IPAI

International Primary Aluminium Institute
Health Committee Review

The Measurement of Employee Exposures in Aluminium Reduction Plants

Part I: Chemical Exposures

REVISION

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Preface

Preface to first edition

This review has been prepared by the Health Committee of the International Primary Aluminium Institute under the authority of the Board of Directors of the Institute.

Special recognition is due to the members of the working group who produced the detailed text. The group was led by Mr. Thomas B. Bonney of Aluminum Company of America and also included Mr. Homer M. Cole of Reynolds Metals Company. Mr. Eirik Nordheim representing Norwegian Smelters. Mr. Jan Rob of Elkem a/s. Dr. Alfred Steinegger of Swiss Aluminium Limited and Mr. Thomas J. Walker formerly of Kaiser Aluminum and Chemical Corporation.

Preface to second edition

The revised version was prepared by the working group led by Dr. Alfred Steinegger of Alusuisse-Lonza Services Ltd., and also included Mr. Homer Cole of Reynolds Metals Company, Dr. Peter Cook of Dubai Aluminium, Mr. Joe Damiano of Aluminum Company of America, Mr. Serge Friar of Alcan Smelters & Chemicals Ltd., Mr. Eirik Nordheim representing Norwegian smelters.

Contents

The advances in industrial technology and biological sciences have played a major part in social and economic progress in recent years. These changes have influenced governments, industry and labour both on a national and international scale. The industrial hygienist as a member of the occupational health team has been a part of this change for a number of years. The preventive approach, which today is prominent in legislation from governments and governmental agencies, has been practised by industrial hygienists for some time.

Modern technology may generate health hazards; some of which are easy to identify, but others are only discovered after years of careful study. For each type of industry, it is important to identify and analyse the health factors to determine their biological effects and to develop methods of prevention, both technical and medical. In this situation, cooperation within an industry is essential if the required knowledge is to be accumulated in a timely and efficient manner. The aluminium industry has been able to cooperate on a world-wide basis, and two of the best examples of this in the field of occupational health are the IPAI seminars, 'Health Protection in Primary Aluminium Production', held in Copenhagen in 1977 and in Montreal in 1981. In these seminars, the various aspects of health protection in the aluminium industry were considered with reference to the various pollutants and physical factors encountered in this industry.

One fact that has emerged during these seminars is that, in order to make a detailed study of the environmental conditions and related health problems at a particular working place, there is a definite need for accurate and generally acceptable methods of sampling and analysis. This is particularly important where official limits have to be met and where the question of modernization of a plant may depend on the outcome of the measurements. The present document, which has been prepared by the Health Committee of IPAI, is intended to give a review of the sampling and analytical techniques in general use in the aluminium industry. It is not the intention to give a detailed description and critical review of all known techniques, but there is an extensive list of references which can be consulted for more detailed information. The document will give a description of the most common pollutants found in the various departments of an aluminium plant. Further, it will deal with the actual sampling and analytical techniques, giving a description of the various sampling strategies and methods for sampling and analysis of the principal contaminants.

Progress and innovations in the technology of aluminium production on the one hand, new results / knowledge gained in measuring technology and assessment of possible health hazards on the other made, in the opinion of the Health Committee, a partial upgrading of the first edition necessary. Essential changes and additions have been made in Section 1 "Principal contaminants at the workplace" and Section 4 "Occupational exposure limits of airborne toxic substances". Section 3 "Air sampling and analysis" was re-written. In Section 2 "Toxicology of principal environmental contaminants" no classification into substances of different importance was made any more.
Part two to be published will describe the measurement of employee exposure to physical agents.

ASBESTOS — Recognized human carcinogen

CAS: 1332-21-4
Amosite—CAS: 12172-73-5;
1987 TLV = 0.5 fiber/ml
Chrysotile—CAS: 12001-29-5;
1987 TLV = 2 fibers/ml
Crocidolite—CAS: 12001-28-4;
1987 TLV = 0.2 fiber/ml
Other forms: 1987 TLV = 2 fibers/ml

Synonyms: Asbestos is a generic term applied to a number of hydrated mineral silicates.

Physical Form. Fibers of various sizes, colors, and textures

~~**Uses: Thermal and electrical insulation; fireproofing; cement products**~~

Exposure. Inhalation

Toxicology. Asbestos causes chronic lung disease (asbestosis), inflammation of the pleura, and certain cancers of the lungs and digestive tract.

Asbestosis is a disorder characterized by a diffuse interstitial pulmonary fibrosis, at times including pleural changes of fibrosis and calcification.[1] Chest x-ray reveals a granular change chiefly in the lower lung fields; as the condition progresses, the heart outline becomes shaggy, and irregular patches of mottled shadowing may be seen. Typically, the patient exhibits restrictive pulmonary function. Accompanying clinical changes may include fine rales, finger clubbing, dyspnea, dry cough, and cyanosis.

The onset of asbestosis probably depends upon asbestos dust concentration, fiber morphology, and the length of exposure. There may also be some factors of individual susceptibility, although it is not possible to identify persons who may have undue susceptibility or resistance to developing asbestosis.[2] It is a progressive disease that may develop fully in 7 to 9 years and may cause death as early as 13 years after first exposure. Usually, pneumoconiosis becomes evident 20 to 40 years after the first exposure to asbestos. Once established, asbestosis progresses even after exposure has ceased.[1]

There is often thickening of the visceral pleura from extension of the parenchymal inflammation. The parietal pleura may show patches of severe thickening, particularly over the diaphragm and the lower portions of the chest wall, resulting in the so-called pleural hyaline plaques. These may be seen by x-ray, especially if calcified. Pleural plaques, which produce no symptoms, may develop from asbestos bronchogenic carcinoma and mesothelioma of the pleura and peritoneum are causally associated with asbestos exposures; excesses of cancer of the stomach, colon, and rectum have also been observed.[4] Among 632 asbestos workers observed from 1943 to 1967, there were 99 excess deaths (above that expected on the basis of the US white male population) for three types of malignancies: (1) bronchogenic (63), (2) gastrointestinal, (26) and (3) all other sites combined (10).[1]

Mesothelioma, a relatively rare and rapidly fatal neoplasm seen chiefly in crocidolite workers, may occur without radiologic evidence of asbestosis at exposure levels lower than those required for prevention of radiologically evident asbestosis.[1] Mesothelioma can occur after a short intensive exposure; cases in patients younger than 19 years of age indicate that the latent time period for development may be shorter than first estimated, although the disease may occur following a very limited exposure 20 to 30 years earlier.

Cigarette smoking is strongly implicated as a cocarcinogen among asbestos workers.[5] The incidence of lung carcinoma among nonsmoking asbestos workers is not significantly greater than that of nonasbestos workers, whereas asbestos workers who smoke have a much higher incidence. Cigarette-smoking asbestos workers have approximately 15 times the risk of developing lung cancer compared with nonsmoking asbestos workers.[6]

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Sources
Electrical insulators between pot anodes and cathodes, or ground; thermocouple wire; Marinite; spacers in casting furnaces; brake linings

(remainder of outdated technology)