Don Smith statement

I am currently an Emeritus Distinguished Professor of Microbiology and Environmental Toxicology at the University of California, Santa Cruz. I served on the faculty at UCSC for over 30 years, recently retiring as a Distinguished Professor in June, 2025. My research career throughout this time has focused on the environmental and human health associated with exposure to potentially toxic metals, including exposure assessment and adverse health outcomes in humans, wildlife, and laboratory animal models. I have published over 150 peer-reviewed research articles and several book chapters on this topic, received multiple professional awards recongnizing my research accomplishments, and have served in various professional leadership positions, including President of the Society of Toxicology Metals Specialty Section.

My professional opinion is that large industrial-scale lithium battery energy storage facilities may pose unacceptable risks for environmental and human exposure to toxic levels of metals, including cobalt, nickel, manganese, and aluminum in the event of thermal runaway and facility fire. This opinion is informed directly from the catastrophic fire at the Vistra battery storage facility fire in Moss Landing, California in January 2025.

Specifically, energy storage facilities using lithium - NCA or NMC battery technologies pose significant potential environmental and human health risks due to their potential susceptibility to thermal runaway and fire. Combustion of Li-NCA or Li-NMC batteries generates plumes/emissions of small (sub-PM_{10}) particulates highly enriched in nickel, cobalt, and aluminum (or manganese). These metal-enriched particulates may be distributed over large areas (10's of square miles) or more, depending on weather conditions. These airborne metal-enriched particulates will serve as a potential source of elevated inhalation exposure to these metal-enriched particulates, as well as contribute to deposition and increased loading of outdoor and indoor dust enriched in these metals. Moreover, outdoor deposited dust particles can be readily resuspended into airborne dust, posing ongoing repeated inhalation exposure risk. Outdoor deposited dust also serves as the primary source of indoor residential dust, and both outdoor and indoor dust may pose a particularly important source of metal exposure to infants and young children, who have higher respiratory rates compared to adults, and who also commonly exhibit hand to mouth activity, increasing risk of oral ingestion of metals associated with dust particulates.

Regarding the Vistra battery facility fire, I have reviewed various datasets that were shared with me, including:

1) A file labeled "Aiello__ElkhornSLough_2023-2025_XRF (002)", which contains surface soil XRF data along the Elkhorn Slough Reserve transect studies by Dr. Aiello, measured in 2023 and again in 2025 (post-fire). All three transects (3, 8, 12) show clear 3 – 5-fold increases in cobalt, nickel, manganese, and possibly for copper as well from 2023 to post-fire 2025, but no similar increases in iron. The results also show a significant correlation between cobalt and nickel for the 2025

data, but not the 2023 data, consistent with a common source for the elevated cobalt and nickel levels in the 2025 data. Scanning electron microscopy analyses shows examples of a nanoparticle enriched in cobalt, nickel, and manganese, again consistent with a common combustion source.

2) Two files labeled "Ghost Wipe Sampling_2025_Jan_With Stardard Limits" and "NAML Ghost Wipe Sampling_anonymized (2)" that contain surface dust wipe metal loadings from various locations in the vicinity of the Vistra facility and beyond. The data show clear evidence of elevated cobalt, nickel, and manganese surface loadings at locations within 6 to 7 miles of the Vistra facility, and a few cases of elevated surface dust metal levels as far away as ~13 miles, compared to sites distant from facility.
3) A file labeled "NAML Draft Clearance Levels sample data table for all metals 3-9-25" that contains summary tables of measured dust wipe metal levels vs recommended clearance levels, showing several samples with elevated dust cobalt and nickel levels exceeding clearance thresholds.
4) A document labeled "SC Agricultural Commissioner Plant Results". The file contains metal levels in strawberries and various vegetable greens collected January 30 or Feburary 5, 2025 (collection locations unclear). The data show cases of elevated produce levels for cobalt, manganese, and nickel that exceed recommended intake levels for humans (manganese and cobalt) or the Tolerable Upper Intake Level (nickel), given assumptions of daily consumption amounts.
5) A document labeled "Evaluation of Heavy Metals in Agricultural Fields following the Moss Landing Battery Fire" (dated March 26, 2025). The document contains data from soil samples collected February 10 and 11, and include soil surface (0-1") and at depth (3-4", or 0-12") samples (specific sampling locations unclear). Samples were analyzed at UC Davis via total digestion and ICP-AES analyses. Sites 1-6 were within 2 miles of the Vistra battery facility, and site 7 was ~17 mi from facility. All metal levels (cobalt, nickel, manganese, copper) appeared within background levels for all sites and soil depths.
6) Data from OEHAA (Part of CalEPA group, dated Feb 14, 2025) showing soil and surface water metal levels in samples collected from DTSC. Data were from nine collection stations, and all metal levels in soil and water did not appear to be significantly above background levels, though the sampling approach (bulk soil) and limited number of sampling locations were likely inadequate to draw meaningful conclusions.
7) The Moss Landing Fire Update: Soil Screening Data Summary, County of Monterey Health Department, Environmental Health Bureau (dated Feb 13, 2025). This report presents DTSC analyses of surface soil samples collected January 24, 2025 by XRF from eight sites. Site 8 (closest to the plant) shows two (out of five) subsamples with very elevated cobalt, manganese, nickel, and copper (no other metals were included). Metal levels at all other sites appear to be within background levels.
8) The CTEH Preliminary Report (dated Jan 23, 2025) providing real time air monitoring data for CO, HF, HCN, HCl, PM2.5. Gasses below DLs, and PM2.5 data in avg and range only, not individual values. The highest side-pak PM2.5 value (0.088 mg/m3) is elevated, but it is not clear whether the locations of the air monitors

sufficiently captured the fire plume. As such, the data appear inadequate to reach meaningful conclusions.

9) A report labeled "HunterBrook - BREAKING: NEW DATA INDICATES ELEVATED HEAVYMETAL AFTER VISTRA FIRE". The report contains summary figures of geolocated elevated nickel, manganese, and cobalt levels in surface (soil, dust? Not clear) samples in the vicinity of the Vistra battery facility, but it is not clear how the data were generated.

Conclusions regarding data from the Vistra facility fire:
Altogether, these data support cause for concern of elevated human exposures to toxic cobalt, nickel, and manganese, with potential increased risk for adverse health effects. However, while in some cases alarming, these data are insufficient to establish actual exposure risk to humans in the communities surrounding the Vistra facility, and they are insufficient to conclude that there are no health risks from the Vistra facility fire. This is because of the limited scope of environmental sampling by local and state agencies, and, apparently, the complete absence of any human exposure assessment. The seeming absence of a comprehensive exposure assessment is very concerning, as residents are not getting the answers they deserve, and at the same time are being told that there is not a problem, when in fact the existing data that I have reviewed are insufficient to reach a conclusion on exposure risk.

To assess actual human exposures and risk for potential adverse health effects, a comprehensive exposure assessment study should (or should have) been conducted, involving the collection and analyses of exposure biomarkers (e.g., blood, hair, etc.) along with comprehensive sampling of residential indoor and outdoor dust samples using a cyclone sampler or equivalent that would allow for quantification of metal concentrations in collected dust as well as surface dust loadings in areas impacted by the fire plume, in addition to unimpacted control areas and subjects for appropriate comparisons. Such an assessment is needed to determine whether elevated exposures occurred during the peak of the fire event, and are possibly continuing to occur due to resuspension and inhalation/ingestion of deposited dusts from the fire.

In the absence of human exposure assessment, I have recommended that inhabitants in the vicinity of the Vistra facility that are concerned about potential exposures follow heightened residential hygiene practices, including a thorough cleaning of dust on indoor and outdoor surfaces, perferrably using a HEPA-certified vacuum and wet wipes while wearing appropriate face covering to reduce inhalation exposure risk while cleaning. The potential risks of elevated exposure to children, who are typically the most susceptible segment of the population to the adverse outcomes from elevated metal exposure, should also be carefully considered.

Background on the potential toxicity of cobalt, nickel, manganese, and aluminum: Below I summarize briefly the potential toxicity of cobalt, nickel, manganese, and aluminum that could result from elevated exposure. These summaries are based on publically-available materials from the Agency for Toxic Substances Disease Registry (ATSDR) and other health summaries.

Cobalt

While small amounts of cobalt, most notably as cobalamin (vitamin B12), is essential for some biological functions, elevated levels of cobalt exposure are known to be toxic. Sub-micron particles of cobalt, such as would result from a Li ion battery storage facility fire, would be inhaled and absorbed through the respiratory tract, whereas larger particles would be deposited in the respiratory tract and removed by mucociliary clearance and swallowed. Inhaled cobalt absorption into the body ranges from 52 to 78%.

Cobalt can participate in redox cycling, leading to an excess of free radicals leading to tissue damage. This is the likely mechanism resulting in pulmonary toxicity. Elevated inorganic cobalt exposure is known to inhibit various enzymes responsible for protein synthesis and RNA synthesis. This is the likely underlying pathophysiology resulting in cardiomyopathy and other adverse health outcomes. For example, $CoCl_2$ inhibits tyrosine iodinase, which leads to decreased thyroid hormone (T3, T4) and hypothyroidism. Also, inorganic cobalt is well-known to promote erythropoiesis (increased production of red blood cells) Co^{2+} may bind to transferrin resulting in impaired oxygen transport to renal cells through induction of hypoxic inducible factor-1 alpha and likely increased iron availability for erythropoiesis. This leads to the development of reticulocytosis and polycythemia.The average U.S. soil cobalt concentration in 2022 was 7.34 ug/g (parts per million).

Nickel

Nickel does not play any essential role in biological function. Toxicity from nickel can occur through various routes of exposure, including inhalation, ingestion, and dermal absorption. Chronic nickel toxicity can manifest as sinusitis, asthma, and dermatitis. Prolonged exposure to nickel has also been linked to lung and nasal cancer development. Dermal nickel exposure is a well-known to produce contact dermatitis. Once sensitized, the hypersensitivity to nickel can persist indefinitely.

The symptoms of nickel toxicity can occur immediately after exposure or be delayed. Inhalation can cause respiratory, neurologic, and hepatic dysfunction. Pulmonary findings include pneumonitis and acute lung injury. Other findings include cerebraledema, myocarditis, altered mental status, and seizures. Multiple mechanisms have been investigated to explain the toxic effects of nickel on the human body. Inhalational and absorbed nickel appears to form a complex with peptide and protein thiols. A reaction occurs between nickel-thiol complexes and oxygen, producing free radicals, which can create reactive oxygen species, ultimately can lead to intracellular hypoxia. In addition, through epigenetic alterations, nickel can substitute for iron, causing alterations in DNA and histone demethylation.

Manganese

Relatively small amounts of manganese are essential for some biological functions, but elevated exposures are well-known to cause toxicity. In particular, elevated levels of manganese exposure, either through inhalation or ingestions, are well-known to cause oxidative stress, cell and tissue damage, and increase the risk of neurodevelopmental and neurodegenerative disorders. Young children are particularly susceptible to elevated manganese exposure because they absorb and retain greater proportions of ingested or inhaled manganese compared to adults, and because of their rapidly developing central nervous system.

Chronic environmental manganese exposure is associated with reduced IQ, fine motor function disturbances, and increased risk for behavioral alterations, including inattention and hyperactivity, and increased risk of ADHD diagnosis. There are few well-accepted biomarkers of elevated environmental manganese exposure, though some studies have reported that elevated levels of manganese in hair are associated with adverse health effects in children and adolescents.

Aluminum

Aluminum is ubiquitous in the environment, and adverse health effects from environmental (versus occupational) aluminum exposure are uncommon. Per ATSDR (2008), aluminum is poorly absorbed following either oral or inhalation exposure and is essentially not absorbed dermally. Aluminum binds to various types of molecules in the blood and distributes to every organ, with highest concentrations ultimately found in bone and lung tissues.

While aluminum can be measured in the blood, bone, urine, and feces, there are insufficient data to relate aluminum exposure levels with blood or urine levels. The most sensitive target of aluminum toxicity is the nervous system. Impaired performance on neurobehavioral tests of motor function, sensory function, and cognitive function have been observed in animal models. Respiratory effects, such as impaired lung function and fibrosis have been observed in occupationally exposed aluminum workers. Children who are exposed to high levels of aluminum exhibit symptoms similar to those seen in adults, including neurological effects and skeletal effects.