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REVIEW ARTICLE

Exposure to Ambient Ultra-Fine Particles and Stroke

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ABSTRACT

Stroke is one of the main causes of death attributed to air pollution. Significant research has now shown that urban air pollutants exposure has been established as a source of neuroinflammation and oxidative stress that causes Central Nervous System (CNS) disease. Transition metals, Particulate Matter (PM) including fine particles (PM ≤ 2.5 μm, PM 2.5) and ultra-fine particles (UFPs, PM <0.1 μm, PM 0.1), nitrogen oxide, and ozone are potent or oxidant that capable of producing Reactive Oxygen Species (ROS) can reach the brain and affect CNS health. Numerous biological mechanisms are responsible that are not well understood. Recent studies suggest that changes in the Blood-Brain Barrier (BBB) and or leakage and transmission along the olfactory nerve into the Olfactory Bulb (OB) and microglial activation are the key factors of CNS damage following exposure to air pollution. This preliminary review cites evidence that ambient PM exposure is one of the causes of stroke.

INTRODUCTION

Stroke, one of the leading causes of disability and death, is a serious illness characterized by severe side-affects and poor survival. Estimated that stroke can be lead to approximately 10% of all deaths and 5% of all the Disability-Adjusted Life Years (DALY) worldwide [1], with the highest incidence of stroke in middle and low-income countries [2]. The stroke global burden has been increasing rapidly over the past decades, as differences between high, middle, and low-income countries increase [2]. The harmful effect of air pollutants exposure on stroke, as an emerging risk factor, is currently very worrying. By the modifiable risk factors controlling, such as air pollution exposure, the stroke high burden can be reduced by up to 90% [2,3]. Some research has examined the relationship between short-term ambient PM exposure and ischemic stroke over the past two decades [4]. Although previous studies have shown that short-term PM exposure is associated with an ischemic stroke higher risk [5,6], several study gaps remain. For instance, what is the relationship between ischemic stroke and short-term exposure to PM at the national or regional level and what is the role of different parts of PM in the association? The PM1 is a main component of PM 2.5. Some of the studies have shown that PM1 has stronger effects than PM2.5 on health outcomes [7,8], but its relationship is unclear with ischemic stroke. Besides, most previous findings have been conducted impact of air pollution exposure on stroke in a local city. Inconsistencies in results, involving estimates of different pollutant effects and latency time, due to the various factors (such as designs and study methods, different study areas), remain.

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Therefore, studies focusing on the short-term impact of fine particles on stroke using an advanced multi-urban plan and statistical method are needed, to reduce air pollution PM and prevent ischemic stroke.

Stroke

A stroke in the brain is caused by ischemia that led to sensorimotor disorders that could potentially brain damage or cause dementia. The stroke can be classified according to the cause of ischemia. Ischemic stroke results from atherothrombotic blockage of blood vessels inside the brain or vessels that lead to the brain (such as carotid arteries), or bleeding from the brain due to rupture of cerebral arteries and cerebral hemorrhage. In any case, oxidative stress is one of the major features of ischemia, and the ischemia caused damage by sudden tissue reperfusion and pathophysiological changes in the cells of the affected brain area.

Oxidative stress following air pollution exposure in the stroke

Overview of epidemiological evidence: Some of prior epidemiological surveys have shown fine PM acute effects on ischemic stroke. A survey examined the relationship between ischemic stroke and short-term air pollutants exposure (PM2.5 and PM10) using 94 studies in 28 countries. The findings of this study showed that the increase in stroke was related to an increase of 10 µg/m3 in PM2.5 and PM10 [4]. Many epidemiological evidences in different parts of the world show that air pollution exposure is associated with an increase in stroke [9,10]. PM communications are more consistent than communications related to air pollution gaseous [4] and in many guidelines, PM levels are under levels of air pollutants [11]. The production of free radicals from PM has been shown to increase stroke by increasing coagulation and blood coagulation [12], which corresponds to stronger links between ischemic stroke and air pollution exposure rather than hemorrhagic stroke [11]. In the survey of oxidative stress biomarkers in blood samples of non-smokers cerebrovascular disease patients, in all subjects NO_2 and PM2.5 were associated with methylation of genes associated with inflammation and oxidative stress [13].

Animal study: While challenging that *in vitro* and *in vivo* models have provided witness that exposure to air pollutants exacerbates stroke and that oxidative stress and inflammation are its main mechanisms [9]. Ambient UFPs, with their known ability to produce in vitro free radicals, has been shown to cytokine release and alter glutamate signaling in brain after inhalation in rats [14]. After the instillation of the PM10 in mice, was observed the upregulation of homoxygenase expression in the brain [15]. The effects of UFPs on brain are related to the level of metals transition in PM, potentially playing an important role in the production of metal-derived free radicals [16,17]. It was found that PM due to oil combustion causes great changes in markers of oxidative stress in hearts and lungs of hypertensive rats prone to stroke compared to healthy or hypertensive cases that are not susceptible to stroke [18]. Was suggested that after inhaling vehicle exhaust gases, oxidative stress plays an important role in disrupting the BBB [19-21]. Indeed, the photochemical induction of the thrombosis in cerebral arteries was intensified after pulmonary DEPs exposure. The effect can be prevented after co-administration of the antioxidant cysteine [22]. Our recent findings support the presence of the inflammatory effects in the brain tissue pathological samples whit cognitive and emotional impairment following exposure to DEPs. For example, anxiety and depression in male mice [23], hippocampal inflammatory cytokine response, and altered morphology [19,24], and disorders in learning and memory [25,26].

***In vitro* study:** The CNS cells antioxidants reserve have a less than many other cells types [27,28]. To demonstrate that your PM can induce cytotoxicity and oxidative stress, cultured neurons and microglial cells (cells such as macrophages of the nervous system) have been used [9]. Free radical production from ambient PM regulated channel of N-Methyl-D-Aspartate (NMDA) (which is involved in hypoxia neuronal damage) can be cultured in glial and neuronal cells [14]. In the PC-12 cells (as a cell model for the neural utilization) have been found similar observations for direct action of DEPs on dopamine signaling [29]. In addition, can be induced the ischemic periods in cultured brain sections that cause molecular changes because they have the demonstrative properties of a response to cerebral ischemia in vivo. The use of this tissue preparation with different types of UFPs can cause changes of cellular–molecular characteristics of stroke response (such as changes in the NMDA channel activity, disruption in glutamatergic and dopaminergic). In most cases, such factors are associated with oxidative stress and inflammation [19]. For the cerebral vessels model, can be used primary cultures of microvascular cells. The production of ROS is the prominent mechanism in that DEPs can disrupt the function of the endothelial cells which line these vessels inner surface [30]. A potential primary target of the underlying disease course of cerebrovascular disease represents by these cells. Direct exposure to DEPs to isolated capillaries in the brain increases inflammation and oxidative stress and may alter the permeability of the blood-brain barrier [31]. While there is discussion as to whether the inhaled air pollution particles in sufficient numbers can reach the brain and penetrate throughout the brain tissue, in the laboratory this means that inhalation of UFPs can increase be susceptibility to consequences of the stroke.

Exposure to PM and ischemic stroke

Exposure to air pollution PM increases antioxidant activity, inflammation, and activates circulating platelets, and decreases enzyme activity and vascular endothelial function, the latter of which may increase blood clotting and peripheral thrombosis [32,33]. Evidence has shown that NO_2

is linked with plasma fibrinogen [34] and also associated with PM2.5, which causes increased coagulability through secretion of cytokines [35]. There is a concern that the observed health effects related to NO_2 may be the result of air pollution UFPs exposure or traffic-related emissions [36]. Exposure to PM2.5 leads to oxidative stress and atherosclerosis in the heart. These conditions may reduce the heart rate variability, modulate visfatin, and induce the CD36-dependent 7-ketocholesterol that accumulates in macrophages [37-40].

The short-term exposure effects of environmental PM on stroke have been investigated by several surveys. For example, a cross-sectional multi-urban Case-crossover showed that per10 µg / m3 increase in concentration of PM10 exposure during 0-6 days, was associated with 0.7% increase in ischemic stroke [41]. Another study conducted showed that increasing every 10 µg/m3 of PM2.5 exposure per day, was significantly related with 0.31 in ischemic stroke [6]. PM1 may be harmful more than PM2.5 because it is characterized by a smaller size and a higher ratio of surface-to-volume. PM1 contains more toxicant effects and able to penetrate deep into the lungs [42,43]. Previous findings indicate a strong effect of PM1 on cardiovascular mortality relative to PM2.5 [7,44]. The underlying biological mechanisms about the association of ischemic stroke and PM exposure are not fully understood. Previous surveys have indicated some of the potential pathways. The evidence suggests that PM exposure may cause endothelial dysfunction and further lead to small-vessel stroke [45]. Exposure to environmental PM pollution is related to increased ischemic stroke risk by thrombosis promotion via several pathways including inflammation and oxidative stress, platelet activation, fibrinolysis disorder, and epigenetic changes [46]. Air pollution PM was related to a wide range of the health consequences, including some diseases associated with stroke (e.g., diabetes and hypertension) [47]. The other study findings show that the relationship between air pollution exposure and stroke morbidity was greater in the elderly [48]. Thus, the effect of the age modification on the relationship between stroke morbidity and air pollutants needs further research. Additionally, vascular risk factors, for example hyperlipidemia and hypertension, also may modify these relationships, which should be considered in future study. Seasonal analysis showed that a statistically significant relationship was between the concentration of ambient air pollutants and stroke only in winter; also observed that the harmful effects of SO_2 and NO_2 are stronger in the cold season [49]. Due to the possibility of temperature inversion, the concentration of air pollutants is usually higher in winter than in summer. The component of substances carried by the particles may be vary between different seasons, which influence this effect. Air pollutants exposure, a risk factor for stroke, has received much attention in recent years [50]. Effective policies to improving air quality, according to the PM-stroke association, can help reduce the stroke burden.

Although some of the findings have shown the adverse effects UFPs on various health outcomes [51,52], many study gaps still remain. The long-term UFPs exposure effects on the health of at-risk populations are significant. Therefore, further studies need to be conducted on exposure to air pollution PM and health effects. Also, UFPs measurement monitoring technology and tools need to be improved to give more information about the temporal and spatial trends of UFPs concentrations.

CONCLUSION

Exposure to PM10, PM2.5 and UFPs showed acute side effects of ischemic stroke. Due to the occurrence of temperature inversion in the cold seasons of the year, exposure to UFPs will increase and will have more severe effects. Implementing effective air pollution control policies can be helpful in reducing the stroke burden. Further researches are needed to discover the UFPs effects on human health. Also, advanced technology and tools for PM monitoring and control need to provide a little more information about the sources of PM pollution.

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