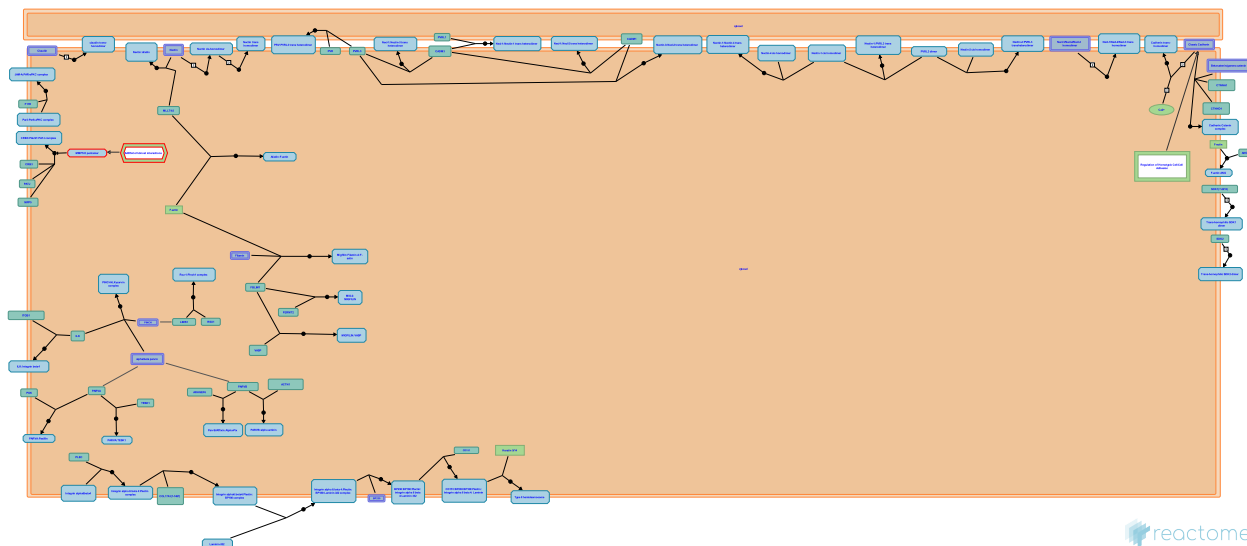


Cell junction organization



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This is just an excerpt of a full-length report for this pathway. To access the complete report, please download it at the [Reactome Textbook](https://reactome.org/page/about-us).

10/10/2024

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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Sidiropoulos, K., Viteri, G., Sevilla, C., Jupe, S., Webber, M., Orlic-Milacic, M. et al. (2017). Reactome enhanced pathway visualization. *Bioinformatics*, 33, 3461-3467. [↗](#)

Fabregat, A., Jupe, S., Matthews, L., Sidiropoulos, K., Gillespie, M., Garapati, P. et al. (2018). The Reactome Pathway Knowledgebase. *Nucleic Acids Res*, 46, D649-D655. [↗](#)

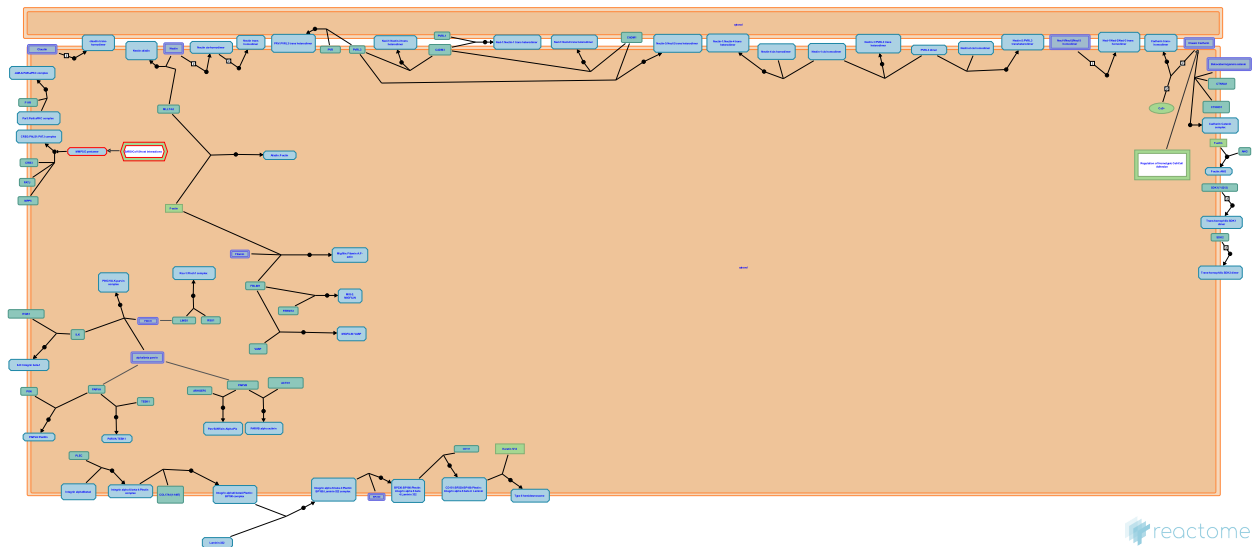
Fabregat, A., Korninger, F., Viteri, G., Sidiropoulos, K., Marin-Garcia, P., Ping, P. et al. (2018). Reactome graph database: Efficient access to complex pathway data. *PLoS computational biology*, 14, e1005968. [↗](#)

Reactome database release: 90

This document contains 4 pathways ([see Table of Contents](#))

Cell junction organization ↗

Stable identifier: R-HSA-446728



Cell junction organization in Reactome currently covers aspects of cell-cell junction organization, cell-extracellular matrix interactions, and Type I hemidesmosome assembly.

Editions

2009-11-17

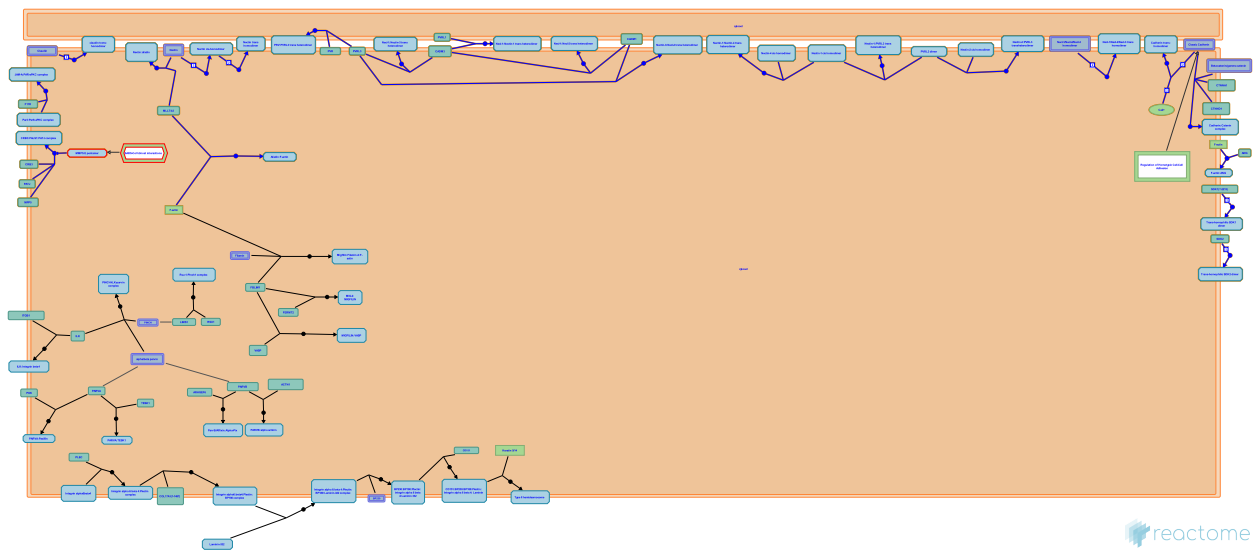
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Cell-cell junction organization ↗

Location: [Cell junction organization](#)

Stable identifier: R-HSA-421270



Epithelial cell-cell contacts consist of three major adhesion systems: adherens junctions (AJs), tight junctions (TJs), and desmosomes. These adhesion systems differ in their function and composition. AJs play a critical role in initiating cell-cell contacts and promoting the maturation and maintenance of the contacts (reviewed in Ebnet, 2008; Hartsock and Nelson, 2008). TJs form physical barriers in various tissues and regulate paracellular transport of water, ions, and small water soluble molecules (reviewed in Rudini and Dejana, 2008; Ebnet, 2008; Aijaz et al., 2006; Furuse and Tsukit, 2006). Desmosomes mediate strong cell adhesion linking the intermediate filament cytoskeletons between cells and playing roles in wound repair, tissue morphogenesis, and cell signaling (reviewed in Holthofer et al., 2007).

Literature references

Matter, K., Balda, MS., Aijaz, S. (2006). Tight junctions: molecular architecture and function. *Int Rev Cytol*, 248, 261-98. ↗

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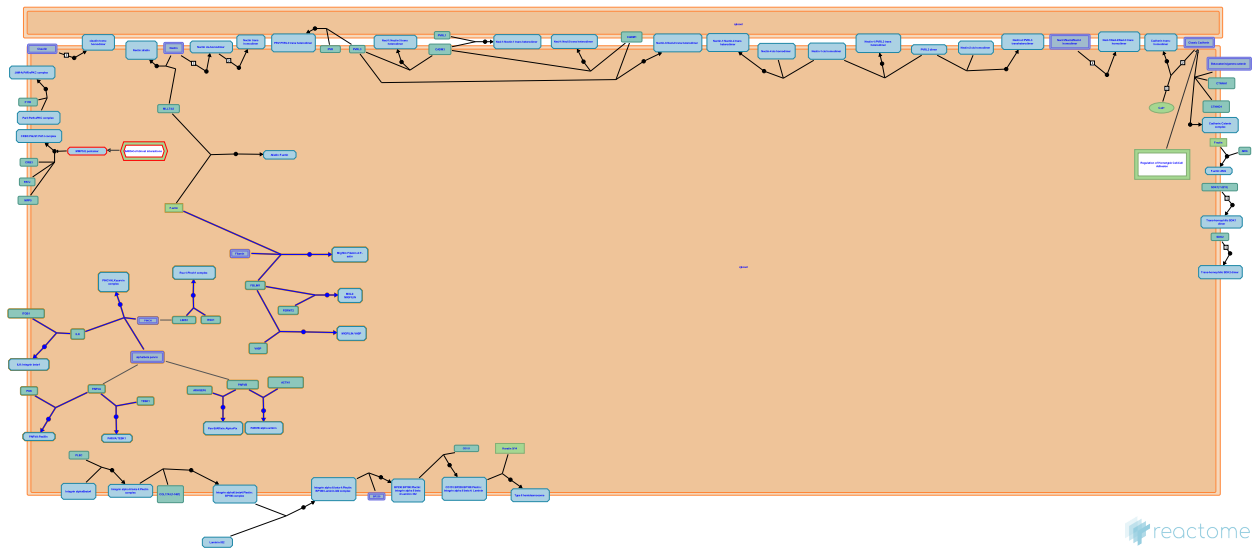
Editions

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Cell-extracellular matrix interactions ↗

Location: [Cell junction organization](#)

Stable identifier: R-HSA-446353



Cell-extracellular matrix (ECM) interactions play a critical role in regulating a variety of cellular processes in multicellular organisms including motility, shape change, survival, proliferation and differentiation. Cell-ECM contact is mediated by transmembrane cell adhesion receptors, such as integrins, that interact with extracellular matrix proteins as well as a number of cytoplasmic adaptor proteins. Many of these adaptor proteins physically interact with the actin cytoskeleton or function in signal transduction.

Several protein complexes interact with the cytoplasmic tail of integrins and function in transducing bi-directional signals between the ECM and intracellular signaling pathways (reviewed in Sepulveda et al., 2005).

Early events that are triggered by interactions with ECM, such as formation/turnover of Focal Adhesions, regulation of actin dynamics and protrusion of lamellipodia to promote cellular spreading and motility are modulated by PINCH- ILK- parvin complexes (see Sepulveda et al., 2005). A number of partners of the PINCH-ILK-parvin complex components have been identified that regulate and/or mediate the functions of these complexes (reviewed in Wu, 2004). Interactions with some of these partners modulate cytoskeletal remodeling and cell spreading.

Literature references

Wu, C. (2004). The PINCH-ILK-parvin complexes: assembly, functions and regulation. *Biochim Biophys Acta*, 1692, 55-62. ↗

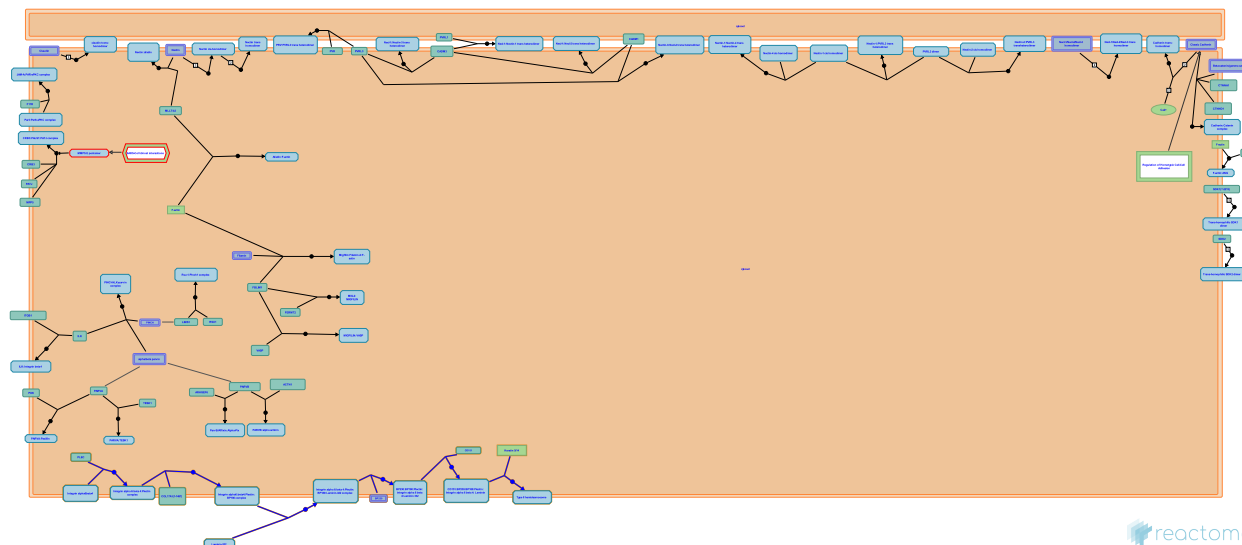
Editions

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2009-11-12	Reviewed	Wu, C.

Type I hemidesmosome assembly [↗](#)

Location: Cell junction organization

Stable identifier: R-HSA-446107



Hemidesmosomes (HDs) are specialized multiprotein junctional complexes that connect the keratin cytoskeleton of epithelial cells to the extracellular matrix and play a critical role in the maintenance of tissue structure and integrity (reviewed in Litjens et al., 2006). HDs mediate adhesion of epithelial cells to the underlying basement membrane in stratified squamous, transitional and pseudostratified epithelia (Jones et al., 1994 ; Borradori and Sonnenberg, 1996). Classical Type I HDs are found in stratified and pseudo-stratified epithelia, such as the skin, and contain $\alpha 6\beta 4$, plectin, tetraspanin CD151 and the bullous pemphigoid (BP) antigens BP180 and BP230 (reviewed in Litjens et al., 2006). While HDs function in promoting stable adhesion, they are highly dynamic structures that are able to disassemble quickly, for example, during cell division, differentiation, or migration (see Margadant et al, 2008).

Literature references

Litjens, SH., Sonnenberg, A., de Pereda, JM. (2006). Current insights into the formation and breakdown of hemidesmosomes. *Trends Cell Biol.* 16, 376-83. [↗](#)

Editions

2009-11-04	Authored, Edited	Matthews, L.
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