These exposures, in combination with germ-line genetic constitution, alter lung cancer risk through several etiologic pathways including but not limited to: increased levels of DNA adduct formation, increased angiogenesis and altered anti-apoptotic signaling.

Dr. Brenner will provide an overview of previous and current international collaborative research efforts examining the role of inflammation in the development of lung cancer. Research targeting modifiable lifestyle risk factors, molecular studies and large-scale genomic initiatives, as well as future avenues for additional research and collaboration will be discussed.