BIOGRAPHICAL SKETCH

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NAME: Bergman, Richard N.

eRA COMMONS USER NAME (credential, e.g., agency login): richardnbergman

POSITION TITLE: Director, Cedars-Sinai Diabetes and Obesity Research Institute; Alfred Jay Firestein Chair in Diabetes Research, Cedars-Sinai

EDUCATION/TRAINING *(Begin with baccalaureate or other initial professional education, such as nursing, include postdoctoral training and residency training if applicable. Add/delete rows as necessary.)*

INSTITUTION AND LOCATION	DEGREE *(if applicable)*	Completion Date MM/YYYY	FIELD OF STUDY
Case Western Reserve University, Cleveland, OH	B.S.E.	1965	Biomedical Engineering
University of Pittsburgh, Pittsburgh, PA	Ph.D.	1971	Physiology

A. Personal Statement

Dr. Richard Nathan Bergman is an internationally respected expert in the areas of diabetes and obesity research. He holds an undergraduate degree in Engineering from Case-Western Reserve University (Case Alumni Scholar) and the Ph.D. in Physiology from the University of Pittsburgh. Professor Bergman has been contributing important research in these areas for over 30 years, and has published over 400 articles. Bergman was the Keck Foundation Chair at the USC Keck School of Medicine, and was Professor of Medicine and Biomedical Engineering and Chairman of Physiology/Biophysics for 17 years. In 2011 Dr. Bergman moved from USC to Cedars-Sinai Medical Center. He is founding Director of the Cedars-Sinai Diabetes and Obesity Research Institute. He holds the Alfred Jay Firestein Chair in Diabetes. Dr. Bergman has mentored directly 50 individuals at various levels including Predoctoral, Postdoctoral, Faculty and Sabbatical. Many of these individuals are still involved in diabetes research. Some outstanding examples include Diane Finegood, Jay Taborsky, Marilyn Ader, Aage Volund, and Jang Youn. Bergman developed and headed the MD/PhD program at USC for 12 years. He served on the NIH metabolism study section for 6 years, and has reviewed for many organizations including the American Diabetes Association, the JDRF, the Obesity Society, and the NIH (Director's New Innovator Award, and SCOR on gender differences for Northwestern University). In 2015 he was awarded the Albert Renold Award from the American Diabetes Association; this award is specifically given for mentoring of diabetes researchers and establishing an outstanding environment for training in diabetes research.

B. Positions and Honors.

Positions and Employment

1971-1976 Instructor/Assistant/Associate Professor, Biomedical Engineering, University of Southern California (USC)
1977-1979 Associate Professor, Biomedical Engineering and Biological Sciences, Northwestern University
1980-June 2011 Professor, Physiology/Biophysics and Medicine and Biomedical Engineering, Keck School of Medicine, University of Southern California (KSOM of USC)
1993-June 2011 Chairman, Physiology and Biophysics, University of Southern California School of Medicine

2003-June 2011 Keck Professor of Medicine (endowed)
June 2011- Professor Emeritus, Physiology/Biophysics and Medicine and Biomedical Engineering, Keck School of Medicine, University of Southern California (KSOM of USC)
July 2011- Director, Cedars-Sinai Diabetes and Obesity Research Institute (DORI)
August 2011- Alfred Jay Firestein Chair in Diabetes Research, Cedars-Sinai Medical Center
April 2013- Professor-in-Residence, Department of Medicine, University of California, Los Angeles (UCLA)

Selected Honors

1989 **Outstanding Researcher in Diabetes (Lilly Award)**, American Diabetes Association (ADA)
1995 USC Associates Award for Research Creativity and Scholarship
1998 Mosenthal Award Lecture, New York Diabetes Association
1999 Plenary Lecturer, Association of Chairs of Departments of Physiology (ACDP)
2000 Levine-Williams Award, American Federation for Clinical Research (AFCR; Western)
2000 Irving Schwartz Endowed Professorship, Mount Sinai School of Medicine
2003 Joseph Globus Award, Mount Sinai School of Medicine
April 2004 Novo-Nordisk Endowed Lectureship, McGill University
July 2004- Fellow, N. American Assoc for Study of Obesity (NAASO)
July 2004 **TOPS Research Award**, N. American Assoc for Study of Obesity (NAASO)
2004 **Man of Distinction Lifetime Achievement Award**, American Diabetes Association (ADA)
April 2005 USC Mellon Award for Excellence in Mentoring
April 2006 Solomon Berson Lecturer, Federation of American Societies for Experimental Biology (FASEB)
June 2006 Stefan Schuy Research Award, Austrian Society for Biomedical Engineering (Österreichische Gesellschaft für Biomedizinische Technik, ÖGBMT)
June 2006 **Banting Medal for Scientific Achievement**, American Diabetes Association (ADA)
2007-2017 **MERIT Award ("Method to Extend Research in Time")**, National Institutes of Health (NIH)
2009 **Naomi Berrie Award**, Columbia University Medical Center
March 2010 **Kroc Lecturer**, Baylor College of Medicine
June 2015 **Albert Renold Award**, American Diabetes Association
2016 **Alexander Marble Lecturer**, Joslin Diabetes Institute

Other Experience and Professional Memberships

2004-2006 Advisory Board, Case Western Reserve University Center for Modeling Integrated Metabolic Systems (MIMS)
2004-2006 President-Elect, Association of Chairs of Departments of Physiology (ACDP)
2006-2007 President, Association of Chairs of Departments of Physiology (ACDP)
2007-2012 Editor-in-Chief, *Obesity (a Research Journal)*

C. Contribution to Science

I. The Minimal Model. Methods for assessment of insulin resistance and beta-cell function were in their infancy when we proposed the most parsimonious mathematical representation of glucose and insulin dynamics. We devised a test which allowed for measurement of insulin sensitivity, beta-cell response, insulin clearance and glucose effectiveness from a single test. The method has been used in hundreds of clinical, epidemiologic and genetic studies. Dr. Bergman was the originator of this method.

1. Toffolo G., R.N. Bergman, D.T. Finegood, C.R. Bowden, and C. Cobelli. Quantitative estimation of beta cell sensitivity to glucose in the intact organism: A minimal model of insulin kinetics in the dog. Diabetes. 29:979-990, 1980.
2. Bergman R.N., L.S. Phillips, and C. Cobelli. Physiologic evaluation of factors controlling glucose tolerance in man: Measurement of insulin sensitivity and beta-cell glucose sensitivity from the response to intravenous glucose. J. Clin. Invest. 68:1456-1467, 1981.
3. Bergman R.N., R. Prager, A. Volund, and J.M. Olefsky. Equivalence of the insulin sensitivity index in man derived by the minimal model method and the euglycemic clamp. J. Clin. Invest. 79:790-800, 1987.
4. Bergman R.N. Banting Lecture 2006: Orchestration of glucose homeostasis – From a small acorn to the California oak. Diabetes. 56:1489-1501, 2007.

II. The Disposition Index (DI). Dr. Bergman proposed that there is a hyperbolic relationship between insulin sensitivity and insulin response, such that SI x AIRglucose = DI. The ability of the beta-cells of the pancreas to compensate for insulin resistance is given by DI, which is the most powerful predictor known of conversion from normal glucose tolerance to Type 2 diabetes.

1. Bergman R.N., L.S. Phillips, and C. Cobelli. Physiologic evaluation of factors controlling glucose tolerance in man: Measurement of insulin sensitivity and beta-cell glucose sensitivity from the response to intravenous glucose. J. Clin. Invest. 68:1456-1467, 1981.
2. Bergman R.N., M. Ader, K Huecking, and G. Van Citters. Accurate assessment of beta-cell function: The hyperbolic correction. Diabetes. 51 (Suppl 1):S212-S220, 2002.
3. Lorenzo C., L.E. Wagenknecht, M.J. Rewers, A.J. Karter, R.N. Bergman, A.J.G. Hanley, and S.M. Haffner. Disposition Index, glucose effectiveness, and conversion to type 2 diabetes: The Insulin Resistance Atherosclerosis Study (IRAS). Diabetes Care. 33:2098-2103, 2010. PMCID: PMC2928371.
4. Watkins S.M., M.W. Rowe, J.A. Kolberg, L.E. Wagenknecht, and R.N. Bergman. Biomarker models as surrogates for the Disposition Index in the Insulin Resistance Atherosclerosis Study. Diabet. Med. 29:1399-1406, 2012. PMID: 22413950.

III. Importance of transendothelial insulin transport to insulin action. Utilizing lymph sampling from the canine hindlimb, we were able to assess the rate of transport of insulin across the endothelial barrier in skeletal muscle. We proved that said transporter is rate-limiting for insulin action, and that the transport rate is reduced in obesity, partially explaining insulin resistance in the moderately obese state.

1. Chiu J.D., J.M. Richey, L.N. Harrison, E. Zuniga, E. Kirkman, M. Ellmerer, and R.N. Bergman. Direct administration of insulin into skeletal muscle reveals that the transport of insulin across the capillary endothelium muscle limits the time course of insulin to activate glucose disposal. Diabetes. 57:828–835, 2008. PMID: 18223011.
2. Chiu J.D., C.M. Kolka, J.M. Richey, L.N. Harrison, E. Zuniga, E.L. Kirkman, and R.N. Bergman. Experimental hyperlipidemia dramatically reduces access of insulin to skeletal muscle. Obesity (Silver Spring). 17:1486-1492, 2009. NIHMSID: NIHMS168988.
3. Kolka C.M., L.N. Harrison, M. Lottati, J.D. Chiu, E.L. Kirkman, and R.N. Bergman. Diet-induced obesity prevents interstitial dispersion of insulin in skeletal muscle. Diabetes. 59:619-626, 2010. PMCID: PMC2827487.
4. Kolka C.M. and R.N. Bergman. The barrier within: Endothelial transport of hormones. Physiology (Bethesda). 27:237-247, 2012. PMID: 22875454.

IV. Role of free fatty acids (FFA) in insulin suppression of liver glucose output. We proved that the effect of insulin to control liver glucose output was indirect and controlled by plasma FFA. Thus insulin resistance of the liver is due in whole or part an inability of insulin to suppress liver glucose production.

1. Bergman R.N., D.C. Bradley, and M. Ader. On insulin action in vivo: The single gateway hypothesis. Adv. Exp. Med. Biol. 334:181-198, 1993.
2. Bradley D.C., R.A. Poulin, and R.N. Bergman. Dynamics of hepatic and peripheral insulin effects suggest common rate-limiting step in vivo. Diabetes. 42:296-306, 1993.
3. Rebrin K., G.M. Steil, S.D. Mittelman, and R.N. Bergman. Causal linkage between insulin suppression of lipolysis and suppression of liver glucose output in dogs. J. Clin. Invest. 98:741-749, 1996.
4. Ellmerer M., S.P. Kim, M. Hamilton-Wessler, K. Hucking, E. Kirkman, and R.N. Bergman. Physiological hyperinsulinemia in dogs augments access of macromolecules to insulin sensitive tissues. Diabetes. 53:2741-2747, 2004.

V. Role of liver insulin clearance in pathogenesis of Type 2 diabetes. We showed that the metabolic clearance rate of insulin (MCR) changes in the insulin resistant situation, and that this change is an important compensatory mechanism whereby an increased fraction of insulin secreted from the beta-cell enters the systemic circulation in the insulin resistant situation, this helping to compensate for the insulin resistant condition.

1. Mittelman S.D., G.W. Van Citters, S.P. Kim, D.A. Davis, M.K. Dea and R.N. Bergman. Longitudinal compensation for fat induced insulin resistance includes reduced insulin clearance and enhanced β□cell response. Diabetes. 49:2116-2125, 2000.

2. Kim S.P., M. Ellmerer, E.L. Kirkman, and R.N. Bergman. β-cell "rest" due to reduced first-pass hepatic insulin extraction in the insulin resistant, fat-fed model. Am. J. Physiol. Endocrinol. Metab. 292:E1581-E1589, 2007.
3. Goodarzi M.O., C.D. Langefeld, A.H. Xiang, Y.D. Chen, X. Guo, A.J. Hanley, L.J. Raffel, F. Kandeel, J.L. Nadler, T.A. Buchanan, J.M. Norris, T.E. Fingerlin, C. Lorenzo, M.J. Rewers, S.M. Haffner, D.W. Bowden, S.S. Rich, R.N. Bergman, J.I. Rotter, R.M. Watanabe, and L.E. Wagenknecht. Insulin sensitivity and insulin clearance are heritable and have strong genetic correlation in Mexican Americans. Obesity (Silver Spring). 22:1157-1164, 2014. doi: 10.1002/oby.20639. [Epub 2013 Jun 13]. PMCID: PMC3968231.
4. Ader M., D. Stefanovski, S.P. Kim, J.M. Richey, V. Ionut, K.J. Catalano, K. Hucking, M. Ellmerer, G. Van Citters, I.R. Hsu, J.D. Chiu, O.O. Woolcott, L.N. Harrison, D. Zheng, M. Lottati, C.M. Kolka, V. Mooradian, J. Dittmann, S.Yae, H. Liu, A.V. Castro, M. Kabir, and R.N. Bergman. Hepatic insulin clearance is the primary determinant of insulin sensitivity in the normal dog. Obesity (Silver Spring). 22:1238-1245, 2014. doi: 10.1002/oby.20625. [Epub 2013 Dec 3]. PMCID: PMC3969862 [Available on 2014/11/1].

Complete List of Published Works on NCBI MyBibliography:
http://www.ncbi.nlm.nih.gov/sites/myncbi/richard.bergman.1/bibliography/40360206/public/?sort=date&direction=ascending

D. Research Support

Ongoing Research Support

R01 DK 29867-A1 Bergman (PI) 07/01/15 06/30/19
NIH/NIDDK
Quantitation of Factors Regulating Glucose Tolerance
The major goal of this project is to explain mechanisms of hyperinsulinemic compensation for insulin resistance.
Role: PI

5 R01 DK27619 Bergman (PI) 12/01/07 11/30/17
NIH/NIDDK
*Quantitative Studies of Metabolic Organ Dynamics (**M.E.R.I.T award**)*
The major goal of this project is to examine the role of the liver in glucose tolerance.
Role: PI

5 R01 DK062370 Boehnke (PI) 05/01/09 05/31/16 *(Dr. Bergman no longer receives funding)*
NIH/NIDDK/University of Michigan
Identifying Genes for Type 2 Diabetes: FUSION
The major goal of this project is to map and clone susceptibility genes for type 2 diabetes and diabetes-related traits.
Role: PI Subcontract

U1111-1138-72234 (UTN) Bergman (PI) 11/01/14 05/31/16
Novo-Nordisk
Liraglutide Actions on the Liver: Effects on Glucose Phosphorylation
The major goal of this project is to examine the acute and chronic effect of liraglutide on liver glucose uptake by quantifying the effect of liraglutide on liver glucose phosphorylation (GCK activity) in vivo using the lactate model of liver glucose uptake.
Role: PI

2 T32 DK007770-11 Melmed (PI) 07/01/12 06/30/17
NIH/NIDDK
Training Program in Endocrinology, Diabetes, and Metabolism
The major goal of this program is to train scientists to study thyroid, reproductive endocrinology, diabetes/metabolism, endocrine oncology, endocrine hypertension, and neuroendocrinology.
Role: co-PI

5 P30 DK063491-11 Olefsky (PI) 05/01/13 04/30/18
NIH/NIDDK
Diabetes Endocrinology Research Center (DERC)
The major goal of the UCSD/UCLA/Salk/Cedars Sinai DERC is to foster research in the prevention and treatment of diabetes and its complications to ultimately improve the lives of patients.
Role: Member

Completed Research Support *(past 3 years)*

2 R01 HL060944 Wagenknecht (PI) 01/01/10 12/31/14
NIH/NHLBI/Wake Forest University
IRAS Family Study-2: Genetics of Adiposity and Glucose Homeostasis
The major goal of this project is to target the further exploration of genomic regions and positional cloning of genes contributing to variation in adiposity and glucose homeostasis. We will re-contact participants from the Insulin Resistance and Atherosclerosis Study to repeat some of the primary phenotypes for measures of change (abdominal CT scan and fasting insulin). New phenotypes (total body fat by DXA and adipocytokines, including adiponectin and soluble TNF-α receptors 1 and 2), as well as a panel of nutritional, dietary, and eating behaviors will also be assessed.
Role: PI Subcontract