Chapter 5
Occupational Exposure to Aluminum and Cognitive Impairment

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Abstract Aluminum industry has been producing more than a century of history. Aluminum is a light metal which is widely used in industrial applications. Occupational aluminum workers are an important source for high exposure Al population. With the world's demand for aluminum and the increasing production, between 2007 and 2016, primary Al production in China increased by 251% which shows that a growing number of workers are being exposed to Al. Occupational aluminum exposure to the health of the population has become increasingly prominent. Al is a well-established neurotoxicant and is suspected to be linked with various neurodegenerative diseases including Alzheimer's disease (AD) and MCI. Studies on workers exposed to aluminum have revealed disturbances in cognitive function. This chapter reviews the relationship between occupational exposure to aluminum and cognitive impairment.

Keywords Aluminum · Occupational exposure · Biological monitoring · Cognitive function

5.1 Introduction

Aluminum (Al) is a ubiquitous element in the Earth. Therefore, exposure is unavoidable even in the general population. Exposure that greatly exceeds that of the general population is experienced owing to occupation and drinking water. Occupationally, Al can be found in various industries, e.g., the Al powder industry and the metal industry, and in Al foundries. Aluminum resistance to chemicals is due to a protective layer of Al oxide that spontaneously forms on the surface. Aluminum and its alloys with other elements (copper, magnesium, manganese, silicon, and zinc) are used in vehicles, electric devices and wiring, building materials, packaging, and for corrosion protection of structural steel. Aluminum powders are

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used in pigments, automotive paints, rocket propellants, explosives, and fireworks. Bauxite is the main raw material for aluminum production. It is refined to Al hydroxide and further to Al oxide, which is electrolytically reduced to aluminum (primary aluminum production). Large volumes of aluminum are produced by recycling scrap aluminum in the smelter (secondary aluminum production) [22]. Neurotoxicity is the critical effect of exposure to aluminum. Al is a well-established neurotoxicant and is suspected to be linked with various neurodegenerative diseases including Alzheimer's disease (AD) and MCI [14, 17, 20]. In recent years, studies suggested an association between cognitive impairment and other neurological effects and occupational exposure to aluminum dust or fume. This chapter reviews the relationship between workers' exposure to aluminum and cognitive impairment.

5.2 Exposure Assessment

Occupational exposures during the primary smelting of aluminum, foundry work, production of aluminum flake powder, or the welding of aluminum with the metal inert gas (MIG) method can be associated with an increased uptake of aluminum [23]. The existing form of aluminum in the workplace is mainly fume and dust. So that it is primarily absorbed from the air via the lungs at the workplace. Ingestion is possible, but according to current scientific knowledge, the inhalation route dominates.

Data on the Al half-times of occupationally exposed persons varies widely in the literature, from days to months, depending on the duration of the exposure. Certainly, the bioavailability of different types of Al has to be considered. The magnitude of Al exposure and work conditions varied widely among the different job types. At electrolysis worksites, the main pollutants were aluminum oxide and fluorides, and workers were exposed to relatively high levels of aluminum fume and dust. However, the smelting and welding workers were much less exposed to these pollutants, and many welding workers worked outdoors. The workers studied have been aluminum production workers employed in either the foundry or potroom [3, 11, 16, 24] and aluminum welders [9, 26] with normal, slightly, or moderately increased measures of body burden of aluminum. Among industrially exposed workers, welders who use the metal inert gas (MIG) technique have the highest concentrations of aluminum in urine and serum [25].

Biological monitoring of aluminum is based on the analysis of aluminum concentration in blood/serum or in urine. Al is conventionally determined in serum, plasma, whole blood, or urine after it enters the human body. Hosovski et al. found in an aluminum foundry that the mean U-Al was 1.7 μmol/L among 87 workers with a job seniority of about 10 years [11]. In a group of 17 welders (job seniority about 4 years), the mean S-Al and U-Al concentrations were 0.21 and 2.8 μmol/L, respectively [9]. In another study, the high-exposure group of 30 welders mainly working on aluminum tanks and ships for about 14 years revealed higher levels: the mean serum Al and urine Al concentrations were 0.46 and 7.1 μmol/L, respectively [23].

Bast-Pettersen et al. reported a median urinary Al concentration of 40.5 μg/l in 20 Al welders aged 21–52 years with an average exposure time of 8.1 years [4]. In a foundry, among 119 workers with a mean exposure history of 15 years, low concentrations of aluminum were measured in blood (B-Al) (median 0.037 μmol/L) and urine (median 0.15 μmol/L) [12]. Similar observations were made among welders of train bodies and truck trailers with a job seniority of over 10 years three times for 4 years: in the first examination, the mean P-Al and U-Al concentrations of 44 welders were 12.5 μg/l and 110.7 μg/g Cr, respectively; in the second examination, the mean P-Al and U-Al concentrations of 33 welders were 13.8 μg/l and 120.0 μg/g Cr, respectively; in the third examination, the mean P-Al and U-Al concentrations of 20 welders were 13.9 μg/l and 81.5 μg/g Cr, respectively [13]. Sjorgen et al. reported 38 welders (age = 39 years, work years = 17 years) whose median urinary Al concentration was 24 μg/g Cr and blood Al concentration was 3.0 μg/l [26]. Altogether, the exposure time and Al levels in serum and urine were within the range of studies on occupational exposed Al workers. The obvious conclusion to be drawn is that occupational exposed aluminum can with time lead to significantly increased levels in aluminum biomarkers.

5.3 Cognitive Impairment

There is no doubt that aluminum accumulation and its subtle effects occur among occupationally exposed individuals. Studies on workers exposed to aluminum have revealed disturbances of cognitive processes. Occupational exposure to Al seems to have different effects on workers in different industries. The measurable effects on the central nervous system might only develop after a protracted exposure, and the intellectual domain, mainly affected, varies. Many of the studies concerned exposures in primary aluminum production, foundries, or smelting of scrap aluminum.

The first studies on neurotoxic effects of Al exposure in humans were carried out on patients who had died of dialysis-related encephalopathy. Alfrey et al. found, for example, elevated levels of Al in the brains of patients that had died of dialysis-related encephalopathy [2]. A case study by McLaughlin et al. first suggested that occupational aluminum exposure can cause both lung and brain effects. The patient was a 49-year-old man who had worked for 13 years as a ball-mill operator in a flake powder factory. He was diagnosed with lung fibrosis and rapidly progressing deterioration of CNS functions: forgetfulness, speech disorder, myoclonic jerks, hemiparesis, convulsive attacks, and dementia. Postmortem analysis of aluminum in the brain yielded 5 mg/kg (wet weight) which was a 10–20 times higher concentration than the levels found in brain samples from nonexposed individuals, which were analyzed in parallel [18]. Longstreth reported three patients with a progressive neurologic disorder. The exposure years of three patients were 12, 15, and 16, respectively. Neurologic findings showed that all had incoordination and an intention tremor. Neuropsychological test scores found that two patients had cognitive

deficits, and the most severely affected patient also had spastic paraparesis. The author believed that these patients' diseases probably are related to an occupational exposure in the potroom [16]. After that a few epidemiological studies were conducted on workers occupationally exposed to Al. In Canadian miners who used McIntyre powder by inhalation as prophylaxis against silicosis, Rifat et al. [21] conducted a morbidity prevalence study on Al-produced neurotoxic effects in these mines between 1988 and 1989. The exposed group was consisted of 261 miners and the control was 346 unexposed miners. Cognitive test scores and proportions impaired indicated a disadvantage for exposed miners. With adjustment confounding, the estimated relative risk of impairment of cognitive function among exposed miners was 2.6 [21]. Hosovski investigated 87 workers (age = 41 years, job seniority = 19 years) in an aluminum foundry and 60 nonexposed workers (age = 42 years, job seniority = 18 years). Al values in blood in exposed group were 136.85 μg/l and that in the control was 58.09 μg/l. Al values in urine in exposed group were 45.38 μg/l and that in the control was 7.25 μg/l. The author measured the psychic abilities of subjects with psychomotor ability tests, Wechsler's test of intelligence, and Bender's test for estimation of cerebral damage. Slower psychomotor reaction and dissociation of oculomotor coordination were found in the exposed workers. Exposed workers had reduced memory ability and their mental and emotional balance was disturbed. The observed changes in psychomotor and intellectual abilities could be a consequence of the long-lasting toxic effects of aluminum [11]. White reported the investigation of 25 symptomatic workers from an aluminum smelting plant. The mean age was 47 years of 25 workers and the average duration of employment was 19 years. Neuropsychological test results showed preservation in certain spheres of functioning, such as verbal IQ, with substantial impairment in others, particularly memory functioning. On memory tests, 70–75% showed mild or greater impairment. This study supported the existence of a syndrome characterized by incoordination, poor memory, impairment in abstract reasoning, and depression. Aluminum exposure in the potroom was considered the most likely cause [27]. A cross-sectional study was conducted at a Norwegian primary aluminum plant. Thirty-eight retired workers aged 61–66 years comprising 14 potroom workers, 8 foundry workers, and 16 controls volunteered to participate. They were tested with a neuropsychological test battery. Workers in potrooms with Søderberg electrolytic cells were found to show signs of impairment of the nervous system. A test for tremor discriminated significantly between the potroom group and the controls. There was a suggestion of increased risk of impaired visuospatial organization and a tendency to a decline in psychomotor tempo in the potroom workers. Bast et al. suggest that the above findings may be related to long-term occupational exposure in the potroom and further to chronic low-dose exposure to aluminum [3]. Sjogren studied the effect on the nervous system among welders exposed to aluminum and manganese. This study chose 38 welders (age = 39 years, work years = 17 years) as exposed group and 39 nonexposed Al welders (age = 40 years, work years = 14 years) as control group. The blood Al concentration in exposed group was 3.0 μg/l and that in the control was 1.0 μg/l. The urinary Al concentration in exposed group was 24 μg/gCr and that in the control was 4.7 μg/gCr. Nervous symptom showed that the

welders exposed to Al reported more symptoms from the central nervous system at the time of the test and the most prominent symptom was fatigue. Psychological examination showed that the welders exposed to Al achieved a significantly lower score in non-dominant hand tapping speed, Luria-Nebraska motor scale task item3 and item4, and dominant hand pegboard than did the control group. This study suggested the neurotoxic effects in the welders exposed to aluminum and manganese are probably caused by the aluminum and manganese exposure, respectively [26]. The investigation in 1999 was a cross-sectional study of asymptomatic aluminum welders and a reference group of mild steel welders. Based on urinary aluminum concentrations, welders were classified into a reference (n = 28, average age 38 years), low (n = 27, average age 37 years)-, and high (n = 24, average age 41 years)-exposure group. The mean urinary aluminum concentrations were 0.46, 2.25, and 9.98 μmol/l, respectively. A comprehensive neuropsychological examination was undertaken to assess psychomotor function, simple visual reaction time, attention-related tasks, verbal and visual or visuospatial abilities, as well as verbal and visual learning and memory. The result showed that the low exposed group performed poorer on the memory for designs and on more difficult block design items demanding preliminary visuospatial analysis. The time-limited synonym task, embedded figures, digit symbol speed, and the backward counting component of the divided attention task showed exposure-response relations. In general terms, therefore, the present results suggest that aluminum is associated with detrimental effects on certain cognitive functions. What seems common to the tasks showing impairments is the involvement of time-limited processing in visuospatial tasks where working memory demands are great [1]. Workers from one of the largest aluminum production plants in China founded in 1958 were studied; 167 male workers aged 25–60 years (mean age 37.6 years) were selected to use the WHO-recommended neurobehavioral core test battery (NCTB). An Al-exposed group included 104 workers who had been exposed to aluminum, while working in electrolysis, smelting, or welding, for at least 5 years. Al urine concentration in exposure group was 41.79 μg/g Cr, and that in non-exposure workers was 17.73 μg/g Cr. In this study, all results were adjusted for education and duration of employment to reduce the effects of these factors. After adjustment for work duration and educational level, notable changes in mood as well as neurobehavioral performance still existed in the Al-exposed groups, and age-dependent characteristics were obvious. Younger Al-exposed workers had short memory and elderly workers an impairment of motor activity and accuracy to a certain extent. It should be noted that in the present study, we only found that the young Al-exposed workers had considerably impaired cognitive functions and the elderly notably retarded motor ability, instead of cognitive impairment. These results indicate that occupational aluminum exposure, at the measured level, might interfere with normal behavioral functions. These effects seem to be age-dependent, which might be attributable to age-related changes in susceptibility to environmental chemicals as well as the duration of aluminum exposure [8]. Pollizi conducted a cross-sectional case-control study in northern Italy. The group of 64 exposed workers was to be retired from work for at least 10 years and composed of former aluminum dust-exposed workers with long-term exposure

to the metal from an aluminum remelting plant, and the control group of 32 blue collar workers was composed of demographically similar subjects. The median exposure level of aluminum, in the respirable fraction, was 14.70 μg/m^3 with a range of 7.46–39.26 μg/m^3. Mean serum Al in exposed group (14.1 μg/L) is significantly higher than that of the control group (8.2 μg/L). The neuropsychological tests resulted there is a significant difference in the latency of P300, MMSE score, MMSE-time, CDT score, and CDT-time between the exposed and the control population. P300 latency correlates positively with Al-s and MMSE-time. Al-s has significant effects on all tests: a negative relationship was observed between internal Al concentrations, MMSE score, and CDT score; a positive relationship was found between internal Al concentrations, MMSE-time, and CDT-time. The authors suggest aluminum may be an essential hazard for the central nervous system and raise the question whether preclinical detection of aluminum neurotoxicity and consequent early treatment might help to prevent or retard the onset of AD or AD-like pathologies [20]. Buchta et al. surveyed the longitudinal study included 98 Al welders (mean age 37 years) in the car-body construction industry, with a median of 6 years of occupational exposure to Al welding fumes, and an education-matched, gender-matched, age-matched control group of 50 car-production workers (mean age 36 years) at the same plant. Two cross-sectional studies were done in 1999 and 2001. In the second cross-sectional study, 97 welders and 50 controls could be examined. Al concentration in plasma and urine was measured. All subjects were tested the neurobehavioral, which was included a symptom questionnaire, modified Q16, and computerized and non-computerized tests: psychomotor performance (steadiness, line tracing, aiming, tapping), verbal intelligence (WST), simple reaction time, digit span, block design (HAWIE), symbol-digit substitution, switching attention (European neurobehavioral evaluation system, EURO-NES), and standard progressive matrices. The median Al urine concentration in exposure group was 57.6 μg/gcr (1999) and 52.4 μg/gcr (2001), and median plasma Al level in exposure group was 10.3 μg/L (1999) and 4.3 μg/L (2001). Median respirable air dust was 0.47 mg/m^3 (1999) and 0.67 mg/m^3 (2001). Significant difference in reaction time was seen between welders and non-welders. Regression analyses reveal a significant relationship between reaction time and Al excretion in urine that was confounded by other factors. The results suggest that reaction time could be a first indicator for possible neurological changes in Al welders, as it is significantly related to exposure and age [5]. Buchta conducted the longitudinal study comprised of two cohorts, Al welders and controls in 1999 and 2000. A group of 33 aluminum welders (age = 43 years, Al welding = 11 years) and a control group of 26 production workers (age = 40 years) participated in two examinations in this longitudinal study. In the first examination, Al-preshift and Al-postshift in plasma of exposed workers were 9.6 μg/l and 11.6 μg/l. Al-preshift and Al-postshift in urine of exposed workers were 92.1 μg/gCr and 97.0 μg/gCr. In the second examination, Al-preshift and Al-postshift in plasma of exposed workers were 10.6 μg/l and 14.3 μg/l. Al-preshift and Al-postshift in urine of exposed workers were 90.1 μg/gCr and 143.9 μg/gCr. Cognitive performance showed that welders conducted significantly poorer performance in symbol-digit substitution, block design, and to some extent

in switching attention [6]. He et al. conducted the study on the alteration of neurobehavioral parameters, autonomic nervous function, and lymphocyte subsets in aluminum electrolytic workers of long-term aluminum exposure. Thirty-two exposed workers came from an aluminum plant and 32 control workers were selected. The age of exposed group was 35 years and the length of service was 15 years. Urinary Al in exposed group was 40.08 μg/gCr and that in the control was 26.84 μg/gCr. All subjects were tested nervous function by NCTB (neurobehavioral core test battery) and autonomic nervous function test battery. NCTB result showed that there are significant differences in POMSC, POMST, SRT, SRTF, DSY, PAC, and PA between two groups. This study suggests that Al exposure exerts adverse effects on neurobehavioral performances, especially movement coordination and negative mood [10]. Monika conducted and summarized a meta-analysis of data on the effect of occupational Al exposure on cognitive and motor performance. The final sample consisted of nine studies examining 449 exposed and 315 control subjects. The study found that urinary Al concentration below 135 μg/l has an impact on cognitive performance and cognitive performance was negatively related to U-Al [19]. N.H. Zawilla undertook this research to test the cognitive status of workers ($n = 54$) exposed to Al dust and matched control workers ($n = 51$) by using the ACE-R. The serum Al in the exposed workers was 20.27 μg/l and that in the control was 4.43 μg/l. The ACE-R is a brief cognitive test battery that includes five cognitive functions, namely, attention, memory, verbal fluency, language, and visuospatial abilities. There are significant difference in total ACE-R test score between exposed group and control group [29]. Concetto et al. investigated exposure sample of 86 male Al welders and control group of 90 clerical staff came from the same company. The median age of exposure workers is 38 years and length of service is 22 years. The serum Al in the exposed workers was 24.19 μg/l and that in the control was 6.93 μg/l. The results showed exposed workers decreased cognitive with response with regard to attention and memory performance using WMS and the Stroop test. This study confirmed that occupational exposure to Al causes alteration in cognitive responses that are more evident in complex functions [7]. Lu et al. analyzed the relation between cognitive functions and tau-protein expression in peripheral blood lymphocytes of retired aluminum (Al)-exposed workers. Sixty-six retired Al potroom workers (age = 62 years, length of service = 30 years) and 70 unexposed controls (age = 61 years) were investigated. The serum Al in the exposed workers was 25.18 μg/l and that in the control was 9.97 μg/l. There is significant difference in total MMSE scores, orientation in time and place, short-time memory, and calculation ability. This study suggests that long-term exposure to Al may cause cognitive disorders [17]. Yang investigated 366 Al-exposed workers in aluminum potroom and assessed their cognitive function with Mini-Mental State Examination (MMSE). Serum Al in Al-exposed workers was 48.99 μg/l. This study suggested the total scores of MMSE decreased with the increase of serum Al level and long-term exposure to Al may cause MCI. MCI induced by Al was significantly associated with global DNA methylation in blood [28]. The above study of occupational population in various industries has found that occupational exposed Al may cause different degree of cognitive impairment in different aspects (see Table 5.1).

Table 5.1 Occupational exposure aluminum and cognitive impairment

Author	Publication year	Work type	Exposure years(years)	Sample size(population age)	Blood aluminum	Urine aluminum	Cognitive function impairment
Rifat	1990	Miners		Control 346			MMSE
				Exposed 261			CPM (colored progressive matrices test)
							SDMT(symbol digit modalities test)
Hosovski	1990	Foudary	19	Control 60 (42 years)	Control 58.09 μg/l	Control 7.25 μg/l	Complex reaction time
				Exposed 87(41 years)	Exposed 136.85 μg/l	Exposed 45.38 μg/l	Oculomotor coordination
							Sum of manipulative test
							Memory
							Coding
							Picture completion
							Object assembling
Sjogren	1996	Welder	17	Control 39 (40 years)	Control 1.0μg/l	Control 4.7μg/gCr	Tapping speed(non-dominant hand)
				Exposed 38 (39 years)	Exposed 3.0μg/l	Exposed 24.0 μg/gCr	Luria –Nebraska motor scale item3, item4
							Pegboard (Dominant hand)
Ritva Akila	1999	Welder	23	Reference 28 (38 years)	None	Reference 0.46 μmol/l	The memory for designs
				Low exposure 27 (37 years)		Low exposure 2.25 μmol/l	Difficult block design items
				High exposure 24 (41 years)		Highexposure 9.98 μmol/l	

Guiwen Guo	1999	Electrolysis, smelting, welding	17	Control 64 (40 years)	None	Control 17.73 μg/g Cr	Tension, depression, anger and fatigue
				Exposure 103 (37 years)		Exposure 41.79 μg/g Cr	Digit span forward
							Digit symbol
							Total pursuit aiming
Salvatore Polizzi	2002	Foundry	25(exposure years)	Control 32 (67 years)	Control 8.2 μg/l	None	The latency of P300, MMSE score, MMSE-time, CDT score and CDT-time
			11 (retirement years)	Exposure 64(68 years)	Exposure 14.1 μg/		
M. Buchta	2003	Welder	6	1999	1999	1999	Reaction time
				Reference 50	13.47μg/l	57.77μg/g Cr	
				Exposure 98	2001	2001	
				2001	6.4μg/l	52.4μg/g Cr	
				Reference 50 (36 years)			
				Exposure 97(37 years)			
He	2003	Electrolytic	15	Control 32		Control 26.84 μg/gCr	POMSC
				Exposed 34(35 years)		Exposed 40.08 μg/gCr	POMST
							SRT
							SRTF
							DSY
							PAC
							PA

(continued)

Table 5.1 (continued)

Author	Publication year	Work type	Exposure years(years)	Sample size(population age)	Blood aluminum	Urine aluminum	Cognitive function impairment
Buchta	2005	Welder	11	1999	Exposed	Exposed	Symbol-digit substitution
				Reference26	1999	1999	Block design
				Exposure 33	Preshift 9.6 μg/l	Preshift 92.1 μg/g Cr	Switching attention
				2001	Postshift 11.6 μg/l	Postshift 97.0 μg/g	
				Reference 26 (40 years)	2001	Cr	
				Exposure 33(43 years)	Preshift 10.6 μg/l	2001	
					Postshift14.3 μg/l	Preshift 90.1 μg/g Cr	
						Postshift 143.9 μg/g	
						Cr	
N. H. Zawilla	2014	Smelter	22	Control 51(46 years)	Control 4.43 μg/l		ACE-R
		Other production line					
				Exposed 54(46 years)	Exposed 20.27 μg/l		
Concetto	2014	Welder	16	Control 90(38 years)	Control 6.93 μg/l		Wechsler Memory Scale
				Exposed 86(46 years)	Exposed 24.19 μg/l		The Stroop Test
Lu xiaoting	2014	Potroom	30	Control 70 (61 years)	Control 9.97 μg/l		MMSE
				Exposed 66 (62 years)	Exposed 25.18 μg/l		Orientation in time and place
							Short-time memory
							Calculation ability
Yang xiaojuan	2015	Potroom	21	45	48.99		MMSE
							Orientation in time and place
							Short-time memory
							Attention and language skills

However, some studies are inconsistent with the above content. The role of Al in neurology is controversial. Letzel et al. showed no measurable cognitive decline in 32 dust-exposed workers in a German Al powder plant. He conducted two cross-sectional studies at an Al powder plant to evaluate possible nervous system effects from occupational Al exposure. The first study selected 32 Al dust-exposed workers and 30 unexposed control workers to test biological monitoring, neuropsychological testing, and evaluation of P300 potentials. Five years later, in the second investigation, all available workers from both groups (15 still exposed workers, 6 formerly exposed workers, and 15 unexposed workers) were reassessed using the same methods except for the P300 potentials. In the first study, Al concentration in plasma of exposed workers was 8.7 μg/l and that of the control was 4.3 μg/l. The urinary Al concentration in exposed group was 87.6 μg/g Cr and that in the control was 9.0 μg/g Cr. In the second study, Al concentration in plasma of exposed workers was 6.7 μg/l and that of the control was 4.3 μg/l. The urinary Al concentration in exposed group was 19.8 μg/g Cr and that in the control was 4.5 μg/g Cr. In the two cross-sectional studies, no significant exposure-related differences between the two study groups were found for the psychometric test and the P300 parameters [15]. Iregren et al. studied effects on the nervous system in a group of potroom and foundry workers, Al welders, and a small group of flake powder production workers. There were 119 smelters (age = 46 years, length of service = 15 years), 16 flake powder production workers (age = 35 years, length of service = 8 years), and 38 welders (age = 38 years, length of service = 15 years) as groups exposed to aluminum and 39 mild steel welders (age = 39 years) as control group. The serum Al in the smelters, flake powder production workers, and welders was 1.0 μg/l, 9.0 μg/l, and3.0 μg/l, respectively. That in the control was 1.0 μg/l. Al urine concentration in the smelters, flake powder production workers, and welders was 4.2 μg/g Cr, 59 μg/g Cr, and 24 μg/g Cr, respectively. That in non-exposure workers was 4.7 μg/g Cr. In the potroom and foundry workers, no effects on the nervous system related to Al exposure were detected, whereas the welders, who had been exposed to high levels of Al, showed a reduced performance, though not significant, in four tests of motor function and one pegboard test. However, in the highly exposed flake powder production workers, no effect on the central nervous system was seen [12]. Bast investigated 20 Al welders (age = 33 years, exposed to Al years = 8 years) and 20 construction workers as control group. The urinary Al concentration in welders was 0.18 μmol/lCr. Neuropsychiatric symptoms showed that welders reported more symptoms than referents did. Results of the static steadiness test showed that the welders performed statistically significantly better than the reference. The author explained the result may be a positive selection of workers with high manual skills into welding working and a possible job-related training effect on hand steadiness [4].

Variation in findings may be due to differences in the methods of assessment and the magnitude of exposure to aluminum. Certain methodological weaknesses have made it difficult to identify the role of aluminum in some of the conclusions drawn. For example, workers have been exposed to several potential toxicants other than aluminum, no measures of aluminum uptake or body burden were reported, no reference groups were used, or findings based on very small samples have been reported.

5.4 Conclusion

Occupational exposure to Al seems to have different effects on workers in different industries. The measurable effects on the central nervous system might only develop after a protracted exposure, and the intellectual domain, mainly affected, varies. The current focus in most neurotoxicological research is on low-level exposure, and consequently the impairments reported are often subtle because they reflect marginal or subclinical changes. The present study suggests that to detect, and more importantly understand, the earliest signs of central nervous system dysfunction it is necessary to apply a theoretically based cognitive approach to the analysis of performance especially for empirically sensitive tasks. The selection of test methods allowing component analysis to be undertaken offers the most likely prospect of showing the elementary cognitive processes underlying impaired performance. For the future, a cohort study of large sample occupational population exposed aluminum will be established, and neurobehavioral test will be standardized, so that the relation of occupational exposure aluminum and cognitive function is more convincing.

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