www.Arterez.com

Q1, 2020

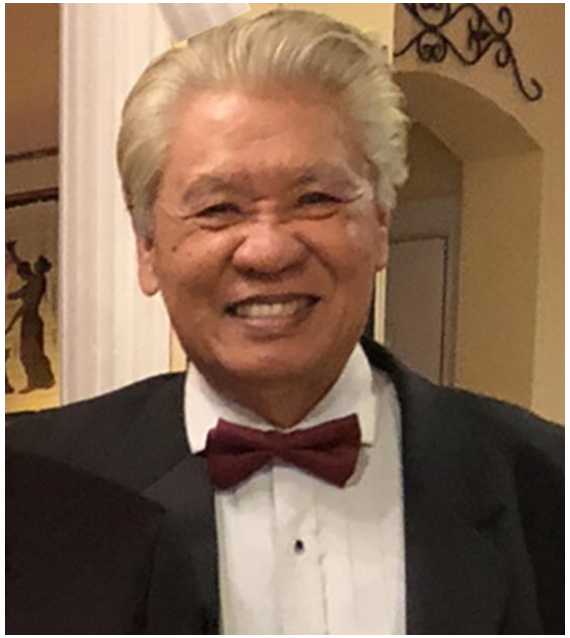
MISSION/VISION

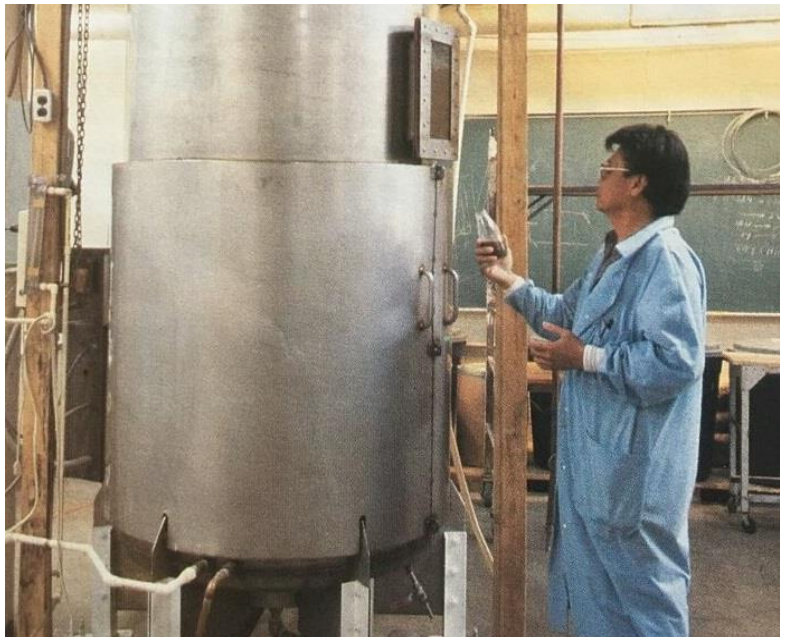
To develop companion and predictive diagnostic panels as well as preventive and curative oral therapies targeting the multifactorial root causes of CVD, supported by a patented portfolio of novel, synthesized compounds, methods and biomarkers.

MISSION/VISION

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 Waksmar 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 antifunga 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 funair flask system, keep climbing. Tunac, an internationally recognized Detroit-area microbiologist said his heat-resistant shake-flasks "are destined to revolutionize the fermentation process for microbiologists around the globe. "For microbiologist fermentation is a crucial part of their research. The

EDUCATION & CAREER

Education

U of Philippines – BS, Plant Pathology
So. Dakota State – Masters, Plant Pathology

Penn State – Microbiology Ph.D. program

Rutgers (Waksman Instit) – Ph.D. & 1st drug
world center for antibiotic research

Merck – Dir of Research

Avermectin (*Ivomec*: *2015 Nobel Prize*)

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 "hunting," discovery

Tunair Labware flasks & bioreactor
– still sold worldwide today

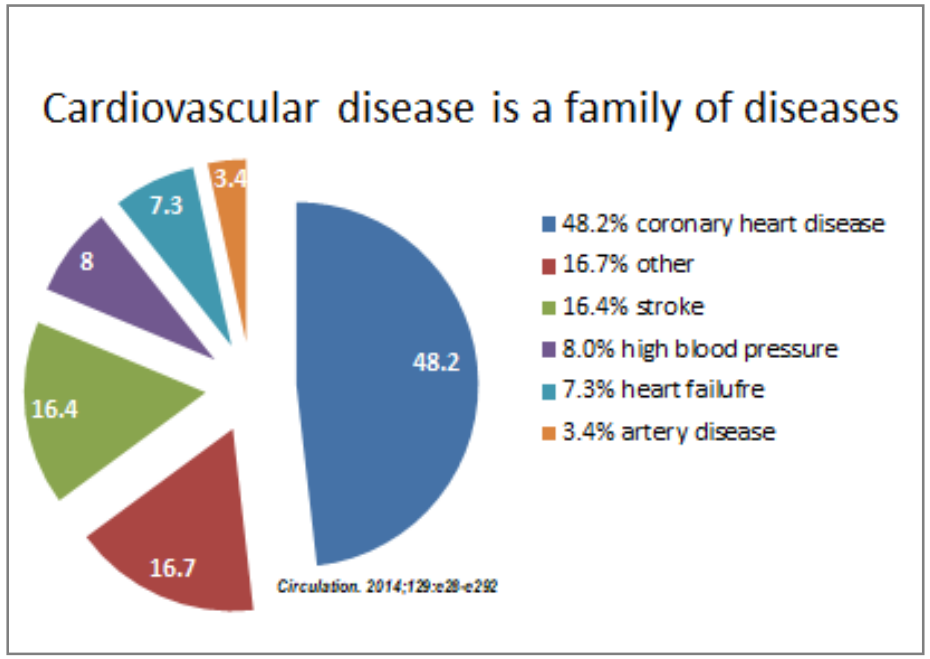
Supergen (SUPG: NASDAQ) – Dublin, CA
Anticancer (Mitomycin)
Licensed to Astex Pharma (ASTX)
Sold to Otsuka Pharma for $886M

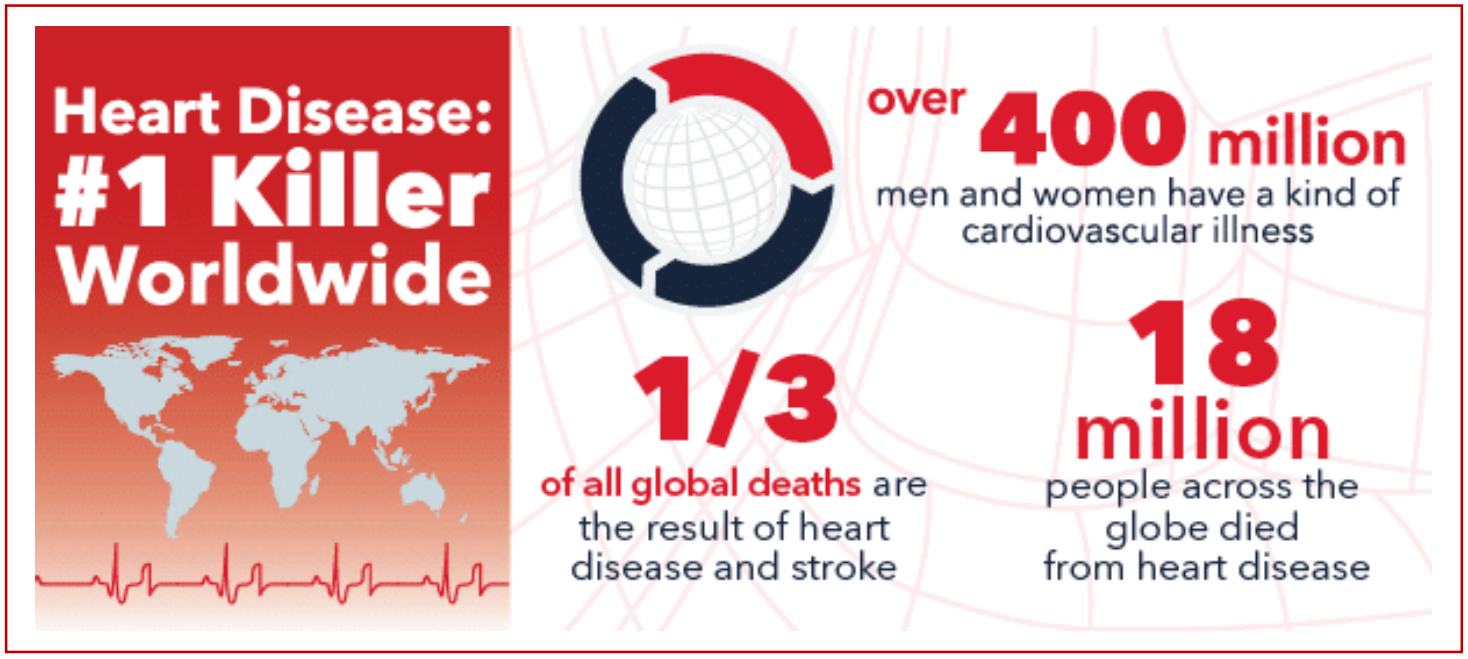
JJ Pharma, Inc – San Ramon, CA
Anti-arthritis Drugs

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

Farmaceutix, LLC – Ferndale, MI (2012-2018)
Anti-Embolic™ drugs

CVD LEADING KILLER

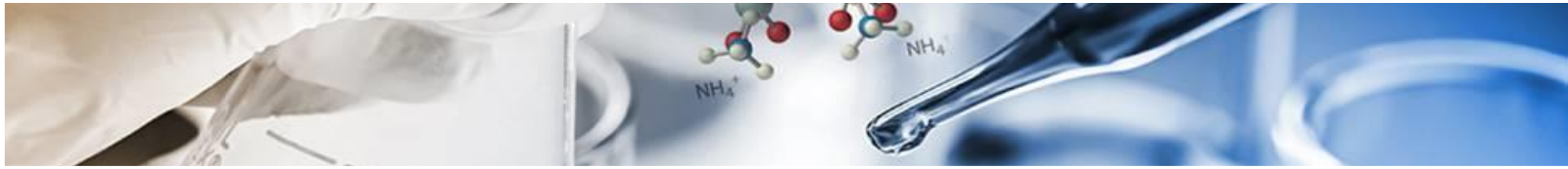




- 400 M with CVD illness at any given time
- 1/3 of all global deaths
- In all developing nations, CVD is largest health risk and cost
- US costs rising to an est. $1.5 trillion by 2030
- No predictive diagnostics or preventive, curative therapy exist

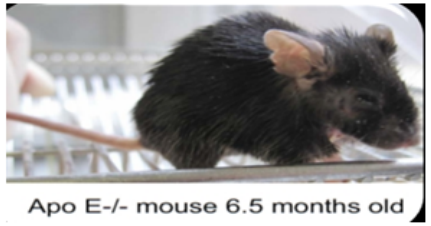
Statins, most prescribed drug in US: > 350 M filled in 2018

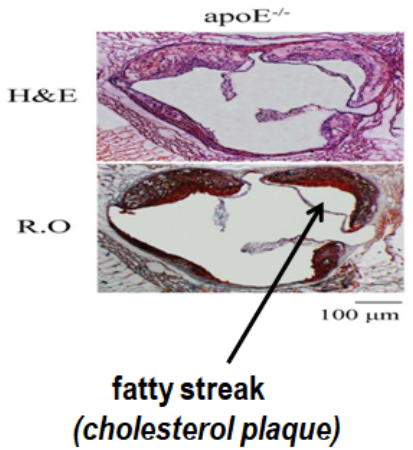
APO E MODEL: ***A NIH-funded genetically altered mouse to support the "cholesterol hypothesis" linking high cholesterol to plaque formation and atherosclerosis.***



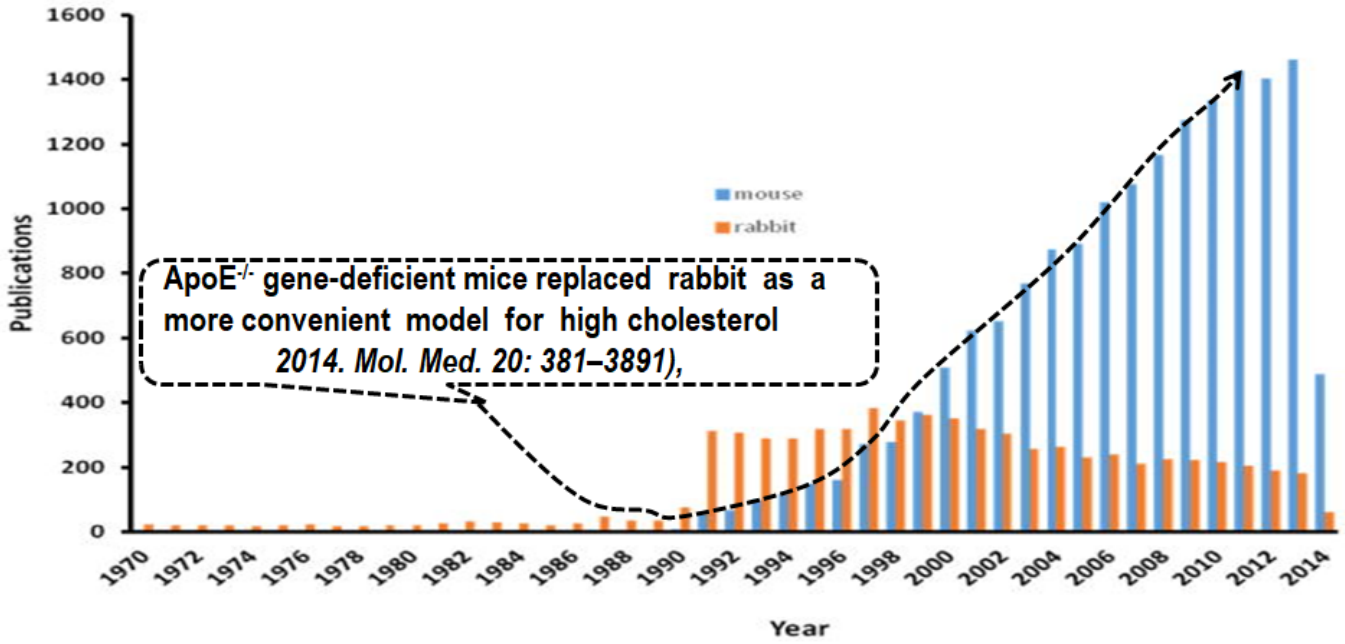
Creation of apoE high-cholesterol mouse

- 1992: ApoE$^{-/-}$ knock-out (KO) mouse – *apoE gene (responsible for cholesterol absorption) was removed (1992. Cell. 71: 343–353 and; 1992. Science. 258: 468–471).*
- thus, dietary cholesterol accumulates in the artery and produce 'fatty streaks' or 'cholesterol plaque'


Apo E-/- mouse 6.5 months old

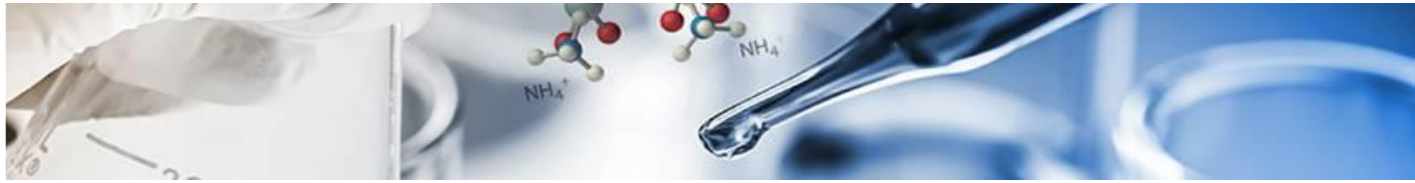


fatty streak
(cholesterol plaque)



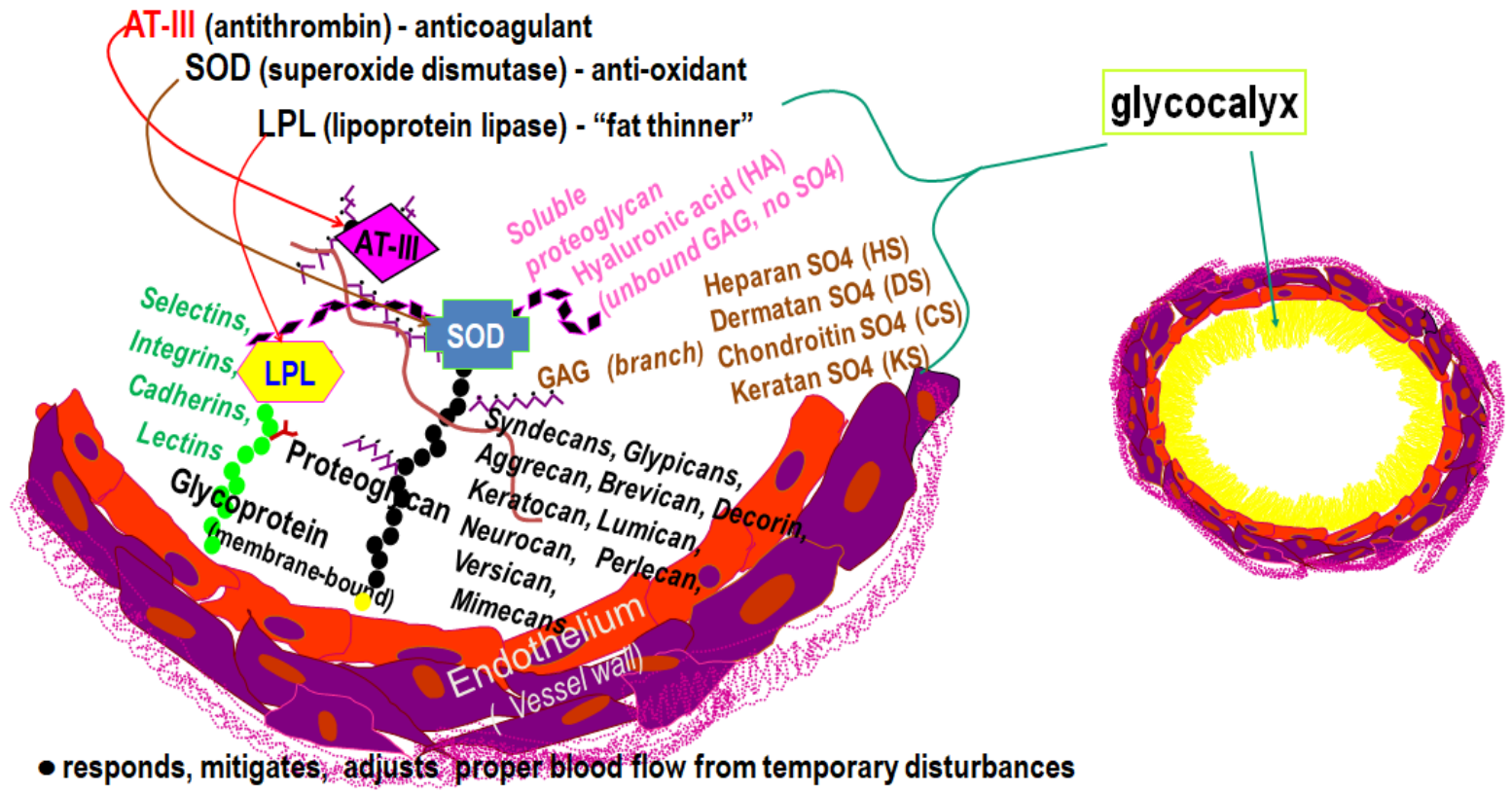
In an ApoE mouse, the absence of the apoE binding protein allows lipoproteins to circulate in the blood stream and eventually "stick" on the arterial wall and accumulate resulting in 'cholesterol' plaques or hypercholesterolemia. This is not how plaques are formed in humans, yet the ApoE remains the model of choice in the study of atherosclerosis.

FACT A: *CHOLESTEROL IS NOT THE CAUSE OF CVD, rather It begins with disruption of the glycocalyx which is the protective coating of the vasculature essential to maintain healthy blood flow.*



Glycocalyx protects endothelium

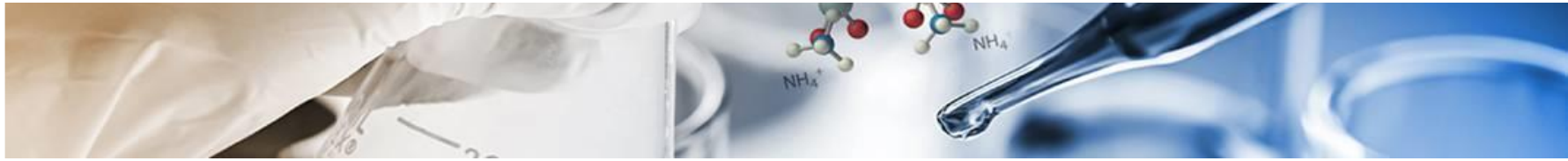
Provides a 'nest' to 3 key enzymes that regulate blood flow



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

In humans, the endothelial glycocalyx (GCX) is the fine protective inner layer of the artery and serves as a nest for important glycoprotein and proteoglycan components.

FACT B: ***Glycocalyx disruption triggers CVD and a plethora of other chronic diseases.***



Disruption of glycocalyx triggers diseases

- The pathological changes to glycocalyx are manifested in both cardiovascular and non-cardiovascular 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)

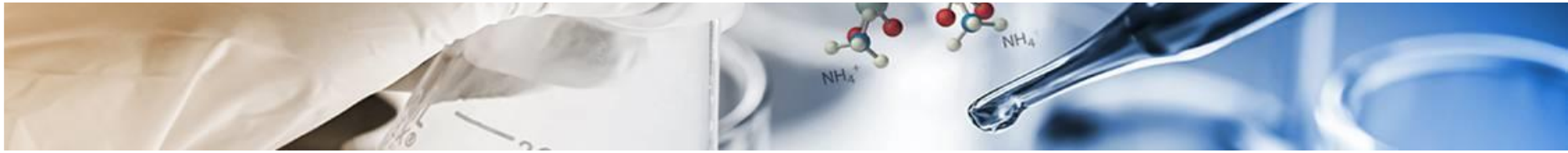
Epithelial diseases *(non-endothelial and/or endothelial)*

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)*

It is the disruption of the glycocalyx, not cholesterol, that is the cause and thus key to solving vascular-related 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-disease.'

FACT C: *A high Fat diet thickens the blood thus slowing blood flow.*



Fat triggers CVD, not cholesterol

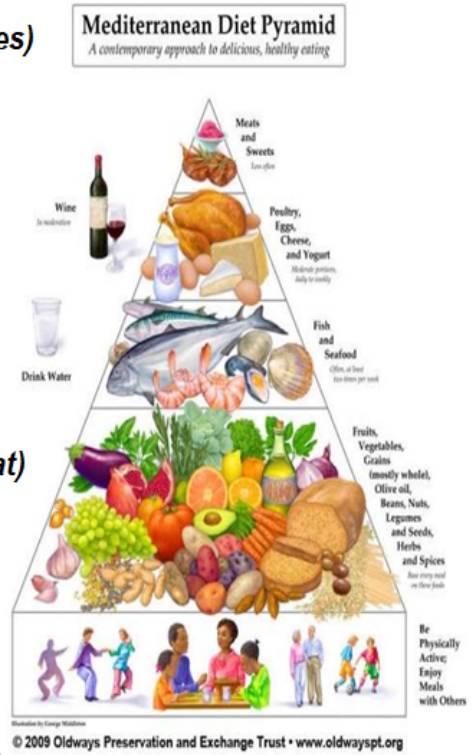
1958: Ancel Key's Mediterranean diet "Seven Countries Study" showed low CVD *because of less dietary fat*

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)*

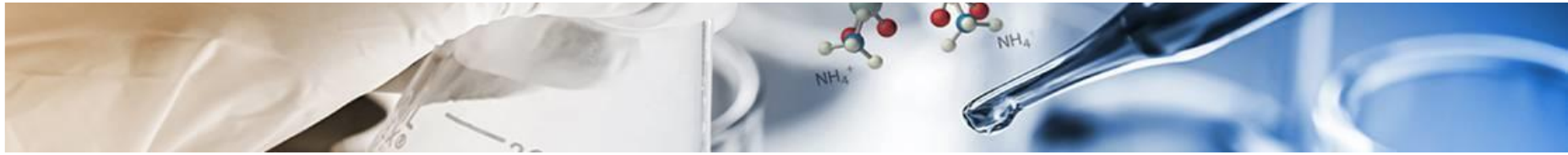


Fate of cholesterol and fat in diet:

- *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*

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 notoriously associated with heart disease contains 21% fat.

FACT D: ***Pollutants trapped in a thick blood-flow, triggers inflammation.***



Pollutants are oxidative, inflammatory: create gaps

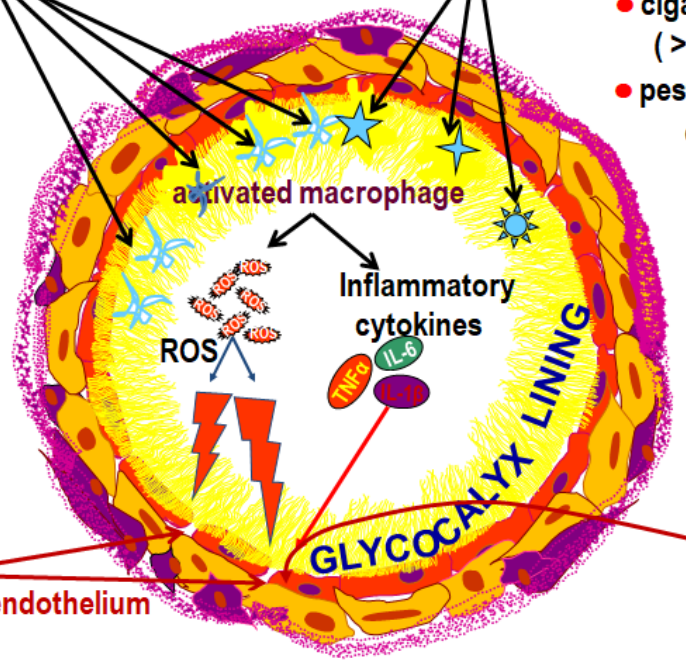
Whirlpool pockets accumulate pollutants, debris

Biological pollutant

- Dead human cells *shed ~ 2M/sec*
- Gut microbes *Chlamydia sp., H. pylori, Enterobacter sp., Cytomegalovirus,*
- Dental microbes *Porphyromonas gingivalis, Prevotella sp., Tannerella sp., Aggregatibacter sp.*

Chemical pollutant

- cigarette smoke *(> 4,700 compounds)*
- pesticides, xenobiotics *(drugs, PCB, dioxins, aromatic hydrocarbons, chlorinated wastes, heavy metals)*



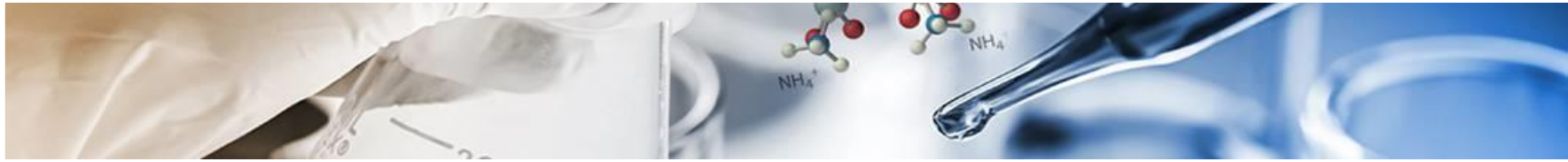
Reactive oxygen species (ROS) and cytokines trigger 'tiny gaps' in endothelium

Plaque *(underneath endothelium)*

- debris infiltration *(plaque)* = ischemia (stenosis), myocardial infarct (heart attack), CHD,
- electrolyte leakage = hypertension, atrial fibrillation, congestive heart failure (CHF)

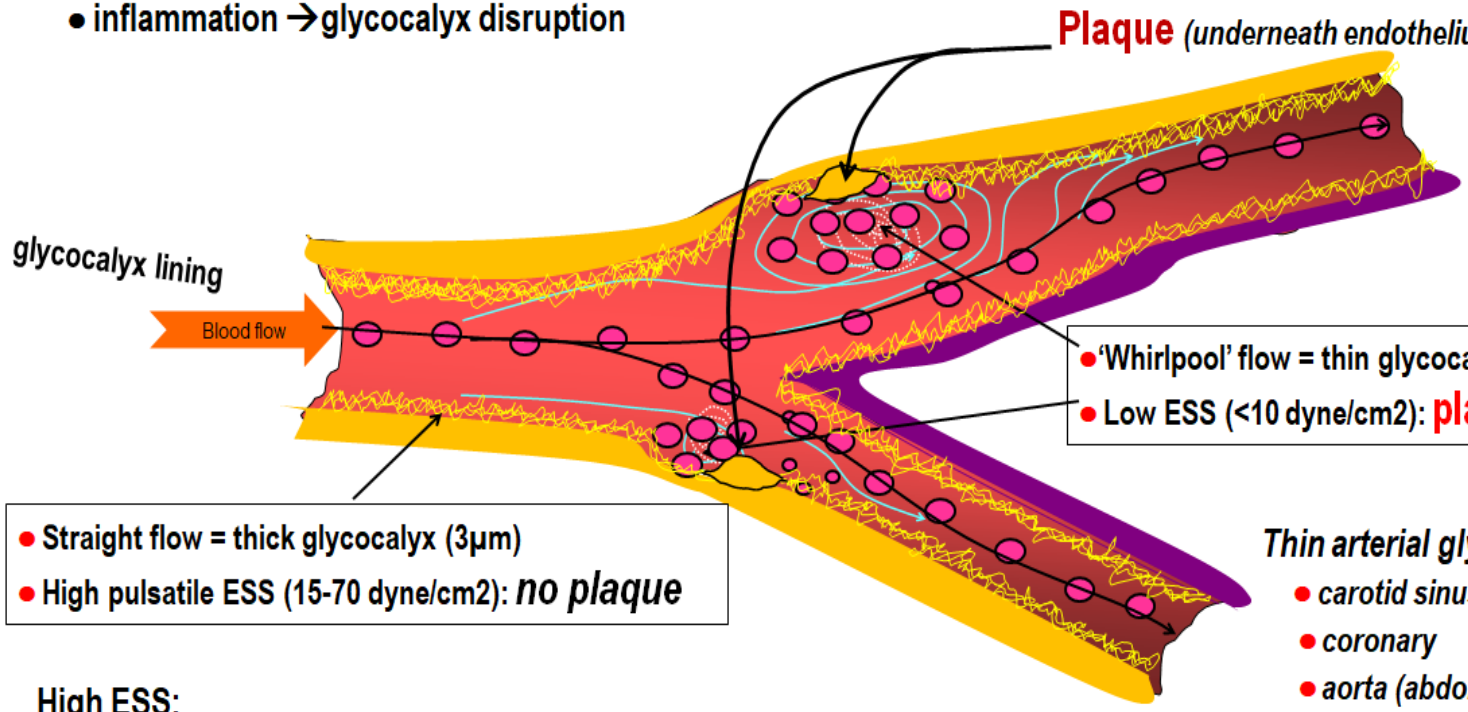
Biological and chemical pollutants in the arterial bends triggers inflammation and tiny endothelial gaps creating electrolyte leakage (hypertension) and debris infiltration (plaque). Cholesterol packaged in lipoproteins (made of fatty acids and prone to oxidation) fill the gaps, preventing osmotic imbalance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

FACT E: ***Arterial bends and bifurcations are sites of stagnant blood flow and plaques.***



'Whirlpool' pockets at forks, bends: plaque sites

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

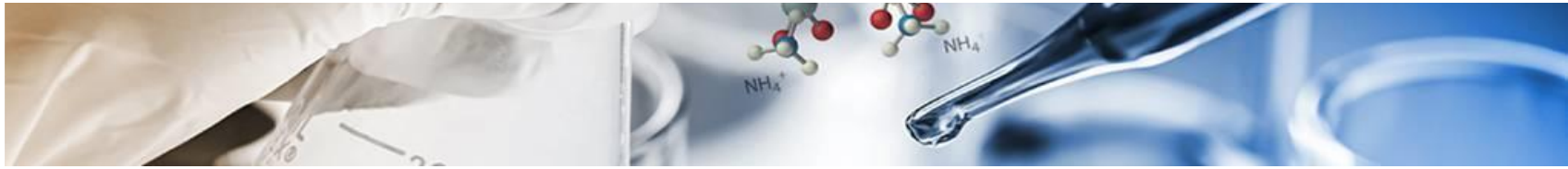


High ESS:
- **high nitric oxide (NO),** ***vasodilation, anti-inflammatory***
- **high antioxidants;** ***suppress reactive oxygen species (ROS)***

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

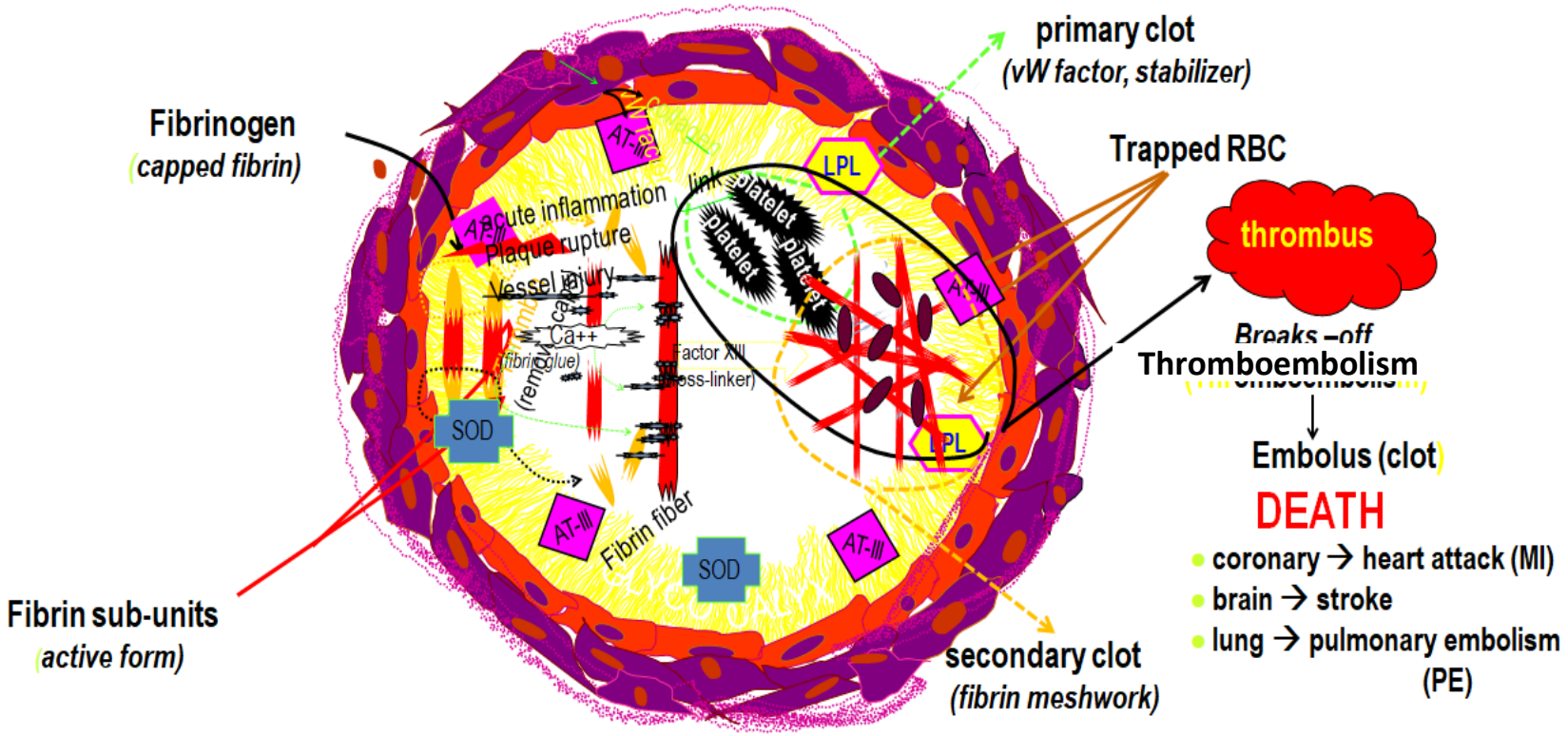
A high fat diet, sugars, pollutants and sedentary lifestyle all contribute to viscous blood and blood flow naturally slows at arterial forks and bends, creating a "whirlpool pocket." Stagnant blood concentrates debris, mobilizing macrophoges (foam cells) to engulf and remove debris.

FACT F: *Acute inflammation triggers plaque rupture leading to fatal clot (thromboembolism)*



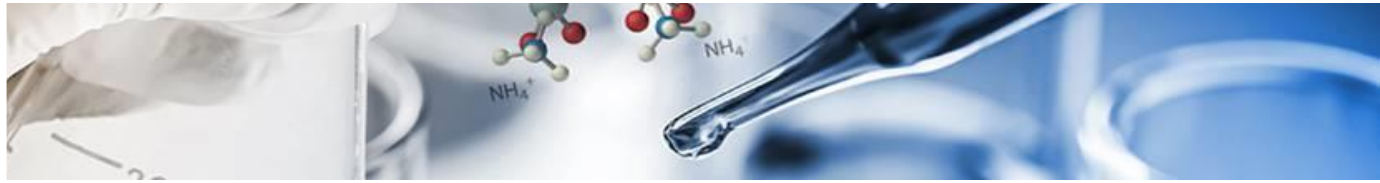
Thromboembolism, fatal process in CVD

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



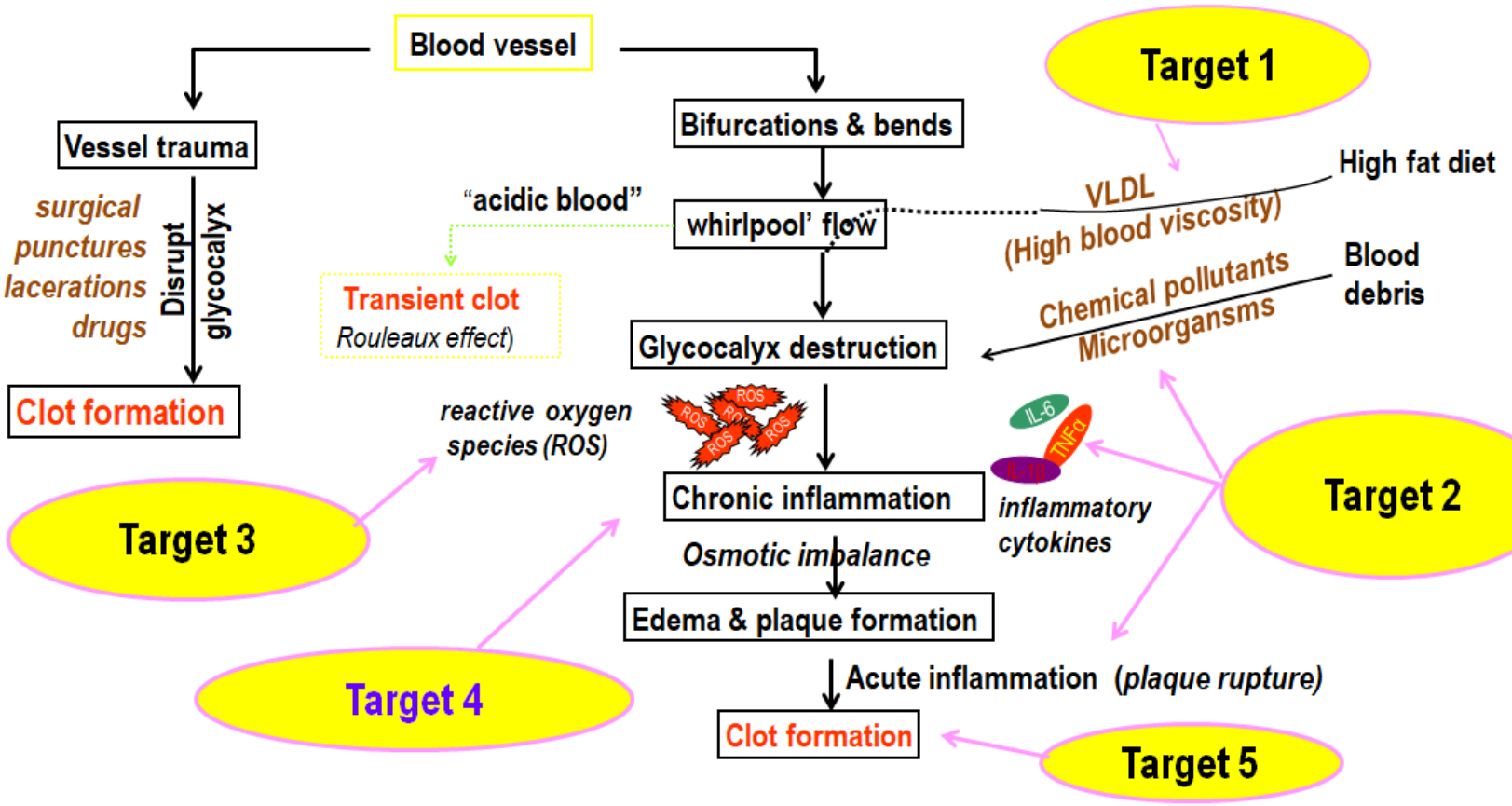
Clot formation starts with a disrupted glycocalyx (primary clot) and progresses into a secondary clot (embolus). This is the fatal component of CVD.

PROOF 1: *Identified multiple biochemical sites as 'druggable targets.'*



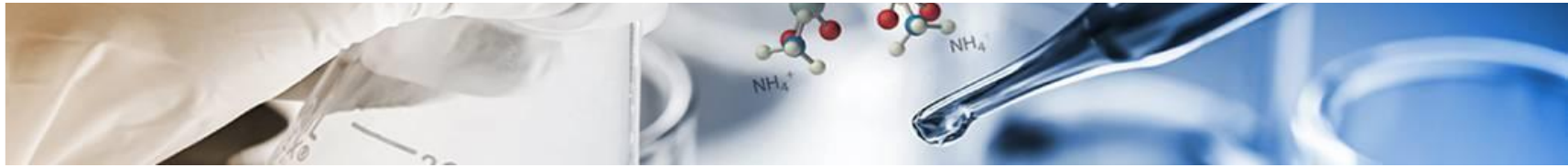
Quest for curative CVD drug

Piecing together a thromboembolic cascade and possible drug targets



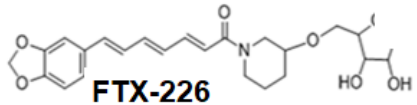
Dr. Tunac carefully studied the thromboembolic cascade and identified 'druggable' targets.

PROOF 2: ***Rationally synthesized 9 drugs for the identified targets.***



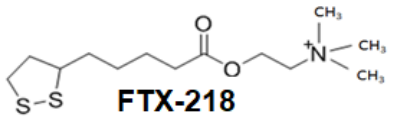
Designed & synthesized drugs for specific targets

Target 1 (VLDL)

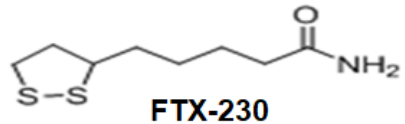


FTX-226
trans-trans-trans piperine-ribose

Target 2 (cytokines)

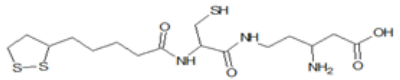


FTX-218
lipoate choline

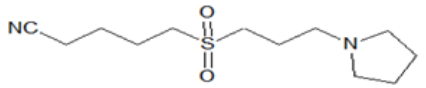


FTX-230
ammonium lipoate

Target 3 (ROS)

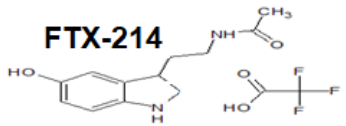


FTX-219
lipoate-cysteine-glutamate

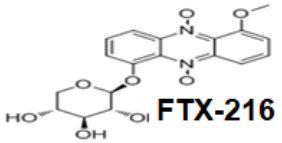


FTX-224
di-oxide isothiocyanate-pyrrole

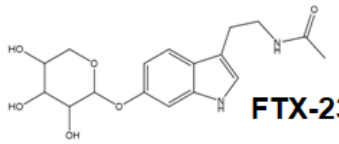
Target 4 (gycocalyx)



FTX-214
indole-acetamide

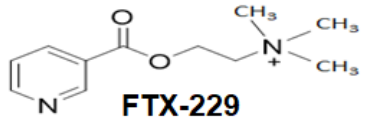


FTX-216
myxin-1β xyloside



FTX-231
Melatonin-6, β-xyloside

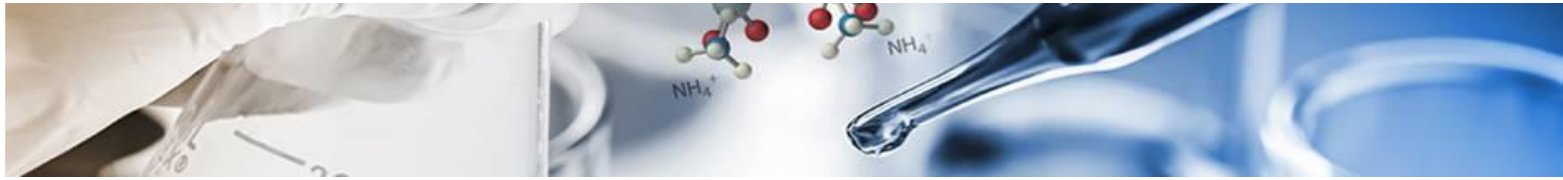
Target 5 (thrombin)



FTX-229
nicotinyl choline

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

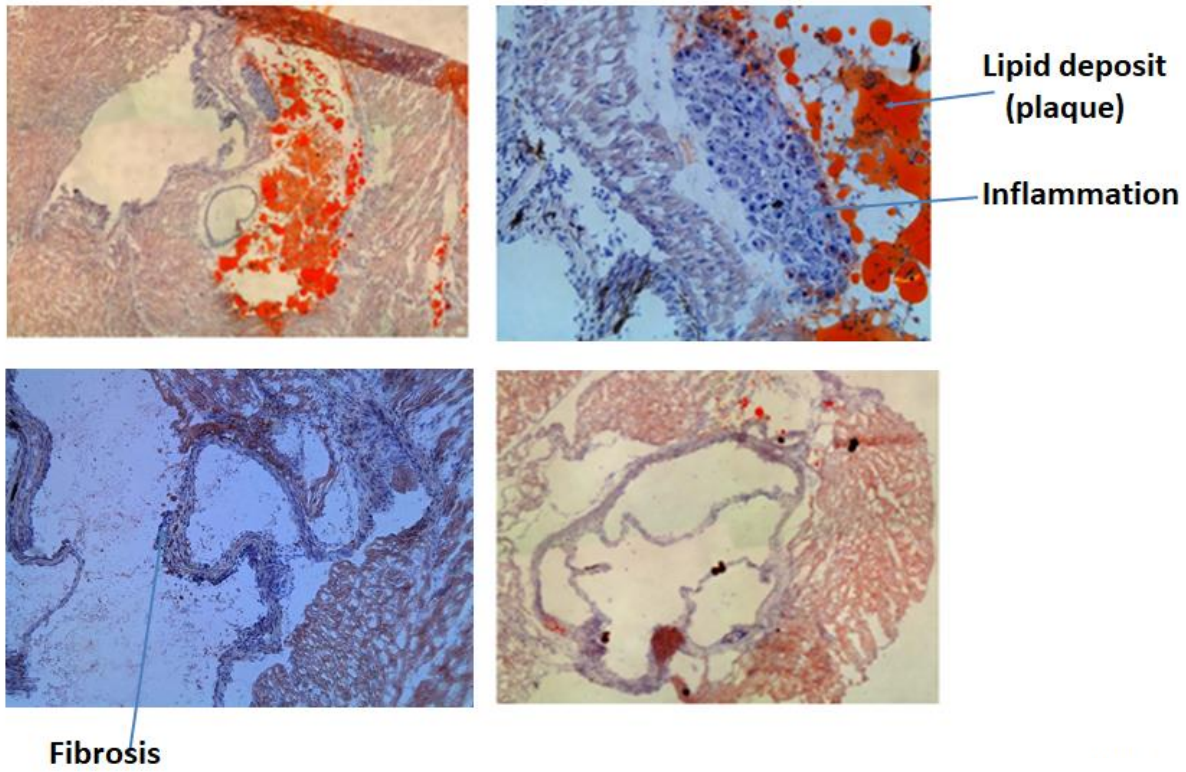
PROOF 3: ***Developed a natural mouse model that produces arterial plaques when subjected to CVD risk factors.***



Created an animal model for atherosclerosis

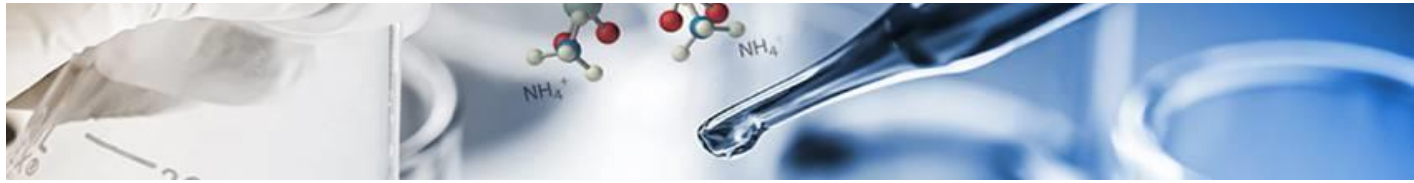
The Tunac Arterial Plaque ™(TAP) model

- The TAP mouse model produced plaques by treatment with agents that disrupt blood flow:
  1. *High fat diet , to create low ESS*
  2. *Polychlorinated biphenyl (PCB), an oxidative agent*
  3. *P. gingivalis, inflammatory infectious agent*
- 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



Recognizing that cholesterol is not a cause, and that the ApoE model is therefore not useful in the study of atherosclerosis, Dr. Tunac developed the Tunac Aterial Plaque (TAP) mouse™ model. The first to create arterial plaques in a natural mouse.

PROOF 4: *Multifactorial disease requires a multi-compound drug; The triple combo K (Embotricin™) was found most effective.*



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

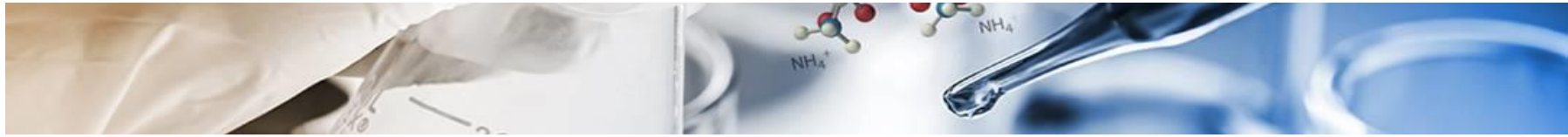
Curative/preventive

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

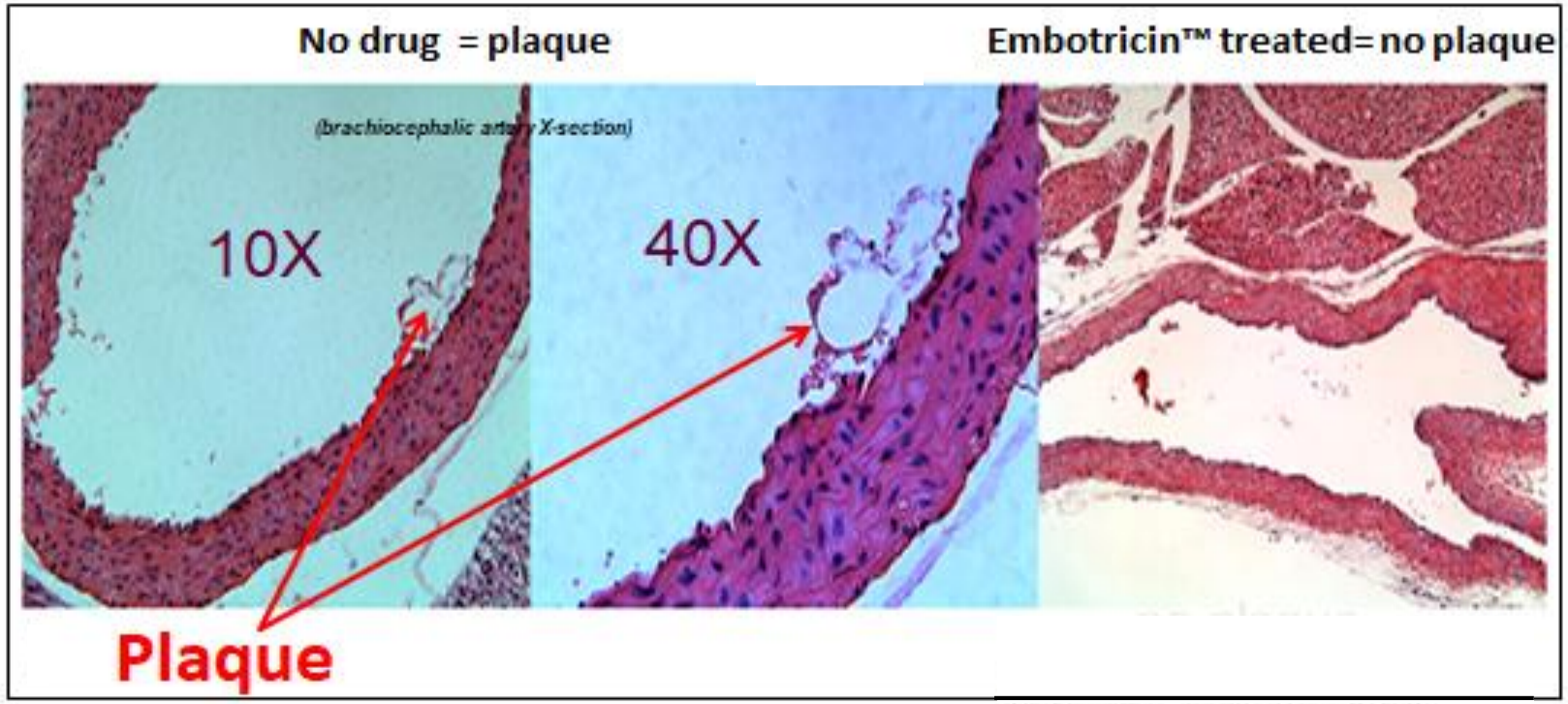
Anti-embolic™ *– compound that prevents formation of emboli (clots) involving plaque reduction and/or restoration of disrupted endothelial glycocalyx*

This process led to an abbreviated factorial 3-drug combo design. Note, individual drugs showed activity but one combo proved curative and preventive of arterial plaques.

PROOF 5: ***Embotricin™ proved to prevent and reverse plaque formation.***

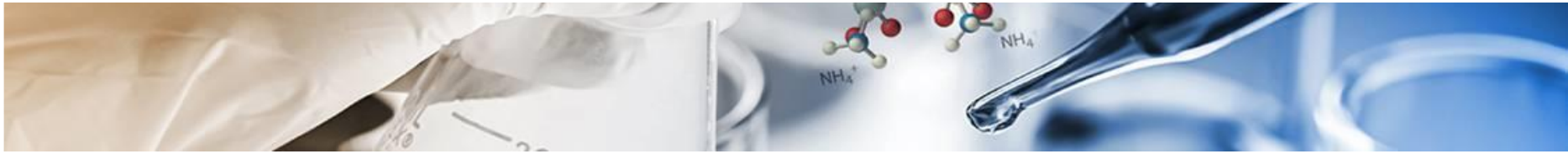


- Embotricin™ was administered before or after the mouse was made atherosclerotic.
- Embotricin™ added to an atherosclerotic mouse reversed plaque formation: curative
- Embotricin™ administered before mouse was made atherogenic prevented plaque formation



This is a micrograph of a mouse brachiocephalic artery showing plaque in a non-treated TAP™ mouse.

PROOF 6: *Embotricin™ also restored (healed) the disrupted glycocalyx.*



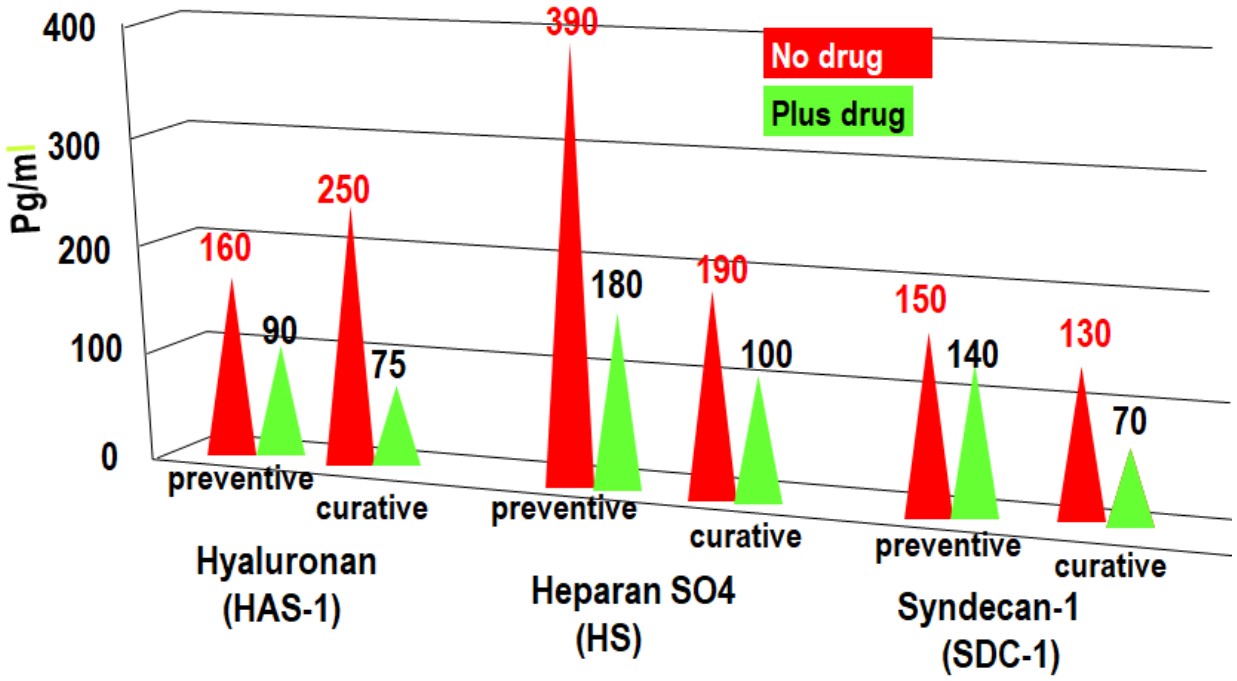
Embotricin™ restored glycocalyx

Preclinical data:

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

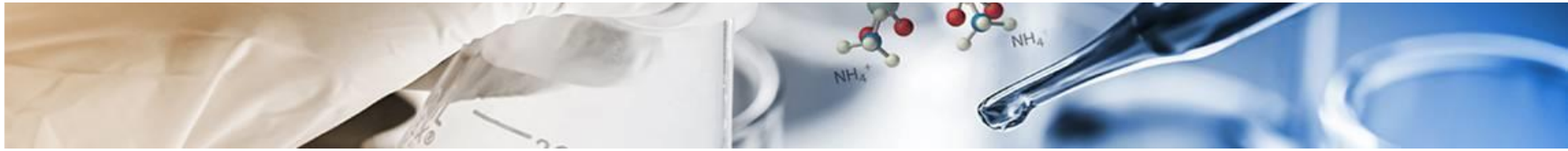
Corroborating clinical data:

- *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™ prevented and restored shedding of glycosaminoglycans, which corroborates published clinical data.

PROOF 7: *Embotricin™ prevented clotting (embolism).*



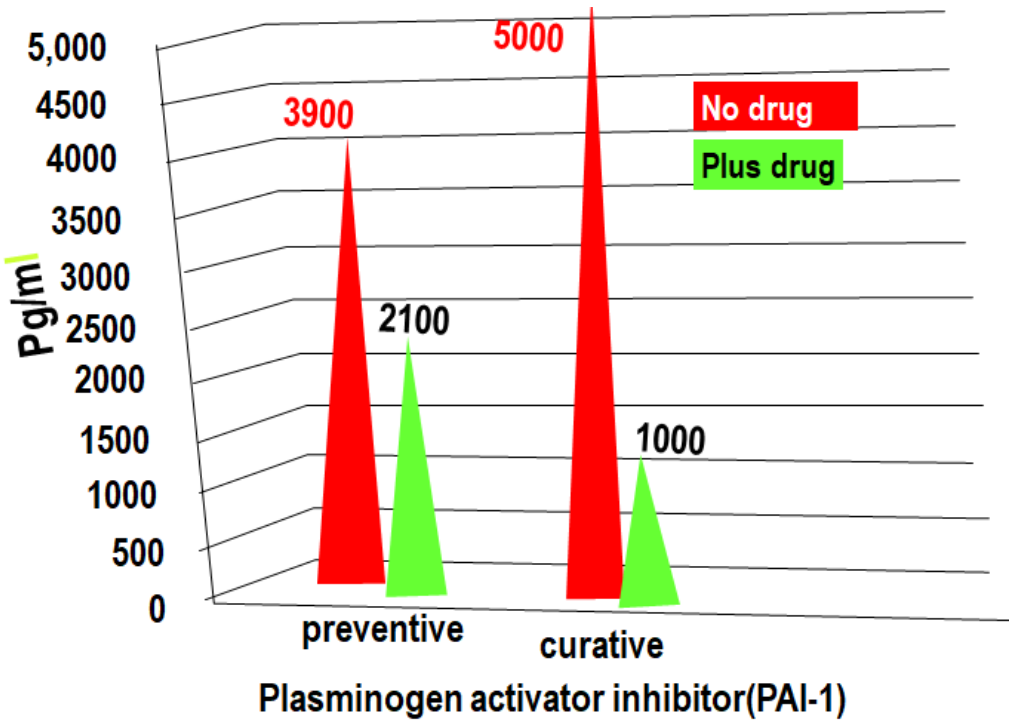
Embotricin™ reduced PAI-1 & embolism

Preclinical data:

- *Embotricin™* red

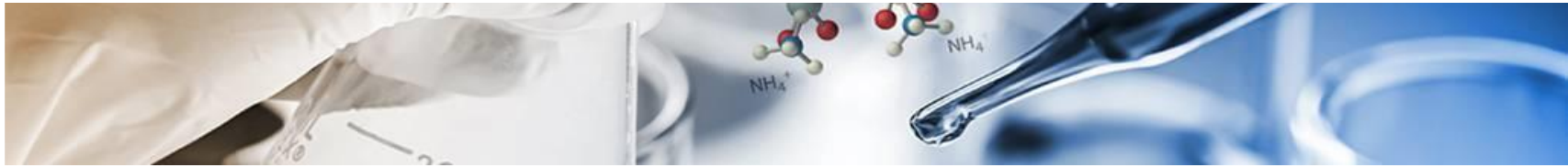
Corroborating clinical data:

- *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

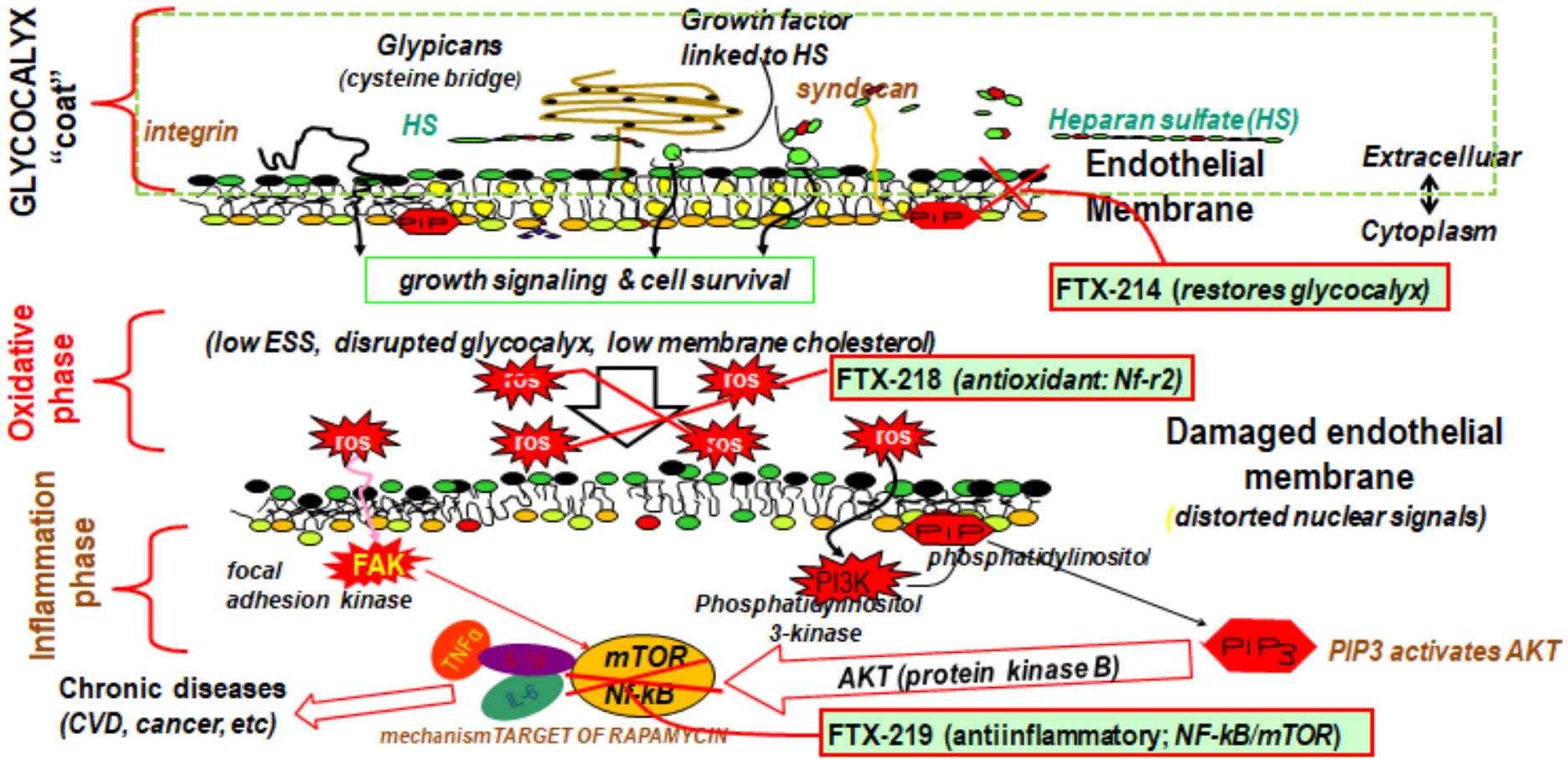


Embotricin™ proved curative and preventive of clot (embolus) formation as evidence by the marker plasminogen activator inhibitor-1 (PAI-1) in early mouse studies. Confirmatory MRI/Histophathology to be published Q1. 2020.

PROOF 8: *Embotricin™ effectively treats CVD due to its multifactorial mode of action.*



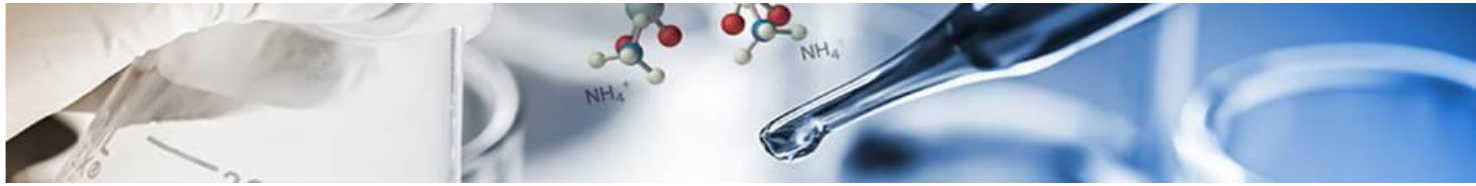
Embotricin™ proposed action sites



Embotricin™ , a triple combo drug consisting of FTX-214 , FTX-218, and FTX-219 act synergistically to restore the health of the vascular system: FTX-214 restores glycocalyx, FTX218 an antioxidant and FTX-219 an anti-inflammatory.

DEVELOPMENT OF DIAGNOSTIC BLOOD MARKERS:

Glycocalyx debris or detritus is the foundation for our novel "fingerprint" diagnostic technology targeting chronic disease.

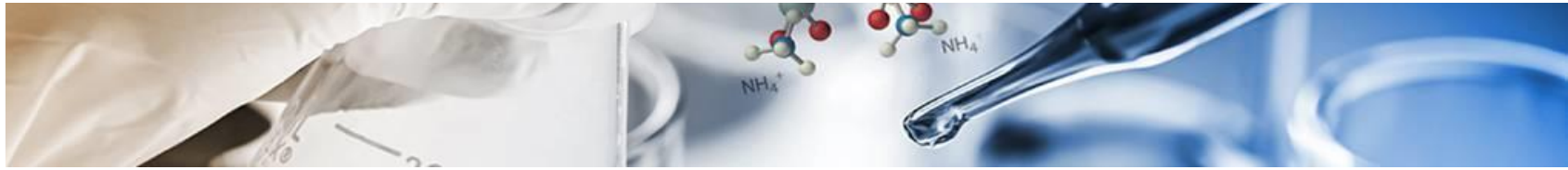


Glycocalyx Detritus Fingerprint - 2012: Dr. J. B. Tunac (US) introduced glycocalyx detritus *(rubbed or worn off glycocalyx debris)* as component blood biomarkers for a biological fingerprinting system. Currently, there is no equivalent fingerprint system developed for disease diagnosis. In this regard, the glycocalyx detritus pattern present an equivalent to the physical patterns found on finger tips as a basis for the classic fingerprint or the nucleotide microsatellites bands that describe a DNA fingerprint. The classic fingerprint and DNA fingerprint do not diagnose diseases and are used only to identify individuals. On the other hand, the Glycocalyx Detritus Fingerprint™ technology will be the first of its kind to identify, predict diagnose and treat chronic disease, a discovery that could revolutionize or mark a new era in healthcare.

In Summary:

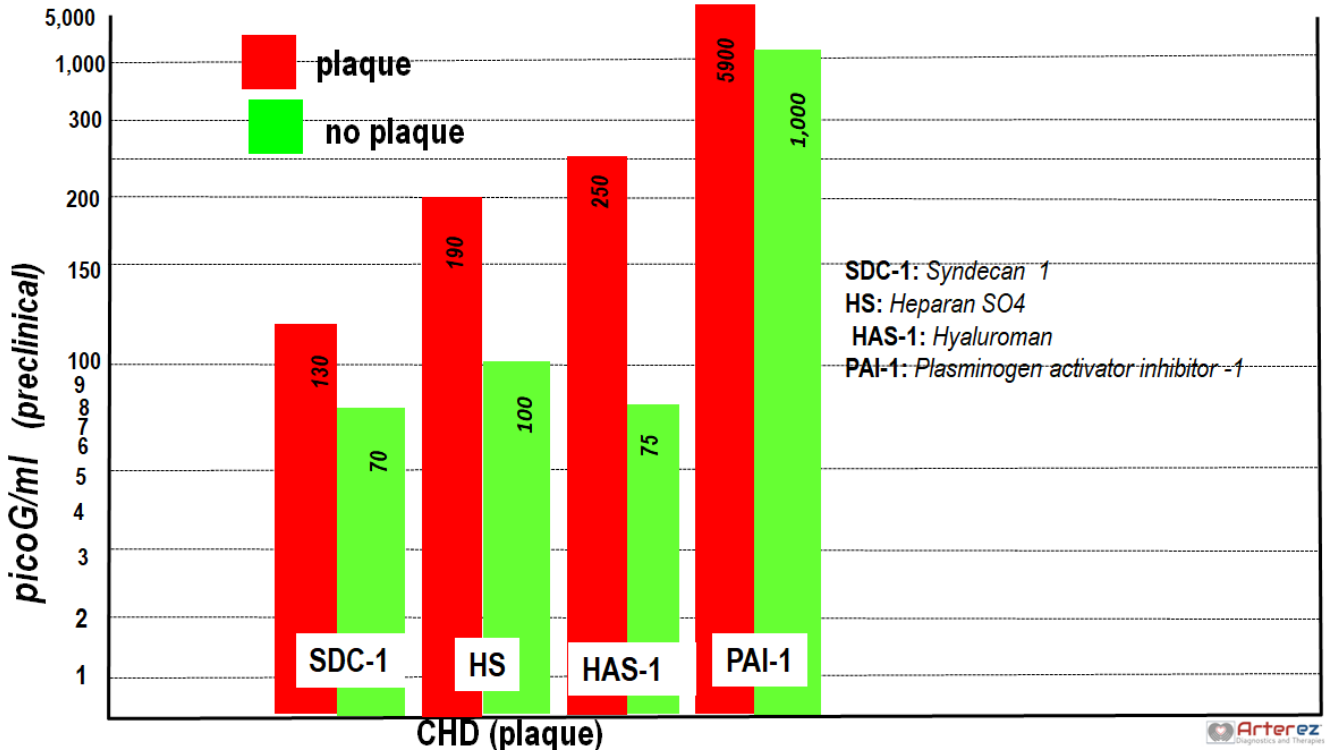
The use of our Glycocalyx Detritus Fingerprint™ technology, as opposed to a single biomarker, we believe will both increase the frequency and accuracy of early disease identification and diagnosis as well as enable disease identification, classification and staging, thus serving as a guide for improved therapies. Dr. Tunac has long noted that the historical development of antibiotics targeting microorganisms led to a cure of infectious disease. His hypothesis began with the idea that an equivalent approach would be to target the endothelial glycocalyx to cure CVD by an anti-embolic™ mechanism. We believe this may well represent a new paradigm, a true breakthrough in medical science, and may well become 'the' benchmark for predicting, preventing and treating chronic disease.

PROOF 1: ***A 4-panel diagnostic panel correlated with plaque formation.***



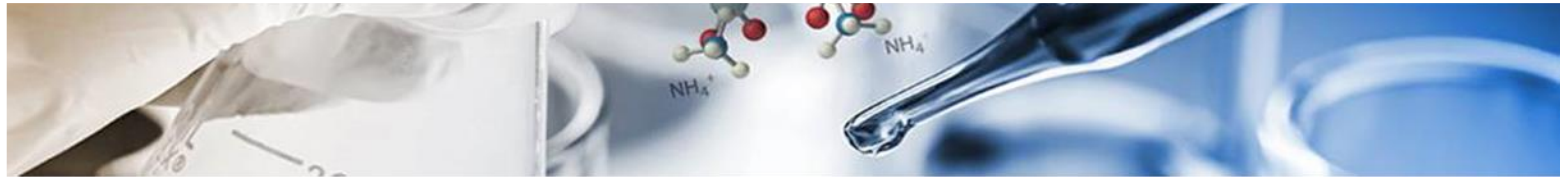
Proof-of-Principle - 4-panel glycocalyx detritus fingerprint (Glycocardia^{CHD}) as a companion diagnostic for plaque formation.

Fingerprint of a 4-panel glycocalyx detritus showing elevated blood levels in the presence of plaque and a corresponding decrease without plaque.

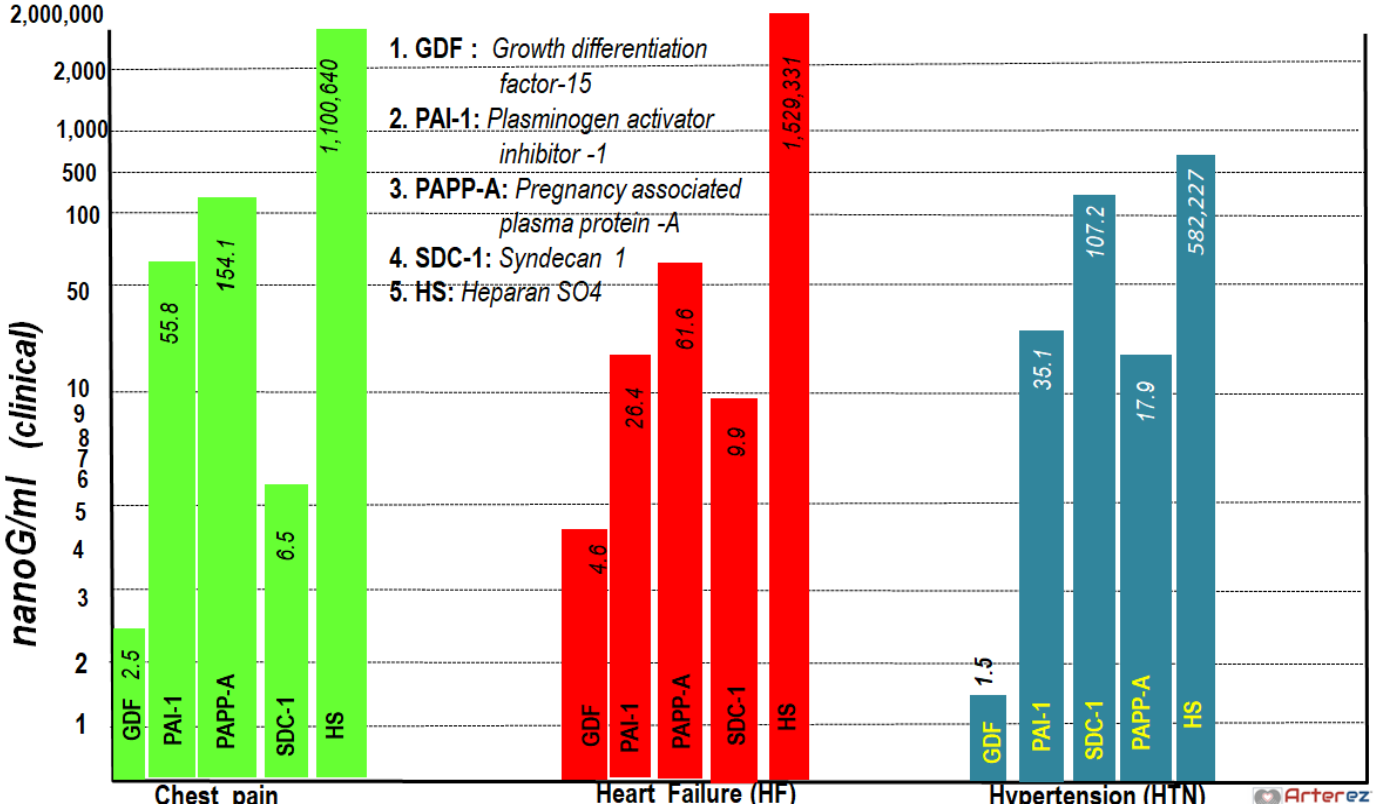


Coronary heart disease (CHD) is a member of the cardiovascular family (CVD) and the leading CVD killer. The characteristic feature of CHD is plaque formation resulting in atherosclerosis or hardening of the arteries. Plaque formation is triggered by glycocalyx disruption and the shedding of glycocalyx detritus. In this regard 4 glycocalyx detritus (Glycocardia^{CHD}) 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). A mouse (TAP™ model) was used to model plaque formation. Indeed the blood levels of the 4 detritus correlated with plaque formation.

PROOF 2: ***Our 5-marker panel ELISA proved effective in identifying patients diagnosed with different CVD diseases.***

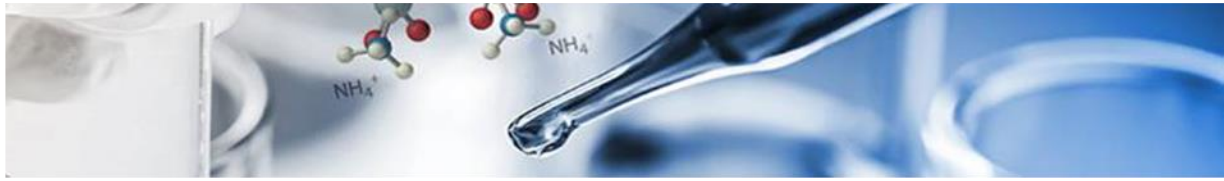


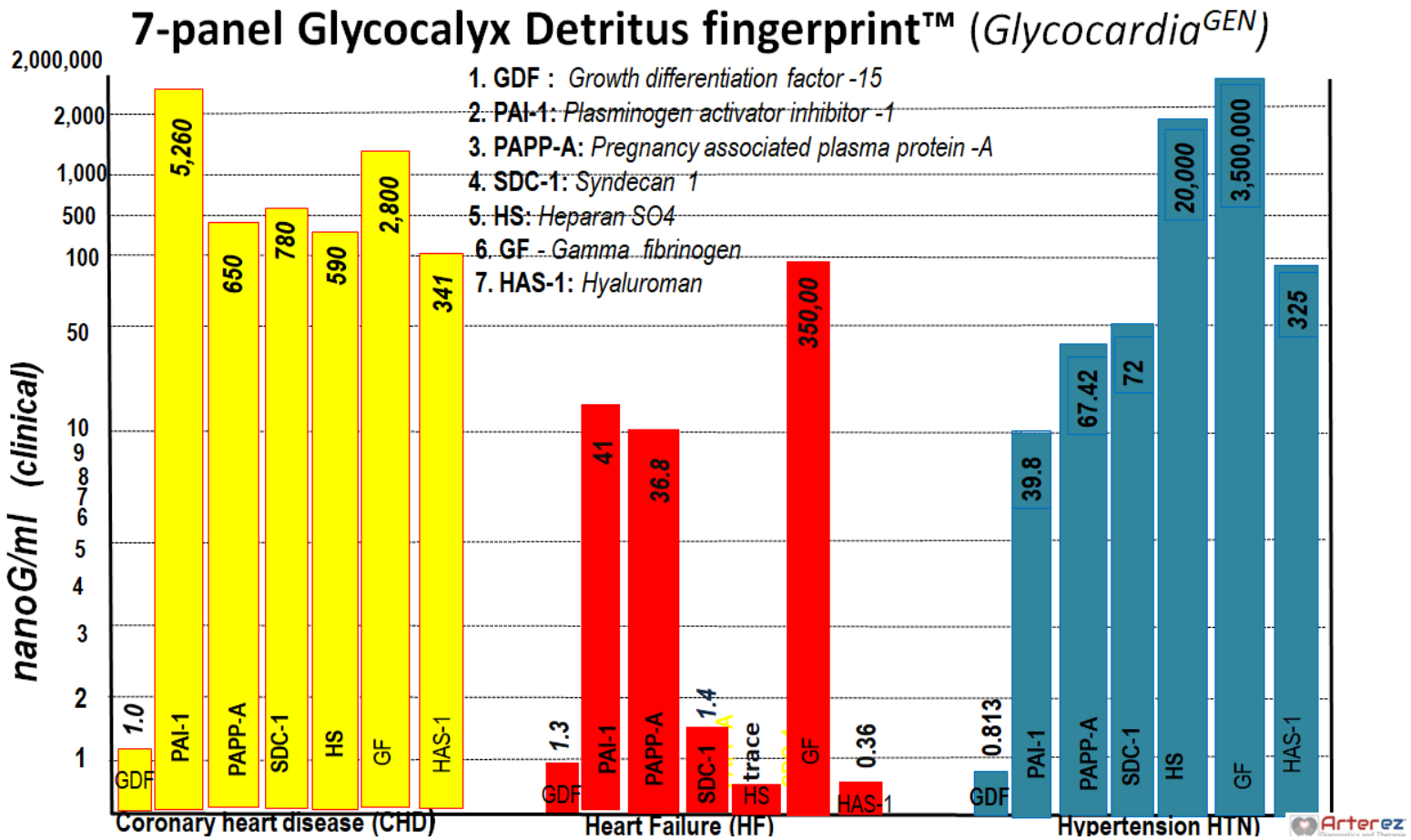
5-panel Glycocalyx Detritus fingerprint™ (*Glycocardia^{HF}*): clinical



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.

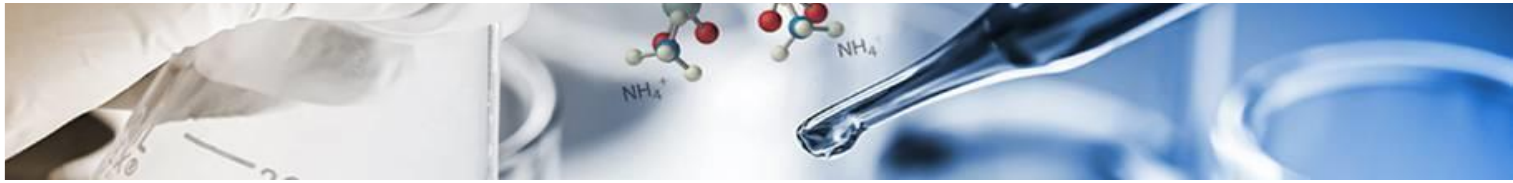
PROOF 3: ***Expansion to a 7-marker panel incorporating published clinical data proved a fingerprint for chronic disease can in fact be identified and thus serve to predict, diagnose and be used as a tool to treat chronic disease.***



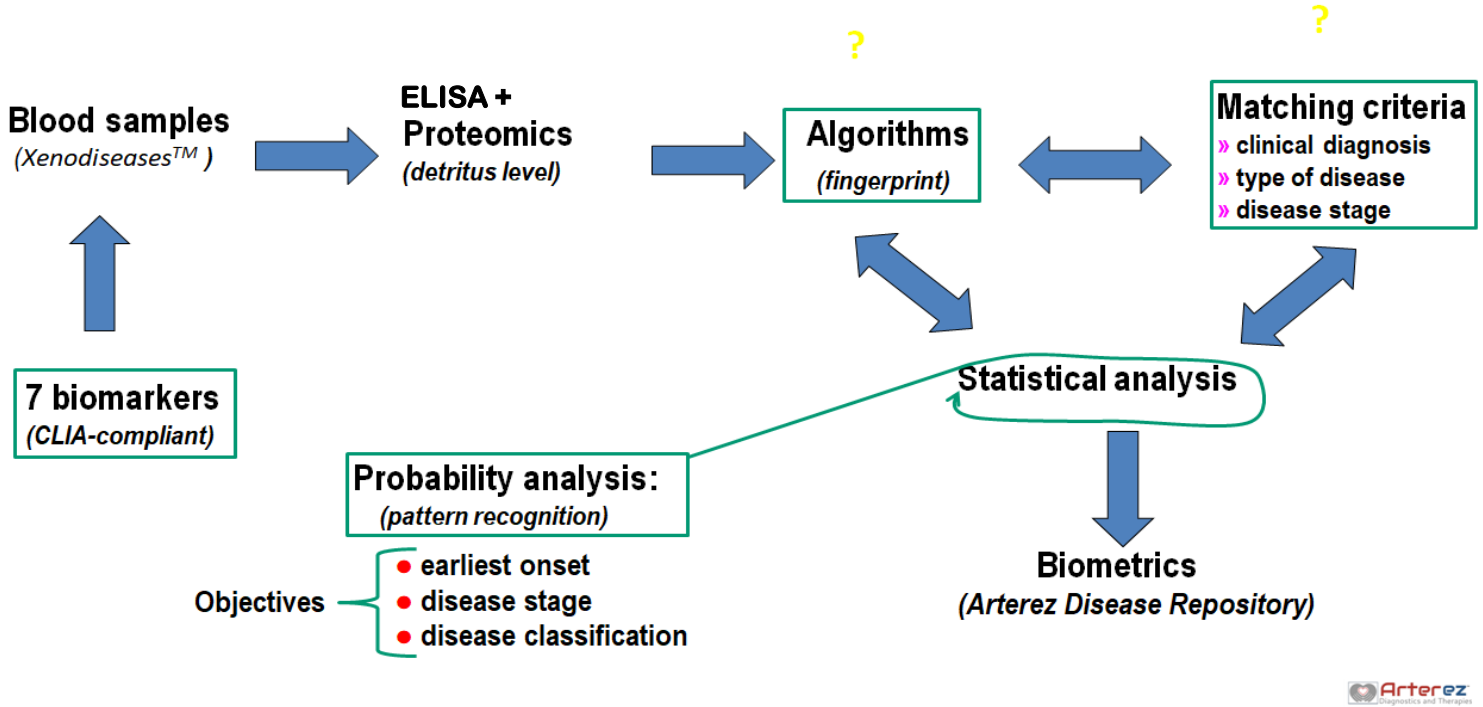


Blood levels of 7 detritus components were obtained from published literature of patients with coronary heart disease (CHD), heart failure (HF), and hypertension and a virtual fingerprint was constructed. Each disease showed a unique fingerprint, which confirms the hypothesis of the Glycocalyx Detritus Fingerprint™ as a unique tool for identifying diseases currently in development.

DEVELOPMENT OF A CENTRAL REPOSITORY OR PROPRIETARY DATABANK FOR CHRONIC DISEASE.



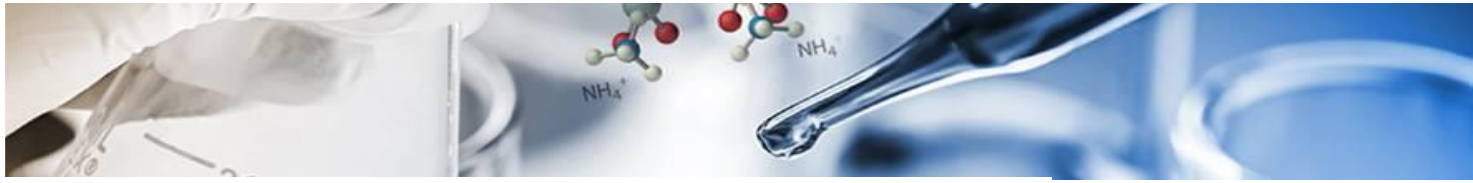
Arterez Disease Repository data base



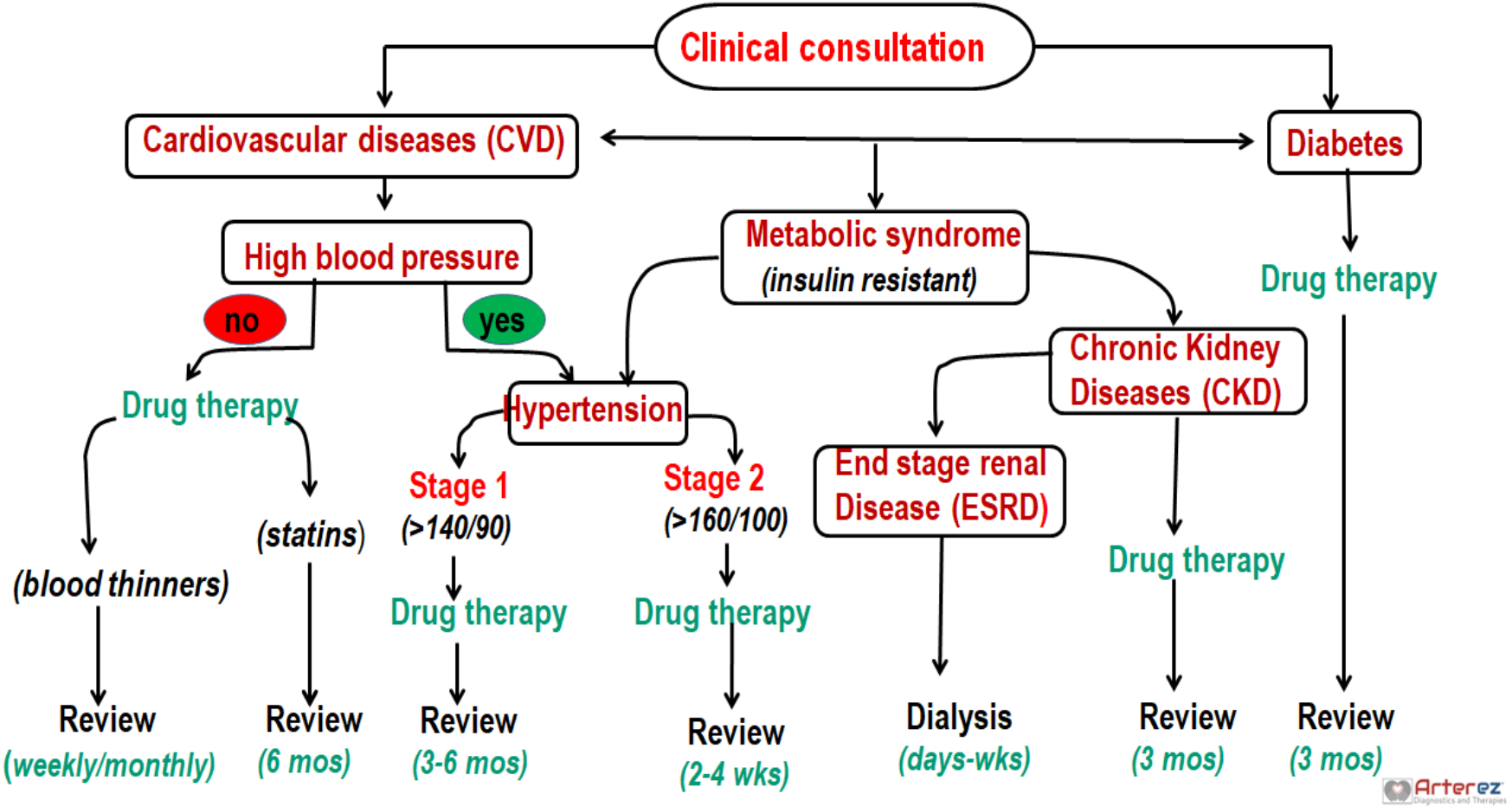
The Glycocalyx Detritus Fingerprint™ proved to be effective in creating distinct patterns or fingerprints of the different family members of CVD tested. This indicates it will effectively identify, predict and ultimately be used to diagnose diseases. As previously stated, we believe the Glycocalyx Detritus Fingerprint™ technology and corresponding proprietary algorithms will be the first analytical tool to diagnose chronic disease, beginning with the CVD family, representing a new paradigm for diagnosis that will also assist in targeted treatment.

A fingerprint is useful only as a matching tool; on its own it is useless. Arterez is building a robust Glycocalyx Detritus Fingerprint™ database and series of algorithms for individual diseases (white paper to be published Q1, 2020).

GLYCOCALYX DETRITUS FINGERPRINT AS A GUIDE FOR TREATMENT AND SCHEDULING OF THERAPIES.



Glycocalyx Detritus fingerprint™ treatment guide



The Glycocalyx Detritus Fingerprint™ can be used twofold: 1) as a companion diagnostic for custom therapies (e.g., Embotricin™), or 2) 'stand-alone' predictive diagnostic to monitor or evaluate the traditional symptom-targeted therapies.

BUSINESS MODEL 1

Arterez wholly owns two complimentary product companies, GlycoTrx and ComboRx

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

BUSINESS MODEL 2

FUTURE DEVELOPMENTS

Chronic Disease
Diagnostic Tools

GlycoDiabx™
Diabetes Diagnostic

GlycoArthx™
Arthritis Diagnostic

Chronic Disease
Therapies

Metabotricin™
Diabetes Oral Therapy

Arthritricin™
Arthritis Oral Therapy

INTELLECTUAL PROPERTIES

Docket #: ARTZP003PUS

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

PCT/US2016/015015

Biomarkers of Vascular Disease

US 9,867,842 B2

Methods and Compositions for Reversing Disruption of the Glycocalyx, Inflammation and Oxidative Damage.

International PCT IP in process (to be submitted by November, 2020).

Trademark applications in process:

- Arterez
- GlycoCardia
- Embotricin
- Tunac Arterial Plaque Animal Model (TAP)
- Anti-Embolics
- Detritus Fingerprinting Technology

CLINICAL ROADMAP

Q1 2020- Q4 2022

1 . FDA/IRB compliant toxicity study for 3-combo drug *Embotricin™* supported by our companion diagnostic *GlycoCardia* ^{CVD}

2 . IRB-initiated proof-of-principle clinical evaluation of *Embotricin™ v*s patients with CHD.

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

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

3 . IRB-initiated proof-of-principle clinical evaluation of *Embotricin™* vs patients with hypertension (HTN)

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

4. 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}

SCIENCE SUMMARY

COMBORX, LLC

Embotricin™ (1st 3x combo anti-embolic™ drug)

Targeting multi-factorial root causes of CVD*

- Repair/maintenance of GCX
- Minimizing oxidation
- Reducing inflammation

Curative, preventive of arterial plaques

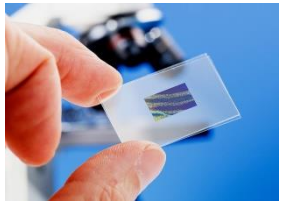
Non-toxic up to 3,000 mg/kg in combination

GLYCOTRX, LLC

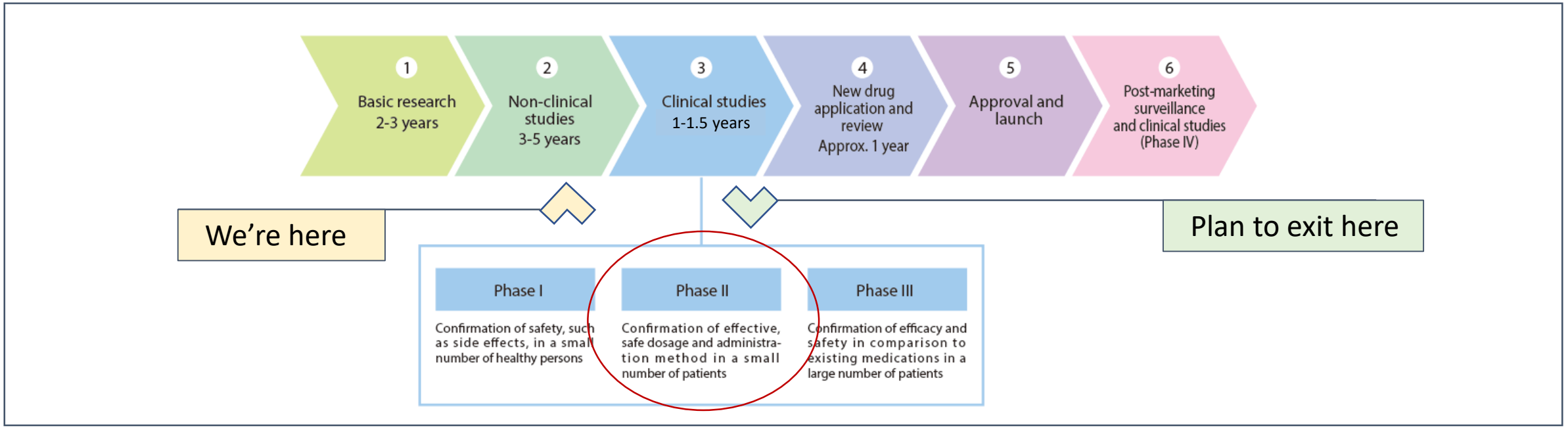
GlycoCardia™ (1st 'jury-panel' for CVD)

Companion and predictive 3-7 marker kits targeting individual CVD diseases –

Future Predictive Diagnostics:



- GlycoCardia HF
- GlycoCardia HTN
- GlycoCardia CHD



BREAKTHROUGH?

Embotricin™, next breakthrough akin to penicillin!

Infectious diseases are curable, targets causative agent

	Infectious diseases	**vs.**	***Cardiovascular diseases***
Death statistics	**162 AD – 1930s** * *162 AD - killed ~40% Chinese soldiers* * *1200 – 1393: 2/3 of Chinese population* * *1346 – 1350: half of Europe's population* * *1520: wiped out Aztecs population* * *1860s: killed Civil War soldiers*		***1230 BC - to date*** * *1230 BC - Pharaoh Merenptah died from CVD* * *Currently # 1 killer globally:* *2008 - 17.3 M /yr (30% of all deaths)* *by 2030 - 23.6 million M deaths/year*
Historical treatment *(symptom-target)*	**soybean curd, wine, myrrh, opium, iodide, mercury, arsenic, sulfa**		***anti-lipidemics*** *(cholesterol-lowering),* ***anti-hypertensives, anti-coagulants*** *(blood thinners)*
Predisposing conditions	**poor hygiene, unsanitary environment**		***sedentary lifestyle, high-fat diet, preservatives, pollution, smoking***
Causative agent *(root cause)*	**1930s: microorganisms** (*pathogenic species*)		***2010s: xenobiotics*** *(endothelial glycocalyx breakdown)*
Medical breakthrough	**1940s: antibiotics** (*penicillin, first curative drug*)		**2020s: *anti-embolic™*** *(anti-clot)* (*Embotricin™, first CVD cure*)

Historical development of antibiotic 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.

THANK YOU FOR
YOUR CONSIDERATION

Arterez™
Diagnostics and Therapies

www.Arterez.com