

# PILRA binds PIANP, COLLEC12 trimer, NP-DC1, CLEC4G

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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

Fabregat, A., Sidiropoulos, K., Viteri, G., Forner, O., Marin-Garcia, P., Arnau, V. et al. (2017). Reactome pathway analysis: a high-performance in-memory approach. *BMC bioinformatics*, 18, 142. [↗](#)

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Reactome database release: 90

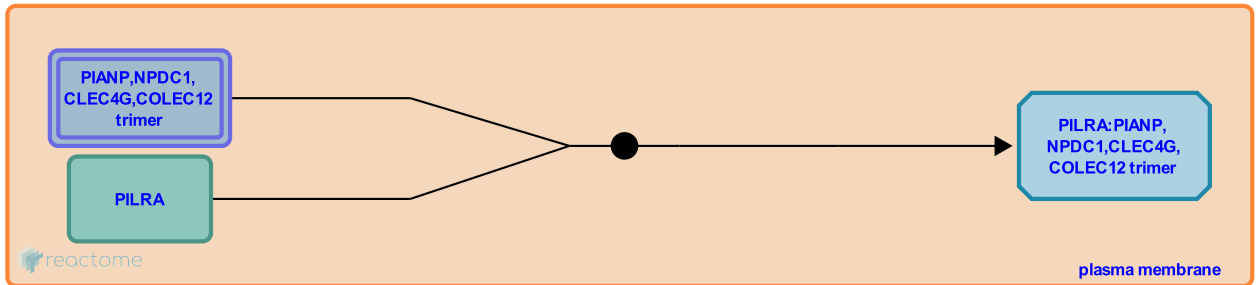
This document contains 1 reaction ([see Table of Contents](#))

**PILRA binds PIANP, COLLEC12 trimer, NPDC1, CLEC4G** [↗](#)

**Stable identifier:** R-HSA-8862090

**Type:** binding

**Compartments:** plasma membrane



Paired immunoglobulin-like type 2 receptor alpha (PILRA) binds to multiple ligands including CD99 (Shiratori et al. 2004), PILR-associating neural protein (PIANP, PANP) (Kogure et al. 2011), Herpes simplex virus-1 glycoprotein B (Sato et al. 2008), Collectin-12 (COLEC12), Neural proliferation differentiation and control protein 1 (NPDC1) and C-type lectin domain family 4 member G (CLEC4G) (Sun et al. 2012). Binding studies suggest that PILR recognizes a complex ligand domain involving both silica acid and protein motif(s). Thus, PILR is evolved to engage multiple ligands with common molecular determinants to modulate myeloid cell functions in anatomical settings where PILR ligands are expressed. The precise function of PILRa-Ligand interaction is not well understood (Sun et al. 2012). PILRa negatively regulates inflammation and keeps myeloid system in check. Pilra KO mice produce more pathogenic cytokines during inflammation and are prone to enhanced autoimmune arthritis. Correspondingly, anti-PILRa mAb ameliorated inflammation in mouse arthritis models and suppressed the production of proinflammatory cytokines (Sun et al. 2014).

**Literature references**

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Arase, H., Ogasawara, K., Saito, T., Lanier, LL., Shiratori, I. (2004). Activation of natural killer cells and dendritic cells upon recognition of a novel CD99-like ligand by paired immunoglobulin-like type 2 receptor. *J. Exp. Med.*, 199, 525-33. [↗](#)

**Editions**

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