The GETA Fall meeting exceeded expectations, as we had to bring in extra chairs to accommodate all the last minute participants. The half-day conference entitled “Toxicity to the Developing Brain: Recent Research and Mechanisms of Effect” was held at the Elihu Harris State Building in Oakland on November 9, 2004, focused on current research in California on neurodevelopmental toxicity. Over 80 scientists representing industry, national laboratories, academia and government attended. The meeting highlighted research on how early-life exposures to very different chemicals or other environmental insults alter proper brain development. Chemicals discussed included organophosphate pesticides, PCBs and manganese. Researchers are trying to understand the underlying mechanisms of toxicity to help determine if early-life chemical exposures in humans are involved in outcomes such as hearing loss, attention deficit hyperactivity disorders, autism or other long-term learning and perception deficits.

Four distinguished speakers provided updates on their research. First, Dr. Frank Crinella of UC Irvine gave a talk on soy consumption, manganese toxicity and studies of hyperactivity. Soy infant formula contains fairly high levels of naturally occurring manganese and food companies historically supplemented formula with additional manganese. Dr. Crinella reviewed the human and animal evidence indicating that high intake of manganese consumption early in life is associated with hyperactivity later in life. He presented his research in rats indicating clear dose-response relationships with manganese intake and hyperactivity. Mechanistic studies suggested that alterations to the dopamine neurotransmitter systems may be responsible. Second, Dr. Isaac N. Pessah of UC Davis shared his work on chemically induced alterations of calcium channel proteins (the ryanodine receptor) in nerve cells. These large receptors control calcium homeostasis that is essential for proper nerve cell growth and interconnections. He discussed data on how non-coplanar PCBs and other compounds altered calcium signaling at very low concentrations. Third, Dr. Tal Kenet of UC San Francisco shared her work on environmental influences of proper brain development in the rat. She showed amazing images of how environmental insults such as repetitive noise or PCB exposure could physically alter the patterns of brain organization in the rat. Finally, Dr. Gary Quistad of UC Berkeley discussed work conducted in his laboratory on the toxicity of organophosphates and their effects on secondary non-acetylcholinesterase targets. For example, one mechanism by which these pesticides may cause neurodevelopmental effects is through inhibition of enzymes that remove lysophospholipids from cell membranes. The audience was treated to some very detailed information on myriad ways that environmental exposures can disrupt the intricate dance that is brain development.

As was the case with all of our meetings this year, the presentations were followed by good food! Attendees continued the lively discussion at a reception in the Golden Bear Café, where the caterer served a delicious spread of Indian food. It was an excellent meeting — start to finish.