



## Phospho-AKT Pathway Sampler Kit

E051002

Kits Includes	Cat.	Quantity	Application	Reactivity	Source
<a href="#">Akt (Phospho-Ser473) Antibody</a>	E011054-2	50µg/50µl	IHC, WB	Human, Mouse, Rat	Rabbit
<a href="#">p53 (Phospho-Ser15) Antibody</a>	E011094-2	50µg/50µl	IHC, WB	Human,	Rabbit
<a href="#">IKKα (Phospho-Thr23) Antibody</a>	E011129-2	50µg/50µl	IHC, WB	Human, Mouse, Rat	Rabbit
<a href="#">FAK (phospho-Tyr925) Antibody</a>	E011123-2	50µg/50µl	WB	Human, Mouse, Rat	Rabbit
<a href="#">p27Kip1 (Phospho-Thr187) Antibody</a>	E011208-2	50µg/50µl	WB	Human, Mouse, Rat	Rabbit

**Akt** is involved in mediating various biological responses, such as inhibition of Apoptosis and stimulation of cell proliferation. Activation of Akt can begin with several events, mainly the binding of a Ligand to a Receptor in the cell membrane. Most common Ligands activating Akt include Growth factors, Cytokines, Mitogens and Hormones. Insulin and a variety of Growth factors bind to RTK (Receptor Tyrosine Kinase) and cause autophosphorylation of tyrosine residues on the intracellular domain of the receptor. PI3K (Phosphoinositol 3-Kinase) is recruited to the phosphotyrosine residues (consensus sequence pYXXM) via SH2 domains in the regulatory domain (p85), and is therefore targeted to the inner cell membrane. Binding of the p85 subunit of PI3K to the phosphorylated RTK leads to conformational changes in the catalytic domain of PI3K (p110) and consequent kinase activation. PI3K can be activated by Ras. Insulin can also activate PI3K via IRS1 (Insulin Receptor Substrate-1). GPCR (G-Protein-Coupled Receptor) also activates PI3K through GN-Beta (Guanine Nucleotide-Binding Protein-Beta) and GN-Gamma (Guanine Nucleotide-Binding Protein-Gamma) subunits of G-proteins. Cytokines can also activate PI3K via JAK1 (Janus Kinase-1). In B-Cells, PI3K is activated by BCR (B-Cell Receptor) via SYK (Spleen Tyrosine Kinase) and BCAP (B-Cell Receptor Associated Protein). PI3K then phosphorylates membrane bound PIP2 to generate PIP3. The binding of PIP3 to the PH domain anchors Akt to the plasma membrane and allows its phosphorylation and activation by PDK1 (Phosphoinositide-Dependent Kinase-1). DNA-PK, CDC37 (Cell Division Cycle-37), HSP90 (Heat Shock Protein-90KD) and PKC $\beta$  (Protein Kinase-C-Beta) are also reported to phosphorylate Akt. Integrins also activates Akt via **FAK** (Focal Adhesion Kinase), Paxillin and ILK (Integrin-Linked Kinase). Akt can also be activated in response to a variety of cellular stress, such as heat shock, administration of ultra violet light, ischemia (a decrease in blood supply), hypoxia (oxygen deficiency), hypoglycemia (abnormally low level of glucose in the blood) and oxidative stress. The activity of Akt is negatively regulated by PTEN (Phosphatase and Tensin Homolog), SHIP (SH2-Containing Inositol Phosphatase) and CTMP (Carboxyl- Terminal Modulator Protein). The actions of Akt in the cell are numerous and diverse, but all result in anti-apoptosis, or pro-cell proliferation effects. These physiological roles of Akt include involvement in metabolism, protein synthesis, apoptosis pathways, transcription factor regulation and the cell cycle. Akt exerts its effects in the cell by phosphorylating a variety of downstream substrates. The downstream targets of Akt include BAD (BCL2 Antagonist of Cell Death), Caspase9, FKHR (Forkhead Transcriptional Factor),