



## Perceptions on Cholesterol Management and Cholesterol Absorption and Production Inhibition

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### Abstract

**Introduction:** Clinical trial evidence over the last two decades shows that reducing cholesterol levels reduces the risk of Cardiovascular (CV) disease, particularly coronary heart disease, which is a leading cause of death and morbidity in the United States and Europe [1]. The findings of these research are used to develop national and international treatment guidelines that define lipid thresholds and treatment goals for a number of patient groups [2]. These guidelines are a critical management tool for clinicians to use in medical practice to help them lessen the effect of atherosclerotic-related CV disease [3].

Despite the fact that 5-hydroxy-3-methylglutaryl-coenzyme A (HMG-CoA) reductase inhibitors (statins) are widely suggested in various guidelines, statins alone (even at higher doses) are sometimes insufficient for many patients to achieve low-density lipoprotein cholesterol (LDL-C) objectives[12]. According to a recent European survey, over 60% of hyper cholesterolemic patients fail to meet their cholesterol objectives based on national guidelines. 13 Failure to titrate statin doses, in part due to worries about the likelihood of adverse events at higher statin doses when only a modest 6% reduction in LDL-C levels may be predicted, is one of the main reasons for this poor level of goal achievement. As a result, there is a significant disconnect between what is advised and what is actually accomplished in practise.

In light of findings from large outcome studies, this disparity is anticipated to widen as guidelines adopt the premise that lower LDL-C is healthier.

By delivering a single tablet of ezetimibe, simvastatin or co-administering ezetimibe with a statin, a new strategy to cholesterol control is targeting cholesterol absorption in the gut from both biliary and dietary sources, as well as cholesterol synthesis in the liver.

Several studies have shown that blocking cholesterol absorption and synthesis results in significantly better LDL-C reduction and more individuals reaching their LDL-C objectives than doubling the baseline statin dose. The surveys were designed to gauge cardiologists and general practitioners (GP) opinions on cholesterol management, cholesterol absorption

and production inhibition, the extent to which treatment guidelines are being followed, and potential barriers to prescribing high-dose statins for high-risk patients across Europe.

### Discussion

The majority of doctors polled believed that statin medication alone might not be enough to cure hypercholesterolemia in patients with CV risk, and that blocking cholesterol absorption and synthesis reduces LDL-C levels more effectively than treating only one source. That is, treating cholesterol absorption by the gut as well as generation by the liver is expected to result in better cholesterol reduction than treating production with statins alone. This viewpoint is supported by a plethora of clinical trial evidence demonstrating that blocking cholesterol absorption and synthesis results in consistently superior LDL-C lowering efficacy and enhanced LDL-C goal accomplishment.

When compared to utilising high-dose statins, the majority of physicians believed that limiting cholesterol absorption and synthesis would reach higher efficacy faster and with fewer adverse effects.

For patients with CV risk factors, most physicians chose to employ cholesterol-lowering therapies that were more acceptable than high-dose statins, with side effects influencing physicians' reluctance to prescribe high-dose statins rather than worries about efficacy.

When choosing a treatment technique for limiting absorption in patients with CHD or diabetes, many cardiologists believed that treating biliary cholesterol was more significant than treating dietary cholesterol, showing the increased awareness of the impact of biliary cholesterol on LDL-C levels.

Cholesterol in the body comes through biliary and dietary cholesterol absorption in the gut, as well as production in the liver and peripheral tissues, with biliary cholesterol accounting for roughly two-thirds of intestinal cholesterol. Because, unlike dietary cholesterol, the amount of cholesterol in bile is essentially unaffected by dietary alteration or medications such as statins, fibrates, or nicotinic acid, inhibiting biliary cholesterol absorption is an important therapeutic target.

Almost all doctors agreed that decreasing LDL-C and total cholesterol was their primary goal. More than 80% agreed that preventing a primary or secondary CV event, as well as lowering LDL-C and total cholesterol, was important, and that assisting patients with diabetes or CHD achieve LDL-C objectives was also important. Despite this, only a small percentage of physicians were aware of the optimum LDL-C values for patients with CHD or diabetes, and the majority believed that only half of their patients had achieved target LDL-C levels. Nearly half of doctors were unaware of the impact of doubling a statin's dose on LDL-C lowering.

GPs, in particular, were ignorant of critical worldwide standards for at-risk patients and instead tended to adopt country-specific guidelines. The majority of doctors felt that guidelines are not followed to their full potential, owing to financial restrictions.

Finally, the results of the 2005 and 2006 surveys show an increasing belief among European practitioners that many patients with hypercholesterolemia require more advanced cholesterol-lowering medication to achieve LDL-C targets.

Many practitioners are depending more on blocking both cholesterol absorption in the intestine and cholesterol production in the liver to achieve better LDL-C control (especially in patients with CV risk factors). Lowering LDL-C to the target level is now commonly regarded as improving patient outcomes. Our findings show that many doctors are hesitant to achieve these aims by raising statin dosage. When a cholesterol absorption inhibitor is used with a statin, greater LDL-C management is achieved without increasing the statin dose.

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