

Occupational and Environmental Health in the Aluminum Industry

Key Points for Health Practitioners

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Aluminum, the most abundant metal in the earth's crust (making up approximately 8%), has a multitude of uses ranging from aerospace to construction through manufacturing to food and pharmaceutical applications. The production of primary aluminum begins with the mining of raw ore and is followed by the extraction of aluminum metal through a series of long established and vertically integrated industrial processes. In this issue of the Journal, these processes and their technologies—both contemporary and innovative—are fully described. The basic chemical process produces, for every 4 to 6 kg of bauxite, approximately 2 kg of alumina and 1 kg of aluminum. Other raw materials include carbon, aluminum fluoride, cryolite, and electrical energy. The chemical, physical, biological, psychosocial, and ergonomic hazards related to primary aluminum production are also characterized, in the context of both occupational and community health. Epidemiological analyses detailing known health risks are comprehensively summarized. Emerging issues of interest and study, such as environmental and community health in relation to aluminum production facilities, are also discussed. Public and consumer health considerations, focused on hypotheses about aluminum and neurocognitive impacts, are systematically reviewed and assessed.

BAUXITE MINING PROCESS AND ASSOCIATED OCCUPATIONAL HEALTH HAZARDS AND RISKS

Although a very small percentage (less than 1% globally) of aluminum is derived from ores such as nepheline and from alternative sources, such as fly ash from coal-fired power stations, bauxite mining represents, by far, the most common initial upstream step in the eventual production of aluminum metal. A brief historical perspective, process overview, and sustainable bauxite mining report are available via the International Aluminium Institute's (IAI's) Aluminium for Future Generations project.¹

Bauxite ore, named for the town of Les Baux, France, is one of the earliest sources of bauxite and contains alumina (Al₂O₃), the immediate precursor of aluminum (Al) in the production cascade. The overwhelming majority of known global bauxite reserves are found in the geographic band bounded by the Tropics of Cancer and Capricorn. Key deposits are found in West Africa, South America and the Caribbean, South and Southeast Asia, and Australia.

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Most bauxite is very near the surface and thus easily mined relative to other ores. Nevertheless, a number of physical, chemical, biological, ergonomic, and psychosocial hazards exist throughout the mining process, as described in the article by Donoghue et al.²

Physical hazards include noise, heat and humidity, ergonomics including vibration, naturally occurring radioactive material, and ultraviolet radiation. The potential for traumatic injury is a reality that compels operating locations to assure the proximate availability of skilled emergency medical response capability. Noise from sources such as earth-moving equipment, blasting, drilling, and crushing is ever present. Consequently, noise-induced hearing loss is a significant risk to be managed, and robust hearing conservation programs are essential. The climate of tropical mining locations, with its inherent high ambient heat and humidity, can lead to heat-related illnesses; thus, appropriate employee awareness and control strategies are necessary.

Operators of heavy equipment and machinery are subject to whole-body vibration, which can contribute to or exacerbate spinal disorders. Naturally occurring radioactive material is present in bauxite at very low levels and transfers to the solid residue stream during refining, being absent in the alumina end product. This latter hazard must be considered and monitored; however, occupational monitoring data from the bauxite mining and alumina refining sector indicate personal dose levels less than applicable public exposure limits; thus, it is unlikely to be of significant human health concern.³ Ultraviolet radiation exposure, logically more pronounced, given the concentration of bauxite mining activity in tropical zones, can contribute to the occurrence of both squamous and basal cell carcinoma; however, it is reassuring to note that prolonged occupational outdoor work does not seem to confer an increased risk for melanoma, as noted by Donoghue et al.² Generally accepted control measures include enclosed mobile equipment cabs, creative scheduling to avoid midday sun exposure, and proper protective clothing and sunscreen.

Chemical hazards are few, because bauxite per se is generally considered to be biologically inert. In the occupational hygiene context, it is best categorized as a nuisance dust, or particle not otherwise specified. Although Donoghue et al.² point out that there has been one reported case of mild pulmonary fibrosis in an individual exposed to bauxite crushing and transport over several decades more than 50 years ago, epidemiological studies of contemporary dust exposures in well-managed mining operations seem not to be associated with either clinically significant negative lung function impacts or pneumoconiosis. Trace quantities of beryllium and other metals are present in some bauxites; however, these have not been associated with adverse health impacts in those involved in the mining industry.

Additional hazards include biological, ergonomic, fatigue, and psychosocial factors. Communicable diseases like malaria and dengue fever, in some areas, must be anticipated and addressed through appropriate mosquito and other vector control efforts, chemoprophylaxis, early diagnosis and treatment, employee education, and travel medicine consultation. Ergonomic hazards are minimized, because mining has matured to a highly mechanized state. Fatigue, however, is an increasingly relevant concern owing to extended shifts and overtime, and the implementation of fatigue risk management programs is growing within the industry. Recent

concerns raised by the International Agency for Research on Cancer about shiftwork that involves circadian disruption are another area that deserves further consideration.⁴ Psychosocial factors—including alcohol and drug abuse—are compounded by isolation, social change, and the lack of health care and other usual social amenities in some mining settings and should also be considered and addressed.

ALUMINA REFINING PROCESS AND ASSOCIATED OCCUPATIONAL HEALTH HAZARDS AND RISKS

Alumina refining transforms bauxite ore to calcined alumina (Al_2O_3). This extraction method, known as the Bayer process, occurs through a progressive series of steps: crushing of the ore, digestion in a sodium hydroxide solution, clarification to remove solid waste materials, precipitation of solid hydrated alumina, and calcination to drive off water. A brief process overview and an alumina technology road map are available via the IAI's Aluminium for Future Generations project.⁵ Digestion requires large quantities of caustic soda, the signature chemical hazard linked to the refining process. Several other important physical, chemical, biological, ergonomic, and psychosocial hazards exist, which are also described in the article by Donoghue et al.²

Physical hazards include noise, heat and humidity, vibration, ergonomic, and ultraviolet radiation exposure. Minor traumatic injuries, particularly to hands and fingers, are not uncommon; however, the occurrence of major traumatic events is rare. The presence of adequately equipped on-site emergency response and medical personnel is therefore highly desirable. Noise is a ubiquitous hazard throughout aluminum refineries, and noise-induced hearing loss remains an unfortunate but still prevalent occupational illness for refinery workers. Aggressive hearing conservation programs are essential. Best-practice programs incorporate quantitative hearing protection fit-testing and emerging technologies that use personal noise dosimetry with real-time notification of daily exposure limit exceedance. Vibrating hand tools are frequently used within refineries, with hand-arm vibration syndrome occasionally manifesting in the workforce.

Chemical hazards include alumina and bauxite dusts, caustic soda, and diesel exhaust fumes. Donoghue et al² indicate that while Western Australia-based epidemiological studies hint at increased respiratory symptoms, such as wheeze and rhinitis, among production workers, no clinically significant lung function decrements have been observed.

Cancer incidence and mortality studies are very limited. Published data from Western Australia indicate no increased risk for all-cause and all-combined cancer mortality compared with an external reference population, and no trends with employment duration. Analyses using an internal comparison population showed no excess cancer risk of any type with bauxite or alumina exposure.

With strong alkalis (mostly NaOH) present throughout the refining process, chemical splashes and spills remain a concern. Serious burns of skin and eyes are possible. Traditional emergency showers and eye-wash stations using water are being augmented with more contemporary first aid agents, which are establishing evidence for safety and efficacy.

Confined space entry hazards abound at refineries; thus, conventional health and safety practices to control for these risks are paramount. Diesel-powered mobile equipment—used, for example, inside refinery tanks for descaling—generates diesel particulates, exposure to which must be controlled. Welding-fume control through standard ventilation and respiratory protection is important. Residual asbestos may be present in older refineries and must be managed appropriately to protect against worker exposures. The presence of organic matter in some bauxites can produce volatile organic compounds during the digestion process; volatile organic compounds, with associated odor and irritation, are issues of potential concern

both to employees and to local communities. Because alumina refineries tend to be colocated in relative geographic proximity to bauxite mines, the same environmental ambient concerns relating to heat and humidity, communicable disease, psychological factors, and ultraviolet light, apply, as do their respective control strategies.

ALUMINUM SMELTING PROCESS AND ASSOCIATED OCCUPATIONAL HEALTH HAZARDS AND RISKS

Aluminum is extracted from alumina through the Hall-Héroult process. In brief, this entails an electrochemical process involving long lines (some more than a kilometer in length) of electrolytic cells, or pots, that use low-voltage, high-amperage electricity to produce the aluminum from the raw material or alumina (aluminum oxide). The Hall-Héroult process was invented in 1886 and has remained fundamentally unchanged since that time. A more-detailed graphic overview and description are available via the IAI's Aluminium for Future Generations project.⁶

In this process, molten aluminum is produced by electrolytic reduction of alumina, dissolved in a molten fluoride electrolyte consisting mainly of cryolite, within a series of electrolytic cells or "pots." The pots are carbon lined and serve as the cathode to the electrolysis process. Carbon anodes have to be produced by a separate process. There are two main types of cells used in aluminum production. The Söderberg cell was the dominant technology for many years since its initial use in the early part of the twentieth century; however, the more common type in operation today is known as the prebake cell. In the former, the anode is made from a mixture of petroleum coke and coal tar pitch, and contains about twice the pitch content used for making prebaked anodes. Small briquettes of anode paste are added regularly at the top of the Söderberg cell and the anode bakes in situ. In the prebake cell, anodes are made from a mixture of petroleum coke, coal tar pitch, and anode material termed "butts" that represents remnant anode removed from the cell during anode changing. Anodes are consumed during the electrolytic process and must be removed from the cell before they are completely used up. Prebake anodes derive their name from the fact that such anodes are prebaked in special anode-baking furnaces at about 1150°C to 1200°C, causing the pitch to carbonize and forming strong and dense anode blocks.

Söderberg cells have lower current efficiency and greater difficulty in collecting anode-baking fumes, especially polycyclic aromatic hydrocarbons (PAHs). These hydrocarbons are mainly volatiles from the pitch used in the anode paste. Polycyclic aromatic hydrocarbon consists of many different organic compounds, which have been shown to be carcinogenic.

Alumina has three basic roles in the smelting process:

1. As a feed for the cell, alumina may be added intermittently (in older cell types) or in measured quantities at short intervals usually via a point breaker feeder system.
2. As a thermal insulator on top of both the cell crust and the carbon anodes.
3. To help as a scrubbing medium in the cleaning of captured cell gases. Activated alumina, which contains the residual fluoride, is then used as a feed for the reduction process; in fact, it has a positive impact on cell chemistry and process efficiency over nonactivated alumina.

Power, a major input for the electrolytic cells, comes from four main generating sources. Coal (50%) and hydroelectric (40%) provide the greatest contribution, with natural gas (8%) and nuclear (2%) sources contributing the remainder.

The electrolyte consists of mainly cryolite and smaller amounts of aluminum fluoride, calcium fluoride, and alumina, and can be the source of various fluorinated compounds, such as hydrogen fluoride (HF) and sodium tetrafluoroaluminate (NaAlF_4). The article by Kvande and Drabløs⁷ fully describes the operation of the

cells and potlines, and the reader is referred to that article for a detailed review of operational considerations, including specifics on the magnetic fields and cell start-up considerations.

Physical Hazards

Physical hazards associated with all heavy manufacturing are common during aluminum smelting. Chief among these hazards are heat stress, noise, and ergonomic and electromagnetic fields (EMFs). Within smelting operations, heat stress occurs because of high levels of process-generated heat, which is compounded by ambient heat exposure, metabolic demands associated with the most common tasks involved (particularly within potrooms), and the requisite use of personal protective equipment and clothing.

Studies of US smelter workers suggest that heat stress levels may exceed defined occupational exposure limits (OELs) and guidelines.^{8,9} There is at least some evidence supporting the protective nature of heat stress OELs in relation to anode setters within the aluminum industry as well as other workers.^{10,11} This evidence, derived from investigations of heat-related acute incidents and ambient heat measured as a function of OEL (below, above low, and above high), showed statistically significant increases in incidents as temperature increases.

Both general and job-specific controls for heat stress exposures should be considered and implemented based on the results of exposure assessments. Acclimatization protocols remain an important component of heat stress management. Baseline and preheat season medical screening to identify personal risk factors for heat intolerance, emergency response planning, and worker training on early recognition of heat-related illness symptoms are additional components to a comprehensive program. For job-specific exposures, engineering and administrative controls are often necessary, and may include the use of personal cooling methods of heat stress control.

Noise is arguably the most prevalent occupational hazard within the aluminum industry, as it is for most other manufacturing settings. Yet, noise is often subordinated to other hazards perhaps because of its omnipresence within industry, the delayed onset of perceptible impact, perceived mild impact as an occupational illness, and difficulty in differentiating occupational from nonoccupational impacts.

Both auditory (noise-induced hearing loss, acoustic trauma, and tinnitus) and, with increasing recognition, nonauditory effects (communication interference, risk for injury, and cardiovascular impacts) can result from excessive occupational noise exposure. It is the auditory effects that serve as the basis for current occupational noise regulation globally. Studies of noise-exposed individuals within aluminum production facilities indicate an approximate 2% annual rate of standard threshold shifts.¹² A standard threshold shift is viewed as a significant change and is defined by the US Occupational Safety and Health Administration as an average decline of 10 dB or more at 2000, 3000, and 4000 Hz in a given ear, relative to a baseline audiogram; age correction is optional.¹³

Noise-related injury risk is thought primarily to be related to communication interference and inability to detect safety warning signals.¹⁴ Both animal studies and epidemiological studies of noise-exposed workers show acute physiological effects on the cardiovascular system—increased blood pressure and heart rate, cardiac rhythm disturbances, and increased cardiovascular workload—via stimulated stress responses and cortisol/catecholamine releases and are hypothesized to increase risk for acute myocardial infarction or sudden death.¹⁵

Contemporary aluminum production technology requires high levels of direct-current electric power to drive the electrochemical reduction process within the electrolytic cells (pots) found in potrooms. This necessary low-voltage, high-amperage current, used to create the electric charge between the anodes and cathodes of the

pots, is generated within smelter colocated rectifier yards through the conversion of incoming alternating current electricity. Potroom workers, electricians, and maintenance personnel are thus potentially exposed to EMFs as an unavoidable physical hazard; however, measured levels of EMF exposure in potrooms and rodding areas are below existing OELs. Epidemiological studies exploring the potential health impacts on aluminum industry workers with EMF exposure are very limited; but, to date, no relationships have been identified in preliminary studies looking at EMF exposure and cancer risk, sick leave because of musculoskeletal disorders, and reproductive outcomes.^{16–18} Administrative controls to restrict individuals with certain medical devices from potrooms and other high-field strength environments are prudent. Magnetophosphenes, a visual phenomenon manifesting as the sensation of light flashes in ones vision caused by induced electric currents stimulating the retina,^{19–21} have also been noted as occurring in aluminum smelter workers.

A number of traditional ergonomic stressors exist within aluminum smelting and refining; however, there are few published studies detailing the nature, extent, and programmatic intervention focused on such risks. Nevertheless, published data do exist within the industry demonstrating robust injury risk reduction when targeted ergonomic hazards are identified and systematically controlled.²² In addition, the IAI, through a collaborative effort with the International Council on Mining and Metals, has produced health metrics guidance, which recognizes the importance of including ergonomics as a key leading indicator.²³

Respiratory Disorders

As with other industrial settings, inhalation hazards represent the most common mechanism for potentially injurious exposure within aluminum production. There is a long history of academic study relative to respiratory disorders among aluminum smelter workers, particularly for those whose primary tasks occur in potrooms. Thus, the literature base is replete.

Much of the earlier literature has its origins in the study of Norwegian and Canadian smelter workers, as primary aluminum production has been a long-standing core industry in these regions. These were primarily case reports and prevalence studies. More recently, Australian, European, and US researchers have contributed additional important data to the overall understanding of the respiratory health of aluminum production workers.

Respiratory health endpoints of interest have centered on the symptoms of cough, wheeze, and rhinitis, as well as the more quantifiable presence of obstructive or restrictive lung disease. To date, reactive airways dysfunction syndrome has not been reported among aluminum smelter workers. Asthma in the primary aluminum industry, colloquially called “potroom asthma,” however, has been of particular interest and study and is recognized as a condition somewhat specific to the industry.

In this issue of the Journal, Kongerud and Søyseth²⁴ provide an excellent historical and contemporary review of respiratory disorders that have been investigated in association with occupational hazards present within the aluminum production industry. Cross-sectional studies have suggested a higher prevalence of generic respiratory symptoms, often correlated with increasing exposure or employment duration; however, the usual limitations of such studies preclude drawing specific causal inferences.

Robust longitudinal studies have more consistently shown elevated rates of both nonspecific respiratory complaints as well as diagnosable asthma in potroom versus nonpotroom workers. Statutory occupational disease reporting in many countries—asthma in this context—has been useful in corroborating epidemiological findings and suggests that potroom asthma cases continue to occur, albeit at much reduced rates as compared with historical levels. This reduced incidence over the last decade or so directly parallels

concurrent reductions in potroom dust and gas exposures over the same time course.

A number of mortality cohort studies of aluminum production workers have been ongoing. Chronic obstructive pulmonary disease is the cause-specific respiratory disease of interest. An increased chronic obstructive pulmonary disease-related mortality rate has been observed, particularly for workers involved in potroom employment during the era of less-mature hygiene and respiratory protection practices.

Mechanistically, potroom asthma has most consistently been linked with fluoride exposures in dose-response fashion, dating to the 1930s. Nevertheless, the definitive causative agent or agents have not been unequivocally determined. The relative contribution of isolated peak, repeated peak, or chronic low-level exposures to fluorides on the development of potroom asthma remains uncertain.

Predisposing host factors have been studied and debated. Atopy does not seem to confer an increased likelihood for the development of asthma symptoms among potroom workers. Once clinically manifest, there are no discernible differences between potroom-associated asthma and asthma in the general population.

Within the broad construct of occupational asthma definitions, potroom asthma is generally viewed as an irritant, nonimmunological form of work-related or work-exacerbated asthma. The IAI Health Committee has developed criteria for the diagnosis of this condition.²³

Follow-up studies of subjects with potroom asthma indicate that, as with other forms of work-related asthma, symptoms may persist even after removal from further potroom exposure. Thus, worker education, rigorous medical surveillance, and prompt removal from exposure on early symptom recognition are essential to the most favorable prognosis.

Various forms of diffuse parenchymal lung disease have also been associated with aluminum production and aluminum itself, as presented in the accompanying article by Taiwo.²⁵ Recognition that metallic aluminum powder and aluminum oxide can lead to the development of lung disease dates to the 1930s. Historical references to aluminosis (Shaver disease) attributed to high concentrations of alumina and silica among alumina abrasive manufacturing workers appeared in 1947. Aluminum-induced granulomatous lung disease has also been reported and is distinguished from sarcoidosis by the presence of aluminum within the granulomas.

Epidemiological evidence for interstitial lung disease in primary aluminum production workers (mining, refining, and smelting) within the United States and, more recently, Australia is scant. Because of the rarity of such case reports, controversy remains as to the causal connection with aluminum. Coexistent exposures to more plausible fibrogenic materials such as asbestos and silica in the primary production work environment are common.

Taiwo²⁵ concludes that, altogether, the existing human epidemiological data suggest aluminum is—in the vast circumstances—only a nuisance dust (occupationally), with rare idiosyncratic occurrences of pulmonary fibrosis in susceptible individuals.

Beryllium-related disease (beryllium sensitization and/or chronic beryllium disease) has now been demonstrated at very low incidence rate in aluminum smelter workers, owing to naturally present beryllium in substrate ores that progresses through, and concentrates during, the production process.

Cancer

There is epidemiological evidence for a causal connection between exposures to specific agents during primary aluminum production processes and certain cancers. Most of what is known relates to Søderberg operations or mixed Søderberg/prebake operations. There have been significantly increased lung and bladder cancer risks reported in Søderberg workers from several countries, but not all. Polycyclic aromatic hydrocarbons have been the putative exposure agent linked to these cancers. In prebake smelters, the main

exposure to PAHs occurs during manufacture of the anode, and once in situ within the pot additional release of PAHs from prebake anodes is low. In contrast, exposure to PAHs within Søderberg smelters occurs during operation of the cell; thus, all potroom workers are potentially exposed, including during the relining of cathodes. Many PAHs are present and arise mainly from the coal tar binder or pitch. Benzo(a)pyrene (BaP) is often used as a good measure of exposure for the common PAHs seen in potrooms. Observed lung and bladder cancer risks increase with cumulative exposure to BaP even after adjustment for smoking.

Limited evidence exists in several cohorts for an increased risk of tumors at other sites, including stomach, pancreas, rectum/rectosigmoid junction, esophagus, larynx, buccal cavity/pharynx, kidney, brain/nervous system, prostate, and lymphatic/hematopoietic tissues (in particular non-Hodgkin lymphoma, Hodgkin disease, and leukemia). For most of these tumor sites, the relationship with specific exposures has not been demonstrated clearly, and further follow-up of workers is warranted.

It is clear that exposures to PAHs are a significant cause of certain cancers. The etiology of other cancer types, however, is not as obvious, and while some risks are considered as significant ones, there is a definite need for further research in this area. Other potential exposures can include some metals (beryllium, mercury, nickel, vanadium, etc) and crystalline silica. Workers involved in pot lining can be exposed to tars and fluorides, and in some cases, asbestos.

In 2012, the International Agency for Research on Cancer published volume 100F “A Review of Human Carcinogens: Chemical Agents and Related Occupations,” including a monograph titled “Occupational Exposures During Aluminium Production.”²⁶ This monograph notes that in *IARC Monograph* volume 92: “There was *sufficient evidence* from epidemiological studies of a carcinogenic effect of occupational exposure in aluminium production based on a relatively large number of studies that showed a consistent excess of cancer of the bladder and a somewhat less consistent excess of lung cancer.” After further reviews of new data, the 2012 monograph concludes, in the final evaluation: “There is *sufficient evidence* in humans for the carcinogenicity of occupational exposures during aluminium production. Occupational exposures during aluminium production cause cancer of bladder, and of the lung . . . Occupational exposures during aluminium production are carcinogenic to humans (Group 1).” It is important to note, however, that the aluminium production exposure agent of focus in International Agency for Research on Cancer’s determination is, specifically, PAHs.

Globally, many studies have been carried out with respect to cancer in the aluminum smelting industry. Gibbs and Labrèche²⁷ sum up the data in three tables that are, however, too extensive to review in full detail in the present article. The first table provides a brief authors’ annotated description of “Published Cohort Studies on Workers in Aluminum Reduction Plants.” The second table summarizes mortality and incidence data for various cancers with respect to whether the results show a statistically significant excess or a not statistically significant excess. In Table 3, the authors comment on the “available evidence of a causal relationship between work in aluminium production and selected cancer sites.”

NEW TECHNOLOGIES⁷

Inert Anodes

One of the long-standing wishes of the industry has been to invent and use “inert” anodes, one that is chemically nonreactive. Any future inert anode material must have several key characteristics: conduct electricity, have low solubility, have low reactivity in the electrolyte, show good chemical resistance, be physically stable, robust, and resistant to thermal shock. Other important factors include low wear rates, so that there is little need for repeated

replacement—ideally it should last the average length of the cell itself and have a purity level that prevents aluminum contamination.

Ongoing work on the quest for an inert anode is fueled by cost savings, when compared with the need for repeated replacement of consumable anodes, as well as potentially lower chronic material costs, improved cell environmental profiles, and fewer issues related to employee health through a lower job risk profile. Nevertheless, a direct replacement of inert anodes in existing electrolysis cells would lead to higher electricity consumption.

Two areas of interest remain in the forefront of inert anode development—metal alloy anodes, and “cermet” or ceramic/metal alloy mixtures. Unfortunately, issues still remain to be resolved before either of these approaches can be considered as a proven technology. The potential advantages are so great, however, that serious work continues to be done toward the successful development of the inert anode.

Carbothermic Production of Aluminum

The carbothermic process uses carbon and heat in a three-step process, rather than an electrolytic reduction process, to reduce alumina to aluminum. Like the inert anode, the carbothermic process has gone through several iterations, starting with the initial attempts that produced Al–Cu alloys (1886) and later efforts that led to Al–Si alloys (from the 1920s to 1945). More recent efforts in several countries have focused on the production of pure aluminum. Currently, work is underway in Norway to produce an Al–C alloy, which could then be reduced to aluminum. These steps, the production of the Al–C alloy and the reduction of the alloy to aluminum, pose the greatest challenges.

To further complicate matters in the search for a successful carbothermic process, considerable carbon monoxide (CO) will be released, leading to a 60% increase in overall CO₂ over current methods, through natural degradation. Even with a potential saving on electrical power, and assuming such savings would be applied to power generation from coal sources, a net increase of 40% will occur unless the CO₂ can be captured and used in other processes/uses.

COMMUNITY AND ENVIRONMENTAL HEALTH CONSIDERATIONS

Health Risk Assessment Around Alumina Refineries

Health risk assessment (HRA) is a tool that can be used to estimate or predict the current or future health impact of chemical exposures on a population, such as the communities in which industrial operations are present. In recent years, HRA has been increasingly used to characterize potential health impacts to the citizenry in proximity to alumina refineries, and Australia-located refineries in particular—as noted by Donoghue and Coffey²⁸ in this issue of the Journal—have cultivated significant experience and expertise in the HRA process.

Because the overriding type of industrial emission for refineries is airborne chemicals, the centerpiece component of an HRA is air dispersion modeling. Such modeling requires significant professional input to execute and interpret properly, and this methodology must be used to fully assess and characterize potential health impacts.

The starting point for a rigorous HRA is the identification of all key or potential emission sources (point and fugitive), followed by an inventory of substances emitted and estimates of emission rates. Single-source emission sources are far more easily modeled than when there are multiple overlapping emissions from different industry or ambient sources, which introduce significant complexity. A key output of the modeling process is the development of ground-level concentrations for each chemical of interest. In areas with difficult topography, direct measurement should be used to verify the model.

Many assumptions relative to exposure estimates inform the air dispersion modeling process; thus, uncertainties are inherent in predicted ground-level concentrations. The ratio of each chemical specific ground-level concentration to available acute and chronic guideline values produces acute and chronic hazard quotients, respectively. Individual hazard quotients are then summed to generate an Acute Hazard Index (AHI) and a Chronic Hazard Index (CHI). Additive relationships are assumed, building further conservatism into the analysis. The AHIs capture the 1- and 24-hour health risks, whereas CHIs reflect annual average health risk. Incremental cancer risk is determined in a similar fashion. Incremental cancer risks describe cancer risk above background, typically using the de minimus level of 1×10^{-6} set out by authorities such as the US Environmental Protection Agency and the World Health Organization.

Overall, only a few substances predominate in the calculated AHIs and CHIs. For acute health effects, these are mostly the criteria pollutants; however, AHIs in completed HRAs have only rarely shown values in excess of 1.0, the threshold for potential elevated risk to the community, even at the most sensitive receptor sites. Likewise, CHIs in these same locales have consistently been less than 1.0. Observation of values more than 1.0 warrants dialogue among the professional team commissioned for the HRA to revisit the a priori modeling assumptions and emission data, given the conservatism embedded in the methodology. The process is extremely iterative.

Incremental cancer risk estimates approached the US Environmental Protection Agency de minimus level, supported as well by separate but parallel epidemiological investigation of workers at the assessed refineries in Australia showing no increased cancer incidence or mortality.

In Australia, best-practice HRA approaches and modeling techniques have been developed under the auspices of the Australian Aluminium Council. Health risk assessments are quite useful in assessing the potential impacts of process or facility changes, and implemented engineering control measures, and also for engaging appropriate community stakeholders.

HRA Around Aluminum Smelters

Community health risks from smelter emissions, including noise, can be roughly assessed on the basis of existing occupational health studies and on other community-based studies. Potential community health effects from aluminum smelters arise from the use of the Hall–Héroult production process, a process with well-defined hazards that have the potential to impact on the health of smelter workers.

The article by Martin and Larivière²⁹ reviewed 298 studies published in peer-reviewed journals, using keywords relevant to communities and the aluminum smelting process. They were also able to include other data gathered as a result of personal experiences in the aluminum smelting industry. A number of community studies were reviewed with respect to potential “outside the fence” health risks from smelter-generated emissions. This enabled them to develop a HRA by using “comparisons between actual or estimated community exposure levels and those associated with adverse outcomes in the smelter studies and/or nonsmelter communities” to “assess risk in aluminium smelter communities.”

Multiple hazards were identified in aluminum smelter workplaces, and their risks to workers were defined. Mirroring what is described in earlier sections of this article, the hazard profile included aluminum, aluminum oxide (Al₂O₃), beryllium, carbon monoxide (CO), carbon dioxide (CO₂), dust (respirable and inhalable), fluorides (particulate and gaseous), EMFs (static [direct current] and variable [alternating current]), nitrogen dioxide (NO₂), noise, PAHs (as benzene soluble matter and BaP), and sulfur dioxide (SO₂). Identified hazards were then assigned “to one of five risk categories: none; uncertain; low; medium; high.”

To estimate the contribution of an industrial site to health impacts in a community is a challenge. There is first a need to understand the industrial processes themselves, then to have good data related to emissions to the community, and finally to be able to define the contribution of the specific industrial site being studied. (See methodology described earlier in relation to HRAs around alumina refineries). In general, the more urban the area, the greater is the likelihood that there will be multiple industrial, and perhaps natural (crustal) environmental, sources for the emissions under study. Another confounder is the use of occupational health data extrapolated to the general population, as these populations may differ significantly (eg, workplace populations usually consist of more men, fewer women, and no children when compared with local communities).

The authors have reviewed available articles on potential health hazards that may also pose community health risks because of the proximity of the communities to aluminum smelters. They note that there is the potential for materials such as BaP, sulfur dioxide, particulate matter, fluorides, beryllium, and noise to pose community health risks. The range of risk magnitude, however, is widely dispersed on the basis of a number of both controllable and noncontrollable factors, including emission-control technologies, dispersion patterns, and proximity to the facility.

Public Health Considerations—The Aluminum Hypothesis

The Aluminum Hypothesis (AH) espouses the theory that aluminum exposure is involved in the etiology of Alzheimer disease (AD). As excellently chronicled in the article by Lidsky,³⁰ this hypothesis owes its origins to three articles from the mid-1960s. One of these articles demonstrated that aluminum causes neurofibrillary (NF) tangles in the brains of rabbits when injected intracerebrally. This led to an initial focus on the role of aluminum in AD. Although it still continues to attract the attention of a small group of scientists and aluminum continues to be viewed with concern by some of the public, the AH has gradually been abandoned by most researchers.

The reasons for this abandonment of the AH and for the ongoing public concern are worth exploring further. Although many laboratories continue to investigate the etiology of AD, very few scientists are looking at the AH. Consequently, there is a paucity of peer-reviewed articles on the subject. The original research by Wisniewski and colleagues^{31,32} and Terry and Peña³³ showed that aluminum injected into the brains of rabbits induced neurofibrillary tangles that, with silver staining, looked like the neurofibrillary tangles seen in AD patients. The rabbits also showed signs of cognitive deficits. This study was, in fact, done in response to an accidental finding in another study that antigens bound with Holt adjuvant (containing alum phosphate) led to convulsions and NF degeneration. Later research showed elevated aluminum levels in brains of patients with AD and dialysis encephalopathy due to contamination of dialysates by aluminum.

The AH arose on the basis of these studies. According to Lidsky,³⁰ the observations in these articles do not relate aluminum to AD for several reasons:

- Aluminum salts do not lead to NF changes that are similar to those seen in AD.
- The symptoms and pathology of dialysis encephalopathy—while caused by aluminum—are not the same as those of AD.
- Increasing levels of aluminum in the brain with aging have no known functional significance.

The Bradford Hill Criteria (BHC) are frequently used to determine whether there is solid scientific evidence suggesting a causative role between diseases and occupation and lifestyle. Although Dr Hill outlined nine criteria, the following four criteria subsets have been identified as “necessary criteria” to establish causation with respect to neurocognitive disorders such as AD;

BHC 1—Strong association between the causative agent and the outcome:

- Aluminum elevations in the brain do not lead to the AD clinical signs or neuropathology.
- Individuals with high levels of aluminum in their brains due to chronic renal impairment did not show the clinical patterns of AD.

BHC 2—Consistency of findings

- There is an ongoing and consistent lack of agreement between studies conducted by different investigators, as well as, often, between findings reported by the same investigators.

BHC 3—Appropriate temporal sequence of exposure to agent and outcome

- Cognitive symptoms of AD occur long after the onset of the disease—often decades later.
- Many epidemiological studies have insufficient lapse time between exposure and onset of AD.

BHC 4—Biological plausibility

- Examining *in vitro* impacts of aluminum, one review of more than 100 articles noted that aluminum can impact “more than 200 biologically important reactions and cause various adverse effects on the mammalian central nervous system,” however:
 - Aluminum concentrations far exceeded “those seen in normal individuals or even those persons with disturbed renal function,” and
 - None of the studies cited indicated “that *in vitro* aluminum can induce pathological changes in animal models that are qualitatively similar to those of AD.”
- *In vivo*, the toxicokinetics of rats (and possibly other animals) differ significantly from those of humans, and the renal system of rats is very sensitive to aluminum toxicity—a factor seldom taken into account; *in vivo* studies that discuss the renal issue have produced contradictory results.

Lidsky³⁰ concludes that:

- “Consideration of the published research concerning aluminum’s role in Alzheimer’s disease indicates that not one of the four Bradford Hill criteria deemed necessary to establish causation with respect to neurocognitive disorders such as Alzheimer’s disease has been satisfied.”

Until the etiology of AD is clearer and treatments are more effective, the increasing public familiarity (and fear about) AD is likely to continue to lead to concerns about the relationship between aluminum and AD and perpetuate the AH.

CONCLUSION

There are many well-studied and characterized occupational health hazards and risks within the primary aluminum production industry. On the basis of various environmental and technical factors, some of these risks may, in select circumstances, also extend to local communities—although the evidence for this is less clear. Rigorous health protection programs have generally been implemented throughout the industry to address and control, to the extent feasible, the identified hazards and risks. These include comprehensive industrial hygiene and medical surveillance programs and the use of state-of-the-art technologies and approaches wherever possible. Improved process control, positive technological changes, and better planning have also lowered potential risks for local communities.

The Health Committee of the IAI has been a key catalyst and facilitator in driving continual improvement and technological changes for the protection of workers in the aluminum industry and those people resident in surrounding communities, including the

development of applicable industry-relevant health indicators. The IAI's Health Committee has also had, and continues to have, an interest in enhancing the scientific understanding of the roles played by aluminum with respect to human health.

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