Casual Friday Series

# Regen Support Kit 101: Multiple Applications

A BIOGENETIX CLINICAL PRESENTATION

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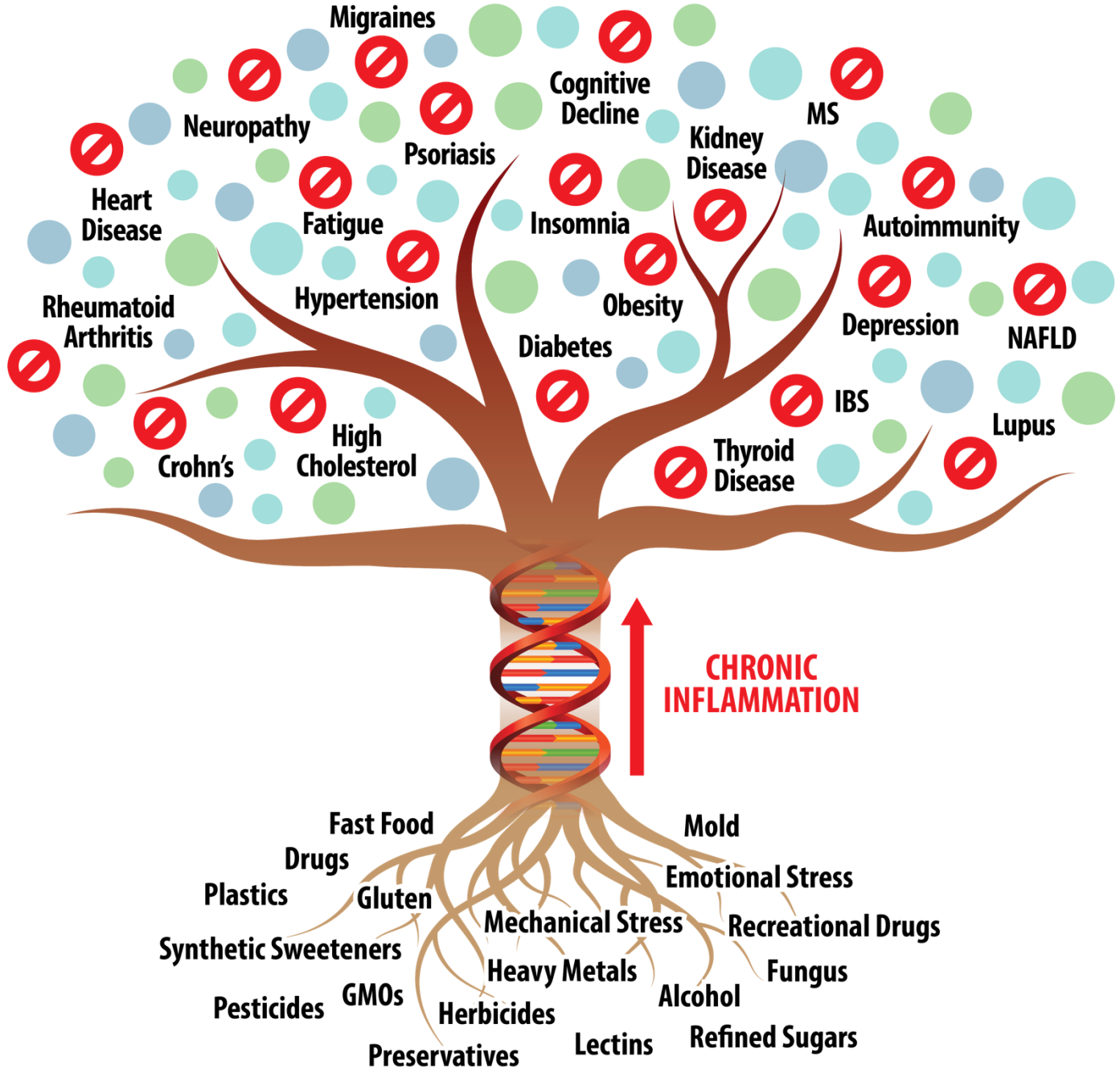
Migraines
Neuropathy
Psoriasis
Cognitive Decline
MS
Kidney Disease
Heart Disease
Fatigue
Insomnia
Autoimmunity
Rheumatoid Arthritis
Hypertension
Obesity
Depression
NAFLD
Diabetes
IBS
Lupus
Crohn's
High Cholesterol
Thyroid Disease
CHRONIC INFLAMMATION
Fast Food
Drugs
Plastics
Gluten
Mold
Emotional Stress
Mechanical Stress
Recreational Drugs
Synthetic Sweeteners
Heavy Metals
Fungus
Pesticides
GMOs
Herbicides
Alcohol
Preservatives
Lectins
Refined Sugars

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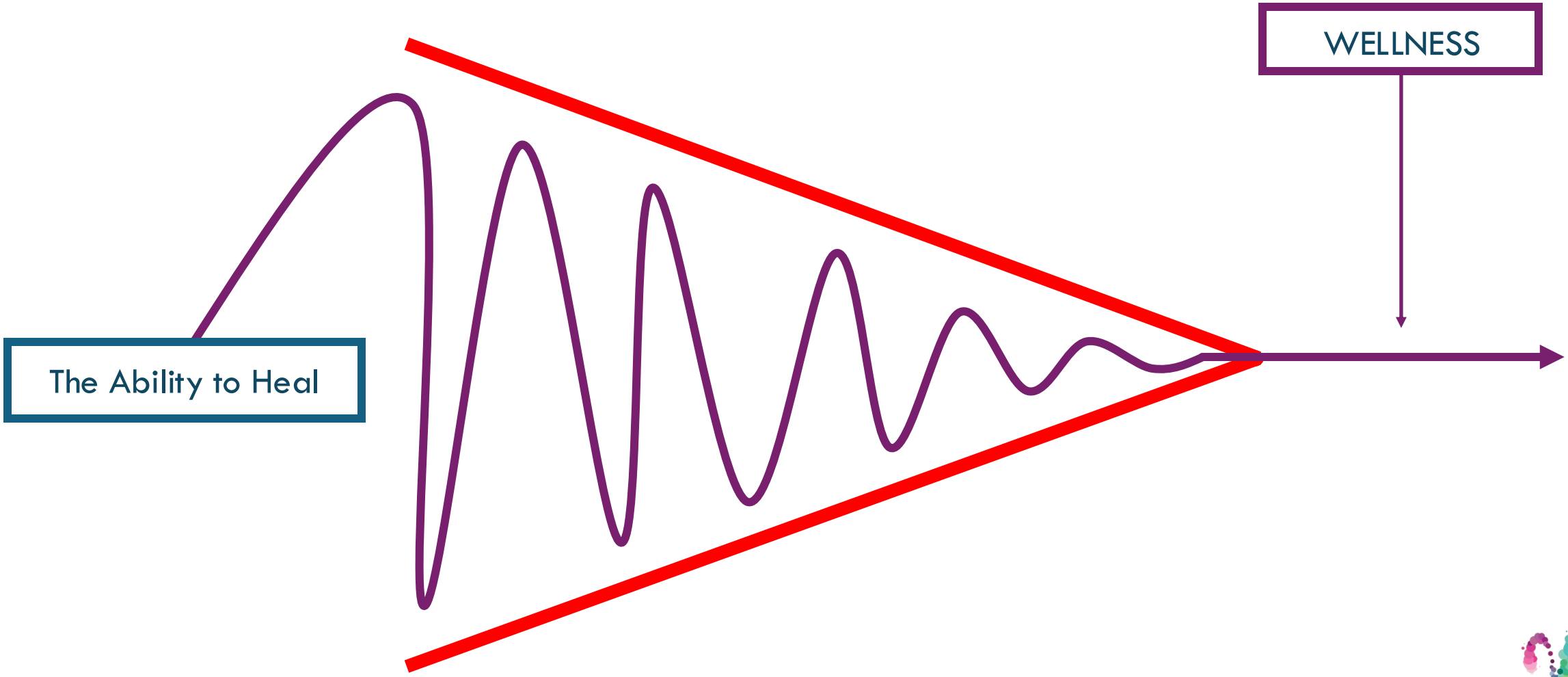
# The Wedge Protocol

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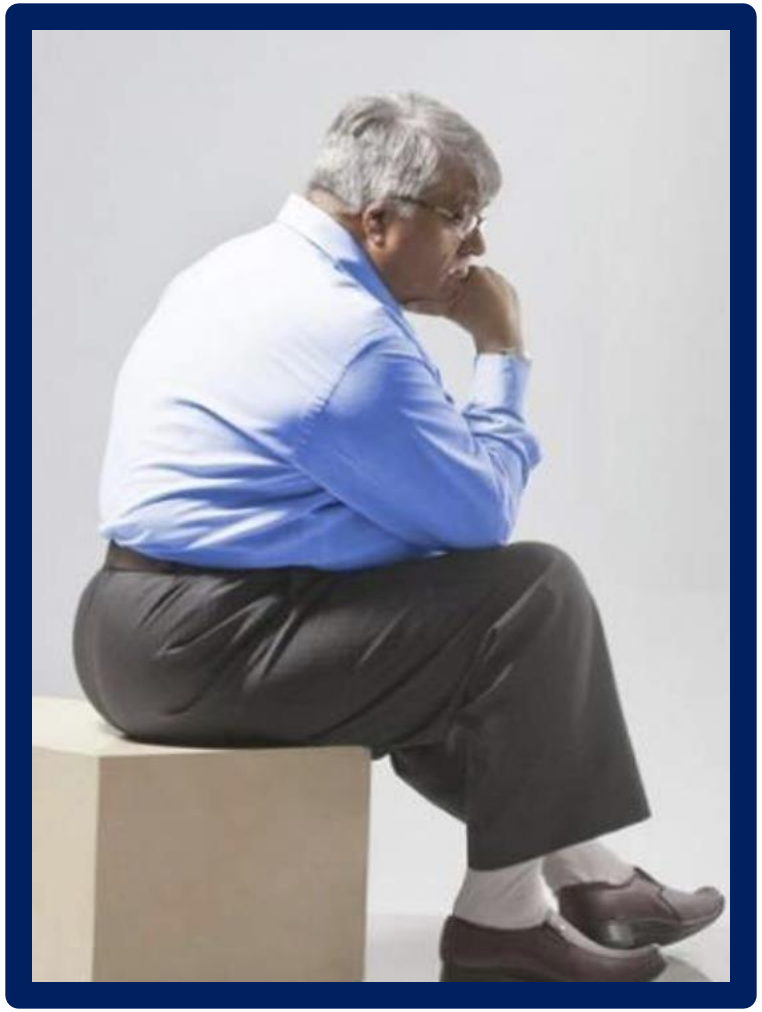


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CONCISE REVIEW | Free Access

# Stem cell homing: From physiology to therapeutics

Jane L. Liesveld ✉, Naman Sharma, Omar S. Aljitawi

First published: 11 June 2020 | https://doi.org/10.1002/stem.3242 | Citations: 156

Stem cell homing is a multistep endogenous physiologic process that is also used by exogenously administered hematopoietic stem and progenitor cells (HSPCs). This multistep process involves cell migration and is essential for hematopoietic stem cell transplantation. The process can be manipulated to enhance ultimate engraftment potential, and understanding stem cell homing is also important to the understanding of stem cell mobilization. Homing is also of potential importance in the recruitment of marrow mesenchymal stem and stromal cells (MSCs) to sites of injury and regeneration. This process is less understood but assumes importance when these cells are used for repair purposes. In this review, the process of HSPC and MSC homing is examined, as are methods to enhance this process.

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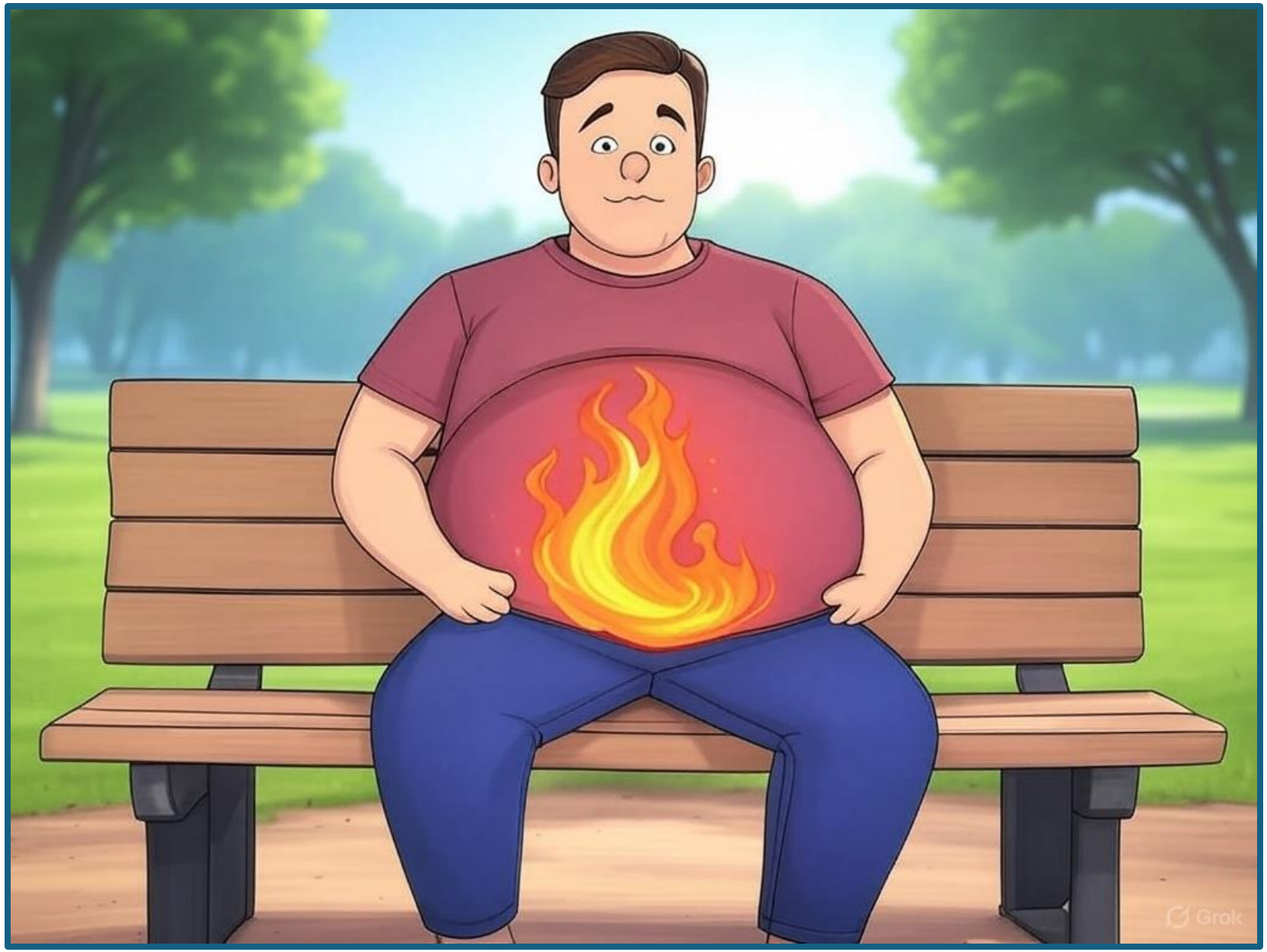


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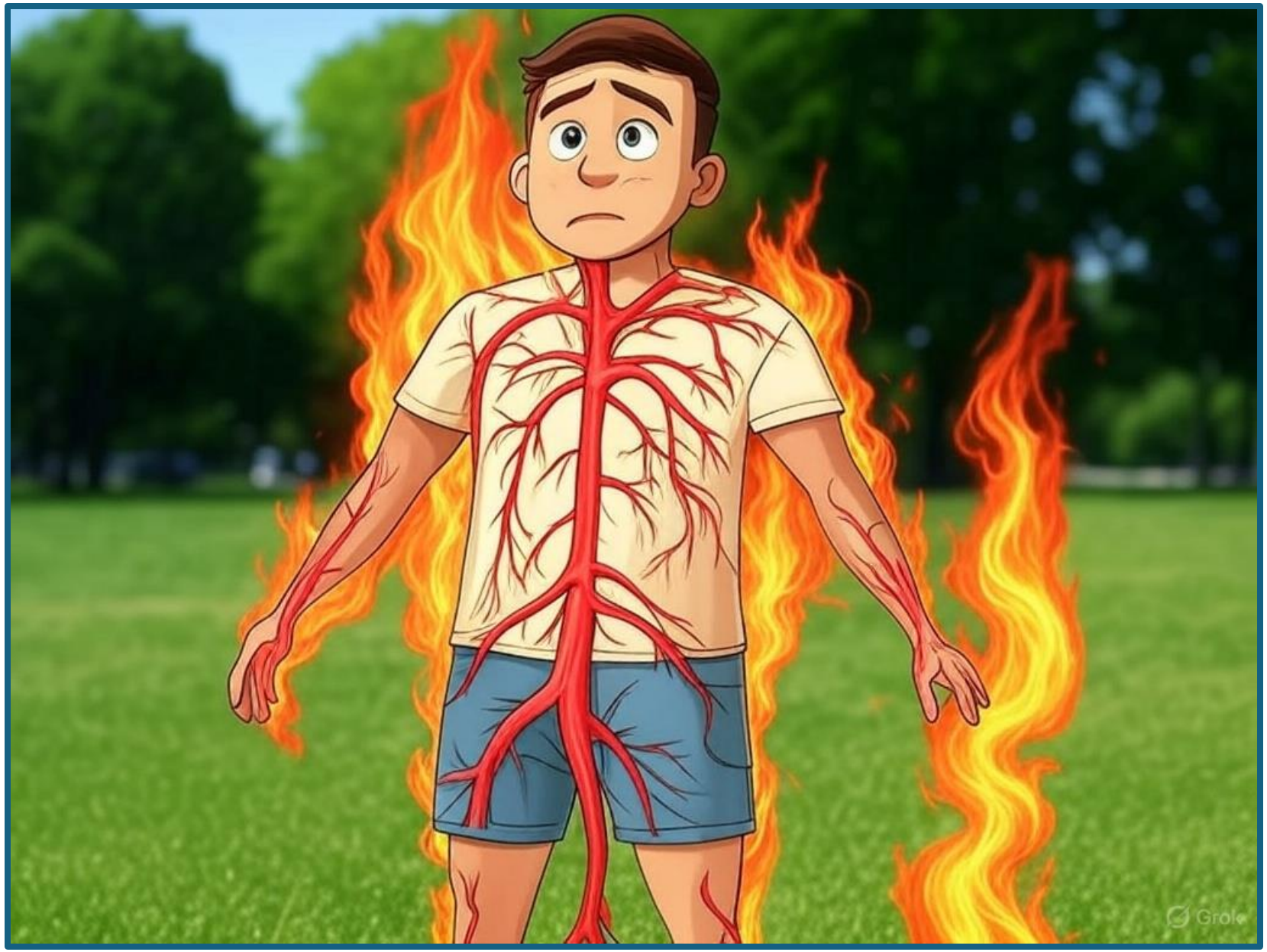


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| TEST NAME | CURRENT RESULT | PREVIOUS RESULT | CURRENT RESULT / PREVIOUS RESULT | REFERENCE |
|---|---|---|---|---|
| Arsenic* | 48.62 | | 0 11.9 52 | ≤52 ug/g |
| Beryllium* | 0.25 | | 0 0.2 0.76 | ≤0.76 ug/g |
| Cadmium* | 0.63 | | 0 0.29 0.8 | ≤0.8 ug/g |
| Cesium* | 6.82 | | 0 6.37 10.3 | ≤10.3 ug/g |
| Lead* | 0.72 | | 0 0.52 1.16 | ≤1.16 ug/g |
| 2,2-bis(4-Chlorophenyl) acetic acid (DDA) | 14.56 | | 0 7.9 19 | ≤19 ug/g |
| Butylparaben* | 0.47 | | 0 0.25 4.39 | ≤4.39 ug/g |
| Dimethyl phosphate (DMP)* | 28.57 | | 0 9.1 33.6 | ≤33.6 ug/g |
| Dimethylthiophosphate (DMTP)* | 8.54 | | 0 5.91 33.7 | ≤33.7 ug/g |
| Triclosan (TCS)* | 66.49 | | 0 29.9 358 | ≤358 ug/g |

* Indicates NHANES population data reference ranges.

72 yo male, dm2, hbp, hypercholesterolemia, NAFLD

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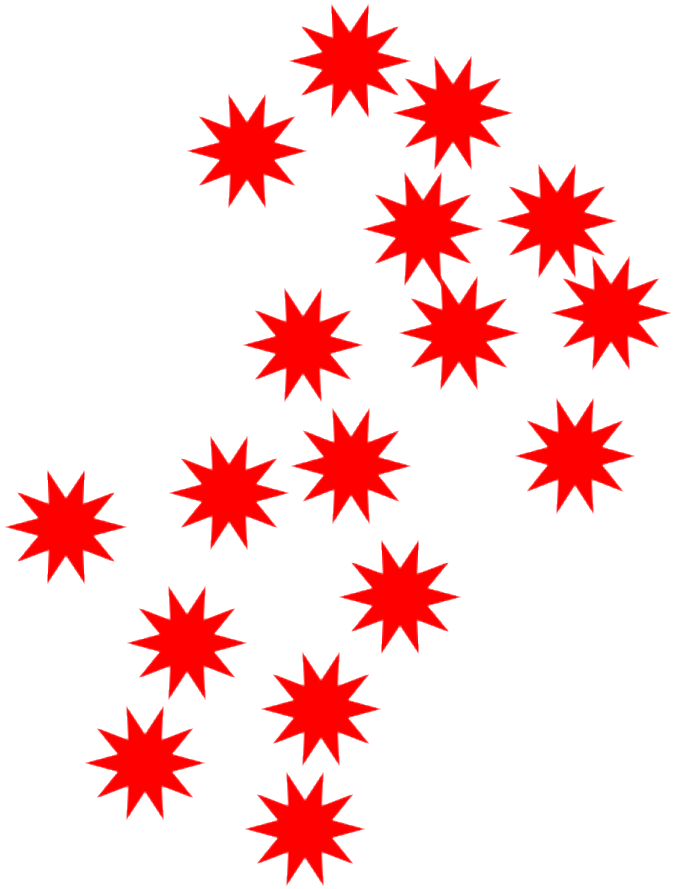
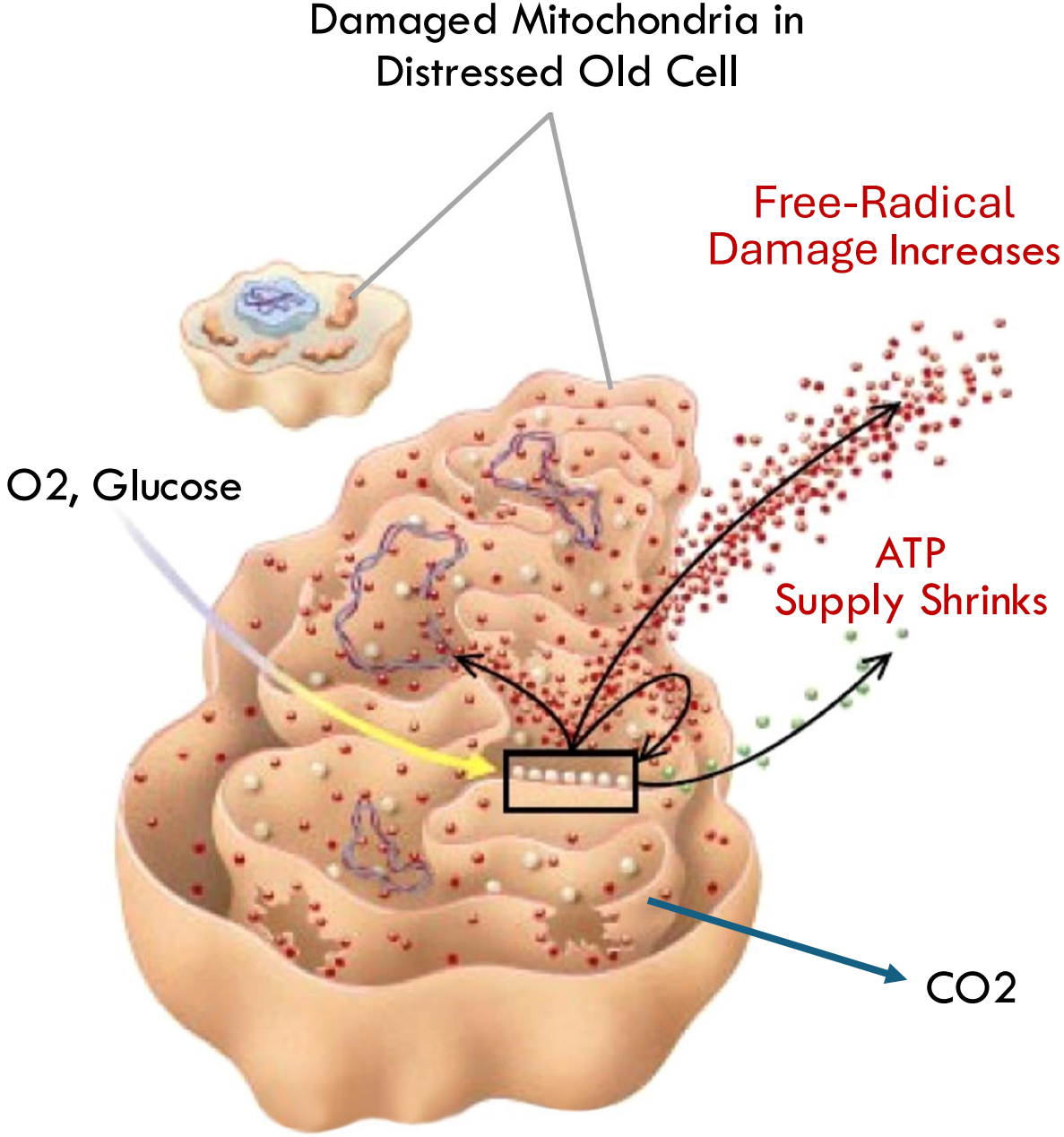
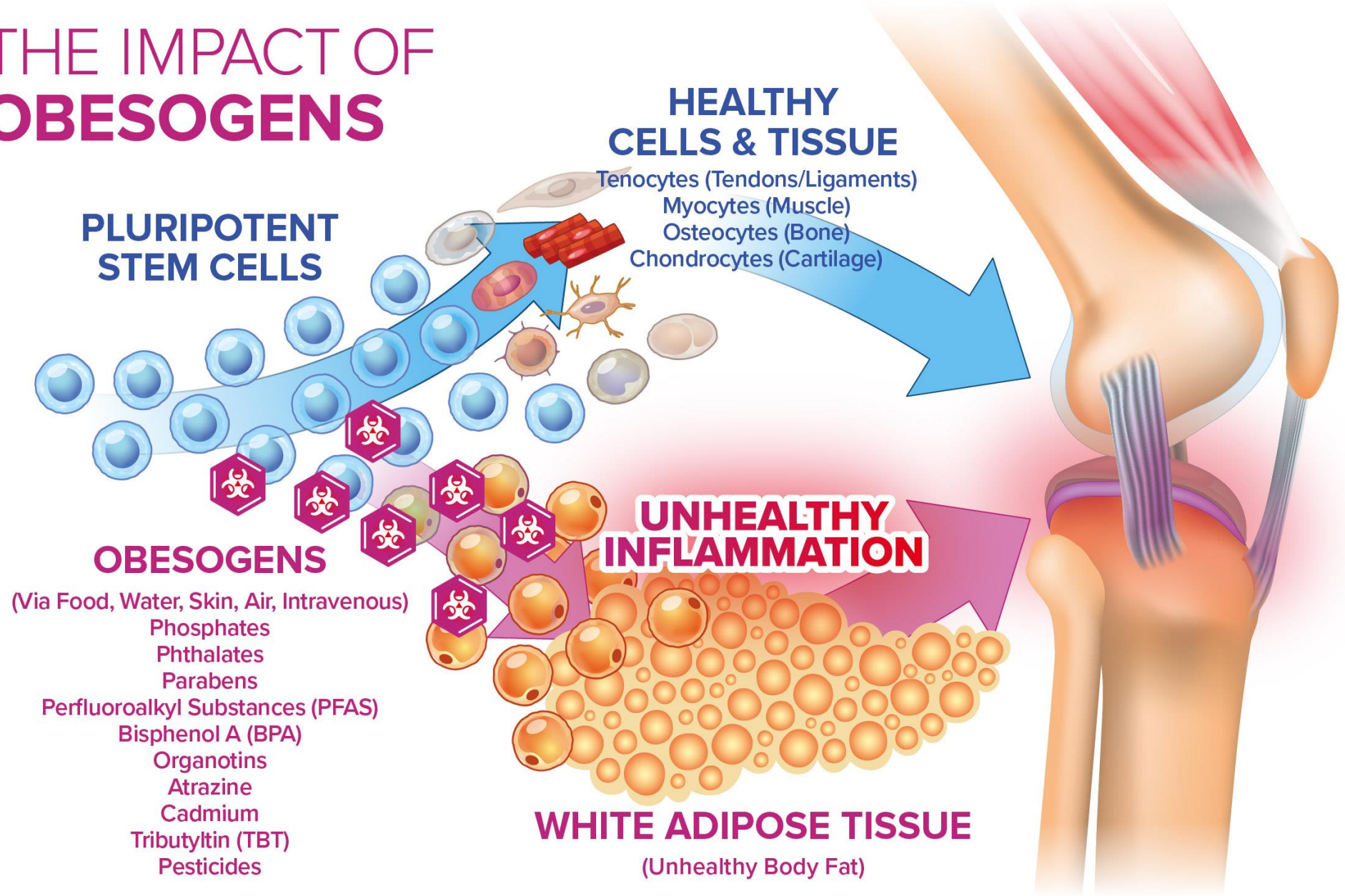
= Glucose Molecule



INSULIN



# THE IMPACT OF **OBESOGENS**

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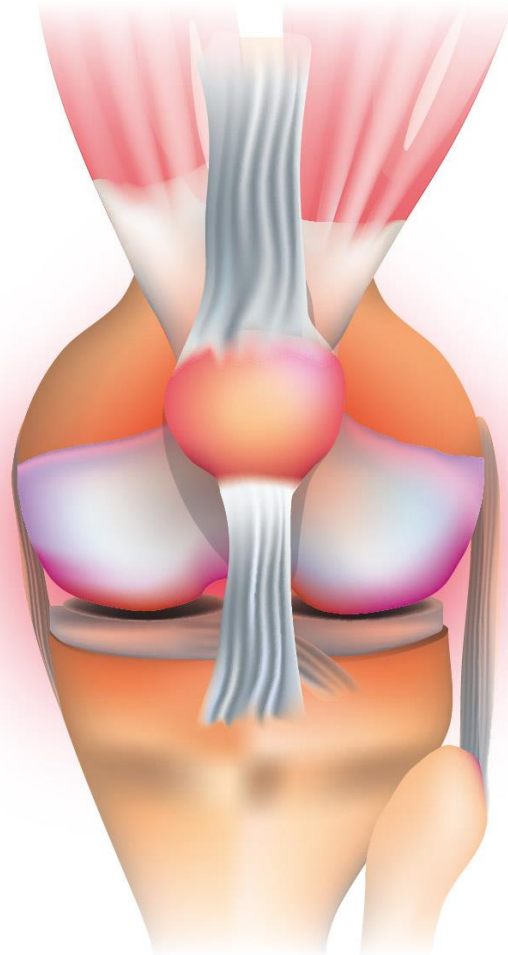
# THE **ADIPOKINE** SPIRAL



1
Obesogens Drive
Stem Cells into Adipocytes

2
Adiposity Increases

3
Increased
Adipokine Activity

4
Triggering
Increased
Unhealthy Inflammation

5
Ongoing
Tissue Damage

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# Adipokines

Adipokines are proteins produced by adipose tissue that can be pro- or anti-inflammatory. Some examples of adipokines include:

- **Leptin**: A pro-inflammatory adipokine that comes from adipocytes
- **Resistin**: A pro-inflammatory adipokine that comes from adipocytes and peripheral blood mononuclear cells
- **Adiponectin**: An adipokine that can be anti-inflammatory
- **Visfatin**: An adipokine that can be pro-inflammatory
- **Lipocalin-2**: A pro-inflammatory adipokine that comes from adipocytes and macrophages
- **Interleukin 6**: An adipokine that can be pro-inflammatory
- **Tumor-necrosis factor**: An adipokine that can be pro-inflammatory
- **Interleukin 10**: An adipokine that can be anti-inflammatory
- **Transforming growth factor-β**: An adipokine that can be pro-inflammatory

Adipokines can affect insulin resistance, lipid and glucose metabolism, and inflammation. An imbalance of adipokines can lead to metabolic syndrome, type 2 diabetes, and cardiovascular disease.

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# Biogenetix Regenerative support Kit+


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Arthritis Res Ther. 2009 Mar 18;11(2):R43. doi: 10.1186/ar2651

# Oral phosphatidylcholine pretreatment alleviates the signs of experimental rheumatoid arthritis

The present results provide evidence that an increased dietary PC uptake prior to CIA is associated with significantly enhanced anti-inflammatory protection. CIA is a widely used, standardized tool for the investigation of chronic, autoimmune RA with polyarthritis and subsequent cartilage and bone erosions [32]. In our experiments, the RA model provided accurate measures for clinical and histological signs of joint inflammation and for simultaneous quantification of the microhemodynamics in the synovial microcirculation. The effects of PC intake were observed at different stages of the disease, and the results revealed that prophylactic oral PC supplementation ameliorated the CIA-induced pain and many of the clinical signs of inflammation. Moreover, histological evaluation indicated considerably

My Summary: investigated the impact of phosphatidylcholine on the microcirculation in arthritic joints. It was observed that phosphatidylcholine could prevent the neovascularization associated with arthritis, thereby possibly reducing further joint damage. The compound also ameliorated tissue damage and reduced the expression of inducible nitric oxide synthase, which is linked to inflammation.

critical time for PC involvement is during the onset of CIA.

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J Cell Physiol. Author manuscript; available in PMC: 2013 Sep 1.

*Published in final edited form as:* J Cell Physiol. 2012 Sep;227(9):3216–3224. doi: 10.1002/jcp.24012

# Vitamin C Treatment Promotes Mesenchymal Stem Cell Sheet Formation and Tissue Regeneration by Elevating Telomerase Activity

FL Wei [1], CY Qu [2], TL Song [1], G Ding [1], ZP Fan [1], DY Liu [1], Y Liu [1], CM Zhang [1], S Shi [2,*], SL Wang [1,3,*]

Cell sheet engineering has been developed as an alternative approach to improve mesenchymal stem cell-mediated tissue regeneration. In this study, we found that vitamin C (Vc) was capable of inducing telomerase activity in periodontal ligament stem cells (PDLSCs), leading to the up-regulated expression of extracellular matrix type I collagen, fibronectin, and integrin β1, stem cell markers Oct4, Sox2, and Nanog as well as osteogenic markers RUNX2, ALP, OCN. Under Vc treatment, PDLSCs can form cell sheet structures because of increased cell matrix production. Interestingly, PDLSC sheets demonstrated a

My Summary: vitamin C was found to induce telomerase activity in ligament stem cells, leading to improved extracellular matrix production, which is essential for tissue regeneration. The study also showed that vitamin C treatment could enhance the regeneration of soft tissue defects.

tissue regeneration.

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PeerJ. 2018 Apr 27;6:e4641. doi: 10.7717/peerj.4641

# Collagen overlays can inhibit leptin and adiponectin secretion but not lipid accumulation in adipocytes

Sherri L Christian [1,✉], Nikitha K Pallegar [1], Robert J Brown [1], Alicia M Viloria-Petit [2]

Regardless of the mechanism, we have clearly shown that manipulations to the preparation of Type I collagen can result in significant and specific alterations to adipocyte physiology and function. Thus, selective alteration of adipokine secretion without impacting TG storage may be possible using collagen matrices. Adapting this method to *in vivo* procedures could potentially be used to modify appetite-regulating hormones without diminishing the essential role of adipocytes to store free fatty acids, which are toxic in high abundance or when deposited ectopically (Bays, Mandarino & Defronzo, 2004).

My Summary: this research has shown that Type I collagen overlays can selectively alter adipokine secretion in adipocytes without significantly affecting lipid accumulation. Important implication when combined with vit C for both ligamentous and tendon-based intervention.

manipulated while not substantially impairing TG synthesis in developing adipocytes by use of different collagen preparations and cell culture plates. These findings may provide researchers a new way to affect adjacent cells or tissue by selectively manipulating adipocyte function *in vitro* or, potentially *in vivo*.

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