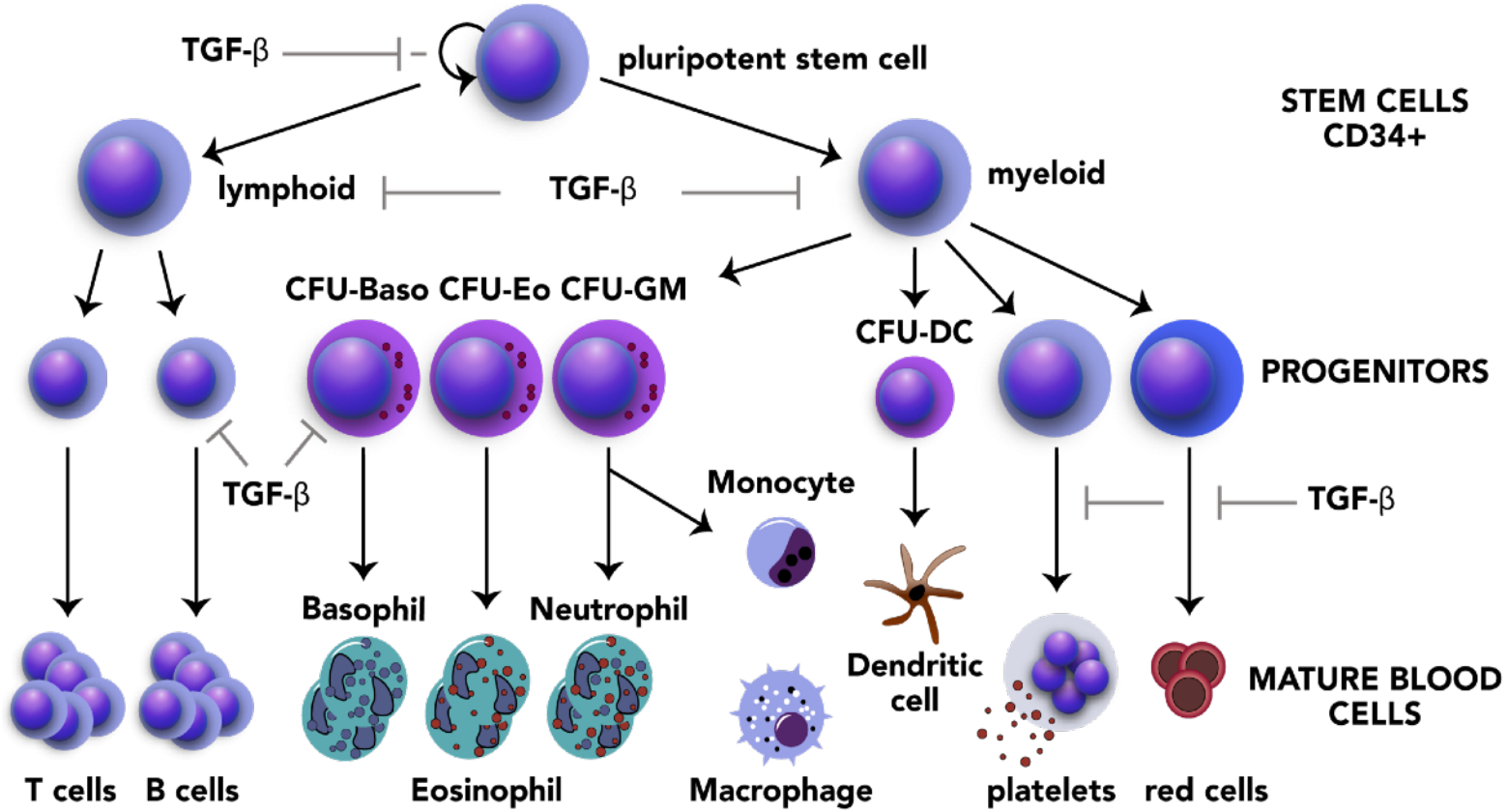
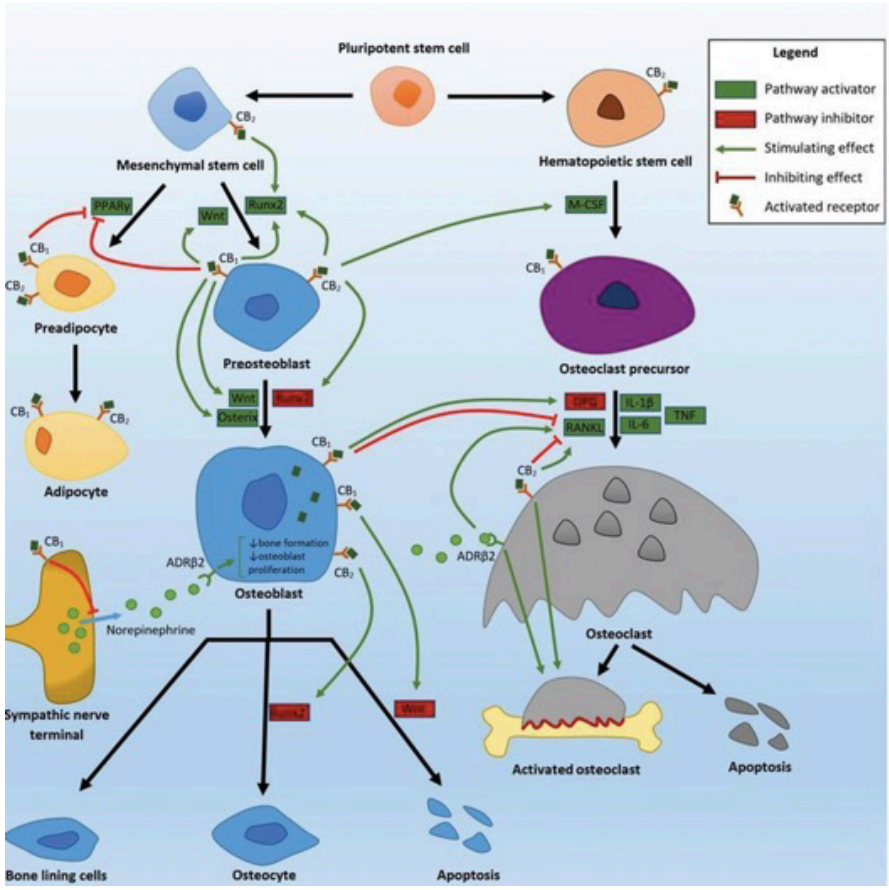
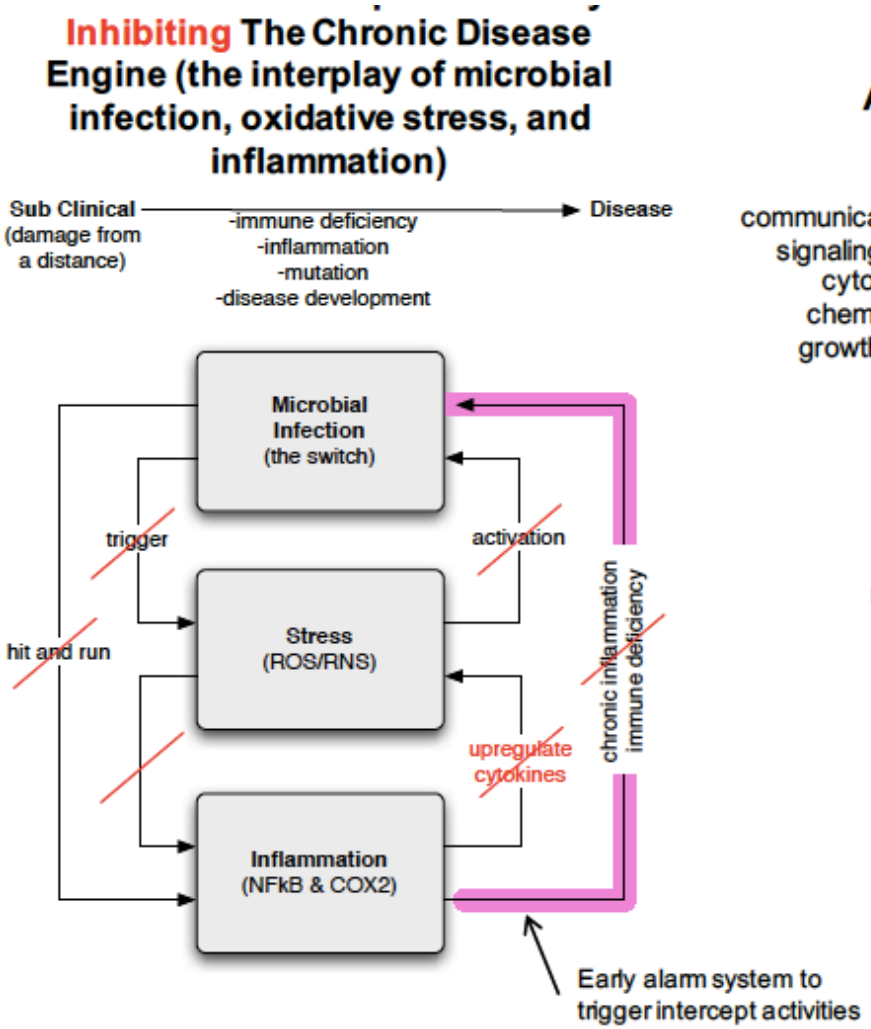
TGF-B Master regulator of Hematopoietic Stem Cell

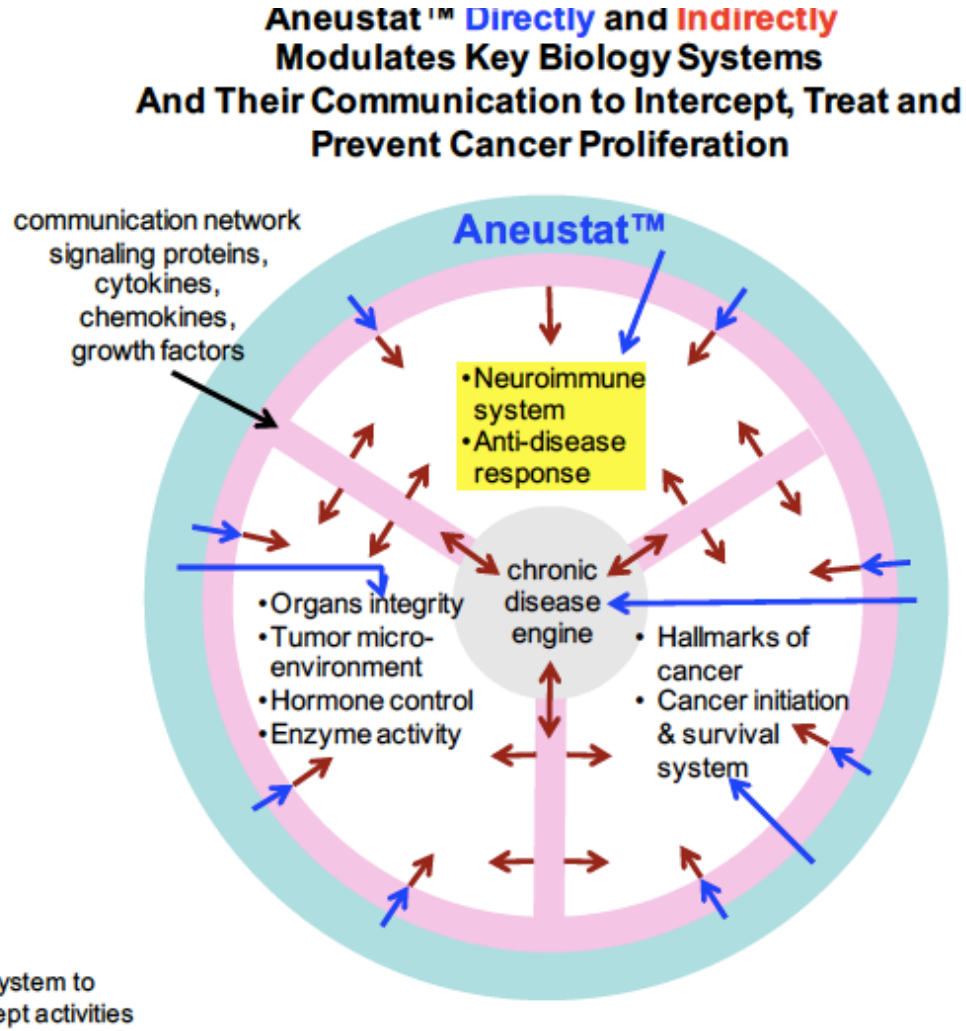
Accelerated Myelopoiesis=INFLAMMAGING=AIDS= COVID 19





GENYOUS/OMNITURA Anuestat™: An Improved Pharmacological Paradigm THE SMART Platform for combination therapy for Cancer Cardiovascular & Neuroimmune Disease





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- Multivalent MOA
- Adaptive Arsenal
- Regulation/restore Homeostasis
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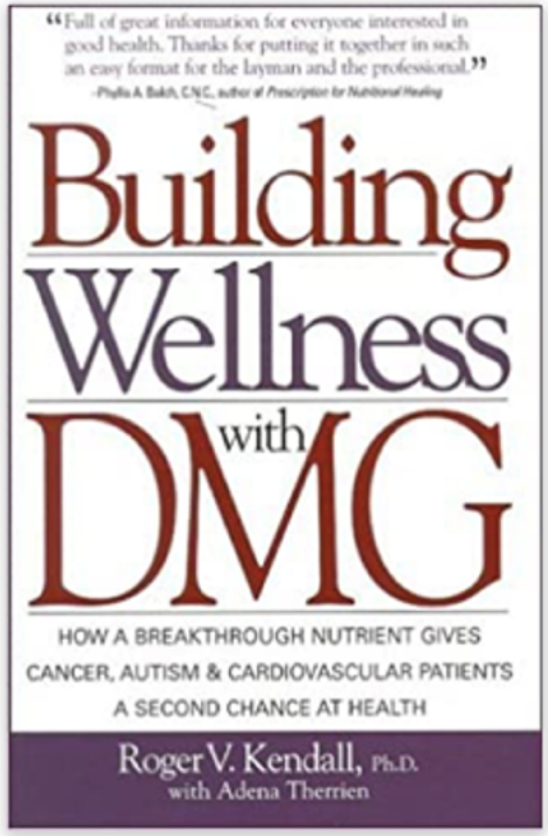
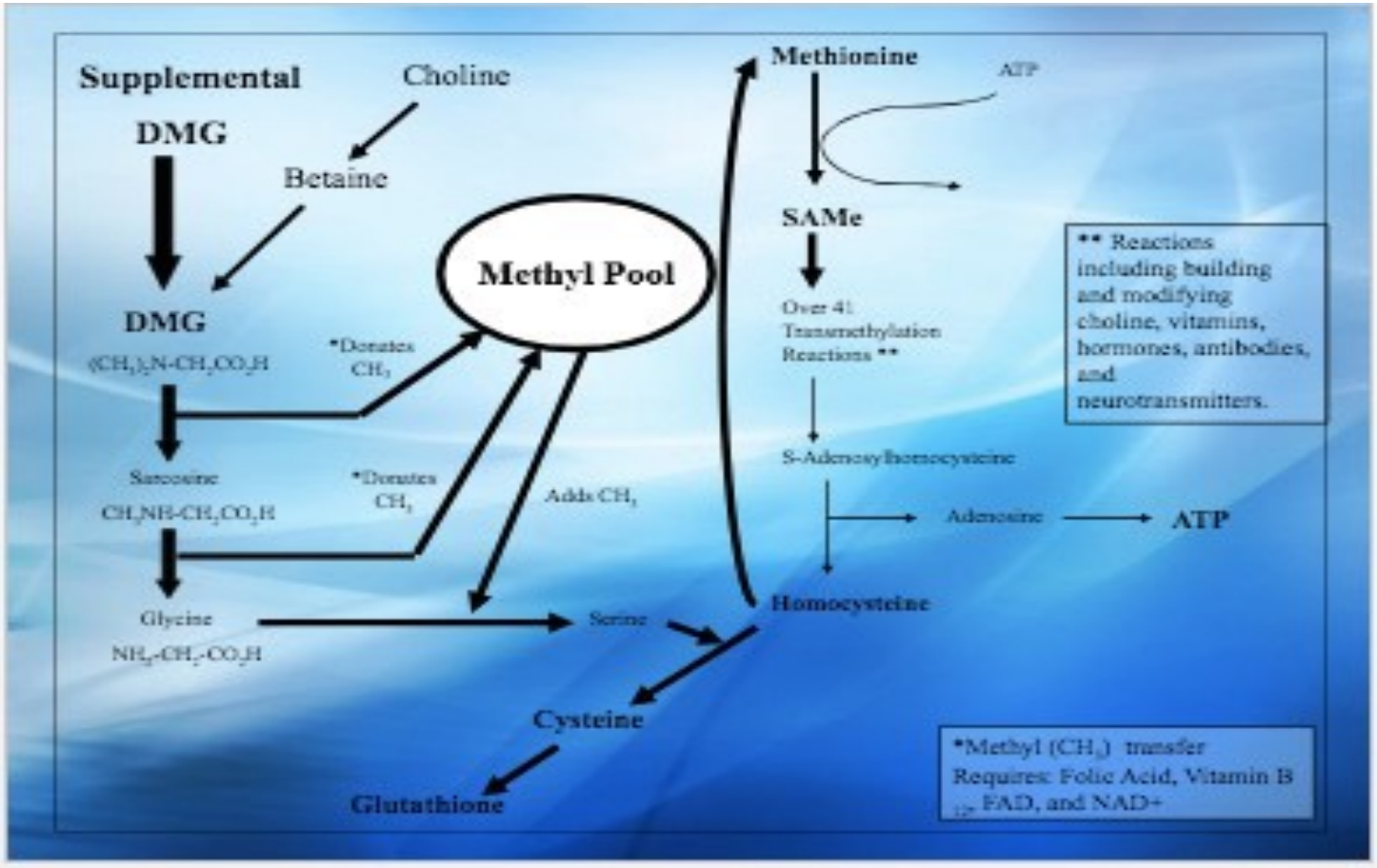
- Amino Acid – Intermediary metabolite of the human body
- Important nutrient found in low levels in our food
- As a Key Nutrient DMG PROTECTS OUR DNA

CH_3

$CH_3NCH_2C0_2$





Nattokinase is neither NATTO nor a Kinase
It's a drug a serine protease that destroys the body's ability to make glutathione

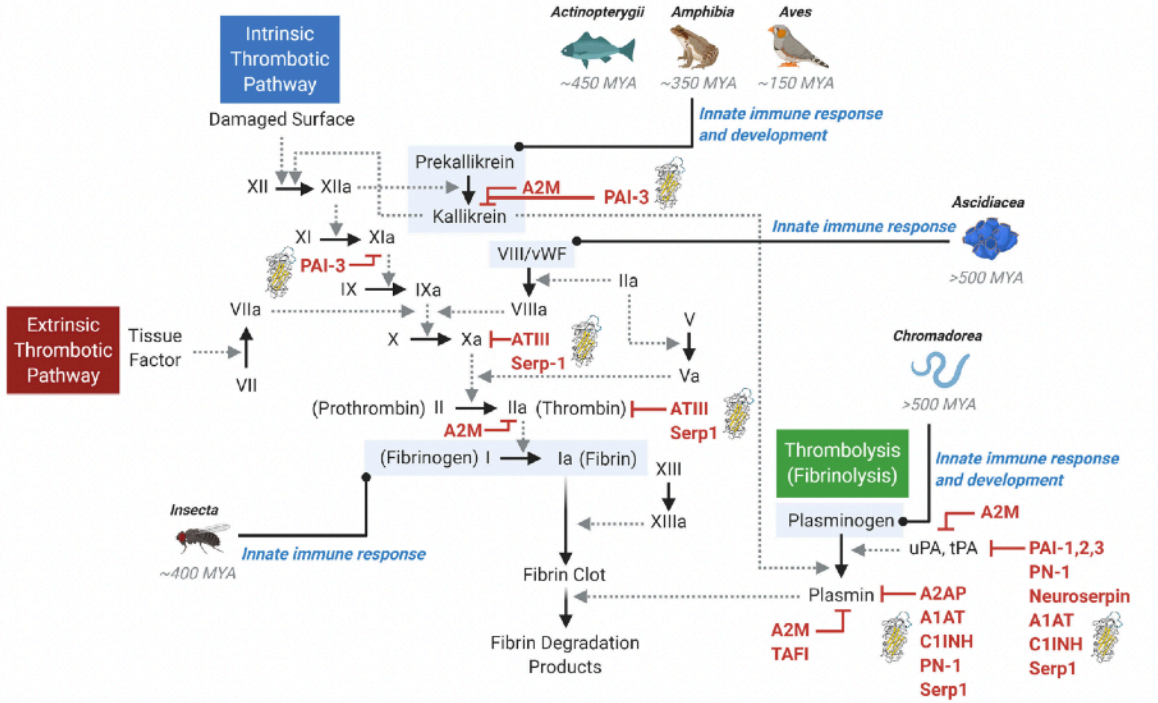


FIGURE 1 | The thrombotic and thrombolytic cascades and primordial immune response. The thrombotic pathways (intrinsic and extrinsic) and the thrombolytic (fibrinolysis) pathway involve a complex cascade of protease activation. Solid arrows indicate the conversion to an active protease, while dotted line arrows indicate the activity of the activating upstream protease. A variety of inhibitors are shown, with serpin inhibitors denoted by a serpin protein structural image. Examples of early primordial immune response origins are noted in context of the pathways. MYA, million years ago.

frontiers in Cardiovascular Medicine

REVIEW
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Fibrinolytic Serine Proteases, Therapeutic Serpins and Inflammation: Fire Dancers and Firestorms

*Jordan R. Yaron[1,2], Liqiang Zhang[1], Qiuyun Guo[1], Shelley E. Haydel[3,4] and Alexandra R. Lucas[1]**

[1] Center for Personalized Diagnostics and Center for Immunotherapy, Vaccines and Virotherapy, The Biodesign Institute, Arizona State University, Tempe, AZ, United States, [2] School for Engineering of Matter, Transport and Energy, Ira A. Fulton Schools of Engineering, Arizona State University, Tempe, AZ, United States, [3] Center for Bioelectronics and Biosensors, The Biodesign Institute, Arizona State University, Tempe, AZ, United States, [4] School of Life Sciences, Arizona State University, Tempe, AZ, United States

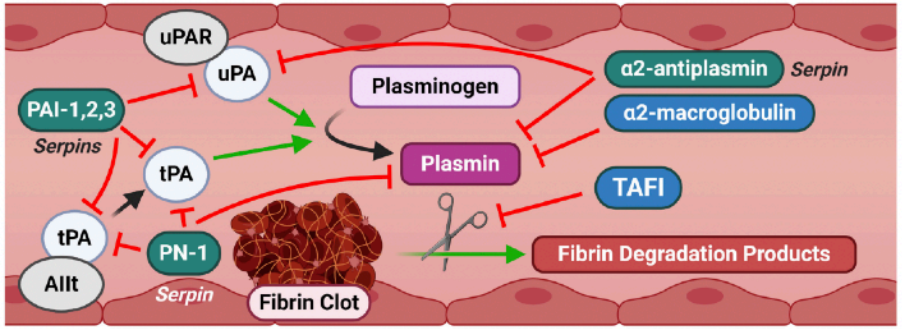


FIGURE 3 | Canonical signaling of the fibrinolysis pathway. Fibrinolysis is characterized by the degradation of a fibrin clot into degradation products by plasmin. Plasmin is generated from plasminogen by uPA and tPA. Several serpins and other inhibitors provide a tight regulation of this cascade. Substantial promiscuity exists across multiple elements of the pathway, providing redundant controls against inappropriate activation. AIIt and uPAR are shown as representative canonical fibrinolytic receptors for brevity. AIIt, Annexin II tetramer; PAI-1,2,3, Plasminogen Activator Inhibitor-1, 2, 3; PN-1, Protease Nexin-1; TAFI, Thrombin activatable fibrinolysis inhibitor; tPA, Tissue-type plasminogen activator; uPA, Urokinase-type plasminogen activator; uPAR, Urokinase-type plasminogen activator receptor.

Published OnlineFirst September 25, 2012; DOI: 10.1158/2159-8290.CD-12-0345

GENES & DEVELOPMENT 26:877–890 © 2012 by Cold Spring Harbor Laboratory Press ISSN 0890-9369/12; www.genesdev.org

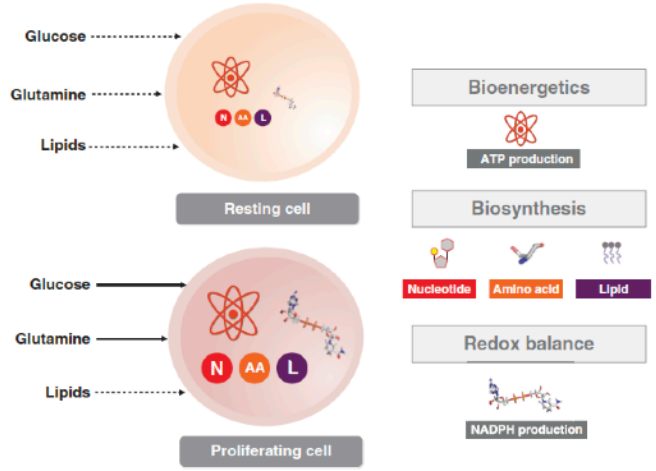
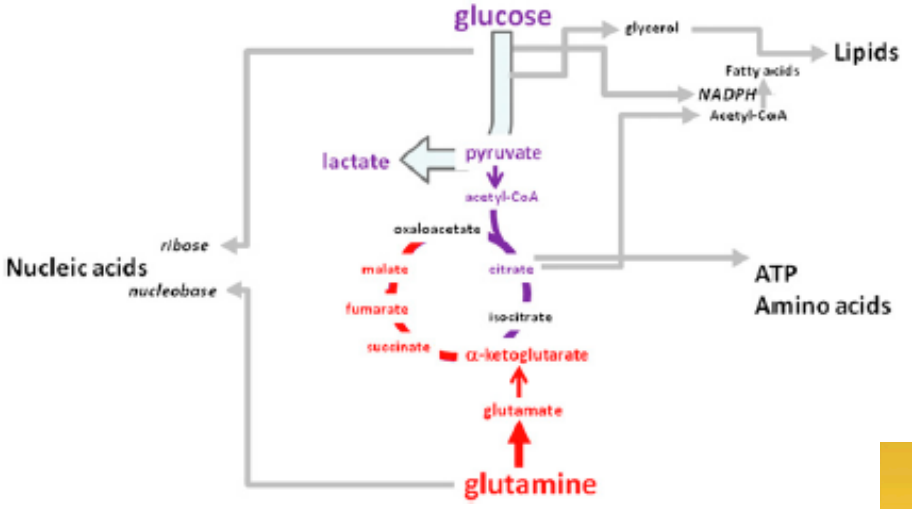
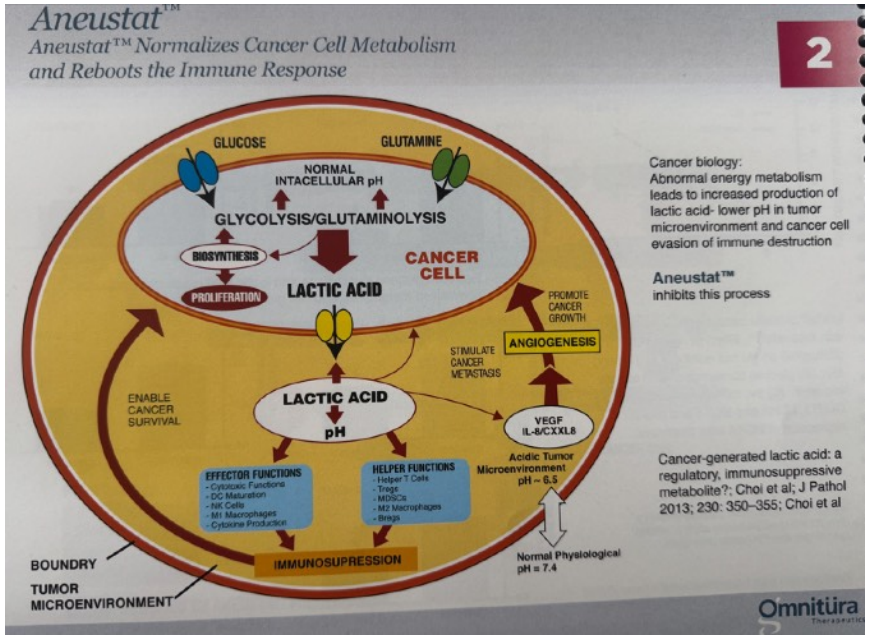


Figure 1. Metabolism: resting versus proliferating cells. Normal resting cells use a catabolic metabolism to satisfy the energetic requirements of homeostasis. This demand is met through fatty acid oxidation and the oxidative metabolism of glucose. Proliferating cells, however, must rewire their metabolic program to not only meet various energetic requirements, but to also satisfy the anabolic demands of macromolecular biosynthesis (nucleotides, lipids, and proteins), as well as the maintenance of redox homeostasis. Upon growth factor–mediated stimulation, proliferating cells increase their uptake of glucose and glutamine, which are the 2 primary substrates that fuel cell growth. Solid lines are indicative of increased cellular uptake.





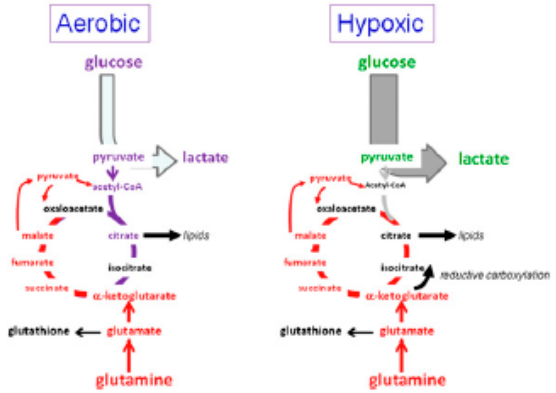
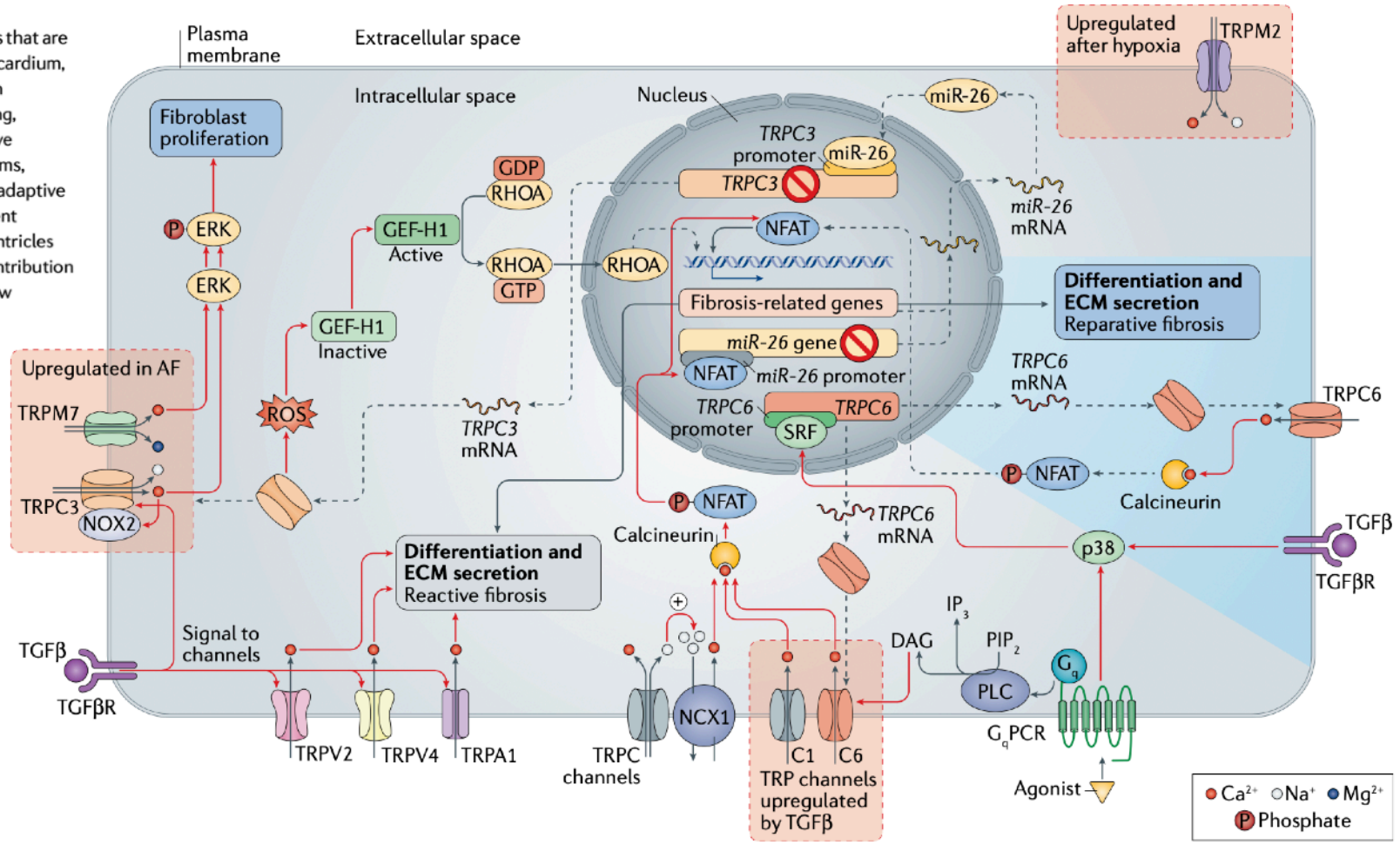


Figure 2. Hypoxic rewiring of metabolism. While aerobic proliferating cells use glucose and glutamine for biomass production through the TCA cycle, hypoxic cells shunt glucose to lactate and rewire glutamine metabolism. Glutamine can be used to drive the TCA cycle independently of glucose or contribute to lipid synthesis via IDH-mediated reductive carboxylation of ketoglutarate generated from glutamine.

Transient receptor potential channels in cardiac health and disease

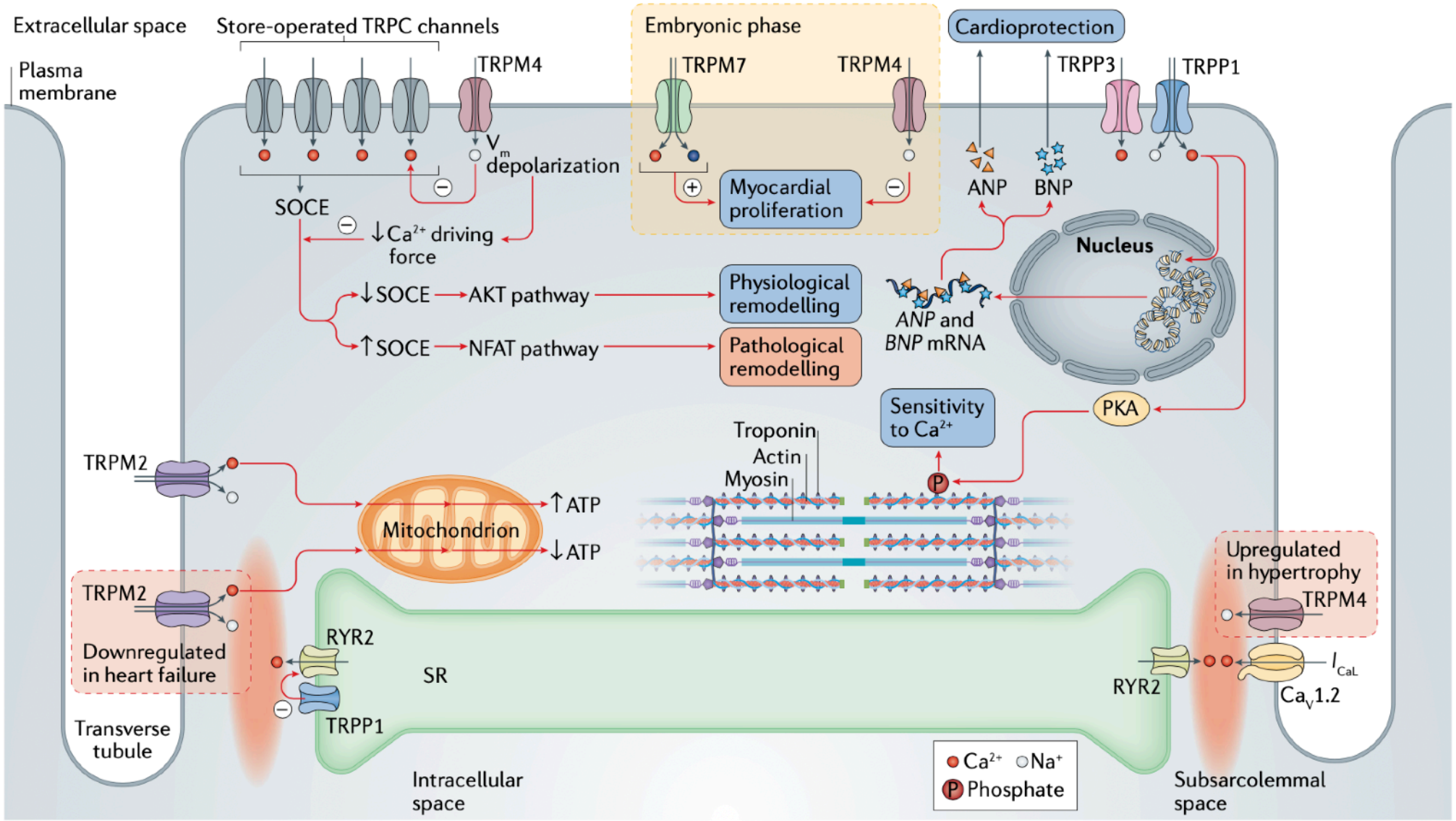
*Thomas Hof[1,2,3], Sébastien Chaigne[1,2,3], Alice Récalde[1,2,3], Laurent Sallé[4], Fabien Brette[1,2,3] and Romain Guinamard[4]**

Abstract | Transient receptor potential (TRP) channels are nonselective cationic channels that are generally Ca^{2+} permeable and have a heterogeneous expression in the heart. In the myocardium, TRP channels participate in several physiological functions, such as modulation of action potential waveform, pacemaking, conduction, inotropy, lusitropy, Ca^{2+} and Mg^{2+} handling, store-operated Ca^{2+} entry, embryonic development, mitochondrial function and adaptive remodelling. Moreover, TRP channels are also involved in various pathological mechanisms, such as arrhythmias, ischaemia–reperfusion injuries, Ca^{2+}-handling defects, fibrosis, maladaptive remodelling, inherited cardiopathies and cell death. In this Review, we present the current knowledge of the roles of TRP channels in different cardiac regions (sinus node, atria, ventricles and Purkinje fibres) and cells types (cardiomyocytes and fibroblasts) and discuss their contribution to pathophysiological mechanisms, which will help to identify the best candidates for new therapeutic targets among the cardiac TRP family.



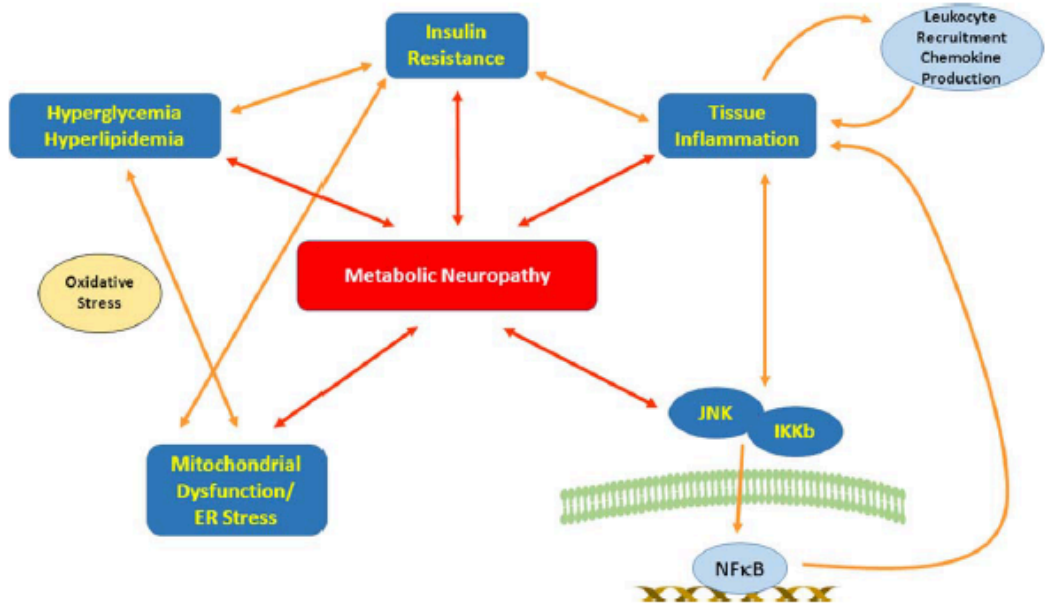
| Roles of TRPC channels in ventricular cardiomyocytes.

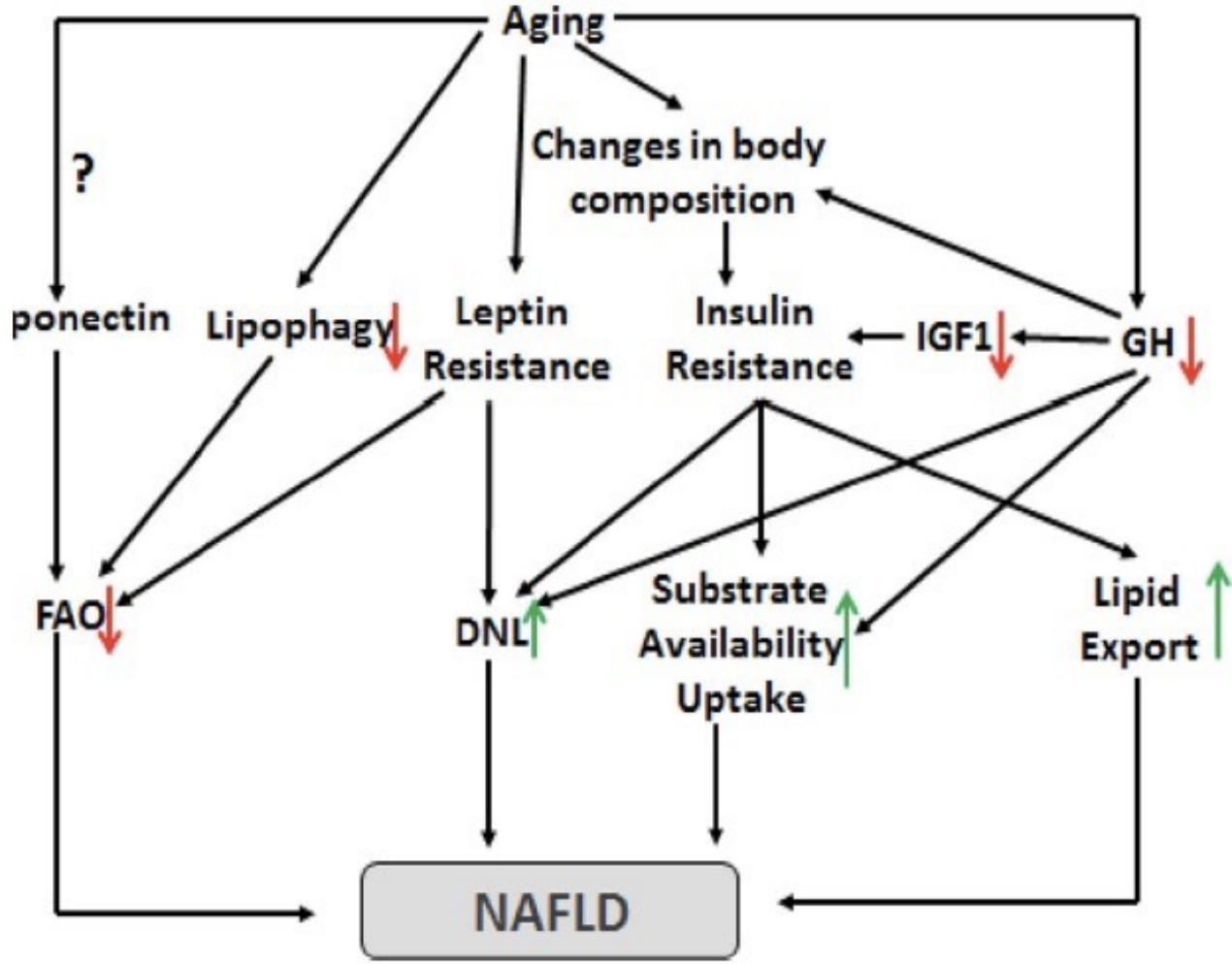
Fig. 4 | **Roles of TRPM and TRPP channels in ventricular cardiomyocytes.** TRPM2 channels participate in the maintenance of mitochondrial function. TRPM4 channels attenuate store-operated Ca^{2+} entry (SOCE) by depolarizing the membrane



Inflammaging is associated with dysregulation of Glucose and lipid metabolism

ANNALS *of Neurology*





mTOR nutrient sensing Dysregulation
Neuroimmune, Cardiovascular Disease & cancer?

http://www.discoverymedicine.com/David-Fernandez/files/2010...

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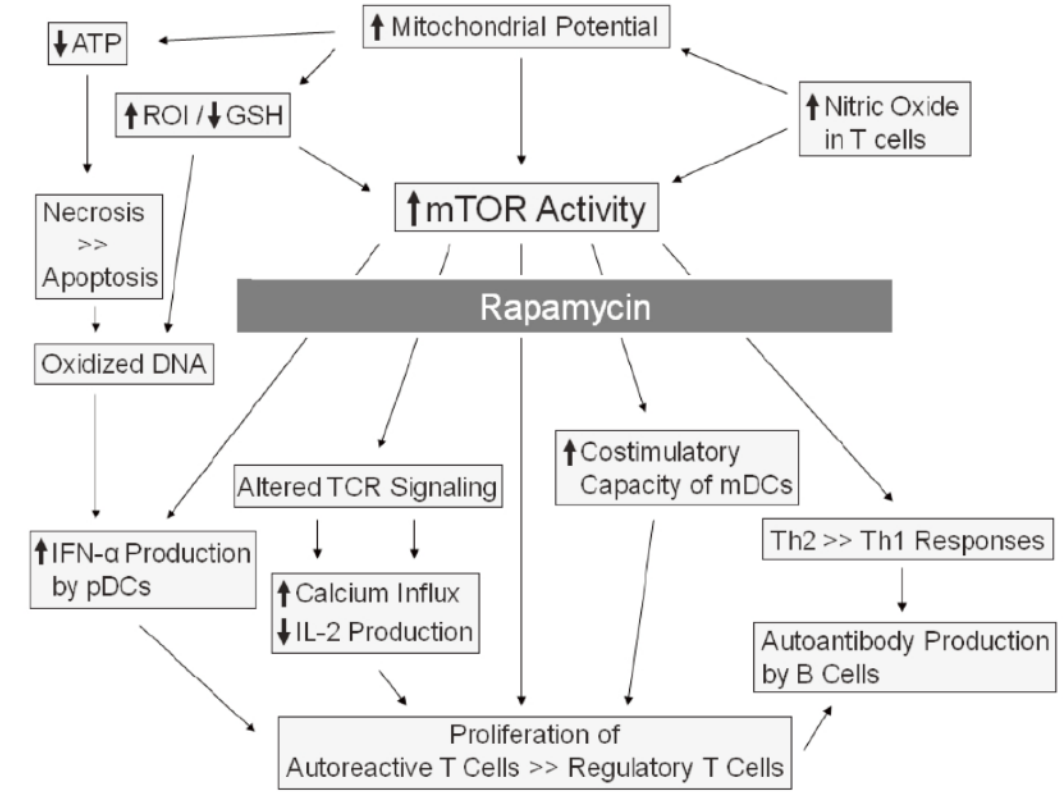

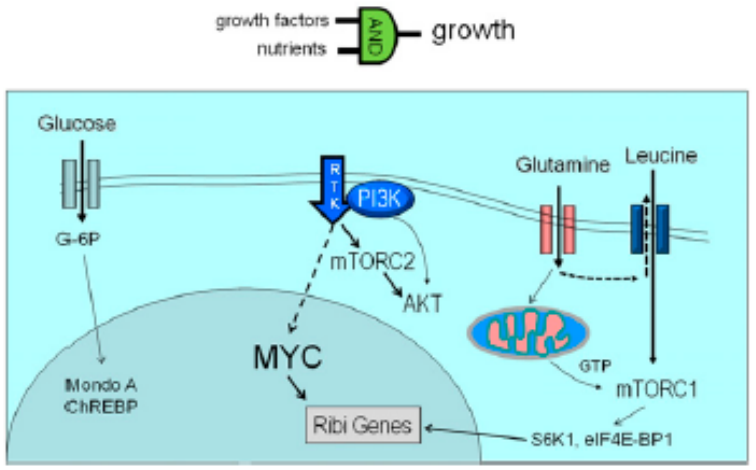

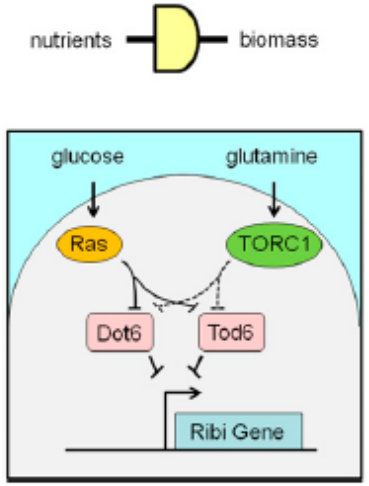

Figure 4. Nutrient sensing and yeast cell growth. Glucose and glutamine are depicted to signal via Ras and TORC1, respectively, to inhibit repressors (Dot6 and Tod6) of ribosomal biogenesis (Ribi) genes.

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- If you cannot take aspirin or blood-thinning medications (also called antiplatelet or anticoagulant therapy)

Abbott

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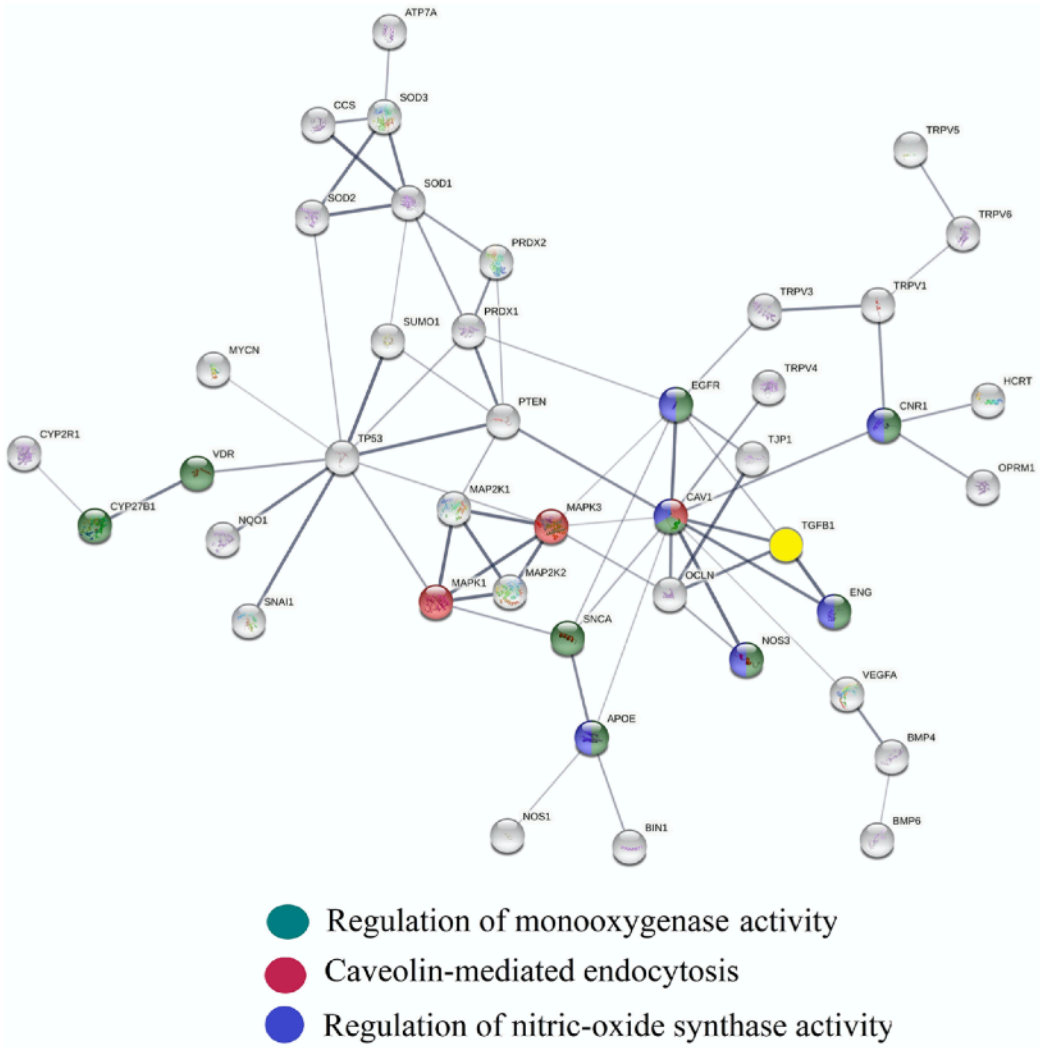

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Functional characterization of nutraceuticals using spectral clustering: Centrality of caveolae-mediated endocytosis for management of nitric oxide and vitamin D deficiencies and atherosclerosis

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Emergent System Analytics LLC, Clinton, CT, United States

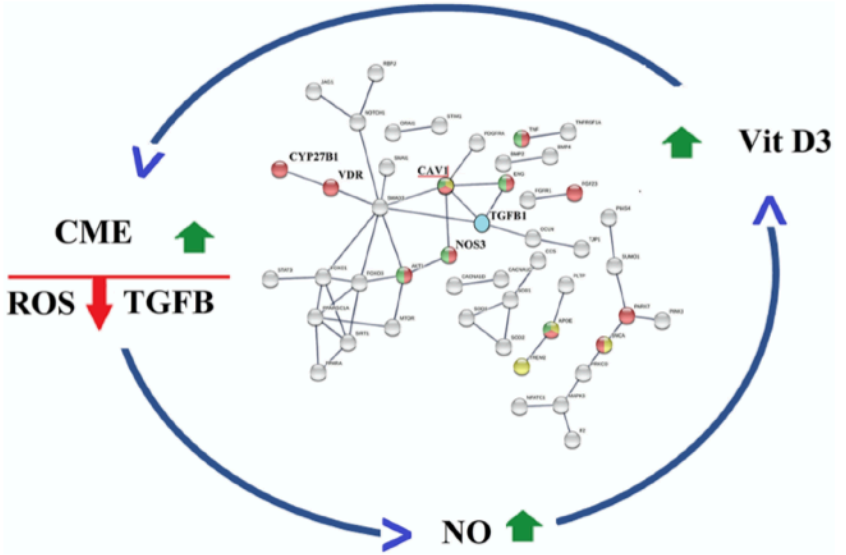
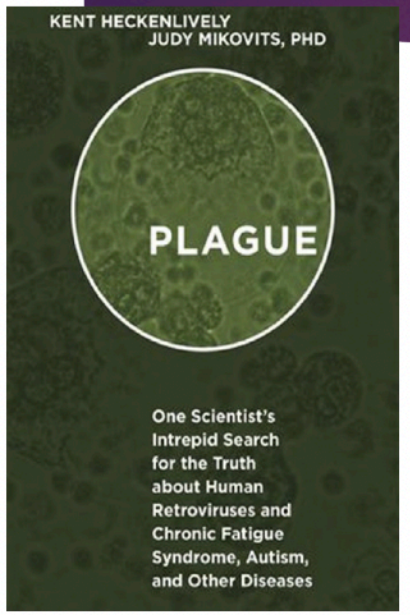
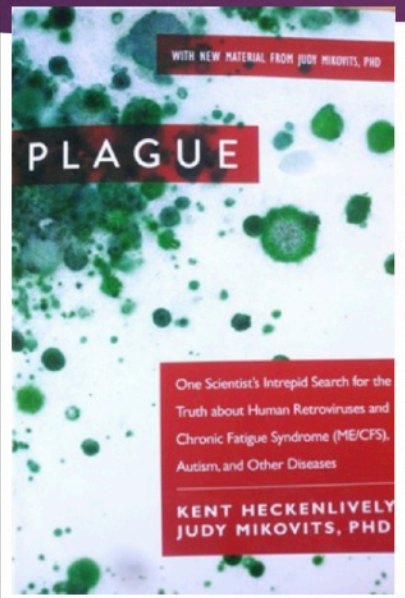


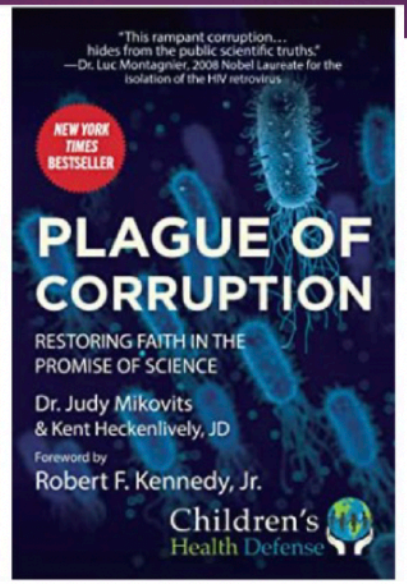
FIGURE 4
Caveola mediated endocytosis (CME) modulates activities of a reciprocal feedback loops that finetunes ROS production, TGF beta activity, Nitric oxide levels O and Calcitriol production.

GOD's People are destroyed from lack of Knowledge (Hosea 4:6)
THE FEAR OF THE LORD is the Beginning of Knowledge but Fools Despise Wisdom & Instruction (PROVERBS 1:7)



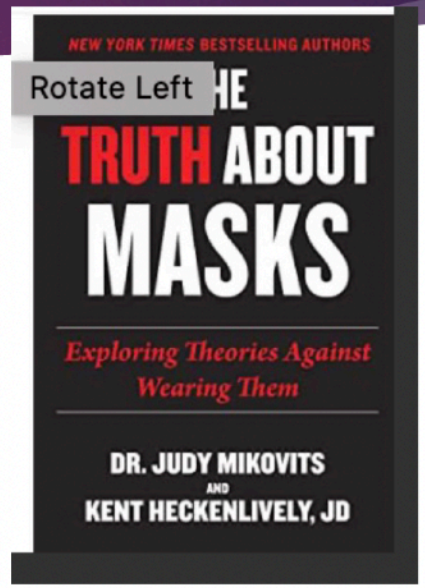


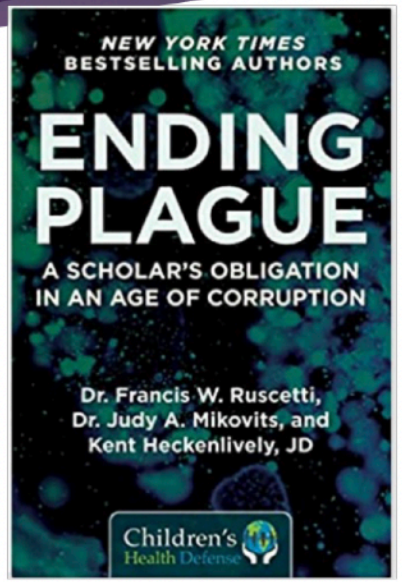
2014 (James 1:19-22)



2017

April 2020 Psalm 91



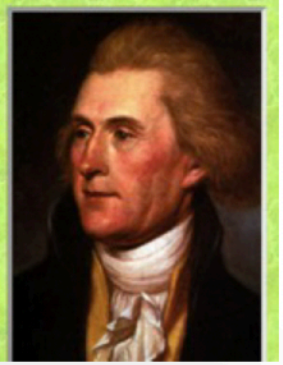


2021(Ephesians 5:11

2021(2 Chronicles 7:14

Quote by Thomas Jefferson

"If people let the government decide what foods they eat and what medicines they take, their bodies will soon be in as sorry a state as are the souls who live under tyranny." -- Thomas Jefferson



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