Cognitive Vitality Reports® are reports written by neuroscientists at the Alzheimer's Drug Discovery Foundation (ADDF). These scientific reports include analysis of drugs, drugs-in-development, drug targets, supplements, nutraceuticals, food/drink, non-pharmacologic interventions, and risk factors. Neuroscientists evaluate the potential benefit (or harm) for brain health, as well as for age-related health concerns that can affect brain health (e.g., cardiovascular diseases, cancers, diabetes/metabolic syndrome). In addition, these reports include evaluation of safety data, from clinical trials if available, and from preclinical models.

Air Pollution: Risk and Avoidance

Evidence Summary

Air pollution is a significant health hazard that is modifiable by both individual and societal action.

Brain health risk: Air pollution is one of the top modifiable risk factors for cognitive decline and dementia. It is associated with increased risk of dementia and potentially a faster rate of decline.

Aging and related health risk: Air pollution is a leading risk factor for death and increases risk of a variety of health conditions including cardiovascular disease, respiratory ailments, and certain kinds of cancer.

Safety concerns: Air pollution poses a threat to human health. Individual action and governmental regulation can effectively reduce air pollution levels.

Route(s) of exposure: Inhalation	**Dose**: Preferred air quality index (AQI) at 50 or below	**Common subtypes** Ground-level ozone (O3), also known as smog Gases such as CO, nitrogen oxides (NOX, NO2), sulfur oxides (SO2) Particulate matter (PM) of varying sizes (ultra-fine PM, PM2.5, PM10)
	BBB: Many air pollutants are BBB penetrant	
Clinical trials: N/A	**Observational studies**: Largest study included over 63 million Medicare patients	

What is it?

Air pollution has many components including particulate matter (PM), gases, metals, and organic compounds. Harmful components may include sulfur dioxide, nitrogen oxides, acid aerosols, carbon monoxide, and ozone (O_3). Major components of PM include "transition metals, ions (sulfate, nitrate), organic compound, quinoid stable radicals of carbonaceous material, minerals, reactive gases, and materials of biologic origin." (Valavandis et al., 2008). In urban areas, vehicle exhaust is the primary source of PM ambient air pollution, but air pollution can also be generated by cooking indoors, smoke, industry, and natural causes of pollution such as forest fires. Particulate matter has been heavily implicated in cardiovascular and respiratory premature mortality, particularly the fine particulates below 2.5 um in diameter ($PM_{2.5}$) that deeply penetrate and lodge within the lungs (up to 50% are retained in the lung parenchyma). Ultrafine particles (<100nm) are also a concern.

Air pollution is one of the leading risk factors for death in the world; the 2018 Lancet Commission Report on Pollution and Health estimated that air pollution is responsible for 16% of all deaths worldwide (Landrigan et al., 2018). It is thought to affect human health in numerous ways, from cardiovascular and cardiopulmonary health to cancer to cognitive health to premature death and beyond. It has also been linked to disorders as diverse as diabetes to osteoporosis to systemic inflammation (reviewed by Forum of International Respiratory Societies' Environmental Committee, Part I and Part II).

Air pollution exposure in utero and in children has been associated with cognitive deficits, autism-spectrum disorders, schizophrenia, and low birth weight (Woodward et al., 2015, Clifford et al., 2016, Calderón-Garcidueñas et al., 2016). Similarly, indoor air pollution, primarily from burning wood and other solid fuel, is estimated to be a major health risk (WHO Fact Sheet). This report, however, focuses primarily on ambient (outdoor) air pollution effects in older adults.

Brain Health Risk: Air pollution is one of the top modifiable risk factors for cognitive decline and dementia. It is associated with increased risk of dementia and potentially a faster rate of decline.

Types of evidence:

- 1 commission report
- 4 meta-analyses and systematic reviews
- 6 systematic reviews
- 2 clinical trials
- 15 observational studies
- 3 reviews
- 2 laboratory studies

Human research to suggest risk of dementia, accelerated decline, or impaired cognitive function:

In 2020, air pollution was added to the list maintained by the Lancet Commission on Dementia Prevention, Intervention, and Care of potentially modifiable risk factors for dementia. In 2023 the WHO also added air pollution to their list of modifiable risks. As per the Lancet Report, eliminating late-life exposure to air pollution would decrease the prevalence of dementia by 2% worldwide. For comparison, eliminating midlife obesity or late life diabetes would reduce prevalence of dementia by 1% each, and eliminating midlife hypertension would reduce the prevalence of dementia by 2%. The report found that high NO_2 increased risk of dementia (adjusted HR=1.2; 95% CI, 1.0 to 1.3), as did high $PM_{2.5}$ levels (adjusted HR=1.1; 95% CI, 1.0 to 1.2). While air pollution does not appear to be the greatest risk factor for dementia in terms of hazard ratio, the size of the population exposed to this risk factor renders air pollution a serious public health issue (Livingston et al., 2020).

Several systematic reviews and/or meta-analyses have examined the association between exposure to air pollution and dementia, including four systematic reviews & meta-analyses published in 2023. These publications utilized different approaches to answer slightly different questions, and thus their included studies and overall findings are not identical. The authors report some differences in which pollutant is most deleterious to neurological health. The reports all grapple with some of the same limitations, such as the complexity of accounting for exposure to air pollutants when these concentrations can vary even within zip codes and exposure to the air pollution varies based on human behavior. Nonetheless, the studies consistently find associations between exposure to air pollution, particularly $PM_{2.5}$ and SO_2, and incidence of dementia. The findings of the 2023 systematic reviews & meta-analyses are included below.

Wilker et al., 2023 examined the association between exposure to air pollution and dementia diagnosis. The meta-analysis included 16 studies of more than 16 million adults (18+ years of age) with exposure to air pollutions durations of at least a year and clinical dementia diagnoses. Their results found consistent associations between dementia and air pollution, even when the levels of pollutants were below current regulatory standards (12 $\mu g/m^3$ in the US; 20 $\mu g/m^3$ in the UK; 25 $\mu g/m^3$ in the EU). There were 14

included studies that examined $PM_{2.5}$, and the authors found that the overall hazard ratio per 2 µg/m^3 $PM_{2.5}$ was 1.04 (95% CI, 0.99 to 1.09). Seven of these studies used active case ascertainment and their calculated hazard ratio was 1.42 (95% CI, 1.00 to 2.02) The overall hazard ratio per10 µg/m^3 nitrogen dioxide was 1.02 (95% CI, 0.98 to 1.06; nine studies), and per 10 µg/m^3 nitrogen oxide was 1.05 (95% CI, 0.98 to 1.13; five studies). They found no clear association with dementia for ground-level ozone (HR per 5 µg/m^3=1.00; 95% CI, 0.98 to 1.05; four studies).

Abolhasani et al., 2023 investigated the association between exposure to air pollution and dementia diagnosis in cohort studies of adults who are more than 40 years of age. This meta-analysis included 17 studies comprising 91,391,296 patients, 5,521,111 of whom were diagnosed with dementia. Their results indicated that risk of dementia increased 3% per 1 µg/m^3 increment in $PM_{2.5}$ (HR=1.03; 95% CI, 1.02 to1.05). They found trends towards associations between dementia per 10 µg/m^3 increase increment in NO_X, NO_2, and ozone levels, though they were not statistically significant. This paper found high heterogeneity in the data.

Tang et al., 2023 analyzed cohort studies that looked at association between air pollution and dementia diagnosis in adults. They included 15 previously published studies along with new data from two cohort studies in China that the authors were involved with. When they compared the highest levels of pollutant exposure to the lowest, they found that the association of dementia was increased for $PM_{2.5}$ exposure (RR=1.11; 95% CI, 1.06 to 1.17), CO exposure (RR=1.60, 95% CI, 1.39 to 1.84) and NO_2 exposure (RR=1.14, 95% CI, 1.03 to 1.27).They did not find significant associations for PM_{10}, NO_x, or ozone.

Gong et. al., 2023 assessed 30 papers for association between particulate pollution specifically and dementia, and 19 papers for association between particulate pollution and cognitive function. They found that exposure to higher levels of $PM_{2.5}$ was associated with increased risk of AD (OR=1.65; 95% CI, 1.37 to 1.94). They also found significant associations between $PM_{2.5}$ exposure and all-cause dementia (pooled OR=1.30; 95% CI, 1.14 to 1.47) and Parkinson's disease (PD) (OR=1.17; 95% CI, 1.0 to 1.33). They found high heterogeneity in these results. They also found trends towards associations between exposure to particulate matter and cognitive function.

Studies have also indicated that air pollution can increase rate of cognitive decline in cognitively intact individuals, as reviewed in Kilian & Kitazawa, 2018. One study published after the review, Kulick et al., 2020, details results from a prospective study of aging and dementia in 4,821 cognitively intact older adult (65+ years of age) participants in northern Manhattan. Participants undergo cognitive testing every 18-24 months, and the mean follow-up is 6 years. The authors found a faster rate of cognitive decline in participants exposed to higher levels of air pollution, as did several of the other studies review by Kilian & Kitazawa, 2018.

Residual confounding is possible, as with all epidemiologic studies. Most epidemiologic studies did control for socioeconomic status, however, and a sensitivity analysis from a 2016 systemic review

concluded that socioeconomic differences are unlikely to be sufficient to account for the observed correlations (Power et al., 2016).

While much of the literature on the cognitive effects of air pollution has focused on elderly adults, there are also studies that suggest that air pollution can affect cognitive function in children and non-elderly cognitively intact adults, though the evidence is less clear for the latter group. A blinded study of 24 adult participants tested whether indoor air quality in terms of volatile organic compounds (VOCs), CO_2, and ventilation affected cognitive function. Participants in the study spent all day working in an office where the air quality was varied each day for six total days spread out over two weeks, and underwent cognitive testing each day. They found that cognitive performance was significantly higher on days when participants were exposed to better air quality as measured by lower CO_2 and lower VOCs; on average, scores were 61% higher on days with lower VOC concentrations, and 101% higher on days with lower VOC, CO_2 levels, and increased ventilation (Allen et al., 2016). A systematic review by Power et al., 2016 and another by Chandra et al., 2022 are both excellent reviews of the state of the research on the associations between air pollution and cognitive function / impairment.

Human research to suggest increased risk to patients with dementia:

While air pollution is considered a modifiable risk factor for dementia, it is less clear how air pollution affects patients already diagnosed with dementia.

In Lee et al., 2023, the authors analyzed the association between air pollution and cognitive decline in 269 patients with MCI or AD for a mean follow-up period of 4 years. The authors found that higher chronic exposure to SO_2 was associated with faster decline of memory, and that higher chronic exposure to $PM_{2.5}$ was associated with faster decline of visuospatial scores in patients that were also APOE4 carriers. They found no association between rate of cognitive decline and CO, PM_{10}, or NO_2.

A study in Taiwan compared cognitive decline of AD patients who lived in a city with higher pollution levels compared to AD patients who lived in a less polluted city. The authors found that patients living in the city with higher levels of CO, NO_2, PM_{10}, and SO_2 air pollution had faster rates of cognitive decline, with SO_2 having the greatest impact (Lin et al., 2022). However, other studies have not found effects of air pollution levels on rate of cognitive decline (Cleary et al., 2018).

Mechanisms of action for neurological harm identified from laboratory and clinical research:

Given the many components of air pollution, and even $PM_{2.5}$ it is not surprising that many mechanisms of neurological harm have been suggested (Levy et al., 2012; Schraufnagel et al., 2019 Part I and Part II). First, air pollution may impact brain health indirectly, through increased risk or exacerbation of health conditions that in turn increase risk of neurodegeneration. Air pollution can increase risk of cardiovascular conditions and stroke (Shah et al., 2015, Yusuf et al., 2020). Damage to the blood-brain

barrier and endothelial cells have been reported in animal models and some human pathology (e.g. Block & Calderon-Garciduenas, 2009, Calderon-Garciduenas et al., 2015). Air pollutants such as $PM_{2.5}$ have been linked to systemic inflammation which can affect health in a variety of ways.

Evolving evidence indicates air pollution may also have direct effects on the brain. In vivo rodent studies have reported that exposure to air pollutants can cause changes consistent with Alzheimer's disease such as an increase in Aβ42, tau hyperphosphorylation, neurofibrillary tangles, neuroinflammation and microglial activation, oxidative stress, DNA damage, damage to the blood-brain barrier, changes in neurotransmitter levels, and neurodegeneration. Studies have also found effects on rodent cognition such as learning and memory (Weuve 2014, Fougere et al., 2015, Yan et al., 2016, Cheng et al., 2016, Costa et al., 2018, Wilker et al., 2023, Kilian et al., 2023). For example, just 3 hours of exposure to air pollution led to 129% increases in Aβ40 & Aβ42 levels in wildtype mice (Kim et al., 2012). Changes in Aβ deposition have been observed in both wildtype and in AD model mice and have been accompanied by synaptic alterations such as decreased neurite density in the CA1 region of the hippocampus (Cacciottolo et al., 2017). $PM_{2.5}$ or ultrafine particles are thought to reach the brain via olfactory and cranial nerves and possibly via transfer from circulation to brain endothelial cells or trafficking of macrophage-like cells from the lung capillary bed to systemic circulation (Calderon-Garciduenas et al., 2013, Genc et al., 2012). In autopsy studies, PM has been found in olfactory bulb neurons (Calderon-Garciduenas et al., 2008) and magnetite nanoparticles have been broadly detected in the CNS (Maher et al., 2016).

Studies in humans on neurodegenerative pathology are also evolving. A long series of papers from Lilian Calderón-Garcidueñas at the University of Montana indicate that children and young adults in Mexico City, exposed to high air pollution levels, exhibit unusually early pathological signs of Alzheimer's and Parkinson's disease (e.g. Calderon-Garciduenas et al., 2016 & 2013). These signs have included cognitive impairment, prefrontal white matter lesions, proinflammatory cytokine and COX2 expression in the CNS, frontal tau hyperphosphorylation, beta-amyloid diffuse plaques, reduced CSF levels of A□1-42 & BDNF, and aggregated hyperphosphorylated alpha-synuclein in the brainstem and olfactory nerves. Despite these alarming results, studies from other laboratories or on older adults are limited. Smaller brain volumes or white matter hyperintensities have reported in some cohorts (Chen et al., 2015, Casanova et al., 2016, Wilker et al., 2015) but not all (Wilker et al., 2016), with air pollution measured by proximity to major roads or $PM_{2.5}$ exposure.

APOE4 interactions:

APOE4 status did not influence the relationship between dementia and air pollution in one case-control study (Wu et al., 2015) but APOE4 carriers were more likely to show a negative correlation with air pollution and cognitive function in another study (Schikowski et al., 2015). A series of studies from young people exposed to pollution in Mexico City report that APOE4 exacerbates sensitivity to air pollution, for example with worsened cognitive deficits and more extensive Aβ42 immunoreactivity (e.g. Calderon-Garciduenas et al., 2016, 2008).

Cacciottolo et al., 2017 examined whether air pollution has a differential effect on APOE4 carriers. This study involved both APOE transgenic mice as well as a prospective study cohort of 65- to 79-year-old women living in the US and enrolled in the Women's Health Initiative Memory Study (WHIMS). After controlling for factors including education, income, and health, the authors found that exposure to high levels of $PM_{2.5}$ was associated with increased risk of cognitive decline and dementia diagnosis, as compared to living in an area with low level of $PM_{2.5}$. This effect varied based on APOE genotype. Carrying APOE4 allele(s) was associated with higher risk of cognitive decline (ε3/3: HR=1.65; ε3/4: HR=1.93; ε4/4: HR=3.95) and dementia (ε3/3: HR=1.68; ε3/4: HR=1.91; ε4/4: HR=2.95.)

Using wildtype and transgenic AD model mice expressing human APOE3 or APOE4, the researchers also assessed the effects of repeated exposure to $PM_{2.5}$. For this study, exposure was 5 hours a day, 3 days a week, for 15 weeks. The mice who were exposed to $PM_{2.5}$ had significantly increased Aβ deposition, and this was exacerbated in the APOE4 mice as compared to the APOE3 mice. There were also synaptic aberrations in both wildtype and transgenic animals in the hippocampus after exposure.

Other studies have found that exposure to higher concentrations of air pollution is associated with more rapid cognitive decline, and that this association is particularly prominent in APOE4 carriers (Kulick et al., 2020; Lee et al., 2023).

Aging and related health risks: Air pollution is a leading risk factor for death and increases risk of a variety of health conditions including cardiovascular disease, respiratory ailments, and certain kinds of cancer.

Types of evidence:

- 1 systematic analysis
- 2 systematic review and meta-analyses or meta-analyses alone
- 1 Joint Professional Association Statement
- 1 Lancet Commission Report
- 1 actuarial modeling study
- 9 observational studies
- 5 reviews

Air pollution has been associated with a dizzying variety of health conditions, including obesity, chronic obstructive pulmonary disorder, diabetes, osteoporosis, allergies, asthma, skin aging, and a variety of cancers. The links between air pollutants and non-communicable diseases has been reviewed in depth in Schraufnagel et al., 2019, Part II.

Premature Death: INCREASED RISK WITH HIGHER AIR POLLUTION EXPOSURE

The WHO estimates that, worldwide, 6.7 million people a year die prematurely due to air pollution (WHO Fact Sheet). Globally, air pollution reduces life expectancy by an average of 20 months; for comparison, tobacco usage reduces average global life expectancy by 22 months. The reduction of average life expectancy by air pollution is not as great in high-income countries, where the reduction is calculated to be 5 months. For comparison in this high-income country context, this reduction in average life expectancy is greater than the reduction resulting from breast cancer in these countries (Apte et al., 2018).

Cardiovascular and Cardiopulmonary Health: INCREASED RISK WITH HIGHER AIR POLLUTION EXPOSURE

As detailed in a joint statement in 2021 from the World Heart Federation, American College of Cardiology, American Heart Association, and the European Society of Cardiology, air pollution increases risk of cardiovascular and cardiopulmonary diseases such as ischemic heart disease, stroke, and chronic obstructive pulmonary disease (COPD). It is thought that particulate matter is particularly associated with cardiovascular risk, whereas ozone and nitrogen oxides are particularly associated with respiratory issues. The proposed underlying mechanism is that particulate matter causes oxidative stress and inflammation. This oxidative stress and inflammation, along with the physical presence of particulate matter in the circulatory system, promotes a number of pathologies such as hypertension, vasoconstriction, and atherosclerosis, which can lead to cardiovascular events.

In 2018, it was estimated that air pollution accounts for 19% of all cardiovascular deaths, 23% of all ischemic heart disease deaths, and 21% of all stroke deaths (Hadley et al., 2018).

A 2020 prospective cohort study of 155,722 participants from around the world examined the associations between modifiable risk factors and cardiovascular disease and mortality. They found that air pollution accounted for 13.9% of the risk of cardiovascular disease (Yusuf et al., 2020).

Cancer: INCREASED RISK OF CERTAIN SUBTYPES OF CANCER WITH HIGHER AIR POLLUTION EXPOSURE

Exposure to higher levels of air pollution is associated with a variety of cancers. Particulate matter is particularly linked with lung cancer, with one 2014 systematic review and meta-analysis finding that the meta-relative risk for lung cancer was1.09 (95% CI, 1.04 to 1.14) for each increase of 10 $\mu g/m^3$ of $PM_{2.5}$ (Hamra et al., 2014). Air pollution overall is also associated with increased incidence of and/or mortality from cancer of the bladder and kidney, as well as colorectal cancer and gastric cancer. Preclinical evidence has indicated that certain pollutants such as ground-level ozone and nitrogen oxides may influence cancer-related cellular events such as inflammation, changes in telomere length, altered expression of genes involved in DNA damage and repair, and epigenetic changes (reviewed by Schraufnagel et al., 2019, Part II).

Safety concerns: Air pollution poses a threat to human health. Individual action and governmental regulation can effectively reduce air pollution levels.

Types of evidence:

- 1 systematic review and meta-analysis
- 2 meta-analyses
- 1 modeling study
- 2 observational studies
- 2 reviews
- 2 laboratory studies

Details: In 2017, 2.6 million premature deaths worldwide were estimated to be caused by ambient air pollution, largely due to exposure to PM below 10 microns (PM_{10}). Of these deaths, 72% related to ischemic heart disease and stroke, 14% to lung cancer, and 14% to respiratory illness. The list of diseases includes atherosclerotic cardiovascular disease, COPD, lung cancer and lung infection, myocardial infarction, stroke, and others under investigation (Laumbach et al., 2015). In 2019, an estimated 4.2 million premature deaths were due to outdoor air pollution alone. The WHO estimates that 37% of outdoor air pollution-related premature deaths were due to ischemic heart disease and stroke, 18% and 23% of deaths were due to chronic obstructive pulmonary disease and acute lower respiratory infections respectively, and 11% of deaths were due to cancer within the respiratory tract. As of 2023, the WHO estimates that outdoor and household air pollution combined cause 6.7 million premature deaths annually (WHO Fact Sheet).

There is no lower threshold for risk to $PM_{2.5}$. In European countries where many cities have air quality levels within WHO recommended guidelines, exposure to human-generated PM has been estimated to shorten average life expectancy by 8.6 months (WHO Fact Sheet). Similarly, in Canadian regions with low average exposure to $PM_{2.5}$, the risk of non-accidental mortality increases by 26% with each 10ug/m^3 increase in exposure (HR=1.26; 95% CI, 1.26 to 1.84) (Pinault et al., 2016). Short-term changes in exposure have been temporally linked to cardiovascular and stroke-related hospital admissions and mortality (Levy et al., 2012, Shah et al., 2015).

Susceptibility to health risks from air pollution can vary. The elderly and those with asthma, COPD, diabetes, and cardiovascular diseases are thought to be vulnerable. Children and the developing fetus may be particularly susceptible as well. Children and young adults appear susceptible to subclinical effects like inflammation and deposits of $PM_{2.5}$ in the lung during exercise. In one study, people who have never smoked were particularly harmed by air pollution (mortality HR=1.76; 95% CI, 1.15 to 2.69) although the difference from smokers was not significant (Pinault et al., 2016). The same study also reported a non-significant trend for increased harm in obese individuals. Elderly people may be particularly sensitive. It has been proposed to exacerbate pathological aging and frailty through chronic

inflammation (e.g. increased CRP), systemic oxidation from PAHs and transition metals, metabolic disorders caused by $PM_{2.5}$ and dioxin, DNA damage from PM and other toxins, and epigenetic effects such as DNA methylation and increased histone acetyl transferase activity (Fougere et al., 2015).

Which toxins are most harmful for air pollution? Ozone, also known as smog, is less likely to contribute to mortality rates according to a meta-analysis of data from 8 cohorts (Atkinson et al., 2016). $PM_{2.5}$ and sulfate exposure share similar associations with increased mortality from all-cause, lung cancer, or cardiopulmonary causes (Pope et al., 1994). For stroke-related mortality or hospitalizations, the associations appear to be similar for carbon monoxide, nitrogen dioxide, sulfur dioxide, and $PM_{2.5}$ (Shah et al., 2015). $PM_{2.5}$ itself is a complex and varied mixture of molecules but evidence is very limited on which types are most harmful (Levy et al., 2012). The organic components of PM have been suggested to be involved with atherosclerotic disease based on preclinical research (Keebaugh et al., 2015).

Actions to reduce exposure:

Some actions can reduce an individual's exposure (Laumbach et al., 2015).

- Air pollution levels vary locally, daily, seasonally, and even by time of day. They can be monitored online at AirNow, with apps (e.g. PlumeAirReport, BreezoMeter, AirVisual), and even handheld devices. For example, community efforts using the AirCasting platform have been used to record $PM_{2.5}$ levels across NYC, observing up to 6 fold differences in $PM_{2.5}$ concentration (e.g. Wired article, TakingSpace.org). Consider staying inside on days with particularly bad air pollution. Shift exercise routes away from polluted areas such as roads – even moderate exercise can cause a 5 fold increase in the deposition of ultrafine PM in the lungs – or exercise indoors if possible. Do not avoid exercise, however. The cardiovascular benefits of exercise are likely to outweigh the risks of air pollution exposure, (for example commuting via bicycle) (Laumbach et al., 2015).
- Wear a mask if you must go outside when the air quality is poor. Well-fitting N95 masks and respirators can significantly reduce exposure to pollutants, including $PM_{2.5}$. Cloth and surgical masks do not offer the same kind of protection, as the former does not filter the small-size particles as well, and there can be too much leak around the latter (Kodros et al., 2021).
- If you do need to go outside on a poor air quality day, change your clothing when you return home. Showering can further reduce the amount of particles on your person.
- Breathing through the nose instead of the mouth can somewhat reduce lung penetration of particles (Laumbach et al., 2015).
- For many people, the highest exposure to PM occurs while driving. Exposure may be reduced by closing car windows and vents while in heavy traffic, installing a high efficiency particle filter, avoiding idling and long-warm-ups, especially in enclosed spaces, avoiding smoking in the car, keeping the vehicle tuned and maintained, and buying a low-emitting vehicle such as electric or hybrid. (Laumbach et al., 2015, CA Fact Sheet)

- Indoors, cooking is a major source of PM. The use of exhaust fans that vent outdoors may reduce exposure. Other common indoor sources include smoking, fires in wood stoves or fireplaces, candles, and incense (Fact Sheet).
- Staying indoors and closing the window can reduce but not avoid outdoor pollution without additional efforts. $PM_{2.5}$, ozone, and sulfate toxins penetrate into buildings and $PM_{2.5}$ specifically can remain in circulation. However, effective portable or central air cleaning systems are available such as in-duct HEPA filtration systems or stand-alone air purifiers (Laumbach et al., 2015).
- Indoor air quality monitors are available, allowing personalized efforts to reduce exposure in the home.
- Purchase lower-emitting items, including Energy Star certified appliances and items made with low- or non-VOC emitting materials, can reduce production of air pollutants.
- Organic waste degradation in landfills leads to methane production which can increase ground-level ozone levels. Composting organic waste, on the other hand, avoids methane production and thus can reduce air pollution. Individuals can compost at home or collect organic waste, which can then be picked up by composting services like NeighborhoodCompost, based in New Jersey or brought to compost collection sites like those run by GrowNYC.
- Plant-based diets are beneficial for human health and also for environmental health, as they generate less air pollutants like $PM_{2.5}$. Decreasing meat consumption, particularly of red meat, and increasing the portion of your diet that is plant-based can help reduce air pollution (Domingo et al., 2021).
- As some sources of air pollution can be driven by climate change, such as forest fires, individual actions to reduce consumption, both in terms of energy and material items, can add up to long-term useful effects.

The EPA has further guides for both ground-level ozone and particulate matter.

Despite all of these options to reduce personal exposure, air pollution is and will remain largely influenced by societal actions. History has shown that emissions can be successfully reduced. Average $PM_{2.5}$ levels declined by 37% from 2000-2015 in the USA (EPA). In Los Angeles from 1960 to 2010, maximum ozone levels reduced from 600 to 150 ppbv and VOC concentrations (volatile organic compounds, a source of PM) decreased by a factor of 50 (EOS.org).

Policies aimed at reducing factory emissions, managing agricultural waste in way that reduce emissions, ensuring access to clean energy sources for homes, shifting transport towards lower-emitting options such a bicycles or electric vehicles, utilizing energy generated from low-emissions fuel or combustion-free sources such as solar or wind, and reducing overall energy consumption and waste production all can have powerful effects on reducing air pollution (WHO Fact Sheet).

Research underway:

The evidence for dementia risk is rapidly evolving. More is undoubtedly underway. To date, no drugs or supplements have been shown to be effective at protecting against related toxicity, despite some claims for PUFAs and antioxidants.

Technology continues to innovate, lowering emissions from industry and vehicles and enabling individuals to reduce their exposure. A passive transparent air filter for high efficiency $PM_{2.5}$ capture was reported in 2015 (Liu et al., 2015). Vegetation in urban planning is another interesting possibility. $PM_{2.5}$ and NO_2 deposit onto vegetation at much higher rates than on hard non-porous surfaces. While common urban planning with occasional trees has minimal influence, more extensive vegetation planting in street canyons was predicted in one model to reduce street-level concentrations by 40-60% (Pugh et al., 2012, Salmond et al., 2016).

There are approximately 90 ongoing studies that involve air pollution based on ClinicalTrials.gov; no study is currently specifically examining dementia and air pollution. Several studies are investigating effects on risk factors for dementia, like cardiovascular health.

Search terms:
Pubmed, Google: air pollution

- Dementia, Alzheimer's, APOE4, human health, cancer, cardiovascular, premature death

Websites visited for air pollution:

- Clinicaltrials.gov