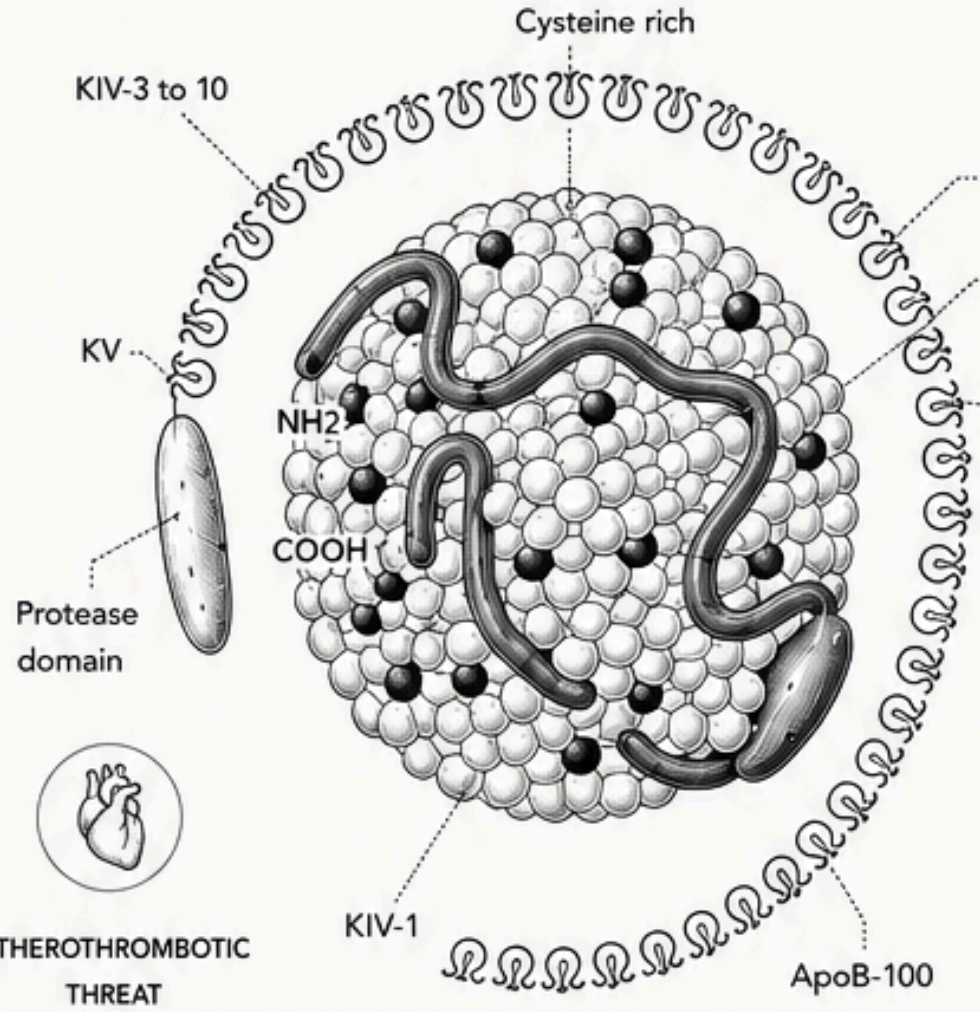
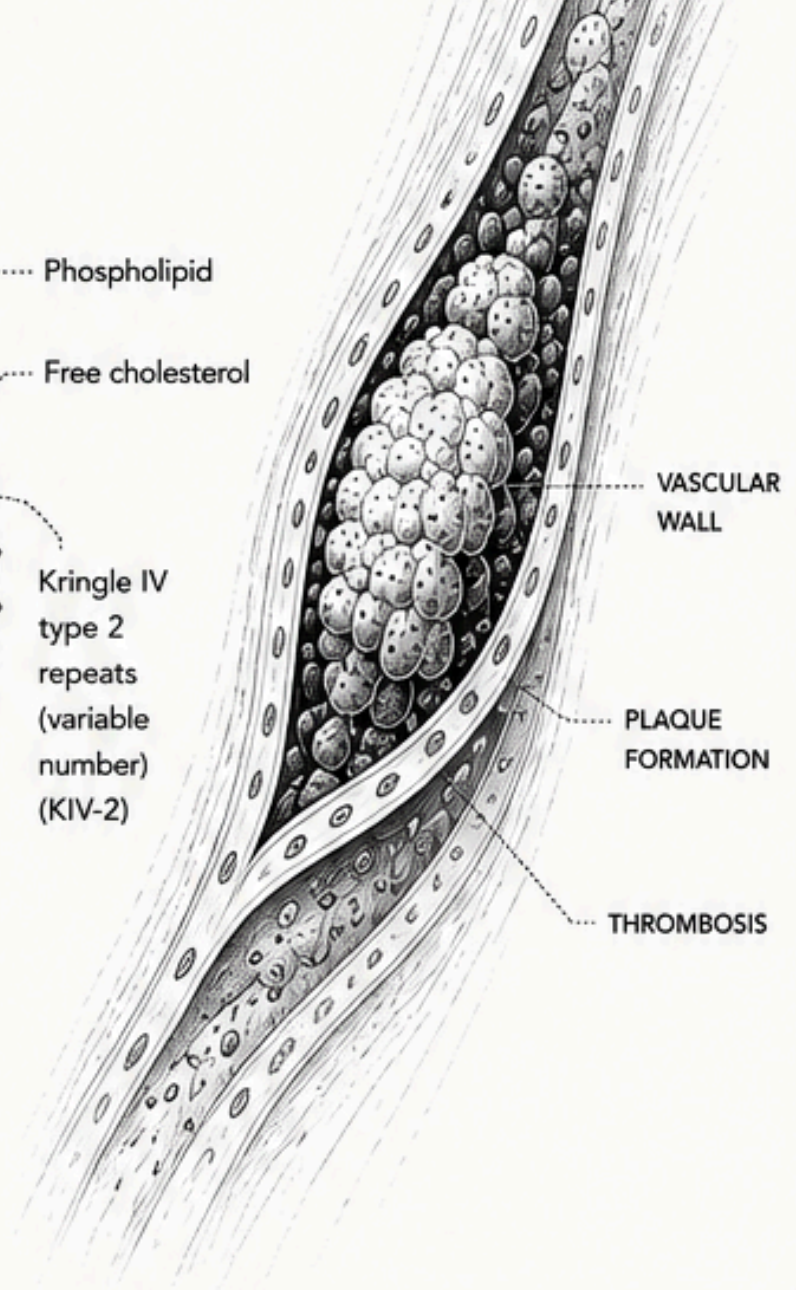
Lp(a): The Hidden Cardiovascular Risk

Normal cholesterol does **not** always mean normal cardiovascular risk.

LIPOPROTEIN(a) STRUCTURE





GENETICALLY DETERMINED

Largely non-modifiable with lifetime exposure

HIDDEN RISK FACTOR

Can be normal lipids, high risk

ATHEROTHROMBOTIC THREAT

Promotes atherosclerosis and thrombosis

Adapted from Cegla J et al, 2019

HOW Lp(a) INCREASES CARDIOVASCULAR RISK

1 GENETICALLY DETERMINED

Lp(a) levels are set by genetics and remain stable throughout life.

2 STRUCTURE

Lp(a) is an LDL-like particle with an additional Apo(a) attached.

3 PRO-ATHEROGENIC & PRO-INFLAMMATORY

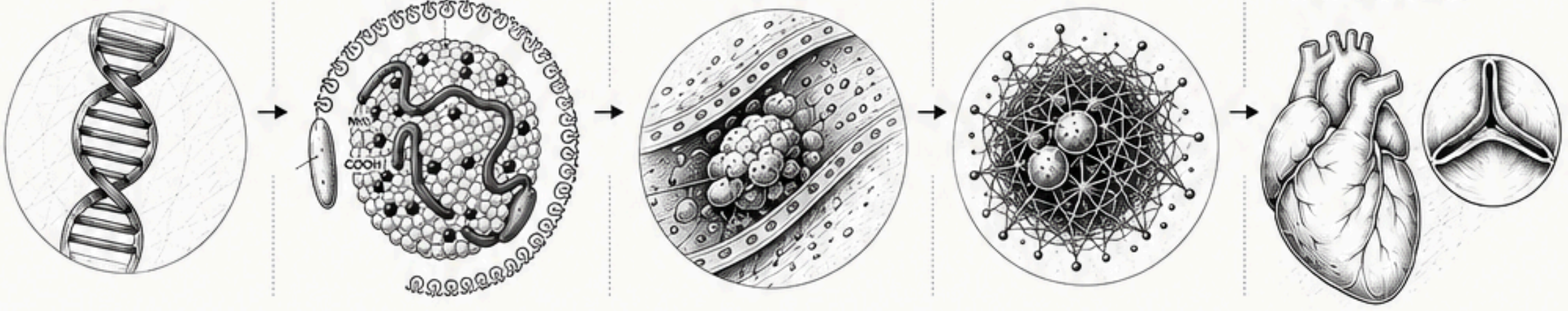
Enhances cholesterol deposition, inflammation and plaque growth.

4 PRO-THROMBOTIC

Interferes with fibrinolysis and promotes clot formation.

5 INCREASED CARDIOVASCULAR RISK

Higher Lp(a) levels are associated with higher risk of ASCVD, stroke and aortic valve stenosis.



INDEPENDENT • CAUSAL • CONTINUOUS RISK

WHEN SHOULD YOU TEST Lp(a)?

PREMATURE CARDIOVASCULAR DISEASE
Men <55 years
Women <65 years

FAMILY HISTORY of early heart disease

FAMILIAL HYPERCHOLESTEROLEMIA (FH)

RECURRENT EVENTS despite optimal lipid-lowering therapy

AOOTIC VALVE DISEASE (calcific stenosis)

Guidance from **HEART UK** and **NEELI** recommends at least one lifetime measurement in most adults – earlier if high risk.

MANAGEMENT STRATEGIES

1. OPTIMISE TRADITIONAL RISK FACTORS

- LDL-C lowering (BP control)
- Smoking cessation
- Diabetes management
- Healthy lifestyle

2. LIPID-LOWERING THERAPIES

- Statins – do not lower Lp(a)
- PCSK9 inhibitors – ↓ Lp(a) ~20–30%
- Inclisiran – modest ↓
- Obicetrapib – ↓ Lp(a) ~57%

3. EMERGING THERAPIES

Antisense oligonucleotides and siRNA therapies in development with reductions up to 80–90% in early trials.