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BACKGROUND

VTE is a serious medical condition characterized by the formation of a thrombus in a vein that obstructs blood flow. VTE may progress from deep vein thrombosis (DVT) to pulmonary embolism, in which a fragment of the clot travels to the lungs as an embolus. VTE affects approximately one million individuals each year in the United States and remains a significant cause of preventable morbidity and mortality worldwide.

While traditional risk factors such as immobility, malignancy, and inherited thrombophilia are well established, emerging evidence suggests that environmental exposures play an important but often overlooked role in thrombotic disease. Increasing air pollution, widespread microplastic contamination, and extreme weather conditions driven by climate change may contribute to prothrombotic states through inflammatory, endothelial, and microvascular mechanisms.

METHODS

A focused review of peer-reviewed literature was conducted evaluating the relationship between environmental factors and venous thromboembolism. Emphasis was placed on population-based cohort studies assessing long-term air pollution exposure, experimental and epidemiologic studies examining microplastic exposure, and clinical studies evaluating the effects of extreme weather and cold temperatures on coagulation and blood rheology. Findings were assessed for epidemiologic associations, biological plausibility, and proposed mechanistic pathways.

MECHANISTIC EFFECTS OF AIR POLLUTION

Mechanistic studies support epidemiologic findings, demonstrating that air pollution promotes endothelial dysfunction, systemic inflammation, platelet activation, and altered fibrin clot structure. These effects create a prothrombotic environment that increases susceptibility to venous clot formation.

EXTREME WEATHER AND COLD-INDUCED THROMBOSIS

Extreme weather conditions, particularly cold exposure, have been associated with physiological changes favoring thrombosis. Cold temperatures induce peripheral vasoconstriction, hemoconcentration, increased blood viscosity, elevated platelet activity, and inflammatory shifts. These mechanisms may explain the higher incidence of VTE observed during winter months.

AIR POLLUTION AND VENOUS THROMBOEMBOLISM RISK

Strong evidence linking exposure to air pollution with an increased risk of VTE was identified. In the Multi-Ethnic Study of Atherosclerosis and Air Pollution (MESA Air), 6,651 adults were followed for up to 18.5 years (Figure 1). Higher long-term exposure to fine particulate matter (PM_{2.5}), nitrogen dioxide (NO₂), and nitrogen oxides (NO_x) was associated with a significantly increased incidence of VTE. Ozone exposure was not significantly associated with VTE risk.

After multivariable adjustment, hazard ratios for VTE increased per interquartile rise in PM_{2.5}, NO₂, and NO_x, as shown in Figure 2. These findings were robust across multiple sensitivity analyses.

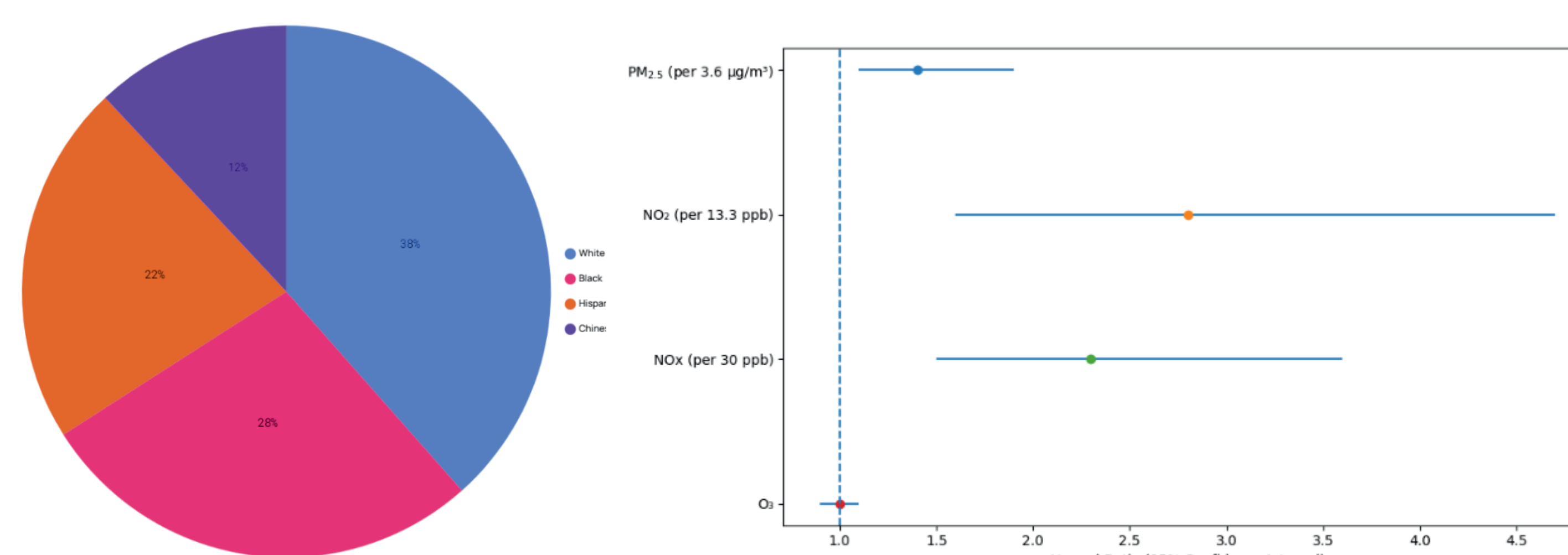


Figure 1: Racial Cohorts from the MESA Study

Figure 2: VTE Risk Interquartile Increase in Air Pollutants (MESA Air, Continuous Models)

MICROPLASTICS AS EMERGING THROMBOTIC RISK FACTORS

Emerging evidence implicates microplastics (MPs) as a novel thrombotic risk factor. Experimental studies have shown that inhaled or ingested MPs can enter systemic circulation, where they are phagocytosed by immune cells and form MP-labeled cells that obstruct capillaries, reduce blood flow, and mimic thrombus formation in animal models (Figure 3)

Epidemiologic analyses have associated higher environmental MP concentrations with increased prevalence of cardiovascular and metabolic diseases closely linked to thrombosis. Strategies to reduce MP exposure are summarized in Table 1.

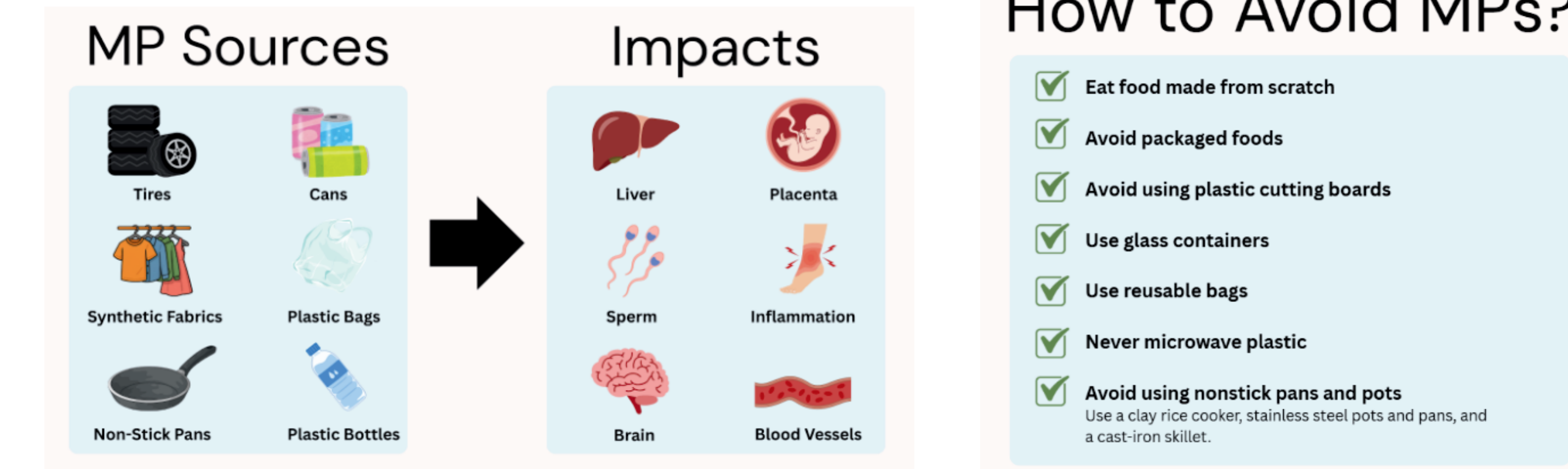


Figure 3: Different ways of exposure to microplastics, impact on health

How to Avoid MPs?

- Eat food made from scratch
- Avoid packaged foods
- Avoid using plastic cutting boards
- Use glass containers
- Use reusable bags
- Never microwave plastic
- Avoid using nonstick pans and pots
Use a clay rice cooker, stainless steel pots and pans, and a cast-iron skillet.

Table 1: How to avoid MPs?

CONCLUSIONS

Environmental factors, including air pollution, microplastic exposure, and extreme weather conditions, appear to meaningfully contribute to the development and incidence of venous thromboembolism through procoagulant, inflammatory, and microvascular mechanisms. Although establishing direct causality is challenging, the consistency of epidemiologic findings combined with strong mechanistic plausibility highlights environmental exposure as an important and potentially modifiable risk factor. Incorporating environmental considerations into thrombosis research, clinical risk assessment, and public health policy is essential to reducing the thrombotic disease burden amid rising pollution and climate change.