Supp. Table I: Molecules regulating Akt activity and cardiac functions.

Molecules	Action	Cardiovascular functions	Refs
PI (3,4,5) P3-dependent protein kinase-1 (PDK1)	PDK1 phosphorylates Akt at T308 residue and increases its activity.	PDK1 deficiency induces heart failure in mice due to reduced cardiomyocyte size, cardiac muscle mass and increased sensitivity of cardiomyocytes to hypoxia.	1-3
Mammalian target of rapamycin Complex 2 (mTORC2)	mTORC2 phosphorylates Akt at S473 residue and enhances its catalytic activity.	mTORC2 mediates pro-survival signaling in adult cardiomyocytes.	4, 5
Inositol hexakisphosphate kinase 1 (IP6K1)	IP6K1 produces diphosphoinositol pentakiphosphate which competes with Akt PH domain for binding to PIP3.	Inhibition of IP6Ks enhances Akt activity in mesenchymal stem cells to improve their therapeutic efficacy for treating myocardial infarction.	6, 7
Inositol polyphosphate multikinase (IPKs)	IPK physiologically generates PIP3 to activate Akt.	Expresses mostly in the developing heart. Homozygous IPK2-null mice are smaller than normal controls, and they die embryonically during E9.5–E10.	8, 9
PTEN	PTEN negatively regulates intracellular levels of PIP3 and thereby inhibits Akt activity.	Muscle-specific deletion of PTEN induces basal cardiac hypertrophy, accompanied with mild reduction in LV systolic function. However, cardiac specific deletion of PTEN protects mice from post MI cardiac remodeling.	10-14
SH2 domain-containing inositol 5′-phosphatases (SHIP); Inositol polyphosphate-5-phosphatase (INPP5)	Inositol 5- phosphatases hydrolyze PI (3,4,5) P3 and thereby negatively regulates the growth factor-mediated activation of Akt.	INPP5f knockout mice exhibit exaggerated hypertrophy with reactivation of the fetal genes during cardiac stress.	15-18
PH domain leucine-rich repeat protein phosphatases (PHLPP-1 and -2)	PHLPP protein phosphatases inhibit Akt by dephosphorylating it at S473 residue.	PHLPP-1 knockout cardiomyocytes show increased survival during ischemia/ reperfusion injury due to increased activity of Akt.	19-23
Protein kinase C-related kinase 2 (PRK2)	PRK2 directly binds and inhibits Akt by preventing phosphorylation at T308 and S473 residues.	The functional role of PRK2 in the heart is not known.	24
Protein phosphatase 2A (PP2A)	PP2A dephosphorylates Akt at T308 residue.	PP2A hyper activation leads to contractile dysfunction in the heart	25-28
PHLDA3	PHLDA3, the PH domain-only protein, directly interferes with binding of membrane lipids to Akt, thereby inhibiting Akt activity.	Not studied in cardiomyocytes.	29

TCL1	TCL1 oncogene binds to the PH domain of Akt, promotes nuclear transport, and enhances its kinase activity.	Not studied in cardiomyocytes	30, 31
Zyxin	Zyxin, a cytoskeletal LIM-domain protein targets Akt into the nucleus and promotes Akt activity.	Zyxin promotes cardiomyocyte survival.	32, 33
Carboxyl-terminal modulator protein (CTMP)	CTMP binds to the carboxyl-terminal of Akt at the plasma membrane and inhibits phosphorylation of Akt at T308 and S473.	Not studied in cardiomyocytes.	34, 35
TRAF6 E3 Ligase	TRAF6-mediated lysine-63 ubiquitination of the PH domain promotes Akt membrane recruitment, and phosphorylation upon growth-factor stimulation of cells.	Muscle specific deletion of TRAF6 inhibits skeletal muscle wasting in mice. Endothelial deficiency of TRAF6 attenuated the development of atherosclerosis in a mouse model.	36-38
Skp2-SCF E3 ligase	Skp2-SCF E3 ligase poly-ubiquitinates Akt to promote membrane recruitment in response to EGF stimulation of cells.	Not studied in cardiomyocytes	39
NEDD4-1 E3 ligase	Controls lysine-63 ubiquitin-dependent trafficking of phosphorylated AKT to perinuclear region, where it is released into cytoplasm or imported into the nucleus.	Not studied in cardiomyocytes	40
Tetratricopeptide repeat domain 3 (TTC3) E3 ligase	TTC3 binds to phosphorylated Akt, facilitates its ubiquitination and degradation within the nucleus.	Not studied in cardiomyocytes	41
Poly(ADP-ribose) polymerase-1 (PARP1)	Inhibition of PARP-1 increases Akt phosphorylation.	PARP-1 deficiency protects mice from angiotensin II-induced cardiac hypertrophy, ischemia reperfusion injury and diabetic cardiomyopathy.	42-47
BSD domain–containing signal transducer and Akt interactor (BSTA)	BSTA-Akt1 interaction promotes the mTORC2 - Akt1 association and phosphorylation of Akt1 at S473 during growth factor stimulation.	Not studied in cardiomyocytes	48
Cylindromatosis factor (CYLD)	Deubiquitination of Akt by CYLD suppresses growth factor–mediated ubiquitination, membrane recruitment and activation.	CYLD activation inhibits inflammation and proliferation of vascular cells	49, 50
SIRT1	SIRT1 mediated deacetylation promotes Akt-PIP3 binding, membrane recruitment, and phosphorylation upon growth-factor stimulation.	Chronic SIRT1 activation induces cardiac hypertrophy and heart failure in mouse models. However, short term activation protects the heart from ischemia reperfusion injury.	51-54
SIRT2	SIRT2-mediated deacetylation of Akt	SIRT2 depletion reduces TNFα	

	promotes its phosphorylation.	stimulated necrosis, thus reduces ischemia reperfusion injury in the mouse heart.	55, 56
O-GlcNAcase	O-GlcNAcylations of Akt disrupts its interaction with PDK1, thereby inhibiting Akt phosphorylation at T308 residue.	O-GlcNAcase activation improves contractile function of the diabetic heart.	57-61
Glutaredoxin	Glutaredoxin, a protein disulfide oxidoreductase reduces oxidative modification of Akt, thereby maintaining Akt phosphorylation status.	Glutaredoxin activation protects the heart from adverse ventricular remodeling induced by chronic MI and doxorubicin-induced cardiotoxicity.	62-65

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