TEST ID: SLC1B
SOLUTE CARRIER ORGANIC ANION TRANSPORTER FAMILY MEMBER 1B1 (SLCO1B1) GENOTYPE, STATIN, BLOOD

USEFUL FOR
- Aiding risk prediction for statin-associated myopathy in patients beginning statin therapy, especially simvastatin therapy
- Determining a potential genetic effect related to statin intolerance in patients with statin-associated myopathy, especially related to simvastatin

CLINICAL INFORMATION

SLCO1B1 encodes the organic anion-transporting polypeptide 1B1 (OATP1B1) influx transporter located on the basolateral membrane of hepatocytes. OATP1B1 facilitates the hepatic uptake of statins as well as other endogenous compounds (eg, bilirubin). Changes in the activity of this transporter (eg, through genetic variations or drug-drug interactions) can increase the severity of statin-associated myopathy (ie, statin intolerance).1

The most common adverse drug reaction associated with statins is skeletal muscle toxicity, which can include myalgia (with and without elevated creatine kinase levels), muscle weakness, muscle cramps, myositis, and rhabdomyolysis.2 Rhabdomyolysis, while rare, is of clinical concern because of the risk for death as a result of cardiac arrhythmia, renal failure, and disseminated intravascular coagulation. While the underlying causes of statin-associated myopathy are not known, several hypotheses have been formulated, including those related to the biochemical pathway of cholesterol synthesis inhibition and statin metabolism.

The SLCO1B1*5 (c.521T>C, p.V174A; rs4149056) allele interferes with localization of the transporter to the plasma membrane, and can lead to increased systemic statin concentrations.3 All statins are substrates of OATP1B1, but the association with SLCO1B1*5 and statin intolerance varies depending on statin and dose, and is most pronounced with higher doses of simvastatin therapy. A case-control study of simvastatin-induced myopathy observed an odds ratio (OR) for myopathy of 4.5 per copy of the *5 allele in patients receiving high-dose (80 mg/day) simvastatin therapy (the OR was 16.9 in *5 homozygotes compared to individuals who did not carry *5).4 Also demonstrated was a dose relationship in a replication cohort of patients taking 40 mg/day simvastatin with a relative risk of 2.6 per copy of the *5 allele. While SLCO1B1 genotype has been shown to affect systemic exposure of other statins (eg, atorvastatin, pravastatin, rosuvastatin) in addition to simvastatin,3 there is less evidence demonstrating a clinical association between SLCO1B1 genotype and myopathy with statins other than simvastatin.1

Frequency of the SLCO1B1*5 allele varies across different racial and ethnic groups. The *5 allele occurs in the homozygous or heterozygous state in approximately 20% to 28% of Caucasians and Asians, and 8% of Africans.

MOBILE APPS FROM MAYO MEDICAL LABORATORIES

Lab Catalog for iPad and Lab Reference for iPhone and iPod Touch
Requires iOS 5.1+

REFERENCE VALUES

An interpretive report will be provided.

ANALYTIC TIME

1 day (Not reported on Saturday or Sunday)

CONTENT AND VALUES SUBJECT TO CHANGE. SEE THE MAYO MEDICAL LABORATORIES TEST CATALOG FOR CURRENT INFORMATION.
**INTERPRETATION**

Heterozygosity and homozygosity for the \( SLCO1B1^*5 \) allele is associated with decreased organic anion-transporting polypeptide 1B1 (OATP1B1) activity and an increased risk for simvastatin-associated myopathy.

Absence of the \( SLCO1B1^*5 \) allele decreases, but does not rule-out, the risk of simvastatin-associated myopathy. For additional information regarding pharmacogenomic genes and their associated drugs, please see the Pharmacogenomic Associations Tables in Special Instructions. This resource also includes information regarding enzyme inhibitors and inducers, as well as potential alternate drug choices.

**CLINICAL REFERENCE**