

## Richard Roth

Professor of Chemical and Systems Biology, Emeritus

### Bio

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#### ACADEMIC APPOINTMENTS

- Emeritus Faculty, Acad Council, Chemical and Systems Biology

#### PROFESSIONAL EDUCATION

- PhD, Brown University

#### LINKS

- <http://molepharm.stanford.edu/faculty/homepages/roth.html>: <http://molepharm.stanford.edu/faculty/homepages/roth.html>

### Research & Scholarship

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#### CURRENT RESEARCH AND SCHOLARLY INTERESTS

Insulin is one of the primary regulators of rapid anabolic responses in the body. Defects in the synthesis and/or ability of cells to respond to insulin results in the condition known as diabetes mellitus. To better design methods of treatment for this disorder, we have been focusing our research on how insulin elicits its various biological responses. We are utilizing the techniques of immunology, molecular biology, and biochemistry to study:

- (i) How does the insulin receptor initiate the response to insulin? Like various oncogenes, the insulin receptor has an intrinsic enzymatic activity; it phosphorylates various proteins on tyrosine residues. This enzymatic activity has been found to be critical for insulin to elicit its various responses. The receptor kinase tyrosine phosphorylates various endogenous proteins. These proteins bind and activate a lipid kinase called a phosphatidylinositol 3-kinase. This kinase activates a serine/threonine kinase called Akt or PKB. A major focus is to understand the role of this serine kinase in eliciting various biological responses. Novel substrates for this kinase are being isolated and genes regulated by this kinase are being identified.
- (ii) How is the response to insulin modulated? Cells from non-insulin dependent diabetics (the most common form of diabetes, ~5 million in the US) exhibit a profound resistance to insulin. This resistance can be mimicked in cell cultures by stimulating the serine phosphorylation of the insulin receptor and/or various substrates of the insulin receptor tyrosine kinase. We are therefore exploring the hypothesis that excessive serine phosphorylation of the insulin receptor and/or insulin receptor substrates in these individuals causes this insulin resistance. We are determining the serine residues phosphorylated in the receptor and insulin receptor substrates, the enzymes response for this phosphorylation, and the consequences of these phosphorylations;
- (iii) How is the response to insulin terminated? We have purified to homogeneity a protease with a high specificity for insulin and capable of cleaving insulin at the same sites as those identified in insulin cleaved in intact cells. We have also isolated the cDNA which encodes for this protease and are overexpressing this protease in mammalian cells to determine whether it will affect the termination of the insulin response; and
- (iv) What is the relationship of the insulin receptor to the receptor for other insulin-like growth factors? We are comparing the abilities of these different receptors to stimulate various biological responses.

## Teaching

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### GRADUATE AND FELLOWSHIP PROGRAM AFFILIATIONS

- Cancer Biology (Phd Program)
- Chemical and Systems Biology (Phd Program)

## Publications

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### PUBLICATIONS

- **Pitriysin** *HANDBOOK OF PROTEOLYTIC ENZYMES, VOLS 1 AND 2, 3RD EDITION*  
Roth, R. A., Rawlings, N. D., Salvesen, G. S.  
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- **Leucine-stimulated mTOR signaling is partly attenuated in skeletal muscle of chronically uremic rats** *AMERICAN JOURNAL OF PHYSIOLOGY- ENDOCRINOLOGY AND METABOLISM*  
Chen, Y., Sood, S., McIntire, K., Roth, R., Rabkin, R.  
2011; 301 (5): E873-E881
- **Akt Isoforms Differentially Protect Against Stroke-induced Neuronal Injury in Vitro and in Vivo**  
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- **Increased workload fully activates the blunted IRS-1/PI3-kinase/Akt signaling pathway in atrophied uremic muscle** *KIDNEY INTERNATIONAL*  
Chen, Y., Sood, S., Biada, J., Roth, R., Rabkin, R.  
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- **PRAS40 regulates mTORC1 kinase activity by functioning as a direct inhibitor of substrate binding** *JOURNAL OF BIOLOGICAL CHEMISTRY*  
Wang, L., Harris, T. E., Roth, R. A., Lawrence, J. C.  
2007; 282 (27): 20036-20044
- **Protein kinase B/Akt phosphorylation of PDE3A and its role in mammalian oocyte maturation** *EMBO JOURNAL*  
Han, S. J., Vaccari, S., Nedachi, T., Andersen, C. B., Kovacina, K. S., Roth, R. A., Conti, M.  
2006; 25 (24): 5716-5725
- **PKC delta-mediated IRS-1 Ser24 phosphorylation negatively regulates IRS-1 function** *BIOCHEMICAL AND BIOPHYSICAL RESEARCH COMMUNICATIONS*  
Greene, M. W., Ruhoff, M. S., Roth, R. A., Kim, J., Quon, M. J., Krause, J. A.  
2006; 349 (3): 976-986
- **Mechanism of Akt1 inhibition of breast cancer cell invasion reveals a protumorigenic role for TSC2** *PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA*  
Liu, H., Radisky, D. C., Nelson, C. M., Zhang, H., Fata, J. E., Roth, R. A., Bissell, M. J.  
2006; 103 (11): 4134–39
- **On the mechanism for neomycin reversal of wortmannin inhibition of insulin stimulation of glucose uptake** *JOURNAL OF BIOLOGICAL CHEMISTRY*  
Shimaya, A., Kovacina, K. S., Roth, R. A.  
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- **Inhibition of mTOR activity restores tamoxifen response in breast cancer cells with aberrant Akt activity** *CLINICAL CANCER RESEARCH*  
DeGraffenried, L. A., Friedrichs, W. E., Russell, D. H., Donzis, E. J., Middleton, A. K., Silva, J. M., Roth, R. A., Hidalgo, M.  
2004; 10 (23): 8059–67
- **Modulation of human insulin receptor substrate-1 tyrosine phosphorylation by protein kinase C delta** *BIOCHEMICAL JOURNAL*  
Greene, M. W., Morrice, N., Garofalo, R. S., Roth, R. A.  
2004; 378: 105-116
- **Akt promotes increased mammalian cell size by stimulating protein synthesis and inhibiting protein degradation** *AMERICAN JOURNAL OF PHYSIOLOGY-ENDOCRINOLOGY AND METABOLISM*

- Faridi, J., Fawcett, J., Wang, L. H., Roth, R. A.  
2003; 285 (5): E964-E972
- **Inorganic polyphosphate stimulates mammalian TOR, a kinase involved in the proliferation of mammary cancer cells** *PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA*  
Wang, L. H., Fraley, C. D., Faridi, J., Kornberg, A., Roth, R. A.  
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  - **Expression of constitutively active Akt-3 in MCF-7 breast cancer cells reverses the estrogen and tamoxifen responsiveness of these cells in vivo** *CLINICAL CANCER RESEARCH*  
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  - **Gene expression profiling in prostate cancer cells with Akt activation reveals Fra-1 as an Akt-inducible gene** *MOLECULAR CANCER RESEARCH*  
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  - **Identification of a proline-rich Akt substrate as a 14-3-3 binding partner** *JOURNAL OF BIOLOGICAL CHEMISTRY*  
Kovacina, K. S., Park, G. Y., Bae, S. S., Guzzetta, A. W., Schaefer, E., Birnbaum, M. J., Roth, R. A.  
2003; 278 (12): 10189-10194
  - **Modulation of insulin-stimulated degradation of human insulin receptor substrate-1 by serine 312 phosphorylation** *JOURNAL OF BIOLOGICAL CHEMISTRY*  
Greene, M. W., Sakaue, H., Wang, L. H., Alessi, D. R., Roth, R. A.  
2003; 278 (10): 8199-8211
  - **Akt modulates STAT3-mediated gene expression through a FKHR (FOXO1a)-dependent mechanism** *JOURNAL OF BIOLOGICAL CHEMISTRY*  
Kortylewski, M., Feld, F., Kruger, K. D., Bahrenberg, G., Roth, R. A., Joost, H. G., Heinrich, P. C., Behrmann, I., Barthel, A.  
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  - **Protein kinase B/Akt is essential for the insulin- but not progesterone-stimulated resumption of meiosis in Xenopus oocytes** *BIOCHEMICAL JOURNAL*  
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  - **Reduced hippocampal insulin-degrading enzyme in late-onset Alzheimer's disease is associated with the apolipoprotein E-epsilon 4 allele** *AMERICAN JOURNAL OF PATHOLOGY*  
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  - **Regulation of the forkhead transcription factor FKHR (FOXO1a) by glucose starvation and AICAR, an activator of AMP-activated protein kinase** *ENDOCRINOLOGY*  
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  - **Reduced hippocampal insulin degrading enzyme in late-onset Alzheimer's disease is associated with the apolipoprotein E-epsilon 4 allele**  
McMillan, P., Cook, D., Leverenz, J., Kulstad, J., Schellenberg, G., Craft, S., Ericksen, S., Jin, L. W., Roth, R., Kovacina, K.  
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  - **Concentration-dependent stimulatory and inhibitory effect of troglitazone on insulin-induced fatty acid synthase expression and protein kinase B activity in 3T3-L1 adipocytes** *NAUNY-SCHMIEDEBERG'S ARCHIVES OF PHARMACOLOGY*  
Barthel, A., Kruger, K. D., Roth, R. A., Joost, H. G.  
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Barthel, A., Schmoll, D., Kru Ger, K. D., Roth, R. A., Joost, H. G.  
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- **Ligand-dependent interaction of estrogen receptor-alpha with members of the forkhead transcription factor family** *JOURNAL OF BIOLOGICAL CHEMISTRY*  
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- **Differential regulation of endogenous glucose-6-phosphatase and phosphoenolpyruvate carboxykinase gene expression by the forkhead transcription factor FKHR in H4IIE-hepatoma cells** *BIOCHEMICAL AND BIOPHYSICAL RESEARCH COMMUNICATIONS*  
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- **Activation of protein kinase B/cAkt in hepatocytes is sufficient for the induction of expression of the gene encoding glucokinase** *BIOCHEMICAL JOURNAL*  
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- **Oncogenic transformation of cells by a conditionally active form of the protein kinase Akt/PKB** *CELL GROWTH & DIFFERENTIATION*  
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- **The MMAC1 tumor suppressor phosphatase inhibits phospholipase C and integrin-linked kinase-activity** *ONCOGENE*  
Morimoto, A. M., Tomlinson, M. G., Nakatani, K., Bolen, J. B., Roth, R. A., Herbst, R.  
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- **Heregulin regulation of Akt/protein kinase B in breast cancer cells** *BIOCHEMICAL AND BIOPHYSICAL RESEARCH COMMUNICATIONS*  
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Nakatani, K., Thompson, D. A., Barthel, A., Sakaue, H., Liu, W., Weigel, R. J., Roth, R. A.  
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- **Regulation of GLUT1 gene transcription by the serine threonine kinase Akt1** *JOURNAL OF BIOLOGICAL CHEMISTRY*  
Barthel, A., Okino, S. T., LIAO, J. F., Nakatani, K., Li, J. P., Whitlock, J. P., Roth, R. A.  
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- **The role of glycogen synthase kinase 3 beta in insulin-stimulated glucose metabolism** *JOURNAL OF BIOLOGICAL CHEMISTRY*  
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- **Identification of a human Akt3 (protein kinase B gamma) which contains the regulatory serine phosphorylation site** *BIOCHEMICAL AND BIOPHYSICAL RESEARCH COMMUNICATIONS*  
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- **Modulation of insulin receptor substrate-1 tyrosine phosphorylation by an Akt/phosphatidylinositol 3-kinase pathway** *JOURNAL OF BIOLOGICAL CHEMISTRY*  
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- **Muscle fiber type-specific defects in insulin signal transduction to glucose transport in diabetic GK rats** *DIABETES*  
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  - **Activation of protein kinase B/Akt is sufficient to repress the glucocorticoid and cAMP induction of phosphoenolpyruvate carboxykinase gene** *JOURNAL OF BIOLOGICAL CHEMISTRY*  
LIAO, J. F., Barthel, A., Nakatani, K., Roth, R. A.  
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  - **Divergent effects of exercise on metabolic and mitogenic signaling pathways in human skeletal muscle** *FASEB JOURNAL*  
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  - **Protein kinase B/akt and Rab5 mediate ras activation of endocytosis** *JOURNAL OF BIOLOGICAL CHEMISTRY*  
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  - **Modulation of insulin receptor substrate-1 tyrosine phosphorylation and function by mitogen-activated protein kinase** *JOURNAL OF BIOLOGICAL CHEMISTRY*

- DeFea, K., Roth, R. A.  
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