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Personalized medicine and statin side effects: Fact or fiction?

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Cardiovascular disease is the number one killer in the United States. Statins reduce low-density lipoprotein cholesterol (LDL-C) and decrease cardiovascular events. About 43 million Americans currently take statins, and the 2013 cardiovascular disease prevention guidelines will increase statin drug prescriptions for approximately 13 million more Americans. The FDA warns that statins may induce skeletal muscle side effects, cognitive changes and increase fasting glucose levels. Observational studies show that the incidence of skeletal muscle side effects has been underestimated in randomized trials and may affect up to 20% of patients taking statins. Some individuals are more susceptible to statin myopathy because of female gender, advanced age, drug-drug interactions or underlying genetic polymorphisms. Currently five lipophilic and two hydrophilic statins are on the market. Statin effects on cardiac muscle are currently unknown. Healthy mice received either the lipophilic atorvastatin or the hydrophilic pravastatin daily for seven months and were compared to vehicle treated animals. Atorvastatin and pravastatin reduced LDL-C compared to vehicle. Long-term atorvastatin treatment altered the ultrastructure of cardiac muscle in healthy mice, but pravastatin did not. Only atorvastatin administration increased the mortality of mice prone to heart failure, and repressed mitochondrial genes by genome-wide expression profiling. In cultured cardiac myocytes atorvastatin treatment down-regulated survival pathways, decreased RhoA activation, and induced apoptosis. Personalized medicine in CVD prevention should; 1) focus on lifestyle changes, 2) add investigation and documentation for possible statin-induced adverse effects, and 3) determine a treatment regimen that does not impair the patient's quality of life during healthy aging.

Biography

Alice E Zemljic-Harpf obtained her medical degree at the University of Graz, Austria, in 1997. She completed her postdoctoral training at the Cedars-Sinai Medical Center, Los Angels, and at the University of California, Los Angeles. In 2003 she moved to the University of California, San Diego. She currently holds a faculty level position in the Department of Anesthesiology, and Cardiac and Neuro Protection Laboratories, at the University of California, San Diego. Her research investigates molecular pathways leading to heart failure, with the ultimate goal to design novel approaches for the prevention and treatment of cardiac dysfunction.

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