Lithium Extends Lifespan

Lithium is an essential nutrient

Most people know lithium as the “drug” given to bipolar patients. In reality it is not a drug but a mineral, and in bipolar disorder it’s given in high doses: the target dose is usually 900 to 1,800 mg a day.

However, lithium is a required nutrient. “The available experimental evidence now appears to be sufficient to accept lithium as essential; a provisional RDA for a 70 kg adult of 1000 μg/day is suggested.” (1000 μg = 1 mg.)

This is, as can be seen, much lower than the dose given in bipolar.

Furthermore, low levels of lithium in drinking water have been associated with violence, suicide, and homicide. See, for example, Lithium in Tap Water and Suicide Mortality in Japan.

Lithium increases lifespan in humans and animals

A study looked at epidemiological evidence of an effect of lithium on human lifespan, and found it. Low-dose lithium uptake promotes longevity in humans and metazoans. Mortality rates were inversely associated with lithium concentrations in tap water; furthermore, the lower mortality rate remained after adjusting for suicide, showing that lithium provides some other health
benefit not strictly related to mental health.

Since this association does not show causality, the same authors used low-level lithium, at about the same concentration found in the tap water, and tested it on the worm \textit{C. elegans}. It extended their lifespan, showing causality.

\textbf{Lithium promotes autophagy}

\textit{Lithium seems to extend lifespan by promoting autophagy}, the cellular self-cleaning process that rids cells of junk and is crucial to lifespan extension. It does this by an mTOR-independent mechanism, meaning that it does not depend on fasting. Through autophagy, lithium has been found to \textit{delay progression of amyotrophic lateral sclerosis}.

\textbf{How much lithium do you need?}

As stated above, about 1 mg a day is a suggested RDA for lithium. Dose for bipolar patients are hundreds or thousands times higher, but there’s considerable risk of toxicity at those doses, while there appears to be little for low doses. A common formulation, \textit{lithium orotate}, provides 5 mg lithium.

I generally take one 5 mg tablet of lithium orotate once every few days.

\textbf{Lithium delays ALS progression through autophagy promotion}

\textit{Lithium delays progression of amyotrophic lateral sclerosis}

ALS is a devastating neurodegenerative disorder with no effective treatment. In the present study, we found that daily doses of lithium, leading to plasma levels ranging from 0.4 to 0.8 mEq/liter, delay disease progression in human patients affected by ALS. None of the patients treated with lithium died during the 15 months of the follow-up, and disease progression was markedly attenuated when compared with age-, disease duration-, and sex-matched control patients treated with riluzole for the same amount of time. In a parallel study on a genetic ALS animal model, the G93A mouse, we found a marked neuroprotection by lithium, which delayed disease onset and duration and augmented the life span. These effects were concomitant with activation of autophagy and an increase in the number of the mitochondria in motor neurons and suppressed reactive astrogliosis. Again, lithium reduced the slow
necrosis characterized by mitochondrial vacuolization and increased the number of neurons counted in lamina VII that were severely affected in saline-treated G93A mice. After lithium administration in G93A mice, the number of these neurons was higher even when compared with saline-treated WT. All these mechanisms may contribute to the effects of lithium, and these results offer a promising perspective for the treatment of human patients affected by ALS.

Full paper at the link. While none of the patients treated with relatively low-dose lithium died, 30% of the controls not treated with lithium died. Lithium works this way by the promotion of autophagy, which is the self-clearance mechanism of cells. In effect, the lithium allows the ALS patients to clear the pathological junk from their cells.

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**Low-dose lithium**

**Lithium: Occurrence, Dietary Intakes, Nutritional Essentiality**

Abstract

Lithium is found in variable amounts in foods; primary food sources are grains and vegetables; in some areas, the drinking water also provides significant amounts of the element. Human dietary lithium intakes depend on location and the type of foods consumed and vary over a wide range. Traces of lithium were detected in human organs and fetal tissues already in the late 19th century, leading to early suggestions as to possible specific functions in the organism. However, it took another century until evidence for the essentiality of lithium became available. In studies conducted from the 1970s to the 1990s, rats and goats maintained on low-lithium rations were shown to exhibit higher mortalities as well as reproductive and behavioral abnormalities. In humans defined lithium deficiency diseases have not been characterized, but low lithium intakes from water supplies were associated with increased rates of suicides, homicides and the arrest rates for drug use and other crimes. Lithium appears to play an especially important role during the early fetal development as evidenced by the high lithium contents of the embryo during the early gestational period. The biochemical mechanisms of action of lithium appear to be multifactorial and are intercorrelated with the functions of several enzymes, hormones and vitamins, as well as with growth and transforming factors. The available experimental evidence now appears to be sufficient to accept lithium as essential; a provisional RDA for a 70 kg adult of 1000 μg/day is suggested.
In a subsequent study [19], the mean scalp hair Li levels of incarcerated violent offenders in California were found to be 0.028 ± 0.029 μg/g, significantly lower than the 0.099±0.126 μg/g observed in hair of nonincarcerated controls, although this does not necessarily establish a causal relationship.