Which Technique for Plaque Evaluation: Stable vs Vulnerable

Near Infrared Spectroscopy

Akiko Maehara, MD

Cardiovascular Research Foundation

Columbia University Medical Center

New York City, NY

CARDIOVASCULAR RESEARCH FOUNDATION
A Passion for Innovation

COLUMBIA UNIVERSITY MEDICAL CENTER
NewYork-Presbyterian
The University Hospital of Columbia and Cornell

Disclosure Statement of Financial Interest

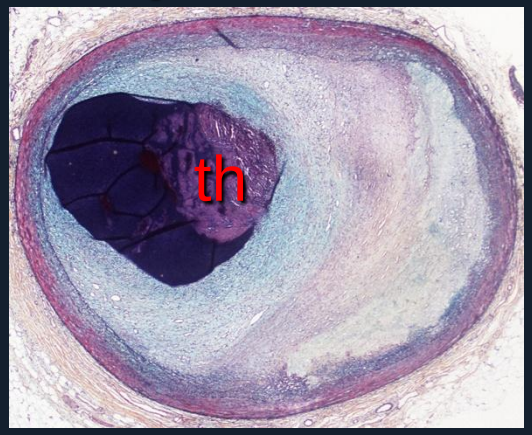
Within the past 12 months, I or my spouse/partner have had a financial Interest /arrangement or affiliation with the organization(s) listed below

Affiliation/Financial Relationship	Company
Grant/ Research Support:	Boston Scientific Corp.
Consultant:	Boston Scientific Corp.
Speaker Fee:	St Jude Medical, Volcano Corporation

Unstable Plaque=Causing Thrombosis

Plaque Rupture

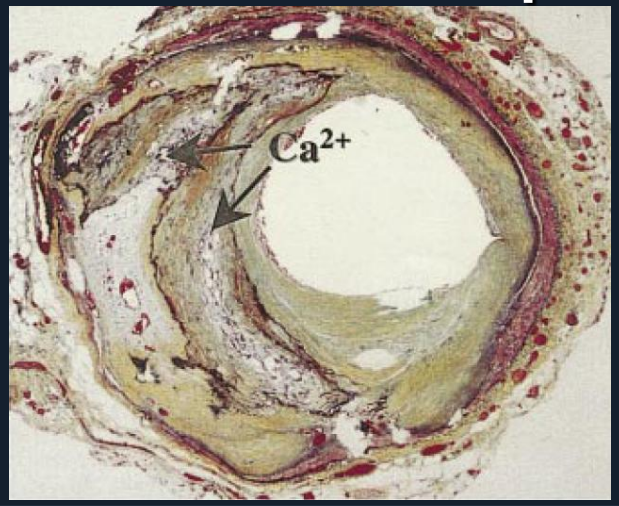
Plaque Erosion



Calcified Nodule

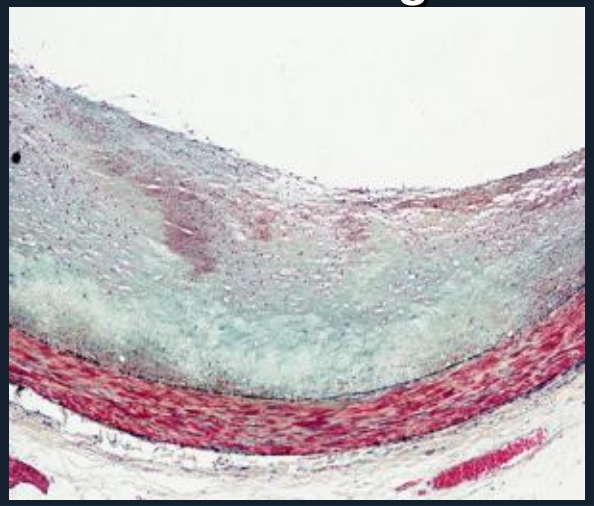
Stable Plaque=Not Causing Thrombosis

Fibrocalcific Plaque

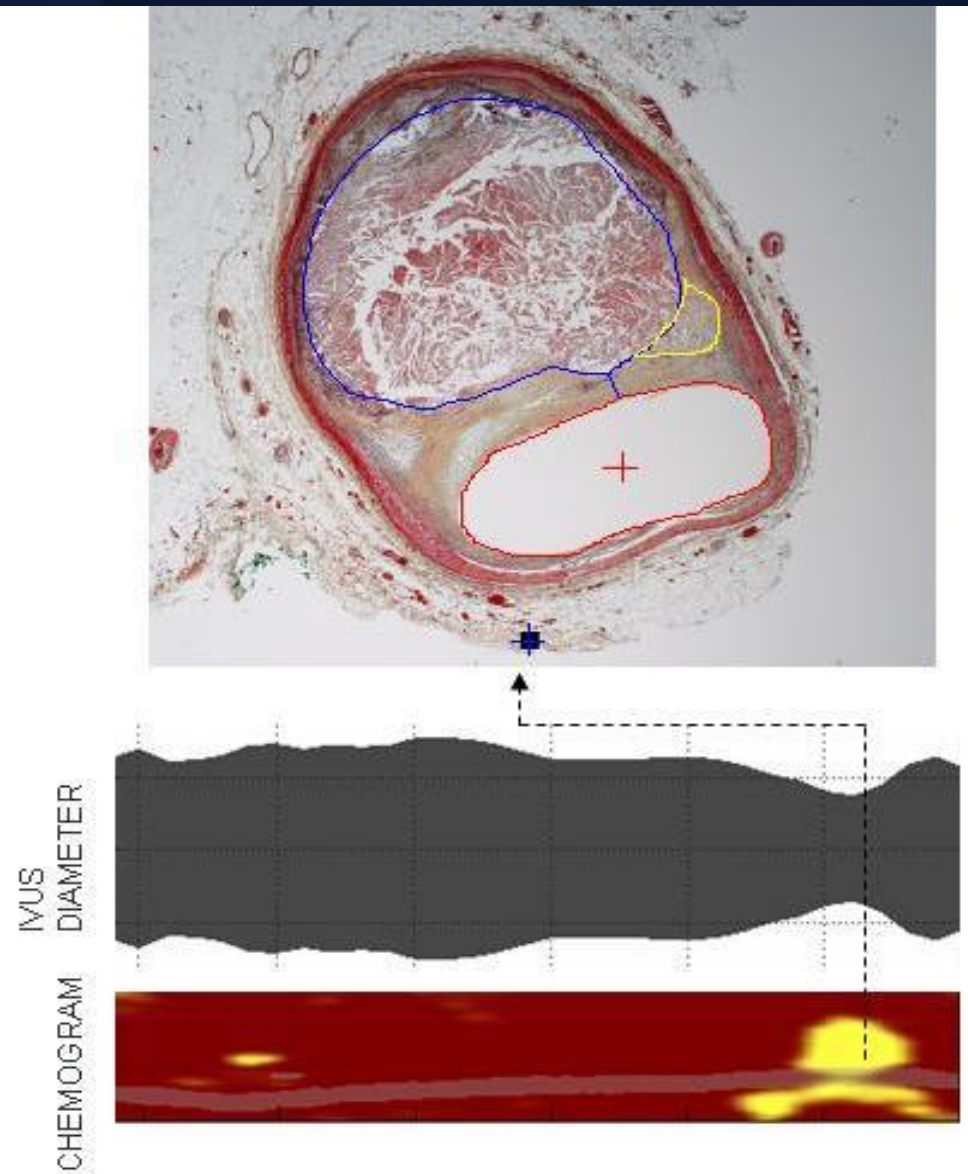


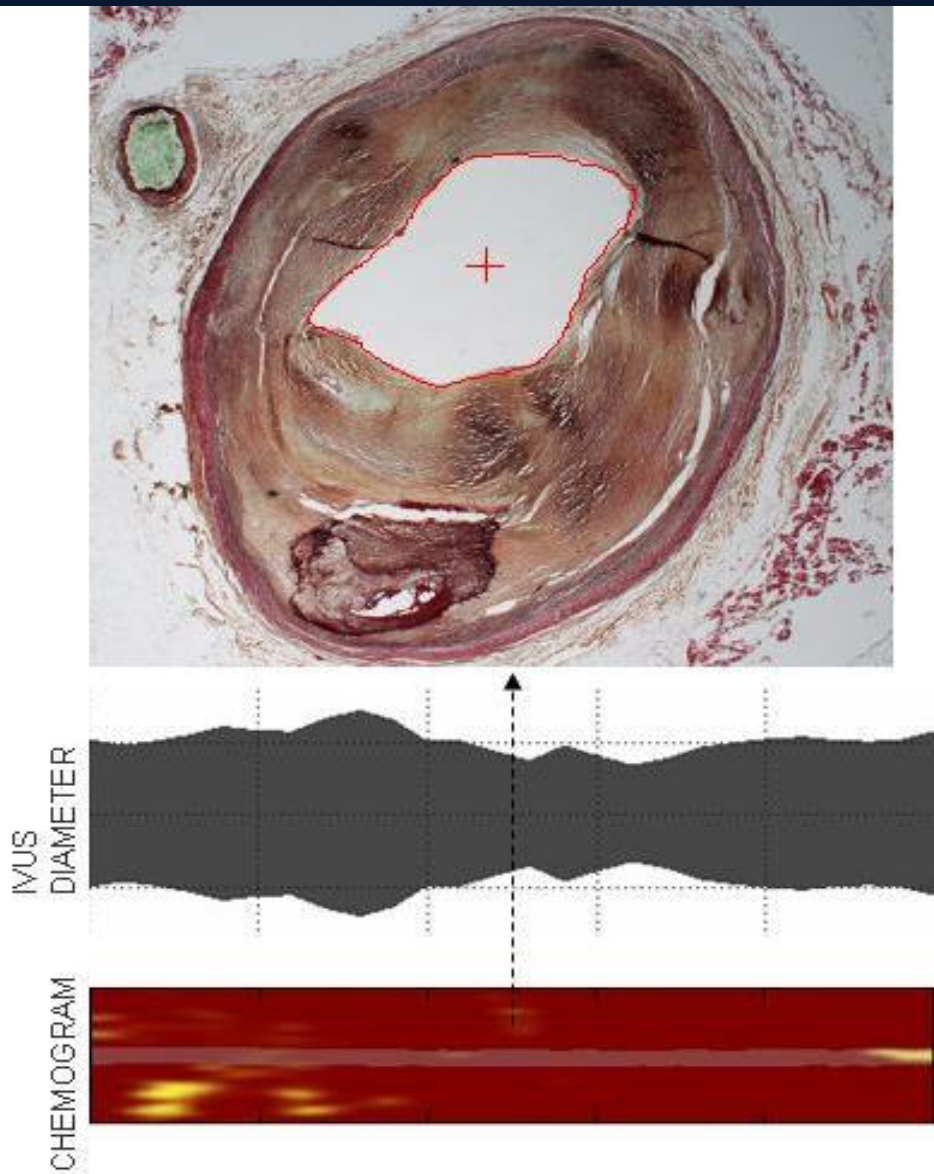
Healed Rupture

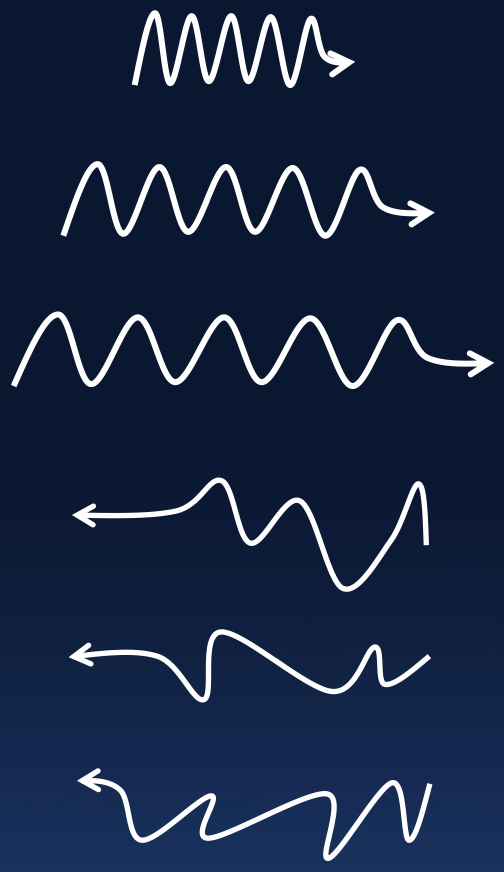
Pathological Intimal Thickening

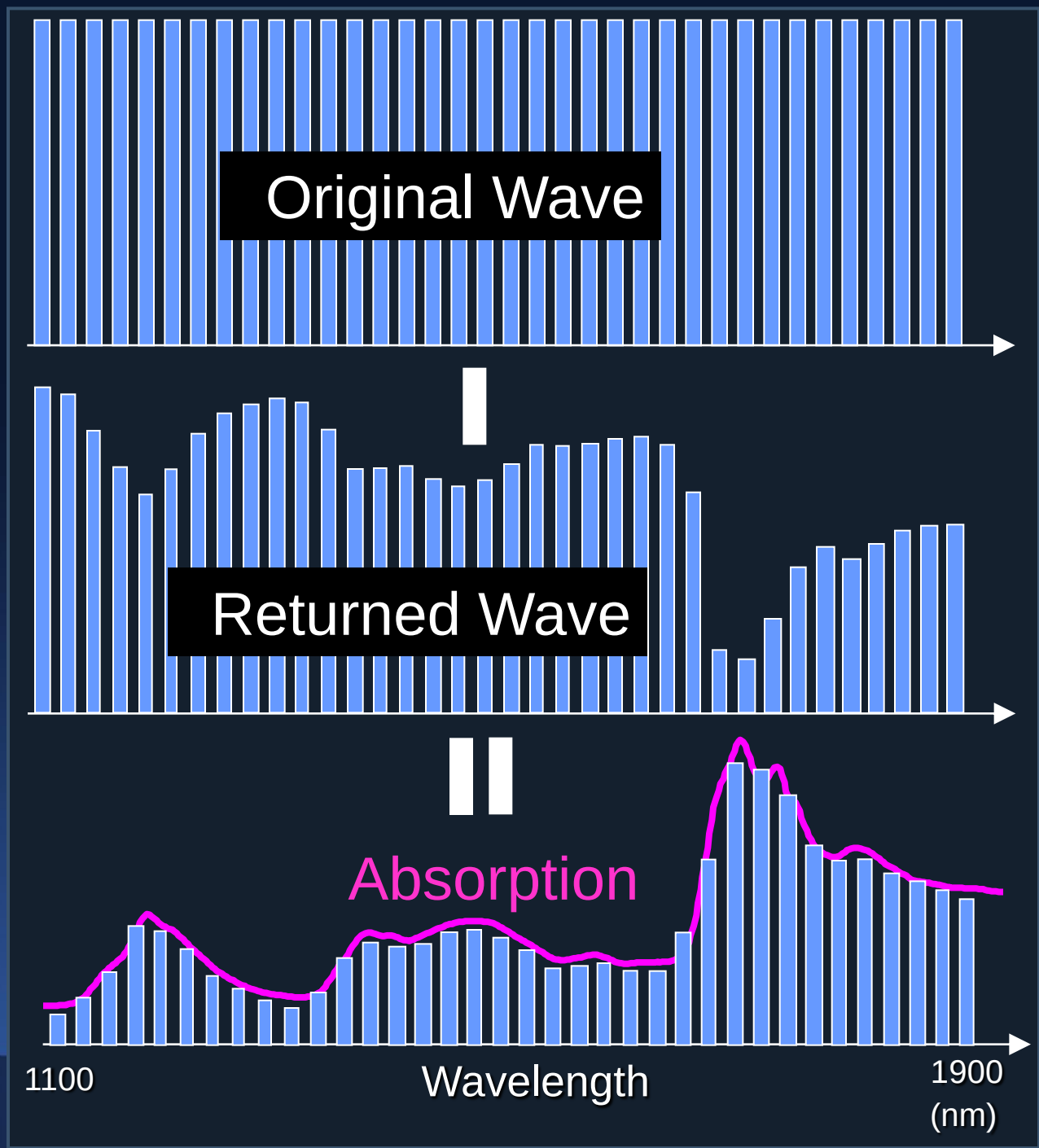


NIR can Distinguish Lipid-rich from Fibrotic Plaques





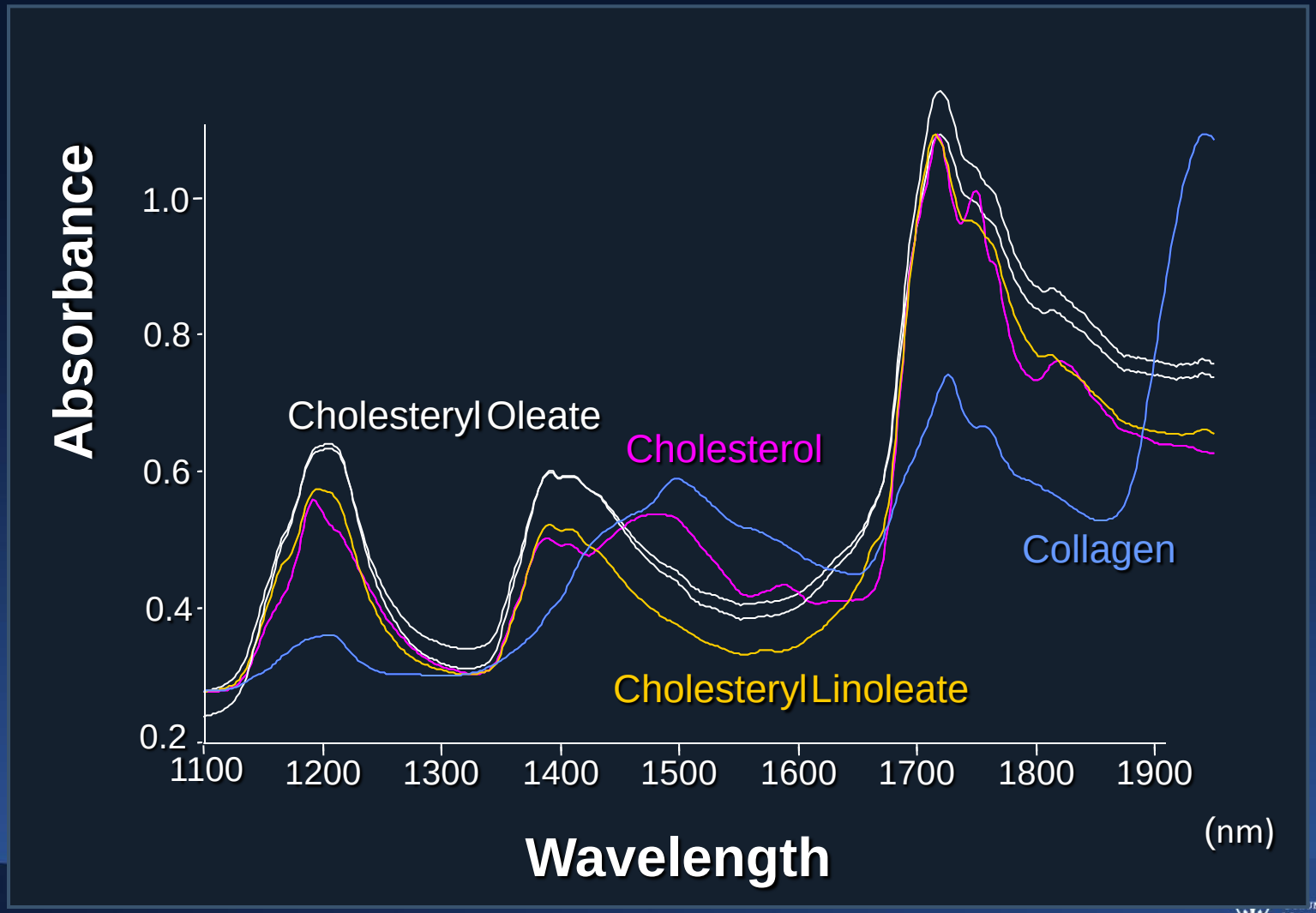




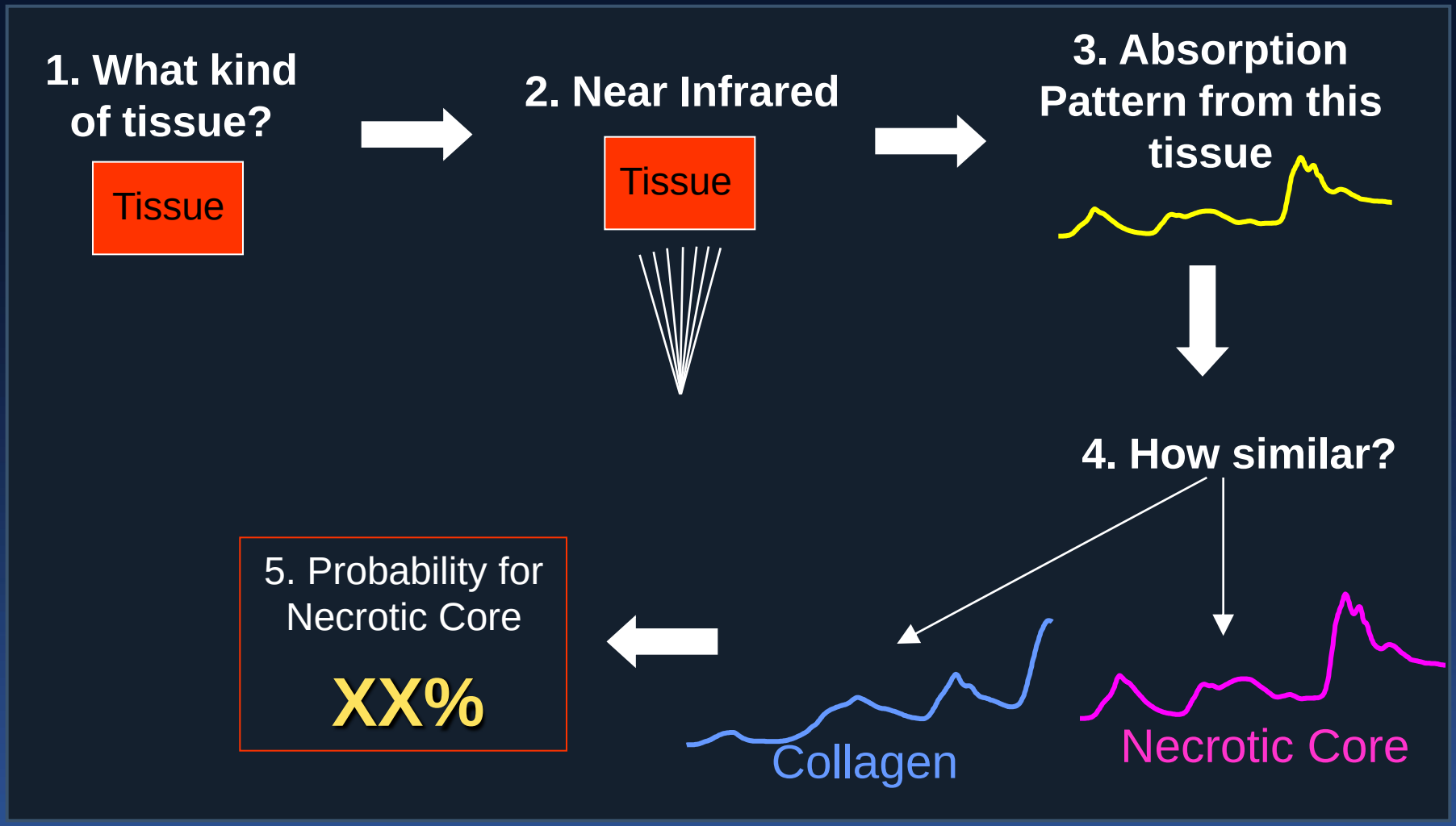
CARDIOVASCULAR RESEARCH FOUNDATION
A Passion for Innovation

NIR Spectroscopy

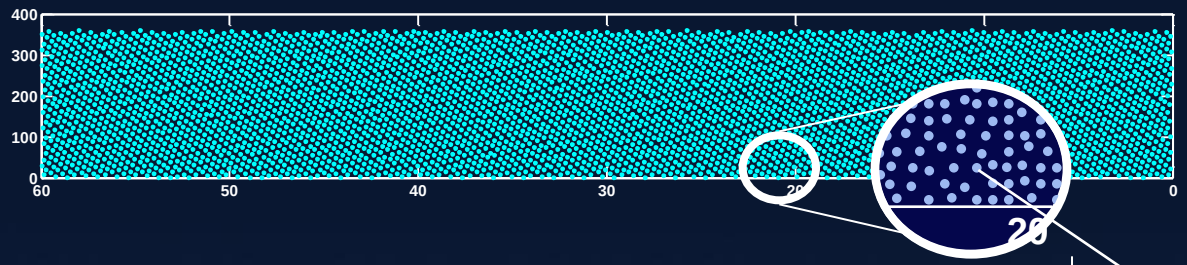
- Necrotic Core>0.2mm thick, >60°, Cap<0.45mm



Process of NIR Spectroscopy

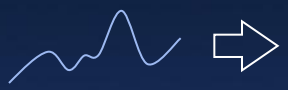


Formation of the Chemogram LCP Prediction



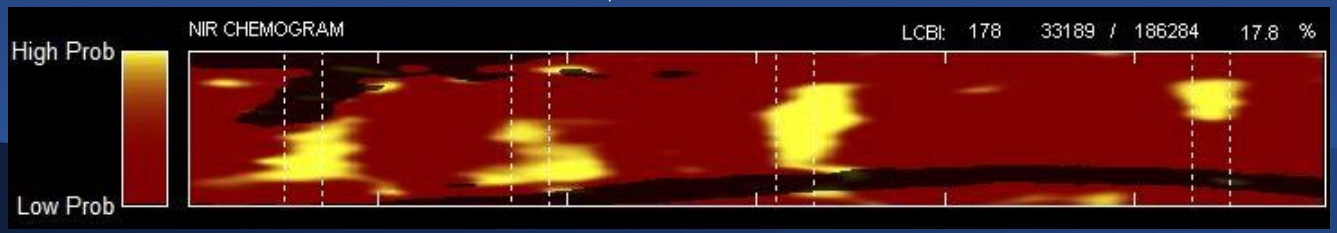
Spectra acquired at discrete pullback and rotation positions

Spectra transformed into posterior probability of LCP

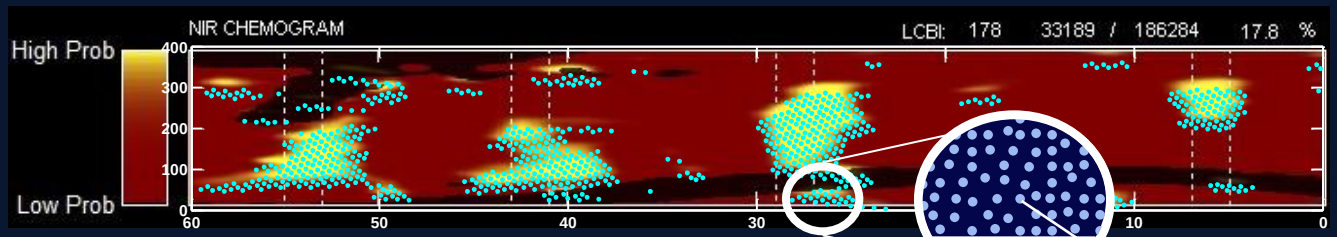


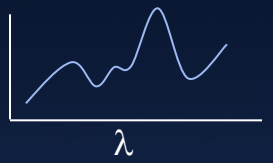
Probability mapped to a color

Pixels formed into an image



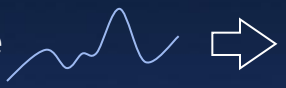
Formation of the Cap Thickness Prediction Image





Spectra acquired at discrete pullback and rotation positions

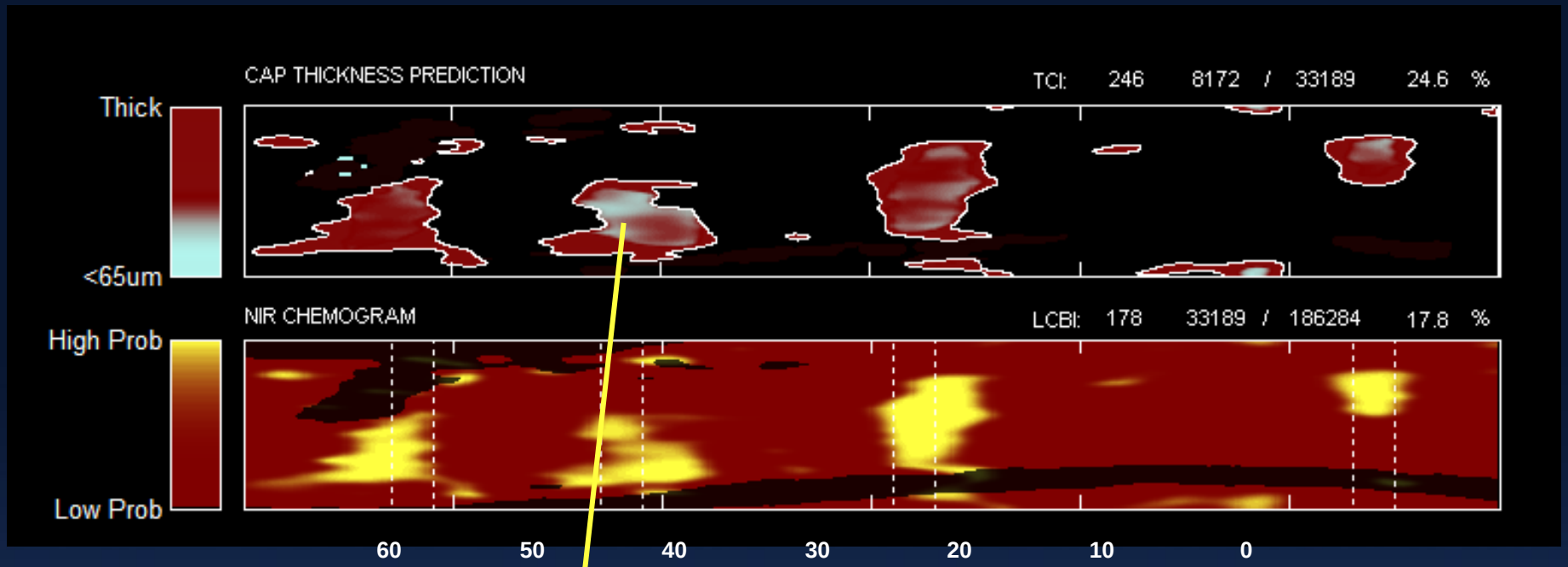
LCP Spectra transformed into posterior probability of thin cap presence



0→1

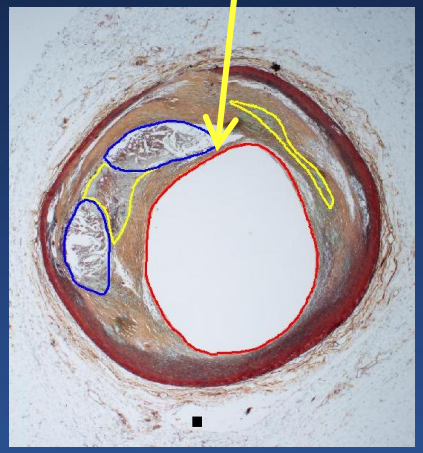
Probability mapped to a color

Pixels formed into an image

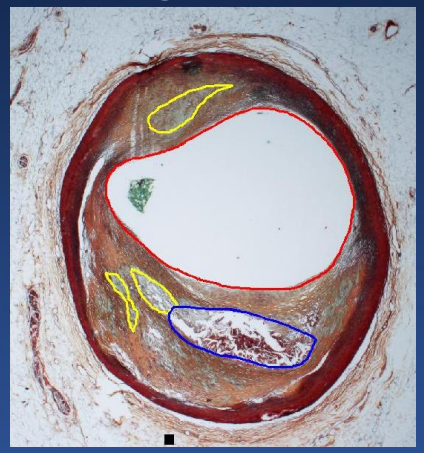


52mm

42mm

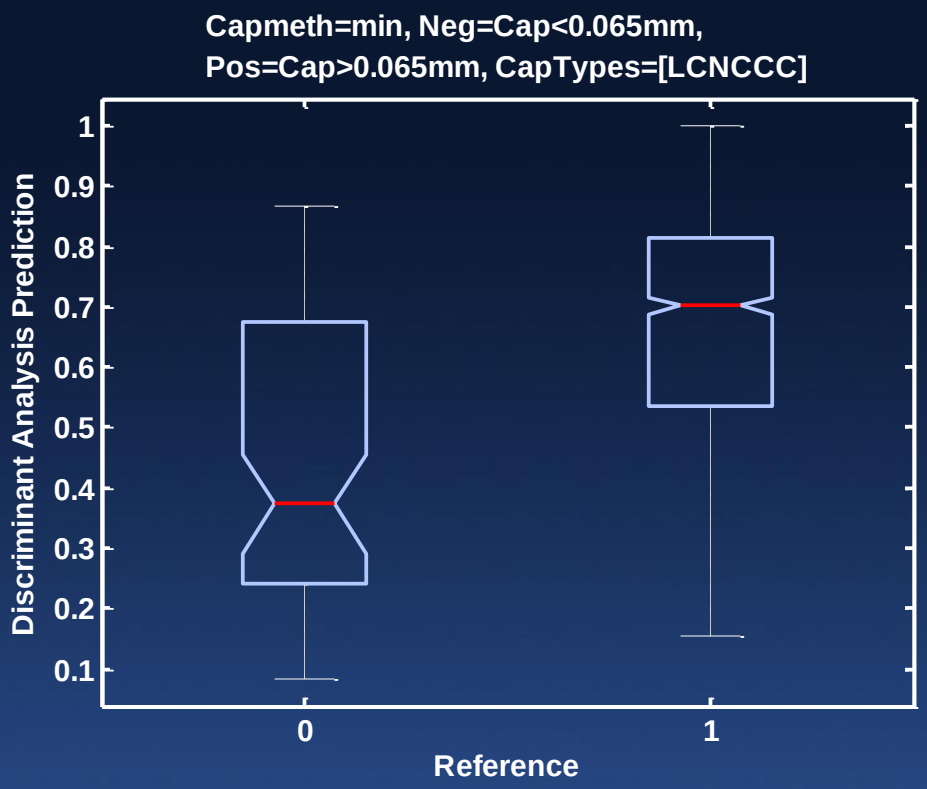


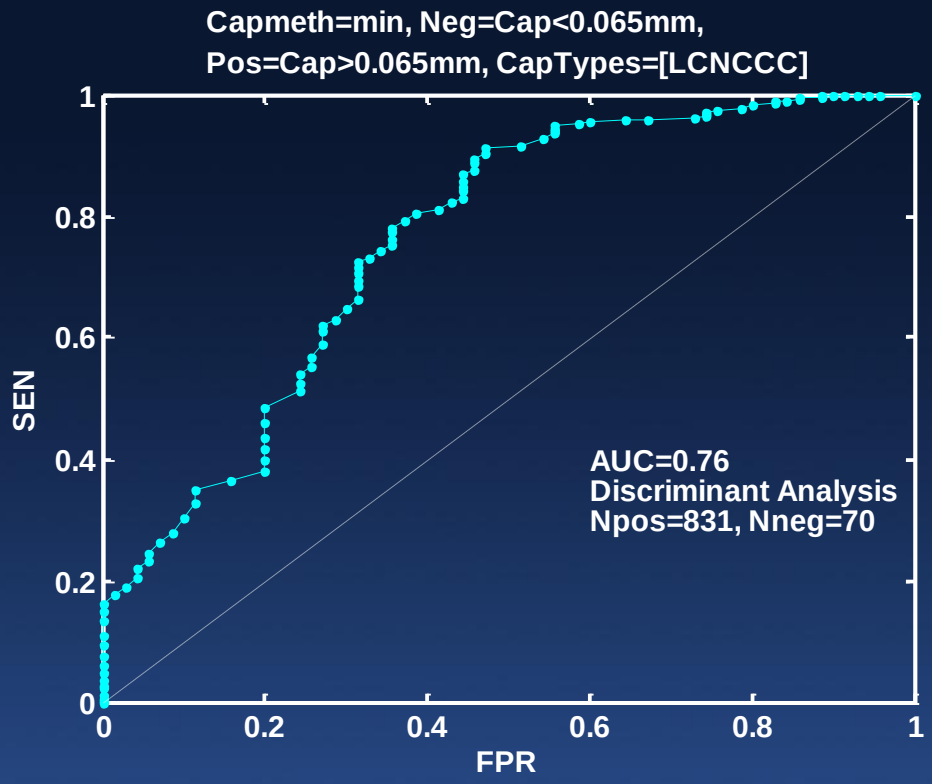
28mm

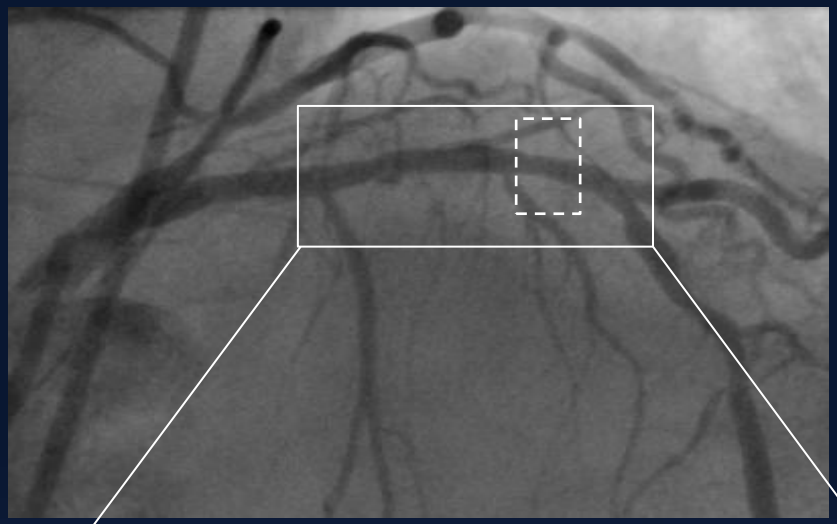


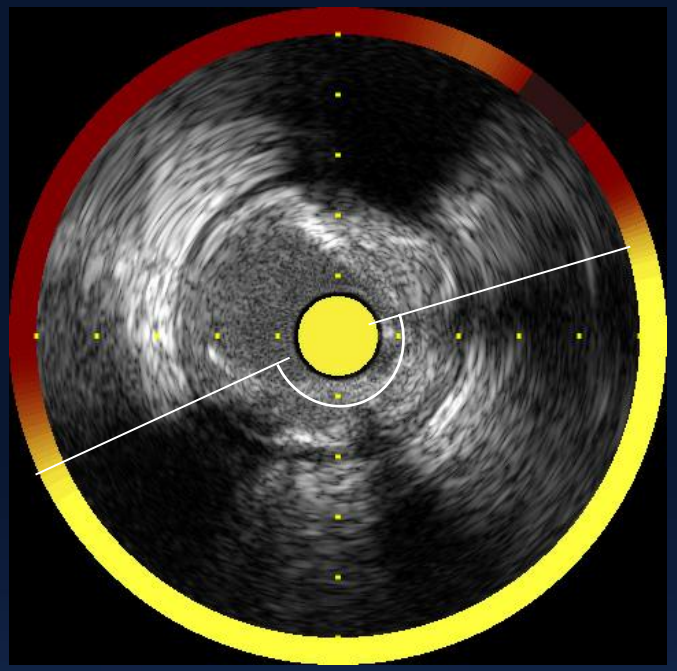
6mm

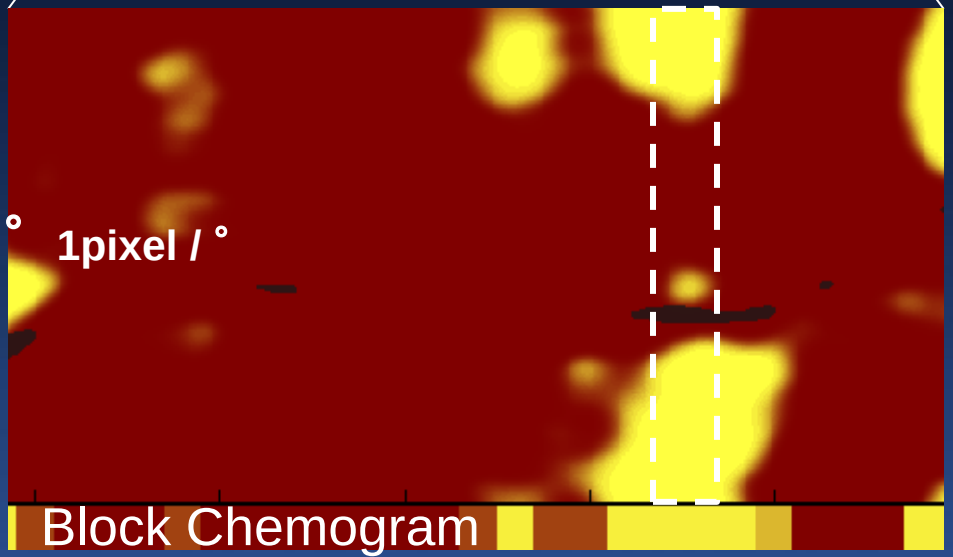
Ability to Predict Thin Cap (<0.065mm)











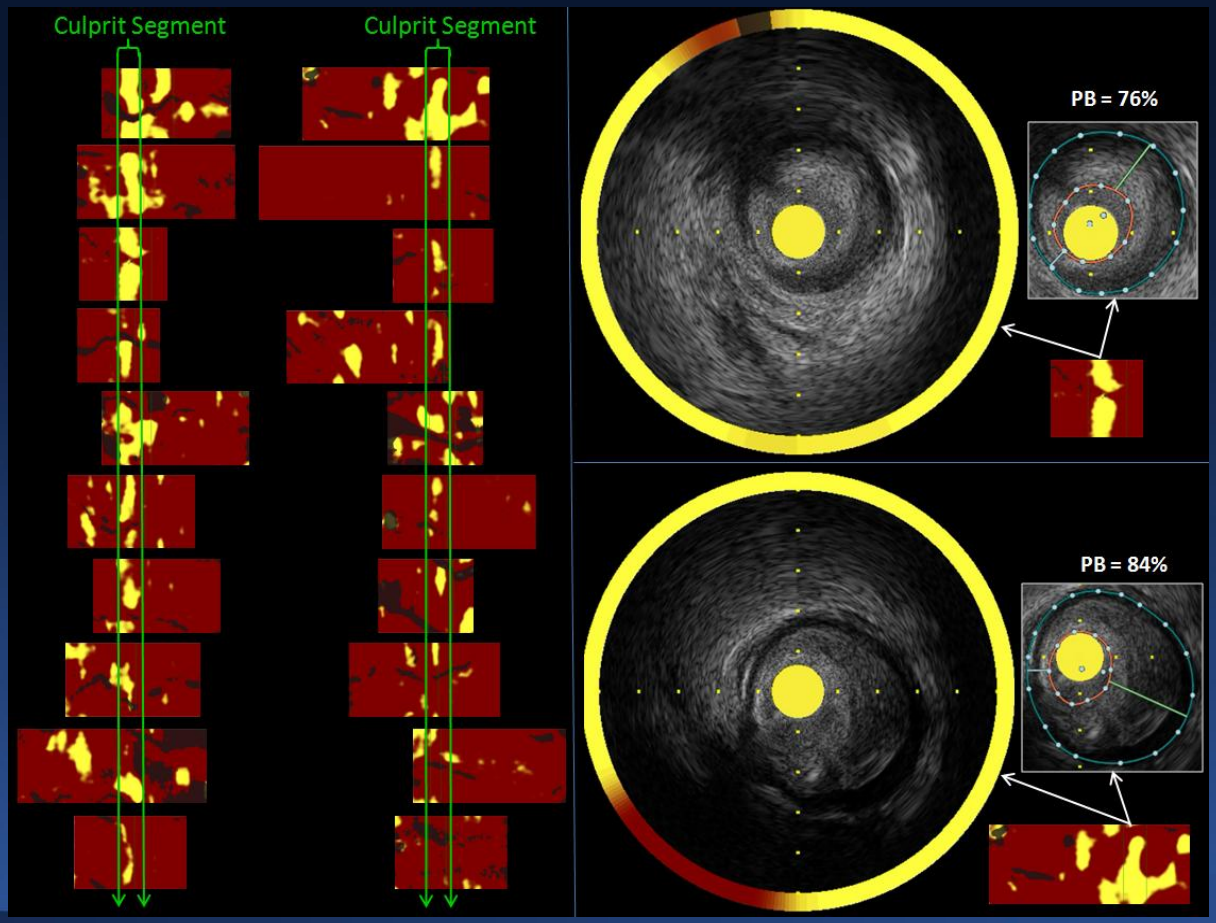
Lipid core burden index

- Lesion LCBI =28499/178923*1000 =**159**
- Max_{4mm} LCBI =8515/13951=**610**

Is there a characteristic signal of lesions that cause STEMI?

Near infrared spectroscopy (InfraReDx) was performed immediately after infarct artery recanalization in 20 pts with STEMI

The NIRS chemograms of all 20 STEMI pts. The culprit segments contain LCP in 19 cases (95%), all with large plaque burden.



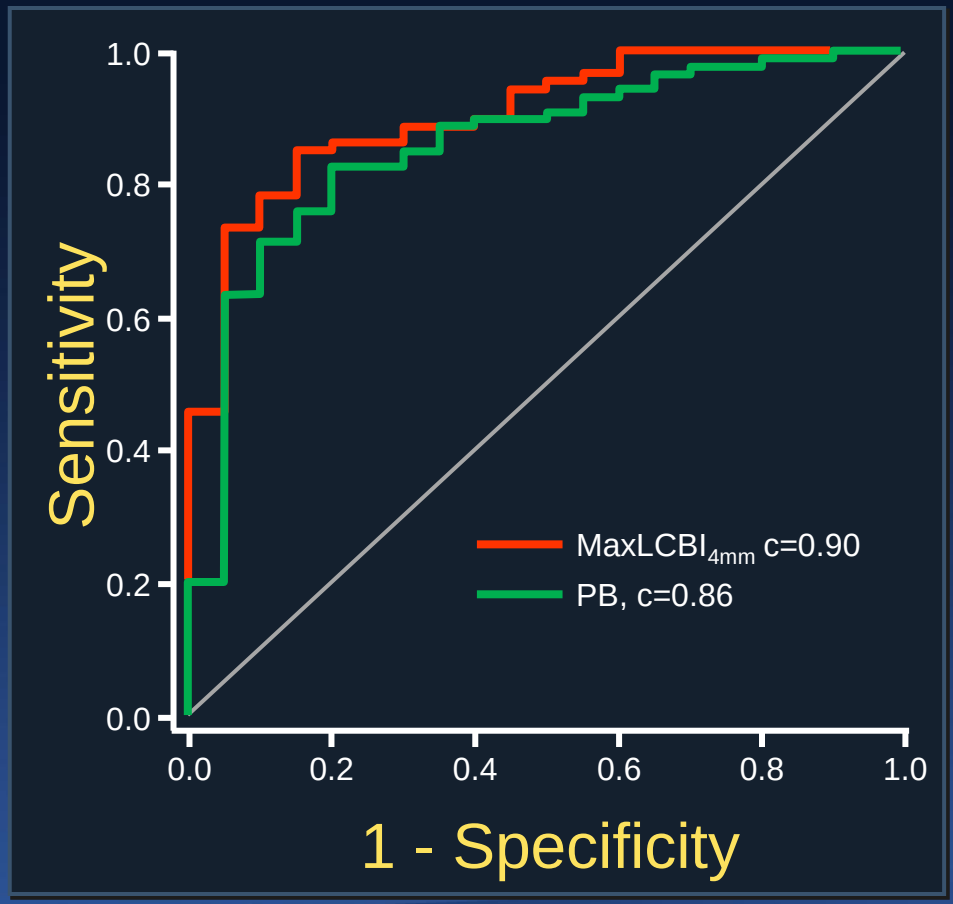
Madder RD. JACC Interv 2013

Is there a characteristic signal of lesions that cause STEMI?

Near infrared spectroscopy (InfraReDx) was performed immediately after infarct artery recanalization in 20 pts with STEMI

Ability of NIRS ($maxLCBI_{4mm}$) and IVUS (plaque burden and calcification) to distinguish the culprit segment from non-culprit segments of the STEMI culprit vessel:

- AUC for $maxLCBI_{4mm}$ = 0.90
- AUC for plaque burden = 0.86



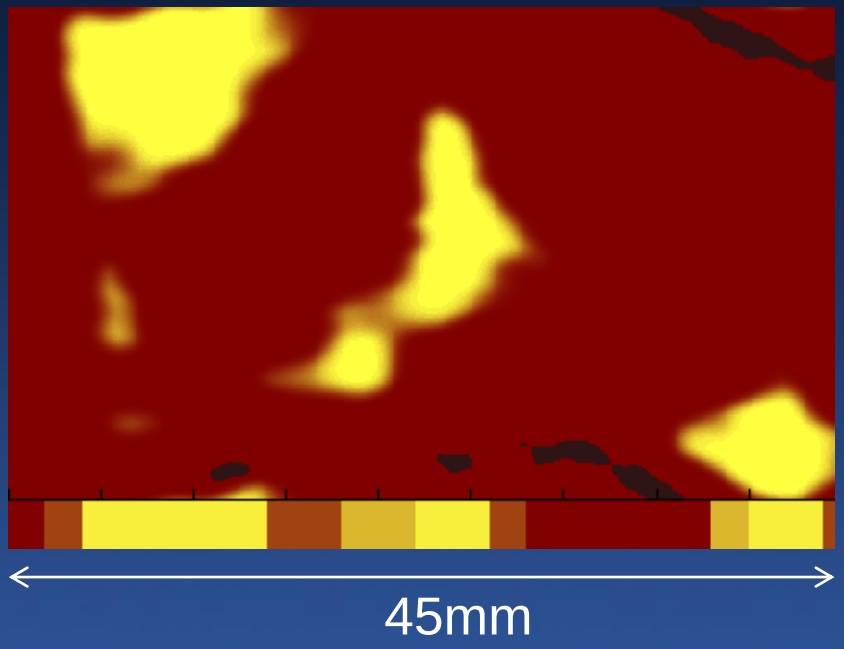
Madder RD. JACC Interv 2013

CARDIOVASCULAR RESEARCH FOUNDATION
A Passion for Innovation
COLUMBIA UNIVERSITY MEDICAL CENTER
NewYork-Presbyterian
The University Hospital of Columbia and Cornell

Relationship between Lipid Rich Plaque detected by NIRS and Outcomes

- Prospective Single Center Study, 206 patients (ACS47%)
- Primary Endpoint: Composite of all-cause mortality, non-fatal ACS, stroke and unplanned PCI during one-year FU
- >40mm non culprit segment of NIRS

Lipid Core Burden Index (LCBI)=188



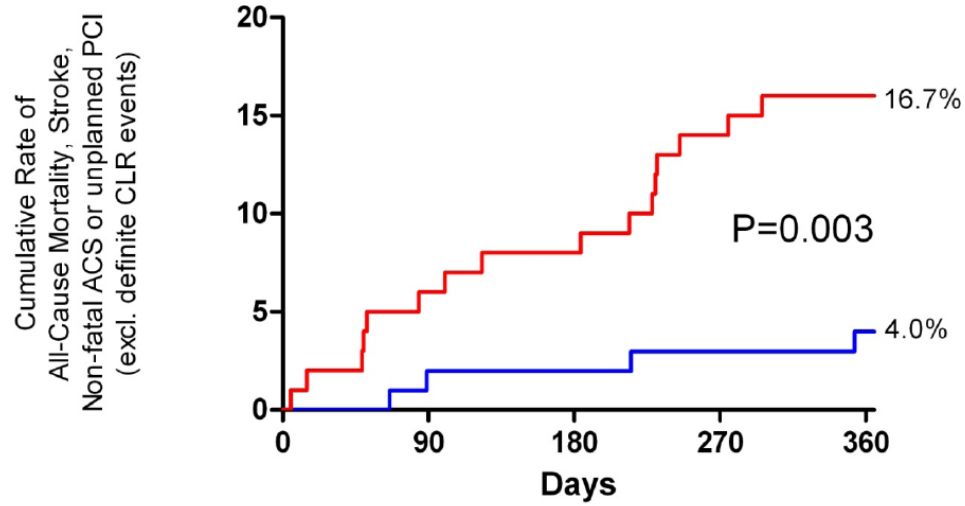
Oemrawsingh RM et al, *ESC2003*

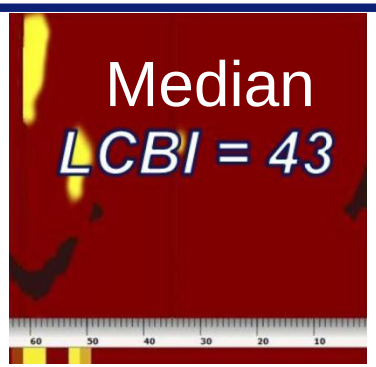
CARDIOVASCULAR RESEARCH FOUNDATION
A Passion for Innovation
COLUMBIA UNIVERSITY MEDICAL CENTER
NewYork-Presbyterian
The University Hospital of Columbia and Cornell

Relationship between Lipidic Plaque detected by NIRS and Outcomes

Primary endpoint

Erasmus MC



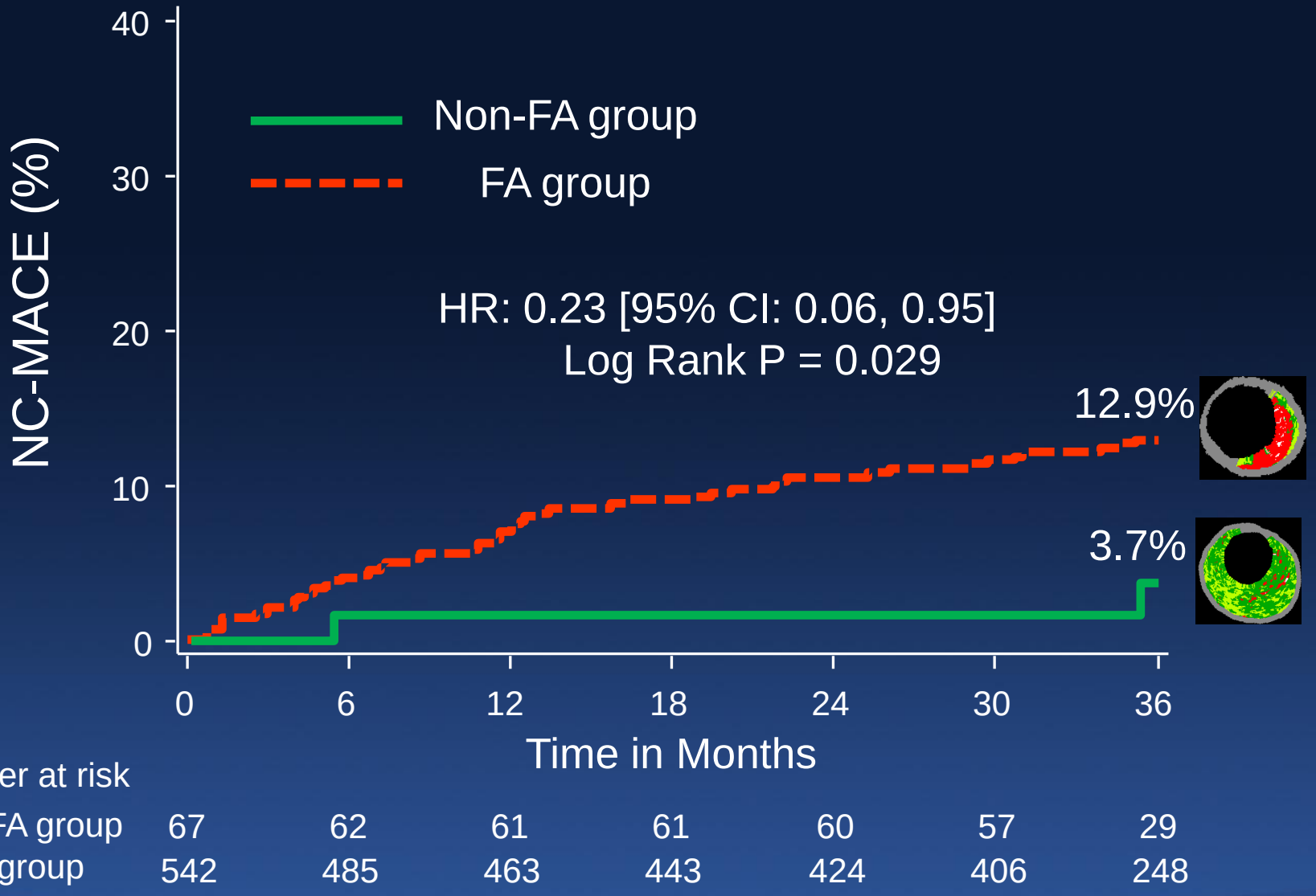


LCBI ≥ median

LCBI < median

Adjusted HR: **4.04** 95% CI: 1.3-12.3 P=0.01

PROSPECT: Non-FA Lesions



Number at risk

Non-FA group	67	62	61	61	60	57	29
FA group	542	485	463	443	424	406	248

CARDIOVASCULAR RESEARCH FOUNDATION
A Passion for Innovation

Dohi et al, *JACC Img* 2013; 6: 908-16.

COLUMBIA UNIVERSITY MEDICAL CENTER
NewYork-Presbyterian
The University Hospital of Columbia and Cornell

Two/Three Vessel CAD
(n= 87)

After stenting the target vessel
The non-target lesion underwent FFR

FFR≤0.8 ➔IVUS, NIRS

Randomized

Standard	Aggressive
n = 43	n = 44
Continue statin the patient was taking	Rosuvastatin 40 mg daily
Dual antiplatelet therapy for 1 year	Dual antiplatelet therapy for 1 year

Follow up Cath (6-8 weeks)
FFR, IVUS and NIRS repeated.
If FFR ≤0.8, lesion stented and imaging repeated.
If FFR > 0.8 the patient was treated medically.

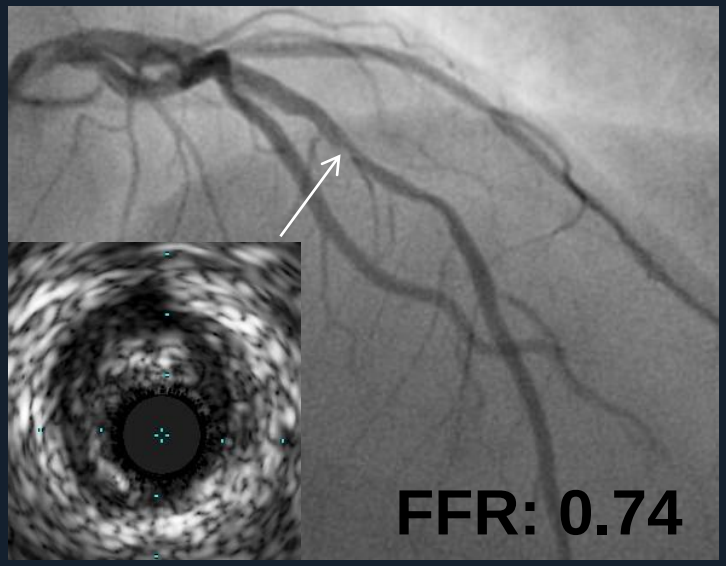
Imaging data analyzed by CRF Core Lab
Data analysis for primary outcome analyzed by MSH independent Core Lab

***Optimal medical therapy for all patients**

Kini A et al. *JACC* 2013; 62: 21-9.

Case Example

Baseline

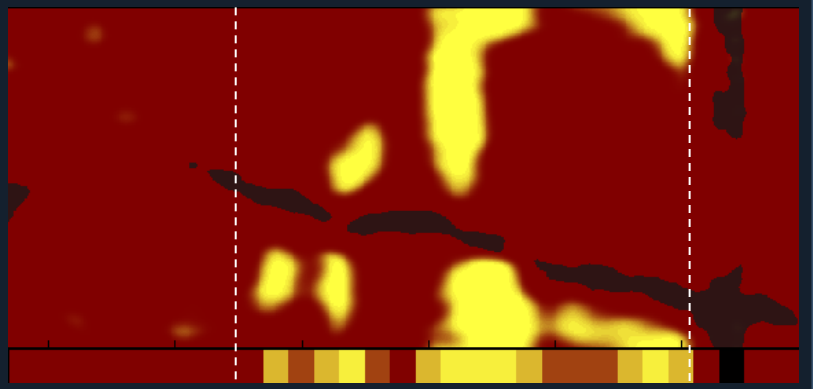


Plaque Area
5.6mm^2

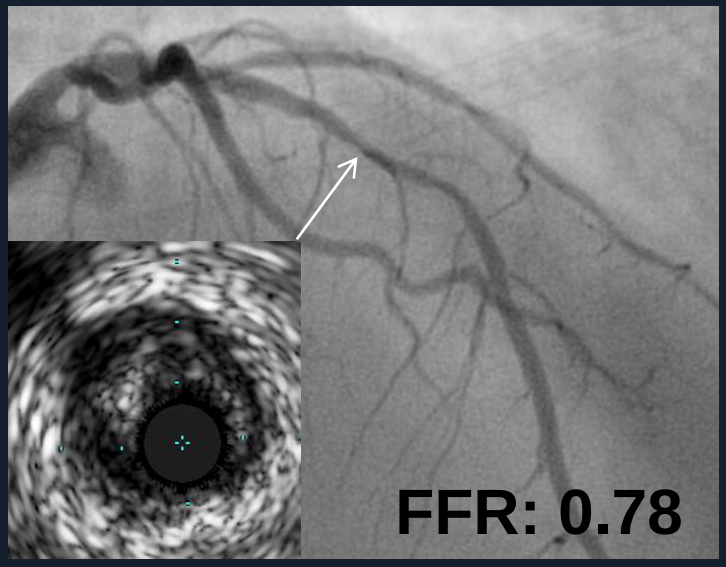
Lesion LCBI: 259

Max10mm LCBI: 511

Max4mm LCBI: 802



Follow-up

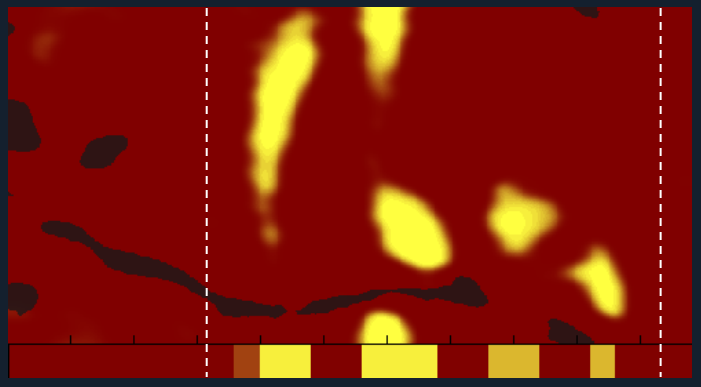


Plaque Area
5.5mm^2

Lesion LCBI: 177

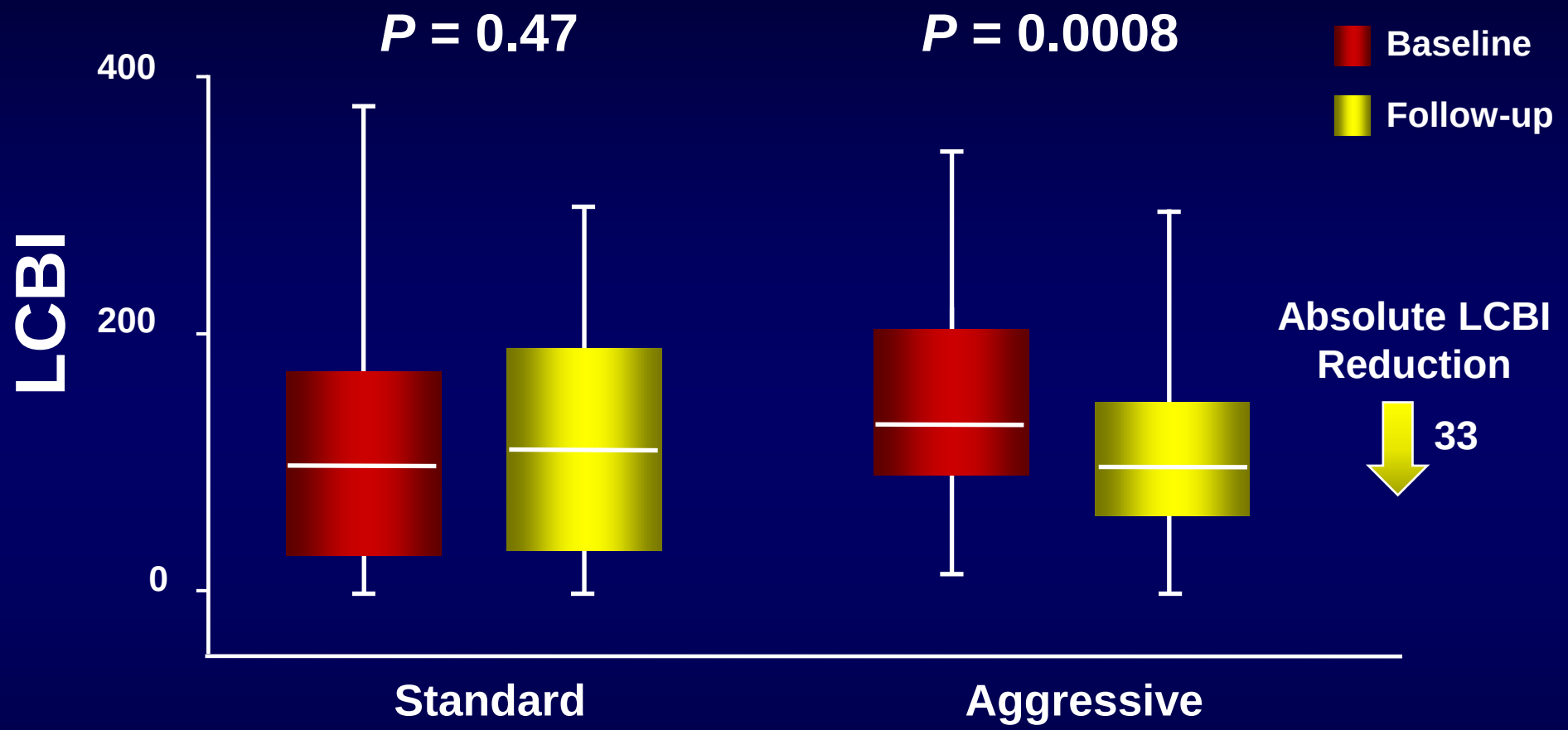
Max10mm LCBI: 289

Max4mm LCBI: 474



Kini A et al. *JACC* 2013; 62: 21-9.

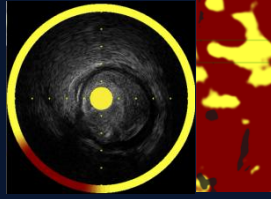
Paired Analysis – Lesion LCBI



Variable	Standard (n = 43)	Aggressive (n = 44)	*P*
Percent atheroma volume	0.26%	0.24%	0.98

Kini A et al. *JACC* 2013; 62: 21-9.

PROSPECT II Study



900 pts with ACS at up to 20 hospitals in Sweden, Denmark and Norway (SCAAR)

NSTEMI or STEMI >12º

IVUS + NIRS (blinded) performed in culprit vessel(s)

Successful PCI of all intended lesions (by angio ±FFR/iFR)

↓

Formally enrolled

↓

3-vessel imaging post PCI

Culprit artery, followed by non-culprit arteries

Angiography (QCA of entire coronary tree)

IVUS + NIRS (blinded) (prox 6-8 cm of each coronary artery)

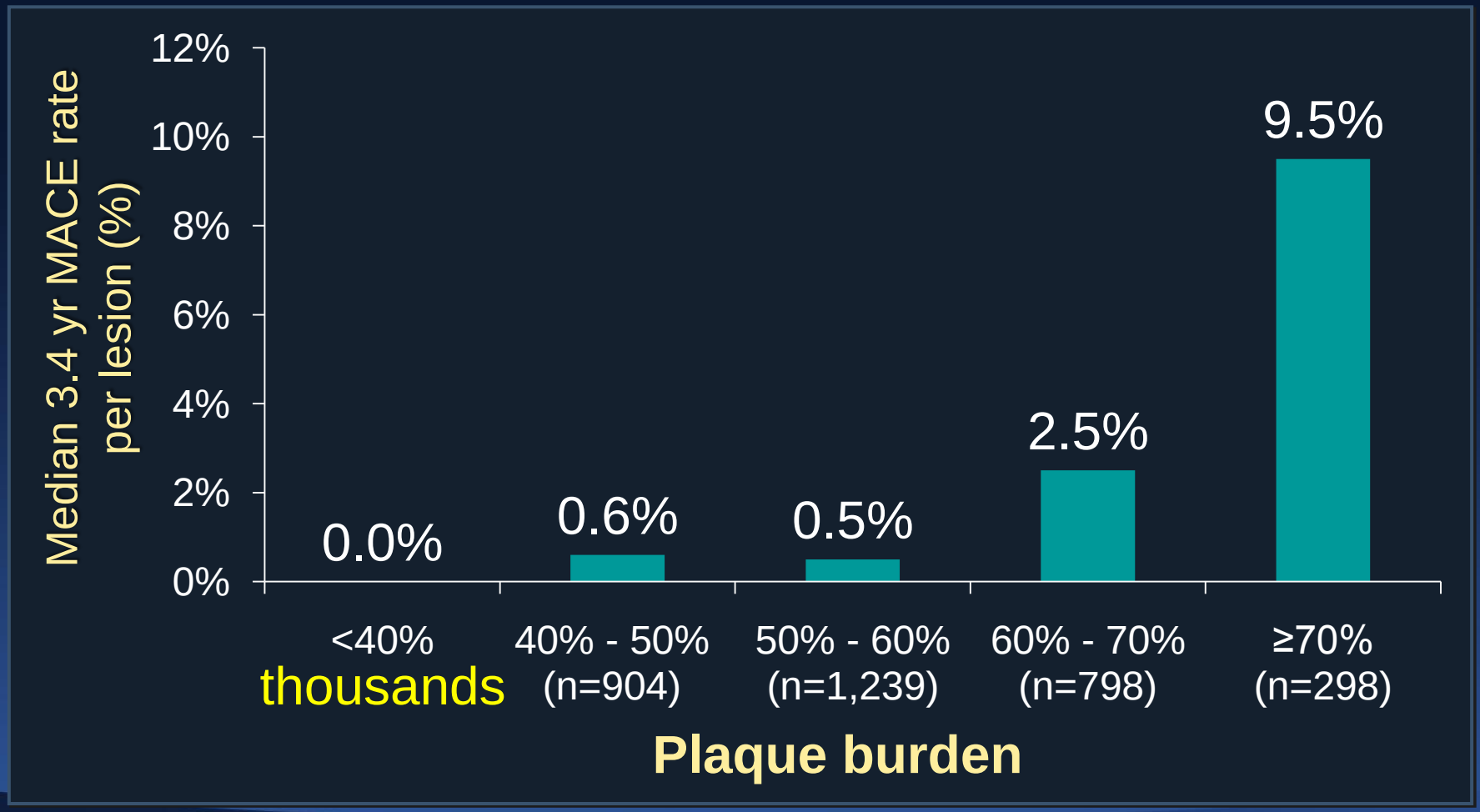
↓

CARDIOVASCULAR RESEARCH FOUNDATION
A Passion for Innovation

COLUMBIA UNIVERSITY MEDICAL CENTER
NewYork-Presbyterian
The University Hospital of Columbia and Cornell

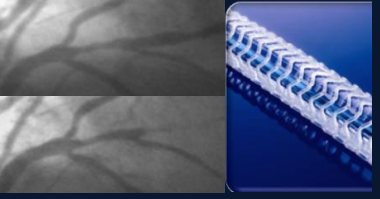
PROSPECT: Correlates of Non-Culprit Lesion Related Events

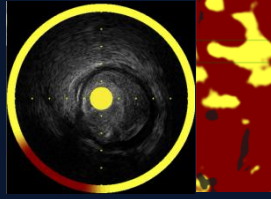
Impact of plaque burden



McPherson JA et al. JACC Img 2012;5:S76–85

CARDIOVASCULAR RESEARCH FOUNDATION
A Passion for Innovation
COLUMBIA UNIVERSITY MEDICAL CENTER
NewYork-Presbyterian
The University Hospital of Columbia and Cornell





PROSPECT II Study

PROSPECT ABSORB RCT

900 pts with ACS after successful PCI

3 vessel IVUS + NIRS (blinded)

≥1 IVUS lesion with ≥70% plaque burden present?

Yes
(N=300)

No
(n=600)

R
2:1

ABSORB BVS + GDMT (N~200)

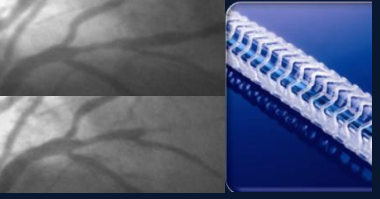
GDMT
(N=100)

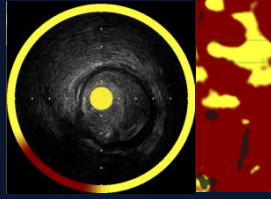
Routine angio/3V IVUS-NIRS FU at 2 years

Clinical FU for ≥3 years

CARDIOVASCULAR RESEARCH FOUNDATION
A Passion for Innovation

COLUMBIA UNIVERSITY MEDICAL CENTER
NewYork-Presbyterian
The University Hospital of Columbia and Cornell





PROSPECT II Study
PROSPECT ABSORB RCT
- Primary endpoints and analysis -

PROSPECT II

Endpoints: Composite MACE (cardiac death, cardiac arrest, MI, or unstable or progressive angina requiring rehosp or revasc) adjudicated to non-culprit lesions

Analysis: Multivariable predictors, including clinical, QCA, IVUS and NIRS (patient and lesion level)

PROSPECT ABSORB

Endpoints and analysis: IVUS MLA at 2 years (superiority, powered); Death, TV-MI, TLR (noninferiority, not powered)

Summary

1. Algorithm was validated for lipid rich plaque (LRP) and show the distribution of LRP.
2. Algorithm for thin cap fibroatheroma seems to have good probability and will be available soon.
3. NIRS shows good reproducibility and robust for evaluation of evolution of LRP.
4. NIRS seems to have high negative predictive value for stable plaque and clinical impact of positive prediction will be answered in PROSPECT2.