Hypertension - Cause and Cure

Given the fact that sFlt1 can be verified as being IL-12 derived and having two other forms (Flt-2 and Flt-3) with the amino acids being phenylalanine - tyrosine and tryptophan, these findings enabled the use of quantum biology modeling to identify other near certain epigenetic equivalents; e.g. DNAJB3 and VEGF.

The ability to use bioelectric signaling (vibrations from magnetic beads) prompted us to conduct bioinformatic search to determine if vagus and/or trigeminal nerve stimulation was linked to preeclampsia in an ample number of research studies. It was! DIY!
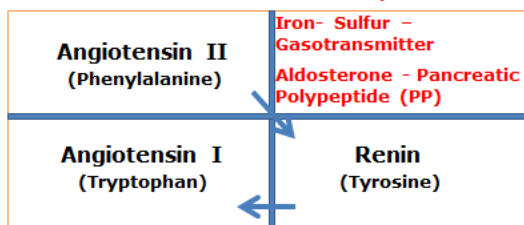
Summary

Our findings provide compelling evidence for an easily testable theory that vagus and/or trigeminal nerve stimulation can activate autophagy for mitigating kinase-like activities of these three aromatic amino acids that can result in hypertension.

Refer to the following for discussion with a qualified bioinformatics professional.

Modeling of Brain - Gut Conversions

Hormone Biosynthesis: Blood Pressure Regulation
Draft: For Discussion Only

Angiotensin II (Phenylalanine)	**Iron- Sulfur – Gasotransmitter** **Aldosterone - Pancreatic Polypeptide (PP)**
Angiotensin I (Tryptophan)	**Renin** (Tyrosine)

One of the primary causes of hypertension can be verified as the renin- angiotensin – aldosterone path. The fact is known but the sources and configuration has been an unknown.

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Magnetic Beads Hold Promise as Potential Preeclampsia Tx

Proof-of-concept study shows beads reduced sFlt-1 in blood of women with preeclampsia

- by Molly Walker, Staff Writer, MedPage TodayMay 13, 2019

Magnetic beads may have the potential to treat a molecule in the blood seen in high abundance in pregnant women with preeclampsia, an in vitro proof-of-concept study found.

Working with blood samples from women with preeclampsia, researchers were able to substantially reduce levels of soluble endothelial growth factor (sFlt-1), which is responsible for both angiogenic imbalance and endothelial dysfunction, reported Vassilis Tsatsaris, MD, PhD, of Cochin Hospital in Paris, in *Hypertension*.

They noted that during preeclampsia, a "massive amount" of sFlt-1 is released by the placenta, which inhibits both vascular endothelial growth factor (VEGF) and placental growth factor (PlGF), which are not only essential to the endothelium, or cells that line the interior surface of blood vessels and lymphatic vessels, but explain the endothelial dysfunction during preeclampsia.

"This was a proof of concept study and our approach aims to restore physiologic levels of angiogenic factors," Tsatsaris said in a statement.

The authors said their objective was to develop and provide a proof of concept of a selective and competitive apheresis -- or separating out one particular constituent from the blood. In this case, it was reducing sFlt-1 and increasing

free PlGF "to restore the physiological angiogenic balance in the maternal circulation."

Researchers grafted magnetic beads with molecules of sFlt-1, which competes with PlGF. Then to "increase sFlt-1 capture and optimize equilibrium displacement," they also used VEGF as a competitive ligand -- which has a more than 10 times greater affinity for sFlt-1 than PlGF, the authors said.

"This competitive biomimetic binding approach captures circulating sFlt-1 while releasing endogenous PlGF, thereby increasing the bioavailability of PlGF and potentiating its proangiogenic effects on maternal endothelial function," they wrote.

To test this approach in humans, researchers used plasma samples from pregnant women with preeclampsia. They found around a 40% reduction in sFlt-1 and an increase in free PlGF concentration from one-sixth to more than double in all the tested samples, the authors said.

"Thus, our microfluidic system with V-beads decreased the sFlt-1/PlGF ratio in plasma samples by 63% on average," they wrote.

The authors noted that the flow rates and fluid volumes used in their study are not compatible with application in pregnant women, but that the device could be of interest for a proof-of-concept in animal models. They said that their next steps with this research includes developing apheresis columns based on the ligand-based approach, "and usable in vivo with an animal model."