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PATHOPHYSIOLOGICAL MECHANISMS OF ALUMINIUM TOXICITY

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Aluminium forms about 8% of the earth's crust. It is most commonly found as bauxite ore, which is used for extraction of this metal. Aluminium has a high reactivity and forms compounds such as aluminium oxide, aluminium hydroxide, and potassium aluminium sulfate. Exposure of these compounds to oxidants leads to the formation of a superficial coating of aluminium oxide, which is highly resistant to corrosion and insoluble in water. However, acid rains have allowed the dissolution of these compounds and the entry of aluminium into biological systems. It can enter in human body through water, food, drugs, and inhalation of polluted air.

Once when accumulates in the body aluminium exhibits toxic effects on different organ systems: central nervous, respiratory, skeletal, hematopoietic, reproductive, digestive (liver), and integumentary system. Toxic systemic effects of aluminium are first observed in patients with kidney failure treated with medicines containing aluminium compounds which manifest as: dialysis encephalopathy syndrome, osteomalacia with osteodystrophy and microcytic anaemia.

Aluminium is on the top of a surprisingly short list of neurotoxic inorganic elements and their compounds. It is linked with development of neurodegenerative diseases, including autism, attention deficit disorders, amyotrophic lateral sclerosis, Alzheimer's disease, dementia, Gulf war syndrome, and Parkinsonism. Clinical and experimental studies suggest several possible mechanisms of toxic aluminium action on cells. Those are: increased production of oxidative stress, alteration of membrane function, disruption of intracellular signaling, and alteration or inhibition of enzyme functions.

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