



MEDICAL POLICY

MEDICAL POLICY DETAILS	
Medical Policy Title	GENOTYPING - CYTOCHROME P450 (CYP450) FOR DRUG METABOLISM
Policy Number	2.02.30
Category	Laboratory Tests
Effective Date	12/20/07
Revised Date	12/18/08, 12/17/09, 02/17/11, 12/15/11, 12/20/12, 12/19/13, 12/18/14, 02/18/16, 02/16/17, 01/18/18, 02/21/19, 2/20/20
Product Disclaimer	<ul style="list-style-type: none"> • If a product excludes coverage for a service, it is not covered, and medical policy criteria do not apply. • If a commercial product (including an Essential Plan product) or a Medicaid product covers a specific service, medical policy criteria apply to the benefit. • If a Medicare product covers a specific service, and there is no national or local Medicare coverage decision for the service, medical policy criteria apply to the benefit.

POLICY STATEMENT

- I. Based upon our criteria and review of peer-reviewed literature, cytochrome p450 (CYP450) CYP2D6 genotyping to determine drug metabolizer status is considered **medically necessary** for patients with either of the following:
 - A. Gaucher type I disease being considered for treatment with eliglustat; OR
 - B. Huntington disease being considered for treatment with tetrabenazine in a dosage greater than 50 mg per day.
- II. Based upon our criteria and review of peer-reviewed literature, cytochrome p450 (CYP450) CYP2C19 genotyping is considered **investigational** for the purpose of aiding in the choice of clopidogrel versus alternative anti-platelet agents, or in decisions on the optimal dosing for clopidogrel.
- III. Based upon our criteria and review of peer-reviewed literature, genotyping to determine cytochrome p450 (CYP450) genetic polymorphisms for the purpose of aiding in the choice of drug or dose to increase efficacy and/or avoid toxicity is considered **investigational**. This includes, but is not limited to, CYP450 genotyping for the following applications:
 - A. selection or dosing of selective serotonin reuptake inhibitors (SSRI);
 - B. selection or dosing of antipsychotic drugs;
 - C. selection and dosing of selective norepinephrine reuptake inhibitors;
 - D. selection and dosing of tricyclic antidepressants;
 - E. dosing of efavirenz (common component of highly active antiretroviral therapy for human immunodeficiency virus (HIV) infection);
 - F. deciding whether to prescribe codeine for nursing mothers;
 - G. dosing of immunosuppressant for organ transplantation; and
 - H. selection or dose of beta blockers (e.g., metoprolol).
- IV. Based upon our criteria and review of peer-reviewed literature, the use of genetic testing panels that include multiple CYP450 mutations is considered **investigational**.

Refer to Corporate Medical Policy # 2.02.33 regarding Genotyping - Cytochrome p450 2C9 (CYP2C9) and Vitamin K Epoxide Reductase subunit CI (VKORC) that Effect Response to Warfarin.

Refer to Corporate Medical Policy #11.01.03 regarding Experimental and Investigational Services.

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POLICY GUIDELINES

The Federal Employee Health Benefit Program (FEHBP/FEP) requires that procedures, devices, or laboratory tests approved by the U.S. Food and Drug Administration (FDA) may not be considered investigational, and thus, these procedures, devices, or laboratory tests may be assessed only on the basis of their medical necessity.

DESCRIPTION

Drug efficacy and toxicity vary substantially across individuals. Because drugs and doses are typically adjusted, if needed, by trial and error, clinical consequences may include a prolonged time to optimal therapy. In some cases, serious adverse events may result. Various factors may influence the variability of drug effects, including age, liver function, concomitant diseases, nutrition, smoking, and drug-drug interactions. Inherited (germline) DNA sequence variation (polymorphisms) in genes coding for drug metabolizing enzymes, drug receptors, drug transporters, and molecules involved in signal transduction pathways may also have major effects on the efficacy or toxicity of a drug.

The cytochrome p450 (CYP450) family is a major subset of all drug-metabolizing enzymes; several CYP450 enzymes are involved in the metabolism of a significant proportion of currently administered drugs. Some CYP450 enzyme genes are highly polymorphic, resulting in some enzyme variants that have variable metabolic capacities among individuals, and some with little to no impact on activity. Thus, CYP450 enzyme variants constitute one important group of drug-gene interactions influencing the variability of effect of some CYP450 metabolized drugs.

Individuals with two copies (alleles) of the most common DNA sequence of a particular CYP450 enzyme gene are termed extensive metabolizers. Poor metabolizers lack active enzyme gene alleles and intermediate metabolizers, who have one active and one inactive enzyme gene allele, may suffer to a lesser degree some of the consequences of poor metabolizers. Ultrarapid metabolizers are individuals with more than two alleles of an active enzyme gene. There is pronounced ethnic variability in the population distribution of metabolizer types for a given CYP enzyme.

Ultrarapid metabolizers administered an active drug may not reach therapeutic concentrations at usual, recommended doses of active drugs, while poor metabolizers may suffer more adverse events at usual doses due to reduced metabolism and increased concentrations. Conversely, for administered prodrugs that must be converted by CYP450 enzymes into active metabolites, ultrarapid metabolizers may suffer adverse effects and poor metabolizers may not respond.

However, it is important to realize that many drugs are metabolized to varying degrees by more than one enzyme, either within or outside of the CYP450 superfamily. In addition, interaction between different metabolizing genes, interaction of genes and environment, and interactions among different non-genetic factors also influence CYP450-specific metabolizing functions. Thus, identification of a variant in a single gene in the metabolic pathway may be insufficient in all but a small proportion of drugs to explain inter-individual differences in metabolism and consequent efficacy or toxicity.

Diagnostic genotyping tests for some CYP450 enzymes are available. Some tests are offered as in-house laboratory services, which do not require FDA approval but which must meet general laboratory quality standards for high complexity testing. The AmpliChip® (Roche Molecular Systems, Inc.) is the only FDA-approved test and was cleared through the 510(k) process. The AmpliChip tests the DNA from a patient's white blood cells collected in a standard anticoagulated blood sample for 29 polymorphisms and mutations for the CYP2D6 gene and two (2) polymorphisms for the CYP2C19 gene.

Other tests include the GeneSight® Psychotropic test (AssureRx Health), the Millenium PGT® test (Millenium Health), and the Proove® Drug Metabolism, Opioid, Pain Perception test (Proove Biosciences), which analyze genes that may affect a patient's response to antidepressant, antipsychotic, anticonvulsant medications, and narcotics. The tests include genotyping the pharmacokinetic genes from the CYP450 family and other pharmacodynamic genes related specifically to a system such as, the serotonin system. Results of the testing are used by clinicians to guide therapy, determine response to therapy, and determine risk of adverse events from drug dosage.

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RATIONALE

Validation of genotyping to improve pharmacologic treatment outcomes is a multistep process. In general, important steps in the validation process address the following:

- I. Analytic validity which measures technical performance (e.g., whether the test accurately and reproducibly detect the gene markers of interest).
- II. Clinical validity which measures the strength of the associations between the selected genetic markers and dose, therapeutic efficacy, and/or adverse events.
- III. Clinical utility which determines whether the use of genotyping for specific genetic markers to guide prescribing and/or dosing improves patient outcomes such as therapeutic effect, time to effective dose, and/or adverse event rate compared to standard treatment without genotyping.

Further discussion of the validation process is provided in a 2004 BlueCross BlueShield Association Technology Evaluation Center (TEC) Special Report. The purpose of the report was to provide background information on CYP450 enzymes; genotyping applications for currently available drugs, examples of companies and products, evaluation of clinical utility, examples and the current state of evidence, regulatory issues, and cost-effectiveness analysis. The report offered the following observations and conclusions:

- I. Although a genotyping assay may be designed to determine metabolizer status for a variety of enzymes and need only be performed once per patient to generate results relevant to a variety of drugs, whether or not the information is relevant for a particular drug must be validated for each drug of interest. A pharmacogenomic approach is most likely to be beneficial when the drug has a narrow therapeutic dose range (or some patients are non-responders), when the consequences of treatment failure are severe, and/or when serious adverse reactions are more likely in patients with certain gene variants.
- II. The analytical validity of pharmacogenomic testing is likely to be high but should be evaluated for each marker of interest.
- III. In several cases there is good retrospective evidence to support clinical validity. Data suggest a strong association between specific variant alleles and increased adverse events related to specific drugs or between specific variant alleles and final doses for specific drugs that are significantly different from expected. But such associations may not explain the majority of inter-individual variability in drug response. For example, although CYP2C9 genotype is an independent predictor of final warfarin dose, genotype in combination with known significant confounders explains only 30%-40% of the variation in the final dose.
- IV. Reduced activity in a particular CYP450 enzyme because of genotype may not affect outcomes when other metabolic pathways are available and when other confounders influence drug metabolism. Therefore, prospective studies of clinical utility are important to validate hypotheses generated by associational studies.
- V. Without prospective evidence defining the effect of genotyping on such outcomes, there are few dosing recommendations based on genotype. In one example, Kirchheiner, et. al. (2004) reviewed CYP2D6 and CYP2C19 polymorphisms and pharmacokinetic data for several antidepressants and antipsychotics to provide dose recommendations for use in prospective clinical trials. In general, tricyclic antidepressant dose reductions to about 30% to 60% of standard were recommended for poor metabolizers and increases of up to 250% were recommended for ultrarapid metabolizers. SSRIs have a wider therapeutic range and generally fewer adverse events; maintenance doses of 60% to 100% of standard, depending on the drug, were recommended for poor metabolizers. However, these recommendations were largely extrapolated from data on genotype-dependent pharmacokinetics for use in future clinical trials; efficacy of the recommendations in routine clinical use has not been established.

Articles published since the TEC Special Report tend to be studies of the effect of variability in single genetic components in complex, multiple-component metabolic systems. Several studies show that a strong association of genotype with metabolizing enzyme activity does not necessarily lead to differences in clinical outcomes. For example:

- I. High variability in the metoprolol area under the plasma concentration-time curve was associated with CYP2D6 genotypes, but there was no association between CYP2D6 genotype and drug-associated adverse effects or antihypertensive response rate/blood pressure changes. (Zineh, et al. 2004)
- II. Cyclosporine oral clearance is correlated with CYP3A4 genotype, but the effect on cyclosporine dose requirement during maintenance therapy in transplant patients was small. (Hesselink, et al. 2004)

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- III. Among more than 2,300 children, adolescents, and adults participating in clinical trials of atomoxetine (Strattera), there were no differences between “normal” metabolizers (extensive metabolizers or EMs) and slow metabolizers (poor metabolizers or PMs) in serious adverse events experienced. While the FDA label describes associations of genotype with metabolizing enzyme activity, the FDA actively decided not to include a recommendation to perform genotyping prior to prescribing atomoxetine. Thus, genotyping for CYP2D6 poor metabolizers of atomoxetine is not recommended because the margin of safety is not exceeded and evidence to support guidelines for dosing such that patient outcomes are improved has not been collected.
- IV. A review of pharmacogenomic studies of antidepressants and antipsychotics (Kirchheiner, et al. 2004) concluded that it is not yet possible to translate pharmacogenetic parameters into therapeutic recommendations, and that multigenic interactions and gene-environment interactions will need to be considered.

Other studies show promising associations between genotype and clinical parameters or outcomes, but none studies prospective use of genotyping to direct patient management and determine effect on outcomes. For example:

- I. In transplant patients, tacrolimus has a narrow therapeutic window and consequences of severe toxicity or acute rejection on either side of the target blood concentration range. Daily dose required to achieve the target blood concentration varied significantly according to CYP3A5 genotype. (Tsuchiya, et al. 2004)
- II. CYP2D6 genotype is associated with risperidone adverse reactions and discontinuation due to adverse events. (de Leon, et al. 2005)
- III. Combination CYP2C9 and CYP2C19 genotypes subdivide patients into five groups for which recommended phenytoin dose ranges have been calculated; prospective verification of improved outcomes awaits study. (Hung, et al. 2004)

In January 2007, an Agency for Healthcare Research and Quality (AHRQ) report on CYP450 testing for adults treated with SSRIs for non-psychotic depression concluded “There is a paucity of good-quality data addressing the questions of whether testing for CYP450 polymorphisms in adults entering SSRI treatment for non-psychotic depression leads to improvement in outcomes, or whether testing results are useful in medical, personal, or public health decision making.”

On March 12, 2010, the FDA approved a new label for clopidogrel with a “boxed warning” about the diminished effectiveness of the drug in patients with impaired ability to convert the drug into its active form. The boxed warning is based on the concern that the antiplatelet effect of clopidogrel depends primarily on its activation by the CYP450 system. Patients with decreased CYP2C19 function because of genetic polymorphisms metabolize clopidogrel poorly and have higher rates of cardiovascular events after acute coronary syndrome (ACS) and percutaneous coronary interventions (PCIs) than patients with normal CYP2C19 function. The warning also notes that tests are available to identify patients with genetic polymorphisms, and that alternative treatment strategies should be considered in poor metabolizers of the drug.

In response to the FDA “Boxed Warning” for clopidogrel use, the American College of Cardiology Foundation Task Force on Clinical Expert Consensus Documents and the American Heart Association published a report - ACCF/AHA Clopidogrel Clinical Alert: Approaches to the FDA “Boxed Warning” (2010). The report was developed to help guide practitioners and patients in prescribing pharmacogenomic testing to identify patients with altered clopidogrel metabolism which has been shown to increase patient’s risk for a suboptimal clinical response to clopidogrel thus changing their treatment. The report emphasized that the FDA warning originated from a small unpublished crossover trial of 40 healthy patients receiving clopidogrel which evaluated pharmacokinetic and antiplatelet response. The chief findings were decreased active metabolite exposure and increased platelet aggregation in the poor metabolizers when compared with the other groups. Seven recommendations for practice were put forward. One recommendation was that careful clinical judgment is required to assess the importance of the variability in response to clopidogrel for an individual patient and its associated risk to the patient. It has been shown that the genetic variability in CYP causes variable response to clopidogrel however the specific impact of the individual genetic polymorphisms on clinical outcome remains to be determined. Information regarding the predictive value of pharmacogenomic testing is very limited at this time. Multiple studies are ongoing to address this issue. Until these studies are complete, the evidence base is insufficient to recommend either routine genetic or platelet function testing. There is no information that routine testing improves outcome in large subgroups of patients. In addition, the clinical course of the majority of patients treated with clopidogrel without either genetic testing or functional testing is excellent. Genetic testing to determine if a patient is predisposed to poor clopidogrel

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metabolism (“poor metabolizers”) may be considered before starting clopidogrel therapy in patients believed to be at moderate or high risk for poor outcomes. Patients believed to be at moderate or high risk for poor outcomes might include, among others, patients undergoing elective high-risk PCI procedures (e.g., treatment of extensive and/or very complex disease). For patients who experience poor response or adverse effects when taking clopidogrel, other treatment options are available such as using higher loading doses or switching for clopidogrel to prasugrel.

The evidence for testing for CYP2C19 metabolizer status by CYP2C19 genotyping in individuals with need for antiplatelet therapy who are undergoing or being considered for clopidogrel therapy includes one randomized controlled trial (RCT) of CYP2C19 genotype-directed antiplatelet therapy, observational studies and analyses or RCTs of clopidogrel therapy, and meta-analyses of these studies. Relevant outcomes include morbid events and treatment-related morbidity and mortality. Systematic reviews of observational studies report that genetic variants may be associated with a modest increase in the rate of stent thrombosis and clinical endpoints. CYP2C19 genotype has been associated with increased risk of thrombosis in patients with coronary disease or cardiac interventions being considered as candidates for clopidogrel treatment. This observation is most pronounced for stent thrombosis in patients undergoing percutaneous coronary intervention (PCI). The evidence addressing whether the use of CYP2C19 genotype-directed therapy improves outcomes is limited. One RCT comparing CYP2C19 genotype-directed antiplatelet therapy reported that patients receiving genotype-directed therapy had higher on treatment platelet reactivity. However, the effect on clinical endpoints is not well understood. The evidence is insufficient to determine the effects of the technology on health outcomes.

Gaucher disease is a rare autosomal recessive lipid storage disease in which deficiency or absence of the enzyme β -glucocerebrosidase leads to lysosomal accumulation of the glycosphingolipid glucosylceramide. Untreated, this accumulation can lead to a range of effects, including anemia and thrombocytopenia, splenomegaly, bone disease, pulmonary fibrosis, and central nervous system involvement. Gaucher disease has been treated through enzyme replacement or substrate reduction therapy. Eliglustat tartrate is an orally-administered selective inhibitor of glucosylceramide synthase. It received FDA approval in 2014; and, in three phase III clinical trials, has been found to lead to improvements in hematologic metrics and organomegaly. Eliglustat tartrate is metabolized by CYP2D6 and CYP3A. FDA labeling requires that patients be selected on the basis of CYP2D6 metabolizer status as determined by genotype, with recommendations based on genotype about dosage and concomitant use of CYP2D6 and CYP3A inhibitors.

Huntington’s Disease is an autosomal dominant genetic neurodegenerative disorder characterized by progressive cognitive and motor dysfunction, including chorea. In 2008, the FDA approved tetrabenazine as an orphan drug for the treatment of chorea in Huntington’s Disease based on RCT evidence of improved chorea symptoms in ambulatory patients with Huntington’s Disease. FDA labeling for tetrabenazine includes recommendations for genotyping for CYP2D6 for patients who are being considered for doses above 50 mg per day. The labeling states: “Patients requiring doses above 50 mg per day should be genotyped for the drug metabolizing enzyme CYP2D6 to determine if the patient is a poor metabolizer (PM) or an extensive metabolizer (EM). Maximum daily dose in poor metabolizers (PM): 50 mg with a maximum single dose of 25 mg. Maximum daily dose in EMs and intermediate metabolizers (IMs): 100 mg with a maximum single dose of 37.5 mg.

The FDA labeling for the orphan drugs eliglustat (for Gaucher disease) and tetrabenazine (for Huntington’s disease) recommends CYP2D6 genotyping before use. There is limited published evidence about outcome changes associated with genotype-directed therapy for these medications. However, given the FDA labeling and the high variation in drug exposure based on metabolizer status, genotyping of CYP2D6 to determine metabolizer status before the use of these drugs is reasonable and may be considered medically necessary.

The evidence for CYP450 genotyping in individuals with a variety of clinical conditions undergoing or being considered for treatment with a drug that is metabolized by CYP450 enzyme(s) includes prospective and retrospective observational studies reporting associations with CYP450 metabolizer status and medication response or adverse effects. Relevant outcomes include symptoms, change in disease status, morbid events, medication use, and treatment-related morbidity and mortality. Most published studies of CYP450 pharmacogenomics are retrospective evaluations of CYP450 genotype association with intermediate (e.g., circulating drug concentrations) or, less often, final outcomes (e.g., adverse events or efficacy) and are largely small and underpowered or not designed to examine the clinical effects of homozygous variant poor metabolizers (PMs) and of ultrarapid metabolizers (UM), where the strongest effects, if any, would be seen. The hazards associated with different metabolizer status are therefore uncertain. Decision-making regarding dose or

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medication selection changes in response to CYP450 metabolizer status is poorly defined, and outcome changes are uncertain. The evidence is insufficient to determine the effects of the technology on health outcomes.

CODES

- *Eligibility for reimbursement is based upon the benefits set forth in the member's subscriber contract.*
- **CODES MAY NOT BE COVERED UNDER ALL CIRCUMSTANCES. PLEASE READ THE POLICY AND GUIDELINES STATEMENTS CAREFULLY.**
- *Codes may not be all inclusive as the AMA and CMS code updates may occur more frequently than policy updates.*

CPT Codes

Code	Description
81225	CYP2C19 (cytochrome P450, family 2 subfamily C, polypeptide 19) (e.g., drug metabolism), gene analysis, common variants (e.g., *2, *3, *4, *8, *17)
81226	CYP2D6 (cytochrome P450, family 2, subfamily D, polypeptide 6 (e.g., drug metabolism), gene analysis, common variants (e.g., *2, *3, *4, *5, *6, *9, *10, *17, *19, *29, *35, *41, *1XN, *2XN, *4XN)
81227	CYP2C9 (cytochrome P450, family 2, subfamily C, polypeptide 9) (eg, drug metabolism), gene analysis, common variants (e.g., *2, *3, *5, *6)
81230	CYP3A4 (cytochrome P450 FAMILY 3 subfamily A member 4) (eg, drug metabolism), gene analysis, common variants (e.g., *2, *22)
81231	CYP3A5 (cytochrome P450 FAMILY 3 subfamily A member 5) (eg, drug metabolism), gene analysis, common variants(e.g., *2, *3, *4, *5, *6, *7)
81479	Unlisted molecular pathology procedure
81599	Unlisted multianalyte assay with algorithmic analysis
0070U	CYP2D6 (cytochrome P450, family 2, subfamily D, polypeptide 6) (eg, drug metabolism) gene analysis, common and select rare variants (ie, *2, *3, *4, *4N, *5, *6, *7, *8, *9, *10, *11, *12, *13, *14A, *14B, *15, *17, *29, *35, *36, *41, *57, *61, *63, *68, *83, *xN), (Mayo Clinic, Laboratory Developed Test)
0071U	CYP2D6 (cytochrome P450, family 2, subfamily D, polypeptide 6) (eg, drug metabolism) gene analysis, full gene sequence (List separately in addition to code for primary procedure), (Mayo Clinic, Laboratory Developed Test)
0072U	CYP2D6 (cytochrome P450, family 2, subfamily D, polypeptide 6) (eg, drug metabolism) gene analysis, targeted sequence analysis (ie, CYP2D6-2D7 hybrid gene) (List separately in addition to code for primary procedure) (Mayo Clinic, Laboratory Developed Test)
0073U	CYP2D6 (cytochrome P450, family 2, subfamily D, polypeptide 6) (eg, drug metabolism) gene analysis, targeted sequence analysis (ie, CYP2D7-2D6 hybrid gene) (List separately in addition to code for primary procedure), (Mayo Clinic, Laboratory Developed Test)
0074U	CYP2D6 (cytochrome P450, family 2, subfamily D, polypeptide 6) (eg, drug metabolism) gene analysis, targeted sequence analysis (ie, non-duplicated gene when duplication/multiplication is trans) (List separately in addition to code for primary procedure), (Mayo Clinic, Laboratory Developed Test)
0075U	CYP2D6 (cytochrome P450, family 2, subfamily D, polypeptide 6) (eg, drug metabolism) gene analysis, targeted sequence analysis (ie, 5' gene duplication/multiplication) (List separately in addition to code for primary procedure), (Mayo Clinic, Laboratory Developed Test)

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Code	Description
0076U	CYP2D6 (cytochrome P450, family 2, subfamily D, polypeptide 6) (eg, drug metabolism) gene analysis, targeted sequence analysis (ie, 3' gene duplication/multiplication) (List separately in addition to code for primary procedure) (Mayo Clinic, Laboratory Developed Test)

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HCPCS Codes

Code	Description
None	

ICD10 Codes

Code	Description
I20.0	Unstable angina
I21.01-I21.5	ST elevation (STEMI) and non-ST elevation (NSTEMI) myocardial infarction (code range)
I25.110-I25.799	Chronic ischemic heart disease (code range)
I63.40-I63.9	Cerebral infarction (code range)
I66.01-I66.9	Occlusion and stenosis of cerebral arteries, not resulting in cerebral infarction (code range)
I73.9	Peripheral vascular disease, unspecified

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KEY WORDS

Amplichip, CYP450, Cytochrome p450 Genotyping

CMS COVERAGE FOR MEDICARE PRODUCT MEMBERS

There is currently a Local Coverage Determination (LCD) for Molecular Pathology Procedures. Please refer to the following LCD website for Medicare Members: https://www.cms.gov/medicare-coverage-database/details/lcd-details.aspx?LCDId=35000&ver=131&CntrctrSelected=298*1&Cntrctr=298&s=41&DocType=2&bc=AAgAAAQBAA&AA&

There is currently no National Coverage Determination (NCD) or Local Coverage Determination (LCD) for the GeneSight® Psychotropic (AssureRx Health, Inc, Mason, OH) gene panel. However, effective 10/01/15 the Medicare

Medical Policy: GENOTYPING - CYTOCHROME P450 (CYP450) FOR DRUG METABOLISM

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Part-B carrier for Ohio, CGSAdministrators, LLC, established a favorable LCD for the GeneSight® Assay for Refractory Depression. This covers most of Medicare beneficiaries in all 50 states since the Assurex Health reference laboratory in Mason, Ohio, is within the sole jurisdiction of NHIC for purposes of Part-B coverage. Please refer to:

[https://www.cms.gov/medicare-coverage-database/details/lcd-details.aspx?LCDId=35443&ver=21&CntrctrSelected=+238*2&Cntrctr=238&name=+\(15202%2c+MAC+-+Part+B\)&s=42&DocType=Active&bc=AggAAAQBAAAA&](https://www.cms.gov/medicare-coverage-database/details/lcd-details.aspx?LCDId=35443&ver=21&CntrctrSelected=+238*2&Cntrctr=238&name=+(15202%2c+MAC+-+Part+B)&s=42&DocType=Active&bc=AggAAAQBAAAA&)