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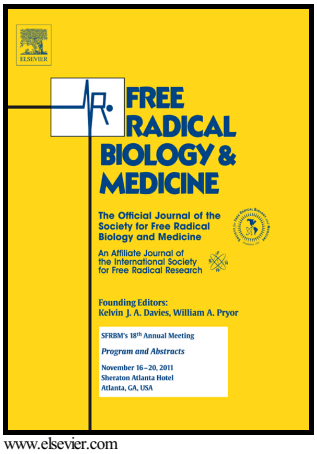

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Role of oxidative stress in cardiovascular disease outcomes following exposure to ambient air pollution

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Abstract

Exposure to ambient air pollution is associated with adverse cardiovascular outcomes. These are manifested through several, likely overlapping, pathways including at the functional level, endothelial dysfunction, atherosclerosis, pro-coagulation and alterations in autonomic nervous system balance and blood pressure. At numerous points within each of these pathways, there is potential for cellular oxidative imbalances to occur. The current review examines epidemiological, occupational and controlled exposure studies and research employing healthy and diseased animal models, isolated organs and cell cultures in assessing the importance of the pro-oxidant potential of air pollution in the development of cardiovascular disease outcomes. The collective body of data provides evidence that oxidative stress (OS) is not only central to eliciting specific cardiac endpoints, but is also implicated in modulating the risk of succumbing to cardiovascular disease, sensitivity to ischemia/reperfusion injury and the onset and progression of metabolic disease following ambient pollution exposure. To add to this large research effort conducted to date, further work is required to provide greater insight into areas such as (a) whether an oxidative imbalance triggers and/or or worsens the effect and/or is representative of the consequence of disease progression, (b) OS pathways and