***Review Article***

# Geoengineering, Coal Fly Ash, and the New Heart-Iron Connection: Universal Exposure to Iron Oxide Nanoparticulates


## ABSTRACT

Globally, air pollution is the leading environmental cause of human disease and death, and it is a major contributor to cardiovascular disease. Air pollution damages the cardiovascular system by oxidative stress, inflammation, endothelial dysfunction, and pro-thrombotic changes. Ultrafine particulate matter from the combustion of fossil fuels delivers the most potent and harmful elements of air pollution. Coal fly ash is a rich source of nano-sized metal, iron oxide, and carbonaceous particles. Previous findings revealed that coal fly ash is widely utilized in undisclosed tropospheric aerosol geoengineering. Proper iron balance is central to human health and disease, and the harmful effects of iron are normally prevented by tightly controlled processes of systemic and cellular iron homeostasis. Altered iron balance is linked to the traditional risk factors for cardiovascular disease. The iron-heart hypothesis is supported by epidemiological, clinical, and experimental studies. Biogenic magnetite ($Fe_3O_4$) serves essential life functions, but iron oxide nanoparticles from anthropogenic sources cause disease. The recent finding of countless combustion-type magnetic nanoparticles in damaged hearts of persons from highly polluted areas is definitive evidence of the connection between the iron oxide fraction of air pollution and cardiovascular disease. Spherical magnetic iron oxide particles found in coal fly ash and certain vehicle emissions match the exogenous iron pollution particles found in the human heart. Iron oxide nanoparticles cross the placenta and may act as seed material for future cardiovascular disease. The pandemic of non-communicable diseases like cardiovascular disease and also rapid global warming can be alleviated by drastically reducing nanoparticulate air pollution. It is crucial to halt tropospheric aerosol geoengineering, and to curb fine particulate emissions from industrial and traffic sources to avoid further gross contamination of the human race by iron oxide-type nanoparticles.



***Keywords:*** *cardiology, particulates, aerosols, coal fly ash, climate intervention, particulate air pollution, magnetite, nanoparticles, geoengineering*


## 1. INTRODUCTION

Iron comprises nearly one-third of our planet's mass with the great majority existing in the core in highly reduced chemical state, and a lesser amount in oxidized form in the outer portions [1,2]. Iron is a critical element for the origin and development of life [3], but one that necessitates maintaining a delicate balance. Iron imbalance, including excessive or misplaced iron in the body, can promote a vast array of acute and chronic illnesses. It can truly be said that "*life exists at the interface of iron deficiency and iron sufficiency*" [4].

Iron in its various forms constitutes a significant component of air pollution [5]. Poor air quality is the greatest threat to human health, and currently over 95% of the world's population resides in areas with polluted air [6,7]. In 2004 the first American Heart Association scientific statement on "Air Pollution and Cardiovascular Disease" concluded that exposure to

particulate matter (PM) in air pollution contributes to cardiovascular morbidity and mortality [8]. Since that time the body of evidence has grown and strengthened, and the particulate matter ≤2.5 μm across (PM2.5) in air pollution is now considered a modifiable risk factor for cardiovascular disease [9].

Recently published data show independent associations between short-term exposure to particulate matter ≤10 μm across (PM10) and ≤2.5 μm across (PM2.5) and daily all-cause, cardiovascular, and respiratory mortality in more than 600 cities across the globe [10]. Ambient PM2.5 poses a major threat to global health, and fine particulate matter (less than 2.5 um) is considered the most important environmental risk factor for cardiovascular disease and death [11]. Air pollution is a major contributor to the global epidemic of non-communicable disease; it is also controllable and therefore a great many adverse health effects could be prevented [12].

Air pollution produces harmful effects on the heart and cardiovascular system by the development of pulmonary inflammation, secondary systemic inflammation, oxidative stress, endothelial dysfunction, and pro-thrombotic changes [13]. Radical oxygen species induce activation of monocytes and produce pro-atherogenic changes in lipoproteins involved in plaque formation. Air pollution predisposes to thrombus formation due to increases in coagulation factors and platelet activation and aggregation [14]. Due to their size, charge, and chemical composition of ultrafine particulate matter ≤0.1um or ≤=100 nm (UFP), it is much easier UFP to cross the pulmonary epithelium and the lung-blood barrier than PM2.5 and larger particles [15]. The translocation of UFP to the bloodstream has multiple detrimental effects on the cardiovascular system. After depositing on vascular epithelium, UFP enhances oxidative stress, resulting in plaque instability which can result in hemorrhage or thrombus (clot) formation [16].

## 2. METHODOLOGY

Review articles are written in different ways to achieve different results [17,18]. The approach we use in this review is to gather seemingly unrelated facts scattered through the literature, which when arranged logically, reveal causal relationships and lead to new understanding [19].

## 3. PRINCIPAL ORIGIN OF AIR-POLLUTION PARTICULATE IRON

Air pollution particles are typically solids ≤ 10 μm across that occur in the troposphere and originate from a variety of sources including incomplete biomass burning, combustion-ash of fossil fuels, soot, volcanic eruptions, wind-blown road debris, sand, sea salt, biogenic material [20] and, significantly, pyrogenic coal fly ash from unfiltered industrial exhaust [21-24] and from geoengineering applications [25-31]. Tropospheric particulates have short atmospheric residence times ranging from days to a few weeks before they settle to ground [32-35]. The residence time of stratospheric air pollution particulates [36], on the other hand, is considerably longer, on the order of months [32-35,37], but these pollution particulates in falling to ground briefly become tropospheric particulates. Tropospheric mixing by convection assures pollution-exposure to virtually everyone [38].

Coal is the most abundant fossil fuel on earth, and when burned, its heavy ash settles beneath the burner while its light “fly ash”, coal fly ash (CFA), condenses and accumulates in the hot gases above the burner producing the characteristic spherical morphology of CFA particles resulting from the surface tension of the melt [39]. Coal fly ash contains at least 39 elements that were occluded when coal formed and became concentrated when the coal burned [40]. The most volatile elements, which are the last to condense, are strongly enriched on surfaces of the smallest CFA particles [41].

Coal fly ash is a significant source of the coarse, fine, and ultrafine particulate air pollution particles that directly threaten human and environmental health [42]. Emissions of CFA from Western power plants, unlike many in China [43] and India [44], are reduced by use of electrostatic precipitators or filters, but the collection efficiency of these technologies is lowest for ultrafine and nanoparticles. As a result, industrial CFA emissions are dominated by particles that can travel long distances in the atmosphere and be inhaled directly into the lung [45].

Coal fly ash is an abundant and cheap waste product that requires little additional processing for use in climate engineering operations [25-31,46]. Jet-sprayed into the troposphere, the CFA represents a deliberate and severe form of air pollution [47,48]. CFA particles directly affect climate because they not only scatter, but absorb incoming solar radiation and outgoing terrestrial radiation. The particles thus become heated and transfer that heat to the surrounding atmosphere, which reduces atmospheric convection, concomitantly reducing surface heat loss, and leading to global warming [47].

The upper portion of Figure 1 is a photograph showing geoengineering particulate-pollution trails that are typical of those now observed on a near-daily, near-global basis. The white color is the result of scattered sunlight [47]. There has been a concerted effort to deceive people into believing that these particulate-pollution trails are ice-crystal contrails formed from

the moisture is jet exhaust [49-51]. Moreover, two publishers and their editors were coerced into retracting peer-reviewed and published public health articles without due process being afforded the author [52]. Clearly, those who order the aerial particulate spraying are aware of the adverse health risks [53-56] and are intent on deceiving those who must breathe the contaminated air.

The lower portion of Figure 1 is a photograph of geoengineering particulate-pollution trails that show, not only the characteristic white trails, but black trails as well, presumably from jet-sprayed carbon black, which is an efficient absorber of radiation that scatters little [47]. The black trails are conclusive evidence that the aerial trails are not ice-crystal contrails which never form black trails. Recently, one of us (JMH) in departing Frankfurt, Germany observed numerous white trails below the cloud level, but observed black trails above the clouds, presumably so that this sprayed material was more out of public view.

**Figure 1.** Upper: White particulate-pollution trails above Soddy-Daisy, Tennessee, USA. Lower: Mixture of white and black particulate-pollution trails above Danby, Vermont, an impossible combination for alleged ice-crystal ‘contrails’.

The United Nations' Intergovernmental Panel on Climate Change (IPCC) promotes the idea that anthropogenic carbon dioxide is causing global warming while never acknowledging the fact that geoengineering, altering Earth's natural processes, has been ongoing for years and is, in fact, contributing to global warming.

The Director-General of the United Nations' World Health Organization recently noted [57], that the simple act of breathing is killing seven million people a year and injuring billions more. "No one, rich or poor, can escape air pollution", he acknowledged, "Despite this epidemic of needless, preventable deaths and disability, a smog of complacency pervades the planet." Yet, the Director-General does not mention the deliberate, geoengineering jet-sprayed particulate-pollution that evidence indicates is consistent with CFA [28-30].

The primary elements in CFA are oxides of silicon (Si), aluminum (Al), iron (Fe) and calcium (Ca), with lesser amounts of magnesium (Mg), sulfur (S), sodium (Na), chlorine (Cl), and potassium (K). The many trace elements in CFA include arsenic (As), barium (Ba), beryllium (Be), cadmium (Cd), chromium (Cr), copper (Cu), lead (Pb), manganese (Mn), mercury (Hg), nickel (Ni), phosphorus (P), selenium (Se), strontium (Sr), thallium (Tl), thorium (Th), titanium (Ti), uranium (U), vanadium (V), and zinc (Zn) [40]. Burning coal concentrates these trace elements in CFA, which are typically of higher relative proportion than those found in Earth's crust [58].

Coal fly ash forms in an anhydrous chemical environment in the hot gases above the combustion burner which renders its chemical behavior quite different than similar-sized particles from Earth's crust. At least 39 elements can be partially extracted from CFA by exposure to water [40]. Elements that can be extracted include aluminum in a chemically mobile form which is toxic to plants and other biota [59,60]. A host of toxic elements can be extracted by bodily fluids from inhaled CFA [28].

Coal fly ash contains substantial quantities of iron oxides, hematite and magnetite, as well as spherical carbonaceous particles [61]. Iron in the form of maghemite ($\gamma$-$Fe_2O_3$) crystals often forms on the surface of microspheres ("ferrospheres") in CFA [62]. Iron speciation by Mossbauer spectroscopy indicates that ferric iron in an aluminosilicate glass phase is the source of bioavailable iron in CFA and that this iron species is associated with combustion particles, but not with crustal dust derived from soil minerals [63].

Detailed analysis of the ultrafine fraction of coal fly ash leads to the following conclusions [64-67]:

- Greater concentrations of hazardous and volatile elements are contained in the ultrafine fraction compared to the coarse fraction, with enrichments over 50 times observed for some elements;
- Iron oxides present in the ultrafine fraction are highly reactive and likely produce oxidative stress in tissue; and,
- Soot (originating from tars or other carbonaceous components) comprises a significant fraction of ultrafine particulate matter and it also contributes to toxicity. Increased toxicity of this carbon fraction is consistent with theories in which carbon mediates transition metal (e.g. iron) complexes [64].

## 4. COAL FLY ASH ADVERSE HEALTH MPLICATIONS

Fine particulate matter derived from combustion of fossil fuels delivers the most potent and harmful elements of air pollution. The main vehicle of these adverse effects is most likely the combustion-derived UFP's and nanoparticles that incorporate reactive organic and transitional metal components [68]. Ambient particles in air pollution contain numerous soluble transitional metals capable of producing redox cycling and oxidative stress. Residual oil fly ash (ROFA) containing water-soluble iron and other transition metals lead to acute inflammatory effects in animals. The bioavailable transition metal, rather than the particulate mass, may be the primary determinate of inflammation [69].

A recent review of health outcomes in communities impacted by coal fly ash include higher rates of all-cause mortality, premature death, chronic respiratory disease, lung cancer, and cardiovascular disease [42,64,70]. Sub-micron, ultrafine-type spherical pollution particles were shown to cause delayed cardiovascular effects compared to larger (coarse) particles [71]. Our work suggests that aerosolized coal fly ash, including its use in geoengineering operations, is a significant risk factor for chronic lung disease [56], lung cancer [54], and neurodegenerative disease [55]. We have shown that the size and morphology of the damaging magnetite pollution particles found in brain tissue of persons with dementia [72] is most consistent with an origin in coal fly ash [55].

There is strong evidence from both epidemiologic and experimental studies that that PM-associated injuries to the cardiovascular system of both healthy and compromised hosts are mediated by soluble metals [73]. Coal fly ash is a fine-mode aerosol that is rich in metal contaminants [73]. The determination of metal levels in ambient air pollution is important because absorption rates into the blood for many metals are higher by inhalation (up to 50-60%) than those by ingestion (5-10%) [74].

Coal combustion produces detectable levels of As, Fe, Co, Mn, and Antimony (Sb) in ambient air [75]. Metals in CFA that have been linked to cardiovascular disease include iron, arsenic, lead, cadmium, and mercury [76]. Magnetic measurements of PM2.5 can be an efficient process for the assessment of trace metal levels in air pollution. The dominant magnetic minerals found in PM2.5 are magnetite and hematite which are comprised of both angular and spherical particles of anthropogenic origin [77].

The atmospheric burden of anthropogenic combustion iron is now estimated to be many (eight or more) times greater than previous measurements based on more accurate measurements of magnetite on a global level [23]. Experimental studies [78] show that:

- PM particles contain iron that can be mobilized from the particle in vitro and inside human lung epithelial cells;
- Mobilized iron catalyzes reactive oxygen species (ROS); and,
- Iron in CFA induces pro-inflammatory markers like cytokine interleukin-8 (IL-8).

A mechanism of biological effect common to many ambient air pollution particles is a disruption of iron homeostasis in cells and tissues [79]. Following endocytosis, functional groups at the surface of retained particles complex available-iron to the cell and can result in a functional deficiency of requisite iron inside the cell. Superoxide production by the cell exposed to the particle facilitates import of iron into the cell with the objective of reversing the metal deficiency. Failure to resolve the functional iron deficiency inside the cell following particle exposure activates kinases and transcription factors resulting in further inflammation and tissue damage [80].

## 5. IRON BALANCE/HOMEOSTASIS

Iron is an essential element for nearly all living organisms. Iron's participation in heme and iron-sulfur cluster proteins is vital to oxygen transport, DNA synthesis, metabolism, and both cellular respiration and signaling. However, iron's redox potential and participation in Fenton chemistry can trigger oxidative stress, lipid peroxidation, DNA damage, and ultimately cell viability and apoptosis (programmed death) [81].

Harmful effects of iron are normally prevented by tightly controlled regulatory mechanisms which maintain systemic and cellular iron homeostasis [82]. Systemic iron homeostasis is controlled by the hepcidin-ferroportin axis. Hepcidin is the primary regulator of iron absorption and tissue distribution [82]. Hepcidin and its cellular iron exporter ferroportin control the major fluxes of iron into serum via intestinal iron absorption, the delivery of recycled iron from macrophages, and release of stored iron from hepatocytes. Hepcidin and ferroportin play important roles in host defense and immunity, for example, hepcidin synthesis is stimulated by inflammation [83]. Hepcidin controls iron entry into circulation from absorptive enterocytes, iron recycling macrophages, and hepatocytes by binding to ferroportin and inducing its degradation. Stimulating hepcidin inhibits iron absorption and release, while lowering hepcidin promotes iron availability [84].

Cellular iron homeostasis is regulated by factors including iron regulatory proteins (IRP1 and IRP2), which are RNA - binding proteins which control intracellular iron metabolism, transferrin receptor 1 (TR1), divalent metal transporter, ferritin subunits (which regulate iron storage), and ferroportin, which exports iron via the plasma membrane. These mechanisms keep the cytoplasmic labile iron pool (LIP) strictly controlled [85]. An imbalance of these homeostatic factors can lead to an accumulation of intracellular iron, increased reactive oxygen species, and eventually cell death [86].

## 6. IRON AND THE HEART/CARDIOVASCULAR SYSTEM

The iron-heart hypothesis was formulated by Dr. Jerome L Sullivan, who proposed that the lower incidence of cardiovascular disease in premenopausal women compared to men was because they had lower iron stores due to regular blood loss [87]. The iron hypothesis has been controversial since this time, with many studies supporting the role of iron in cardiovascular disease, while others have not shown a definite relationship [88]. Ferritin (the main iron storage protein in the body) levels are not consistently elevated in atherosclerotic vascular disease. However, iron in ferritin is bound, and only free or loosely bound iron participates in redox reactions. In other words, total body iron is not necessarily related to the level of biologically active iron. However, catalytic (reactive) iron is associated with cardiovascular disease [89]. Most intracellular iron is found in ferritin, while the redox active iron forms the labile iron pool. Pro-inflammatory and anti-inflammatory macrophages inside arterial plaque differ as to their amount of intracellular iron and their labile iron pool [90].

Sullivan himself pointed out that cardiovascular disease associated with inflammation may be caused by elevated hepcidin levels that cause retention of iron within plaque macrophages. Although there is defective retention of iron within

arterial macrophages from down-regulated hepcidin in hemochromatosis, as with iron deficiency anemia this macrophage iron is mobilized elsewhere in the body to support erythropoiesis. These observations may explain why atherosclerosis is not increased in homozygous hemochromatosis, an iron-overload disease [91]. It is noteworthy that patients with cardiovascular disease have significantly higher concentrations of iron in their intracellular monocytes/macrophage labile iron pool than do healthy controls [92].

Macrophages are mononuclear phagocytic cells found throughout the body. They participate in immunity and play a key role in senescent red blood cell recycling, free heme detoxification, and provision of iron for hemoglobin synthesis [93]. High turnover of iron is necessary for continuous erythropoiesis and tissue integrity but it challenges the macrophage's ability to maintain cellular iron homeostasis [92,93].

Macrophages play a central role in the progression of atherosclerosis, and different subtypes of these cells are found in atherosclerotic plaques [94]. Lipid ingestion is the primary stimulus for M1 macrophage differentiation in plaques which induces inflammatory cytokines and production of foam cells. M2 macrophages are thought to produce anti-inflammatory agents that counter balance this inflammation [94]. The loading of macrophages with iron leads to oxidation of low density lipoprotein (LDL) - (the "bad cholesterol") and increased cellular cholesterol accumulation and scavenger receptor expression [95]. There is a growing body of evidence that labile (reactive) iron is involved in cardiovascular disease by direct oxidative damage to cellular components and/or oxidation of extracellular biomolecules including LDL [96].

Elevated hepcidin increases iron in macrophages and inhibits gastrointestinal uptake. Hepcidin may promote plaque destabilization by preventing iron mobilization from macrophages within atherosclerotic lesions; the absence of this mobilization may result in increased cellular iron loads, lipid peroxidation, and progression to foam cells. Increases in iron concentration are found in atherosclerotic plaques in comparison to healthy arterial tissue [97]. Iron within atherosclerotic plaque is implicated as a catalyst of oxidative stress capable of causing plaque progression and rupture. Iron in asymptomatic plaques is found in macrophages as ferritin or hemosiderin. Iron in symptomatic plaque is found within thrombus. The abundance of iron in symptomatic plaques is associated with the source patient's LDL level [98].

## 7. IRON AND RISK FACTORS FOR CARDIOVASCULAR DISEASE

Altered iron homeostasis is a common factor that links all the major risk factors for atherosclerotic vascular disease; i.e. hypertension, smoking, lipid abnormalities, diabetes, obesity, and heavy alcohol intake. PM2.5 air pollution is known to be related to the development of cardio-metabolic conditions including atherosclerotic heart disease, hypertension, and diabetes [99]. Epidemiological evidence provides strong evidence supporting the association of iron and an increased risk of diabetes [100] and heart disease [100]. Insulin-resistance associated hepatic iron overload syndrome (IRHIO) is characterized by high serum ferritin and the type of insulin resistance that characterizes adult onset diabetes [101]. Increased serum ferritin predicts the development of hypertension in middle-aged men [102]. Arterial stiffness is associated with increased ferritin and deposition of iron in vessel walls [103].

Men with high body iron stores have a two to three times risk of myocardial infarction (heart attack) compared to men with low body iron [104]. Iron in excess increases lipid peroxidation that modifies the fatty acid profile of cell membranes and leads to damage of organelles and mitochondrial dysfunction [105]. Iron-mediated alterations in lipid metabolic pathways are involved in the initiation and progression of cardiovascular disease in complex ways that are still poorly understood [106]. Increased dietary availability of iron through supplementation and fortification of processed foods is a factor linked to the global epidemic of obesity [107].

Smoking cigarettes is a major risk factor for cardiovascular disease. The amount of iron in a single cigarette is about 0.4 mg, and nearly all of this amount can be transferred into the body of the smoker [108]. Smokers have a several fold (4-9X) increase in the iron content of their alveolar macrophages compared to nonsmokers [4]. Serum iron and ferritin levels are increased in smokers, supporting systemic accumulation of this metal after cigarette smoke exposure. Cigarette smoke particles alter iron homeostasis, both in the lung and systemically [109]. Magnetic analysis shows cigarette ashes contain a significant amount of very fine, nanometer-sized magnetic particles, as well as larger, magnetically stable grains. Magnetization data indicates that cigarette ashes contain about 0.1 wt% quantity of magnetite, depending on the brand [110]. Iron oxide nanoparticles are even generated from the heating coils of electronic cigarettes [111].

Both heavy alcohol and smoking cigarettes can worsen iron accumulation in the body [112]. Heavy alcohol use is associated with increased cardiovascular risk, and excessive alcohol is the third leading cause of premature death (after smoking and obesity) in the U.S. [113]. Iron regulation is disturbed in patients with chronic liver disease, including alcoholic liver disease. Liver disease decreases the production of hepcidin, which leads to iron deposition in the liver and higher levels of non-transferrin bound (reactive) iron in the bloodstream [114]. Acute infections are known frequent

antecedents to cardiovascular events like myocardial infarction [115]. Iron is an essential growth factor for most pathogenic organisms. Stored iron can be mobilized by microorganisms like Chlamydia pneumoniae which can acquire the ability to colonize coronary arteries and trigger heart attacks [116].

Iron metabolism is a balancing act and complex biological systems have developed to maintain this balance, or homeostasis [117]. Once iron is absorbed by the gut enterocyte, it is bound to iron transport protein (transferrin) and iron storage protein (ferritin) that control iron availability to cells and tissues throughout the body. Disruption of iron balance in heart muscle has dramatic effects: iron excess leads to cardiomyopathy and iron deficiency exacerbates heart failure [117].

Normal heart function requires the balance of iron supply for oxidative phosphorylation and redox signaling with tight control of intracellular iron to below levels that generate reactive oxygen species. Iron deficiency can be absolute (e.g. due to blood loss), or functional, where iron is kept out of the circulation due to the effect of inflammation on hepcidin levels [118]. Recent evidence suggests that heart cell (cardiomyocyte) iron levels are balanced by a separate cardiac hepcidin-ferroportin axis operating independently of the systemic hepcidin-ferroportin axis [119].

The high rate of cardiomyopathy in patients with hemosiderosis and transfusional iron overload indicates that iron build-up in the heart plays a major role in the development of heart failure [120]. Abnormal sites of deposition of iron in heart tissue and cardiomyocytes contribute to this pathology [120]. Disruption of links in these systemic or cellular iron networks can lead to increased cellular iron, either from circulatory sources or mismatches in cellular iron distribution. Heart cells must cope with fluctuations of labile iron by balancing iron intake for utilization and safely storing “surplus” iron within the cell [121,122]. Ferroptosis is a recently described form of cell death mediated by iron and it is characterized by an accumulation of lipid peroxidation products within the cell. Ferroptosis has been shown to be involved in cell death associated with neurodegenerative disease (dementia and Parkinson’s disease) and cardiomyocyte death following reperfusion injury [123].

## 8. MAGNETITE – IRON OXIDE NANOPARTICLES – AND THE HEART

Magnetite is an iron oxide ($Fe_3O_4$) mineral occurring naturally in Earth’s surface rocks and sand [124]. Biogenic magnetite crystals occur in the bodies of a wide range of organisms including man. Magnetite likely has several vital life functions, for example, the detection of magnetic fields [125]. Results from analysis of human tissue show the presence of ferromagnetic, fine-grained, magnetically interacting particles in tissue including the heart, spleen, and liver [126].

Magnetite biomineralization in the human brain was first described in the 1990’s [127]. Brain tissue contains biogenic magnetite between 5 and 100 million single-domain crystals per gram. These biogenic ferrimagnetic crystals are known to be exquisitely sensitive to external electromagnetic fields via a resonance/vibrational (vs thermal) mechanism [127].

Human biogenic magnetite nanoparticles tend to be single domain size, high chemical and crystalline purity, arranged in chains, and associated with lipid coatings near the cell membrane [128]. In 2016 Maher et al. [72] showed two types of magnetite in the brains of persons with dementia: euhedral biogenic particles and spherical exogenous particles most likely arising from air pollution. Magnetite in tissue paradoxically serves essential life functions while in excess or from external sources causes harmful effects and disease [129].

An excess of labile iron related to the inability of ferritin to retain iron in its core may represent an additional iron supply for magnetic nanoparticle biomineralization [129]. Iron oxide nanoparticles, but not zerovalent iron nanoparticles or coated iron oxide particles can cross plasma membranes. Electrophysiology shows a transient, small, but detectable increase of membrane conductance associated with this nanoparticle crossing of the plasma membrane [130]. It was recently discovered that nanoparticles added to stem cells are first degraded, but then new magnetic nanoparticles are synthesized in situ from the released iron. This “re-magnetization” process involves ferritin protein, and it is apparently a mechanism of detoxification in a situation of iron excess [131].

Magnetite is a major anthropogenic component in ambient PM2.5 and it is derived mainly from industrial sources [132]. Iron oxide nanoparticles in cultured human umbilical endothelial cells produce oxidative stress and apoptosis, suggesting that that mechanism might play a key role in downstream cardiovascular effects, e.g. atherosclerotic disease, hypertension, and myocardial infarction [133].

A recent study showed that Mexico City residents have up to 22 billion magnetic nanoparticles per gram of ventricular tissue, and that these particles were directly responsible for early and significant cardiac damage [134]. The finding of countless combustion-type magnetic nanoparticles in the hearts of persons (including those with dementia) from highly

polluted areas is definitive evidence of the link between the iron fraction of air pollution and heart disease [134]. Magnetite and maghemite nanoparticles showing the typical round or spherical morphology of a combustion source were present in numbers 2-10 times that of controls and they were found inside mitochondria in ventricular cardiomyocytes, in endoplasmic reticulum, intercalcated discs, and both endothelial and mast cells [134]. The health consequences of these pollution particles likely reflects the combination of surface charge, ferrimagnetism, redox activity and the potential for disruption of the heart's electrical activity [15,134].

This indisputable evidence of cardiac damage by exogenous magnetic nanoparticles [134] is supported by experimental work showing uptake of ultrafine iron oxide particles into atherosclerotic lesions in animals. USPIO's (ultra-small superparamagnetic particles of iron oxide) are phagocytosed by macrophages in atherosclerotic plaques of the aortic wall of hyperlipidemic rabbits in quantities sufficient to detect by MRI [135]. The IV administration of USPIO's in mice causes thrombosis, cardiac oxidative stress, and DNA damage [136]. Iron oxide nanoparticles that are taken up by macrophages in atherosclerotic plaque can be detected by differential phase optical coherence tomography (DP-OCT).

## 9. SOURCES OF MAGNETIC NANOPARTICLES

The primary source of magnetic pollution particles in the human brain and heart is assumed to be roadside air pollution arising from vehicle combustion and frictional brake-wear, and secondarily from industry and coal-fired power plants [72,134,137]. However, it is more likely that coal combustion products including coal fly ash are the primary source of these spherical magnetic particles, with a secondary contribution from vehicle (especially diesel) emissions. Detailed analyses of coal fly ash including its magnetic component confirm an abundance of spherical magnetite nanoparticles matching the exogenous magnetic particles found in human tissue [55,138].

Many metallic nanoparticles collected from roadside sites are rich in iron composed of various iron oxides (including magnetite) in the form of spheres or multifaceted polyhedra. The smallest particles (10 nm or less) often agglomerate into larger clusters [139]. Scanning electron microscope studies show that particles of roadside mineral dust and brake dust consist predominately of angular particles in irregular shapes [140]. Particles of car exhaust contain mostly carbonaceous material in agglomerated chains with metallic ash components from fuel and lubricant oil. These particles are irregular, with some spherical soot particles in the smallest fraction [141].

Diesel and biodiesel exhaust structures reveal soot agglomerates with some metal-containing fly ash particles from lube oil. Newer diesel engines actually emit more nanoparticles than old diesel engines, and they are considered more harmful to human health [142].The identification of magnetic particulates in roadside snow shows that angular-shaped particles are derived from vehicle emissions and that spherule-shaped particles occur mostly from emissions from industrial activities [143].

Diesel emissions do produce magnetite nanoparticles - spherical and often in aggregates [144,145]. What is unknown is whether such particles are indigenous products of diesel hydrocarbon combustion coupled with engine wear, or whether those particles originate from residues of catalysts used in fuel production [146], from the debris of catalytic converters [147] or from the consequence of fuel additives as various nanoparticles, including magnetite, are used as diesel fuel additives to improve combustion and reduce certain emissions [148].

There are several commonalities between coal fly ash and diesel/biodiesel type emissions and aerosols. Coal fly ash and diesel/biodiesel emissions contain ultrafine particles of both carbon and metals which can cause oxidative stress. The production of oxidants, either directly by air pollution particles or by host response to those particles, is central to their biological effect [149]. ). Co-exposure to carbon black and $Fe_2O_3$ particles causes a synergistic oxidative effect that is significantly greater than the additive effects of exposure to either particle type alone [150]

Magnetite ($Fe_3O_4$) is often used as a catalyst because it has a permanent magnetization and contains iron in both divalent and trivalent forms. Hematite and goethite have also been used as catalysts, often in composites with carbon, aluminum and zeolites [151]. Fly ash waste can function as a catalytic converter for reduction of HC and CO emissions [147]. Coal fly ash can be used as a catalyst in the production of biodiesel [146].

Studies show that iron oxide ($Fe_2O_3$) nanoparticles blended into biodiesel fuel can enhance engine performance, while reducing certain harmful emissions from compression ignition (CI) engines [152]. Nanofluid using magnetite is synthesized by reacting iron II ($FeCl_2$) and iron III ($FeCl_3$) in aqueous ammonia solution to form magnetite ($Fe_3O_4$) (Ferrofluid). One percent Ferrofluid mixed with biodiesel decreases HC, CO, and NO2 emissions in CI engines [148].

The effects of various metal oxide nanoadditives (e.g. $CeO_2$, $TiO_2$, MnO, $Al_2O_3$, and iron) on the performance and emission characteristics of CI have been reviewed and indicate that these nanoparticles promote complete combustion, increase engine efficiency, and lower smoke, CO, and other gaseous emissions. However, this same review reveals that "safety criteria for public concern during production and use are rarely attempted" [153]. Magnetite has been detected in the particulate matter collected from diesel engine exhaust using a total exhaust dilution tunnel [145].

It is now known that inhaled diesel emissions generated with cerium oxide ($CeO_2$) nanoparticle fuel additives induce adverse pulmonary and cardiovascular effects [154].

Human exposures to nano-size (< 100 nm) particulates have increased dramatically over the past century. Unlike fine or coarse PM particles, there is no effective monitoring or regulation of nanoparticles. The main anthropogenic sources of these particles are from vehicles and industrial emissions, including coal combustion products [155]. Besides carbonaceous matter, both CFA and diesel emissions contain nanosized metal oxide particles as free particles and also as attached to other particles including soot [156].

Particles originating from combustion processes can be divided into three categories: 1. Primary nanoparticles formed in high temperature, 2. Delayed primary particles formed as gaseous compounds nucleate during the cooling and dilution phase and 3. Secondary nanoparticles formed from gaseous precursors via atmospheric photochemistry [141].

Nanoparticles in the roadside environment are usually a complex mixture of particles from different sources that are affected by atmospheric processing [141]. In the atmosphere, most nucleation mode particles consist of sulfates, nitrates and organic compounds.

Delayed primary particles are often created by sulfuric acid which is in gaseous phase under tailpipe conditions but will condense or nucleate immediately when the exhaust is released and cooled [157]. Acids formed from anthropogenic pollutants dissolve iron in airborne CFA particles. Spherical iron-rich fly ash particles internally mixed with sulfate are the most important source of bioavailable iron to ocean systems [158]. Atmospheric deposition of bioavailable iron has drastically shifted the global plankton community in the direction of harmful algae blooms [159]. The soluble iron fraction derived from vehicle particulate matter is strongly associated with the production of reactive oxygen species [160].

Secondary nanoparticles can form at rates of tens of thousands per second with an atmospheric lifetime up to several days [161]. Nanoparticles from anthropogenic sources usually vastly outnumber the larger accumulation mode particles and they are far more dangerous than micron-sized particles [162].

## 10. HUMAN EFFECTS OF IRON NANOPARTICLES

Regardless of source, the presence of iron-rich spherical pollution particles in the hearts of young (average age 24) urbanites with associated cognitive deficits from highly polluted areas indicates that these particles are important markers for cardiovascular disease [134].

Various nanoparticles have negative effects of male germ cells, the female reproductive system, and the fetus [163]. The presence of metals including cadmium, chromium, cobalt, lead, mercury, and nickel in amniotic fluid has negative impacts on cognitive skills and the overall health status of children at age 3 [164].

Early exposure to aerosol particulate matter may have adverse consequences later in life. Animal studies show that early life exposure to PM2.5 air pollution induces adult cardiac disease [165].

In utero exposure in mice to ultrafine particles consisting of spherical-type carbon particles and metals (e.g. Fe, Cr, Ni, and Ti) causes placental inflammation, oxidative stress, and altered blood pressure in the offspring [166]. It was recently shown that combustion-derived particulate matter accumulates on the fetal side of the human placenta. The placental load of black carbon is positively associated with the mothers' residence and level of exposure to PM2.5 during pregnancy [167].

Iron oxide particles are commonly used for biomedical, industrial, and commercial applications due to their unique properties and potential biocompatibility. However, higher levels of exposure to iron oxides in mice leads to charge-dependent fetal loss and changes in reproductive organs of the offspring [168].

The balance of essential micronutrients like iron is critical during rapid growth and differentiation in the fetal and neonatal stages. Both iron deficiency and iron excess can have severe effects on normal human development that may persist into

adulthood [169]. The fetal origins hypothesis [170] states that fetal undernutrition or malnutrition in middle to late gestation leads to disproportionate fetal growth and predisposes to future coronary artery disease.

The developing fetus and young child are more biologically and psychologically vulnerable than adults to the effects of toxic air pollutants. Toxic pollutants from combustion sources can affect cardiovascular health and functioning over the course of a lifetime after initial "seeding" early in life [171]. The unequivocal finding of exogenous iron oxide pollution nanoparticles in heart tissue of children as young as 3 strongly suggests that combustion-derived magnetic iron particles could be the seed for future cardiac dysfunction. Given the extraordinary number of pollution particles in the hearts of healthy "controls," it is likely that the great majority of mankind has already been contaminated with iron-rich magnetic nanoparticles [134].

## 11. CONCLUSION

Iron is an essential metal for nearly all living organisms. However, excess or misplaced iron in the body causes organ dysfunction by oxidative stress and reactive oxygen species. Complex systemic and intracellular regulatory mechanisms are involved for iron metabolism, storage, and transfer. As there is very limited excretion of iron, iron can be loaded by ingestion, transfusions, disease states, and exogenous sources.

Free iron, non-transferrin bound iron, and labile iron in the circulatory system and within cells are responsible for iron's toxicity. Scavenger macrophage cells are found throughout the body, including the heart, and they play a key role in iron balance, recycling, free heme detoxification, and provision of iron for hemoglobin synthesis. Disruption of iron homeostasis can damage both the heart and the entire cardiovascular system. Disruption of iron homeostasis is associated with the traditional risk factors for atherosclerotic heart disease.

The finding of countless combustion-derived, spherical, magnetic air-pollution nanoparticles in human hearts is irrefutable evidence of gross contamination of the body by the iron oxide/magnetite fraction of air pollution. These particles match precisely the iron/magnetite nanoparticles in coal fly ash and certain combustion engine/diesel fumes. While traffic-related air pollution (TRAP) is undoubtedly a major contributor to these magnetic nanoparticles, it is presumably eclipsed by coal fly ash from industrial sources, coal-fired power plants, and especially by the aerosolized coal fly ash usage in ongoing undisclosed tropospheric aerosol geoengineering.

Exposure to iron oxide nanoparticles in humans now occurs from "womb to tomb," is cumulative over time, and is related not only to cardiovascular disease later in life, but a host of other non-communicable diseases including neurodegeneration and dementia. Chronic inhalation of iron oxide nanoparticles should now be considered one of the most important modifiable risk factors for cardiovascular disease. The best "treatment" is prevention; i.e. provision of clean air containing minimal PM2.5 air pollution and nanoparticles.

The finding of countless magnetic air-pollution particles from combustion sources in human hearts underscores the urgent need to reduce particulate air-pollution on a global basis. There must be international study, quantification, and regulation of ultrafine/nanoparticle air pollution. The current pandemic of non-communicable diseases, including cardiovascular disease, and rapid global warming can both be alleviated by reducing particulate pollution. Complacency over the dangers of air pollution and the deadly "code of silence" regarding the subject of ongoing, "covert," climate engineering must be broken if the world is to have a realistic chance of combatting these public health emergencies. Immediate steps that must be taken include:

- Halt the "hidden in plain sight" tropospheric aerosol geoengineering operations,
- Improve the control of small particle emissions from coal-fired industrial and power plant sources, especially in developing countries where ambient air pollution is greatest,
- Reduce ultrafine/nanoparticle vehicle emissions,
- Strive to eliminate harmful nanoparticulate-type fuel additives.

These actions collectively constitute a moral imperative if humanity, even our children, are to have a viable future. The behind-the-scenes push for weather control, "climate intervention," and geoengineering schemes has come to threaten not only humans but the entire web of life on Earth.

**DISCLAIMER:**

Authors have declared that no competing interests exist. The products used for this research are commonly and predominantly use products in our area of research and country. There is absolutely no conflict of interest between the authors and producers of the products because we do not intend to use these products as an avenue for any litigation but for the advancement of knowledge. Also, the research was not funded by the producing company rather it was funded by personal efforts of the authors.

## CONSENT

Not Applicable.

## ETHICAL APPROVAL

No human or animal subjects were involved.

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