Factor V Leiden and Factor II

Factor II and V are blood clotting proteins, and alterations in either can increase the chance of developing dangerous cardiovascular events caused by venous thrombosis. Healthcare providers should strongly consider testing patients for these alterations if they have:

- A first-time venous thrombosis.
- Recurring venous thrombosis
- Family history of venous thrombosis
- Venous thrombosis while pregnant
- Venous thrombosis before the age of 50
- Venous thrombosis in an unusual site
- If the patient is taking medications which increase the risk for blood clots

APOE

The APOE gene encodes instructions for lipid transport through the bloodstream. Changes to the APOE e3 allele are found in 25% of the Caucasian populations and are highly associated with cardiovascular disease.

Statins are typically prescribed in cases of high cholesterol and triglyceride levels to decrease the chance of developing cardiovascular disease. Genetic changes in the APOE gene may influence how patients respond to these medications. Thus, pharmacogenetic testing may be of use when deciding if statins would be beneficial to patient treatment plans.

PLEASE CALL OR SEND US AN EMAIL TODAY FOR MORE INFORMATION AND TO ORDER SAMPLE TEST KITS.

References

1. http://emedicine.medscape.com/article/1733331-overview#a2
2. http://www.mayomedicallaboratories.com/articles/features/cyp2c19/index.html
3. Wilcken B, Bamforth F, Li Z, et al. Geographical and ethnic variation of the 677C>T allele of 5,10 methylenetetrahydrofolate reductase (MTHFR):
4. Findings from over 7000 newborns from 16 areas worldwide. J Med Genet 2003; 40(8):619–25. Accessed 8/23/2016.

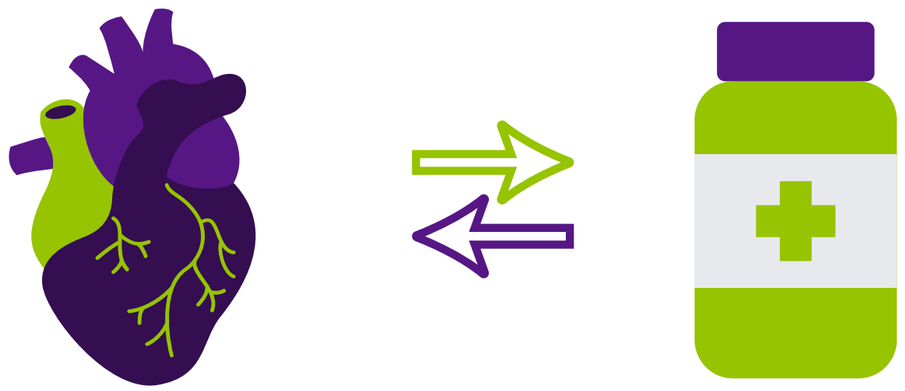
GENESYS Diagnostics INC.

CAP ACCREDITED COLLEGE of AMERICAN PATHOLOGISTS

Clinical Laboratory Improvement Amendments

PHARMACOGENETICS IN CARDIOLOGY

Precision Prescribing



Genesys Diagnostics Inc. provides one of the most comprehensive pharmacogenetic panels available. This panel identifies genetic mutations with direct adverse implications for cardiological medications. This information is essential for healthcare providers when prescribing medications or making changes to a patient's treatment plan.

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ACHIEVE OPTIMAL PRESCRIBING

Healthcare providers can personalize drug therapy by identifying a patient's drug metabolizing status for improved efficacy and a reduced number of adverse drug events (ADEs). Studies have shown that genetic mutations in the APOE and MTHFR genes may be key components to the development of hypertension and abnormal blood lipid levels.

Many medications used to treat cardiac conditions are metabolized by five novel genes: CYP2D6, CYP2C9, CYP2C19, CYP3A4, and CYP3A5. Determining patients' genotypes is crucial to determining how they may react to certain medications and developing individualized personal treatment plans.

CARDIOVASCULAR PATIENT PANELS

Cardiovascular Panel	APOE, CYP2C9, CYP2C19, CYP2D6, CYP3A4, CYP3A5, Factor II, Factor V, MTHFR, SLCO1B1, VKORC1
Comprehensive Panel	ABCB1, APOE, COMT, CYP1A2, CYP2B6, CYP2C9, CYP219, CYP2D6, CYP3A4, CYP35, DRD2, Factor II, Factor V, GLP1R, MTHFR, OPRM1, PNPLA5, SLCO1B1, SULT4A1, VKORC1

Genesys tests for all clinically significant genetic variants in the genes responsible for metabolizing most medications: CYP3A4, CYP3A5, CYP2C9, CYP2C19, CYP2D6, and VKORC1.

Coumadin (Warfarin) Dosing

Genetic alterations detected by these tests are found in approximately 50% of patients. Standard doses in patients with specific alterations may lead to adverse outcomes because of their sensitivity to the drug [1]. Changes to the CYP2C9 gene, which is responsible for the metabolism of Coumadin, and the VKORC1 gene, which is responsible for activating the pathway that forms blood clots, may influence how an individual responds to this medication. Adverse effects associated with Coumadin may include minor wounds that will not stop bleeding, major bleeding, and fatal hemorrhage. To accurately measure and establish a pattern in blood clotting time of patients on Coumadin, pharmacogenetic testing may be necessary.

Plavix (Clopidogrel Bisulfate) Dosing

Plavix does not have its antiplatelet effects until it is metabolized into its active form by CYP2C19. The CYP2C19 gene may have changes that prevent the gene from functioning properly. In these cases, individuals may not benefit from taking Plavix as they may be unable to convert Plavix into the active form. These "poor metabolizers" may not receive the full benefit of Plavix treatment and may still have a higher risk of heart attack, stroke, and cardiovascular death. It is estimated that 2-14% of the US population are poor metabolizers [2].

Beta-Blockers

The CYP2D6 gene is responsible for metabolizing more than 25% of all medications, including beta-blockers. Certain changes to this gene may have a significant effect on how individuals metabolize these medications. Changes to the CYP2D6 gene may cause an inability to metabolize the standard dose of beta-blockers, which can lead to a build up of un-metabolized medication in the bloodstream. These genetic changes may put patients at an increased risk to have adverse drug effects, particularly in patients taking multiple medications. Pharmacogenetic testing should be performed prior to prescribing beta-blockers to determine if the patient is able to metabolize beta-blockers properly.

MTHFR

Roughly 33% of Americans carry one copy of a common MTHFR gene mutation [3]. As each person has 2 copies of the MTHFR gene, people can inherit 1 copy of the MTHFR mutation or 2 copies (one from each parent). People who inherit 2 copies of a common MTHFR gene mutation may have an increased chance to develop conditions such as coronary artery disease, blood clots, and stroke compared to those without these changes [4]. Coronary artery disease, blood clots, and stroke are all complex conditions and are caused by a combination of both genetic and environmental factors.