



September 17, 2025, Monthly Update

Eureka, We Found It!

Cellular Rejuvenation!

Preface

Since the mid-1980s, when FGF-1 was first discovered, many researchers have been amazed by what this naturally occurring human molecule can do. It can grow new blood vessels (angiogenesis), new neurons (neurogenesis), new skin, new hair follicles, and much, much more. Dr. Jacobs was at Washington University as part of the team that discovered FGF-1 in the 1980s. I became involved in 1998 with FGF-1 when medical doctors in Fulda, Germany, used FGF-1 to trigger the growth of new blood vessels to treat the number one cause of death in the world, small vessel heart disease. Merck, who patented FGF-1 in the 1980s, used it to heal wounds and more. Merck worked with FGF-1 for 20 years with no adverse events reported for the molecule and got amazing results. We and others used it in the brains of animals to reverse Parkinson's disease, to enhance recovery from stroke, and more. Three years ago, we started dosing Parkinson's disease patients with FGF-1 and have seen excellent improvements in most patients. Of the over 200 Parkinson's disease patients we dosed, 25 were also type 2 diabetics. After dosing with FGF-1, all ceased to be diabetic. We found many animal studies (conducted by others) that reported that type 2 diabetic animals reversed their diabetes in days and, in some cases, within hours with FGF-1. Zhittya Genesis Medicine has now decided to sponsor a 100-person type 2 diabetes Medical

Research Study in a Las Vegas clinic to reverify our data from the British Virgin Islands that FGF-1 can reverse this disease. Our Protocol was submitted and approved by an Institutional Review Board (IRB), as required by the US Government Health and Human Services Dept. (HHS) regulations. We should be dosing the first type 2 diabetic patients within the next few weeks.

While we have seen FGF-1 reverse type 2 diabetes in 25 humans and many animals, we still wonder WHY? Why does FGF-1 reverse type 2 diabetes? That question of “Why?” led us to the discovery of “glucose-sensing neurons in the brain.” These glucose-sensing neurons in the brain measure and manage our body's responses to blood sugar levels. These amazing glucose-sensing neurons measure our blood sugar levels, and through direct neural pathways directly to the pancreas and liver, they direct our body's reaction to balance the blood sugar levels. In last Month's Update, I reported on glucose-sensing neurons, and I recommend you search “glucose-sensing neurons and FGF-1.” I must have read 20 papers on glucose-sensing neurons and FGF-1. I think there may be several hundred more reports, if not thousands more medical research papers, on the topic of glucose-sensing neurons and FGF-1. I had my answer on why FGF-1 appears to reverse type 2 diabetes by it repairing the function of glucose-sensing neurons. Satisfied with myself one night while I was sleeping at 2:00 AM, I woke up remembering all the papers that I had read where the authors stated that FGF-1 was able to modulate the cell activities of the glucose-sensing neurons. My brain asked me, “What is cellular modulation?” I got up and started researching cellular modulation, and WOW, what I found!

Eureka! We found that FGF-1 rejuvenates cell function back to normal. If a cell is dysfunctional, that cell captures and takes FGF-1 into the cell to rejuvenate its intracellular functions.

FGF-1 repairs intracellular dysfunction. For years I have reported to you that our original math to treat Parkinson's disease patients was off, since the trillions of FGF-1 molecules we are introducing into the brain should have created millions of new blood vessels that dramatically enhanced the brain's function. In one prior Update, I stated we cannot figure out where 90% of the FGF-1 has gone. Now we have an idea. It is reported that the average human brain contains around 100 billion neurons. Estimates say that our brains could also contain 1 trillion glial cells! Our estimate is that we introduce about 3 trillion FGF-1 molecules when we dose up the nose. It is believed 10,000 FGF-1 molecules on a stressed endothelial cell's FGF-1 receptors will trigger a “foci” (new blood vessel growth). Dosing twice

a day is 6 trillion molecules, and for 6 days, is 36 trillion FGF-1 molecules. Divide that by the 10,000 needed to trigger a foci, and you have a potential 360 billion shots of growing a lot of new blood vessels to feed the starving areas of the brain. Take the same 36 trillion and divide by 100 billion neurons, and that is only 360 FGF-1 molecules per neuron, or only 36 per glial cell. That may explain why it is taking longer and taking much more FGF-1 than we originally thought. The aged brain seems to be starving for FGF-1.

FGF-1 is made in the brain by everyone. As we get older, does the brain make less FGF-1, therefore leading to cellular dysfunction and what we call “aging”? Are neurodegenerative diseases caused by FGF-1 deprivation?

For those of you who do not appreciate how blessed I am, on Monday, September 8, 2025, the Wall Street Journal had an article entitled “Ultrawealthy Pile Money Into Longer Lifespans.” It discusses the billions of dollars being invested by the ultra-rich to extend life. The second-to-last paragraph in the article states how their search for **rejuvenation of cells is the biggest buzz in this search.** Please tell those ultrawealthy billionaires, **“Eureka, Zhittya has found it!”**

Here is the link to the Wall Street Journal story: [zgm.care/cellularmodulation](https://www.wsj.com/articles/zgm-care/cellularmodulation)

Do you think I am lucky or just blessed?

Report from AI search

Question: What is, in biological terms, cellular modulation?

Cellular modulation, in biological terms, refers to the process by which cells regulate or adjust their functions, behaviors, or responses through various molecular and biochemical mechanisms. It involves the dynamic alteration of cellular processes such as gene expression, signaling pathways, metabolism, or membrane potential in response to internal or external stimuli. This allows cells to adapt to changing environments, maintain homeostasis, or perform specialized functions.

Key Mechanisms of Cellular Modulation:

1. Gene Expression Modulation:

- Cells modulate gene expression by upregulating or downregulating the transcription of specific genes in response to signals (e.g., hormones, growth factors, or stress).
- This can involve transcription factors, epigenetic modifications (e.g., DNA methylation, histone acetylation), or non-coding RNAs like

microRNAs.

2. Signal Transduction Modulation:

- Cells adjust their signaling pathways to fine-tune responses to external stimuli. For example, ligand binding to receptors can activate or inhibit pathways like MAPK, PI3K/Akt, or cAMP signaling.
- Modulation may involve feedback loops, cross-talk between pathways, or changes in receptor sensitivity.

3. Metabolic Modulation:

- Cells alter metabolic pathways to meet energy demands or adapt to nutrient availability. An example is switching between glycolysis and oxidative phosphorylation in response to oxygen levels.
- Enzymes, transporters, or cofactors may be regulated to modulate metabolic flux.

4. Membrane Potential and Ion Channel Modulation:

- In excitable cells (e.g., neurons, muscle cells), modulation occurs through changes in ion channel activity, affecting membrane potential and signal transmission.
- This can involve neurotransmitters, voltage-gated channels, or ligand-gated channels.

5. Immune Modulation:

- Immune cells (e.g., T-cells, macrophages) modulate their activity through cytokine signaling or receptor interactions to balance inflammatory or anti-inflammatory responses.
- This is critical in processes like immune tolerance or pathogen clearance.

Examples of Cellular Modulation:

- **Neuronal Modulation:** Neurotransmitters or neuromodulators (e.g., dopamine, serotonin) alter synaptic strength, affecting learning and memory.
- **Immune Response:** T-cells modulate their activity based on antigen presentation, adjusting proliferation or cytokine release.
- **Hormonal Regulation:** Insulin modulates glucose uptake in cells by triggering GLUT4 transporter translocation to the cell membrane.

In essence, cellular modulation is a broad concept encompassing the intricate ways cells fine-tune their activities to respond to physiological needs, environmental cues, or pathological conditions. It ensures cellular adaptability and coordination within multicellular organisms.

Question: Is there evidence of FGF-1 improving or being used during cellular modulation?

Yes, there is substantial evidence from the scientific literature that fibroblast growth factor 1 (FGF-1) is involved in cellular modulation, where it regulates and often improves cellular functions such as proliferation, differentiation, survival, and regeneration in response to stimuli. This occurs through activation of signaling pathways like Ras/Raf/MEK/ERK, PI3K/AKT, JNK/p38 MAPK, and STAT3/NF- κ B, which fine-tune gene expression, metabolic adjustments, and signal transduction. Below, I'll outline key areas with supporting evidence.

Role in Stem Cell Modulation

FGF-1 promotes self-renewal and inhibits senescence in stem cells, enhancing their adaptability and longevity. In mesenchymal stem cells (MSCs), it acts as a mitogen to maintain an undifferentiated state and multipotentiality, primarily via the PI3K/AKT-MDM2 pathway, which degrades p53 to prevent growth arrest and senescence. It also induces Sox2 expression to inhibit Wnt signaling, supporting proliferation without differentiation. In hematopoietic stem cells (HSCs), FGF-1 enhances proliferation and reconstitution potential when combined with factors like thrombopoietin (TPO) and stem cell factor (SCF), expanding multipotent progenitors. For neural stem cells (NSCs), it supports proliferation in NSC assays and increases NSC activity in brain regions like the subventricular zone, countering age-related declines in neurogenesis. Overall, FGF-1's modulation helps mitigate aging by preserving stem cell function and reducing senescence across tissues.

Role in Tissue Repair and Regeneration

FGF-1 improves cellular modulation during injury repair by promoting regeneration and functional recovery. In axonal regeneration, particularly after spinal cord injury (SCI), FGF-1 levels decrease post-injury, but exogenous administration or gene transfer enhances neuroprotection, axon regrowth, and remyelination. A clinical trial (NCT03229031) involving 46 SCI patients showed that FGF-1 treatment improved motor, sensory, and functional independence scores without major side effects. Studies in rat models confirm FGF-1 facilitates vagus nerve regeneration after crush injuries and promotes recovery via regulation of autophagy and reactive oxygen species. In broader tissue contexts, FGF-1 supports wound healing, angiogenesis, and organ development (e.g., heart, lens, liver, and lung), where it modulates cell survival and differentiation thresholds to specify tissue patterns.

Other Cellular Processes Modulated by FGF-1

- **Proliferation and Survival:** Via the PI3K/AKT pathway, FGF-1 regulates cell survival, migration, and processes like angiogenesis and neuroprotection in various cell types.
- **Differentiation and Gene Expression:** It influences adipogenesis, chondrocyte differentiation, and neurite outgrowth through pathways like MEK-ERK1/2-STAT3, while upregulating genes such as APOE, CCND1, and MMP9.
- **Cell Cycle and Apoptosis:** FGF-1 induces transient G2 arrest in chondrocytes and mediates apoptosis under stress via JNK and p38 MAPK pathways.

In summary, FGF-1 is actively used in cellular modulation across developmental, regenerative, and pathological contexts, often improving outcomes like enhanced proliferation, reduced senescence, and better tissue repair. Its therapeutic potential is evident in applications such as SCI treatment and stem cell therapies.

End of AI Report

Zhittya's Medical Research Studies Funding

Funding for the Medical Research Study, to “Reverse Type 2 Diabetes,” has started. We have about 70% committed of the \$5 million we wish to raise to fund the first 100-person study in Las Vegas. I believe we will be able to finish the funding for the type 2 diabetes study within 90 days. As we develop more data that Zhittya may succeed in reversing type 2 diabetes, investors are liking the potential 10 times return on their money more. I hope to have results in the 1st or 2nd quarter of 2026.

With the warm reception we have had from investors about the type 2 diabetes medical research funding, I have decided to start now the Medical Research Study for healing chronic wounds. We are offering the same ten times upside as a royalty of the cash Zhittya earns from the sale of the FGF-1 medicine to heal chronic wounds. The difference is that we will pay 50% of the cash receipts to the investors, until they max out at 10 times their investment. We are doing this since I believe, (1) we have 40 years of data, which shows me a 99.9% likelihood of success; (2) nonhealing wounds are a major unmet medical need (estimated 6 million in the USA every year alone); and (3) the drug is delivered topically, which should allow it to move more quickly to approval. I believe if we start dosing in October 2025 and dose the 100th patient in January 2026 (with an estimated 4 to

5 months to heal the worst of the worst wounds), that should give us results by July of 2026. Zhittya should have a wonderful announcement for the millions of people being harmed by a lack of treatment for their non-healing chronic wounds.

Medical Research Studies in Nevada

If you have an interest in learning about the Medical Research Study Funding investment opportunity (minimum investment \$25,000), please email me at: Dan333@aol.com or call me at 702-802-8373.

Cross Border Medical Tourism

Cross Border Medical Tourism services are for those people who want to be considered for Zhittya's future clinical trials or its current Medical Research Studies. Cross Border's focus is to fill all available slots in the Medical Research Studies in the British Virgin Islands and The Bahamas, while we wait for other clinics to come online.

Cross Border is working on filling the available slots for the next BVI Medical Research Studies (intranasal FGF-1 for Parkinson's disease) on the following dates:

Cohort #41: October 24 – 29, 2025

Cohort #42: December 3 – 8, 2025

Please get in touch with Sam Cross at Cross Border Medical if you wish to be considered for the BVI cohorts. Sam's email address is:

sam.crossbordermedical@gmail.com.

In addition, if you are interested in participating in a functional MRI brain scan study, which will take place at Washington University in St. Louis, please let Sam know. There are a finite number of slots for the imaging studies. These studies will look at brain blood flow before and after treatment with FGF-1.

Conclusion by Dan Montano

This is a short Update for Zhittya this time. We have so much going on we are operating over our limits. We are creating a new manufacturing lab in Reno, Nevada (to make Zhittya the biggest manufacturer of FGF-1 in the world), new devices for dosing FGF-1 up the nose, medical research studies for Parkinson's disease, stroke recovery, dementia, type-2 diabetes, chronic wounds, and a lot more. Every time we chase a question regarding the utilization of FGF-1, we are overwhelmed with the data that we find. Search "biologic cell modulation & FGF-

1” and have a shock at how much data there is. We have missed that data for the last 27 years, and it seems that many others have also. In our type 2 diabetes study, we have added tests of liver and pancreas function, since a few months ago a study (done by others) said that the FGF-1 enhanced the functions of both of those organs. They speculated that the FGF-1 had enhanced the cellular modulation of those two organs. How did the FGF-1 get there? The blood system? Not likely, as FGF-1 has a short half-life in the bloodstream. We introduce FGF-1 into the brain by FGF-1 crawling along the nerves, like the olfactory nerve. It is speculated that FGF-1 may crawl down the nerves that go from the glucose sensing neurons in the brain directly into the pancreas. We do not know if that is true, but we know we need to find out.

We have so much going on, and I believe that in the new year, 2026, when you learn what we have accomplished, you will be amazed. It is great to have so many wonderful things to work on.

Eureka, we found it!

FGF-1 is the molecule that rejuvenates, modulates, or, in just plain words, “fixes your individual cells.” I believe repairing brain cells is a good thing.

I pray we all succeed! Bless you all!

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