PTK2 autophosphorylates downstream of EGFR

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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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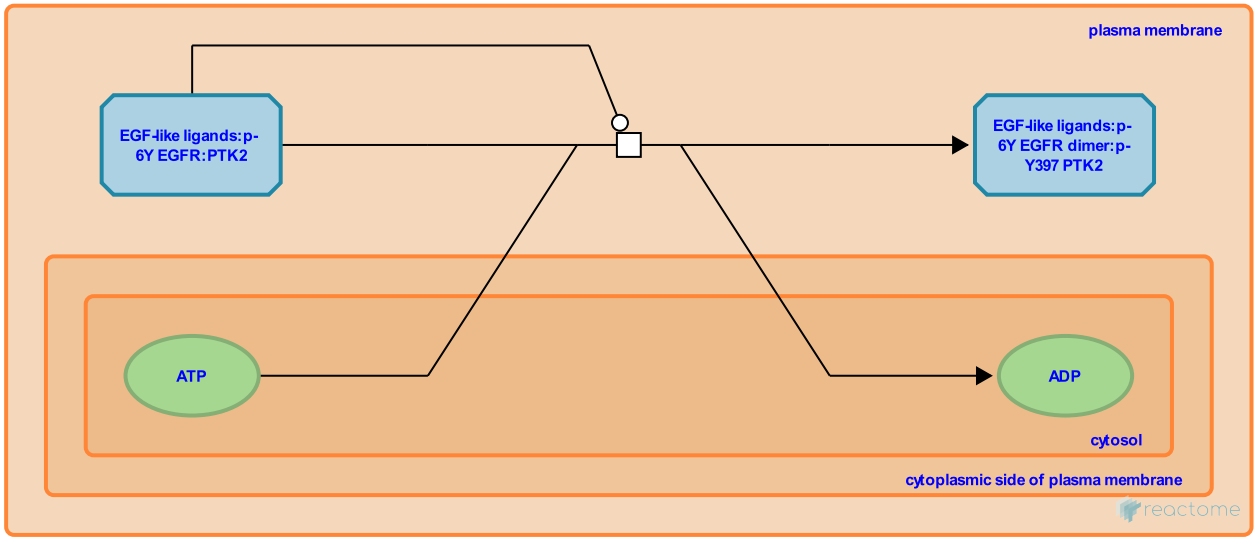
Stable identifier: R-HSA-9625487

Type: transition

Compartments: plasma membrane, cytosol



Stimulation of cells with either E2 (beta-estradiol), tamoxifen or G1 (a GPER1 agonist) enhances EGFR-dependent FAK autophosphorylation at Y397 (Sieg et al, 2000; Liu et al, 2010; Tsai et al, 2013). Signaling occurs through both GPER1 and ER alpha (ESR1) and induces cell proliferation and migration through the EGFR-PI3K-ERK pathway (Liu et al, 2010; Tsai et al, 2013; reviewed in Zhu et al, 2018). FAK autophosphorylation is also required for FOS (c-fos) induction downstream of E2 (Liu et al, 2010; Tsai et al, 2013; Maggiolini et al, 2004; Vivacqua et al, 2006a,b).

Literature references

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Editions

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