Part IV
Bioavailability and Toxicology of Airborne Particulate Matter

Clare L. S. Wiseman and Fathi Zereini

As discussed previously, there is strong evidence for an association between ambient PM exposures in urban populations and patterns which have been observed in cardiopulmonary mortality and morbidity. Fine PM exposures have been particularly identified as posing a risk, especially in the elderly, those with a compromised health status and children with their developing respiratory systems. Less is known regarding the chemical constituents of PM and their bioavailability and the toxicological mechanisms through which they exert a negative health effect; crucial issues in the development of air quality policy and emissions regulations which are protective of human health. Part IV contains three sections which discuss important aspects of the bioavailability of PM exposures and the toxicological mechanisms involved in the induction of negative health outcomes.

In the first chapter, Leopold and Schuster discuss a method for the preparation and use of a standardized material to investigate the chemical behavior and bioavailability of palladium (Pd) emitted from traffic, a metal which has been found in increasingly greater amounts in the environment due to its popular use as a catalyst in automotive catalytic converters. In presenting the results of a study they conducted on barley plants, they provide an empirical example as to how the environmental bioavailability and impacts of PM constituents can be assessed. The final two sections address issues related to the toxicological mechanisms through which airborne PM and its chemical constituents may exert an effect in humans and their assessment. Specifically, Valavanidis et al. discuss the primary mechanisms involved in the generation of reactive oxygen species (ROS) in their research using traffic-related PM and exhaust soot from diesel and gasoline powered vehicles in the second chapter. As part of this, they detail the role of stable free radicals, polyaromatic hydrocarbons (PAHs) and transition metals and their interactions in contributing to the cytotoxic and carcinogenic potential of PM. In the third chapter, Akhtar et al. provide a comprehensive overview of the results and methods used to assess the toxicology of PM both in vivo and in vitro. As they discuss, in vivo and in vitro toxicology studies have significantly contributed to our understanding of the pathophysiological mechanisms underlying PM-induced health effects. In particular, they have revealed the importance of oxidative

stress-initiated inflammation as being a major pathway of PM-induced respiratory and cardiovascular disease. They conclude, however, that knowledge gaps remain; most notably those that pertain to the mechanisms responsible for the induction of human health effects at low dose PM exposures.