INTRODUCTION

It is only 100 years since a practical procedure for the large scale production of aluminum was developed. Since that time, aluminum has found applications in construction, electrical equipment, furnishings, transportation, containers, pharmaceuticals, and many other items encountered in daily life. Exposure to aluminum is unavoidable since it is the third most common element in the Earth's crust, and industrial use is in the tens of millions of tons per year.

HUMAN EXPOSURE

In the 1920's attention was drawn to the potential health hazards of the aluminum present in city drinking water and various medicines, and possible industrial exposure (Spofforth, 1921; Betts, 1926).

Industrial aluminum exposure has not been extensively studied. There was one report over twenty years ago (McLaughlin et al., 1962) of a ball-mill room worker who developed encephalopathy and pulmonary fibrosis associated with inhalation of aluminum dust. Increased aluminum concentrations in urine of industrially-exposed individuals have been documented (Sjogren et al., 1983). A higher incidence of respiratory complaints has been reported among aluminum smelter workers (Chan-Yeung et al., 1983a). No significant effects have been found on musculoskeletal or hematological parameters in these workers (Chan-Yeung et al., 1983b). Studies of the incidence of cancers among aluminum industry workers have been inconclusive (Andersen et al., 1982; Rockette and Arena, 1983). Most reviewers of industrial aluminum exposure have concluded that the aluminum industry entails no greater occupational health hazard than other comparable industries (DeHamel, 1983).

Some domestic tap-water contains aluminum in high concentration, either naturally or because aluminum has been added as a flocculant in the purification process. Acid rain markedly increases the "natural" aluminum content of water.

In chronic renal failure, Mayor et al. (1981) have proposed that parathyroid hormone may contribute to the hyperaluminemia by increasing...
intestinal absorption and by influencing tissue distribution. The interrelations between parathyroid hormone and the homeostasis of aluminum and calcium remain to be defined. In aluminum poisoning, the secretion rate of parathyroid hormone may be either reduced (Canata et al., 1983) or unchanged (Biswa et al., 1982).

The impact of environmental aluminum on human health was comprehensively reviewed by Sorenson et al. (1974). In their review, they evaluated the total human exposure to aluminum from food, water, and atmospheric sources in a wide variety of geographical locations and concluded that the daily intake of aluminum from environmental sources ranges from 1 to 100 milligrams (mean = 5 mg).

METABOLIC MODEL

Gastrointestinal Absorption

Absorption of aluminum from the intestinal tract and its subsequent effect on mineral metabolism have been studied in rats (Berlyne et al., 1972; Feinroth et al., 1982); dogs (Henry et al., 1982); cows (Kappel et al., 1983); and rabbits (Thornton et al., 1983). These animal studies, as well as human studies (Gorsky et al., 1979; Greger and Baier, 1983a), have demonstrated a small net positive balance of ingested aluminum in mammals. In one metabolic balance study it was reported that aluminum was retained only when large (>125 mg/day) amounts were ingested and that there was no retention of aluminum at dietary intakes of 5 mg/day (Greger and Baier, 1983a). The effect of an increased dietary intake of aluminum on mineral metabolism in humans has been studied (Greger and Baier, 1983b; Spencer and Lender, 1979), but there is little agreement on the health effects of low to moderate exposure to aluminum.

Because intestinal absorption of aluminum at high levels of intake has been documented, the propriety of cookware has been questioned (Levick, 1980). The aluminum content of food has been shown to increase after cooking in aluminum pots (Koning, 1981; Lione, 1984; Trapp and Cannon, 1981), but the contribution from this source to total dietary intake is minimal. There has been recent concern over the effect of acid rain on the environmental availability of aluminum and resulting increased human exposure (Jackson and Huang, 1983). There is some evidence of higher incidences of certain neurological disorders in geographical areas that are rich in environmental aluminum particularly in drinking water. Non-environmental sources of aluminum exposure include surgical implants, industry, and pharmaceuticals (Drummond et al., 1983).

Aluminum-containing pharmaceuticals are an important source of aluminum. Significant amounts of aluminum are found in antacid preparations, which may contain up to 700 mg of Al(OH)3 containing 242 mg of elemental aluminum. This amount, in a single dose of antacid, represents about 50 times the average normal daily ingestion of aluminum from other sources. Berlyne et al. (1970) were the first to draw attention to aluminum-containing pharmaceuticals when they reported the finding of hyperaluminemia in renal failure patients who were being treated with aluminum hydroxide gels. The values were increased in some patients on oral treatment with aluminum-cycle resins and aluminum hydroxide, and in some patients on dialysis treatment who were not taking oral aluminum salts but who had been exposed to a dialysate with a relatively high aluminum content. Berlyne and co-workers proposed that the increased values in these patients was probably accounted for by considerable absorption of aluminum ions from the intestine and impairment of the normal efficient renal mechanism of excretion of aluminum. They subsequently reported the effects