Patient ID	Patient Name		Birth Date	Sex	Age
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Antidepressants

⚠ Pharmacogenomic Interactions

None

✓ Standard Prescription Recommended

amitriptyline *Elavil*
amoxapine *Asendin*
bupropion *Wellbutrin*
citalopram *Celexa*
clomipramine *Anafranil*
desipramine *Norpramin*
doxepin *Sinequan*
duloxetine *Cymbalta*
escitalopram *Lexapro*
fluoxetine *Prozac*
fluvoxamine *Luvox*
imipramine *Tofranil*
levomilnacipran *Fetzima*
maprotiline *Ludiomil*
mirtazapine *Remeron*
nortriptyline *Pamelor*
paroxetine *Paxil*
protriptyline *Vivactil*
sertraline *Zoloft*
trazodone *Desyrel*
trimipramine *Surmontil*
venlafaxine *Effexor*
vilazodone *Viibryd*
vortioxetine *Trintellix*

Insufficient Pharmacogenomic Evidence

brexanolone *Zulresso*
desvenlafaxine *Pristiq*
ketamine *esketamine, Spravato*
milnacipran *Savella*
nefazodone *Serzone*

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Antipsychotics

⚠ Pharmacogenomic Interactions

None

✓ Standard Prescription Recommended

aripiprazole *Abilify*
asenapine *Saphris*
brexpiprazole *Rexulti*
cariprazine *Vraylar*
chlorpromazine *Thorazine*
clozapine *Clozaril*
haloperidol *Haldol*
iloperidone *Fanapt*
lurasidone *Latuda*
olanzapine *Zyprexa*
perphenazine *Trilafon*
pimozide *Orap*
quetiapine *Seroquel*
risperidone *Risperdal*
thioridazine *Mellaril*
ziprasidone *Geodon*

Insufficient Pharmacogenomic Evidence

fluphenazine *Prolixin*
loxapine *Adasuve, Loxitane*
paliperidone *Invega*
thiothixene *Navane*
trifluoperazine *Stelazine*

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Anxiolytics And Hypnotics

⚠ Pharmacogenomic Interactions

None

✓ Standard Prescription Recommended

alprazolam *Xanax*
buspirone *BuSpar*
clonazepam *Klonopin*
diazepam *Valium*
eszopiclone *Lunesta*
lorazepam *Ativan*
oxazepam *Serax*
triazolam *Halcion*
zolpidem *Ambien*

Insufficient Pharmacogenomic Evidence

chlordiazepoxide *Librium*
clorazepate *Tranxene*
flurazepam *Dalmane*
temazepam *Restoril*

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Mood Stabilizers

⚠ Pharmacogenomic Interactions

None

✓ Standard Prescription Recommended

carbamazepine *Tegretol*

oxcarbazepine *Trileptal*

Insufficient Pharmacogenomic Evidence

gabapentin *Neurontin*

lamotrigine *Lamictal*

lithium *Eskalith*

topiramate *Topamax*

valproic acid/divalproex *Depakote*

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Other Medications

⚠ Pharmacogenomic Interactions

nicotine replacement therapy

✓ Standard Prescription Recommended

amphetamine
atomoxetine *Strattera*
buprenorphine *Buprenex, Butrans, Probuphine*
bupropion *Wellbutrin*
deutetrabenazine *Austedo*
lofexidine *Lucemyra*
methadone *Methadose, Diskets, Dolophine*
naltrexone *Revia, Vivitrol*
valbenazine *Ingrezza*

Insufficient Pharmacogenomic Evidence

acamprosate *Campral*
benztropine *Cogentin*
disulfiram *Antabuse*
varenicline *Chantix*

⚠ Pharmacogenomic Interaction Details

Medication	Comment
nicotine replacement therapy	Individuals with this CHRNA3 genotype have been reported to demonstrate comparable likelihood of end-of-treatment abstinence when treated with nicotine replacement therapy as compared to placebo; however, data are limited and other genetic and clinical factors may influence an individual's likelihood of smoking cessation.

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Genotype Findings

Analyte	Result	Performing Site
ADRA2A rs1800544 Genotype	G/G This individual is negative for the c.–1252G>C (rs1800544) variant, which is located in a transcription factor binding site in the 5'UTR of the ADRA2A gene and may impact gene expression.	MCR
ANKK1 rs1800497 Genotype	G/G This individual is negative for the c.2137G>A (rs1800497) variant. Although this variant (which this patient does not have) is in the ANKK1 gene, it is associated with the TaqIA polymorphism of the dopamine D2 receptor gene and a reduced number of dopamine binding sites in the brain.	MCR
CHRNA3 rs1051730 Genotype	C/C This individual is negative for the c.645C>T (rs1051730) variant. This variant (which this patient does not have) is in in tight linkage disequilibrium with rs16969968 variant in CHRNA5 among multiple populations that have been studied, and may be associated with CHRNA5 expression and function.	MCR
COMT rs4680 Genotype	G/G This individual is negative for the c.472G>A (rs4680) variant and is therefore expected to be a normal metabolizer (Val/Val).	MCR
CYP1A2 Genotype	*1/*1	MCR
CYP1A2 Phenotype	Normal (extensive) metabolizer This individual is expected to metabolize CYP1A2 substrates normally.	MCR
CYP2B6 Genotype	*1/*1	MCR
CYP2B6 Phenotype	Normal (extensive) metabolizer This individual is expected to metabolize CYP2B6 substrates normally.	MCR
CYP2C19 Genotype	*1/*1	MCR
CYP2C19 Phenotype	Normal (extensive) metabolizer With prodrugs that are activated by CYP2C19, normal drug activation by CYP2C19 is expected to occur. With drugs that are inactivated by CYP2C19, normal inactivation is expected.	MCR
CYP2C9 Genotype	*1/*1	MCR
CYP2C9 Phenotype	Normal (extensive) metabolizer This patient has a genotype associated with normal CYP2C9 enzymatic activity.	MCR
CYP2C9 Activity Score	2.00	MCR

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Analyte	Result	Performing Site
CYP2D6 Genotype	*1/*1	MCR
CYP2D6 Phenotype	Normal (extensive) metabolizer Prodrugs are converted to their active metabolite at a normal rate and drugs that are inactivated by CYP2D6 are metabolized at a normal rate.	MCR
CYP2D6 Activity Score	2.00	MCR
CYP3A4 Genotype	*1/*1	MCR
CYP3A4 Phenotype	Normal (extensive) metabolizer Normal metabolism of drugs that are inactivated or activated by CYP3A4 is expected.	MCR
CYP3A5 Genotype	*3/*3	MCR
CYP3A5 Phenotype	Poor metabolizer Note: This is the most common phenotype among Caucasian individuals. While this individual is expected to have minimal, if any, CYP3A5-mediated metabolism, standard dosing may be appropriate for many medications metabolized by CYP3A5. Original dosing recommendations for some CYP3A5-metabolized medications were determined based on populations primarily comprised of CYP3A5 poor metabolizers (i.e. CYP3A5*3/*3). Therapeutic drug monitoring is recommended for applicable medications.	MCR
DRD2 rs1799978 Genotype	A/A This individual is negative for the c.–585A>G (rs1799978; also known as -241A>G) variant, which is located in the DRD2 promoter region. The DRD2 gene encodes the D2 subtype of the dopamine receptor.	MCR
EPHX1 rs2234922 Genotype	A/A This individual is negative for the c.416A>G (rs2234922) EPHX1 variant, suggesting that normal EPHX1-related metabolism is expected; however, the influence of EPHX1 variants on enzyme activity may be substrate-specific.	MCR
GRIK4 rs1954787 Genotype	T/T This individual is negative for the c.83–10039T>C (rs1954787) variant in the GRIK4 gene, which encodes the kainic acid-type glutamate receptor KA1. This variant (which this individual does not have) disrupts a putative enhancer element and may impact gene expression.	MCR
HLA-A*31:01 Genotype	Negative This individual is negative for the HLA-A*31:01 allele.	MCR
HLA-B*15:02 Genotype	Negative This individual is negative for the HLA-B*15:02 allele.	MCR

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Analyte	Result	Performing Site
HTR2A rs7997012 Genotype	T/T This individual is negative for the c.614–2211T>C (rs7997012) variant. The HTR2A gene encodes the 5-hydroxytryptamine (serotonin) receptor 2A.	MCR
HTR2C rs3813929 Genotype	C/C This individual is negative for the c.–759C>T (rs3813929) variant. The HTR2C gene encodes the 5-hydroxytryptamine (serotonin) receptor 2C.	MCR
HTR2C rs1414334 Genotype	C/C This individual is negative for the c.551–3008C>G (rs1414334) variant. The HTR2C gene encodes the 5-hydroxytryptamine (serotonin) receptor 2C. Of note, the C allele is considered the ancestral allele, but in most populations G is more common at this locus.	MCR
MTHFR Genotype	677CC; 1298AA This individual is negative for both the c.665C>T (also known as 677; rs1801133) and the c.1286A>C (also known as 1298; rs1801131) variants and is expected to have normal MTHFR enzyme activity. Given the lack of evidence to support the use of MTHFR genotype to determine treatment plan, this result does not exclude supplementation with L-methylfolate or folic acid as a potential treatment consideration.	MCR
OPRM1 rs1799971 Genotype	A/A This individual is negative for the c.118A>G (rs1799971) variant, suggesting that this individual displays normal cell surface mu-opioid receptor expression.	MCR
SCN1A rs3812718 Genotype	G/G This individual is negative for the c.603–91G>A (rs3812718, also known as IVS5–91G>A) variant. This gene encodes the sodium voltage-gated channel alpha subunit 1.	MCR
SLC6A4 5HTTLPR Genotype	L/L This individual is homozygous for the long promoter polymorphism allele of the serotonin transporter gene. The long promoter allele is reported to express normal levels of the serotonin transporter.	MCR
UGT2B15 rs1902023 Genotype	G/G This individual is negative for the c.253G>T (rs1902023) variant and is expected to metabolize UGT2B15 substrates at a normal rate.	MCR

Interpretation

MCR

Gene specific results and interpretation provided above.

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Additional Information

MCR

Medications and recommendations included in this report are based on the Clinical Pharmacogenetics Implementation Consortium Guidelines www.cpicpgx.org/guidelines/, information publicly available in PharmGKB www.pharmgkb.org , Dutch Pharmacogenetics Working Group Recommendations www.pharmgkb.org , FDA medication labels, and available literature. For additional information regarding pharmacogenomic genes and their associated drugs, please see the Pharmacogenomic Associations Tables on the Mayo Clinic Laboratories webpage,https://www.mayocliniclabs.com/it-mmfiles/Pharmacogenomic_Associations_Tables.pdf . This resource also includes information regarding enzyme inhibitors and inducers, as well as potential alternate drug choices. Please note that the information at this link is educational material intended for health care professionals and may not be comprehensive. This educational material is not intended to supersede the care provider's experience and knowledge of her/his patient to establish a diagnosis or a treatment plan. All medications require careful clinical monitoring. Please contact the laboratory at 1–800–533–1710 for further information about pharmacogenomic testing. For additional information on star allele nomenclature for the Cytochrome P450 enzymes, see the Pharmacogene Variation Consortium website www.pharmvar.org.

Method

MCR

Genotyping is performed using a PCR-based 5'-nuclease assay. Fluorescently labeled detection probes anneal to the target DNA. PCR is used to amplify the segment of DNA that contains the polymorphism. If the detection probe is an exact match to the target DNA, the 5'-nuclease polymerase degrades the probe, the reporter dye is released from the effects of the quencher dye, and a fluorescent signal is detected. Genotypes are assigned based on the allele-specific fluorescent signals that are detected. (TaqMan SNP Genotyping Assays User Guide, Applied Biosystems)

For SLC6A4, PCR amplification is performed for the region surrounding the polymorphism and is followed by size separation of the products. (Lesch KP, Bengel D, Heils A, et al: Association of anxiety-related traits with a polymorphism in the serotonin transporter gene regulatory region. Science 1996;274:1527–1530)

Amplification for the HLA-B*15:02 and HLA-A*31:01 alleles and an internal control gene is performed by real-time PCR in the presence of SYBR Green, which fluoresces when bound to double-stranded DNA. A genotype is assigned based on the allele-specific SYBR Green fluorescent signals that are detected. (Unpublished Mayo method)

Disclaimer

1 MCR

A combination of a polymerase chain reaction (PCR)-based 5'-nuclease assay using fluorescently labeled detection probes, PCR amplification followed by size separation of the products, and real-time PCR in the presence of SYBR Green was performed to test for the presence or absence of the following specific pharmacogenomic variants:
ADRA2A rs1800544
ANKK1 rs1800497
CHRNA3 rs1051730
COMT rs4680
CYP1A2 *1F, *1K, *6, and *7
CYP2B6 *4, *5, *6, *7, *8, *9, *11, *12, *13, *14, *15, *16, *18, *19, *20, *22, *23, *25, *26, *27, *28, *35, *36, and *38 CYP2C9 *2, *3, *4, *5, *6, *8, *9, *11, *12, *13, *14, *15, *16, *17, *18, *25, *26, *28, *30, *33, and *35 CYP2C19 *2, *3, *4, *5, *6, *7, *8, *9, *10, *17, and *35 CYP2D6 *2, *3, *4, *4N, *5, *6, *7, *8, *9, *10, *11, *12, *13, *14,*15, *17, *29, *35, *36, *41, *59, *68, *114, and CYP2D6 gene duplication. Additional CYP2D6 variants may be detected through the cascade testing process. CYP3A4 *8, *11, *12, *13, *16, *17, *18, *22, and *26
CYP3A5*3, *6, *7, *8, and *9
DRD2 rs1799978
EPHX1 rs2234922
GRIK4 rs1954787
HLA-A*31:01
HLA-B*15:02
HTR2A rs7997012
HTR2C rs3813929 and rs1414334
MTHFR rs1801133 (677C>T) and rs1801131 (1298A>C)
OPRM1 rs1799971
SCN1A rs3812718
SLC6A4 5HTTLPR promoter genotype
UGT2B15 rs1902023
A two-tiered testing approach was used to identify CYP2D6

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variants (including copy number variants). Tier 1 (always performed) consists of targeted variant analysis and analysis of copy number variation (CNV) using CYP2D6-specific probes for the promoter, intron 6 and exon 9. Detectable CNVs include duplications, multiplications and deletions (*5) of CYP2D6, as well as CYP2D6::CYP2D7 (i.e. *4N, *36, *68) and CYP2D7::CYP2D6 (i.e. *13) hybrid genes. Tier 2, which is performed if the Tier 1 testing results in an ambiguous phenotype, consists of comprehensive Sanger sequencing of the CYP2D6 gene and/or CYP2D7::CYP2D6 and CYP2D6::CYP2D7 hybrid genes. For additional clinical information, see Cytochrome P450 2D6 (CYP2D6) Comprehensive Cascade (test ID 2D6Q).

CAUTIONS:

CLINICAL CORRELATIONS

This test is not designed to provide specific dosing recommendations and is to be used only as an aid to clinical decision making. Drug-label guidance should be used when dosing patients with medications regardless of the predicted phenotype. Test results should be interpreted in the context of clinical findings, family history, and other laboratory data.

TECHNICAL LIMITATIONS

Pharmacological phenotype prediction and interpretation is based on the limited dataset of the specific variants/alleles interrogated. This test will not detect all variants that may cause abnormal drug metabolism or drug response. Rare variants may be present that could lead to false negative or positive results. The methods used may not identify all inactive alleles or the precise configuration of variants (or alleles in the case of CYP2D6) on a patient's chromosomes; therefore, this lab will report the most likely diplotype based upon reported or observed frequencies. Note that this ambiguity can be clarified by family studies, if desired. If results obtained do not match the clinical findings (phenotype), additional testing should be considered.

Assignment of a *1 allele indicates that none of the alleles of interest in that gene were detected, but does not rule out the presence of other variants in the gene. For additional information regarding the standard Human Genome Variation Society (HGVS) nomenclature associated with these variants, please see the Mayo Clinic Laboratories test catalog for this test.

For the HLA portion of this test, alleles known to potentially interfere include other HLA-A*31 alleles (including HLA-A*31:01:23), HLA-B*15:13, HLA-B*15:31, HLA-B*15:55, HLA-B*15:88, HLA-B*15:89, HLA-B*18:20, HLA-B*15:112, HLA-B*15:121, HLA-B*15:144, and HLA-B*15:170. However, most of these alleles are rare, and it is not known if any of these related alleles are associated with drug-associated cutaneous adverse reactions.

Samples may contain donor DNA if obtained from patients who received non-leukoreduced blood transfusions or allogeneic hematopoietic stem cell transplantation. Results from samples obtained under these circumstances may not accurately reflect the recipient's genotype. For individuals who have received blood transfusions, the genotype usually reverts to that of the recipient within 6 weeks. For individuals who have received allogeneic hematopoietic stem cell transplantation, a pre-transplant DNA specimen is recommended for testing. Genetic test results in patients who have undergone liver transplantation may not accurately reflect the patient's genetic status for some genes on this panel.

Reviewed by MCR

Ann Marie Moyer, M.D., Ph.D.

Received: 15 Apr 2025 10:57 **Reported:** 16 Apr 2025 17:52

Laboratory Notes

1 This test was developed and its performance characteristics determined by Mayo Clinic in a manner consistent with CLIA requirements. This test has not been cleared or approved by the U.S. Food and Drug Administration.

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