What would you observe if you fused a G1 cell with a S cell?

A. Mitotic and pulverized chromosomes.

B. Mitotic and compact G1 chromosomes.

C. Mostly non-compact G1 chromosomes.

D. Compact G1 and G2 chromosomes.

What would you observe if you created a transgenic mouse that over expressed p27

A. Normal size mouse with large thymus.

B. Normal size mouse with normal thymus.

C. Normal size mouse with small thymus.

D. Small size mouse with small thymus.

Sister chromatids are held together by

A. Condensin and Cohesin.

B. Condensin.

C. Cohesin.

D. Smc4 and Smc2.

The presence of the MAD2 checkpoint protein on a chromatid occurs due to

A. Unattached Ndc80 proteins

B. An unassembled kinetochore

C. Unattached microtubules

D. Unattached microtubule depolymerase

The separation of chromosomes during telophase is driven by

A. Ndc80 motor proteins

B. Plus end depolymerization of microtubles

C. Minus end depolymerization of microtubules

D. Plus and minus end depolymerization of microtubules

Checkpoints in the cell cycle occur

A. In G1

B. In G1 and G2

C. In G1, G2 and M

D. In G1, G2, M and S

Formation of the contractile ring and cleavage furrow requires

A. Tubulin

B. ATP

C. Actin and Myosin

D. B and C

EGF signaling is transmitted across the plasma membrane by

A. Lipids.

B. Ras.

C. A heterotrimeric G protein.

D. A receptor tyrosine kinase.

E. An SH2 domain contain protein.

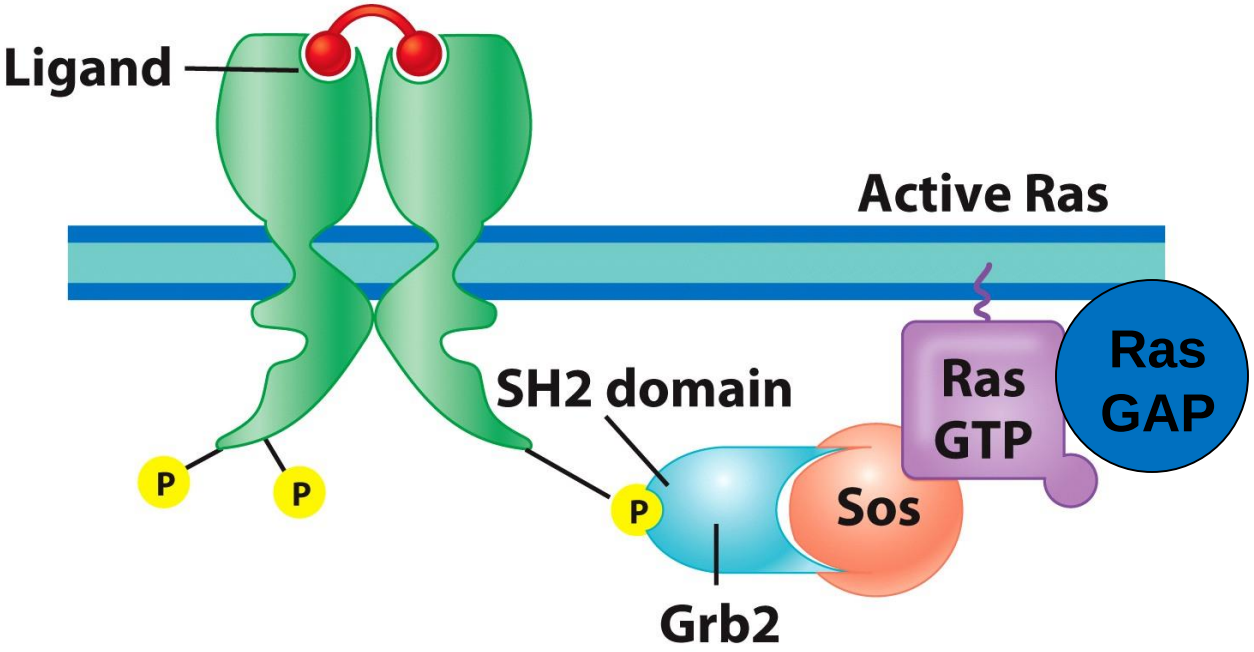
Glucagon is a hormone secreted by the pancreas. It then travels in the blood stream and alters glucose metabolism in the liver. What type of signaling is this?

A.Endocrine signaling.

B.Paracrine signaling.

C.Autocrine signaling.

D.Signaling via cell-cell contact.



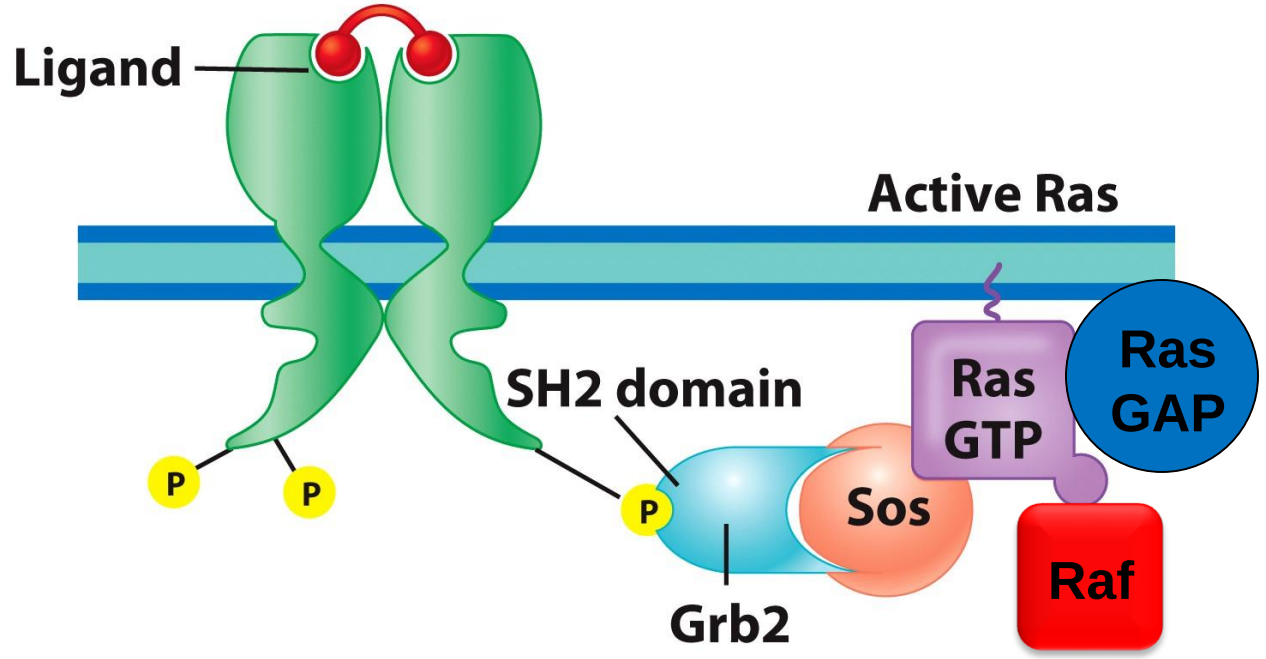

What protein-protein interaction is missing?

A. RasGEF

B. RasGAP

C. ERK

D. MAPKK





What protein-protein interaction is missing?

A. RAF

B. RTK

C. ERK

D. MAPKK

What would cause the same phenotype as inactivation of SOS, the Ras GEF?

A. Ras that is stuck in a GTP bound state.

B. Grb2 (the SH2 containing protein) can bind to EGFR even when EGFR is not phosphorylated.

C. An EGF receptor that dimerizes even in the absence of ligand.

D. Raf is inactive.

E. A drug that mimics the effect of EGF on EGFR.

Which feature is found in the Insulin receptor, but NOT in EGF receptor?

A. Trans-autophosphorylation.

B. Generation of phosphotyrosine residues.

C. A disulfide linked receptor dimer.

D. Recruitment of downstream signaling molecules.

You have created a muscle cell line in which PKB is locked in a phosphorylated state. What would you likely observe in this cell line?

A. Increased PI3Kinase activity.

B. Glycogen synthesis is arrested.

C. Increased glucose uptake.

D. Decreased fusion of Glut4 vesicles with the PM.

Insulin receptors are found on

A. Liver cells.

B. Muscles cells.

C. Fat cells.

D. All of the above.

Type I diabetes is an autoimmune disease that results in loss of the beta cells of the pancreas. These patients could be treated by

A. Insulin injection

B. Drugs that phosphorylate IR

C. Drugs that phosphorylate PKB

D. All of the above.

Type II diabetes is gradual resistance to insulin. These patients could be treated by

A. Insulin injection

B. Glucagon injection

C. Drugs that dephosphorylate PKB

D. A and C.

The insulin receptor activates

A. PKB

B. RAF

C. PI3K

D. All of the above

PKB increases cellular glucose uptake by upregulating

A. Glycogen synthesis

B. Glucose transporter activity

C. Fusion of transporter vesicles

D. A and C.

G proteins are turned off by?

A. The production of cAMP.

B. Hydrolysis of bound GTP.

C. The dissociation of the Beta and Gamma subunits.

D. Ligand binding to receptor.

E. phosphorylation.

GPCRs are turned off by?

A. Binding of cAMP.

B. Hydrolysis of bound GTP.

C. Arrestin binding

D. Ligand binding to receptor.

E. Phosphorylation

G protein alpha most tightly binds

A. GPCRs

B. Ligand bound GPCRs

C. Ligand bound GPCRs when they are in the GDP state

D. Ligand bound GPCRs when they are in the GTP state

GPCRs and G protein alpha function together like

A. RAS and RAF

B. RTK and SH2

C. RasGEF and RAS

D. MAPKK and MAPK

E. PI3K and PI(4,5)P2

Adenylate cyclases are?

A. soluble proteins.

B. usually membrane bound.

C. can be directly activated.

D. Are activated by GPCRs.

E. B and C.

G proteins are turned off by?

A. The production of cAMP.

B. Hydrolysis of bound GTP.

C. The dissociation of the Beta and Gamma subunits.

D. Ligand binding to receptor.

E. phosphorylation.

Glucagon and Epinephrine do not share this feature?

A. Bind and activate a GPCR.

B. Cause a G protein to exchange GDP for GTP.

C. Binding results in glycogen breakdown.

D. Made by the pancreas.

E. Leads to an increase in cAMP.

PKB/AKT activates the Rab protein involved in GLUT4 exocytosis. If you use a chemical to inhibit PKB activity, what would you likely observe in insulin-treated cells?

A. PI3 kinase is activated.

B. GLUT4 vesicles tether to the plasma membrane.

C. GLUT4 vesicles fuse with the plasma membrane.

D. Increased glucose uptake into the cells.

PKA cellular specificity is determined by?

A. PI3 kinase

B. GPCRs

C. PLCs

D. AKAPs

E. A and D

Arrestin regulates GPCR signaling by?

A. Inactivation

B. Promoting GPCR endocytosis

C. Intracellular signaling

D. All of the above

E. None of the above

All of the following are second messengers except

A. cAMP

B. Nitric Oxide

C. GTP

D. DAG

E. IP3

The action of PI-PLC$\beta$ can

A. hydrolyze PI(4,5)$P_2$

B. be activated by G$\alpha$

C. produce IP3

D. produce DAG

E. all of the above

Inhibition of PI-PLCβ will

A. produce more DAG

B. decrease cystosolic Ca++

C. increase IP3 receptor activity

D. produce more IP3

E. all of the above

GPCRs can activate ERK

A. When bound to arrestin

B. When bound to arrestin if endocytosed

C. Via PI3K

D. Via RAS

E. B and D

GPCRs can inhibit RTK signaling

A. by activating RAS

B. by inhibiting RAS

C. by inhibiting RAF

D. by inhibiting ERK

Divergent signaling occurs

A. when receptors activate the same pathway

B. when receptors inhibit the same pathway

C. when a receptor activates different pathways

D. any of the above

You are studying the effect of 2 recently discovered growth factors that bind to different receptors and activate PKA. It is likely that

A. the two signaling pathways are the same

B. the two signaling pathways are convergent

C. the two signaling pathways are divergent

D. the two signaling pathways experience cross talk

E. none of the above

Apoptosis occurs during

A. Embryo development

B. Cell turnover in adult tissue

C. T-cell clonal selection

D. Tissue atrophy

E. All of the above

Apoptosis causes the following

A. RNA fragmentation

B. DNA fragmentation

C. Mitochondrial division

D. Membrane blebs

E. B and D

During development apoptotic cells are phagocytosed by

A.Immune cells

B. Macrophages

C. Microphages

D. B cells

E. A and B

Apoptotic release of cytochrome c occurs

A. via the Bcl-2 MOMP complex

B. during extrinsic apoptosis

C. after caspase 9 activation

D. After caspase 8 activation

E. A and D

Both extrinsic and intrinsic apoptosis require

A. caspase 8 activation

B. caspase 9 activation

C. cytochrome c release

D. caspase 3 activation

E. B and D

You have isolated a mutant human cell line that releases cytochrome c from the mitochondria but does not undergo apoptosis. You could conclude that

A. caspase 8 activation is defective

B. caspase 9 activation is defective

C. Bcl-2 is defective

D. caspase 3 activation is defective

E. either B or D

You have isolated a mutant human cell line that undergoes apoptosis but cannot be phagocytosed. Treating these cells with GFP annexin V will

A. highlight the nuclear membrane

B. show a diffuse cytochrome c distribution

C. show nothing

D. highlight the plasma membrane

E. either B or D

Apoptotic cells show a loss of phosphotidylserine asymmetry at the plasma membrane. This most likely occurs due to

A. activation of a flippase

B. inactivation of a flippase

C. activation of a lipid transferase

D. inactivation of a lipid transferase

E. either A or D