Edexcel International Advanced Level (IAL) Biology Revision Series

1C: Cardiovascular Health and Risk Factors

Topic 1: Molecules, Transport and Health

COMPREHENSIVE HIGH-YIELD LECTURE NOTES & SPEC COMPANION

1. Understanding Cardiovascular Disease (CVD)

Cardiovascular Diseases (CVDs) encompass a broad category of multi-factorial pathologies that impair the structural and functional integrity of the heart, its valvular structures, and the entire systemic peripheral blood vessel network. In affluent global populations, a significant shift toward sedentary recreation (such as prolonged internet use, television watching, and regular eating out) has driven up the incidence of these chronic vascular diseases.

At the center of most clinical forms of CVD is **atherosclerosis**—a progressive, localized disease process affecting intermediate and large-diameter systemic arteries. Atherosclerosis hardens arterial walls and narrows their internal lumens, which drastically restricts downstream blood flow and can lead to myocardial infarctions (heart attacks) or ischemic strokes.

2. The Pathogenesis of Atherosclerosis

Atherosclerosis develops over decades through a complex, multi-stage inflammatory cascade within the arterial wall. This process follows a specific series of pathological steps:

1. **Endothelial Damage:** The single-cell layer lining the arterial lumen (the *endothelium*) is damaged or disrupted. This initial injury is typically caused by constant mechanical shear stress from chronic **high blood pressure (hypertension)** or toxic chemical irritants from tobacco smoke.
2. **Inflammatory Response:** Damage to the endothelium triggers a localized inflammatory defense cascade. The compromised endothelial cells express surface adhesion molecules, recruiting circulating white blood cells (specifically **monocytes**) to the site of injury. These cells migrate through the gaps in the endothelial layer into the underlying tunica intima.
3. **Atheroma Formation (Fatty Streak):** Once inside the inner lining of the artery wall, the accumulated monocytes transform into active macrophages. These macrophages ingest large quantities of modified, oxidized **Low-Density Lipoproteins (LDLs)** circulating in the blood. As they become overloaded with cholesterol lipids, they transform into swollen **foam cells**. The accumulation of these foam cells creates a visible, sub-endothelial lipid deposit called a *fatty streak*.
4. **Plaque Development & Fibrous Capping:** Local smooth muscle cells migrate from the middle layer of the artery wall into the inner lining, where they multiply and secrete a tough matrix of connective tissue fibers, primarily collagen. This creates a hard **fibrous plaque** with a dense cap that seals in the core of

lipid waste. This lesion bulges into the arterial lumen, narrowing its diameter, increasing resistance to blood flow, and elevating localized blood pressure.

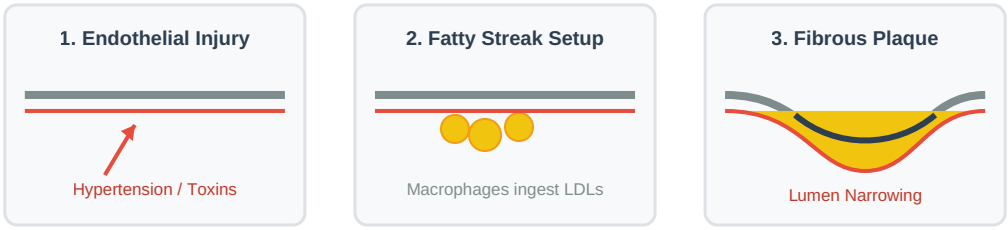

Figure 1: Pathological stages of plaque accumulation leading to arterial lumen narrowing.

3. The Clinical Consequences of Atherosclerosis

As plaques grow, they compromise blood circulation, which can cause several acute, life-threatening cardiovascular conditions:

Myocardial Infarction (Heart Attack)

If advanced plaque forms within the **coronary arteries** (the vessels supplying oxygenated blood directly to the heart muscle), the cardiac tissue downstream is deprived of oxygen (*ischemia*). If a plaque ruptures, it can trigger an acute blood clot that completely blocks the artery. This cuts off oxygen delivery, causing permanent ischemic necrosis (death) of the affected heart muscle tissue.

Ischemic Cerebrovascular Stroke

A stroke occurs when the blood supply to part of the brain is suddenly restricted or cut off. This is typically caused by an atheroma or blood clot blocking the **carotid arteries** or smaller cerebral vessels. Deprived of oxygen, brain cells quickly die, leading to rapid loss of neurological functions.

4. Scientific Evaluation of Risk & Epidemiology

A **risk** is defined scientifically as the mathematical probability that a specific unwanted event or outcome will occur within a given timeframe. In medicine, epidemiological studies identify risk factors and evaluate their impacts. These studies generally fall into two categories:

- **Cohort Studies:** Large groups of healthy individuals are monitored over an extended period. Researchers track their lifestyle choices, diet, and environmental exposures to see who develops a specific disease, allowing them to directly link risk factors to health outcomes over time.

- **Case-Control Studies:** A group of individuals who already have the disease (the *cases*) is compared directly to a matched group of healthy individuals (the *controls*). Researchers analyze their past choices and histories to identify retrospective correlations.

> **DATA ANALYSIS: EVALUATING EPIDEMIOLOGICAL STUDIES**
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> To be scientifically valid, epidemiological data must meet strict design criteria: the study sample must be large enough to minimize the role of chance, potential confounding variables must be strictly controlled, and the selection process must avoid bias to ensure the results genuinely reflect the wider population.

Correlation versus Causation

A common pitfall in epidemiological analysis is confusing a statistical **correlation** with true **causation**. A correlation exists when a change in one variable matches a change in another variable. However, this does not prove that the first variable directly caused the second; both could be influenced by a third, unmeasured confounding factor. Causation can only be confirmed when a clear biological mechanism explains exactly how one factor triggers the disease process.

5. Categorization of Cardiovascular Risk Factors

Cardiovascular disease is multi-factorial, driven by a combination of non-modifiable traits and modifiable lifestyle risk factors:

Risk Factor Category	Specific Risk Element	Underlying Biological Influence & Mechanism
Non-Modifiable Risk Factors (Cannot be altered by intervention)	**Advanced Age**	As organisms age, blood vessels naturally lose their elasticity and stiffen. This increases peripheral resistance, elevating blood pressure and making the endothelium more vulnerable to damage.
	Biological Sex	Pre-menopausal women exhibit lower statistics of CVD than men. Endogenous **oestrogen** levels improve lipid profiles by raising HDLs and lowering LDLs, while also helping protect endothelial linings.
	Genetic Predisposition	Inherited alleles can predispose individuals to CVD by causing structural weaknesses in arterial walls, poor cholesterol metabolism, or familial hypercholesterolaemia.
Modifiable Lifestyle Risk Factors (Can be managed or altered)	**Dietary Profiling**	Diets high in saturated fatty acids raise circulating blood **LDL cholesterol** levels, accelerating fatty streak deposition and plaque development.
	Chronic Hypertension	Consistently high blood pressure generates elevated mechanical shear stress against arterial surfaces, causing direct endothelial damage that initiates atherosclerosis.
	Tobacco Consumption	Nicotine stimulates adrenaline release, narrowing blood vessels and raising blood pressure. Carbon monoxide binds to hemoglobin, reducing oxygen transport, while toxic chemicals cause oxidative damage to the endothelium.
	Physical Inactivity	Sedentary lifestyles lower metabolic efficiency, reduce high-density lipoprotein (HDL) levels, and increase the risk of obesity and secondary type-2 diabetes.

6. Quantitative Energy Balance and Body Mass Index (BMI)

Maintaining a healthy weight depends on a balanced energy ledger: **energy input** (calories consumed through food and drink) must match **energy output** (calories expended through basal metabolism and

physical activity). If energy intake consistently exceeds output, the body stores the excess calories as adipose tissue, leading to weight gain and obesity.

Calculating Body Mass Index (BMI)

In clinical practice, body composition and weight-related health risks are monitored using the **Body Mass Index (BMI)**. This index evaluates an individual's weight relative to their height using a standard mathematical formula:

$$\text{Body Mass Index (BMI)} = \text{Weight (kg)} \div [\text{Height (m)}]^2$$

The resulting values categorize clinical weight status and align with specific thresholds of cardiovascular risk:

- **Underweight:** BMI values below 18.5 kg m^{-2}.
- **Normal WNL (Within Normal Limits):** BMI values ranging from $18.5 \text{ to } 24.9 \text{ kg m}^{-2}$.
- **Overweight:** BMI values ranging from $25.0 \text{ to } 29.9 \text{ kg m}^{-2}$.
- **Clinical Obesity:** BMI values ranging from $30.0 \text{ to } 39.9 \text{ kg m}^{-2}$.
- **Morbid Obesity:** BMI values equal to or exceeding 40.0 kg m^{-2}.

> **EXAM HINT: AVOIDING CALCULATION MISTAKES**
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> When calculating BMI in exams, a common mistake is forgetting to square the height value, or using height in centimeters rather than meters. Always double-check your units (kg and meters) before finalizing your answer.

Alternative Metric: Waist-to-Hip Ratio

While BMI is a useful general tool, it has limitations—for example, it can misclassify highly muscular athletes as overweight because it does not distinguish between muscle mass and fat tissue. Because of this, clinicians often use the **waist-to-hip ratio** alongside BMI. This metric measures abdominal obesity directly, which serves as a highly accurate indicator of visceral fat accumulation and an independent predictor of future cardiovascular disease.