BIOGRAPHICAL SKETCH

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NAME: KOCH, BRUCE D.

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

POSITION TITLE: Senior Director

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

INSTITUTION AND LOCATION	DEGREE (if applicable)	Completion Date MM/YYYY	FIELD OF STUDY
Bates College, Lewiston, Maine	BS	06/1979	Biology
Harvard University, Cambridge, Mass.	PHD	09/1986	Cell and Developmental Biology
University of California, Berkeley, Berkeley, California	Postdoctoral Fellow	01/1991	Cell Biology

A. Personal Statement

I have 19 years of drug discovery experience in the pharmaceutical industry, first at Syntex and then at Roche. During that time, I've helped lead a number of drug discovery projects and was the Project Leader for two of those projects. I was part of several management teams which decided upon the progression of candidate molecules into entry-into-humans enabling studies. For ten of those years I also oversaw a High Throughput Screening group and was deeply involved in company-wide efforts to improve the value and efficiency of lead identification at Roche. As with much of the work done in the pharmaceutical industry, our results were considered proprietary and confidential and could not be published except as limited abstracts and presentations at scientific meetings.

1. Koch B. A Practical Guide to Drug Development in Academia. Mochly-Rosen D, Grimes K, editors. Cham: Springer International Publishing; 2014. Developing Assays for High-Throughput Screening (HTS). ; p.40-45. 176p.
2. Dietrich PS, Koch B, Guthrie H, Gubler UA. , inventors. Stable cell lines expressing hERG. United States US8,349,572. 2013.
3. Dietrich PS, Koch B, Guthrie H, Gubler UA. , inventors. Stable cell lines expressing hERG. United States US7,776,590B2. 2010.
4. Giannetti AM, Koch BD, Browner MF. Surface plasmon resonance based assay for the detection and characterization of promiscuous inhibitors. J Med Chem. 2008 Feb 14;51(3):574-80. PubMed PMID: 18181566.

B. Positions and Honors

Positions and Employment

1991 - 1995	Research Scientist I, Syntex Discovery Research, Institute of Pharmacology, Palo Alto, CA
1995 - 1999	Research Scientist II and Site Technology Officer, Roche Palo Alto, New Leads Discovery, Palo Alto, CA
2000 - 2004	Principal Research Scientist, Roche Palo Alto, Lead Discovery Group, Palo Alto, CA
2004 - 2009	Associate Director and Head, High Throughput Screening Group, Roche Palo Alto, High Throughput Screening, Palo Alto, CA
2009 - 2010	Director, Roche Palo Alto, Discovery Technologies, Palo Alto, CA
2011 -	Senior Director, Stanford University School of Medicine, Office of the Senior Associate Dean for Research, Discovery and Technological Service Centers, Stanford, CA

Other Experience and Professional Memberships

1997 -	Member, Biophysical Society
1999 -	Member, Society for Laboratory Automation and Screening
2003 - 2003	Member, NIH Study Section, NIH Roadmap Assay Development R25
2010 -	Advisor, SPARK Program, Stanford University
2010 - 2010	Member, NIH Study Section, NIH Roadmap HTS Assay for MLPCN R03
2013 -	Member, Association of Biomolecular Resource Facilities
2014 -	Member, NIH Study Section, Drug Discovery for Aging, Neuropsychiatric and Neurologic Disorders (SBIR/STTR)

Honors

1985	Fellow, Albert J. Ryan Foundation
1986	Postdoctoral Fellow, Jane Coffin Childs Memorial Fund for Cancer Research

C. Contribution to Science

1. Following up on the observations of Brian Shoichet's lab, we used surface plasmon resonance to directly observe high-stoichiometry interactions of promiscuous compounds with proteins.

    a. Giannetti AM, Koch BD, Browner MF. Surface plasmon resonance based assay for the detection and characterization of promiscuous inhibitors. J Med Chem. 2008 Feb 14;51(3):574-80. PubMed PMID: 18181566.

2. The Selective Sodium Channel Blocker program, of which I eventually became the leader, sought to identify NaV1.8 subtype-selective sodium channel blockers for the treatment of pain. As part of that program, we cloned several novel sodium channels, including human and rat NaV1.8. I did the initial expression and electrophysiological and pharmacological characterization of those cloned channels. I oversaw the development of a miniaturized assay and the execution of a 650,000 compound HTS screen against this ion channel. My group also developed and ran the selectivity assays against other sodium channels.

    a. Koch BD, Faurot GF, McGuirk JR, Clarke DE, Hunter JC. Modulation of mechano-hyperalgesia by clinically effective analgesics in rats with a peripheral mononeuropathy. Analgesia (Elmsford, N.Y.). 1996; 2:157-164.
    b. Sangameswaran L, Delgado SG, Fish LM, Koch BD, Jakeman LB, Stewart GR, Sze P, Hunter JC, Eglen RM, Herman RC. Structure and function of a novel voltage-gated, tetrodotoxin-resistant sodium channel specific to sensory neurons. J Biol Chem. 1996 Mar 15;271(11):5953-6. PubMed PMID: 8626372.
    c. Sangameswaran L, Fish LM, Koch BD, Rabert DK, Delgado SG, Ilnicka M, Jakeman LB, Novakovic S, Wong K, Sze P, Tzoumaka E, Stewart GR, Herman RC, Chan H, Eglen RM, Hunter JC. A novel tetrodotoxin-sensitive, voltage-gated sodium channel expressed in rat and human dorsal root ganglia. J Biol Chem. 1997 Jun 6;272(23):14805-9. PubMed PMID: 9169448.
    d. Dietrich PS, McGivern JG, Delgado SG, Koch BD, Eglen RM, Hunter JC, Sangameswaran L. Functional analysis of a voltage-gated sodium channel and its splice variant from rat dorsal root ganglia. J Neurochem. 1998 Jun;70(6):2262-72. PubMed PMID: 9603190.

3. For my postdoc (in the laboratory of Randy Schekman), I studied factors involved in the post-translational translocation of yeast pre-pro-alpha factor. One of the factors was a set of HSP70s.

    a. Deshaies RJ, Koch BD, Werner-Washburne M, Craig EA, Schekman R. A subfamily of stress proteins facilitates translocation of secretory and mitochondrial precursor polypeptides. Nature. 1988 Apr 28;332(6167):800-5. PubMed PMID: 3282178.

4. During my graduate work, I explored the mechanisms by which somatostatin signaled through it's receptor (a GPCR) and found that it activated two mechanisms: 1. inhibition of adenylate cyclase via Gi and 2. a

cAMP-independent mechanism that appeared to be due to an increase in K+ conductance. This work relied heavily upon immunoassays to measure both secreted hormone (prolactin) and second messenger levels.

a. Koch BD, Schonbrunn A. Characterization of the cyclic AMP-independent actions of somatostatin in GH cells. II. An increase in potassium conductance initiates somatostatin-induced inhibition of prolactin secretion. J Biol Chem. 1988 Jan 5;263(1):226-34. PubMed PMID: 2891696.
b. Koch BD, Blalock JB, Schonbrunn A. Characterization of the cyclic AMP-independent actions of somatostatin in GH cells. I. An increase in potassium conductance is responsible for both the hyperpolarization and the decrease in intracellular free calcium produced by somatostatin. J Biol Chem. 1988 Jan 5;263(1):216-25. PubMed PMID: 2891695.
c. Koch BD, Dorflinger LJ, Schonbrunn A. Pertussis toxin blocks both cyclic AMP-mediated and cyclic AMP-independent actions of somatostatin. Evidence for coupling of Ni to decreases in intracellular free calcium. J Biol Chem. 1985 Oct 25;260(24):13138-45. PubMed PMID: 2865257.
d. Koch BD, Schonbrunn A. The somatostatin receptor is directly coupled to adenylate cyclase in GH4C1 pituitary cell membranes. Endocrinology. 1984 May;114(5):1784-90. PubMed PMID: 6143660.

My full bibliography is available here:
http://www.ncbi.nlm.nih.gov/sites/myncbi/bruce.koch.2/bibliography/47404990/public/?sort=date&direction=descending