- This text consists of multiple-choice questions and answers related to atherosclerosis, its risk factors, and pharmacological treatments.

- It covers the characteristics of atherosclerotic plaques, non-modifiable risk factors, classification of cholesterol levels, and the transport of fats in the blood.

- The questions also delve into various drug classes used to manage atherosclerosis, including Statins, Niacin, Fibrates, and Bile Acid Resins, detailing their mechanisms of action, effects on lipid levels, common side effects, and specific applications.

- Atherosclerosis refers to "gruel or paste." Statins lower LDL by increasing liver LDL receptor expression due to decreased hepatic cholesterol synthesis.

- Statins effectively reach the liver due to extensive presystemic biotransformation.

- Beyond cholesterol lowering, Statins do not increase blood pressure.

- Rhabdomyolysis from Statins is exacerbated by Fibrates, increasing risk by 10+ fold.

- Niacin inhibits diacylglycerol acetyltransferase 2, an enzyme in triglyceride synthesis.

- The HMG-CoA reductase reaction inhibited by Statins releases NADPH.
- Niacin can cause amblyopia/blurred vision.
- Foam cell formation begins with modified LDL and a monocyte differentiating into a macrophage.
- Fibrates lower VLDL and LDL by reducing hepatic triglyceride synthesis.
- Fibrates and Statins interact, increasing myopathy/ rhabdomyolysis risk.
- Fibrates should be used cautiously in patients with renal failure.
- Bile Acid Resins reduce LDL-C.
- Bile Acid Resins cause strong and rapid HMG-CoA reductase upregulation.
- Ezetimibe's inhibition of sterol absorption is offset by increased cholesterol synthesis.
- Ezetimibe indirectly stimulates hepatic LDL receptor expression.
- Atherosclerosis plaques are on large and medium-sized arteries.

- Endogenous cholesterol production is approximately 1 g/day.
- Statins alone achieve 30-50% LDL-C reduction.
- Statins are used for primary and secondary prevention of atherosclerosis.
- Niacin reduces triglyceride synthesis by reducing free fatty acid transport to the liver.
- Niacin increases plasminogen activator, contributing to fibrinolysis.
- Niacin can cause angio-oedema.
- Fibrates increase LPL synthesis, decreasing lipoprotein triglycerides.

#
Question
A
B
C
D
Answer
1
Atherosclerosis is characterized by the deposition of plaques on the innermost layer of arteries, also known as the:
Media
External lamina
Intima
Fibrous cap
C
2
The word component sclerosis in Atherosclerosis means:
Gruel
Paste
Hardness
Lipid
C
3
The fibrous cap of an atherosclerotic plaque overlies which component?
Intima
Media
Lipid Core
Lumen

C
4
Which cell type is often referred to as a "foam cell" within an atherosclerotic plaque?
Endothelial cells
Smooth muscle cell
Macrophages
T-lymphocytes
C
5
Which of the following is considered a non-modifiable risk factor for atherosclerosis?
Hypertension
Cigarette smoking
Age
Obesity
C
6
Which risk factor is unique in its ability to drive atherosclerosis even in the absence of other risk factors?
Diabetes Mellitus
Elevated serum cholesterol levels
Sedentary lifestyle
Elevated Homocysteine
B
7
LDL cholesterol levels of \mathbf{< 100} mg/dL are classified as:
Very high
High

Borderline high
Optimal
D
8
LDL cholesterol levels of \mathbf{190} mg/dL and above are classified as:
High
Borderline high
Optimal
Very high
D
9
Cholesterol and fats are transported in the blood via:
Albumins
Bilirubins
Lipoproteins
Cytokines
C
10
Lipoproteins are classified based on the type and ratio of:
\text{DNA} and protein
\text{RNA} and fats
Protein and fats
Water and salt
C
11
Which of the following is a class of lipoproteins?
\text{HMD}
\text{VFL}
VLDL

\text{AML}
C
12
Which lifestyle modification is linked with a lower risk of heart disease and increases \text{HDL}?
Diet low in cholesterol
Physical activity
Decreasing body weight
Decreasing stress
B
13
What is the mechanism by which Statins primarily lower cholesterol?
Increasing bile acid reabsorption
Inhibiting LDL receptor synthesis
Blocking HMG CoA reductase
Activating \text{PPAR}-\alpha
C
14
Which drug class typically achieves a \mathbf{30-50\%} decrease in \text{LDL}?
Fibrates
Niacin
Statins
Bile Acid Resins
C
15
A consequence of inhibiting \text{HMG-CoA} reductase is the decreased synthesis of:
Bile Acids

\text{CHO} (Cholesterol) in the liver
\text{LPL}
Sterol Hormones
B
16
The conversion of \text{HMG-CoA} to mevalonic acid is catalyzed by:
Mevalonate kinase
\text{HMG-CoA} reductase
Geranyl transferase
\text{Acetoacetyl-CoA} synthetase
B
17
Which of the following is a common adverse effect of Statins?
\text{GI} bleeding
Myopathy
Angio-oedema
Hyperuricemia
B
18
Statins should not be given during:
Active infection
Diabetes Mellitus
Pregnancy
Combination with \text{CCBs}
C
19
Which drug listed is an inactive pro-drug that requires biotransformation?

Atorvastatin
Rosuvastatin
Simvastatin
Pravastatin
C
20
Statins are used for secondary prevention in patients with symptomatic atherosclerotic disease, such as:
Acute gastritis
Angina
Bronchitis
\text{BPH}
B
21
Niacin is also known as:
Vitamin \text{B}1
Vitamin \text{B}2
Vitamin \text{B}3
Vitamin \text{B}6
C
22
Which drug is considered the best agent to increase \text{HDL-C} (\sim 35-40\%)?
Statins
Niacin
Fibrates
Ezetimibe
B
23
Niacin is a water-soluble \text{B}-complex vitamin that

functions only after conversion to \text{NAD} or:
\text{CoA}
\text{AMP}
\mathbf{\text{NADP}^{\mathbf{+}} \text{ Nicotinamide}}
\text{ATP}
C
24
A harmless but common side effect of Niacin related to cutaneous vasodilation is:
Rashes
\mathbf{\text{Sensation of warmth (Flushing)}}
Pruritus
Diarrhea
B
25
The class of drugs known as \text{PPARs} Activators includes:
Statins
Bile Acid Resins
Fibrates
Niacin
C
26
\text{PPARs} Activators stimulate:
\text{CHO} synthesis
\text{LDL} receptor synthesis
Fatty acid oxidation
\text{HMG-CoA} reductase
C
27

Fibrates are the drugs of choice in the treatment of severe:
Hypercholesterolemia
Hypertriglyceridemia
Atherosclerosis
Hypertension
B
28
The Bile Acid –Binding Resins are insoluble in:
Lipid
Protein
\text{Alcohol}
Water
D
29
Which effect allows Bile Acid-Binding Resins to lower
\text{LDL-C} levels?
\text{LPL} activation
\text{PPAR- }\alpha activation
Increased \text{LDL} receptor production
\text{NPC1L1} inhibition
C
30
Which drug is an Inhibitor of Sterol Absorption?
Simvastatin
Fenofibrate
Cholestyramine
Ezetimibe
D
31
Ezetimibe specifically inhibits the transport process in jejunal

enterocytes involving the transporter:
\text{HMG-CoA}
\text{LDL-R}
\mathbf{\text{NPC1L1}}
\text{LPL}
C
32
A positive pharmacodynamic action of Statins is the stabilization of:
\text{VLDL}
\text{HDL}
Atherosclerotic plaques
\text{T}-lymphocytes
C
33
The two major sources of cholesterol in the body are endogenous production (liver) and:
Vegetables
Fruits
Food (animal sources)
Water
C
34
What is the term for the breakdown of muscle fibers releasing contents like myoglobin into the bloodstream, a severe \text{ADR} of Fibrates/Statins?
Myalgia
Myositis
Rhabdomyolysis
Angio-oedema

C
35
Statins increase the expression of \text{LDL} receptors primarily in the:
Muscle tissue
\text{CNS}
Liver
Adipose tissue
C

#
Question
A
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Answer
71
The Greek word athero in Atherosclerosis refers to:
Hardness
\text{Cholesterol}
Gruel or paste
Artery wall
C
72
The primary mechanism by which Statins \downarrow circulating \text{LDL} is by causing the liver to increase expression of \text{LDL} receptors, driven by the \downarrow in:
\text{FFA} flux

Hepatic \text{CHO} synthesis
Plasma \text{VLDL}
\text{LPL} activity
B
73
Which drug property of Statins helps them reach their target tissue (the liver) effectively?
Inactive pro-drug status
Low molecular weight
Extensive presystemic biotransformation
High lipid solubility
C
74
Which of the following is NOT listed as a promising pharmacodynamic action of Statins beyond \text{CHO} lowering?
Immune suppression
\uparrow \text{neovascularisation}
\mathbf{\text{Increased blood pressure}}
\uparrow \text{synthetic activity in osteoblasts}
C
75
The severe side effect of Rhabdomyolysis from Statins is exacerbated by the combination with Fibrates, which increases the risk by approximately:
2 fold
5 fold
10+ fold
20 fold
C

76
Niacin's mechanism of action may involve inhibiting a rate-limiting enzyme of triglyceride synthesis, specifically:
\text{LPL}
\text{HMG-CoA} Reductase
Diacylglycerol acetyltransferase 2
\text{Adipocyte adenylyl cyclase}
C
77
The \text{HMG-CoA} reductase reaction that Statins inhibit converts \text{HMG-CoA} to mevalonate through an intermediate. What molecule is released during this conversion (seen in the image)?
\text{ATP}
\text{ADP}
\text{NADPH}
\text{NADH}
C
78
Which is a possible toxicity of Niacin that affects the eyes?
Diplopia
Amblyopia, blurring of vision
Conjunctivitis
Glaucoma
B
79
The initial step in the formation of a foam cell involves \text{LDL} being modified, then a Monocyte crossing the endothelium and differentiating into a:
Fibroblast

\text{T}-lymphocyte
Macrophage
Smooth muscle cell
C
80
Fibrates primarily lower VLDL and LDL levels by enhancing \text{LPL} activity and stimulating fatty acid oxidation, which results in a reduction of hepatic:
Triglyceride synthesis
\text{HDL} synthesis
\text{LDL} receptor synthesis
\text{CHO} absorption
A
81
\text{Fibrates} and Statins interact, leading to increased levels of both drugs. This is primarily a concern for which \text{ADR}?
Gallstones
\text{GI} disturbances
Myopathy/Rhabdomyolysis
Hyperuricemia
C
82
\text{Fibrates} should be used with caution in which patient population due to potential accumulation or altered response?
Diabetic patients
Pregnant women
Patients with renal failure
Patients with angina

C
83
The Bile Acid –Binding Resins cause hepatic \text{CHO} content to decrease, which triggers two simultaneous opposing effects: \uparrow \text{LDL} receptors and \uparrow \text{HMG-CoA} reductase. The net effect is a reduction in:
\text{HDL-C}
\text{Triglycerides}
\text{LDL-C}
\text{VLDL}
C
84
Which drug class causes a strong and rapid upregulation of \text{HMG-CoA} reductase as a compensatory mechanism?
Statins
Niacin
Bile Acid Resins
\text{Fibrates}
C
85
Although Ezetimibe can \downarrow \text{LDL-C} by 15-20\ %, its inhibition of sterol absorption (54%) is offset by a compensatory increase in:
\text{LPL} synthesis
\text{FFA} flux
Cholesterol synthesis
\text{LDL} receptor expression
C
86

The effect of Ezetimibe, reducing delivery of cholesterol to the liver by chylomicron remnants, indirectly stimulates the expression of hepatic genes regulating:
\text{NPC1L1}
\text{Triglyceride} synthesis
\mathbf{\text{LDL receptor expression}}
\text{HMG-CoA} reductase
C
87
The definition of Atherosclerosis states that the plaques contain cholesterol and lipids, which are deposited on the walls of:
Veins
Capillaries
Large and medium-sized arteries
Arterioles
C
88
The endogenous production of cholesterol in the liver is approximately:
100 \text{ mg/day}
1 \text{ g/day}
10 \text{ g/day}
100 \text{ g/day}
B
89
What percentage of \text{LDL-C} reduction is achieved when Statins are used alone?
15-20\%
\mathbf{30-50\%}

60\%
90\%
B
90
The clinical use of Statins includes \text{secondary prevention} of \text{MI} and stroke in patients with symptomatic atherosclerotic disease, as well as \text{primary prevention} for those with high \text{CHO} and other risk factors for:
\text{COPD}
\text{BPH}
Atherosclerosis
\text{RA}
C
91
Niacin reduces triglyceride synthesis in the liver by reducing the transport of which molecules from adipose tissue to the liver?
\text{Triglycerides}
Free fatty acids (\text{FFA})
\text{VLDL}
\text{HDL}
B
92
In addition to its \text{HDL}-increasing, \text{TG}-lowering, and \text{LDL}-decreasing effects, Niacin also \uparrow plasminogen activator, which contributes to:
Anticoagulation
Fibrinolysis
Vasoconstriction

\text{CHO} absorption
B
93
Which side effect of Niacin, common to other drugs like \text{ACE} inhibitors, involves the skin/mucous membranes?
Hemolytic anemia
\text{Hyperuricemia}
Angio-oedema (rare)
Constipation
C
94
\text{Fibrates} increase \text{LPL} synthesis, resulting in enhanced lipolysis of lipoprotein triglycerides; this primarily decreases the levels of which lipoproteins?
\text{LDL} and \text{HDL}
\text{HDL} and \text{Chylomicrons}
\text{VLDL} and \text{LDL}
\text{IDL} and \text{HDL}
C
95
In the flow chart, the activation of \text{PPAR}-\alpha by \text{Fibrates} leads to \uparrow \text{apoA-I} and \text{apoA-II} synthesis in hepatocytes, which results in \uparrow:
\text{VLDL} synthesis
Plasma \text{HDL}
\text{Triglyceride} synthesis
\text{LDL} catabolism
B
96

Which drug can improve insulin resistance, a potentially beneficial secondary effect of the drug class?
Simvastatin
\text{Cholestyramine}
Fenofibrate (\text{Fibrates})
Niacin
C
97
The Bile Acid –Binding Resins are large polymeric, water-insoluble, anionic-exchange resins. This implies they have what charge?
Neutral
Positive
Negative
Both positive and negative
B
98
Ezetimibe's mechanism is complementary to Statins primarily because it acts on absorption in the jejunum, while Statins act on \text{CHO} synthesis in the:
Jejunum
Adipose tissue
Liver
Vascular wall
C
99
Which drug class is most likely to counteract the effect of Bile Acid-Binding Resins by increasing the enzyme that synthesizes \text{CHO}?
\text{Fibrates}

Niacin
\text{Ezetimibe}
Bile Acid Resins themselves
D
100
A common \text{ADR} for both Statins and Ezetimibe, especially when combined, is:
Hyperuricemia
Myopathy
Cutaneous vasodilation
Gallstones
B