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Opinion Article

SLCO1B1 Polymorphisms and Statin-Induced Myopathy: A Clinical Pharmacogenomic

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DESCRIPTION

Statins are the most widely prescribed class of lipid-lowering medications for the primary and secondary prevention of cardiovascular disease. While generally well-tolerated, a subset of patient's experience Statin-Associated Muscle Symptoms (SAMS), ranging from mild myalgia to severe rhabdomyolysis. This variability in drug response and toxicity has prompted investigations into genetic factors influencing statin pharmacokinetics. Among these, polymorphisms in the *SLCO1B1* gene, which encodes the hepatic transporter *OATP1B1*, have emerged as key determinants of statin disposition and toxicity.

Multiple pharmacogenomics studies have substantiated the link between the *SLCO1B1*5* polymorphism and statin-induced adverse effects. The SEARCH trial, one of the largest pharmacogenetic investigations in cardiovascular pharmacotherapy, demonstrated that individuals homozygous for the C allele had a 16-fold increased risk of simvastatin-induced myopathy compared to non-carriers. This finding has been replicated in various ethnic populations, including European, Asian, and African cohorts, although allele frequencies and effect sizes vary.

In Portuguese patients, recent data from the Gen Cardio study indicated a 14.3% frequency of the *5 allele, with a higher prevalence in statin-intolerant individuals. Furthermore, patients with the TC or CC genotype reported higher rates of muscle pain and discontinuation of therapy. This has direct clinical implications, as adherence to statin therapy is critical for long-term cardiovascular risk reduction, and early identification of at-risk individuals could prevent avoidable morbidity.

In response to accumulating evidence, the Clinical Pharmacogenetics Implementation Consortium (CPIC) has published guidelines recommending *SLCO1B1* genotyping for patients prior to initiating simvastatin, particularly at high doses. For patients with decreased or poor function alleles, lower doses or alternative statins such as pravastatin or rosuvastatin are advised due to their reduced dependence on *OATP1B1*-mediated transport.

Despite the clarity of the genotype-phenotype relationship, clinical implementation of *SLCO1B1* testing remains limited. Barriers include lack of reimbursement policies, clinician unfamiliarity with genetic results, and the perception that alternative statins can be trialed without the need for genetic confirmation. Nonetheless, institutions such as the University of Porto Medical Center have begun integrating preemptive *SLCO1B1* testing into their cardiovascular clinics, with early reports showing a decrease in statin discontinuation rates and improved patient satisfaction.

Emerging research is also exploring the broader implications of *SLCO1B1* polymorphisms beyond statins. The transporter is involved in the hepatic uptake of various other drugs, including certain antidiabetics, antivirals, and chemotherapeutics. Thus, *SLCO1B1* genotyping could evolve into a multipurpose pharmacogenomics marker, especially in patients receiving polypharmacy.

Pharmacoproteomic analyses are further enhancing our understanding of *OATP1B1* expression and function. Mass spectrometry-based assays are now capable of quantifying transporter abundance in liver biopsy samples, revealing inter individual variability that sometimes exceeds that predicted by genotype alone. These findings suggest that integrating proteomic data with genotyping could refine risk stratification for SAMS and guide more nuanced clinical decisions.

It is also important to consider the pharmacoeconomic implications of implementing *SLCO1B1* testing. Simulation models suggest that genotyping, when applied selectively to patients starting high-dose simvastatin or with prior intolerance, is cost-effective and may reduce long-term healthcare expenditures by avoiding hospitalizations due to rhabdomyolysis or emergency visits for muscle-related complaints.

In summary, *SLCO1B1* polymorphisms, particularly the *5 variant, play a pivotal role in determining individual susceptibility to statin-induced myopathy. Incorporating

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SLCO1B1 genotyping into clinical practice offers a tangible opportunity to improve the safety and tolerability of statin therapy, thereby enhancing adherence and clinical outcomes. As the field of pharmacogenomics matures, transporter polymorphisms such as *SLCO1B1* should be considered essential components of personalized cardiovascular medicine.