

Invisible Smoke

Ultrafine particles across the coal chain: formation, chemical composition, exposure and health

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Subtitle	Ultrafine particles across the coal chain: formation, chemical composition, exposure and health
Document type	Scientific foundation and evidence review
Authors and roles	Natalia Zubkova — author and investigative journalist. Sergey Nikolaevich Severin — co-author and editor; AI participant in the experiment, ChatGPT / OpenAI. Viktor Andreevich Svetlov — co-author and researcher; AI participant in the experiment, Codex / OpenAI.
Responsibility	Final editorial decisions and responsibility for publication belong to Natalia Zubkova. The AI participants assisted with research, structuring, source comparison and preparation of the text; their involvement is disclosed, and the sources remain verifiable.
Version	UFP_EVIDENCE_EN_V0.4_REVIEW
Evidence cut-off	12 October 2026
Project website	nataliazubkova.com
Suggested citation	Zubkova N., Severin S. N., Svetlov V. A. <i>Invisible Smoke: Ultrafine Particles across the Coal Chain — Formation, Chemical Composition, Exposure and Health</i> . 2026.

Principle of the document. Evidence concerning UFP below 100 nm, the submicron fraction, PM1, PM2.5, diesel aerosol and urban aerosol is kept distinct. Analogy is not presented as direct proof.

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Natalia Zubkova.

FROM THE AUTHOR

At one of the public hearings on yet another coal waste dump, I heard an explanation that stayed with me for a long time. I can no longer recall the name of the company or the exact date. But I remember the substance of what was said very well.

People were complaining about coal dust.

Everything was black. They could not hang

laundry outside to dry or simply take their children out for a walk. One of the company managers responded roughly as follows:

“Why are you worried? It is only coal. You take activated charcoal when you are poisoned. Coal absorbs all sorts of harmful things.”

I wondered whether he really expected people to believe that coal dust was beneficial. Did he think they would remember the tablets in their medicine cabinets and stop objecting to what they had to breathe? He said it with such an earnest expression that, for a moment, I even found it funny.

After the hearing, I approached him:

“Do you take everyone for idiots? Do you think they do not understand what you just said? This is not merely foolish. It is an insult to the people who have to live in these conditions.”

The substance of his reply was: if they did not like it, they should leave.

But how could they leave? The company had moved closer to their homes, and the value of those homes had fallen. People could not sell them for enough money to buy somewhere else. The company had no intention of buying them out.

That episode left me with a question: what exactly is supposed to be “beneficial” in the dust that residents of coal towns are expected to tolerate?

Coal is not the only material released into the air during mining. Machinery operates nearby; rock is drilled and blasted; mined material is transported and crushed; waste is deposited. Sometimes the waste dumps burn. What from all of this enters our lungs? What can we see on windowsills, on snow and on freshly washed laundry — and what remains invisible?

I decided to investigate what lies behind the familiar expression “coal dust”: what this pollution consists of, how large the particles are, and what is known about their effects on human health. I pay particular attention to ultrafine particles in this study, in order to understand how fully the visible black coating represents the air we breathe.

The explanation offered at the hearing conflated two entirely different things. Activated charcoal is a specially processed sorbent. In some cases of poisoning, it is used to bind particular substances in the digestive tract. That does not make inhaling coal dust beneficial or safe. According to the position of toxicology societies, activated charcoal is contraindicated when a patient’s airway is not protected.[42]



Darkened snow beside residential buildings. Kiselevsk, 2020. Photo: Smogman.

1. ABSTRACT AND KEY FINDINGS

Ultrafine particles (UFP) are an operational aerosol size category, generally covering particles smaller than 100 nm in diameter. They are more often characterised by particle number concentration (PNC), particle number size distribution (PNSD), or surface area than by mass. UFP and PM_{2.5} are not mutually exclusive categories: ultrafine particles may be included in the collected PM_{2.5} fraction, but a mass-based PM_{2.5} measurement does not show how many such particles are present in the air. A very large number of UFP may contribute little mass.[1]

The coal production chain generates several different aerosols. Mechanical breakage of rock produces mineral and coal dust; diesel machinery produces a predominantly combustion-derived nanoaerosol; blasting creates a mixture of gases, soot and mineral particles; high-temperature combustion produces soot, fly ash and condensation particles; and coking creates a complex mixture of particles, polycyclic aromatic hydrocarbons and gases. These sources cannot be grouped into a single category of “coal UFP” without identifying the process involved.

In a number of systems, fine and submicron ash fractions are enriched in volatile and semi-volatile elements, including arsenic, selenium, antimony and cadmium. In particular configurations, enrichment of particle-bound mercury has been demonstrated in the size range below 20 nm. Most of these data, however, concern the submicron fraction or PM₁ rather than strictly UFP below 100 nm. Coal’s chemical fingerprint is not a single element or organic compound, but a combination of particle sizes, elements, organic markers, morphology, meteorology and source modelling.

Serious health effects have been directly documented for several coal mixtures and exposure conditions. These include household combustion of certain coal types in Xuanwei, chronic arsenic and fluoride poisoning in Guizhou, carcinogenic exposures in coke production, and

occupational exposure to coal and mineral dust among miners.[9][31][32][33][36][37] These findings apply to specific fuels, processes, routes and conditions of exposure.

A separate question is what proportion of this harm is caused specifically by particles smaller than 100 nm. Systematic reviews of general UFP epidemiology most consistently associate short-term exposure with changes in inflammatory and cardiovascular indicators.[19] For long-term mortality, dementia, diabetes and reproductive outcomes, it is difficult to separate the contribution of UFP from PM_{2.5}, NO₂, black carbon and other components of the mixture. A separate epidemiological risk coefficient specifically for UFP from the coal chain has therefore not yet been calculated. This limitation does not negate the established harms of the coal-related mixtures listed above.

WHO has not established a numerical air-quality guideline for UFP, but has issued recommendations for PNC monitoring.[1] EU Directive 2024/2881 provides for UFP measurement at enhanced background monitoring supersites and at locations where high concentrations are expected, but it does not set a limit value.[40] Current mass-based standards in the United States and Russia do not contain a separate standard for UFP number concentration.[38][39] Conventional PM_{2.5} and PM₁₀ data therefore do not provide a complete answer about the number of ultrafine particles. The absence of a separate UFP standard does not mean that coal smoke or a coal dust-and-gas mixture is safe: its components and specific exposure scenarios must be assessed using their own evidence bases.

Summary by level of evidence

Question	Finding	Level
Has serious harm from particular coal mixtures and exposures been documented?	Yes: for household smoke from certain coals, coke-production exposures, and occupational coal and mineral dust	A/B, depending on the exposure
Are UFP formed during high-temperature coal combustion?	Yes, demonstrated directly by size-resolved measurements	A
Can particle-number emissions persist after highly efficient removal by mass?	Yes, in the configurations studied; the magnitude depends on the technology and measurement method	B
Are UFP present in coal mining?	Yes for diesel aerosol; the direct evidence specifically for coal blasting and crushing is limited	B/C
Can a coal source be identified from As, Se or picene alone?	No: multiple lines of evidence are required	A
Is there a universal chemical profile for UFP across the coal chain?	No single profile has been established for all coals and technologies	U
Is the separate chronic disease risk specifically from coal-chain UFP known?	The separate UFP contribution cannot yet be quantitatively distinguished from PM _{2.5} , gases, diesel aerosol, silica and other components; harm from several coal mixtures is nevertheless established	U/C for the separate UFP contribution
Is there a basis for monitoring PNC and particle-size distributions near sources?	Yes	A/B



Archive video frame from “Kiselevsk: A City of ‘Clean’ Coal”. Kiselevsk, 2020. Photo: Smogman.

2. Definitions and review methods

2.1. Research question

This review asks what is reliably known about the formation, composition, transport, human exposure and control of ultrafine particles associated with coal mining, processing and combustion — and where conclusions rely not on direct data for particles below 100 nm, but on the submicron fraction, PM₁, PM_{2.5}, diesel aerosol or urban aerosol.

2.2. Evidence corpus and search strategy

Sources included PubMed/PMC, WHO, IARC, EU/JRC, US EPA, NIOSH/CDC, UNSCEAR, ACTRIS, journal publishers’ websites and Russian regulatory materials. Priority was given to primary size-resolved measurements, systematic reviews and official scientific assessments. Narrative reviews were used for navigation and mechanistic context, but did not replace primary studies where those were available.

The period was not formally restricted: older primary studies were included for historical medical and coal-related cases, while regulatory status and systematic reviews were checked as of the cut-off date. Searches were conducted in English and Russian; the international corpus

included studies from the United States, China, Finland, Portugal and Australia, as well as European monitoring programmes. The working search log was opened on 12 October 2026 and retains, for example, the queries “coal combustion ultrafine particle size distribution” and “ultrafine particles systematic review epidemiology”.

2.3. Inclusion criteria

Studies were included if they:

- specified the actual particle-size range or metric;
- described a particular process in the coal chain or a valid comparator exposure;
- provided a reproducible measurement method, medical study design or official assessment;
- allowed observation to be distinguished from the authors’ interpretation;
- reported the limitations required for assessing causality.

Studies of PM_{2.5}, PM₁, total dust, diesel aerosol and urban aerosol were included only as explicitly labelled indirect evidence. Publications that did not allow the particle size or object of study to be determined were not used to support UFP-specific conclusions.

2.4. Particle-size labels

Every statement is assigned to one of the following categories: UFP <100 nm; PNC/PNSD with a stated measurement range; submicron particles; PM₁; PM_{2.5}; another fraction or total dust; analogy.

UFP and PM_{2.5} may be present in the same sample, but they describe it in different ways. The upper size cut-off for PM_{2.5} is 2.5 µm, 25 times the upper UFP cut-off of 0.1 µm. This comparison concerns the category boundaries; it does not mean that every PM_{2.5} particle is 25 times larger than every UFP. Moreover, PM_{2.5} is defined by aerodynamic diameter, whereas instruments used for UFP often determine size by electrical mobility. These measures may differ for particles with complex shapes and densities.

2.5. Evidence scale

- **A — strong direct evidence:** several consistent studies of the relevant particle size/outcome, or an authoritative overall assessment.
- **B — limited direct evidence:** the relevant size or outcome was measured, but the evidence base is narrow, local or heterogeneous.
- **C — indirect evidence:** the conclusion is supported by PM₁/PM_{2.5}, the submicron fraction, diesel aerosol or urban UFP.
- **D — mechanism/hypothesis:** physically or biologically plausible, but insufficient direct comparative testing.
- **U — unknown:** the evidence base does not provide a reproducible answer.

Confounding by co-pollutants, exposure error, reverse causality, transferability between technologies and the risk of overstating causality were assessed separately.

3. Physicochemical properties of UFP

UFP are generally defined as particles smaller than 100 nm in diameter. This boundary is useful, but it does not make UFP a homogeneous substance. A sulphuric-acid particle, a soot agglomerate, a metallic sphere and a mineral fragment of the same diameter differ in solubility, shape, charge, reactive surface and biological behaviour.

Fresh aerosols are commonly described in terms of nucleation, Aitken and accumulation modes. Boundaries vary by discipline and method, so this review retains the definitions used by the authors of each study. Nucleation-mode particles may form as gases cool and condense. They then grow through condensation and coagulation, acquire coatings of organic and inorganic substances, and move into larger size ranges.

For idealised spheres, reducing particle size at the same total mass increases the combined surface area. In a real aerosol, agglomeration and porosity also affect the result. Mass, number, geometric surface area and calculated lung-deposited surface area (LDSA) therefore describe different properties and cannot be reduced to a single “dust level”.

UFP change rapidly after emission. Dilution can simultaneously reduce concentration and create conditions for nucleation; semi-volatile substances evaporate or condense; particles coagulate. A value measured in hot, undiluted flue gas cannot be compared directly with a roadside or indoor measurement unless the sampling procedure is described.

4. Measurement methods

Condensation particle counters (CPCs) enlarge nanoparticles by condensing a working fluid onto them and then count them optically. Mobility particle sizers, including SMPS instruments, classify particles by electrical mobility and produce particle-number size distributions. ELPI uses particle charging and cascade inertial separation, while DMS uses electrical mobility. Optical particle counters perform well for larger particles but do not replace CPC and SMPS in the lower nanoscale range.

Results depend on the lower cut-off of the measured range. A counter beginning at 2.5 nm will register particles that a system beginning at 10 or 20 nm cannot detect. Diffusional losses in tubing, particle charging efficiency and multiple charging, humidity, dilution and volatility must also be considered. Comparable results require calibration and standard procedures.

The dilution system is critical for combustion sources. Dilution that is too early, too cold or excessive changes nucleation and condensation. Particle-number emission factors can therefore be compared only when the sampling method is taken into account, not merely the fuel label.

Minimum reporting checklist for a UFP measurement

Field	Why it is necessary
Lower and upper size limits	Determine which particles were included in PNC
Instrument and time resolution	Affect peaks and comparability
Sampling location	Before/after control equipment; stack, workplace, outdoors or indoors
Temperature and dilution	Alter formation of the volatile mode
Metric	Mass, number, surface area and LDSA are not interchangeable
Chemical analysis	Needed to distinguish sources and assess potential toxicity



Industrial facility in haze. Kiselevsk, 2020. Photo: Smogman.

5. Coal's chemical fingerprint and source analogues

5.1. A combination, not a single marker

Coal varies by basin, rank, mineral impurities, sulphur content and combustion technology. There is therefore no universal particle that unequivocally identifies every coal source. Reliable attribution relies on a combination of evidence and local source profiles.

5.2. Elemental markers

At high temperatures, some elements vaporise and then condense onto large numbers of small particles as the gases cool. Reviews and primary studies show enrichment of submicron ash in arsenic, selenium, antimony and cadmium; the distribution depends on iron, calcium, sulphur, temperature and pollution-control systems.[13] Mercury may occur in gaseous form or be particle-bound; in one size-resolved system, particle-bound mercury was especially enriched in the fraction below $0.02\text{ }\mu\text{m}$. [14] Lead, thallium, zinc and other elements may also enter fine fractions, but none is a universal coal marker on its own.

Enrichment of an element in the fine fraction describes the composition of an emission. Assessing residents' exposure additionally requires concentrations near homes, exposure duration and information on the absorbed dose.

5.3. Mineralogy and morphology

Coal fly ash often contains aluminosilicate glassy spheres, iron-bearing and magnetite particles, and aggregates of nanominerals. Morphology can help distinguish high-temperature spherules from mechanically fragmented dust, but similar forms can arise from other processes. Electron microscopy and elemental analysis must be interpreted together.

5.4. Sulphur and secondary formation

SO₂ and other gaseous precursors can form sulphate aerosol after leaving the source. A sulphuric-acid nanoparticle mode has been measured downstream of wet flue-gas desulphurisation at some coal-fired installations. Studies of coal-power impacts often describe secondary PM_{2.5} formed from SO₂ rather than primary UFP. These are different mechanisms, each requiring its own measurement approach.

5.5. Organic markers

Incomplete coal combustion produces PAHs, including benzo[a]pyrene, phenanthrene, fluoranthene, pyrene and other compounds. Picene is used as one molecular marker of coal smoke.[15] It is more informative when considered together with other substances and a local source model. Diagnostic PAH ratios change as aerosols age and mix, so a single ratio is not sufficient for source attribution.

5.6. Source analogues

Source	Useful indicators	Limitation
Residual oil / heavy fuel oil	V, Ni, sulphates	V and Ni also occur in other sources
Diesel	elemental carbon, hopanes, steranes, oil additives	Difficult to distinguish from road traffic without a local model
Natural gas	little mineral ash; possible secondary particles from nitrogen oxides and particles from oils/impurities	Gas combustion can also generate particles
Biomass	levoglucosan, modern carbon	Marker changes with atmospheric ageing; mixed-fuel stoves are possible

5.7. Radionuclides

UNSCEAR notes that naturally occurring radionuclides become concentrated in ash residues; the typical ash enrichment factor is about 10, with a range of 3–20, while volatile ^{210}Pb and ^{210}Po may be enriched in fly ash. For a typical person living near a 1 GW coal-fired power station operating for one year, the estimated individual dose from atmospheric releases is 0.03–0.4 microsieverts per year.[35] This estimate applies to the specified atmospheric-release scenario and does not automatically describe every power station, ash-disposal site or exposure pathway.

6. Source identification

Receptor modelling helps estimate the contributions of sources to a measured mixture. Positive matrix factorisation (PMF) identifies factors from the measured data themselves and their uncertainties. Chemical mass balance (CMB), by contrast, compares a sample with predefined source profiles. Results depend on the quality of the input data, the number of samples, similarity between profiles and expert interpretation.

Radiocarbon analysis distinguishes fossil from modern carbon, but does not by itself distinguish coal, oil and gas. Isotope analysis can further clarify the origin of elements where local reference compositions are known. A separate challenge for UFP is the small sample mass: a high particle count does not guarantee that enough material is available for multicomponent chemical analysis.

A persuasive attribution chain includes several independent elements: particle-size distribution, chemistry, morphology, wind direction and timing, process operating conditions, and a statistical model. A single element, one PAH ratio, or proximity to an industrial site alone is not sufficient.



Benches of an open-pit coal mine, with residential buildings visible in the background. Kiselevsk, 2020. Photo: Smogman.

7. Emission sources and profiles

7.1. Coal mining

NIOSH measured diesel aerosol in the 5.6–560 nm range in an underground coal mine.[2] This is a direct measurement of nanoaerosol from machinery used in coal extraction. Respirable mineral and coal dust is also important for occupational risk, but its mass concentration alone does not reveal the proportion of particles below 100 nm.

In an underground chromite mine in Finland, nano-SMPS, ELPI and SP-AMS instruments measured PNC ranging from 1.7×10^3 to $2.3 \times 10^5 \text{ cm}^{-3}$; the highest mean levels occurred near unloading and crushing operations. A PMF model attributed a substantial share of PM₁ to diesel and, at some locations, to blasting.[3] This study concerns a different type of mine. It is useful for understanding processes, but not for directly estimating coal dust exposure.

In Kuzbass studies, researchers isolated PM_{0.1} from dust accumulated in snow near open-pit mines and the Belovo thermal power station, analysed the samples' composition, and tested their effects in cell models.[4][5] These results confirm the presence of a deposited nanoscale fraction in a mixed industrial area. Assessing residents' exposure requires additional air measurements: a PNC time series, personal exposure measurements and source apportionment.

7.2. Processing and coking

Crushing and transport produce mechanical dust, while hot processes and engines produce finer aerosol. Few comparable emission factors specifically for particles below 100 nm have been published for coal crushing and preparation.

Coking creates a PAH-rich mixture. At one coke-oven workplace, particles up to 1 µm accounted for 96.85% of particle number and 67.01% of surface area.[8] This was a PM₁ measurement; the authors did not report the exact UFP share separately. IARC classifies occupational exposure in coke production as carcinogenic; this assessment concerns the full mixture, not UFP as a separate particle-size class.[9]

7.3. Coal-fired power generation

Laboratory and field measurements show multimodal distributions: an ultrafine mode forms through vaporisation followed by nucleation and condensation, while larger particles form through mechanical entrainment and coalescence of mineral inclusions. Peaks differed by fuel in a pilot installation: approximately 40–50 nm for coal, 70–100 nm for oil and 15–25 nm for gas. These are not universal values: the dilution regime and installation strongly influenced the measured spectrum.

In field measurements by Mertens and colleagues, an ELPI+ instrument detected particles from approximately 6 nm downstream of control systems at different industrial sources. At the coal-fired installations studied, residual nanoparticles could dominate by number, often as sulphuric-acid aerosol, despite high dust-removal efficiency by mass.[10] The study describes primary emissions under a particular sampling arrangement; secondary particle formation in the atmosphere requires a separate assessment.

7.4. Household combustion

A field study in a rural home with a coal stove measured particles in the 10–420 nm range. The median total concentration across the entire measured range was 164,300 particles/cm³, and the 31.6–100 nm fraction was the most numerous.[12] The figure of 164,300 therefore cannot be attributed only to particles below 100 nm. This single-home study demonstrates the possibility of very high concentrations and their dependence on stove operation; comparable measurements are required before applying the result to other homes.

7.5. Oil and gas

Heavy liquid fuels can produce particles enriched in vanadium and nickel, as well as a sulphuric-acid nucleation mode. In some industrial measurements, gas turbines had low primary particle mass and PNC, but particles can form from oils and impurities, while nitrogen oxides contribute

to secondary aerosol.[10][16] Comparisons of coal, oil and gas should cover not only the stack, but also extraction, flaring and atmospheric transformation.

7.6. Comparative table

Process	Directly measured size range	Main finding	Level for coal-chain UFP
Coal boiler / power station	from a few nanometres to micrometres	Nucleation and ash modes; high technological variability	A/B
Household coal stove	10–420 nm and other ranges	High PNC is possible; combustion conditions are critical	B
Diesel in a coal mine	5.6–560 nm	Direct nanoaerosol from machinery	B
Crushing/blasting	direct data from a non-coal mine	Particles are generated; a comparable coal emission factor has not yet been established	C
Coking	predominantly PM1 and mixed exposure	Fine, numerically dominant fraction and PAHs	C/U for strict UFP evidence
Burning waste dumps	nanoparticles in material and individual laboratory aerosols	Nanominerals and soot form; resident exposure requires field measurements	C/U



A smoking section of a waste dump, general view. Kiselevsk, 2020. Photo: Smogman.

8. Pollution-control efficiency

Electrostatic precipitators, fabric filters, scrubbers, flue-gas desulphurisation systems and wet electrostatic precipitators can remove a large proportion of particle mass. In a study of Chinese power stations, the reported overall collection efficiencies were 96.38–99.89% for electrostatic precipitators and 99.94% for a fabric filter.[11] The lowest size-dependent efficiency occurred in the 0.1–1 μm range; the fabric filter still achieved 99.54% efficiency in that range. The 0.1–1 μm range lies predominantly above the UFP boundary. A local minimum on an efficiency curve therefore does not mean that collection is ineffective.

High removal efficiency by mass can coexist with a substantial number concentration of residual particles: mass and number answer different questions. Assessing potential harm also requires information on composition, size, exposure duration and particle deposition in the respiratory tract.

Wet flue-gas desulphurisation reduces SO_2 but, under some operating conditions, has been associated with sulphuric-acid nuclei; a wet electrostatic precipitator can provide additional removal of fine aerosol. Results from one control train cannot be generalised to every power station.



Industrial area beneath a broad dark plume. Kiselevsk, 2020. Photo: Smogman.

9. Atmospheric transport and exposure

UFP generally show steeper spatial gradients than PM_{2.5}: concentrations change rapidly near a road, stack, stove or other source. During transport, particles coagulate, grow and change in composition. A single urban monitoring station may therefore poorly represent personal exposure.

Assessment requires fixed-site PNC/PNSD monitoring, mobile measurements, personal sensors, meteorological data, the timing of source activity and dispersion models. Indoors, exposure depends on outdoor infiltration, building filtration and indoor sources such as cooking, candles, smoking and heating.

Dose in the respiratory tract depends not only on outdoor PNC, but also on time, pulmonary ventilation, particle size and hygroscopicity. For studies to be comparable, they need to retain not only concentration, but also duration, breathing pattern and the deposition model used.



A playground in winter. Kiselevsk, 2020. Photo: Smogman.

10. Toxicology and mechanisms of harm

Coal smoke and coal dust-and-gas emissions act as mixtures. They can simultaneously contain particles of different sizes, PAHs, metals, sulphates, silica and gases. The toxicity of several of these components and the severe consequences of certain coal-related exposures are established

independently of whether a particular study succeeded in isolating the separate role of UFP.[9][31][32][33][36][37]

10.1. Deposition in the respiratory tract

Deposition means particles settling in the respiratory tract. Diffusional motion allows very small particles to deposit in the nose, bronchi and alveoli. The deposited fraction varies with particle size, breathing route, physical activity, anatomy and lung condition. In a controlled study of 23 nm carbon particles, deposition was higher in adults with asthma, particularly during exercise.[17] The magnitude must be measured separately for children and for specific coal mixtures.

10.2. Translocation

An experiment with inhaled gold nanoparticles in humans and mice found that some particles reached the bloodstream; translocation was greater for 5 nm particles than for 30 nm particles.[18] This experiment demonstrates that transfer across the lung barrier is possible. The actual amount for an atmospheric coal mixture depends on particle composition, solubility and surface properties.

An olfactory pathway to the brain has been demonstrated in rodents using labelled nanoparticles.[24] Its quantitative importance for humans is still under study. Black-carbon particles and nanoaggregates have been detected in human placentas.[27][28] These studies demonstrate that particle transfer is possible, but do not determine the clinical outcome or the source of any particular particle.

10.3. Oxidative stress and inflammation

A large reactive surface, transition metals, quinones and PAHs can contribute to the formation of reactive oxygen species. Cellular and animal models demonstrate activation of inflammatory pathways, impaired endothelial function, changes in blood coagulation and damage to genetic material. In a mouse experiment using size-fractionated fly ash, pulmonary inflammatory responses differed by particle size and coal source.[48] In a separate study of human lung epithelial cells, IL-8 release varied with fly-ash particle fraction and coal source and was associated with bioavailable iron.[49]

These experiments identify biologically plausible mechanisms of harm. Moving from a cellular response to a quantitative estimate of disease risk in humans also requires real-world concentrations and exposure duration. Equal masses of different aerosols may have different biological activity.

10.4. The “Trojan horse” as a limited metaphor

Particles can carry PAHs, sulphates and metals on their surfaces; uptake of a particle by a cell can alter where and how quickly these components are delivered. At the same time, substances may detach from the surface, dissolve and undergo chemical transformation. The “Trojan horse” metaphor helps illustrate the mixture, but actual risk is determined by bioavailability and dose.

10.5. Toxicity of individual components

The hazard posed by coal aerosol is determined not only by particle size but also by chemical composition. Benzo[a]pyrene and several other PAHs are covered by IARC carcinogenicity assessments.[9] Chronic arsenic exposure is associated with cancer and vascular effects; in Guizhou, severe poisoning was linked to household use of coal with a high arsenic content.[31] Excess selenium is toxic, although selenium is also an essential trace element.[44] Lead primarily harms the nervous system, especially in children, while mercury can damage the nervous, digestive and immune systems, lungs and kidneys.[45][46]

Carcinogenicity or toxicity is an established property of the relevant exposure, but individual risk depends on concentration, chemical form, route and duration. Chemical analysis is therefore essential: equal numbers of particles with different compositions may represent different toxic burdens.



A winter skating rink; smoke from a coal-fired boiler house is visible in the background. Kiselevsk, 2020. Photo: Smogman.

11. Clinical and epidemiological evidence

Clinical and epidemiological studies answer two different questions. The first is whether particle pollution and specific coal mixtures cause harm. For a number of exposures, the answer is yes. The second is the independent contribution of UFP after other mixture components are taken into account. For many outcomes, this separate contribution still cannot be quantified reliably.

11.1. Cardiovascular system

A 2019 systematic review found the most consistent short-term associations of UFP with heart-rate variability, blood pressure and markers of systemic inflammation.[19] Controlled exposures have identified changes in repolarisation, endothelial function or platelet activation.

The evidence base for myocardial infarction, stroke and long-term cardiovascular mortality is much stronger for PM_{2.5} than for UFP. After adjustment for NO₂, black carbon and PM_{2.5}, findings for PNC differ across studies.[19] Overall assessment: **B** for some short-term markers; **C/U** for coal-chain UFP and long-term clinical events.

11.2. Respiratory system

The overall UFP evidence base supports short-term irritation and inflammatory changes, but findings for lung function vary. A Canadian cohort of 160,641 births and a US cohort reported an association between prenatal UFP exposure and childhood asthma.[21][22] In the Dutch PIAMA study, the association was no longer statistically significant after adjustment for NO₂ and black carbon in PM_{2.5}. [23] A Copenhagen time-stratified case-crossover study identified a short-term association between UFP and childhood asthma hospitalisations.[20]

Occupational coal and mineral dust is associated with chronic lung disease among miners.[33] There is also a strong broader evidence base for PM in chronic obstructive pulmonary disease and chronic bronchitis. In these mixtures, the independent long-term contribution of UFP has not yet been separated from larger particles, silica, diesel aerosol and gases.

11.3. Cancer

In the relevant assessments, IARC classifies outdoor air pollution and its particulate matter, diesel-engine exhaust, emissions from household coal combustion, and coke production as carcinogenic to humans.[9][36] These classifications concern the named mixtures and exposures. UFP as a single particle-size class do not have a separate classification of this kind.

In its 1997 monograph, IARC placed coal dust in Group 3, “not classifiable as to its carcinogenicity to humans”; an advisory group later recommended it as a priority for re-evaluation, but that recommendation does not itself change the classification.[9] Crystalline

silica and individual chemicals have their own assessments.

11.4. Nervous system and cognitive function

The Medicare study used modelled UFP exposure for 2017 and followed incident dementia diagnoses in 2018–2020. The authors reported a small positive association.[25] In the prospective Adult Changes in Thought cohort of 4,283 participants, no elevated risk was found after adjustment for several pollutants.[26] These findings diverge; conclusions are influenced by the exposure model and co-pollutants. The corpus contains no dementia studies specific to coal-chain UFP.

11.5. Pregnancy and children

There is extensive evidence linking PM_{2.5} with preterm birth and low birth weight.[29] Fewer studies address UFP, and their exposure models differ. Detection of black carbon on the fetal side of the placenta confirms that particles can reach this tissue.[27] Assessing clinical risk and coal's contribution requires studies that measure the source, particle size and pregnancy outcome together.

11.6. Metabolic disorders

In a controlled UFP study of people with type 2 diabetes, researchers observed transient platelet activation and a possible endothelial response.[30] The study tested vascular responses in people who already had diabetes; it did not study the onset of diabetes or obesity. Metabolic outcomes therefore remain an active field of research.

11.7. Mortality

Particulate air pollution is associated with premature mortality in the broader PM evidence base.[1] Findings for UFP are less consistent: studies differ in measured size ranges, exposure models and sets of adjusted co-pollutants.[19] The available evidence therefore does not permit calculation of a separate mortality risk specifically from coal-chain UFP.



Smoke emerging between fragments of rock. Kiselevsk, 2020. Photo: Smogman.

12. Specific coal-related cases

12.1. Xuanwei

In Xuanwei, a high risk of lung cancer among never-smoking women is associated with household combustion of particular “smoky” coals. In a case-control study of 1,015 cases and 485 female controls, risk was associated with groups of PAHs and NO₂.^[37] Mineralogical and size-resolved studies show the presence of nanoscale components and strong dependence of emissions on fuel and stove design.

This is strong evidence for the carcinogenicity of a particular household coal mixture and provides important information about emission chemistry. The study assessed a mixture, not a separate PNC measure or fraction below 100 nm. It therefore confirms the hazard of that specific coal smoke, but does not provide a separate risk coefficient for UFP.

12.2. Guizhou

Chronic arsenic poisoning in coal-burning areas of Guizhou involved several exposure routes: contaminated food accounted for the largest share in the source assessment, air for a smaller share, and water for a small share.^[31] Fluorosis was likewise associated with drying and storing food above coal fires and with mineral impurities in the coal.^[32]

This case directly demonstrates that fuel chemistry and household practices can cause severe chronic exposure and disease.[31][32] The studies describe several routes by which substances entered the body; the independent contribution of inhaled UFP to this harm was not calculated separately.

12.3. Communities near coal-fired power stations and coke plants

A systematic review of populations living near coal-fired power stations described respiratory, childhood, perinatal and other outcomes.[47] Closure of individual facilities or installation of controls created natural-experiment conditions in some studies. Exposure was usually defined by proximity, PM_{2.5} or pollutant mixtures: these findings concern coal-power exposure as a whole, and the independent UFP contribution was not calculated.

Coke-oven worker cohorts and IARC assessments establish the carcinogenicity of the complex occupational exposure.[9] It includes PAHs, coal-tar volatiles, particles and gases simultaneously; the contribution of UFP was not calculated separately in these studies.

12.4. Burning waste dumps and mine fires

Materials-science studies have identified carbon nanoparticles and nanominerals in burning coal waste.[6] Laboratory studies show how soot changes with spontaneous-combustion temperature.[7] Assessing residents' exposure requires long-term PNC and PNSD measurements near homes, chemical source attribution and personal monitoring. The central point is already established: a burning waste dump produces more than mechanical dust — gaseous combustion products and the aerosol that forms are added to it.

13. Occupational medicine

Coal workers' pneumoconiosis, progressive massive fibrosis, reduced lung function and chronic obstructive pulmonary disease are well documented among miners.[33] These diseases are primarily associated with respirable coal and mineral dust, including crystalline silica. The occupational harm is established; quantifying the separate UFP contribution requires size-resolved data.

Diesel machinery adds a nanoaerosol rich in elemental carbon. The Diesel Exhaust in Miners Study was conducted in non-coal mines and confirmed an association between occupational diesel-exhaust exposure and lung cancer.[34] For coal mines, this provides a direct warning about the diesel component of the mixture; it does not determine the contribution of coal-derived UFP themselves.

Occupational exposures in coke production are recognised as carcinogenic.[9] Workers at power stations and coke plants encounter mixtures of dust, PAHs, metals and gases. To estimate the contribution of each component, studies account for employment duration, tasks, protective equipment and the healthy-worker effect — the systematic difference between an employed group and the general population.



Open-pit machinery and residential buildings in the same field of view. Kiselevsk, 2020. Photo: Smogman.

14. Vulnerable groups

Children have developing lungs and higher minute ventilation relative to body weight; older people and those with cardiovascular or respiratory disease may have less physiological reserve. Pregnancy creates a distinct window of exposure, while workers may receive higher occupational doses. Each group requires measurements that reflect its real conditions.

Exposure means a person's contact with a pollutant over time. Residents living near open-pit mines, power stations, coke plants and homes heated by stoves may encounter several sources simultaneously. Social conditions, housing quality, ventilation and access to healthcare can affect both the level of exposure and its health consequences.

15. Separating the effects of UFP from PM2.5

UFP and PM2.5 may share sources, but their concentrations do not always vary in the same way across space and time. A multi-pollutant model tests whether an association with UFP remains after adjustment for NO₂, PM2.5, black carbon and noise. When indicators are closely correlated, statistical uncertainty increases.

The most informative studies combine within-city UFP models, repeated measurements, information on people's movements, personal monitoring and markers of specific sources. Observations before and after a source closes or controls are installed are especially useful if other changes occurring at the same time are taken into account.

Differences between asthma studies, and between the Medicare and ACT dementia findings, demonstrate why multi-pollutant models and precise exposure descriptions are necessary.



An open excavation behind private homes. Kiselevsk, 2020. Photo: Smogman.

16. Regulatory framework

16.1. WHO

The 2021 WHO air-quality guidelines did not establish a numerical guideline for UFP because the available data were insufficient for a reliable concentration–response function. WHO instead recommended monitoring PNC with a lower size cut-off no higher than 10 nm, developing spatial monitoring networks and reducing emissions.[1] A 24-hour concentration below 1,000 particles/cm³ was described provisionally as low and above 10,000 particles/cm³ as high; for an hourly average, a level above 20,000 particles/cm³ was described as high. These are reference values for describing concentration levels, not statutory limits or established safe thresholds.

16.2. European Union

EU Directive 2024/2881 provides for UFP monitoring at enhanced background monitoring supersites and at locations where high concentrations are expected, including traffic, industrial, port and airport areas.[40] The deadline for transposition of the relevant provisions into national law is 11 December 2026. This is a requirement of the directive; it should not be read as evidence that a fully operational monitoring network already exists in every country. The directive does not establish a UFP limit value.

16.3. United States

US EPA air-quality standards regulate PM_{2.5} and PM₁₀ by mass; there is no separate standard for UFP or PNC.[39] UFP are considered within the scientific evidence on particle pollution, but no separate limit value has been established.

16.4. Russia

SanPiN 1.2.3685-21, Chapter I, Table 1.1, in the version dated 24 December 2025, contains mass-based standards for PM_{2.5}, PM₁₀ and individual substances. The table contains no separate standard for UFP number concentration.[38] It was checked on 12 October 2026. Standards describing workplace nanoaerosols and methods for assessing them do not replace an ambient-air standard for populated areas.

17. Overall assessment of the evidence

One boundary is essential when reading this section. The hazards of several coal mixtures are already established: household smoke from particular coals is associated with lung cancer and chronic poisoning; coke-production exposures are recognised as carcinogenic; and coal and mineral dust cause severe occupational disease.[9][31][32][33][36][37] The table below addresses a narrower question: how confidently can the independent contribution of UFP within these mixtures be identified?

17.1. Health outcomes: the independent UFP contribution

Outcome	Direct UFP evidence	Additional evidence base	Conclusion for coal-chain UFP
Pulmonary and systemic inflammation	the most consistent short-term signal	PM2.5, diesel aerosol, fly-ash toxicology	C
Blood pressure and heart-rate variability	recurring short-term changes; effect size varies	strong general PM evidence base	C
Myocardial infarction and stroke	independent UFP contribution has not yet been consistently isolated	strong PM2.5 evidence base	U/C
Asthma	several positive studies; some associations weakened after adjustment for other pollutants	PM2.5 and NO ₂	U/C
COPD	separate UFP contribution has not been calculated	established harm from occupational coal dust; diesel evidence	U/C
Lung cancer	UFP as a size class have not been classified separately	carcinogenicity of several specific mixtures and agents is established	U/C
Dementia	large studies diverge	PM2.5	U
Pregnancy/children	fewer direct studies	PM2.5 and placental mechanisms	U/C
Diabetes and obesity	mechanisms and individual associations remain under study	growing PM2.5 evidence base	U/D

17.2. Substances

Component	What is established	What is required to assess human exposure
Arsenic and selenium	enrichment of fine and submicron ash in a number of systems	size-resolved measurements near homes and personal dose
Mercury	enrichment of particle-bound mercury in one system	UFP contribution to total mercury exposure
PAHs	carriage on particles and strong evidence for coal smoke	separate assessment of UFP and the other mixture components
Silica	strong occupational risk in the respirable fraction	size distribution and UFP contribution
Sulphates	primary and secondary formation; a nanoparticle mode in some installations	linking chemical composition to particle-number size distribution
Radionuclides	enrichment of ash residues	assessment of the specific power station and all exposure pathways

18. Limitations and uncertainties

1. **Incompatible size metrics.** Many coal studies use PM₁ or PM_{2.5}. In this review, they provide additional evidence, not a direct substitute for UFP measurements.
2. **Technological heterogeneity.** Coal type, boiler or stove, control equipment and sampling procedure can radically alter the particle spectrum.
3. **Low UFP mass.** Chemical analysis requires accumulation of material and can miss short-lived peaks.
4. **Source mixtures.** Diesel, roads, heating and secondary chemistry coexist with coal sources.
5. **Exposure error.** A central monitoring station does not always represent an address or personal dose.
6. **Co-pollutants.** UFP correlate with NO₂, black carbon and other indicators; close correlations make it difficult to separate their effects statistically.
7. **Transferability of toxicology.** High cellular doses and purpose-made nanoparticles differ from atmospheric mixtures.
8. **Occupational and residential exposure.** Mine concentrations and composition require separate assessment and cannot serve as direct estimates for residents.
9. **Russian evidence gap.** No representative public PNC/PNSD time series was found for coal towns in Kuzbass.
10. **Publication cut-off.** Regulations and studies published after 12 October 2026 are not included.

19. Conclusions and recommendations

The absence of a separate UFP standard or risk coefficient does not allow coal pollution to be regarded as safe. Serious health effects have been documented for a number of specific exposures: household smoke from particular coal types, mixtures encountered in coke production, occupational coal and mineral dust, and diesel exhaust.[9][31][32][33][34][36][37]

The uncertainty concerns a narrower question: what share of the overall risk is caused specifically by particles smaller than 100 nm after PM_{2.5}, gases, silica, PAHs, metals and other components are separated. Answering it requires particle-number and particle-size measurements alongside chemical analysis and exposure data.

For regulators

- Supplement mass-based PM monitoring with PNC/PNSD measurements with a documented lower size cut-off.
- Select sites at expected concentration maxima as well as background sites; do not substitute one for the other.
- Publish raw time series, instrument metadata, QA/QC information and accompanying meteorological data.
- Distinguish primary from secondary particles and occupational from residential exposure.

For industry

- Assess control systems simultaneously by mass, number and particle-size distribution.
- Measure before and after each control stage under stable and transitional operating conditions.
- Include diesel machinery, blasting, crushing, waste dumps and uncontrolled burning in emission inventories.
- Publish number-based efficiency and residual particle-size distributions alongside mass-based efficiency.

For clinicians and public-health professionals

- Consider the combined exposure rather than searching for a “disease caused by one particle”.
- Do not conflate miners’ occupational diseases with risks to residents.
- Report the level of evidence and possible co-pollutants.

For researchers

- Conduct long-term studies using local and personal UFP measurements and attribution of coal sources.
- Combine PNC/PNSD, chemistry, PMF/CMB, meteorology and process logs.
- Publish null findings and sensitivity analyses.
- Build a comparable Russian evidence base for Kuzbass.

For residents

HEPA filters do not capture particles by acting simply as a sieve: diffusion is important for the smallest particles, while interception and inertia are important for larger particles.[43] Household filtration can reduce particle concentrations in a room, including UFP, but it does not remove every gas or eliminate the source.[41] Select an air cleaner with a clean air delivery rate (CADR) appropriate for the room size, and avoid devices designed to generate ozone. If smoke is visible outdoors, there is a strong smell of burning or an obvious emission event is under way, it is reasonable to reduce time outside, close windows for the duration of the episode and use filtration where possible. This precaution does not require the smoke's composition to be determined in a laboratory first.

Author's conclusion

After that public hearing, I was left with a simple question: what is actually in the dust that people are told to tolerate? Research answers it clearly enough that the dust cannot be called "beneficial". The coal chain produces not one substance but a mixture: mechanical dust, diesel aerosol, products of blasting and combustion, soot, PAHs, metals and secondary particles. The smallest particles cannot be assessed from a visible black coating or from PM_{2.5} mass alone. They need to be counted, measured by size and analysed chemically.

Scientific caution here means precision, not inaction. Where data for a particular town are lacking, measurements are needed. Without such monitoring, part of the exposure remains unrecorded in official statistics, and residents do not receive a complete picture of the pollution.

20. Appendices

20.1. How to read a measurement result

Before interpreting a result, check: size range; metric; units; instrument; location; duration; dilution; humidity; chemical analysis; background control; co-pollutants.

20.2. Mini-glossary

- **UFP** — ultrafine particles, generally smaller than 100 nm.
- **PNC** — particle number concentration, generally particles/cm³.
- **PNSD** — particle number size distribution.

- **PM_{0.1}** — a mass-based size fraction with an upper cut-off of approximately 0.1 µm. PM_{0.1} and UFP number concentration are different measures and are not necessarily directly comparable.
- **PM₁ / PM_{2.5} / PM₁₀** — mass-based fractions up to the stated aerodynamic diameter.
- **LDSA** — estimated lung-deposited surface area.
- **Nucleation** — formation of new particles from gas molecules.
- **Coagulation** — particles joining together, reducing number and increasing size.
- **PMF / CMB** — receptor models for source apportionment.

20.3. Measurements still needed in Kuzbass

- What are the annual mean PNC and PNSD in residential areas of Kuzbass?
- What proportion of UFP comes from coal, diesel, heating and transport?
- How does the particle-size distribution change during mass blasting and spontaneous combustion?
- What is the cumulative personal dose received by residents and workers?
- What clinical risk remains after adjustment for PM_{2.5}, NO₂ and black carbon?

21. References and source register

1. World Health Organization. 2021. *WHO Global Air Quality Guidelines: Particulate Matter (PM_{2.5} and PM₁₀), Ozone, Nitrogen Dioxide, Sulfur Dioxide and Carbon Monoxide*. WHO. <https://www.who.int/publications/i/item/9789240034228>.
2. Bugarski A.D. et al. 2021. *Diesel Aerosols in an Underground Coal Mine*. In: *Proceedings of the 18th North American Mine Ventilation Symposium*. DOI: 10.1201/9781003188476-10; NIOSH Stacks.
3. Saarikoski S. et al. 2019. *Sources and Characteristics of Particulate Matter at Five Locations in an Underground Mine*. *Aerosol and Air Quality Research*, 19, 2613–2624. DOI: 10.4209/aaqr.2019.03.0118.
4. Larionov A. et al. 2022. *Chemical Composition and Toxicity of PM₁₀ and PM_{0.1} Samples near Open-Pit Mines and Coal Power Stations*. *Life*, 12(7), 1047. DOI: 10.3390/life12071047.
5. Leshukov T. et al. 2022. *Fractional Composition and Toxicity Coal-Rock of PM₁₀–PM_{0.1} Dust near an Opencast Coal Mining Area and Coal-Fired Power Station*. *Sustainability*, 14(24), 16594. DOI: 10.3390/su142416594.
6. Ribeiro J. et al. 2010. *Identification of Nanominerals and Nanoparticles in Burning Coal Waste Piles from Portugal*. *Science of the Total Environment*, 408(23), 6032–6041. DOI: 10.1016/j.scitotenv.2010.08.046.
7. Tan B. et al. 2026. *Experimental Study on Emission Characteristics of Soot Particles during Spontaneous Combustion of Coal*. *Fuel*, 403, 136139. DOI: 10.1016/j.fuel.2025.136139.
8. Xie Q. et al. 2015. *Size Distribution Characteristics of Particulate Matter in the Top Areas of Coke Oven*. *Chinese Journal of Industrial Hygiene and Occupational Diseases*, 33(3), 161–165. PubMed 25916437.
9. International Agency for Research on Cancer. No date. *Agents Classified by the IARC Monographs*. IARC Monographs. Accessed 12 October 2026. <https://monographs.iarc.who.int/agents-classified-by-the-iarc/>.
10. Mertens J. et al. 2020. *Fine and Ultrafine Particle Number and Size Measurements from Industrial Combustion Processes: Primary Emissions Field Data*. *Atmospheric Pollution Research*, 11(4), 803–814. DOI: 10.1016/j.apr.2020.01.008.
11. Yi H. et al. 2006. *Characteristics of Inhalable Particulate Matter Concentration and Size Distribution from Power Plants in China*. *Journal of the Air & Waste Management Association*, 56(9), 1243–1251. DOI: 10.1080/10473289.2006.10464590.
12. Luo Z. et al. 2022. *Impacts of Household Coal Combustion on Indoor Ultrafine Particles—A Preliminary Case Study and Implication on Exposure Reduction*. *International Journal of Environmental Research and Public Health*, 19(9), 5161. DOI: 10.3390/ijerph19095161.
13. Senior C., Granite E., Linak W., Seames W. 2020. *Chemistry of Trace Inorganic Elements in Coal Combustion Systems: A Century of Discovery*. *Energy & Fuels*, 34(12), 15141–15168. DOI: 10.1021/acs.energyfuels.0c02375.
14. Peng Y. et al. 2021. *Mercury Speciation and Size-Specific Distribution in Filterable and Condensable Particulate Matter from Coal Combustion*. *Science of the Total Environment*, 787, 147597. DOI: 10.1016/j.scitotenv.2021.147597.
15. Oros D.R., Simoneit B.R.T. 2000. *Identification and Emission Rates of Molecular Tracers in Coal Smoke Particulate Matter*. *Fuel*, 79(5), 515–536. DOI: 10.1016/S0016-2361(99)00153-2.
16. Chang M.-C.O. et al. 2004. *Measurement of Ultrafine Particle Size Distributions from Coal-, Oil-, and Gas-Fired Stationary Combustion Sources*. *Journal of the Air & Waste Management Association*, 54(12), 1494–1505. DOI: 10.1080/10473289.2004.10471010.
17. Chalupa D.C. et al. 2004. *Ultrafine Particle Deposition in Subjects with Asthma*. *Environmental Health Perspectives*, 112(8), 879–882. DOI: 10.1289/ehp.6851.
18. Miller M.R. et al. 2017. *Inhaled Nanoparticles Accumulate at Sites of Vascular Disease*. *ACS Nano*, 11(5), 4542–4552. DOI: 10.1021/acsnano.6b08551.

19. Ohlwein S. et al. 2019. *Health Effects of Ultrafine Particles: A Systematic Literature Review Update of Epidemiological Evidence*. *International Journal of Public Health*, 64(4), 547–559. DOI: [10.1007/s00038-019-01202-7](https://doi.org/10.1007/s00038-019-01202-7).
20. Bergmann M. et al. 2025. *Short-Term Exposure to Ultrafine Particles and Asthma Hospital Admissions in Children in Copenhagen, Denmark*. *Thorax*, 80(11), 796–802. DOI: [10.1136/thorax-2024-222465](https://doi.org/10.1136/thorax-2024-222465).
21. Lavigne E. et al. 2019. *Spatiotemporal Variations in Ambient Ultrafine Particles and the Incidence of Childhood Asthma*. *American Journal of Respiratory and Critical Care Medicine*, 199(12), 1487–1495. DOI: [10.1164/rccm.201810-1976OC](https://doi.org/10.1164/rccm.201810-1976OC).
22. Wright R.J. et al. 2021. *Prenatal Ambient Ultrafine Particle Exposure and Childhood Asthma in the Northeastern United States*. *American Journal of Respiratory and Critical Care Medicine*, 204(7), 788–796. DOI: [10.1164/rccm.202010-3743OC](https://doi.org/10.1164/rccm.202010-3743OC).
23. Yu Z. et al. 2022. *Ambient Ultrafine Particles and Asthma Onset until Age 20: The PIAMA Birth Cohort*. *Environmental Research*, 214(1), 113770. DOI: [10.1016/j.envres.2022.113770](https://doi.org/10.1016/j.envres.2022.113770).
24. Oberdörster G. et al. 2004. *Translocation of Inhaled Ultrafine Particles to the Brain*. *Inhalation Toxicology*, 16(6–7), 437–445. DOI: [10.1080/08958370490439597](https://doi.org/10.1080/08958370490439597).
25. Zhu Q. et al. 2025. *Associations between Ultrafine Particles and Incident Dementia in Older Adults*. *Environmental Science & Technology*, 59(11), 5443–5451. DOI: [10.1021/acs.est.4c10574](https://doi.org/10.1021/acs.est.4c10574).
26. Blanco M.N. et al. 2024. *Traffic-Related Air Pollution and Dementia Incidence in the Adult Changes in Thought Study*. *Environment International*, 183, 108418. DOI: [10.1016/j.envint.2024.108418](https://doi.org/10.1016/j.envint.2024.108418).
27. Bové H. et al. 2019. *Ambient Black Carbon Particles Reach the Fetal Side of Human Placenta*. *Nature Communications*, 10, 3866. DOI: [10.1038/s41467-019-11654-3](https://doi.org/10.1038/s41467-019-11654-3).
28. Bongaerts E. et al. 2020. *Translocation of (Ultra)fine Particles and Nanoparticles across the Placenta: A Systematic Review on the Evidence of In Vitro, Ex Vivo, and In Vivo Studies*. *Particle and Fibre Toxicology*, 17, 56. DOI: [10.1186/s12989-020-00386-8](https://doi.org/10.1186/s12989-020-00386-8).
29. Yuan L. et al. 2019. *Maternal Fine Particulate Matter (PM_{2.5}) Exposure and Adverse Birth Outcomes: An Updated Systematic Review Based on Cohort Studies*. *Environmental Science and Pollution Research*, 26(14), 13963–13983. DOI: [10.1007/s11356-019-04644-x](https://doi.org/10.1007/s11356-019-04644-x).
30. Stewart J.C. et al. 2010. *Vascular Effects of Ultrafine Particles in Persons with Type 2 Diabetes*. *Environmental Health Perspectives*, 118(12), 1692–1698. DOI: [10.1289/ehp.1002237](https://doi.org/10.1289/ehp.1002237).
31. Liu J. et al. 2002. *Chronic Arsenic Poisoning from Burning High-Arsenic-Containing Coal in Guizhou, China*. *Environmental Health Perspectives*, 110(2), 119–122. DOI: [10.1289/ehp.02110119](https://doi.org/10.1289/ehp.02110119).
32. Guo J. et al. 2021. *Review on Health Impacts from Domestic Coal Burning: Emphasis on Endemic Fluorosis in Guizhou Province, Southwest China*. *Reviews of Environmental Contamination and Toxicology*, 258, 1–25. DOI: [10.1007/398_2021_71](https://doi.org/10.1007/398_2021_71).
33. Almberg K.S., Cohen R.A. 2023. *Modern Coal Miners Have Higher Death Rates from Lung Diseases Than Their Predecessors*. NIOSH Science Bulletin. <https://www.cdc.gov/niosh/bulletin/2023/mining-lung-disease.html>.
34. Attfield M.D. et al. 2012. *The Diesel Exhaust in Miners Study: A Cohort Mortality Study with Emphasis on Lung Cancer*. *Journal of the National Cancer Institute*, 104(11), 869–883. DOI: [10.1093/jnci/djs035](https://doi.org/10.1093/jnci/djs035).
35. United Nations Scientific Committee on the Effects of Atomic Radiation. 2025. *Sources, Effects and Risks of Ionizing Radiation: UNSCEAR 2024 Report, Volume II, Scientific Annex B*. United Nations. https://www.unscear.org/unscear/uploads/documents/publications/UNSCEAR_2024_Annex-B.pdf.
36. International Agency for Research on Cancer. 2010. *Household Use of Solid Fuels and High-Temperature Frying*. IARC Monographs, Volume 95. <https://publications.iarc.who.int/113>.
37. Vermeulen R. et al. 2019. *Constituents of Household Air Pollution and Risk of Lung Cancer among Never-Smoking Women in Xuanwei and Fuyuan, China*. *Environmental Health Perspectives*, 127(9), 097001. DOI: [10.1289/EHP4913](https://doi.org/10.1289/EHP4913).
38. Chief State Sanitary Physician of the Russian Federation. 2021. *SanPiN 1.2.3685-21: Hygiene standards and requirements for ensuring the safety and/or harmlessness of environmental factors to humans*, Chapter I, Table 1.1; version dated 24 December 2025, amendments effective 1 March 2026. Accessed 12 October 2026. https://www.consultant.ru/document/cons_doc_LAW_375839/fa69e15a74de57cbe09d347462434c11fcfeeaca/.
39. U.S. Environmental Protection Agency. No date. *NAAQS Table*. Accessed 12 October 2026. <https://www.epa.gov/criteria-air-pollutants/naaqs-table>.

40. European Parliament and Council of the European Union. 2024. *Directive (EU) 2024/2881 of 23 October 2024 on Ambient Air Quality and Cleaner Air for Europe (Recast)*. *Official Journal of the European Union*. <https://eur-lex.europa.eu/legal-content/EN/TXT/?uri=CELEX%3A32024L2881>.

41. U.S. Environmental Protection Agency. 2024. *Guide to Air Cleaners in the Home*. EPA 402-F-24-002. <https://www.epa.gov/indoor-air-quality-iaq/guide-air-cleaners-home>.

42. Chyka P.A., Seger D. 1997. *Position Statement: Single-Dose Activated Charcoal*. *Journal of Toxicology: Clinical Toxicology*, 35(7), 721–741. DOI: 10.3109/15563659709162569.

43. Alonso M., Alguacil F.J. 2001. *An Introduction to Aerosol Filtration*. *Revista de Metalurgia*, 37(6), 693–712. DOI: 10.3989/revmetalm.2001.v37.i6.536.

44. National Institutes of Health, Office of Dietary Supplements. No date. *Selenium: Fact Sheet for Health Professionals*. Accessed 12 October 2026. <https://ods.od.nih.gov/factsheets/Selenium-HealthProfessional/>.

45. World Health Organization. No date. *Lead Poisoning and Health*. Accessed 12 October 2026. <https://www.who.int/news-room/fact-sheets/detail/lead-poisoning-and-health>.

46. World Health Organization. No date. *Mercury and Health*. Accessed 12 October 2026. <https://www.who.int/news-room/fact-sheets/detail/mercury-and-health>.

47. Amster E. 2021. *Public health impact of coal-fired power plants: a critical systematic review of the epidemiological literature*. *International Journal of Environmental Health Research*, 31(5), 558–580. DOI: 10.1080/09603123.2019.1674256.

48. Gilmour M.I., O'Connor S., Dick C.A.J., Miller C.A., Linak W.P. 2004. *Differential pulmonary inflammation and in vitro cytotoxicity of size-fractionated fly ash particles from pulverized coal combustion*. *Journal of the Air & Waste Management Association*, 54(3), 286–295. DOI: 10.1080/10473289.2004.10470906.

49. Smith K.R., Veranth J.M., Hu A.A., Lighty J.S., Aust A.E. 2000. *Interleukin-8 levels in human lung epithelial cells are increased in response to coal fly ash and vary with the bioavailability of iron, as a function of particle size and source of coal*. *Chemical Research in Toxicology*, 13(2), 118–125. DOI: 10.1021/tx9901736.

Authors and AI involvement

- **Natalia Zubkova** — author.
- **Sergey Nikolaevich Severin** — co-author and editor.
- **Viktor Andreevich Svetlov** — co-author and researcher.

OpenAI models were used in preparing this material: ChatGPT in Sergey Nikolaevich Severin’s editorial and analytical work, and Codex in Viktor Andreevich Svetlov’s research and technical work. The first-person account, final wording and decision to publish belong to Natalia Zubkova.

Appendix A. Full annotated source register

For every source, the actual particle-size fraction, its role in the study and the limit of the permissible conclusion are retained.

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1. Definitions, measurement and atmospheric behaviour

M01 · WHO. *Global Air Quality Guidelines* (2021). <https://www.who.int/publications/i/item/9789240034228>

Size / metric	PNC; lower cut-off ≤ 10 nm, no upper cut-off specified in the good practice statement	Type	official
Role in this study	Defines good practice for UFP monitoring; absence of a numerical AQG; distinction between low and high PNC	Does not support	Treating the recommended levels as statutory health limits

M02 · WHO. *Executive Summary of the Air Quality Guidelines*. <https://iris.who.int/bitstream/handle/10665/345334/9789240034433-eng.pdf>

Size / metric	PNC	Type	official
Role in this study	Concise wording of the good practice statements	Does not support	Inferring an individual medical risk from a single PNC value

M03 · EU Directive 2024/2881. <https://eur-lex.europa.eu/legal-content/EN/TXT/?uri=CELEX%3A32024L2881>

Size / metric	UFP/PNC	Type	legislation
Role in this study	EU obligations for UFP monitoring at supersites and near expected high-concentration sources	Does not support	Claiming that the EU has set a UFP concentration limit

M04 · GOST R 54597-2011 / ISO/TR 27628:2007. https://meganorm.ru/mega_doc/norm/gost-r_gosudarstvennyj-standart/18/gost_r_54597-2011_ISO_TR_27628_2007_natsionalnyy_standart.html

Size / metric	workplace nanoaerosols	Type	standard
Role in this study	Russian terminology and measurement methods for nanoscale aerosols	Does not support	Claiming that Russia has an ambient air-quality standard based on UFP number

M05 · SanPiN 1.2.3685-21, version dated 24 December 2025, with amendments effective in 2026. https://www.consultant.ru/document/cons_doc_LAW_375839/fa69e15a74de57cbe09d347462434c11fcfeeaca/

Size / metric	mass-based limits, including PM2.5/PM10	Type	legislation
Role in this study	The version in force at the cut-off date regulates mass concentrations of PM2.5/PM10 and individual substances; no separate PNC/UFP limit was found in the ambient-air section	Does not support	Treating the absence of a separate UFP limit as absence of risk, or claiming that UFP are entirely outside broader mass- and substance-based indicators

M06 · HEI Review Panel. <i>Understanding the Health Effects of Ambient Ultrafine Particles</i> (2013). https://www.healtheffects.org/system/files/Perspectives3.pdf			
Size / metric	UFP/PNC	Type	expert review
Role in this study	Foundational framework for sources, exposure, toxicology and epidemiology	Does not support	Presenting a 2013 review as the current overall conclusion without later studies
M07 · Schraufnagel et al. <i>Ultrafine particles: unique physicochemical properties relevant to health and disease</i> (2020). https://pmc.ncbi.nlm.nih.gov/articles/PMC7156720/			
Size / metric	UFP	Type	review
Role in this study	Number versus mass, surface area, dynamics, coagulation and source gradients	Does not support	Assigning one universal atmospheric lifetime to UFP under all conditions
M08 · JRC. <i>Overview of ultrafine particles...</i> https://publications.jrc.ec.europa.eu/repository/bitstream/JRC128188/JRC128188_01.pdf			
Size / metric	UFP/PNC	Type	official scientific review
Role in this study	Definitions, instruments, chemical composition and regulatory gaps	Does not support	Treating all 50–100 nm particles as chemically identical
M09 · Wiedensohler et al. <i>Harmonisation of mobility particle size spectrometers</i>. https://publications.jrc.ec.europa.eu/repository/handle/JRC70396			
Size / metric	approximately 20–200 nm	Type	interlaboratory study
Role in this study	Comparability of SMPS/MPSS after calibration and correction for losses	Does not support	Assuming that measurements from different instruments are automatically comparable
M10 · ACTRIS. <i>Standard procedures for aerosol in-situ measurements</i>. https://www.actris-ecac.eu/files/ACTRIS_standard_procedures_for_aerosol_in-situ_measurements.pdf			
Size / metric	PNSD	Type	technical guidance
Role in this study	Quality control for size-resolved measurements	Does not support	Substituting a procedural description for evidence of actual exposure
M11 · Rissler et al. <i>A work group report on ultrafine particles</i> (2016). https://pmc.ncbi.nlm.nih.gov/articles/PMC4976002/			
Size / metric	UFP, number/volume/surface	Type	expert review
Role in this study	Distinguishes metrics and discusses biological plausibility	Does not support	Declaring one metric proven superior for every health effect

M12 · Kreyling et al. / Critical review of lung deposition measurements. https://pubmed.ncbi.nlm.nih.gov/24151837/			
Size / metric	size-resolved deposition	Type	critical review
Role in this study	Dependence of deposition on size, breathing pattern and method	Does not support	Assigning one deposition fraction to the entire UFP range
M13 · HEI workshop on metrics. https://pubmed.ncbi.nlm.nih.gov/27801854/			
Size / metric	mass/number/surface	Type	expert report
Role in this study	Metrics describe different aerosol properties	Does not support	Conflating PNC, mass and surface area
M14 · <i>Mobile-source ultrafine particles: definitions...</i> https://pubmed.ncbi.nlm.nih.gov/35530659/			
Size / metric	UFP; different lower cut-offs	Type	review/methodology
Role in this study	Demonstrates the dependence of PNC on the instrument's measurement range	Does not support	Comparing values without the lower size cut-off
M15 · US EPA. NAAQS Table. https://www.epa.gov/criteria-air-pollutants/naaqs-table			
Size / metric	PM2.5/PM10 mass	Type	official
Role in this study	Current US standards list mass-based PM2.5/PM10, with no separate UFP/PNC NAAQS	Does not support	Concluding that UFP are safe or are not studied
M16 · US EPA. PM ISA 2019 and 2022 supplement. https://www.epa.gov/naaqs/particulate-matter-pm-standards-integrated-science-assessments-current-review			
Size / metric	PM, including UFP literature	Type	official scientific assessment
Role in this study	EPA scientific evidence on PM components, sources and UFP	Does not support	Treating the ISA as a UFP regulation

2. Coal combustion, ash and pollution control

C01 · *Measurement of ultrafine particle size distributions from coal-, oil-, and gas-fired stationary combustion sources.* <https://pubmed.ncbi.nlm.nih.gov/15648387/>

Size / object	UFP, stationary combustion	Type	primary
Role in this study	Comparison of three fuels; influence of dilution and sample ageing	Does not support	A universal ranking for all technologies

C02 · *Yi et al. Full-scale lignite power plant after ESP (2008).* <https://pubs.acs.org/doi/10.1021/ef700527y>

Size / object	0.03–10 µm	Type	primary, one power station
Role in this study	Two number peaks downstream of an ESP; UFP can be nearly invisible by mass	Does not support	Generalising the profile to every coal and power station

C03 · *Yi H. et al. Characteristics of inhalable particulate matter concentration and size distribution from power plants in China (2006).* <https://pubmed.ncbi.nlm.nih.gov/17004679/>

Size / object	0.03–10 µm	Type	primary, five Chinese power stations
Role in this study	Bimodal distribution; ESP/fabric-filter efficiency; minimum efficiency at 0.1–1 µm	Does not support	Applying the percentages to any control system

C04 · *Sun et al. (2014).* <https://pubmed.ncbi.nlm.nih.gov/25826918/>

Size / object	5.6–560 nm	Type	laboratory
Role in this study	Nucleation modes during pulverised-coal combustion	Does not support	Treating a laboratory emission factor as an emission factor for an operating power station

C05 · *Ultrafine ash aerosols from coal combustion: characterization and health effects.* <https://www.sciencedirect.com/science/article/abs/pii/S154074890600349X>

Size / object	ultrafine ash	Type	primary/mechanistic review
Role in this study	Vaporisation, nucleation and condensation; distinctive composition of the ultrafine mode	Does not support	Assigning the same composition and toxicity to all particles

C06 · *Chemistry of Trace Inorganic Elements in Coal Combustion Systems*. <https://pmc.ncbi.nlm.nih.gov/articles/PMC8048144/>

Size / object	submicron; below 0.2 µm in some studies	Type	review
Role in this study	As, Se, Sb, Cd and others in fine fractions; roles of Fe, Ca, S and coal type	Does not support	Labelling every cited result as strictly UFP below 100 nm

C07 · Wang et al. As/Se in PM1 (2022). <https://www.sciencedirect.com/science/article/abs/pii/S0048969722008154>

Size / object	PM1	Type	primary
Role in this study	Enrichment and bioavailability of As/Se in the fine fraction; boiler differences	Does not support	Applying the results directly to UFP or population dose

C08 · As/Se speciation in coal fly ash (2021). <https://www.sciencedirect.com/science/article/abs/pii/S0016236121001046>

Size / object	fine ash	Type	primary
Role in this study	Chemical forms of As and Se	Does not support	Identifying an urban aerosol source without receptor analysis

C09 · *Formation and control of primary PM from coal combustion*. <https://www.tandfonline.com/doi/full/10.1080/10962247.2014.951744>

Size / object	three modes	Type	review
Role in this study	Mechanisms of ultrafine, intermediate and coarse modes; controls	Does not support	Presenting modal boundaries as identical across installations

C10 · ULE plant with ESP+FGD+WESP. <https://www.sciencedirect.com/science/article/pii/S001623611630789X>

Size / object	fine PM	Type	primary, one configuration
Role in this study	High overall removal and composition of residual emissions	Does not support	Concluding that UFP are completely eliminated

C11 · *Fine and ultrafine particle number... industrial combustion processes* (2020). <https://www.sciencedirect.com/science/article/pii/S1309104220300180>

Size / object	particle number downstream of control systems	Type	field study, different installations
Role in this study	Compliance with mass limits does not preclude large numbers of nanoparticles	Does not support	Comparing installations without accounting for dilution and instruments

C12 · Gilmour M.I. et al. *Differential pulmonary inflammation and in vitro cytotoxicity of size-fractionated fly ash particles from pulverized coal combustion* (2004). <https://pubmed.ncbi.nlm.nih.gov/15061611/>

Size / object	below 0.2 µm / fine / coarse	Type	mouse instillation and in vitro toxicology
Role in this study	Pulmonary inflammatory responses differed by particle size and coal source	Does not support	Claiming that the effect is clinically established in humans

C13 · Gasparotto et al. (2013). <https://pubmed.ncbi.nlm.nih.gov/23856402/>

Size / object	ash containing ultrafine particles/nanoparticles	Type	cellular
Role in this study	Oxidative stress and inflammatory responses in macrophages	Does not support	Attributing the effect solely to coal: the fuel contained 2–3% tyres

C14 · Smith K.R. et al. *Interleukin-8 levels in human lung epithelial cells are increased in response to coal fly ash and vary with the bioavailability of iron, as a function of particle size and source of coal* (2000). <https://pubmed.ncbi.nlm.nih.gov/10688536/>

Size / object	below 1 µm, below 2.5 µm and larger	Type	human lung epithelial-cell model
Role in this study	IL-8 release varied with particle fraction and coal source and was associated with bioavailable iron	Does not support	Calling the below-1 µm fraction UFP or treating a cell response as clinical evidence

C15 · Nano-mineralogy of coal fly ash. <https://pubmed.ncbi.nlm.nih.gov/30711584/>

Size / object	5–500 nm	Type	primary, materials
Role in this study	Forms, minerals and potentially hazardous elements in ash nanoparticles	Does not support	Establishing their atmospheric concentration or human dose

C16 · *Impacts of Household Coal Combustion on Indoor Ultrafine Particles* (2022). <https://pmc.ncbi.nlm.nih.gov/articles/PMC9101610/>

Size / object	10–420 nm; separate analysis of 20–100 nm	Type	field case study
Role in this study	Direct simultaneous indoor/outdoor PNC measurement; very high peak during coal combustion	Does not support	A universal profile for every home, coal or stove

C17 · Qin et al. *Hourly Emission of Submicron PM From Residential Coal Combustion* (2026). <https://agupubs.onlinelibrary.wiley.com/doi/10.1029/2026JD046630>

Size / object	PNC/surface area, submicron	Type	primary + inventory
Role in this study	Differences between flaming and smouldering; PNC and lung-deposited surface area	Does not support	Treating a risk model as observed disease

C18 · Bond et al. *Primary particle emissions from residential coal burning* (2002). <https://agupubs.onlinelibrary.wiley.com/doi/full/10.1029/2001JD000571>

Size / object	size distributions	Type	primary
Role in this study	Different coals produce different modes and optical properties	Does not support	A single household-coal profile

C19 · *Significant UFP emissions from residential solid fuel combustion* (2020). <https://pubmed.ncbi.nlm.nih.gov/32023515/>

Size / object	UFP	Type	laboratory + real-world
Role in this study	Direct particle-number emission factors for coal and biomass	Does not support	Comparing values measured under different combustion conditions

C20 · Xuanwei household coal pollution and fuel efficiency (2021). <https://www.sciencedirect.com/science/article/pii/S0269749121015311>

Size / object	size distribution, PM _{0.1}	Type	primary
Role in this study	Coal source and stove alter the size profile; improved stoves reduce emissions	Does not support	Attributing a medical outcome to one size fraction

C21 · Xuanwei C1 coal nano-mineralogy. <https://hero.epa.gov/reference/3363746/>

Size / object	20–200 nm; PM ₁	Type	primary
Role in this study	Mineral composition of fine/ultrafine emissions from a particular coal	Does not support	A general signature for Xuanwei or all coals

3. Mining, diesel, processing, coking and fires

D01 · NIOSH. *Diesel aerosols in an underground coal mine*. https://stacks.cdc.gov/view/cdc/215443/cdc_215443_DS1.pdf

Size / object	5.6–560 nm (FMPS)	Type	field
Role in this study	Direct size-resolved measurements of diesel aerosol in a coal mine	Does not support	Attributing all aerosol to coal dust

D02 · MSHA. *Estimation of Diesel Particulate Concentrations in Underground Mines*. <https://arlweb.msha.gov/S%26HINFO/TECHRPT/DIESEL/DIESEL3.pdf>

Size / object	diesel PM	Type	official field report
Role in this study	Scale of occupational diesel exposure in mines	Does not support	Separating UFP without a size-resolving instrument

D03 · NIOSH workshop on diesel equipment in underground coal mines. <https://www.cdc.gov/niosh/docs/82-122/pdfs/82-122.pdf>

Size / object	diesel/mineral dust	Type	official historical source
Role in this study	Context for mixed exposure	Does not support	Current levels or causality of individual components

D04 · Coke-oven emissions, NTP Report on Carcinogens. <https://www.ncbi.nlm.nih.gov/books/NBK590934/>

Size / object	mixture of dust, gases and PAHs	Type	official review
Role in this study	Composition of coke-production emissions and carcinogenic exposure hazard	Does not support	Attributing the entire effect to UFP

D05 · IARC. *Chemical Agents and Related Occupations, Volume 100F* (2012), including coke production, occupational exposures during coal-tar distillation and coal-tar pitch. <https://publications.iarc.who.int/123>; National Toxicology Program. *Coke-Oven Emissions, 15th Report on Carcinogens* (2021). <https://www.ncbi.nlm.nih.gov/books/NBK590934/>

Size / object	mixture	Type	official assessments
Role in this study	Carcinogenicity of specific occupational exposures and coke-oven emissions	Does not support	Applying the classifications to any particle carrying PAHs or to UFP as a separate size class

D06 · PAHs near a modern coke battery. https://www.sciencedirect.com/science/article/abs/pii/S0016236114012782			
Size / object	TSP/PAH	Type	field
Role in this study	PAH profiles near coke production	Does not support	Calling a TSP study a UFP study
D07 · Nanomineralogy... coal fire. https://hero.epa.gov/reference/3356411/			
Size / object	nano/UFP in burnt waste	Type	materials science
Role in this study	Nanominerals and elements in spontaneous-combustion waste	Does not support	Establishing the dose inhaled by residents
D08 · Ordos coal mine/waste-dump monitoring. https://pubs.rsc.org/en/content/articlehtml/2021/ra/d0ra10586d			
Size / object	atmospheric pollutants	Type	field
Role in this study	Context for mine fires and waste dumps	Does not support	Automatically treating measured PM as strict UFP
D09 · Saarikoski et al. <i>Sources and Characteristics of Particulate Matter at Five Locations in an Underground Mine</i> (2019). https://aaqr.org/articles/aaqr-19-03-0a-0118			
Size / object	nano-SMPS, ELPI+, OPC; PM1/PM10	Type	field, underground chromite mine
Role in this study	Direct measurements near the crusher, unloading, conveyor and blast area; PNC 1.7×10^3 – 2.3×10^5 cm ⁻³ ; PMF attributed 35–84% of PM1 to diesel and 7–60% to blasting	Does not support	Calling these values representative of a coal mine or a universal emission factor; applying PM1 composition to all nanoparticles by number
D10 · Larionov et al. <i>Chemical Composition and Toxicity of PM10 and PM0.1 Samples near Open-Pit Mines and Coal Power Stations</i> (2022). https://doi.org/10.3390/life12071047			
Size / object	PM10 and PM0.1 separated from snow	Type	cumulative field sampling + in vitro, Kuzbass
Role in this study	Confirms a separable nanoscale fraction in accumulated dust near three open pits and the Belovo power station; compares composition and cellular responses	Does not support	Calling the result an instantaneous atmospheric PNC, personal dose, established share from a specific facility, or disease among residents

D11 · Leshukov et al. *Fractional Composition and Toxicity Coal–Rock of PM10–PM0.1 Dust near an Opencast Coal Mining Area and Coal-Fired Power Station* (2022). <https://doi.org/10.3390/su142416594>

Size / object	dust deposited over winter; 10–0.1 µm and below 0.1 µm	Type	cumulative field sampling + in vitro, Kuzbass
Role in this study	Size separation of coal-rock dust near open pits and a power station; emphasises mixed contributions from mining, transport, dumps, machinery and the station	Does not support	Establishing the source of each particle, airborne concentration or causation of disease

D12 · Xie et al. *Size distribution characteristics of particulate matter in the top areas of coke oven* (2015). PMID 25916437. <https://pubmed.ncbi.nlm.nih.gov/25916437/>

Size / object	aerodynamic fractions, predominantly PM1 rather than strict UFP	Type	field, coke-oven workplace
Role in this study	96.85% of number and 67.01% of surface area were attributable to particles ≤1 µm in the surveyed area	Does not support	Calling PM1 ultrafine particles or generalising from one battery to the entire coke industry

D13 · Ribeiro et al. *Identification of nanominerals and nanoparticles in burning coal waste piles from Portugal* (2010). <https://pubmed.ncbi.nlm.nih.gov/20855106/>

Size / object	nanominerals/nanoparticles in material from a burning waste pile	Type	materials science
Role in this study	Directly demonstrates carbon nanoparticles, agglomerates and mineral phases in burning coal waste	Does not support	Establishing airborne PNC, transport distance or resident dose

D14 · Tan et al. *Experimental study on emission characteristics of soot particles during spontaneous combustion of coal* (2026). <https://doi.org/10.1016/j.fuel.2025.136139>

Size / object	laboratory particles from self-heating/combustion	Type	laboratory
Role in this study	Temperature dependence of soot-particle release and composition during spontaneous combustion	Does not support	Equating a laboratory reactor with an open waste heap or calculating population risk

4. Oil, gas and transport as comparators

001 · González & Rodríguez. *Refinery, ships, traffic*. <https://www.sciencedirect.com/science/article/abs/pii/S0169809512002633>

Size / object	>2.5 nm; PNC	Type	field
Role in this study	UFP episodes from a refinery and ships; distinction from PM2.5	Does not support	Universal concentrations for all refineries

002 · <i>Climate-relevant properties... oil and natural gas combustion.</i> https://www.sciencedirect.com/science/article/abs/pii/S1352231006000410			
Size / object	size distribution	Type	primary, one boiler
Role in this study	Gas particle mass below detection does not mean zero particle number; oil produces a UFP peak	Does not support	A general conclusion about every gas-fired installation
003 · Natural-gas turbine PM2.5/UFP. https://www.sciencedirect.com/science/article/pii/S1352231015305550			
Size / object	UFP/PM2.5	Type	primary, 100 MW
Role in this study	High nanoparticle number in the exhaust of a modern turbine	Does not support	Comparing with coal without identical methods
004 · Natural gas/biogas/biomethane UFP. https://pubs.acs.org/doi/10.1021/acs.est.8b04170			
Size / object	UFP	Type	primary
Role in this study	Semi-volatile and non-volatile fractions, secondary aerosol, role of S/siloxanes	Does not support	Saying that gas “does not generate particles”
005 · Singapore petroleum-industry sulphur and UFP. https://aaqr.org/articles/aaqr-22-07-0a-0265			
Size / object	10–190 nm	Type	field
Role in this study	Association of industrial sulphur with PNC and new particle formation	Does not support	Attributing every UFP to petrochemicals
006 · Heavy residual oil markers / source-apportionment review. https://pmc.ncbi.nlm.nih.gov/articles/PMC7456793/			
Size / object	generally PM2.5/PM1	Type	review
Role in this study	V and Ni as residual-oil markers within a source profile	Does not support	Treating V or Ni as unique proof
007 · Kleeman et al. fuel and lubricating oil. https://pubmed.ncbi.nlm.nih.gov/18350902/			
Size / object	size-resolved PM	Type	primary
Role in this study	Contributions of fuel and lubricating oil; hopanes/steranes	Does not support	Treating hopanes/steranes as unique to diesel

008 · Riddle et al. roadside source apportionment. <https://pubmed.ncbi.nlm.nih.gov/18800533/>

Size / object	PM0.1 and other fractions	Type	primary
Role in this study	Direct source apportionment of roadside PM0.1	Does not support	Applying the shares to a mine or a different vehicle fleet

009 · IARC Monograph 105: Diesel and gasoline engine exhausts. <https://publications.iarc.who.int/129>

Size / object	complex exposure	Type	official assessment
Role in this study	Diesel exhaust is carcinogenic to humans	Does not support	Using this assessment to label every individual UFP component carcinogenic

5. Source identification and chemical fingerprints

S01 · Hopke et al. *Global review of recent source apportionments*. <https://pmc.ncbi.nlm.nih.gov/articles/PMC7456793/>

Fraction	mainly PM2.5	Type	review
Role in this study	PMF/CMB, local specificity of profiles, combinations of markers	Does not support	Applying a PM2.5 profile directly to UFP

S02 · *Source Apportionment: Findings from the U.S. Supersites Program*. <https://www.tandfonline.com/doi/pdf/10.3155/1047-3289.58.2.265>

Fraction	PM2.5	Type	review
Role in this study	Organic markers, CMB/PMF and uncertainties	Does not support	Calling picene an absolutely specific marker

S03 · Szidat et al. review of radiocarbon apportionment. <https://pubmed.ncbi.nlm.nih.gov/24136253/>

Fraction	carbonaceous PM	Type	review
Role in this study	Separation of fossil and non-fossil carbon using ¹⁴ C	Does not support	Distinguishing coal, oil and gas by radiocarbon alone

S04 · 2026 PM1 marker + PMF study. <https://pubmed.ncbi.nlm.nih.gov/42323424/>

Fraction	PM1	Type	primary
Role in this study	Picene as part of a locally calibrated diagnostic system	Does not support	A universal threshold for other regions or UFP

S05 · Beijing 14C + organic markers. <https://boris-portal.unibe.ch/bitstreams/399da76e-a62a-4935-b44c-ab0002fdc208/download>

Fraction	carbonaceous PM	Type	primary
Role in this study	Combination of 14C, picene and hopanes for separating sources	Does not support	Drawing a conclusion from picene alone

S06 · PAH diagnostic ratios / coke-profile study. <https://www.sciencedirect.com/science/article/abs/pii/S0016236114012782>

Fraction	TSP	Type	primary
Role in this study	Possibilities and limitations of PAH profile comparison	Does not support	Strict unique identification without considering alternative sources

6. General UFP epidemiology and medical mechanisms

H01 · Ohlwein et al. (2019). <https://pubmed.ncbi.nlm.nih.gov/30790006/>

Design / fraction	85 UFP studies	Type	systematic review
Role in this study	Short-term inflammatory and cardiovascular changes are the most consistent; other outcomes are inconclusive or insufficient	Does not support	Declaring every listed disease proven specifically for UFP

H02 · Long-term UFP systematic review/meta-analysis (2026). <https://pubmed.ncbi.nlm.nih.gov/41605538/>

Design / fraction	85 studies through December 2024	Type	systematic review/meta-analysis
Role in this study	Current assessment of long-term evidence; overall independent evidence rated low/inadequate	Does not support	Extracting one pooled HR without co-pollutant caveats

H03 · Short-term UFP mortality review (2025/26). https://pubmed.ncbi.nlm.nih.gov/40939674/			
Design / fraction	21 studies	Type	systematic review
Role in this study	Finds no clear association between short-term PNC and all-cause/CV/respiratory mortality	Does not support	Claiming a proven UFP effect on mortality
H04 · Frampton et al. controlled carbon UFP. https://pubmed.ncbi.nlm.nih.gov/15768531/			
Design / fraction	humans, 10–25 µg/m ³ UFP	Type	controlled
Role in this study	Subtle changes in HRV/repolarisation; absence of several expected inflammatory effects	Does not support	Extrapolating to chronic coal exposure
H05 · UFP in type 2 diabetes. https://pubmed.ncbi.nlm.nih.gov/20822968/			
Design / fraction	humans, 32 nm EC UFP	Type	randomised crossover
Role in this study	Transient platelet activation/possible endothelial activation	Does not support	Proving that UFP cause type 2 diabetes
H06 · Miller et al. inhaled gold nanoparticles. https://pubmed.ncbi.nlm.nih.gov/28443337/			
Design / fraction	humans + mice, 5/30 nm Au	Type	tracer experiment
Role in this study	Direct evidence that some inhaled nanoparticles enter the blood; size dependence	Does not support	Assuming the same translocation fraction for ambient coal UFP
H07 · Oberdörster et al. olfactory route. https://pubmed.ncbi.nlm.nih.gov/15204759/			
Design / fraction	rats, 36 nm 13C	Type	animal model
Role in this study	Possible olfactory pathway to the brain	Does not support	Claiming an established quantitative pathway in humans
H08 · Placental translocation systematic review. https://pubmed.ncbi.nlm.nih.gov/33138843/			
Design / fraction	73 studies, few human	Type	systematic review
Role in this study	Biological possibility and substantial limitations of human evidence	Does not support	Claiming proven transfer of every UFP to the fetus

H09 · Black carbon on the fetal side of the placenta. https://pubmed.ncbi.nlm.nih.gov/31530803/			
Design / fraction	human placenta, BC	Type	observational/analytical
Role in this study	BC particles detected on the fetal side; association with maternal exposure	Does not support	Proving a coal source or clinical outcome
H10 · Air-pollution nanoparticles in placental cells. https://pubmed.ncbi.nlm.nih.gov/33181987/			
Design / fraction	15 placentas	Type	analytical
Role in this study	Nanoaggregates and metallic components in placental macrophages	Does not support	Claiming direct entry into the fetus
H11 · Airport UFP controlled exposure. https://pubmed.ncbi.nlm.nih.gov/32402984/			
Design / fraction	21 people, 5 h	Type	controlled field study
Role in this study	Short-term changes in lung function/QTc in a specific exposure	Does not support	Applying the effect to coal UFP
H12 · UFP and endothelial NO. https://pubmed.ncbi.nlm.nih.gov/23751346/			
Design / fraction	cells; UFP <200 nm	Type	mechanistic
Role in this study	Oxidative stress/endothelial dysfunction as a mechanism	Does not support	Calling it clinical proof of myocardial infarction
H13 · UFP dementia, ACT cohort. https://pubmed.ncbi.nlm.nih.gov/38185046/			
Design / fraction	4,283 people	Type	prospective cohort
Role in this study	Null result after multi-pollutant adjustment — an important boundary	Does not support	Claiming an unequivocal association between UFP and dementia
H14 · U.S. Medicare UFP dementia study. https://pubmed.ncbi.nlm.nih.gov/40079183/			
Design / fraction	approximately 20–21 million, UFP model	Type	large cohort
Role in this study	Positive association; contrast with H13	Does not support	Proving causality or a coal source

H15 · PM2.5/dementia Burden of Proof. https://pubmed.ncbi.nlm.nih.gov/40119171/			
Design / fraction	PM2.5	Type	systematic review
Role in this study	Indirect evidence for general PM2.5; must be explicitly labelled as analogy	Does not support	Attributing the finding to UFP
H16 · Yuan L. et al. <i>Maternal fine particulate matter (PM2.5) exposure and adverse birth outcomes: an updated systematic review based on cohort studies</i> (2019). https://pubmed.ncbi.nlm.nih.gov/30891704/			
Design / fraction	PM2.5; 42 cohort studies	Type	systematic review
Role in this study	Evidence on birth weight, low birth weight, preterm birth and small for gestational age for the mass-based PM2.5 fraction	Does not support	Calling PM2.5 evidence direct evidence for coal-related UFP
H17 · Lavigne et al. <i>Spatiotemporal Variations in Ambient Ultrafine Particles and the Incidence of Childhood Asthma</i> (2019). https://pubmed.ncbi.nlm.nih.gov/30785782/			
Design / fraction	UFP model; 160,641 births	Type	cohort
Role in this study	Positive association of second-trimester exposure with childhood asthma after adjustment for PM2.5 and NO ₂	Does not support	Applying to a coal source or treating all pregnancy windows as equivalent
H18 · Wright et al. <i>Prenatal Ambient Ultrafine Particle Exposure and Childhood Asthma</i> (2021). https://pubmed.ncbi.nlm.nih.gov/34018915/			
Design / fraction	UFP model; 376 mother–child pairs	Type	small cohort
Role in this study	Positive association and possible windows of susceptibility	Does not support	Ignoring the small sample, wide confidence intervals and modelled exposure
H19 · Yu et al. <i>Ambient ultrafine particles and asthma onset until age 20: PIAMA</i> (2022). https://pubmed.ncbi.nlm.nih.gov/35777436/			
Design / fraction	UFP model; 3,687 participants	Type	birth cohort
Role in this study	Single-pollutant association weakened after adjustment for NO ₂ and PM2.5 absorbance; an important negative boundary for an independent effect	Does not support	Claiming that UFP independently cause asthma
H20 · Copenhagen child asthma admissions (2025). https://pubmed.ncbi.nlm.nih.gov/40274412/			
Design / fraction	monitored UFP; 15,903 admissions	Type	case-crossover
Role in this study	Short-term positive association with asthma hospital admissions	Does not support	Establishing long-term causality or a coal source

H21 · *Ultrafine particle deposition in subjects with asthma (2004).* <https://pubmed.ncbi.nlm.nih.gov/15175176/>

Design / fraction	23 nm carbon; 16 adults	Type	controlled dosimetry
Role in this study	Measured deposition fraction was higher in adults with asthma, particularly during exercise and for the smallest particles	Does not support	Applying exact fractions to children, different compositions or ordinary ambient exposure

7. Specific coal-related medical cases

K01 · *Xuanwei indoor smoky-coal constituents and lung cancer.* <https://pmc.ncbi.nlm.nih.gov/articles/PMC6792381/>

Exposure	household smoke, chemical constituents	Type	case-control
Role in this study	Association of lung-cancer risk with a PAH cluster/NO ₂ among never-smoking women	Does not support	Claiming that the effect was caused specifically by UFP

K02 · *IARC Monographs: household coal combustion.* <https://publications.iarc.who.int/101>

Exposure	household coal combustion	Type	official assessment
Role in this study	Carcinogenicity of the specific exposure	Does not support	Applying it to every combustion method and to UFP separately

K03 · *Guizhou endemic arsenic poisoning.* <https://pmc.ncbi.nlm.nih.gov/articles/PMC1240722/>

Exposure	coal + contaminated food/air	Type	field
Role in this study	Demonstrates multiple exposure routes; food dominated	Does not support	Presenting it as a purely inhalational UFP case

K04 · *Guizhou fluorosis review.* <https://pubmed.ncbi.nlm.nih.gov/34625836/>

Exposure	household coal/clay, food contamination	Type	review
Role in this study	Endemic fluorosis and the food-drying pathway	Does not support	Presenting it as UFP epidemiology

K05 · **Childhood skeletal fluorosis.** <https://pmc.ncbi.nlm.nih.gov/articles/PMC2778178/>

Exposure	household coal, food	Type	cross-sectional
Role in this study	Clinical case of chronic mixed exposure	Does not support	Causation specifically by particle inhalation

K06 · **IARC coal dust Vol. 68.** <https://publications.iarc.fr/86>

Exposure	occupational coal dust	Type	official 1997 assessment
Role in this study	Coal dust was Group 3 at the time of assessment; silica separately Group 1	Does not support	Calling coal dust IARC Group 1 without a new monograph

K07 · **IARC priorities 2025–2029.** https://monographs.iarc.who.int/wp-content/uploads/2024/11/AGP_Report_2025-2029.pdf

Exposure	coal dust	Type	advisory report
Role in this study	New evidence justifies priority re-evaluation	Does not support	Presenting the recommendation as an already completed classification

K08 · **NIOSH coal-miner lung-disease bulletin.** <https://www.cdc.gov/niosh/bulletin/2023/mining-lung-disease.html>

Exposure	mixed occupational dust	Type	official
Role in this study	Pneumoconiosis, PMF, COPD and loss of lung function	Does not support	Attributing the effects to UFP or one component

K09 · **DEMS cohort.** <https://pubmed.ncbi.nlm.nih.gov/22393207/>

Exposure	diesel in non-coal mines	Type	cohort
Role in this study	Occupational diesel exhaust and lung cancer	Does not support	Calling it a coal cohort or UFP-specific study

K10 · **DEMS II COPD.** <https://pubmed.ncbi.nlm.nih.gov/38422874/>

Exposure	diesel + respirable dust	Type	cohort
Role in this study	COPD mortality with separate exposure assessments	Does not support	A direct conclusion about residents of mining towns

K11 · **Coke-oven workers meta-analysis (2026).** <https://pubmed.ncbi.nlm.nih.gov/42050638/>

Exposure	COE/PAH/PM2.5/CTPV	Type	systematic review
Role in this study	Occupational respiratory disease/cancer	Does not support	A UFP-specific effect

K12 · **Amster. *Public health impact of coal-fired power plants: a critical systematic review* (2019).** <https://pubmed.ncbi.nlm.nih.gov/31617747/>

Exposure	pollutant mixtures/PM, not UFP	Type	systematic review of 40 studies
Role in this study	Supports adverse population effects of power-station emissions, while noting heterogeneous exposure and confounding	Does not support	Attributing the effect to UFP or one station without source-specific exposure assessment

K13 · ***Exposure to air pollution from coal-fired power plants and impacts on human health: a scoping review* (2025).** <https://pubmed.ncbi.nlm.nih.gov/40042098/>

Exposure	proximity/pollutant mixtures	Type	scoping review of 56 studies
Role in this study	Organises respiratory, perinatal and childhood outcomes near power stations and effects of closures/controls	Does not support	Treating proximity as an exact dose or isolating UFP

K14 · **Henneman et al. *Mortality risk from United States coal electricity generation* (2023).** <https://pubmed.ncbi.nlm.nih.gov/37995235/>

Exposure	coal PM2.5 estimated with an SO ₂ model	Type	Medicare cohort, 650 million person-years
Role in this study	Direct source-specific evidence on secondary coal PM2.5 and mortality	Does not support	Calling the finding UFP, primary ash or an individual risk without the model

K15 · **Costantino et al. *Occupationally related cancer risk among coke oven workers: 30 years of follow-up* (1995).** <https://pubmed.ncbi.nlm.nih.gov/7640988/>

Exposure	complex coke-oven emissions	Type	cohort of 15,818 workers
Role in this study	Long-term follow-up of occupational exposure and respiratory cancer	Does not support	Isolating UFP as the causal agent

8. Protection and home filtration

P01 · EPA *Guide to Air Cleaners in the Home*. <https://www.epa.gov/indoor-air-quality-iaq/guide-air-cleaners-home>

Object	household filters	Type	official guidance
Role	Selecting CADR/HEPA; limitations; ozone-generating devices	Does not support	Promising that one HEPA unit will remove every pollutant or gas

P02 · Lowther et al. HEPA and UFP. <https://pubmed.ncbi.nlm.nih.gov/32739515/>

Object	18–514 nm	Type	chamber study
Role	Size-resolved efficiency; minimum near 200–250 nm	Does not support	Applying laboratory efficiency to every home

P03 · Systematic review of real-world filtration. <https://pubmed.ncbi.nlm.nih.gov/34374125/>

Object	predominantly PM2.5	Type	systematic review
Role	Filtration reduces indoor PM, but confidence is low/heterogeneous	Does not support	Guaranteeing clinical benefit or a UFP effect

P04 · Sultan et al. portable air cleaners. <https://hero.epa.gov/reference/1729721/>

Object	UFP	Type	chamber study
Role	HEPA/ESP/electret devices reduce UFP; technologies differ	Does not support	Recommending ionisers/ozone generators as a safe substitute