

Exercise walk-through

1.5

Atherosclerosis, blood clotting and CVD risk (statements 1.10–1.13, core practical 2)

Pearson Edexcel · International A-Level · Biology

Atheroma, clot, catastrophe

Damage : smoking, hypertension, LDL cholesterol harm the endothelium

Plaque : inflammation and LDL deposition narrow and stiffen the artery

Clot : thromboplastin converts prothrombin to thrombin; fibrinogen becomes fibrin

Read a health study like an examiner

Your turn

Gum disease correlates with endothelial damage. List three checks before claiming causation.

- A. Who was sampled, how selected, how many?
- B. Were confounders like smoking, age, diet controlled?
- C. Is there a mechanism and a dose-response pattern?

DNA copies itself semi-conservatively

Unzip : helicase breaks the hydrogen bonds between the strands

Template : each old strand pairs with free nucleotides by complementary base pairing

Join : DNA polymerase links the nucleotides into a new strand

Exercises 2.1·2.3

2.1 Put these in order: fibrin forms, thromboplastin released, prothrombin converted, endothelium damaged. [2]

2.2 Explain why a clot is more likely to form where an atherosclerotic plaque has developed. [2]

2.3 State two ways in which the structure of an artery changes as a plaque develops. [2]

Exercises 2.1·2.3

2.1 Put these in order: fibrin forms, thromboplastin released, prothrombin converted, endothelium damaged. [2]

2.1 Endothelium damaged → thromboplastin released → prothrombin converted to thrombin → fibrin forms.

2.2 Explain why a clot is more likely to form where an atherosclerotic plaque has developed. [2]

2.2 The plaque narrows the lumen, so blood flows faster and more turbulently past it, damaging the already-weakened endothelium and exposing collagen, which triggers platelet adhesion and the clotting cascade.

2.3 State two ways in which the structure of an artery changes as a plaque develops. [2]

2.3 Any two: lumen narrows; wall loses elasticity / hardens as smooth muscle and calcium-rich plaque accumulate; endothelium damaged.

Exercises 3.1·3.2

3.1 A man has a blood pressure of 17.2/11.4 kPa and a family history of heart disease. Explain how his hypertension contributes to the development of atherosclerosis, and state why his family history counts as a risk factor even though he cannot change it. [5]

3.2 Haemophilia reduces the conversion of prothrombin to thrombin. Explain how this leads to prolonged bleeding, referring to the molecules that are affected and the step that fails. [4]

Exercises 3.1·3.2

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3.1 High pressure forces blood against the endothelium, damaging its cells; the damage begins the inflammatory response and lets LDL-cholesterol enter the artery wall; plaque grows, narrowing the artery further and raising pressure again – a positive feedback of deterioration. Family history indicates inherited alleles that raise cholesterol handling, blood-pressure regulation or vessel-wall susceptibility, so his baseline risk is higher whatever his behaviour.

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3.2 With little thrombin formed, fibrinogen is not converted to fibrin, so no insoluble fibrin mesh forms; platelets arrive but are not bound into a stable mesh by fibrin, so the platelet plug washes away and bleeding continues.

Exercise 3.3

3.3 A study of 10 000 people records for each participant: age, BMI, smoking, blood pressure and LDL-cholesterol. The risk of a heart attack in the next ten years is 1 % for non-smokers with normal BMI and pressure, 3 % for smokers with normal BMI and pressure, and 11 % for smokers with high BMI and high pressure. Explain what these figures show about how risk factors combine, and calculate how many times greater the combined risk is than the baseline risk. [5]

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3.3 Smoking alone multiplies the baseline risk by 3; adding high BMI and high blood pressure raises it to 11 times baseline (11%/1%, A1) – far more than the sum of the separate effects would suggest. Risk factors interact: each one multiplies the effect of the others on the same arterial system.