Tryptophan for insomnia but also for headaches and strokes?

A SHORT PERSPECTIVE

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Insomnia is a multi-faceted disease which affects the worldwide population nearing a pandemic. Anything that amounts to less than 8 hours sleep nightly is considered chronic sleep disturbance. Sleep disturbances are not only characterised by reduced sleep but also by increased sleep and roots of the ailment are to be associated to multiple comorbidities. Often insomnia used to impact the geriatric over 60 years population, but it is now widespread across all ages. The most common causes in youth are iron-deficiency anemia, therefore mineral and vitamin deficiencies (especially B₁₂, B₆, B₃, D and E), but also anxiety, depression, trauma, serotonin and estrogen imbalances (due not only to menopause) or other conditions. The consequences of the lack of sleep are even greater in effect than the causes and include cognitive decline from neuronal loss but also vessel fragility which can lead to dementia and strokes, but also to fatigue, headaches, irritability, low mood and exhaustion. Genetics can be a factor, but stress is a major culprit too. Lifestyle change certainly improves and is an ignition to reducing stress, because as the famous stoic Seneca said "*mens sana in corpore sano*". With regards to lifestyle change, below is presented advice on dietary routines that could improve sleep disturbances.

Tryptophan an amino acid

Tryptophan is an amino acid found in a variety of foods: milk, chocolate, yogurt, tuna, nuts, strawberries and so on. It is also a precursor of serotonin a neurotransmitter which is associated with sleep, since its metabolic product is melatonin. Serotonin is also involved in emesis, and vessel constriction.

Tryptophan, is essential (Figure 1) and thus acts as the primary source for endogenous serotonin and melatonin. Bioavailability of tryptophan is a challenge for dietary supplements in the sense that delivering tryptophan in healthy amounts to the brain is not simple, not as an active ingredient 'enveloped' within a dosage form anyway. This is because although Tryptophan is relatively "unique" (there are isomers, but it is unique in its specific anabolism), its progenic forms from 'a couple' enzymatic reactions, serotonin and melatonin, are aspecific in their physiological metabolic pathways; both serotonin and melatonin receptors are in fact heterogenous, whence endogenous tryptophan molecules function unidirectionally and indirectly, they necessitate the conversion into serotonin (5-Hydroxytryptamine via the 5-hydroxytryptophan pathway) and later to melatonin to produce sleep or vessel dilation (reduced migraine) effects.

FIGURE 1. Structure of tryptophan. The charges across the structure of tryptophan are fairly distributed and counterbalance the electronegativity of the carboamidic bond atoms, i.e. the carboxy- and aminic groups^[1]. Serotonin, its biosynthetic product, has a hydroxyl group hanging from the C5 position and reduced carboxyl- to alkyl- group thus slightly increasing hydrosolubility, ultimately favouring brain-barrier crossings (as it is well known).

The tryptophan aspecificity can also lead to potentially toxic effects. It is why instead of producing or misusing supplements integrating tryptophan containing foods within the diet would be more beneficial. An exemplary change could be sipping a light hot chocolate or having a yogurt before bedtime instead whilst leaving fruits for lunch or break hour; or, having a cup of semi-skimmed milk at night.

By improving the quality and quantity of sleep, migraines would be reduced by the "natural" increase of serotonin, but fewer free radicals also would be released increasing the quality of oxygen. Balanced sleep hours would lead to improved mood, signifying less stress and anxiety, which would lead to the strengthening of small vessels, important in maintaining cognition and in the prevention of dementia.

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References

1. Pardis Tabaee Damavandi, Martin T. Dove & Richard W. Pickersgill (2017) A review of drug therapy for sporadic fatal insomnia, *Prion*, 11:5, 293-299, DOI: 10.1080/19336896.2017.1368937