1. [The Roles of Tricellular Tight Junction Protein Angulin-1/Lipolysis-Stimulated Lipoprotein Receptor (LSR) in **Endometriosis** and Endometrioid-Endometrial Carcinoma.](https://pubmed.ncbi.nlm.nih.gov/34944960/)

Shimada H, Kohno T, Konno T, Okada T, Saito K, Shindo Y, Kikuchi S, Tsujiwaki M, Ogawa M, Matsuura M, Saito T, Kojima T.Cancers (Basel). 2021 Dec 17;13(24):6341. doi: 10.3390/cancers13246341.PMID: 34944960 **Free PMC article.** Review.

## Abstract

Tight junction proteins play roles beyond permeability barriers functions and control cell proliferation and differentiation. The relation between tight junctions and the signal transduction pathways affects cell growth, invasion and migration. Abnormality of tight junction proteins closely contributes to epithelial mesenchymal transition (EMT) and malignancy of various cancers. Angulin-1/lipolysis-stimulated lipoprotein receptor (LSR) forms tricellular contacts that has a barrier function. Downregulation of angulin-1/LSR correlates with the malignancy in various cancers, including endometrioid-endometrial carcinoma (EEC). These alterations have been shown to link to not only multiple signaling pathways such as Hippo/YAP, HDAC, AMPK, but also cell metabolism in ECC cell line Sawano. Moreover, loss of angulin-1/LSR upregulates claudin-1, and loss of apoptosis stimulating p53 protein 2 (ASPP2) downregulates angulin-1/LSR. Angulin-1/LSR and ASPP2 concentrate at both midbody and centrosome in cytokinesis. In EEC tissues, angulin-1/LSR and ASPP2 are reduced and claudin-2 is overexpressed during malignancy, while in the tissues of endometriosis changes in localization of angulin-1/LSR and claudin-2 are seen. This review highlights how downregulation of angulin-1/LSR promotes development of endometriosis and EEC and discusses about the roles of angulin-1/LSR and its related proteins, including claudins and ASPP2.

**Keywords:**AMPK; ASPP2; EMT; HDAC; Hippo pathway; angulin-1/LSR; claudins; endometrioid-endometrial carcinoma; endometriosis; tricellular tight junctions.