Stanford

Richard Roth

Professor of Chemical and Systems Biology, Emeritus

Chemical and Systems Biology Operations

Bio

ACADEMIC APPOINTMENTS

- Emeritus Faculty, Acad Council, Chemical and Systems Biology Operations

PROFESSIONAL EDUCATION

- PhD, Brown University

LINKS

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

Research & Scholarship

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 recpetor 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

GRADUATE AND FELLOWSHIP PROGRAM AFFILIATIONS

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

Publications

PUBLICATIONS

- **Pitrilysin** *HANDBOOK OF PROTEOLYTIC ENZYMES, VOLS 1 AND 2, 3RD EDITION*
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- **On the mechanism for neomycin reversal of wortmannin inhibition of insulin stimulation of glucose uptake** *JOURNAL OF BIOLOGICAL CHEMISTRY*
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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.
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  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.
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- **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*
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