



# Medical Coverage Policy

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## Pharmacogenetic Testing

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### Related Coverage Resources

- [Inflammatory Bowel Disease - Testing for the Diagnosis and Management](#)
- [Lab Management Guidelines](#)
- [Laboratory Testing for Transplantation Rejection](#)
- [Laboratory Testing Services](#)
- [Molecular and Proteomic Diagnostic Testing for Hematology and Oncology Indications](#)

### INSTRUCTIONS FOR USE

The following Coverage Policy applies to health benefit plans administered by Cigna Companies. Certain Cigna Companies and/or lines of business only provide utilization review services to clients and do not make coverage determinations. References to standard benefit plan language and coverage determinations do not apply to those clients. Coverage Policies are intended to provide guidance in interpreting certain standard benefit plans administered by Cigna Companies. Please note, the terms of a customer’s particular benefit plan document [Group Service Agreement, Evidence of Coverage, Certificate of Coverage, Summary Plan Description (SPD) or similar plan document] may differ significantly from the standard benefit plans upon which these Coverage Policies are based. For example, a customer’s benefit plan document may contain a specific exclusion related to a topic addressed in a Coverage Policy. In the event of a conflict, a customer’s benefit plan document always supersedes the information in the Coverage Policies. In the absence of a controlling federal or state coverage mandate, benefits are ultimately determined by the terms of the applicable benefit plan document. Coverage determinations in each specific instance require consideration of 1) the terms of the applicable benefit plan document in effect on the date of service; 2) any applicable laws/regulations; 3) any relevant collateral source materials including Coverage Policies and; 4) the specific facts of the particular situation. Each coverage request should be reviewed on its own merits. Medical directors are expected to exercise clinical judgment where appropriate and have discretion in making individual coverage determinations. Where coverage for care or services does not depend on specific circumstances, reimbursement will only be provided if a requested service(s) is submitted in accordance with the relevant criteria outlined in the applicable Coverage Policy, including covered diagnosis and/or procedure code(s). Reimbursement is not allowed for services when billed for conditions or diagnoses that are not covered under this Coverage Policy (see “Coding Information” below). When billing, providers must use the most appropriate codes as of the effective date of the submission. Claims submitted

*for services that are not accompanied by covered code(s) under the applicable Coverage Policy will be denied as not covered. Coverage Policies relate exclusively to the administration of health benefit plans. Coverage Policies are not recommendations for treatment and should never be used as treatment guidelines. In certain markets, delegated vendor guidelines may be used to support medical necessity and other coverage determinations.*

## Overview

This Coverage Policy addresses pharmacogenetic testing. Pharmacogenetics is the study of gene variations within an individual's deoxyribonucleic acid (DNA) and how these differences influence an individual's response to medications.

Please see the Related Coverage Resources section above for additional information regarding pharmacogenetic testing for oncologic, hematologic, transplant, and other conditions.

## Coverage Policy

**Coverage for genetic testing varies across plans. Refer to the customer's benefit plan document for coverage details.**

**Pharmacogenetic testing (e.g., genotyping, pathogenic/likely pathogenic variant analysis) is considered medically necessary when ALL of the following criteria are met:**

- The individual is a candidate for a targeted drug therapy associated with a specific gene biomarker or gene pathogenic/likely pathogenic variant.
- The results of the pharmacogenetic test will directly impact clinical decision-making.
- The testing method is considered to be scientifically valid to identify the specific gene biomarker or gene pathogenic/likely pathogenic variant.
- **EITHER** of the following:
  - Identification of the specific gene or biomarker for use with a specific drug target has been demonstrated to improve clinical outcomes for the individual's condition being addressed.
  - Identification of the gene biomarker is noted to be clinically necessary prior to initiating therapy with drug target as noted within the U.S. Food and Drug Administration (FDA)-approved prescribing label.

**Pharmacogenetic screening in the general population is considered not medically necessary.**

**Gene expression classifiers for pharmacologic response are not covered or reimbursable.**

## Coding Information

### Notes:

1. This list of codes may not be all-inclusive since the American Medical Association (AMA) and Centers for Medicare and Medicaid Services (CMS) code updates may occur more frequently than policy updates
2. Deleted codes and codes which are not effective at the time the service is rendered may not be eligible for reimbursement.

**Not Covered or Reimbursable:**

<b>CPT®* Codes</b>	<b>Description</b>
81230	CYP3A4 (cytochrome P450 family 3 subfamily A member 4) (eg, drug metabolism), gene analysis, common variant(s) (eg, *2, *22)
81231	CYP3A5 (cytochrome P450 family 3 subfamily A member 5) (eg, drug metabolism), gene analysis, common variants (eg, *2, *3, *4, *5, *6, *7)
81283	IFNL3 (interferon, lambda 3) (eg, drug response), gene analysis, rs12979860 variant
81355	VKORC1 (vitamin K epoxide reductase complex, subunit 1) (eg, warfarin metabolism), gene analysis, common variant(s) (eg, -1639G>A, c.173+1000C>T)
81418	Drug metabolism (eg, pharmacogenomics) genomic sequence analysis panel, must include testing of at least 6 genes, including CYP2C19, CYP2D6, and CYP2D6 duplication/deletion analysis
0029U	Drug metabolism (adverse drug reactions and drug response), targeted sequence analysis (ie, CYP1A2, CYP2C19, CYP2C9, CYP2D6, CYP3A4, CYP3A5, CYP4F2, SLC01B1, VKORC1 and rs12777823)
0030U	Drug metabolism (warfarin drug response), targeted sequence analysis (ie, CYP2C9, CYP4F2, VKORC1, rs12777823)
0031U	CYP1A2 (cytochrome P450 family 1, subfamily A, member 2)(eg, drug metabolism) gene analysis, common variants (ie, *1F, *1K, *6, *7)
0032U	COMT (catechol-O-methyltransferase) (eg, drug metabolism) gene analysis, c.472G>A (rs4680) variant
0033U	HTR2A (5-hydroxytryptamine receptor 2A), HTR2C (5-hydroxytryptamine receptor 2C) (eg, citalopram metabolism) gene analysis, common variants (ie, HTR2A rs7997012 [c.614-2211T>C], HTR2C rs3813929 [c.-759C>T] and rs1414334 [c.551-3008C>G]) (Code deleted 12/31/2025)
0173U	Psychiatry (ie, depression, anxiety), genomic analysis panel, includes variant analysis of 14 genes
0175U	Psychiatry (eg, depression, anxiety), genomic analysis panel, variant analysis of 15 genes
0345U	Psychiatry (eg, depression, anxiety, attention deficit hyperactivity disorder [ADHD]), genomic analysis panel, variant analysis of 15 genes, including deletion/duplication analysis of CYP2D6
0347U	Drug metabolism or processing (multiple conditions), whole blood or buccal specimen, DNA analysis, 16 gene report, with variant analysis and reported phenotypes
0348U	Drug metabolism or processing (multiple conditions), whole blood or buccal specimen, DNA analysis, 25 gene report, with variant analysis and reported phenotypes
0349U	Drug metabolism or processing (multiple conditions), whole blood or buccal specimen, DNA analysis, 27 gene report, with variant analysis, including reported phenotypes and impacted gene-drug interactions
0350U	Drug metabolism or processing (multiple conditions), whole blood or buccal specimen, DNA analysis, 27 gene report, with variant analysis and reported phenotypes
0392U	Drug metabolism (depression, anxiety, attention deficit hyperactivity disorder [ADHD]), gene-drug interactions, variant analysis of 16 genes, including

<b>CPT®*</b> <b>Codes</b>	<b>Description</b>
	deletion/duplication analysis of CYP2D6, reported as impact of gene-drug interaction for each drug
0411U	Psychiatry (eg, depression, anxiety, attention deficit hyperactivity disorder [ADHD]), genomic analysis panel, variant analysis of 15 genes, including deletion/duplication analysis of CYP2D6
0419U	Neuropsychiatry (eg, depression, anxiety), genomic sequence analysis panel, variant analysis of 13 genes, saliva or buccal swab, report of each gene phenotype
0423U	Psychiatry (eg, depression, anxiety), genomic analysis panel, including variant analysis of 26 genes, buccal swab, report including metabolizer status and risk of drug toxicity by condition
0434U	Drug metabolism (adverse drug reactions and drug response), genomic analysis panel, variant analysis of 25 genes with reported phenotypes
0437U	Psychiatry (anxiety disorders), mRNA, gene expression profiling by RNA sequencing of 15 biomarkers, whole blood, algorithm reported as predictive risk score
0438U	Drug metabolism (adverse drug reactions and drug response), buccal specimen, gene-drug interactions, variant analysis of 33 genes, including deletion/duplication analysis of CYP2D6, including reported phenotypes and impacted gene-drug interactions
0460U	Oncology, whole blood or buccal, DNA single-nucleotide polymorphism (SNP) genotyping by real-time PCR of 24 genes, with variant analysis and reported phenotypes
0461U	Oncology, pharmacogenomic analysis of single-nucleotide polymorphism (SNP) genotyping by real-time PCR of 24 genes, whole blood or buccal swab, with variant analysis, including impacted gene-drug interactions and reported phenotypes
0476U	Drug metabolism, psychiatry (eg, major depressive disorder, general anxiety disorder, attention deficit hyperactivity disorder [ADHD], schizophrenia), whole blood, buccal swab, and pharmacogenomic genotyping of 14 genes and CYP2D6 copy number variant analysis and reported phenotypes
0477U	Drug metabolism, psychiatry (eg, major depressive disorder, general anxiety disorder, attention deficit hyperactivity disorder [ADHD], schizophrenia), whole blood, buccal swab, and pharmacogenomic genotyping of 14 genes and CYP2D6 copy number variant analysis, including impacted gene-drug interactions and reported phenotypes
0516U	Drug metabolism, whole blood, pharmacogenomic genotyping of 40 genes and CYP2D6 copy number variant analysis, reported as metabolizer status

<b>HCPCS</b> <b>Codes</b>	<b>Description</b>
G9143	Warfarin responsiveness testing by genetic technique using any method, any number of specimen(s)

**\*Current Procedural Terminology (CPT®) ©2025 American Medical Association: Chicago, IL.**

## **General Background**

Pharmacogenetics, or pharmacogenomics, is the study of gene variations within an individual's deoxyribonucleic acid (DNA) and how these differences influence an individual's response to medications. An individual's unique genetic makeup helps determine how he or she responds to a drug, and whether or not side effects or adverse reactions may be experienced. Variations in genes may also cause an individual to metabolize a drug more quickly, more slowly or at the same rate as anticipated, based on dosage.

Pharmacogenetics encompasses variations in genes that encode drug transporters, drug-metabolizing enzymes and drug targets, as well as specific genes related to the action of drugs. A slight variation in DNA can result in a subtle change in a protein which translates into major differences in how the protein functions. A pharmacogenetic test is meant to guide treatment strategies, patient evaluations and decisions based on its ability to predict response to treatment in particular clinical contexts.

A particular variant is not always phenotype-specific, in that it may have a different impact depending on the drug in question. Racial and ethnic differences in the frequency and nature of genetic variants are also possible and should be recognized in translating outcomes from one population to another (Fohner, et al., 2013). The relation of a gene or gene biomarker and a drug target must be validated for each therapeutic indication in different racial and ethnic groups, as well as in different treatment and disease contexts (Crews, et al., 2012).

Although genetics has an impact on genes related to inter-individual differences in drug response, it is only one of the many variables affecting these genes. Other factors include the characteristics of the condition for which the drug is prescribed, co-administration of other drugs, and non-genetic factors, including the individual's diet, weight, and smoking habits. Identification of gene variations may be clinically useful in a small number of drugs; however, it may be insufficient in others to explain complex differences in metabolism, efficacy and toxicity. The presence of polymorphisms alone may be a poor predictor of phenotype because of variability.

Clinical laboratories may develop and validate tests in-house and market them as a laboratory service; however, laboratories offering such tests as a clinical service must meet the general regulatory standards of the Clinical Laboratory Improvement Act (CLIA) and must be licensed by CLIA for high-complexity testing. High complexity techniques used for pharmacogenetic testing include immunohistochemistry (IHC), fluorescent in situ hybridization (FISH), polymerase chain reaction (PCR) and microarray assays. According to the U.S. Food and Drug Administration (FDA) (2007), diagnostic tests that assay the presence of a particular pattern (e.g., single nucleotide polymorphism [SNP] set, haplotype pattern) should ideally be validated in a prospective clinical trial.

An increasing number of multigene genotyping panels with the goals of detecting inter-individual differences in drug metabolism and response to a variety of drug targets are commercially available. The number of gene biomarkers and gene mutations and associated drug targets which are tested for vary widely between tests; some tests evaluate for a few biomarkers and associated drug targets, while others may include hundreds of biomarkers within the test. Some multigene assays assess for the presence or absence of multiple biomarkers and provide lists of potential therapeutic agents, clinical trials, and review of published literature associated with the biomarkers that are identified in the patient sample.

### **Clinical Utility**

The clinical use of a genetic test should be based on analytical validity (i.e., analytical sensitivity and specificity), and clinical validity (i.e., clinical sensitivity and specificity), and both positive and negative predictive value. Before a genetic test can be generally accepted in clinical practice, data

must be collected to demonstrate the benefits and risks from both positive and negative results (i.e., the test must have clinical utility).

The clinical usefulness or utility of pharmacogenetic testing is the extent to which results of testing will impact clinical decision-making and improve health outcomes. Pharmacogenetic test results are meant to guide patient evaluation and treatment strategy and decisions based on the ability to predict response to treatment in particular clinical contexts, and to allow the clinician to predict an individual's response to specific pharmacotherapy, assist in making treatment choices, individualize drug dosages in order to maintain a consistent drug level in the body and avoid adverse reactions from overdose or suboptimal effects from under medication (Cicali, et al., 2025). The integration of genomic data in individual treatment requires evidence of consistency and size of measured effects, medication compliance, and phenoconversion. The effects of ethnicity must be evaluated, especially in the context of global drug development and extrapolation of clinical trial genomic data from one population to another (Ehmann, et al., 2014).

When applied in a clinical setting, the information from these tests can potentially identify individual variability in drug response, including both effectiveness and toxicity. The individual for whom testing is proposed should be a candidate for a targeted drug therapy associated with a specific gene biomarker or gene mutation, and the results of testing must directly impact clinical decision making. The identification of the specific gene or biomarker for use with a specific drug target must also be demonstrated by published, peer-reviewed clinical trial data to improve clinical outcomes for an individual receiving that specific treatment and be considered scientifically valid to identify the biomarker.

### **Gene Expression Classifiers**

The genetic basis for disease is determined by the inheritance of genes containing specific sequences of DNA. The phenotypic expression of these genes, through the synthesis of specific proteins, involves interaction with environmental signals that trigger activation of particular genes. Ribonucleic acid (RNA) is transcribed from a DNA template, and messenger RNA (mRNA) is then translated into protein. Transcription and translation underlie gene expression. Three to five percent of genes are active in a particular cell, even though all cells have the same information contained in their DNA. Most of the genome is selectively repressed, a property that is governed by the regulation of gene expression, mostly at the level of transcription (i.e., the production of messenger RNA from DNA). In response to a cellular perturbation, changes in gene expression take place that result in the expression of hundreds of gene products and the suppression of others. This molecular heterogeneity can affect when and how a disease presents clinically in an individual with a genetic predisposition to a condition, and how individuals with a given disease will respond to specific treatments. Analyses of gene expression can be clinically useful for disease classification, diagnosis, prognosis, and tailoring treatment to underlying genetic determinants of pharmacologic response (e.g., breast cancer classification assays performed on tumor biopsy specimens) (Steiling and Christenson, 2025). However, there is a lack of evidence to support the use of gene expression classifiers and profiling (i.e., via whole blood specimen) for pharmacologic response.

Please see the Related Coverage Resources section of this policy for additional coverage policies and guidelines which address gene expression classifiers/gene expression profiling for transplant rejection, hematologic/oncologic indications, and other conditions.

### **U.S. Food and Drug Administration (FDA)**

The FDA considers the use of genomic information in drug labels either to require a genetic test for prescribing a drug, to recommend the use of a genetic test prior to drug therapy, or simply to provide information about the current knowledge of genomics that is relevant to drug therapy without the requirement or recommendation of a specific action. While the clinical utility of

genotyping prior to treatment is not proven for all medications for which genomic information is included (Slavin, et al., 2015), clinical utility is established when identification of a specific gene biomarker is noted to be clinically necessary prior to initiating therapy with a specific drug target, as noted within the FDA-approved prescribing label. The drug label will indicate that results from the specific genetic test listed may be required for safe and/or effective use of the medication, and recommend specific actions based on the genotype results (e.g., drug may be contraindicated; dosage adjustments may be needed).

An FDA Safety Communication issued in 2018 warned against the use of many genetic tests with unapproved claims to predict patient response to specific medications. The communication's intent was to alert patients and healthcare providers that for many genetic tests, claims to predict a patient's response to specific medications have not been reviewed by the FDA, and may not have the scientific or clinical evidence to support this use for most medications. Changing drug treatment based on the results from such a genetic test could lead to inappropriate treatment decisions and potentially serious health consequences for the patient. The FDA specifically noted the relationship between DNA variations and the effectiveness of antidepressant medication has never been established. According to the FDA, there are a limited number of cases for which at least some evidence does exist to support a correlation between a genetic variant and drug levels within the body, and this is described in the labeling of FDA-cleared or -approved genetic tests and FDA-approved medications.

### **Literature Review**

Increasingly, published peer-reviewed scientific evidence regarding the clinical utility of pharmacogenetic testing informs the ability of such testing to benefit health outcomes. Prospective clinical trials of standard management procedures compared with genetic test-directed management offers the highest level of evidence. Evidence may also be derived using banked samples from already-completed clinical trials, or by constructing an indirect chain of evidence linking test results to clinical outcomes. To date, much of the existing research in the area of pharmacogenetic testing has been limited by study design, including uncontrolled and poorly defined case and control groups, presence of confounding variables, high risk of bias, and the use of retrospective and non-blinded study protocols.

Although genome-wide association studies report inter-individual variability, high-quality randomized controlled trial data demonstrating improved clinical outcomes are lacking. Many early phase clinical trials are exploratory, with no formal genomic hypothesis, and have small sample sizes that make it difficult to identify important gene variants influencing pharmacokinetics and pharmacodynamics (Lesko and Schmidt, 2014). However, clinical utility has been established for pharmacogenetic testing for a number of gene biomarkers and their specific drug targets.

Zhang et al. (2025) conducted a systematic review and meta-analysis of 13 randomized controlled trials comparing pharmacogenomic (PGx)-guided antidepressant treatment with treatment-as-usual (TAU) in individuals with major depressive disorder (MDD). Trials involved participants age 14–80 years across Asia, Europe, North America, and Australia, using commercial and laboratory-developed pharmacogenomic tests. Included were randomized controlled trials of individuals with MDD based on Diagnostic and Statistical Manual of Mental Disorders (DSM) or International Classification of Diseases (ICD) criteria, and without significant suicide risk, severe cooccurring psychiatric disorders, cognitive impairment, or unstable or serious medical conditions; comparing PGx-guided treatment versus TAU without PGx guidance; and reporting severity of depressive symptoms on validated clinical scales. Outcome measures included response and remission at eight weeks and 12 weeks. Follow-up durations ranged from four to 24 weeks. Attrition or subjects lost to follow-up were not reported. Compared to TAU, PGx-guided treatment was associated with improved response at eight weeks (RR 1.23; 95% CI 1.05 to 1.43) and 12 weeks (RR 1.29; 95% CI 1.17 to 1.43), and improved remission at eight weeks (RR 1.37; 95% CI 1.19

to 1.57); however remission at 12 weeks did not reach statistical significance (RR 1.56; 95% CI 0.93 to 2.61). Subgroup analyses suggested larger effects in Asian populations and in difficult-to-treat major depressive disorder at eight weeks. Adverse events were not analyzed due to inconsistent measurements across studies. Limitations of the meta-analysis included heterogeneity in MDD not specified groups; variable blinding; non-standardized TAU; unknown impact of concurrent nonpharmacologic interventions; and use of modified intention-to-treat and per-protocol analyses. The authors recommended further studies to validate ethnic differences, examine long-term remission, and evaluate panel-size cost-effectiveness.

Yang et al. (2021) conducted a systematic review and meta-analysis of five randomized controlled trials (n=684) comparing genotype-guided versus conventional tacrolimus dosing in kidney transplantation. The intervention compared initial dosing guided by CYP3A5 genotype with conventional dosing strategies. Outcome measures including tacrolimus concentrations within the therapeutic range, time to achieve the therapeutic range, delayed graft function, acute rejection, graft survival censored for death, and adverse events. Results indicated that genotype-guided dosing increased the proportion of individuals achieving therapeutic tacrolimus concentrations at steady state (risk ratio [RR] 1.40, 95% confidence interval [CI] 1.14-1.72; p=0.001), and generally shortened the time to therapeutic range. However there were no statistically significant differences between the groups for delayed graft function, acute rejection, graft survival censored for death, or adverse events. Limitations noted by the authors included less rigid inclusion criteria attributable to the limited number and breadth of trials, and variability in ethnic origin with differing CYP3A5 polymorphism frequencies. The authors concluded that pharmacogenetic-guided initial tacrolimus dosing based solely on CYP3A5 genotype did not demonstrate clinical endpoint benefits and recommended further studies in Chinese and African American populations.

Tse et al. (2018) conducted a systematic review and meta-analysis of 16 randomized controlled trials and two conference abstracts (n=5230) comparing genotype-guided warfarin dosing with conventional dosing strategies. The review included 2626 individuals in the genotype-guided arm and 2604 in the conventional arm, with a mean follow-up of 64 days. The interventions contrasted genotype-informed dosing algorithms (incorporating CYP2C9, VKORC1, and CYP4F2 polymorphisms) against fixed-dose or clinical information-guided regimens. The predefined endpoints were time to first therapeutic international normalized ratio (INR), time to first stable INR, time in therapeutic range (TTR), excessive anticoagulation (INR  $\geq$ 4), bleeding, thromboembolism, and mortality. Results demonstrated statistically significant reductions in time to first therapeutic INR (mean difference 2.6 days, standard error 0.3 days; p<0.0001) and time to first stable INR (mean difference 5.9 days, standard error 2.0 days; p<0.01), as well as increased TTR (mean difference 3.1%, standard error 1.2%; p<0.01) for genotype-guided warfarin dosing compared with conventional dosing strategies. Genotype-guided dosing was associated with lower risks of excessive anticoagulation (risk ratio [RR] 0.87; 95% confidence interval [CI] 0.78, 0.98; p<0.05) and bleeding (RR 0.82; 95% CI 0.69, 0.98; p<0.05), while no significant differences were observed for thromboembolism or mortality. When comparing the genotype-guided groups and the clinical information-guided groups specifically, there was no apparent improvement in TTR, excessive anticoagulation, risk of thromboembolism or mortality. The meta-analysis was limited by heterogeneity in some outcomes, limited follow-up duration, inclusion of conference abstracts, and variable genotype algorithms.

Zeier et al. (2018) reviewed the evidence for several combinatorial pharmacogenetic test decision support tools whose potential utility to improve antidepressant treatment response or side effect burden has been evaluated in clinical settings. The authors noted available literature suffers from publication bias, because some products garner more investment than do others, and questions about scientific integrity are inherent in studies conducted by or reports authored by personnel with significant financial interests in the outcome. Although some of the preliminary published

data sound promising, particularly with regard to the CYP450 gene variants and side effect burden, the authors concluded that there is insufficient evidence to support widespread use.

Wang et al. (2014) published results of a study evaluating the evidence that supports pharmacogenomic biomarker testing in drug labels and how frequently testing is recommended. Using guidelines published by the Evaluation of Genomic Applications in Practice and Prevention Working Group and FDA databases, the authors reviewed drug labels that described the use of a biomarker for reference to clinical validity and clinical utility. Of 119 notations in drug labels, 36.1% provided evidence of clinical validity while 15.1% provided evidence of clinical utility. Sixty-one labels (51.3%) made recommendations regarding clinical management based on the results of a biomarker test. Of these, 30.3% provided clinical utility data. A full description of supporting studies was included in 13 labels (10.9%). The authors noted that it may be premature to include biomarker recommendations in drug labels when data regarding patient outcomes are not available.

Pharmacogenetic testing is not currently recommended for general population screening. Clinical trials regarding the use of pharmacogenetic testing for screening in the general population are lacking in the published, peer-reviewed scientific literature and the role of such testing has not been established.

### **Professional Societies/Organizations**

The 2022 Department of Veterans Affairs/Department of Defense clinical practice guideline for the management of major depression disorder found insufficient evidence to recommend for or against pharmacogenetic testing to help guide the selection or dosing of antidepressants.

In a 2020 policy statement, the American Academy of Child and Adolescent Psychiatry (AACAP) recommended that clinicians “avoid using pharmacogenetic testing to select psychotropic medications in children and adolescents”. The AACAP further recommended additional high quality prospective studies to assess the clinical significance of pharmacodynamic and combinatorial pharmacogenomic testing in this population.

## **Health Equity Considerations**

Health equity is the highest level of health for all people; health inequity is the avoidable difference in health status or distribution of health resources due to the social conditions in which people are born, grow, live, work, and age.

Social determinants of health are the conditions in the environment that affect a wide range of health, functioning, and quality of life outcomes and risks. Examples include safe housing, transportation, and neighborhoods; racism, discrimination and violence; education, job opportunities and income; access to nutritious foods and physical activity opportunities; access to clean air and water; and language and literacy skills.

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## Revision Details

Type of Revision	Summary of Changes	Date
Annual Review	<ul style="list-style-type: none"><li>No clinical policy statement changes.</li></ul>	5/16/2026

Type of Revision	Summary of Changes	Date
Focused Review	<ul style="list-style-type: none"> <li>No clinical policy statement changes.</li> </ul>	10/15/2025
Annual Review	<ul style="list-style-type: none"> <li>No clinical policy statement changes.</li> </ul>	2/15/2025
Focused Review	<ul style="list-style-type: none"> <li>No clinical policy statement changes.</li> </ul>	11/1/2024
Annual Review	<ul style="list-style-type: none"> <li>Revised policy statement for biomarker genotyping/mutation analysis.</li> </ul>	2/15/2024

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