March 19th, 2025

Potential Medical Consequences of Lithium-ion Electric Battery Fire

Literature review

Methodology:

For the purpose of this review the National Library of Medicine was searched with the following **Keywords**:

Battery electric vehicles; Lithium-ion battery; Lithium-ion battery explosions; Lithium-ion battery fire; Car fire; Heavy metal injury; Cobalt, Nickel, Chromium; Trace elements; Toxic fluoride gas emissions; Nanoparticles; Gas and fume inhalation; Toxic metal fumes (aluminum, cadmium, chromium, nickel, iron); Fire smoke; Lithium-Iron-Phosphate Battery thermal runaway

More than 200 citations of Lithium-ion battery thermal runaway were identified, 65 citations of lithium battery fire.

Thousands of citations were identified reporting on heavy metals and toxic metal fumes exposure.

Introduction:

Lithium-ion-Nickel-Manganese-Cobalt (NMC) and Lithium-Iron-Phosphate (LFP) batteries are considered to be one of the most important energy storage technologies. As he energy density of batteries increases, battery safety becomes even more critical if the energy is released unintentionally. In the last few years, the medical community has encountered increasing episodes of burn injuries secondary to Lithium-ion battery explosion. Explosions occur through a process known as a "thermal runaway." This process occurs when the battery overheats and the internal battery temperature increases dangerously high, to the point of inner fire and explosion. Overcharge, puncture, external heat, short circuit, amongst others, are conditions that cause a "thermal runaway". Accidents related to fires and explosions of Lithium-ion batteries occur frequently worldwide. Some have caused serious threats to human life and health and have led to numerous product recalls by manufacturers. These incidents are reminders that safety is a prerequisite for batteries, and serious issues need to be resolved before the future application of high-energy battery systems. Lithium-ion battery fires generate intense heat and considerable amounts of gas and smoke. The emission of toxic gases, nanoparticles, heavy metal particulates can be a larger threat than heat, the knowledge of such emissions is limited. Additionally, the long-term medical effects of Lithium-ion batteries fire have not been studied. Most of the literature citations report on the short-term medical effects of Lithium-ion batteries in small devices such as e-cigarette, electric vehicles and electric motorcycles/scooters. Data on fire in a large electric batteries storage system is scant and long-term medical adverse effects are not available yet. Recently, a major fire erupted in a battery storage system located at Moss-Landing California. Short-term medical adverse effects due to the fire were recorded but long-term effects are still unknown.

Results:
Hydrofluric Acid

The flouride ions in hydrochloric acid are strong scavengers of bivalent cations, such as calcium and magnesium, and can slowly penetrate skin tissue and when inhaled respiratory tissue, resulting in necrosis.

Use of Lithium-ion batteries has raised safety issues owing to chemical leakage. Hydrofluoric acid
(Hydrogen Fluoride, HF) which potentially might leak from lithium-ion batteries may be extremely toxic. The inhalation no-observed-adverse-effect-level
(NOAEL) for HF is 0.75 mg/kg/d. When a lithium-ion battery explodes in a limited space, HF emissions amount to 10-100 ppm calculated to be 81.8 mg/m^3, and the average daily dose (ADD) was 19.5
mg/kg/d.

The potential effects of HF exposure may result in skin burns, inhalation injury to the respiratory tract and to the eyes.

Heavy metals exposure

Data on heavy metal exposure of Lithium-ion batteries fire is not yet available. However, environmental and occupational data on the potential medical consequences of such exposure is very much available.

Most of the medical consequences take time to present itself. Diseases such as Interstitial Fibrosis, lung cancer and bronchiectasis, bronchiolitis obliterans-organizing pneumonia may take years to manifest itself and /or to be diagnosed (similar to smoking induced lung disease).

Below is a table of potential **pulmonary** pathologic consequences of exposure to several agents:

Metals	**Pulmonary Pathologic Reactions**
Aluminum	Interstitial pneumonia, Interstitial fibrosis, Pulmonary alveolar proteinosis
Cadmium	Diffuse alveolar damage, Lung cancer
Chromium	Lung cancer, Pulmonary fibrosis, intestinal cancer

Cobalt	Lung Cancer, Fibrosing alveolitis, Interstitial fibrosis
Iron	Nodular fibrosis
Nickel	Lung cancer; lung inflammation and fibrosis
Gases and Fumes	
Toxic metal fumes	
Aluminum, Cadmium, Chromium, Nickel	Tracheobronchitis, bronchiolitis, Diffuse alveolar damage, bronchiolitis obliterans
Fire smoke	Necrotizing tracheobronchitis, Diffuse alveolar damage, Bronchiolitis obliterans, Bronchiectasis

Above tablet modified from Murray and Nadel's 4th edition Textbook of respiratory medicine

Cobalt Inhalation

Chronic inhalation exposure to cobalt metal and cobalt sulfate has caused **lung cancer** in rats and mice, as well as systemic tumors in rats. Cobalt compounds are listed as probable or possible human carcinogens by some agencies, and there is a need for quantitative caner toxicity criteria. The U.S. Environmental Protection Agency

has derived a provisional inhalation unit risk (IUR) of 0.009 per µg/m^3 based on a chronic inhalation study of soluble cobalt sulfate heptahydrate; however, a recent 2-year cancer bioassay affords the opportunity to derive IURs specifically for cobalt metal. The mechanistic data supports that the carcinogenic mode of action (MOA) is likely to involve oxidative stress, and thus, **non-linear/threshold mechanisms**. Several additional analyses resulted in an IUR of 0.003 per µg/m^3 for cobalt metal, which is ~3-fold less potent than the provisional IUR.

Preclinical data (rodents) regarding long-term inhalation exposure to poorly soluble cobalt are available. Pulmonary effects of the poorly soluble tricobalt tetraoxide (C_3O_4) (dose ranging of 5, 20, 80 mg/m^3) in a 28-day inhalation exposure study revealed **lung tissue inflammation** at the mid-dose with increasing severity in the high-dose group and post-exposure persistency. Markers for cellular damage and cell proliferation were statistically significantly increased.

Cobalt exposure in the hard metal and bonded diamond tool industry is a well-established cause of **Interstitial Lung Disease** (ILD). The primary theories regarding **Interstitial Lung Disease** (ILD). The primary theories regarding the underlying mechanism of cobalt related ILD include an immunologic mechanism and an oxidant injury mechanism. Cobalt related ILD may present in subacute and chronic forms and often has associated upper respiratory symptoms. The finding of cannibalistic multinucleated giant cells in lung tissue samples is diagnostic. **Giant cell interstitial pneumonia** is the classic pathologic pattern, but cobalt related ILD may also present with pathologic findings of **Usual Interstitial Pneumonia**, **Desquamating Interstitial Pneumonia**, or **Hypersensitivity Pneumonia**.

Nickel Exposure

Nickel contact can cause a variety of side effects on human health, such as allergy, **cardiovascular** and kidnesy diseases, lung fibrosis, lung and nasal cancer. Although the molecular mechanisms of nickel-induced toxicity are not yet clear, mitochondrial dysfunctions and oxidative stress are thought to have a primary and crucial role in the toxicity of this metal. Recently, researchers, trying to characterize the capability of nickel to induce cancer, have found out that epigenetic alterations induced by nickel exposure can perturb the genome. Several research groups have demonstrated that exposure to nickel nanoparticles (Nano-Ni) results in severe and persistent lung inflammation and fibrosis. Nano-Ni caused extensive **pulmonary fibrosis** and **chronic inflammation** in mouse lungs. Potential inflammatory mechanisms to this injury were explored and documented.

Exposure of Nickel Sulfate $NiSO_4$ to human umbilical vein endothelial cells (HUVECs) suggest that $NiSO_4$ can inhibit cell growth, induce oxidative stress, and trigger subsequent inflammatory responses and apoptosis in HUVECs. These findings may explain the growing evidence that Nickel and its compounds have adverse effects on the human **cardiovascular system**.

Nickel has been identified as a human group I carcinogen; however, the underlying mechanisms governing Ni-induced carcinogenesis are still being elucidated. However, recent information demonstrates that Ni exposure mediates carcinogenesis and cancer progression. Exposure to nickel significantly increases mortalitiy from **lung**, **rectal** and **kidney cancers**.

Lung cancer associated with Nickel exposure is observed in people who were exposed to inhaled nickel compounds. Exposure to Nickel through other routes such as water, food and skin may lead to increased cancer risks in other organs. A systematic review and meta-analysis to evaluate the association between serum and hair nickel levels and breast cancer was published in recent years.

Nine studies determining the serum nickel levels and six studies evaluating the hair nickel levels were identified in a systematic literature search. Findings showed that high serum nickel levels were associated with breast cancer, and nickel exposure might be a risk factor for breast cancer.

Heavy Metal Exposure

Heavy metals are harmful environmental pollutants that have attracted widespread attention due to their health hazards to **human cardiovascular disease**. Heavy metals, including lead, cadmium, mercury, arsenic, and chromium, are found in various sources such as air, water, soil, food, and industrial products. Recent research strongly suggests a connection between cardiovascular disease and exposure to toxic heavy metals. Epidemiological, basic, and clinical studies have revealed that heavy metals can promote the production of reactive oxygen species, which can then exacerbate reactive oxygen species generation and induce inflammation, resulting in endothelial dysfunction, lipid metabolism distribution, disruption of ion homeostasis, and epigenetic changes. Over time, heavy metal exposure eventually results in an increased risk of hypertension, arrhythmia, and atherosclerosis.

Chromium Exposure

Public health concerns are centered on the presence of chromium (Cr) hexavalent that is classified by The International Agency for Research on Cancer as a known human carcinogen. Under certain conditions trivalent Cr can be oxidized in the soil to hexavalent Cr which has high environmental mobility. Epidemiological surveys of chromium compounds have shown strong associations between exposure to Cr hexavalent and mortality die to lung cancer, as well as positive associations with **cancers of the nose and nasal cavity**. Cr hexavalent exposure in the workplace remains a serious problem as a cause of lung cancer and cancers of nose and nasal cavity. Epidemiological evidence for Cr hexavalent ingestion is suggestive of elevated risks for **stomach cancers**. Exposure of animals to Cr hexavalent in drinking water induced tumors in the alimentary tract, with linear and supralinear responses in the mouse small intestine. The most abundant form of DNA damage induced by Cr hexavalent is Cr-DNA Adducts, which cause mutations and chromosomal breaks. Extensive formation of DNA Adducts has a high predictive value for carcinogenicity, the shape of tumor-dose responses in mice, and a biological signature of mutagenic carcinogens (multispecies, multisite, and trans-sex tumorigenic potency) strongly support the importance of the DNA-reactive mutagenic mechanisms in carcinogenic effects of Cr hexavalent.

Manganese Exposure

Exposure to manganese (Mn) causes clinical signs and symptoms resembling, but not identical to,

Parkinson's disease. Emerging data suggest that beyond traditionally recognized occupational manganism, Mn exposures and the ensuing toxicities occur in a variety of environmental settings, nutritional sources, contaminated foods, infant formulas, and water, soil, and air with natural or man-made contaminations. Upon fast absorption into the body via oral and inhalation exposures, Mn has a relatively short half-life in blood, yet fairly long half-lives in tissues. Recent data suggest Mn accumulates substantially in bone, with a half-life of about 8-9 years expected in human bones. At elevated levels, the essential element manganese (Mn) is neurotoxic and increasing evidence indicates that environmental Mn exposure early in life negatively affects neurodevelopment. Mn exposure early in life may cause toxic effects later in life. Studies investigated the correlation between blood Mn concentration and **sarcopenia** (loss of muscle mass) risk in the National Health and Nutrition Examination Survey (NHANES) from 2011 to 2018. A positive association between the $\log_{10}$ Mn concentration and the prevalence of sarcopenia in the logistic regression model. Moreover, **heavy metals** mixtures were positively correlated with the prevalence of sarcopenia, with Mn identified as the main contributor to this. Furthermore, inflammation mediated the relationship between Mn exposure and the prevalence of sarcopenia. These resluts revealed that excessive Mn exposure is a contributing factor for sarcopenia.

Lithium-Iron-Phosphate Batteries

Lithium iron phosphate (LFP/(LiFePO(4))) batteries contain metals, toxic electrolytes, organic chemicals and plastics that can lead to serious safety and environmental problems when they are improperly disposed of, during burning/external fire or damaged (e.g. crushed during an earthquake or accidently damaged). Numerous studies have identified the primary gases produced during battery Thermal Runaway as H_2, CO, CO_2, CH_4, C_2H_6, C_2H_4, C_3H_8, among others. The formation of Hydrogen Fluoride (HF) during fire (which is a toxic gas) is dependent on the state of charge of the batteries, the number of battery cells affected and the rate of energy release. From laboratory experiments it seems that the rate of HF formed during fire is higher with LFP in comparison with Lithium-ion Nickel-Manganese-Cobalt (NMC) battery. However, as other factors are involved (as mentioned above), it is difficult to draw firm conclusions. Nevertheless, the release of HF during LFP battery fire may be at least comparable to the release of HF during NMC battery during fire. For the toxic effects of HF please see above. Other gases such as carbon monoxide (CO) binds to hemoglobin forming carboxyhemoglobin preventing oxygen from reaching vital organs (brain, heart) and causing damage or death.

Although we could not find any publication of LFP battery iron release during fire or damage, but we did find documentation of iron dust and nanoparticles exposure which may result in lung nodular fibrosis (scarring), and when extensive may lead to pulmonary hypertension and abnormal gas exchange (see above table). Iron overload by ingestion has been documented in rural sub-Saharan African populations that have the custom of drinking a traditional fermented beverage with high iron content and in population drinking beer/alcohol from iron-cast vessels. Iron overload in these populations is a sequel to these drinking habits and may result in hepaticportal fibrosis and micronodular cirrhosis and eventually liver failure and/or liver cancer. Iron content post LFP explosion may contaminate drinking water sources and soil resulting over time with iron overload of the exposed population.

Summary

We conducted a review of published literature with key interest in the potential of toxic gases and heavy metals contamination of air, water and soil post Lithium-ion NMC and Lithium-Iron-Phosphate (LFP) Electric Battery Fire. Lithium-ion and lithium-ion-iron-phosphate battery storage technology of electric energy has its advantages but has its risks and concerns. The most significant concerns are based on the numerous incidents of battery fire due to thermal runaway. So far there is no ideal solution to how to prevent such fires and the medical consequences (not to mention the economic consequences) of such a fire.
The review included some of the fumes, gases, and heavy metals potentially discharged to the environment during such a fire. Obviously, this review is not a conclusive search of all potential environmental contamination. As we could not identify long-term observation of the population exposed to NMC and LFP battery fire we had to focus on other forms of exposure to several heavy metals, mainly occupational exposures. We would like to emphasize that exposure to several of the elements may lead to no or little acute medical symptoms. However, it is very possible (and one may say probable) that long-term consequences such as chronic lung diseases, cancer (i.e. lung, head and neck, gastrointestinal), Parkinson's like disease and neurodevelopment abnormalities of young children may affect the exposed population years after exposure.

Based on the literature review we recommend the following:

1. Pause all construction of electric battery storage systems based on Lithium-ion Nickel-Manganese-Cobalt and Lithium-Iron-Phosphate battery storage systems until further information regarding potential short and long-term health effects are investigated.

2. Develop state standards for construction and safety measures of energy storage systems.

3. Develop new energy storage technologies which have minimal risks to the environment and population.

4. Short and long-term medical follow up of population exposed to electric energy storage systems fire.

5. Build electric energy storage systems which are non-lithium ion or LFP based which are proven to be non-fire toxic prone and are away from populated areas.

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References may be provided upon request