Available lipid-modifying drugs used in the management of dyslipidemia include 3-hydroxy-3-methyl-glutaryl-CoA reductase inhibitors (statins), the cholesterol absorption inhibitor, ezetimibe, and bile acid sequestrants (BAS).

In hypophysectomized rats, hepatic 3-hydroxy-3-methylglutaryl coenzyme A (HMG-CoA) reductase activity, immunoreactive 97-kilodalton (97-kDa) protein, and mRNA were all reduced to undetectable levels. Administration of triiodothyronine (T3) resulted in large increases in all three after a 36-h lag period. HMG-CoA reductase activity, immunoreactive 97-kDa protein levels, and reductase mRNA levels were tightly correlated. Feeding hypophysectomized rats diets containing the bile acid sequestrant colestipol, together with the potent reductase inhibitor mevinolin, resulted in an increase in HMG-CoA reductase activity similar to that seen with T3 but a lesser stimulation of reductase mRNA levels. These results suggest that agents which cause depletion of mevalonate-derived products may share in part with T3 a common mechanism for increasing levels of HMG-CoA reductase activity in order to satisfy cellular needs for these products. Dexamethasone treatment, which is known to prevent the T3-mediated stimulation of reductase activity, caused a marked decrease in 97-kDa immunoreactive material but had little effect on reductase mRNA levels [1)].

1)
Sample CE, Pendleton LC, Ness GC. Regulation of 3-hydroxy-3-methylglutaryl coenzyme A reductase mRNA levels by L-triiodothyronine. Biochemistry. 1987 Feb 10;26(3):727-31. doi: 10.1021/bi00377a011. PMID: 3567145.