There is scientific consensus that air pollution exacerbates childhood asthma, but there is continued controversy about the role of both regional and local traffic-related pollution in causing the disease. *In vitro* and animal studies have shown that diesel and ambient particulate matter has pro-oxidant and inflammatory activity relevant to the pathogenesis of asthma. Epidemiological studies of children have shown associations between residential exposure to near roadway air pollutants and wheeze and asthma. In addition, the pattern of dispersion of near roadway exposures to ultrafine particulate matter and other traffic-related pollutants has been shown to correspond to the near-roadway residential distance gradient in prevalence of asthma in the southern California Children’s Health Study. However, epidemiological associations vary based on the traffic metric used, and it has been argued that confounding may explain the associations observed. Additional research is required to resolve these uncertainties by developing better assessment of exposure to toxicologically relevant components of the ambient mixture, including the contribution of time-activity patterns in relevant microenvironments, and by better specifying potential confounders. However, an emerging literature showing susceptibility to oxidant air pollutants based on co-exposures and genetic variation in biological pathways predicted from toxicological studies provides strong evidence of a causal relationship between these pollutants and the development of new onset asthma. The presentation will make the case that intervention is warranted, because the economic and social costs of asthma are large.