hosts, expanding our understanding of cross-species common receptors.

5. Antiviral strategies based on mosquito-borne viral receptors

Receptors play a critical role in the entry of viruses into host cells, making them prime targets for therapeutic interventions. By understanding and identifying these receptors, researchers can design inhibitors that block viral entry, preventing infection and subsequent disease progression. One widely used approach is receptor decoys, which compete with receptors on the cell membrane. Some studies have shown that soluble receptors (ectodomain fusion proteins) can be developed as decoys. These soluble receptors mimic the virus's natural binding sites, thereby inhibiting its ability to attach to actual cell surface receptors. This strategy has proven effective in blocking the entry and subsequent infection of various mosquito-borne viruses, including DENV, CHIKV, SFV and EEEV (Chen et al., 2017; Clark et al., 2021; Ma et al., 2024; Zhang et al., 2018). Antibodies that disrupt viral-receptor interactions could also reduce viral infectivity. For instance, blocking Mxra8 with anti-Mxra8 monoclonal antibody reduced CHIKV and ONNV infection and disease symptoms in C57BL/6 mice (Zhang et al., 2018). Small molecule inhibitors designed to interfere with receptor binding sites on the virus could prevent viral attachment and entry, thus inhibiting viral reproduction (Abdelnabi & Delang, 2020). Moreover, targeting key factors in the cell signaling pathways associated with these receptors also presents a promising approach to reducing or blocking viral infection. For example, inhibiting specific kinases in the TAM receptor signaling pathways has been shown to impede viral replication (Wang et al., 2021).

Developing control strategies targeting universal receptors in both vertebrate hosts and insect vectors is particularly valuable for managing mosquito-borne viruses. Such strategies could offer dual benefits: 1) Reducing viral load in vertebrate hosts: Targeting universal receptors in vertebrates can alleviate symptoms and reduce disease severity. 2) Interrupting viral acquisition in mosquitoes: Targeting universal receptors in mosquitoes can disrupt the viral transmission cycle and reduce the number of infected vectors in nature. Although not widely implemented, these dual-targeted control strategies represent a promising area for future research. In mammals, many therapeutic strategies targeting mosquito-borne virus receptors have proven effective. However, research on blocking mosquito receptors remains relatively limited. The antiserum against several mosGCTLs can interrupt the acquisition of DENV, WNV, and JEV by *Aedes* or *Culex* mosquitoes (Cheng et al., 2010; Liu et al., 2014, 2017a). Targeting the conserved regions of C-type lectin receptors shared by both mammals and mosquitoes can lead to the development of dual-action strategies for controlling mosquito-borne flaviviruses. Additionally, LDL class-A (LA), which serves as a receptor for alphaviruses in both mammals and mosquitoes, is another ideal candidate for dual-effect therapies. More functional experiments using "vertebrate-mosquito-vertebrate" transmission animal models are needed to demonstrate the feasibility and potential of these new strategies. Targeting both vertebrate and mosquito receptors could improve therapeutic outcomes for infected individuals and reduce disease incidence through vector control, offering significant potential for future advancements.

6. Conclusion and perspective

Viral receptors are pivotal in influencing infection and transmission, making them a central focus in virology research. The identification of new viral receptors is expected to accelerate with technologies like CRISPR-Cas9 screening libraries, AI, and big data models. However, it is crucial to validate all identified receptors through comprehensive biochemical and biological functional experiments. Some receptors mentioned in this review still require further functional validation and cellular tropism analysis of viral infection in future studies. In addition, more future work should be conducted to elucidate the mechanisms of how the Mosquito-Borne Arbovirus entry/fusion machine is activated following the engagement of entry receptors. In summary, the continuous discovery of new receptors for mosquito-borne viruses will enhance our understanding of virus-host interactions. Developing receptor-targeted therapeutic strategies, especially those targeting universal entry receptors conserved across species, will significantly improve the control and prevention of mosquito-borne diseases in nature.

CRediT authorship contribution statement

Jianying Liu: Writing – original draft. **Yixin Quan:** Resources, Data curation. **Hua Tong:** Resources, Formal analysis. **Yibin Zhu:** Formal analysis. **Xiaolu Shi:** Funding acquisition. **Yang Liu:** Writing – review & editing, Writing – original draft. **Gong Cheng:** Writing – review &

editing.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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