HNP1-3 bind CD4

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Introduction

Reactome is open-source, open access, manually curated and peer-reviewed pathway database. Pathway annotations are authored by expert biologists, in collaboration with Reactome editorial staff and cross-referenced to many bioinformatics databases. A system of evidence tracking ensures that all assertions are backed up by the primary literature. Reactome is used by clinicians, geneticists, genomics researchers, and molecular biologists to interpret the results of high-throughput experimental studies, by bioinformaticians seeking to develop novel algorithms for mining knowledge from genomic studies, and by systems biologists building predictive models of normal and disease variant pathways.

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Literature references

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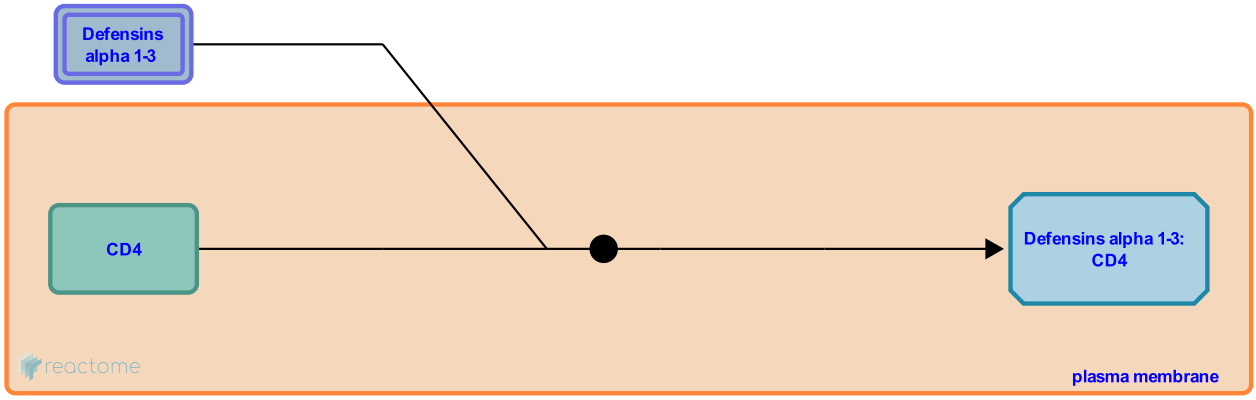
Stable identifier: R-HSA-1471314

Type: binding

Compartments: extracellular region, plasma membrane



Alpha-defensins, theta-defensins and their synthetic analogues the retrocyclins have been shown in numerous studies to have anti-HIV-1 activity (Chang & Klotman 2004). This appears to be mediated via multiple mechanisms including direct viral inactivation and down regulation of host-cell target co-receptors important for viral entry (Furci et al. 2007, Seidel et al. 2010). Further, HNPs 1 3, act as lectins and bind with relatively high affinity to gp120 (KD range, 15.8-52.8 nM) on the HIV-1 envelope and CD4 (KD range, 8.0-34.9 nM) on host target cells, both important molecules for viral entry (Wang et al. 2004). Artificial theta defensins, the retrocyclins, predicted from the human pseudogenes bind with even higher affinity whereas HNP-4 binding is much weaker (Wu et al. 2005). Alpha defensins have been demonstrated to inhibit the binding of gp120 to CD4 thus blocking HIV-1 fusion with its target cells (Furci et al. 2007).

Literature references

Waring, AJ., Lal, RB., Hong, T., Cole, AM., Owen, SM., Wang, W. et al. (2004). Activity of alpha- and theta-defensins against primary isolates of HIV-1. *J Immunol, 173*, 515-20. ↗

Editions

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