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Understanding Chemical-induced Adverse Effects with Neuroproteomics

Prasada Rao S. Kodavanti[1], Joyce E. Royland[1], Witold Winnik[1] and Oscar Alzate[2]

[1]National Health and Environmental Effects Research Laboratory, U.S. Environmental Protection Agency, USA and [2]University of North Carolina at Chapel Hill, USA.

Abstract

Technological advances have supplied neuroproteomics with refined tools for the study of the expression, interaction, and function of proteins in the nervous system. Using bioinformatics, neuroproteomics can reveal the organization of dynamic, functional protein networks, and macromolecular structures that are the basis of behavioral, anatomical, and functional processes. Neuroproteomics promotes the understanding of post-translational modifications by which proteins are chemically modified after synthesis affecting the 3D conformation of the proteins, which can determine protein function. The most common techniques used in proteomics are two-dimensional differential gel electrophoresis (2D DIGE) and protein arrays, which are powerful tools to separate complex protein mixtures; mass spectrometry for protein identification and quantitation, and multiple databases and computer programs able to elucidate the protein interaction networks. Proteomics allows the identification of all proteins in a particular sample, the components of biochemical pathways, and potential post-translational modifications. Neuroproteomics has already demonstrated its value by identifying early biomarkers following neuronal damage in drug addiction or brain injury, and in monitoring nerve growth. In regards to chemically-induced adverse effects on the nervous system, proteomics can aid in understanding the mode of action of particular chemicals as well as identifying biomarkers for the toxic effects. The identification of mode-of-action-based-biomarkers can aid in the development of *in vitro* models to screen and prioritize chemicals for further neurotoxicity testing. Studies of two well-established neurotoxicants in both humans and animal models (Aroclor 1254, a commercial polychlorinated biphenyl mixture; and DE-71, a commercial polybrominated diphenyl ether mixture) using 2D DIGE have revealed that proteins related to energy metabolism in mitochondria (ATP synthase, sub unit β (ATP5β), creatine kinase and malate dehydrogenase), calcium signaling (endoplasmic reticulum ATPase, voltage-dependent anion-selective channel protein 1 (VDAC1) and Ryanodine receptor type II) and growth of the nervous system (valosin-containing protein (VCP), collapsin response mediator protein 3 (CRMP-3)) may be involved in the developmental neurotoxicity of these persistent chemicals. Studies are underway on other neurotoxic chemicals to identify common protein signatures and pathways.

Biography

Prasada R. Kodavanti is a Senior Research Toxicologist at the Neurotoxicology branch of US EPA in Research Triangle Park, NC. Dr. Kodavanti has published over 100 peer-reviewed journal articles, 13 book chapters, and 8 review articles. His publications have appeared in a wide variety of leading scientific journals. In 1996, one of his papers won the prestigious Society of Toxicology's Board of Publications Award for the Best Paper in *Toxicology and Applied Pharmacology*. He has received several USEPA's Scientific and Technological Achievement (STAA) Awards. In 2009, he received STAA level 1 award (highest scientific award by USEPA) for highlighting genomics in understanding the mode of action in developmental neurotoxicity. Dr. Kodavanti is a member of the Advisory Board for the journals "Pharmacology and Toxicology", "Toxicological Sciences", and "Neurotoxicology".