Joseph Knoll, M.D. is a Hungarian neurochemist, pharmacologist and Nazi concentration camp survivor. Dr. Knoll is best known for developing the drug l-deprenyl (Selegiline), the first selective monoamine oxidase-B (MAO-B) inhibitor. L-deprenyl is derived from the endogenous trace amine beta-Phenylethylamine (PEA). Both of them are powerful mesencephalic enhancers, neuromodulators and neuroprotectors.

For more than half a century Dr. Knoll has conducted research and published numerous articles about his research with PEA as an endogenous mesencephalic activity enhancer key role in the operation of innate and acquired drives and its synthetic derivatives. They modulate, amplify and protect our brain’s circuitry. According to Knoll, enhancer regulation can be defined as the existence of enhancer-sensitive neurons in the brain capable of working in a split-second on a significantly higher activity level due to endogenous enhancer substances from his testing of beta-Phenylethylamine (PEA).

The endogenous enhancer substance PEA enhances the impulse-propagation mediated release of catecholamines and serotonin. Enhancer substances may have their own receptors on specific enhancer-sensitive neurons that facilitate the release of neurotransmitters depending on neuronal firing activity.

**Neuroamplifier of neurotransmitter signal strength activity**

Knoll and colleagues discovered through experiments is PEA and its cousin deprenyl and PEA are “Neuroamplifiers”. They enhance the electronic coupling in the synaptic gap junction of linked regions of cells for greater signal strenght in the pulses of neurotransmitter release by increasing the signal-to-noise ratio for stronger signal firing. In other word, PEA more efficiently couples the release of neurotransmitters to the electrical impulse that triggers their release. In so doing, this turns up the volume level of catecholamine nerve activity for enhancing their overall effects.

This can be of great importance for cognitive enhancement, of clinical importance in Parkinson’s disease and Alzheimer’s disease, where the nigrostriatal tract and mesolimbic-cortical circuits under-function and for effectively treating depression due to an under-activity of both dopamine and noradrenalin neurons. This is an important mechanism responsible for the stimulating effects of Modafinil and other stimulant drugs.

**Catecholamines keep the higher brain centers active**

After 45 years of research, Knoll has concluded that the regulation of lifespan must be located in the brain. His research convinced him that the role of the catecholamnergic neurons is to keep the higher brain centers in a continually active state. Their intensity dynamically changes within broad limits according to physiological requirements. Knoll’s research shows that catecholaminergic nerve activity peaks at sexual maturity, and then begins a long, gradual decline thereafter.

Knoll’s animal research has shown catecholaminergic activity, learning ability, sexual activity and longevity to be inextricably interlinked. According to Dr. Knoll continuous decline of the mesencephalic enhancer regulation during the post-developmental phase of life is in causal relationship with the age-related decline performance and contributes to the manifestation of age-related diseases.
Improve performance at any age, delay decline and extend health lifespan

Knoll argues that the quality and duration of life is a function of the inborn efficiency of the catecholaminergic brain machinery, “A high performing longer living individual has a more active, more slowly deteriorating catecholaminergic system than low performing, shorter living peers.” He states that the activity of our catecholaminergic system can be improved at any time during life. Therefore it is feasible to transform a lower performing, shorter living individual to a better performing, longer living one. It does matter how old you are, for performance improvement.

To postpone age related decline, extend lifespan and improve performance, Dr. Knoll recommends enhancing the activity of the catecholaminergic engine of the brain from sexual maturity until death via the administration of a daily dose of a mesencephalic enhancer substance. And PEA is the brains endogenous mesencephalic enhancer substance.

References


These statements have not been evaluated by the Food and Drug Administration. This product is not intended to diagnose, treat, cure, or prevent any disease.