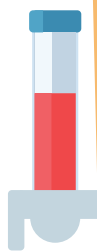
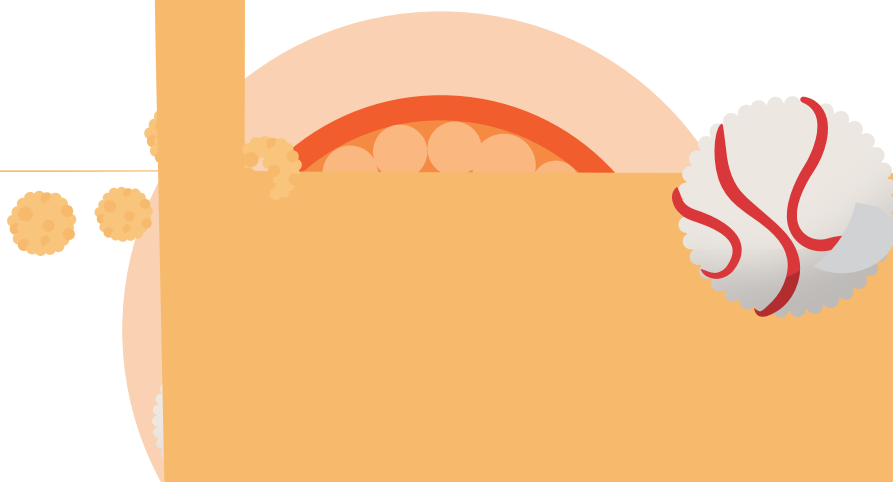
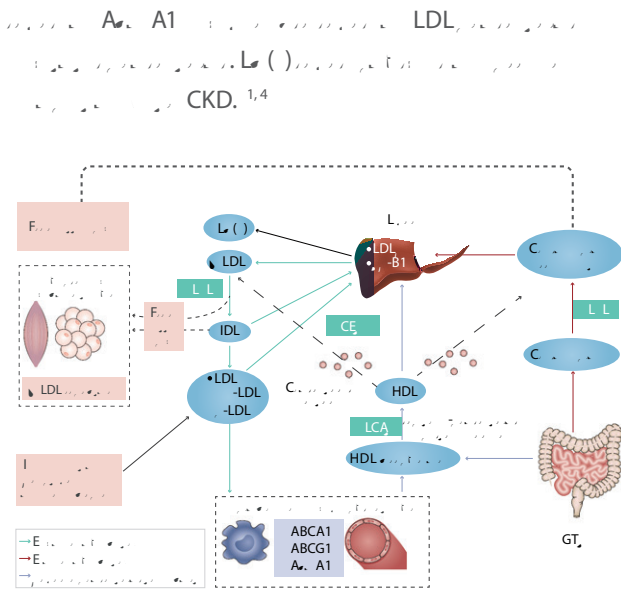


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Hypertriglyceridemia in CKD results from the delayed catabolism of triglyceride-rich lipoproteins. Apo A1 synthesis is reduced in patients with CKD, and the activation of LCAT by Apo A1 is also decreased. The maturation of HDL precursors is consequently affected. Increased Lp(a) is caused by post-translation modification of lipoproteins related to CKD.

Figure 3. Dyslipidemia in CKD

CKD is associated with a dyslipidemic profile characterized by elevated levels of LDL-C, IDL-C, and Lp(a), and decreased levels of HDL-C.

The KDIGO guideline (KDIGO) (2013) recommends a target LDL-C of 50 mg/dL for patients with CKD.

LDL-C is the primary target for treatment in CKD. The target LDL-C level is 50 mg/dL for patients with CKD.

Statins are the first-line treatment for LDL-C in CKD. The target LDL-C level is 50 mg/dL for patients with CKD.

For patients with CKD who are not on statins, the target LDL-C level is 50 mg/dL.

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