

M.L. Green, M.M. Pisano, R.A. **Prough**, and T.B. Knudsen, (2013) Release of targeted p53 from the mitochondrion as an early signal during mitochondrial dysfunction, Cellular Signalling, 25, 2383-90. [PMID: 23899557; PMCID: Pending]

Michael-Miller, K.K., Al-Rayyan, N., Ivanova, M.M., Mattingly, K.A., Ripp, S.L., Klinge, C.M. and **Prough**, R.A. (2013). DHEA and its metabolites directly activate estrogen receptors alpha and beta, Steroids, 78, 15–25. [PMID: 23123738: PMCID: PMC3529809]

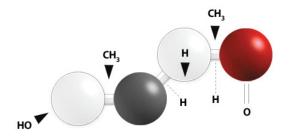
Xu, X., Powell, D.W., Lambring, C.J., Puckett, A.H., Deschenes, L., **Prough**, R.A., Poeschla, E.M., and Samuelson, D.J. (2014) Human MCS5A1 candidate breast cancer susceptibility gene FBXO10 is induced by cellular stress and correlated with lens epithelium-derived growth factor (LEDGF), Molecular Carcinogenesis, 53, 300–313. [PMID: 23138933; PMCID: Pending]

Xu, X., **Prough**, R.A. and Samuelson, D.J. (2014) Differential 12-O-tetradecanoylphorbol-13-acetate-induced activation of rat mammary carcinoma susceptibility Fbxo10 gene promoter region via a PKC-AP1 pathway, Molecular Carcinogenesis, 54, 134–147. [PMID: 24008983; PMCID: Pending]

Makia, N.L., Surapureddi, S., Monostory, K., **Prough**, R.A. and Goldstein, J.A. (2014) Activator Protein 1 regulation of human CYP2C9 Expression by electrophilic stress involves MAPK activation and DNA Looping, Molecular Pharmacology, 86, 125-137. [PMID: 24830941; PMCID: PMC4127925]

B Wahlang, K. C. Falkner, H.B. Clair, L.A. Al-Eryani, R.A. **Prough**, J.C. States, D. Coslo, and C. Omiecinski, and M. Cave (2014) Human Receptor Activation By Aroclor 1260, A Polychlorinated Biphenyl Mixture", Toxicological Sciences, 140, 283-297 (2014). [Epub ahead of print] [PMID: 24812009; PMCID: PMC4176050].

DHEA (Déhydroépiandrostérone)



B. Wahlang, K.C. Falkner, J.C. States, R. A. **Prough**, and M. Cave (2014) Arochlor 1260 Exposure Worsens Hepatic and Systemic Inflammation in an Animal Model of Diet-Induced Obesity and Nonalcoholic Fatty Liver Disease, Toxicology & Applied Pharmacology, 279, 380-390 (2014) [PMID: 24998970; PMCID: PMC4225625]

External Professional Activities (2013-2014):

Editorial Board Membership:

Jan. 1981 - Present: Archives of Biochemistry and Biophysics.

Jan. 1977-Dec 2011: Drug Metabolism and Disposition; Associate Editor,

January 1, 1994-2011.

Jan. 1983 - Present: Journal of Pharmacology and Experimental

Therapeutics.

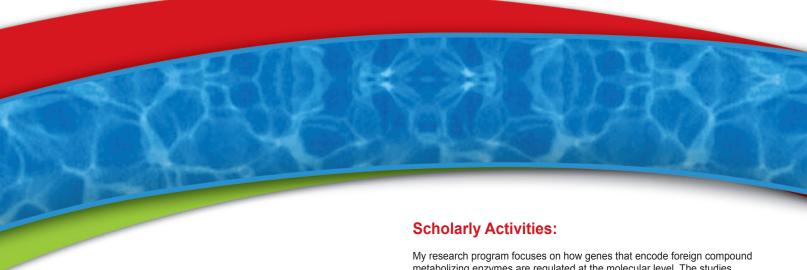
Jan. 1984 - Present: Xenobiotica.

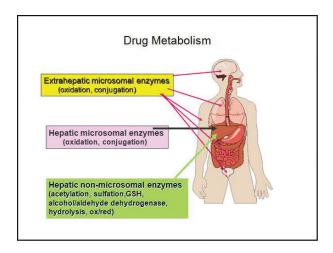
Oct. 2002 - Present Drug Metabolism Reviews, Associate Editor
Jul 2004 - Present: Pharmacology & Therapeutics, Associate Editor

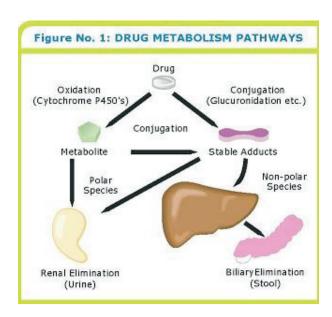
Review Panels:

K99/R00 Review Panel National Institutes of Environmental Health Sciences (Fall 2014)

United Kingdom Cancer Research Foundation (February 2015)







metabolizing enzymes are regulated at the molecular level. The studies focus on metabolism of aldehydic products of volatile organic compounds, sterols, and polychlorinated hydrocarbons. Specifi cally, we are addressing the regulation of these genes through action of nuclear receptors such as AhR, CAR, PXR, LXR, and FXR, as well as other transcription factors such as Nrf2 and AP-1. Many aldehyde metabolizing enzymes are regulated through action of the reactive processes of the aldehydes themselves on nuclear receptors, serving as electrophilic metabolites. The studies continue with our ongoing collaboration with Aruni Bhatnagar, Daniel Conklin, and Sanjay Srivastava in Cardiology, Department of Medicine. Currently, I am working with Matthew Cave in the Division of Gastroenterology looking at how PCBs alter intermediary metabolism through the nuclear receptors that utilize bile acids as ligands (PXR, LXR, FXR). The work over the past 30 years allows my program to assess effects of obesity on the expression and regulation of genes encoding foreign compound metabolizing enzymes. A third study is focused upon a new human gene of the 11β-hydroxysteroid dehydrogenases that Boaz Robinzon, Hebrew University, Jerusalem, and I discovered in human liver nuclei and microsomes that catalyzes the oxidation of glucocortoids by NADP+. This gene is not expressed in rodents. We are expressing the gene product in E. coli and characterizing its biochemical properties related to organelle localization in the human hepatocyte and its substrate specifi city. We are also studying its expression during the obese state, in collaboration with Stephen Winters, M.D., Division of Endocrinology, Department of Medicine.

Grants:

Co-P.I. on "UofL Environmental Health Sciences Training Program" National Institutes of Environmental Health Sciences, T32 ES11564, 07/01/2004-06/30/2015, 07/01/04-06/30/09, TDC \$348,149

P.I. on "Summer Environmental Health Sciences Training Program"
National Institutes of Environmental Health Sciences. 1 T35 ES 014559-01
04/01/11-03/30/16, TDC \$163,640

Mentor on "Summer Endocrinology Research Training Program" National Institutes of Diabetes and Digestive Diseases and Kidney, 1 T35 DK072923-01, C.M. Klinge (P.I.) 05/01/11-04/30/16; TDC \$143.635

Co-P.I. on "PCBs Worsen Obesity/Metabolic Syndrome Through Toxic Metabolic Endotoxemia"

National Institutes of Environmental Health Sciences, 1R01ES021375-01 M. Cave (P.I.)

03/01/12-02/28/16, TDC \$1,125,000