



## Association of Plasma Membrane Microdomains with HIV-1 Assembly

Sakurai Roy\*

Department of Virology, University of Tokushima, Tokushima, Japan

### DESCRIPTION

The plasma membrane's micro domain-based compartmentalization is involved in many stages of the HIV-1 life cycle. In particular, it has been proposed that these micro domains act as preformed platforms that facilitate the concentration of viral components (such as Gag and Env) or the delivery of these proteins to particular locations in cells during events in the late phase of the HIV-1 life cycle, such as assembly and cell-to-cell transmission. Nevertheless, current research suggests that Gag, by its membrane-binding and multimerization activities, actively participates in structuring microdomains rather than simply being a passive passenger of those structures. In this article, we concentrate on current research on the active function that Gag plays during the association of microdomains. We will also go through the significance of plasma membrane microdomains and large-scale domains in cell-to-cell transmission in light of this new perspective [1-3]. The distribution and activity of Env, Nef, and viral receptors are hypothesised to be modulated by microdomains, which may also have an impact on virion infectivity, virion attachment to target cells, and virus-cell fusion.

The viral structural polyprotein Gag is both required and sufficient for the formation of virus-like particles. According to cleavage by the viral protease, HIV-1 Gag is produced as a 55 kDa polyprotein with the following four key structural domains: Matrix (MA), Capsid (CA), Nucleocapsid (NC), and P6. To drive particle formation, its components must cooperate within the context of the full-length Gag polyprotein since proteolytic cleavage mostly takes place after virion assembly and release [4-7]. After cytosolic production, Gag traffics to the site of assembly, attaches to cellular membranes, multimerizes, buds through the membrane, and enlists host components to facilitate membrane scission, releasing an immature particle.

The C-terminal portion of the CA domain (CA-CTD) and NC are two important functional domains that support Gag multimerization. Gag homodimerization is mediated *via* an interface formed by CA-CTD. The NC domain's capacity to bind RNA is considered to play a role in Gag multimerization.

Interestingly, NC may be replaced by heterologous leucine zipper dimerization motifs in Gag multimerization and particle formation. Our results point to a structural role for RNA binding to NC, either as a scaffold or a catalyst for CA dimerization. The Spacer Peptide 1 (SP1) between CA and NC, in addition to CA and NC, is crucial in controlling the multimerization process [8].

The plasma membrane region where the Gag multimers is attached undergoes an outward curvature as a result of higher-order Gag multimerization. The Gag hexameric lattice's natural curvature, which depends on CA for creation, is probably what motivates this phase. This is supported by the fact that certain CA mutations cause a phenotype known as budding arrest, which is characterized by numerous electron-dense patches underneath the plasma membrane. The cellular ESCRT (Endosome Sorting Complexes Needed for Transport) is recruited to assembly virions by interactions with the NC and P6 domains, and it is this complex that drives the release of nascent particles [9].

There are several different microdomains in the plasma membrane. Lipid-lipid, protein-protein, and protein-lipid interactions control the partitioning of membrane components, which compartmentalizes cellular functions. HIV-1 was initially thought to assemble at lipid rafts, as is the case with many different enveloped viruses, because of its sensitivity to cellular cholesterol depletion and the cofractionation of viral components with Detergent-Resistant Membranes (DRM). On the basis of microscopy, it was subsequently suggested that HIV-1 assembly takes place at microdomains that are rich in tetraspanin [10].

### REFERENCES

1. Thormann A, Teuscher N, Pfannmüller M, Rothe U, Heilmann A. Nanoporous aluminum oxide membranes for filtration and biofunctionalization. *Small*. 2007;3(6):1032-1040.
2. Michalewska Z, Michalewski J, Adelman RA, Nawrocki J. Inverted internal limiting membrane flap technique for large macular holes. *Ophthalmol*. 2010;117(10):2018-2025.

**Correspondence to:** Sakurai Roy, Department of Virology, University of Tokushima, Tokushima, Japan, E-mail: Sakuriroy@gmail.com

**Received:** 27-Jan-2023, Manuscript No. JMST-23-20343; **Editor assigned:** 30-Jan-2023, Pre QC No. JMST-23-20343 (PQ); **Reviewed:** 13-Feb-2023, QC No. JMST-23-20343; **Revised:** 20-Feb-2023, Manuscript No. JMST-23-20343 (R); **Published:** 02-Mar-2023, DOI: 10.35248/2155-9589.23.13.332

**Citation:** Roy S (2023) Association of Plasma Membrane Microdomains with HIV-1 Assembly. *J Membr Sci Technol*. 13:332.

**Copyright:** © 2023 Roy S. This is an open access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

3. Hoess A, Teuscher N, Thormann A, Aurich H, Heilmann A. Cultivation of hepatoma cell line HepG2 on nanoporous aluminum oxide membranes. *Acta Biomater.* 2007;3(1):43-50.
4. Heilmann A, Teuscher N, Kiesow A, Janasek D, Spohn U. Nanoporous aluminum oxide as a novel support material for enzyme biosensors. *J Nanosci Nanotechnol.* 2003;3(5):375-379.
5. Shimada N, Sugamoto Y, Ogawa M, Takase H, Ohno-Matsui K. Fovea-sparing internal limiting membrane peeling for myopic traction maculopathy. *Am J Ophthalmol.* 2012;154(4):693-701.
6. Ma Y, Kaczynski J, Ranacher C, Roshanghias A, Zauner M, Abasahl B. Nano-porous aluminum oxide membrane as filtration interface for optical gas sensor packaging. *Microelectron Eng.* 2018;198:29-34.
7. Iwasaki M, Miyamoto H, Okushiba U, Imaizumi H. Fovea-sparing internal limiting membrane peeling *versus* complete internal limiting membrane peeling for myopic traction maculopathy. *Jpn J Ophthalmol.* 2020;64(1):13-21.
8. Wakatsuki Y, Nakashizuka H, Tanaka K, Mori R, Shimada H. Outcomes of Vitrectomy with Fovea-Sparing and Inverted ILM Flap Technique for Myopic Foveoschisis. *J Clin Med.* 2022;11(5):1274.
9. Jani AM, Kempson IM, Losic D, Voelcker NH. Dressing in layers: layering surface functionalities in nanoporous aluminum oxide membranes. *Angew Chem Int Ed Engl.* 2010;49(43):7933-7937.
10. Iida Y, Hangai M, Yoshikawa M, Ooto S, Yoshimura N. Local biometric features and visual prognosis after surgery for treatment of myopic foveoschisis. *Retina.* 2013;33(6):1179-1187.