Editorial

Is NAD(P)H Oxidase a Missing Link for Air Pollution–Enhanced Obesity?

Ming-Hui Zou

Obesity has reached epidemic proportions in the developed and developing world. The rise in obesity prevalence has resulted in an increase in the myriad serious medical problems associated with excess body fat. With a body mass index of >30, there appears to be a detectable increase in risk from obesity-related illnesses.1 It is the most important common risk factor for type 2 diabetes, and the incidence of this disease has closely tracked the increase in obesity rates worldwide.2 Troublingly, obesity is also related to many other serious human health outcomes, and the list includes malignant disease, liver disease, polycystic ovary syndrome, and osteoarthritis.1 Thus, the overall healthcare burden attributable to obesity is immense. Although the most commonly advanced reasons for obesity are the widening availability of low-cost high-fat diets, certain food marketing practices, and institutionally driven reductions in physical activity,3 alternative hypotheses have been posited. The multifactorial causality for obesity is well established and includes genetic, dietary, economic, psychological, reproductive, pharmacological, and environmental factors.4

Airborne pollution as a risk for human health other than pulmonary/respiratory disorders is an emerging concept, with the recognition of novel mechanisms having been discovered in recent decades. Particulate air pollution or the presence of particulate matter (PM) in inspired air has received attention in recent decades. Particulate air pollution is an emerging concern, with the recognition of novel mechanisms having been discovered in recent decades. Particulate air pollution or the presence of particulate matter (PM) in inspired air has received attention in recent decades. Particulate air pollution is an emerging concern, with the recognition of novel mechanisms having been discovered in recent decades. Particulate air pollution is an emerging concern, with the recognition of novel mechanisms having been discovered in recent decades. Particulate air pollution is an emerging concern, with the recognition of novel mechanisms having been discovered in recent decades. Particulate air pollution is an emerging concern, with the recognition of novel mechanisms having been discovered in recent decades. Particulate air pollution is an emerging concern, with the recognition of novel mechanisms having been discovered in recent decades.

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None.

Figure. Hypothesized mechanism of insulin resistance and adipocyte hypertrophy in mice exposed to PM2.5. PM in inspired air can activate immune-competent cells such as monocytes and macrophages. Xu et al8 report that such exposure induces increased adipocyte diameter via NAD(P)H oxidase activation and insulin resistance, ostensibly through immune activation and cytokine release. The authors also document changes in vasomotor responses in response to agonists, as well as insulin. The dashed arrow represents a less-established mechanistic link between inspiring PM2.5 and endothelial dysfunction. Insulin resistance and obesity, attributable to changes in adipocyte size and numbers, can in turn feed back to the vasculature compounding the deleterious phenotype.

this regard between wild-type mice and p47phox knockout animals, suggesting other sources of reactive oxygen species or alternative mechanisms for abnormal endothelial functions caused by PM2.5. Indeed, this result would also not reconcile or alternative mechanisms for abnormal endothelial functions caused by PM2.5. Indeed, this result would also not reconcile with the previous findings of the group that both p22phox and p47phox mRNA levels were increased in aortic tissues harvested from Sprague–Dawley rats that had been infused with angiotensin II and exposed to PM2.5 in inspired air. Whether the difference can be explained by interspecies or different age of mice for PM2.5 exposure is unknown. In addition, why global improvement of insulin signaling in p47phox knockout mice had minimal effects on endothelial function in PM2.5 exposed mice warrants further investigation.

The study by Xu et al8 has provided novel biological insights into the molecular basis of PM2.5-induced intracellular events, highlighting the activation of NAD(P)H oxidase on PM2.5 exposure. More specifically, it points to delayed effects of air pollution with consequences that may remain obscure for decades, adding to the insidious nature of this proposed hazard. Thus, this study might be potentially important because it has addressed the impacts of airborne PM2.5 on the functioning of signaling pathways with significant roles in redox homeostasis and inflammation.

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