**Lithium sulfate**

CAS: 10377-48-7  
MF: Li$_2$SO$_4$  
FW: 109.9  
Solubility: soluble in water (1 part in 2.6 parts of water); almost insoluble in alcohol.

Li$^+$  
MW: 6.94

**Major uses**  
Lithium (Li) is used in psychiatry for the treatment of mania, endogenous depression and psychosis; and also for treatment of schizophrenia. Usually lithium carbonate (Li$_2$CO$_3$) is applied, but sometimes lithium citrate (Li$_3$C$_6$H$_5$O$_7$), lithium sulfate, or lithium oxybutyrate are used as alternatives [1, 2].  
Li has also industrial use, e.g. in alloys, as a catalytic agent, and as a lubricant [3].

**Human toxicity**  
Poisoning is categorized as being acute, chronic (when the patient was taking drug therapeutically), or acute-on-chronic. Daily therapeutic oral doses of lithium carbonate or lithium citrate range from 600 mg to 2400 mg [4].  
The symptoms of intoxication include nausea, vomiting, diarrhea, drowsiness, weakness, ataxia, blurred vision, tinnitus and seizures. At the severe intoxication, CNS depression, cardiac arrhythmias, epileptic seizures, blood circulation collapse, hypotension, kidney failure, coma, and death can occur [4, 5].  
The therapeutic blood plasma level of Li is between 4.2 and 9.7 mg/l; the toxic level is about 14 mg/l; and the lethal level is exceeding 35 mg/l [6].

*Carcinogenicity:* Li and its salts are not classified as a human carcinogens.

**Kinetic data**  
*Absorption:* Gastrointestinal absorption of lithium salts is rapid (within a few minutes) and complete following oral administration of tablets or the liquid form of lithium salt [2].  
*Kinetics* is biphasic [7].

*Bioavailability:* over 95% [5].

*Volume of distribution:* 0.6 l/kg [5].

*Distribution:* lithium diffuses quickly into the liver and kidney, but up to 8-10 days is required to reach equilibrium between serum and brain, bone and muscle (reviewed in [5]).  
*Therapeutic peak plasma levels* are reached within 30 min to 2 h; in overdose situation – up to 72 h [5].  
*The plasma half-life* at the overdose situation can vary between 30 and 100 h [2].  
*Plasma-protein binding:* <10% [5].
Elimination half-life: 3-12 h; 8-65 h at the overdose situation [7].

Passage of blood-brain barrier is restricted [7]. Distribution of Li to the brain can be delayed up to 24 h [8].

Li passes freely through the placenta membrane [9].

**Metabolism and excretion**

Li is not metabolized.

Because Li chemical similarity to sodium (Na\(^+\)) and potassium (K\(^+\)), it may interact or interfere with biochemical pathways for these substances and displace these cations from intra- or extracellular compartments of the body [2].

Li seems to be transported out of nerve and muscle cells by the active sodium pump, although inefficiently [9].

**Excretion** is predominantly by the kidney: about 80% of Li is reabsorbed at the proximal renal tubule [2]. At the therapeutic doses, approximately 97% of a single dose of Li is excreted in the urine within 10 days [4].

**Mechanisms of action**

Li produces many metabolic and neuroendocrine changes, but no conclusive evidence favors one particular mode of action [2]. For example, Li interacts with neurohormones, particularly the biogenic amines, serotonin (5-hydroxy tryptamine) and norepinephrine, which provides a probable mechanism for the beneficial effects in psychiatric disorders, e.g. manias (reviewed in [10]).

In the CNS, Li affects nerve excitation, synaptic transmission, and neuronal metabolism [10]. Li stabilizes serotoninergic neurotransmission [2].

Li may influence water balance in the body by reducing the capacity of the renal tubule to react to vasopressin [11].

Partial substitution for Na\(^+\) and K\(^+\) ions by Li\(^+\) ion can, possibly, disturb energy processes in the cells [7].

The exact mechanisms of poisoning are still poorly understood.

**Target organs**: CNS, kidney, cardiovascular system [7].

**References**


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