

***Lathyrus sativus* and human health**

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Grass pea (*Lathyrus sativus* L.), consumed since the Neolithicum, has unique qualities and poses unique risks. It can adapt to drought, moderate salinity and marginal soils and can fix nitrogen better than other food or feed crops, producing the cheapest protein. During famine it is a mixed blessing as a lifesaver but also as the cause of neurolathyrism when consumed as an exclusive staple food for over 2 months. In grass pea seed the level of ODAP (beta-N-oxalyl-L-alpha,beta-diaminopropionic acid), blamed as the causal agent of neurolathyrism, is greatly influenced by environmental factors.

Prolonged consumption of grass pea seed deficient in sulfur amino acids may deplete the body of antioxidants and increase the susceptibility for neurolathyrism. In young chicks, addition of methionine to the grass pea feed can prevent the neurological symptoms. In human neuronal cell lines, the neuro-activity of ODAP can be reduced by methionine. In newborn rats stress increases the incidence of neurolathyrism. Ample evidences indicate oxidative stress as a factor in the etiology of this disease.

Besides ODAP, grass pea seed also contains high amounts of homoarginine which sustains the production of nitric oxide longer than arginine and is considered as stamina strengthening. In combination with cereals or other foodstuffs richer in sulfur amino acids and antioxidants, grass pea can be a safe and perfectly healthy food, but like other legumes it should not be a staple food. The reputation of grass pea as “toxic plant” and ODAP as “neurotoxin” should be seriously re-evaluated.