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How Much Chronic Exposure to Aluminum and its Toxicity Should be Concerned in Current Human Life?

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From the earliest days of food and environment regulation, the use of Aluminum (Al) in foods and water has been condemned. Al is an environmentally abundant metal entering various food additives and medicines which accumulates in the organism body (Pournourmohammadi *et al.*, 2008). Al has been suggested as a toxic agent in several pathological processes, including neurodegenerative diseases, mycrocytic anemia and osteoporosis (Mohammadirad and Abdollahi, 2011). Al causes its chronic toxicity through oxidative stress by generating reactive free radicals and disruption of cellular lipids, proteins and DNA. Generally, oxidative stress represents an imbalance between the production of reactive oxygen species and a biological system's capability to cleanse free radical intermediates or to repair the induced damages to cell wall and its nucleus. In the body, there are antioxidants that act against oxidants including non-enzymatic and enzymatic types. Non-enzymatic antioxidants include vitamins like C and E, selenium, zinc, glutathione, uric acid and ubiquinol. Enzymatic antioxidants include Superoxide Dismutase (SOD), Catalase (CAT) and Glutathione Peroxidase (GPx) (Abdollahi *et al.*, 2004). Therefore, the evaluation of oxidative stress has become an important aspect of investigation of Al toxicity (Malekirad *et al.*, 2010).

Recently, Mohammadirad and Abdollahi (2011) conducted a systematic review on Al and evaluated studies on oxidant/antioxidant imbalance induced by Al toxicity. They concluded that disruption of balance between body oxidant and antioxidant system plays a predominant role in Al toxicity, although they stated that exact mechanism of Al toxicity is not yet known. In the recent years, involvement of oxidative stress in the pathogenesis of many diseases including inflammatory bowel diseases (Rezaie *et al.*, 2007; Hosseini-Tabatabaei and Abdollahi, 2008), diabetes (Rahimi *et al.*, 2005), osteoporosis (Yousefzadeh *et al.*, 2006) and other oxidant-related diseases (Hasani-Ranjbar *et al.*, 2009, 2010; Ranjbar *et al.*, 2008) has been described.

As is often the case, these findings raise new concerns. For instance, if human life is inevitably exposed to environmental Al and if Al acts through oxidative stress and if oxidative stress is involved in pathogenesis of many human debilitating diseases, thus we have to expect involvement of Al in pathogenesis of much more diseases. Proving human exposure to Al by means of measuring its blood concentration and finding sources of Al exposure in the life of people suffering from debilitating diseases and then exploring the link between disease incidence and severity with exposure to Al will lead us to better definition of human diseases and their prognosis. Fortunately, Al has antidote that if used in proper time and dosage can control toxicity of Al (Nikfar *et al.*, 2011), if the source of exposure ends. Taking collectively, exposure to Al is an important concern in the present industrial life of human.

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