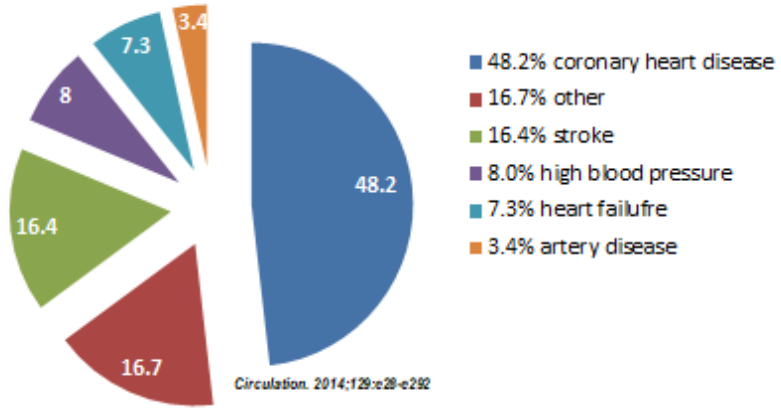
Introducing
Arterez, Inc.

CVD #1 KILLER WORLDWIDE

Arterez™ Diagnostics and Therapies

SLIDE 2

Cardiovascular disease is a family of diseases



- 400 million people with CVD illness at any given time
- 1/3 of all global deaths
- In all developing nations, CVD is largest health risk and cost
- US costs rising to an est. $1.5 trillion by 2030
- No predictive diagnostics or preventive, curative therapy exist

Statins, most prescribed drug in US: > 350 M filled in 2018

Introducing
Arterez, Inc.

VISION

Arterez™
Diagnostics and Therapies

SLIDE 3

To develop multi-compound oral therapies, diagnostic panels and therapeutic monitoring aides targeting the multifactorial root causes of cardiovascular disease leading to predictive, preventive and curative outcomes, supported by a patent portfolio of novel compounds, methods and biomarkers.

More information available here:
https://www.arterez.com/

About Arterez

- Arterez, Inc. is a pharmaceutical platform technology company developing therapeutics and diagnostics for vascular diseases particularly due to the breakdown of the endothelial glycocalyx initially targeting cardiovascular indications
- Glycocalyx (GCX) is an extracellular matrix that covers body surfaces, lines internal closed cavities, glands, body tubes and the vascular system. GCX disruption and shedding signal the onset of diseases
- Diseases, including cardiovascular disease (CVD) are multifactorial including multilevel oxidation and inflammation of cell components and inability of the cell to recover
- Complex diseases are better addressed by polypharmacology or combo drugs rather than the current “one drug-one target-one disease" philosophy permeating the standard of care worldwide

"Historical development of antibiotics targeting microorganisms led to a cure of infectious disease. An equivalent approach is to target the endothelial glycocalyx to cure CVD by an anti-embolic™ mechanism."
Dr. Joe Tunac

Preclinical proof-of-principles/milestones:

- Developed a natural animal model which mimics glycocalyx shedding and disruption
- Demonstrated that high fat diet and environmental pollutant trigger CVD
- Synthesized 9 new chemical entities (NCEs), and discovered a triple NCE combo (Embotricin™) that cured and prevented plaque
- Demonstrated that the polypharmacology platform of Embotricin™ cured coronavirus
- Embotricin™ is nontoxic: effective dose of 3.0 mg/kg and maximum tolerated dose of ~1,000 mg/kg
- Developed a first in class fingerprinting system of diseases (akin to the classic fingerprint and DNA-fingerprint), based on glycocalyx components (detritus) shedding: “Glycalyx Detritus Fingerprint™ (GDF) and GlycoCardia™ for the identification, characterization and early diagnosis of CVD and other vascular disease indications as well as a therapeutic aide for Embotricin™

"Historical development of antibiotics targeting microorganisms led to a cure of infectious disease. An equivalent approach is to target the endothelial glycocalyx to cure CVD by an anti-embolic™ mechanism."
Dr. Joe Tunac

Clinical development:

- IND-compliant toxicology study including general (14-day) rodent toxicokinetic (TK), genotoxicity, pharmacokinetic (PK), pharmacodynamics (PD), absorption distribution metabolism excretion (ADME) and chemistry manufacturing and control (CMC) to determine the maximum recommended starting dose (MRSD) for first-in-human clinical. (Note: pre-consultation with FDA suggests an allowance of a fixed-dose IND-toxicology protocol)
- Planned clinical Embotricin trials to target 3 of 5 of the following disease indications:
    a. Hypertension,
    b. Pulmonary Hypertension,
    c. Dialysis Fistula,
    d. Heart Failure and
    e. Coronary Heart Disease.

"Historical development of antibiotics targeting microorganisms led to a cure of infectious disease. An equivalent approach is to target the endothelial glycocalyx to cure CVD by an anti-embolic™ mechanism."
Dr. Joe Tunac

Arterez™ Diagnostics and Therapies

A Delaware Corporation

GlycoTRx
Chronic Disease Diagnostics
A Michigan LLC

GlycoCardia™
Cardiovascular Diagnostic

ComboRx
Chronic Disease Therapeutics
A Michigan LLC

Embotricin™
Cardiovascular Oral Therapy

Arterez wholly-owns GlycoTrx, LLC and ComboRx, LLC

FUTURE PRODUCT DEVELOPMENTS

Chronic Disease
Diagnostic Tools

GlycoDiabx™
Diabetes Diagnostic

GlycoArthx™
Arthritis Diagnostic

Chronic Disease
Therapies

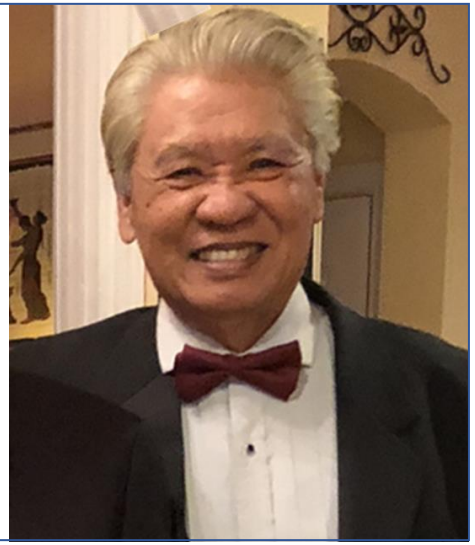
Metabotricin™
Diabetes Oral Therapy

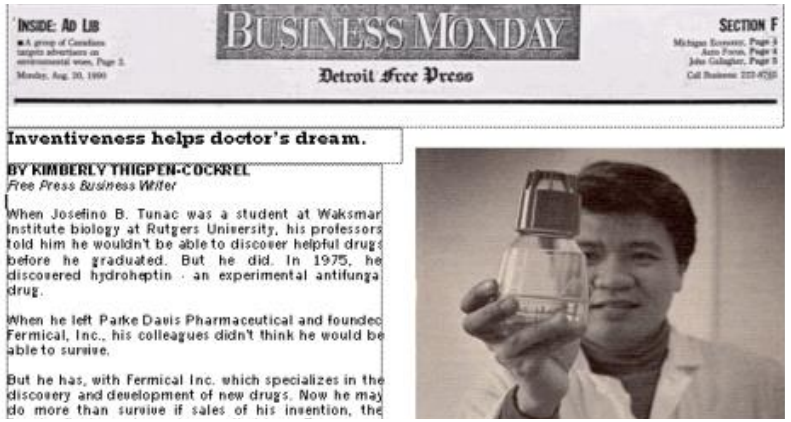
Arthritricin™
Arthritis Oral Therapy

Arterez' pre-clinical data in addition to independent research focused on the endothelial glycocalyx demonstrate a root cause correlation between vascular pathophysiologies.

That bold vision began with
Dr. Joe Tunac

40 Yr. 'Drug Hunter'





INSIDE: AD LIB

BUSINESS MONDAY

Detroit Free Press

SECTION F

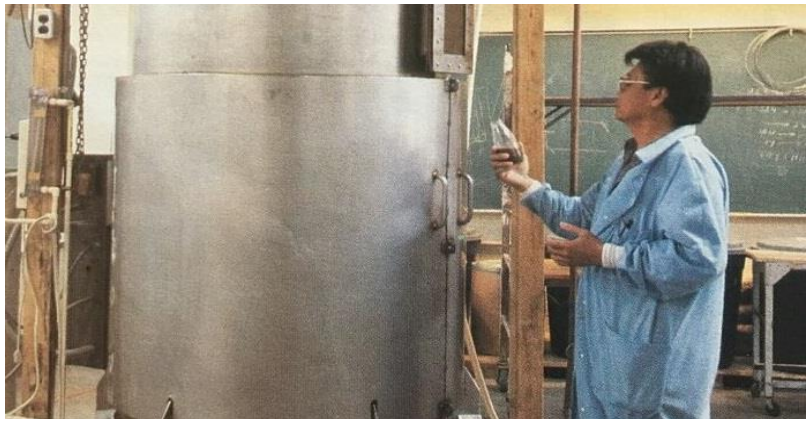
Inventiveness helps doctor's dream.

BY KIMBERLY THIGPEN-COCKREL
Free Press Business Writer

When Josefino B. Tunac was a student at Waksman Institute biology at Rutgers University, his professors told him he wouldn't be able to discover helpful drugs before he graduated. But he did. In 1975, he discovered hydroheptin - an experimental antifungal drug.

When he left Parke Davis Pharmaceutical and founded Fermical, Inc., his colleagues didn't think he would be able to survive.

But he has, with Fermical Inc. which specializes in the discovery and development of new drugs. Now he may do more than survive if sales of his invention, the



Education

U of Philippines – BS, Plant Pathology
So. Dakota State – Masters, Plant Pathology
Penn State – Microbiology Ph.D. program
Rutgers (Waksman Institute; world center for anibiotic research) – Ph.D.
1st student to successfully develop a drug

Merck – Dir of Research

Avermectin (*Ivomec*: *2015 Nobel Prize*)
1st billion-dollar drug
Cefoxitin (*Mefoxin*)
Primaxin (*Imipenem*)

Parke-Davis/W-Lambert – Dir, Antibiotics & Chemo

Pentostatin (*Nipent*)
Daunorubicin (*Cerubidine*)
Vidarabine (*Vira-A*)

Founder & Co-Founder

Fermical – Ferndale, MI
Biotech Lab: drug discovery & development
Tunair Labware flasks & bioreactor
Still sold worldwide today

Supergen (SUPG: NASDAQ) – Dublin, CA
Anticancer Drug (Mitomycin)
Licensed to Astex Pharma (ASTX)
Sold to Otsuka Pharma for $886M

JJ Pharma, Inc – San Ramon, CA
Anti-arthritis Drugs

Acea Biotech, Inc – San Francisco, CA
Anti-fungal (*Corifungin*)
Designated orphan drug by FDA

Farmaceutix, LLC – Ferndale, MI (2012-2018)
Anti-Embolic™ drugs
All IP now wholly owned by Arterez, Inc.

ISSUED – US 9,867,842 B2
Methods and Compositions for Reversing Disruption of the Glycocalyx, Inflammation and Oxidative Damage.

PENDING – ARTZP003PUS
Drug Treatment and Biomarker Panel Targeted to Diseases due to Multifactorial Ontology of Glycocalyx Disruption

PENDING – PCT/US2016/015015
Biomarkers of Vascular Disease

PENDING - International PCT
Filed November 2020

DRAFTED – Provisional to be filed Mar 2021
Natural Arterial Plaque Mouse Model

Issued Trademarks

- Arterez™
- GlycoCardia™
- Embotricin™

Applications in process

- Natural Arterial Plaque Mouse Model (filed Aug, 20)
- 'Glycalyx Detritus Fingerprint' (GDF) technology
- Anti-Embolic
- Embotriocin
- EmboRx
- Zeno-Disease

NOTE:
Issued and pending patents are available for download at via the following website link:

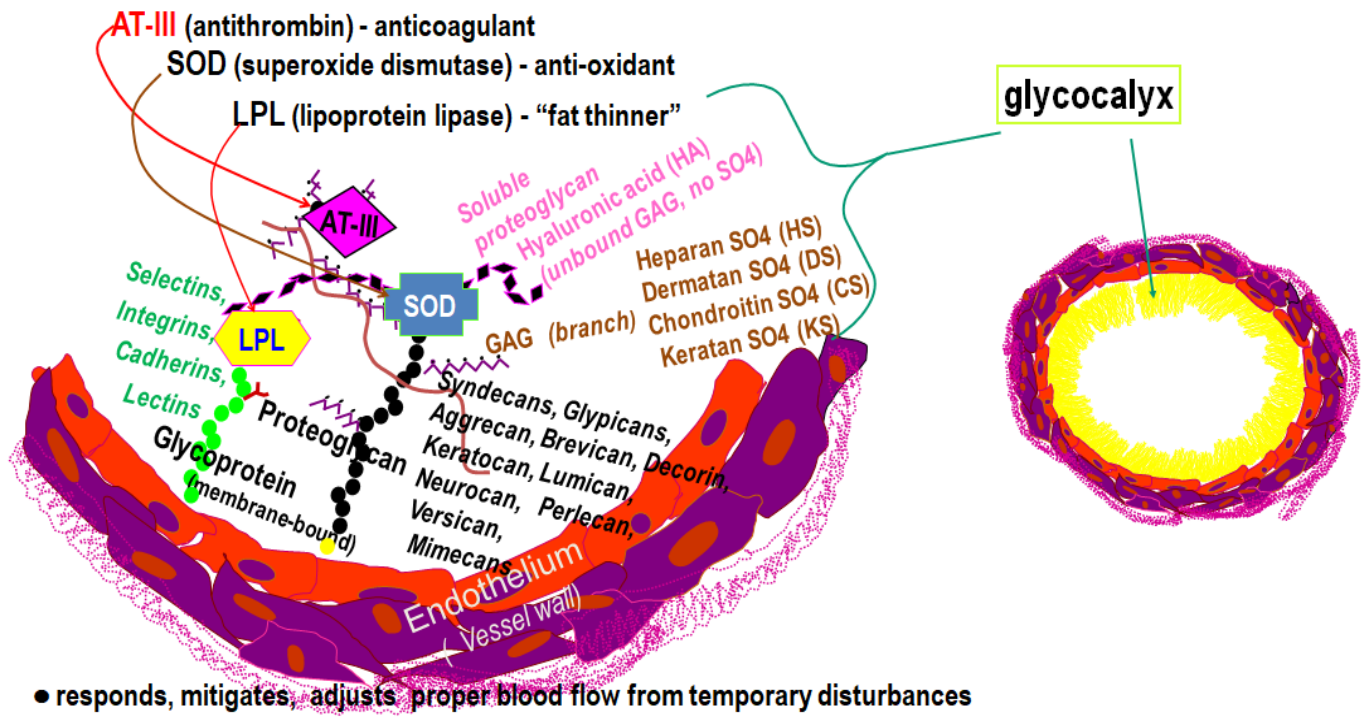
https://www.arterez.com/investors

FACT A:

CVD begins with disruption of the glycocalyx which is the protective coating of the vasculature essential to maintain healthy blood flow and serves as a nest for important glycoprotein and proteoglycan components.

Glycocalyx protects endothelium

Provides a 'nest' to 3 key enzymes that regulate blood flow



- responds, mitigates, adjusts proper blood flow from temporary disturbances
- stagnant blood flow *(low shear)*, glycocalyx disruption, chronic shedding → chronic diseases, CVD

FACT B:

The disruption of the glycocalyx, not cholesterol is key to solving vascular pathophysiologies, including CVD.

Cholesterol is essential for healing, critical to good health and well being and while statins effectively reduce LDL cholesterol, they also reduce coQ10 equally (thus starving heart muscle), among other elements in the mevalonate pathway leading to side effects and new diseases we've coined 'Xeno-diseases.'

Disrupted Glycocalyx

Vascular diseases *(endothelial)*

Cardiovascular disease (CVD)

- Stroke
- Angina (pectoris)
- Coronary heart disease (CHD)
- Myocardial infarction (MI)
- Heart failure *(swelling, heart attack)*
- Peripheral arterial disease (PAD)
- Rheumatic heart disease
- Atrial fibrillation *(arrhythmia)*
- Deep vein thrombosis (DVT)
- Atherosclerosis *(hardened artery)*
- Hypertension *(high blood pressure)*
- Ischaemia *(restricted blood supply)*
- Aneurysm (cerebral, abdominal)

Epithelial diseases *(non-endothelial and/or endothelial)*

Non-CVD chronic diseases

- Cancer
- Arthritis *(eczema, psoriasis, vitiligo)*
- Diabetes
- Neuropathy *(Alzheimers, MS, Parkinsons, ALS)*
- Obesity
- Metabolic syndrome
- Eye diseases *(macular degeneration, glaucoma, cataract, dry eye)*
- Irritable bowel syndrome (IBS)
- Crohn's disease
- Gastrointestinal diseases *(ulcer, GERD)*
- Sepsis
- Respiratory diseases *(Asthma, COPD, cystic fibrosis)*

FACT C:

A high Fat diet thickens the blood thus slowing blood flow.

An animal-based diet contains both cholesterol and fat, yet cholesterol is fairly constant while fat content varies (cheese, beef the highest).

The typical western diet associated with heart disease contains 21% fat.

Fat triggers CVD, not cholesterol

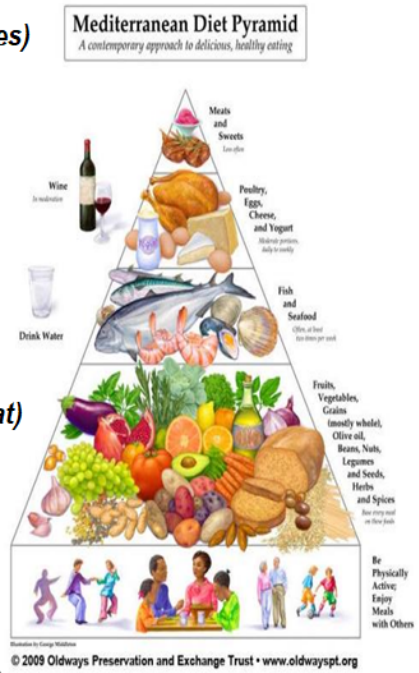
1958: Ancel Key's Mediterranean diet "Seven Countries Study" showed low CVD *because of less dietary fat*

Every animal-based food contains cholesterol and fat *(cholesterol almost constant, but fat varies)*

Food type	% Cholesterol / fat
seafood	*0.046 / 1.37*
(scallop, lobster, clam, shrimp, crab)	
chicken	*0.042 / 2.50*
pork	*0.036 / 6.16*
beef	*0.049 / 9.62*
egg	*0.340 / 2.50*
milk (whole)	*0.016 / 4.00*
cheddar cheese	*0.107 / 32.00*

0.09% cholesterol, 7.95 % fat

- **Western Type Diet:(WTD):** *0.15% cholesterol , 21% fat*
- **Mediterranean lifestyle:** *low in fat, plenty of exercise (foundation of fruits, vegetables, grains, fish; low poultry & red meat)*



Cholesterol and fat in the diet

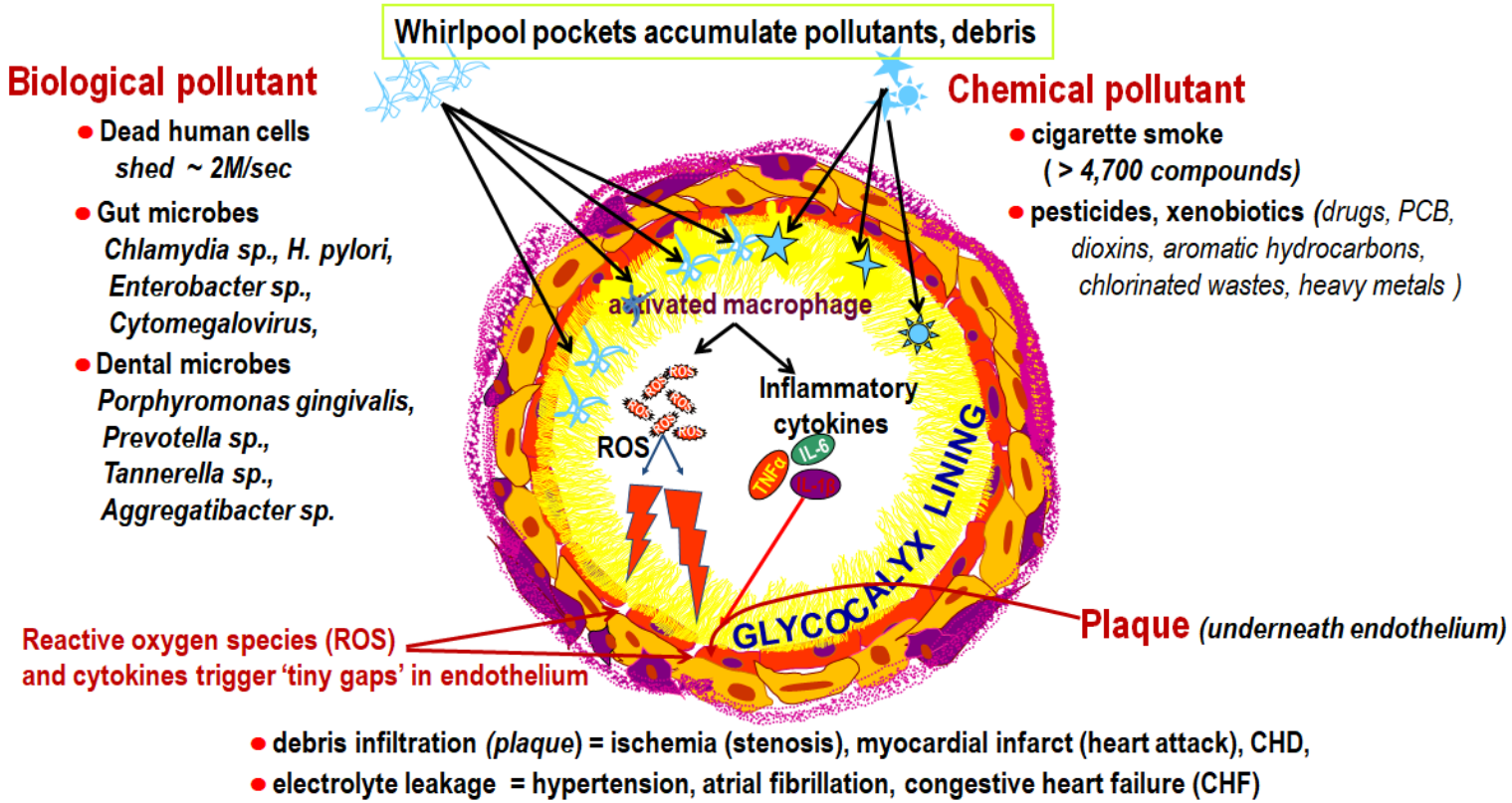
- *diet cholesterol and fat packaged in lipoprotein (chylomicron) for delivery to liver*
- *cholesterol converted to bile; fat repackaged into VLDL for delivery to blood stream*
- *VLDL increase blood viscosity, create stagnation.*
  - *» less fat, less VLDL , better blood flow*
  - *» seafood contains as much cholesterol as beef, poultry and pork, but less fat*

FACT D:

Pollutants trapped in thickening blood-flow, triggers inflammation.

Biological and chemical pollutants in the arterial bends triggers inflammation and tiny endothelial gaps creating electrolyte leakage (hypertension) and debris infiltration (plaque). Cholesterol packaged in lipoproteins (made of fatty acids and prone to oxidation) fill the gaps, preventing osmotic imbalance and bleeding.

Pollutants are oxidative, inflammatory: create gaps



FACT E:

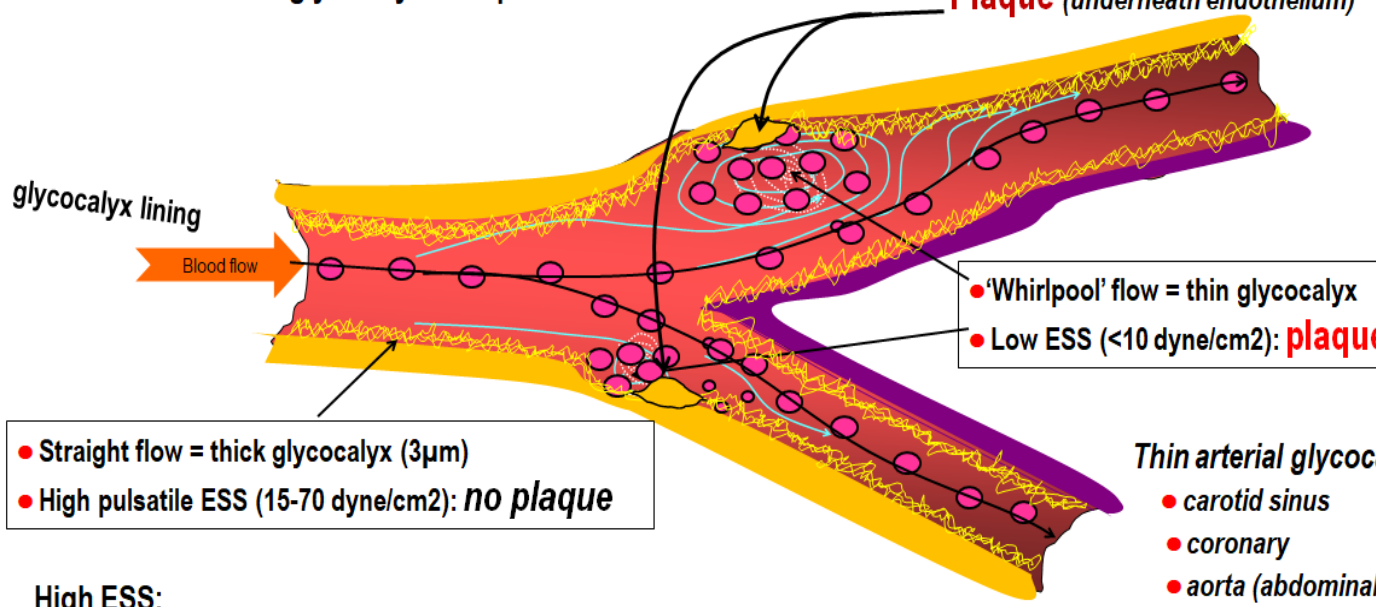
Arterial bends and bifurcations are sites of stagnant blood flow and plaques.

A high fat diet, sugars, pollutants and sedentary lifestyle all contribute to viscous blood and blood flow naturally slows at arterial forks and bends, creating a "whirlpool pocket."

Stagnant blood concentrates debris, mobilizing macrophoges (foam cells) to engulf and remove debris.

'Whirlpool' pockets at forks, bends: plaque sites

Stagnated blood flow, accumulates debris
- **inflammation → glycocalyx disruption**



Thin arterial glycocalyx
- *carotid sinus*
- *coronary*
- *aorta (abdominal, thoracic Circle of Willis)*
- *peripheral arteries*

High ESS:
- **high nitric oxide (NO), *vasodilation, anti-inflammatory***
- **high antioxidants; *suppress reactive oxygen species (ROS)***

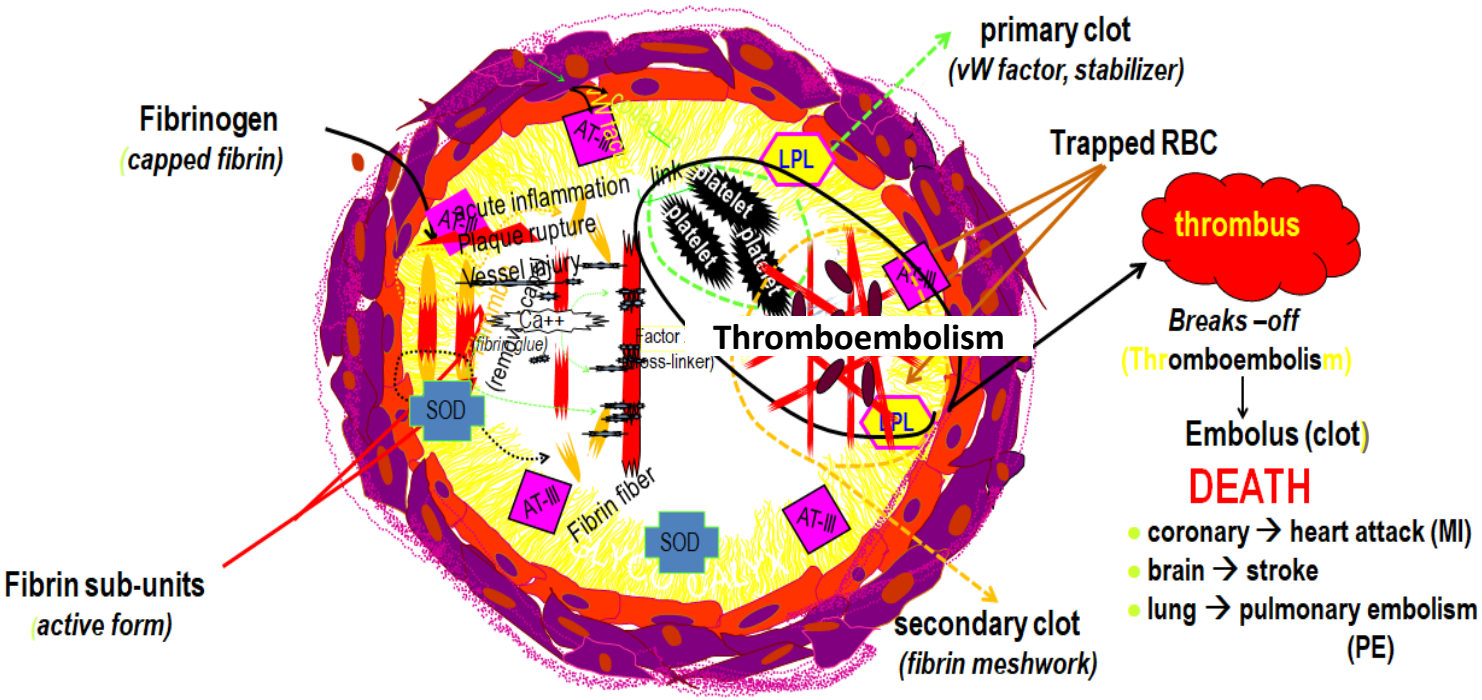
FACT F:

Acute inflammation triggers plaque rupture leading to fatal clot (thromboembolism).

Clot formation starts with a disrupted glycocalyx (primary clot) and progresses into a secondary clot (embolus). This is the fatal component of CVD.

Thromboembolism, fatal process in CVD

Disruption of protective glycocalyx: *exposes collagen; release tissue factor (TF) binds platelets* → **primary clot**
Removal of SOD, LPL, & AT-III: *prone to inflammation* → *thromboembolism*
Fibrinogen exposed to thrombin → *thrombin produces fibrin* → **secondary clot**



Embotricin™ effectively and specifically protects, repairs and prevents damage to the arterial lining which is now widely recognized as a therapeutic and diagnostic target for vascular disease – the endothelial glycocalyx. In addition, Embotricin™ also addresses inflammation and oxidation.

More information available here:
https://www.arterez.com/therapeutics

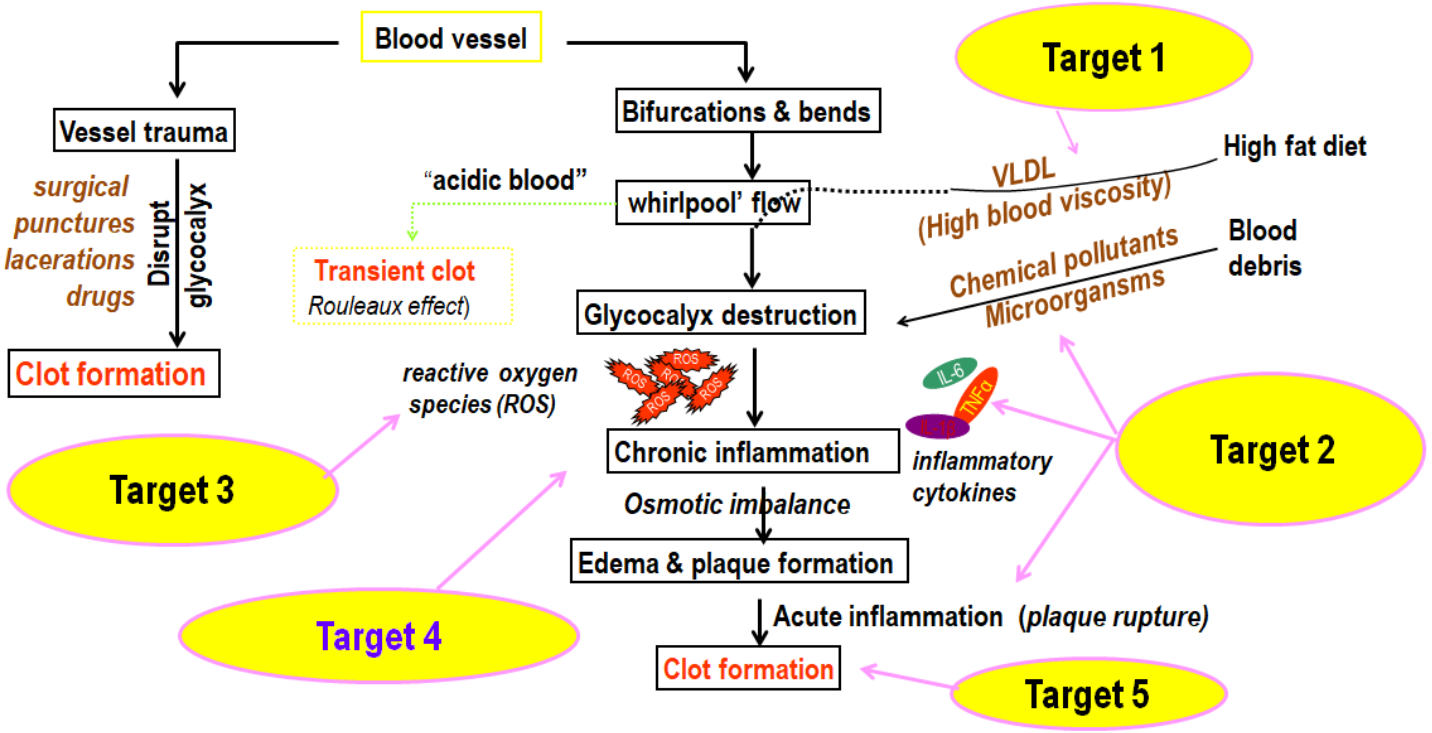
THERAPEUTICS (Preclinical)

PROOF 1:

Dr. Tunac carefully studied the thromboembolic cascade and Identified multiple biochemical sites as 'druggable targets.'

Quest for curative CVD drug

Piecing together a thromboembolic cascade and possible drug targets

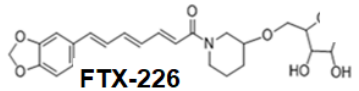


PROOF 2:

Structures of 9 proprietary compounds were then defined and subsequently rationally synthesized for specific targets.

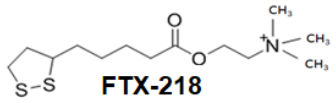
Designed & synthesized drugs for specific targets

Target 1 (VLDL)

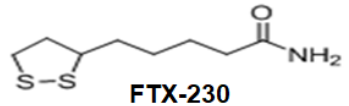


FTX-226
trans-trans-trans piperine-ribose

Target 2 (cytokines)

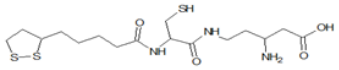


FTX-218
lipoate choline

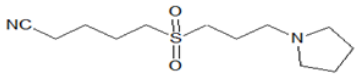


FTX-230
ammonium lipoate

Target 3 (ROS)

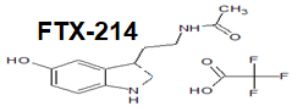


FTX-219
lipoate-cysteine-glutamate

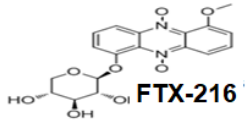


FTX-224
di-oxide isothiocyanate-pyrrole

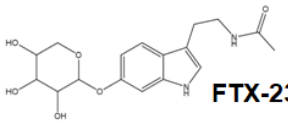
Target 4 (gycocalyx)



FTX-214
indole-acetamide

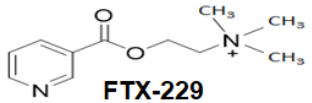


FTX-216
myxin-1β xyloside



FTX-231
Melatonin-6, β-xyloside

Target 5 (thrombin)



FTX-229
nicotinyl choline

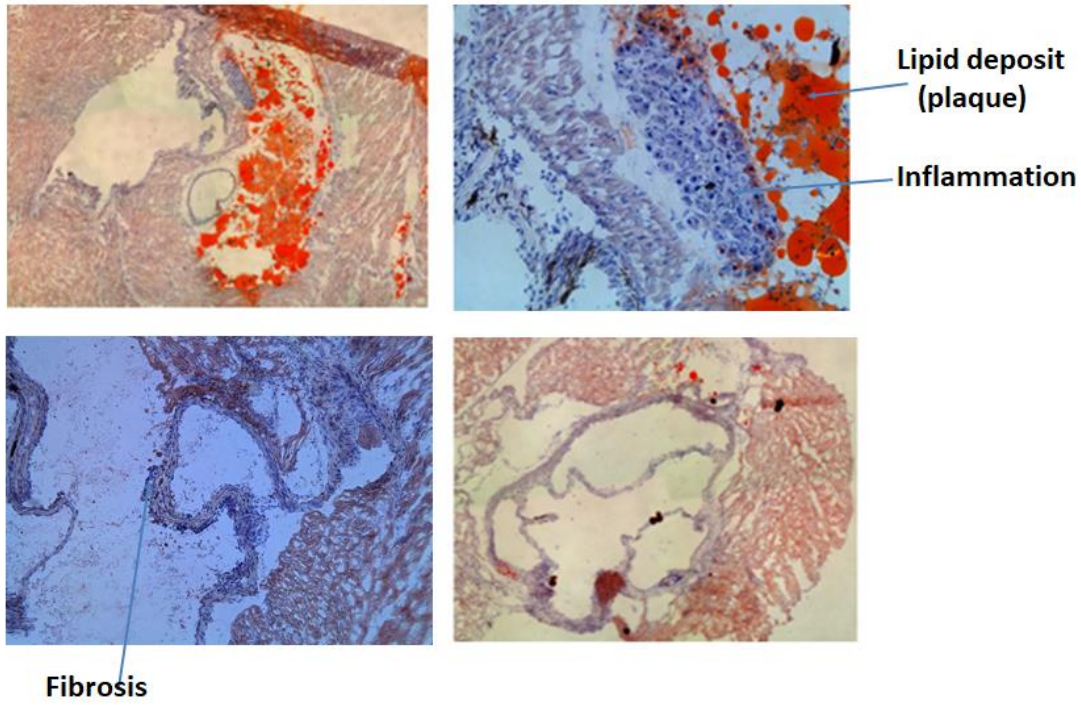
Created an animal model for atherosclerosis

PROOF 3:

Recognizing that the APO-E mouse model is only useful in the study of cholesterol and not the multi-factorial root causes of CVD, Dr. Tunac developed a first-in-kind natural mouse model for atherosclerosis that produces arterial plaques when subjected to common CVD risk factors.

- The new mouse model produced plaques by treatment with agents that disrupt blood flow:
  - *High fat diet*
  - *Proprietary oxidative agent*
  - *Proprietary inflammatory agent*

  (MRI with Iron oxide contrast used to identify endothelial gaps, etc)
- Animals were sacrificed; the hearts and aortic sinus were frozen, sectioned (10 µm) and examined for fibrous tissue, inflammation and plaques
- First time regular mouse produced plaques



PROOF 4:

The discovery process led to a 3-active compound drug design.

Multifactorial disease requires a multi-compound drug.

Note, while several combination drugs showed activity, one combo proved curative and preventive of plaques.

The triple combo K (Embotricin™) was found most effective and has since proved non-toxic in toxicology studies.

The discovery process!

- ***Drugs tested in 3-combo to address multifactor nature of CVD***
- ***Drugs active individually, but curative/preventive only in combo!***

3-combo drugs (FTX)	Blood markers					
	'Hyaluronan'		'Heparan sulfate'		'PAI-1'	
	Preventive	Curative	Preventive	Curative	Preventive	Curative
A. 226/229/216	-	+	-	+	-	+
B. 226/229/214	-	+	-	+	+	+
C. 226/229/218	-	+	-	+	-	+
D. 226/229/219	-	-	-	-	+	+
E. 226/229/230	-	-	-	-	+	+
F. 224/216/214	-	+	+	+	+	-
G. 224/216/219	+	-	+	-	-	-
H. 224/216/219	-	-	+	-	+	+
I. 216/214/218	+	+	+	-	+	+
J. 216/214/219	-	+	-	+	+	-
K. 214/218/219	+	+	+	+	+	+

- **Curative:** *atherosclerotic animal, then drug treatment*
- **Preventive:** *drug introduced before animal made atherosclerotic*

Curative only

Curative/preventive

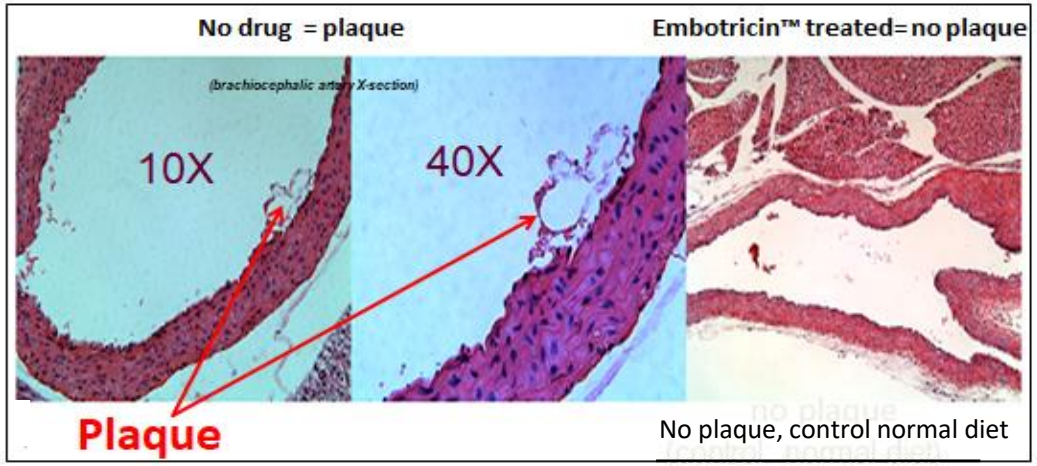
'Combo K' = Embotricin™ (anti-embolic™ drug)

Anti-embolic™ – compound that prevents formation of emboli (clots) involving plaque reduction and/or restoration of disrupted endothelial glycocalyx

PROOF 5:

Embotricin™ proved to prevent and reverse plaque formation. This is a micrograph of a mouse brachiocephalic artery showing plaque in a non-treated natural mouse.

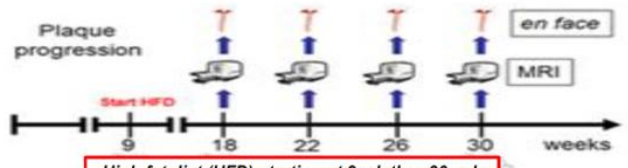
- Embotricin™ was administered before or after the mouse was made atherosclerotic.
- Embotricin™ added to an atherosclerotic mouse reversed plaque formation: curative
- Embotricin™ administered before mouse was made atherogenic prevented plaque formation

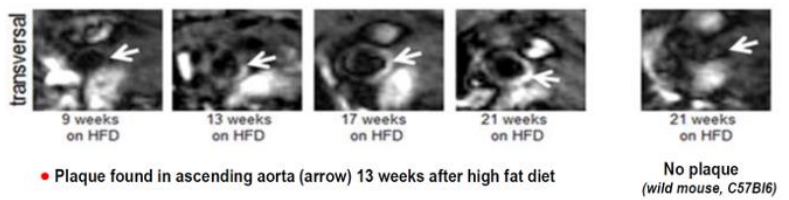


ApoE -/- Mouse MRI Predicate

Published MRI scan in mouse (ApoE-/- mouse)

- ApoE-/- mouse fed with high fat diet (HFD) *2017. PLoS ONE 12(8):e0180407)*





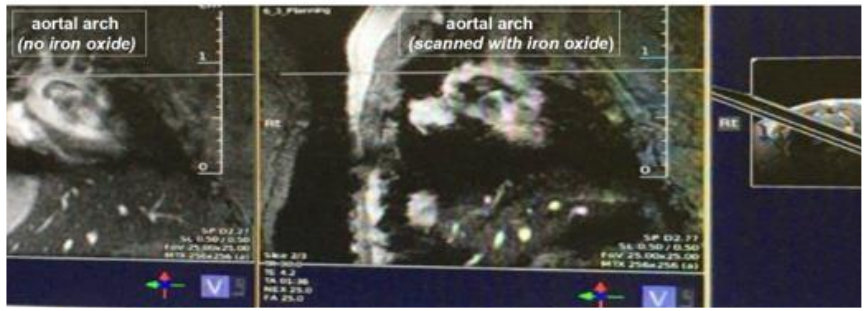
- Plaque found in ascending aorta (arrow) 13 weeks after high fat diet

No plaque
(wild mouse, C57Bl6)

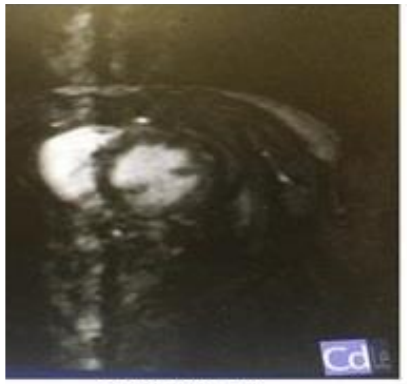
Natural arterial plaque mouse model pre-clinical studies are on-going – patent to be filed Sep,20

Confirmed plaques In natural mouse with MRI

- **Plaques in aortal arch is subendothelial, mimicking human condition**
- **Plaques formed 2wks after PCB treatment** *(vs 13 wks in the (ApoE-/- mouse)*



Established protocol to measure ventricular ejection volume



Ejection fraction volume
(left ventricle)

Henry Ford Health System 7-Tesla preclinical
(vs 1.5 Tesla clinical)

PROOF 6:

Embotricin™ restored (healed) the disrupted glycocalyx lining and prevented further shedding of glycosaminoglycans, which also corroborates published study data. https://www.arterez.com/glycocalyx-studies

Current ongoing translational studies evaluating additional end points such as ejection fraction also prove promising.

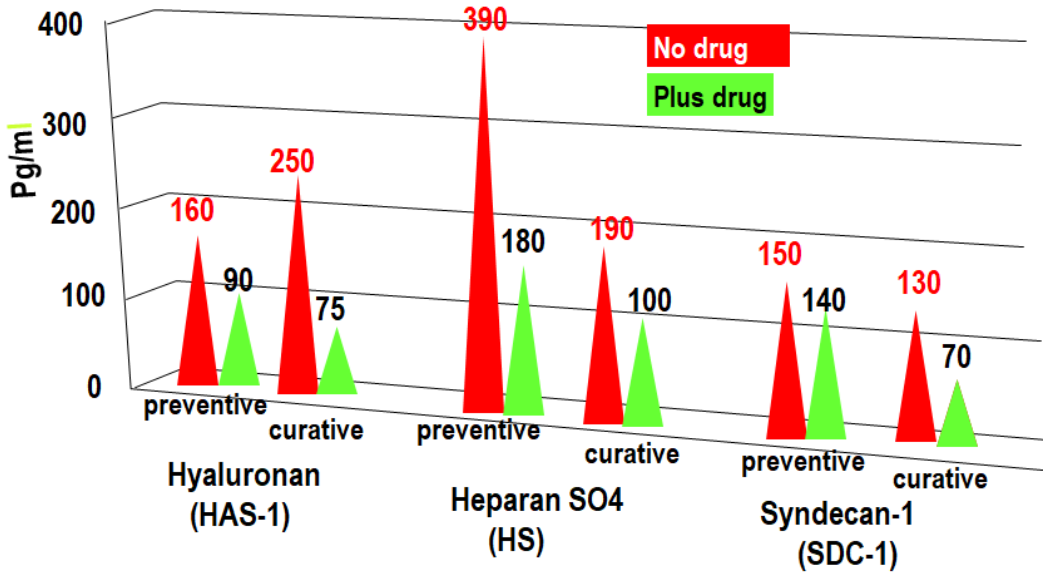
Embotricin™ restored glycocalyx

Preclinical data:

- *Embotricin™* prevented & restored shedding of glycosaminoglycans (GAG), and preventive/curative of plaques

Corroborating clinical data:

- *2011. Ann Surg 254:194–200*: levels of syndecan-1 and heparan SO4 proportional to glycocalyx damage associated with thrombosis & mortality
- *2015. Br J Clin Pharmacol 80: 389–402* shedding of syndecans, heparan SO4 and hyaluronan result in ischaemia, atherosclerosis, diabetes, & renal disease



Embotricin™ reduced PAI-1 & embolism

PROOF 7:

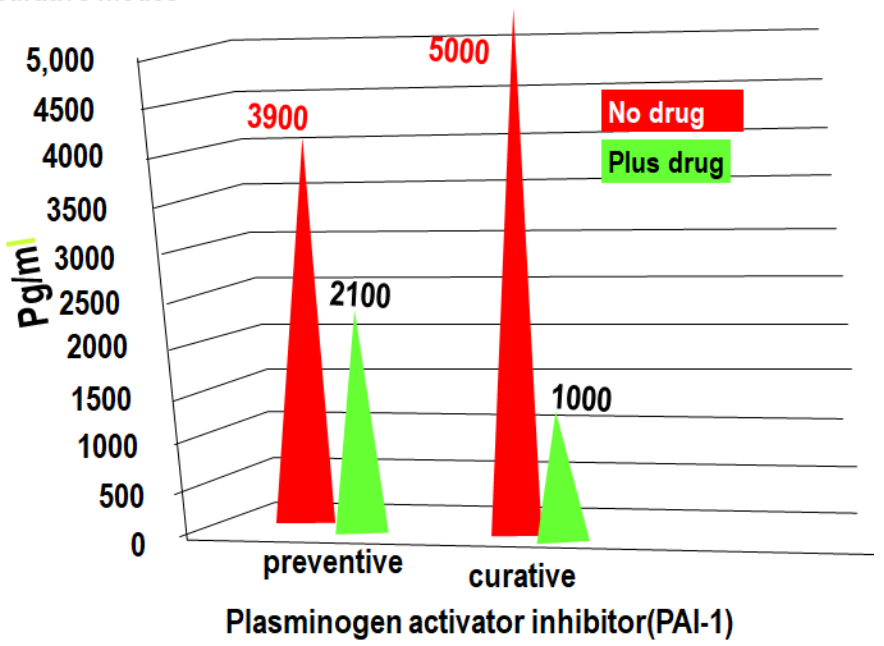
Embotricin™ proved curative and preventive of clot (embolus) formation as evidenced by the marker plasminogen activator inhibitor-1 (PAI-1) in early mouse studies and was subsequently confirmed in histophathology and MRI.

Preclinical data:

- *Embotricin™* reduced PAI-1 levels in both preventive and curative modes

Corroborating clinical data:

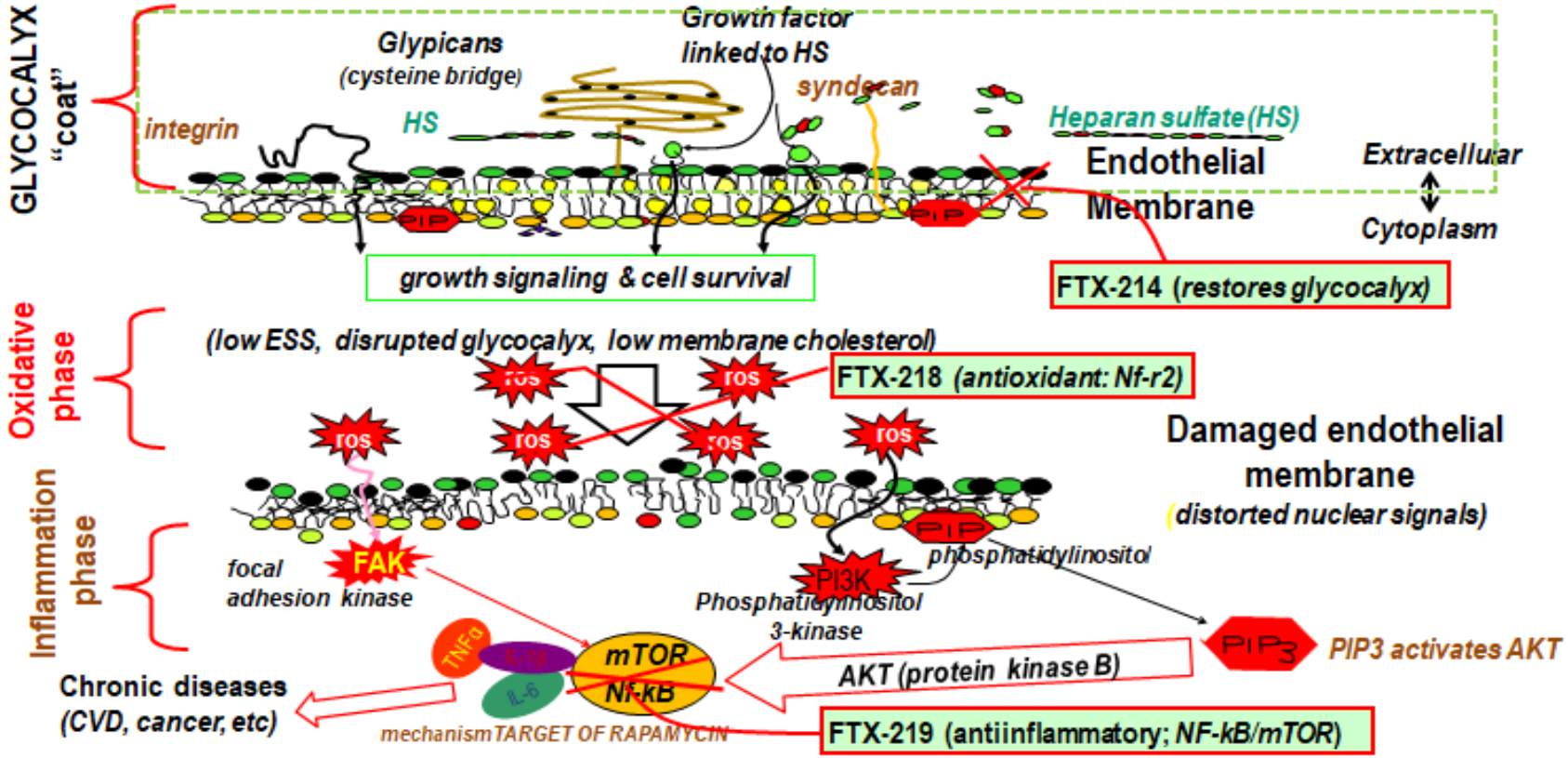
- *1996. Circulation 94:2057–2063:* high levels of plasminogen inhibitor activator-1 (PAI-1) predict onset of myocardial infarction
- *1999. Circulation 99:2496–2498:* ruptured plaque releases PAI-1, which triggers thromboembolism
- *2003. Circulation 108:391–394:* ruptured plaque, poor prognosis for survival
- *2004. J Histochem Cytochem 52:1091– 1099:* increasing PAI-1 levels promote plaque rupture



PROOF 8:

Embotricin™ effectively treats CVD due to its multifactorial mode of action. FTX-214 , FTX-218, and FTX-219 act synergistically to restore the health of the vascular system by (1) restoring the glycocalyx, while (2) simultaneously addressing oxidation and (3) reducing inflammation.

Embotricin™ proposed action sites



PROOF 9:

Dosing Toxicology: FTX 214, 218, 219 were well tolerated when administered up to a dose of 200 mg/kg. Adverse effects were observed only at the 1000 mg/kg dose (3,000 mg/kg in combination) with one animal dying and the other five showing lethargy immediately after dosing, thus the maximal tolerated dose is between 200 and 1000 mg/kg for each compound when administered in combination by a single oral gavage.

TREATMENT NUMBER	DOSE ADMINISTERED (MG/KG)	CONCENTRATION OF EA TEST ARTICLE IN DOSING SOLUTIONS	STUDY DAYS
1	10	0.8	1
2	50	4	4
3	200	16	8
4	1000	80	16

Arterez' diagnostics involve the use of biomarkers to diagnose and assess the risk for cardiovascular disease, which are important aspects in clinical decision making and setting therapeutic strategies, including the use of biomarkers as companion diagnostics to assess the efficacy of compounds and formulations (Embotricin or other) to treat vascular diseases, particularly thromboembolism.

More information available here:
https://www.arterez.com/diagnostics

Context

In the last couple of years, a record number of personalized therapeutic products were approved in the US, establishing an urgent need for companion diagnostics and causing many drug developers to actively seek external support and expertise

Growing Companion Diagnostics Market

>40% Of drugs approved by the FDA in 2018 were personalized therapies

100+ Companion diagnostic tests are currently approved / under investigation

USD 6+ Billion Anticipated net worth of the companion diagnostics market, by 2030

Companion diagnostics have become an indispensable part of modern healthcare; given the growing focus on personalized medicine, the demand for such tools is likely to increase

Need for Contract Services

Cater to Rising Demand for **Diagnostics**

Resolve Technical **Complexities**

Expedite Development **Timelines**

The need for multidisciplinary expertise is anticipated to be the key driver for drug developers to rely on outsourcing the development of companion diagnostics

Current Landscape and Recent Developments

200+ Companies Presently claim to offer services for companion diagnostic development

300+ Trials Focused on potential biomarkers are being conducted by the top 20 pharma players

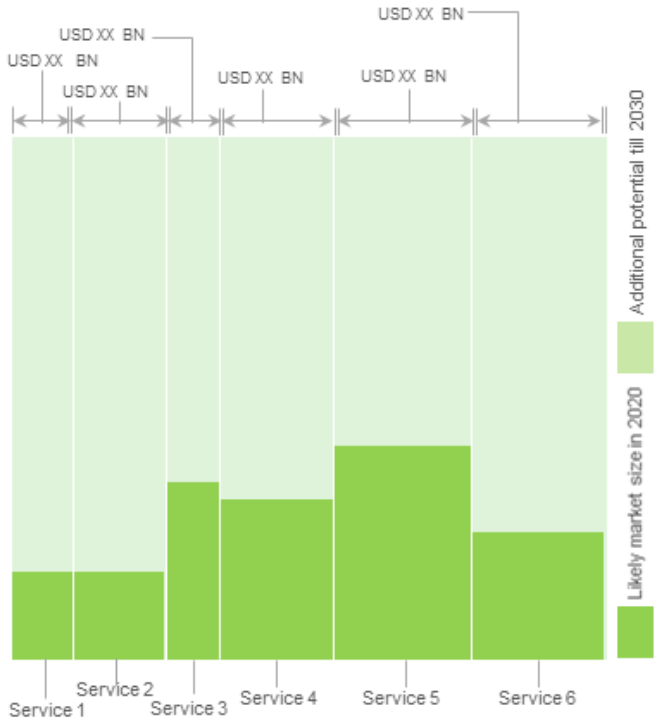
140+ Partnerships Have been inked between stakeholders between 2017 to 2019

Service providers that are active in this field are presently focusing on expanding existing capacities and service portfolios in order to cater to the evolving needs of sponsor companies

Given the growing demand for precision medicine, the companion diagnostics services market is expected to witness a double digit growth; the opportunity is likely to be distributed across different regions and techniques

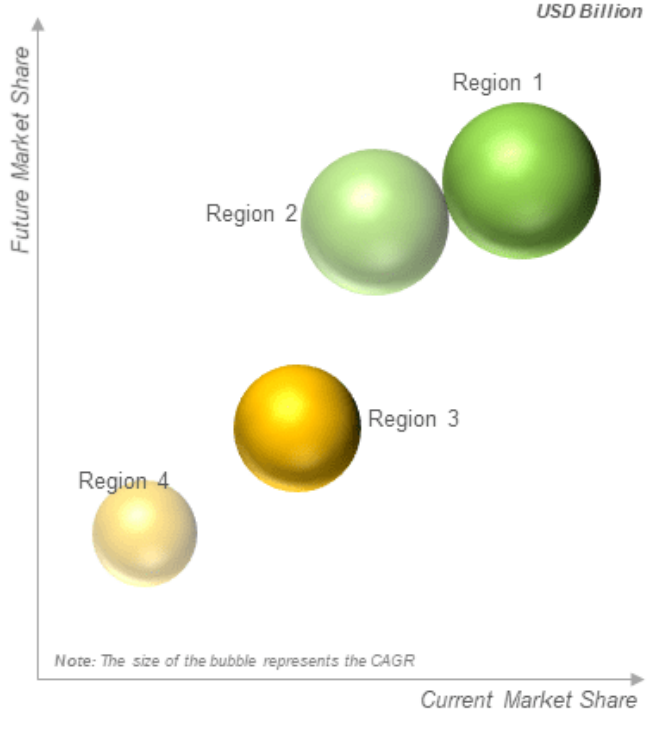
Companion Diagnostics Services Market

Distribution by Service Type: 2020 and 2030[1,2]



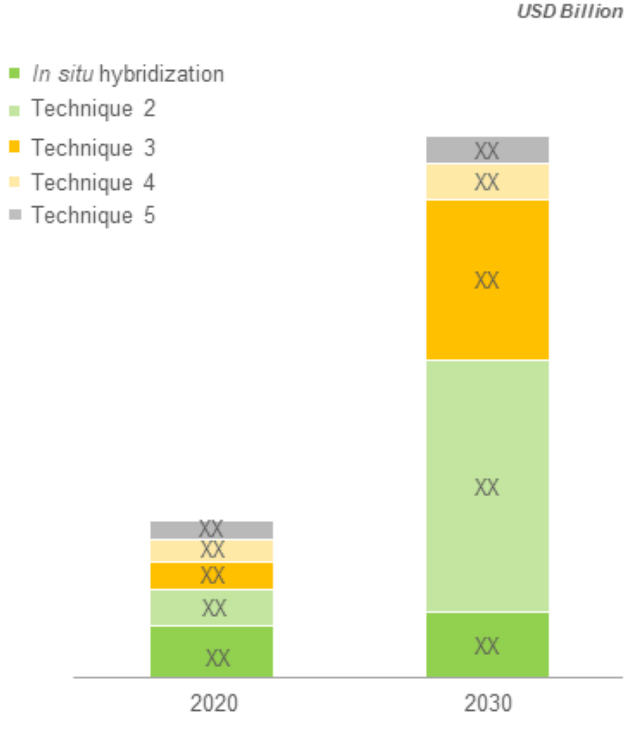
Companion Diagnostics Services Market

Market Attractiveness Analysis by Region[1,3]



Companion Diagnostics Services Market

Distribution by Techniques: 2020 and 2030[1,4]

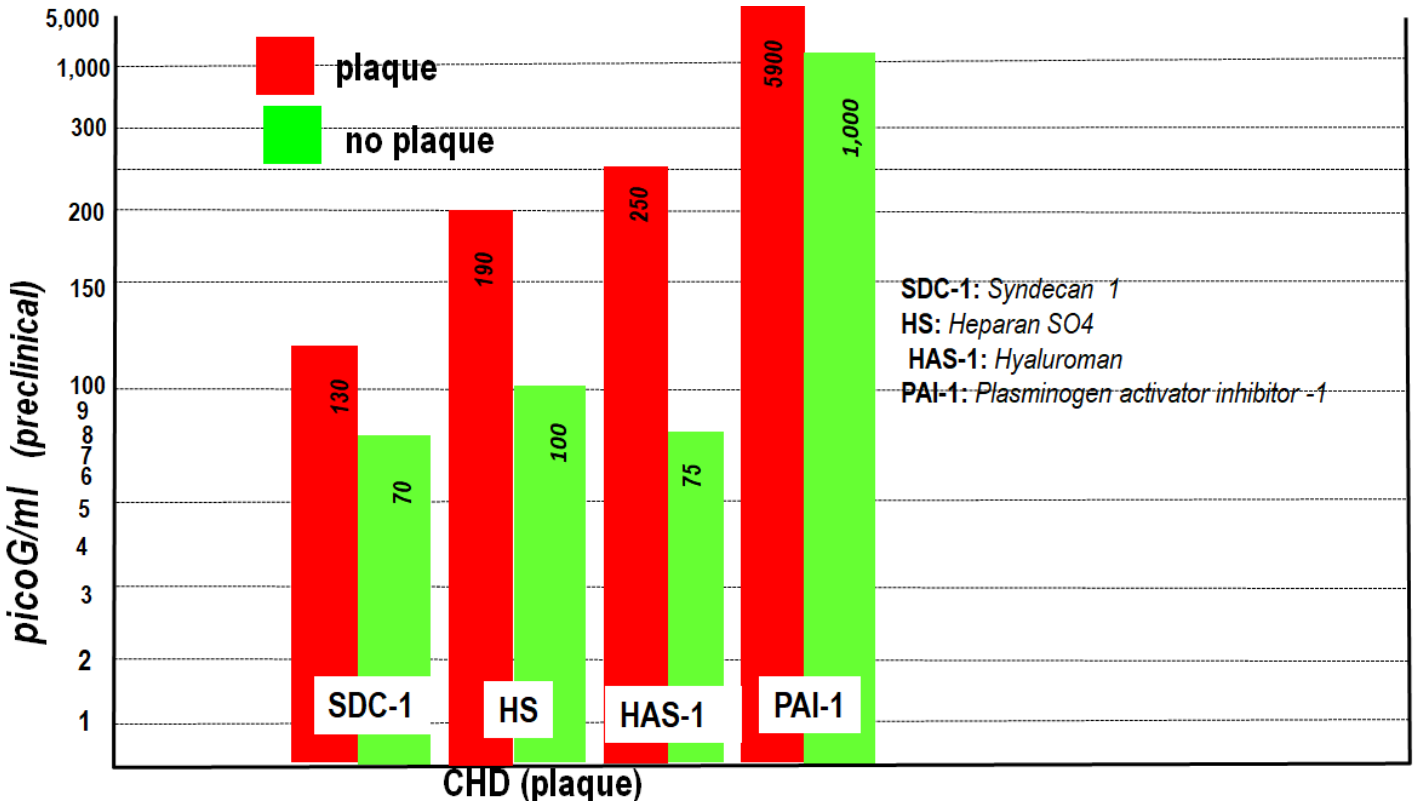


Note 1: Illustrations are not as per actual scale
Note 2: The *Companion Diagnostics Services Market, 2020-2030* report takes into consideration the following services: biomarker discovery, feasibility studies, assay development, clinical validation, analytical validation and manufacturing
Note 3: The *Companion Diagnostics Services Market, 2020-2030* report takes into consideration the following regions: North America, Europe, Asia and rest of the world
Note 4: The *Companion Diagnostics Services Market, 2020-2030* report takes into consideration the following analytical techniques: ISH, NGS, IHC, PCR and others

PROOF 1:

The characteristic feature of CHD is plaque formation resulting in atherosclerosis or hardening of the arteries. Plaque formation is triggered by glycocalyx disruption and the shedding of glycocalyx detritus. In this regard 4 glycocalyx detritus were selected as components of the fingerprint, namely: syndecan-1 (SDC-1), heparan SO_4 (HS), hyaluroman-1 (HAS-1 :), and plasminogen activator inhibitor -1 (SDC-1). Our mouse model was used to model plaque formation. Indeed the blood levels of the 4 detritus correlated with plaque formation.

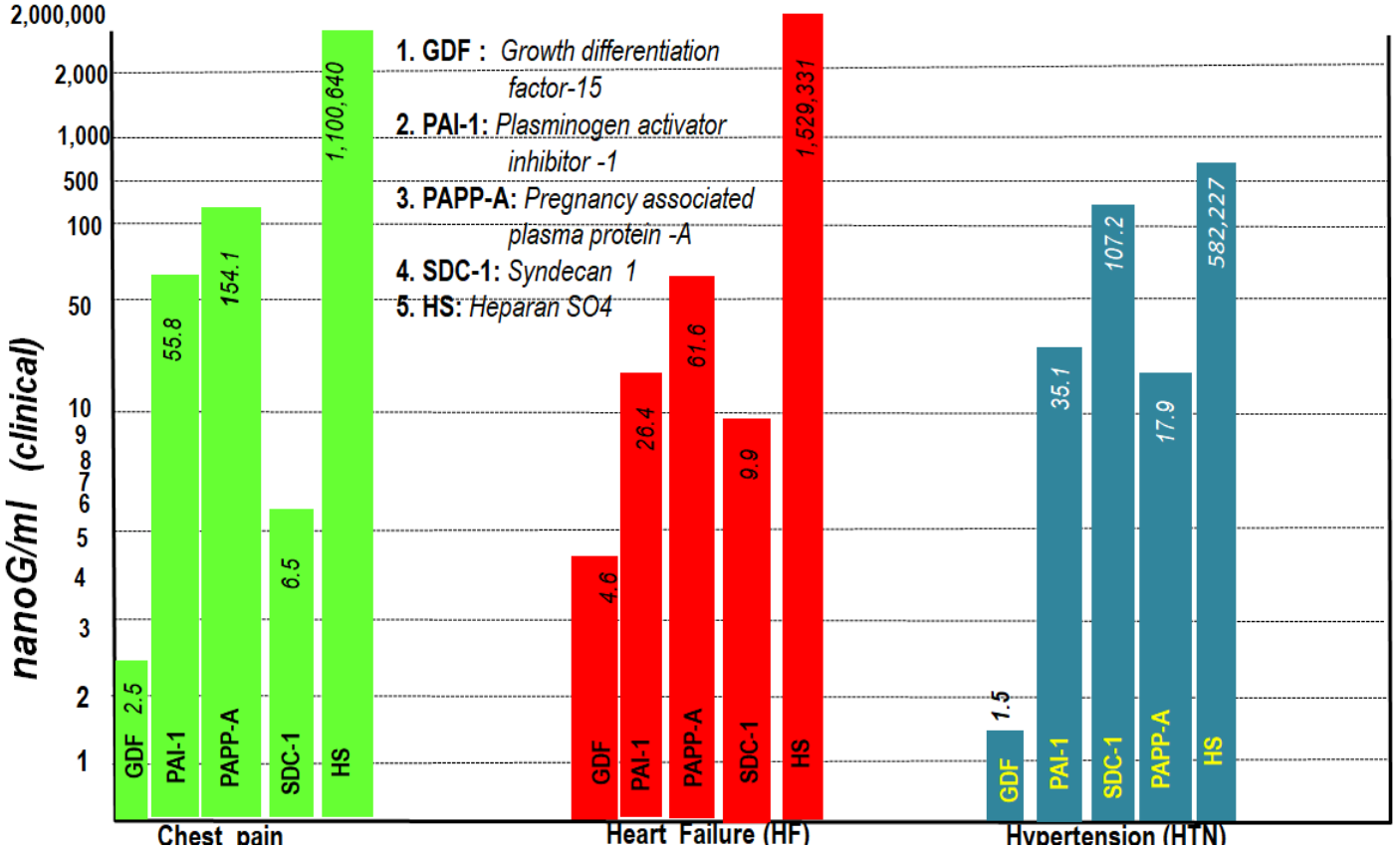
4-panel GlycoCalyx Detritus Fingerprint™ (*Glycocardia^{CHD}*): preclinical



PROOF 2:

The correlation of blood levels of the 4 glycocalyx detritus to plaque formation prompted the evaluation of IRB clinical samples. These clinical samples represented blood withdrawn from patients suffering from chest pain, heart failure (HF) and hypertension (HTN); Fingerprint of 3 diseases (chest pain, heart failure, hypertension) which are members of the CVD family, showed significantly different levels of each of the biomarkers, differentiating each disease from the other.

5-panel GlycoCalyx Detritus Fingerprint™ (*Glycocardia^{HF}*): clinical



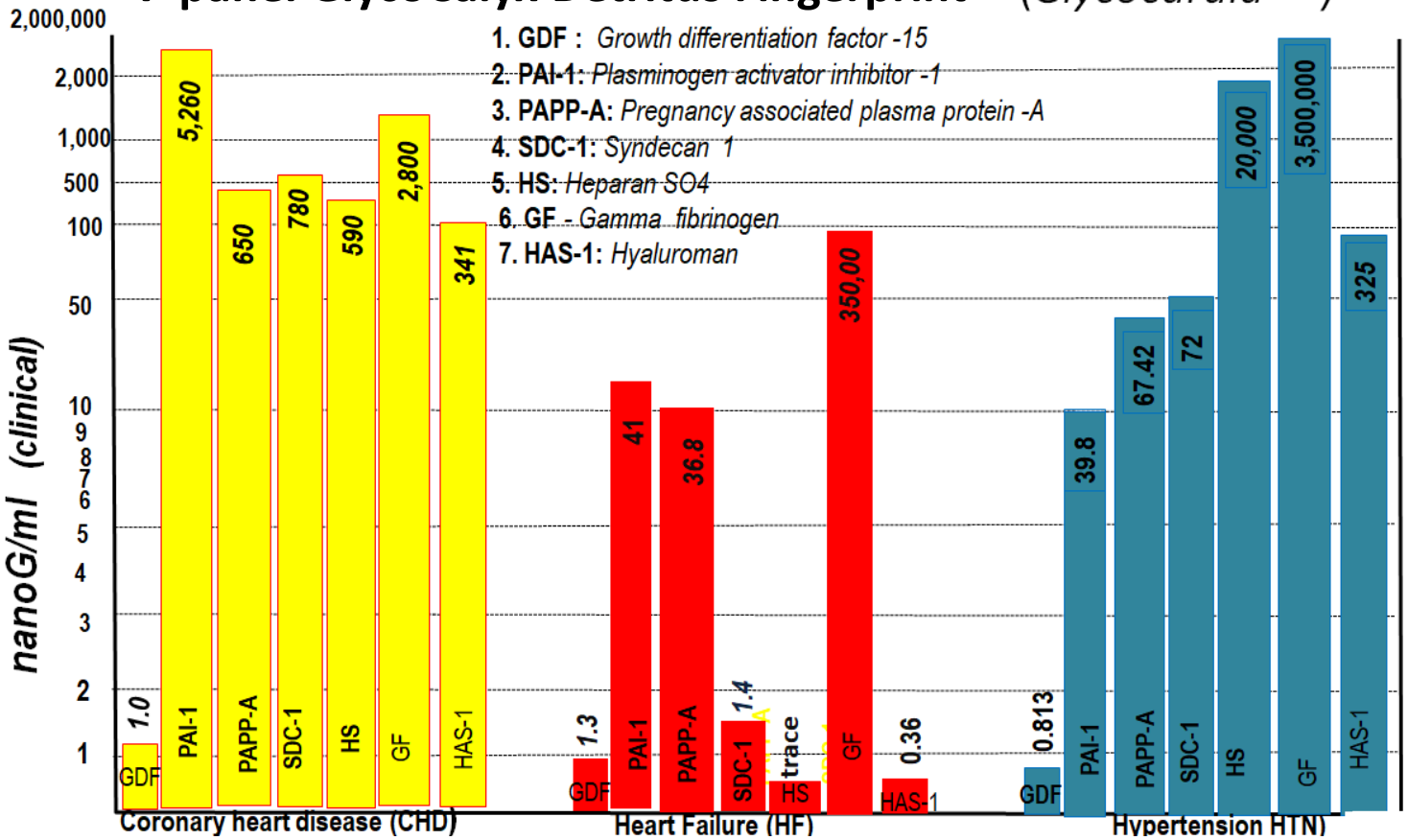
PROOF 3:

ELISA Blood levels from the 7 detritus components in our pending patents were obtained from published literature of patients with coronary heart disease (CHD), heart failure (HF), and hypertension and a virtual fingerprint was constructed.

Each disease showed a unique fingerprint, which confirms the hypothesis of the Glycocalyx Detritus Fingerprint™ as a unique tool for identifying diseases currently in development.

More information can be found here:
https://www.arterez.com/glycocalyx-studies

7-panel GlycoCalyx Detritus Fingerprint™ (*Glycocardia^{GEN}*)



3-Disease Remnant Study (SBIR)

AIM 1: Qualify ELISA assays for quantitating 7-marker levels using 250 human serum (remnant) samples with a clinical diagnosis of coronary artery disease (CAD), hypertension and heart failure.

AIM 2: We have designed a statistical process and methodology to develop the foundation of disease algorithms. This will be accomplished by using the proposed study data to arrive at a path analysis to establish causality between and amongst the biomarkers and indications, beginning with CAD.

6 Disease IRB Pilot Study (ARBOR ASSAYS)

AIM 1: Qualify ELISA assays (and Mass Spec if feasible) for quantitating 7-marker levels using 21 IRB approved human serum samples (plus control group) - with a clinical diagnosis of CAD, hypertension, heart failure, diabetes type II, rheumatoid arthritis and stroke.

AIM 2: Utilize same statistical process and methodology as above to develop the foundation of disease algorithms for further ELISA focus and development across multiple vascular diseases beyond the family of CVD. This will be accomplished by using the proposed study data to arrive at a path analysis.

USES:

GlycoCardia can be used twofold:

1) As a companion diagnostic for custom therapies (e.g., Embotricin™),
2) 'Stand-alone' predictive diagnostic to monitor or evaluate the traditional symptom-targeted therapies.

HIGHLIGHTS:

1) Provides specific information concerning the associated pathology and risk factors for the CVD family
2) Unique and proprietary blood test algorithm
3) Adoptable ELISA test structure; utilizes existing equipment found in both acute and chronic care settings
4) Proved to predict onset of clot and production of plaques in tested animals
5) Currently no diagnostic test available providing patient specific information concerning the pathology or the absolute risk factors for the family of CV Diseases.

Note: companion diagnostics guided drug development efforts have demonstrated to effectively reduce clinical trial costs by almost 60%
--Root Analytics, Jun20.

Glycalyx Detritus Fingerprint™ Technology (GDF) – Proof of Principle The rapid and correct identification of diseases is crucial and important as a guide for appropriate therapy. This involves the correlation of various pieces of information followed by the recognition and differentiation of patterns or ‘fingerprints’. The GDF is a first in kind technology that could mark a new era in healthcare.

This “biomarker panel” technology, as opposed to a single biomarker, increases the accuracy of diagnosis and enables disease identification, classification and disease staging serving as guide for improved therapies that target the multi-factorial root causes of chronic disease, specifically damage to the glycocalyx.

More information available here:
https://www.arterez.com/diagnostics

Glycalyx Detritus Fingerprint™ (GDF) – Glycocalyx debris or detritus is the foundation for GlycoCardia™ and a novel, evolving "fingerprint" diagnostic technology targeting chronic disease.

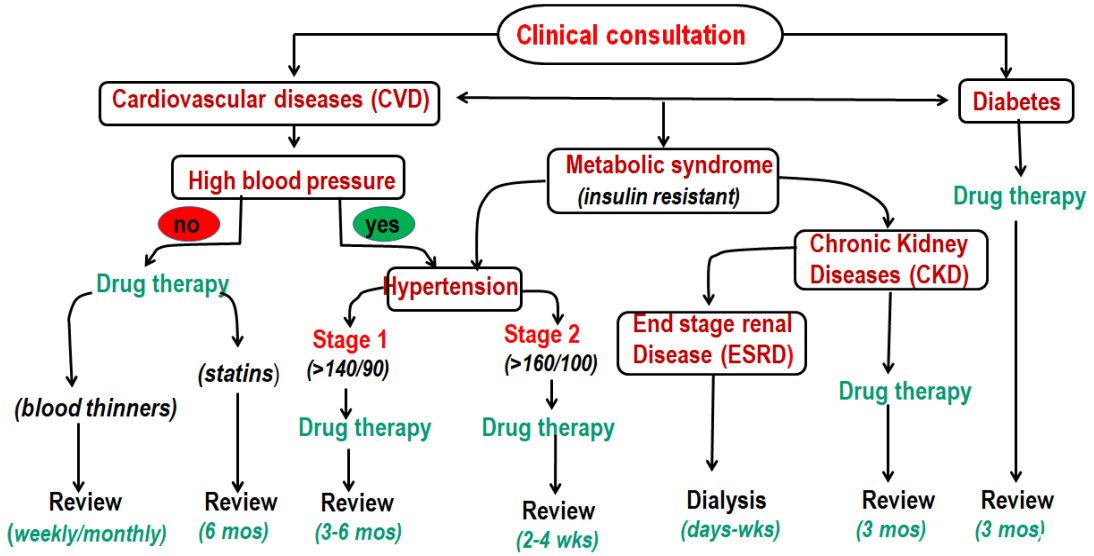
Currently, there is no equivalent technology developed for early or predictive disease diagnosis.

The GDF™ technology will be the first to identify, predict, diagnose and treat chronic disease, *a discovery that could offer significant clinical value.*

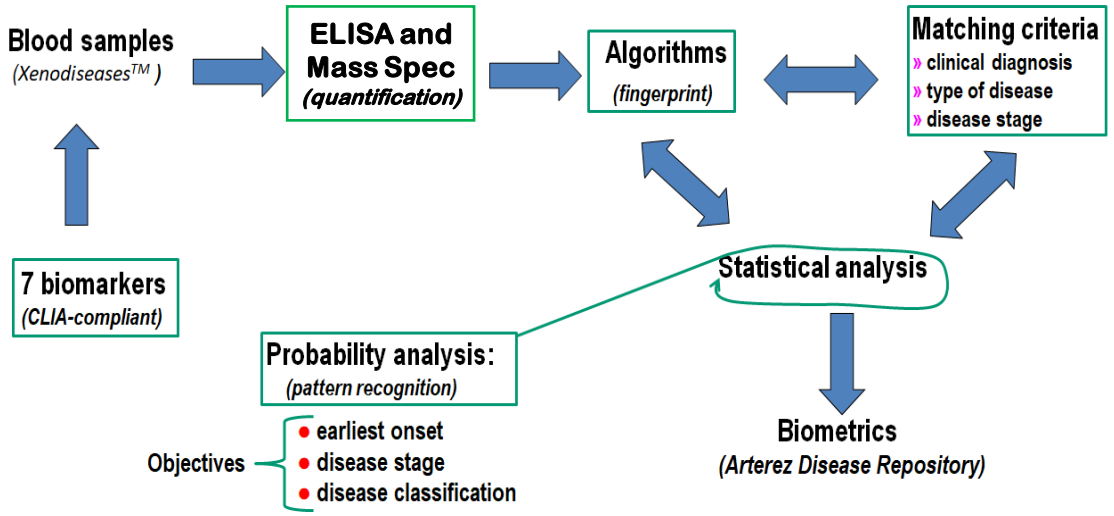
Clinical ELISA and Mass Spec studies in progress.

In Q1 2021, Arterez will engage diagnostic partners to co-develop the technology.

Glycalyx Detritus Fingerprint™ (GDF) Treatment Guide



Arterez Disease Repository Database Development

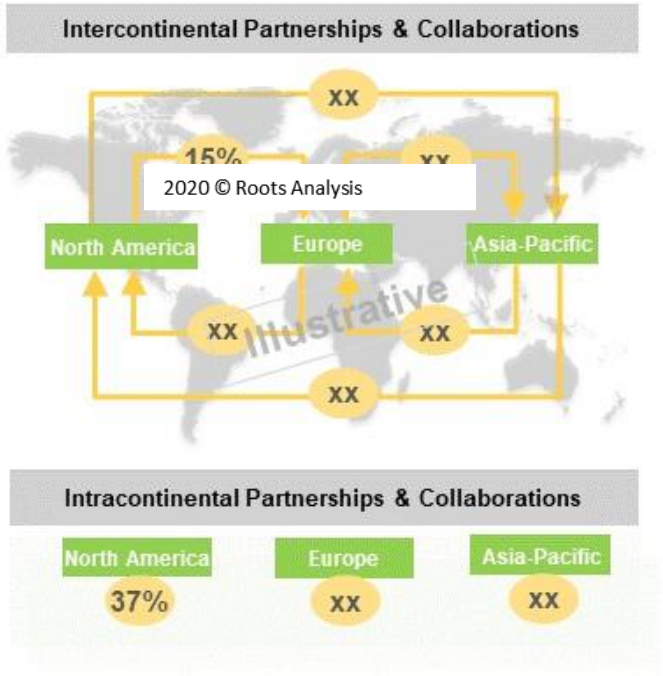


The rise in interest in this field is reflected in the number of partnerships established in the recent past, involving both international and indigenous stakeholders, and focused on a variety of end objectives

Partnerships and Collaborations
Distribution by Type of Partnership

During 2017-2019, partnership activity in this domain has increased at a CAGR of 25%. In fact, maximum activity being reported in 2018

Partnerships and Collaborations
Geographical Activity



Firms that have signed multiple deals within North America include Covance and Illumina; active players in other regions include Almac, R-Biopharm ad Roche

Partnerships and Collaborations
Key Value Drivers[1]

Co-Development
Recent Example: Lucence Diagnostics and MEDx co-develop the companion diagnostic tests for PD-L1 (Nov 2019)

Geographical Expansion
Recent Example: Acquisition of N-of-One by QIAGEN (Jan 2019)

Geographical Consolidation
Recent Example: Acquisition of uBiome by Psomagen (Dec 2019)

Strategic Alliances
Recent Example: PPD and NeoGenomics Strategic Alliance for Pathology and Molecular Testing Solutions (Jun 2018)

Access to Innovative Technologies
Recent Example: Acquisition of Provista Diagnostics by Ascenda Biosciences (Jun 2019)

Note 1: The filled (grey) arrows represent the relative popularity of different value drivers.

Company Summary

DEVELOPMENT TIMELINE

Arterez™ Diagnostics and Therapies

SLIDE 39

2012-2017

2012

- Dr. Josefino Tunac launches Farmaceutix, LLC (FTX)
- R&D for anti-embolic™ markers & compounds*

2013

- Surrogate markers identified for Development/testing of compounds
- Developed natural arterial plaque mouse model

2014

- Rationally synthesized 9 synthetic compounds
- 3x combo proved to prevent & reverse arterial plaques

2015

- Proved non-toxic up to 20x effective dose
- Engaged firm to assist with Platform development and funding

2016

- New studies launched for Natural model, biomarkers
- Compounds/methods, biomarker patent filed

2017

- FTX acquires control & rights to all intellectual properties
- Patents amended, audit completed, development pathway established

2018-2023

2018

- Arterez launched, FTX IP assigned, team developed
- Website built, new IP filed & issued, providers engaged

2019

- Seed raise, biomarkers added, ELISA & HFHS** studies engaged
- Glycalyx Detritus Fingerprint™ and natural model validated

2020

- 6 disease IRB pilot study, NCE's manufactured to GLP
- ELISA, pre-clinical studies, GAP analysis, plan, series A

2021

- Formulation (individual & combo), chronic toxicology, Pre-IND
- Series B, patient recruitment, Australia review + IND/IRB filings

2022

- Phase 1 healthy, limited 1B studies vs CHD, HF, HTN
- IP valuation, engage licensing partner/s and/or suitors

2023

- Complete co-dev, licensing or other for diagnostic and therapy
- Consider IPO to continue development of platform

*4 surrogate markers identified, 9 compounds designed and rationally synthesized to target multi-factorial root causes of CVD – also proved interesting for other vascular pathophysiologies.

***University Research Study – 7 marker ELISA 32 patients vs HF, HTN, Chest Pain*
***Henry Ford Health Systems (HFHS) MRI translational studies using TAP model.*

Company Summary

CLINICAL PROOF OF PRINCIPLE

Arterez™ Diagnostics and Therapies

SLIDE 40

IRB-initiated proof-of-principle clinical evaluation of *Embotricin™* vs patients with coronary artery disease

Compare GlycoCardia ^{CVD} vs arteriographic imaging techniques, e.g.,CCTA (coronary computed tomography angiography), MRA (magnetic resonance angiography) or MRI (magnetic resonance imaging)

Expected outcome *"Embotricin™ reduces plaque in CHD patients, monitored by GlycoCardia^{CHD}*

IRB-initiated proof-of-principle clinical evaluation of *Embotricin™* vs patients with hypertension

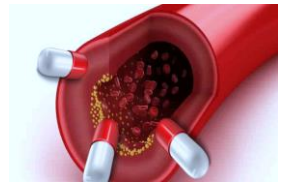
Expected products *"Embotricin™ improves HTN in patients, monitored by GlycoCardia^{HT}*

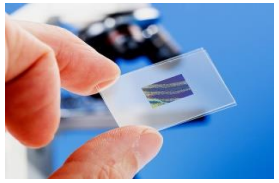
IRB-initiated proof-of-principle clinical evaluation of *Embotricin™* vs patients with heart failure

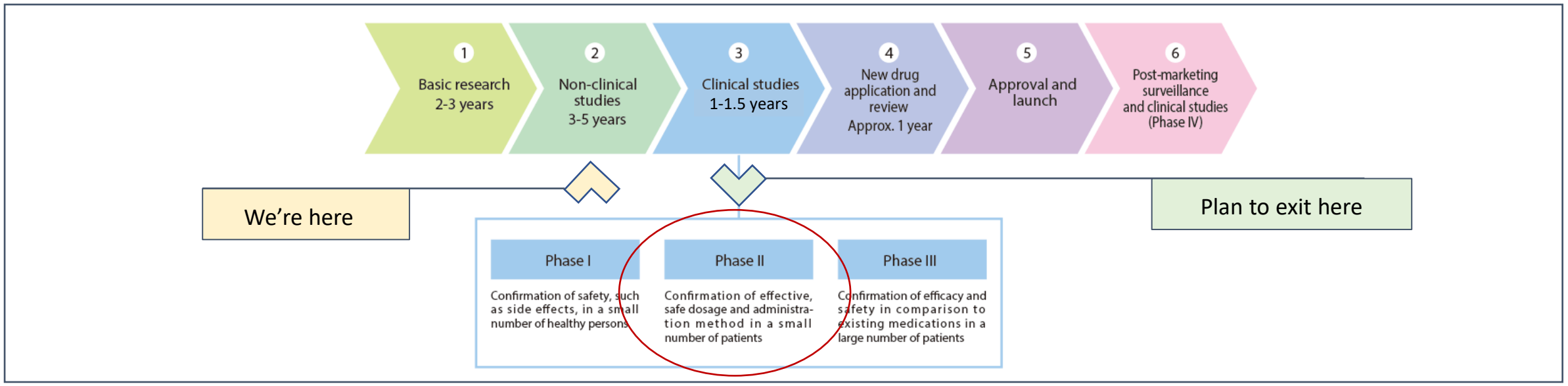
Systolic dysfunction (SD, HFrEF) or diastolic dysfunction (DD, HFpEF)

Compare "GlycoCardia ^{CVD} vs echocardiogram (Doppler) and cardiac magnetic resonance (CMR)

Expected products *"Embotricin™ improves ventricular ejection fraction, monitored by GlycoCardia^{HF}*







THANK YOU FOR TIME
AND CONSIDERATION.

www.Arterez.com