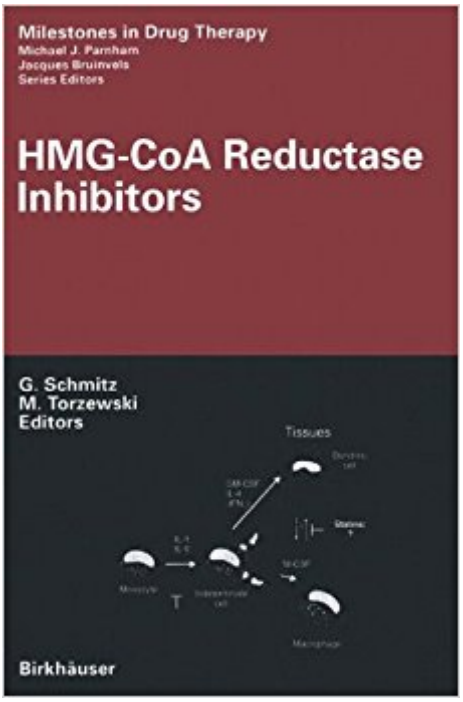
HMG-CoA Reductase Inhibitors (Milestones In Drug Therapy)



Synopsis

HMG-CoA reductase inhibitors (statins) are established drugs for the treatment of hypercholesterolemia. Furthermore, they induce regression of vascular atherosclerosis as well as reduction of cardiovascular-related morbidity and death in patients with and without coronary artery disease. This book deals with statins which have substantially altered the approach to therapy of atherosclerosis and its sequelae. Emphasis is placed on the scientific background to the discoveries and the development of the therapy, including an overview of the current state of knowledge of the drugs. Clinical data are reviewed extensively. This book not only provides the reader with valuable information but also stimulates further research into the pathogenesis of atherosclerosis and the mechanisms behind the action of effective statins. It sets the stage for creative thinking among scientists of many disciplines for the accomplishment of our ultimate goals in treating atherosclerosis and its sequelae. This topical volume...

Book Information

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Customer Reviews

HMG-CoA reductase inhibitors are essentially the statin drugs: atorvastatin (LipitorÃƒâ Ã â„¢), cerivastatin (BaycolÃƒâ Ã â„¢,withdrawn 8/01), fluvastatin (LescolÃƒâ Ã â„¢), lovastatin (MevacorÃƒâ Ã â„¢), pravastatin (PravacholÃƒâ Ã â„¢), simvastatin (ZocorÃƒâ Ã â„¢),pitavastatin and rosuvastatin (CrestorÃƒâ Ã â„¢), which were introduced to lower total cholesterol (TC) levels, and especiallyLDL-cholesterol (LDL) levels, ostensibly to prevent

coronary heart disease (CVD). The book consists of 8 chapters by nominally highly qualified authors in the form of review articles of the sortnormally found in medical journals. These are devoted to the pharmacology and supposed benefits of statin drugs.The writing is in expert medical language and is consistent, well-written, well-edited and very well-referenced, inquantity if not in quality. The index is inadequate. All chapters attempt to justify the wide use of the statin drugs to lower TC and LDL by citing references in supportof the claims that high levels have been correlated with cardiovascular disease (pp1,19,35,81,84,99,121,126). Suchclaims are unfounded (Ravnskov U, The Cholesterol Myths, Washington, DC, New Trends, 2000). The supposed benefits of the statins, beyond a large, but meaningless lowering of TC and LDL, are usually givenas lowered relative risks (RR) of mostly non-fatal heart attacks without the slightest indication of the magnitude of themore meaningful reduction of absolute risk (pp101,103,106,115,122,124,137). This misrepresentation has beennoted (Ravnskov, 2000; Gigerenzer G, Calculated Risks: How to Know When Numbers Deceive You, New York,NY, Simon & Schuster, 2002). So the usual tout of pravastatin in the WOSCOPS trial of a 22% drop in all-causemortality was noted without the information that this was only an 0.9% drop absolute in the 5-year trial period(p106). The higher all-cause death rates in 2 of the big trials were ignored, as was the higher breast cancer rate (RR= 1500%) in the CARE trial (Ravnskov, 2000). Besides cancer, the other side effects of statins listed were incomplete, and should have included myalgia,myopathy, polyneuropathy, liver and kidney damage, congestive heart failure and amnesia. Side-effects were said toaffect 2% of patients (p115-6) and 2-6% (p123). In fact, a recent review noted side-effects in 20% of patients abovethe placebo rate, and no change whatever in the all-cause death rate for atorvastatin (Newman CB, Palmer G,Silbershatz H, Szarek M. Safety of Atorvastatin Derived from Analysis of 44 Completed Trials in 9,416 Patients.Am J Cardiol 2003;92:670-6). Statins decrease the body’s production of the essential coenzyme Q-10 and dolichol, among other things. Thiswas not mentioned as a problem in any chapter. While this was shown in one biochemical diagram (p65), it was notin another (p82). Low Q-10 levels are strongly associated with congestive heart failure. "Statins are contra-indicated during pregnancy and breastfeeding. The reason for this is that cholesterol is anessential component for fetal development, including the synthesis of steroids and cell membranes" (p116). Theauthors seem unable to comprehend that cell membranes, steroids and coenzyme Q-10 are needed by all humans. The rare familial hypercholesterolemia, in which TC > 400 mg/dL, was represented as more deadly than it reallyis (p99,111), (Ravnskov, 2000). There was some recognition that statins operate to lower non-fatal heart attack rates by mechanisms other thancholesterol lowering, but not that their desirable effect on thromboxane A2 is less than men can obtain with bufferedaspirin (p71),

or that the desirable effect of raising nitric oxide (NO) levels is less than one can obtain with thesupplement L-arginine with no side-effects. There was no understanding that these effects of statins are independentof initial or final TC or LDL levels (Nielsen JV. Serun lipid lowering and risk reduction? Where is the connection?Br Med J Rapid Response, 19 Nov 01, to Kmietowicz Z. Statins are the new aspirin, Oxford researchers say. BrMed J 2001;323:1145), and thus there is no way to determine who should be treated, or what the dose should be. An entire chapter is devoted to the cost-benefits of statin use (p138ff). Since the use of statins for primaryprevention of CVD has been shown to increase all-cause mortality by 1% over a 10-year period (Jackson PR, et al.Statins for primary prevention: at what coronary risk is safety assured? Br J Pharmacology 2001;52:439-446), and statinshave very little effect in secondary prevention, it would seem that there is no cost-benefit (Kauffman JM, "DoHypolipidemic Drugs Lower Medical Expenses?" Pharmacotherapy 2001;22(12),1583-1586). This book may be of use for a pharmacologist looking for an overview, however narrow in outlook, with literature citations.---Joel M. Kauffman, 6 Oct 03

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