LDL–C is lowered. However, no variation in the relative reduction in ASCVD risk was observed after the data were adjusted for LDL–C reduction. Furthermore, there is no differentiation between the specific statins and doses used in primary and secondary prevention RCTs, based on a high level of evidence that statins reduce ASCVD risk similarly in both populations.

Percent reductions in LDL–C for a specific statin and dose were calculated for the RCTs included in individual meta-analyses conducted by the Cholesterol Treatment Trialists (CTT) in 2010 (20) in which statin therapy reduced ASCVD events. High-intensity statin therapy on average lowers LDL–C by approximately ≥50%, moderate-intensity statin therapy lowers LDL–C by approximately 30% to <50%, and lower-intensity statin therapy lowers LDL–C by <30% (Table 5).

Table 5. High- Moderate- and Low-Intensity Statin Therapy (Used in the RCTs reviewed by the Expert Panel)*

High-Intensity Statin Therapy	Moderate-Intensity Statin Therapy	Low-Intensity Statin Therapy
Daily dose lowers LDL–C on average, by approximately ≥50%	Daily dose lowers LDL–C on average, by approximately 30% to <50%	Daily dose lowers LDL–C on average, by <30%
Atorvastatin (40†)–80 mg **Rosuvastatin 20** *(40)* **mg**	**Atorvastatin 10** *(20)* **mg** **Rosuvastatin** (*5*) **10 mg** **Simvastatin 20–40 mg‡** **Pravastatin 40** *(80)* **mg** **Lovastatin 40 mg** *Fluvastatin XL 80 mg* **Fluvastatin 40 mg bid** *Pitavastatin 2–4 mg*	*Simvastatin 10 mg* **Pravastatin 10–20 mg** **Lovastatin 20 mg** *Fluvastatin 20–40 mg* *Pitavastatin 1 mg*

Specific statins and doses are noted in bold that were evaluated in RCTs (17,18,46-48,64-67,69-78) included in CQ1, CQ2 and the CTT 2010 meta-analysis included in CQ3 (20). All of these RCTs demonstrated a reduction in major cardiovascular events. Statins and doses that are approved by the U.S. FDA but were not tested in the RCTs reviewed are listed in *italics*.

*Individual responses to statin therapy varied in the RCTs and should be expected to vary in clinical practice. There might be a biologic basis for a less-than-average response.

†Evidence from 1 RCT only: down-titration if unable to tolerate atorvastatin 80 mg in IDEAL (47).

‡Although simvastatin 80 mg was evaluated in RCTs, initiation of simvastatin 80 mg or titration to 80 mg is not recommended by the FDA due to the increased risk of myopathy, including rhabdomyolysis.

bid indicates twice daily; FDA, Food and Drug Administration; IDEAL, Incremental Decrease through Aggressive Lipid Lowering study; LDL–C, low-density lipoprotein cholesterol; and RCTs, randomized controlled trials.

4.2. LDL–C and Non-HDL–C Treatment Goals

The Expert Panel did not find evidence to support titrating cholesterol-lowering drug therapy to achieve optimal LDL–C or non-HDL–C levels because the clinical trials were essentially fixed dose trials (CQ1 and CQ2). Dosage increases did occur in a few RCTs with the intent of maximizing statin therapy. Therefore, these were not truly tests of defining optimal goals for LDL–C in primary and secondary prevention