

## Session 2: Airway, Pathogens & Plaque TRIAD with Gina & Lora



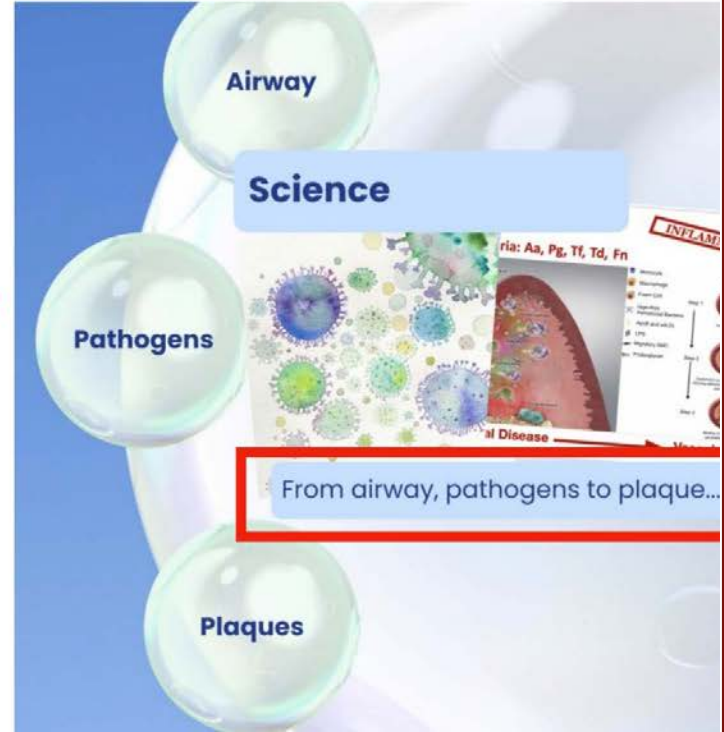
$O_2$



**anaerobic  
oral  
pathogens**

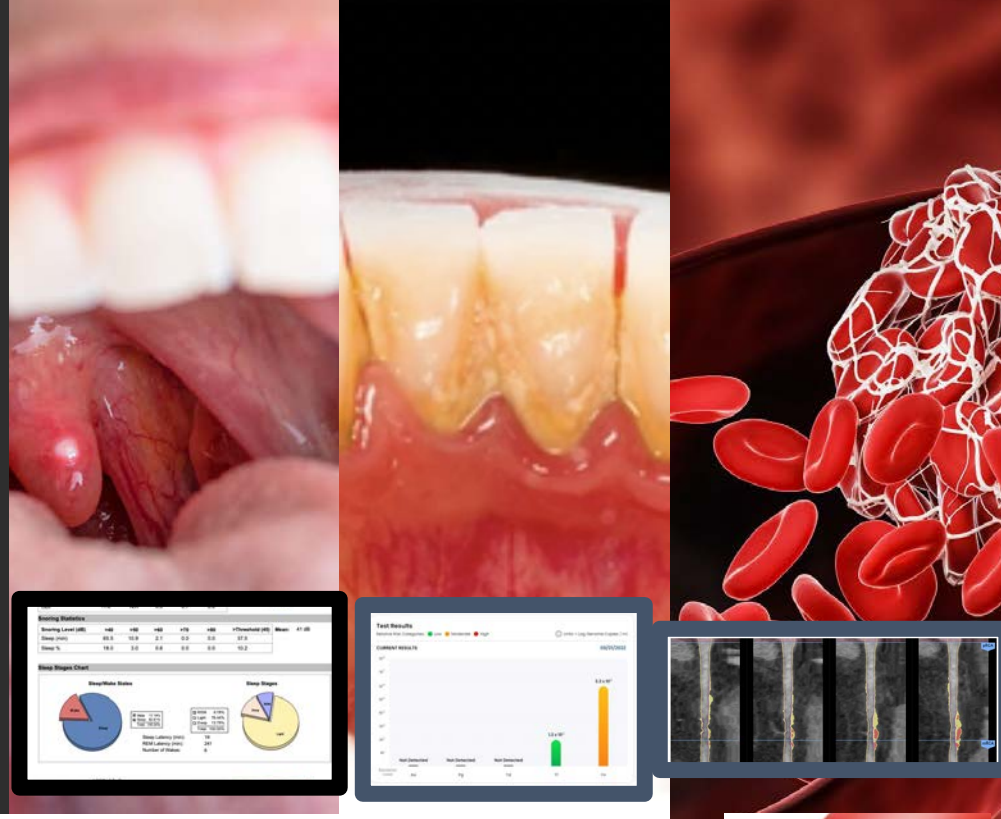


**arterial  
plaque**



# Airway to Pathogens to Plaque

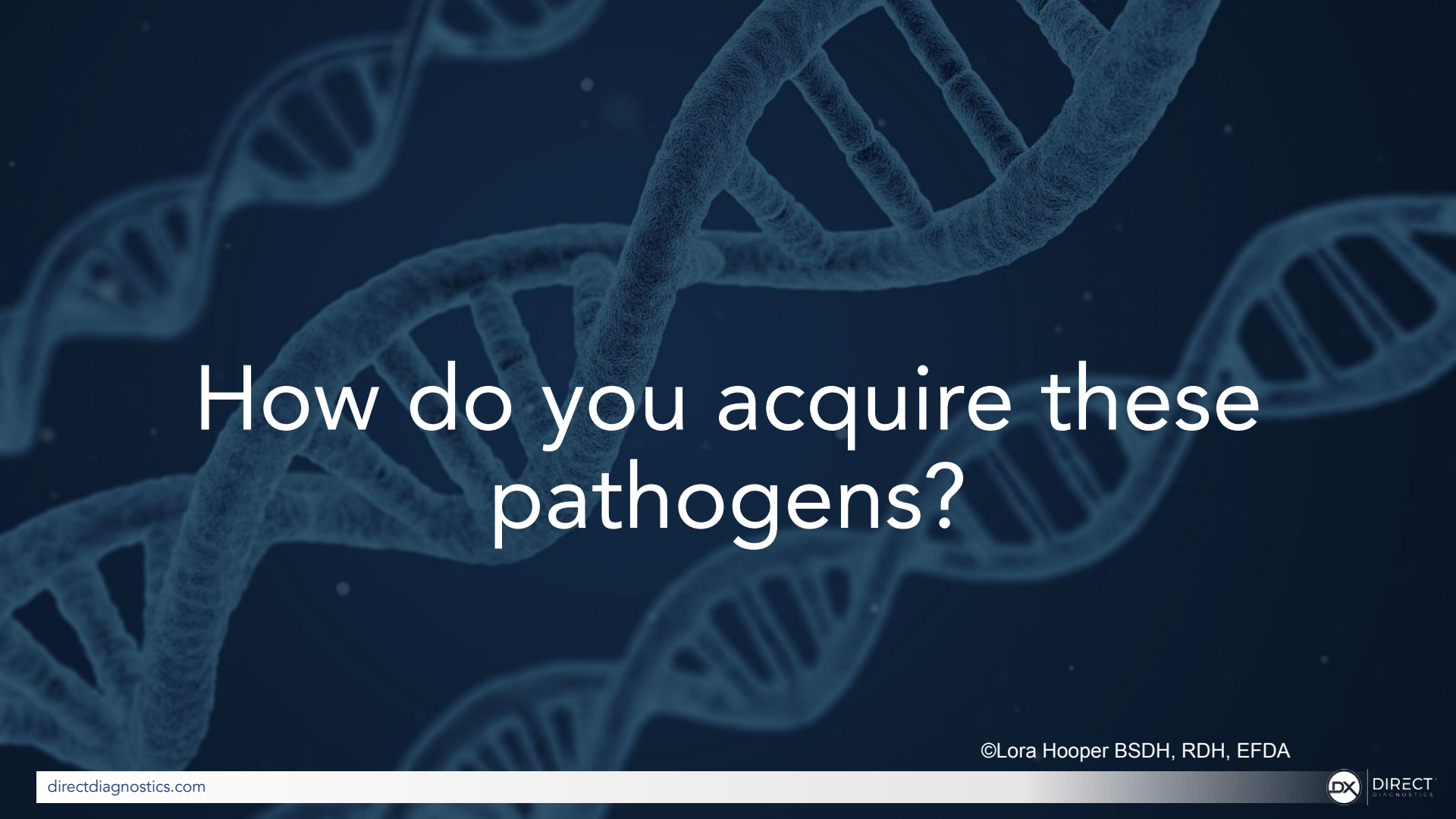
Constant desaturation, constant microbial challenge,  
constant inflammation



**Ask...what are the root causes**


# Clinical presentation is only part of the story

- What is subclinical?



# How do you acquire these pathogens?

©Lora Hooper BSDH, RDH, EFDA



# Bacterial challenge Airway development





### Is your kid a mouth breather? It might be time for an intervention

The term may induce stickers, but the actual habit can cause—and indicate—a surprising number of medical issues.



Sharing food & drink  
First kiss...continual microbial challenge

Continue to desaturate and bacteria maturation...



The background of the slide is a composite image. On the left, there are several blue, rod-shaped bacteria, likely representing oral pathogens, set against a dark blue background. On the right, there is a close-up of a tooth. The crown of the tooth is white, while the root and the area just below the gum line are covered in a thick, yellowish-brown layer, representing dental decay or plaque. The entire scene is overlaid with a large, dark circular area in the center where the text is located.

# Pathogens

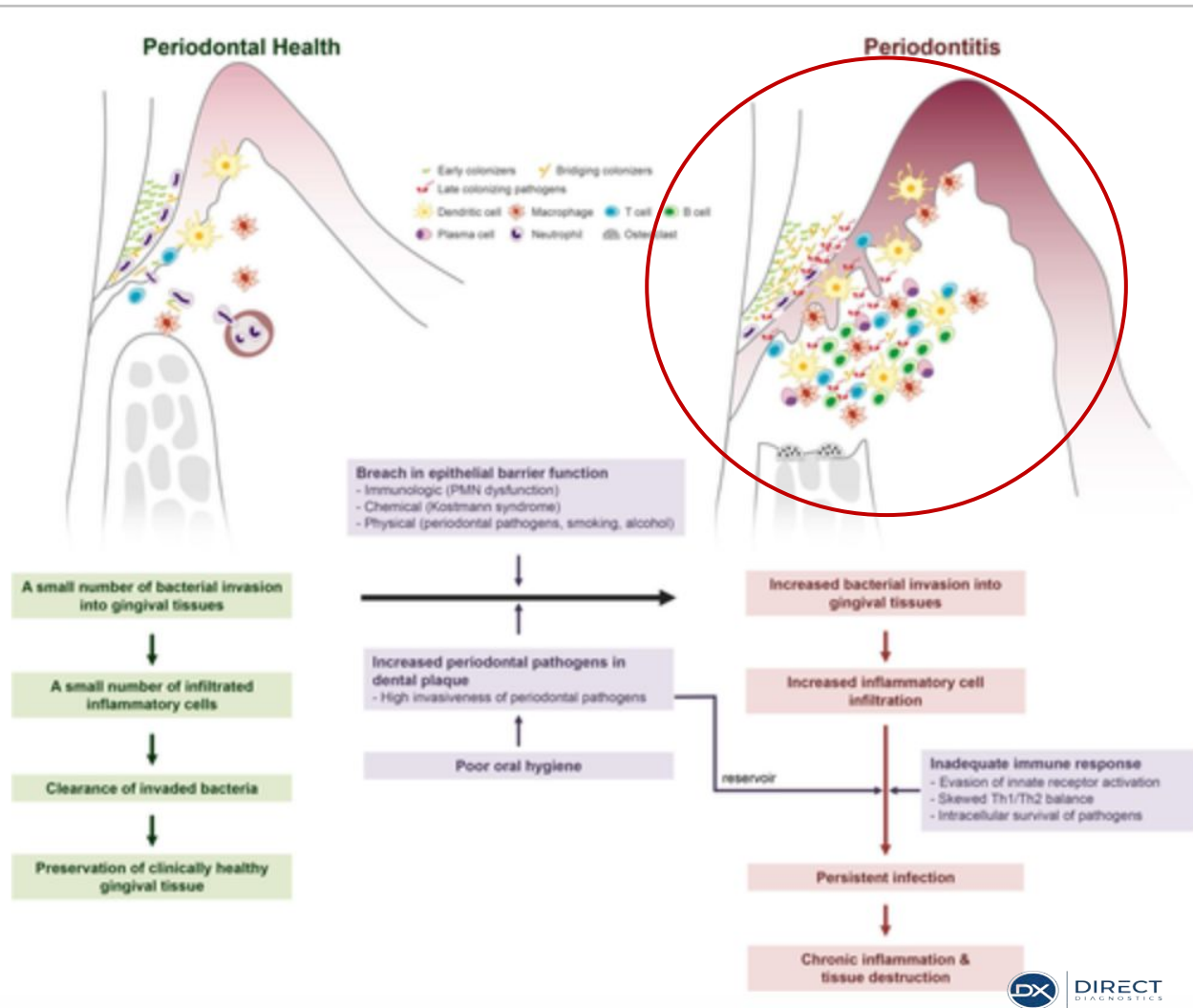
Microbial challenge

"Red zone"



# Health to disease

Subclinical



# Heart

## Bacteria enter

The Bloodstream

Gum Tissue →

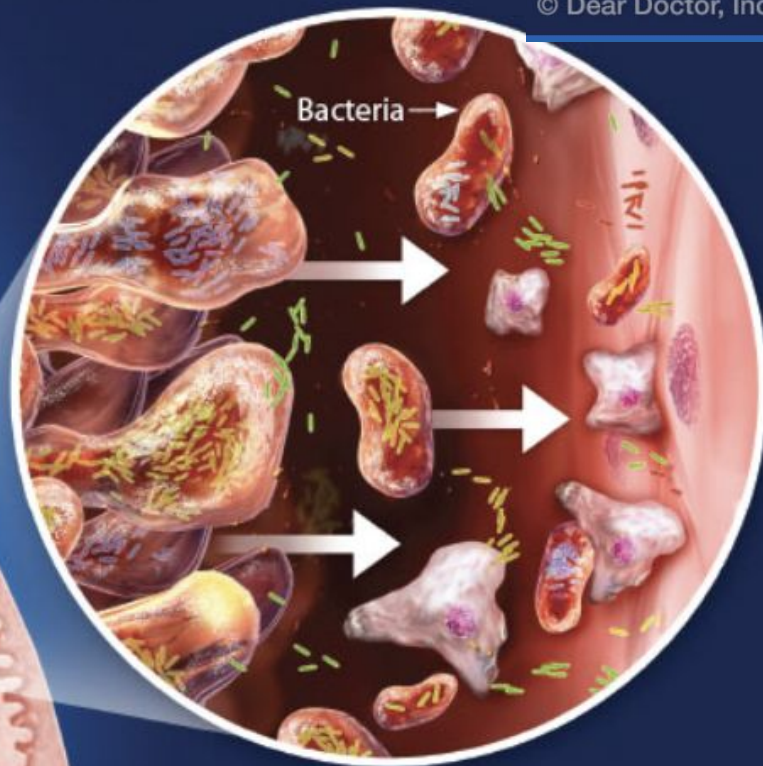
Periodontal  
Ligament

Bone →

Root

Plaque  
(Biofilm)  
and Tartar  
(Calculus)

Inflammation



Harmful bacteria can enter the bloodstream through the gums, causing localized and systemic inflammation.

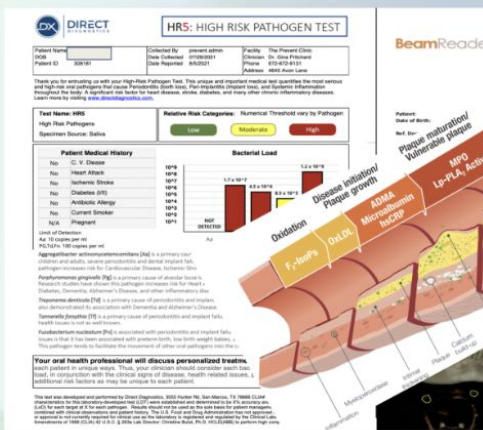
©Lora Hooper BSDH, RDH, EFDA

Lack of  
oxygen

Airway  
Pathogens  
Plaque

Increase  
pathogens

PH  
Acidity



80%

of American adults over 35 have  
some form of Gum Disease.  
- The American Academy for Oral Systemic  
Health (AAOSH)

**HR5™**  
HIGH RISK PATHOGEN TEST

50%

Up to 50% of heart attacks and stroke  
are triggered by oral pathogens  
Source: Circulation (2013)

Oral bacteria have been implicated in the development of  
**Alzheimer's Disease and Dementia**  
- AAOSH

Oral bacteria have been implicated in the development of  
**Adverse Pregnancy Outcomes**  
such as low birth weight babies, early birth, and stillbirth  
- AAOSH

## MEET THE PATHOGENS



**Aa**

Aggregatibacter  
actinomycetemcomitans

Primary cause of **rapid alveolar bone loss** in both children and adults, **severe periodontitis** and **dental implant failure**. Research shows this pathogen increases risk for **Cardiovascular Disease, Ischemic Stroke, Brain Abscesses, and Heart Infections**.



**Pg**

Porphyromonas  
gingivalis

Primary cause of **alveolar bone loss, periodontitis and implant failure** (peri-implantitis). Research shows this pathogen increases risk for **Heart Attack, Ischemic Stroke, Type 2 Diabetes, Dementia, Alzheimer's Disease, and other inflammatory diseases**.



**Td**

Treponema  
denticola

Primary cause of **periodontitis and implant failure** (peri-implantitis). Research has also demonstrated its association with **Dementia and Alzheimer's Disease**.



**Tf**

Tannerella  
forsythia

Primary cause of **periodontitis and implant failure** (peri-implantitis). It's effect on other health issues is not as well known.



**Fn**

Fusobacterium  
nucleatum

Associated with **periodontitis and implant failure**. Research shows it has also been associated with **preterm birth, low birth weight babies, and fetal death**. This pathogen facilitates the movement of other oral pathogens into the circulatory system.



# Oral is Systemic



A background image of a person's torso, wearing a grey t-shirt over a red garment. Their hands are clasped together over their chest, suggesting chest pain or discomfort.

# **50% of Heart Attacks are Triggered by Oral Pathogens!**

Pessi T, Karhunen V, Karjalainen PP, et al. Bacterial signatures in thrombosis aspirates of patients with myocardial infarction. *Circulation*. 2013;127(11):1219-1228. doi: 10.1161/CIRCULATIONAHA.112.001254.

©Lora Hooper BSDH, RDH, EFDA





## OPEN ACCESS

# High-risk periodontal pathogens contribute to the pathogenesis of atherosclerosis

Bradley Field Bale,<sup>1</sup> Amy Lynn Doneen,<sup>1</sup> David John Vigerust<sup>2</sup>

<sup>1</sup>Texas Tech Health Science Center, School of Nursing, Lubbock, Texas, USA

<sup>2</sup>Department of Neurological Surgery, Vanderbilt University School of Medicine, Nashville, Tennessee, USA

### Correspondence to

Dr Bradley Field Bale,  
1002 Montrose Dr, Gallatin,  
TN 37066, USA;  
bbale@baleedoneen.com

Received 7 June 2016

Revised 1 November 2016

Accepted 5 November 2016

### ABSTRACT

Periodontal disease (PD) is generated by microorganisms. These microbes can enter the general circulation causing a bacteraemia. The result can be adverse systemic effects, which could promote conditions such as

cardiovascular disease. Level A evidence supports that PD is independently associated with arterial disease. PD

is a common chronic condition affecting the majority of Americans 30 years of age and older. Atherosclerosis remains the largest cause of death and disability. Studies indicate that the adverse cardiovascular effects from PD are due to a few putative or high-risk bacteria:

*Aggregatibacter actinomycetemcomitans*, *Porphyromonas gingivalis*, *Tannerella forsythia*, *Treponema denticola* or *Fusobacterium nucleatum*. There are three accepted

The most common were *Pg* and *Aa*. Sixty-four per cent of those atheromas had two or more pathogens. Only one of the atheroma from a patient without PD demonstrated any oral pathogens.<sup>3</sup> In 2011, 42 carotid endarterectomy specimens were analysed for oral pathogen DNA. Every atheroma had at least one pathogen, and many had multiple pathogens. Again, the most common bacteria were *Pg* and *Aa*.<sup>4</sup> Oral pathogens create bacteraemia, and those bacteria, especially the high-risk microbes, are frequently associated with atherosclerotic lesions.

The American Heart Association (AHA) stated after an extensive review of the literature that PD was independently associated with arteriosclerotic vascular disease (ASVD). This relationship was

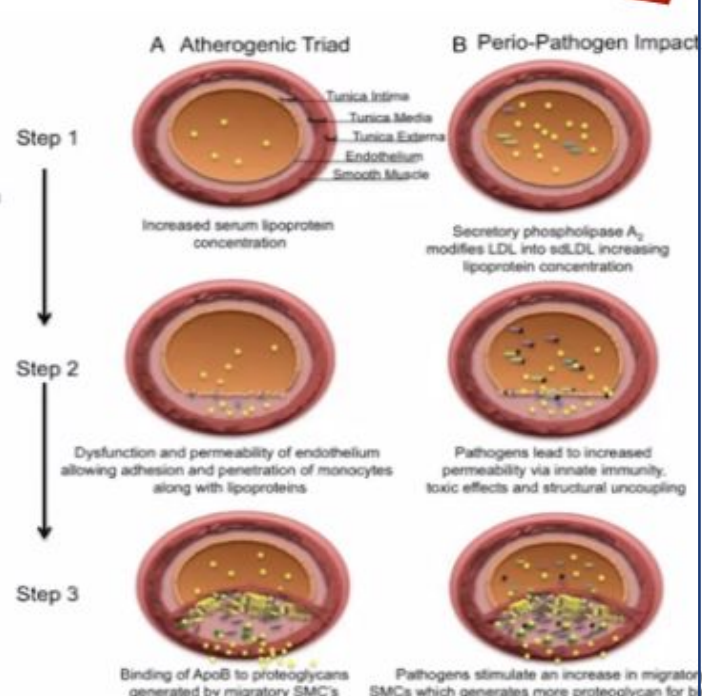
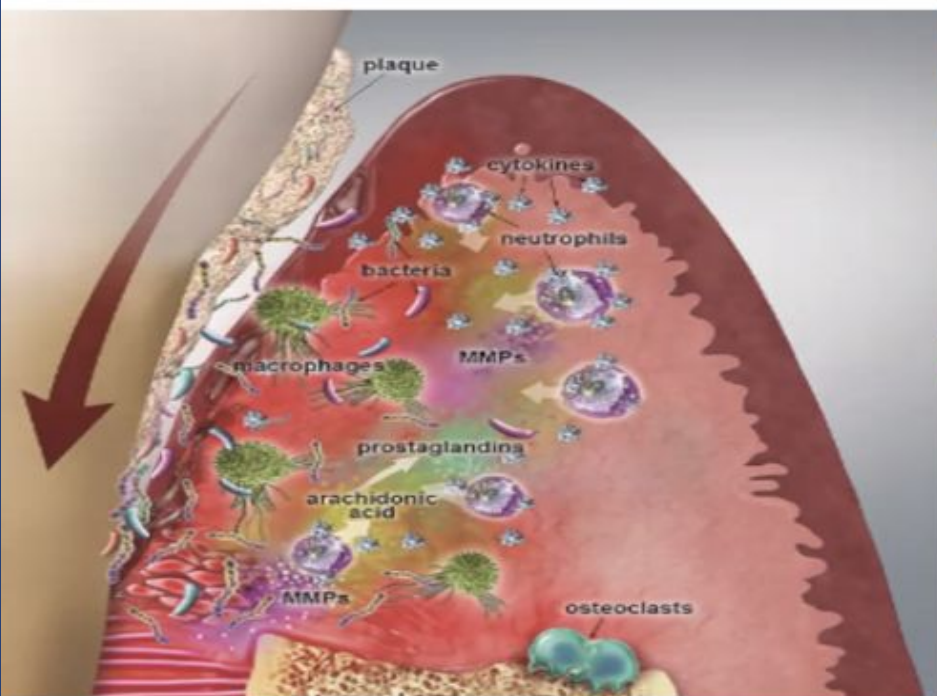
# LEAKY SYNDROME



DIRECT  
DIAGNOSTICS

## INFLAMMATORY CASCADE

High risk bacteria: Aa, Pg, Tf, Td, Fn



Periodontal Disease

Vascular Disease

# Inflammation Testing

Total Cholesterol

Triglycerides

HDL

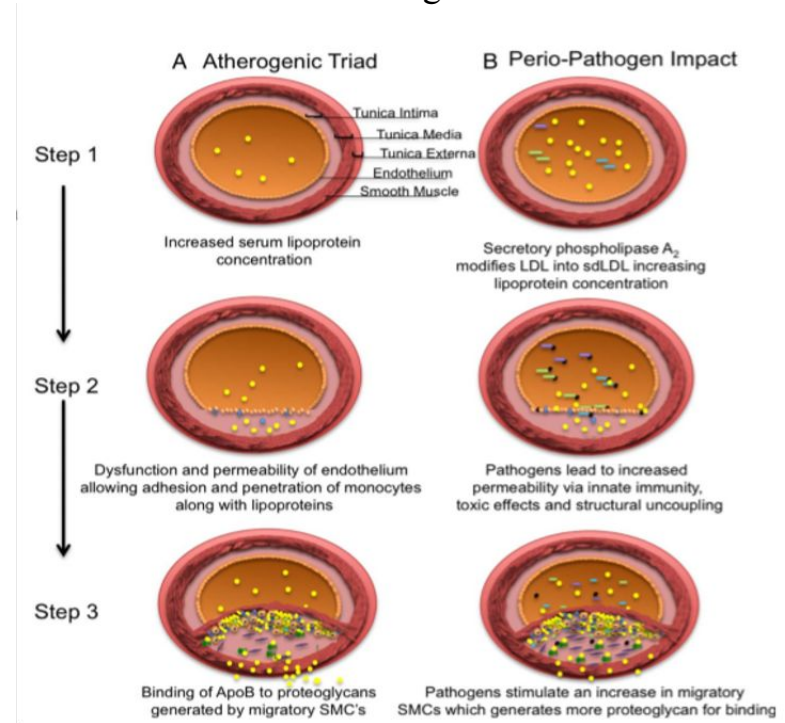
Apolipoprotein B

LDL

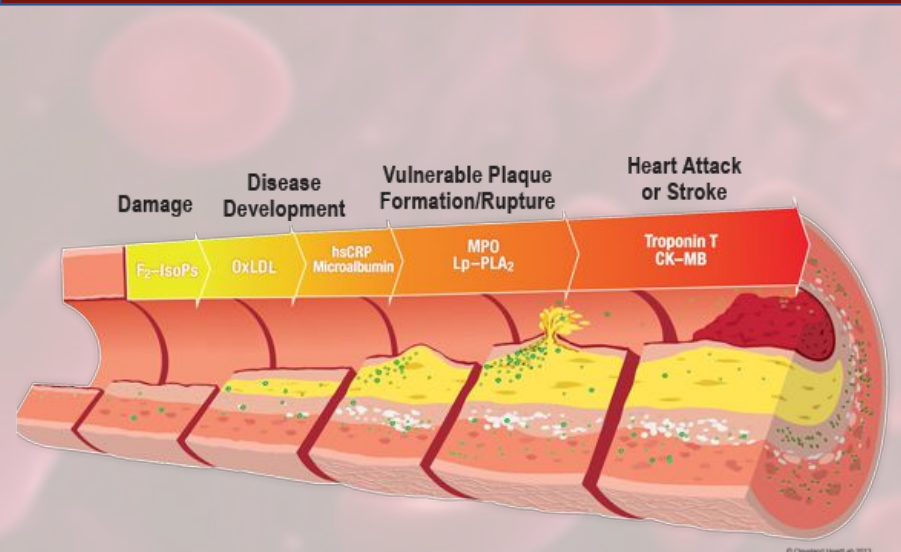
Apolipoprotein A1

Lipoprotein (a)

Leaky Syndrome  
Leaky Gums, Leaky Gut, Leaky Gutter  
Atherogenic Triad



# CIMT The 5 High Risk Bacteria Have a Direct & Deadly Impact in and on the Arterial Wall

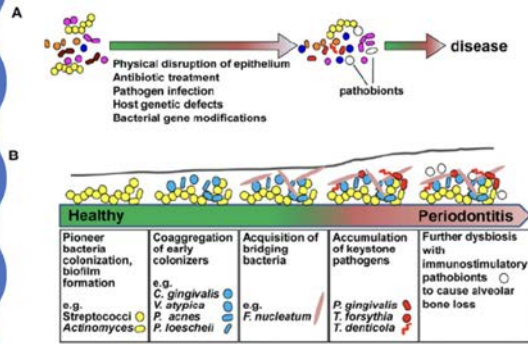


1. Our results are consistent with Pussinen et al's<sup>29</sup> report that serum antibody levels to periodontal bacteria (and thus systemic translation of local infection) **are more related to bacterial levels than overt clinical disease**. Hence, as an exposure of significance for systemic disease, "preclinical" periodontal disease cannot be ignored.<sup>30</sup>
2. We have found an % 0.1 mm difference in IMT change among participants **with deteriorating versus improving periodontal health** during a relatively short time (ie, 3 years).

**Changes in Clinical and Microbiological Periodontal Profiles Relate to Progression of Carotid Intima -Media Thickness: The Oral Infections and Vascular Disease Epidemiology Study**  
Moïse Desvarieux, Ryan T. Demmer, David R. Jacobs, Panos N. Papapanou, Ralph L. Sacco and Tatjana Rundek  
*J Am Heart Assoc.* 2013;2:e000254; originally published October 28, 2013;



# HOW BIOFILM GROWS



The Role of Oral Pathobionts in Dysbiosis during Periodontitis Development

March 2014, *Journal of Dental Research* 93(6), DOI:10.1177/0022034514528212, PubMed

## PERIO PATHOGENS FOUND IN CAROTID ATHEROMA

## Bacteria Resistant to Scaling & Root planing

- *A.a.* (66.67%, 28/42)
- *P.g.* (78.57%, 33/42),
- *T.f.* (61.90%, 26/42)
- *F.n.* (50.00%,) 21/42

42 carotid endarterectomy specimens analyzed via DNA for PD pathogens

Figuerio, E. DDS, et al. *Journal of Periodontology*, 8/2011. DOI: 10.1902/jop.2011.100719

- *A.a.*
- *P.g.*
- *T.f.*

Source: *Periodontology* 2000, vol.28 2002, 106-176, slots, Ting

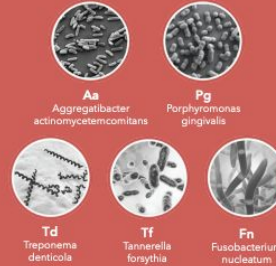
## AIRWAY

- Airway to pathogens to plaque
- Scalloped tongue
- Narrow palate
- Overlapping / crooked teeth
- Mallampati score (how much of the back of the throat can you see?)
- Tongue-tie
- Worn / cracked teeth
- Neck size / BMI / weight
- Snoring
- Waking up at night / not able to fall asleep / insomnia / nightly urination
- Morning or frequent headaches
- Puffy / red gums

## AIRWAY

## PATHOGENS PLAQUE

## PATHOGENS



## PLAQUE

- Pathogens build up in the lining of the artery wall. As oxygen desaturates, pathogens and plaques increase.
- CIMT to measure thickness of artery wall, presence of plaque, and arterial age
- Pathogens cross the blood brain barrier, increasing the production of beta amyloid plaques





# Airway to GI

Lack of nitric oxide

Lack of healthy commensals

Dysbiosis of mouth and gut

# J Oral Microbiol 2019

11(1): 1586422. Published online March 2019 doi:

Here are some of the findings of the study, published by the National Center for Biotechnology Information, a division of the U.S. National Institute of Health:

- 1) Bleeding gums that occur because of gingivitis and gum disease, can give oral microbes access to the **bloodstream**. This allows these microorganisms to circulate systemically.
- 2) Microbes from the mouth directly penetrate the **esophagus**, which is the muscular tube that connects the throat with the stomach. This can be enough to unbalance the ecosystem of the digestive tract.
- 3) There have been recent advances in identifying microbial metabolites that directly affect the **gastrointestinal tract**. Metabolites is a fancy word for bacteria poop and other by-product of microbial metabolism. These metabolites can lead to various chronic diseases of the digestive tract and they are also absorbed into the blood stream, causing a low grade inflammatory state.

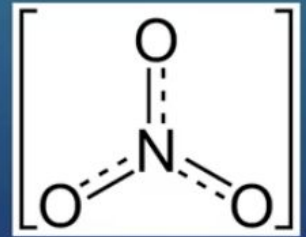
# Nitrate is inert in Humans.

# Nitrate must be reduced to nitrite by commensal bacteria.

200 million Americans use mouthwash daily

200 million Americans use antacids daily

200 million prescriptions for antibiotics every year

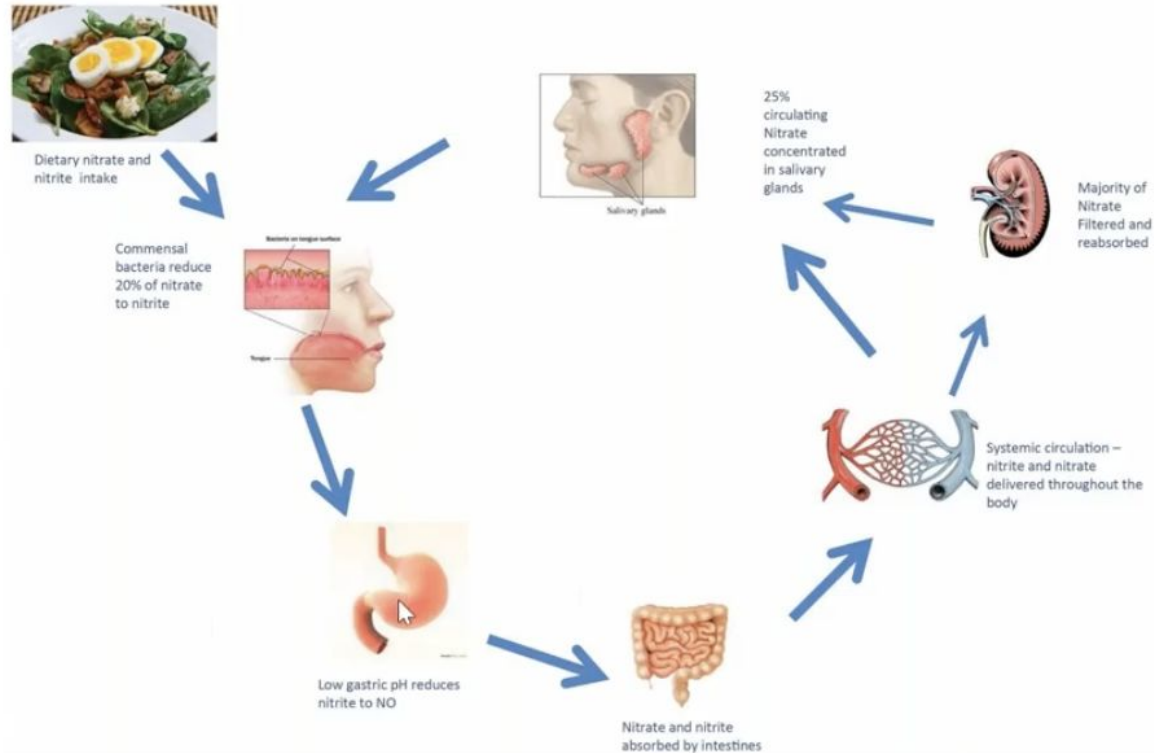


# Disruption of **Nitrate-Nitrite-NO** Pathway

1. Insufficient dietary intake of nitrate/nitrite rich foods  
(green leafy vegetables, beets, etc.)
2. Problems with nitrate uptake in duodenum  
(sialin (SLC17A5) transporter mutations – Salla Disease)
3. Insufficient saliva production  
(Sjogrens syndrome)
4. Lack of oral commensal bacteria to reduce nitrate to nitrite  
(use of antibiotics (over 200M users)/antiseptic mouthwash  
(over 200M users), poor oral hygiene)
5. Insufficient stomach acid production – Achlorhydria  
(use of PPI's (over 200M), H. Pylori infection, iron overload)
6. Increased oxidative stress that scavenges NO

NO THERAPEUTIC INTERVENTION

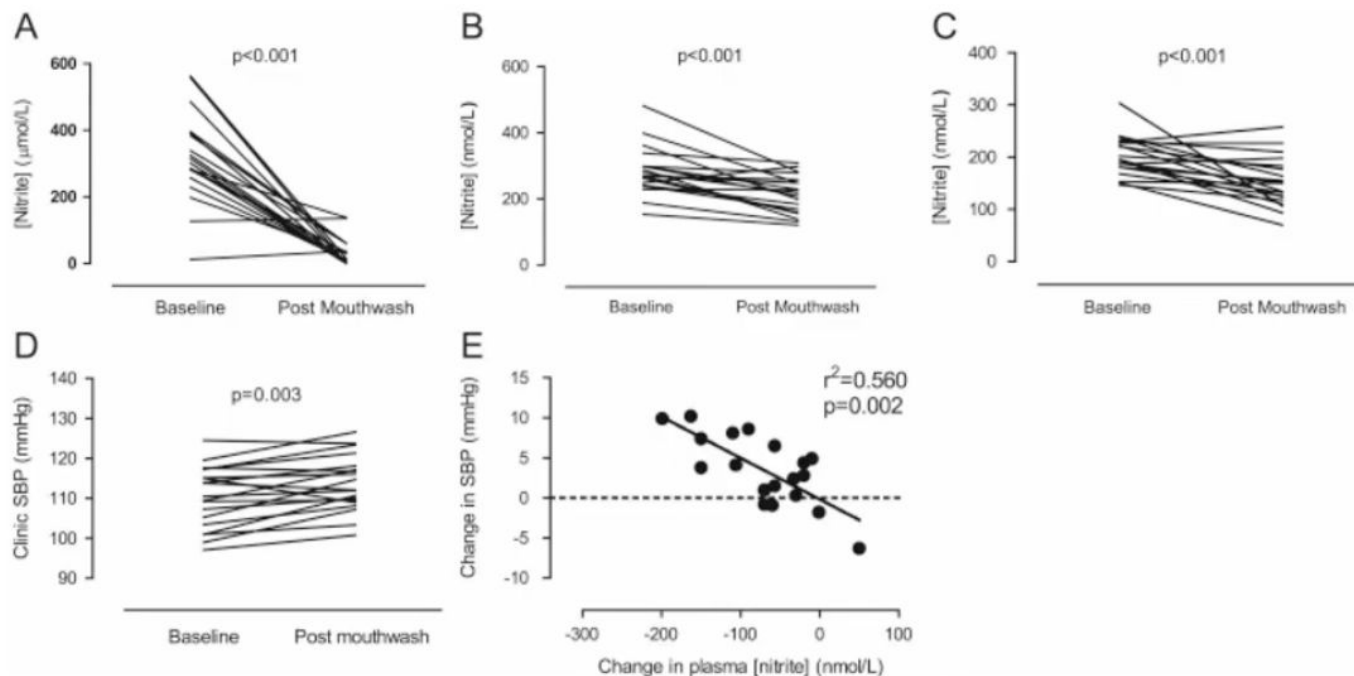
# New Paradigm - Human Nitrogen Cycle



NO THERAPEUTIC INTERVENTION

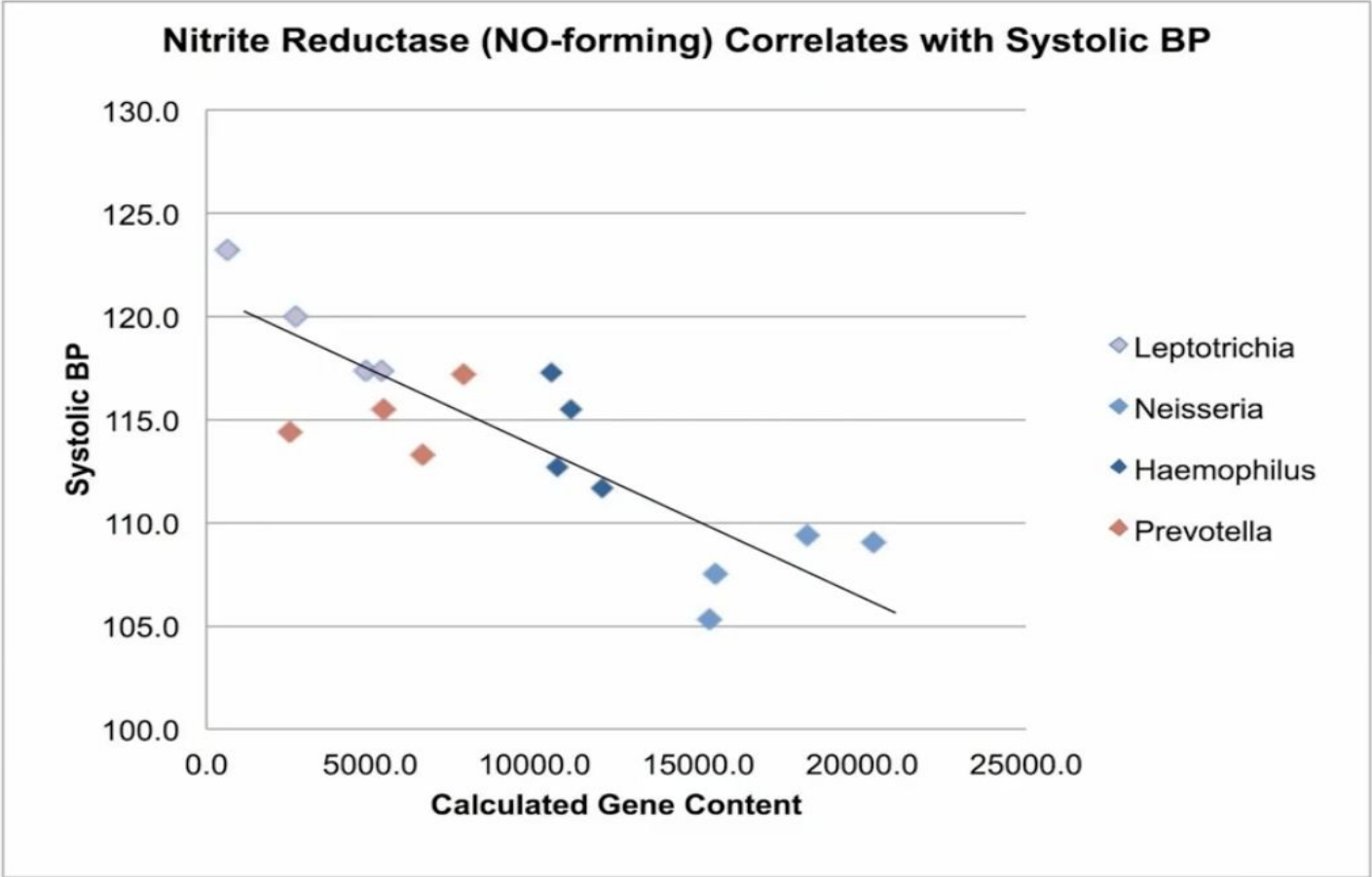


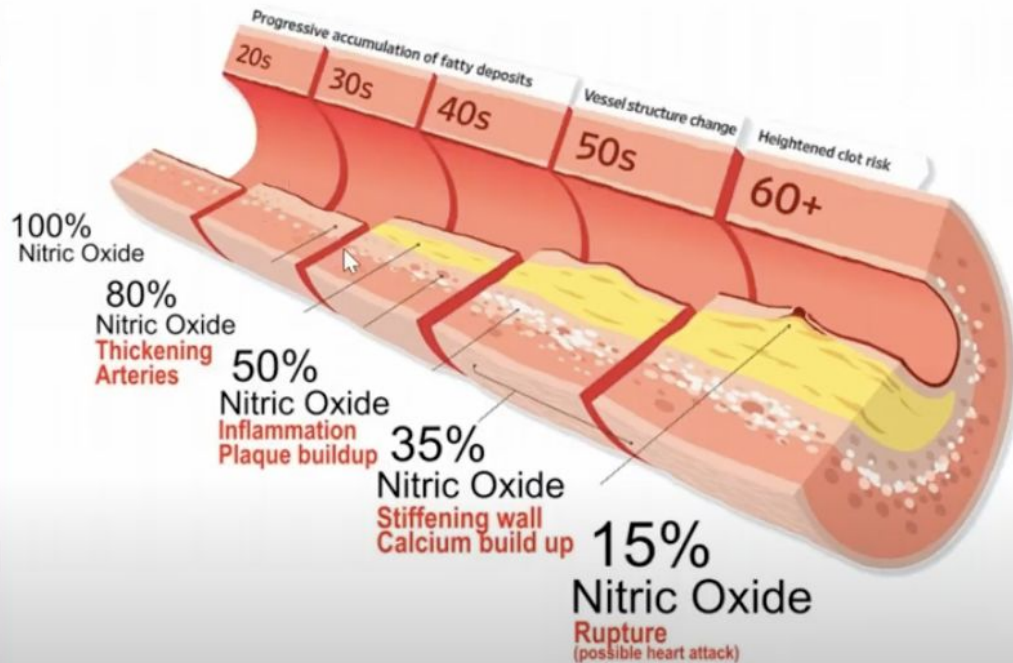
# Physiological Role for Nitrate-Reducing Oral Bacteria in **Blood Pressure Control**



Kapil et al Free Radic Biol Med. 2013 Feb;55:93-100

NO THERAPEUTIC INTERVENTION





Loss of **NO** is  
Associated with  
**Atherosclerosis.**

As we age, we lose 85%  
of our ability to make  
Nitric Oxide.

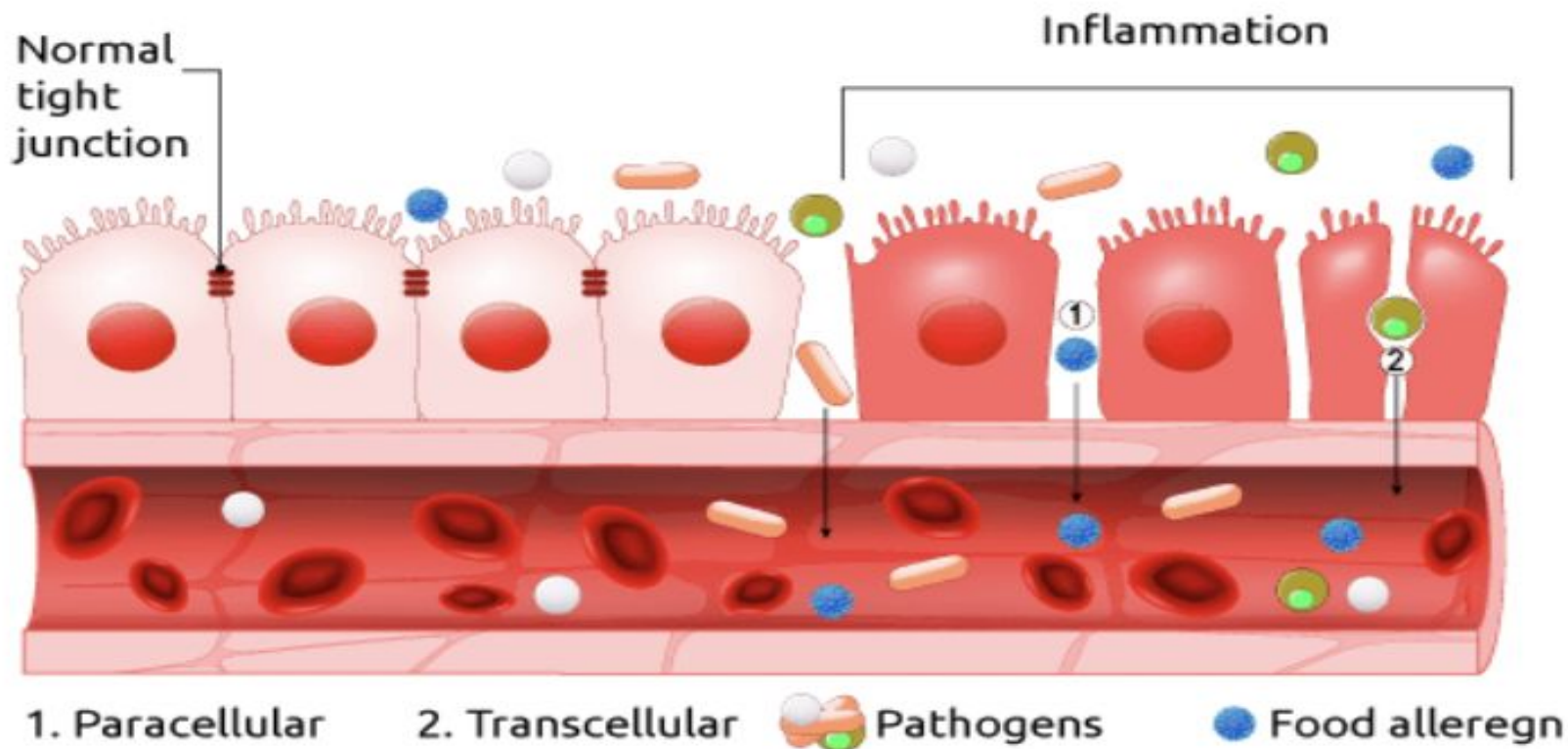
“© Copyright Nathan S. Bryan, Ph.D. 2019. All Rights Reserved.”

# Dysbiosis

Nitrates to nitric oxide



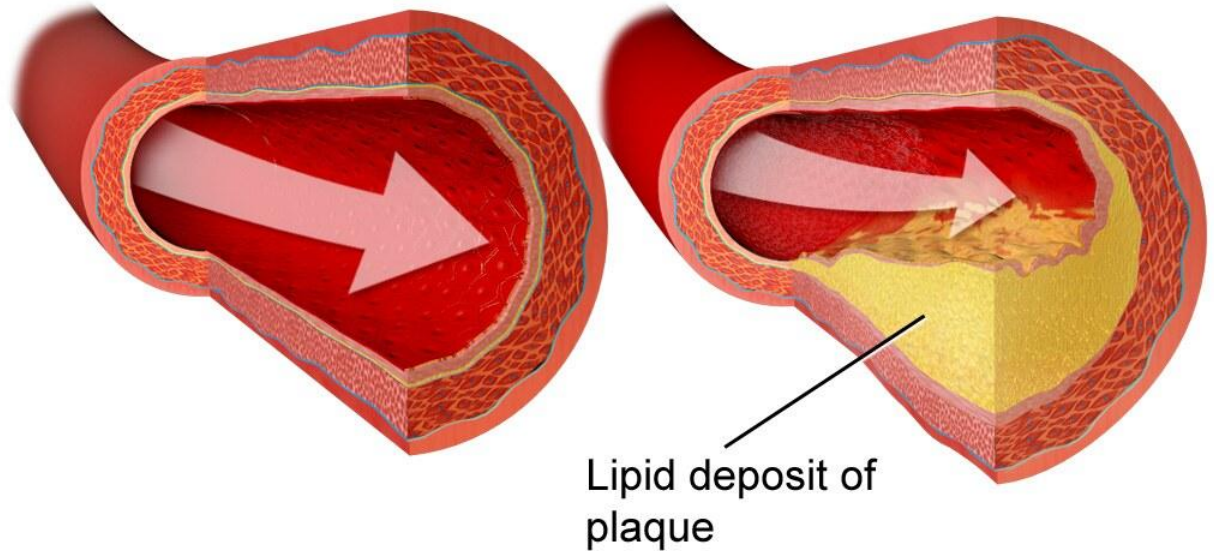
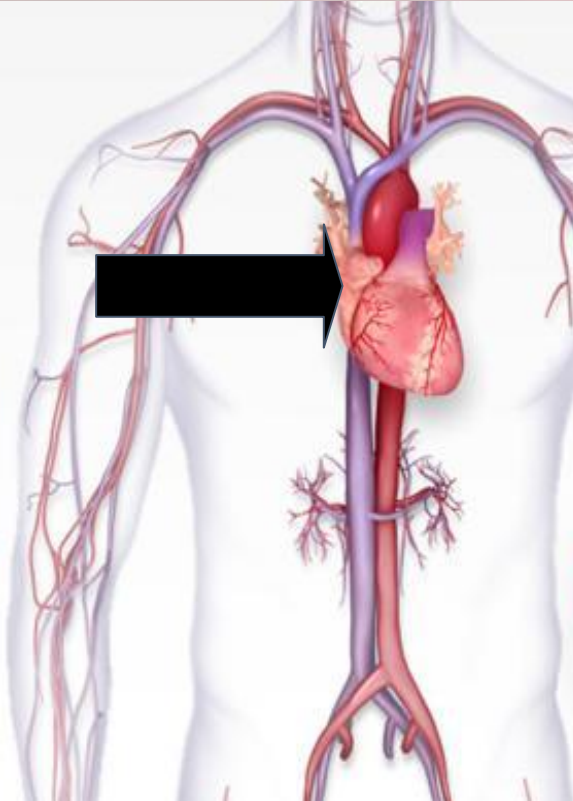
# Leaky Gut



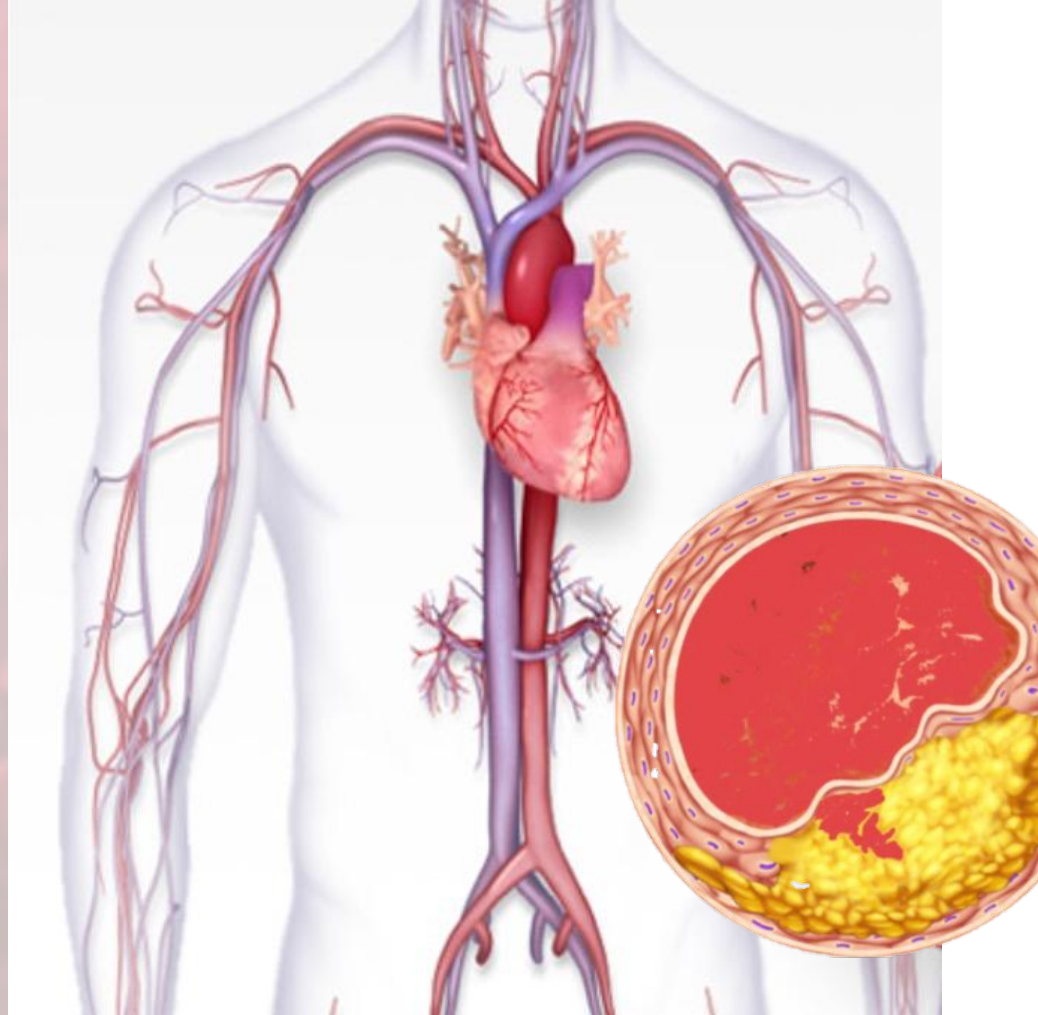
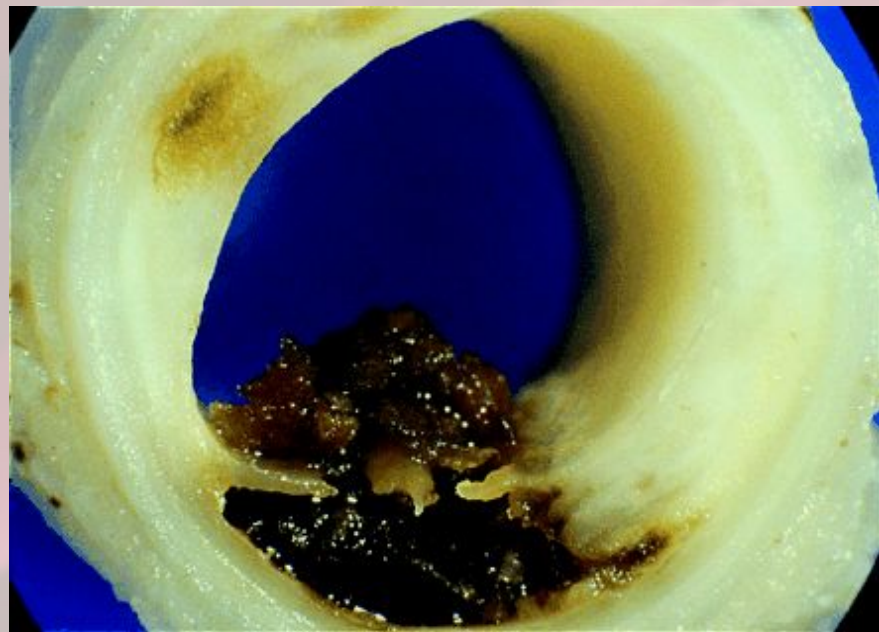




# Subclinical Disease EVALUATION BEFORE SYMPTOMS



## Coronary Artery Disease





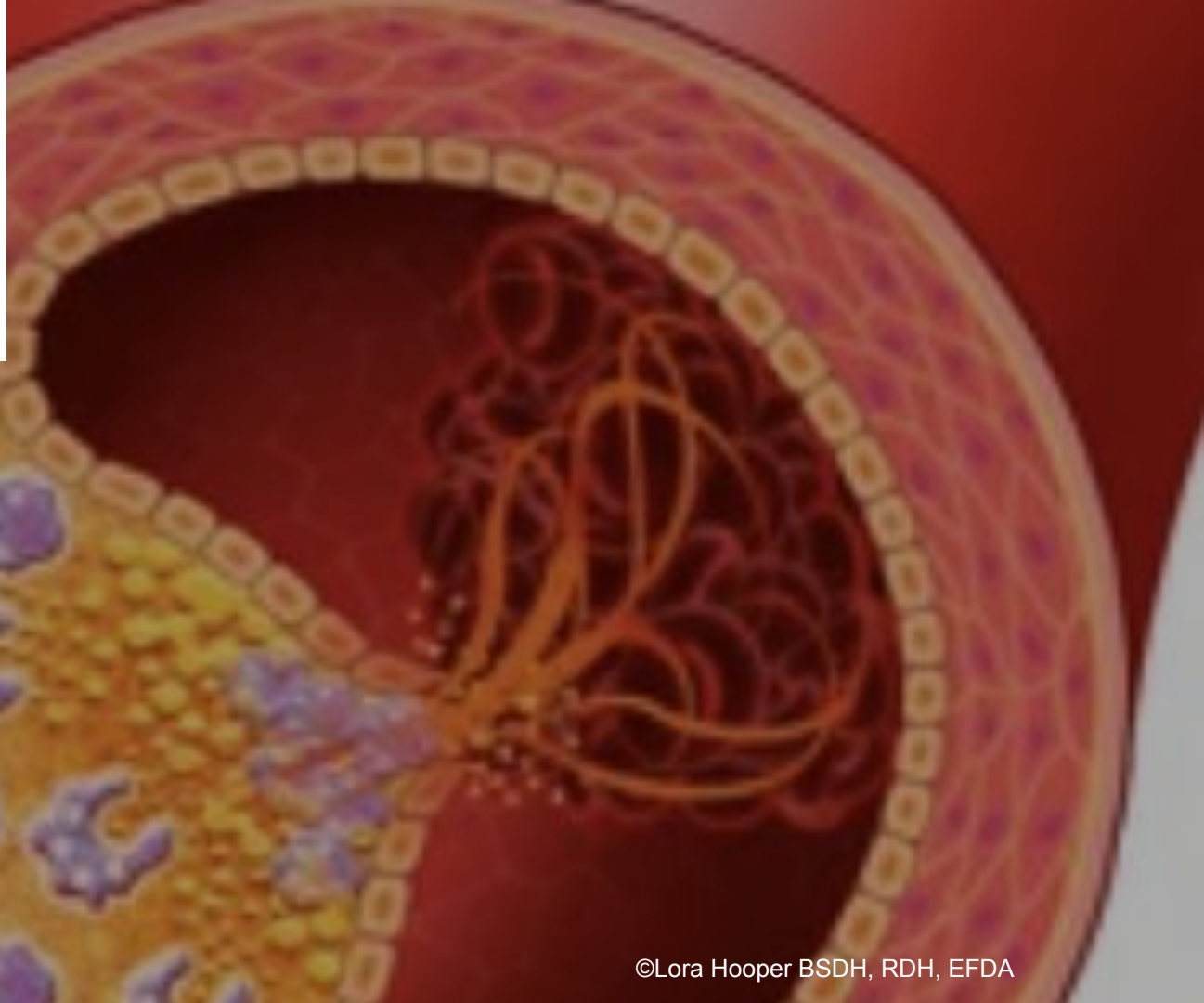
**Ultrasound:**

**Aorta**

**Carotid (CIMT)**

**CT:**

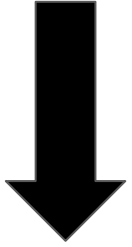
**Coronary (CCTA)**





# LEAKY SYNDROME

Leaky Gums

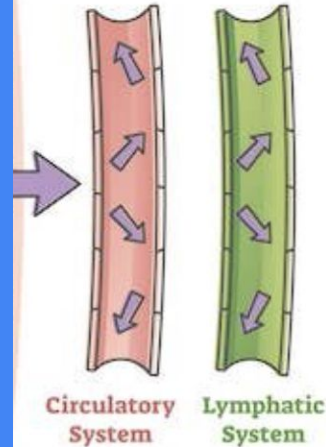
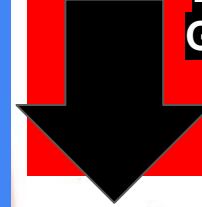


Leaky Gut



**Airway to  
Pathogens to  
Plaque**

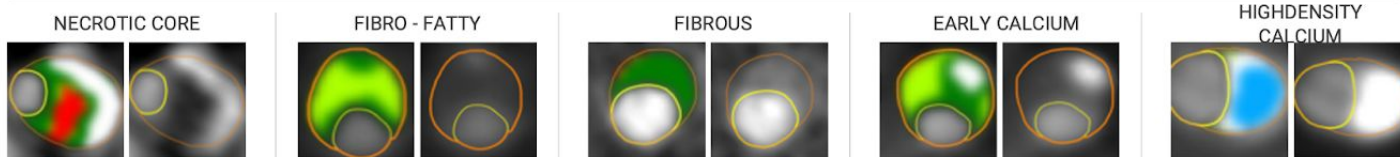
Leaky  
Gutter



# Not all plaque is the same.

It's not just about identifying the presence of plaque. Determining the amount and type of atherosclerosis (plaque) present enables earliest diagnosis and precision treatment possible.

Plaques that look different behave differently.



HIGH RISK (~10%)

INTERMEDIATE RISK (~60%)

LOW RISK (~30%)

## 1. Dark Plaques are Dangerous.

Dark (non-calcified) plaques are the strongest predictor of heart attack risk.

## 2. Bright Plaques are Stable.

Bright (calcified) plaques are protective against heart attacks.

## 3. Plaques Change Over Time.

Medicine, diet and exercise transform dark plaques to bright plaques.

## 4. Prevent Heart Attacks.

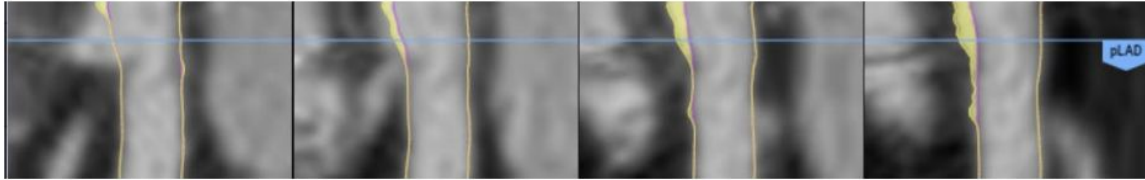
Heart attack prevention is not through regression, but through plaque transformation.

Transforming plaque type is the only trackable approach to personalizing heart attack prevention.

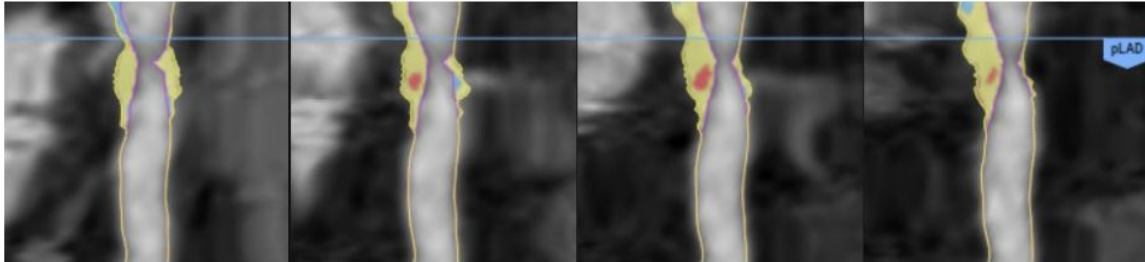


# Examples of Baseline and Follow-up Comparison Results

**Initial: 2010**

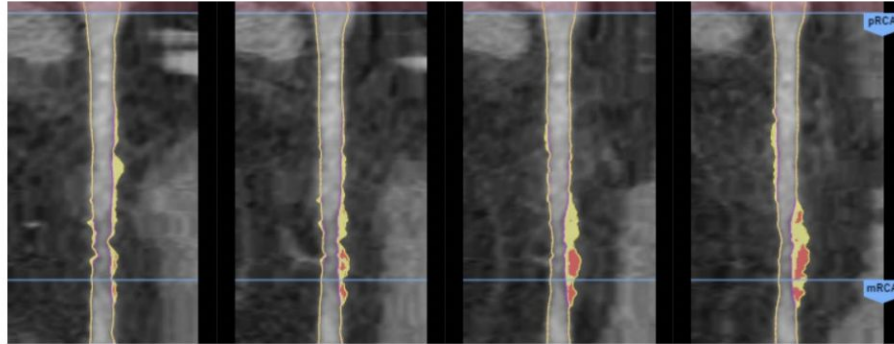


**Follow up: 2017**

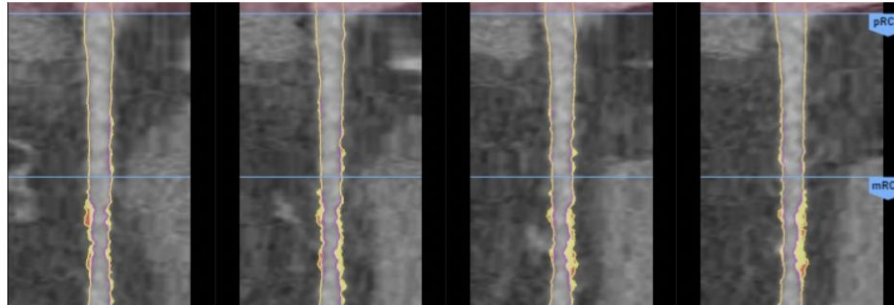


# Examples of Baseline and Follow-up Comparison Results

Initial: 2017

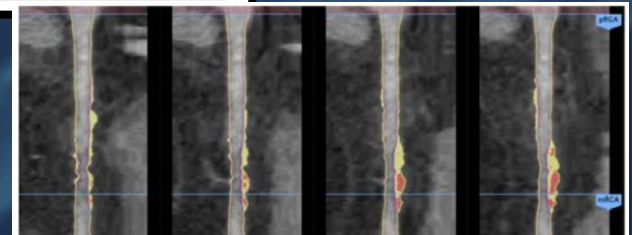
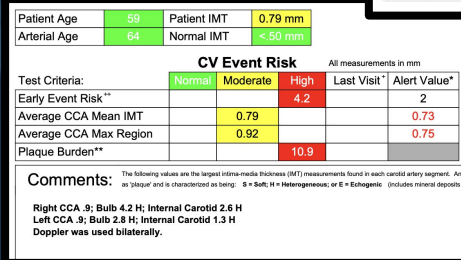
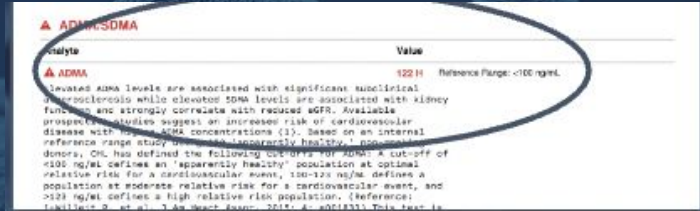
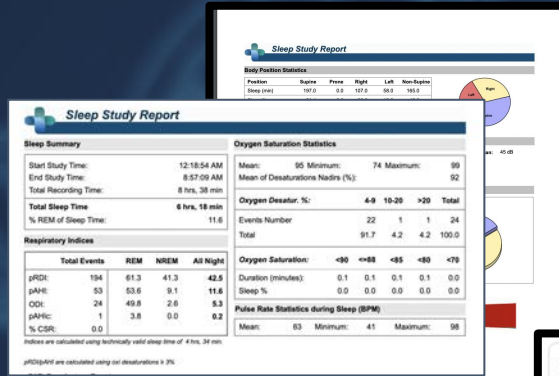


Follow up: 2018

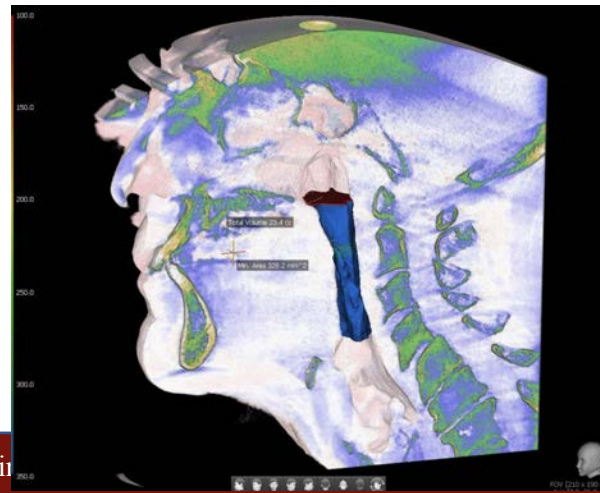




# Put the story together: Airway to Pathogens to Plaque



# Case



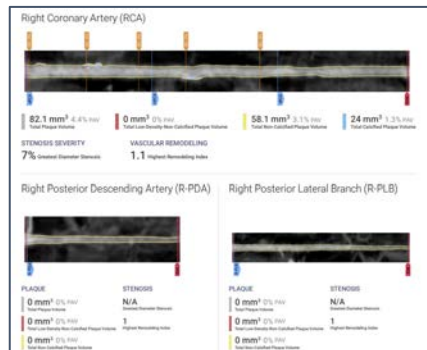
1/2022

6/2022

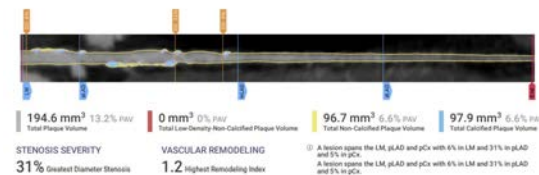
3/2022

INFLAMMATION	
Myeloperoxidase <sup>(B)</sup>	378
Lp-PLA <sub>2</sub> Activity <sup>(7)</sup>	100
hs-CRP	2.1
Microalbumin/Creatinine	UND
Microalbumin	<3.0
Creatinine, Urine, Random	31
ADMA (Asymmetric dimethylarginine) <sup>(1)</sup>	94
SDMA (Symmetric dimethylarginine)	102
F <sub>2</sub> -Isoprostane/Creatinine <sup>(6)</sup>	0.94
F <sub>2</sub> -Isoprostane	0.29
Creatinine, Urine, Random	31
LIPIDS	

<b>▲ ADIPOPROTEIN S</b>	
Reference Range: <10	87 H
Risk Category:	
Optimal: < 10	
Moderate: 10 - 150	
High: > 150	
Cardiovascular event risk category cut points (optimal, moderate, high) are based on national lipid association recommendations (Lipid 16 et al, J Clin Lipid. 2013;17(1):108-128 and Sirtori CR et al, Endocr Pract. 2017;23(1):21-37).	
<b>▲ Lp-PLA<sub>2</sub> ACTIVITY</b>	
Analyte	Value
▲ Lp-PLA <sub>2</sub> ACTIVITY	128 H
Reference Range: <125 nmol/mL	
This test was developed and its analytical performance characteristics have been determined by Quest Diagnostics Nichols Institute San Juan Capistrano. It has not been cleared or approved by FDA. This assay has been validated pursuant to the CLIA regulations and is used for clinical purposes.	
<b>▲ ADMA/SDMA</b>	
Analyte	Value
ADMA/SDMA	102 H
Reference Range: <100 ng/mL	
Elevated ADMA levels are associated with significant nonclinical atherosclerosis while elevated SDMA levels are associated with kidney function and strongly correlate with reduced eGFR. Available prospective studies suggest an increased risk of cardiovascular disease with higher ADMA concentrations (1). Based on an internal reference range study using 300 "apparently healthy," non-smoking donors, Qn has defined the following cutpoints for ADMA: a cutpoint of <100 ng/mL defines an "apparently healthy" population at optimal relative risk for a cardiovascular event, 100-120 ng/mL defines a population at moderate relative risk for a cardiovascular event, and >120 ng/mL defines a high relative risk population. (Reference: 1. J Intern Med. 2014;275(1):1-11).	



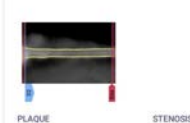
Left Main and Left Anterior Descending (LM+LAD)



First Diagonal Branch (D1)

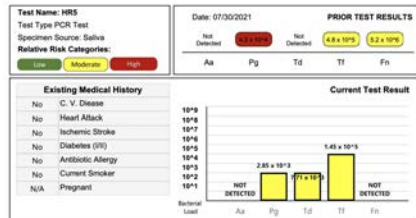
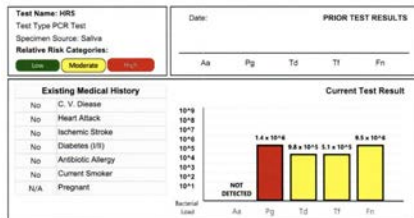


Second Diagonal Branch (D2)



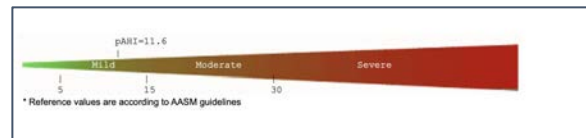
7/2021

3/2022



Patient: 11061794 Patient ID: 3145234 Accession Risk Score: Patient Info:	Provide: Vending No. 0000 Prices: TX 75030 Account No: 1300	Specimen:	Report Date & Time: 05/14/2022 3:00 AM Received Date & Time: 05/11/2022 4:00 PM Collection Date & Time: 05/10/2022 01:00 PM						
Test Name	Test Result	Interpretation	Reference	Test Name	Test Result	Interpretation	Reference		
<b>Genetic Tests by Genotyping<sup>14</sup></b>									
Reported Date: 05/14/22	Appt	L513	Most common genotype.  If cholesterol/lipid levels is indicated and/or if patient is on statin therapy, 1.5-fold increased CVD risk. Optimal all CVD Risk factors.		Reported Date: 05/14/22	MTFR	677 G/T 1290 A/C  677 G/T genotype - single copy of 1750A/G genotype - single copy of 1750A/G  Hypertension is elevated consider nutritional therapy and/or statin and other a statin supplementation	10	
Reported Date: 05/14/22	9p21 CVD Risk	n1979728 A/G	1.5-fold increased CVD risk. Optimal all CVD Risk factors.	11	Reported Date: 05/14/22	KIF5B Status	1/T	Normal CVD Risk.	11
Reported Date: 05/14/22	Hypertension	n1333048 G/G	1.5-fold increased CVD risk. Optimal all CVD Risk factors.		Reported Date: 05/14/22	4q25 AFib Fibrillation Risk	n1933844 T/G  1.4-fold increased risk of atrial fibrillation and stroke. Monitor for all and if develops, use 1.6	11	

Sleep Study Report			
Sleep Summary		Oxygen Saturation Statistics	
Start Study Time:	12:18:54 AM	Mean:	95 Minimum: 74 Maximum: 99
End Study Time:	8:57:09 AM	Mean of Desaturations Nadir (%):	92
Total Recording Time:	8 hrs, 38 min	Oxygen Desatur. %:	4.9 10-20 >20 Total
Total Sleep Time:	6 hrs, 18 min	Events Number:	22 1 1 34
% REM of Sleep Time:	11.6	Total:	91.7 4.2 4.2 100.0
Respiratory Indices		Oxygen Saturation	
Total Events	REM NREM All Night	<90	<88
pRDI:	194 61.3 41.3 42.5	<85	<80
pAHI:	53 53.6 9.1 11.6	<75	<70
ODI:	24 45.8 2.6 5.3	Duration (minutes):	0.1 0.1 0.1 0.1 0.0
pAHC:	1 3.8 0.0 0.2	Sleep %:	0.0 0.0 0.0 0.0 0.0
% CSR:	0.0	Pulse Rate Statistics during Sleep (BPM)	
		Mean:	83 Minimum: 41 Maximum: 98



# Tests in Both Medical and Dental

## 1. Airway:

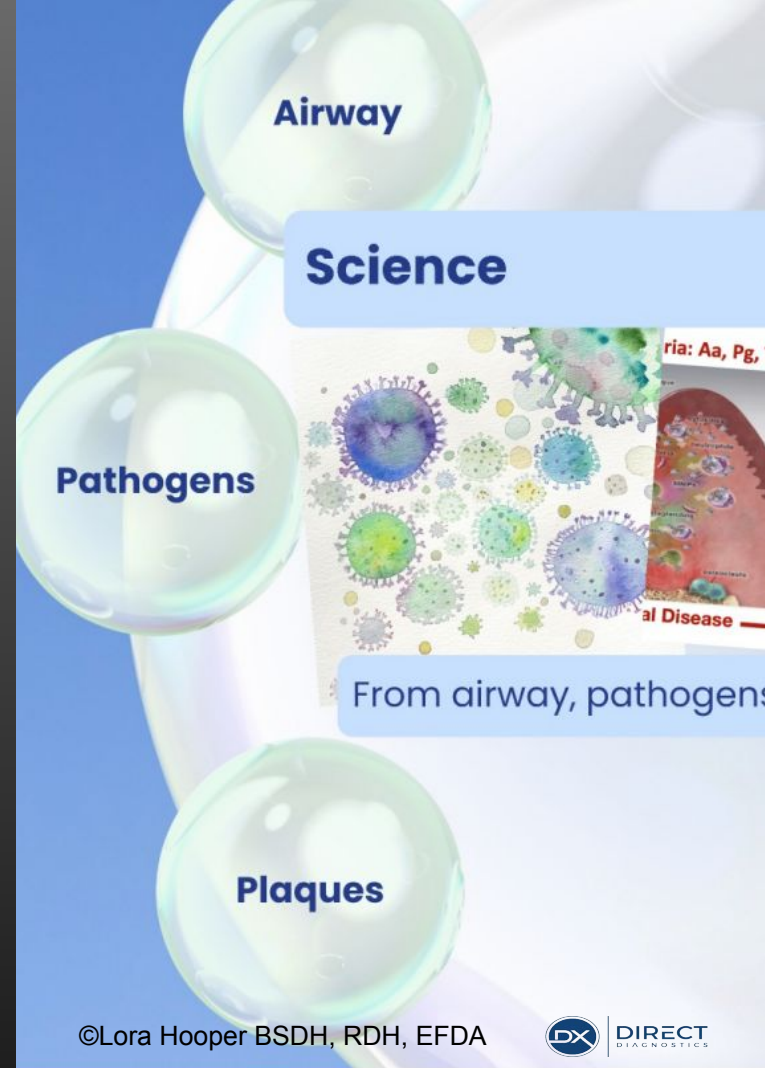
- Sleep test- WatchPat
- Nitric oxide test
- ADMA

## 2. Pathogens:

- Saliva test: HR5 test

## 3. Plaque:

- CMT Scan / Cleerly AI (CT angiogram)







# Airway

Sleep, nitric oxide, CBCT, & ADMA

©Gina Pritchard, DNP, RN, CNS, ACNP

[directdiagnostics.com](http://directdiagnostics.com)



DIRECT  
DIAGNOSTICS

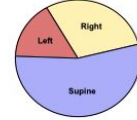
# Sleep test



## Sleep Study Report

### Body Position Statistics

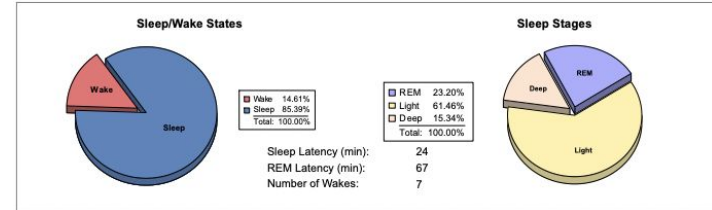
Position	Supine	Prone	Right	Left	Non-Supine
Sleep (min)	197.0	0.0	107.0	58.0	165.0
Sleep %	54.4	0.0	29.6	16.0	45.6
pRDI	50.3	N/A	29.1	22.2	25.9
pAHI	48.4	N/A	29.1	22.2	25.9
ODI	31.6	N/A	18.8	11.1	15.3



### Snoring Statistics

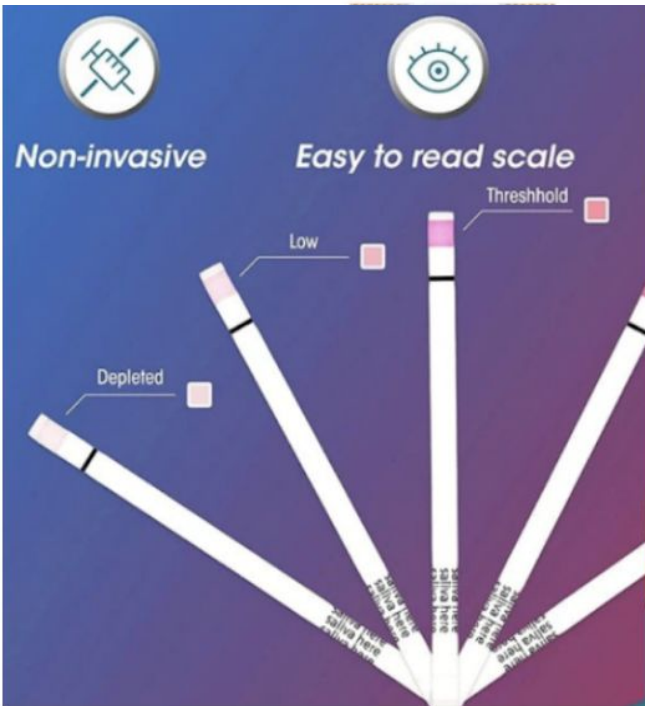
Snoring Level (dB)	>40	>50	>60	>70	>80	>Threshold (45)	Mean: 45 dB
Sleep (min)	186.1	80.7	10.1	0.0	0.0	126.7	
Sleep %	51.4	22.3	2.8	0.0	0.0	35.0	

### Sleep Stages Chart



\* Reference values are according to AASM guidelines

# New Paradigm - Human Nitrogen Cycle



## Directions for Use:

1. Place the test strip with the “saliva here” side on your tongue and hold for 5 seconds.
2. Fold the strip over and gently press the two sides together and hold for 10 seconds.
3. Separate and compare your results using the Berkeley Test Nitric Oxide Scale on the side of the package.

Nitrate and nitrite  
absorbed by intestines

NO THERAPEUTIC INTERVENTION

© Copyright Nathan S. Bryan, Ph.D. 2019. All Rights Reserved.

©Lora Hooper BSDH, RDH, EFDA



## Know Your Risk

for Blood Vessel Damage.

### ADMA/SDMA

#### What are ADMA and SDMA?

ADMA (asymmetric dimethylarginine) and SDMA (symmetric dimethylarginine) are made by your body when proteins are broken down. Too much ADMA and SDMA can make it hard for your body to make nitric oxide, which is a chemical needed to keep blood vessels healthy.

#### Why check my ADMA and SDMA levels?

Picture wasps and honey bees competing for nectar. They may look similar, but only the honey bees can use nectar to make honey. This is like how ADMA can compete with structures in our bodies for certain proteins that make nitric oxide. Only special structures, like L-arginine (found in foods like seeds, nuts, fish, soy, meat, and dairy), can work with these proteins to make nitric oxide. ADMA looks like these structures, but it cannot be used to make nitric oxide. Without enough nitric oxide, blood vessels can become damaged, which increases your risk for heart and blood vessel disease.

High levels of ADMA may suggest your body isn't making enough nitric oxide and there could be a problem with your blood vessel health. SDMA looks a lot like ADMA and it can let your doctor know if your kidneys are working properly.

Your doctor may want to check your ADMA and SDMA levels if you don't have good eating or lifestyle habits, or if you have major risk factors for heart attacks, such as smoking, high blood pressure, high blood sugar, or high cholesterol levels, or your kidneys are damaged. Ask your doctor if this test is right for you.

#### What can I do to improve my ADMA/SDMA levels?

There are a number of things you can do help keep your blood vessels healthy and lower your risk of heart disease, as well as your ADMA/SDMA levels.

- **Eat a healthy diet.** A heart-healthy, Mediterranean diet – that is low in saturated fat and cholesterol, full of high-fiber foods (such as fresh fruits, vegetables, and whole grains), and has very little added salt and sugars – can help you control cholesterol levels, blood pressure, and blood sugar.

- **Exercise more.** Talk with your doctor about exercises that would be safe for you to do.
- **If you smoke, you should quit.** Smoking damages the walls of blood vessels and increases both heart attack and stroke risk, partly because smoking can also make clots form faster and bigger.
- **Take your medications,** if told by your doctor, to lower your blood pressure, blood sugar, and/or blood cholesterol levels.

With heart disease being the #1 killer of Americans, it's important to develop a plan with your doctor to lower your risk of a heart attack or stroke before one happens.

#### Additional Need-to-Knows:

The ADMA/SDMA test can be done at the same time you have your standard cholesterol test. When getting ready for the ADMA/SDMA test:

- Keep taking your medications as directed.
- Fasting is not required.

#### ADMA Relative Risk

<b>&lt;100</b> Low	Your ADMA result is in the desirable range, suggesting that you have good nitric oxide levels and lower risk of blood vessel damage.
<b>≥100</b> Moderate/High	You have moderate/high levels of ADMA, suggesting that you may have low nitric oxide levels and higher risk of blood vessel damage.

#### SDMA Reference Range

<b>&lt;135</b> Low	Your result is in the desirable range, suggesting that you have lower risk for kidney problems.
<b>≥135</b> Moderate/High	You have high levels of SDMA, suggesting that you have higher risk for kidney problems.

#### What do my results mean?

#### What do my results mean?

### INFLAMMATION

Myeloperoxidase <sup>(10)</sup>	213
Lp-PLA <sub>2</sub> Activity <sup>(9)</sup>	153
hs-CRP	0.3
Microalbumin/Creatinine	UND
Microalbumin	<3.0
Creatinine, Urine, Random	122
ADMA (Asymmetric dimethylarginine) <sup>(1)</sup>	109
SDMA (Symmetric dimethylarginine)	87
CxLDL	61
F <sub>2</sub> -Isoprostane/Creatinine <sup>(8)</sup>	0.24
F <sub>2</sub> -Isoprostane	0.29
Creatinine, Urine, Random	122





# Pathogens

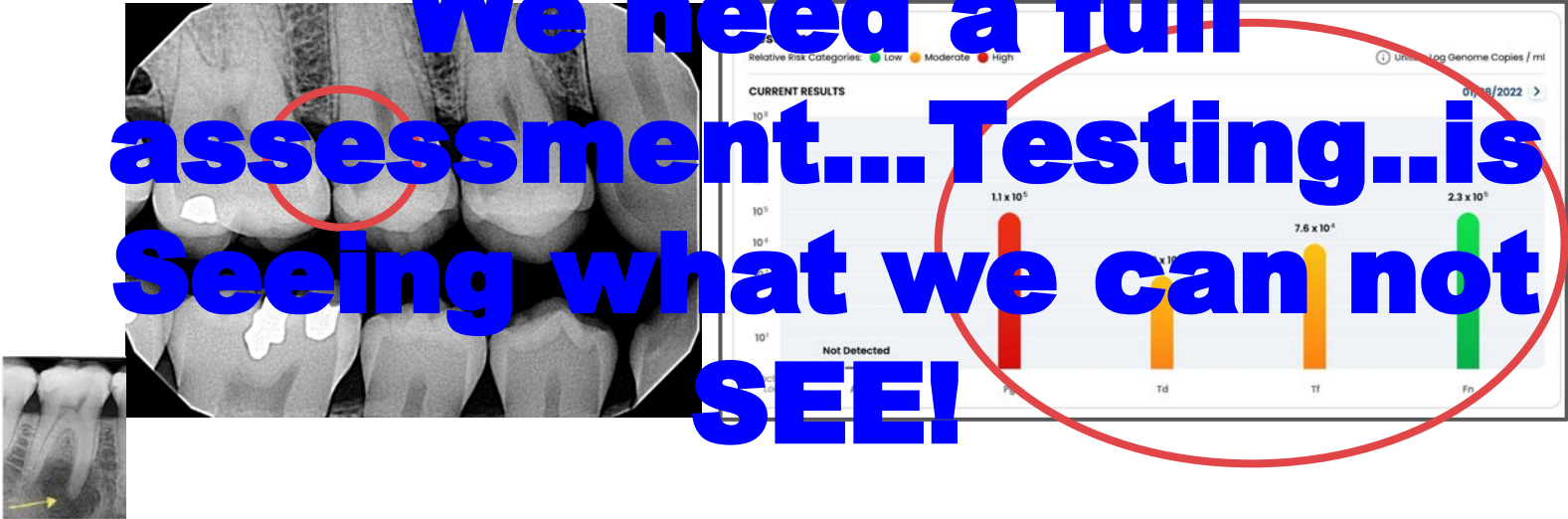
5 High Risk Pathogens HR5 saliva test

©Gina Pritchard, DNP, RN, CNS, ACNP

# Who is 80% or who is 20%... how would you know?

- HR5 test is comparable to taking x-rays-

**We need a full assessment... Testing..is Seeing what we can not SEE!**



# IT'S YOUR MOUTH TELLING YOU YOUR BODY A

Evidence shows that 5 high risk oral pathogens are the causative drivers of inflammation and disease

Aa



## Aggregatibacter

- Heart Disease • B
- Blocks Immune Sy
- Oxidative Stress •
- Rapid Bone Loss

Pg



## Porphyromonas

- Rheumatoid Arthr
- Blocks Immune Sy
- Appendicitis • O
- Cancer • Implant

13.6x increased risk of a ca

Td



## Treponema de

- Diabetes • Joint f
- Aneurysm • Heart
- Cancer • Stroke

FEEDS OFF STRESS!

Tf



## Tannerella fors

- Artery Plaque • H
- Joint Replacemen
- Implant Failure • I

Fn

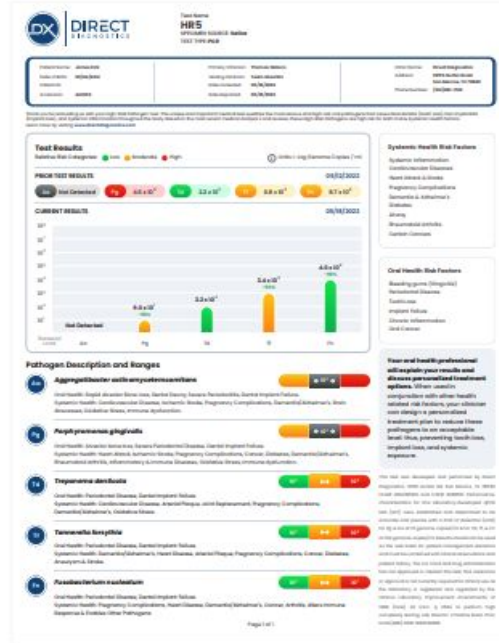


## Fusobacterium

- Alzheimer's • Der
- Cancer • Implant
- Birth Weight, Prete
- Allows Pg and other high

## Why These 5?

- ✓ Periodontal disease starts as an asymptomatic sub-clinical disease.
- ✓ Oral is systemic. These 5 high-risk pathogens enter the bloodstream by crossing the epithelial and endothelial linings throughout the body.
- ✓ 3 of the 5 high risk pathogens are resistant to traditional treatments and products.
- ✓ These 5 control the entire oral microbial community.



HR5<sup>®</sup>  
HIGH RISK PATHOGEN TEST



Aa

Aggregatibacter  
actinomycetemcomitans



Pg

Porphyromonas  
gingivalis



Td

Treponema  
denticola



Tf

Tannerella  
forsythia



Fn

Fusobacterium  
nucleatum

# Get the 5 high risk anaerobic pathogens

# Why these 5...

1. Control the oral microbial community: gene transfer, etc.
2. Most destructive in oral cavity: alveolar bone loss
3. They are systemic: cross epithelium & endothelium
4. Resistant species: to treatment & products



This is typical nitric oxide- sleep disorder breathing  
- Is this acceptable and willing to be at goal?

## 1. Source of infection:

- a. Airway
  - i. [Nitric oxide test](#)

## 2. Resistant strains: Yes

- a. SRP: Tf

## 3. Systemic antibiotics: Timing

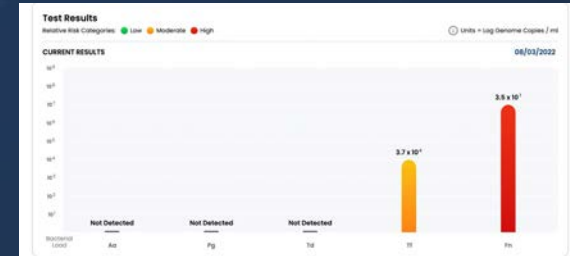
- a. Phase 1: airway, Nitric Ox-NO2U, or berkley Life
- b. Phase 2: repopulation
- c. Phase 3: antibiotics \* if needed

## 4. Help healing? Yes

- a. StellaLife

## 5. Repopulation: Yes

- a. Tf: Pro Biora Pro or Blis-Burst / BioGaia, etc: one or other (never both)
- b. GI: Probiotic / GI Prevent Protocol





# Plaques

CIMT & Cleerly

©Gina Pritchard, DNP, RN, CNS, ACNP

[directdiagnostics.com](https://directdiagnostics.com)



DIRECT  
DIAGNOSTICS

# CIMT

Patient Age	59	Patient IMT	0.79 mm
Arterial Age	64	Normal IMT	<.50 mm

## CV Event Risk

All measurements in mm

Test Criteria:	Normal	Moderate	High	Last Visit*	Alert Value*
Early Event Risk**			4.2		2
Average CCA Mean IMT		0.79			0.73
Average CCA Max Region		0.92			0.75
Plaque Burden**			10.9		

## Comments:

The following values are the largest intima-media thickness (IMT) measurements found in each carotid artery segment. Any measurement is characterized as being: **S = Soft**; **H = Heterogeneous**; or **E = Echogenic** (includes mineral deposits like

**Right CCA .9; Bulb 4.2 H; Internal Carotid 2.6 H**

**Left CCA .9; Bulb 2.8 H; Internal Carotid 1.3 H**

**Doppler was used bilaterally.**

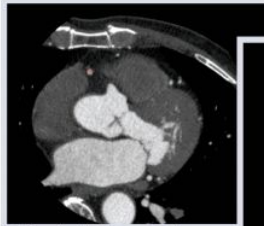
# Cleerly

## Know The Problem: Coronary Artery Disease

### How do you differentiate “higher risk” plaques from potentially more “stable” plaques?

Heart specialists know that there is no single way to determine which plaque is going to cause a heart attack. But findings that may increase the risk of a heart attack can be visualized using advanced imaging with the Cleerly analyses.

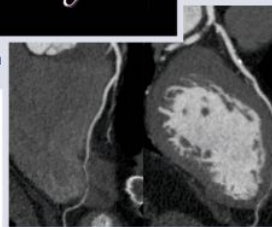
Coronary CT angiography, or advanced “cat scans” of the heart arteries, are the most accurate non-invasive method to quantify and characterize atherosclerosis (plaques). It is the test of choice for evaluation of suspected coronary artery disease in many professional societal guidelines.<sup>5,6,7</sup>



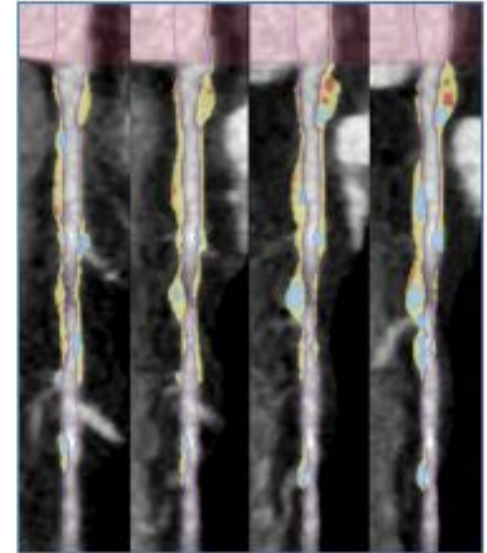
The figure above illustrates a cross-section of the heart.



The figure above depicts coronary arteries that have been extracted and illustrated in 3D.

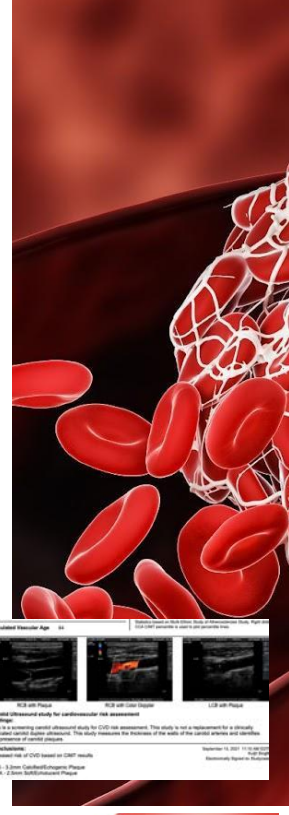
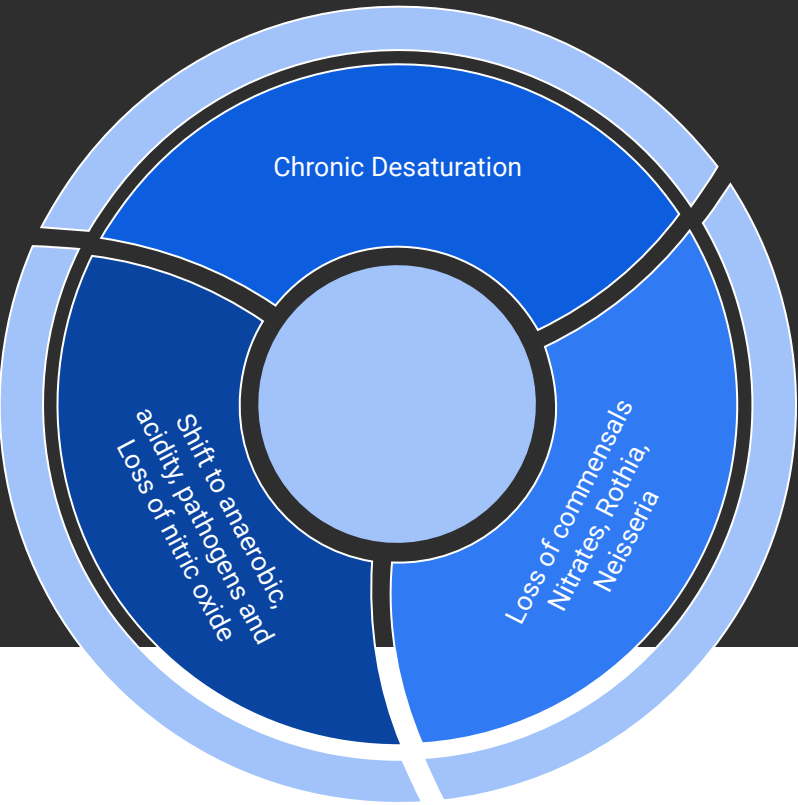


The figure above illustrates 2 images of the same coronary artery in the heart depicted in different views.





# Airway to Pathogens to Plaque



---

Discover what the triple threat is and how you can know if you have a problem lurking in one or more of these top contributors to health and longevity (or disease and early death). Understand the tests and tactics needed to discover your problem areas and begin to eliminate the threat.

# The Triple Threat

Airway/SDB

Oral Pathogens

(Low N.O./Sleep QQ)

Leads to Plaque

Home Sleep Study Watch Pat

HR5 high risk pathogens

Nitric Oxide

Wearable Technology

Oura Ring

Sleep

Latency, Total, Deep REM

O2

Breathing Regularity

Readiness

Resting Heart Rate

HRV

Body Temperature



The background of the slide is a microscopic view of blood, showing numerous red blood cells (erythrocytes) in various shades of red and pink. The cells are biconcave and appear to be floating in a fluid medium. The lighting creates a sense of depth, with some cells in sharp focus and others blurred in the background.

# Thank You!

The Dangerous Mouth & The Dangerous Duo = The Triple Threat